Intracardiac electrogram method of VV-delay optimization in biventricular pacemakers

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Abstract

Ventricle to ventricle (VV) delay optimization can provide an additional benefit to cardiac resynchronization therapy, but the methods currently used for optimization are time consuming and operator-dependent. We present two cases of VV-delay optimization with the use of a new intracardiac electrogram method. (Cardiol J 2007; 14: 305–310)

Key words: cardiac resynchronization therapy, heart failure

Introduction

Cardiac resynchronization therapy (CRT) became the standard treatment of severe heart failure with left ventricular systolic dyssynchrony [1, 2]. Although large clinical trials, which proved the effectiveness of this therapy, evaluated only simultaneous biventricular pacing [3, 4], recent smaller studies demonstrated that sequential biventricular pacing with individualized ventricle to ventricle (VV) delay optimization may provide further benefit [5–8]. Nowadays, the widely available and most commonly used tool for VV optimization is echocardiography; either standard (left ventricular outflow tract velocity-time integral (VTI LVOT) used for calculation of stroke volume), myocardial performance index (MPI), or dP/dT from the spectrum of mitral regurgitation or tissue Doppler imaging [6–8]. Echocardiographic methods, however, have several limitations: they are time consuming, require two persons and are operator-dependent. The optimal VV delay varies over time and should be re-evaluated during follow-ups [9]. Therefore, in routine CRT pacemaker follow-up, there is a strong need for an easier, quicker and more cost-effective method of VV-delay optimization.

A novel method of determining optimal VV delay using intracardiac electrogram (IEGM) signals has recently been described [10]. This method assumes that optimal VV timing occurs when the paced activations from right (RV) and left ventricular (LV) leads meet in the intraventricular septum [11]. In this method, first the delay in milliseconds (ms) between RV and LV intrinsic depolarization (D) is measured on the real-time IEGM from the LV and RV. Afterwards, the different wave front velocities left to right (IVCD-LR: pacing LV, sensing in RV and measuring the distance between the two events in ms on IEGM) and right to left (IVCD-RL: pacing RV, sensing in LV) are measured, and then the two values are subtracted one from the other (ε = IVCD-LR – IVCD-RL). The optimal VV timing is calculated: \( VV_{\text{opt}} = 0.5 \times (\Delta + \varepsilon) \). In most cases, this so-called correction coefficient (ε) is equal or close to 0 ms, and the formula can be simplified: \( VV_{\text{opt}} = 0.5 \times \Delta \), with the ventricle which was...
later on IEGM paced first. This method is implemented in the automated optimization algorithms in the new range of CRT devices [12], but the ‘manual’ use of this method is possible in every device allowing simultaneous registration of intracardiac electrograms from the left and right ventricle.

We present two patients who had their CRT devices optimized by this method in our department.

Case 1

The first patient, ZC, 57 years old female with dilated cardiomyopathy, was admitted to our department because of increasing dyspnoea (NYHA III). ECG at admission showed sinus rhythm with first degree A-V block (PR 240 ms), left bundle branch block pattern (LBBB) of QRS complexes and QRS width 180 ms. In echocardiography, dilated LV dimensions with severely impaired left ventricular systolic function (LVEF 22%), severe functional mitral regurgitation (grade III), estimated pulmonary artery pressure 55 mm Hg and marked inter- and intra-left ventricular dyssynchrony were noted (interventricular mechanical delay 110 ms, difference in time to onset and time to peak between interventricular septum and lateral wall in Pulsewave TDI were 155 and 165 ms, respectively). Despite the fact that she remained on optimal medical therapy using ACE-inhibitor, furosemide, carvedilol and spironolactone, she was admitted to the hospital three times in the last six months because of worsening heart failure. Therefore, a biventricular pacing system was implanted (FRONTIER™ II Model 5596, St. Jude Medical), with LV lead (QuickSite™ 1056T 86 cm, St. Jude Medical) in the lateral vein. Implantation and post-implantation period was uncomplicated. CRT significantly reduced the degree of mitral regurgitation, pulmonary artery pressure decreased (48 mm Hg) and LVEF improved, but still, a slight intra-left ventricular dyssynchrony persisted. On the third day, optimization of VV-delay and AV delay was accomplished in a blinded fashion. One person optimized the VV-delay with the IEGM method and the AV delays using surface ECG method described by Koglek [11, 13]. First, we measured the intrinsic conduction delay between the right and the left ventricle IEGM: \( \Delta = 40 \) ms (Fig. 1). Second, we measured the IVCD-LR = 140 ms (Fig. 2A) and the IVCD-RL = 140 ms (Fig. 2B). The optimal delay was calculated \( VV_{\text{opt}} = 0.5 \times (40 \text{ ms}) = \) LV first 20 ms. Optimal VV-delay proved to be 20 ms. LV paced first and optimal paced and sensed AV-delay were 120 and 80 ms, respectively.

The other person, unaware of the results, performed the optimization using the echocardiographic method. Aortic outflow tract velocity-time integral (VTI_LVOT) was recorded over the same range of VV-delay settings. Several beats were recorded and the average VTI of the last five beats was calculated after determining the optimal VV-delay setting, which in this case was 20 ms LV activated first (Fig. 3A–D). Next, optimizing AV-delay using
the Ritter method [14] was performed, and in this case optimal paced and sensed AV-delay were 110 and 60 ms, respectively. Programming the optimal AV-
delay gave additional benefits (Fig. 3E). Table 1 compares the time spent on the two methods used for optimization. IEGM/ECG methods for AV and VV delay

table 1. Results and the time spent on the IEGM/ECG and echocardiographic optimization of the AV-delay and VV-delay.

<table>
<thead>
<tr>
<th></th>
<th>Value obtained in ECG/EGM method</th>
<th>Value obtained in echo method</th>
<th>Time required for ECG/IEGM optimization</th>
<th>Time required for echo optimization</th>
</tr>
</thead>
<tbody>
<tr>
<td>AV-delay</td>
<td>AV 120 ms/PV 80 ms</td>
<td>AV 110 ms/PV 60 ms</td>
<td>8 min</td>
<td>10 min</td>
</tr>
<tr>
<td>VV-delay</td>
<td>LV 20 ms</td>
<td>LV 20 ms</td>
<td>3 min</td>
<td>30 min</td>
</tr>
<tr>
<td>Total time</td>
<td></td>
<td></td>
<td>11 min</td>
<td>40 min</td>
</tr>
</tbody>
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optimization required a quarter of the time when compared to the echocardiographic method, with good correlation.

The patient was programmed according to the obtained results and after six months remains in NYHA class I/II; echocardiography shows LVEF about 35% and markedly decreased mitral regurgitation (grade I–II) and almost no intra-left ventricular dyssynchrony.

Case 2

The second patient, KB, a 75-year-old male, was admitted to our Department for the replacement of a permanent VVI pacemaker implanted in 1979 because of permanent atrial fibrillation with symptomatic bradycardia. During the years since the first implantation, the patient gradually developed dilated, pacing-induced cardiomyopathy. Recently, his clinical status remained in stable New York Heart Association (NYHA) class III for about 6 months under treatment with an ACE-inhibitor, beta-blocker, spironolactone, digoxin and coumarine derivative. In echocardiography, his left ventricular function was moderately impaired (LVEF 35%), his left ventricle dilated (left ventricular end-diastolic diameter of 71 mm) and relative moderate-severe mitral regurgitation (grade II–III) was present. Pacing the apex of RV resulted in a QRS width of 220 ms and marked dyssynchrony in echocardiography (interventricular mechanical delay 70 ms, difference in time to onset and time to peak between interventricular septum and lateral wall in Pulse-wave TDI were 100 and 80 ms, respectively). Therefore, we decided to upgrade the VVI pacemaker to a biventricular system. The biventricular pacemaker (FRONTIER™ II Model 5596, St. Jude Medical) was connected to the existing right ventricular lead and the LV lead (QuickSite™ 1056T 86 cm, St. Jude Medical) was implanted in the postero-lateral vein. Implantation and postoperative period were uncomplicated. Immediately after implantation, we observed a narrowing of the paced QRS complexes to 150 ms. The patient improved clinically to NYHA class II and was discharged from the hospital 4 days after the procedure with LVEF about 38% and non-optimized VV delay.

After six weeks, he returned to our department for pacemaker follow-up, still in NYHA class II and LVEF similar to the value at discharge. We decided to optimize the VV delay to enhance the clinical benefit of CRT. First, the optimal VV delay was determined using the IEGM method. We measured the intrinsic conduction delay between the right and the left ventricle IEGM (Δ = 90 ms). The IVCD had not been measured in this patient and therefore was assumed to be zero. The optimal VVopt was determined as $VV_{\text{opt}} = 0.5 \times (90 + 0) = \text{LV first 45 ms}$ (Fig. 4).

Next, optimal VV-delay was evaluated in standard Doppler echocardiography by measuring left ventricular outflow tract velocity time integral at seven different settings: 65 ms, 45 ms, 25 ms RV activated first, simultaneous biventricular pacing, then 25 ms, 45 ms and 65 ms LV activated first. Five minutes were allowed to stabilize the heart rhythm and hemodynamics and an average of five $VTI_{LVOT}$ was calculated for each setting. The values obtained are summarized in Table 2. The most important $VTI_{LVOT}$ with the shortest LPEI was noted for a VV-delay of 45 ms (Fig. 5), which was consistent with the IEGM method. The optimal value was programmed, and in the following days, the clinical status of the patient improved. After three months, he remains in NYHA I class and further progress in the reverse remodelling of heart cavities is observed in echocardiography.
Table 2. Results of the echocardiographic optimization of the VV-delay in the Patient 2. Average of 5 measurements for each setting was calculated. Optimal VV-delay = 45 ms LV first.

<table>
<thead>
<tr>
<th>VV-delay LV first</th>
<th>LPEI</th>
<th>Aortic VTI</th>
</tr>
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<tbody>
<tr>
<td>VV = 0 ms</td>
<td>163.0 ms</td>
<td>19.3 cm</td>
</tr>
<tr>
<td>VV = 25 ms</td>
<td>147.9 ms</td>
<td>19.3 cm</td>
</tr>
<tr>
<td>VV = 45 ms</td>
<td>133.1 ms</td>
<td>20.6 cm</td>
</tr>
<tr>
<td>VV = 65 ms</td>
<td>133.1 ms</td>
<td>19.55 cm</td>
</tr>
</tbody>
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VTILVOT — left ventricular outflow tract velocity-time integral; LPEI — left pre-ejection interval

Conclusion

In our patients, the IEGM method gave consistent results with echocardiography; therefore, it has potential practical impact. VV optimization using this method takes less than five minutes, which significantly shortens the biventricular device follow-up even if it is not equipped with any automated optimization algorithm.

References

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Figure 5. Case 2. Left ventricular outflow tract velocity-time integral (VTI\textsubscript{LVOT}) measurements for the VV-delay optimization (only 4 settings, the whole optimization results with the average of 5 measurements presented in the Table 2). VTI\textsubscript{LVOT} marked as AV VTI. Three measurements taken and the average of them calculated. A. Pre-excitation of the left ventricle by 45 ms: average of 3 VTI\textsubscript{LVOT} 20.6 cm (Optimal VV-delay). B. Pre-excitation of the left ventricle by 25 ms: average of 3 VTI\textsubscript{LVOT} 19.3 cm. C. Pre-excitation of the left ventricle by 65 ms: average of 3 VTI\textsubscript{LVOT} 19.6 cm. D. Simultaneous biventricular pacing: average of 3 VTI\textsubscript{LVOT} 19.3 cm.