

Original Articles

Perfusion Techniques for Pulmonary Thromboendarterectomy Under Deep Hypothermia Circulatory Arrest: A Case Series

Bingyang Ji, MD;*† Jinping Liu, MD;* Yongbo Wu, MD;‡ Guyan Wang, MD;§
Zhengyi Feng, MD;* Mingzheng Liu, MD;§ Cun Long, MD;* Yunhu Song, MD‡

*Department of Cardiopulmonary Bypass, Cardiovascular Institute and Fuwai Hospital, PUMC and CAMS, Beijing, China; †Department of Pediatrics, Hershey Medical Center, Penn State College of Medicine, Hershey, Pennsylvania; ‡Department of Cardiac Surgery, Cardiovascular Institute and Fuwai Hospital, PUMC and CAMS, Beijing, China; and §Department of Anesthesiology, Cardiovascular Institute and Fuwai Hospital, PUMC and CAMS, Beijing, China

Abstract: Pulmonary thromboendarterectomy (PTE) is a complicated surgical procedure that is an effective treatment in reducing pulmonary artery pressure and pulmonary vascular resistance for chronic thromboembolic pulmonary hypertension. Chronic thromboembolic pulmonary hypertension usually results from incomplete lysis of a large organized thrombus in the main pulmonary artery and secondary branches, leading to pulmonary hypertension, right ventricular failure, and subsequent death because of heart failure. Between March 1997 and April 2005, 30 PTE operations were performed in Fuwai Hospital, Beijing, China. They were 24 men and 6 women, with an average age of 45.7 ± 11.4 years and average disease history of 48 ± 12.6 months. Twelve of them were in New York Heart Association (NYHA) class 4, and 18 were in class 3. Seventeen cases were found with deep venous thrombosis (DVT), and inferior vena cava filters were implanted before surgery. The mean systolic pulmonary pressure was 91.4 ± 22.4 mmHg, mean pressure of arterial oxygen (PaO_2) was 56.2 ± 8.6 mmHg, mean cardiac index (CI) was 1.64 ± 0.47 L/min/m², and mean saturation of arterial oxygen (SaO_2) was 0.90 ± 0.05 . All operations were performed using the PTE procedure under deep hypothermia and intermittent circulation arrest. Perfusion management consisted of myocardial, cerebral protection, lung protection, and deep hypother-

mia with multiple periods of circulatory arrest and reperfusion at hypothermia, ultrafiltration, and cell-saving techniques. One patient died of infective shock post-operatively. Four cases experienced complications of the central nervous system. The mean cardiopulmonary bypass time was 191.1 ± 34.4 minutes, the mean aortic clamping time was 95.1 ± 27.8 minutes, and mean circulation arrest time was 47.7 ± 12.9 minutes. Improvement of hemodynamic status occurred immediately after surgery. Mean pulmonary artery pressure decreased from 91.4 ± 22.4 to 48.3 ± 10.7 mmHg, and CI increased from 1.64 ± 0.47 to 2.58 ± 0.51 L/min/m². PaO_2 increased from 56.2 ± 8.6 to 88.9 ± 6.0 mmHg and SaO_2 increased from 0.90 ± 0.05 to 0.97 ± 0.01 . Twenty-six cases were followed for 36.8 months: 22 in NYHA class 1, 3 in class 2, and 1 in class 3. PTE is an effective treatment for chronic thromboembolic pulmonary hypertension. The key to success is to adopt synthesized measures to protect the vital organ under deep hypothermic circulatory arrest (DHCA) from ischemia and reperfusion injury. Appropriate patient selection, perioperative management, improved techniques, and experience can optimize outcome. **Keywords:** pulmonary thromboendarterectomy, cardiopulmonary bypass, deep hypothermic circulatory arrest. *JECT. 2006;38:302–306*

Chronic thromboembolic pulmonary hypertension is a rare disease but is the only type of pulmonary hypertension that can be successfully treated with surgery. Pulmonary thromboendarterectomy (PTE) is a complex but ef-

fective surgical procedure for significantly reducing pulmonary artery pressure and pulmonary vascular resistance. The reduction of right ventricular after-load results in a decrease of right heart dimensions and functional improvement immediately after PTE (1–5). From March 1997 to April 2005, 30 PTE procedures were performed in Fuwai Hospital, Beijing, China. The purpose of this paper is to summarize the perfusion techniques for PTE that may involve the protection of multiple organ systems, including the brain, renal, heart, and lung, and deep hypothermia with multiple periods of circulatory ar-

Address correspondence to: Bingyang Ji, MD, Senior Researcher Assistant of Pediatrics, Penn State College of Medicine, Department of Pediatrics-085, 500 University Drive, PO Box 850, Hershey, PA 17033-0850. E-mail: bingyangji@psu.edu.

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rest and reperfusion using hypothermia, cell-saving, and ultrafiltration techniques.

Epidemiology and Pathophysiology of Chronic Thromboembolic Pulmonary Hypertension

The reason that most patients with chronic thromboembolic pulmonary hypertension present late in the course of the disease is because an embolic event can occur without symptoms so that symptomatic pulmonary embolism is often overlooked or misdiagnosed (6,7). Although its thromboembolic basis has been questioned, some evidence still supports this reason regardless of whether there is a documented history of acute venous thromboembolism (8,9). Some factors also could be considered such as local cellular abnormalities (10), hereditary risk factors, coagulopathies, and vascular abnormalities.

The extent of vascular obstruction is a major determinant of pulmonary hypertension. In the majority of patients, >40% of the pulmonary vascular bed is obstructed (11). Hemodynamic progression may involve recurrent thromboembolism or in situ pulmonary artery thrombosis. Chronic thromboembolic pulmonary hypertension causes right ventricular pressure overload and reduced left ventricular pre-load. Right ventricular enlargement causes a leftward shift of the inter-ventricular septum. The change impairs diastolic and systolic function of the left ventricle. Finally, the cardiac index decreases and results in heart dysfunction (12). The dilatation and alteration of right ventricular geometry leads to annular dilatation of the tricuspid valve, causing more strain on the right ventricle, further increasing tricuspid valve incompetence.

Clinical Manifestations and Diagnosis

Similar to other types of pulmonary hypertension, progressive exertional dyspnea and exercise intolerance are characteristic of thromboembolic pulmonary hypertension. History, physical examination, chest radiography, pulmonary function tests, transthoracic echocardiography, and computed tomography (CT) scans are all important routine tests to establish diagnosis. A non-invasive lung perfusion scan plays a pivotal role in differentiating between primary vs. obstructive pulmonary hypertension. Pulmonary angiography seems to be the key diagnostic procedure, to confirm the diagnosis and establish surgical feasibility (7). When diagnosis remains unclear, pulmonary angiography can contribute to the diagnostic evaluation (11). Right heart catheterization should be considered in any patient with unexplained dyspnea and segmental or larger defects on ventilation-perfusion scanning, especially if there is echocardiographic evidence of right atrial or right ventricular dysfunction (11).

Incidence and Prognosis

The real incidence of chronic thromboembolic pulmonary hypertension is unknown. Because of the nonspecific

nature of the symptoms and manifestations and the complexity of diagnosis, many patients with chronic thromboembolic pulmonary hypertension are overlooked and misdiagnosed. In Fuwai Hospital, there were 442 patients diagnosed with PTE from 1974 to 2002. Both the number of PTE in-patients in every year and their proportion of all the in-patients in Fuwai Hospital increased, from <5 cases averaging 0.1152% every year in the 1970s, to 48–79 cases averaging 0.5866% every year in the 2000s. The trend is still increasing (13).

If the condition of chronic thromboembolic pulmonary hypertension is not treated surgically, survival rates are low. The in-hospital mortality of patients with PTE decreased, from 20% in the 1970s to 5.8% in the 2000s in Fuwai Hospital. Patients in the study of Riedel et al. (14) had a 5-year survival rate of 30% when mean pulmonary artery pressure was >40 mmHg at the time of diagnosis and only 10% when mean pulmonary artery pressure exceeded 50 mmHg. Medically treated patients of Lewczuk et al. (15) had a 12% and 50% mortality rate when mean pulmonary artery pressures were <30 and >30 mmHg, respectively.

Treatment of PTE

Treatment options are limited, and survival rates are inferior to those achieved with pulmonary thromboendarterectomy. Treating patients with continuous anticoagulation, vasodilators, or thrombolytic agents is seldom effective and leads to poor prognosis (15,16). These patients die, mostly as a result of right ventricle failure.

Indications and Contraindications

PTE is considered in symptomatic patients who have hemodynamic or ventilatory impairment at rest or with exercise. Fedullo et al. (11) states that the only absolute contraindication to thromboendarterectomy is the presence of severe underlying lung disease, either obstructive or restrictive. Thrombi should be considered surgically accessible, pulmonary vascular resistance should be 300 dynes s/cm (17), and patients must be New York Heart Association (NYHA) classes 2 to 4. In Leuven, three contraindications are considered: unrepairable coronary atheromatosis, significant comorbidity, and septic, parasitic, or neoplastic pulmonary emboli (18) Jamieson et al. (19) recently considered that there is no degree of embolic occlusion that is inaccessible, and there is no degree of right ventricular impairment or any level of pulmonary vascular resistance considered inoperable.

MATERIALS AND METHODS

Patients

The study was approved by Fuwai Hospital, Beijing, China. From March 1997 to April 2005, 30 patients were diagnosed with PTE in Fuwai Hospital, Beijing, China.

There were 24 men and 6 women, with an average age of 45.7 years and an average of history of 48.0 months. Twelve were in NYHA class 4 and 18 in were in class 3. Seventeen cases were found with deep venous thrombosis (DVT) and were implanted with inferior vena cava filters before surgery. The mean systolic pulmonary arterial pressure (SPAP) was 91.4 ± 22.4 mmHg, mean pressure of arterial oxygen (PaO_2) was 56.2 ± 8.6 mmHg, cardiac index (CI) was 1.64 ± 0.47 L/min/m², and mean saturation of arterial oxygen (SaO_2) was $90 \pm 5\%$ (Table 1).

Anesthesia and Monitoring

Anesthesia was induced by intravenous administration of fentanyl citrate and vecuronium bromide and was maintained with intravenously administered propofol and inhalation of isoflurane. Preparation was the same as a normal cardiac procedure in terms of monitoring: radial artery line, central venous line, Swan-Ganz catheter, electrocardiography, rectal and esophageal thermometer, and transesophageal echocardiography.

Surgery Procedure

The chest was opened through a median sternotomy. Cardiopulmonary bypass (CPB) was established by bicaval and aortic cannulation, and the body was cooled to a nasopharyngeal temperature of 18°C. The central pulmonary arteries were opened, and a dissection plane developed that was followed to segmental level. Deep hypothermic circulatory arrest (DHCA) was used in all procedures to achieve clear visualization during peripheral dissection. Repeated periods of circulatory arrest limited to 20 minutes were used; usually endarterectomy of one pulmonary arterial bed could be accomplished within one 20-minute period. After complete endarterectomy, extracorporeal circulation was resumed, and the patient was rewarmed. Concomitant cardiac procedures (closure of patent foramen ovale, tricuspid valve reconstruction, or

coronary artery bypass grafting) usually were performed during the rewarming period. Post-operatively, hemodynamic monitoring was continued for 48 ± 72 hours after the operation. Decreased systemic vascular resistance was treated with norepinephrine if necessary. Residual pulmonary hypertension was treated with nitrates, inhalation of prostacyclin, or iloprost. After hemodynamic stabilization, the patients were weaned from ventilator support and extubated once normal gas exchange was present. Anticoagulation was started with intravenous heparin 6 hours after surgery.

Perfusion Management

CPB Preparation and Circuit Priming: The Stockert heart-lung machine (Munich, Germany) with roller pump and membrane oxygenator, a tubing pack with a cardioplegia delivery (blood: crystalloid = 4:1) system (Perfect, Beijing, China), and an arterial filter (Xi Jing, Xi an, China) were used. Three suction lines were placed on the pump: two standard and one additional suction line to a blood collection reservoir from which the suctioned blood was processed during or after the CPB by cell-saving. Cell-saver was applied from "skin to skin" in all cases. A standard aortic cannula and two venous cannulae for bicaval cannulation were used. Filter for ultrafiltration was also routinely primed before bypass in all 30 cases. Prime for the circuit consisted of 1.0 L lactated Ringer solution and 1 L Gelofusion, 100 mL of albumin solution 20%, 250 mL of mannitol 20%, 1 mL of heparin, 2.5 million KIU aprotinin, and 1 g of antibiotics. During CPB, all patients received a high-dose anti-fibrinolytic and anti-inflammatory response treatment with aprotinin (5 million KIU: 2.5 million in prime; 2.5 million KIU intravenously after induction). A heating/cooling blanket under the patient was used for cooling and rewarming during the bypass. During CPB, we monitored the following parameters: continuous inline venous oxygen saturation, hemoglobin and hematocrit, nasopharyngeal and rectal temperatures, ECG, and arterial, central venous, and CPB pressures. Times of CPB, aortic cross-clamping, circulatory arrest, and rewarming were recorded. We use the blood gas pH-stat method for our arterial blood gases from -18°C and converted to α -stat during rewarming (20).

CPB Procedure: CPB was started under full heparinization (4 mg/kg heparin) and when activated clotting time (ACT) reached 760 seconds (Celite ACT). This level of anticoagulation was maintained during the entire process. Standard aortic and bicaval cannulation was performed. Separate caval cannulation was necessary for multiple reasons. CPB was started, and cooling was immediately started to decrease temperature to 18°C rectal temperature. During the cooling period, as much of the dissection as possible of the right pulmonary artery was performed. When visibility was too limited by bronchial arterial back-

Table 1. Patient characteristics (mean \pm SD) (n = 30).

Age (years)	45.7 \pm 11.4
Weight (kg)	72 \pm 19.4
BSA (m ²)	1.74 \pm 0.4
Sex (male/female)	24/6
History (Mos)	48 \pm 12.6
NYHA	3 18/4 12
Dyspnea	29
Low extremities edema	21
Syncope	5
Hemoptysis	11
DVT (IVC filters)	17
Associate procedure	
TV annuloplasty	3
CABG	1
PV annuloplasty	1

BSA, body surface area; NYHA, New York Heart Association functional class; DVT, deep venous thrombosis; IVC, inferior vena cava; TV, tricuspid valve; CABG, coronary artery bypass graft; PV, pulmonary valve.

Table 2. Perioperative characteristics (mean \pm SD) (n = 30).

CPB time (min)	191.1 \pm 34.4
Cross-clamp time (min)	95.1 \pm 27.8
Circulatory arrest time (min)	47.7 \pm 12.9
Hypothermic reperfusion (min)	34 \pm 8
Rewarming time (min)	71 \pm 17
Mechanical ventilation time (h)	85.1 \pm 24.6
Operative death	1/30
Central nerve system complication	4/30
Reperfusion pulmonary edema	7/30
Follow-up (cases)	26/30
Follow-up time (mos)	36.8 \pm 9.8
After surgery NYHA class	
	Follow up 26 cases
	22 1
	3 2
	1 3

CPB, cardiopulmonary bypass; NYHA, New York Heart Association functional class.

bleeding, the endarterectomy was halted until an adequate temperature was reached to apply DHCA. At an esophageal temperature of 18°C, we changed from α -stat blood gas monitoring to pH-stat. An iced cover was placed around the patient's head for cerebral protection. Before reaching a temperature of 18°C, the aorta was cross-clamped, and cold blood cardioplegia was perfused; the route of delivery was exclusively antegrade. The initial dose of cardioplegia was 15 mL/kg of body weight. The rest of the time, the heart was protected by systemic and local hypothermia. A second dose of cardioplegia was administered before the second circulatory arrest. The arterial pump was stopped, and the arterial line was clamped. When no more blood returned to the venous reservoir, the venous line was clamped, and the patient's blood was recirculated through the oxygenator and arterial filter through a bypass line. When the left endarterectomy was completed, CPB was restarted. When inline venous oxygen saturation reached 85%, rewarming began. It was important to keep the differences between water and blood temperature at <5°C (20,21). The iced cover around the head was removed. During rewarming, additional procedures were performed. When an esophageal temperature of 30°C was reached, we returned from pH-stat blood gas monitoring to the α -stat method. When the aorta was unclamped and the heart began to beat, 250 mL of mannitol 20% was administered as an oxygen free radical scavenger. This also increased the volume of urine output. Blood collected in the blood collection reservoir was processed by cell-saver, either to optimize hematocrit levels during CPB or to be donated after CPB. When the patient's rectal temperature reached 37.5°C, the patient was weaned from the heart-lung machine.

RESULTS

One patient died of infective shock post-operatively. Operative mortality was 3.3% (1/30). Complications of the

Table 3. Comparison of PaO₂, SaO₂, and SPAP between the preoperative and postoperative time.

	Pre-operative	Post-operative
PaO ₂ (mmHg)	56.2 \pm 8.6	88.9 \pm 6.0
SaO ₂ (%)	90 \pm 5	97 \pm 1
CI (L/min/m ²)	1.64 \pm 0.47	2.58 \pm 0.51
SPAP (mmHg)	91.4 \pm 22.4	48.3 \pm 10.7

PaO₂, pressure of arterial oxygen; SaO₂, saturation of arterial oxygen; CI, cardiac index; SPAP, systolic pulmonary arterial pressure.

central nervous system occurred in 4 cases (13.3%), and reperfusion pulmonary edema occurred in 7 cases (23.3%). The mean CPB time was 191.1 \pm 34.4 minutes, the mean aortic clamp time was 95.1 \pm 27.8 minutes, and the mean circulation arrest time was 47.7 \pm 12.9 minutes. The mechanical ventilation time was 85.1 \pm 24.6 hours. Hemodynamic improvement occurred immediately after surgery. Mean pulmonary artery pressure decreased from 91.4 \pm 22.4 to 48.3 \pm 10.7 mmHg, PaO₂ and SaO₂ increased from 56.2 \pm 8.6 to 88.9 \pm 6.0 and from 0.90 \pm 0.05 to 0.97 \pm 0.01 mmHg (Table 2), respectively. Twenty-six cases were followed over 36.8 months: 22 in NYHA 1, 3 in class 2, and 1 in class 3 (Tables 2 and 3).

DISCUSSION

Chronic thromboembolic pulmonary hypertension has a poor spontaneous prognosis with high morbidity and mortality. The aim of PTE is to reduce pulmonary vascular resistance and improve long-term outcome (3). Although this procedure can significantly reduce elevated pulmonary arterial pressures and vascular resistance, it is not without substantial risk, because the perioperative morbidity and mortality after PTE are high in comparison with other, more routine DHCA procedures (20–22). Although the implication of surgery should not be underestimated, appropriate patient selection, perioperative management, improved techniques, and experience in perfusion can optimize outcome.

In the literature, there are some modified techniques during DHCA in PTE for avoiding the central nerve system complications; low flow perfusion (500–1000 mL/min) (23), 20-minute selective antegrade cerebral perfusion (24), placement of aortic occlusion balloons to occlude the ostia of the bronchial arteries (25), and normothermic bypass with beating heart or electrically induced ventricular fibrillation (26) have all been performed to avoid the harmful effects of DHCA. In our experience (20–22), if cooling is deep enough and circulatory arrest times are kept to a minimum, the neurologic outcome should be controlled. After surgery, four cases presented short-lived delirium with recovery in a couple of days. A mechanism for post-operative delirium after DHCA has yet to be found. Although the pre-operative pulmonary vascular re-

sistance and the mean pulmonary artery pressure were high in our patient population, the operative mortality was low. Circulatory arrest time was limited to 20 minutes. If the endarterectomy cannot be performed within this period, hypothermic reperfusion was performed for at least 10 minutes, or venous saturation was allowed to return to at least 85%. When the right endarterectomy was completed, CPB was restarted, the cross-clamp was taken away, and hypothermic reperfusion was performed to repay the oxygen debt, allowing venous saturation to return to at least 85%, and then rewarming was started (20–22).

There are two immediate concerns after the PTE procedure that should be taken into consideration. The first is the redistribution of pulmonary blood flow away from previously well-perfused lung segments into the freshly endarterectomized segments. This can lead to reperfusion pulmonary edema (RPE), which occurs some 24–72 hours after surgery (11,27) and is associated with prolonged mechanical ventilation and increased inspired oxygen concentrations. There were seven cases in our group that experienced RPE. Treatment options are supportive, including adequate sedation, prolonged mechanical ventilation with positive end-expiratory pressure (PEEP), diuretics application, fluid restriction, and hemofiltration. Later, a bolus of steroids and inhaled NO may be used to improve oxygenation (18) or prostacyclin transfusion. The second major problem is the persistence of pulmonary hypertension (18,27). This, together with reperfusion pulmonary edema, is a significant cause of mortality after PTE. Treatment options are aggressive right ventricular afterload reduction, inhaled NO, and prostacyclin to reduce pulmonary vascular resistance.

In conclusion, PTE under DHCA is a favorable therapeutic procedure in selected patients with chronic thromboembolic pulmonary hypertension to achieve an improvement and decrease the related complications of clinical symptoms. The benefit of this procedure is long term, and the results suggest that a longer life expectancy can be anticipated in these patients.

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