



Fluid management in the thoracic surgical patient: where is the balance?

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Pulmonary complications (PPCs) following lung resection are common and have a significant negative impact on the patients recovery after lung resection surgery as well as economic effect on health resource usage (1,2). A restrictive fluid strategy has been a long-standing dogma of such surgery which has been repeatedly challenged not only in terms of causality but because of potential deleterious effects on kidney function. The optimal fluid balance in the thoracic surgical patient during the perioperative period and its possible association with the development of PPCs has generated a long debate (3-5).

This analysis by Wu *et al.* was a retrospective observational study at a single institution between May 2016 and April 2017 that included 446 adult patients who underwent minimally invasive lobectomy, either robotic or VATS. Patients older than 70 years and those with renal dysfunction, ischemic heart disease, congestive heart failure, history of thoracic surgery, intraoperative bleeding >100 mL, second surgery, sleeve lobectomy, bronchiectasis, tuberculosis, chronic inflammatory disease and high or low BMI were excluded which may make some of the findings difficult to extrapolate to 'real life'. Despite this selection of patients and restriction to patients undergoing minimally invasive surgery the authors report a high rate of PPC of 38.5% compared to similar studies (6,7).

The patients were divided into 4 groups post HOC [restrictive (Q1) ≤ 9.4 mL/kg/h; moderate (Q2) =9.4–11.8 mL/kg/h, moderately liberal (Q3) ≥ 11.8 –14.2 mL/kg/h and liberal

(Q4) >14.2 mL/kg/h] depending on the incremental quartiles of the exposure variable—*intraoperative total fluid infusion rate*—to assess the impact on postoperative outcomes. Similarly, to analyses the effect of intraoperative colloid on post-operative outcomes, patients were classified into 3 groups—no intraoperative colloid, restrictive (up to 3.8 mL/kg/h) and moderate (>3.8 mL/kg/h). The colloid used was hydroxyethyl starch (HES). The observation outcomes were: PPCs, AKI, in hospital mortality, post-op length of stay and costs.

The incidence of postoperative pneumonia and PPCs was lowest in the moderate administration rate groups. Both restrictive and liberal fluid administration regimens were associated with poorer postoperative outcomes. Significant pre- and peri-operative differences between each group were noted; for example, patients in the restrictive group were mostly male, smokers and with a higher BMI. When looking into a possible association between the infusion rate and postoperative AKI, no significant difference between groups was demonstrated. The low rate of AKI reported by authors (1.8%) might explain this result.

These findings are interesting for several reasons. Firstly, the authors attempt to provide a basis for an intraoperative fluid threshold that can be used to minimize the risk of PPC. This is very challenging, mainly due to a lack of evidence of causality; in this study the driver for type of fluid and rate is left preference of the anesthetist and the clinical situation.

Is it that patients who have a higher requirement for fluid intraoperatively are just 'sicker' and so more likely to develop a PPC—a chicken and egg scenario? A randomized controlled trial by Matot *et al.* looking into the fluid management during VATS for lung resection showed that, within the range of 2–8 mL/kg/h of intraoperative fluid, PPC rate was not significantly different between the high (8 mL/kg/h) and low volume groups (2 mL/kg/h). But Matot's trial was not powered to look at this outcome (PPCs) and the rate of PPC was much lower than the one reported by Wu *et al.*, so we must interpret these results with caution (8).

Since the heterogeneity of our patient population precludes us from using a general approach to fluid management, some have suggested that fluid therapy should be individualized and based on objective feedback on one's fluid responsiveness. The benefit of goal directed therapy is controversial and most of the evidence comes from non-thoracic surgical trials (9–12). Although the literature suggests an association between the fluids infusion rate and the development of PPCs, to our knowledge, no causative relationship between the aforementioned parameters has been demonstrated.

Secondly, Wu *et al.* showed that an intraoperative colloid infusion rate over 3.8 mL/kg/h was associated with a lower incidence of PPCs without increasing the risk of post-operative AKI. Several randomized controlled trials in patients being administered HES and undergoing thoracic or abdominal aortic surgery, did not find any evidence for renal impairment (13–15). Although the evidence pertaining to the impact of intraoperative colloid on the development of PPCs is unclear, we do know that colloid solutions can keep the lung 'dry' by increasing the intravascular osmotic pressure and that a hyperoncotic state can result in osmotic necrosis-based kidney injury (16,17). Studies in abdominal aortic surgery which showed improvement in selected outcomes such as AKI and PPC, used a combination of colloid and crystalloid solutions (14,15), whilst the studies demonstrating that colloids are harmful only used HES as the sole fluid agent and included unwell, septic patients on ITU (18,19). These findings have not been validated in thoracic surgery.

In conclusion, a balanced approach to administering fluids intraoperatively seems to be the most sensible option for patients. PPCs and AKI development is multifactorial and the blame cannot only be solely placed on restrictive or liberal of fluid administration. Furthermore, this study re-emphasises that a euvolaemic strategy is likely the best approach to take peri-operative fluid management and this

recommendation is expressed in recent enhanced recovery guidelines for thoracic surgery (20).

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Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

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