Fluid physiology, tissue compliance, and colloids

Editor—I would like to thank the authors for their immensely informative article on transvascular fluid exchange.\(^1\) It is apparent that advances in this area of science are now starting to explain many of the clinically observed phenomena in critically ill patients, such as the broad equivalence of crystalloids and colloids, and the failure of albumin infusions to improve tissue oedema.

A phenomenon familiar to many of us is the turning point when a sick patient in intensive care exhibits spontaneous diuresis, thereby excreting their iatrogenic excess body water—at this stage, we ‘know’ that the patient will get better. Do the authors think that this occurrence represents the juncture at which tissue compliance returns to normal, and the increased hydrostatic pressure drives fluid back into the circulation? Certainly, the components of the inflammatory response that control these changes hold great promise as targets for future therapies.

Despite the admirable explanation of the revised Starling equation in this article, I am not sure I agree with the conclusion that a rational use of colloids should be restricted to euvolaemic or hypervolaemic haemodilution. This statement is based on the premise that patients who are profoundly hypovolaemic will have an equivalent plasma expansion with either class of fluid, that is, they are below the J-point on the graph provided (Fig. 4 in the original article). Above the J-point, colloids do stay in the circulation for longer, and I would argue that this applies to fluid optimization in the perioperative period, where patients are only moderately hypovolaemic. Thus, over several days of flow-guided optimization, the total volume of colloids used will be less compared with crystalloids for the same effect. However, the long debated question remains as to whether the advantage of less tissue oedema outweighs the other adverse effects (and cost) associated with colloids.

Declaration of interest

None declared.

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Reply from the authors

Editor—Dr Toner raises some interesting points regarding our article on the revised Starling equation and glycocalyx model paradigm (RSE&GM) which we are pleased to respond to.\(^1\) We recognize the turning point Dr Toner describes when a recovering patient sheds litres of oedema, and it seems to be a sign of recovery rather than the cause of it. We could not do more than speculate on the mechanism, but normalization of interstitial collagen fibre conformation and reversal of GAG hydration must be involved. The relevance of RSE&GM to the practice of perioperative goal-directed fluid therapy is immense. First, we need to consider where the patient is on the J-curve. We suspect that general anaesthesia tends to lower venous resistance more than arterial resistance, lowering both arterial and capillary pressure and so moving the J-point to the left. We know that the endothelial glycocalyx layer is reduced or compacted during anaesthesia and surgery, and it therefore seems prudent to prefer a colloid-free isotonic salt solution for stroke volume optimization until the transendothelial pressure difference has been restored to the J-point. Dr Toner correctly observes that we would only choose to include colloid molecules in our isotonic salt solution if we intend to take the patient substantially above the J-point and retard Jv, but if stroke volume optimization can be achieved around or below the J-point, a colloid-free isotonic salt solution will suffice. On Dr Toner’s concern about tissue oedema, many anaesthetists use phenylephrine to preserve the mean arterial pressure after recovering from circulatory side-effects of anaesthesia, phenylephrine is weaned off. With judicious vasopressor and diuretic therapy, it is our practice to take care not to allow patients to become fluid-overloaded in the days after surgery, whether or not the anaesthetist or surgeon chooses to use an intraoperative colloid solution. Dr Toner’s assertion that the total volume of colloid used for optimization over several days is less compared with crystalloids is worth quantifying in his practice. It is widely asserted that colloid boluses are essential for goal-directed intra-operative fluid therapy, in spite of published evidence to the contrary. Future clinical studies need to include a group in which the haemodynamic goal is targeted by variable infusion of a colloid-free isotonic salt solution, and in which we allow excessive anaesthetic-induced vasodilation to be minimized by a judicious dose of an α-adrenoceptor agonist to protect capillary pressure. We should be concerned that there is very little published safety information on the effects of starches or gelatines on the endothelial glycocalyx layer.