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CRITICAL CARE

LETTER

# Relation between mean arterial pressure and renal function in the early phase of shock: a prospective, explorative cohort study

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See related research by Badin *et al.*, http://ccforum.com/content/15/3/R135

We read with interest the paper by Badin and colleagues [1] in a recent issue of *Critical Care*. The paper concerns the relation between arterial pressure and renal function in the early phase of shock. The authors found that a threshold of mean arterial pressure (MAP) of 72 to 82 mm Hg could be necessary to avoid acute kidney injury (AKI) in septic shock. This result was not found when sepsis was not the cause of shock. The authors state that this may be related to the loss of renal autoregulation ability in the early course of sepsis, thus explaining the absence of MAP threshold in non-septic shock. However, human studies on this subject are lacking and this hypothesis remains mainly theoretical.

In this study, a difference exits between patients with AKI and those without AKI. Patients with AKI at 6 hours have a significantly higher volume expansion in the last 6 hours before inclusion. Volume expansion (a cornerstone of sepsis treatment) can lead to an increase in abdominal pressure [2]. This condition is not infrequent, even among medical patients [3], particularly during early septic shock [4].

Thus, the beneficial effect on renal function of a higher MAP may be linked to the preservation of abdominal perfusion pressure and filtration gradient (FG) rather than to the impairment of renal autoregulation. We have shown (though only with preliminary results) that even a very moderate elevation of intra-abdominal pressure (IAP) can lead to a significant decrease of FG when the systemic hemodynamic is profoundly altered [5].

Thus, it cannot be excluded that the higher MAP threshold in the septic group is linked to a higher IAP value. The relevant goal may be not to aim for a minimal MAP of more than 65 mm Hg to prevent AKI at the early phase of septic shock but to target a sufficient MAP value to preserve abdominal perfusion pressure and FG by monitoring IAP.

## Authors' response

Julie Badin and Thierry Boulain

We thank Dr. Jacobs for his relevant remarks concerning our work [1]. Although we did not measure IAP, we examined the influence of the amount of fluid administered on AKI: the amount of fluid, administered either before or during the first 72 hours and examined by quartiles, was not linked to the proportion of AKI at 72 hours in the whole population or in patients with sepsis or AKI at 6 hours ($P$ >0.1 in all cases by chi-square test). Furthermore, in the population that Dr. Jacobs refers to (septic shock and initial AKI), the areas under the receiver operating characteristic curve of time-averaged MAP to predict AKI at 72 hours were very similar in patients who received less than 2,000 mL (the median amount of fluid received) before inclusion and those who received 2,000 mL or more: 0.82 versus 0.86, respectively. The best MAP thresholds in the two groups were also similar: 71 versus 73 mm Hg. To explain these results that do not support the hypotheses of a major influence of the amount of fluid received and of the indirect influence of IAP, one might argue that (a) the link between fluids and IAP was not found in all studies [3] and (b) abdominal compartment syndrome secondary to aggressive fluid therapy often occurs after several days [6]. Finally, we acknowledge that randomized interventional studies are necessary to know whether, in order to prevent AKI, resuscitation of shock should target a fixed level of MAP or of MAP *minus* IAP.

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**Abbreviations**
AKI, acute kidney injury; FG, filtration gradient; IAP, intra-abdominal pressure; MAP, mean arterial pressure.

**Competing interests**
The author declares that he has no competing interests.

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