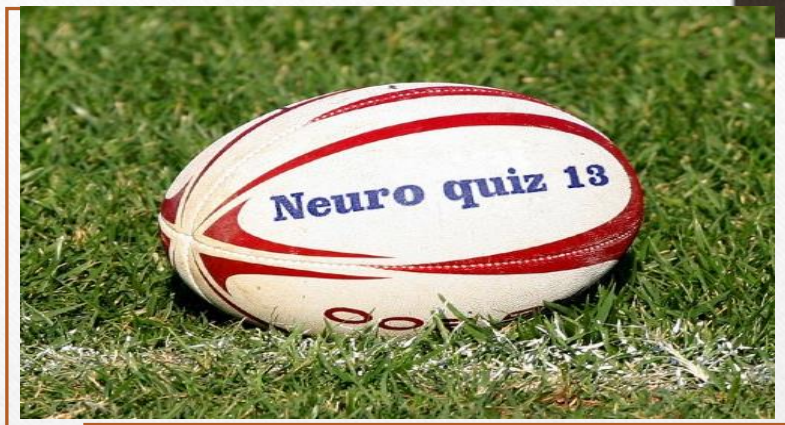

SNACC

SOCIETY FOR NEUROSCIENCE IN ANESTHESIOLOGY AND CRITICAL CARE

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This quiz is being published on behalf of the SNACC Education Commitee

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

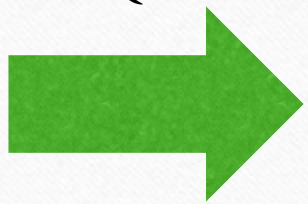
A. They blunt CO2 response.

B. Render CBF pressure passive

C. Uncouple flow and metabolism

D. Decreases CMRO2 while increasing cerebral blood flow

Go to Q 2



A. They blunt CO2 response

Correct answer

Cerebrovascular reactivity to CO_2 (vasodilation with hypercapnia and vasoconstriction with hypocapnia) has been considered to be regulated by the change in extracellular H^+ 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 Pa co_2 are **preserved** by inhalational agents, although the magnitude of response may vary according to agent and anesthetic depth.

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

Back to Q 1

Go to Q 2

B. Render CBF pressure passive

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 a completely abolished autoregulation at 1.5 MAC. Xenon appears to maintain autoregulation. When autoregulation is lost sudden blood pressure changes can lead to cerebral ischemia or brain edema in patients with intracranial pathology like 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

Incorrect

Try again

C. Uncouple flow and metabolism

Cerebral blood flow and metabolism are said to be coupled, and in general under physiologic conditions this coupling is generally preserved. Volatile anesthetic agents in concentrations exceeding 1.5 MAC are associated with increasing CBF, suggesting an uncoupling of flow and metabolism. Nitrous oxide also impairs flow-metabolism coupling. Intravenous anesthetic agents such as propofol seem to preserve flow-metabolism coupling better than volatile agents.

Ref Cottrell and Young's neuroanethesia; 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

Incorrect

Try again

D. Decreases CMRO2 while increasing cerebral blood flow

In general, all inhalational anesthetics are cerebral vasodilators and 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(CMRO2). 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;

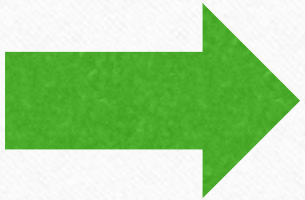
Incorrect

Try again

2. The following situations can disrupt cerebral autoregulation except

A. Large dose volatile agents
B. Hypercapnia
C. Traumatic brain injury
D. Nicardipine infusion
E. Hypocapnia

Go to Q 3



A. Large doses of volatile anesthetics

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.

Ref; ; 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

Incorrect

Try again

B. Hypercapnia

Increases in $PaCO_2$ can lead to a change in cerebrovascular resistance (CVR) 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.

Incorrect

Try again

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

C. Traumatic brain injury

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.

Incorrect

Try again

D. Nicardipine infusion

Nicardipine infusion- nicardipine could significantly impair autoregulation, probably because of its potent dilating effects on cerebral arterioles.

Ref; The Influence of Nicardipine-, Nitroglycerin-, and Prostaglandin E1-Induced Hypotension on Cerebral Pressure Autoregulation in Adult Patients During Propofol-Fentanyl Anesthesia. Hiroshi endoh et al; Anesth Analg 2002;94:169–73

Incorrect

Try again

Correct answer

E. Hypocapnia

Hypocapnia, on the contrary, increases CVR, improves vascular tone, and augments cerebrovascular autoregulation while decreasing CBF.

Back to Q 2

Go to Q 3

3. Cerebral blood flow changes maximum with which one of the following maneuvers?

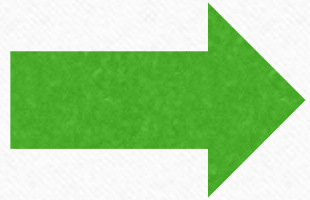
A. Change of $PaCO_2$ from 40-60

B. Change of PaO_2 from 120-70

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/minute

Go to Q 4



Correct answer

A. Change of PaCO2 from 40-60

CBF is profoundly influenced by $PaCo_2$. Within the physiologic range of 20 to 60 mm Hg, CBF changes by 3% to 4% per 1–mm Hg change in CO_2 tension, with an accompanied commensurate change in CBV(cerebral blood volume). CO_2 reactivity is brisk and occurs within seconds of changing the arterial $Pa\ Co_2$

A prolonged change in systemic CO_2 tension is accompanied by active transport of bicarbonate in or out of CSF to restore a normal acid-base balance. Thus, the effects of hyperventilation on CBF are not sustained beyond 24 hours

Back to Q 3

Go to Q 4

B. Change of PaO2 from 120-70

Hypoxemia causes vasodilation of the cerebral vessels and an increase in CBF, but this does not occur until Pa o_2 is less than 50 mm Hg.

Incorrect

Try again

C. Change in the MAC of sevoflurane from 0.5-1.5

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 flow during low-dose inhaled anesthesia but increased flow during high doses above 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

Incorrect

Try again

D. Change in propofol infusion dose from 50mcg/kg/min to 100mcg/kg/minute

Intravenous anesthetic agents like propofol, are indirect cerebral vasoconstrictors that reduce cerebral metabolism coupled with a corresponding reduction in CBF. Both autoregulation and CO_2 reactivity are preserved

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Neuroanesthesia: Preoperative Evaluation;
Deepak Sharma ; Arthur M. Lam; Chapter 21,
Youmans Neurological Surgery, Chapter 21, 395-407.e2

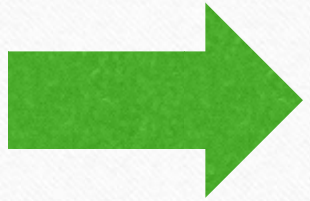
Incorrect

Try again

4. The following agents decrease CBF/CMRO2 and ICP except

A. Dexmedetomidine
B. Propofol
C. Etomidate
D. Nitrous oxide

Go to Q 5



A. Dexmedetomidine

Dexmedetomidine has been reported to decrease both rCBF and global CBF. Dexmedetomidine reduced CMR equivalent (CMRe; this value is calculated by multiplying Vmca 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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Ref 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

Incorrect

Try again

B. Propofol

Propofol produces dose-related decreases in both CBF and CMR o $_2$, with minimum CMR o $_2$ 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.

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

Incorrect

Try again

C. Etomidate

Etomidate, decreases $CMRO_2$ progressively until an isoelectric EEG appears. A maximal decrease in CBF was achieved before the maximal decrease in CMR o $_2$ suggesting that etomidate causes vasoconstriction through a different mechanism (possibly by direct action) as compared to barbiturates which cause decrease in CMRO2 consequentially causing cerebral vasoconstriction. Parallel decreases in ICP and CBF were observed with etomidate.

Ref; 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

Incorrect

Try again

D. Nitrous oxide

Correct answer

It is now generally agreed that N_2O increases CBF, $CMRO_2$, and ICP, although the magnitude varies substantially. N2O, when added to volatile anesthetics, raises both CBF and CMR.

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

Back to Q 4

Go to Q 5

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

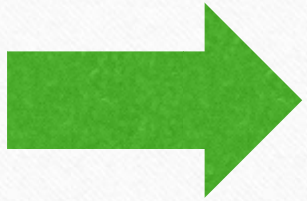
A. Systolic blood pressure between 140 and 180.

B. $PaCO_2$ 35-45

C. Blood glucose of 160mg/dl.

D. Anticonvulsant medications

Back to Q 1



Systolic blood pressure between 140 and 180

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 >140mm Hg (fluids and vasopressors) and <180mm 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.

Ref ; 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.

Incorrect

Try again

PaCO2 35-40

Arterial PCO 2 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.

Ref; 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.

Incorrect

Try again

Blood glucose of 160mg/dl.

Correct answer

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 recommend that insulin treatment of HG 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.

J Neurosurg Anesthesiol Volume 26, Number 2, April 2014 SNACC Consensus Statement

Back to Q 5

End of set

Back to Q 1

Anticonvulsant medications

Seizures can cause rapid neuronal damage and seizures should be actively prevented and treated.

Incorrect

Try again