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# Ventricular Dysrhythmia in the Native Heart after Heterotopic Heart Transplantation: Diagnosis with Selective Electrocardiography

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HETEROTOPIC heart transplantation is an alternative option in end-stage heart disease, particularly in patients with reversible pulmonary hypertension[1,2] and in cases of marked body-weight mismatch.[2] The donor heart may function as a total or partial biventricular assist device.[3] A common occurrence is the presence of malignant ventricular dysrhythmias of the native heart, yet frequently adequate systemic hemodynamics are maintained by heterotopic grafts during malignant native ventricular dysrhythmias, and the latter are not considered a contraindication to heterotopic heart transplantation.[4,5] Identification and treatment of dysrhythmias in patients with an innervated and denervated heart remains an important clinical challenge.[6] We report a case of heterotopic heart transplantation in which ventricular dysrhythmia with significant hemodynamic impact developed in the native heart. The condition was diagnosed by selective right- and left-sided electrocardiograph (ECG), and the dysrhythmia was terminated with left-sided DC countershock.

## Case Report

A 57-y-old white man weighing 55-kg and measuring 173 cm and classified as ASA physical status 4 was admitted to the hospital for a heart transplantation. Sixteen years before admission he suffered an acute myocardial infarction. Five years before admission it became difficult for him to perform his duties as a lifeguard and exertional dyspnea and angina developed. Two years before admission he suffered a second acute myocardial infarction that left him with markedly reduced myocardial function and a left ventricular ejection fraction of 20%. He underwent coronary artery bypass surgery the year before admission, with little improvement in function. In addition, he had thalassemia minor, hypertension (controlled), hypercholesterolemia, and reduced renal function ($Cr^{51}$ ethylenediamine tetraacetic acid clearance of 36 ml/min).

On admission he was in no acute distress but was mildly dyspneic. Physical examination revealed a blood pressure of 110/70 mmHg, central venous pressure of 12 cm $H_2O$, hepatomegaly, a liver pulse, and minimal edema. Chest roentgenograms showed congestion in both lung fields and cardiac enlargement. The admission ECG showed a regular sinus rhythm at a rate of 69 bpm, left axis deviation, and a prolonged PR interval (240 ms). A right heart catheterization 1 month before admission gave the following cavity pressure measurements: right atrium, 17 mmHg; right ventricle, 70/12 mmHg; pulmonary artery, 75/25 mmHg; pulmonary capillary wedge pressure, 22 mmHg; cardiac output, 3.1 $l \cdot min^{-1}$; cardiac index, 1.7 $l \cdot min^{-2}$; pulmonary vascular resistance, 460 dynes $\cdot s^{-1} \cdot cm^{-5}$; and right atrial and pulmonary artery hemoglobin oxygen saturation values, 38%. Ventriculography demonstrated poor left ventricular function and angiography showed total obstruction of the left iliac artery. Pulmonary hypertension was present yet it was found to be reversible with administration of sodium nitroprusside and partially reversible with prostaglandin $E_1$.

Because of rapidly deteriorating heart failure requiring inotropic support, he underwent a heterotopic heart transplantation[1–3] with a donor heart retrieved from a 10-y-old 30-kg boy. The donor heart was implanted in the right chest in the left ventricular assist configuration, where donor and recipient atria, aortae, and superior venae cava were connected. The donor pulmonary artery was connected to the recipient right atrium end to side. Total ischemic time for the donor heart was 314 min. Cardiopulmonary bypass lasted for 168 min, with a cross-clamp time of 80 min. Anesthesia was induced and maintained with 10 mg diazepam, 300 $\mu$g sufentanil, 30 mg atracurium, and 8 mg vecuronium. Muscle relaxation was maintained with pancuronium as needed, and the lungs were mechanically ventilated at an $FIO_2$ of 0.5 with oxygen and air with a minute volume of 6 l at a rate of 12 bpm. The patient also received 500,000 U aprotinin per hour throughout. Anesthesia and surgery were uncomplicated, and myocardial performance after weaning from cardiopulmonary bypass was considered

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excellent. No cardiac pacing was required and the patient maintained a blood pressure of 120/80 mmHg, a heart rate of 80 bpm, a central venous pressure of 12 mmHg, and a left atrial pressure of 15 mmHg. A total of six units of platelets, four units of fresh frozen plasma, and seven units of packed red cells were administered during the procedure. The patient was transferred to the intensive care unit in satisfactory condition, and continuous mechanical ventilation was maintained with a $FIO_2$ of 0.5. Arterial blood gases were pH, 7.45; $P_{O_2}$, 97 mmHg; $P_{CO_2}$, 39 mmHg; bicarbonate, 27.3 meq·l$^{-1}$; $Sa_{O_2}$, 99%; and hemoglobin concentration, 10 g/dl. Transesophageal echocardiography showed poor left ventricular function of the native heart, normal valve anatomy, and a trace of mitral regurgitation. Transthoracic echocardiography of the donor heart showed normal end-diastolic (53 mm) and end-systolic (42 mm) diameters, normal global and regional contractility, normal valve anatomy, and no pericardial fluid or tamponade.

Five hours after admission to the ICU, the patient became hypotensive, with blood pressure of 80/50 mmHg and a heart rate of 50 bpm. Inotropic support, colloid, crystalloid, and packed red cells restored stable hemodynamics, with a blood pressure of 110/70 mmHg and heart rate of 70 bpm. Twelve hours after admission to the intensive care unit, the patient became suddenly hypotensive with a rapid heart rate of apparently about 150 bpm. Blood pressure decreased to 55/46 mmHg with a rapid rhythm, the nature of which was difficult to distinguish from standard ECG leads because of unequal contributions from the native and donor hearts. $CaCl_2$ (500 mg), phenylephrine (200 $\mu$g), verapamil (6 mg), and lidocaine (100 mg twice) were given intravenously with no effect. Blood pressure decreased to 47/38 mmHg. A standard 12-lead ECG was taken, as was an ECG in the dextrocardia configuration (*i.e.,* with all leads swapped from left to right with precordial leads $V_{2R}$–$V_{6R}$ on the right chest to distinguish between electrical activities of the native and donor heart, respectively). The top panel of figure 1 is the standard left-sided 12-lead ECG; the bottom panel is the right-sided ECG reflecting predominantly the donor heart. Figure 1 shows clearly that standard ECG leads (aVR, aVL, aVF, I, II, III) contain information from both the donor and native hearts. Precordial lead $V_1$ of the left side (*upper*) demonstrate a rapid sinus rhythm or supraventricular rhythm, whereas leads $V_2$–$V_6$ (left side) reveal ventricular tachycardia at a rate of 245 bpm. The right-sided ECG (*lower*) demonstrates in lead $V_{1R}$ the native heart ventricular tachycardia and a rapid sinus rhythm of the heterotopic allograft at a rate of 154 bpm in leads $V_{2R}$–$V_{6R}$. The diagnosis of native heart ventricular tachycardia was retained and the patient was given a synchronized DC shock of 50 Joules with one defibrillator paddle under the left clavicle and the other on the left mid-axillary line at the

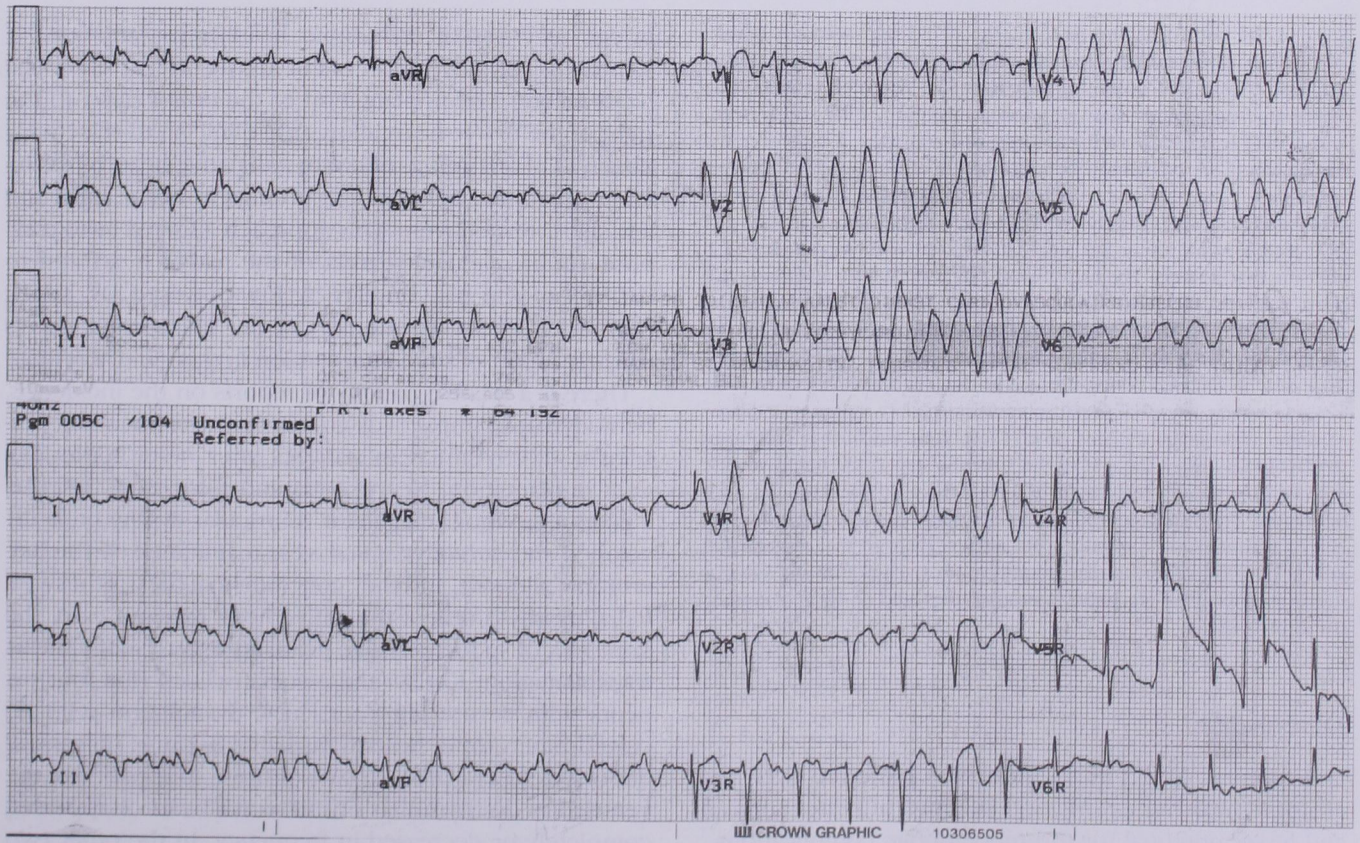


**Fig. 1. (*Upper*) Twelve-lead electrocardiograph taken in the standard manner (left precordials). Precordial leads $V_2$–$V_6$ show rapid ventricular tachycardia of the native heart, whereas lead $V_1$ shows rapid sinus rhythm of the donor heart. (*Lower*) Twelve-lead electrocardiogram taken in mirror image (right sided). Precordial lead $V_{1R}$ shows rapid ventricular tachycardia of the native heart, whereas leads $V_{2R}$–$V_{6R}$ show rapid sinus rhythm of the heterotopic allograft.**



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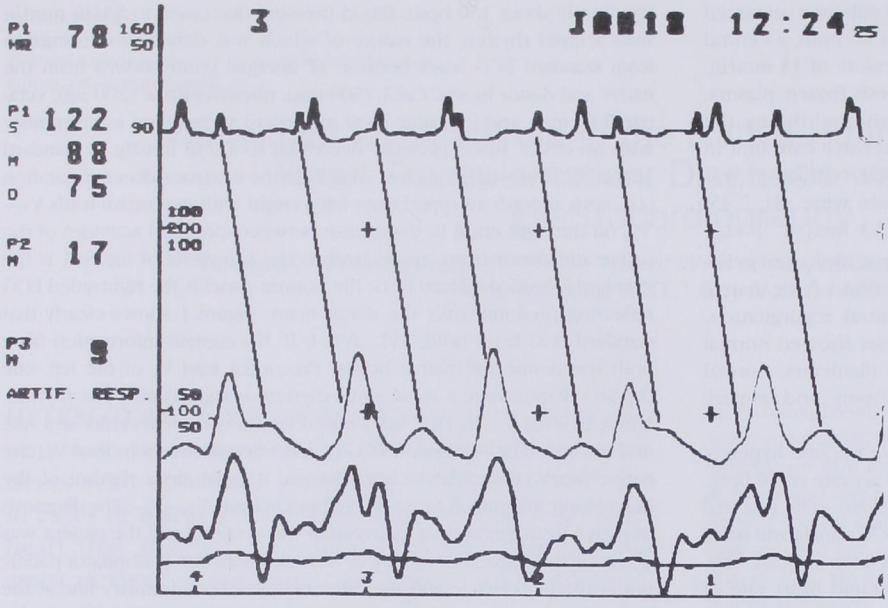


**Fig. 2. From upper to lower: ECG lead II, radial arterial pressure, left atrial pressure, and central venous pressure of the patient 12 h after the native heart ventricular dysrhythmia. The native heart beats at a regular rate of 78 bpm, whereas the donor heart rate is faster at 128 bpm. The native heart generates the larger arterial pressure waves. The diagonal lines illustrate the electromechanical correspondence of the smaller QRS complexes (allograft) to the smaller pressure waves generated by the allograft at this time. The left atrial pressure tracing reflects some degree of mitral regurgitation.**

level of the tenth rib. The first DC shock did not restore a normal rhythm, and a second DC shock of 100 Joules restored sinus rhythm in the native heart and stable hemodynamics. A repeated transthoracic ultrasound examination showed good contractility of the donor heart, with a mild hypokinesis of the anterior wall and apex, a trace of mitral insufficiency, an estimated ejection fraction of 60%, and no signs of tamponade. The patient's condition remained stable and the trachea was extubated 12 h after the dysrhythmia. Figure 2 shows a record of ECG lead II, radial artery, left atrial, and central venous pressures 12 h after the dysrhythmia. The native heart rate was 78 bpm and the donor heart rate was 128 bpm. The smaller QRS complexes belong to the donor heart. The large arterial pressure waves are generated by the native heart, whereas the donor heart generated smaller pressure waves. This pattern reversed during the rest of the postoperative course. The V-waves on the left atrial pressure tracing probably represent a degree of mitral regurgitation at this time. There were no further significant dysrhythmias during the rest of the patient's stay in the intensive care unit, and the patient was discharged from there on the tenth day after operation. Transthoracic and transesophageal echocardiography and heart catheterization 3 months after surgery showed that the allograft generated 100% of the cardiac output, normal ventricular and valvular function of the allograft, stable hemodynamics, and no signs of congestive heart failure.

## Discussion

Orthotopic heart transplantation is preferred over heterotopic heart transplantation because of better survival and lesser potential for thromboembolic complications.[7] Yet heterotopic heart transplantation remains a viable option in the presence of reversible pulmonary hypertension, in cases of marked body-size mismatch, and as a bridge to eventual orthotopic heart transplantation.[1,2,5] Ventricular dysrhythmias occur frequently in end-stage heart failure. The implantation of the donor heart to the right of the native heart allows the clinician to selectively obtain ECG recordings of donor and native heart by examining precordial ECG lead recordings from the right and left chest, respectively. This approach allowed us to diagnose ventricular tachycardia of the native heart and to defibrillate the native heart by low-powered DC shock on the left chest. To the best of our knowledge, this is the first report of such intervention in this clinical setting.

There is a relative paucity of reports on the incidence, hemodynamic impact, diagnosis, and treatment of ventricular dysrhythmias in patients after heterotopic allograft implantation. Neerukonda and associates[4] describe a patient in whom malignant ventricular dysrhythmia developed and hemodynamics were maintained by the heterotopic allograft, and they conclude that these otherwise lethal dysrhythmias of the native heart may not carry the same risk of sudden cardiac death after heterotopic heart transplantation. Kotliar and associates[5] describe four patients with severe left ventricular dysfunction who received heterotopic heart transplants because only small donors were available. In two of these four patients, the ventricular dysrhythmias of the native heart disappeared completely or decreased in frequency. In the other two patients, ventricular dysrhythmias remained, yet they maintained stable hemodynamics by virtue of the function of the heterotopic allograft. These authors suggest



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that heterotopic heart transplantation is not absolutely contraindicated in the face of severe ventricular dysrhythmia, as was previously suggested.

We report a case in which ventricular dysrhythmia of the native heart with important hemodynamic effects was diagnosed with right- and left-sided 12-lead ECG, and in which the dysrhythmia was terminated by low-powered DC shock on the left side of the chest.

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