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The acute hemodynamic effect of dual-chamber pacing: atrioventricular stimulation versus intrinsic atrioventricular conduction

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ABSTRACT

The study aimed to determine whether impedance cardiography-based stroke volume (SV) measurements could be used in optimizing the atrioventricular (AV) interval for dual-chamber pacemakers in patients with preserved AV conduction. The study included 42 consecutive patients (33 males; mean age, 66.8 ± 7.7 years) indicated for a dual-chamber pacemaker or dual-chamber defibrillator. SV was evaluated during intrinsic AV conduction and sequential AV pacing 4 ± 2 days after implantation by using impedance cardiography. During intrinsic AV node conduction, the mean PQ time was 205 ± 61.9 ms. The mean optimal AV delay in the DDD mode was 103 ± 25 ms. The mean SV was 65.8 ± 14.0 ml during intrinsic conduction and increased to 73.6 ± 14 ml ($P < 0.001$) after optimizing the AV interval. The mean increase in SV during optimal AV delay was 17 ± 17% in patients with prolonged AV conduction versus 6 ± 5% in patients with normal AV conduction. Dual-chamber pacing without optimizing AV delay may impair hemodynamics (65.8 ± 14.0 ml for the mean SV of the hemodynamically worst AV delay vs. 61.7 ± 11.7 ml for the mean SV of the intrinsic AV conduction; $P = 0.001$). AV optimizing in patients with a baseline PQ interval of <160 ms did not improve hemodynamics. In patients with dual-chamber pacemakers and a baseline PQ-interval of ≥160 ms, optimizing the AV interval significantly improved the SV. Blindly programming AV delay may be harmful through impairing the hemodynamics.

Keywords: Dual-chamber pacing, Optimization of atrioventricular delay, Impedance cardiography.

INTRODUCTION

Optimal atrioventricular delay (AVD) is critical for cardiac hemodynamics in patients with dual-chamber pacemakers, especially for an appropriately timed atrial systole and an increase in the end-diastolic left ventricular (LV) volume [1-6]. The optimal AVD appears to be determined by a number of cardiac (i.e., hemodynamic and electrophysiological) factors [7-10]. A number of methods has been used to investigate the effects of different AVDs on left atrial and ventricular function, but most are complicated, invasive, or too expensive for clinical routine. Impedance cardiography (IC) and Doppler echocardiography are 2 non-invasive methods for determining the optimal AVD in patients with dual-chamber or atrial-triggered ventricular pacing [2, 11, 12]. However, a disadvantage of echocardiography is that this method is time consuming [13, 14].

Several studies have compared IC with other methods, such as the rmodilution, the indirect Fick (CO_2) method, and radionuclide ventriculography, and found a good correlation in the results between them [15-18]. IC measurements show a high reproducibility and allow the reliable detection of small changes in stroke volume (SV) at various pacemaker settings [19]. Moreover, prior studies have demonstrated that IC was a useful, non-invasive technique for optimizing the AVD and correlated well with Doppler echo in patients with standard DDD pacemakers [3, 11, 19-22]. Uncontrolled studies have shown that symptomatic patients with a PR interval of ≥300 ms and normal LV function improved with dual-chamber pacing; the improvement in this group of patients is considered a Class IIa indication for permanent pacing [23, 24]. When comparing AAI and DDD modes of pacing in patients with sick sinus syndrome, a

normal ejection fraction, and a long PR interval, a previous study found that patients with an AV interval of <270 ms and >270 ms had a higher aortic velocity time integral with AAI pacing and DDD pacing, respectively [25].

The best method for programming AVD in patients with preserved intrinsic AV conduction in the absence of a high degree AV block is still debated. Further, the data conflict on whether intrinsic AV conduction should be preserved in patients paced with a dual-chamber pacemaker in the absence of a high degree AV block [25]. The aim of the current study was to determine whether IC-based SV measurements could be used in optimizing the AV interval in pacemakers for patients with preserved intrinsic AV conduction and in detecting immediate hemodynamic changes with optimized AV delay versus no ventricular pacing.

METHODS

Patients

Forty-two consecutive patients (33 males; mean age, 66.8 ± 7.7 years) with an indication for dual-chamber pacemakers or dual-chamber defibrillators according to current guidelines [26, 27] and sinus rhythm were included in this study between February 2004 and March 2005. The baseline characteristics of the study patients are detailed in Table 1. Evaluation of patients before implantation included a 12-lead surface electrocardiography, as well as echocardiography for measurements of the LV dimensions and LVEF in the apical 4-chamber view.

Device implantation

Dual-chamber pacemakers or dual-chamber defibrillators were implanted using the standard techniques, with atrial and ventricular electrodes positioned at the right appendage and right ventricular apex, respectively.

Pacing study protocol

For optimizing the AVD, all patients were examined in the supine position in a silent environment to reduce the impact of sympathetic activation by external stimuli. A standard protocol involving a period of stabilization and equilibration was performed. Pacemakers were programmed in DDD mode with a lower rate limit of 30 bpm to avoid the effects of atrial pacing on the AV interval [28]. During data acquisition, the telemetry between the implanted device and the programmer was disconnected to prevent interference with the measurement of impedance.

Impedance cardiography

The optimization of AVD was performed using a commercially available system for IC (Task Force Monitor Systems, CN Systems, Graz, Austria). Two electrodes were placed bilaterally to the inferior chest wall in combination with 1 electrode at the neck. A low-amplitude, high-frequency current was delivered via these surface electrodes, and transthoracic impedance (resistance) to this current flow was measured. Changes in transthoracic impedance were measured by means of 4 additional surface electrodes: one pair was placed bilaterally to the sternum, and the second pair was placed bilaterally to the abdomen. SV was calculated on a beat-to-beat basis from the transthoracic impedance signal [29]. SV was measured during intrinsic conduction (VVI mode at 30 beats/min) and AV pacing using a standard protocol. The AV-interval modification involved changes from 80 to 120 ms, in 20-ms steps, and a nominal AV interval of 150 ms. Once the consistent values for SV were confirmed, we proceeded to the next stage of the AV interval in the pacing protocol.

Informed consent

This trial was a prospective one in which all patient had implanted pacemaker. The patients provided informed consent and approval for the study. The study added no harm to the patients either physiologically or psychologically rather than the disease itself. Management was given to them according to the guidelines and standard safety measures.

Statistics

All data are expressed as the mean value ± standard deviation. Statistical analysis was performed using Fisher's exact test comparing more than 2 sets of data. For comparison between 2 sets of data, a student's t-test was used. A P-value of less than 0.05 was considered to be significant. Data processing was done using commercially available software (SPSS version 16.0).

RESULTS

Patients characteristics involved in the study were analyzed and showed that most of them were females (33 females compared to 9 males), with mean age 66.8 years, 59% of them had coronary artery disease (CAD) at baseline, with mean left ventricular ejection fraction (LVEF) 44.8. Detailed characteristics of the patients are shown in Table 1.

Table (1): Patient characteristics.

Characteristic	Mean ± standard deviation
Age (years)	66. 8 ± 7.7
Gender (female/male)	33/9
%CAD	59
QRS (ms)	96.2 ± 30.5
PQ (ms)	205.8 ± 61.9
LVEF %	44.8 ± 16.6

CAD: Coronary artery disease, LVEF: Left ventricular ejection fraction

The mean baseline PQ interval and mean QRS duration during intrinsic AV node conduction were 205 ± 61.9 ms and 96.3 ± 30 ms, respectively. The mean optimal AV delay was 103 ± 25 ms. The mean SV increased from 65.8 ± 14 ml during intrinsic conduction to 73.6 ± 14 ml after optimization of the AVD interval (P < 0.001; Figure 1).

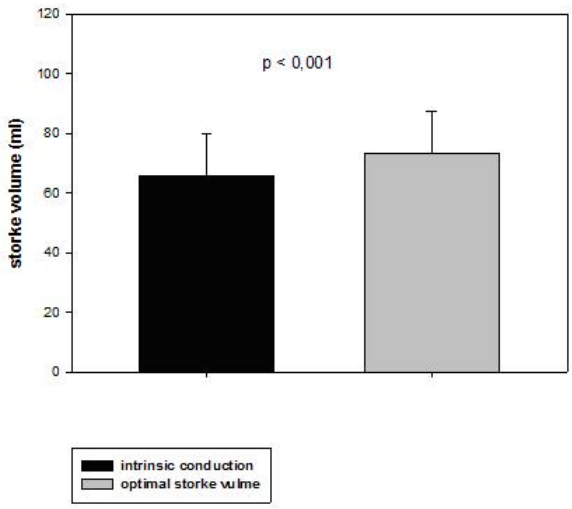

Figure (1): Increase in stroke volume (ml) by intrinsic atrioventricular (AV) node conduction following the use of optimal AV delay measured by impedance cardiography at rest.

This increase in SV during optimal AVD was observed in patients with normal and prolonged AV conduction. In patients with normal AV conduction, the mean SV during intrinsic AV conduction was 71.0 ± 14.8 ml vs. 76 ± 15 ml after optimizing the AVD (P = 0.002). In patients with prolonged AV node conduction, the mean SV during intrinsic AV conduction was 60.4 ± 11.0 ml vs. 71. ± 12 ml after optimizing the AVD (P < 0.0001). However, the worst SV during differently paced AVDs (80–120 ms and 150 ms) was lower than the SV during intrinsic AV conduction (61.7 ± 11.7 ml vs. 65.8 ± 14.0, respectively; P = 0.001; Figure 2).

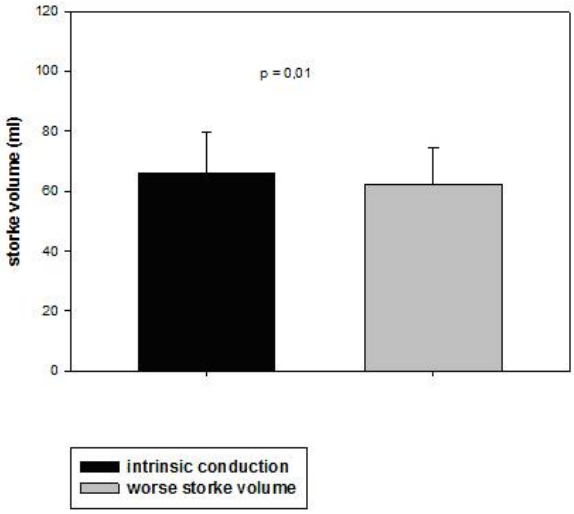

Figure (2): Stroke volume measured by impedance cardiography without ventricular pacing in comparison to SV for the worst AV delay.

Patients with prolonged AV conduction (≥200 ms; 21 patients; mean PQ time, 249.5 ± 45.7 ms) had a significantly lower SV during intrinsic AV conduction than patients with normal AV conduction (<200 ms; 21 patients; mean PQ time, 157.4 ± 16.1 ms). The mean SV was 60.4 ± 11.0 ml and 71.0 ± 14.8 ml in this former and latter groups, respectively (P = 0.014, Table 2).

Table (2): Hemodynamic parameters for optimal AVD and intrinsic AV node conduction.

	SV (ml) with AV intrinsic conduction	SV (ml) with optimal AV Delay	P value
All Patients	65.8 ± 14	73.6 ± 14	<0.001
PQ-Time ≥ 200 ms	60.4 ± 11.0	71 ± 12	<0.0001
PQ-Time < 200 ms	71.0 ± 14.8	76 ± 15	0.002
PQ-Time ≥ 160 ms	62.4 ± 12.5	72 ± 13.6	< 0.0001
PQ-Time < 160 ms	76 ± 14	78.7 ± 14.8	0.1
QRS ≥ 120 ms	64.0 ± 12.4	75.7 ± 9.0	0.01
QRS < 120 ms	66.4 ± 14.6	73 ± 15.4	<0.0001
LVEF ≥ 50%	66.3 ± 17	74.4 ± 16.8	0.004
LVEF < 50%	65.5 ± 11.4	73 ± 11.8	< 0.001

SV (stroke volume), LVEF (left ventricular ejection fraction)

The optimization of the AVD increased the SV in patients with normal and prolonged AV conduction (6 ± 5% vs. 17 ± 17%), with the effect more pronounced in the latter group of patients. In the sub-analysis, we found that patients with a PQ time of <160 ms (mean PQ time, 145 ± 11.5 ms, 10 patients) did not benefit from optimizing the AVD interval (mean SV by intrinsic AV node conduction, 76 ± 14 ml; mean SV by AVD optimization, 78.7 ± 14.8 ml). Optimization of the AV interval significantly improved the SV in patients with bundle branch block (11 patients; mean QRS duration, 139 ± 17 ms), as well in patients with short QRS duration (mean QRS, 81 ± 16.3 ms). The mean SV at the baseline in patients with bundle branch block was 64 ± 12.4 ml and increased significantly to 75.7 ± 9 ml during AVD (P < 0.0001). Meanwhile, the mean SV in patients with the short QRS was 66.4 ± 14.6 ml and increased to 73 ± 15.4 ml during AVD (P = 0.01).

The mean SV during intrinsic AV conduction was 66.3 ± 17.1 ml in patients with a preserved LV ejection fraction (LVEF) (19 patients; mean EF, 61.2 ± 6.2%) and 65.5 ±

11.4 ml in patients with impaired LVEF (23 patients; EF, 31.3 ± 7.9). After optimization of the AV interval, the SV was significantly increased to 74.4 ± 16.8 ml and 73 ± 11.8 ml, respectively (P = 0.004 and P < 0.001, respectively).

DISCUSSION

Hemodynamic measurements 4 ± 2 days after pacemaker implantation demonstrated the hemodynamic benefit of optimizing the AVD. The present study showed that patients with prolonged AV conduction (≥200 ms) had a significantly lower SV than patients with normal AV conduction (<200 ms). This hemodynamic effect with a first AV block has been shown in many previous studies [23] and is associated with exercise intolerance and shortness of breath due to early atrial contraction before complete atrial filling, which leads to a compromising of ventricular filling, an increase in pulmonary capillary wedge pressure, and a decrease in cardiac output follow. This electrical and mechanical remodeling may lead to atrial fibrillation and other atrial arrhythmias. In our study, the SV during the optimal AV interval improved significantly from baseline measurements in patients with AV prolongation. In agreement with our results, a number of studies previously demonstrated improvement in acute cardiac function using DDD pacing compared to AAI pacing with a first degree AV block [25]. Lliev et al. [14] demonstrated that AV interval optimization at a shorter value that forced ventricular pacing was associated with better cardiac performance as compared to normal ventricular activation. Over the past years, there has been a trend to program very long AVDs to achieve functional AAI pacing, even in the presence of a marked first-degree AV block. This trend is based on the results of the DAVID study [24] and a number of other prospective studies (MOST [25], MADIT II [26], Midas 6 [27]. These studies revealed that forced ventricular pacing in the DDD group had resulted in left bundle branch blocks contributing to an increased incidence of congestive heart failure. However, in our study, we found that the optimization of AVD in patients with normal AV conduction (only when AV conduction was ≥160 ms) was associated with significant increase in SV in comparison to their intrinsic AV conduction. Although unnecessary ventricular pacing should be avoided, not all ventricular pacing, even from the RV apex, is automatically bad. The INTRINSIC RV trial demonstrated that DDD pacing with AV hysteresis was superior to intrinsic conduction with VVI at a standby rate of 40 bpm [22]. In the Danpace study, single-chamber pacing was compared with dual-chamber pacing (DDD) in patients with sick sinus syndrome, and was associated with an increased risk of atrial fibrillation when the PQ interval was >180 ms [28]. In agreement with the Danpace trial, our study showed that DDD pacing with an optimization of AV delay was superior to intrinsic conduction when the PQ interval was >160 ms with ventricular pacing showing abnormal ventricular activation sequence.

A major limitation in our study is that we studied only the acute hemodynamic effect but without long-term follow-up. We recommend long-term follow up after AV optimization to avoid deleterious effects of RV apex pacing on myocardial function and to re-establish intrinsic AV conduction by deterioration of LV systolic function. Another potential limitation is that IC can overestimate SV if very short AV intervals are programmed [30]. In our study, we tried to overcome this limitation by programming an AVD of not less than 80 ms.

CONCLUSIONS

In this study, we found that the optimization of AV delay using IC is possible and resulted in significant improvement in acute hemodynamics compared to intrinsic AV conductions, not only in patients with markedly prolonged PR interval but also in those with normal AV conduction when the PQ time was <160 ms with ventricular pacing showing abnormal ventricular activation sequence. We add that blindly programming AVD pacing without optimization of the AV interval may impair the hemodynamics. The mean optimal AV delay in the DDD mode was 103 ± 25 ms. The increase in the mean SV was greater in patients with a prolonged AV interval greater than in those with a normal AV interval.

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COMPETING INTERESTS

The author declares that he has no competing interests.

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