

Rethinking Vasopressor Education: The Need to Avoid Teaching the Bare Minimum

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To the Editor:

As medical educators, we all strive to teach in a manner that facilitates learners' understanding, and optimizing the delivery of educational material is essential in this process. Conveying potentially complex topics to novices in an organized, methodical, and succinct approach helps to ensure that the “how” and “why” of problems are sufficiently communicated. However, the educational content (the “what”) must be sufficiently comprehensive to capture both key physiological concepts and relevant supporting data for those concepts. Although the educational format described in this brief review (1) is excellent, we believe the authors erred in their exclusion of angiotensin II and the renin-angiotensin-aldosterone system (RAAS). With regard to hemodynamic management, the authors chose “... to emphasize the evolutionary basis of human physiology rooted in ensuring

survival, which may not neatly translate to sustaining life amid a prolonged shock state.” However, they preface their discussion with “... we do not discuss ... angiotensin II, or other inotropes (i.e., levosimendan and milrinone), as these topics tend to distract from the core physiologic principles being conveyed” Excluding the RAAS from a contemporary discussion on human hemodynamic physiology and hemodynamic management is to omit one of the few evolutionary pillars of mammalian physiology that ensures adequate end-organ perfusion. It would be unthinkable to exclude the RAAS in a similar discussion about how to teach the management of hypertension. Angiotensin II, an active metabolite of the RAAS, was first described for managing vasodilatory shock in the *Journal of the American Medical Association* in 1961 (2), and we have previously described its more than three decades of clinical use (3).

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We coauthored a 2017 multicenter, intercontinental, placebo-controlled randomized controlled trial published in the *New England Journal of Medicine* highlighting the hormone's ability to raise blood pressure (4), and since then numerous studies have subsequently confirmed those findings (5). To refer to the RAAS as a distraction from the core physiologic principle of hemodynamic homeostasis is a disservice to the next generation of

learners. We, as critical care medicine educators, must fundamentally reconsider the content of what we consider acceptable when teaching hemodynamic management. The day we sacrifice the quality of complex educational content to improve the delivery of oversimplified concepts is the day the bear catches us.

Author disclosures are available with the text of this article at www.atsjournals.org.

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