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Vasopressin Is No Inotrope

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Dear Dr Jacobs,

Response to the letter to the editor by Kaufman et al regarding our article "The Perspective of the Intensivist on Inotropes and Postoperative Care Following Pediatric Heart Surgery: An International and Systematic Review of the Literature" in the *World Journal for Pediatric and Congenital Heart Surgery*. 2018;9(1).

I would like to thank Drs Kaufman and da Cruz for their interest in our article and their comments and would also like to thank the editor for providing us with an opportunity to respond. I certainly agree with our colleagues from Colorado that vasopressin can be a very useful pharmacologic agent to treat vasoplegia of different causes. However, treating vasoplegia and increasing blood pressure is not the same as increasing cardiac output, which was the focus of our review.[1] The use of vasoactive medication, vasodilators, and vasoconstrictors probably merits a survey and review on its own.

Many centers will actually aim to reduce afterload as much as possible after cardiac surgery using vasodilators such as milrinone, low-dose dobutamine, sodium nitroprusside, α-blockers, and/or ACE inhibitors.[2,3] In our survey, 79% of respondents indicated they employ a strategy of decreasing afterload in postoperative Norwood patients specifically.[1] It was recently shown in an animal model with fixed loading conditions that milrinone is very poor at increasing cardiac output.[4] My personal belief is that with milrinone, which owes 30% of its increase in cardiac output due to vasodilatation,[5,6] we often see hypotension which then requires a vasoactive drug to increase/normalize blood pressure. In our survey, 30% of respondents indicated they added norepinephrine for this reason specifically and a total of 55% of respondents indicated the use of norepinephrine and 43% use vasopressin in their prophylactic regimen.[1] Respondents did not specify why a vasoconstrictor was added so frequently, but we assume it was to increase blood pressure as vasopressin has no inotropic properties. The best approach to improve cardiac output is unknown and it still raises many questions. I wonder how much increase in cardiac output can actually be achieved with milrinone when a vasoconstrictive agent is added? Aren't we just increasing afterload again and therefore mitigating the milrinone effect? How useful is milrinone with added vasoconstrictor for the clinically relevant outcomes of our patients? Should we even use milrinone? If we should, in which dose? And if we would not, would we need less vasoconstrictors such as vasopressin? We just don't know and this deserves further research. However, when it comes to treatment of postoperative low cardiac output syndrome (LCOS), only 3 (3.5%) of 86 in our survey indicated the use of vasopressin as a second-tier drug for the treatment of LCOS (unpublished data). This underlines our belief that vasoconstrictors do not have a role in increasing cardiac output. They do have a role in treating (postoperative) vasoplegia, and I thank Dr Kaufman and da Cruz for pointing that out.

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