Reduction of the pace polarization artefact for capture detection applications by a tri-phasic stimulation pulse


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KEYWORDS
capture detection; polarization; evoked response; pacing threshold

Abstract This study investigated the ability to minimize pace polarization artefacts (PPA) by adjusting the post-stimulus pulse duration of a tri-phasic stimulation pulse. Adjustment of the stimulation pulse was enabled by downloading special study software into an already implanted pacemaker. Tests were performed in a total of 296 atrial leads and 311 ventricular leads. Both chronic and acute leads were included in the study. Statistically significant differences were found in the initial PPA (without any adjustment of the stimulus pulse) between atrial and ventricular leads. In addition, significant differences were observed among various lead models with respect to changes over time in the initial ventricular PPA. Successful PPA reduction was defined as a reduction of the PPA below 0.5 mV for atrial leads and below 1 mV for ventricular leads. Results show a success rate for ventricular and atrial PPA reduction of 97.8% and 98.7%, respectively. Threshold tests showed that after reduction of the PPA loss of ventricular capture can be

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Introduction

Reliable detection of evoked responses is a prerequisite for any system, aimed at automatic measurement of pacing thresholds and adjustment of stimulation output parameters. The major factor impeding this detection is the polarization at the tissue–electrode interface following delivery of a stimulation pulse. This phenomenon is referred to as the pace polarization artefact (PPA). A system has been described earlier [1,2] in which a tri-phasic stimulation pulse was applied to reduce this artefact. This system was developed and effectively applied for measurement of the interval between the ventricular stimulus and the evoked T-wave as a rate responsive indicator [1,3].

Currently, application of this principle in a capture detection system is considered. For atrial applications minimal experience with this system is available. Moreover, compared with T-wave detection, detection of evoked atrial and ventricular complexes is expected to be more demanding with respect to the reduction of the polarization artefact, since the signals to be detected are less remote in time from the stimulus than the evoked T-wave. This study was conducted to investigate the ability of adjusting a tri-phasic stimulation pulse to reduce the atrial and ventricular pace polarization artefact to a level that allows reliable detection of the evoked response. In order to evaluate the effect of different electrodes and electrode maturation a wide range of lead models and chronic and acute leads were included in the study.

Materials and methods

Implanted devices

All patients included in this study were implanted with a Diamond II cardiac pacemaker (Vitatron Medical B.V., Dieren, The Netherlands). This device provides a tri-phasic stimulation pulse, consisting of a pre-charge pulse, the actual stimulus and a fast re-charge pulse. Special investigational software was developed to investigate minimization of the pace polarization artefact (PPA) by adjusting the post-stimulus pulse duration while the pre-charge duration remains constant. After downloading this software into the implanted device the post-stimulus pulse duration could be adjusted. Cardiac potential amplitudes were determined by adjusting the sensitivity of the device and monitoring detection of the signal within a pre-defined time window after the stimulus (0–10 ms for polarization artefacts and 0–40 ms for evoked responses). The pacemaker hardware allowed detection of PPA amplitudes and evoked response amplitudes up to 7.5 mV. If the PPA or the evoked response was still sensed at this maximum detection threshold the amplitude was documented as > 7.5 mV. Pacemaker telemetry markers were used to monitor the detection of evoked responses.

Patient inclusion and exclusion

All patients indicated for implantation of a Diamond II device (either a first implantation or a replacement) and atrial and ventricular pacing leads as listed in Table 1 were suitable candidates to be included in the study. Patients with chronic atrial and/or ventricular tachyarrhythmias and patients with renal failure were excluded from the study. All centres received ethical approval for their participation and all patients signed a Patient Informed Consent form prior to their inclusion.

Measurements

Follow-ups were scheduled at typically 2 days, 15 days and 90 days after pacemaker implantation. Patients with chronic leads were not included in the follow-up at 15 days after pacemaker implantation. During these follow-ups the investigational software was downloaded into the device. Atrial and ventricular PPA reduction tests were performed during all follow-ups including:

1. Assessment of the amplitude of the PPA at the standard post-stimulus pulse duration of 6 ms. For leads with an initial PPA amplitude less than 0.5 mV in the atrium or less than 1.0 mV in the ventricle, steps 2, 3 and 4 were omitted.
Although theoretically an initial PPA amplitude less than 0.5 mV or 1 mV would not exclude a negative PPA amplitude, based on current experience the initial and residual PPA amplitudes for these leads were both assumed to be positive and less than 0.5 mV for atrial leads or less than 1.0 mV for ventricular leads.

2. Reduction of the initially positive PPA amplitude by reducing the post-stimulus pulse duration by steps of 0.5 ms. Reduction of the post-stimulus pulse duration was discontinued when loss of detection of the PPA was observed at a sensitivity of 0.5 mV for atrial leads and 1.0 mV for ventricular leads.

3. Increasing the post-stimulus pulse duration by 0.5 ms in order to achieve a small positive residual polarization. Since loss of PPA detection at the 0.5 mV/1.0 mV sensitivity could imply a negative PPA amplitude this increase was required to assure a small positive residual PPA amplitude.

4. Assessment of the residual PPA amplitude.

5. Assessment of the evoked response amplitude.

6. Pulse duration threshold tests at three different output settings.

The investigational software required to adjust the post-stimulus pulse duration and to perform the measurements was automatically downloaded into the device prior to and removed upon completion of each measurement. As a result, full standard functionality was returned to the device at the end of the follow-up visit.

### Analysis

Reduction of the PPA amplitude was considered successful for an individual lead if the PPA amplitude could be reduced below 0.5 mV for an atrial lead and below 1.0 mV for a ventricular lead at all required follow-ups. In order to arrive at a conservative estimation for the success rate of the atrial and ventricular PPA reduction tests, these rates were calculated for the population of all leads for which the PPA reduction test was performed during all required follow-ups. In addition, the proportion of atrial and ventricular leads for which a residual PPA amplitude less than 5 mV could be achieved, either with or without the PPA reduction test, was calculated. Stepwise logistic regression was used to assess the effect of lead model in determining the success rate of the PPA reduction test and the proportion of leads with a residual PPA of less than 5 mV.

Data were analyzed by grouping PPA amplitudes into four categories: less than 2.5 mV, 2.5 mV up to (but not including) 5 mV, 5–7.5 mV, and greater than 7.5 mV.

### Results

#### General

Three hundred and thirty-eight patients were included of whom 198 were male. The average age at the time of pacemaker implant was 71 ± 10.6 years.

The distribution of leads included in the study is presented in Table 2. Lead impedances were within the typical range of the various lead types.

Atrial leads with an initial PPA amplitude less than 0.5 mV and ventricular leads with an initial PPA amplitude less than 1.0 mV were not included in the PPA reduction test. For a total number of 295 atrial leads and 253 ventricular leads (chronic and acute leads) the PPA reduction test was performed at all required follow-ups.

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Lead types to be included in the study</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Atrial lead types</strong></td>
<td>Electrode characteristics</td>
</tr>
<tr>
<td>Chronic leads (&gt; 5 years)</td>
<td>Various</td>
</tr>
<tr>
<td>Medtronic 5534/5034/5554/5054</td>
<td>High impedance, steroid eluting</td>
</tr>
<tr>
<td>Medtronic 4068/4568/5068/5568</td>
<td>Screw-in, steroid eluting</td>
</tr>
<tr>
<td>CPI 4269/4243/4244/4245</td>
<td>Screw-in, non-steroid and steroid eluting</td>
</tr>
<tr>
<td>Vitatron IRP 13B/IRP 13JB</td>
<td>Tined, non-steroid eluting</td>
</tr>
<tr>
<td><strong>Ventricular lead types</strong></td>
<td>Lead characteristics</td>
</tr>
<tr>
<td>Chronic leads (&gt; 5 years)</td>
<td>Various</td>
</tr>
<tr>
<td>Medtronic 5034/5054</td>
<td>High impedance, steroid eluting</td>
</tr>
<tr>
<td>Biotronik SX60BP</td>
<td>High impedance, non-steroid eluting</td>
</tr>
<tr>
<td>Intermedics 430-10</td>
<td>Tined, non-steroid eluting</td>
</tr>
<tr>
<td>Vitatron IRP 13B</td>
<td>Tined, non-steroid eluting</td>
</tr>
</tbody>
</table>

Note: any combination of atrial and ventricular leads was allowed.
Initial PPA amplitude measurements

Figs. 1 and 2 show the initial PPA amplitude at the standard post-stimulus pulse duration for all leads measured during the follow-ups (note that the 15-days post-implant follow-up was not performed for chronic leads).

From the results displayed in Figs. 1 and 2 it is obvious that the vast majority of atrial leads had initial PPA amplitudes of more than 7.5 mV, whereas lower initial PPA amplitudes are more common for ventricular leads. The Medtronic 5034 and 5054 leads and the Vitatron IRP 13B leads implanted in the atrium showed higher initial PPA amplitudes than their ventricular counterparts \((P < 0.0001)\).

Since 99% of all initial atrial PPA amplitudes measured during all three follow-ups were above 7.5 mV (the maximum amplitude that could be measured with the implanted device) no conclusions can be drawn as to any lead type or time dependency of the atrial initial PPA.

For ventricular leads the initial PPA amplitude decreased over time \((P < 0.001)\) and a significant difference among lead models was seen \((P < 0.001)\). The initial ventricular PPA amplitudes measured for the Biotronik SX60BP, the Intermedics 430-10 and the Vitatron IRP 13B leads decreased between the follow-ups 2 days and 90 days post-implant, whereas other leads did not show this decrease. Further analysis of the ventricular results showed significant differences between various lead types. As a typical example, Fig. 3 displays the initial PPA amplitude distribution for ventricular leads at 3 months after implantation. Similar results were obtained during the other follow-ups. The Intermedics 430-10 lead had the lowest mean initial PPA amplitude \((P < 0.001\) compared with other leads) followed by the Biotronik SX60BP lead.

Reduction of PPA amplitudes

For all ventricular leads showing an initial PPA amplitude above 1 mV \((N = 253)\) reduction of the PPA amplitude by adjustment of the post-stimulus pulse duration was successful on all but one occasion (this exception occurred during a single

Table 2  Distribution of lead types in the study

<table>
<thead>
<tr>
<th>Type of lead</th>
<th>Number</th>
<th>Type of lead</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic leads</td>
<td>27</td>
<td>Chronic leads</td>
<td>33</td>
</tr>
<tr>
<td>Medtronic 5534/5034/5554/5054</td>
<td>73</td>
<td>Medtronic 5034/5054</td>
<td>112</td>
</tr>
<tr>
<td>Medtronic 4068/4568/5068/5568</td>
<td>89</td>
<td>Biotronik SX60BP</td>
<td>58</td>
</tr>
<tr>
<td>CPI 4269/4243/4244/4245</td>
<td>32</td>
<td>Intermedics 430-10</td>
<td>42</td>
</tr>
<tr>
<td>Vitatron IRP 13B/IRP 13JB</td>
<td>75</td>
<td>Vitatron IRP 13B</td>
<td>66</td>
</tr>
<tr>
<td>Total</td>
<td>296</td>
<td>Total</td>
<td>311</td>
</tr>
</tbody>
</table>

Note that not all patients had both atrial and ventricular leads from the pre-defined lead types for this study, as listed in Table 1.

Figure 1  Initial PPA amplitude for atrial leads. This figure shows the distribution of atrial PPA amplitudes before the PPA reduction test at several follow-ups during the study. As indicated by this figure the majority of atrial PPA amplitudes were above 7.5 mV.

Figure 2  Initial PPA amplitude for ventricular leads. This figure shows the distribution of ventricular PPA amplitudes before the PPA reduction test at several follow-ups during the study. As indicated by this figure slightly less than 50% of the ventricular PPA amplitudes were above 7.5 mV at subsequent follow-ups.
follow-up only). As a result the 95% lower bound for the success rate of the ventricular PPA reduction test is 97.8% (the observed success rate is 99.6%).

In all atrial leads presenting with an initial PPA amplitude above 0.5 mV ($N = 295$) the PPA amplitude could successfully be reduced. This implies that the 95% lower confidence bound of the percentage of atrial leads allowing successful reduction of the PPA amplitude is 98.7% (while the observed success rate is 100%).

**Residual PPA amplitude**

In order to facilitate the differentiation between the residual PPA and the negative evoked response the residual PPA should have a positive polarity. Therefore, following loss of sensing of the PPA signal the post-stimulus pulse duration was increased by 0.5 ms to obtain a small positive residual PPA amplitude. In Figs. 4 and 5 this positive residual PPA amplitude is compared with the initial PPA amplitude before any polarization reduction was carried out. The results in these figures were obtained during the follow-up 90 days after pacemaker implantation. It should be noted that for eight atrial leads included in the PPA reduction test the residual PPA was not measured.

Expectedly, the 0.5 ms increase in the post-stimulus pulse duration results in an increase in the PPA amplitude. Fig. 5 shows that despite this increase, 99% of the ventricular leads has a residual PPA amplitude that is still less than 5 mV. Again significant differences among the ventricular lead models were present with the Intermedics 430-10 lead having the smallest residual PPA amplitude and the chronic leads the largest amplitude.

As shown in Fig. 4 the 0.5 ms increase in the post-stimulus pulse duration has a more substantial effect on atrial leads. Although PPA amplitude reduction is successful (PPA amplitude less than 0.5 mV) for all atrial leads, for 37% (107 out of 287 leads) of these leads a 0.5 ms increase of the post-stimulus pulse duration resulted in a PPA amplitude of more than 7.5 mV. Regarding the residual atrial PPA amplitude, significant differences among lead models were detected. Specifically, the Vitatron IRP 13B and IRP 13 JB leads had the lowest residual PPA amplitude, significantly below every other model.

![Graph showing the distribution of PPA amplitudes for various ventricular leads.](https://example.com/graph1)

**Figure 3** Initial PPA amplitude for various ventricular leads at the 90 days follow-up. This figure shows the distribution of the PPA amplitudes for various ventricular leads, measured at 90 days after implantation. The Intermedics 430-10 lead showed the lowest mean initial PPA amplitude.

![Graph showing the comparison of initial and residual PPA amplitudes.](https://example.com/graph2)

**Figure 4** Residual atrial PPA amplitude vs. initial PPA amplitude. In this figure the residual and initial atrial PPA amplitudes are compared as they were measured during the follow-up at 90 days after implantation. The figure shows the distribution of the initial PPA amplitudes ("Before") before the PPA reduction test and the distribution of the residual PPA amplitudes ("After") after the PPA reduction test and a single 0.5 ms increase in the post-stimulus pulse duration in order to assure a positive residual PPA amplitude. As shown by this figure the PPA reduction test has reduced the percentage of atrial leads with a PPA amplitude above 7.5 mV from about 75% to slightly less than 40%. 

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As with the initial PPA amplitude, a significant decrease over time in the residual PPA amplitude was observed among the ventricular leads. In this case, the Medtronic 5534 and 5034 leads, the Biotronik SX60BP leads and the Vitatron IRP 13B leads showed a lower residual PPA amplitude at the follow-up 90 days after implant.

**Table 3** shows the proportion of leads for which a residual PPA amplitude of less than 5 mV could be obtained at all required follow-ups, with or without the PPA reduction.

<table>
<thead>
<tr>
<th>Lead type</th>
<th>Total number of leads</th>
<th>Observed proportion</th>
<th>95% Lower confidence bound</th>
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<tr>
<td>Atrial leads</td>
<td>283</td>
<td>13.1%</td>
<td>9.4%</td>
</tr>
<tr>
<td>Ventricular</td>
<td>303</td>
<td>98.7%</td>
<td>96.7%</td>
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Figure 5  Residual ventricular PPA amplitude vs. initial PPA amplitude. In this figure the residual and initial ventricular PPA amplitudes are compared as they were measured during the follow-up at 90 days after implantation. The figure shows the distribution of the initial PPA amplitudes ('Before') before the PPA reduction test and the distribution of the residual PPA amplitudes ('After') after the PPA reduction test and a single 0.5 ms increase in the post-stimulus pulse duration in order to assure a positive residual PPA amplitude. As shown by this figure almost 60% of the ventricular leads had an initial PPA amplitude above 7.5 mV, whereas after the PPA reduction test more than 70% of the leads have a PPA amplitude less than 2.5 mV.

As with the initial PPA amplitude, a significant decrease over time in the residual PPA amplitude was observed among the ventricular leads. In this case, the Medtronic 5534 and 5034 leads, the Biotronik SX60BP leads and the Vitatron IRP 13B leads showed a lower residual PPA amplitude at the follow-up 90 days after implant.

**Table 3** shows the proportion of leads for which a residual PPA amplitude of less than 5 mV could be achieved, either with or without PPA reduction (so, including those leads for which a PPA reduction was not performed since the initial PPA was already sufficiently low). This table includes all leads for which the residual PPA was measured at all required follow-ups.

**Signal detection**

After achieving a small positive residual PPA the atrial and ventricular evoked response amplitudes were measured. Results of these measurements are displayed in **Fig. 6**, together with the amplitudes of the residual PPA. Furthermore, it should be noted that in almost all patients the atrial evoked response amplitude exceeded the measurement limitation of $-7.5$ mV. This means that the actual amplitudes could even be higher, while usually substantially lower amplitudes in the atrium are observed.

In order to find an explanation for the unexpectedly high evoked response amplitudes measured in the atrium additional measurements were performed in eight patients to investigate the influence of the stimulation output voltage on the measurement results. Results of these measurements are displayed in **Fig. 7**. Although the amplitude of the evoked response is expected to be independent of the stimulation output voltage, the results in **Fig. 7** show a reduction in the evoked response amplitude with decreasing output voltage amplitude. This suggests that the evoked response signal, observed during these measurements is actually a combination of the evoked response and an additional component that is influenced by the output voltage amplitude.

**Threshold tests**

A typical result of a ventricular threshold measurement is shown in **Fig. 8**. During this automated measurement the duration of the stimulation pulse is reduced each fourth stimulation and the evoked response detection is monitored by telemetry ECG markers.

**Figure 5**  Residual ventricular PPA amplitude vs. initial PPA amplitude. In this figure the residual and initial ventricular PPA amplitudes are compared as they were measured during the follow-up at 90 days after implantation. The figure shows the distribution of the initial PPA amplitudes ('Before') before the PPA reduction test and the distribution of the residual PPA amplitudes ('After') after the PPA reduction test and a single 0.5 ms increase in the post-stimulus pulse duration in order to assure a positive residual PPA amplitude. As shown by this figure almost 60% of the ventricular leads had an initial PPA amplitude above 7.5 mV, whereas after the PPA reduction test more than 70% of the leads have a PPA amplitude less than 2.5 mV.

**Table 3** Proportion of leads for which a residual PPA amplitude of less than 5 mV could be obtained at all required follow-ups, with or without the PPA reduction.

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**Figure 6**  Amplitudes of evoked responses and residual PPA measured at the 90 days follow-up. This figure shows the mean residual PPA amplitude and the mean evoked response amplitude in the atrium and the ventricle measured during the follow-up at 90 days after implantation. Obviously, the mean residual PPA amplitude is higher in the atrium than in the ventricle, while the mean atrial evoked response amplitude is unexpectedly high.
Given the fact that the first evoked response after a reduction in the pacing pulse duration is not correctly detected (which will be explained in the Discussion), Fig. 8 shows that reliable true positive and true negative detection of the ventricular evoked response is achieved. Similar ventricular results were obtained in all patients.

Although some atrial threshold measurements showed results similar to those obtained in the ventricle, a number of these measurements showed a different behaviour. A typical example of such a result is displayed in Fig. 9.

Discussion

The results of this study demonstrate that the atrial and ventricular pace polarization artefacts can be appropriately reduced by adjustment of the post-stimulus pulse duration, irrespective of the lead type and the amount of polarization that was initially observed. For all ventricular leads except one and for all atrial leads a gradual reduction in the post-stimulus pulse duration resulted in successful reduction of the PPA amplitude. The observation that a 0.5 ms increase in the post-stimulus pulse duration has a substantial effect on the residual PPA in the atrium suggests that at least for atrial PPA reduction the post-stimulus pulse duration should be adjusted in smaller steps in order to arrive at a small positive residual PPA.

In addition, the results of this study show a time dependency in the amplitude of the initial ventricular PPA (before PPA reduction by means of adjusting the post-stimulus pulse duration). Furthermore, significant differences were found in the initial ventricular PPA amplitude between several lead types. The observed differences cannot be explained by the electrode surface area.

For a good understanding of the evoked response measurements and the observations from the threshold measurements it is important to note that actual evoked response has a negative amplitude whereas the reduction of the polarization artefact is aimed at achieving a small positive residual PPA. Furthermore, as long as capture is achieved, the intracardiac signals measured during
this study are always a superposition of the actual PPA and the evoked response, even within the PPA measurement window of 10 ms from the stimulus. The almost immediate presence of an evoked response may explain the difference between initial PPA amplitudes in the atrium and the ventricle. Since the atrial evoked response amplitude is usually smaller than the ventricular evoked response amplitude the summation of the positive PPA and the negative evoked response yields a smaller observed amplitude in the ventricle, compared with the atrium.

To interpret the observations made during the threshold tests as they are displayed in Figs. 8 and 9 it should be noted that after a change in the stimulation pulse duration the tissue-electrode interface needs a certain time to reach a steady state in which the residual PPA amplitude is again appropriately low. This phenomenon is referred to as the dynamic response. Since the residual PPA and the evoked response are opposite in sign a large PPA amplitude in the non-steady state has a strong compensatory effect on the amplitude of the resulting signal which may lead to temporary

Figure 8 Ventricular threshold measurement. This figure shows an ECG recording of a ventricular threshold test. During this test the stimulation pulse duration is reduced every four beats; each arrow indicates the first beat after a pulse duration reduction. The upper trace shows the surface ECG during this test, the lower trace shows the telemetry ECG markers used to monitor capture detection. A single downward marker indicates a ventricular pace without the detection of an evoked response, a double downward marker indicates pacing followed by the detection of an evoked response. The recording shows a typical detection pattern observed during the reduction of the ventricular pacing pulse duration, characterized by a false negative capture detection immediately after the reduction and true positive capture detection in the three remaining beats of each cycle.

Figure 9 Atrial threshold measurement. For explanation see Fig. 8. Atrial pace and capture detection markers are upwards. This figure shows a true positive atrial capture detection immediately after a reduction in the atrial stimulation pulse duration. In the remaining three beats of each cycle a false negative capture detection is observed. After actual loss of capture (LOC) as observed in the surface ECG capture is still detected immediately after the reduction in the pacing pulse duration (false positive detection) and no capture is detected in the three remaining beats of the cycle (true negative detection). The combination of true and false detections as observed in this example suggests that the system is not detecting evoked responses but a polarization phenomenon that is related to the reduction of the pulse duration (dynamic response).
loss of detection of the evoked response as it is observed in the ventricular threshold tests. The atrial threshold tests show a rather inverse dynamic response, characterized by an evoked response detection immediately after a reduction in the pacing pulse duration and a false negative capture detection in the steady state situation. This suggests that the system is not detecting the actual atrial evoked response but an artefact signal caused by the response of the PPA to a reduction in the pacing pulse duration.

The observation that the atrial evoked response amplitude and the amplitude of the residual PPA amplitude increase with increasing stimulation output voltage (as displayed in Fig. 7) is essential for the understanding of the unexpected results obtained from atrial evoked response and threshold measurements. Since the stimulation output voltage cannot influence the evoked response amplitude, the only conclusion can be that the observed amplitude variation is caused by a contribution of the PPA to the observed signal. This conclusion calls for an explanation how the residual PPA, which is supposed to be a positive signal, can result in a more negative amplitude of the evoked response. The most likely explanation for this behaviour is the application of a second order filter in the device’s hardware, used for measurement of the intracardiac signals. Typically, the step-response of such a second order filter shows a negative overshoot. Since the amplitude of this negative overshoot increases with increasing stimulation output voltage the resulting signal composed of the evoked response and the PPA increases with increasing stimulation output voltage.

The overshoot due to the applied hardware also explains the inverted dynamic response observed during atrial threshold measurements. The non-steady state PPA immediately after a change in the stimulation output voltage causes a large negative overshoot. This contribution is responsible for a negative signal, detected during the threshold test and considered an evoked response. After reaching the steady state the PPA amplitude and the resulting negative overshoot have decreased and the resulting signal is no longer detected, causing a false negative evoked response detection.

In the ventricle the actual evoked response has a relatively large amplitude, compared with atrial evoked responses. As a result, the behaviour of the residual PPA and the resulting overshoot due to the input filter have a substantially smaller effect on the observed signal, considered the evoked response. This explains why the observations in the atrium did not show in the ventricular measurements. However, it cannot be excluded that the observed ventricular evoked response amplitude is affected by this phenomenon and that an inverted dynamic response may be observed in case of relatively small ventricular evoked responses.

The results of this study call for further investigation of the behaviour of the PPA and the observed evoked response amplitude in the atrium and the ventricle. In this continued investigation signal overshoot should be limited by applying a first order input filter and adjustments of the post-stimulus pulse duration should be made by smaller steps than the 0.5 ms used in the present study.

Conclusions

- Appropriate reduction of the ventricular PPA, required for detection of ventricular evoked responses could be achieved, independent of the lead type (including chronic leads) and the PPA initial amplitude. Reliable detection of ventricular capture was observed during ventricular threshold tests.
- Although results indicate that the atrial PPA can be successfully reduced, the reliable detection of atrial evoked responses was most likely hampered by the hardware characteristics of the applied pacing device. Further investigation of atrial capture detection with hardware optimized for this application should be carried out.

Acknowledgement

References