Perioperative Fluid Management for Pulmonary Resection Surgery and Esophagectomy

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Abstract

Perioperative fluid management is of significant importance during pulmonary resection surgery and esophagectomy. Excessive fluid administration has been consistently shown as a risk factor for lung injury after thoracic procedures. Probable causes of this serious complication include fluid overload, lung lymphatics and pulmonary endothelial damage. Along with new insights regarding the Starling equation and the absence of a third space, current evidence supports a restrictive fluid regimen for patients undergoing pulmonary resection surgery and esophagectomy. Multiple minimally invasive hemodyamic monitoring devices, including pulse pressure/stroke volume variation, esophageal Doppler, and extravascular lung water measurement, were evaluated for optimizing perioperative fluid therapy. Further research regarding the prevention, diagnosis, and treatment of acute lung injury after pulmonary resection and esophagectomy is required.

Keywords

intraoperative assessment, monitoring, outcome, postoperative complications, thoracic surgery

Introduction

Perioperative fluid management is a key component of anesthetic management during thoracic surgery.[1] On one hand, fluid restriction could compromise perfusion of vital organs and surgical anastomosis. On the other hand, fluid overload could lead to cardiopulmonary complications, notably pulmonary edema, which carries a high mortality rate. Perioperative fluid management to avoid postpneumonectomy pulmonary edema has been previously reviewed.[1,2] In this review, we will provide an update on this topic. Furthermore, fluid management during other thoracic procedures, such as pulmonary resection in general and esophagectomy, will be discussed.

Incidence and Risk Factors of Lung Injury After Pulmonary Resection Surgery and Esophagectomy

Pulmonary edema has long been recognized as a complication after penumonectomy, with symptomatic onset on postoperative day 1 to 4. In one of the earliest and most widely known reports, Zeldin et al[3] described 10 cases of postpneumonectomy edema. Subsequently, this complication has also been found after less invasive pulmonary resection surgery, such as lobectomy, segmentectomy, and esophagectomy.[4-6] It shares the same histological features with acute respiratory distress syndrome (ARDS)[7] and is not associated with cardiogenic pulmonary edema. The majority of recent reports define this complication as acute lung injury (ALI) using the American–European Consensus on ARDS (Table 1),[8] with ARDS representing the most severe form. The incidence of ALI after pulmonary resection surgery ranges from 2% to 7% and up to 15% in penumonectomy (Table 2).[7,9-21] Despite advances in surgical and anesthetic techniques, the mortality rate of this complication remains high between 25% and 64%. Various anesthetic-related risk factors were implicated in the development of ALI. Notably, excessive perioperative fluid administration was consistently found to be one of these risk factors (Table 2).[3,11-14,17]

The incidence of ARDS following esophagectomy was reported in 2 studies to be 16% and 14.5%, with an associated mortality rate of 14% and 50%, respectively.[6,23] Both reports suggest increased fluid administration as a risk factor for lung injury. Furthermore, perioperative fluid balance is associated with increased incidence of postoperative mortality, cardiovascular and pulmonary morbidities.[24,25]

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Table 1. Criteria for ALI as per the American–European Consensus Conference.

1. Acute onset
2. Reduced PaO$_2$/FiO$_2$ ratio: ≤300 mm Hg for ALI and ≤200 mm Hg for ARDS
3. Bilateral infiltrates on chest X-ray
4. Pulmonary artery wedge pressure ≤18 mm Hg or no clinical signs of left atrial hypertension

Abbreviations: ALI, acute lung injury; ARDS, acute respiratory distress syndrome.

In pneumonectomy, Zeldin et al[3] initially reported that an amount of 37 mL/kg of fluids administered intraoperatively and 24 hours postoperatively is associated with postpneumonectomy pulmonary edema. Subsequent studies revealed that the infusion of more than 2000 mL of fluids during pneumonectomy is a risk factor for ALI (Table 3).[11,12,22] Furthermore, the perioperative (intraoperative and 12 hours postoperative) fluid infusion of more than 2500 mL negatively affected postoperative mortality.[26] Similar findings were observed in less invasive pulmonary resection.[14,17] An intraoperative fluid infusion of 9.1 mL/kg/h and a 24-hour fluid balance of 2000 mL are associated with the development of ALI. Last, in esophagectomy, an intraoperative fluid administration of more than 5000 mL is related to ALI.[6,23]

Pathogenesis of Acute Lung Injury After Pulmonary Resection Surgery

To date, there is no unifying theory that explains the pathogenesis of lung injury after pulmonary resection surgery. In the following section, current evidence related to fluid exchange between the intravascular and interstitial compartments will be examined to facilitate our understanding about the development of ALI. The various causes of this complication will also be discussed.

The Revised Starling Equation and the Glycocalyx Model

The fundamental principle governing fluid shifts between the plasma and the interstitial fluid compartments was first established by Starling.[27] In essence, the classic expression of the Starling principle is presented as follows:

$$\text{Filtration force} = (P_c - P_i) - (\pi_p - \pi_i),$$

where P_c and P_i represent the hydrostatic pressure in local capillary and interstitium, respectively, and π_p and π_i represent the oncotic pressure in local capillary and interstitium, respectively. This principle depicts the traditional model in which fluid filtrated into the interstitium at the arteriole

end of the capillary is reabsorbed in the venule end. However, multiple lines of evidence proposed a revision of the traditional Starling principle.[28] First of all, venous reabsorption was not observed when the Starling forces were individually measured in capillaries.[29] Only net filtration into the interstitium was noted, suggesting drainage through the lymphatic system is the primary factor to maintain interstitial volume. However, based on measurements of the 4 classic Starling forces, the predicted lymph flow to prevent interstitial edema in steady state is often much larger than the observed lymph flow.[30] To explain this paradox, the glycocalyx model was proposed. The endothelial glycocalyx layer (EGL) is a web of membrane bound glycoproteins and proteoglycans on the luminal side of endothelial cells.[31] In nonfenestrated capillaries found in the lungs and muscles, the EGL is prominent and continuous along the endothelium. Between the endothelium and EGL is the subglycocalyx space containing fluids with lower oncotic pressure than the interstitium (Figure 1). Because of its proximity to the intravascular space, the oncotic pressure difference that determines fluid exchange should be calculated as $\pi_p - \pi_g$ instead of $\pi_p - \pi_i$, where π_g represents the oncotic pressure in the subglycocalyx space. π_g is much lower than π_i because of the outward flow of transcapillary ultrafitrate preventing protein diffusion equilibrium between the subglycocalyx fluid and the interstitial fluid (Figure 1).[28] The low π_g prevents excessive extravasation of plasma into the interstitium.

The EGL is a fragile structure and is disrupted by inflammatory cytokines, particularly tumor necrosis factor α,[32,33] surgical trauma, ischemia–reperfusion, and hypervolemia,[34-37] leading to increased endothelial permeability and potentially pulmonary edema observed in ALI. In a mouse model, full restoration of the EGL after an inflammatory insult requires 5 to 7 days.[38] Of note, hypervolemia damages the EGL through 2 mechanisms. First, it induces the release of atrial natriuretic peptide, which directly causes shedding of the EGL.[36] Second, if hypervolemia is caused by crystalloids, the dilution of plasma protein concentration hinders the attachment of these proteins to the EGL and thereby the formation of a tight meshwork.

Several strategies to preserve the EGL were proposed based on evidence from animal experiments and can be classified as conservative and pharmacologic. The simplest conservative measure is to avoid hypervolemia. Other potential pharmacologic therapies include albumin infusion,[39] hydrocortisone,[32,40] direct inhibitors of inflammatory cytokines (eg, etanercept, tumor necrosis factor α inhibitor),[41] and antithrombin III.[37] The potential mechanisms of these agents are summarized in Table 4. Recent studies have reported a decrease in the release of inflammatory cytokines during thoracic surgery with one-lung ventilation using sevoflurane compared with total intravenous anesthesia.[42,43] Interestingly, in an

Table 2. Incidence, Risk Factors, and Mortality Rate of Acute Lung Injury After Pulmonary Resection and Esophagectomy.

Authors (Publication Year)	Design	Procedure	Total Patients	Incidence (%)	Risk Factors	Mortality (%)
Blank et al (2011)[10]	Retrospective	Pneumonectomy	129	7	Blood product transfusion	N/A
Marret et al (2010)[11]	Retrospective	Pneumonectomy	129	7	ASA class >2, fluid administration	33
Fernández-Pérez et al (2006)[22]	Retrospective	Pneumonectomy	170	9	Intraoperative tidal volume, preoperative FVC, fluid administration	40
Algar et al (2003)[9]	Retrospective	Pneumonectomy	242	2.5	Prolonged anesthetic time, ppo-FEV$_1$, heart disease, no chest physiotherapy, COPD	N/A
Parquin et al (1996)[12]	Prospective	Pneumonectomy	146	15	Prior radiotherapy, perfusion of remaining lung <55%, fluid administration	9.1
Turnage and Lunn (1993)[7]	Retrospective	Pneumonectomy	806	2.6	Right pneumonectomy	100
Verheijen-Breemhaar et al (1988)[13]	Retrospective	Pneumonectomy	243	4	Right pneumonectomy, repeat thoracotomy, fluid administration	N/A
Sen et al (2010)[15]	Retrospective	Pulmonary resection	143	7.5	Alcohol abuse, ASA score, resection type, FFP transfusion	27.3
Alam et al (2007)[14]	Retrospective case control	Pulmonary resection	1428	3.1	Fluid administration, decreased postoperative predicted lung function	25
Watanabe et al (2004)[18]	Retrospective	Pulmonary resection	3270	N/A	N/A	36
Dulu et al (2006)[16]	Retrospective	Pulmonary resection	2039	2.5	N/A	40
Licker et al (2003)[17]	Prospective	Pulmonary resection	879	3.1	High ventilatory pressure, fluid administration, pneumonectomy, alcohol abuse	26
Ruffini et al (2001)[20]	Retrospective	Pulmonary resection	1221	2.2	N/A	52
Kutlu et al (2000)[19]	Retrospective	Pulmonary resection	1139	3.9	N/A	64.4
Waller et al (1993)[21]	Retrospective	Pulmonary resection	205	4.4	N/A	56

Abbreviations: ASA, American Society of Anesthesiologists; FVC, forced vital capacity; ppo, predicted postoperative; FEV$_1$, forced expiratory volume in 1 second; COPD, chronic obstructive pulmonary disease; FFP, fresh frozen plasma.

Table 3. Fluid Administration in Patients With or Without Acute Lung Injury After Pulmonary Resection and Esophagectomy.

Authors (Publication Year)	Procedure	Timing	Acute Lung Injury	No Acute Lung Injury	P
Marret et al (2010)[11]	Pneumonectomy	Intraoperative infusion	3800 mL	2500 mL	<.0001
Fernández-Pérez et al (2006)[22]	Pneumonectomy	Intraoperative infusion	2200 mL	1300 mL	.001
Parquin et al (1996)[12]	Pneumonectomy	Intraoperative infusion	≥2000 mL	<2000 mL	<.01
Zeldin et al (1984)[3]	Pneumonectomy	24 hours postoperative infusion	37 mL/kg	27 mL/kg	<.05
Alam et al (2007)[14]	Pulmonary resection	Intraoperative and 12 hours postoperative infusion	2775 mL	2500 mL	<.05
Licker et al (2003)[17]	Pulmonary resection	Intraoperative infusion	9.1 mL/kg/h	7.2 mL/kg/h	.005
		Cumulated intraoperative and 24 hours postoperative infusion	2.6 mL/kg/h	2.0 mL/kg/h	.003
		Fluid balance 24 hours postoperation	2000 mL	1520 mL	.026
Casado et al (2010)[6]	Esophagectomy	Intraoperative fluid balance	5415 mL	4174 mL	.01
		Fluid balance 5 days postoperation	7873 mL	5928 mL	.03
Tandon et al (2001)[23]	Esophagectomy	Intraoperative infusion	5000 mL	4400 mL	<.027

animal model of ischemia–reperfusion injury, less destruction of the EGL is observed with sevoflurane compared with total intravenous anesthesia.[44] Although current evidence is limited, volatile anesthetics may be preferred over its intravenous counterpart during thoracic surgery.
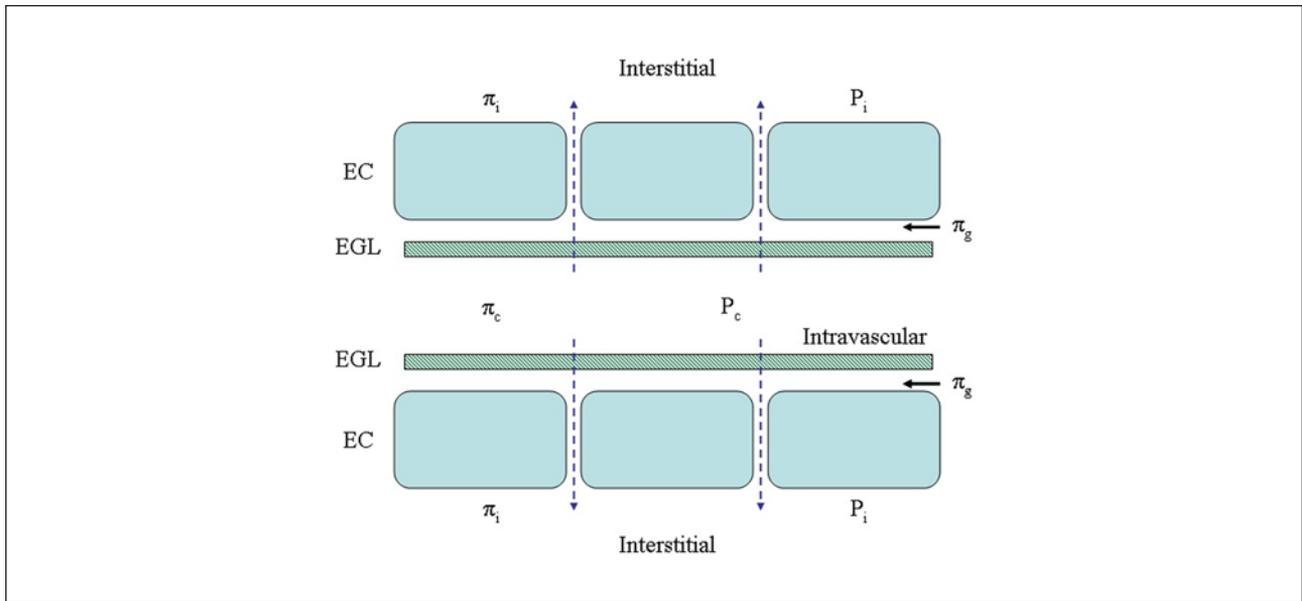
Figure 1. The glycocalyx model for fluid exchange between the intravascular and interstial space. The various components of the glycocalyx model and revised Starling's forces are shown. In steady state, net filtration into the interstitium occurs and is subsequently removed by the lymphatic system.

Abbreviations: EC = endothelial cell; EGL = endothelial glycocalyx layer; π_c = capillary oncotic pressure; π_i = interstitial oncotic pressure; π_g = oncotic pressure in subglycocalyx space; P_c = capillary hydrostatic pressure; P_i = interstitial hydrostatic pressure.

Table 4. Pharmacologic Agents That Protect the Endothelial Glycocalyx Layer (EGL) and Their Proposed Mechanisms of Action.

Pharmacologic Agents	Mechanisms of Action
Albumin	Maintain stability of the EGL through electrostatic binding and formation of a tight meshwork
Hydocortisone	Stabilize mast cells and their release of histamine and cytokines
Etanercept	Direct inhibition of tumor necrosis factor α
Antithrombin III	Inhibit serine proteases and their proteolytic damage to the EGL

The Third Space

Classically, the third space represents a fluid compartment functionally and anatomically separated from the interstitial space.[45] It is not involved in the dynamic exchange of fluid between the intravascular space and the interstitium. It was first described in 1961 by the use of sulfate tracer techniques to measure extracellular fluid volume in patients undergoing major abdominal surgery.[46] The location of the third space remained unidentified to date but was speculated to be in the gastrointestinal tract or traumatized tissues. The third space concept leads to a culture of aggressive fluid repletion for loss into this hypothetical compartment in major gastrointestinal surgery, including esophagectomy. Recently, Brandstrup et al[47] performed a systematic review on trials measuring extracellular fluid volume during surgery or hemorrhage. It was concluded that the evidence supporting the existence of a third space is weak and is likely due to flawed methodology.

As mentioned above, in the setting of major surgery and its resultant inflammatory response, an increase in endothelial permability ensues.[48] The irrational fluid replacement for third space loss may lead to the development of interstitial pulmonary edema.

Probable Causes: Fluid Overload, Lung Lymphatics, and Pulmonary Endothelial Damage

It has been clearly demonstrated that excessive crystalloid infusion in anesthetized dogs in the lateral position rapidly causes fluid accumulation in the dependent lung and hypoxemia.[49] As mentioned earlier, multiple studies report an association between ALI and fluid administration. However, fluid overload was not the sole culprit as ALI still occurred despite severe fluid restriction (<700 mL positive balance 24 hours postoperation).[2]

Lung lymphatics are responsible for the drainage of capillary filtrate in the interstitium. Pulmonary edema results when the amount of fluid filtered by the pulmonary capillaries exceeds the ability of the lung lymphatics to transport it out of the thorax. Surgical trauma during pulmonary resection surgery may impair lymphatic drainage. It has been shown that the lymphatic drainage of the right lung is mainly ipsilateral (>90%) but that of the left lung is contralateral (>55%).[50] Thus, a left pneumonectomy has little effect on the lymphatics of the remaining right lung. In contrast, a right pneumonectomy could potentially decrease the lymphatic drainage of the left lung by half, resulting in decreased clearance of capillary filtrate in the interstitium and pulmonary edema. In fact, right pnemonectomy has been associated with the development of ALI.[7,13]

Endothelial damage is implicated in ALI after pulmonary resection surgery because of findings of low pulmonary artery occlusion pressure, high levels of protein in edema fluid and ARDS pathology. Potential mechanisms of endothelial damage include volume-induced lung injury and ischemia–reperfusion injury.[51,52] These processes induce the release of inflammatory cytokines and increase in pulmonary pressures.[28,53,54] Circulating neutrophils are activated and migrate to the alveolar airspace, releasing proteolytic enzymes and reactive oxygen species.[55] Oxygen toxicity may also contribute to endothelial injury through the formation of reactive oxygen species.[56] The adverse effect of oxygen to the lungs depends on its concentration and duration of exposure.[57,58] Healthy lungs are usually able to sustain an exposure of 100% oxygen for less than approximately 12 hours before demonstrating capillary leak.[59,60] Nevertheless, in thoracic surgical patients presenting with multiple other risk factors for ALI, their tolerance for high oxygen exposure may be significantly reduced.

Possible Causes: Volume-Induced Lung Injury and Right Ventricular Dysfunction

Alveolar overdistension during positive pressure ventilation causes alveolar stretch injury and endothelial damage of similar degree seen in excessive pulmonary vascular pressures.[54] Intraoperative tidal volume was shown to be a risk factor for ALI after pneumonectomy.[22] Patients who developed ALI were ventilated with larger tidal volume than those who did not (median, 8.3 vs 6.7 mL/kg predicted body weight; $P < .001$). Following a left pneumonectomy, the functional residual capacity (FRC) of the residual right lung increases approximately 35%.[61] The increase in FRC of the left lung after a right pneumonectomy may be even larger because of mediastinal shift to the right. If the same tidal volume during 2-lung ventilation is used during 1-lung ventilation, the large tidal volume combined with an increase in FRC could result in volume-induced lung injury.

Right ventricular (RV) dysfunction has been shown to peak on the second postoperative day in patients undergoing lobectomy and pneumonectomy.[62] Potential mechanisms leading to RV dysfunction include an increase in RV afterload and tachycardia.[63,64] RV dysfunction can lead to an increase of central venous pressure which inhibits lymphatic drainage of the lungs.[65]

Acute Kidney Injury After Pulmonary Resection Surgery

Avoiding fluid overload appears to be key in preventing ALI during pulmonary resection surgery and esophagectomy. However, a relatively restrictive perioperative fluid regimen may impair renal perfusion, resulting in acute kidney injury (AKI). The incidence of AKI after pulmonary resection surgery from 2 recent studies were 6.8% and 5.9%, respectively.[66,67] Patients who developed AKI suffered from prolonged hospital stay,[66] increased cardiopulmonary complications,[66,67] and increased mortality.[67]

Licker et al[67] identified 4 risk factors for AKI: American Society of Anesthesiologists classes 3 and 4, forced expiratory volume in 1 second, the use of vasopressor, and duration of anesthesia. Similar quantities of crystalloids were administered perioperatively in patients with and without AKI (4.8 vs 4.9 mL/kg/h intraoperatively; 1.1 vs 1.1 mL/kg/h postoperative day 1). Multiple different risk factors for AKI were reported by Ishikawa et al,[66] including hypertension, peripheral vascular disease, preexisting renal dysfunction, preoperative use angiotensin II receptor blockers, intraoperative use of colloids, and open procedures. The average amount of total intraoperative crystalloid received by patients with and without AKI was similar at 1450 and 1276 mL, respectively. There is a significantly higher percentage of patients with AKI who received colloids intraoperatively (19% vs 6%, $P < .001$). However, there is no significant difference in the volume of colloids administered to the 2 groups of patients. The association between colloid solutions and postoperative AKI is not fully understood. Potential mechanisms have been proposed but further studies are required to examine this issue.[68]

In patients with preexisting kidney dysfunction undergoing pulmonary resection surgery and esophagectomy, several anesthetic goals should be considered to prevent acute on chronic kidney injury while adopting a restrictive fluid regimen: First, avoid hypotension secondary to excessive anesthesia. Second, maintain adequate perfusion pressure through the judicious use of vasopressors. Last, consider invasive hemodynamic monitoring to guide management.

To conclude, it appears that a restrictive fluid regimen during pulmonary resection surgery is not associated with postoperative AKI. The maintenance of adequate intravascular volume and perfusion pressure remain the primary goals to avoid AKI. The measurement of perfusion of individual organs is not easily obtained in routine anesthetic practice. However, emerging evidence regarding the use of minimally invasive device to evaluate hemodynamic status may allow anesthesiologists to optimize fluid therapy during thoracic procedures.

Goal-Directed Fluid Therapy During Pulmonary Resection Surgery and Esophagectomy

Goal-directed fluid therapy (GDFT) involves the monitoring of hemodynamic parameters and rational fluid administration based on the information obtained to optimize tissue perfusion. Many of these hemodynamic parameters measure fluid responsiveness, defined as a significant increase in cardiac output with fluid loading. Predicting fluid responsiveness allows maximizing cardiac output and perfusion while avoiding unnecessary fluid administration. Mortality and morbidity benefits of GDFT has first been reported in high risk surgical patients by Shoemaker et al[69] Subsequently, GDFT was also shown to promote splanchnic circulation and reduce gastrointestinal complications in patients undergoing major surgery.[70,71] GDFT may play an important role in pulmonary resection surgery and esophagectomy because of the detrimental effects of fluid overload associated with these procedures.

In pulmonary resection surgery, the use of transesophageal Doppler and pulse pressure (PPV)/stroke volume variation (SVV) monitoring was examined in several studies.[72-76] Unlike conventional thermodilution technique using the pulmonary artery catheter, these devices are minimally invasive and allow continuous hemodynamic monitoring. Transesophageal Doppler has been validated for cardiac output measurement against reference techniques such as thermodilution and the Fick method.[77,78] Recently, this device was demonstrated to be able to detect low cardiac output during lung surgery when heart rate and blood pressure remained unchanged.[73] PPV/SVV was also studied during one-lung ventilation in thoracic surgery. Both techniques use the heart–lung interaction during mechanical ventilation to assess fluid responsiveness. PPV is derived from analysis of the arterial waveform and SVV is derived from pulse contour analysis. At a tidal volume of 8 to 10 mL/kg during 2-lung ventilation, PPV $\geq 13\%$ and SVV $\geq 12\%$ both highly correlate with fluid responsiveness.[79]

It is unclear whether this correlation remains the same during one-lung ventilation at protective tidal volumes (6 mL/kg). It was suggested that during one-lung ventilation, the amount of shunted blood through the nonventilated lung does not contribute to the generation of PPV/SVV and will result in a lower threshold value.[74] In fact, at tidal volumes of 6 mL/kg, PPV $\geq 6\%$ and SVV $\geq 8\%$ yield moderate correlation with fluid responsiveness.[74,76]

In esophagectomy, SVV monitoring was shown to predict perioperative fluid responsiveness and hypotension.[80] In addition to intraoperative monitoring, there are attempts to use minimally invasive technology for postoperative monitoring as severe hemodynamic changes are common after esophagectomy. Monitoring of extravascular lung water by the PiCCO (pulse contour cardiac output) system, a single transpulmonary thermodilution technique, is validated against gravimetric measurement, the reference technique.[81] The PiCCO system may offer prognostic, diagnostic, and therapeutic information in patients at risk of developing ALI. It has been shown that an increase in extravascular lung water correlates with worsened respiratory function and higher risk for postoperative pulmonary complications in patients undergoing esophagectomy.[82,83] Of note, the use of the PiCCO system in pulmonary resection surgery is controversial as the accuracy in estimating extravascular lung water is affected by a decrease pulmonary blood volume.[81]

Applying minimally invasive devices to monitor hemodynamic status allows a rational approach to perioperative fluid therapy. Transesophageal Doppler, PPV/SVV monitoring, and the PiCCO system appear to be promising technology. Further research regarding the use of these monitoring devices and their effects on patient outcome in pulmonary resection surgery and esophagectomy is required.

Fluid Management Principles for Pulmonary Resection Surgery and Esophagectomy

The following principles of fluid management are suggested for all pulmonary and esophageal resection surgery. For an average adult

1. Total positive fluid balance in the first 24 hours postoperatively should not exceed 20 mL/kg.
2. Crystalloid administration should be limited to <2 L intraoperatively and <3 L in the first 24 hours postoperatively.
3. Colloids should only be used to replace an equivalent volume of blood loss if blood is not required (maintain Hb > 80 g). Maximum colloid volume = 1 L for an adult.
4. There is no third space loss.
5. Urine output >0.5 mL/kg/h is unnecessary in the early postoperative period, unless the patient is at high risk of developing acute kidney injury.

6. If increased tissue perfusion is needed postoperatively, appropriate invasive hemodynamic monitoring should be initiated to guide treatment with vaspressors, inotropes, or fluid administration.

Additional anesthetic management suggestions that may decrease the risk of ALI after thoracic procedures include using lung protective ventilation strategies (low tidal volume and application of peak end-expiratory pressure) and avoiding increase in pulmonary vascular pressures (pain, hypercapnia, hypoxemia, and hypothermia).

Conclusion

Acute lung injury after pulmonary resection and esophagectomy is a severe complication associated with high mortality rate. Excessive perioperative fluid administration is an important risk factor for the development of this complication. The pathogenesis of ALI appears to be multifactorial. Possible mechanisms include fluid overload, trauma to the lymphatics, endothelium and EGL. A restrictive approach of fluid therapy is advocated for pulmonary resection and esophagectomy and there is currently no evidence that minimizing fluid administration compromises distal organ perfusion such as the kidneys. Minimally invasive hemodynamic monitors may play a role in optimizing fluid therapy in patients undergoing thoracic procedures. Further research regarding the prevention, diagnosis and treatment of ALI after pulmonary resection and esophagectomy is required.

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