

LETTER

Target blood pressure in sepsis: between a rock and a hard place

Thiago Domingos Corrêa, Jukka Takala and Stephan Mathias Jakob*

See related commentary by Beloncle *et al.*, <http://ccforum.com/content/17/2/126> and related research by Corrêa *et al.*, <http://ccforum.com/content/17/1/R21>

We acknowledge the constructive comments of Beloncle and colleagues [1] regarding our recently published study. We demonstrated that targeting a mean arterial blood pressure (MAP) between 50 and 60 mmHg (Low-MAP) in porcine fecal peritonitis was associated with increased incidence of acute kidney injury (AKI) in comparison to targeting a MAP between 75 and 85 mmHg (High-MAP), which resulted in increased net positive fluid balance and vasopressor load [2].

Beloncle and colleagues argue that a dilution effect of the higher amount of fluid resuscitation on creatinine concentrations cannot be ruled out. Nevertheless, we report total hemoglobin concentrations in our manuscript [1], and, at study end, they were actually higher in animals in the High-MAP group than in the Low-MAP group (10.0 g/dl versus 8.4 g/dl, respectively, $P = 0.008$; Table 3 in the original manuscript). The assumption that the low incidence of AKI in animals allocated to the High-MAP group could be explained by a dilution effect is, therefore, unlikely [2]. We hypothesize that the circulating blood volume in the High-MAP group was lower at study end as a consequence of norepinephrine-induced vasoconstriction.

Moreover, Beloncle and colleagues suspect that low baseline hemoglobin levels in the Low-MAP group may have contributed to the development of kidney dysfunction [2]. Nevertheless, since the values were virtually identical in the Low- and High-MAP groups (9.3 mg/dl and 9.2 mg/dl, respectively), it seems unlikely that low hemoglobin levels - which were normal for young pigs - explain the observed differences in AKI.

Abbreviations

AKI, acute kidney injury; MAP, mean arterial blood pressure.

Competing interests

The authors declare that they have no competing interests.

Published: 17 May 2013

References

1. Beloncle F, Lerolle N, Radermacher P, Asfar P: Target blood pressure in sepsis: between a rock and a hard place. *Crit Care* 2013, **17**:126.
2. Corrêa TD, Vuda M, Takala J, Djafarzadeh S, Silva E, Jakob SM: Increasing mean arterial blood pressure in sepsis: effects on fluid balance, vasopressor load and renal function. *Crit Care* 2013, **17**:R21.

doi:10.1186/cc12692

Cite this article as: Corrêa TD, *et al.*: Target blood pressure in sepsis: between a rock and a hard place. *Critical Care* 2013, **17**:433.

*Correspondence: stephan.jakob@insel.ch
Department of Intensive Care Medicine, Inselspital, Bern University Hospital and University of Bern, Freiburgstrasse 10. CH-3010, Bern, Switzerland