CRITICAL CARE

LETTER

Re-thinking resuscitation goals: an alternative point of view!

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See related viewpoint by Dünser *et al.*, http://ccforum.com/content/17/5/326

We respectfully disagree with several key assertions made by Dünser and colleagues [1] in this issue of *Critical Care* and consider their approach to resuscitation to be potentially harmful. Septic shock is not primarily a volume-depleted state, and attempts to treat vasoplegic shock with fluids alone will compound the macro- and micro-circulatory abnormalities of sepsis. A vasopressor with both α1 and β1 adrenergic activity (norepinephrine) will increase arterial tone, preload, and cardiac contractility [2]. Early administration of norepinephrine is associated with improved hemodynamics and reduced mortality in patients with sepsis [2,3]. Permissive hypotension is an untested and potentially dangerous concept. When the mean arterial pressure (MAP) falls below an organ's autoregulatory threshold, organ blood flow decreases in an almost linear fashion [4]. Because the autoregulatory ranges of the heart, brain, and kidney are above 45 mm Hg [4], such a blood pressure will, as one would expect, predictably decrease organ blood flow. Lehman and colleagues [5] have convincingly demonstrated that the risk of kidney injury and death increases sharply with an MAP of below 60 mm Hg. The notion that sepsis is associated with tissue hypoxia is unproven and, as Hotchkiss and Karl [6] argued over 20 years ago, is likely to be incorrect. Attempts to titrate therapy to a nonexistent oxygen debt on the basis of an elevated lactate concentration are doomed to fail. Similarly, the use of central venous oxygen saturation to guide the resuscitation of patients with sepsis is problematic. Although urine output may be a valuable marker of renal perfusion in hypovolemic states, this clinical sign becomes unreliable in sepsis-associated acute kidney injury, in which experimental models show that oliguria occurs in the presence of marked global renal hyperemia [7]. In summary, we consider that the first step in the resuscitation of patients with septic shock is to achieve an MAP of at least 60 to 65 mm Hg with the use of vasoactive agents (norepinephrine) and small volumes of balanced fluid. A simultaneous goal would be to ensure adequate flow (cardiac output) as determined by echocardiography and minimally invasive cardiac output monitoring and supported by an integrated assessment that includes monitoring the patient's clinical response to therapy.

Abbreviation
MAP, mean arterial pressure.

Competing interests
The authors declare that they have no competing interests.

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References

1. Dünser MW, Takala J, Brunauer A, Bakker J: **Re-thinking resuscitation: leaving blood pressure cosmetics behind and moving forward to permissive hypotension and a tissue perfusion-based approach.** *Crit Care* 2013, **17:**326.
2. Hamzaoui O, Georger JF, Monnet X, Ksouri H, Maizel J, Richard C, Teboul JL: **Early administration of norepinephrine increases cardiac preload and cardiac output in septic patients with life-threatening hypotension.** *Crit Care* 2010, **14:**R142.
3. Abid O, Akca S, Haji-Michael P, Vincent JL: **Strong vasopressor support may be futile in the intensive care unit patient with multiple organ failure.** *Crit Care Med* 2000, **28:**947-949.
4. Bellomo R, Di Giantomasso D: **Noradrenaline and the kidney: friends or foes?** *Crit Care* 2001, **5:**294-298.
5. Lehman LW, Saeed M, Talmor D, Mark R, Malhotra A: **Methods of blood pressure measurement in the ICU.** *Crit Care Med* 2013, **41:**34-40.
6. Hotchkiss RS, Karl IE: **Reevaluation of the role of cellular hypoxia and bioenergetics failure in sepsis.** *JAMA* 1992, **267:**1503-1510.
7. Wan L, Bagshaw SM, Langenberg C, Saotome T, May C, Bellomo R: **Pathophysiology of septic acute kidney injury: what do we really know?** *Crit Care Med* 2008, **36:**S198-S203.

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