Welcome to the July 2019 installment of SNACC's Article of the Month. This month's selection addresses the effects of common vasopressors on cerebral physiology.

Dr. Riikka Takala offers her expert commentary this month. Dr. Takala became a certified neuroanesthesiologist in 2005 in her home country of Finland. She is the leader of the neuranesthesia and neurointensive care training program at Turku University Hospital. Turku was the European Capital of Culture in 2011 and is the official Christmas City of Finland!

Dr. Takala is one of four national examiners for neuroanesthesia and neurointensive care. Her main research interest concentrates on biomarkers in TBI, a subject she spoke about at the SNACC meeting in 2018.

As always, we encourage our readers to give us their feedback on the SNACC Twitter feed, or on Facebook.

Nina Schloemerkemper, MD; Adrian Pichurko, MD; and Oana Maties, MD

Commentary

Riikka Takala, MD, PhD

Vasopressors are commonly used in neuroanesthesia and neurocritical care in an attempt to optimize cerebral perfusion pressure to match metabolic demand. Despite their wide use, the effects of vasoactive medications on the cerebral physiology are perhaps less known. The article by Thorup et al is a comprehensive review of the anatomy, physiology and pharmacology pertinent to this topic.
The first section of the chosen article covers the anatomy of the pial arteries, arterioles and capillaries and how cerebrovascular tone is neuronally regulated. It also describes how a neurovascular unit is formed by astrocytes, pericytes, neurons, microglia and endothelium. Glycocalyx, which is important in maintaining the vascular wall integrity is also part of the neurovascular unit. The integrity of glycocalyx may be lost in traumatic brain injury (TBI) which could change drug effects.

Cerebral blood flow (CBF) is mostly regulated at the level of pial arterioles and intracerebral resistance vessels. With the aid of a table, the authors describe the distribution of adrenoreceptors in various arteries and arterioles. The same table also summarizes the effects of phenylephrine, ephedrine, norepinephrine and dopamine on the cerebral blood flow, cerebral perfusion pressure, oxygen extraction fraction and brain tissue oxygen saturation in the healthy brain.

The second section offers a more detailed description and discussion of the aforementioned vasoactive medications and their effects on systemic circulation, cerebrovascular physiology and their interactions in the healthy brain.

The third part of the article is dedicated to vasopressors in TBI. Vasoactive drugs may have different effects on cerebral blood flow and cerebral metabolism once the integrity of the blood brain barrier (BBB) is disrupted. In TBI, vasoactive agents may have direct access to brain tissue and could possess direct vasoconstrictive effects on the vessels. E.g. noradrenaline has been observed to reduce regional CBF. In addition, cerebral metabolic rate of oxygen and glucose may increase. Despite a limited number of studies, the article looks at each vasoactive drug in detail.

Lastly, the authors discuss the role of microcirculation and oxygen exchange in head injury. Unfortunately, data on this subject is scarce. Of note, this research group has published several microcirculation studies (1,2,3).

Although no recommendation about the preferred vasoactive agent is made, this nice and comprehensive review from the Danish neuroanesthesiologists is a must-read that should interest not just neuroanesthesiologists and neurointensivists, but all anesthesiologists.

Until more evidence is available, the vasoactive of choice in patients with TBI should perhaps be based on the familiarity of the drug and local practice. More studies are still needed on vasopressors (including arginine vasopressin) and inotropes (dobutamine) and their effects on cerebral physiology in various neurocritical illnesses and on patient outcome.

References

