

Anesthetic management of heterotopic heart transplantation on beating heart

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Introduction

In 1906, Alexis Carrel performed the first experimental heterotopic heart transplantation in animals [1]. The placement of the heart into the thoracic cavity was first described by Demikhov [2]. His experiments were performed between 1940 and 1960, and he described 24 different techniques for the positioning of the two hearts. In 1974, Barnard and Losman performed the first successful clinical heterotopic heart transplantation in a human [3]. Although it was first used for pure left ventricular assistance, this operation was later modified to a biventricular assistance system [4]. Heterotopic heart transplantation is still indicated in selected patients, mainly those with pulmonary vascular bed problems [5]. Cardiopulmonary bypass (CPB) has well-known deleterious effects on the pulmonary vascular bed due to leukocyte sequestration, as well as on the immune response of the patients. The negative effect of cardioplegic arrest on the native heart is another drawback of classical heterotopic heart transplantation with the use of CPB [6–8]. There has been no report of heterotopic heart transplantation in the literature performed on the beating heart, without cardiopulmonary bypass. We present our method of anesthesia and a new technique for heterotopic heart transplantation without CPB used successfully in our institution at Kocaeli University on a patient with resistant pulmonary hypertension and dilated cardiomyopathy.

Case report

A 58-year-old man (weight 55 kg, body surface area 1.58 m²) had end-stage dilated cardiomyopathy. A left ventricular ejection fraction of 20% was measured by two-dimensional echocardiography. Cardiac catheterization documented severe pulmonary hypertension with pulmonary arterial pressures of 86/40 mmHg (mean, 48 mmHg), pulmonary capillary wedge pressure of 29 mmHg, and pulmonary vascular resistance of 7.3 Wood units, which decreased to a level of 4.0 Wood units with multiple drug manipulations (isoproterenol, prostoglandin E₁, nitroprusside, and oxygen inhalation). Cardiac output and cardiac index were 2.4 l·min⁻¹ and 1.51 l·mm⁻², respectively. Coronary angiography was normal, but left ventriculography confirmed moderate mitral regurgitation and dilated cardiomyopathy. The patient was placed on our waiting list and the decision was made to repeat right heart catheterization at the time of transplantation for the definitive operative procedure. Since the patient had NYHA class IV congestive heart failure, he was hospitalized for high-dose inotropic support with (dopamine 10 µg·kg⁻¹·min⁻¹, dobutamine 10 µg·kg⁻¹·min⁻¹, and isoprenaline 0.5 µg·kg⁻¹·min⁻¹). During his hospitalization, a young female donor (52 kg body weight and 1.50 m² body surface area) was referred to us. The patient received premedication with midazolam 5 mg intramuscularly 30 min preoperatively. In the operating room, the patient was monitored with a five-lead ECG, and ST segment analysis, SpO₂, ET CO₂, invasive radial artery blood pressure, and rectal and esophageal temperature were measured. In the operating room, a pulmonary artery catheter was placed. The pulmonary artery pressure and calculated pulmonary vascular resistance were 80/38 (mean, 46 mmHg) and 6.4 Wood units, respectively. Anesthesia was induced with 5 mg midazolam, 1 mg fentanyl, and 0.1 mg·kg⁻¹ vecuronium to facilitate tracheal intubation. Anesthesia was maintained with a

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mixture of air and oxygen (50%/50%), 0.3–0.5 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ infusion of fentanyl and additional vecuronium to sustain paralysis as required. He received infusion of nitroglycerin 0.5 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ during the operation. A total of 1500 ml of 0.9% NaCl, 7 units of whole blood, 1 M KIU of aprotinin, and 500 mg of methyl prednisolone was administered during the operation, which lasted for a total of 6 h. Anesthesia was maintained with a total of 15 mg of midazolam, 21 mg of vecuronium, and 9 mg of fentanyl. The operation was completed without problems. Hemodynamics were stable during both induction and maintenance of anesthesia. The patient was weaned from mechanical ventilation on the first postoperative day (at 21 h). Immunosuppressive therapy included a triple-drug combination: low-dose cyclosporine (3–5 $\text{mg}\cdot\text{kg}^{-1}\cdot\text{day}^{-1}$), azathioprine (0–2 $\text{mg}\cdot\text{kg}^{-1}\cdot\text{day}^{-1}$) and oral methyl prednisolone (0.3 $\text{mg}\cdot\text{kg}^{-1}\cdot\text{day}^{-1}$) were started. Daily leukocyte subtype analysis was used for monitoring of rejection. The pulmonary catheter was withdrawn on day 3 to reduce the likelihood of infection in the immunosuppressed patient. The inotropic doses were adjusted according to output measurements and stopped on day 3. Cardiac functions were monitored via a Swan-Ganz catheter for the first 3 days and by daily echocardiographic contractility measurements of both hearts and aortic flow distal to the anastomosis. On day 1, the pulmonary arterial pressure and pulmonary capillary wedge pressure were 58/26 mmHg (mean, 37 mmHg) and 20 mmHg. The pressures fell further to 44/20 mmHg (mean, 28 mmHg) and 16 mmHg, respectively on day 3. Since the thermodilution catheter was placed only on the native heart, the total cardiac function was assessed by echocardiographic measurements. The fractional shortening of the native heart improved from 10% to 21% during follow-up, while the donor heart remained in good condition through the postoperative monitoring period. The patient died because of acute rejection on day 9 postoperatively.

Technique

After systemic heparinization with 300 units $\cdot\text{kg}^{-1}$, the arterial cannula was placed distally in the ascending aorta and bicaval cannulation was accomplished by direct caval cannulation. The CPB circuit was primed and connected to the patient in the usual manner. The donor heart could be harvested with pulmonary vein cuffs and with a long superior caval vein due to the lack of a pulmonary recipient. The inferior vena cava and pulmonary veins were oversewn with a running 4/0 monofilament suture. The donor left atrium was then opened widely in a transverse fashion, with the pulmonary vein cuffs on both sides extended to create a diamond-

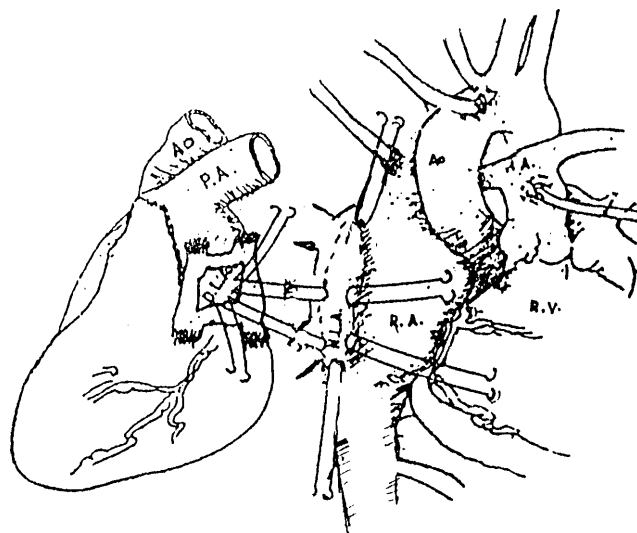


Fig. 1. Placement of left atrial sutures

shaped anastomosis. The right side of the pleura and the pericardium of the recipient was opened 2 cm above the phrenic nerve. The donor heart was placed in the right chest, anterior to the deflated lung. Since the recipient's left atrium was dilated and distended, a wide-mouth Satinsky side-biting clamp was inserted, extending from the right superior pulmonary vein below the inferior pulmonary vein parallel to the right atrium, similar to the clamping of the interatrial groove in the Blalock-Hanlon procedure. Due to the hemodynamic deterioration with this clamping, the clamp was removed, and interrupted U sutures were placed on the circumference of an imaginary ellipsoid placed between the two right pulmonary veins, with its longer axis lying cranio-caudally. The posterior row of sutures was then passed through the donor left atrium inside out and tied (Fig. 1). Then the side clamp was inserted below the suture rows. The recipient's atrium was opened longitudinally, and the atrial tissue between the two suture rows was excised (Fig. 2). The anterior row of sutures was also passed through the donor atrium and tied, and the common left atrium was created. The side clamp at the atrium was removed, and bleeding control was performed. The donor aorta was then anastomosed to the side-clamped recipient aorta by interposing an 18-mm Dacron tubular graft, and the clamps (side clamp on the recipient aorta and cross clamp on the donor aorta) were removed after deairing of the donor left heart through the aortic vent cannula. A right atriotomy about 5 to 6 cm in length was made in the recipient after clamping the right atrial appendix. A linear incision (5 to 6 cm) was made in the posterior aspect of the superior vena cava with extension into the right atrium to create an adequate right atrial orifice in the donor. Both atria

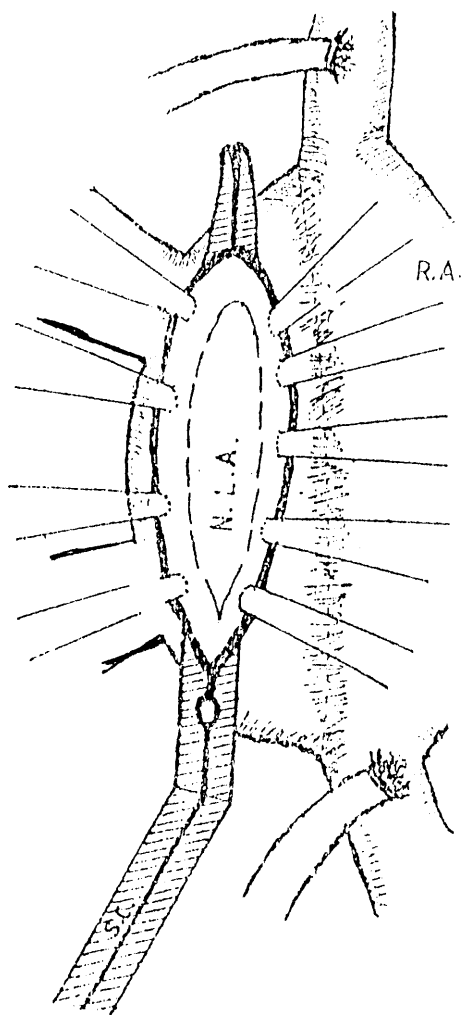


Fig. 2. Clamping of the recipient left atrium

were anastomosed using a polypropylene continuous suture (Fig. 3). Pulmonary artery anastomosis was also performed by interposing a woven Dacron graft between the pulmonary arteries similar to the aortic anastomosis to provide a tension-free anastomosis (Fig. 4). The operation was completed without heparin reversal with protamine due to the presence of Dacron grafts.

Discussion

Two primary indications for heterotopic heart transplantation exist today: longstanding recipient pulmonary hypertension and elevated pulmonary vascular resistance, with a gross weight mismatch (greater than 20%) between the donor and recipient [5]. Heterotopic heart transplantation has been considered advantageous for recipients of cardiac transplants with pulmonary hypertension and elevated pulmonary vascular

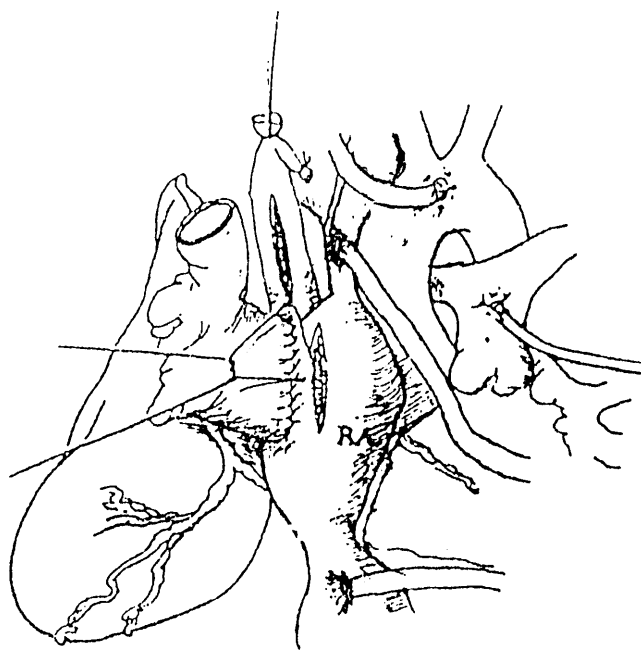


Fig. 3. Right atrial anastomosis

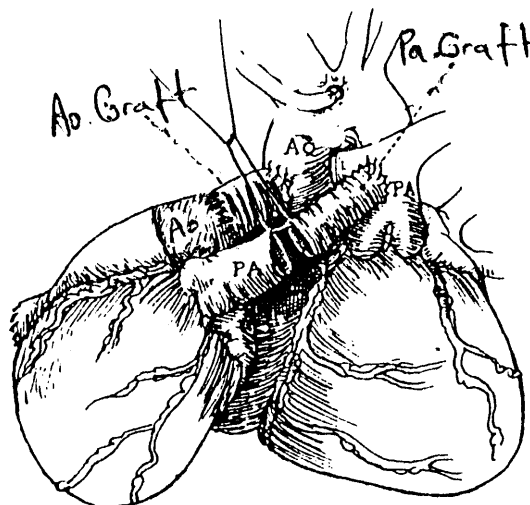


Fig. 4. Completion of the pulmonary anastomosis

resistance [9]. Heterotopic heart transplantation has been associated with an improvement in both systemic and pulmonary circulation because decreases in left ventricular and right ventricular filling pressures lead to an increase in the cardiac index [10]. In heterotopic heart transplantation, the native heart remains as the natural cardiac assist device, especially in the early post-operative period [11]. However, if the native left ventricular functions deteriorate after transplantation, the donor heart provides the patient with the greater percentage of his or her systemic cardiac output [12]. De-

spite recent advances in myocardial protection techniques, heterotopic heart transplantation with CPB in patients with persistent pulmonary hypertension may have a suboptimal outcome due to the damaging effect of CPB, especially after prolonged pump runs. Cardiopulmonary bypass triggers a systemic inflammatory response, which can lead to end-organ injuries affecting postoperative morbidity. Extracorporeal circulation causes hemolysis and activates leukocytes and platelets; after bypass, white cells have reduced opsonization, metabolism, and phagocytosis. Myocardial stunning to some degree is inevitable during aortic cross-clamping. Causative factors include complement activation, surgical trauma, immune responses, contact of blood with the extracorporeal circuit, lung reperfusion injury, coagulation, and cerebral dysfunction. Postoperative renal insufficiency and neurologic problems are seen with CPB [13–17]. Pulmonary interstitial edema increases and atelectasis develops. Compression atelectasis is usually seen in heterotopic heart transplantation. We think that pulmonary complications can be prevented by avoiding extracorporeal circulation during heterotopic heart transplantation. There is a trend toward giving these patients no premedication or smaller doses of premedication. The preparation of anesthetic and cardiovascular drugs for patients undergoing heart transplantation does not differ from that for other cardiac patients. Because immunosuppressed patients are predisposed to infection, efforts are made to organize and use equipment in as sterile a manner as possible. This includes the use of an “intubation pack” that contains all the necessary items, previously sterilized, for laryngoscopy and intubation. Monitoring catheters must be inserted using sterile technique. After placement of monitors, induction of anesthesia follows the basic principles of cardiac anesthesia. Our induction consisted of the administration of fentanyl, vecuronium, and midazolam. The response of the patient was observed in terms of anesthesia, ventilation, blood pressure, and heart rate. If a drastic response occurs, it is usually within 30 s to 1 min after initial dosing. In our patient, the hemodynamics were stable during both induction and maintenance of anesthesia. Anesthesia was maintained with a mixture of air and oxygen, and infusion of fentanyl and additional vecuronium. In the maintenance of anesthesia, the most prevalent problem is vasodilation in the face of a poorly contracting heart and a decreased blood volume. It must be realized that the patient’s blood pressure can be safely allowed to drop as far as a mean arterial pressure of 70 mmHg without concern that the patient will be harmed. This mean arterial pressure falls within the autoregulatory range of both the brain and the kidney and, presumably, of other organs [18]. The overall aim is to maintain an adequate blood pressure, to maintain

urinary output over the longer term, and to ascertain that arterial and venous pH give no evidence of intracellular acidemia. In our patient, the postoperative pulmonary arterial pressure and pulmonary capillary wedge pressure decreased to near normal values postoperatively. With this technique of anesthesia, heterotopic heart transplantation can be performed on the beating heart without the deleterious effects of CPB, preventing potential pulmonary and renal morbidity.

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