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MULTIMEDIA MANUAL OF CARDIO-THORACIC SURGERY

Retrograde pulmonary perfusion as an adjunct to standard pulmonary embolectomy for acute pulmonary embolism

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Summary

Mortality rates for pulmonary embolectomy in patients with acute massive pulmonary embolism have decreased in recent years. However, it still ranges from 30 to 45% when surgery is performed on critically ill patients, and the mortality rates reach 60% in patients who have experienced a cardiac arrest before the procedure. The causes of death in these patients are generally attributed to right heart failure due to persistent pulmonary hypertension, intractable pulmonary oedema, and massive parenchymal and intrabronchial haemorrhage. Clinical and experimental findings indicate that venous air embolism causes severe or even lethal damage to the pulmonary microvasculature and the lung parenchyma consequent to the release of endothelium-derived cytokines. These findings are similar to those observed when severely compromised patients undergo pulmonary embolectomy for air entrapped in the pulmonary artery during embolectomy, which may lead to fatal outcomes. Retrograde pulmonary perfusion (RPP), besides enabling the removal of residual thrombotic material from the peripheral branches of the pulmonary artery, fills the pulmonary artery with blood and prevents pulmonary air embolism. We believe that the use of RPP as an adjunct to conventional pulmonary embolectomy decreases the morbidity and mortality rates associated with pulmonary embolectomy in critically ill patients.

Keywords: Pulmonary embolism • Pulmonary embolectomy • Retrograde pulmonary perfusion

BACKROUND

The recently observed improvement in the results of surgical pulmonary embolectomy for massive pulmonary embolism has largely been credited to recent advances in diagnostic techniques and the identification of criteria for the timely recognition of critical factors, thus allowing for the judicious selection of patients and early surgery [1–8]. However, reported mortality rates range from 30 to 45% when embolectomy for massive pulmonary embolism is performed on haemodynamically unstable patients, reaching 60% when those patients have experienced cardiac arrest before the procedure [1, 2]. Furthermore, an extensive review of the literature [9] has shown that the mortality rate for pulmonary embolectomy performed under these circumstances has not changed over the last 50 years, measuring, on average, 74%. The causes of death are usually attributed to right ventricular failure, persistent pulmonary hypertension, pulmonary oedema or massive parenchymal and intrabronchial haemorrhage [8, 10–13]. In addition to the incomplete removal of thrombotic material in the pulmonary circulation being held responsible for persistent pulmonary hypertension and the consequent right ventricular failure, we believe that pulmonary air embolism should be considered a contributor to negative outcomes in these patients [14–19]. We have developed a simple technique of retrograde pulmonary perfusion (RPP) as an adjunct to conventional pulmonary embolectomy that facilitates both the removal of residual thrombotic material from the peripheral branches of the pulmonary artery and, by filling the arterial tree with blood, eliminates the entrapped air and the consequent negative effects that it may induce. This technique has been previously reported by us [20].

SURGICAL TECHNIQUE

A median sternotomy is performed and a standard normothermic cardiopulmonary bypass with bicaval cannulation is adopted. The arterial line is connected to a Y connector. One branch of the connector is joined to the arterial cannula, which is inserted into the ascending aorta. The other branch of the connector is joined to a 20-Fr clamped plastic cannula, which is inserted into the left atrium through a purse-string suture placed on the right upper pulmonary vein. The set-up of the extracorporeal circuit is shown in Figure 1. After institution of normothermic cardiopulmonary bypass, cross-clamping of the aorta and antegrade infusion of warm blood cardioplegic solution, a longitudinal incision 4–5 cm in length is made in the pulmonary artery trunk distal to the pulmonary valve. In cases in which the pulmonary artery is particularly short, the incision is extended beyond the bifurcation into the proximal right and left pulmonary arteries. The thrombotic material is carefully extracted by means of forceps and suction (Video 1). The right atrium and ventricle are then explored, and all visible clots are removed. Then, while the pulmonary artery is still open, the clamp

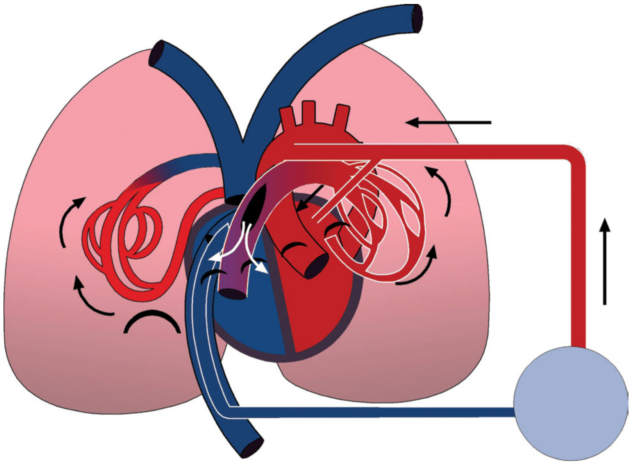
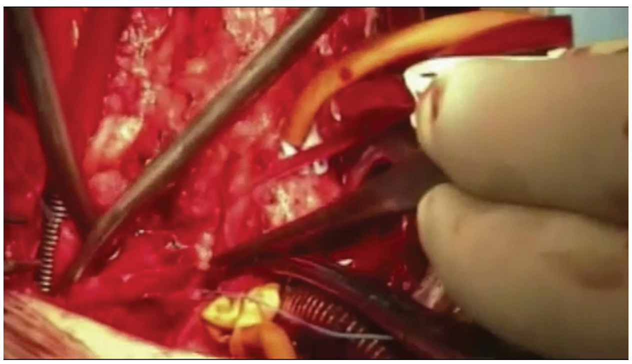
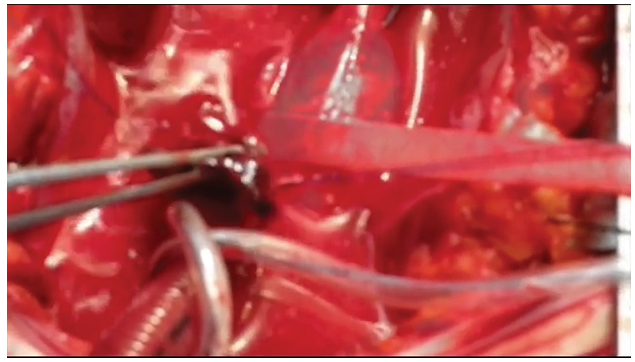


Figure 1: Extracorporeal circuit set-up for retrograde pulmonary perfusion. A 20-Fr cannula connected to the arterial line is inserted into the left atrium and it is released, allowing blood to flow at a pressure of 40 mmHg into the left atrium after standard pulmonary embolectomy is performed. Blood flows into the pulmonary circulation in a retrograde fashion, expelling residual thrombotic material and air from the pulmonary arteriotomy.

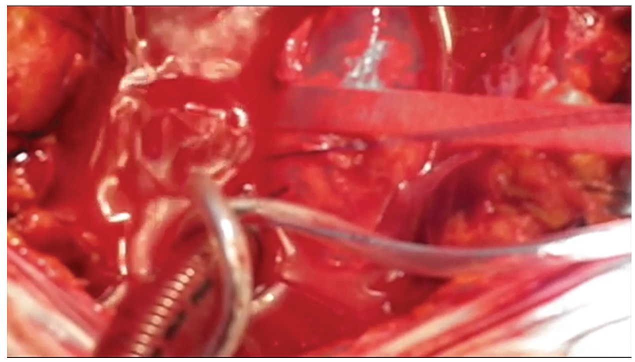


Video 1: Standard pulmonary embolectomy. Thrombotic material is extracted through a pulmonary arteriotomy.

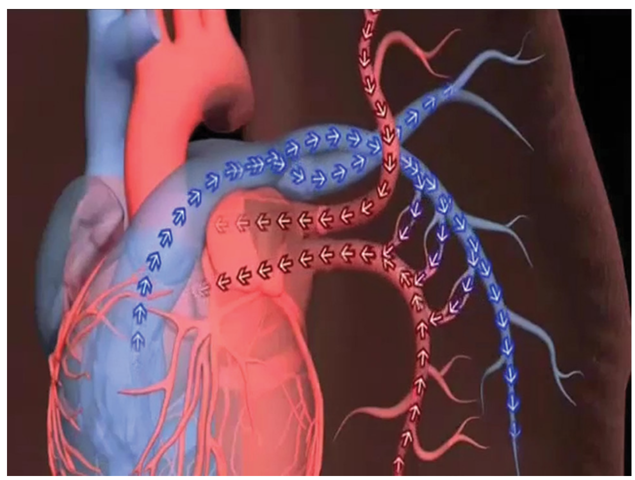
on the cannula connecting the arterial line in the extracorporeal circuit with the left atrium is released. Blood fills the left atrium; after ~1 min, the blood begins flowing into the pulmonary artery in a retrograde fashion at a pressure of ~40 mmHg (Video 1). While most of the retrograde perfusate flows externally through the pulmonary arteriotomy, in order to prevent left ventricular distension, manual compression and suction through a vent placed into the ascending aorta are performed. The lungs are repeatedly inflated in order to mobilize any residual fragment of thrombotic material that may be lodged in the distal branches of the pulmonary artery and to facilitate the elimination of residual air bubbles. Residual clots expelled by the retrograde flow of blood are aspirated (Video 2). Subsequently, with the retrograde blood flow, all air is progressively eliminated from the pulmonary circulation (Video 3). After ~5 min, the retrograde flow is interrupted and the pulmonary arteriotomy is closed. In most cases, it is possible to perform the closure of the pulmonary arteriotomy by means of a direct suture; however, in a limited number of cases, in the presence of a particularly thin and fragile pulmonary artery, a bovine pericardial patch was employed. The left atrial cannula is disconnected from the arterial line and is used as a vent. The aorta is declamped, and the patient is weaned from cardiopulmonary bypass by the standard method.



Video 2: Retrograde pulmonary perfusion. Residual thrombotic material is expelled from the pulmonary circulation by the retrograde pulmonary perfusion.



Video 3: Retrograde pulmonary perfusion. Retrograde pulmonary perfusion eliminates air from the pulmonary circulation.

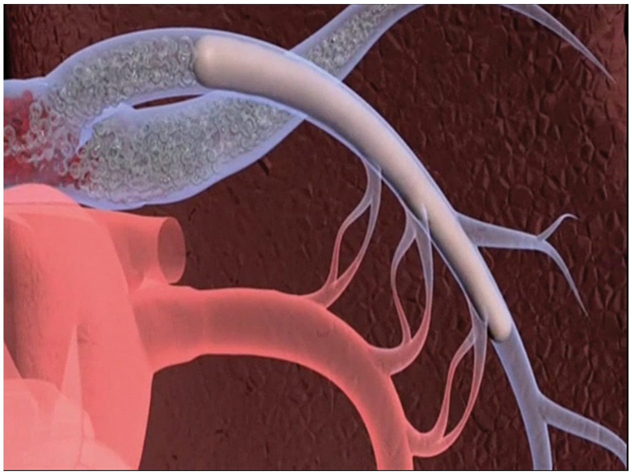


Video 4: Retrograde pulmonary perfusion. Animation scheme: normal pulmonary circulation.

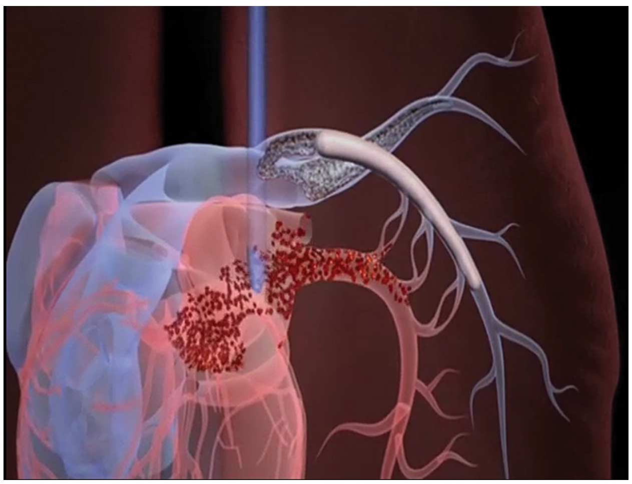
Animated schemes of the normal pulmonary circulation, of its variations secondary to the pulmonary embolism and the effect of RPP are shown in Videos 4–6.

RESULTS

Our experience, dating from January 1985 to June 2005, with a series of 21 consecutive critically ill patients with massive



Video 5: Retrograde pulmonary perfusion. Animation scheme: acute pulmonary embolism.



Video 6: Retrograde pulmonary perfusion. Animation scheme: effect of retrograde pulmonary perfusion.

pulmonary embolism who underwent pulmonary embolectomy supplemented by RPP has been previously reported [20]. Of this series, 2 patients had a cardiopulmonary arrest in the ward and were taken to the operating theatre under external cardiac massage. Each underwent emergency surgery on the sole basis of a presumptive clinical diagnosis of pulmonary embolism, which was later confirmed at the operating table. The other 19 patients exhibited acute, severe haemodynamic and respiratory compromise that required inotropic support. These patients presented with either a contraindication or a failure of response to the use of thrombolytic agents and were referred for emergent surgical pulmonary embolectomy. In these 19 cases, the diagnosis was established by transthoracic echocardiography and pulmonary angiography. Three patients belonging to this group of 19 experienced a cardiac arrest during induction of anaesthesia, and surgery was initiated while resuscitative manoeuvres were undertaken. There were neither in-hospital deaths, nor major postoperative complications. All patients were discharged from the hospital on anticoagulant medication by the 10th postoperative day.

Since the publication of that report, a further series of 13 patients with acute massive pulmonary embolism, with clinical presentation analogous to that exhibited by the patients belonging to the previous group, were operated upon in our institution with the technique herein described. Of these, 9 were men and 4 were women; the average age was 65, 75 ± 15, 50 (24–79) years. Two patients had experienced a cardiac arrest prior to surgery. There was 1 death secondary to an uncontrollable haemorrhage from a massively infarcted lung. The other patients did not present any significant postoperative complications and experienced a regular postoperative course. The hospital mortality rate for the total group of 34 was 2.9%.

DISCUSSION

Recently published reports state that results of open pulmonary embolectomy have improved, with mortality rates ranging from 8 to 27% [1, 2, 5–7]. However, these series consisted primarily of patients who were not in a critical condition at the time of surgery [1–4]. Mortality rates for open pulmonary embolectomy remain high, ranging from 30 to 45%, when surgery is performed on critically ill patients who have massive pulmonary embolism [1, 5]. It has been suggested that this high death rate is the consequence of reserving pulmonary embolectomy as a last-resort treatment for patients in severe shock and for those patients in whom less invasive forms of treatment have failed [2, 3, 8].

Recent advances in establishing diagnostic criteria allowing early recognition of critical factors prior to the development of an irretrievable clinical condition have improved results of open surgical embolectomy [5–7]. However, mortality rates for pulmonary embolectomy in patients who experienced cardiac arrest before surgery remain around 60% [2, 8].

The causes of death in patients who undergo pulmonary embolectomy have been attributed to right heart failure secondary to persistent pulmonary hypertension [8, 11], intra-alveolar and interstitial pulmonary oedema with normal left-sided pressures, and massive parenchymal and intrabronchial haemorrhage [10, 12, 13]. The common pathological finding is pulmonary haemorrhagic infarction [10, 12].

Incomplete removal of thrombotic material lodged in the distal pulmonary arterial tree is considered an important cause of persistent pulmonary hypertension [8, 11]. The extraction of clots from the distal branches of the pulmonary artery is commonly performed through an extended pulmonary arteriotomy by suction and the use of standard or gallbladder-stone forceps, Fogarty catheters or similar instruments. Mechanical injury to the pulmonary arterial wall by these means is thought to be responsible for the parenchymal and endobronchial bleeding [3, 13]. The danger of injury to distal vessels has prompted recommendations to avoid blind instrumentation and to limit extraction to visible clots [3].

Scant attention has been devoted to the role of air embolism in causing these adverse, often fatal effects during pulmonary embolectomy. In fact, abundant experimental [15, 16] and clinical [17, 19] evidence indicates that pulmonary air embolism releases endothelium-derived cytokines, damaging and occluding the microvasculature, with consequent pulmonary hypertension, pulmonary oedema and injury to the lung parenchyma. These findings are strikingly similar to those presented by critically ill patients who undergo pulmonary embolectomy. We believe that air entrapped in the pulmonary artery during pulmonary embolectomy is likely a major cause of the negative outcomes in these patients [20].

RPP performed as an adjunct to pulmonary embolectomy appears to confer two benefits: it helps to flush out residual thrombotic material lodged in the distal pulmonary arterial branches, and it prevents air embolism within the pulmonary artery, thus helping to prevent the associated detrimental effects.

Since the initial report in 1966 of the clinical use of RPP for pulmonary embolism, in which the authors described the use of retrograde injection of a fibrinolysin solution into the pulmonary veins in 3 patients with 1 long-term survivor [21], RPP has been successfully used as an aid to treat acute pulmonary embolism in a few isolated cases [22]. In an additional instance, RPP was used to flush a cyanoacrylate glue obstruction from the distal pulmonary artery after its embolization from a cerebral arterio-venous malformation in a 3-year-old boy [23]. Recently, Zarrabi *et al.* reported the use of retrograde pulmonary embolectomy in a series of 30 patients: 11 had developed severe haemodynamic and/or respiratory compromise; of these, 3 had experienced a cardiac arrest. The total in-hospital mortality rate was 6.6%. In contrast to the simple irrigation of the left atrium with oxygenated blood derived from the arterial line of the extracorporeal circulation that we employ, these authors have resorted to individual irrigation of each pulmonary vein after having incised the interatrial septum [24].

The technique of RPP is simple, and it appears effective in reducing the morbidity and mortality that have accompanied pulmonary embolectomy. Should further clinical investigation confirm that RPP improves the outcomes of surgical pulmonary embolectomy, wider implementation of the procedure would be warranted.

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