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Pulmonary Embolism

Fayaz Ahmed and Ahmed Elsayed Mahmoud

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Abstract

Pulmonary embolism is sudden occlusion of pulmonary arteries, usually by a clot arising in the lower limb veins. The majority of pulmonary emboli are silent, and it is only when the embolus burden is substantial that the patient becomes symptomatic. Mortality after an acute, major thromboembolic episode is significantly high. Pulmonary embolism which causes hemodynamic instability is usually associated with occlusion of more than 50% of the pulmonary vasculature. Associated severe pulmonary hypertension may cause cardiac arrest. The precipitation of RV failure is also affected by the degree of preexisting right ventricular hypertrophy or dilatation, tricuspid valve regurgitation, and the presence of coronary artery disease. Aggressive therapy is needed in this subgroup of patients. Unfortunately, surgical embolectomy is seldom even entertained as an option in the management of these patients. A critical assessment of the data reveals that there is in fact a definite place for surgical therapy in the management of massive pulmonary embolism.

Keywords: pulmonary embolism, pulmonary hypertension, surgical embolectomy

1. Introduction

Pulmonary embolism is sudden occlusion of pulmonary arteries, usually by a clot arising in the lower limb veins. It is not a disease by itself but rather a complication of this venous thrombosis.

Pulmonary embolism is commonly mislabeled, more likely an unrecognized phenomenon particularly in hospitalized individuals.

The majority of pulmonary emboli are silent, and it is only when the embolus burden is substantial that the patient becomes symptomatic. Mortality after an acute, major thromboembolic episode reaches about 15–20% of patients within 48 hours [1].

Aggressive therapy is needed in this subgroup of patients. Unfortunately, surgical embolectomy is seldom even entertained as an option in the management of these patients, published mortality rates for acute pulmonary embolectomy have ranged from 20 to 60%, making it difficult to argue that the surgical results were any better than the natural history. A critical assessment of the data reveals that there is in fact a definite place for surgical therapy in the management of massive pulmonary embolism [2].

2. Practice essentials

Pulmonary embolism usually presents with dyspnea, tachypnea, dull chest pain, and cardiovascular changes such as tachycardia, mild-to-moderate hypotension, and distended neck veins. Most pulmonary embolism patients are hemodynamically stable and have adequate cardiac output.

Pulmonary embolism which causes hemodynamic instability is termed massive pulmonary embolism. It is usually associated with occlusion of more than 50% of the pulmonary vasculature [3]. Pulmonary angiograms demonstrate a unique degree of blockage of the pulmonary vasculature.

The severity of symptoms may not be related to the embolus burden, particularly in patients with preexisting cardiac or pulmonary disease. Cardiac arrest may occur. The precipitation of RV failure is also affected by the degree of right ventricular hypertrophy or dilatation, tricuspid valve regurgitation, and the presence of coronary artery disease. Pulmonary hypertension is also influenced by many factors. Humoral factors such as serotonin, adenosine diphosphate (ADP), platelet-derived growth factor (PDGF), and thromboxane are released from platelets attached to the thrombi. Anoxia and tissue ischemia downstream from emboli inhibit endothelium-derived relaxing factor (EDRF) production and enhance release of superoxide anions by activated neutrophils [4].

2.1. Anatomy

The anatomy of the pulmonary vasculature should be familiar to all cardiothoracic surgeons. What may be less well appreciated, however, is the remarkable access available to the lobar vessels via median sternotomy. All lobar and segmental vessels can be accessed via incisions in the pulmonary arteries from within the pericardial space as one would during pulmonary thrombo-endarterectomy [5].

2.2. Pathophysiology

Pathophysiology of pulmonary hypertension in acute pulmonary embolism entails the release of serotonin from platelets, histamine from tissues, and circulating thrombin. Hypoxia due to ventilation/perfusion mismatch and increased dead space will also worsen pulmonary vasoconstriction and set a vicious cycle. Persistent systemic hypotension or refractory hypoxemia is an indication for aggressive interventional, surgical, or thrombolytic management. Operative risk is markedly elevated once the patient is in cardiogenic shock [6].

Right ventricular dysfunction is a harbinger of hemodynamic decompensation, an event that may unfold quite precipitously, abruptly closing the window of opportunity on a patient that has been otherwise holding on for several hours. Thrombolytics have taken center stage in the aggressive treatment of the unstable patient [7]. This is in part due to their wide availability and the familiarity many physicians have with their use in the context of treating acute coronary syndromes. However, study reports found no improvement in mortality rate when thrombolytics were used in unselected patients as compared with heparin but an almost two-fold increased risk of hemorrhage. Catheter embolectomy is another option. Endovascular techniques include clot fragmentation, clot aspiration, and rheolytic therapy. The mortality rate associated with these interventions, however, has been 25–30% [8]. Surgical intervention performed before hemodynamic collapse has an operative risk no higher than that of thrombolytic therapy in most cases. Surgery is clearly the option of choice when there is a clot in transit, in the right atrium, or trapped in a patent foramen ovale [7, 9].

3. Diagnosis

Diagnosis is suspected with a history consistent with massive pulmonary embolism. Symptoms and signs vary with the extent of blockage, the magnitude of humoral response, and the cardiac and pulmonary reserve of the patient. Routine laboratory tests are usually normal. Serum D-dimer is almost always elevated in the presence of acute pulmonary embolus and is frequently used in emergency rooms as a screening test. The most common electrocardiographic abnormalities of acute pulmonary embolism are tachycardia and nonspecific ST- and T-wave changes [5]. The major value of the electrocardiogram is excluding a myocardial infarction. A minority of patients with massive embolism may show evidence of corpulmonale, right axis deviation, or right bundle branch block. Chest X-ray may show oligemia (Westermark's sign) or linear atelectasis (Fleischner lines), both of which are nonspecific findings. Ventilation-perfusion (V/Q) scans may be used for their negative predictive value and may be unreliable because of pneumonia, atelectasis, previous pulmonary emboli, and other conditions may cause a ventilation and perfusion mismatch. In general, negative V/Q scan may rule out significant pulmonary embolism. Pulmonary angiogram provides the most definitive diagnosis for acute pulmonary embolism. Contrast-enhanced high-resolution computerized tomographic (CT) scanning is most commonly diagnostic (**Figure 1**).

Advantages of MDCTPA in the diagnosis of acute PE are as follows:

• Widely available, especially after day hours

• Rapid interpretation

• Direct visualization of embolus

• Noninvasive

• Highly accurate

• Evaluation of alternative causes of chest pain

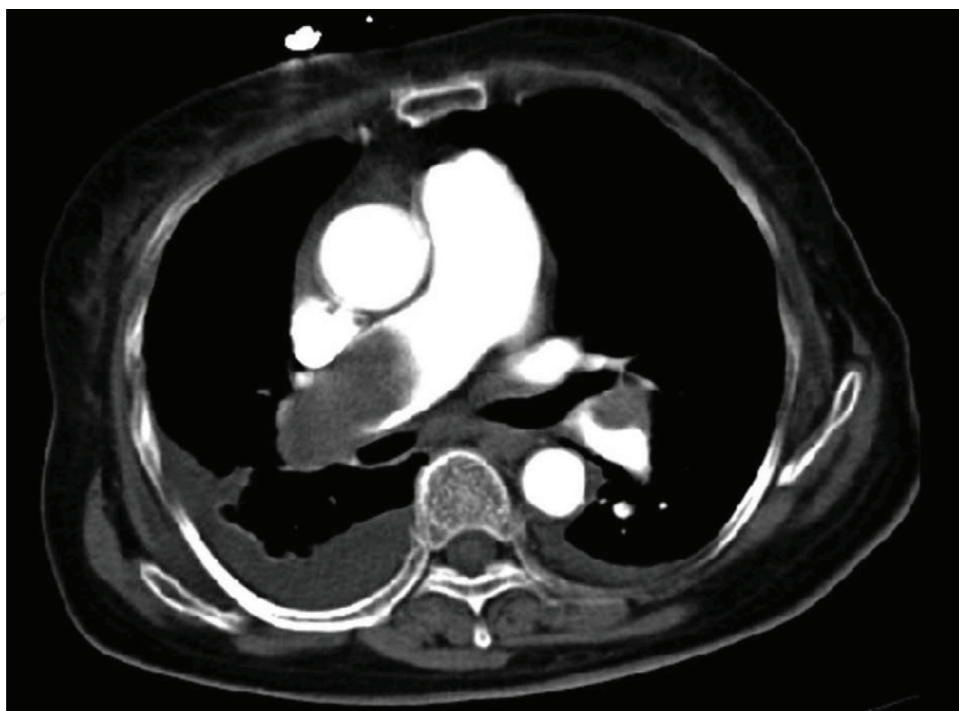• Possible concurrent performance of lower extremity

Figure 1. Massive PE as appears in contrast enhanced CT chest.

- venous imaging with CTV

- Ability to provide ancillary findings, which may affect management and prognosis [CTV (computed tomographic venography) and MDCTPA (multidetector computed tomographic pulmonary angiography)].

The most important recent diagnostic development from a surgical standpoint is transesophageal echocardiography. This modality may not visualize distal embolic material in the pulmonary vasculature but is more important to identify thrombus in transit, including paradoxical embolus in transit, and to permit evaluation of right ventricular function (**Figure 2**) [10].

4. Treatment

Oxygen should be administered to alleviate hypoxic pulmonary vasoconstriction, and it is likely that a severely affected patient will require intubation and ventilator support. Pharmacological agents, including vasopressors, may be instituted to stabilize the patient.

Pulmonary artery catheters, although obviously helpful in management, may occasionally emboli more thrombi because of the risk of dislodging further thromboembolic material [11].

4.1. Thrombolysis

Natural history of the clot in survivors of acute embolic events is fragmentation and progressive lysis. Thrombolytic agents dissolve thrombi by activating plasminogen to plasmin.
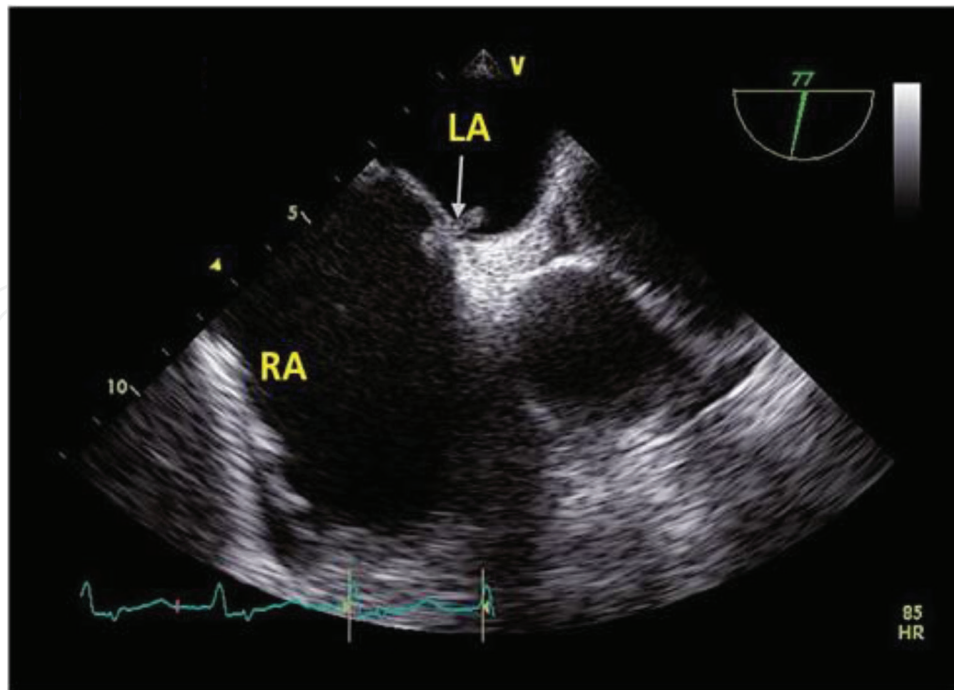
Figure 2. Echocardiogram demonstrating thrombus in transit across PFO.

Plasmin, when in proximity to a thrombus, degrades fibrin to soluble peptides. Circulating plasmin also degrades soluble fibrinogen and, to variable degrees, factors II, V, and VIII. In addition, increased concentrations of fibrin and fibrinogen degradation products contribute to coagulopathy by both inhibiting the conversion of fibrinogen to fibrin and interfering with fibrin polymerization. The thrombolytic agents currently reported for the treatment of acute pulmonary embolism include streptokinase, urokinase, recombinant tissue plasminogen activator (rt-PA, alteplase), anisoylated plasminogen streptokinase activator complex (APSAC, anistreplase), and reteplase [12]. There are newer agents arriving, like tenecteplase, lanoteplase, staphylokinase, and saruplase and are undergoing clinical testing.

Trials suggest a trend toward better results with thrombolytic therapy because of a more rapid diminution in right ventricular afterload and dysfunction. Compared with heparin therapy alone, thrombolytic agents carry a higher risk of bleeding problems, with up to 20% of patients experiencing a significant bleeding complication [13]. In general, thrombolytic therapy is contraindicated in patients with fresh surgical wounds, anemia, recent stroke, peptic ulcer, or bleeding dyscrasias.

4.2. Anticoagulation

Patients should be heparinized to prevent further propagation of thrombus at its origin and also in the pulmonary arterial tree. Intravenous (IV) heparin is started with an initial bolus dose of 70 U/kg followed by 18–20 U/kg/h. Heparin prevents propagation and formation of new thrombus. It rarely dissolves existing clot. Intrinsic fibrinolytic system will lyse fresh thrombi over a period of days to weeks. Evidence suggests early treatment of stable patients with acute pulmonary embolism with subcutaneous low-molecular-weight heparin (tinzaparin) given

once daily has been shown to be as effective and safe as IV heparin with respect to recurrent thromboembolism, major bleeding, and death [14].

4.3. Surgical embolectomy

Emergency pulmonary embolectomy was first described by Trendelenburg in 1908, using pulmonary artery and aortic occlusion, through a transthoracic approach. There were no surviving patients [14]. Later on, the first successful open embolectomy was performed and described by Sharp using cardiopulmonary bypass [15].

If a patient has been taken directly to the operating room without a definitive diagnosis, trans-esophageal or epicardial echocardiography and color Doppler mapping can confirm or refute the diagnosis in the operating room.

4.3.1. Indication for surgery

Emergency pulmonary thromboembolectomy is indicated for suitable patients with life-threatening circulatory insufficiency, where the diagnosis of acute pulmonary embolism has been established. Indications for acute surgical intervention include the following: hemo-dynamic instability, definitive diagnosis of pulmonary embolism in the main or lobar pul-monary arteries with compromise of gas exchange, unstable patients in whom thrombolytic or anticoagulation therapy is absolutely contraindicated, thrombus in transit or thrombus trapped within the right atrium, patent foramen ovale, or right ventricle.

Surgical embolectomy, as initial therapy for massive PE compared to thrombolytic therapy, has less early mortality rates and significantly less bleeding complications.

Patients who undergo surgical embolectomy after the failure of lysis clearly demonstrate a critically high-mortality rate.

CT-derived RV/LV ratio could be a useful parameter to identify candidates who might benefit from direct surgical therapy instead of thrombolysis [16].

4.3.2. Operative procedure

Intraoperative transesophageal echocardiography is now a routine in modern practice and greatly facilitates intraoperative decision-making particularly with regard to exploration of the right atrium and evaluation for clot in transit [10]. The groin vessels should be prepped into the field in case postoperative extracorporeal membrane oxygenation is necessary. Poor venous return from the inferior vena cava line only can be a result of clot in transit impacted in the cannula orifice (**Figure 3**). For this reason, the superior vena cava cannula is placed first so that partial bypass may be initiated if clot is dislodged from the inferior vena cava. Routine massage of the lower extremities and abdomen and open aspiration of the inferior vena cava return with cardiotomy suckers is better to extract additional material in transit. Tapes are passed around the superior vena cava and inferior vena cava, if patent foramen ovale or para-doxic embolus in transit has been identified by transesophageal echo. A brief episode of car-dioplegic arrest should be instituted to examine the left atrium (**Figure 4**).
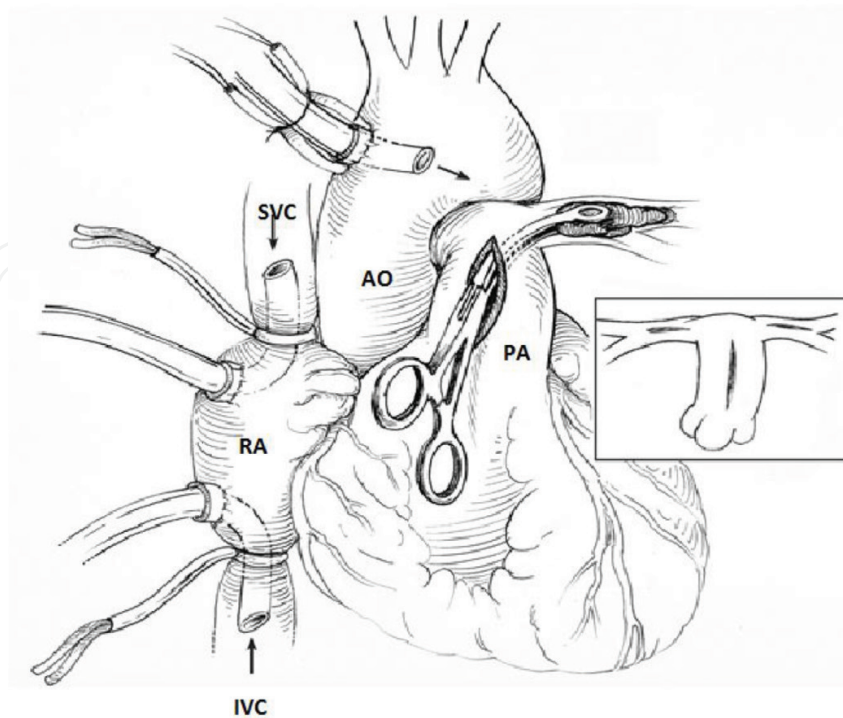
Figure 3. The pulmonary arteriotomy sites.

If embolus in transit is identified in the right atrium, this can be extracted via a standard right atrial incision without cardioplegic arrest (**Figure 4**). This approach provides optimal protection of the right ventricle during the procedure. If right atrial exploration is not required,
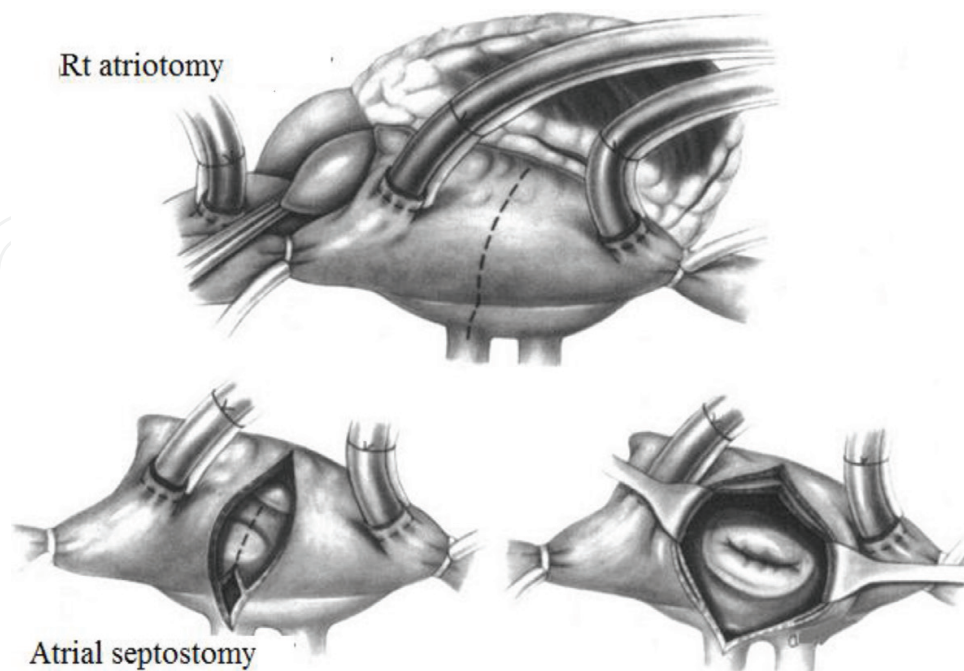

Figure 4. Approaches to explore thrombus in transit.

a bullet-tip sucker can be dropped into the right atrium via a stab wound with a purse-string suture. This will reduce the amount of blood passing through the right ventricle and into the pulmonary artery [11, 17].

The pulmonary vessels are extraordinarily fragile and rapidly taper in diameter, making rupture of the vessels a very real possibility. Thrombus in the left pulmonary artery is accessed via an incision beginning in the main pulmonary artery. Adequate access permitting direct visualization of the segmental vessels requires extension of the incision onto the left pulmonary artery itself. This may require division of the pericardial reflection over the ventral surface of the pulmonary artery (**Figure 5**).

A linear incision first in the posterior pericardium overlying the vessel and then in the vessel itself provides ready access to all of the lobar and segmental vessels. An incision such as this permits direct inspection of the right upper lobe branch, right middle lobe branch, and the segmental vessels to the right lower lobe. A flexible suction catheter passed in the pulmonary arteries with massage of the lungs, helps to dislodge smaller thrombi in the distal branches (**Figure 6**). The arteriotomies are primarily closed with running 4-0 Prolene. A final step in the procedure is insertion of an inferior vena cava filter via a purse-string suture on the right atrial appendage.
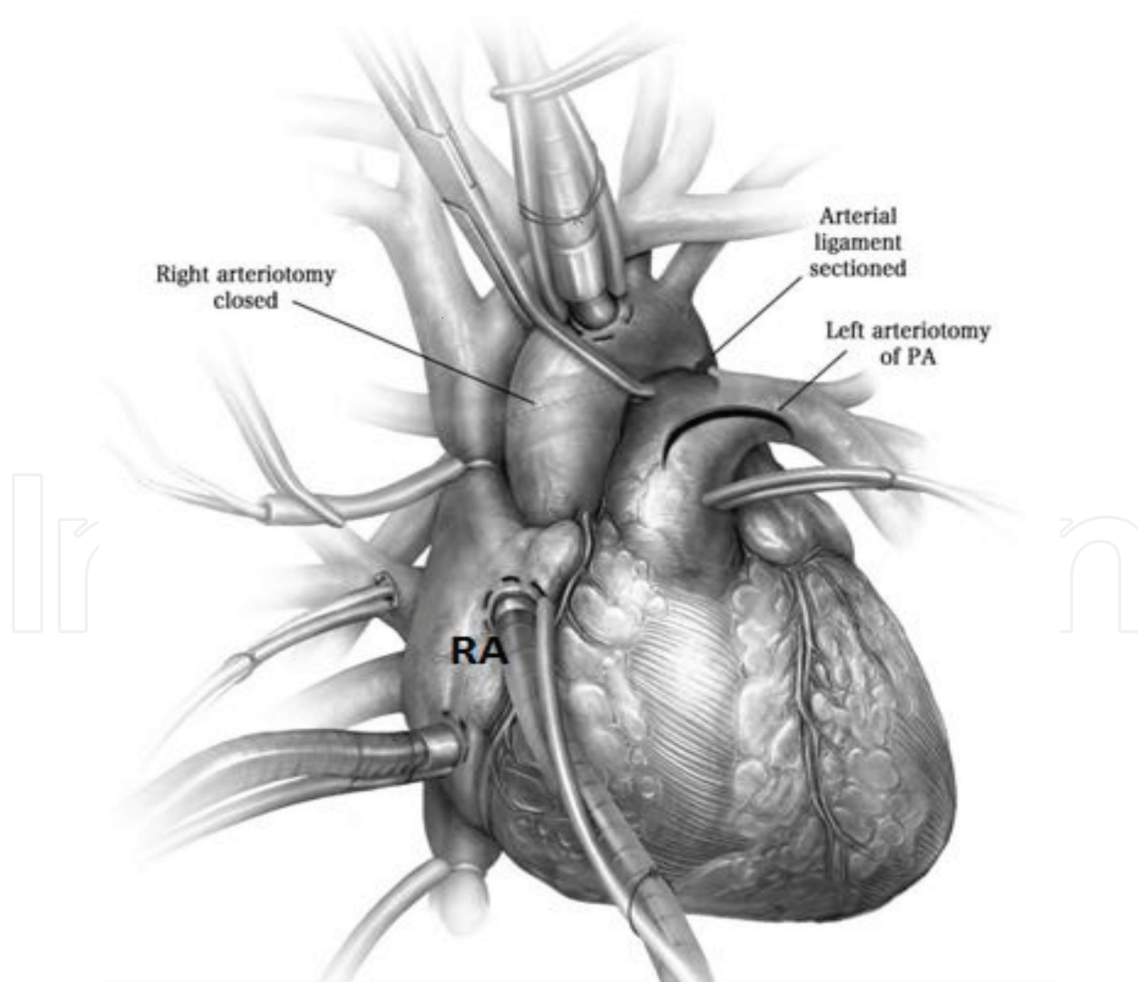

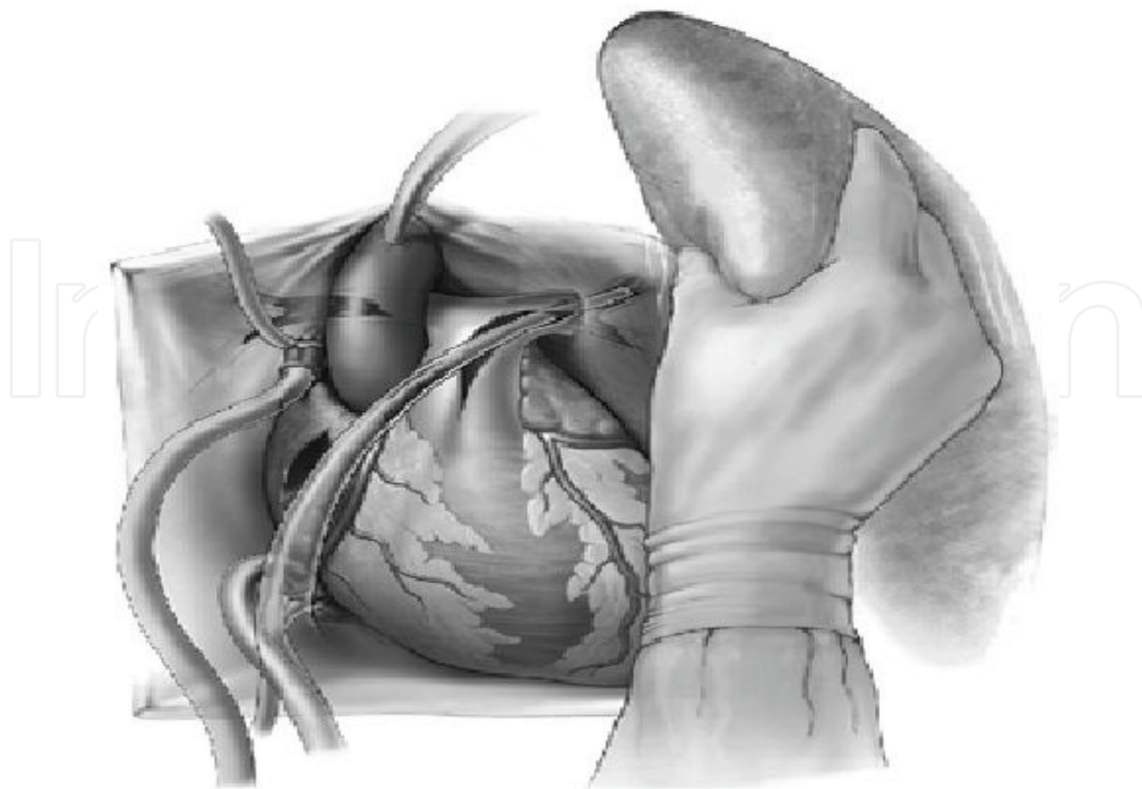**Figure 5.** Incision to explore distal pulmonary arteries.

Figure 6. Lung massage to mobilise peripheral thrombi.

4.3.3. Benefits of surgery

Pulmonary embolectomies demonstrate excellent early and late survival rates for patients with stable PE and unstable PE. These findings confirm pulmonary embolectomy as a beneficial therapeutic option for central PE, especially during the postoperative period when thrombolytic therapy is often contraindicated. Procoagulant risk factors such as endothelial injury, malignancy, and decreased mobility are common among postoperative patient populations across surgical specialties, but surgical options for the treatment of PE remain underappreciated and underutilized. With increasing surgical experience and improved outcomes, the role for pulmonary embolectomy in the acute setting may be expanding [18, 19]. All PE patients with imminent risk of hemodynamic decompensation due to severe RV impairment and central clot burden should be evaluated for surgical treatment [20]. Surgical embolectomy although normally confined to the most critical PE patients can be done with good long-term survival comparable to active medical treatment with thrombolysis despite the mortality risk inflicted by the surgical procedure. High-risk PE patients treated with surgical embolectomy had a significantly lower amount of residual emboli and pulmonary diffusion impairment than patients treated with thrombolysis. The clinical consequences of residual emboli were identified significant shorter 6-MWT, a higher mean pulmonary arterial pressure, and more dyspnea when compared to PE patients without residual emboli [21]. Residual clot burden is an independent risk factor for increased mortality at long-term follow-up [22]. Residual emboli after acute PE were found to be an independent predictor for chronic thromboembolic pulmonary hypertension, a severe late complication of acute PE [21, 22].

Pulmonary diffusion impairment after acute PE occurs more frequently in high-risk patients treated with thrombolysis compared to surgical embolectomy and was correlated with residual emboli. The surgical superiority on pulmonary morbidity is due to rapid removal of the total emboli resulting in fast restoration of normal pulmonary circulation, while thrombolysis either is unable to resolve the emboli due to its size or fractionates it into smaller parts which are carried to the peripheral pulmonary vasculature.

Current American Heart Association, European Society of Cardiology, and American College of Chest Physicians guidelines reserve surgical pulmonary embolectomy for central PE with hemodynamic instability and a contraindication for thrombolytic therapy, or when thrombolysis has failed [23, 24]. These treatment algorithms are based on limited data from small surgical series, and these practice patterns may be more reflective of scarce surgical expertise. Increasing evidence suggests that pulmonary embolectomy might be considered first-line therapy for select patients [25, 26]. This has resulted in the extension of eligibility for pulmonary embolectomy to include those who are hemodynamically stable but with demonstrative evidence of impending right ventricular failure [27].

5. ECMO in pulmonary embolism

Extracorporeal membrane oxygenation use (ECMO) for selected patients with massive PE is associated with good outcomes. Patients presenting in cardiac arrest have worse outcomes. Survival rates and neurological recovery are better when the cause of cardiac arrest is pulmonary embolism compared to other causes of cardiac arrest [11].

As an emergency procedure, standard femoro-femoral, venoarterial ECMO is instituted to ensure rapid and effective CPR in arrest or pre arrest patients, this can be achieved by use of smaller percutaneous cannulas limited to basic one arterial and one venous cannula (**Figure 7**) [5].

The tip of the venous catheter is advanced into the right atrium to obtain a flow rate of 2.5–4.0 l/min using an emergency pump-oxygenator circuit primed with crystalloid.

Mortality rates for emergency pulmonary thromboembolectomy vary widely between 11 and 92% in retrospective studies with varying operative techniques, preoperative hemodynamic state of the patient populations, and treatment plans. In general, greater surgical mortality was encountered if a patient had a preoperative cardiac arrest or required ECMO support [9, 28].

Prevention of recurrence should always be stressed upon in patients with successful outcomes, by addressing factors such as obesity, tobacco abuse, use of oral contraceptives, or postmenopausal hormone replacement. Consideration should be given to a search for occult malignancy. Consultation with a hematologist and systematic search for a prothrombotic state is routine. If no treatable cause is identifiable or patients have evidence of a hypercoagulable state, warfarin therapy is indicated for life.
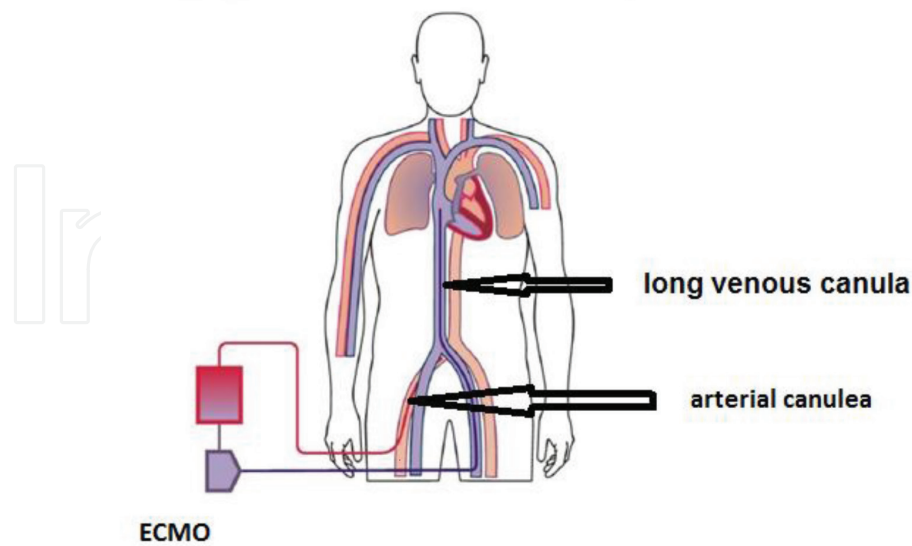
Figure 7. Arteriovenous ECMO circuit.

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