Reasons for Loss of Cardiac Resynchronization Therapy Pacing
Insights From 32 844 Patients

Alan Cheng, MD; Sean R. Landman, MS; Robert W. Stadler, PhD

Background—The efficacy of cardiac resynchronization therapy (CRT) is associated with the amount of CRT pacing delivered. The specific causes of CRT pacing loss and their relative frequencies remain poorly defined.

Methods and Results—CRT patients who transmitted device data from 2006 to 2011 were screened for inclusion. Device diagnostics were analyzed using an automated algorithm to categorize CRT loss into 10 different causes. The algorithm was validated against manual adjudications using a portion of the entire cohort. There were 80 768 patients analyzed with a median time of 594 (interquartile range, 294–1003) days from implant to time of analysis. In this cohort, 40.7% of patients had <98% pacing, and 11.5% of patients had <90% pacing. For patients with <98% pacing, device diagnostics explained 55.8% of pacing loss: 30.6% atrial tachycardia/atrial fibrillation; 16.6% premature ventricular contractions; and 8.6% captured as episodes with at least 10 consecutive beats of CRT loss (ventricular sensing episodes). Inappropriately programmed sensed and paced atrioventricular (AV) intervals (SAV/PAV) accounted for 34.5% of all ventricular sensing episodes. As the severity of CRT loss increased, the contribution of atrial tachycardia/atrial fibrillation and SAV/PAV to the loss increased. Atrial tachycardia/atrial fibrillation accounted for >50% and premature ventricular contractions accounted for <10% of CRT loss in those with <90% CRT pacing.

Conclusions—CRT pacing <98% was observed in 40.7% of patients. Among those with suboptimal pacing, atrial tachycardia/atrial fibrillation was the most common reason for CRT pacing loss. Inappropriately programmed SAV/PAV intervals was the most common reason for episodes of sustained loss of CRT pacing. This information can help in defining more effective treatments to improve CRT delivery. (Circ Arrhythm Electrophysiol. 2012;5:884-888.)

Key Words: atrial fibrillation • cardiac resynchronization therapy • heart failure • pacing • ventricular arrhythmia

Cardiac resynchronization therapy (CRT) reduces morbidity and mortality in individuals with systolic heart failure and electric dyssynchrony.1,2 Unfortunately, ≈30% to 40% of CRT patients do not show clinical improvement.3 Several factors have been identified to explain this observation.4 More recently, attention has focused on the importance of maintaining a high degree of CRT pacing.5 A recent study of a large cohort showed the greatest benefit of CRT in patients receiving an excess of 98% CRT pacing.6 When loss of CRT pacing is seen, it often occurs as a result of intrinsic ventricular activation superseding biventricular resynchronization. The reasons for intrinsic ventricular activation and their relative prevalence are poorly understood and important to assess because some of them may be amenable to simple treatments that could improve CRT pacing delivery. In addition, developing an automated process with the capability of adjudicating the reasons for intrinsic ventricular activation could facilitate the application of treatments to reduce CRT pacing loss and perhaps improve CRT response. Using a large real-world cohort of individuals with CRT systems enrolled in a remote monitoring program, we leveraged information provided by the device diagnostics related to CRT pacing behavior in an effort to better understand the reasons for CRT pacing loss and their relative prevalence.

Clinical Perspective on p 888

Methods

Data Collection and Clinical Database
This was a retrospective, cross-sectional analysis using the Medtronic Discovery Link database.7 All patients with CRT pacing enabled that transmitted data before September 21, 2011, were screened for inclusion. The CRT devices with the latest pacing diagnostics (eg, Concerto, Concorde II, Consulta, Protecta, Protecta XT, and Syncra; Medtronic Inc, Minneapolis, MN) were included. Patients without an atrial lead were excluded. For each patient, data were collected through remote telemetry, with information directly transferred to the Medtronic CareLink network. The information was deidentified and entered in the Discovery Link database. The most recent data transmission that included at least 24 hours of monitoring was selected for analysis. Device diagnostics were processed by an automated algorithm to...
determine reasons for loss of CRT pacing. The CRT pacing percentage did not include CRT triggered by ventricular sensed events.

Description of Algorithm and Definitions for Reasons for Loss of CRT Pacing

The selected CRT devices have automated diagnostics designed to document CRT pacing delivery and inhibition. The diagnostics include (1) percentage of time with CRT pacing (%CRT) not including left ventricular (LV) pacing triggered by ventricular sensing; (2) percentage of CRT pacing during atrial tachycardia/atrial fibrillation (AT/AF); (3) hours in AT/AF; (4) average number of premature ventricular contractions (PVCs) and runs of 2 to 4 PVCs per hour; (5) number of occasions where CRT pacing is inhibited for at least 10 consecutive beats (stored as ventricular sensing episodes [VSEs]); and (6) total duration of VSEs. The device is designed to specifically capture VSEs because these episodes represent more sustained periods of CRT loss. See online-only Data Supplement Methods for complete details of the definition of these diagnostics. From these diagnostics, the relative contributions to the total loss of CRT pacing (ie, 100–%CRT) were estimated for each of the 3 broad categories: (1) AT/AF; (2) PVCs, and (3) VSEs. The percentage loss of CRT pacing that occurred during AT/AF was defined as the percentage of CRT pacing loss during AT/AF multiplied by the fraction of time that the patient was in AT/AF (ie, the hours in AT/AF since last session divided by the hours since last session). The total number of PVCs per hour was estimated by adding the number of single PVCs per hour and 3× the number of PVC runs per hour. (The device detects a PVC run if 2–4 sequential PVCs occur; hence, 3× the number of PVC runs is an estimate of the number of PVCs that occurred for each run.) To estimate the percentage of time without CRT pacing because of PVCs, the total number of PVCs per hour was divided by 3600 seconds per hour and multiplied by an estimated duration of 0.59 seconds per PVC (based on analysis of previously published data).³ The percentage of time without CRT pacing that was captured in VSEs was determined by dividing the total duration of VSEs by the hours since last session.

Because VSEs are a documentation of lost CRT pacing but not a specific reason for loss of CRT, the algorithm processed the 60 atrial or ventricular event markers stored with each VSE to determine the cause of each VSE (for algorithm details, see the online-only Data Supplement Methods). Table 1 lists the 9 categories of VSE classification. Note that one of the VSE classifications is AT/AF. These are a subset of the broader category of loss of CRT because of AT/AF; specifically, the broader category includes all CRT lost during AT/AF, whereas VSEs classified as AT/AF include episodes with at least 10 consecutive beats of inhibited CRT because of AT/AF. Thus, overall there were 10 different categories of CRT loss: the 9 categories are listed in Table 1 (with AT/AF considered in a broader context), and the 10th category is PVCs. The VSE classification portion of the algorithm was developed from a database of 418 VSE episodes from 65 patients, which were adjudicated manually according to the classifications in Table 1. Performance of automated VSE classification was determined based on a separate database of 1207 VSEs from 190 patients, which were adjudicated manually according to the classifications in Table 1. The algorithm provided classifications for 97.4% of the duration of VSEs, and 98.8% of the total classified duration agreed with manual adjudication.

Statistical Methods

Statistical comparisons between patient subpopulations were conducted via 2-sample t tests for numerical variables and via 2-proportion tests with normal approximation for categorical variables. P<0.05 was considered statistically significant. All statistical computations were performed using SAS version 9.2 statistical software (SAS Institute Inc, Cary, NC).

Results

There were 80768 patients who met the inclusion criteria for the database. The data transmissions occurred between October 7, 2006, and September 20, 2011. The patients were predominantly men (73.1%) with an average age of 72.3±11.6 years. The median follow-up duration per transmission was 76.0 (interquartile range, 28.1–93.9) days, and there was a broad distribution of durations between device implant and the selected transmission date for analysis (median, 594 [interquartile range, 294–1003] days). CRT-P devices accounted for 0.2% of the cohort. Rate-responsive pacing was enabled in 68.5% of patients, and pacing modes with atrial tracking (DDD/DDDR) were programmed in 92.5% of patients. An AT/AF burden of <1% was observed in 78.0% of the patients, and an AT/AF burden of >90% was observed in 14.7% of the patients. Figure 1 summarizes the %CRT pacing in the entire cohort: 40.7% of patients had <98% CRT pacing, and 11.5% of patients had <90% CRT pacing.

For the 32844 patients with <98% CRT pacing, the automated analysis of device diagnostics accounted for 55.8% of the total loss of CRT. As shown in Figure 2, 30.6% of lost CRT occurred during AT/AF, PVCs accounted for 16.6% of lost CRT, and 8.6% of lost CRT was captured in VSEs. Interestingly, the contribution of AT/AF, PVCs, and the combined reasons for VSEs to the loss of CRT varied according to the categorized percent of CRT pacing. AT/AF was a larger contributor to the loss of CRT in those patients with a greater reduction of CRT. In patients with 95% to 98% CRT, 19.2% of the loss of CRT occurred during AT/AF, whereas in

<table>
<thead>
<tr>
<th>Table 1. Reasons for Loss of CRT Pacing as Captured in Ventricular Sensed Episodes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Classification</strong></td>
</tr>
<tr>
<td>AT/AF</td>
</tr>
<tr>
<td>SAV/PAV</td>
</tr>
<tr>
<td>SVT/ST</td>
</tr>
<tr>
<td>Idioventricular/VT</td>
</tr>
<tr>
<td>Junctional rhythm</td>
</tr>
<tr>
<td>Atrial undersensing</td>
</tr>
<tr>
<td>PVC/PAC</td>
</tr>
<tr>
<td>Nontracking PAC</td>
</tr>
<tr>
<td>Other</td>
</tr>
</tbody>
</table>

CRT indicates cardiac resynchronization therapy; AT, atrial tachycardia; AF, atrial fibrillation; SAV, sensed atrioventricular interval; PAV, paced atrioventricular interval; SVT, supraventricular tachycardia; ST, sinus tachycardia; VT, ventricular tachycardia; PVC, premature ventricular contractions; PAC, premature atrial contractions; AV, atrioventricular.
patients with <90% CRT, 51.8% of the loss of CRT occurred during AT/AF. In contrast, PVCs were a smaller contributor to the loss of CRT in those patients with a greater reduction of CRT. PVCs contributed an estimated 20.9% of the loss of CRT in patients with 95% to 98% CRT pacing but only 9.5% of the loss of CRT in patients with <90% CRT pacing. This trend occurred despite an increase in the number of PVCs per hour from 45.0 for patients with 95% to 98% CRT to 103.3 for patients with <90% CRT. The proportion of loss of CRT that was captured in VSEs increased slightly from 7.2% for patients with 95% to 98% CRT to 12.2% for patients with <90% CRT.

Sustained Periods of CRT Loss (VSEs)

A total of 193,737 VSEs were obtained in patients with <98% CRT pacing. Figure 3 summarizes the duration-weighted automated classifications of the VSEs. Overall, intrinsic ventricular conduction because of long programmed values of sensed atrioventricular (SAV)/paced atrioventricular (PAV) interval accounted for the largest portion of VSE duration (34.5%). Looking over the categories of %CRT pacing, the contribution of long programmed values of SAV/PAV to the duration of VSEs was minimal for patients with 95% to 98% CRT (5.4%) but became dominant for patients with <90% CRT (45.6%). In contrast, the contribution of supraventricular tachycardia/sinus tachycardia to the duration of VSEs was dominant for patients with 95% to 98% CRT (26.9%) but became minimal for patients with <90% CRT (3.6%). The overall contribution of AT/AF to the duration of VSEs was 19.6%. The VSEs that were obtained during AT/AF were a subset of the overall loss of CRT during AT/AF; specifically, VSEs contain the subset that included at least 10 consecutive beats without CRT pacing. Idioventricular rhythm and ventricular tachycardia accounted for 9.9% of overall VSE duration but peaked at 19.1% for patients with 90% to 95% CRT pacing. Loss of CRT pacing because of a programmed nontracking mode of pacing (DDI/R or VVI/R) accounted for 7.8% of the duration of VSEs, almost all occurring in patients with <90% CRT. This latter category was included as a reason because it reflected a potential error of device programming, similar to programming a suboptimal upper tracking rate or SAV/PAV interval.

Given the predominant role of SAV/PAV in accounting for periods of at least 10 beats of CRT loss, we further evaluated whether the programmed SAV/PAV intervals and use of rate-adaptive atrioventricular (RAAV) pacing differed between patients who did and patients who did not have VSEs caused...
Table 2. Device Parameters for Patients With and Without VSEs That Were Caused by SAV/PAV Programming

<table>
<thead>
<tr>
<th></th>
<th>Patients With VSEs Caused by SAV/PAV</th>
<th>Patients Without VSEs Caused by SAV/PAV</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAV (nominal=100 ms)</td>
<td>138.6±50.1</td>
<td>109.3±23.4*</td>
</tr>
<tr>
<td>PAV (nominal=130 ms)</td>
<td>169.7±48.5</td>
<td>139.1±25.1*</td>
</tr>
<tr>
<td>Rate-adaptive AV on</td>
<td>84.0%</td>
<td>91.8%*</td>
</tr>
</tbody>
</table>

VSE indicates ventricular sensed episode; SAV, sensed atrioventricular interval; PAV, paced atrioventricular interval; AV, atrioventricular interval.

*P<0.00001 vs patients with VSEs caused by SAV/PAV.

by SAV/PAV (Table 2). Patients who had VSEs caused by SAV/PAV had substantially longer programmed SAV and PAV (P<0.00001) and were less likely to have RAAV programmed on (P<0.00001).

Discussion

By analyzing existing device-based diagnostics, our efforts to better understand the reasons for loss of CRT pacing resulted in 3 important findings. In a large cohort of real-world patients with CRT systems, 40.7% of patients exhibited <98% CRT pacing. Second, the specific reasons for the loss of CRT pacing varied depending on the overall percentage of pacing. Although AT/AF accounted for the largest portion of CRT loss in all patients with <98% CRT pacing, its contribution to CRT pacing loss diminished as the amount of CRT pacing approached 98%. In fact, PVCs played a larger role in the loss of CRT pacing as overall pacing percentages approached 98%. Last, the largest contributor to more sustained loss of CRT pacing (ie, VSEs containing at least 10 consecutive beats without CRT) was inappropriate programming of SAV/PAV intervals and lack of using RAAV. These findings help shed light on the most common causes for suboptimal CRT pacing as a function of the degree of lost CRT pacing. More importantly, these data could lead to corrective actions and possibly improved CRT response. To facilitate corrective actions, automated algorithms like the one used here could be applied to each data transmission, resulting in a report of the severity of CRT loss and the determined reasons for CRT loss. Currently, observations are made automatically only if CRT pacing is <90%. In addition, no further assistance is provided for interpreting CRT pacing diagnostics.

Efforts Aimed at Improving CRT Delivery

Appropriate corrective actions exist for certain causes of CRT loss, and our data suggest that the majority of CRT loss episodes can be readily corrected. AT/AF can be ablated or pharmacological rhythm or rate control may be effective for improving the CRT pacing percentages. PACing algorithms that minimally overdrive ventricular rate during AT/AF may also increase CRT pacing during AT/AF. Atrioventricular node ablation is a final option, and recent reports suggest that increasing CRT pacing through ablation of the atrioventricular node incrementally improves CRT outcomes, despite a lack of atrioventricular synchrony.9–11 CRT loss from sinus tachycardia can be resolved by increasing the upper tracking rate. CRT loss from atrial undersensing, ventricular oversensing, or nontracking pacing modes can be resolved by appropriate device programming or lead revisions. CRT loss from PVCs may also respond to ablation or antiarrhythmic medications.12–13 Last, CRT loss because of SAV/PAV can be readily resolved by modifying the programmed SAV/PAV intervals or perhaps application of RAAV. Based on our analysis, inappropriately programmed SAV/PAV intervals was the predominant reason for sustained episodes in which loss of CRT pacing was observed. It is interesting to speculate on the reasons for this. In many cases, adjustment of the programmed SAV/PAV intervals is guided by hemodynamic data derived from echo-based modalities or automated device-based systems. When changes are made, they more commonly result in lengthening of the SAV/PAV intervals from their nominal settings.14 Although these changes may be beneficial in a resting state, this may undermine the ability of CRT systems to pace in the setting of increased adrenergic tone. It remains unclear whether hemodynamic optimization at rest, done at the expense of potentially reducing overall CRT pacing percentages, justifies this practice in the future. As our data suggest, perhaps the use of applications such as RAAV may play a greater role in the future management of these patients.

Limitations

Several limitations affected our analysis. Because this was a database analysis from CareLink, we do not have extensive information about clinical patient demographics, such as heart failure status, nor what interventions were used in each patient. By analyzing a cohort of >80000 individuals, we believe that these results remain pertinent to the greater population of patients with these devices. Although safety margins for LV pacing and predominant use of daily automated LV capture management minimized loss of LV capture, it was not possible to account for loss of LV capture as a possible reason for loss of CRT pacing. Inherent limitations in the device diagnostic algorithms also prevented us from explaining every beat without CRT. Specifically, VSEs capture any occurrence of ≥10 consecutive beats of inhibited CRT pacing. However, episodes with <10 consecutive beats of inhibited CRT are not documented. Also, PVC detection in these devices requires short coupling intervals (R-R intervals that are <69% of prior intervals). PVCs with longer coupling intervals are not documented. In addition, a given episode of CRT loss was attributed to a single reason, and it is possible that some occasions of CRT loss occurred for >1 reason (ie, PVCs occurring during AT/AF or rapidly conducted AT/AF during a nontracking pacing mode were both categorized simply as AT/AF). Our manual review of 1207 VSEs suggests that this is rare. Finally, device diagnostics for CRT pacing describe the quantity of CRT pacing but do not address the quality of CRT pacing. Varying degrees of fusion and pseudofusion may provide variable benefit of CRT pacing but are all reported equally as CRT pacing by device diagnostics.

Conclusions

In a large cohort of CRT patients, 40.7% of patients had <98% pacing. AT/AF accounted for the largest portion of CRT loss, a portion that increased as the overall % CRT pacing decreased. The largest contributor to sustained periods without CRT was inappropriate programming of SAV/PAV intervals. Many
causes of CRT loss have corrective actions that are potentially easy to implement and could result in improved CRT response.

Sources of Funding
This work was funded by Medtronic, Inc. Dr Cheng did not receive compensation for work presented in this article.

Disclosures
Dr Cheng has received honorarium from Biotronik, Boston Scientific, Medtronic, and St. Jude Medical. Mr Landman was an employee of Medtronic, Inc, at the time this work was performed. Dr Stadler is an employee of Medtronic, Inc.

References

CLINICAL PERSPECTIVE
Cardiac resynchronization therapy (CRT) reduces morbidity and mortality in patients with systolic heart failure and electric dyssynchrony. Despite our best efforts at optimizing these systems, some patients fail to demonstrate ventricular remodeling or improvements in clinical symptoms. The percentage of beats that receive biventricular pacing has been inversely associated with heart failure hospitalizations and death. This observation has led to recent recommendations that every effort should be made to target 100% biventricular pacing. Although there may be several reasons why pacing is lost, no study has systematically analyzed the reasons and their relative frequencies. This study is the first to describe common reasons for loss of biventricular pacing in a large cohort. The authors developed a novel automated algorithm to determine reasons for loss of pacing by processing CRT device diagnostics and applied this algorithm to >32,000 real-world CRT patients. The results provide a greater understanding of the reasons for loss of CRT pacing and pave the way for development of novel strategies to improve patient response to CRT.
Reasons for Loss of Cardiac Resynchronization Therapy Pacing: Insights From 32 844 Patients
Alan Cheng, Sean R. Landman and Robert W. Stadler

Circ Arrhythm Electrophysiol. 2012;5:884-888; originally published online August 26, 2012; doi: 10.1161/CIRCEP.112.973776

Circulation: Arrhythmia and Electrophysiology is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2012 American Heart Association, Inc. All rights reserved.
Print ISSN: 1941-3149. Online ISSN: 1941-3084

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/5/5/884

Data Supplement (unedited) at:
http://circ.ahajournals.org/content/suppl/2012/08/26/CIRCEP.112.973776.DC1

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation: Arrhythmia and Electrophysiology can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation: Arrhythmia and Electrophysiology is online at:
http://circ.ahajournals.org//subscriptions/
SUPPLEMENTAL MATERIAL

MS ID# CIRCAE/2012/973776
Reasons for Loss of CRT Pacing:
Insights from 32,844 Patients

Loss of CRT during AT/AF
The device documented the duration of AT/AF and the percentage of CRT pacing during AT/AF. This allowed for calculation of the proportion of lost CRT pacing that occurred during AT/AF. Because reasons other than AT/AF can contribute to the loss of CRT during AT/AF (such as PVCs, runs of VT, and oversensing), the computed loss of CRT pacing during AT/AF is likely an overestimate of the loss of CRT that is caused by AT/AF.

Loss of CRT from PVCs
The device detected PVCs as R-R intervals that are ≤ 69% of the average of the preceding 4 R-R intervals (not including those R-R intervals that were classified as PVCs). The number of PVCs per hour and the number of runs of 2-4 PVCs per hour are stored by the device. The total number of PVCs per hour was estimated by adding the number of PVCs per hour and 3 times the number of PVC runs per hour. To estimate the percentage of time without CRT pacing because of PVCs, the total number of PVCs per hour was divided by 3600 seconds per hour and multiplied by an estimated duration of 0.59 seconds per PVC. The estimated 0.59 seconds for the average coupling interval of a PVC was determined by analyzing the R-R intervals of 420,638 PVCs from Holter recordings of 282 patients in the CARISMA study (8). The resulting loss of CRT pacing from PVCs is likely an underestimate because it does not count PVCs with R-R intervals > 69% of the average of the preceding 4 R-R intervals.

Loss of CRT from VSEs
The device documents episodes of 10 or more consecutive sensed ventricular events as Ventricular Sensing Episodes (VSE). The device stores the total duration of VSEs since last follow-up, as well as specific information for up to 8 VSEs from the follow-up period (the 7 most recent and the longest). Each stored VSE includes 60 atrial or ventricular event markers and additional diagnostics such as flags for device-detected AT/AF or VT/VF during the episode. The total duration of VSEs since last follow-up divided by the total hours of follow-up is an estimate of the loss of CRT pacing during VSEs.

Each VSE was then assigned by a classification algorithm to one of the 9 classifications in Table 1. Note that VSEs that were obtained during AT/AF were a subset of the overall loss of CRT during AT/AF. Specifically, the subset of loss of CRT during AT/AF that included at least 10 consecutive beats without CRT pacing is also recorded as VSEs. The VSE classification algorithm was developed on a database of 418 VSE episodes from 65 patients, which were adjudicated manually according to the classifications in Table 1. The first step in the classification algorithm was to determine the average ventricular and atrial rates during the VSE. A rate-branching approach was then applied as detailed below.

A rate > V Rate: If the atrial rate during the VSE was at least 15% greater than the ventricular rate, or if the VSE was detected while the rhythm was classified as AT/AF, the VSE was classified as AT/AF if: 1) The atrial rate during the VSE was at least 160 BPM, and 2) Since the last programmer session, the device must have had either less than 2% occurrence of far-field R-wave sensing or at least 0.01% of time spent in device-defined AT/AF. Otherwise, the processing continued as described below.
**V rate > A Rate:** If the ventricular rate during the VSE was at least 15% greater than the atrial rate, and the VSE had long A-A intervals (at least 1500ms) and did not have a sudden rate acceleration at the start of the VSE, then the VSE was classified as Atrial Undersensing if the device was not programmed to a tracking pacing mode and classified as Non-Tracking if the device was programmed to a non-tracking pacing mode. Otherwise, if the ventricular rate during the VSE was at least 15% greater than the atrial rate, the VSE was classified as Idioventricular/VT.

**V rate ≈ A Rate:** The remaining VSEs were subjected to the following classification steps:
1. If the device was programmed to a non-tracking mode, the VSE was classified as SAV/PAV if fast AV conduction was detected; otherwise, the VSE was classified as Non-Tracking.
2. If the atrial rate during the VSE was less than the programmed Upper Tracking Rate and the VSE contained a pattern of alternating ventricular sensing and atrial refractory sensing that was initiated by a premature beat, then the following sub-steps were applied to classify the VSE:
   a) If nearly coincident atrial and ventricular events were detected, the VSE was classified as Junctional Rhythm.
   b) If the VSE began with a sudden rate acceleration and the first short interval was V-V, then the VSE was classified as Idioventricular/VT.
   c) Otherwise, the VSE was classified as PVC/PAC.
3. If both the atrial and ventricular rates during the VSE were greater than or equal to the programmed Upper Tracking Rate, the VSE was classified as SVT/ST.
4. If fast AV conduction was detected, the VSE was classified as SAV/PAV.
5. If the VSE had long A-A intervals (at least 1500ms) and did not have a sudden rate acceleration at the start of the VSE, then the VSE was classified as Atrial Undersensing.
6. If nearly coincident atrial and ventricular events were detected, the VSE was classified as Junctional Rhythm.
7. All remaining VSEs were classified as Other. The algorithm is designed to provide an “other” classification in cases where it cannot provide a confident classification.

**VSE Classification Performance:** VSE classification performance was determined on a separate validation database of 1207 VSE episodes from 190 patients. These patients had a Medtronic Concerto, Concerto II, Consulta, Protecta, Protecta XT, or Syncra CRT device, had CRT pacing programmed on, and had an atrial lead implanted. The VSE episodes were reviewed manually to classify the true reason for loss of CRT pacing, according to the categories of Table 1. Of the 1207 episodes, the algorithm provided specific classifications for 1044 episodes (86.5%) and the remaining 163 episodes were classified as “Other”. Of the 1044 classified episodes, 990 (94.8%) were classified correctly by the algorithm. Clinically, correct classification of episodes with longer duration is more important than correct classification of episodes with minimal duration. Therefore, a second, duration-based, performance assessment involved weighting the classifications by VSE duration. For example, the classification of a 50 second VSE contributed 10 times that of a 5 second VSE toward the overall duration-weighted performance. The algorithm provided classifications for 97.4% of the duration of VSEs and 98.8% of the total classified duration was correct.