Neuroanesthesia Question of the Month: Which vasopressor best preserves cerebral blood flow?
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Cerebral blood flow (CBF) is preserved via a dynamic and complex interplay of autoregulatory systems. Vasopressors are commonly used in neuroanesthesia to optimize cerebral perfusion and match cerebral blood supply to demand. CBF is mostly regulated at the level of the pial arterioles and intracerebral resistance vessels.[1] Studies suggest the presence of neurovascular units formed from astrocytes, pericytes, neurons, microglia and endothelium.[2] These functional units, along with the BBB, transduce neuronal signals into vasomotor responses.[3] At these vascular beds $\alpha_1\alpha_2$ mediate vasoconstriction and $\beta_1\beta_2$ mediate vasodilation.[4] The distribution of these adrenergic receptors varies throughout the cerebral vasculature.[1] Vasopressor treatment influence on cerebral blood flow (CBF) and brain tissue oxygenation ($SctO_2$) is an ongoing area of investigation.

Phenylephrine, in those with preserved autoregulation, is associated with an increase in MAP and simultaneous reductions In CO and $SctO_2$ as measured by NIRS.[5] Phenylephrine maintains CBF when CO is constant. This relationship could be explained by reflex bradycardia and the dynamic cardiovascular interaction.[6] It has also been suggested that phenylephrine constricts cerebral resistance vessels indirectly through sympathetic nervous system activation subsequent to decreased CO.[5]

Ephedrine reliably was associated with increased MAP, CO and $SctO_2$. CBF was more consistently preserved with ephedrine as opposed to phenylephrine.[5] While both vasopressors augmented MAP, ephedrine restored CO and SctO2.[7]

The evidence around norepinephrine infusion and influence on CBF is controversial. Original research associated norepinephrine with mild reduction in CBF. These studies have been met with conflicting evidence showing neutral effects on brain tissue oxygenation by both NIRS $SctO_2$ and middle cerebral artery (MCA) flow velocity.[8,9]

Despite our ubiquitous use of vasopressors, more research is required to ascertain which vasopressors preserve CBF. There are many limitations to these studies. Surrogate markers (e.g. MAP/CPP, MCA flow velocity transcranial doppler, SctO2 via NIRs and PET) are used to help extrapolate CBF which uses many assumptions about the global blood flow to the microvasculature in the brain. In addition, there are multiple other control variables that have significant effects on CBF that are not uniform in these studies. Lastly, there are individual variabilities in the complex interplay of cardiac, respiratory and neuro hormonal effects that change autoregulation in health and in disease. In particular, patients with brain injury have been shown to have the most variation in CBF due to the shedding of glycocalyx and increased BBB permeability which influences drug delivery and effect.[1] Therefore, in patients with TBI, there is no evidence to support whether the choice in vasopressor can influence outcome by predictably preserving CBF. After discussion with the neuroanesthesiologists at our institution, care should be taken to augment overall cardiac output to maintain end organ perfusion which will be reflected in the CBF and global brain tissue oxygenation. The vasopressor used to accomplish this will be individual to the patient and their physiology.

References

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