Effects of Different Ventricular Pacing Modes on Ventricular Repolarisation in Patients Undergoing Cardiac Resynchronisation Therapy: A Single-centre Study



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Aims	The aims of this study were to compare ventricular repolarisation parameters in patients who underwent cardiac resynchronisation therapy (CRT) at Sun Yat-sen University Memorial Hospital under different ventricular pacing modes and to understand effects of epicardial pacing on ventricular repolarisation.
Methods	The study included 55 patients who underwent CRT. During follow-up outpatient visits three months after CRT implantation, the CRT devices were programmed to deliver no pacing (with the exception of patients with third-degree atrioventricular block), biventricular pacing (BivP), right ventricular endocardial pacing (RV-EndoP), and left ventricular epicardial pacing (LV-EpiP). Signals from the standard 12-lead ECG were recorded simultaneously to measure the QT interval, JT interval, and Tp-e interval, from which the heart rate-corrected QTc interval, JTc interval, and Tp-ec interval were determined.
Results	The JTc interval and Tp-ec interval were prolonged during LV-EpiP and BivP compared with those during spontaneous cardiac rhythm and RV-EndoP. The JTc dispersion and Tp-ec dispersion were not significantly different among the four pacing modes.
Conclusion	Epicardial pacing prolongs myocardial repolarisation time and increases transmural dispersion of repolarisation. Epicardial pacing has no significant effect on the dispersion of regional ventricular repolarisation.
Keywords	Chronic heart failure • Epicardial pacing • Transmural dispersion of repolarisation • Arrhythmia • Cardiac resynchronisation therapy

Introduction

Cardiac resynchronisation therapy (CRT) is a relatively new approach to the treatment of chronic heart failure. Multiple clinical trials have confirmed that CRT can reduce total mortality in patients with heart failure, in particular, the risk of cardiac function deterioration-induced death. However, the impact of CRT on sudden cardiac death is still uncertain [1,2].

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We found in our clinical practice that cardiac function is improved in some patients receiving CRT, but a minority of patients experience an increase in malignant ventricular arrhythmias. Such malignant ventricular arrhythmias are not responsive to anti-arrhythmic drugs and can only be treated with frequent implantable cardioverter defibrillators (ICD) shocks until, ultimately, the only available option is to terminate left ventricular pacing [3-5]. After CRT, the endocardial to epicardial left ventricular activation sequence reverses from epicardial to endocardial. The left ventricular epicardial pacing mode adopted by CRT changes the ventricular depolarisation and repolarisation sequence, and this non-physiologic pacing mode may have a potential arrhythmogenic effect, thereby increasing the risk of sudden cardiac death (SCD). This increased risk may, to some extent, offset the beneficial effect of cardiac resynchronisation.

Some investigators have found that left ventricular epicardial pacing can also prolong repolarisation time in patients with heart failure and increase transmural dispersion of repolarisation (TDR) [6,7]. However, other investigators have obtained different results in similar studies. In a study of 28 heart failure patients undergoing CRT, van Huysduynen et al. [8] showed that TDR during simple left ventricular pacing or biventricular pacing was not increased compared with that during right ventricular pacing.

Therefore, we aimed to compare ventricular repolarisation parameters in patients who underwent CRT at our hospital under different ventricular pacing modes in order to understand the effects of epicardial pacing on ventricular repolarisation. Our results may provide a basis for future improvements in CRT.

Subjects and Methods

Subjects

Sixty-four patients with heart failure underwent CRT involving the implantation of either standard CRT pacemakers or CRT devices with defibrillation capability (CRT-D) at Sun Yat-sen University Memorial Hospital from June 2008 to May 2012. Six patients with persistent atrial fibrillation were excluded and three patients were lost to follow-up. A total of 55 patients were included in this study. All patients met class I and class II indications for CRT/CRT-D according to ACC/AHA/HRS 2008 Guidelines for Device-Based Therapy of Cardiac Rhythm Abnormalities [9].

Data collection

At the follow-up visit in the outpatient clinic three months after CRT implantation, the CRT devices of all the patients were programmed to deliver a sequence of various pacing parameters: no pacing (with the exception of patients with third-degree atrioventricular block), biventricular pacing (BivP), right ventricular endocardial pacing (RV-EndoP), and left ventricular epicardial pacing (LV-EpiP). Signals from the standard 12-lead ECG (Nihon Kohden ECG-9020 P) were recorded simultaneously at a paper speed of 50 mm/s. In

order to reduce the impact of heart rate on the measurement results, the pacing rate of all the CRT devices was set at 80 beats/min during ECG recording. If the patient's spontaneous heart rate was more than 80 beats/min, the pacing rate was set over the spontaneous heart rate to ensure complete capture. ECG recording was initiated 60 minutes after the start of each pacing mode to reduce the influence of cardiac memory.

The QRS duration, QT interval, JT interval, and Tp-e interval were measured in the twelve-lead ECG. The QT interval was taken as the time between the start of the ORS complex and the end of the T wave. In cases where the end of the T wave was not clear, it was defined by the point of intersection between the tangent line of the end of the T wave and the equipotential line, and represents the sum of the ventricular depolarisation time and repolarisation time. The JT interval was defined as the time between the J point to the end of the T wave, representing ventricular repolarisation time. Finally, the Tp-e interval was measured from the peak to the end of the T wave and represents the difference between the epicardial repolarisation time and the midmyocardial repolarisation time. In cases in which there was T wave inversion or a two-way T wave was observed, the T-wave interval was taken as the time from the lowest point of the T wave to the end of the T wave. A T-wave interval was not recorded if the T-wave amplitude was less than 1.5 mV. Each measurement represents the average value of the measurements taken in three consecutive cardiac cycles. All the indicators were measured independently by two cardiologists. If the measurement results differed by more than 20 ms, the two cardiologists would discuss the results or another cardiologist would repeat the measure. The general condition of the patient, NYHA functional classification, left ventricular ejection fraction (EF), and the placement of the left ventricular electrode were reviewed.

Data analysis

The QT interval, JT interval, and Tp-e interval were corrected for heart rate to give the following values. QTc was obtained using Bazett's formula: QTc = QT / (R-R) $^{1/2}$, and QTcd was defined as the difference between the longest and shortest QTc in the 12-lead ECG, which provides a measure of the heterogeneity of ventricular depolarisation and repolarisation. JTc was calculated as JTc = JT / (R-R) $^{1/2}$, and JTcd was defined as the difference between the longest and shortest JTc in the 12-lead ECG, reflecting heterogeneity of ventricular repolarisation. Tp-e represented the longest Tp-e interval in the precordial lead, and Tp-ec = Tp-e / (R-R) $^{1/2}$. Tp-ecd was defined as the difference between the longest Tp-ec interval and the shortest Tp-ec in the precordial lead in the same cardiac cycle.

Statistical analysis

Single-blind data analysis was performed. All data were shown as mean \pm standard deviation, and data were analysed using the SPSS16.0 software. Differences in the ECG indicators under different pacing modes were analysed using the univariate analysis of variance (ANOVA, F test), and all tests were two-sided. The between-group differences

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