Author response

We share your understanding that fluid responsiveness is NOT equivalent to hypovolaemia, and that it also does not equate to fluid administration being of benefit. We offer our apologies should our article have suggested otherwise. Indeed, this is one of the key reasons why we advocate fluid administration against measurable goals, such as stroke volume, as well as blood pressure and pulse. Research suggests that around 30-70% of patients presenting to the ED or ICU are fluid responders,[1-2] i.e. they increase stroke volume by > 10% following a fluid bolus of 250-500 ml delivered in 15-30 minutes[3] (most commonly). It is likely that most patients with acute hypovolaemia are fluid responders. Euvolaemic or hypervolaemic patients may or may not be fluid responders. Goal directed therapy was based on the premise that continued fluid administration to hypervolaemic levels, coupled with red cell transfusion and vasopressors, would increases oxygen delivery and that this would improve organ function/reverse organ hypoperfusion. This approach does not improve outcomes in sepsis.[4-6] As such we agree that the presence of fluid responsiveness does not imply the requirement for fluids or that fluid administration improves outcomes. Sepsis is also not a hypovolaemic state but accounts for around two thirds of shock seen acutely in the Emergency Departments, and is the patient group from which the bulk of research subjects are recruited. We propose fluid administration against stroke volume as we believe this is the current best approach, in part to at least protect non-responders from excess fluid administration as this is known to be harmful.

In point 2 you quite rightly point out the contradiction between data suggesting central venous pressure (CVP) is not a useful endpoint for fluid resuscitation (although low CVP and low blood pressure do suggest fluid will improve both stroke volume and blood pressure) and some studies suggesting inferior vena cava collapsibility index (IVCCI) may have a role.[7] In part this is explained by IVCCI being a dynamic marker whereas most work with CVP was based on it used as a static marker. We suggested that a small and collapsing IVC indicates fluid tolerance – in that fluid will be unlikely to cause harm. We do not suggest the IVC or IVCCI to be used as guides to titrate fluid administration. The literature on IVC and IVCCI is limited and in some cases contradictory. We agree more work is required.

In point 3 you touch on the very important point that if fluid responsiveness is measured directly after fluid administration then measured changes do not allow for subsequent fluid redistribution and excretion. Most studies look at fluid responsiveness immediately post-delivery. A meta-analysis indeed suggests that blood pressure, pulse, stroke volume and cardiac output all decrease post this maximum with time.[8] The clinical relevance remains unknown. Recurrent fluid boli will inevitably risk oedema, indeed this is again why we advocate use of ECHO to define the patients where fluid does not improve physiology and may be detrimental.

Fluid administration has been with us for nearly 200 years. Doctors have assumed benefits and fluid responsiveness in patients acutely unwell with sepsis, hypovolemia and organ failure. We are now learning of the risks of fluid administration and overload. We applaud our colleagues in intensive care medicine for their research into fluid therapy and hope to join Dr Seccombe in unravelling some of the issues that apply to patients who are not critically ill but still are managed with fluid administration. We would welcome collaboration and the development of a cross specialty fluid research group.

Parulekar P, Harris T.

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