The Influence of Brain Death and Donor Heart Re-Transplantation on Intramyocardial Electrograms

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Summary

During heart transplantation (HTX), we routinely implant a telemetric pacemaker and two fractal coated, epimyo-cardial electrodes at the ventricles. It has been shown that parameters of the intramyocardial electrogram (IEGM), recorded for non-invasive rejection monitoring, correlate to the results of an endomyocardial biopsy and that rejection-independent factors may also influence the electrical activity of the heart. In the following report, the behavior of IEGMs of a transplanted heart before and during brain death and, subsequently, after re-transplantation (re-HTX) is described. After a primarily successful orthotopic HTX, subarachnoidal bleeding caused the first recipient's brain death; this was accompanied by changes of the IEGM. After re-HTX to a second recipient, the signals recorded during pacing from the right ventricular electrode position in recipient 2 were found to show a fairly similar signal morphology as compared to those obtained from a comparable electrode position in recipient 1 prior to brain death. Donor heart re-HTX may be feasible, thus expanding the donor pool. Intramyocardial electrograms recorded during pacing and from a certain ventricular area yield a highly reproducible signal morphology - even after brain death, re-HTX, and slight changes in lead position.

Key Words
Donor heart retransplantation, intramyocardial electrogram, brain death, pacemaker telemetry

Introduction

It has been shown that parameters of the intramyocardial electrogram (IEGM), recorded for non-invasive rejection monitoring after heart transplantation (HTX), correlate to the results of an endomyocardial biopsy and that rejection-independent factors may influence the electrical activity of the heart as well [1][2]. Only few reports exist about re-HTX after brain death of a heart transplant recipient [3]. In the present paper, we describe the re-HTX of a donor heart after brain death of the first recipient and the influence of electrode implantation site and brain death on IEGMs.

The analysis of IEGMs for repeated non-invasive detection of acute cardiac allograft rejection has been presented by our group and is described in detail elsewhere [4][5][6]. In brief, during HTX, we implant a telemetric pacemaker (Physios CTM 01, BIOTRONIK, Berlin, Germany) and unipolar, fractal coated, epimyocardial electrodes (ELC 54-UP, BIOTRONIK). Two electrodes are implanted in the ventricles, with a distance of some centimeters between them. With notebook-based pacemaker programming and an IEGM recording system (SWD 1000, BIOTRONIK), both electrodes record spontaneous ventricular electrograms (SVE) during spontaneous beating, as well as ventricular evoked responses (VER) during pacing. The signals are recorded under standardized conditions. The sampled data are sent via the Internet to a data analysis workstation for signal processing and report generation [7]. To exclude artifacts and aberrant heartbeats and to reduce noise and beat-to-beat variability, the signal morphology is rigorously checked and averaged. Finally, the diagnostic parameters are extracted from the averaged SVEs and VERs. To date, only some factors that influence IEGMs have been investi-
gated. Previous studies have shown that these signals are influenced by rejection, rejection therapy [8], the time of day [9], the position of the electrode [10], clinically apparent infection, and ventricular dilatation. It has also been shown that VERs show higher long-term reproducibility than SVEs [11].

Case report

Four years after coronary bypass grafting and aortic valve replacement, a 61-year-old man was admitted for orthotopic HTX. The patient was resuscitated after ventricular fibrillation. As a bridge to transplant, we implanted a cardioverter-defibrillator. Later on, the patient was catecholamine-dependent, and low doses of prostaglandin were administered. Nine months after transplant evaluation, the heart of a 19-year-old male donor became available, and HTX was successfully performed in recipient 1. The patient was discharged from the intense care unit on day 7 after HTX. During the transplantation procedure, we routinely implanted a telemetric dual-chamber pacemaker system for non-invasive rejection monitoring. Both electrodes were screwed in at the right ventricular outflow tract near the septum with a distance of 2.5 cm between them (figure 1). Initial postoperative outcome and patient recovery were excellent. But on day 10 after HTX, subarachnoidal bleeding caused brain death in recipient 1. The patient was discharged from the intensive care unit on day 7 after HTX.

Discussion

This case report indicates that the re-HTX of donor hearts is feasible, thus expanding the donor pool. During brain death, changes of the surface electrogram, reduced myocardial contractility, arrhythmia, and cardiac dysfunction have been reported. The behavior of IEGMs during brain death and after re-HTX has not yet been described. Our routinely applied measurements were performed. Signal quality was excellent. No rejection occurred in recipient 1. During brain death, both spontaneous and paced electrograms changed substantially. Signal amplitudes during the depolarization phase increased, and delayed repolarization resulted in signal prolongation, even for the paced signals, which were all recorded with the same pacing frequency of 100 ppm (figure 2). These changes were accompanied by the corresponding changes of diagnostic parameters.

After re-HTX, spontaneous signals and parameters from both leads in recipient 2 were significantly different compared to those of recipient 1 before brain death. The same was true for the signals during pacing from lead E2, the lead being located at a completely different position in both recipients. However, paced signals from lead E1, which had been implanted at a comparable position in both recipients, correlated fairly well. Signal duration as well as the diagnostic parameter values were found to be in good accordance with the values observed in recipient 1 before brain death (figure 3).
method for non-invasive rejection monitoring allowed
IEGMs to be recorded under these circumstances.
Recordings during brain death showed delayed repolarization as compared to signals previously sampled. Since the heart had already been denervated in recipient 1, this occurrence must be due to humoral and hemodynamic effects only. Hence, our findings underline the close relationship between brain function and the cardiovascular system - even in patients with completely denervated hearts. Before brain death and after re-HTX, VERs originating from the similar right ventricular electrode implantation sites for E1 showed similar signal morphologies. On the contrary, SVEs were different, even those measured at the comparable right ventricular lead positions. Electrograms from E2, of course, showed different signal morphologies due to substantially different lead positions for E2 in recipient 1 and recipient 2. These findings support the hypothesis that electrode position in the myocardium influences signal morphology. However, for paced electrograms, a similar signal morphology is seen at corresponding electrode implantation sites, particularly during the repolarization phase, even in different heart recipients.

Figure 2. Comparison of SVEs and VERs obtained from recipient 1 before and during brain death from both right ventricular lead positions, E1 and E2.


Figure 3. Comparison of SVEs and VERs obtained in recipient 1 before brain death and in recipient 2 after re-HTX from both lead positions, E1 and E2.

References


