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ARTICLE OF THE MONTH

A decrease in spatially resolved near-infrared spectroscopy-determined frontal lobe tissue oxygenation by phenylephrine reflects reduced skin blood flow.

Ogoh S, Sato K, Okazaki K, Miyamoto T, Secher F, Sørensen H, Rasmussen P, Secher NH.
Anesth Analg. 2014 Apr;118(4):823-9.
PMID: 24651237

In this installment of the SNACC Article of the Month, we explore a very interesting, and somewhat controversial, topic: maintenance of cerebral normotension with vasopressors and the possible effects of this on cerebral oxygenation. There are 2 basic concerns at play here: 1.) cerebral oximetry has become more widespread in clinical use as a non-invasive measure of brain tissue oxygenation, but there are concerns that this method may be "contaminated" by extradural tissue measurement, and 2.) vasopressors, while increasing cerebral perfusion pressure, may or may not be responsible for a deleterious cerebral vasoconstriction. The article by Ogoh et al. addresses both of these concerns, using transcranial Doppler measurement to parse out the utility of near-infrared technology in healthy volunteers in a resting state and with vasopressor administration. The expert commentary which you will find below, provided by Dr. Arthur Lam, succinctly sheds some light on the subject. Dr. Lam (at the Swedish Neuroscience Institute in Seattle) is a long-standing member and former president of SNACC, with whom we are all very familiar, and whose contributions to our field are too many to mention. We hope you will enjoy this Article of the Month and will make your voice heard on this clinically important topic by visiting us on LinkedIn.

-John F. Bebawy, MD

Commentary

Reviewer: Arthur Lam, MD

To maintain cerebral perfusion during systemic hypotension, it is common clinical practice to raise blood pressure using a vasopressor, and phenylephrine is probably the most frequently used drug. Concerns had been raised in the past about possible direct cerebral vasoconstrictive effect, thus negating the potential benefits from

increasing the systemic blood pressure. More recent studies using transcranial Doppler have suggested that this is not the case, yet studies on near-infrared brain oximetry (ScO2) have consistently demonstrated a decrease in ScO2 values with phenylephrine administration.

What is the physiologic explanation? This study provides a plausible explanation by measuring simultaneously cerebral blood flow (middle cerebral blood flow velocity or Vmca by transcranial Doppler as a surrogate), extracranial cerebral blood flow (internal carotid artery flow or ICA flow, external carotid flow or ECA flow, and vertebral artery flow or VA flow), and skin blood flow. The hypothesis is that since ScO2 reflects both intracranial components and extracranial components, a decrease in ECA flow corroborated by a decrease in skin blood flow would explain the fall in ScO2 with phenylephrine administration. The authors studied 7 healthy volunteers with the above variables including ScO2 monitored, with and without phenylephrine infusion. The authors observed no change in Vmca, ICA flow, or VA flow during phenylephrine infusion. However, there was a decrease in ECA flow velocity without a change in diameter, resulting in a decrease in flow that did not reach statistical significance ($p = 0.073$), although the rank correlation between baseline values and changes in ECA flow was significant. There was no difference in skin blood flow (attributed to a large variation in baseline values). Do these results prove that the decrease in ScO2 during phenylephrine is a result of extracranial vasoconstriction? I think the answer is a "quite possibly but not definitive". In addition to the limitations cited in the paper, it should be pointed out that Doppler measurements were not measured simultaneously but sequentially since the investigators only have access to one Doppler machine. It would be interesting to indirectly verify these results where the potential influence of "extracranial contamination" of ScO2 can be eliminated at baseline, i.e. during carotid endarterectomy after the external carotid artery has been clamped.