Pulmonary Thromboendarterectomy and Acute Pulmonary Embolectomy Anesthetic Management

Objectives

1. **Understand the WHO types of pulmonary hypertension**
2. **Discuss the physiology of thrombotic pulmonary hypertension**
3. **Detail the anesthetic management of patients presenting for pulmonary thromboendarterectomy**
4. **Debate the benefits of an endobronchial blocker versus double lumen endotracheal tube (ETT) for management of post-PTE airway hemorrhage**
5. **Introduce management of patients with acute pulmonary embolism.**

Normal resting mean pulmonary artery pressure is 14 ±3 mmHg. Pulmonary hypertension is generally defined as a mean pulmonary artery pressure (mPAP) more than 25 mmHg at rest with a mean pulmonary capillary wedge pressure and left ventricular diastolic pressure less than 15 mmHg. The World Health Organization (WHO) defines 5 classes of pulmonary hypertension (Table XXX).

Table XXX – WHO Pulmonary Hypertension Classification

Group	Definition	Example	Notes
I	Pulmonary artery hypertension	Idiopathic Familial	Variable etiology and outcomes
II	Pulmonary venous hypertension	Left sided atrial or ventricular disease or left sided vascular disease	Requires treatment of left sided disease
III	Pulmonary hypertension associated with hypoxemia	COPD, ILD, Sleep-disordered breathing, chronic exposure to high altitude, developmental abnormalities	Correcting hypoxemia
IV	Pulmonary hypertension due to chronic thrombotic disease, embolic, or both	Thromboembolic obstruction of proximal arteries, thromboembolic obstruction of the distal pulmonary arteries, non-thrombotic emboli	Treatment includes PTE
V	Miscellaneous	Sarcoidosis, Langerhans'cell histiocytosis, compression of pulmonary vessels	

Pulmonary thromboendarterectomy (PTE) is the primary mode of treatment for patients that develop **chronic thromboembolic pulmonary hypertension (CTEPH)**. Approximately 500,000 patients will suffer symptomatic pulmonary emboli every year. It is estimated that 1-5% will progress on to CTEPH. Five year survival for patients with a mean pulmonary pressure greater than 50 mmHg is only 10%.

Several risk factors are loosely associated with the development of CTEPH (Table XXX).

Table XXX- Risk Factors for CTEPH

Factors Associated with the Development of CTEPH	Factors not associated with CTEPH
Antiphospholipid antibody Increased levels of Factor VIII Myeloproliferative syndromes Chronic inflammatory states Chronic ventriculoatrial shunts Splenectomy Recurrent venous thrombectomy Chronic indwelling catheters	Protein C or S deficiency Factor V Leiden mutation Prothrombin 20210G mutation

Physiology

The pulmonary vasculature generally has a lot of plasmin and clot should undergo fibrinolysis. Unresolved clot results in thrombus organization as well as fibrosis formation. Medial hypertrophy and intimal hypertrophy leads to vasculature narrowing which increases pulmonary vascular resistance. These microvascular changes are indistinguishable from idiopathic pulmonary arterial hypertension. Additional factors associated with increased pulmonary vascular resistance (PVR) include increased endothelin-1 expression which results in pulmonary vasoconstriction and remodeling.

Pre-operative Evaluation

Most patients will present with dyspnea on exertion. This is primarily to limitations on cardiac output associated with pulmonary occlusion as well as increased dead space ventilation. Other symptoms may include hemoptysis, syncope, light-headedness, chest pain, and palpitations. A history of pulmonary emboli (PE) or deep venous thrombosis is found is less than 50% of patients. Diagnostic modalities include cardiac cathetrization, transthoracic echocardiography (TTE), and electrocardiogram (EKG).

Cardiac catheterization allows assessment of many different measures critical for both diagnosis and evaluation of optimal management strategies (Table XXX).

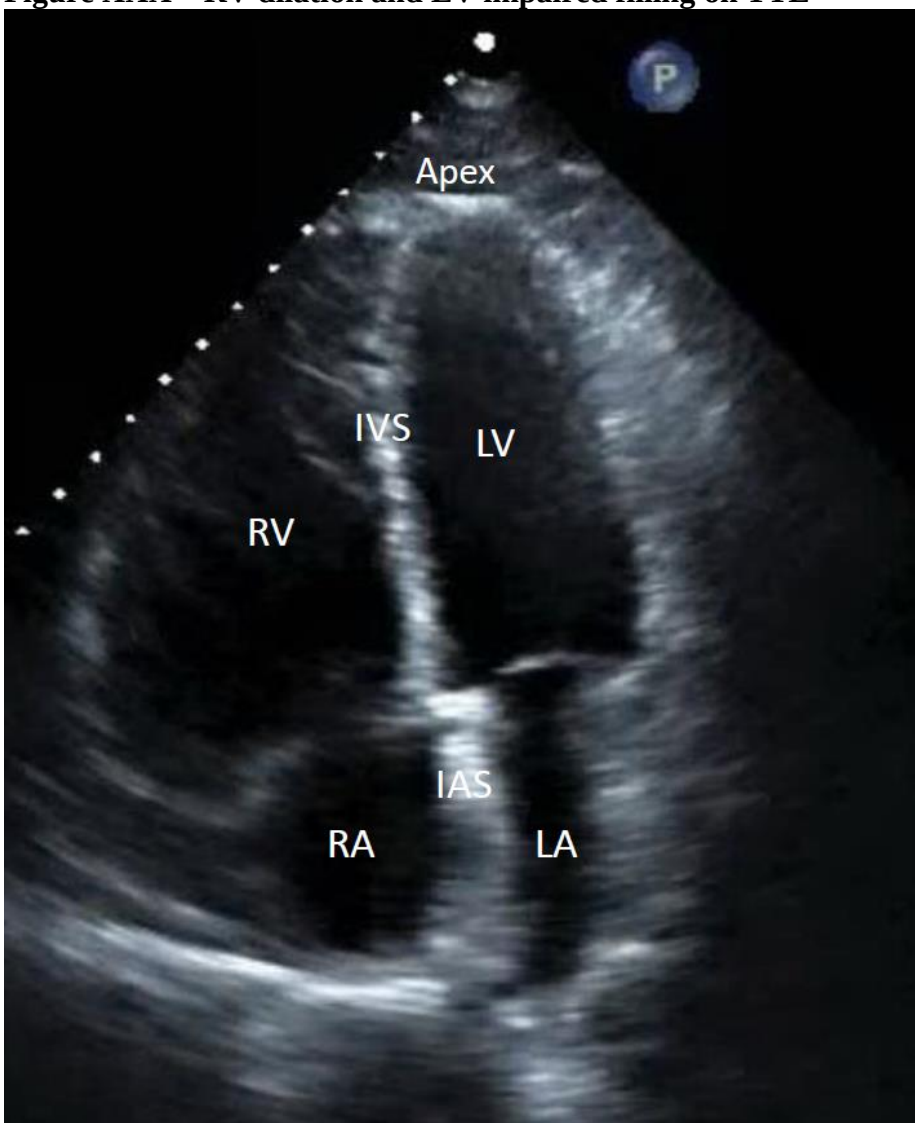
Table XXX- Cardiac catheterization information

Measure	Normal
Right atrial pressure	0-6 mmHg

Right ventricular pressure	12-24/0-6 mmHg
Pulmonary artery pressure (systolic and diastolic)	12-24/6-12 mmHg
Mean pulmonary artery pressure (mPAP)	14 ±3 mmHg
Pulmonary artery occlusion pressure (PAOP)	6-12 mmHg
Pulmonary vascular resistance (mPAP-PAOP)/CO	0.3-1.6 Woods units
Transpulmonary gradient (mPAP-PAOP)	≤12 mmHg
Coronary vascular disease	None or minimal
Atrial and ventricular shunts	None

TTE commonly reveals tricuspid regurgitation, right ventricular and atrial dilatation, right ventricular hypertrophy, and patent foramen ovale.

Figure XXX – RV dilation and LV impaired filling on TTE



EKG findings include right atrial enlargement (RAE), right axis deviation (RAD), right ventricular hypertrophy (RVH), and right bundle branch block (Table XXX). Lead V1 is

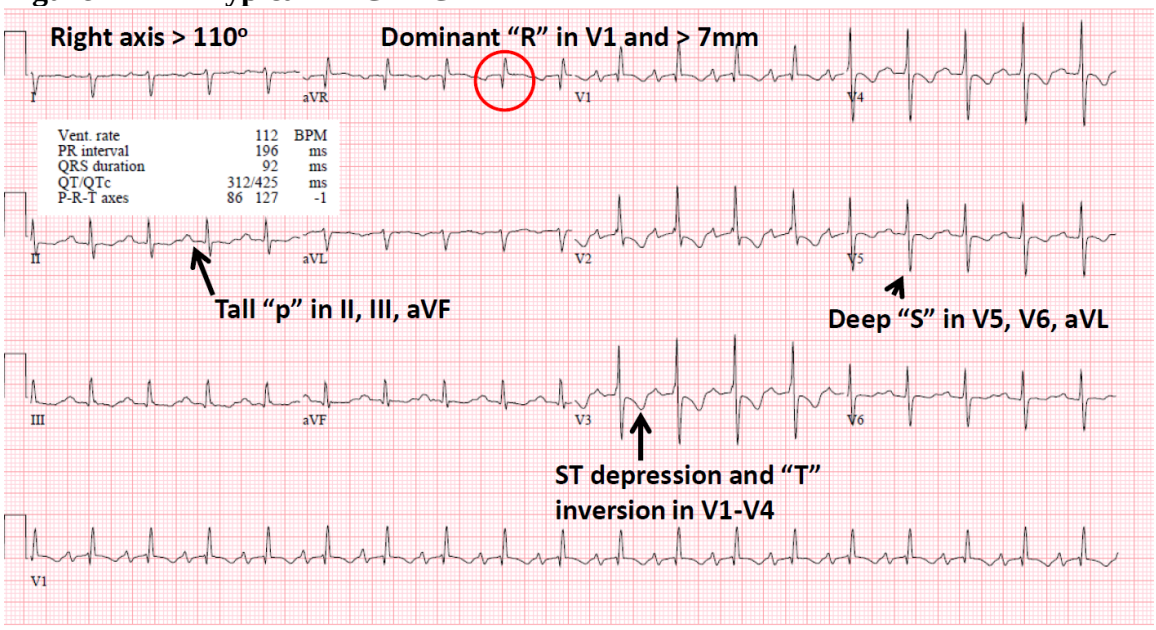
closest to the right ventricle myocardium and is the best lead to assess RVH, a dominant R wave is seen.

Table XXX- EKG changes commonly seen with pulmonary HTN

EKG Finding	Diagnostic Criteria
Right atrial enlargement (RAE)[1]	Tall P (≥2.5 mm) in II, III, AVF
Right ventricular hypertrophy (RVH)	Right axis deviation > 110° Dominant R in lead V1 R wave in lead V1≥7mm[2] ST depression and T wave inversion in V1-V4 (supporting) Deep S waves in V5, V6, and aVL

1. RAE is seen with many conditions such as COPD, pulmonary hypertension, pulmonary stenosis, tricuspid stenosis, and Tetrology of Fallot
2. A dominant R wave in V1 is also seen with a posterior MI, Type A WPW, and a right bundle branch block.

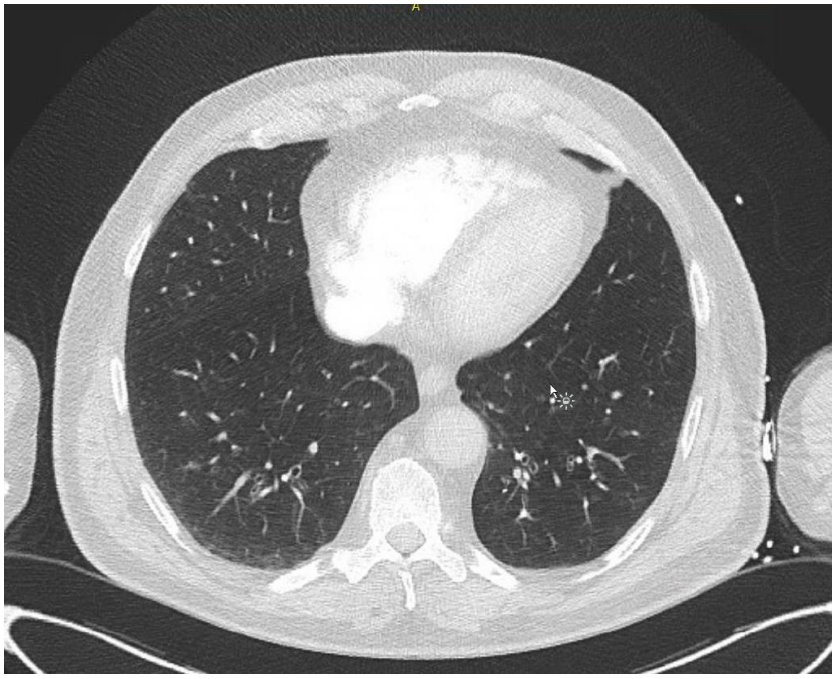
Figure XXX – Typical EKG in CTEPH



Right heart catheterization will allow accurate assessment of the true pulmonary pressures which may exceed 100mmHg (systolic) (Figure XXX). Left heart catheterization is performed to assess for concomitant coronary artery disease.

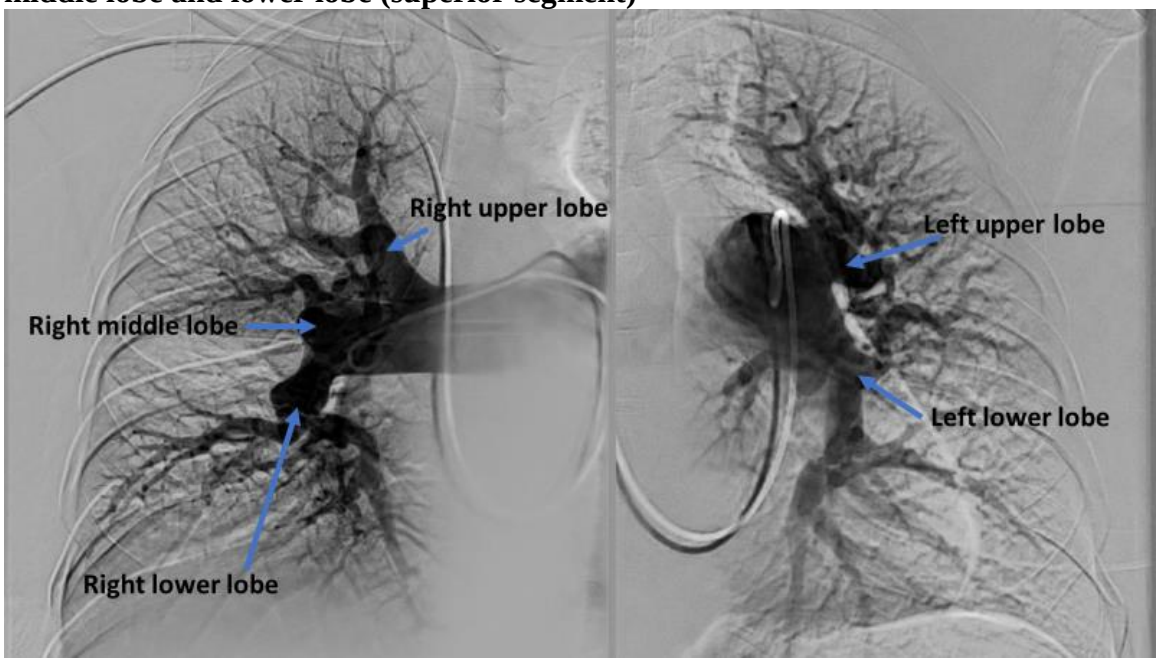
Figure XXX- Near and supra systemic pulmonary pressures

Computed Tomography may demonstrate findings that are pertinent for induction of anesthesia and include RV enlargement and hypertrophy as well as a shift of the interventricular septum towards the left with impaired filling of the left ventricle.



Conventional pulmonary angiography (CPA) is rarely performed anymore. Findings in the setting of PE may include abrupt cut-off, filling defects, slow flow, and regional hypoperfusion. CPA is subject to observer variability and accuracy may be lower than CT.

Image XXX – Pulmonary angiography demonstrating filling defects in the right middle lobe and lower lobe (superior segment)



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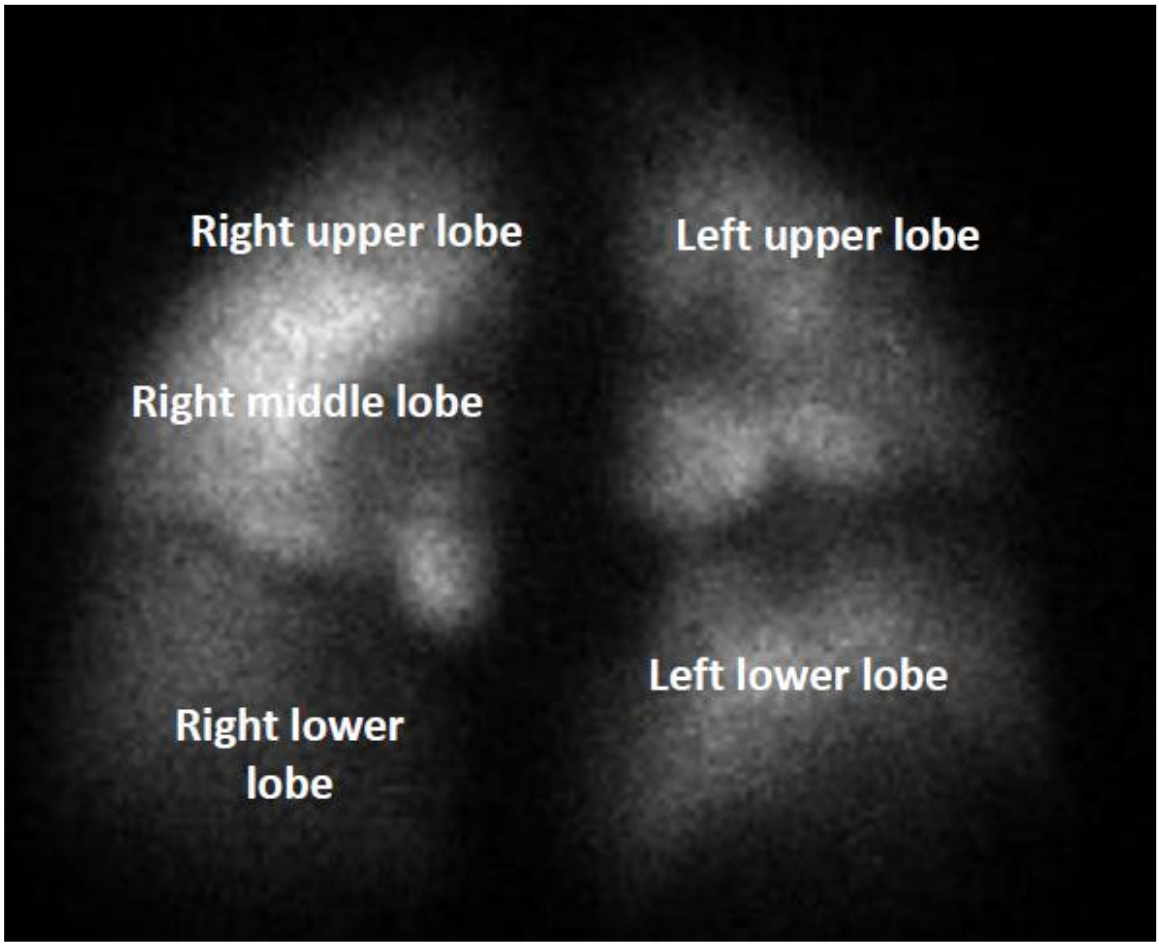


Medical Management

Medical management of CTEPH focus on two areas- preventing clot recurrence and minimizing pulmonary vasoconstriction (Table XXX).

Table XXX- Medical management of CTEPH

Focus	Example	Action
Anticoagulation	Coumadin	Inhibition of formation of Vitamin K dependent clotting factors
Prostanoids	Epoprostenol Treprostinol Ilioprost	Prostacyclin analog that induces pulmonary vasodilation as well as inhibit platelet activation
Endothelin Antagonists	Bosentan	Endothelin-receptor antagonist. Reduces PVR as well as inhibits vascular remodeling
Phosphodiesterase inhibitors	Sildenafil Tadalafil Milrinone	Stabilize cyclic guanosine monophosphate levels which augments nitric oxide-mediated pulmonary vasodilation and ventricular inotropy.
Diuretics	Furosemide	Reduction in RV dilitation

Anesthetic Management

Pre-operative evaluation should include close assessment of the cardiovascular and pulmonary effects of chronic PE.

The **cardiovascular effects** may include right ventricular (RV) hypertrophy, RV dilatation, RV hypokinesis, tricuspid regurgitation, and increased right atrial pressure (RA). Pulmonary regurgitation may be also seen. Patients with chronically elevated right atrial pressure and a patent foramen ovale may exhibit right to left shunting of blood. Systemic manifestations of chronically elevated RA pressure include peripheral edema and hepatic dysfunction.

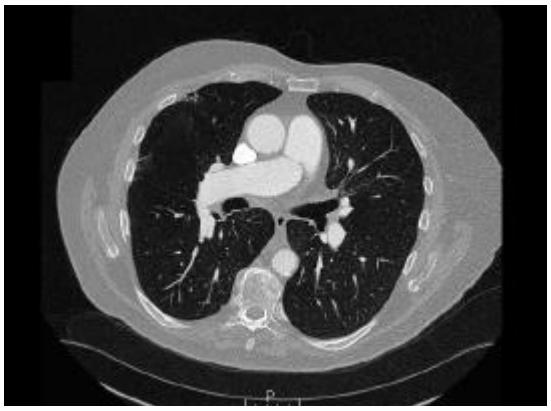
The chronic and progressive pressure overload on the right ventricle from high pulmonary vascular resistance can result in a shift of the interventricular septum to the left. This not only restricts diastolic filling of the heart but also results in worsening function of the right ventricle. This is because appropriate function of the right ventricle depends on the round shape of the left ventricle.

Computed tomography has replaced V/Q scanning and pulmonary angiography as the imaging modality of choice. Diagnostic findings are divided into **vascular signs** and **parenchymal signs** (Table XXX). Enlargement of the PA to greater than a diameter of 20 mm predicts pulmonary hypertension with a sensitivity of 86% and a specificity of 89%. The pulmonary system is involved in blood exchange while the bronchial system is not. When the pulmonary arteries become occluded the bronchial arteries become involved in gas exchange and hypertrophy (Figure 1). Normal bronchial arteries are only 0.5-1.5 mm at origin and are enlarged if the diameter is > 2 mm.

Table XXX - Radiographic Signs of CTEPH

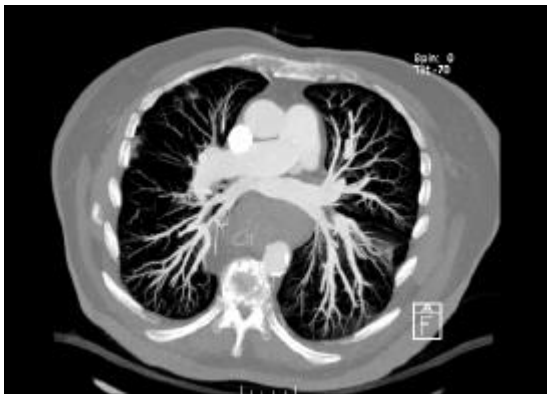
Vascular signs of CTEPH	Parenchymal signs of CTEPH
Complete obstruction Decrease in vessel diameter distal to obstruction Partial filling defects Enlargement of the main PA (size greater than the aorta) Enlargement of the left and right PA branches Right heart chamber enlargement Enlarged bronchial and non-bronchial arteries	Mosaic attenuation pattern

Figure 1 – Occluded right upper lobe vascularity



A mosaic attenuation pattern is a mixture of regions of different attenuation. Low attenuation is caused by regional hypoperfusion (Figure 2). Pulmonary infarction is often seen as peripheral irregular, linear, or wedge shaped densities.

Figure 2 – Hypoperfusion of the right upper lobe with decreased pulmonary venous flow



Interoperative Management

Monitoring will always include an arterial line and central venous line. The benefits of a pulmonary artery catheter (PA line) include beat by beat analysis of right ventricular pressure and intermittent cardiac output. The risks include arrhythmia, dislodgement of clot, and PA rupture. The PA line may be placed before or after induction of anesthesia and should only be advanced slightly into the pulmonary artery. The PA line may need to be pulled back during the procedure. The degree of pulmonary hypertension, right ventricular function, patient comfort and anxiety should be considered. These patients often do not tolerate Trendelenberg positioning well. The degree of sedation to facilitate placement before induction of anesthesia may significantly worsen baseline pulmonary hypertension due to hypercarbia and hypoxemia.

The primary goal of anesthesia is to avoid compromise of right ventricular function and elevations in pulmonary artery pressure (Table XXX).

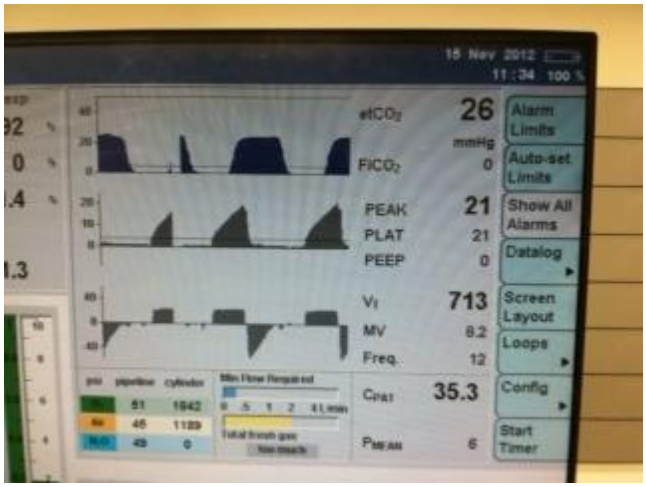
Table XXX – Causes of Increases in Pulmonary Vascular Resistance

Causes of increases in pulmonary vascular resistance
Hypoxemia
Hypercarbia
Pulmonary edema
PEEP, High tidal volume
Pulmonary emboli
Anxiety
Pain
Hypothermia
High doses of alpha (α) agonists

Sedation should only be given while the patient is being closely monitored. Induction an agent such as Etomidate with small doses of fentanyl may maintain hemodynamic function better than large doses of propofol. The change from spontaneous to controlled ventilation may increase pulmonary vascular resistance and worsen right ventricular filling.

These patients will by manifest significant ventilation / perfusion mismatching (V/Q) and the end-tidal carbon dioxide ($ETCO_2$) will not reflect the actual $PaCO_2$ (Figure XXX). Efforts to maintain a normal $ETCO_2$ may result in systemic hypercarbia and worsening pulmonary hypertension. Normocarbia is the goal ($PaCO_2$ 36-38).

Figure XXX – $ETCO_2$ while arterial blood gas analysis reads 7.40/38/279



Tricuspid regurgitation (TR) may be associated with dilatation of the right ventricle. Bradycardia may not be well tolerated.

These patients often have an elevated hematocrit and the removal of 1-2 units of blood for later transfusion may decrease the need for blood from the blood bank. These patients are generally on warfarin prior to surgery and fresh frozen plasma may be needed.

TEE

Transesophageal echocardiography is nearly universally utilized during PTE surgery. Significant pre-operative findings secondary to CTEPH include right ventricular dilitation and dysfunction, tricuspid regurgitation, pulmonary regurgitation, and shifts of the interatrial and interventricular septum. A patent foramen ovale (PFO) may be seen and the direction of blood flow is critical. Clot may occasionally be seen in the cardiac chambers or crossing a PFO. After cardiopulmonary bypass the function of the right ventricle may be compromised. TEE may guide pharmacologic management.

Surgery

The decision whether to operate or not is made by a multidisciplinary team of cardiologists, thoracic and cardiac surgeons, pulmonologists, and radiologists. In 20-40% of cases surgery is not an option.

PTE is performed on cardiopulmonary bypass with deep hypothermic circulatory arrest (DHCA). A median sternotomy is performed. Bicaval cannulation is performed to eliminate blood return to the right atrium and allow performance of procedures such as tricuspid valvuloplasty or closure of a PFO. The aorta is cannulated. Antegrade cardioplegia is administered and cooling is initiated to 18^{o}C. A left ventricular vent via the right upper pulmonary vein may be necessary. Once adequate cooling has been reached, DHCA is initiated. DHCA helps to eliminate filling of the pulmonary artery from collateral circulation. Application of ice to the head is common. Occasionally magnesium supplementation is administered.

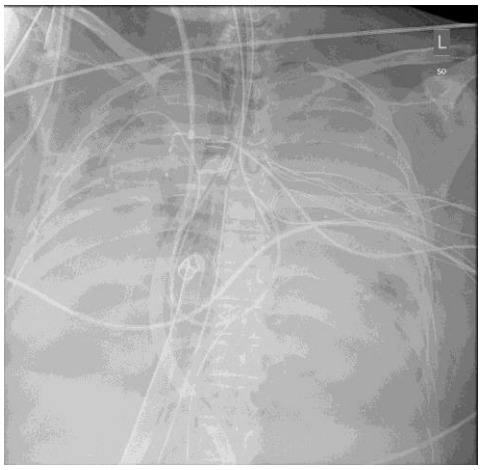
The right pulmonary is opened first. The surgeon will establish a plane for dissection after any gross clot is removed. The plane for the endarterectomy is started on the posterior pulmonary artery wall rather than at the tips of the pulmonary arteriotomy. Care is takes not to damage the phrenic nerve. Plane dissection continues into the distal segments. After the right side is completed circulatory flow is resumed while the arteriotomy is closed. After 20 minutes of circulatory flow, DHCA is initiated again to allow focus on the left pulmonary artery.

Upon completion of the left PTE full circulation and warming is initiated. A warming blanket may be started. During warming tricuspid valve work may be completed although some institutions may not repair the valve with the expectation that remodeling of the right ventricle may return the function to normal.

The airway should be checked for blood as injury is not uncommon. Should significant bleeding and pulmonary compromise be noted, the team should prepare for alternative support i.e. ECMO.

Bleeding into the lung may be so significant that complete that ventilation may be impossible (Figure XXX). Venoarterial (VA ECMO) accomplishes oxygenation and ventilation while allowing nearly completely eliminating blood flow to rest the lungs.

Figure XXX-Complete lung opacification



Weaning from cardiopulmonary bypass may complicated by difficulty with right ventricular function. Nitric oxide (NO) is the preferred pulmonary vasodilator. Respiratory care services should be notified as soon as the need is determined. Epoprostenol (Flolan) may be used or started as NO is weaned.

Reperfusion abnormalities including hypoxemia and hemorrhage are uncommon but a challenge to manage. Reperfusion abnormality without hemorrhage usually occurs in the first 48 hours after the procedure and is indicated by hypoxemia, hypercarbia, and edema. Mechanical ventilation may not be successful.

Table XXX – Benefits of VA ECMO in Pulmonary Hemorrhage After PTE

Provides a circuit for blood to largely bypass PA and vascular injury
Alleviated the impact of right-sided failure on circulation
Provides efficient gas exchange while allowing reduction of ventilator associated lung injury

Adapted from Hou X, Can J Anesth 2013;59:622-623

Management of Bleeding after PTE

Bleeding after PTE is one of the most feared and difficult to manage complications. Our own data reveals that clinically significant bleeding occurs in 2.9% of cases. Reported mortality is 70%. Bleeding generally occurs in the thinner distal vessels where dissection tends to be more difficult.

Risk factors for bleeding associated with PTE have not been clearly defined. Suggested factors include older age, residual pulmonary hypertension, and higher Jameson classification.

Our experience is that bleeding is only recognized during the period of preparation and weaning from cardiopulmonary bypass. We have developed an algorithm for management of airway hemorrhage (Figure XXX). Prior to weaning the airway (ETT) is visually inspected while the lungs are manually expanded. Increased resistance within the airways or the presence of blood should prompt further investigation. It is recommended that the weaning process be suspended during evaluation. Bronchoscopy should be

performed to identify the degree of bleeding (major vs major) and the location. Attempts to manage minor bleeding include weaning from bypass followed by correction of coagulopathy, positive end-expiratory pressure (PEEP), and application of topical vasoconstrictors (epinephrine, phenylephrine). Problems associated with major bleeding include hypovolemia as well as compromise of oxygenation and ventilation. The priority is protection of areas of the lung that are not bleeding. Lung isolation may be accomplished by either placement of a bronchial blocker or a double lumen endotracheal tube. There are advantages and disadvantages to each device (Table XXX).

Device	Advantages	Disadvantages
Endobronchial blocker	No ETT change Selective lobe isolation possible	Easy to dislodge Requires bronchoscopy for positioning Less effective isolation
Double lumen endotracheal tube	Does not require bronchoscopy for initial placement Better lung isolation	Lager outer size (higher risk of vocal cord injury) Smaller bronchial and tracheal lumen Effective suction difficult

Extracorporeal membrane oxygenation (ECMO) has been utilized to support patients with pulmonary hemorrhage (Pretorius 2009) (Berman 2008). Venoarterial (VA) ECMO provides three benefits in pulmonary edema (Hou 2012) (Table XXX).

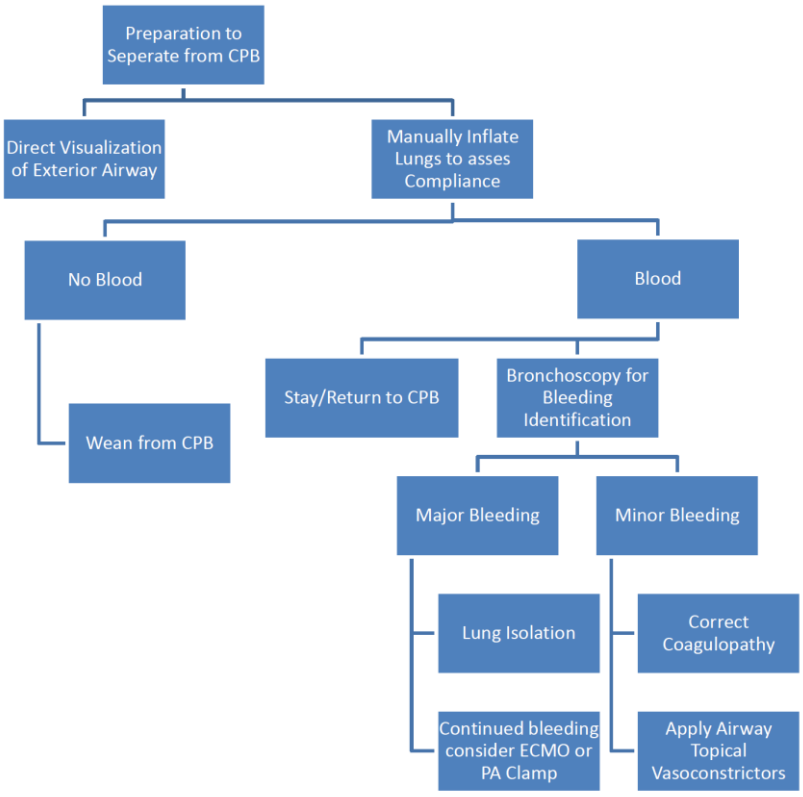


Figure XXX- Management of airway bleeding during PTE

Transport

Transport is critical as with all patients. Transport with a mechanical ventilator may more accurately reproduce the parameters determined as correct compared with manual ventilation. Hypercarbia, acidosis, and hypoxemia must be avoided.

Pathology

The thrombus findings during PTE fall into 4 categories of the Jamieson classification (Table XXX)

Table XXX – Jamieson Classification

Type	Description of inter-operative findings	Occurrence	Prognosis (One-month survival post op)
Type 1	Proximal clot (classical)	20%	98.7%
Type 2	No visible clot but endarterectomy performed along planes	60%	97.5%
Type 3	No visible thrombus but distal intimal thickening	15%	86.8%
Type 4	Pulmonary hypertension not due to CTEPH	5%	85.7%

Thrombus and fibrotic debris removed during PTE is generally subacute (Figure XXX) or chronic (Figure XXX).

Figure XXX – Subacute thrombosis removed from pulmonary artery

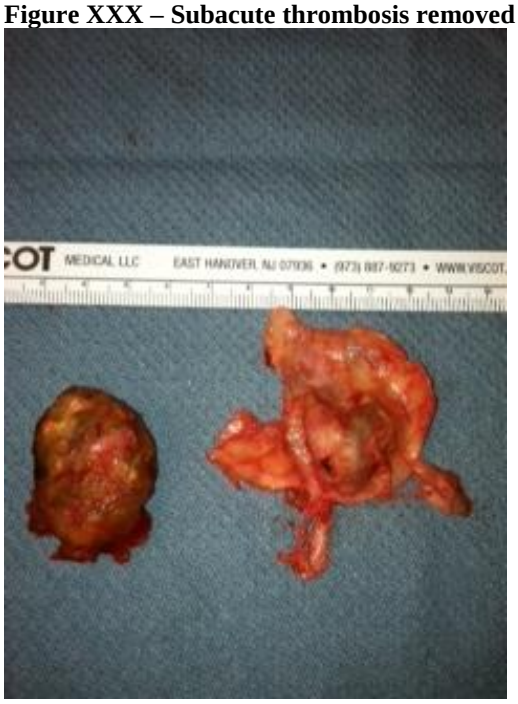
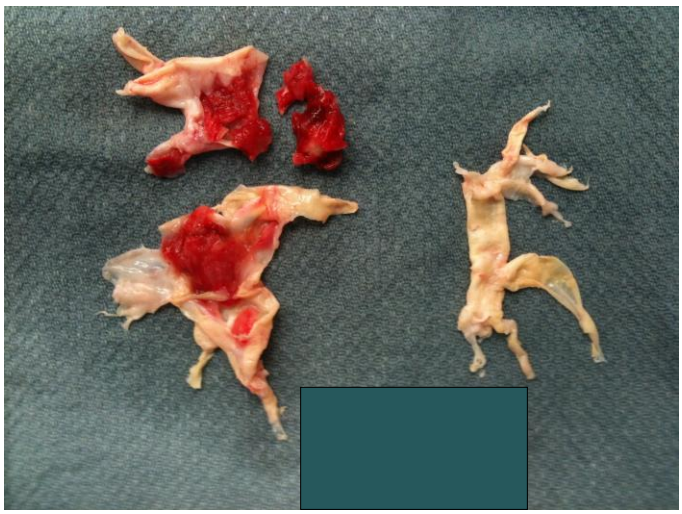


Figure XXX – Chronic thrombus



Acute Pulmonary Embolectomy

Acute massive pulmonary thromboembolism is associated with significant mortality as high as 25% in patients without associated cardiac arrest and as high as 65% in those that experience cardiac arrest. Patients that remain hemodynamically unstable and who have a contraindication to thrombolysis or have failed catheter-based therapy or thrombolysis may benefit from early surgical intervention.

Frederich Trendelenberg, M.D. attempted an surgical embolectomy in 1908 but the patient succumbed to bleeding it was not until 1924 that the first successful surgical embolectomy was reported by Martin Kirschner.

Table XXX – History of Surgical Embolectomy

Surgeon (Year)	Event / Procedure
Frederich Trendelenberg (1906)	Ligation of IVC to prevent progression and embolism of septic thrombi
Frederich Trendelenberg (1908)	First attempt at off-pump embolectomy vial left thoracotomy in a patient with femoral neck fracture
Martin Kirschner (1924)	First successful surgical embolectomy
John Gibbon (1931)	Concept of cardiopulmonary bypass in setting of acute PE
Heparin (1936)	First use of heparin for acute PE
Denton Cooley (1961)	First use of bypass for acute pulmonary embolectomy
Greenfield (1969)	Catheter based clot removal
Thrombolytic therapy (1971)	First use of thrombolysis for treatment of acute PE.

Diagnosis of Acute PE

Symptoms of massive pulmonary emboli are often non-specific and include chest pain, shortness of breath, and occasionally syncope. Tachycardia is commonly present. Arterial blood gas analysis shows hypoxemia with hypocapnia.

The Willis score is a clinical risk stratification model that grades the likelihood of PE based upon clinical presentation and history (Table XXX).

Table XXX- Willis score

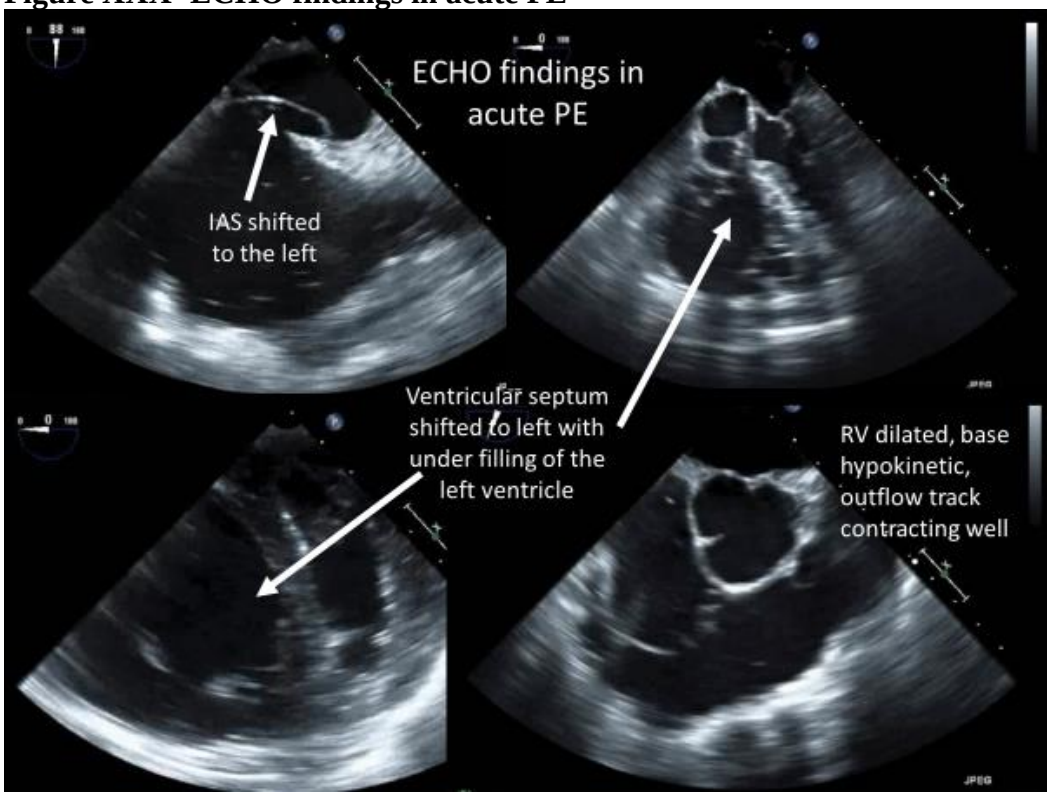
Parameter	Score
Malignancy	1.0 point
Hemoptysis	1.0 point
History of PE or DVT	1.5 points
Tachycardia (>100)	1.5 points
Immobilization (≥3 days) or recent surgery (within 4 weeks)	1.5 points
Most likely diagnosis is a pulmonary emboli; no alternative diagnosis better explains the illness	3.0 points
Clinical signs and symptoms of deep venous thrombosis	3.0 points

Three-tier scale -Low risk (0-1), Moderate risk (2-6), High risk (>6)
Two-tier scale- PE Unlikely (≤4) obtain D-dimer, PE likely (≥4) obtain PE CTA

Computed tomography (CT) serves two purposes, to identify pulmonary emboli and also to rule out other causes of symptoms including aortic dissection, pericardial effusions, rib fractures, pneumonia, and coronary calcification, all of which may mimic symptoms of a PE. CT has an 83% sensitivity and a specificity of 96% and higher when combined with clinical probability. A central filling defect surrounded by contrast is diagnostic of PE.

Echocardiography may demonstrate a shift of the interatrial and interventricular septum to the left, dilation of the right ventricle and a small underfilled left ventricle. Additional findings may include mobile clot in the right atrium, right ventricle, pulmonary artery, or transversing the interatrial septum. Ultrasound can also be used to evaluate the presence of thrombus in the femoral veins.

Figure XXX- ECHO findings in acute PE



Management of an acutely unstable patient with a PE

There are three critical components to management of an unstable patient with acute pulmonary emboli. **Cardiopulmonary resuscitation** includes the administration of oxygen and initiation of inotropic agents for the management of right ventricular failure. Pulmonary artery vasodilators (iNO or Veletri) may reduce pulmonary vascular resistance. If pharmacologic intervention is not successful, extracorporeal membrane oxygenation (ECMO) allows stabilization. Initiation of anticoagulation to prevent or reduce extension is critical. Anticoagulation reduces the extension of clot within the pulmonary vasculature and embolization of the venous thrombosis. Reperfusion of the pulmonary vasculature may be accomplished by surgical embolectomy or thrombolysis if not contraindicated.

Emergent Surgical Embolectomy Indications

Emergent surgical embolectomy is indicated for patients with a massive or submassive PE associated with a (1) contradiction to thrombolysis, (2) failed thrombolysis or catheter extraction, or (3) shock likely to cause death before thrombolysis can take effect (Fukuda 2017).

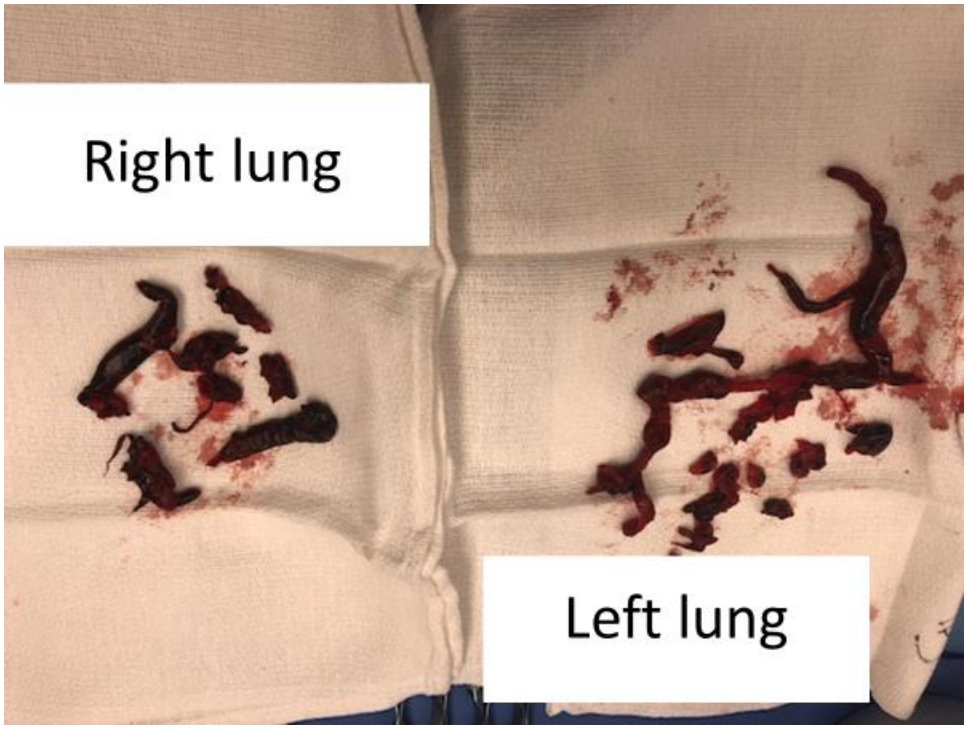
Surgical embolectomy may also be indicated in patients in the post-operative period in whom thrombolysis is contraindicated due to concerns for bleeding. Such patients include those with a recent craniotomy or spinal surgery.

Pulmonary emboli in the peripartum period is a complicated problem. The incidence I slow but increases in pregnant women over age 35. One reported experience included a combined c-section and pulmonary embolectomy (survived) and 2 surgical embolectomies where the pregnancy continued. One child was stillborn while the other survived.

Surgical Embolectomy

Surgical embolectomy occurs on cardiopulmonary bypass. The main PA is opened and clot is removed (Figure XXX). A Fogarty thormbectomy catheter may be utilized to remove more distal clot but this may injury pulmonary vascular tissue resulting in pulmonary hemorrhage. Some have recommended squeezing the lungs to milk back clot. This may induce more bleeding.

Image XXX – Acute biventricular clot



Anesthesia for Acute Surgical Embolectomy

Acute massive pulmonary embolism results in acute right ventricular pressure overload. The right ventricle (RV) is generally dilated with a shift of the interventricular septum to the left. This may be associated with significant tricuspid regurgitation (TR). TR associated with RV dilatation and septal shift limits filling of the left ventricle. Cardiac output (CO) decreases. If CO decreases enough, shock occurs. If blood pressure decreases, perfusion of the RV falls and function further decreases resulting a clinical spiral.

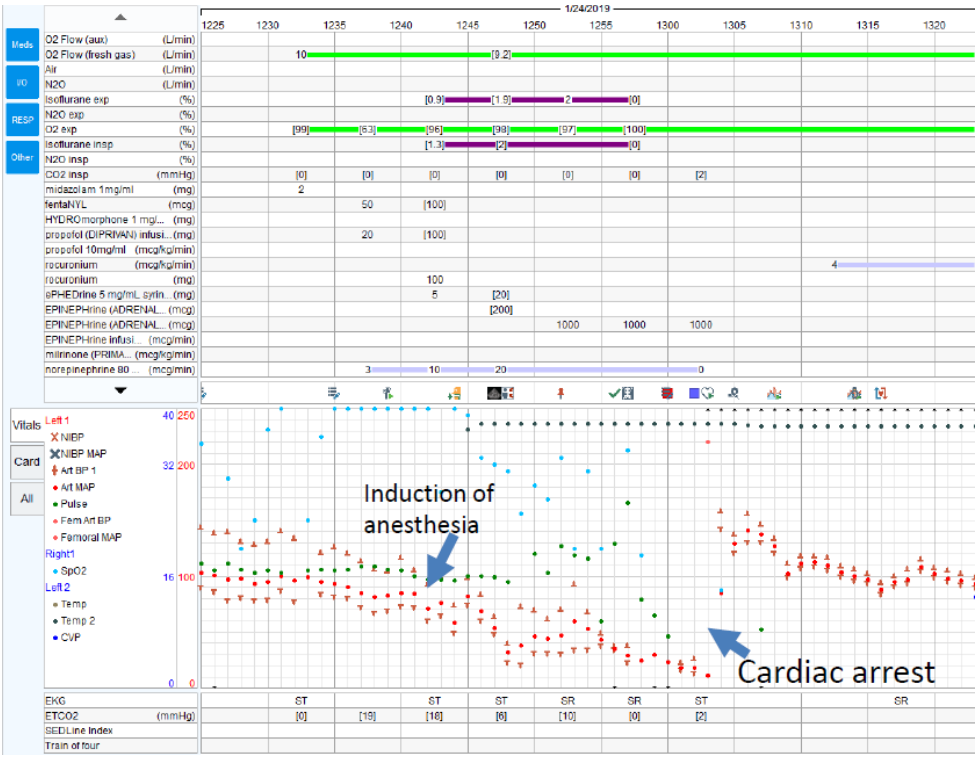
Induction of anesthesia may result in additional hypotension and RV malperfusion. PEEP, hypoxemia, sympathetic discharge, and straining will further increase pulmonary vascular resistance and LV filling. Complete cardiovascular collapse may occur.

An arterial line prior to induction is necessary. Placement of a central line can allow assessment of central venous and pulmonary pressures. Placement of a pulmonary catheter (PAC) is a concern due to risk of displacement of clot. Additional safety factors to consider include surgical presence in the operating room along with the presence of a member of the perfusion team, catheter placement in the femoral vessels for emergent initiation of bypass or ECMO.

Plans for induction of anesthesia should include use of an agent not likely to disturb hemodynamics (Etomidate). Use of inotropic agents or vasopressors to achieve a safety margin prior to induction may reduce the risk of hemodynamic collapse.

Weaning after surgical embolectomy may be challenging due to factors such as right ventricular dysfunction, residual pulmonary hypertension, or airway hemorrhage associated with surgery. Inotropic support with epinephrine or milrinone may be necessary. Inhaled nitric oxide (iNO) or Epoprostenol (Veletri) may reduce pulmonary vascular resistance. Failure to wean from cardiopulmonary bypass may require management with ECMO.

Figure XXX – Cardiac arrest associated with induction in a patient with acute pulmonary embolism



Outcomes after surgery for acute pulmonary embolectomy

Multiple studies have been performed assessing outcomes after surgical embolectomy. Mortality rates range from 3.6% to 27.2% (Fukuda 2017). Mortality is highest in patients that suffer significant arrest prior to surgery.

Table XXX- Studies of Acute Pulmonary Embolectomy

Study (year)	Patients	Outcomes	Notes
Gray (1988)	71 patients between 1964 and 1986	16/15 patients (64%) of those suffering significant arrest prior to surgery died 5/46 (11%) that did not have arrest died Overall 50/71 (70%) and did well without morbidity	Most of those that died did so due to neurologic compromise
Aklog (2002)	29 17 men, 12 women	Survival 26 (89%) Mortality 3 (11%)	
Neely (2015)	115 patients between 1999 and 2013	Central, unstable 49/115 (43%) Stable 56/115 (49%) Other indication 10/115 All Operative mortality 6.6% (Unstable 10.2%, stable 3.6%)	46 (44%) had surgery within 5 weeks
Elssal (2016)	5 in 18 months	Survival 3 (66.6%) Mortality 2 (33%)	1 patient died due to complications associated with

			IVC filter, 1 died interop, 2 patients developed ARF
Keeling (2016)	214 patients between 1998 and 2014	Mortality 25 (11.7%)	
Kalra (2017)	Meta-analysis of 56 studies with 1579 patients	All-cause mortality 26.3% Surgical site complication 7.0%	

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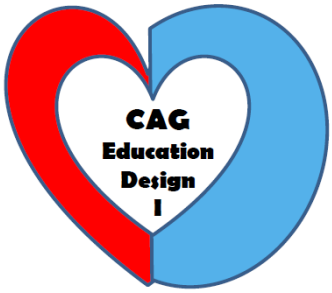
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