Should we use the rate-adaptive AV delay in cardiac resynchronization therapy-pacing?

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Aims
Recommendations for programming the rate-adaptive AV delay in CRT.

Methods and results
In cases of continual biventricular pacing, the optimal AV delay in CRT (AVD\textsubscript{opt}) is the net effect of the pacemaker-related interatrial conduction time (IACT), duration of the left-atrial electromechanical action (LA-EAC\textsubscript{long}), and the duration of the left-ventricular latency period (SV-EAC\textsubscript{short}). It can be calculated by AVD\textsubscript{opt} = IACT + LA-EAC\textsubscript{long} − SV-EAC\textsubscript{short}. We measured these three components in 20 CRT–ICD patients during rest and submaximal ergometric exercise (71 ± 9 W) resulting in a 22.5 ± 9.6 bpm rate increase. IACT and SV-EAC\textsubscript{short} did not reveal significant differences. LA-EAC\textsubscript{long}, however, varied significantly by 2 ± 10.7 ± 16.1 ms (P = 0.008) during exercise. In contrast to AVD\textsubscript{opt-VDD}, there was a significant difference in AVD\textsubscript{opt-DDD} of 2 ± 8.8 ± 14.5 ms (P = 0.014) between the resting and submaximal exercise conditions. In DDD pacing, AVD\textsubscript{opt} was shortened by 2.6 ms/10 bpm.

Conclusion
In consideration of the findings of the studies performed to date, the rate-adaptive AV delay should be deactivated.

KEYWORDS
Rate-adaptive AV delay; CRT; AV delay optimization

Introduction
In patients with CRT and congestive heart failure, the AV delay must be programmed to allow for continual biventricular stimulation. Consequently, the AV delay in CRT is usually programmed to be shorter than the AV delay in standard DDD pacing (100–120 ms).1–4

Clinical studies have shown that the principles of the haemodynamic optimization of the AV delay used in anti-bradycardia pacemaker systems for resting conditions can also be used under specific conditions for CRT.5–11

Modern CRT devices, such as the standard DDD pacemakers, are designed to integrate rate-adaptive AV delay programming because such programming has proven benefits.12–16 However, there is a lack of clear recommendations for effective programming of this parameter in DDD pacing as well as in CRT.17,18 This is assumedly the reason why the recommendations for the programming of the rate-adaptive AV delay for CRT devices varies among the different manufacturers (Table 1).

Studies in healthy subjects have shown that the natural AV delay decreases with increasing heart rate (4 ms/10 bpm).19,20

Melzer et al.21 came to a similar finding in a study of DDD pacemaker patients with an EF > 45%, although this finding was not statistically significant.

Scharf et al.22 published the first results of a study of the rate-adaptive optimal AV delay in patients with CRT. Astonishingly, the optimal AV delay increased with increasing heart rate (20 ms/10 bpm).

In consideration of these studies, our goal in this study was to determine the AVD\textsubscript{opt} using the method described by Ismer et al.23 to establish a clear recommendation for the programming of the rate-adaptive AV delay in CRT.

Methods
Inclusion criteria
All patients had been treated with a CRT-ICD according to the current guidelines.17

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During exercise cardioverter-defibrillator programming et al. Ritter delay in cardiac resynchronization therapy Components and calculation of the optimal AV rate of 60–70 bpm. In order to determine the AVDopt in atrial sense mode at a heart rate >90 bpm. The AV delay was optimized in atrial sense mode at a heart rate >90 bpm.

We programmed all the devices to turn the rate responsive mode off. The AV delay was optimized in atrial sense mode at a heart rate >90 bpm. Consequently, the study was restricted to CRT-responders.

Exclusion criteria

Exclusion criteria were heart failure categorized as NYHA class III–IV, unstable angina pectoris, atrial fibrillation, and device malfunction. Furthermore, patients were excluded with any type of disorder of the esophagus because in such cases an esophageal electrode could be harmful to the patient and therefore cannot be used.

Study equipment

We performed simultaneous recordings of transmural flow, the left-atrial esophageal electrogram, and the real-time sense-event markers as proposed by Ismer et al. and Melzer et al. This approach required the placement of a bi-polarr esophageal electrode to provide a filtered left-atrial electrogram (LAE). Therefore, we applied a 5F esophageal electrode (Ospyka T02/5F) in the position of maximal left-atrial deflection and connected it to a filter amplifier (Fiab Rostockfilter, by Vicchio-Firenze, Italy). The filtered esophageal electrogram along with telemetric real-time pacemaker markers provided by the programmers analogue output were superimposed into the display of transmural flow velocity on the Doppler-echo system (Sonolayer SSH-140A/Toshiba®). Therefore, telemetric output of the programmer and output of the Rostockfilter were connected to the DC inputs of the echo device.

Cardiac resynchronization therapy–implantable cardioverter-defibrillator programming during exercise

We programmed all the devices to turn the rate responsive mode off. The AV delay was optimized in atrial sense mode at a heart rate of 60–70 bpm. In order to determine the AVDopt in atrial paced mode, the right atrial heart rate was set ~10 bpm above the sinus rate.

Components and calculation of the optimal AV delay in cardiac resynchronization therapy

Ritter et al. and Ismer et al. define the optimal AV delay for any heart rate, based on diastolic optimization, as the net effect of the individual pacemaker-related interatrial conduction time (IACT: M₀–LA or S₀–LA) and the left-atrial electromechanical action (LA-EAC_long) reduced by the left-ventricular latency period (SV–EAC_short). The four determinants of the optimal AV delay were measured as follows using two screenshots (Figure 1).

Left-atrial electromechanical action (LA-EAC_long) and pacemaker-related interatrial conduction time (IACT) in VDD pacing
To determine the left-atrial electromechanical action (LA-EAC_long), we programmed a maximally long AV delay defined as IACT + 150 ms (averaging 220 ± 34 ms) during which proper biventricular stimulation still occurs in VDD pacing and measured the time between the beginning of left-atrial deflection (LA) in the esophageal electrogram and the end of undisturbed left-atrial contribution (EAC_long) in transmural flow.

In the same screenshot, pacemaker-related IACT in VDD pacing was measured between right-atrial sense-event marker (M₀) and

<table>
<thead>
<tr>
<th>CRT–ICD model</th>
<th>Standard programming of the rate-adaptive AV delay</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lumax HF-T/ Biotronic®</td>
<td>Between 50 and 150 min⁻¹: −5 ms/10 bpm</td>
</tr>
<tr>
<td>Concerto CRT-D/ Medtronic®</td>
<td>Between 80 and 120 min⁻¹: −10 ms/10 bpm</td>
</tr>
<tr>
<td>Contac Renewal 3/Guidant®</td>
<td>Steps of 2 ms between maximal and minimal AV delay</td>
</tr>
<tr>
<td>Epic HF/St. Jude®</td>
<td>Between 90 bpm and maximal sensor or tracking rate: either −10, −20, or −30 ms/10 bpm</td>
</tr>
<tr>
<td>Ovatio CRT-D/Sorin Group®</td>
<td>Between 60 and 120 min⁻¹: −10 ms/10 bpm</td>
</tr>
</tbody>
</table>

Table 1 Standard programming of the rate-adaptive AV delay in cardiac resynchronization therapy–implantable cardioverter-defibrillators

In order to carry out the multiple complex measurements under controlled exercise conditions, it was necessary for the patients to be in relatively good physical condition. Consequently, the study was restricted to CRT-responders.

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In the same screenshot, pacemaker-related IACT in VDD pacing was measured between right-atrial sense-event marker (M₀) and
the beginning of left-atrial deflection (LA) in the esophageal electrogram (IACT = Mₘ-LA).

**Left-ventricular electromechanical latency period** (SV-EAC_short) **and pacemaker-related interatrial conduction time (IACT)** in DDD pacing

To determine the left-ventricular electromechanical latency period (SV-EAC_short), we programmed an unphysiologically short AV delay defined as IACT = 20 ms (averaging 80.5 ± 19.6 ms) measured during DDD pacing (rate = 10 bpm above sinus rate) and measured the duration between ventricular pacing stimulus (SV) and the end of the truncated left-atrial contribution (EAC) in transmitral flow.

In the same screenshot, IACT in DDD pacing was measured between right-atrial pacing stimulus (SA) and the beginning of left-atrial deflection (LA) in the esophageal electrogram (IACT = SA-LA).

Based on these measurements, optimal AV delays were calculated for VDD (atrial-triggered, AVD_optVDD) and DDD (atrial-paced, AVD_optDDD) modes using the following formulas:

\[
AVD_{optVDD} = Mₘ-LA + LA-EAC_{long} - SV-EAC_{short}
\]

and

\[
AVD_{optDDD} = Sₛ-LA + LA-EAC_{long} - SV-EAC_{short}
\]

**Programming of VV delays**

Since the VV delay could not be optimized in all of the devices, we did not optimize this component in any of the cases. The VV delay was left as programmed without any alterations (0–5 ms).

**Exercise test**

In order to enable echo measurements immediately after exercise, this study had to be performed in a supine position on a bicycle ergometer. We began with 25 W and increased the workload by 22.5 W every 2 min until the individually determined submaximal exercise level was reached. The patients were asked to aim for an intensity of exercise, which they could maintain steadily for 3 min in order to have enough time during the exercise period to carry out the complex measurements. For this reason, the measurement of the components of the AV delay was taken under conditions of submaximal and not maximal exercise.

**Statistical analysis**

The data are expressed as mean ± standard deviation. Individual differences between resting and exercise conditions were statistically analysed by the Student’s t-test using SPSS 12.0 Base System and Professional Statistics for Windows® (SPSS, Inc., San Rafael, CA, USA). P < 0.05 was considered statistically significant.

**Ethics**

The study was conducted in accordance with Helsinki guidelines and was approved by the local ethics committee. Patients provided written informed consent prior to inclusion in the study.

**Results**

**Patients**

Twenty patients were included in our study, 6 women and 14 men with mean age of 64.04 ± 10.66 years. Table 2 shows the clinical characteristics of the patients. The mean interval between CRT implantation and inclusion in the study was 1.3 ± 2.7 years. Before CRT implantation 17 patients were NYHA III (85%) and 3 patients were NYHA IV (15%). At the beginning of the study, 19 patients were NYHA II (95%) and 1 patient was NYHA I (5%).

The following types of CRT–ICDs had been used: 6 Kronos LV-T (Biotronik® Inc; Berlin, Germany), 2 Tupos LV (Biotronik® Inc; Berlin, Germany), 4 Lumax 300 HF-T (Biotronik® Inc; Berlin, Germany), 3 Insync III Marquis (Medtronic® Inc; Minneapolis, USA), and 5 Insync Sentry (Medtronic® Inc; Minneapolis, USA).

**Exercise test**

All 20 patients fulfilled the criteria of the study protocol. Their resting rate was 73.4 ± 10.0 bpm. A submaximal exercise load of 50 and 75 W (71 ± 9 W) resulted in a significant rate increase (P = 0.000) between 7 and 43 bpm with a mean increase of 22.5 ± 9.6 bpm which correlates to an exercise heart rate of 95.8 ± 10.8 bpm. Seven of the patients reached a subjective submaximal exercise level at 75 W, 13 of them at 50 W.

**Components and total duration of the optimal AV delay**

In all 20 subjects, it was possible to measure the components of the optimal AV delay in rest as well as during exercise.

**Pacemaker-related interatrial conduction time**

Comparing exercise with rest, changes of IACT were not significant. The right-atrial sense event marker (Mₘ) and left-atrial deflection in the esophageal electrogram (LA) were left as programmed without any alterations (0–5 ms). The VV delay did not optimize this component in any of the cases. The VV delay was left as programmed without any alterations (0–5 ms).

**Table 2** Clinical characteristics of the patients

<table>
<thead>
<tr>
<th>Clinical characteristics of the patients</th>
<th>Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (mean ± SD)</td>
<td>64.04 ± 10.66</td>
</tr>
<tr>
<td>Male/female</td>
<td>14/6</td>
</tr>
<tr>
<td>Coronary artery disease, n (%)</td>
<td>7 (35%)</td>
</tr>
<tr>
<td>Dilated cardiomyopathy, n (%)</td>
<td>12 (60%)</td>
</tr>
<tr>
<td>Arterial hypertension</td>
<td>1 (5%)</td>
</tr>
<tr>
<td>Left-ventricular ejection fraction before CRT (mean ± SD)</td>
<td>19.15 ± 8.32</td>
</tr>
<tr>
<td>Left-ventricular ejection fraction under CRT (mean ± SD)</td>
<td>23.24 ± 7.64</td>
</tr>
<tr>
<td>Interval in years between testing und CRT–ICD implantation (mean ± SD)</td>
<td>1.3 ± 2.7</td>
</tr>
<tr>
<td>Intrinsic QRS duration in ms (mean ± SD)</td>
<td>152 ± 6.7</td>
</tr>
<tr>
<td>QRS duration under CRT in ms (mean ± SD)</td>
<td>129 ± 9.5</td>
</tr>
<tr>
<td>Medication</td>
<td></td>
</tr>
<tr>
<td>ACE inhibitors, n (%)</td>
<td>15 (75%)</td>
</tr>
<tr>
<td>ARB, n (%)</td>
<td>4 (20%)</td>
</tr>
<tr>
<td>Beta-blockers, n (%)</td>
<td>18 (90%)</td>
</tr>
<tr>
<td>Digitalis, n (%)</td>
<td>14 (70%)</td>
</tr>
<tr>
<td>Diuretics, n (%)</td>
<td>20 (100%)</td>
</tr>
<tr>
<td>Spironolactone, n (%)</td>
<td>17 (85%)</td>
</tr>
</tbody>
</table>

AR, angiotensin-receptor blockers; SD, standard deviation.
Left-atrial electromechanical action (LA-EAC\textsubscript{long}) and left-ventricular electromechanical latency period (SV-EAC\textsubscript{short})

The most significant exercise-related changes concerned the left-atrial electromechanical action (LA-EAC\textsubscript{long}). During exercise, this interval varied significantly by $-10.7 \pm 16.1$ ms ($P = 0.008$). In comparison, the left-ventricular latency period (SV-EAC\textsubscript{short}) varied only by $-1.3 \pm 15.1$ ms ($P = 0.705$). The difference between these intervals represents the electromechanical component of the optimal AV delay.

Calculation of the optimal AV delay

We succeeded in defining the optimal AV delays for each patient for rest and submaximal exercise in both modes. The results are shown in Table 3.

As a consequence of the above results, compared to the resting values ($AVD_{optVDD} = 101.9 \pm 33.0$ ms and $AVD_{optDDD} = 172.4 \pm 48.3$ ms), the mean value of the optimal AV delay during submaximal exercise conditions in VDD mode was found to be $6.2 \pm 16.1$ ms shorter ($AVD_{optVDD} = 95.7 \pm 39.2$ ms, $P = 0.101$) without statistical significance. In DDD operation however, the optimal AV delay of $-8.8 \pm 14.5$ ms during submaximal exercise conditions was significantly shorter ($AVD_{optDDD} = 163.7 \pm 48.3$ ms, $P = 0.014$). In DDD pacing, $AVD_{opt}$ was shortened by 2.6 ms/10 bpm.

Discussion

In order to determine whether it makes sense to activate the rate adaptive AV delay in CRT, we determined the $AVD_{opt}$ in resting as well as exercise conditions. We are the first to implement the method described by Ismer et al.\textsuperscript{23} to calculate $AVD_{opt}$ in 20 patients with CRT. The findings regarding the electrical component of the optimal AV-interval were in accordance with formerly published data. These studies showed that the pacemaker related IACT in VDD and DDD pacing can be seen as a constant for each individual.\textsuperscript{16,21,23} Consequently, only the electromechanical component of the AV delay can be responsible for the heart rate dependent changes in $AVD_{opt}$. The electromechanical component of $AVD_{opt}$ is the difference between the two electromechanical intervals: left-atrial electromechanical action (LA-EAC\textsubscript{long}) and left-ventricular electromechanical latency period (SV-EAC\textsubscript{short}). We found a significant reduction of the interval LA-EAC\textsubscript{long} (left-atrial electromechanical action) by $-10.7 \pm 16.1$ ms ($P = 0.008$) during exercise, while the interval of SV-EAC\textsubscript{short} (left-ventricular electromechanical latency period) was only shortened by $-1.3 \pm 15.1$ ms ($P = 0.705$), which lacks statistical significance.

The only other study to date of LA-EAC\textsubscript{long} and SV-EAC\textsubscript{short} during exercise was performed in patients with AV block and a normal EF (55 ± 8%), and in accordance with our findings resulted in a non-significant shortening of SV-EAC\textsubscript{short} ($-2.6 \pm 21.8$ ms; $P = 0.604$).\textsuperscript{21} The same study found that the left-atrial electromechanical action (LA-EAC\textsubscript{long}) varied the most of all the various components of $AVD_{opt}$, although statistical significance was not achieved ($-8.4 \pm 32.7$ ms; $P = 0.277$).\textsuperscript{21} A review of all of the published findings on the electromechanical component of AV delay to date suggests that LA-EAC\textsubscript{long} is the component which is most influenced by the heart rate, especially in CRT patients as we show here for the first time. A definitive statement, however, cannot be given because some of the findings were not statistically significant.

Fittingly, Ismer et al.\textsuperscript{26} found that the electromechanical component of $AVD_{opt}$ correlated to the ejection fraction, which would explain the differences in the findings in patients with heart failure compared to patients with almost normal ejection fractions.

Consideration of the $AVD_{opt}$ reveals that the adaptations under exercise with DDD-stimulation, in contrast to VDD-pacing, are significant, with a change in $AVD_{opt}$ of $-8.8 \pm 14.5$ ms with an increase in heart rate of $22.5 \pm 9.6$ bpm which translates into a shortening by 2.6 ms per 10 bpm.

Two primary reasons for the lack of significant findings regarding the changes in $AVD_{opt}$ under VDD-stimulation could be: (i) the reduced accuracy in the measurements of the interatrial intervals, which depend on the exact triggering of the right-atrial deflection; (ii) the relatively small increases in heart rate because of the reduced capacity for exercise in our patient collective with heart failure which results, subsequently, in only small changes in the various measured parameters.

In healthy subjects, the duration of the natural atrioventricular conduction interval has been reported to decrease with exercise-increased heart rate.\textsuperscript{19,20} Daubert et al.\textsuperscript{19} observed a physiological shortening of the atrioventricular conduction delay in 10 healthy subjects with a decrease of $4 \pm 2.1$ ms per 10 bpm, at mean. Hence, our results are congruent with those of Daubert et al.\textsuperscript{19} regarding the shortening of $AVD_{opt}$ during exercise.

To our knowledge, the only other study of rate-adaptive AV delay in CRT patients is a study by Scharf et al.\textsuperscript{22} In

### Table 3 Results of resting and submaximal exercise measurements

<table>
<thead>
<tr>
<th></th>
<th>n = 20</th>
<th></th>
<th></th>
<th></th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>Exercise</td>
<td>Difference exercise-rest</td>
<td>P-value</td>
<td></td>
</tr>
<tr>
<td>Heart rate (bpm)</td>
<td>73.4 ± 10.0</td>
<td>95.8 ± 10.8</td>
<td>22.5 ± 9.6</td>
<td>0.000</td>
<td></td>
</tr>
<tr>
<td>$M_\text{LA}$ (ms)</td>
<td>41.6 ± 26.3</td>
<td>44.8 ± 24.1</td>
<td>3.1 ± 12.0</td>
<td>0.254</td>
<td></td>
</tr>
<tr>
<td>$S_\text{V-LA}$ (ms)</td>
<td>112.1 ± 27.4</td>
<td>112.7 ± 27.8</td>
<td>0.6 ± 4.9</td>
<td>0.594</td>
<td></td>
</tr>
<tr>
<td>LA-EAC\textsubscript{long} (ms)</td>
<td>180.6 ± 19.2</td>
<td>170.0 ± 22.1</td>
<td>-10.7 ± 16.1</td>
<td>0.008</td>
<td></td>
</tr>
<tr>
<td>SV-EAC\textsubscript{short} (ms)</td>
<td>120.3 ± 30.1</td>
<td>119.0 ± 30.6</td>
<td>-1.3 ± 15.1</td>
<td>0.705</td>
<td></td>
</tr>
<tr>
<td>LA-EAC\textsubscript{long} - SV-EAC\textsubscript{short} (ms)</td>
<td>60.3 ± 66.9</td>
<td>51.0 ± 63.4</td>
<td>9.4 ± 14.5</td>
<td>0.008</td>
<td></td>
</tr>
<tr>
<td>$AVD_{optVDD}$ (ms)</td>
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</table>
this study, the AV delay optimization was performed in 36 patients using the maximum LVOT-VTI determined by echocardiography. In the investigation, the authors came to the conclusion that the optimal AV delay increased during exercise (20 ms per 10 bpm). The authors suggest that the improvement of diastolic filling under the conditions of a longer AV-interval is a possible explanation for this unexpected result, although diastolic parameters were not examined in the study. Furthermore, it has been speculated that a progressive fusion at longer AVDs might increase ventricular resynchronization during exercise.

The results of Scharf et al. and our findings cannot be brought into congruence, but our results are in congruence with the other to date published findings regarding the rate-adaptive AV delay showing a shortening of optimal AV delay with increased heart rate. The shortcoming of our investigation is that significant changes were only found for AVDoptDDD, while no significant change in AVDoptVDD between rest and exercise was established. Consequently, the shortening of AVDopt by 2.6 ms/10 bpm can only be applied to DDD pacing.

According to our results, the recommendation by Scharf et al. to set the rate adaptive AV delay to +20 ms/10 bpm in CRT patients should be followed only with extreme caution.

 Until further investigations have been performed, for example ones using the myocardial performance index, to clarify whether AVDopt in CRT patients is shortened or lengthened under exercise conditions, the rate-adaptive AV delay parameter in CRT patients should be turned off.

Study limitations

In order to carry out the multiple complex measurements under controlled exercise conditions, it was necessary to carry out this study under submaximal, instead of maximal, exercise conditions. Maximal exercise conditions with greater increases in heart rate would potentially result in larger changes in AVDopt. A further limitation is that in the method we used, only an optimization of the diastolic function was considered, the systolic function was not examined.

Conclusions

Our study of CRT patients with an average heart rate increase of 22 bpm under submaximal exercise conditions did not reveal significant difference in pacemaker-related IACT and left-ventricular electromechanical latency period (SVtEACshort). However, the left-atrial electromechanical action (LA-EAClong) significantly varied by –10.7 ± 16.1 ms (P = 0.008) during exercise. Consequently, the whole electromechanical component of AVDopt (LA-EAClong – SVtEACshort) was shortened significantly (P = 0.008) between 60.3 ± 36.9 ms and 51.0 ± 33 ms.

In contrast to AVDoptVDD, AVDoptDDD revealed a significant change between rest and submaximal exercise conditions of –8.8 ± 14.5 ms (P = 0.014). Therefore, for DDD pacing, the resulting shortening of AVDopt is 2.6 ms/10 bpm.

Considering the studies performed to date, the rate-adaptive AV delay parameter should be deactivated in CRT.

Conflict of interest: none declared.

References


