

Intraoperative Hypotension in Thoracic Surgery: Limits to the Transportability of Evidence from Non-Thoracic Surgery

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Abstract

Hypotension during non-cardiac surgery is associated with myocardial injury, acute kidney injury, cerebral infarction and death; therefore, different mean arterial pressure thresholds have been set for various surgeries. We will determine why the results and treatment plans of thoracic surgery cannot be applied to other diseases of the chest easily, especially in cases of one-lung ventilation (OLV). Thoracic anaesthesia is a type of intrapulmonary shunt that causes hypoxic pulmonary vasoconstriction (HPV) and redistribution of pulmonary blood flow. Lateral positioning, conditions of the thoracic chamber and lung artery obstruction all increase the afterload of the right ventricle (RV), and the time to administer fluid is short. The mean arterial pressure should be less than 60 - 65 mmHg; if it is still this low or even lower, further investigation and treatment will be necessary. Monitor blood pressure, right ventricular (RV) function, pulmonary vascular resistance (PVR), oxygenation, airway pressure, haemorrhage and fluid responsiveness. Although the non-thoracic data have biological significance, random studies in the chest should be carried out before setting general blood pressure goals or haemodynamic models.

Keywords

Intraoperative Hypotension, Thoracic Surgery, One-Lung Ventilation, Right Ventricular Function, Pulmonary Vascular Resistance, Vasopressors, Fluid Therapy, Lung Resection

1. Introduction

In non-cardiac surgery, healthcare providers are now aware that intraoperative

hypotension can be managed and is not necessarily a result of anaesthesia. Prospective studies and guidelines have shown that a mean arterial pressure (MAP) of 60 - 70 mmHg or lower is associated with myocardial injury, acute kidney injury (AKI) and death, and there is an increased risk in cases of prolonged or severe hypotension [1] [2]. Not all of the above are goals for blood pressure after a procedure.

Most of the high-quality studies are about abdominal, orthopedic, vascular and gynecological diseases. Most of these patients are in the supine position, are on two-lung ventilation, and have relatively normal lungs and hearts. Physiological Changes after Thoracotomy: OLV will deliberately create a large shunt, the patient will be frequently in a lateral position, the chest may be open, and the pulmonary artery may be clamped or compressed.

This article is a narrative review rather than a systematic review. The literature base was assembled through a focused, non-systematic search and citation chaining from key trials, reviews, consensus statements, and guidelines, with priority given to thoracic-specific evidence addressing intraoperative hypotension, OLV, pulmonary vascular physiology, right-ventricular function, ventilation, and fluid therapy. No prespecified protocol, formal risk-of-bias assessment, or meta-analysis was undertaken. Non-thoracic evidence is used primarily to define established associations and to examine where biological and treatment-effect transportability to thoracic surgery may be limited.

2. What Non-Thoracic Evidence Has Established

According to the Perioperative Quality Initiative, the intraoperative mean arterial pressure (MAP) of 60 - 70 mmHg or lower is associated with myocardial injury, acute kidney injury (AKI) and death; the extent of injury and how long it lasts determine the seriousness [1]. All the research papers also showed that various criteria for hypotension are associated with damage to the kidneys, heart, brain and other organs after surgery [2].

The INPRESS randomised controlled trial has shown that individualised blood pressure control in the first few days after a heart attack can reduce the risk of organ damage [3]. Among the 7490 patients with non-cardiac surgery in POISE-3 who are taking continuous antihypertensive drugs, the two groups were divided into hypotensive-avoidance (target MAP $\geq$ 80 mmHg) and hypertension-avoidance (target MAP $\geq$ 60 mmHg), and the proportion of major vascular complications was the same in both groups [4]. OPTIMISE also did not significantly reduce the three main problems of cardiac-output-directed care in major gastrointestinal surgery [5]. The above studies have not dealt with OLV, problems in the pulmonary circulation, right ventricular failure or fluid obstruction after lung resection.

3. Why Thoracic Surgery Is Physiologically Different

During OLV, the non-ventilated lung continues to receive some blood flow, creating a right-to-left shunt. Hypoxic pulmonary vasoconstriction (HPV) and grav-

ity redirect flow toward the dependent ventilated lung. Some physiological reports describe pulmonary artery pressure increases on the order of 25% - 35% and pulmonary vascular resistance (PVR) increases of roughly 20% - 50% during OLV [6] [7]. These figures were obtained under particular anesthetic, ventilatory, positional and surgical conditions, and are best read as illustrative rather than as fixed responses. The magnitude is patient-specific: it varies with baseline pulmonary vascular reserve and the HPV response, with lung volume and gas exchange, and with when during OLV the measurement is made [6]-[9].

The RV is vulnerable to acute afterload increases from HPV, increased dependent-lung flow, and alveolar overdistension. Hypercapnia and acidosis add further stress, as do pulmonary artery clamping, surgical retraction, and pre-existing pulmonary hypertension or RV dysfunction [7] [10] [11]. Acute RV failure can reduce left-ventricular (LV) preload and systemic cardiac output. A vasoconstrictor may raise MAP while failing to restore RV output if the dominant problem is pulmonary vascular or mechanical.

Lateral position, open-chest conditions, mediastinal shift and carbon dioxide accumulation can all increase venous return and affect heart function. Central venous pressure is generally relatively high due to increased intrathoracic pressure, it does not reflect preload accurately, and therefore, the functional parameters obtained in OLV, open-chest surgery, arrhythmias, spontaneous breathing or reduced tidal volumes may be inaccurate.

Oxygenation in OLV is related to the mixed venous oxygen content. A reduction in cardiac output will decrease oxygenation in the arterial blood of the obligatory shunt, and raising the cardiac output is not an all-encompassing remedy [12]. Thoracic anaesthesia has many contradictory factors: Fluids can increase perfusion but cause pulmonary oedema; high airway pressure can increase recruitment but reduce venous return and raise PVR; and vasoconstriction can increase MAP but decrease RV output.

4. Why Treatment Evidence Has Limited Transportability

Fluid is a very clear illustration. A lung that has been collapsed and directly manipulated, then re-expanded, cannot tolerate volumes that are considered excessive in an abdominal case, and these people frequently suffer from parenchymal injury and thus have a poor prognosis. Ischaemia-reperfusion injury and disruption of the endothelial barrier are the reasons. Recommendations from ERAS/ESTS are also in line with this; they recommend euvolemia, avoid fluid overload, and only use vasopressors for hypotension due to vasodilation. It does not have a general aim-oriented haemodynamic plan for thoracic diseases [13].

The reasons for choosing vasoconstrictors are also the same. The first is to reduce the vasodilation caused by anaesthesia with noradrenaline. Phenylephrine will raise blood pressure, but it can also cause a decrease in heart rate and stroke volume, thus worsening the symptoms of right ventricular failure. If hypotension is caused by acute right ventricular collapse, then to reduce the right ventricular af-

terload and improve contractility, we should correct hypoxemia, hypercapnia and acidosis, reduce airway pressure, and investigate pulmonary vascular obstruction. Inotrope and inhaled pulmonary vasodilator drugs are occasionally employed.

Ventilator Parameters will also be mentioned. PROTHOR did not decrease the rate of postoperative pulmonary morbidity by continuously increasing PEEP and combining it with OLV; rather, it lowered the intraoperative blood pressure in 37.3% of patients to 14.3% by reducing PEEP and not regularly recruiting [14]. Therefore, the first PEEP is chosen in advance to avoid affecting blood pressure without improving lung function. We would adjust it based on compliance and driving pressure, as well as oxygenation, and also according to pressure and RV reaction.

Epidural analgesia will cause a prolonged vasoconstriction of the sympathetic nerves. In a randomised trial of open thoracic surgery, the subjects under epidural anaesthesia needed more colloid and more phenylephrine to maintain the desired oxygen delivery ratio than those under paravertebral blockade, but the analgesia was the same [15]. The results are small but significant: after epidural placement, there is a problem with blood vessel tone and a large bolus of fluid has been added to someone who did not need it before. **Table 1** shows the kinds of restrictions on the direct submission of non-thoracic evidence.

Table 1. Research directions for limitations in the immediate application of non-thoracic hypotension data in thoracic surgery.

Domain	Common non-thoracic assumption	Thoracic-specific modifier
Ventilation	Two-lung ventilation with relatively stable pulmonary load.	OLV creates shunt, redistributes pulmonary blood flow, and can increase PVR/RV afterload.
Position/chest	Usually supine with a closed chest.	Lateral position, open chest, mediastinal shift, and CO ₂ insufflation can alter venous return and ventricular interaction.
Fluid therapy	Fluid bolus is a common response to low MAP.	Post-resection lungs are vulnerable to edema; fluid must be selective, reassessed, and distinguished from vasodilatory hypotension.
Pressure target	MAP is used as a practical surrogate for perfusion.	MAP may not reflect flow, RV output, venous congestion, or oxygen delivery during rapid thoracic physiological changes.

5. Thoracic-Specific Outcome Evidence

Direct thoracic signs are infrequent but serious. Among the 676 patients with lung cancer surgery, intraoperative hypotension was defined as a drop in systolic blood

pressure to less than 100 mmHg for more than 5 minutes, as it is likely to cause long-term survival problems. Total follow-up for all subjects was 515. The five-year mortality rate in the group with hypotension was 41.2%, and it was also higher than that for those with only hypertension (HR: 1.736, CI: 1.218 - 2.475; $P = 0.002$) [16].

The variables in this model are the attributes of the patient (age, BMI, sex, smoking history, hypertension) and all the interventions before and after treatment (preoperative chemotherapy, perioperative drugs, blood transfusion, and postoperative chemoradiotherapy). Surgical and histopathological factors included the type of surgery, evaluation of mediastinal lymph nodes, tumour histology and grade, pathological staging and postoperative complications [16]. However, this paper has not identified the exact causes and effects of high blood pressure, nor has it shown that strict control can extend life. Data-driven threshold identification and the observational nature of this study have left some residual confounding; for example, changes in the complexity of surgery, intraoperative bleeding and initial cardiopulmonary reserve may affect both hypotension and long-term cancer outcomes.

6. A Practical Framework for Managing Hypotension during Thoracic Surgery

Confirm the accuracy of the pressure reading, check the quality and damping of the waveform, and compare the catheter position with the cuff. Level and zero the transducer before taking the measurement. The Data will be verified, and then the surgery will be restricted. Induction, Assessment of Anesthetic Vasodilation, Hypovolaemia, Neuraxial Anaesthesia and Occult Right Ventricular Dysfunction. Shortly after OLV, there is decreased venous return, increased PVR, hypoxemia, hypercapnia or double-lumen tube malposition. Hilum dissection or pulmonary artery occlusion will cause an abrupt increase in right ventricular afterload, bleeding or mechanical obstruction. Recruitment, auto-PEEP, tension pneumothorax and re-expansion injury at chest closure and lung re-inflation are all related factors.

MAP gives only one direction for the question. Heart rate and rhythm, end-tidal CO_2 , lactate and urine excretion all indicate changes in the body. Parameters of arterial wave shape and echocardiography can distinguish between flow obstruction and vasodilation. Bleeding, oxygenation, airway pressure and driving pressure are related to surgery, the lungs and breathing. Decrease excessive anesthesia or neuraxial vasodilation and confirm airway-device location. Correct Gas-Exchange Disorders. If there is suspected overdistension, decrease the high PEEP or tidal volume. Only give a small reassessment fluid bolus if there is bleeding, preload deficiency or fluid unresponsiveness. Norepinephrine is a strong vasodilator, so if there is right ventricular failure, we should add inotropes or pulmonary vasodilators. Now, most of the reasons for high perioperative pressure have been identified [17].

If MAP is less than 60 - 65 mmHg, be prepared to quickly address any signs of

hypotension or hypoperfusion. Given that this patient has low blood pressure and a history of chronic hypertension, the purpose here is to treat cerebral and renal diseases. Pulmonary hypertension, right ventricular function and cardiac output are also factors that determine the aim of surgery [17]. The Mechanism-driven response summary is shown in **Table 2**.

Table 2. Mechanism-based response to hypotension in thoracic surgery.

Likely phenotype	Clues	Priority actions
Vasodilation	Recent induction or epidural; preserved RV/LV function.	Reduce vasodilatory dose; titrate norepinephrine; avoid reflexive fluid loading.
Preload deficiency/bleeding	Blood loss, low filling, plausible fluid responsiveness, rising lactate.	Control bleeding; use small reassessed fluid or blood aliquots.
RV afterload crisis	High airway pressure, hypoxemia, hypercapnia, rising pulmonary pressure/PVR, RV dilation.	Reduce overdistension; correct gas exchange/acidosis; consider inotropy or inhaled pulmonary vasodilator; resume two-lung ventilation if feasible.
Mechanical obstruction	Abrupt onset during retraction, clamping, insufflation, or closure.	Ask the surgeon to pause; exclude tension pneumothorax, cardiac/pulmonary-vessel compression, obstruction, and airway-device problems.

7. Definitions and Phenotypes of Intraoperative Hypotension

Most of the confusion in this literature is due to treating intraoperative hypotension as a single exposure and having only one biological explanation. It is not the case. Depth, length, time, fluctuation and reason are all different from one another. If the same threshold is used in both cases, the MAP after intubation will be 62 mmHg for a short time and then drop to 48 mmHg after pulmonary artery clamping for 20 minutes. The former is usually characterised by vasodilation and reduced blood flow. The latter may be acute RV collapse, haemorrhage or mechanical obstruction. Most of the non-thoracic studies have combined the two events into one outcome, so their results cannot be applied to thoracic surgery.

Absolute Benchmarks are impractical and insufficient. For a person with chronic hypertension, the decrease from the baseline may be more serious than a particular value, and a MAP of 65 mmHg that a young person can tolerate may be too low for someone with cerebral or renal diseases. Clinical studies have shown that there are considerable differences in which threshold is selected, how depth, duration and area under the curve are included in the model, and what kind of link to organ damage occurs [18] [19]. The general recommendations at present are

not specific, so practitioners should not aim for a low MAP but rather set individualised goals according to the circumstances and perfusion of the patient [17].

Cardiac surgery is the second axis. The pressure-flow dynamics are very unstable, and many surgical factors affect them as frequently as changes in anaesthetic conditions do, such as the start of mechanical ventilation, traction, hilar ligation, changes in PEEP, etc. Each can set its own preload or RV afterload, and there will be no large variation in the recorded pressure; therefore, a pressure-based end point will be unreliable in this case. A reasonable definition of thoracic hypoxemia should include MAP, as well as cardiac output or stroke volume (if available), oxygenation and airway pressure, vasopressor volume, and the stage of surgery at the time of the episode.

Another kind of study is a time-distributed, mechanism-labelled exposure that is the sum of minutes below a personal threshold, and by phenotype it is divided into vasodilatory, hypovolemic, RV-afterload or mechanical. The above labels can be employed to identify causes, and it is possible to learn how different groups respond to treatment without neglecting this effect.

8. Induction of Anesthesia and the Transition from Two-Lung to One-Lung Ventilation

Induction is a dangerous and insufficiently studied period in thoracic anaesthesia. People who have undergone lung resection are often in poor health, have reduced functional residual capacity, increased intra-pulmonary pressure, impaired left-ventricular relaxation, decreased right ventricular volume, and so on. Induction will reduce sympathetic activity and decrease venous return; thus, positive-pressure ventilation will be used to keep breathing and increase intrathoracic pressure. The drop in mean arterial pressure (MAP) will be greater than the one specified.

To switch to OLV, pulmonary blood flow needs to be redirected to the dependent lung, and the degree of this change cannot be known from a single anaesthesia record. Gravity and the surgical side determine its position; the structure of the pulmonary artery and the HPV reaction change this; how severe the collapse, emphysema and vascular disease are will affect how much residual circulation can be maintained [8] [9]. The first few minutes of OLV are used to assess the function of the lungs. All the following conditions need to be ruled out for reduced pulmonary blood flow or impaired right ventricular output before systemic vasodilation is performed: a drop in end-tidal CO₂, an increase in the arterial-to-end-tidal gradient, high airway pressure, or a new need for vasopressors.

A displaced double-lumen tube can obtain much of this picture and is suitable for thoracic work. Ventilation-lung obstruction increases the pressure in the airways and causes hypoxia, hypercapnia and an increase in pulmonary vascular resistance. If not isolated, the shunt will be low and the ventilatory pressure will be high. Place the guide wire, then adjust it again to regulate blood supply and ensure the airway is open, and finally conduct a fibre-optic examination.

We will consider the transition to OLV an intentional physiological treatment,

not merely an item on a list. First, the group will agree on the upper limit of PEEP, oxygenation goals, an emergency plan for low oxygen levels and when to switch back to two-lung ventilation. Given the changes in the area of thoracic surgery, it is more appropriate to proactively raise MAP rather than waiting for it to drop significantly.

9. Hypoxic Pulmonary Vasoconstriction and Pulmonary Vascular Reserve

HPV suppresses Shunt in OLV. Pulmonary arteries in hypoxic alveoli narrow in milliseconds to divert blood to oxygenated regions. The two reactions are a rapid one that appears early and a slower one that becomes more prominent after prolonged hypoxia. Its intensity is related to gas concentration and lung volume, and it can be influenced by cardiac output, vasoactive drugs, inhaled anesthetics, other existing respiratory diseases, etc. [8] [9].

HPV will increase oxygenation, but it will also raise PVR and RV afterload. It is the pulmonary vascular reserve. Healthy Pulmonary Arteries can reduce increased dependent-lung flow. Pulmonary hypertension, emphysema, fibrosis, prior pulmonary embolism or a small predicted postoperative lung volume may all be present. Thus, OLV-related hypoxia can lead to very different systemic blood pressure responses [11].

Airway Pressure and HPV Interaction. Excessive overdistension can increase PVR, and derecruitment may also have this effect; the connection is U-shaped, meaning that both too little and too much PEEP are harmful. Hypercapnia and Acidosis Increase Pulmonary Vasoconstriction. An increase in PVR reduces RV output and therefore decreases oxygen supply; thus, acidosis further increases PVR.

Pulmonary vascular assessment will be added to all guidelines for thoracic hypotension. Dimensions and function of the RV, septal anatomy, and estimated pulmonary pressures are known. Central venous pressure is only a relative measurement; thus, small changes in the load of the airway or oxygenation may be more indicative of physiological responses. A pressure goal that does not account for the pulmonary vascular capacity will only deal with the results and not the root cause.

10. Right-Ventricular-Pulmonary-Artery Coupling

RV-pulmonary-artery coupling is the capacity of the RV to produce flow against afterload. At the time of OLV and lung removal, there is a sudden increase in afterload due to HPV, blood redistribution, pulmonary artery occlusion and surgical compression; thus, the mean airway pressure and hypercapnic acidosis work together [7] [10]. The RV first increases contractility, but then dilation and tricuspid regurgitation occur after the contractile reserve has been exceeded. Leftward Septal Displacement Decreases LV Filling and Systemic Output.

It is not the usual form of vasoplegic hypotension that occurs in abdominal sur-

gery. A person with vasoplegia will only be affected by norepinephrine. A patient with an RV-afterload crisis in the middle of this will have an elevated blood pressure because of increased systemic vascular resistance. At present, the only good interventions are ventilatory and surgical; that is to say, reducing PEEP, correcting hypercapnia and increasing oxygenation, or requesting that traction be stopped and two-lung ventilation be resumed, but not vasoactive agents. Inotropes or inhaled pulmonary vasodilators are needed for some people.

Preoperative risk assessment should consider more than just left ventricular ejection fraction. Cardiac imaging can be used to detect dilatation or dysfunction of the right ventricle, increased pulmonary pressure, abnormal septal motion; pulmonary hypertension and reduced right ventricular reserve are signs that a person will have a lower tolerance for sudden changes in afterload [7] [11]. Cardiopulmonary exercise testing and diffusion capacity can offer information about the reserve of certain lung resection candidates.

Focused echocardiography is carried out in the operating room to determine whether there is sudden or prolonged hypotension and to find the cause, such as OLV or hilar dissection. The goal is not to conduct a full-scale echocardiography; rather, we want to know: Is there right ventricular dilation? Is LV filling decreased? Is there a septum? Is there systolic function? Is the heart affected by the surgery? Therefore, there will be no need to replace the fluid or vasoconstrict.

11. Ventilator-Induced Hemodynamic Effects

Fluctuations in Tidal Volume, PEEP, recruitment, plateau pressure and driving pressure of OLV can affect both lung function and cardiovascular conditions. An increase in tidal volume or PEEP will raise the intrathoracic pressure, decrease venous return, and may increase the afterload of RV if the alveolar pressure exceeds the limit. A small tidal volume can reduce airway pressure but will increase atelectasis, shunt and HPV. Most of the perioperative data show that the tidal volume is small, but the best PEEP strategy is still relatively situation-dependent [20].

Thoracic and abdominal examinations have come to the same conclusion. PROTHOR found that fixed high PEEP did not improve postoperative pulmonary function and was associated with more intraoperative hypotension than low PEEP [14]. PROVHILO had the same effect in open abdominal surgery; increasing PEEP and recruitment did not reduce pulmonary complications but increased circulatory depression and the need for vasoactive drugs [21]. Therefore, in the above research, we have been studying ventilatory gains and haemodynamic losses together.

Additional Chest X-rays will be taken. A way to reduce driving pressure reduced the risk of pulmonary complications in a randomised trial [22], and although iPROVE-OLV had good intraoperative respiratory dynamics, it did not improve this postoperative outcome [23]. A separate study on lung resection compared low tidal volume with PEEP and recruitment to one with high tidal volume and found that the incidence of acute respiratory distress syndrome (ARDS) and

postoperative pulmonary complications was the same [24]. Therefore, the improved mechanics are not thought to have any clinical benefit.

Mechanical energy includes tidal volume, airway pressure, respiratory rate and flow. Post-hoc analysis of iPROVE-OLV showed that personalised PEEP increases the total mechanical power slightly, and different parts of this power are associated with different kinds of pulmonary complications [25]. How it relates to RV function and to systemic hypotension during OLV has not been determined.

We will monitor all changes in PEEP and recruitment, oxygenation, compliance or driving pressure, arterial blood pressure and RV response. If oxygenation is good but MAP or RV performance drops after increasing PEEP, do not change it. Severe hypoxemia may need recruitment or brief two-lung ventilation instead of prolonged increase in airway pressure.

12. Fluid Restriction, Pulmonary Edema, and Renal Perfusion

Management of the Thoracic Space Shows the Limits of Traditional Surgery. After surgery, there is a reduced pulmonary vascular network, an organised lung, and an organised lymphatic system. OLV and ischaemia-reperfusion injury are also harmful to the vascular endothelial barrier. Severe pulmonary oedema increases the shunt fraction and results in respiratory failure, but it also decreases renal blood flow and worsens vasodilatory hypotension.

A group of 1442 people had an incidence of AKI of 5.1%, and although crystalloid caps below 3 mL/kg/h were not associated with AKI, hydroxyethyl starch increased the risk in susceptible individuals [26]. Subsequent systematic reviews and meta-analyses have found that the combined incidence of AKI after standard thoracic surgery is about 8%, and this rate is higher for more extensive operations and varies according to different clinical circumstances [27].

Abdominal data do not specify the thoracic part. Within RELIEF, the restrictive protocol did not reduce disability-free mortality and was associated with more AKI than the liberal one [28]. Thoracoscopic lobectomy: Both the goal-directed and the restrictive strategies had the same rate of postoperative pulmonary complications and AKI, but the restrictive group required additional norepinephrine [29]. Therefore, a weak crystalloid solution is chosen to avoid starch and is customised according to the person's condition.

The actual difference is between promptness and robustness. A patient may increase cardiac output after a bolus but still have too high pulmonary hydrostatic pressure. At the same time, although a person has reduced arterial pressure, their venous pressure is high and there is not a significant increase in blood volume. Minimal fluid stimulation, dynamic response assessment in cases that are technically feasible, directed echocardiography and multimodal perfusion patterns are preferred over repeated empirical boluses [30].

Blood loss is to be managed in an emergency. Late detection will need more vasopressor doses and will harm the local tissue. Under general anaesthesia, con-

trol blood loss, distinguish between bleeding due to surgery and vasodilation caused by anaesthesia, etc.

13. Monitoring and the Problem of Measurement Error

The Measurement Environment of the Thorax is very difficult to obtain. A non-invasive cuff can be placed on the dependent limb and the arterial probe at a different site on the body. Both the field and the instrumentation are sources of signal distortion; there are open-chest settings and electrical cautery on one side, and damping and catheter whip on the other, all at the same time fluctuations in arterial tone are occurring. The measured MAP of 58 mmHg may be true systemic hypotension, an artefact, or a real difference between central and peripheral pressures.

Regularly measure the blood pressure at different times and adjust the medicine according to the results. Randomized studies of non-thoracic procedures have found that continuous non-invasive monitoring can reduce the time-weighted proportion of MAPs below 65 mmHg, but it has not improved clinical outcomes [31]. Thoracic areas need to be monitored continuously because of frequent changes in OLV and surgery; at the same time, one must know exactly where the reference point of the transducer is.

Cardiac output monitoring is used in the event of prolonged hypotension or when the cause of hypotension is unknown, but the traditional devices have problems during OLV and open-chest surgery. Small tidal volume, vasoconstriction, arrhythmias and changes in arterial compliance can all affect the pulse-contour method. Focused echocardiography is generally better at showing the main reasons for right ventricular dysfunction, left ventricular underfilling or mechanical restriction than a single number of cardiac output [7] [30].

Monitor Perfusion as well as Pressure. End-tidal carbon dioxide will fall if there is a decrease in pulmonary blood flow or cardiac output; lactate will only rise slightly and is considered favourable; urine output is neither immediate nor specific for intraoperative renal perfusion; regional oximetry can detect cerebral or somatic desaturation. The Trend of Multimodality is relatively certain compared with other thresholds.

14. Procedure-Specific Modifiers of Hypotension

Cardiothoracic surgery is not a specific operation, and the item in the list is far less than a particular type of surgery. A VATS and a robot will fix the chest wall and add CO₂ inflation to raise the body. Thoracotomy will raise the intrathoracic pressure, increase blood loss and cause parasympathetic denervation. The other two are the types of resection; lobectomy, bilateral lobectomy and segmentectomy affect the pulmonary circulation and right ventricle differently than pneumonectomy, and esophagectomy, mediastinal surgery and pleural intervention are also different [7] [13].

Pneumonectomy is not mandatory. Cutting off a blood supply means all the heart's output has to go through the remaining organ, and thus the right ventricle

bears an excessive load. Esophagectomy has some problems, such as the combination of OLV and abdominal dissection, fluid redistribution, anastomotic complications and pain; thus, neuraxial or regional approaches are often used. Hilal clamp injury and tumour invasion are not usual for a normal VATS lobectomy and cause sudden changes.

In addition, the operating department is also relatively large. Distal and proximal OLV have different bronchial topographies, degrees of dependent lung, and what happens when the tube is displaced. Collapse may compress the heart and pulmonary arteries to a certain extent and reduce venous return. Therefore, rather than a single thoracic approach, a modular one has been chosen; that is, common parts will be kept and each operation will have its own alerts and emergency procedures.

The Degree of Detail in the Research Must be Consistent. Risk assessment should cover the method and plan, the range of OLV, the expected blood loss, and when to clamp and deflate in the event of hypotension. Otherwise, the general effect of all the treatments will not be clear; some may be beneficial and others harmful.

15. Postoperative Continuation of the Thoracic Hemodynamic Problem

Thoracic function is still not normal after skin closure. After lung removal, the remaining pulmonary circulation will be altered, and chest tubes may change the pressure in the chest. Inflammatory pain or immobility can cause hypoxemia and hypercapnia; therefore, epidural or regional anaesthesia can reduce sympathetic activity and thus prevent this. Additional atrial fibrillation, haemorrhage, bronchial occlusion and acute right ventricular impairment can all cause postoperative hypotension.

Previous non-thoracic studies have found that postoperative hypotension may be persistent, unnoticed and associated with myocardial injury [32] [33]. It is particularly serious after thoracic surgery because residual anaesthetic can harm oxygenation and other changes in the chest area. The end of a good intraoperative plan should be a handover, not just an extubation. What the receiving unit needs is the baseline pressure, the total hypotension exposure and vasopressor requirements, the RV outcomes and OLV duration, as well as the fluid balance and post-operative risks due to these factors.

Surgical Post-operative blood pressure goals should be personalised. A low MAP with conscious mentation, warm limbs, balanced lactate levels and preserved urine excretion is not the same as high blood pressure and increased lactate, oliguria, reduced oxygenation or right ventricular enlargement. Regulation is based on physiology, not on indicators; adjust analgesia according to changes in physiology, correct hypoxia or hypercapnia, give fluids, etc. Echocardiography, diuresis or progression to intensive care may be necessary; routine fluid administration is usually not needed.

The perioperative evaluation should cover the entire time of exposure from induction to the first post-operative period. Focusing only on intraoperative MAP may fail to capture how long hypotension occurs and where the various reasons for it are concentrated.

16. Research Priorities and a Transportability Framework

The following is the direction of future research; avoid speculation. Before using a non-thoracic treatment for thoracic diseases, it is necessary to determine whether there are sufficient similarities in the cohort, therapy, control group, endpoint and causal pathway. Some of the possible effect modifiers in this group are physiological, such as pulmonary vascular disease, mechanical ventilation, lateral positioning and open-chest mechanics; others are procedural, including surgical technique, ventilation tactics and fluid resistance. We would prefer the former over the latter after the clinical trial has published its results.

Distinguish between VATS and robotic surgery, as well as among different kinds of thoracotomy; distinguish between lobectomy and esophagectomy or pneumonectomy. Record the length of ventilation, airway pressure, PEEP, pulmonary artery occlusion pressure, blood loss, use of vasopressors and echocardiographic RV parameters. The groups mentioned are pulmonary hypertension, chronic obstructive pulmonary disease (COPD), essential hypertension, right ventricular dysfunction and pneumonectomy.

A thoracic blood pressure study should have a plan, not just one value. The three options are to set a personalised MAP goal and use RV-guided ventilation; to add active norepinephrine and targeted fluid therapy; or to constantly monitor MAP, echocardiography, blood pressure, oxygenation and the stage of surgery together. Determine whether there has been a decrease in organised tension and whether RV afterload, pulmonary oedema and ventilator-induced lung injury have fallen.

The results should include heart and chest problems. The main reasons are myocardial injury, AKI, stroke and RV failure. Pulmonary diseases refer to respiratory failure, prolonged ventilation, re-intubation and pulmonary oedema. Atrial fibrillation, ICU admission, hospitalisation and death are also problems. Mechanistic data should include vasopressor volume, fluid balance, OLV duration, PVR indices and time-adjusted MAP exposure.

Essays need to have enough details for the reproduction and transportability assessments. Specify the place of observation and the sensor base of the researcher, and then list the hypotension threshold and retention rules used. Please specify when the intervention and resuscitation started, what the respiratory conditions were before and after surgery, and how you plan to continue caring for the patient post-surgery.

17. Conclusions

Intraoperative hypotension is common in non-thoracic surgery, but it has not

been properly addressed or controlled during thoracic operations yet. Arterial pressure, pulmonary vascular resistance, right ventricular function, oxygenation, airway pressure and fluid balance are all related to VATS and lung resection.

We have studied non-thoracic papers and found some biological relevance and transferability. A threshold in abdominal or orthopedic surgery should trigger a mechanism-based evaluation and is not to be used alone as an algorithm. There are no randomised controlled trials on the thorax, but physicians should avoid prolonged hypotension, promptly find out why, administer fluids appropriately, adjust vasopressors with the RV in mind, and maintain perfusion in the face of RV collapse and pulmonary oedema.

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Use of AI Software

At the same time, a creative AI engine was employed to help revise the language and increase the expressiveness of the English in this paper. The researchers have verified and corrected all the generated results, cross-checked each reference and statistic with the original materials, and taken full responsibility for the content of this paper. No AI software was used to build the concept framework or scope, choose or assess the cited materials, or create any content. No AI platform is listed as a co-author.

Author Contributions

Sen Wang and Jiapeng Dan started the review and set the scope. Sen Wang, Shiyao Zhao and Wanrong Huang carried out the targeted literature search and selected the referenced articles. Benxin Zhu, Fuquan Zhu and Jialing Liao analysed and organised the cited materials and prepared the tables. Sen Wang wrote the first draft. Jiapeng Dan carefully revised the paper for important intellectual content and served as the corresponding author. All the contributors have approved the final edition.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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Abbreviations

AKI, acute kidney injury;
ARDS, acute respiratory distress syndrome;
CI, confidence interval;
CO₂, carbon dioxide;
COPD, chronic obstructive pulmonary disease;
ERAS, enhanced recovery after surgery;
ESTS, European Society of Thoracic Surgeons;
HPV, hypoxic pulmonary vasoconstriction;
HR, hazard ratio;
ICU, intensive care unit;
LV, left ventricle;
MAP, mean arterial pressure;
OLV, one-lung ventilation;
PEEP, positive end-expiratory pressure;
PVR, pulmonary vascular resistance;
RV, right ventricle;
VATS, video-assisted thoroscopic surgery.