Safe fluid management of major surgical cases demands cardiac output measurement

James MFM, Department of Anaesthesia, University of Cape Town

Correspondence to: Prof Mike James, e-mail: Mike.James@uct.ac.za

Controversies regarding fluid management remain unresolved mainly due to the lack of good quality data on which firm conclusions may be based regarding the crystalloid-colloid debate, the choice between colloid solutions and the best way to assess adequate volume therapy.

It is generally accepted that perioperative hypovolaemia may lead to significant organ dysfunction and a reduction in the quality of outcome. Appreciation of this has led to various approaches to avoid hypovolaemia, including replacement of third space losses, the maintenance of tissue perfusion (largely judged by a determined urine output) and the use of various haemodynamic targets against which to judge the adequacy of volume therapy. Early perioperative fluid therapy in the 1950s was based on a realisation that the stress response limited the ability of the body to clear sodium and water and consequently this approach focused on avoiding fluid overload. Subsequently, however, it was shown that goal directed therapy aimed at improving tissue oxygen delivery resulted in improved surgical outcomes.1 However, a number of subsequent studies failed to confirm these results. In a systematic review of volume optimisation strategies performed in 2002, the University College London group evaluated 12 studies that had studied fluid optimisation and concluded that these studies demonstrated a significant reduction in mortality that was best seen in elective general surgery although there was a trend towards similar outcomes in vascular and cardiac surgery. In this meta-analysis there was no obvious benefit in terms of length of hospital stay or days spent in intensive care. A study in 2002 examined the effects of targeted fluid therapy in patients who suffered a hip fracture and examined outcome in terms of length of hospital stay in three groups. The control group was allowed a free fluid with the aim of maintaining haemodynamic targets within 20% of baseline. The two study groups received additional colloids to leave either a predetermined central venous pressure (CVP) or maximum stroke volume. The two study groups received significantly more colloid volume, in the region of 600 mL, and both groups had a significant reduction in time to fit the hospital discharge.2

Recently, however, there have been several studies that have questioned the validity of generous fluid loading. These studies have demonstrated an increase in complication rates in patients undergoing abdominal surgery who were fluid overloaded.3 On the opposite side of the coin, other studies have shown that moderate crystalloid loading improved a number of markers of patient well-being.4 These controversies led to the concept of fluid optimisation and the view that too little and too much fluid were equally harmful.5 A Cochrane database review of fluid therapy in patients with femoral fractures concluded that there was evidence of a reduction in the length of hospital stay, but no other risks or benefits associated with fluid optimisation. The authors concluded that there were inadequate data on which to base firm conclusions.6 A meta-analysis of optimisation in a variety of cases concluded that severely ill patients where the expected mortality was > 20% would benefit from fluid optimisation, but that there was no benefit demonstrable in patients where the mortality was < 15%. These authors also concluded that haemodynamic optimisation had to be performed prior to onset of organ failure in order to be effective.7 Given then that there is good evidence that fluid optimisation can improve outcome, how should optimal fluid volume in any given set of circumstances be assessed. It is clear that static haemodynamic filling pressures, such as CVP, arterial pressure, and PAOP are at best extremely unreliable in terms of judging adequacy of volume loading. Particularly, measures of central venous filling pressures are only of value in indicating the adequacy of vascular filling at the extremes of these measurements. In the mid-range, which is where we most need to understand the dynamics of fluid loading, they are of little value. Recently, the concept of recruitable stroke work has become a popular index of adequate vascular filling. In this model, the view is taken that increasing cardiac output in response to an increased fluid load indicates a central volume deficit and that fluid should be titrated to some such monitor of cardiac responsiveness.8 Attempts are being made to identify a practically useful and clinically valid index of optimal cardiac function including direct measures of cardiac output responsiveness, changes in cardiac performance in response to ventilatory cycles and measures of the adequacy of tissue perfusion including gastric pH, tissue CO₂ levels and muscle oxygen saturation.

Various studies have looked at gastric pH as a target for fluid therapy,9 and most of these have shown a positive outcome, but the technology proved cumbersome, gaining little popularity, and is no longer available. Variation in the arterial pressure
The waveform in response to positive pressure ventilation has also been used as an index of the adequacy of cardiac filling. Measures such as Delta down, pulse pressure variation and systolic pressure variation have been shown to be effective in determining apparent volume deficits, but no outcome data exist for these methods demonstrating a reduction in perioperative morbidity.

More direct measures of cardiac performance, including pulmonary artery catheterisation, trans-oesophageal echocardiography, oesophageal Doppler studies and various forms of algorithm-driven analysis of arterial waveforms have received considerable attention. The cost and complications of pulmonary catheterisation have rendered this much less popular and intermittent injection cardiac output determination by thermodilution is not really suitable for fluid optimisation. Similar objections pertain to echocardiography, and the accuracy of echocardiographically determined cardiac output measurements has been questioned.

The technique that has received the greatest attention so far is that of trans-oesophageal Doppler estimations of aortic flow velocity which can be converted into cardiac output and stroke volume through measurements of aortic diameter and a proprietary algorithm. Several studies have examined this technique for optimisation of cardiac output and these were the subject of a recent meta-analysis. This meta-analysis concluded that oesophageal Doppler-guided intraoperative fluid replacement in patients undergoing major abdominal surgery led to higher colloid fluid loading, leading to better urine output and cardiac output without any difference in central venous pressure with arterial pressure. Clear benefits were demonstrated in terms of length of hospital stay and the overall rate of complications. ICU was also reduced and bowel function recovered earlier. However, mortality was not significantly different, but the numbers were small. The authors also raised the issue of extending such monitoring into the postoperative period but cautioned that the cumbersome nature of the intervention may make this difficult. Alternative forms of assessment of central filling volume that may be less operator-dependent and easier to use remain to be evaluated. These include various analyses of arterial pulse wave contours and pulse pressure variation with positive pressure ventilation.

In conclusion, there can be little doubt that sick patients undergoing major surgery should have their fluid management titrated against some measure of cardiac output. What is the ideal method for such assessment remains to be established, and so far the only one that has been properly validated is trans-oesophageal Doppler.

References