



# CEREBRAL PHYSIOLOGY

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[NeuroAnesthesia Quiz # 13](#)

Quiz Team

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# CONTENT OUTLINE



Please click on any of the following links to proceed to that question/topic.

Question 1: [Volatile anesthetics and CBF](#)

Question 2: [Cerebral autoregulation](#)

Question 3: [Physiological mechanisms which affect CBF](#)

Question 4: [Anesthetic agents affecting CBF, CMRO<sub>2</sub>, and ICP](#)

Question 5: [Ischemic injury](#)

# QUESTION 1

70 year old patient presents with a supratentorial brain tumor and symptoms of raised intracranial pressure. Volatile anesthetics are part of your anesthetic plan. Which of the following is *incorrect* with respect to their use?

Please click on any of the following links to proceed to that question/topic.

A. [They blunt the cerebrovascular response to CO<sub>2</sub>.](#)

B. [They render CBF pressure passive](#)

C. [At 1 MAC, coupling is maintained](#)

D. [Decreases CMRO<sub>2</sub> while increasing cerebral blood flow](#)

# Great Job!! Correct.

## EXPLANATION

Cerebrovascular reactivity to CO<sub>2</sub> (vasodilation with hypercapnia and vasoconstriction with hypocapnia) has been considered to be regulated by the change in extracellular H<sup>+</sup> concentration mediated by nitric oxide, prostanoids, cyclic nucleotides, and intracellular calcium and potassium channel activity. At clinical levels of anesthesia, cerebrovascular responses to alterations in PaCO<sub>2</sub> are **preserved** with the use of inhalational agents, although the magnitude of response may vary according to the agent and anesthetic depth.

*Brian J.E.: Jr: Carbon Dioxide and the cerebral circulation. Anesthesiology 1998; 88: pp. 1365-1386*

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# Sorry! Incorrect.

## EXPLANATION

Autoregulation appears to be impaired with volatiles at **higher** concentrations. Sevoflurane maintains intact cerebral autoregulation up to 1.5 MAC. Desflurane induces a significant impairment in autoregulation, with completely abolished autoregulation at 1.5 MAC. Xenon appears to maintain autoregulation. When autoregulation is lost, CBF becomes pressure passive with sudden blood pressure changes leading to cerebral ischemia or brain edema in patients with intracranial pathology, i.e., space occupying lesions.

*Strebel S, Lam AM et al; Dynamic and static cerebral autoregulation during isoflurane, desflurane and propofol anesthesia; Anesthesiology 83: 66-76, 1995*

*Patel P, Drummond J, Lemkuil. Cerebral Physiology and the effects of Anesthetic Drugs, in Miller's Anesthesia, 8<sup>th</sup> ed. 2015, Elsevier, PA.*

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# Sorry! Incorrect.

## EXPLANATION

Cerebral blood flow and metabolism are said to be coupled (CBF adjustments parallel changes in CMR) and in general under physiologic conditions this coupling is generally preserved. At about 1 MAC, CMR suppression and the vasodilatory effect of volatile anesthetics is balanced or coupled. At >1.5 MAC, the vasodilatory effect of volatile anesthetics predominate increasing CBF despite a decrease in CMR suggesting an uncoupling of flow and metabolism. This reflects an alteration or increase in the CBF/CMR ratio. Nitrous oxide also impairs flow-metabolism coupling. Intravenous anesthetic agents such as propofol seem to preserve flow-metabolism coupling better than volatile agents.

*Cottrell and Young's neuroanesthesia; Fifth ed; ch 2; Cerebral and spinal cord blood flow; page 17-59 Kaisti K.K., Langsjo J.W., Aalto S., et al: Effects of sevoflurane, propofol, and adjunct nitrous oxide on regional cerebral blood flow, oxygen consumption, and blood volume in humans. Anesthesiology 2003; 99: pp. 603-613*

*Patel P, Drummond J, Lemkuil. Cerebral Physiology and the effects of Anesthetic Drugs, in Miller's Anesthesia, 8<sup>th</sup> ed. 2015, Elsevier, PA.*

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# Sorry! Incorrect.

## EXPLANATION

In general, all inhalational anesthetics are cerebral vasodilators. They increase cerebral blood flow and hence possess the capability of increasing ICP. Inhalational anesthetics, with the possible exception of nitrous oxide ( $N_2O$ ), usually depress metabolism ( $CMRO_2$ ). The net effect of inhalational anesthetics is a balance between a reduction in CBF due to CMR suppression and augmentation of CBF due to direct cerebral vasodilation

*Patel P, Drummond J, Lemkuil. Cerebral Physiology and the effects of Anesthetic Drugs, in Miller's Anesthesia, 8<sup>th</sup> ed. 2015, Elsevier, PA.*

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## QUESTION 2

All of the following situations impair cerebral autoregulation Except:

Please click on any of the following links to proceed to that question/topic.

A. [Higher doses of volatile anesthetic agents](#)

B. [Hypercapnia](#)

C. [Traumatic brain injury](#)

D. [Propofol](#)



# Sorry! Incorrect.

## EXPLANATION

Autoregulation is impaired more easily when vasodilatory anesthetics are used. The regulation of CBF is done by altering the resistance of cerebral blood vessels. Cerebral autoregulation is under the influence of myogenic, metabolic, and neurogenic factors. Beyond a dose of 1.5 MAC, cerebral autoregulation is impaired with volatile anesthetics

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*H Prabakr et al, Current concepts of optimal cerebral perfusion pressure in traumatic brain injury. Journal of Anaesthesiology Clinical Pharmacology | July-September 2014 | Vol 30 | Issue 3*

# Sorry! Incorrect.

## EXPLANATION

Increases in PaCO<sub>2</sub> can lead to a change in cerebrovascular resistance (CVR) causing cerebral vasodilation and impacting cerebrovascular autoregulation. Previously conducted studies revealed that hypercapnia impairs dynamic cerebrovascular autoregulation (dCA), as measured using the thigh cuff deflation method as well as by transfer-function analysis of flow velocity changes with spontaneously fluctuating blood pressure.

*Aaslid R, Lindegaard KF, Sorteberg W, Nornes H. Cerebral autoregulation dynamics in humans. Stroke 1989; 20:45–52.  
Incorrect*

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# Sorry! Incorrect.

## EXPLANATION

The homeostatic mechanisms are often lost after head trauma (CVR is usually increased), and the brain becomes susceptible to blood pressure changes. Cerebral ischemia can result from systemic hypotension.

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*Sakabe R, Matsumoto M, Effects of Anesthetic Agents and other drugs on Cerebral Blood flow, metabolism and intracranial pressure, in Cottrell and Young's Neuroanesthesia, 2010, Saunders, PA.*

# Great Job!! Correct.

## EXPLANATION

In neurologically normal patients, autoregulation is preserved with Intravenous anesthetics like Propofol. This finding may be due, in part, to the vasoconstrictive effect of IV anesthetics.

*Sakabe R, Matsumoto M, Effects of Anesthetic Agents and other drugs on Cerebral Blood flow, metabolism and intracranial pressure, in Cottrell and Young's Neuroanesthesia, 2010, Saunders, PA.*

*Strebel S, Lam A, Matta B, Dynamic and static cerebral autoregulation during isoflurane, desflurane, and Propofol anesthesia, Anesthesiology, 1995;83:66-76*

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## QUESTION 3

Cerebral blood flow changes the most with which one of the following maneuvers?

Please click on any of the following links to proceed to that question/topic.

A. [Change of PaCO<sub>2</sub> from 40-60 mmHg](#)

B. [Change of PaO<sub>2</sub> from 120-70 mmHg](#)

C. [Change in the MAC of sevoflurane from 0.5-1.5](#)

D. [Change in propofol infusion dose from 50mcg/kg/min to 100mcg/kg/min](#)

# Great Job!! Correct.

## EXPLANATION

CBF is profoundly influenced by PaCO<sub>2</sub>. Within the physiologic range of 20 to 60 mm Hg, CBF changes by 3% to 4% per 1mm Hg change in CO<sub>2</sub> tension, with an accompanied commensurate change in CBV(cerebral blood volume). CO<sub>2</sub> reactivity is brisk and occurs within seconds of changing the arterial PaCO<sub>2</sub>

A prolonged change in systemic CO<sub>2</sub> tension is accompanied by active transport of bicarbonate in or out of CSF to restore a normal acid-base balance (at 6 hours). Thus, the effects of hyperventilation on CBF are not sustained.

*Nortje, J Cerebral blood flow and its control. In Gupta, Gelb. Essentials of Neuroanesthesia and Neurointensive Care. 2008. Elsevier, PA*

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# Sorry! Incorrect.

## EXPLANATION

Hypoxemia causes vasodilation of the cerebral vessels and an increase in CBF, but this does not occur until  $\text{PaO}_2$  is less than 60 mmHg. The mediators of cerebral vasodilation include neurogenic effects initiated by peripheral and neuraxial chemoreceptors and local humeral influences.

*Patel P, Drummond J, Lemkuil. Cerebral Physiology and the effects of Anesthetic Drugs, in Miller's Anesthesia, 8<sup>th</sup> ed. 2015, Elsevier, PA.*

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# Sorry! Incorrect.

## EXPLANATION

Anesthetic techniques may also affect cerebral physiology. The cerebral effects of inhaled anesthetics are twofold: they are intrinsic cerebral vasodilators, but their vasodilatory actions are partly opposed by flow-metabolism coupling-mediated vasoconstriction secondary to a reduction in CMR. The overall effect is unchanged CBF during low-dose inhaled anesthesia (0.5-1.5 MAC) but increased flow during high doses (> 1.5 MAC).

*Matta BF, Mayberg TS, and Lam AM: Direct cerebrovasodilatory effects of halothane, isoflurane, and desflurane during propofol-induced isoelectric electroencephalogram in humans. Anesthesiology 1995; 83: pp. 980-985*

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# Sorry! Incorrect.

## EXPLANATION

Intravenous anesthetic agents like propofol, are indirect cerebral vasoconstrictors that reduce cerebral metabolism coupled with a corresponding reduction in CBF. Both autoregulation and CO<sub>2</sub> reactivity are preserved.

*Neuroanesthesia: Preoperative Evaluation; Deepak Sharma ; Arthur M. Lam; Chapter 21, Youmans Neurological Surgery Chapter 21, 395-407.*

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## QUESTION 4

The following agents decrease CBF/CMRO<sub>2</sub> and ICP *Except*:

Please click on any of the following links to proceed to that question/topic.

A. [Dexmedetomidine](#)

B. [Propofol](#)

C. [Etomidate](#)

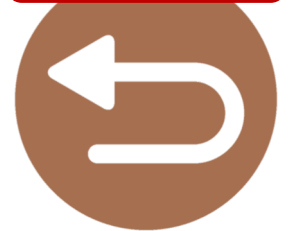
D. [Nitrous oxide](#)

# Sorry! Incorrect.

## EXPLANATION

Dexmedetomidine has been reported to decrease both rCBF and global CBF. Dexmedetomidine reduced the CMR equivalent (CMRe; this value is calculated by multiplying  $V_{mca}$  by the difference between arterial and cerebral jugular venous oxygen contents) in healthy volunteers in a dose-dependent manner. Dexmedetomidine in a small dose decreased both MABP and ICP, and in higher doses it did not influence ICP, despite a significant increase in MABP in halothane-anesthetized rabbits

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*Wang et al; Effects of dexmedetomidine on cerebral blood flow in critically ill patients with or without traumatic brain injury: a prospective controlled trial. Brain Inj. 2013;27(13-14):1617-22. doi: 10.3109/02699052.2013.831130. Epub 2013 Oct 8.*

*Zornow M.H., Scheller M.S., Sheehan P.B., et al: Intracranial pressure effects of Dexmedetomidine in rabbits. Anesth Analg 1992; 75: pp. 232-237*

# Sorry! Incorrect.

## EXPLANATION

Propofol produces dose-related decreases in both CBF and  $CMRO_2$ , with minimum  $CMRO_2$  values being 40% to 60% of control values. In patients with cerebral tumors with midline shift less than 10 mm, ICP was reported to be lower and CPP higher in patients anesthetized with propofol than in those anesthetized with isoflurane or sevoflurane. However careful attention should be paid to the mean arterial pressure which can also decrease with Propofol.

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*Petersen et al; Intracranial pressure and cerebral hemodynamic in patients with cerebral tumors: A randomized prospective study of patients subjected to craniotomy in propofol-fentanyl, isoflurane-fentanyl, or sevoflurane-fentanyl anesthesia. Anesthesiology 2003; 98: pp. 329-336*

# Sorry! Incorrect.

## EXPLANATION

Etomidate, decreases  $CMRO_2$  progressively until an isoelectric EEG appears. A maximal decrease in CBF was achieved before the maximal decrease in  $CMRO_2$  suggesting that etomidate causes vasoconstriction through a different mechanism (possibly by direct action) as compared to barbiturates which cause a decrease in  $CMRO_2$  consequentially causing cerebral vasoconstriction. Parallel decreases in ICP and CBF were observed with etomidate.

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*Milde L.N., Milde J.H., and Michenfelder J.D.: Cerebral functional, metabolic, and hemodynamic effects of etomidate in dogs. Anesthesiology 1985; 63: pp. 371-377*

# Great Job!! Correct.

## EXPLANATION

It is generally agreed that N<sub>2</sub>O increases CBF, CMRO<sub>2</sub>, and ICP, although the magnitude varies substantially. N<sub>2</sub>O, when added to volatile anesthetics, raises both CBF and CMR (increase maybe due in part to the sympathoadrenal-stimulating effect of N<sub>2</sub>O). When administered with IV anesthetics like Propofol, it's cerebral-vasodilating effect is attenuated.

*Kaisti K.K. et al: Effects of sevoflurane, propofol, and adjunct nitrous oxide on regional cerebral blood flow, oxygen consumption, and blood volume in humans. Anesthesiology 2003; 99: pp. 603-613*

*Patel P, Drummond J, Lemkuil. Cerebral Physiology and the effects of Anesthetic Drugs, in Miller's Anesthesia, 8<sup>th</sup> ed. 2015, Elsevier, PA.*

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## QUESTION 5

A 67 year old patient comes to the emergency room due to sudden onset of weakness of the left side of his body. He is being evaluated for prompt reperfusion. Which of the following would possibly be deleterious in his current situation?

Please click on any of the following links to proceed to that question/topic.

A. [Systolic blood pressure between 140 and 180mmHg.](#)

B. [PaCO<sub>2</sub> 35-45mmHg](#)

C. [Blood glucose of 160mg/dl.](#)

D. [Anticonvulsant medications](#)

# Sorry! Incorrect.

## EXPLANATION

Maintenance of CPP(cerebral perfusion pressure) for a patient who is at risk for cerebral ischemic injury is essential. The SNACC consensus statement suggests should be maintained  $>140$ mm Hg (fluids and vasopressors) and  $<180$ mm Hg (with or without IV tPA). Hypotension could be deleterious and causes of hypotension should be investigated (volume depletion, myocardial infarction, cardiac arrhythmia, blood loss, retroperitoneal hemorrhage, and aortic dissection) and treated if possible.

*Society for Neuroscience in Anesthesiology and Critical Care Expert Consensus Statement: Anesthetic Management of Endovascular Treatment for Acute Ischemic Stroke ; J Neurosurg Anesthesiol Volume 26, Number 2, April 2014.*

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## EXPLANATION

Arterial PCO<sub>2</sub> should be maintained in the normal range. Current data in stroke patients suggest that hypocapnia is associated with poor prognosis in stroke. There are no data to support the use of hypocapnia as a therapeutic measure to redistribute cerebral blood flow during focal cerebral ischemia. Hypocapnia may be used temporarily to treat increases in intracranial pressure due to stroke or hemorrhagic conversion thereof. The regional cerebral vasodilatory response to hypercapnia may be impaired in patients with symptomatic cerebral ischemia. Respiratory depression-induced hypercapnia should be avoided during procedural sedation.

*Ruta TS, Drummond JC, Cole DJ. The effect of acute hypocapnia on local cerebral blood flow during middle cerebral artery occlusion in isoflurane anesthetized rats. Anesthesiology. 1993;78:134–140.*

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# Great Job!! Correct.

## EXPLANATION

Although glucose is the main source of energy for neurons in the brain, hyperglycemia is thought to exacerbate ischemic injury due to cellular acidosis. The SNACC consensus statement recommends that insulin treatment of hyperglycemia should be initiated for glucose values of >140 mg/dl. They recommend that glucose concentration is maintained in the range of 70 to 140 mg/dL with treatment for hypoglycemia being initiated for glucose values of <50 mg/dL.

*Society for Neuroscience in Anesthesiology and Critical Care Expert Consensus Statement: Anesthetic Management of Endovascular Treatment for Acute Ischemic Stroke ; J Neurosurg Anesthesiol Volume 26, Number 2, April 2014.*

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## EXPLANATION

Seizures are a known complication of ischemic stroke. Seizures are associated with a increased CBF and CBV and consequent increased ICP which leads to rapid neuronal damage if untreated. Seizures should be actively prevented and treated.

*Patel P, Drummond J, Lemkuil. Cerebral Physiology and the effects of Anesthetic Drugs, in Miller's Anesthesia, 8<sup>th</sup> ed. 2015, Elsevier, PA.*

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