Optimal atrioventricular delay setting determined by evoked QT interval in patients with implanted stimulus-T-driven DDDR pacemakers


Second Department of Internal Medicine, Yokohama City University School of Medicine, Yokohama, Japan

Cardiac function is improved by optimizing the atrioventricular (AV) delay. An automatic optimizing function of AV delay may be necessary to achieve the most favourable haemodynamic state in paced patients. The QT interval may change when cardiac function is improved by optimizing the AV delay. The QT or stimulus-T interval is used as a sensor for rate-responsive pacemakers. Evoked (e) QT interval is measured as the time duration from the ventricular pulse (stimulus) and the T-sense point that is the steepest point of the intracardiac T wave (stimulus-T interval). The relationship between AV delay, eQT interval and cardiac function was studied in 10 patients (73 ± 10 (SD) years old) with an implanted stimulus-T-driven DDDR pacemaker. Cardiac output (CO) and pulmonary capillary wedge pressure (PCWP) were measured by Swan-Ganz catheter. The AV delay was prolonged stepwise by 30 ms. Electrocardiogram event markers which indicated ventricular spike and sensed T wave were recorded, and the interval between two event markers was measured as eQT interval. When AV delay was changed from 240 ms to the AV delay at which CO was maximal (172 ± 33 ms), eQT interval prolonged from 346 ± 60 to 353 ± 62 ms (P<0.01). There was a significant positive correlation between the optimal AV delay at which CO was maximal (172 ± 33 ms) and the optimal AV delay which was predicted from the maximum eQT interval (179 ± 37 ms, r=0.92, P<0.001). When AV delay was changed from 240 ms to the predicted optimal AV delay, CO increased from 4.2 ± 0.7 to 4.5 ± 0.8 l min⁻¹ (P<0.001) and PCWP was decreased from 7.1 ± 4.0 to 5.7 ± 3.1 mmHg (P<0.05). In conclusion, the optimal AV delay can be predicted from the eQT interval which is sensed by an implanted pacemaker. Automatic setting of the optimal AV delay may be achieved by the QT sensor of an implanted pacemaker.

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Key Words: PQ interval, QT interval, DDD pacemaker, atrioventricular block, cardiac output, sensors.

Introduction

Atrioventricular (AV) interval is important for optimization of cardiac function. It has been reported that cardiac function can be improved by implanting a DDD pacemaker and setting a short AV delay in patients with severely reduced cardiac function[1–4]. Optimal AV delay setting is important in patients with implanted DDD pacemakers and cardiac function is improved by optimizing the AV delay[1–1].

When the sympathetic nervous system is activated during exercise, heart rate is increased and QT interval is shortened. The QT or stimulus-T interval has been used as a sensor for rate-responsive pacing[12–18]. It was observed that QT interval, at the same heart rate, was longer with DDD pacing than with VVI pacing (unpubl. data). The QT interval may be changed when cardiac function is improved by optimizing the AV delay[19,20]. The present authors have reported that QT interval on the surface ECG is strongly influenced by the AV delay.
Table 1  Patient's characteristics and results

<table>
<thead>
<tr>
<th>Patients no.</th>
<th>Age (years)</th>
<th>Gender</th>
<th>Underlying heart disease</th>
<th>Ejection fraction (%)</th>
<th>eQTI at different AV delay settings (ms)</th>
<th>Predicted optimal AV delay (ms)</th>
<th>Optimal AV delay (COmax) (ms)</th>
<th>Cardiac output (l. min⁻¹)</th>
<th>PCWP (mmHg)</th>
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*AV delay at which cardiac output was maximal.
eQTI, evoked QT interval; AV, atrioventricular; CO, cardiac output; PCWP, pulmonary capillary wedge pressure.
setting, and changes in QT interval are closely related to changes in cardiac function. The optimal AV delay can be predicted to be the AV delay at which the QT interval is maximal [20].

The QT interval is measured by an implanted QT-driven rate-responsive pacemaker as time duration from the ventricular pace to the T-sense point, which is the steepest point of the intracardiac T wave. Evoked QT (eQT) interval can be measured as the interval between the ECG event markers of an implanted pacemaker which indicates the ventricular spike, and sensed T wave (stimulus-T interval) [12–18].

This study investigated the changes in intracardiac eQT interval sensed by an implanted pacemaker according to the changes in AV delay, and relationships between AV delay, QT interval and cardiac function in patients with implanted DDD pacemakers to evaluate whether the optimal AV delay setting could be achieved by measurement of the QT interval.

Subjects and methods

The subjects were 10 patients ranging in age from 56 to 84 years (73 ± 10 years; mean ± SD, four males and six females) with complete AV block. One patient had had a previous myocardial infarction, and one patient had dilated cardiomyopathy. All patients were implanted with a QT-driven DDDR pacemaker and hospitalized. The ejection fraction estimated by M-mode echocardiography [19] was 31–78 (58.3 ± 16.6)% (Table 1).

Cardiac output (CO) was measured by a Swan-Ganz catheter in all patients, and three values of CO were averaged. Pulmonary capillary wedge pressure (PCWP) was also measured by the Swan-Ganz catheter in nine patients.

Electrocardiogram event markers which indicated ventricular spike and sensed T wave were recorded, and the interval between two event markers (stimulus-T interval) was measured as the eQT interval. Five consecutive values of the eQT interval were averaged. Paper speed of the ECG was set at 100 mm . s⁻¹ (Fig. 1).

With AV sequential pacing, the pacing rate was fixed at 70–80 min⁻¹ to eliminate the influence of heart rate. The AV delay was set at 130 ms and prolonged stepwise by 30 ms from 150 ms. Electrocardiogram event marker function of an implanted pacemaker requires a minimum 130 ms AV delay setting. All measurements were performed after 5 min of pacing.

All examinations were performed by the same observer, and measured by another observer who was blinded to the clinical data.
Optimal AV delay was determined as the AV delay at which CO was maximal. When CO was observed to be maximal at multiple values of AV delay, the average value of the AV delays was defined as the optimal AV delay at which CO was maximal.

Measured variables were expressed as mean ± SD. Statistical analyses were performed by paired Student’s t-test and simple linear regression analysis. Values of P<0.05 were considered to be statistically significant.

**Results**

Evoked QT interval changed according to the change in AV delay. When the AV delay was prolonged, CO and QT interval gradually increased and reached a peak, and then decreased. Tracings from a representative case are shown in Fig. 1. When the AV delay decreased from 130 to 180 ms, CO increased from 3·8 to 4·01 min⁻¹ and eQT interval increased from 346 ± 60 to 353 ± 62 ms (P<0.01, Fig. 2). There was a significant positive correlation between the optimal AV delay at which CO was maximal (172 ± 33 ms) and the optimal AV delay which was predicted from the maximum eQT interval (179 ± 37 ms, r=0·92, P<0.001, Fig. 3). When AV delay was changed from 240 ms to the predicted optimal AV delay, CO increased from 4·2 ± 0·7 to 4·5 ± 0·81 min⁻¹ (P<0·001) and PCWP was decreased from 7·1 ± 4·0 to 5·7 ± 3·1 mmHg (P<0·05, Table 1, Fig. 4).

**Discussion**

The amplitude and duration of the T wave, that is, the period of repolarization, are influenced by heart rate and autonomic nervous tone[12–18]. The ventricular depolarization gradient has been shown to be inversely dependent on sympathetically mediated stress and directly dependent on pacing rate[18]. Activation of the sympathetic nervous system during exercise causes an increase in heart rate and shortening of the QT interval. The QT interval has been used as a sensor for rate-responsive pacing[12–18]. The QT interval is measured by an implanted pacemaker as the time duration from ventricular pace to the T-sense point which is the steepest point of the intracardiac T wave[15–17]. This study investigated the changes in intracardiac eQT interval sensed by an implanted pacemaker according to the changes in AV delay. From the results, eQT interval sensed by an implanted pacemaker also changed according to AV delay variations, and when the AV delay was set to give the maximum value of eQT interval, CO showed its maximum value and PCWP showed its minimal value.

It was observed that QT interval, at the same heart rate, was longer with DDD pacing than with VVI pacing when the QT interval vs heart rate relationship was measured (unpubl. data). It has been reported that the QT interval on the surface ECG was easily influenced by AV delay setting, and changes in QT interval were closely related to changes in cardiac function[19,20]. When CO was increased from the minimum to the maximum value by optimizing the AV delay, the QT interval was significantly prolonged. Cardiac output significantly increased when the AV delay was changed, when QT interval prolonged from the minimum to the maximum value. The QT interval changed according to AV delay variations, and when the AV delay was set to give the maximum value of QT interval, CO showed its maximum value[20].

The reasons why changes in QT interval are closely related to changes in cardiac function are not clear. One possible reason is that it depends on changes in autonomic tone due to changes in cardiac function. When the AV delay setting is inadequate, QT interval may be shortened by activation of the sympathetic nervous system. The interval may be prolonged when cardiac function is improved by optimizing the AV delay. Another possible reason is that it may depend on mechano-electrical feedback. The monophasic action potential is decreased by stretch of the heart muscle[23]. It has been reported that the monophasic action

![Figure 3 Relationship between the optimal atrioventricular (AV) delay at which cardiac output was maximal and the predicted optimal AV delay at which eQT (stimulus-T) interval was maximal.](image-url)

<table>
<thead>
<tr>
<th>AV delay at which cardiac output was maximal (ms)</th>
<th>Optimal AV delay predicted from the maximum evoked QT interval (ms)</th>
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potential is prolonged by afterload reduction, and there was a concomitant change in the QT interval of the epicardial ECG in the pig heart[24]. When the AV delay setting is inadequate, QT interval may be shortened by stretch of the heart muscle according to the increased cardiac load.

As the optimal AV delay may change from time to time with changes in cardiac function, an automatic optimizing function of AV delay is necessary to achieve a favourable haemodynamic state. There was a significant positive correlation between the optimal AV delay at which CO was maximal, and the optimal AV delay predicted by the maximum eQT interval. Therefore, the optimal AV delay can be predicted by measurement of the eQT interval. Automatic setting of the optimal AV delay may be achieved by the QT sensor of the implanted pacemaker.

In this study, left ventricular function in these patients was predominately within the normal range. Relationship between CO and eQT interval during exercise is not clear. Further examinations are required.

In conclusion, eQT interval is strongly influenced by the AV delay setting, and changes in eQT interval are closely related to changes in cardiac function. The optimal AV delay can be predicted as the AV delay at which eQT interval is maximal. As the optimal AV delay can be predicted from the eQT interval which is sensed by an implanted pacemaker, automatic setting of the optimal AV delay may be achieved by the QT sensor of an implanted pacemaker.

References


Figure 4 Changes in cardiac output when atrioventricular (AV) delay was changed from 240 ms to the predicted optimal AV delay. When AV delay was changed from 240 ms to the predicted optimal AV delay, cardiac output increased from 4.2 ± 0.7 to 4.5 ± 0.81 min⁻¹ (P<0.001) and pulmonary capillary wedge pressure decreased from 7.1 ± 4.0 to 5.7 ± 3.1 mmHg (P<0.05).


