Commentary: Angiotensin II: Expanding the rescue options for vasoplegia

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Given the serious effects of vasoplegia on outcomes after cardiothoracic surgery, clinical rescue remains a priority in perioperative management. The knowledge gaps in the pathogenesis of this syndrome have prompted calls for further investigation to solve this complex puzzle, including the roles of the renin-angiotensin-aldosterone system.

In this issue of the Journal, Chow and colleagues evaluate the evidence supporting angiotensin II for vasoplegic rescue in cardiothoracic surgery. The authors highlight the disordered kinetics in the renin-angiotensin-aldosterone system to provide a rationale for support of systemic vascular tone with angiotensin II. They also suggest dosing protocols for angiotensin II in the cardiothoracic setting, as well as avenues for future research.

How might this important work modify our approach to vasoplegia? First, angiotensin II is an important option for clinical rescue in refractory vasoplegic shock. Second, angiotensin II also may facilitate a significant reduction in total vasopressor dose. This vasopressor-sparing effect may minimize the ischemic risk by supporting a more balanced restoration of vascular tone.

Although this report expands the therapeutic armamentarium for vasoplegia, it also highlights some important caveats with this vasopressor. The first caveat is that we know very little about adverse events associated with angiotensin II, owing to the limited evidence base. Although the evidence has established angiotensin II as a therapeutic option in vasoplegia, further clinical trials are needed to delineate its therapeutic window. The second caveat is that the current evidence base has a distinct adult focus. Whereas vasoplegia remains an important syndrome in pediatric practice, the role and safety of angiotensin II in this clinical domain remain largely unknown.

So where do we go from here? There is a critical mass of evidence to support further scientific inquiry both at the biochemical and bedside levels. Detailed investigation of the biochemical mechanisms may explain the observed efficacy of angiotensin II in vasoplegia, likely framed by the kinetics within the renin-angiotensin-aldosterone cascade. The bedside approach to inquiry would include further clinical trials to define the roles and safety of angiotensin II in the management of systemic vascular tone for both pediatric and adult cardiothoracic practice, perhaps with a focus on high-risk patients. These dual approaches are complementary and so together may offer us the best way to integrate angiotensin II into our clinical practice.

In conclusion, Chow and colleagues are to be congratulated for highlighting the promise of angiotensin as a rescue option for vasoplegia in cardiothoracic practice. They have emphasized the role and clinical implications of the renin-angiotensin-aldosterone system in the complex puzzle of vasoplegia associated with cardiac surgery.

References