Review

Technique and Outcomes of Pulmonary Endarterectomy Surgery

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Pulmonary endarterectomy (PTE) is the definitive treatment for chronic pulmonary hypertension resulting from thromboembolic disease. Chronic thromboembolic pulmonary hypertension is estimated to occur in approximately 4% of patients who have developed an acute pulmonary embolism, though the true prevalence is suspected to be much higher. Chronic thromboembolic pulmonary hypertension is characterized by intraluminal thrombus organization, fibrous stenosis, and vascular remodeling of pulmonary vessels. PTE is an operation considered to be a curative for this affliction and is therefore superior to transplantation. The procedure involves the removal of organized and incorporated fibrous obstructive tissue from the pulmonary arterial tree and is a true endarterectomy, not an embolectomy. Surgical outcomes with respect to functional status, quality of life, hemodynamics, right ventricular function, and gas exchange are favorable. Preoperative hemodynamic severity and site of anatomical obstruction are key predictors of postoperative outcome. This article focuses on the surgical techniques and outcomes of patients undergoing PTE for chronic thromboembolic pulmonary hypertension and discusses the currently known factors that affect survival after this operation. (Ann Thorac Cardiovasc Surg 2008; 14: 274–282)

Key words: pulmonary endarterectomy, thromboembolism, pulmonary hypertension

Chronic thromboembolic pulmonary hypertension is a rare disease estimated to result in approximately 3.8% of all cases of pulmonary embolism.[1] It results from an obstruction of the pulmonary arteries (PA) by single or recurrent pulmonary thromboembolisms that do not undergo complete resolution. Several mechanisms have been postulated to cause chronic thromboembolic pulmonary hypertension after an acute embolic event. These include a recurrence of embolism, reported to be 2.5% to 7% of adequately treated pulmonary embolic events,[2] in situ thrombus propagation into branch pulmonary vessels,[3] and failure to resolve the initial embolus, leading to large- and small-vessel vasculopathy.[4] In the proximal pulmonary arterial tree, the unresolved pulmonary emboli cause vascular obstruction of the vessel lumen by two mechanisms: (1) by direct occlusion of the vessel lumen, and (2) by inducing secondary endothelial changes of cellular hyperplasia, webbing, and incomplete clot remodeling.[5] In a subset of patients with chronic thromboembolism, small pulmonary arterioles also manifest a pathological process similar to that seen in idiopathic pulmonary arterial hypertension, whereby these vessels become excessively thickened by muscular hypertrophy and fibrointimal hyperplasia, leading to eventual occlusion.[6] Collectively, this large- and small-vessel pathology results in elevated PA pressures and right heart failure, leading to death if not corrected.

Over the past twenty years, pulmonary endarterectomy (PTE) has evolved to become the treatment of

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choice for patients with chronic thromboembolic pulmonary hypertension. This pioneering surgical procedure has been performed on approximately 3,500 patients with this disease at a limited number of centers around the world. To date, the greatest volume of PTE surgery has been performed at the University of California, San Diego (UCSD). Proper patient selection, meticulous surgical technique, and careful postoperative management have contributed to the effectiveness of this operation. A true endarterectomy (not an embolectomy) of all affected areas in the pulmonary vascular tree is necessary. We and others have shown that PTE ameliorates pulmonary hypertension by improving lung ventilation-perfusion match, arresting right ventricular dysfunction and tricuspid regurgitation, limiting retrograde extension of clot obstruction, and preventing arteriopathic changes in the remaining patent small pulmonary vessels.[7)]

Of the 3,500 cases of PTE performed worldwide, most of them (> 2,000 cases) were done at UCSD. The recent PTE operative experience at UCSD provides the basis of this report.

Patient Selection

The three major reasons to consider a patient for PTE are hemodynamic, respiratory, and prophylactic. The hemodynamic goal is to prevent or ameliorate right ventricular compromise caused by pulmonary hypertension. The respiratory goal is to improve function by the removal of a large ventilated but unperfused physiological dead space. The prophylactic goals are to prevent progressive right ventricular failure, retrograde extension of clot, and secondary vasculopathic changes in the remaining patent vessels. PTE is considered in patients who are symptomatic and have evidence of hemodynamic or ventilatory impairment at rest or during exercise. The selection criteria for surgical intervention have evolved over the years. Recent clinical guidelines from the American College of Chest Physicians recommend that the following four basic criteria should be met: (1) New York Heart Association (NYHA) class III or IV symptoms, (2) preoperative pulmonary vascular resistance (PVR) of more than 300 dynes-sec-cm^{-5}, (3) surgically accessible thrombus in the main, lobar, segmental, or subsegmental arteries, and (4) no significant comorbidities.[8)] Moreover, PTE at UCSD is considered in patients who have only mild symptoms at rest, but significant impairment with exercise. The only absolute contraindication to PTE is the presence of severe underlying lung disease, either obstructive or restrictive. Patients as young as 15 and as old as 84, as well as those with complex coexisting conditions, have successfully undergone the operation. There is no upper limit of PVR or degree of right ventricular failure or tricuspid regurgitation that excludes a patient from operation. Patients with suprasystemic PA pressures can safely undergo PTE.[7)] Furthermore, we do not consider that any patient has disease that is inaccessible at operation—thus if the origin of the pulmonary hypertension is the result of emboli, by definition we consider it "surgically accessible," or operable. Advanced techniques that enable dissection at a subsegmental level make this possible.

According to the usual practice at our institution, a diagnosis of chronic thromboembolic pulmonary hypertension is based on preoperative transthoracic echocardiography, lung ventilation-perfusion scintigraphy, right heart catheterization, and pulmonary angiography. All patients older than 40 undergo preoperative coronary angiography. Patients with coronary or valvular disease (excluding tricuspid regurgitation secondary to pulmonary hypertension) undergo coronary artery bypass grafting (CABG) and/or valve repair or replacement at the time of surgery. An inferior vena cava filter is routinely placed several days in advance of the operation. Patients are treated with warfarin until surgery, and this is continued lifelong after surgery.

Surgical Techniques

PTE is a true endarterectomy and bears little resemblance to pulmonary embolectomy. The technique for this operation has been largely developed by Dr. Stuart W. Jamieson at UCSD.[5,9)] The organized thromboembolic material is fibrotic and adheres to the vessel wall, eventually causing severe intimal thickening and webbing within the lumen of the pulmonary vasculature. After a median sternotomy is made, the pericardium is incised longitudinally and attached to the wound edges. Typically the right heart is enlarged, with a tense right atrium and a variable degree of tricuspid regurgitation. There is usually severe right ventricular hypertrophy, and the patient's condition may become unstable with manipulation of the heart. Anticoagulation is achieved with the use of beef lung sodium (400 units/kg, intravenously) administered to prolong the activated clotting time beyond 400 seconds. Full cardiopulmonary bypass

is instituted with high ascending aortic cannulation and two caval cannulas. These cannulase must be sufficiently inserted into the superior and inferior vena cavae to enable subsequent opening of the right atrium. The heart is emptied on bypass, and a temporary PA vent is placed in the midline of the main PA, one centimeter distal to the pulmonary valve. This site marks the beginning of the left pulmonary arteriotomy.

After cardiopulmonary bypass is initiated, surface cooling with both a head jacket and cooling blanket on the operating room table is begun. The blood is cooled with the pump-oxygenator. During cooling, a 10ºC gradient between blood and bladder or rectal temperature is maintained.[10] Cooling generally takes 45 min to an hour. When ventricular fibrillation occurs, an additional vent is placed in the left atrium through the right superior pulmonary vein. This prevents atrial and ventricular distension from the large amount of bronchial arterial blood flow that is common with these kinds of patients. It is most convenient for the primary surgeon to stand initially on the patient's left side. During the cooling period, some preliminary dissection can be performed with full mobilization of the right PA from the ascending aorta. All dissection of the PA takes place intrapericardially, and neither pleural cavity is entered. An incision is then made in the right PA from beneath the ascending aorta out under the superior vena cava and entering the lower lobe branch of the PA just after the removal of the middle lobe artery.

Any loose thrombus, if present, is removed. The endarterectomy cannot be performed in the presence of thrombus because it obscures the plane and prevents a collapse of the endarterectomized specimen, hindering distal exposure. It is important to recognize that first, an embolectomy without endarterectomy is quite ineffective, and second, in most patients with chronic thromboembolic pulmonary hypertension, direct observation of the pulmonary vascular bed at operation generally shows no obvious embolic material. Therefore to the inexperienced or cursory glance, the pulmonary vascular bed may well appear normal even in patients with severe chronic thromboembolic pulmonary hypertension. If the bronchial circulation is not excessive, the endarterectomy plane can be found during this early dissection. However, although a small amount of dissection can be performed before the initiation of circulatory arrest, it is unwise to proceed unless perfect visibility is obtained because the development of a correct plane is essential.

When the patient's temperature reaches 20ºC, the aorta is cross-clamped and a single dose of cold cardioplegia solution is administered. Additional myocardial protection is obtained by the use of a cooling jacket wrapped around the heart. The entire procedure is now performed with a single aortic cross-clamp period with no further administration of cardioplegic solution. A modified cerebellar retractor is placed between the aorta and superior vena cava. When back-bleeding from bronchial collaterals obscures the direct vision of the pulmonary vascular bed, thiopental is administered (500 mg to 1 g) until the electroencephalogram becomes isoelectric. Circulatory arrest is then initiated, and the patient undergoes exsanguination. It is rare that one 20 min period for each side is exceeded. Although retrograde cerebral perfusion has been advocated for total circulatory arrest in other procedures, it is not helpful in this operation because it does not allow a completely bloodless field, and with the short arrest times that can be achieved, it is unnecessary. Some have advocated the performance of this operation without circulatory arrest; however, we encounter no sequelae from circulatory arrest, and the operation cannot be done thoroughly without the perfect distal visibility afforded by this technique.

Any loose thrombotic debris encountered is removed. A microtome knife and ball-tipped suction cannula are then used to develop the endarterectomy plane posteriorly within the media of the vessel. Dissection in the correct plane is critical because if the plane is too deep, the PA may perforate, with fatal results, and if the dissection plane is not deep enough, inadequate amounts of the partially resorbed thromboembolic material will be removed. Once the plane is correctly developed, a full-thickness layer is left in the region of the incision to ease subsequent repair. For the endarterectomy, gentle traction with forceps while sweeping the outer vessel wall layer away with the ball-tipped suction cannula will result in the progressive withdrawal of the endarterectomy specimen. As each lobar branch appears, it is grasped individually and the specimen withdrawn until each segmental vessel branches again. Each of these subsegmental specimens is then extracted. The removal of each lobar and then the segmental branch makes subsequent distal dissection easier. If a large mass of endarterectomized tissue begins to obscure visibility, it is excised. The entire specimen can thus be removed for a length of approximately 20 cm. The distal-most portion endarterectomy is performed with an eversion

technique. Perforation at the level of the subsegmental vessels will become completely inaccessible later, so care must be taken to remain in the plane of the media for endarterectomy. It is important that each subsegmental branch is followed and freed individually until it ends in a "tail," beyond which is no further obstruction. Residual material should never be cut free; the entire specimen should "tail off" and come free spontaneously. Once the right-sided endarterectomy is completed, circulation is restarted, and the arteriotomy is repaired with a continuous 6-0 polypropylene suture. The hemostatic nature of this closure is aided by the nature of the initial dissection, with the full thickness of the PA being preserved immediately adjacent to the incision.

After repairing the right arteriotomy, the surgeon moves to the patient's right side. The pulmonary vent catheter is withdrawn, and an arteriotomy is made from the site of the pulmonary vent hole laterally to the pericardial reflection, again avoiding entry into the left pleural space. Additional lateral dissection does not enhance intraluminal visibility, it may endanger the left phrenic nerve, and it makes subsequent repair of the left PA more difficult. The left-sided dissection is virtually analogous in all respects to that accomplished on the right. The duration of circulatory arrest intervals during performance of the left-sided dissection is also subject to the same restriction as the right.

After completion of the endarterectomy, the cardiopulmonary bypass is renstituted and warming is commenced. Methylprednisolone (500 mg, intravenously) and mannitol (12.5 g, intravenously) are administered, and during warming a 10°C temperature gradient is maintained between the perfusate and body temperature. If the systemic vascular resistance level is high, nitroprusside is administered to promote vasodilatation and warming. The rewarming period generally takes approximately 90 min, but varies according to the body mass of the patient.

When the pulmonary arteriotomy has been repaired, the PA vent is replaced at the top of the incision. The right atrium is then opened and examined, unless prior to cardiopulmonary bypass, a negative "bubble" test revealed no persistent foramen ovale on transesophageal echocardiography. Otherwise, any intra-atrial communication (present in about 20% of patients) is closed at this point. Although tricuspid valve regurgitation is invariable in these patients and is often severe, tricuspid valve repair is not performed. Right ventricular remodeling occurs within a few days to months, with the return of tricuspid competence.[11] If other cardiac procedures are required, such as coronary artery or mitral or aortic valve surgery, they are conveniently performed during the systemic rewarming period.[12] Myocardial cooling is discontinued once all cardiac procedures have been concluded. The left atrial vent is removed, and the vent site is repaired. Air is evacuated from the heart, and the aortic cross-clamp is removed.

When the patient has been rewarmed, the cardiopulmonary bypass is discontinued. Dopamine hydrochloride is routinely administered at renal doses, and other inotropic agents and vasodilators are titrated as necessary to sustain acceptable hemodynamics. The cardiac output (CO) is generally high, with a low systemic vascular resistance. Temporary atrial and ventricular epicardial pacing wires are placed. Despite the duration of extracorporeal circulation, hemostasis is readily achieved, and the administration of platelets or coagulation factors is generally unnecessary. Wound closure is routine. A vigorous diuresis is usual for the next few hours, which is a result of the previous systemic hypothermia. All patients are subjected to a maintained diuresis with the goal of reaching the patient's preoperative weight within twenty-four hours. Extubation is usually performed on the first postoperative day.

Results

More than 2,200 PTE operations have been performed at UCSD since 1970, and 1,300 cases have been completed at UCSD in the ten-year period since 1997. Data are presented for the most recent 1,100 patients undergoing this operation. Characteristics at the time of operation for patients who underwent PTE are detailed in Table 1. The mean patient age was 51.7 ± 15.5 years, with a range of 8 to 84. A slight predominance of men compared to women was referred for operation, reflecting disease predilection, surgical referral bias, or both. In 10.2% of cases, at least one additional cardiac procedure was performed during the operation (excluding persistent foramen ovale repair). Most commonly, the adjunct procedures were coronary revascularization, aortic valve replacement, or mitral valve repair/replacement. Twenty-four percent of patients underwent closure of a persistent foramen ovale. There was no significant difference between patients undergoing PTE alone or combined with other cardiac operations with respect to cardiopulmonary bypass time, cross-clamp time, or cir-

Table 1. Preoperative patient characteristics

Variable	All patients (n = 1,100)	PTE patients (n = 988)	PTE-CABG patients (n = 94)	PTE-valve patients (n = 18)
Age (y)	51.7 ± 15.5	50.2 ± 15.1	66.6 ± 9.6	53.9 ± 17.2
Male sex (%)	538 (48.9)	459 (46.5)	72 (76.6)	7 (38.9)
PA pressure (mmHg)				
Systolic	75.9 ± 18.6	76.0 ± 18.6	76.1 ± 18.8	71.4 ± 19.9
Diastolic	28.5 ± 9.7	28.8 ± 9.8	26.8 ± 9.5	26.1 ± 6.8
PVR (dynes-sec-cm^{-5})	859.4 ± 439.5	863.5 ± 441.6	845.3 ± 433.2	714.6 ± 334.4
CO (L/min)	3.9 ± 1.3	3.9 ± 1.4	3.8 ± 1.2	3.7 ± 0.8
Pulmonary capillary wedge pressure (mmHg)	9.7 ± 4.7	9.6 ± 4.7	9.6 ± 5.0	13.3 ± 6.4
Tricuspid regurgitation jet (M/s)	4.2 ± 0.7	4.2 ± 0.7	4.0 ± 0.7	3.9 ± 0.7
In hospital >24 h preoperatively	246 (22.4)	216 (21.9)	26 (27.7)	4 (22.2)
In ICU preoperatively	16 (1.5)	14 (1.4)	1 (1.1)	1 (5.6)
On intravenous inotropes preoperatively	30 (2.7)	26 (2.6)	2 (2.1)	2 (11.1)
Mechanical ventilation preoperatively	5 (0.5)	4 (0.4)	1 (1.1)	0 (0.0)
NYHA class				
I	19 (1.7)	17 (1.7)	2 (2.1)	0 (0.0)
II	116 (10.5)	111 (11.2)	5 (5.3)	0 (0.0)
III	874 (79.5)	776 (78.5)	82 (87.2)	16 (88.9)
IV	91 (8.3)	84 (8.5)	5 (5.3)	2 (11.1)
Jamieson disease classification				
I	415 (37.7)	367 (37.1)	42 (44.7)	6 (33.3)
II	469 (42.6)	418 (42.3)	45 (47.9)	6 (33.3)
III	192 (17.5)	182 (18.1)	7 (7.4)	3 (16.7)
IV	24 (2.2)	21 (2.1)	0 (0.0)	3 (16.7)

Data are shown as means ± standard deviation or number (percentage).
PTE, pulmonary endarterectomy; CABG, coronary artery bypass grafting; PA, pulmonary artery; PVR, pulmonary vascular resistance; CO, cardiac output; ICU, intensive care unit; NYHA, New York Heart Association.

culatory arrest time (Fig. 1). Total cardiopulmonary bypass time correlated with body mass and cooling-rewarming intervals and not with specific preoperative hemodynamic parameters.

With this operation, a reduction in PA pressures and PVR to normal levels, and a corresponding improvement in pulmonary blood flow and CO were generally immediate and sustained. Table 2 presents the mean change between preoperative and postoperative hemodynamic parameters of PA pressure, PVR, and CO after PTE. Before the operation, 87.7% of the patients were in NYHA functional class III or IV; at the time of discharge, 81.9% were in NYHA functional class I or II. Moreover, echocardiographic studies from our institution[13] have demonstrated that with the elimination of chronic pressure overload, right ventricular geometry rapidly reverted to normal. In general, right atrial and right ventricular hypertrophy and dilation regressed after operation. Tricuspid valve function (as measured by tricuspid regurgitant velocity) returned to normal within a few days (Table 2) as a result of the restoration of tricuspid annular geometry after the remodeling of the right ventricle. Tricuspid valve annuloplasty was not performed, even when severe tricuspid regurgitation was documented preoperatively.

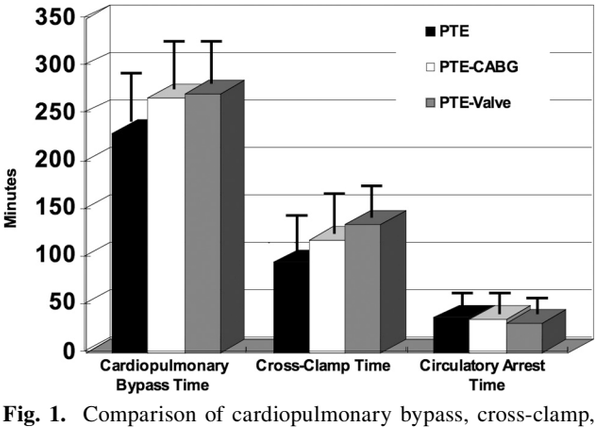

Fig. 1. Comparison of cardiopulmonary bypass, cross-clamp, and circulatory arrest time in patients undergoing pulmonary endarterectomy and combined pulmonary endarterectomy-cardiac operations.

Table 2. Comparison of perioperative hemodynamic parameters

Variable	All patients (n = 1,100)	PTE patients (n = 988)	PTE-CABG patients (n = 94)	PTE-valve patients (n = 18)
Mean decrease in PAS (mmHg)	29.3 ± 19.7	29.4 ± 19.8	28.8 ± 18.5	24.7 ± 20.2
Mean decrease in PAD (mmHg)	10.0 ± 9.7	10.2 ± 9.8	8.0 ± 9.6	8.0 ± 6.7
Mean decrease in PVR (dynes-sec-cm^{-5})	563.6 ± 393.6	566.7 ± 392.2	539.2 ± 411.5	487.9 ± 381.8
Mean increase in CO (L/min)	1.5 ± 1.6	1.5 ± 1.6	1.6 ± 1.6	1.2 ± 1.3
Mean decrease in tricuspid regurgitant velocity (M/s)	1.1 ± 0.8	1.1 ± 0.8	1.1 ± 0.7	0.3 ± 0.9

Data are shown as means ± standard deviation.
PTE, pulmonary endarterectomy; CABG, coronary artery bypass grafting; PAS, pulmonary artery systolic pressure; PAD, pulmonary artery diastolic pressure; PVR, pulmonary vascular resistance; CO, cardiac output.

Table 3. Postoperative outcome

Variable	All patients (n = 1,100)	PTE patients (n = 988)	PTE-CABG patients (n = 94)	PTE-valve patients (n = 18)
Mean/median days intubated	4.8/1.0	4.3/1.0	9.7/3.0	8.4/1.5
Mean/median days in ICU	7.3/4.0	6.7/4.0	13.2/6.0	12.7/4.5
Mean/median days in hospital	14.3/10.0	13.6/10.0	20.8/14.5	20.1/12.5
Mortality (%)	52 (4.7)	40 (4.0)	9 (9.6)	3 (16.7)

Data are shown as means ± standard deviation or number (percentage).
PTE, pulmonary endarterectomy; CABG, coronary artery bypass grafting; ICU, intensive care unit.

The median stay in the intensive care unit for all patients was 4 days, and the median number of days the patients were intubated was 1 day (Table 3). The median length of hospital stay postoperatively was 10 days.

Fifty-two of 1,100 patients died (either in the hospital of within 30 days of discharge), an overall mortality rate of 4.7%. Most cases of death were related to residual high pulmonary pressures and residual high PVR. The mortality rate of patients with preoperative PA systolic pressures (PASs) exceeding 100 mmHg was 10.9% (11 of 101 patients). In individuals in whom the preoperative PAS was less than 100 mmHg, the mortality rate was 4.1% (41 of 999 patients, $p < 0.005$). Failure to lower PA pressures and PVR strongly correlated with mortality rate. Patients with a postoperative PVR higher than 500 dynes-sec-cm^{-5} had a mortality rate of 5.7% (49 of 856), and individuals with a postoperative PVR of less than 500 dynes-sec-cm^{-5} had a mortality rate of 1.2% (3 of 244 patients, $p < 0.005$).

We have previously shown that patient survival and the degree of improvement in pulmonary hypertension and tricuspid regurgitation after PTE are determined by the type and location of pulmonary thromboembolic disease. Four major types of pulmonary occlusive disease, based on anatomy and the location of thrombus and vessel wall pathological change, have been described by our group.[5,14] This intraoperative classification of disease allows the prediction of patient outcome after PTE. For the 1,100 patients reviewed in this article, the results of operation correlated with the classification type. In our cohort of 1,100 patients, 415 (37.7%) were type 1, fresh or organized clot in the main or lobar PA; 469 (42.6%) were type 2, intimal thickening and fibrosis without visible thrombus proximal to the segmental arteries; 192 (17.5%) were type 3, fibrosis, intimal webbing and thickening in segmental and subsegmental arteries only; and 24 (2.2%) were type 4, microscopic distal arteriolar vasculopathy. Type 4 patients have misdiagnosed idiopathic pulmonary arterial hypertension not directly related to pulmonary embolism and are not benefited by PTE.[5,15] Table 4 shows the hemo-dynamic results and mortality for all patients as classified by thromboembolic type.

Reperfusion edema was the single most frequent complication after PTE, resulting in prolonged intubation more than 24 hours postoperatively in 16.2% of patients (Table 5). Of patients with reperfusion injury, most resolved the problem with a short period of ventilatory support and aggressive diuresis. A minority of patients with severe lung reperfusion injury required prolonged periods of ventilatory support, with extreme cases requiring veno-veno extracorporeal support for

Table 4. Thromboembolic classification—hemodynamic results

Variable	All patients (n = 1,100, 100%)	Type 1 (n = 415, 37.7%)	Type 2 (n = 469, 42.6%)	Type 3 (n = 192, 17.5%)	Type 4 (n = 24, 2.2%)
PVR (dynes-sec-cm^{-5})	859.4 ± 439.5	924.2 ± 450.4	799.9 ± 417.2	863.2 ± 454.6	884.6 ± 412.3
	290.4 ± 195.7	269.8 ± 176.6	270.5 ± 191.3	350.8 ± 183.3	595.2 ± 360.2
CO (L/min)	3.9 ± 1.3	3.7 ± 1.4	4.1 ± 1.3	4.0 ± 1.5	3.8 ± 1.2
	5.4 ± 1.5	5.5 ± 1.5	5.5 ± 1.5	5.2 ± 1.4	4.5 ± 1.1
Systolic PA pressure (mmHg)	75.9 ± 18.6	76.8 ± 18.7	75.0 ± 19.5	75.8 ± 16.4	78.4 ± 15.6
	46.4 ± 16.6	44.4 ± 15.1	44.5 ± 15.0	52.7 ± 17.1	73.8 ± 32.1
Diastolic PA pressure (mmHg)	28.5 ± 9.7	29.8 ± 9.6	27.3 ± 10.0	28.3 ± 8.8	32.3 ± 9.5
	18.5 ± 7.2	17.7 ± 6.5	17.9 ± 6.8	20.6 ± 7.9	27.3 ± 12.8
Mean PA pressure (mmHg)	46.2 ± 11.3	47.0 ± 11.4	45.2 ± 11.6	46.5 ± 10.3	50.2 ± 10.5
	28.4 ± 9.6	27.2 ± 8.7	27.5 ± 9.1	31.8 ± 10.1	42.4 ± 15.5
Mortality (%)	52 (4.7)	16 (3.9)	22 (4.7)	12 (6.3)	4 (16.7)

Data are shown as means ± standard deviation or number (percentage).
Top numbers are preoperative values and bottom numbers are postoperative values obtained just prior to removal of the Swan-Ganz catheter.
PVR, pulmonary vascular resistance; CO, cardiac output; PA, pulmonary artery.

Table 5. Perioperative morbidity

Variable	All patients (n = 1,100)	PTE patients (n = 988)	PTE-CABG patients (n = 94)	PTE-valve patients (n = 18)
Reperfusion edema	178 (16.2)	154 (15.6)	21 (22.3)	3 (16.7)
Pneumonia	104 (9.5)	84 (8.5)	16 (17.0)	4 (22.2)
Reoperative bleeding	39 (3.5)	31 (3.1)	7 (7.4)	1 (5.6)
Atrial fibrillation	29 (2.6)	21 (2.1)	7 (7.4)	1 (5.6)
Sternal wound infection	14 (1.3)	13 (1.3)	1 (1.1)	0 (0.0)
Sepsis	13 (1.2)	10 (1.0)	2 (2.1)	1 (5.6)
Pneumothorax	12 (1.1)	12 (1.2)	0 (0.0)	0 (0.0)
Dialysis	11 (1.0)	6 (0.6)	4 (4.3)	1 (5.6)
Pericardial effusion	7 (0.6)	6 (0.6)	0 (0.0)	1 (5.6)
Hemothorax/pleural effusion	8 (0.7)	8 (0.8)	0 (0.0)	0 (0.0)
GI bleeding	7 (0.6)	5 (0.5)	2 (2.1)	0 (0.0)
Sternal dehiscence	6 (0.5)	5 (0.5)	1 (1.1)	0 (0.0)
Pulmonary hemorrhage	4 (0.4)	4 (0.4)	0 (0.0)	0 (0.0)
Chylothorax	4 (0.4)	4 (0.4)	0 (0.0)	0 (0.0)
Cerebrovascular accident	4 (0.4)	3 (0.3)	0 (0.0)	1 (5.6)
Femoral-retroperitoneal hematoma	4 (0.4)	3 (0.3)	0 (0.0)	1 (5.6)
Deep venous thrombosis	2 (0.2)	1 (0.1)	1 (1.1)	0 (0.0)
Drug reaction	1 (0.09)	1 (0.1)	0 (0.0)	0 (0.0)

Data are shown as number (percentage).
PTE, pulmonary endarterectomy; CABG, coronary artery bypass grafting; GI, gastrointestinal.

oxygenation and blood carbon dioxide removal.[16] Neurological complications from circulatory arrest have been eliminated by shorter circulatory arrest periods and the use of a direct cooling jacket placed around the head. The rates of perioperative confusion and stroke for PTE were similar to those seen with conventional open heart surgery. In our series, a reexploration for bleeding was necessary in 3.5% of patients, and only 33.9% required an intraoperative or a postoperative blood transfusion. Despite an average of 6.5 hours for the operation, a wound infection (superficial or deep) occurred in only 1.3% of patients.

In the UCSD experience, overall perioperative mortality has been 9% for the entire cohort of patients encompassing a time span of more than 30 years. In the past 1,100 cases, surgical mortality for PTE was 4.7%. This reflects the learning curve for safely performing this operation and the refinements in surgical technique,

described above, that enhance patient outcome. In the past 200 patients, the mortality has been less than 4%.

Conclusions

PTE is a complex surgical procedure that is considered curative, with significant and sustained functional and hemodynamic improvement in the vast majority of patients with chronic thromboembolic pulmonary hypertension. Medical therapy for this disease is palliative, ineffective in prolonging life, and it only temporarily and sporadically improves symptoms. Lung transplantation for this disease carries higher early and late mortality, along with the risk of lifelong immunosuppression and allograft rejection; it is therefore contraindicated for this disease. Our current mortality rate of less than 4% for PTE is clearly superior to that for lung transplantation. Indeed, these superior mortality rates continue to decrease and are dependent on a high level of surgical expertise and the selection of appropriate patients.

The site of pulmonary arterial obstruction is an important determinant of surgical outcome. We have come to realize that a small subset of patients do not benefit from this operation, because they manifest solely arteriolar-capillary vasculopathy similar to idiopathic pulmonary arterial hypertension (type 4 disease). The risk factors for operation that characterize these patients is a high PVR out of proportion to gross changes seen on angiography. Further refinements in pulmonary waveform analysis,[17] magnetic resonance imaging,[18] and computed tomography scanning[19] may help to further distinguish these patients from those that benefit from operation.

Because of the pioneering efforts to develop and perfect PTE operative technique by Dr. Jamieson and his colleagues at UCSD, this operation may be offered to patients with an acceptable mortality rate and full anticipation of clinical improvement. Currently at our institution, no patient is denied an operation based on severity of pulmonary hypertension or age. Improved awareness of PTE surgery and operative results has ushered in an era whereby patients with chronic thromboembolic disease can expect complete surgical cure.

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