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Current of Injury Predicts Adequate Active Lead Fixation in Permanent Pacemaker/Defibrillation Leads

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OBJECTIVES The aim of this study was to determine whether current of injury can guide adequate placement of active-fixation pacing leads.

BACKGROUND Active-fixation leads cause injury to the myocardium at the time of fixation, manifested as a current of injury (COI) that may result in acute elevation of pacing thresholds. The relationship of COI to subsequent improvement in pacing thresholds is not clear.

METHODS Sixty-five patients undergoing active-fixation lead implantation were enrolled. Current of injury was characterized as the duration of the intracardiac electrogram (EGM) and the magnitude of ST-segment elevation. Pacing parameters were measured up to 10 min after fixation.

RESULTS A total of 96 active-fixation leads were studied, and 76 leads had a current of injury. From baseline to the time of fixation, the duration of the intracardiac EGM in ventricular leads increased from 150 ± 31 ms to 200 ± 25 ms ($p < 0.001$), and the ST-segment increased from 1.5 ± 0.2 mV to 10.0 ± 2.0 mV ($p < 0.001$), with subsequent improvement in pacing thresholds from 1.5 ± 0.4 V to 0.8 ± 0.3 V ($p < 0.001$) at 10 min. Atrial leads with a current of injury had similar findings. Of the 20 leads without a COI, 5 dislodged acutely and 15 had high pacing thresholds at 10 min, requiring repositioning.

CONCLUSIONS The development of a COI indicates that within 10 min of fixation, pacing threshold will return to an acceptable range even if the initial measurement is high. Conversely, without a COI, lead fixation is not adequate and the lead should be repositioned. (J Am Coll Cardiol 2005;45:412–7) © 2005 by the American College of Cardiology Foundation

Permanent pacing and defibrillation leads may be anchored in the heart either passively using tines or by active fixation. Active-fixation leads have either a fixed or an extendable-retractable helix. These leads are initially traumatic to the tissue; therefore, adequate pacing thresholds may not be obtained immediately. The fixation of the lead into the myocardium causes injury to the tissue known as the current of injury (COI). The COI is recognized at the site of tissue injury as an increase in the duration of the intracardiac electrogram (EGM) and elevation of the ST-segment following the QRS signal (1,2). The presence of a COI in retrospective studies has been correlated with adequate lead placement (2,3). Current of injury may be recorded during placement of passive leads as well, presumably because of focally damaged cell membranes consequent to the trauma of electrode pressure against the endocardium (4). With active-fixation leads, the ST-segment elevation is expected to be even more pronounced. The duration of the intracardiac EGM has been reported to increase with lead implantation as well; however, the absolute change from baseline that reflects a significant difference has not been defined in atrial or ventricular leads. In addition, the time course to resolution of COI and its relationship to pacing thresholds have not been studied prospectively, nor has acute dislodgement in the absence of COI been evaluated previously. We investigated the time course of COI with active fixation of atrial and ventricular pacing/defibrillation leads and the improvement in pacing thresholds as an indicator of adequate lead placement. Additionally, the magnitude of COI of active atrial leads that reflects adequate lead placement was evaluated.

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METHODS

Subjects. Subject selection was made from a consecutive sample of patients who were undergoing active-fixation atrial pacing lead and/or ventricular pacing or defibrillator lead implantation for standard clinical indications.

Technique. The patients were brought to the electrophysiology laboratory in a fasting, nonsedated state after having given informed consent. Using standard techniques, access to the subclavian vein was obtained and a sheath was advanced to the right atrium. Either an atrial pacing or a ventricular pacing or defibrillator lead was advanced through the sheath into the right atrium. The peel-away sheath was removed. Using straight and curved stylets under fluoroscopic guidance, ventricular leads were advanced first into the right ventricular outflow tract, and then pulled back into the right ventricular apex. For the atrial leads, the lead was positioned in the right atrial appendage. Once proper location was identified fluoroscopically, the lead was tested before fixation. The leads had an active collar; therefore, measurements could be made before lead fixation. The stylet was in place for recording of the COI before lead fixation, then withdrawn for all subsequent measurements from the time of fixation (0 min) to the 10-min recording time.

The atrial and ventricular pacing/defibrillation leads were all bipolar, extendable-retractable types with an electrically

Abbreviations and Acronyms
COI = current of injury
EGM = electrogram

active helix. The two active-fixation lead models were Medtronic Inc. (Minneapolis, Minnesota) models 6947 (ventricular defibrillation lead) and 5076 (atrial and ventricular pacing leads).

Measurements. A Medtronic pacing system analyzer (model 2090) was used for all acquisition of data. The bipolar intracardiac EGM was recorded at 200 mm/s before fixation, at the time of fixation (0 min), and then at 2, 5, and 10 min after fixation. The specific settings for the pacing system analyzer used included the following features: a sampling rate of 9,862 samples per second per channel, a resolution/accuracy of ±0.2 mV or 10% (whichever is greater), and a bandpass filter setting (atrial and ventricular) of 0.5 to 250 Hz. Once the lead was fixed into the wall (right atrial appendage or the right ventricular apex) as confirmed by fluoroscopy, other pacing parameters were obtained in addition to the intracardiac EGMs. Pacing threshold, impedance, slew rate, current, and R- or P-wave sensing were all measured simultaneously at the time of lead fixation and at 2, 5, and 10 min. The lead was not repositioned until all measurements were made out to 10 min. If the pacing thresholds were not adequate, ≥1.5 mV, at 10 min, the lead was repositioned and the above measurements were obtained in the same manner.

Current of injury was characterized as the duration of the intracardiac EGM and the magnitude of ST-segment elevation. The intracardiac EGM duration, in ms, was hand-measured from the initial deflection to the termination of the last deflection that crossed the baseline (Fig. 1A). The ST-segment elevation, in mV, was measured from baseline (the isoelectric portion before the intracardiac EGM) to the highest voltage following the intrinsic EGM. Therefore, the amplitude of the signal (R- or P-wave amplitude) did not affect the ST-segment measurements.

Statistical analysis. The sample size calculation was based on ST-segment elevation of at least 2.0 mV of the ventricular intracardiac EGM, which has been described to be a significant change from baseline in prior studies (4). The sample size calculation is estimated on the basis of the assumption that the study would have an 80% power (two-sided alpha, 0.025) to detect a difference in ST-segment elevation. Baseline characteristics and results are presented as means and standard deviations for continuous variables. Repeated measures analysis of variance with contrasts was used to test for statistical significance between measurements made before fixation versus 0 and 10 min for the COI, and for 0 versus 10 min for all other continuous variable measurements. A p value of <0.01 was considered significant.

RESULTS

Sixty-five consecutive patients undergoing active-fixation lead implantation were enrolled. There were 18 women and 47 men, with an average age of 60 ± 10 years. Mean

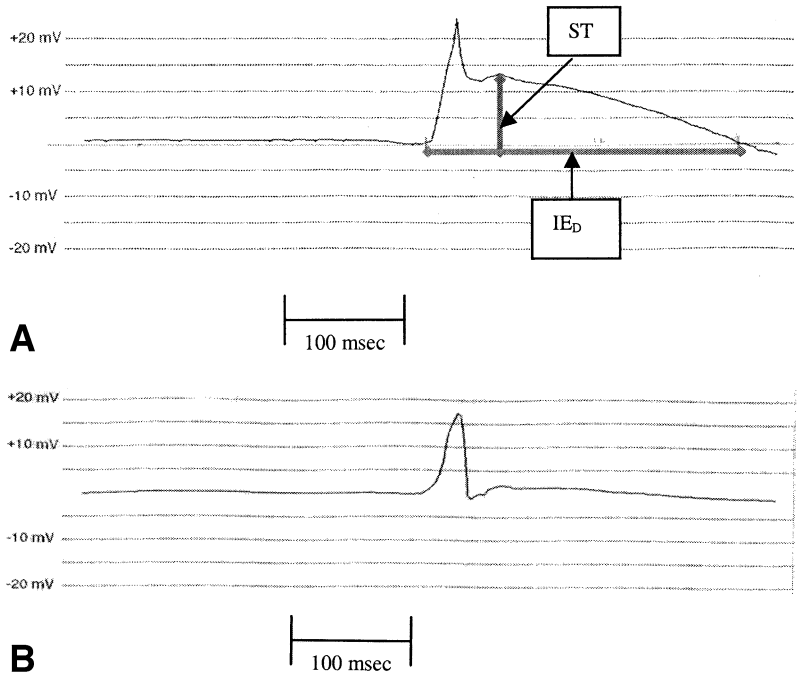

Figure 1. Measurement of current of injury using the intracardiac electrogram as recorded by a Medtronic model 2290 pacing system analyzer. **(A)** Current of injury was hand-measured both as the increase in the duration of the intracardiac electrogram (IE_D) in ms and the magnitude of ST-segment elevation in mV compared to baseline at 200 mm/s. This bipolar ventricular electrogram was recorded at the time of lead fixation with development of a current of injury. **(B)** Ten minutes after lead fixation, with the electrogram having returned to baseline.

Table 1. Acute Measurements of Ventricular Pacing or Defibrillation Leads With Current of Injury

(n = 50)	Before Fixation	0 min	2 min	5 min	10 min	p Value
COI: intracardiac EGM duration (ms)	150 ± 31	200 ± 25	175 ± 25	150 ± 25	138 ± 25	< 0.001
COI: ST-segment elevation (mV)	1.5 ± 0.2	10 ± 2.0	6.6 ± 1.2	3.4 ± 0.8	0.8 ± 0.2	< 0.001
Pacing threshold (V)		1.5 ± 0.4	1.3 ± 0.3	1 ± 0.2	0.8 ± 0.3	< 0.001
Impedance (ohms)		950 ± 24	910 ± 29	880 ± 26	850 ± 25	< 0.001
Current (mA)		1.9 ± 0.2	1.7 ± 0.2	1.5 ± 0.2	1.1 ± 0.1	< 0.001
R-wave sensing (mV)		18 ± 2	18 ± 2	18 ± 2	19 ± 2	NS
Slew rate (V)		2.7 ± 0.3	2.7 ± 0.3	2.8 ± 0.2	2.8 ± 0.2	NS

COI = current of injury; EGM = electrogram; NS = not significant.

ejection fraction was 26 ± 14%. A total of 96 active-fixation leads, all of which had extendable-retractable helices, were studied (65 ventricular and 31 atrial leads). There were 50 Medtronic model 6947 defibrillator leads and 46 Medtronic model 5076 pacing leads implanted.

Ventricular leads. There were a total of 65 ventricular leads; 50 had a COI at the time of fixation, manifested as an increase in the duration of the intracardiac EGM and ST-segment elevation compared to baseline (Fig. 1). The duration of the intracardiac EGM increased from 150 ± 31 ms to 200 ± 25 ms ($p < 0.001$) at the time of fixation and decreased to 138 ± 25 ms ($p < 0.001$) at 10 min (Table 1). The ST-segment elevation increased from 1.5 ± 0.2 mV to 10.0 ± 2.0 mV ($p < 0.001$) at the time of fixation and decreased to 0.8 ± 0.2 mV ($p < 0.001$) at 10 min. The ventricular pacing thresholds decreased significantly from the time of lead fixation to the 10-min recording time, from 1.5 ± 0.4 V to 0.8 ± 0.3 V ($p < 0.001$). Of the 50 ventricular leads that produced a clear COI, 38 had pacing thresholds <1.5 V, 8 had pacing thresholds between 1.5 and 2.0 V, and 4 had pacing thresholds >2.0 V at the time of lead fixation. All leads that had a COI at the time of fixation had adequate pacing thresholds at the end of the 10-min recording period, defined as <1.5 V. Furthermore, current and impedance significantly decreased from the time of fixation to 10 min. There was no significant difference in R-wave sensing or slew rate from the time of fixation to the 10-min recording time. However, consistent desirable R-wave amplitudes ranging between 16 and 21 mV were observed in leads that developed COI after active lead fixation.

The remaining 15 ventricular leads did not have a COI at the time of lead fixation; 10 of these had high pacing thresholds even at the 10-min recording time, ranging between 2.0 and 3.0 V (Table 2). These leads were repositioned after 10 min of fixation and all subsequently developed a COI and adequate pacing thresholds at 10 min. Five other leads did not have a COI at the time of fixation and subsequently dislodged acutely within the 10-min recording period. Pacing thresholds just before dislodgment ranged from 2.4 to 2.7 V (Fig. 2). These leads were also repositioned and subsequently had a COI and adequate pacing thresholds.

Atrial leads. We implanted 31 atrial active-fixation leads; twenty-six leads had a COI at the time of lead fixation (Fig. 3). The duration of the intracardiac EGM increased from 125 ± 25 ms to 175 ± 13 ms ($p < 0.001$) at the time of fixation and decreased to 125 ± 25 ms ($p < 0.001$) at 10 min (Table 3). The ST-segment elevation increased from 0.8 ± 0.1 mV to 2.0 ± 0.3 mV ($p < 0.001$) at the time of fixation and decreased to 0.5 ± 0.1 mV ($p < 0.001$) at 10 min. The atrial pacing thresholds decreased significantly from the time of fixation to 10 min, from 1.5 ± 0.4 V to 0.8 ± 0.2 V ($p < 0.001$). Of the 26 atrial leads that developed an obvious COI, 9 had pacing thresholds <1.5 V and 17 had pacing thresholds between 1.5 and 2.1 V at the time of lead fixation. All leads with a COI at the time of fixation had pacing thresholds <1.5 V at the end of the 10-min recording period. There was also a significant decrease in current and impedance after fixation at the 10-min recording time. No significant difference was noted in P-wave sensing or slew rate in the first 10 min after fixation. Similar to the ventricular leads, consistent desirable P-wave amplitudes ranging between 3.0 and 3.5 mV were observed in leads that developed COI after active lead fixation.

The other five atrial leads did not develop a COI at the time of fixation, and their pacing thresholds remained high, ranging between 2.3 and 3.0 V after the 10-min waiting period (Table 4). These leads were all subsequently repositioned, and a COI with pacing thresholds <1.5 V was obtained.

Table 2. Acute Measurements of Ventricular Pacing or Defibrillation Leads Without Current of Injury

(n = 15)	Before Fixation	0 min	2 min	5 min	10 min	p Value
COI: intracardiac EGM duration (ms)	163 ± 25	150 ± 25	150 ± 25	163 ± 25	163 ± 13	NS
COI: ST-segment elevation (mV)	1.1 ± 0.2	1.1 ± 0.3	1.1 ± 0.2	1.2 ± 0.2	1.0 ± 0.2	NS
Pacing threshold (V)		2.5 ± 0.3	2.4 ± 0.3	2.4 ± 0.3	2.5 ± 0.3	NS
Impedance (ohms)		1,000 ± 33	960 ± 27	930 ± 37	900 ± 32	< 0.001
Current (mA)		1.5 ± 0.2	1.5 ± 0.2	1.5 ± 0.2	1.3 ± 0.2	NS
R-wave sensing (mV)		14 ± 2	13 ± 3	13 ± 2	13 ± 2	NS
Slew rate (V)		2.7 ± 0.3	2.7 ± 0.3	2.7 ± 0.3	2.8 ± 0.3	NS

Abbreviations as in Table 1.

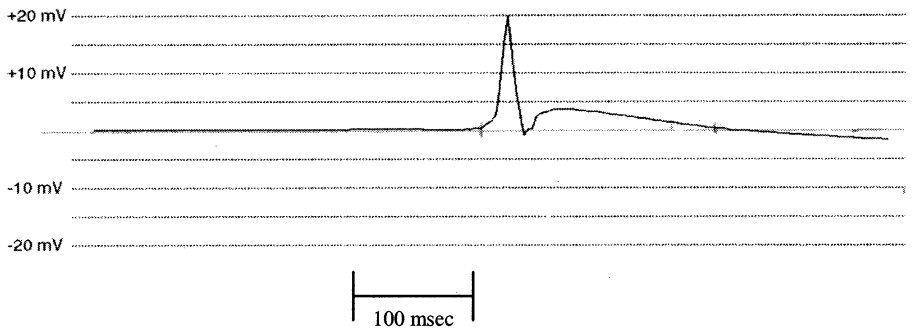

Figure 2. Ventricular lead without a current of injury at the time of fixation. Lead dislodgement occurred after eight minutes from the time of fixation.

DISCUSSION

The major finding of this study is that the presence of an adequate COI at the time of an active-fixation pacing or defibrillation lead placement correlates with adequate lead fixation. We define an adequate COI as an increase in the duration of the atrial or ventricular intracardiac EGM by 50 ms and an increase in ST-segment elevation of at least 5 mV for ventricular leads and 1 mV for atrial leads compared to baseline. Alternatively, an adequate COI could be described as an ST-segment elevation of at least 25% of the intrinsic atrial or the ventricular EGM amplitude. Furthermore, if a COI develops after fixation as seen in our study, but pacing thresholds are transiently elevated (>1.5 V), they will improve within the first 10 min after fixation. One should wait for the improvement in pacing thresholds rather than attempt to reposition the lead immediately. Thus, the intracardiac EGM at the time of lead fixation is a useful tool for guiding lead placement and repositioning.

The presence of a COI is a visual observation of the change in the intracardiac EGM from baseline to the time of fixation. The change in morphology can be described as an increase in duration of the intracardiac EGM and a marked ST-segment elevation from baseline. The morphology of the intracardiac EGM differs between the atrium and the ventricle, mostly because the ventricular EGM has a higher amplitude signal and, subsequently, a greater magnitude of ST-segment elevation in comparison to the atrium. The morphology of the atrial intracardiac signal has been described in the past (5); however, the acute changes related to active-fixation leads and COI have not been studied until now.

As mentioned earlier, an ST-segment elevation of >2.0

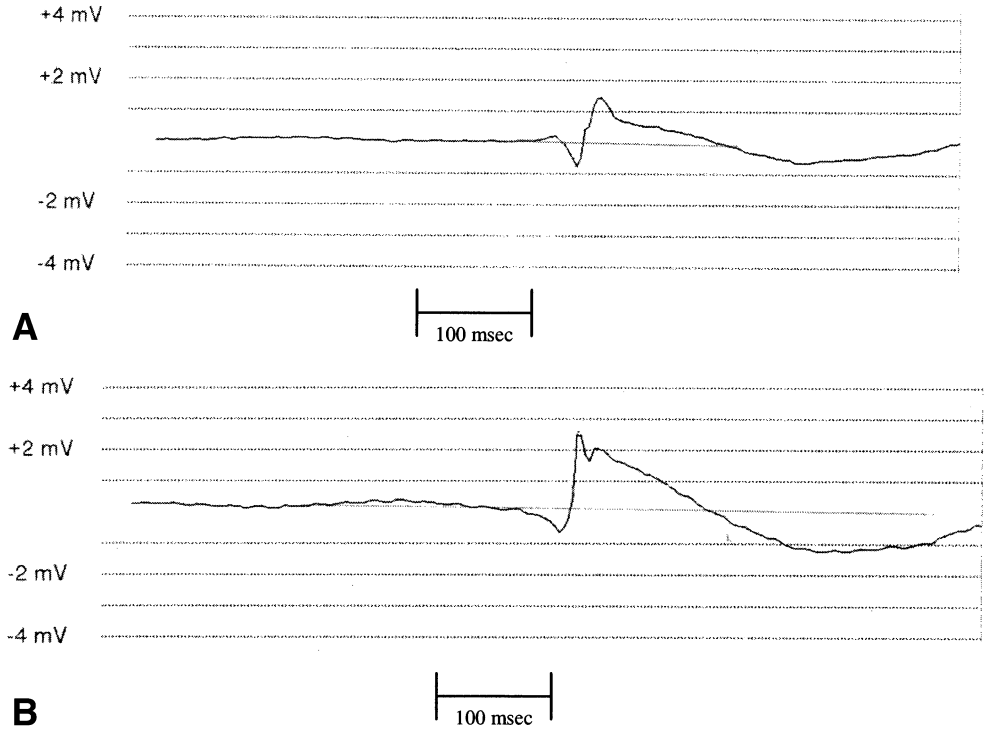

Figure 3. Atrial electrogram recorded at baseline **(A)** and at the time of fixation **(B)**, illustrating a current of injury with prolongation of the intracardiac electrogram and ST-segment elevation.

Table 3. Acute Measurements of Atrial Pacing Leads With Current of Injury

(n = 26)	Before Fixation	0 min	2 min	5 min	10 min	p Value
COI: intracardiac EGM duration (ms)	125 ± 25	175 ± 13	163 ± 13	150 ± 13	125 ± 25	< 0.001
COI: ST-segment elevation (mV)	0.8 ± 0.1	2 ± 0.3	1.4 ± 0.4	1.0 ± 0.3	0.5 ± 0.1	< 0.001
Pacing threshold (V)		1.5 ± 0.4	1.3 ± 0.3	1 ± 0.3	0.8 ± 0.2	< 0.001
Impedance (ohms)		900 ± 21	860 ± 25	830 ± 20	800 ± 25	< 0.001
Current (mA)		1.9 ± 0.2	1.7 ± 0.2	1.5 ± 0.2	1.1 ± 0.1	< 0.001
P-wave sensing (mV)		3.0 ± 0.3	3.0 ± 0.2	3.2 ± 0.2	3.2 ± 0.2	NS
Slew rate (V)		1.0 ± 0.3	1.0 ± 0.3	1.0 ± 0.3	0.9 ± 0.3	NS

Abbreviations as in Table 1.

mV has been considered evidence of an adequate COI in ventricular passive pacing leads (4). However, the magnitude of ST-segment elevation that could constitute a significant COI for atrial leads has not been previously reported. The atrial leads that developed a COI had ST-segment elevation of at least 1 mV, and those that did not have a COI had minimal ST-segment elevation, ≤0.2 mV. On the basis of the results of this study, we define an adequate COI in atrial leads as an increase in ST-segment ≥1 mV and ≥5 mV in ventricular leads. Furthermore, the increase in duration of the intracardiac EGM from baseline has not been defined in both atrial and ventricular active-fixation leads; we noted that an increase of 50 ms from baseline represented a significant change. Although we have presented measurements that are representative of an adequate COI, it should be noted that COI is readily visualized when present without the need for specific measurements.

Other pacing parameters also were measured simultaneously and, as expected, the impedance and the current both decreased significantly over the initial 10-min waiting period after fixation. The slew rate, P-wave, and R-wave sensing did not change significantly during the recording time. Slew rate has been described to decline significantly by about 40% once the lead enters the chronic phase, but this takes up to 4 weeks to occur (6). Therefore, we did not expect an acute decline in slew rate within the first 10 min. Similarly, P- and R-wave sensing also were not significantly different from the time of fixation to 10 min, as it is still in the acute phase.

In the late 1970s, three centers retrospectively reviewed all ventricular lead malfunctions at one-year follow-up. Of the patients who had lead dislodgement as a complication, approximately 50% had demonstrated either no ST-segment elevation or one <2 mV at the time of implantation of passive-fixation leads (4). Therefore, documentation of a discrete COI was a valuable sign of adequate lead placement, and ST-segment elevation of at least 2 mV suggested a satisfactory electrode-tissue interface for passive-fixation leads. A more recent study concluded that atrial lead implantation with the presence of COI predicts improvement of pacing thresholds (7). Furthermore, fixation of active atrial and ventricular leads has been correlated with acute changes in both pacing thresholds and R- or P-wave amplitudes that may improve with time (8). Despite these findings, a prospective evaluation of the COI and its direct relationship to pacing thresholds has not previously been demonstrated in atrial or ventricular leads.

As mentioned earlier, two Medtronic lead models were used in our study. Both have electrically active helices and no differences were noted between COI or other measurements made. However, we cannot predict that similar observations would be valid with inactive helices.

The clinical implications of this study are that COI should be used routinely to assess adequacy of active-fixation lead placement for both atrial and ventricular pacing leads. If a COI is present at the time of fixation but pacing thresholds are not desirable, it is worthwhile to wait, and the threshold will decrease over the next 5 to 10 min. In fact, if an adequate COI is observed, one may consider proceeding with completion of the pacemaker implantation with connecting the leads to the generator and closing the pocket, with final testing of the lead at that time, if the pacing threshold has not already declined to <1.5 V. With this approach, we have had no problems with needing to reopen the pocket and reposition the lead. On the other hand, if we have no COI or minimal changes and a high pacing threshold, we reposition the lead immediately.

Table 4. Acute Measurements of Atrial Leads Without Current of Injury

(n = 5)	Before Fixation	0 min	2 min	5 min	10 min	p Value
COI: intracardiac EGM duration (ms)	125 ± 25	125 ± 13	125 ± 13	125 ± 13	125 ± 13	NS
COI: ST-segment elevation (mV)	0.8 ± 0.2	1.0 ± 0.2	1.0 ± 0.1	0.9 ± 0.1	0.9 ± 0.1	NS
Pacing threshold (V)		3.0 ± 0.4	2.8 ± 0.3	2.8 ± 0.3	2.7 ± 0.3	NS
Impedance (ohms)		800 ± 32	810 ± 26	810 ± 26	800 ± 36	NS
Current (mA)		1.5 ± 0.2	1.5 ± 0.2	1.5 ± 0.2	1.2 ± 0.2	NS
P-wave sensing (mV)		3.2 ± 0.2	3.0 ± 0.3	3.0 ± 0.1	2.8 ± 0.3	NS
Slew rate (V)		1.0 ± 0.4	1.0 ± 0.3	1.0 ± 0.2	0.9 ± 0.3	NS

Abbreviations as in Table 1.

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