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CASE REPORT

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# Successful ablation of atrioventricular accessory pathway after cardiac transplantation

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**Abstract** Liberalization of stringent guidelines regarding donor selection is acceptable in the case of critical recipient condition. Few cardiac allografts with preexisting accessory atrioventricular pathways have been implanted. We describe the successful radiofrequency modification of the atrioventricular node and ablation of an accessory pathway after cardiac transplantation. Although the previously healthy donor had no history of arrhythmia, the recipient's postoperative course was characterized by multiple bouts of reentry tachycardia. The highly successful catheter-based ablation techniques available to cure this condition favor the use of donor hearts with a preexisting accessory pathway.

**Keywords** Heart transplantation · Donor · Wolff-Parkinson-White · Radio frequency ablation

## Introduction

Although much effort has been put into public awareness of the growing donor shortage, this has not resulted in a significant increase in available allografts. The gap between the number of recipients waiting for transplant and the stagnating donor organ supply is still widening [1, 2], resulting in a substantial mortality rate. Attempts to reduce mortality on the heart transplant waiting list have led to the recently published recommendations to improve the yield of donor evaluation [1, 2]. Emphasis is put on both the use of so-called marginal donor hearts and on the hemodynamic and metabolic management in the case of left ventricular dysfunction prior to explantation. Average donor age increased steadily from 1986 and reached 30 years in 1996 [3]. Conflicting results about the long-term outcome of recipients of older donor hearts have been published, the main concern being the transmission of donor atherosclerosis [4, 5, 6, 7, 8]. Most transplant programs are reluctant to accept hepatitis C virus positive donors for a seronegative recipient, unless informed consent can be obtained in a critically ill patient [9].

Few cardiac allografts with preexisting accessory atrioventricular (AV) pathways have been implanted. We report the case of successful radiofrequency modification of the AV node and ablation of an accessory pathway after cardiac transplantation.

## Case report

In October 1998 a 56-year-old woman with endstage idiopathic cardiomyopathy was hospitalized for intractable heart failure. Pretransplant screening revealed no contraindications, and the patient was put on the waiting list. After 3 days the donor heart of a 21-year-old, previously healthy man became available. On arrival at the donor center the cardiac surgeon noted a short PR interval on the electrocardiogram (ECG). Since the recipient was in critical condition, the procedure was continued. During in situ inspection the donor heart developed tachycardia, and cold cardioplegia was rapidly initiated. From the surgical standpoint the transplantation was uneventful. Weaning from extracorporeal circulation, however, was complicated by several episodes of wide complex regular tachycardia. Postoperative ECG demonstrated Wolff-Parkinson-White (WPW) pattern type A (Fig. 1). Despite low-dose flecainide (50 mg twice daily), manipulation of the guidewire and bioptome during biopsy procedures induced tachycardia. After 3 weeks the patient developed grade IIIA rejection and was treated with high-dose intravenous corticoid pulses. During the next few days reentry tachycardia required interruption with intravenous adenosine on several occasions (Fig. 2). Electrophysiological study and ablation of the accessory pathway were scheduled. After introduction of the ablation catheters reentry tachycardia developed which was terminated with intravenous adenosine. After 6 s, during which only atrial complexes were registered, sinus rhythm resumed, thus suggesting antegrade refractoriness of the accessory pathway. Despite thorough electrophysiological mapping, the presumed additional pathway could not be detected. However, circus movement was traced in the AV region and radiofrequency ablation was employed to modify the AV node. The ECG, taken immediately after AV node modification, showed no signs of preexcitation (Fig. 3). Six weeks after the procedure, however, the ECG tracing was comparable to that obtained immediately after transplantation. In January 2000, after an asymptomatic interval of more than 1 year, the patient reported recurrent episodes of palpitations. This time, electrophysiological mapping identified the AV accessory pathway located in the left lateral region, which was subsequently successfully ablated. Until now, no recurrence of tachyarrhythmia has occurred.

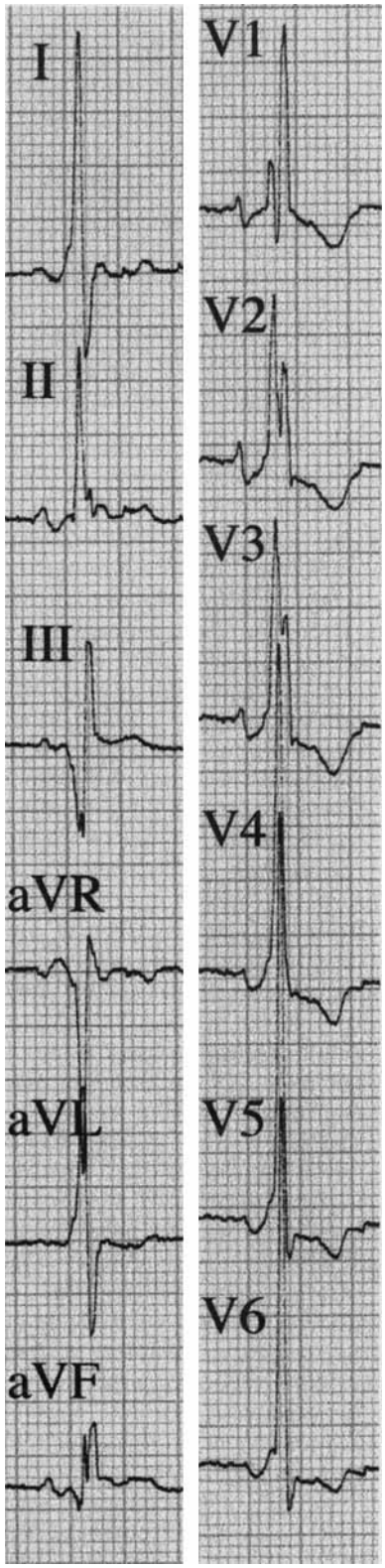


**Fig. 1** Postoperative ECG showing sinus rhythm and the presence of delta waves (leads I and V5 shown)

## Discussion

Several authors have shown that the current rules of size matching can be liberalized if careful hemodynamic guidance in the early postoperative period is guaranteed [10, 11]. Ott and colleagues [12] reported a series of 72

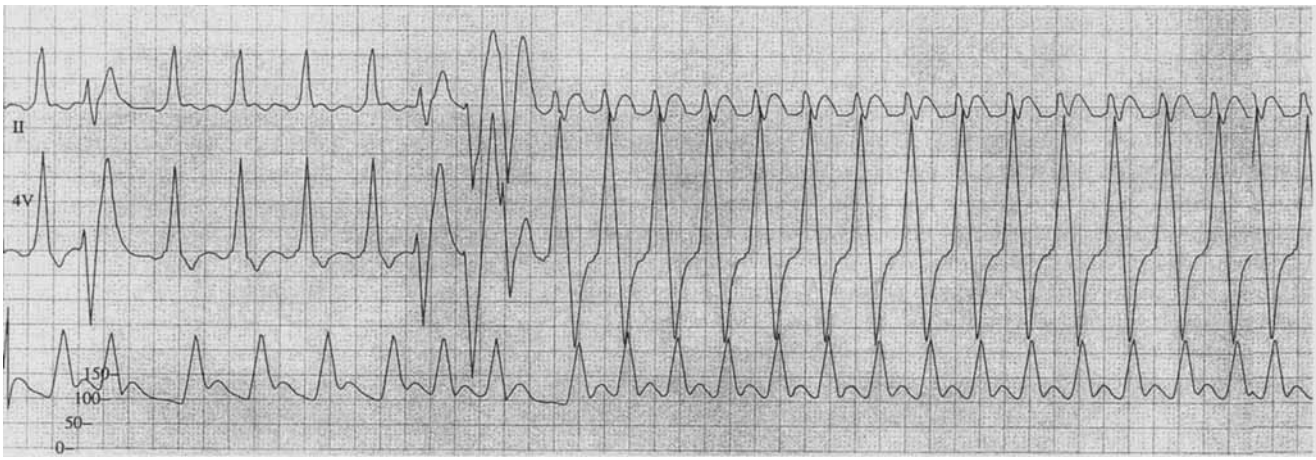


**Fig. 2** ECG showing regular wide-complex tachycardia (leads II and V4 shown) during an episode of grade III rejection

patients receiving hearts from so-called high-risk donors (prolonged cardiopulmonary resuscitation, age >40 years, undersizing >20% body weight, high inotropic requirements, wall motion abnormalities, elevated myocardial enzyme levels, and cold ischemic time >4 h). Although a higher incidence of graft vasculopathy was noted, survival and cardiac function did not differ from those in the low-risk group. In an attempt to resuscitate hearts with myocardial dysfunction, experimental and clinical protocols have shown the value of triiodothyronine in restoring part of the neurohumoral derangements accompanying brain death [13, 14]. The Papworth approach, combining hemodynamic (guidance via pulmonary artery catheter) and metabolic guidelines (triiodothyronine, vasopressin, methylprednisolone, insulin) successfully expanded their donor pool with 30% [15].

Few surgeons have implanted a donor heart with a preexisting accessory AV bundle. A Medline search (heart transplantation, WPW) revealed only seven published cases [16, 17, 18, 19, 20, 21, 22]. In three patients the WPW syndrome was apparent from the ECG prior to explantation of the donor heart. Although donor tachycardia had been documented prior to death in only two, six of the seven recipients developed posttransplant tachycardia that required treatment (catheter ablation in four, pharmacological therapy in two, surgical ablation in one). Ollitrault and colleagues [20] reported a patient in whom the intermittent apparition of the WPW pattern was related to cellular rejection. In two cases surgical ablation of the accessory pathway was attempted during procurement [16, 22]. Unfortunately, Blanche et al. [16] had to resort to radiofrequency ablation afterwards because of erroneous location during the surgical procedure. Since macroscopic identification of the additional pathway is highly inadequate, treatment should be postponed until electrophysiological mapping and radiofrequency ablation are feasible. In the present case report repetitive episodes of reentry tachycardia required both AV node modification and ablation of a bypass tract. The development of reentry tachycardia in recipients of allografts from donors without arrhythmic history suggests that the effect of cardiac denervation on aberrant AV conduction properties is relevant.

Despite limited experience the diagnosis of an accessory pathway at the time of procurement should probably not preclude harvesting of an otherwise structurally and functionally normal donor heart. This point has also been made by other authors [19]. However, to anticipate hemodynamic problems in the peri- and postoperative phase electrophysiological counseling seems the most prudent way to proceed. Arrhythmia at the time of prelevation should lead to prompt cardioplegia. Reentry tachycardia during the early postoperative period and in the intensive care unit can usually be easily interrupted with intravenous adenosine or overdrive pacing. If frequent episodes of arrhythmia occur, treatment with class IC antiarrhythmic drugs can be initiated. If atrial fibrillation with a rapid ventricular response develops, immediate electrical cardioversion is required. To minimize adrenergic stimulation the use of intravenous inotropics and isuprel should be minimized and bradycardic episodes managed using atrial pacing. Of course, drugs promoting conduction through the accessory pathway should be avoided (e.g., verapamil and diltiazem). Although the reported cases are few, the literature suggests that the absence of known arrhythmic antecedents of the donor should not influence the decision of curative electrophysiological treatment in the recipient. The timing of this procedure should, if feasable, be postponed until healing of the cardiac sutures is expected.

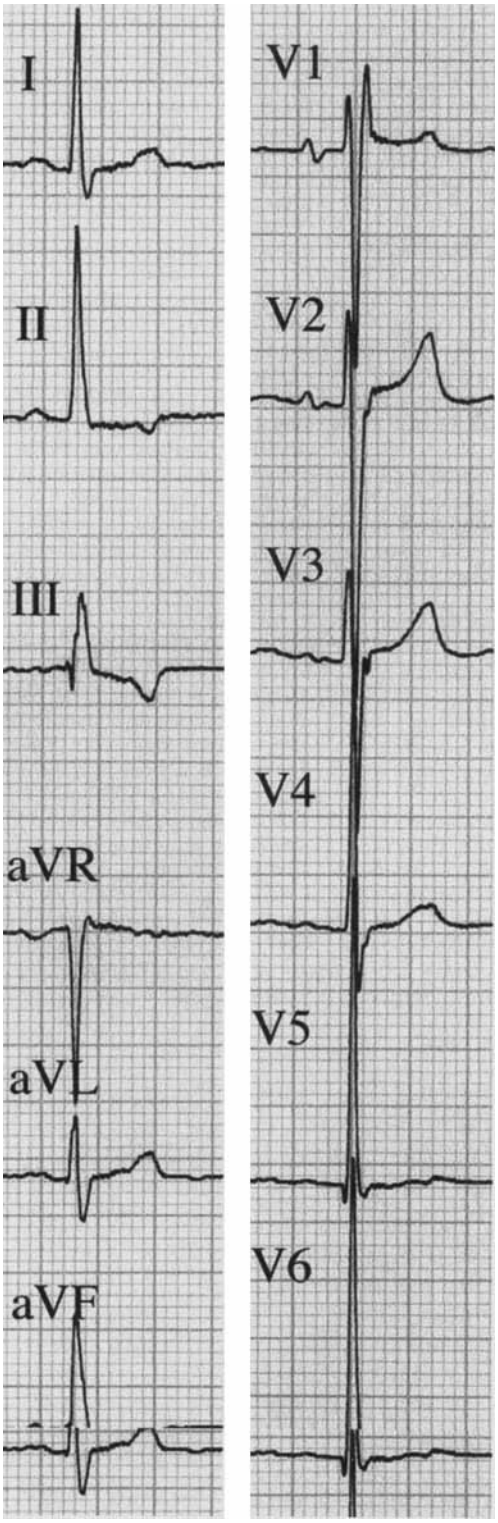


**Fig. 3** ECG recording after radio frequency AV node modification showing disappearance of delta waves in leads I and V5

## Conclusion

Today, the lack of suitable donor hearts is emphasized by an unrelentingly increasing pool of heart failure patients and hence an unacceptable mortality rate on waiting lists. It is therefore important for transplant programs to consider a liberalization of stringent donor criteria, especially when possible recipients are in urgent need. Although not frequently encountered in the general population, our case report adds to the evidence that the existence of a substrate for AV reentry tachycardia should not preclude a potential donor heart of being harvested since electrophysiological treatment is highly effective and save in such cases.

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