## The issue of fluid balance and mortality

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Appropriate use of fluid infusion in cardiac surgery patients is of primary importance in the perioperative period in order to optimize cardiac output and oxygen delivery and to reduce the use of vaspressors and inotropes. Fluid infusion is usually triggered by arterial hypotension, low urine output and signs of inadequate tissue oxygenation (e.g. hyperlactatemia).

This general rule, however, may not be applied when the heart is working in the flat part of the Frank Starling curve, where hypervolemia may cause excessive increase of filling pressures and tissue edema. In this light, it is mandatory to carefully dose the exact amount of fluids to administer in order to avoid the risk of volume overload. *In this issue of HSR Proceedings in Intensive Care and Cardiovascular Anesthesia*, Arora and co-workers shed some light on the issue of perioperative fluid administration to cardiac surgery patients and its correlation to mortality (1). The authors clearly showed how the effects of intravascular filling correlates with mortality, especially if the amount of given fluids exceeded four litres in the perioperative period. This effect remained significant even after adjustment for the presence of acute kidney injury and/or hypotensive events.

The authors did not specify if the nature of infused fluids had a role on patients outcomes nor if specific treatments such as early/aggressive perioperative ultrafiltration might inversely correlate with mortality. More than five years ago, data coming from the Prospective Pediatric Continuous Renal Replacement Therapy registry showed that survival rates in patients with multiorgan dysfunction syndrome were significantly better for patients with less than 20% fluid overload (58% vs 40% survival rate) at continuous renal replacement therapy initiation (2). Fluid balance is probably underestimated in critically ill adults where a huge fluid volume amount is infused in order to target hypovolemia and organ perfusion. Few clinical inves-

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tigations, until now, have evaluated the impact that fluid balance has on clinical outcomes in critically ill adults: the Sepsis Occurrence in Acutely Ill Patients (SOAP) study (3) and the PICARD (Program to Improve Care in Acute Renal Disease) study group (4) recently showed how critically ill patients with acute kidney injury and fluid overload experienced significantly higher mortality with respect of patients without fluid overload apart from the need for RRT. The work from Arora and co-authors seems to support, in the specific setting of cardiac surgery patients, the view that there might be a survival benefit from conservative approach to intra-operative intravascular volume expansion. Early initiation of continuous renal replacement therapies to prevent fluid accumulation and overload in critically ill patients, once initial fluid resuscitative management has been accomplished might be an alternative approach (5) in this light, fluid overload is evolving as a primary trigger/indicator for extracorporeal fluid removal, and this may be independent of dose delivery or solute clearance.

Another final aspect of the study from Arora must be highlighted: if it is true that extra volume provision was detrimental also in patients without acute kidney injury and/or hemodynamic instability. it must be said that in these last patients correlation between fluid overload and mortality was exponentially higher. If it is evident that counterbalancing fluid accumulation, particularly in patients with oliguria or established acute kidney injury might be beneficial, on the other side it is also clear that more severely ill patients might often miss any active attempt of achieving a negative balance and we do not know if increasing vasopressors dose might really improve survival of such patients. Once a need for increased cardiac output is considered, it is helpful to have an indicator of fluid responsiveness. Central venous pressure and pulmonary artery occlusion pressure have limited predictive value as indicators of fluid responsiveness (with respect to volumetric-echocardiographic estimations) due to the existence of different conditions affecting the distribution of blood volumes (6).

Normally, approximately 70% of the total blood resides in the small venules and veins (unstressed volume). Only the remaining 25-30% (1.2-1.4 l) of the total blood volume (stressed volume) determinates, with the elastic recoil of the vasculature, the mean systemic filling pressure that is, with the right atrial pressure, the main determinant of the venous return, and finally of the cardiac output (7). Cardiac surgery procedures and cardiopulmonary bypass deeply influence the venous return and the cardiac output by increasing the venous capacitance and right atrial pressure for different reasons (systemic inflammation, anesthetic drugs, mechanical ventilation). An increase in venous capacitance may cause a reduction in stressed vol-

ume and an increase in unstressed volume: the final result may be a reduction of cardiac output due to a relative hypovolemia.

It seems that some patients (i.e. in case of bleeding) may actually benefit from an increase in mean systemic filling pressure (volume load) whereas in others a venous capacitance reduction (anti-inflammatory strategies, vasopressors, conservative fluid infusion) should be encouraged.

More recently, the use of respiratory variations of arterial pressure (systolic pressure variation, stroke volume variation, pulse pressure variation) to predict fluid responsiveness, have shown some interesting data in both operating theatres and intensive care units (8). Unfortunately, these dynamic indices, robust and reliable under specific conditions (closed chest, controlled mechanical ventilation, sinus rhythm) have not been validated during open chest settings. In this light, low cardiac output in cardiac surgery patients should be managed with a multimodal monitoring (echocardiography, cardiac and vascular filling pressures, dynamic indices of fluid responsiveness) and treatment tailored to the single patient and clinical picture trying to obtain the best balance between fluids, inotropes and vasopressors during the whole intra and post-operative phase.

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