Fluid therapy in 2015 and beyond: the mini-fluid challenge and mini-fluid bolus approach

P. E. Marik

Division of Pulmonary and Critical Care Medicine, Eastern Virginia Medical School, 825 Fairfax Ave Suite 410, Norfolk, VA 23507, USA
E-mail: marikpe@evms.edu

The first description of the use of intravenous fluid in a human is attributed to Dr Thomas Latta during the cholera epidemic in London in 1831–2. Dr Latta described his experience in a letter to the editor of The Lancet.1 Dr Latta first attempted to replace the lost fluid and salts ‘by injecting copiously into the larger intestines warm water, holding in solution the requisite salts, and also administering quantities from time to time by mouth’.2,3 He found there to be no permanent benefit and considered that the unfortunate sufferers’ vomiting and purging were aggravated. Dr Latta wrote ‘finding thus, that such, in common with all the ordinary means in use, was either useless or hurtful, I at length resolved to throw the fluid immediately into the circulation’. The injected solution was made up of ‘two to three draehms of muriate of soda and two scruples of the subcarbonate of soda in six pints of water’ (equivalent to approximately ½ Ringers lactate). His first patient was an elderly woman who had been given all the usual remedies and who had ‘reached the last moments of her earthly existence.’ Dr Latta inserted a tube into the basilic vein and ‘injected ounce after ounce of fluid, closely observing the patient’, at first with no visible effect, but then she began to breathe less laboriously and ‘soon the sharpened features, and sunken eye, and fallen jaw, pale and cold, bearing the manifest imprint of death’s signet, began to glow with returning animation; the pulse returned to the wrist. . .’. After 6 pints (2.8 litre) of fluid had been injected, the woman announced in a strong voice that she was now ‘free from all uneasiness’ and was cured.

The technique of fluid resuscitation described by Dr Latta nearly 200 yr ago has stood the test of time, and appears to be the only logical method to resuscitate patients—give repeated small boluses of fluid and observe the patient closely (what a remarkable concept!). This is best done by giving 200–500 ml boluses of Ringers lactate solution (or 4% human albumin solution) and closely monitoring the response. While the basic concept has not changed, the single most important advancement since the days of Dr Latta is the ability to measure stroke volume (SV) continuously by minimally invasive or non-invasive techniques.4 This allows the clinician to assess the patient’s fluid responsiveness and changes in SV over time. Fundamentally, only patients who are fluid responsive should be treated with fluids.5 Physical examination, chest radiography, central venous pressure (CVP), urine output (particularly in septic patients), and ultrasonography, including the vena caval collapsibility index, have limited value in determining fluid responsiveness and guiding fluid management.6–10 In the intensive care unit (ICU), fluid responsiveness can be determined by a passive leg raising (PLR) manoeuvre coupled with SV monitoring.1 This manoeuvre is a good predictor of fluid responsiveness in both intubated and non-intubated patients.11 The change in the pulse pressure variation (PPV) or stroke volume variation (SVV) following a mini-fluid challenge (100 ml), as elegantly described by Mallat and colleagues12 in this issue of the BJA, is an alternative and/or complementary technique to determine fluid responsiveness in patients in the ICU or operating theatre who are receiving low tidal volume ventilation. However, as demonstrated by Mallat and colleagues and others, the ‘non-challenged’ PPV and SVV have limited diagnostic accuracy (and applicability in ICU patients) for determining fluid responsiveness.13–15

Although still widely recommended,6,16 the idea of giving large boluses of crystalloids (20–30 ml kg⁻¹) is unphysiologic and likely to lead to severe volume overload.17,18 The ability of crystalloids to increase intravascular volume is poor. In healthy volunteers, only 15% of a crystalloid bolus was reported to remain intravascular at 3 h.19,20 In patients with sepsis, <5% of an infused bolus remains intravascular 1 h after the end of the infusion.21 In a caecal ligation model, Bark and colleagues22 demonstrated that the plasma volume expanding effect of normal saline was <1% of the infused volume 20 min after the end of the infusion. In critically ill medical, surgical, and trauma patients, the hemodynamic effects of a fluid bolus are likely to be short lived, with the net effect being the shift of fluid into the interstitial compartment with progressive tissue oedema. Tissue oedema impairs oxygen and metabolite diffusion, distorts tissue architecture, impedes capillary blood flow and lymphatic drainage and disturbs cell–cell interactions, leading to organ dysfunction.23–24 In encapsulated organs such as the kidney, tissue oedema increases interstitial pressure, compromising renal blood flow, which may play a role in the aetiology of acute kidney injury.25 Increased extravascular lung water (EVLW) impairs gas exchange, reduces lung compliance, increases the work of breathing, and is a strong independent predictor of death.26–27

Nunes and colleagues28 assessed the time course of the hemodynamic response of a 500 cc fluid challenge in patients requiring vasopressor support. In this study, 65% of patients were fluid responders; however, the SV increase (in the responders) returned to baseline 60 min after the infusion. Fluid boluses are most frequently given for hypotension or oliguria. While the mean arterial pressure (MAP) may increase immediately following a fluid bolus, this effect is short lived. In a systematic review that investigated the hemodynamic response of fluid boluses in patients with sepsis, Glassford and colleagues29 demonstrated that the MAP increased by 7.8 (3.8) mm Hg immediately following the fluid bolus, by 6.9 (2.7) mm Hg 30 min following the bolus, and by only 2 mm Hg at 1 h, with no increase in the urine output.
following the boluses. These data suggest, as described by Dr Latta more than 200 yr ago, that hemodynamically unstable patients who are fluid responsive should be treated with repeated mini-fluid boluses (200–500 cc) and guided by changes in their hemodynamic profile (including SV). Furthermore, the risk:benefit ratio should be assessed prior to each fluid bolus. It is noteworthy that in the study by Wu and colleagues a mini-fluid bolus of 50 ml crystalloid was associated with a 17% increase in SV. It is likely that the mini-fluid bolus approach will result in smaller increases in cardiac filling pressures with the attenuated release of atrial natriuretic factors and less tissue oedema with a lower cumulative positive fluid balance than large volume fluid resuscitation. In the study by Mallat and colleagues there was a significant decrease in the systemic vascular resistance index in the fluid responders following the fluid bolus. This observation has been reported previously following fluid boluses in patients with sepsis. This suggests that fluid boluses should be considered vasodilator therapy in patients with sepsis and that large volume fluid resuscitation may potentiate the hyperdynamic state. An emerging paradigm in critical care suggests that a ‘less is more’ approach improves patient outcomes, and this approach appears to apply to fluid resuscitation.

Declaration of interest

The author has no financial interest in any of the products mentioned in this article.

References

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