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POINT OF VIEW

Why did arterial pressure not increase after fluid administration?

¿Por qué la presión arterial no aumentó después de la administración de líquidos?

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Introduction

Arterial hypotension (usually defined as systolic blood pressure of <90 mmHg, or mean arterial pressure of <65 mmHg, or a decrease of >40 mmHg),[1] often represents the first clinical sign of an acute decompensated cardiovascular system, and it is the most frequent indication for fluid administration in critically-ill patients.[2] However, the arterial blood pressure (ABP) response to intravenous volume expansion (VE) is somehow unpredictable: some patients exhibit an increase and others not.[3] Therefore, fluid administration, if aimed to restore and maintain ABP, could lead to an unnecessary fluid overload, delayed vasoactive therapy or even an increased mortality.[4]

In the following, we describe the physiological basis of the flow-pressure relationship to understand why, in some hypotensive patients, VE seems to be effective in terms of arterial pressure increase, whereas in others patients ABP remains unchanged, even if cardiac output (CO) increases.

What is arterial hypotension?

Arterial pressure results from the interaction between the blood flow generated by the ventricle and the arterial system.[5] ABP therefore is a regulated variable modulated within narrow limits: as long as the homeostatic autoregulation is preserved, there could be different combinations of arterial system and blood flow conditions for the same level of arterial pressure.[6] Consequently, arterial hypotension should be considered as the consequence of loss of balance between ventricular output and the modulation of the arterial system.[7] Moreover, systemic hypotension is a frequent preamble to organ hypoperfusion, since below a critical arterial pressure level, organ perfusion becomes compromised and regional blood flow will depend directly on systemic pressure.[6] For this reason, a sustained low blood pressure, or even a sole episode of hypotension, has been associated with a poor outcome.[8–11] Similarly,

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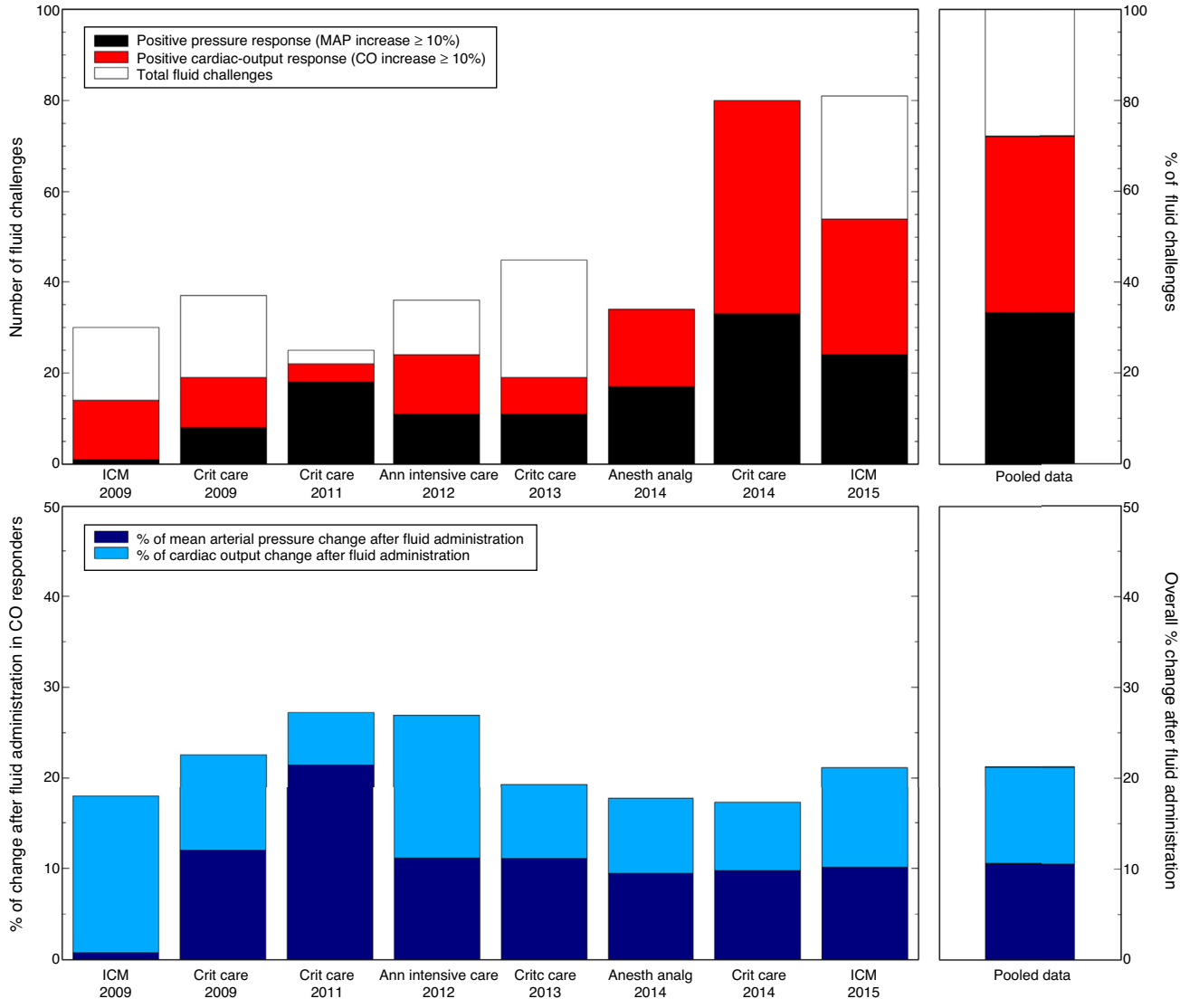

Figure 1 Rate of positive arterial pressure response after fluid administration.
Proportion of pressure-responders (mean arterial pressure, MAP increase ≥10%) and preload-responders (cardiac output, CO increase ≥10%). A ventriculo-arterial coupling ratio 1:1 was assumed. So, for a CO increase of 10%, a MAP increase of 10% should be expected. Please, note that preload-responder and pressure-responder definitions could differ from that defined in the original publication.

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