Atrial lead placement at the lower atrial septum: a potential strategy to reduce unnecessary right ventricular pacing

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Aims

Right ventricular (RV) pacing has been shown to be potentially detrimental to left ventricular function. In conventional dual-chamber pacing the position of the atrial lead could influence duration of the atrio-ventricular (AV) intervals, which is one of the variables that could be associated with an increased percentage of RV pacing. We wanted to see if lead placement at selected atrial septal sites could reduce AV intervals in patients receiving a dual-chamber pacemaker or implantable cardioverter defibrillator.

Methods and results

This was a prospective, acute, randomized single centre study that enrolled 57 patients. The atrial lead was placed in both the right atrial appendage (RAA) and the lower atrial septum (LAS) in each patient in random order. The P-wave durations, PR intervals, A sense–V sense (As–Vs), and A pace–V sense (Ap–Vs) intervals were measured at both atrial lead locations in each patient during device implant. The P-wave durations during sinus rhythm (SR), RAA pacing, and LAS pacing were 113±19, 144±27, and 84±12 ms (RAA vs. LAS, P<0.001), respectively. The PR intervals during SR, RAA pacing, and LAS pacing were 195±47, 230±61, and 167±44 ms (RAA vs. LAS, P<0.001), respectively. The As–Vs interval was 31% shorter in LAS pacing than in RAA pacing (134±44 ms vs. 194±52 ms, P<0.001). The Ap–Vs interval was 24% shorter during LAS pacing than during RAA pacing (195±45 ms vs. 257±63 ms, P<0.001).

Conclusion

When compared with RAA pacing, LAS pacing was associated with a shorter P wave duration, PR interval, As–Vs, and Ap–Vs intervals. The potential long-term impact of the strategy of pacing from LAS in reducing unnecessary RV pacing needs to be explored in future studies.

Keywords

Right ventricular pacing • Atrial lead location • Right atrial appendage pacing • Low interatrial septum pacing

Introduction

Several clinical studies have demonstrated that right ventricular (RV) pacing can be deleterious to left ventricular function1–4 and can lead to atrial and ventricular arrhythmias.5–9 Device manufacturers have developed algorithms to promote intrinsic atrio-ventricular (AV) conduction and avoid or minimize unnecessary RV pacing by prolonging the AV delays as much as possible. Although these programmable features can significantly reduce RV pacing,10–14 they may be insufficient to prevent unnecessary RV pacing and can lead to extremely long AV intervals. Furthermore, long AV intervals have been associated with negative cardiac hemodynamics and with the development of atrial and/or ventricular arrhythmias.15–17 Long AV intervals can also affect device parameters programmability and functionality such as limiting the maximum tracking rate or creating pacemaker-mediated tachycardia. Pacemakers with automatic mode switching to an atrial mode (DDDR → AAIR → DDDR) can be effective in minimizing RV pacing but clinical benefit and long-term results are unknown. A very long A sense–V sense (As–Vs) or A pace–V sense (Ap–Vs) interval could produce unfavourable haemodynamics similar to those in the pacemaker syndrome associated with retrograde conduction.18

Previous studies have demonstrated that pacing from the right atrial appendage (RAA) can increase the atrial activation time.
represented by a longer P wave duration, and can lengthen As–Vs and Ap–Vs intervals, leading to unnecessary and potentially deleterious RV pacing. In contrast, pacing from the lower atrial septum (LAS) has been shown to reduce atrial activation time, represented by a shorter P wave duration. However, no systematic comparison has been made to assess the effect on the AV intervals of the lead placement in the LAS location in relation to the standard RAA location in the same patient. Accordingly, this study was designed to compare the P wave duration, PR interval, As–Vs, and Ap–Vs intervals between the RAA and LAS lead positions. We hypothesized that lead placement at selected atrial septal sites can reduce As–Vs and Ap–Vs intervals in patients receiving a dual-chamber pacemaker or cardioverter defibrillator (ICD).

Methods
This was an acute, prospective, single centre, randomized study in which a total of 57 patients (58% male and 76 ± 11 years) were enrolled over a 16-month period with either a class I or II indication for implantation of a dual-chamber pacemaker or implantable ICD. Patients with permanent second-degree or third-degree AV block or previous open heart surgery (due to possible altered RAA anatomy) were excluded. Either St Jude Medical Tendril DX 1388T (Sylmar, CA, USA) or Medtronic Model 5076 (Minneapolis, MN, USA) bipolar, active fixation leads were used in all patients.

The RV lead was placed first at the septal aspect of the right ventricle outflow tract. Then, the atrial lead was placed in each patient at both the RAA and LAS in random order. Fluoroscopy was used in the frontal, right anterior-oblique (RAO) and left anterior-oblique (LAO) views to ensure a proper placement of the RA lead in either RAA or LAS. A properly placed lead in the RAA and LAS is shown in RAO 10° and LAO 45° projections, respectively, in Figure 1.

The detailed technique used for implanting the RA lead in LAS has been described in a previous study. In a 10° RAO view, the tip of the RA lead was oriented towards the RV cavity perpendicular to the tricuspid valve annulus (Figure 1C). Using a 45° LAO view, counterclockwise torque was applied to orient the RA lead towards the inferior septal area (Figure 1D). A negative P wave morphology in the inferior leads (II, III, and aVF) was used to confirm the position in LAS.

The As–Vs and Ap–Vs intervals for three consecutive cardiac cycles were collected with the RA lead placed in RAA and LAS in each patient. The P wave duration and PR intervals in lead II for

Figure 1 Fluoroscopic images of the atrial and ventricular leads. The right atrial appendage lead is shown in right anterior-oblique 10° projection (A) and left anterior-oblique 45° projection (B), where the right atrial appendage lead is directed superior and anterior. The lower atrial septum lead is shown in right anterior-oblique 10° projection (C) and left anterior-oblique 45° projection (D), where the lower atrial septum lead is directed at 90° angles at the inter atrial septum.
three consecutive cardiac cycles were collected during sinus rhythm (SR) and during pacing (5–10 bpm above the sinus rate) in both the RAA and LAS locations in each patient using a Prucka electrophysiology recording system (Prucka Cradiolab 4.0 Inc., Houston, TX, USA) at recording speeds of 50 and 100 m/s. The average of three consecutive cardiac cycles was used for each measurement.

All data are represented as mean ± SD. The differences between the RAA and LAS locations were calculated for each parameter and compared with the paired t-test analysis. The differences were considered statistically significant when \( P < 0.05 \).

### Results

Demographic data and patient clinical characteristics of the study population are summarized in Table 1. Forty-three patients underwent dual-chamber pacemaker implantation and 14 patients received a dual-chamber ICD. The RA lead was successfully placed in both sites, RAA and LAS, in all patients. The mean procedure times for placing the RA lead at the RAA and LAS locations were 2.5 ± 3 and 4.7 ± 4 min, respectively \( (P < 0.05) \). Far-field R wave oversensing requiring reprogramming of the sensitivity setting was not present while the lead was placed in the RAA location and was present in three patients (5%) in the LAS location. No complications were observed while RA lead was placed in both the RAA and LAS locations.

The P wave duration during SR was 113 ± 19 ms. During pacing from the RAA, the P wave duration increased by 27%, as compared with SR P-wave, to 144 ± 27 ms \( (P < 0.001) \). During pacing from LAS, the P wave duration decreased by 26% and 42% to 84 ± 12 ms as compared with SR P-wave and RAA-paced P wave \( (LAS \text{ vs. } SR, P < 0.001 \text{ and } LAS \text{ vs. } RAA, P < 0.001) \).

Representative PR intervals on 12-lead electrocardiogram (ECG) recording during RAA and LAS pacing and sinus rhythm are shown in Figure 2. The intrinsic PR interval was 195 ± 47 ms.

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**Table 1** Demographics and clinical characteristics of the study population \( (n = 57) \)

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<tbody>
<tr>
<td>Age (year)</td>
<td>76 ± 11</td>
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<tr>
<td>Gender (M/F)</td>
<td>33/24</td>
</tr>
<tr>
<td>Indication for device implantation</td>
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<tr>
<td>PPM=sick sinus syndrome (%)</td>
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<tr>
<td>ICD=LVEF &lt;35% SCD-HeFT criteria (%)</td>
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<td>ICD=LVEF 40% and positive EPS (%)</td>
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<tr>
<td>Paroxysmal atrial fibrillation (%)</td>
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</tr>
<tr>
<td>Medication at implant</td>
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<tr>
<td>Beta-blocker (%)</td>
<td>33</td>
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<tr>
<td>ACE inhibitor (%)</td>
<td>30</td>
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<tr>
<td>Diuretics (%)</td>
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<td>5</td>
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<td>Flecainide (%)</td>
<td>4</td>
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<td>Propafenone (%)</td>
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ACE, angiotensin-converting enzyme; EPS, electro-physiologic study; ICD, implantable cardioverter defibrillator; LVEF, left ventricle ejection fraction; PPM, primary pacemaker; SCD-HeFT, Sudden Cardiac Death in Heart Failure Trial.

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**Figure 2** (A) Twelve-lead surface electrocardiogram recordings showing intrinsic PR interval, (B) prolongation of the PR interval by 71 ms in lead II while pacing from right atrial appendage (RAA), and (C) shortening of PR interval by 42 ms in lead II while pacing from lower atrial septum (LAS).
The PR intervals while pacing from the RAA and LAS locations were 230 ± 61 and 167 ± 44 ms (P < 0.001), representing an 18% increase and a 27% decrease as compared with the intrinsic PR interval, respectively.

Representative As–Vs and Ap–Vs intervals during RAA and LAS pacing are shown in Figure 3. It displays atrial sensing latency in relation to the surface ECG tracing when the lead is positioned in LAS. The As–Vs interval when the lead was positioned at the LAS was 31% shorter when compared with the RAA location (134 ± 44 vs. 194 ± 52 ms, P < 0.001) (Figure 4). Similarly, pacing from LAS produced a 24% shorter Ap–Vs interval than when pacing from RAA (195 ± 45 vs. 257 ± 63 ms, P < 0.001) (Figure 4).

**Discussion**

This acute study demonstrated that pacing from the LAS location produced significantly shorter As–Vs and Ap–Vs intervals compared with RAA pacing. In addition, pacing from LAS resulted in a shorter P wave duration and PR interval as compared with RAA pacing. These findings could have important implications in reducing unnecessary RV pacing and its potential detrimental effect in cardiac synchrony. Current device features and available algorithms to reduce undesirable RV pacing are mainly based on the extension of the AV intervals. Although they reduce the occurrence of unnecessary RV pacing, the resulting AV interval could be excessively long, predisposing the patient to other consequences such as a limitation in programming the upper tracking rate, retrograde activation of the atria with the potential for pacemaker-induced tachycardia, pacemaker syndrome, inappropriate mode switches, worsening of mitral regurgitation, and pacing at the vulnerable period of the T wave, among others. In the case of devices with the managed ventricular-paced feature, the long pauses associated with this method can favour the occurrence of atrial or ventricular arrhythmias.25–27 Therefore, avoiding RV pacing solely based on the extension of the AV interval may not be the optimal strategy. Finding alternative sites for the placement of the atrial lead that can avoid the problems linked to the standard locations such as the RAA and that potentially improve device functionality and programmability is most desired.

Our study showed that pacing from RAA resulted in a longer atrial electrical activation time and prolonged P wave duration as compared with LAS pacing. The effect in the duration of atrial activation time along with the presence of atrial-paced latency accounted for the increased paced P wave duration, PR interval,
and Ap–Vs interval noticed while pacing from RAA. It is well known that an increase in the duration of the atrial electrical activation is strongly related to the predisposition of the development of atrial arrhythmias. Pacing from RAA increases the disparity of atrial activation and repolarization, which provides a substrate that favors reentry and the occurrence of atrial fibrillation.\(^{18,29}\)

The opposite seems to occur with atrial septal pacing.\(^{30–32}\) Pacing from LAS, however, not only reduces the interatrial conduction time, but it also seems to promote a faster AV conduction as reflected in a shorter PR interval and shorter As–Vs interval. The faster AV conduction time noticed while pacing from LAS could be a contributory mechanism to favor the detection of intrinsic ventricular activity before the pacemaker’s sensed and paced AV delay timeout, thus inhibiting the delivery of the ventricular stimuli, and consequently reducing or preventing unnecessary RV pacing. Costeas et al.\(^{31}\) demonstrated that pacing from the high interatrial septum in comparison with RAA pacing significantly reduced RV pacing percentage (22.2%) at 3-month follow-up, even though the As–Vs and Ap–Vs intervals between two groups were not significantly different over the study duration.

In relation to the detection of the intrinsic atrial activity, when the lead is located in RAA, the atrial activation will be sensed early due to the proximity of the tip of the lead to the sinus node area. This early sensing will start the AV timing of the device. Subsequently, the activation has to reach the AV junction and then the right ventricle to meet the tip of the ventricular lead to complete the interval. The resulting delay in detecting the intrinsic ventricular activity would lead to an increased As–Vs interval potentially increasing RV pacing percentage. With the lead positioned in LAS, the detection of the intrinsic atrial activity will be significantly later (increased atrial sensing latency). The late lead positioned in LAS, the detection of the intrinsic atrial activity results in a delay in the initiation of the AV timing of the device, which increases the chances for a relatively early detection of the ventricular activation that leads to a significantly shorter As–Vs interval.

In this study, we demonstrated that the placement of the atrial lead in LAS was readily and safely accomplished in all the patients in a relatively short period of time using the technique described by Acosta et al.\(^{20}\) in which no special tools were required. Other possible benefits that can be attributed to atrial septal pacing are the potential for reduction of the incidence of myocardial perforation seen on lead insertion at standard sites\(^{33}\) as well as the potential for enhancement of the effectiveness of the current device algorithms intending to reduce unnecessary RV pacing percentage. The capacity of shortening the As–Vs and Ap–Vs intervals along with the possible antiarrhythmic effect that has been associated with atrial septal pacing makes it a promising preferred site for atrial lead placement in future clinical practice.

In conclusion, this study demonstrated that LAS pacing produced a significantly shorter P wave duration, PR interval, As–Vs, and Ap–Vs intervals when compared with RAA pacing. A reduction in the As–Vs and Ap–Vs intervals that promote AV conduction may decrease the potential for unnecessary RV pacing. In addition, lead insertion in the LAS could enhance the effectiveness of current algorithms intending to reduce RV pacing percentage. Preventing undesirable RV pacing could reduce pacemaker-induced left ventricular dysfunction and atrial arrhythmias as well as prolong longevity of the devices. Further prospective, randomized studies with long-term follow-ups to explore these potential beneficial effects are warranted.

References


