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Goldilocks, elephants, and surgical fluids

Editor—There is always a risk we approach a clinical problem like the proverbial men approaching an elephant. Fluid management around the time of surgery may be one of these elephants. There is level 1 evidence of at least 29 randomized trials, suggesting that pre- and intraoperative fluid optimization strategies improve morbidity and mortality after major surgery.1 Subgroups in this meta-analysis suggest that targeted/goal-directed therapies are better than other fluid optimization strategies.1 There is also a ‘systematic review’ of seven trials identifying three trials that suggest benefit from fluid restrictive strategies in terms of improving morbidity and length of stay after surgery.2 Further, there are ‘evidenced based guidelines’ recommending optimization strategies and national implementations of such strategies that do not seem to take the fluid restriction evidence base into account.1

Superficially at least, fluid optimization and fluid restriction seem contradictory when considered at any individual time point in care, yet there is evidence they are both likely to be beneficial and possibly more similar than the superficial first glance would suggest.2 This is partly due to terminological issues and requires clarification.3 No one seems to disagree that there is such things as too little fluid (pre and perioperative hypovolaemia/underhydration are associated with inadequate organ perfusion and tissue healing), or too much fluid (associated with organ oedema and surgical complications).3 4 Further, no one would disagree that there is a Goldilocksian ‘just right’ also,3 4 but knowing where this ‘just right’ lies at any one time is more challenging. The problem with the Bellamy model is that it is a static model, when ‘just right’ will likely change between stages of the patient’s care. Varadhan and Lobo3 suggested that fluid balance should be neutral (zero balance) across the entire surgical care period. This may well be correct, but does not guide fluid therapy at any one time point as such.

So maybe we should consider a more dynamic model for ‘just right’ to complement the suggestions of Bellamy (Fig. 1).4 The evidence seems to tell us that, in the immediate preoperative and early intraoperative period, patients benefit from avoidance and correction of hypovolaemia and from fluid optimization (especially when directed at specific goals/targets).5 This strategy will ensure adequate organ oxygen delivery at this critical early period of surgical management. Then, in the later intraoperative period and early postoperative period (exact timing still unclear), we should move to a more restrictive phase of fluid balance in order to avoid tissue oedema and its harmful effects on tissue healing. At this stage, we should remove the accumulated fluid from the preoperative and early intraoperative period and ultimately achieve an overall neutral fluid balance across the operative period.2 It would seem highly probable that the exact volume of fluid and timings of transitions would require titration depending on patient and surgical factors, but the overall pattern would be likely to remain very similar. Clearly, this proposed model would have to be tested in a prospective randomized trial and identify its potential effectiveness and cost-effectiveness, but I believe that it has merit.6

![Fig 1 Dynamic model of perioperative fluid balance.](image-url)
Declaration of interest

None declared.

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4 Bellamy MC. Wet, dry or something else? Br J Anaesthesia 2006; 97: 755–7


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Topical lidocaine to suppress trigemino-cardiac reflex

Editor—A 68-yr-old woman had undergone microvascular decompression (MVD) 2 yr ago for trigeminal neuralgia but presented on this occasion for recurrence of symptoms. She was again scheduled for MVD by the same surgeon. Her medical history was unremarkable and all the laboratory investigations and ECG were within normal limits. Her medications included carbamazepine 200 mg and pregabalin 75 mg once daily.

Standard monitoring was established and anaesthesia was induced with fentanyl 100 μg, propofol 100 mg, and muscle relaxation achieved with vecuronium 5 mg after which the airway was secured with an oral tracheal tube. The lungs were ventilated with 50% O₂ in N₂O and sevoflurane to an ECO₂ 4.0–4.5 kPa. After induction, the left radial artery was cannulated for direct arterial pressure monitoring.

After craniotomy, the surgeon found an additional vascular loop at the root entry zone (REZ) and the graft placed at the previous surgery was in situ. During dissection, there was sudden and profound bradycardia (30 beats min⁻¹) and hypotension (60/40 mm Hg). As the surgeon was alerted to stop the stimulus and atropine 0.6 mg was being given i.v., the monitor showed asystole which lasted for about 10 s and then there was a gradual increase in the heart rate to 70 beats min⁻¹ and arterial pressure increased to 120/70 mm Hg.

After waiting for 5 min, when the surgeon resumed the procedure, similar changes occurred including asystole which reverted to normal rhythm with the removal of the stimulus and without therapeutic intervention. The surgical and anaesthetic team were in a dilemma. Then, one of the authors (N.N.V.) advised the surgeon to place a gauze dipped in 2 ml of 2% lidocaine at the REZ. After 3 min of waiting with the gauze in place, the surgery was resumed and completed without any haemodynamic fluctuations.

The surgeon could not recall any serious haemodynamic changes, nor did the previous anaesthetic chart show any such changes, during the previous surgery.

Residual neuromuscular block was reversed, the patient extubated, and the postoperative ECG was normal.

The afferent arc of the trigemino-cardiac reflex (TCR) is the trigeminal nerve and vagus nerve the efferent. The manifestations range from bradycardia, bradycardia terminating in asystole, hypotension, apnoea, and gastric hypermotility.

In our case, the fifth nerve was approached by the same surgeon using the same surgical approach and a similar anaesthetic technique. There was neither bradycardia nor asystole during the first surgery.

Postulated predisposing factors for TCR include hypotension, hypercapnia, hypoxaemia, light anaesthesia, opioids, calcium channel blockers, β-blockers, abrupt and sustained traction and surgical division of the nerve.

As there was no attempt at sectioning the nerve in this case, the only other factor was the surgery with the previously placed graft in situ, which could have been more reflexogenic.

Most reported cases of TCR reveal that the surgery was completed uneventfully after the administration of i.v. anticholinergic medication. But in our case the reflex returned despite anticholinergics. We thus resorted to topical application of local anaesthetic which proved to be effective. To our knowledge, there has been no case report of the use of topical lidocaine intraoperatively to blunt the afferent arc, although mention has been made of local anaesthetic infiltration or nerve block.

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