

A 34-year-old man is treated in the intensive care unit after being involved in a motorcycle accident. He has multiple injuries, including severe traumatic brain injury. Head CT scan shows areas of contusion and swelling without subdural or epidural hematoma. On the second day of hospitalization, a ventriculostomy is placed for invasive intracranial pressure monitoring. On the third day, his pressure is high despite adequate sedation, elevation of the head of the bed, and removal of cerebrospinal fluid. Hyperventilation would decrease this patient's intracranial pressure by which of the following mechanisms?

- ☐ A. Cerebral vasoconstriction
- ☐ B. Decreased capillary leak
- ☐ C. Decreased sympathetic output
- ☐ D. Increased pO_2
- ☐ E. Increased venous outflow from the head

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- ✓ ☒ A. Cerebral vasoconstriction [84%]
☐ B. Decreased capillary leak [1%]
☐ C. Decreased sympathetic output [4%]
☐ D. Increased pO_2 [4%]
☐ E. Increased venous outflow from the head [7%]

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Explanation:

User Id: [REDACTED]

Interventions for lowering ICP	
Intervention	Mechanism
Head elevation	Increased venous outflow from the brain
Sedation	Decreased metabolic demand & control of hypertension
Intravenous mannitol	Extraction of free water from brain tissue → osmotic diuresis
Hyperventilation	CO_2 washout → cerebral vasoconstriction
Removal of CSF	Reduction of CSF volume/pressure

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Because the brain is an encased organ, relatively small changes in pressure can alter the **intracranial pressure** in important ways. The pressures in 3 distinct compartments of the brain - the brain parenchyma, the cerebrospinal fluid (CSF), and **cerebral blood flow** (CBF) - determine the overall intracranial pressure (ICP). Both brain parenchyma and CSF have relatively constant pressures except in certain circumstances, including space-occupying lesions and obstructions to CSF flow. CBF is influenced by systemic blood pressure and is held constant over a fairly wide range of pressures by cerebrovascular autoregulation. However, the partial pressures of O_2 and CO_2 also play an important role in regulating CBF. In conditions such as stroke or trauma, these systems are disrupted and interventions may be required to lower ICP.

CBF is an important target for therapy, and **$paCO_2$** is a potent regulator of CBF (much greater than paO_2). As levels of cerebral $paCO_2$ rise, so does blood flow. Lowering cerebral arterial $paCO_2$ through hyperventilation results in rapid **vasoconstriction** and a consequent decrease in ICP.

Other interventions to lower ICP do so by lowering systemic pressures and reducing metabolic demand (sedation), or increasing venous outflow (head elevation), reducing brain parenchyma water content/volume (mannitol), or reducing the volume of CSF (therapeutic lumbar punctures).

(Choice B) The CBF system is tightly regulated and mediated by cerebral vasodilation rather than capillary leak.

(Choice C) Hyperventilation does not significantly affect the sympathetic output of the brain, which is mediated predominantly by pressures in the compartments (eg, brain, CSF, blood).

(Choice D) Ventilatory rate has a much greater effect on arterial pCO_2 than on pO_2 , which can be modified by changing the concentration of inhaled oxygen or the end-expiratory pressure in a mechanically ventilated patient.

(Choice E) Increased venous outflow from the head is achieved by elevation of the head rather than hyperventilation.

Educational objective:

Short-term hyperventilation helps lower increased intracranial pressure by causing cerebral washout of CO_2 , leading to vasoconstriction and decreased cerebral blood flow.

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References:

1. [Every breath you take: hyperventilation and intracranial pressure.](#)