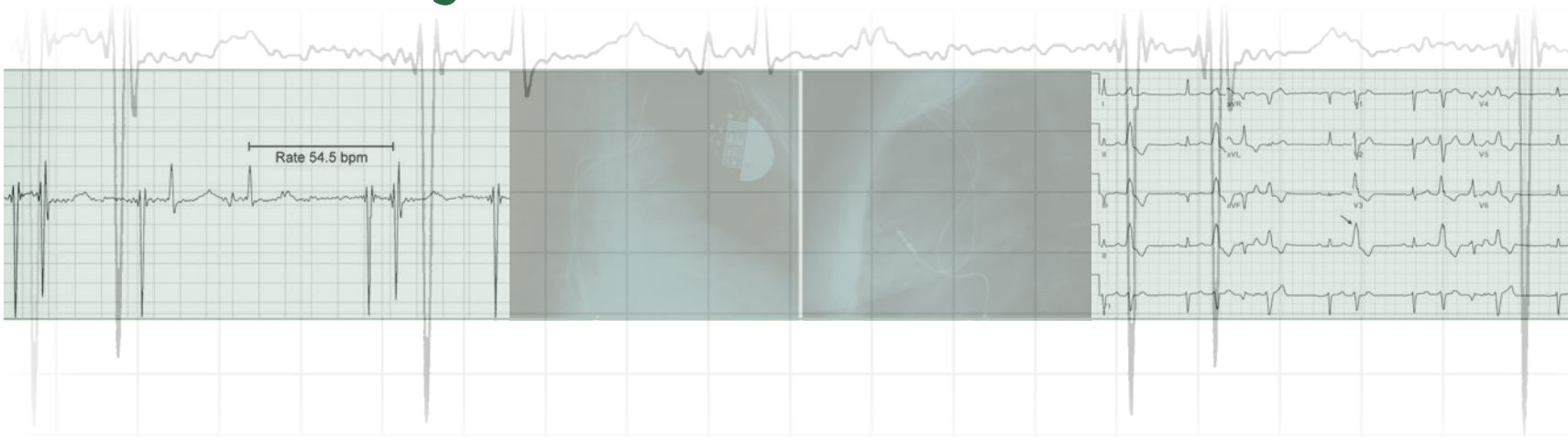
PACEMAKERS, ICDS, AND CARDIAC RESYNCHRONIZATION

A Case-Based Approach to Pacemakers, ICDs, and Cardiac Resynchronization

Volume 2



Advanced Questions for Examination Review and Clinical Practice

Edited by

Paul A. Friedman MD, FACC, FHRS | Melissa A. Rott RN

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Preface

The book that you hold in your hands, *A Case-Based Approach to Pacemakers, ICDs, and Cardiac Resynchronization: Advanced Questions for Examination Review and Clinical Practice*, is a compilation of our favorite teaching cases that were seen at or sent to Mayo Clinic. As our device practice has grown, we have found that one of the best ways to remain current and to educate incoming physicians and nurses is the review of interesting "unknown" clinical cases. Consequently, we established a morning conference in 2008 for the purpose of presenting and discussing interesting or uniquely educational cases. Since learners ranged from cardiology fellows who were new to the device practice to experienced nurses and physicians, group discussion brought out facets of interest at all levels. Cases for this book were selected based on clinical relevance and their usefulness for illustrating general principles, practical tips, or interesting findings in device practice. Occasionally, manufacturer-specific features are discussed, but always with a goal of advancing general concepts in device management.

The cases in this book are presented as a case history, an image when pertinent, and a multiple-choice question. The answer and a detailed explanation is presented on subsequent pages. We've adopted this format to encourage the reader to think through the differential diagnosis and approach the clinical problem based on the information presented. In light of the growing use of pacemakers, defibrillators, and resynchronization devices, we are confident that readers will find this practical means of self-assessment and education useful. Although the questions are designed in a multiple-choice format that may be particularly useful for self-assessment for test-takers, they are not formally validated board questions. This book is for any individual who sees patients with implantable devices, or who will be taking an examination related to device management.

How to Use This Book

The cases generally progress from simpler to more complex, understanding that there will be individual variation in what constitutes a difficult case.

There is no table of contents because the case numbers are clearly marked at the top of each page and we specifically did not want to include in the beginning of the book a listing of the "diagnosis" for each case and therefore limit the ability for the reader to approach the cases as unknowns.

For the reader interested in reviewing a specific type of case (such as "T-wave oversensing" or "inappropriate shock"), two resources are offered. An appendix is provided that identifies the major diagnostic

dilemma presented by each case, and the index will direct the reader to cases and discussions focusing on specific issues. However, we encourage readers to progress sequentially through cases as unknowns to maximize learning and interest.

This book is one of two volumes. The first volume includes introductory and intermediate cases. The second volume includes additional intermediate cases as well as advanced cases. There are more multipart cases in volume 2, to delve more deeply into important concepts.

In various electronic versions of this book, hypertext links and linked indices have been added to facilitate navigation. Also, a combined index that covers both volumes is available at www.cardiotextpublishing.com/titles/detail/9781935395447.

This text includes a collective wisdom of numerous physicians, nurses, technicians, educators, and practitioners. We are indebted to the entire Heart Rhythm services team at Mayo Clinic for identifying and discussing cases, and educating us with them. We have also benefitted greatly from friends and colleagues at other institutions who have kindly shared interesting cases with us, and permitted us to include them in this work. We are grateful for their generosity. If you come across an interesting case that you would like included in a future edition of this book, we would love to discuss it with you. E-mail addresses are listed below for that purpose. Please enjoy the cases! We look forward to your feedback and future contribution.

–Paul Friedman MD and David Hayes MD

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Abbreviations

A	atrial
AF	atrial fibrillation
APC	atrial premature contraction
AS	atrial sensed
ASD	atrial septal defect
AT	atrial tachycardia
ATP	antitachycardia pacing
AV	atrioventricular
AVNRT	atrioventricular nodal reentrant tachycardia
BBB	bundle branch block
CI	confidence interval
CRT	cardiac resynchronization therapy
CT	computed tomographic
ECG	electrocardiogram
EGM	electrogram
EMI	electromagnetic interference
EP	electrophysiological
FFRW	far-field R wave
ICD	implantable cardioverter-defibrillator
IV	intravenous
J	Joules
LAO	left anterior oblique
LBBB	left bundle branch block
LV	left ventricle; left ventricular
LVEF	left ventricular ejection fraction
MRI	magnetic resonance imaging
OR	odds ratio
PA	pulmonary artery
PAC	premature atrial contraction
PMT	pacemaker-mediated tachycardia
PVARB	postventricular atrial blanking period
PVARP	postventricular atrial refractory period
PVC	premature ventricular contraction
RAO	right anterior oblique
RBBB	right bundle branch block
RV	right ventricle; right ventricular
RVOT	right ventricular outflow tract
SVT	supraventricular tachycardia
TARP	total atrial refractory period
TENS	transcutaneous electrical nerve stimulation
V	ventricular
VA	ventriculoatrial
VF	ventricular fibrillation
VRR	ventricular rate regulation
VS	ventricular sensed
VSD	ventricular septal defect
VT	ventricular tachycardia

A Case-Based Approach to Pacemakers, ICDs, and Cardiac Resynchronization

Volume 2

Advanced Questions for Examination Review and Clinical Practice

Case 46

A 74-year-old female with a history of long QT syndrome and cardiac arrest underwent implantation of a dual-chamber defibrillator, the St. Jude Atlas +DR. The RV defibrillator lead is a Riata, which has an integrated bipolar lead. Two years later, the patient is seen in the device clinic and complains of receiving her first and only shock from the device 1 month prior. Portions of the episode are shown in Figure 46.1.

During interrogation, RV sensing and shock coil impedances are normal. The RV lead threshold is normal. The R wave today measures 8.2 mV compared to 9.0 mV at implant. Provocative maneuvers do not impact these values. The ventricular sensitivity setting was set as 0.3 mV. RV sensing parameters are as follows:

	Postsensed	**Postpaced**
Decay Delay	60 ms	Auto
Threshold Start	62.5%	Auto
Refractory Period	125 ms	250 ms

The patient's chest x-ray is normal. Her QT interval is 320 ms. Her potassium is 4.2 mmol/L.

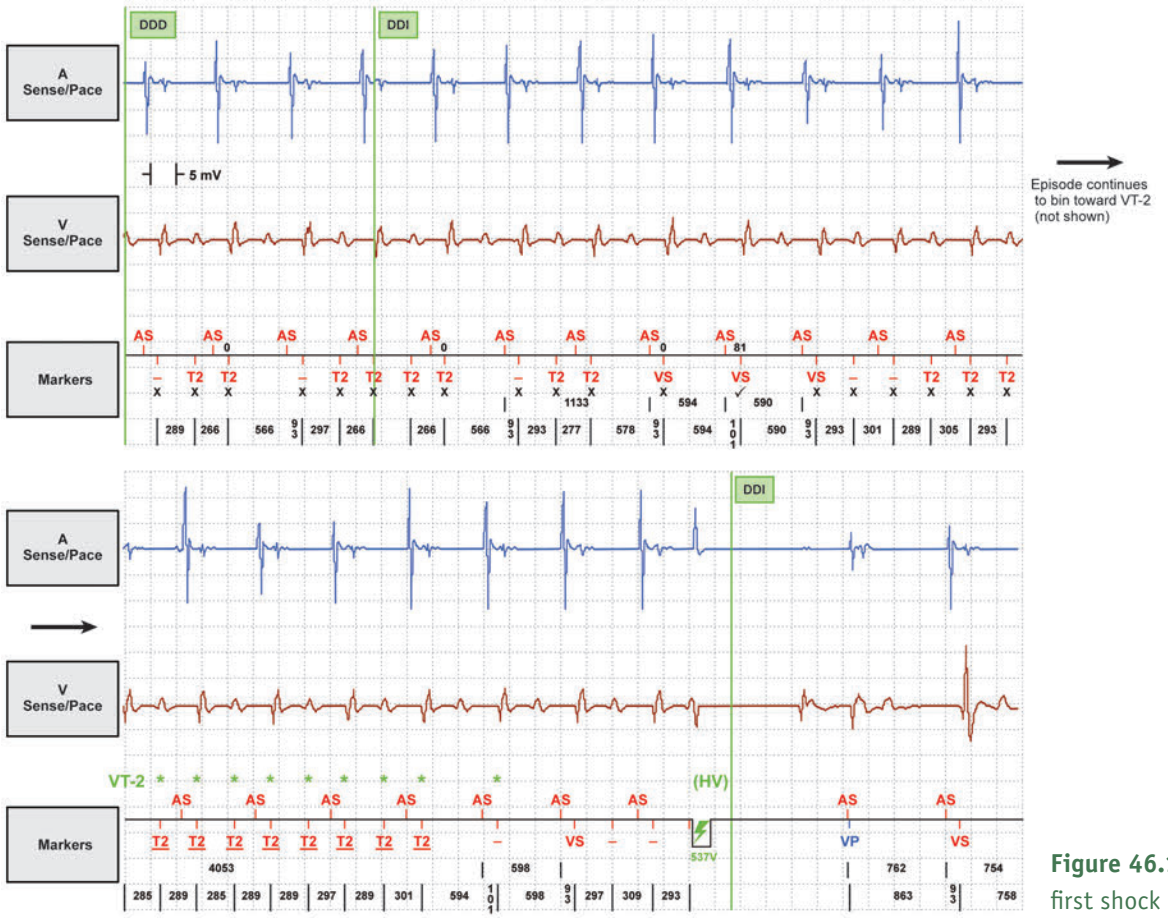

Figure 46.1 Portions of patient's first shock episode.

What would be the next most reasonable management step(s)?

1. Correct electrolytes and initiate the patient on an antiarrhythmic agent
2. Repeat defibrillation threshold testing and consider lead revision
3. Reduce the sensitivity setting to 0.1 mV
4. Program more aggressive antitachycardic pacing therapies

46

2. Repeat defibrillation threshold testing and consider lead revision

This question requires you to recognize T-wave oversensing. Of the choices provided, the most reasonable management option is to repeat defibrillation threshold testing and consider possible lead revision for this patient with T-wave oversensing. In this case, the patient has a history of cardiac arrest. Furthermore, the R-wave amplitude measurement has diminished without a clear etiology. Her QT measures normally at follow-up and is not markedly prolonged in Figure 46.1. Repeat defibrillation threshold testing with possible lead revision represents the most appropriate management step. The ventricular lead should be revised if the safety margin for sensing ventricular fibrillation is insufficient. If the defibrillation lead is replaced, a true bipolar lead may be preferred because T-wave oversensing may be more frequent with integrated-bipolar leads (Weretka S, Michaelsen J, Becker R, et al. Ventricular oversensing: a study of 101 patients implanted with dual chamber defibrillators and two different lead systems. *Pacing Clin Electrophysiol.* 2003;26:65-70).

An annotated version of the episode is shown in Figure 46.2. On the top panel, we see multiple events labeled T2 on the ventricular marker channel, signifying that the device is binning ventricular events that count toward the VT2, or the fast VT, zone. Referring to the ventricular sensing EGM, we see that the T2 labeled events align with both the native R wave and the T wave, resulting in short R-R cycles in the 280 to

300 ms range. Hand calculation of the R-R cycle length (25 mm/s paper speed) demonstrates that the cycle length is actually 590 ms, which is more consistent with sinus tachycardia. The small gray "X's" that align with the V or T2 markers are morphology template match attempts suggesting failure to match T waves *and* intrinsic ventricular EGM to the ventricular morphology template. The checks correspond to a template match. The dashes (-) reflect a cycle length that does not count toward binning. Ventricular sense events are seen for a brief period as well. The device continues to bin events towards the VT2 zone (not shown). In the lower panel, the marker channel shows VT2, meaning that the device has binned enough events toward the VT2 zone to confirm arrhythmia. The asterisks indicate charging. During that charging, the device reconfirms VT2 as denoted by the underlined T2 markers, and ultimately a 15-J shock is delivered.

T-wave oversensing has resulted in the inappropriate detection of VT. The amplitude of the R waves is 3.0 to 3.5 mV during this episode (from baseline to peak), which is markedly reduced from implant. This is probably the reason for the failed morphology match even for the intrinsic QRS complexes (Weretka et al. 2003). The T-wave amplitude measures 1.0 mV, 33% of the R-wave amplitude. Possible reasons for a dimunition in the R-wave amplitude include electrolyte changes, tip fibrosis, infarction, infiltration, or progressive cardiomyopathy in the ventricle, a loosened set screw, or microdislodgment and macrodislodgment of the lead.

46

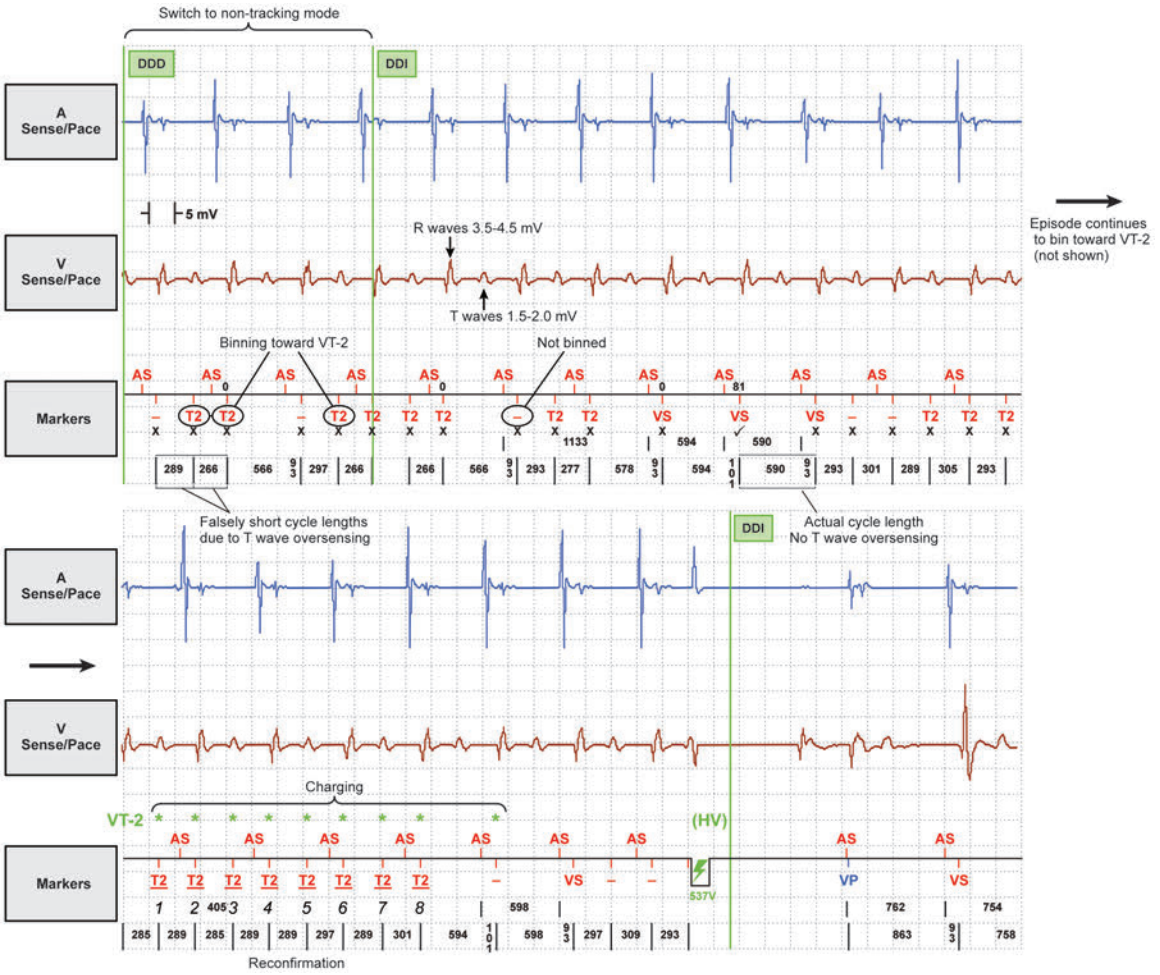

Figure 46.2 Annotated version of patient's first shock episode.

46

The patient wants to defer invasive evaluation or lead revision at this time. In general which is a reasonable set of reprogramming options in patients with this type of oversensing?

1. Lengthen the postsensing Decay Delay and increase the percentage threshold start
2. Increase the postpacing ventricular blanking period
3. Turn off SVT-VT morphology discrimination
4. All of the above

46

1. Lengthen the postsensing Decay Delay and increase the percentage threshold start

This case tests your ability to identify reprogramming options in this patient with an inappropriate shock due to T-wave oversensing.

It is helpful to think about the management of T-wave oversensing in three broad categories: postpacing, large R wave (>3 mV) in spontaneous rhythm, and small R wave (<3 mV) in spontaneous rhythm (Swerdlow CD, Friedman PA. Advanced ICD troubleshooting: part I. *Pacing clin Electrophysiol.* 2005;28(12):1322-46). Typically, the first scenario—oversensing of postpacing T waves—causes inappropriate inhibition of bradycardic pacing or delivery of antitachycardia pacing. It may be corrected by increasing the postpacing ventricular blanking period. In the second scenario in which R waves are greater than 3 mV with a large R/T ratio, reprogramming may be feasible. Some devices allow for adjusting the sensitivity threshold to a higher value. The third scenario of T-wave oversensing in the setting of low-amplitude R waves presents a more challenging situation. Options include:

- St. Jude ICDs provide a programmable Threshold Start, Decay Delay, and the postventricular refractory period designed to reduce oversensing of spontaneous T waves.
- Turning on SVT-VT morphology discrimination to "on," which may classify alternative EGMs associated with intrinsic QRS as sinus and potentially result in withholding therapy.
- If the RT and TR intervals differ sufficiently in the VT zone, the stability algorithm may be used to reject T-wave oversensing.
- Rarely, force ventricular pacing to alter the sequence of depolarization and reduce T-wave amplitude.
- Lead revision of the addition of a second pace/sense RV lead.

Management in this case was particularly difficult because the R-wave dimunition was transient. In general, R-wave amplitudes lower than 5 to 7 mV carry the risk of underdetection of VF and inappropriate shocks due to T-wave oversensing. The T-wave oversensing can often manifest when the ventricular sensitivity or gain is automatically adjusted in relation to the low-amplitude preceding R wave. Responding to this scenario by raising the minimum sensing thresholds carries the risk of undersensing native R waves, as well as underdetection of ventricular

fibrillation. In this case the Decay Delay was extended from 60 to 160 ms and the threshold start was increased from 62.5% to 75.0% (Figure 46.3, adapted from Swerdlow et al. 2005). It is important to recognize that although these changes did not alter the sensitivity threshold, the window to detect VF was made effectively shorter. In some but not all reprogramming situations, repeat ventricular fibrillation induction with defibrillation threshold testing may be warranted to confirm that ventricular fibrillation is reliably detected.

Answer 2, increasing the postpacing ventricular blanking period, is incorrect because over sensing of post pacing T waves is not present. Answer 3, turning off morphology discrimination, likely would have no effect in this case but can be beneficial in patients when the intrinsic QRS matches the morphology template. Answer 4, all of the above, is incorrect.

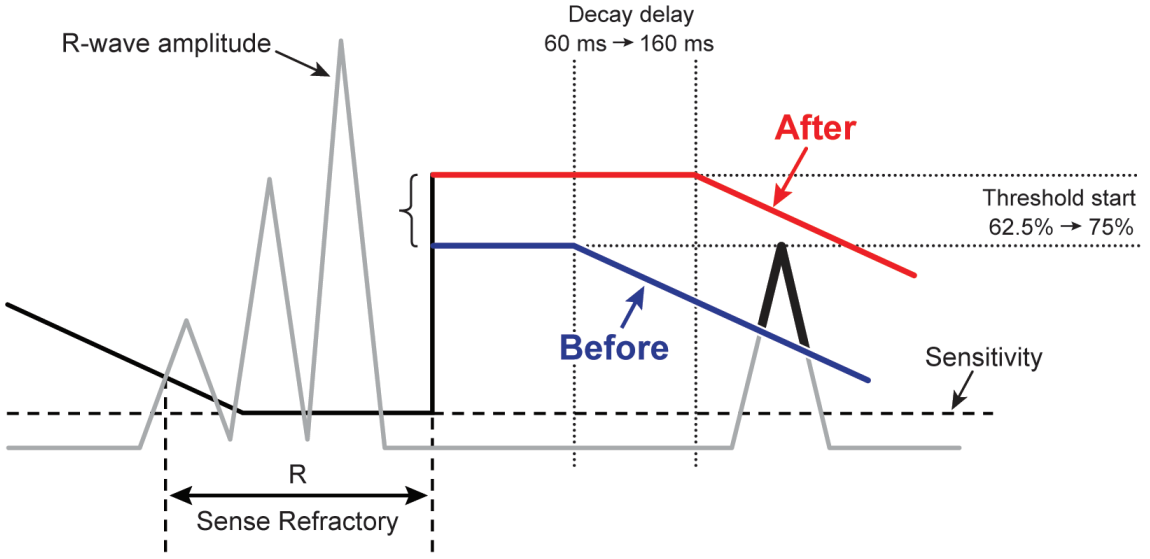

Figure 46.3 Demonstration of adjusted parameters in this patient.

Case 60

A 52-year-old male with dilated cardiomyopathy and ejection fraction of 27% had a resynchronization device implanted a year ago with initial clinical response. His symptoms gradually worsened because of underlying atrial fibrillation and difficult-to-control ventricular rates that resulted in inhibited biventricular rates. An AV node ablation was performed. Over the last month, he has had progressive dyspnea without obvious clinical cause. RV and LV pacing thresholds were unchanged.

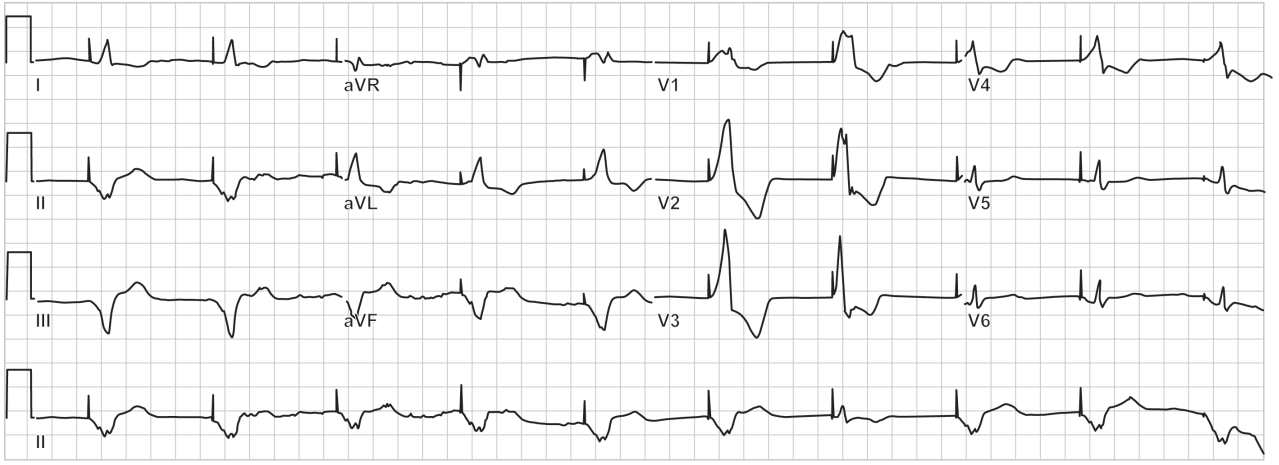

Figure 60.1 Patient's ECG.

60

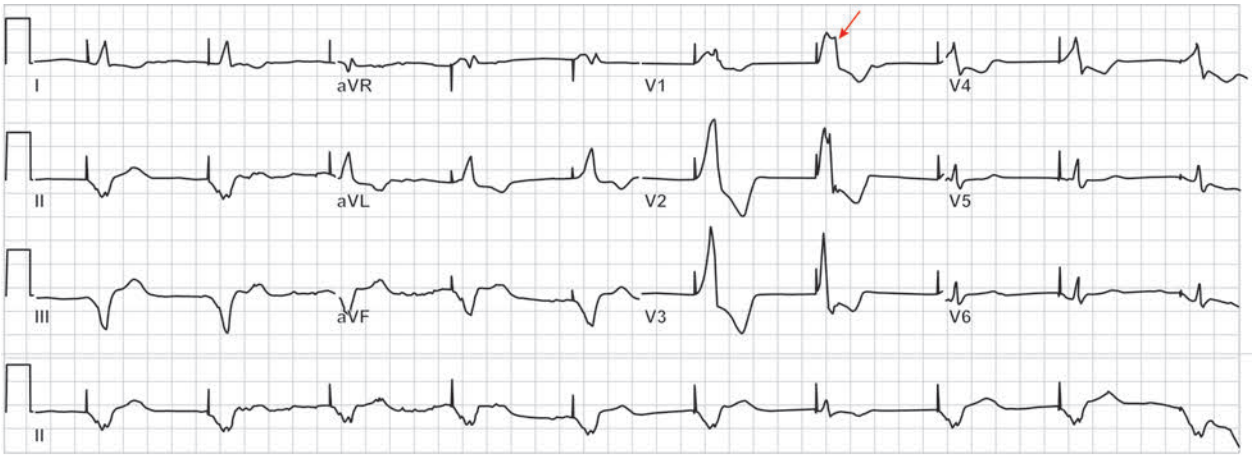

Figure 60.2 Additional ECG showing pseudofused beat.

Q:

Based on the ECG in Figure 60.1, which of the following may be contributing to the patient's clinical deterioration?

1. AF with inhibition of biventricular pacing
2. Frequent PVCs
3. Lead dislodgment
4. Suboptimal LV lead location
5. None of the above

60

2. Frequent PVCs

The key finding seen on this and other ECGs was the presence of PVCs. The arrow in Figure 60.2 points to a pseudofused beat with pacing not contributing to ventricular depolarization. The device counters would consider this as a paced event, and one may be misled into believing that 100% pacing is occurring while a significant number of these complexes may be fused or pseudofused. Although AF may also produce fused beats, the patient has had an AV node ablation, and the morphology of the wide-complex beat is not consistent with antegrade conduction through the AV node. On obtaining a Holter monitor and with manual analysis of wide-complex beats, it was determined that up to 20% of the patient's ventricular beats were PVCs, pseudofused beats, or fusion beats.

We can exclude ventricular lead dislodgment based on the information given that LV lead thresholds were unchanged. With regard to LV lead position, the QRS morphology (RBBB, initial isoelectric in lead I, and negative in leads II, III, and aVF) suggests LV posterior or posterolateral placement. However, the prominent R wave in lead I also suggests that programming an LV offset (LV earlier than RV) could be considered. However, the patient's initial clinical response to CRT makes it likely that the lead positioning was reasonable at implant.

There are several mechanisms by which PVCs give rise to suboptimal resynchronization therapy. When PVCs are sensed, the pacemaker will be inhibited, and the PVCs themselves may worsen cardiomyopathy and produce dyssynchrony to the same or a greater extent than expected with single-site ventricular stimulation or conducted rhythm with BBB.

Several device algorithms have been developed (V-sense response, Medtronic; biventricular trigger mode, St. Jude, Boston Scientific) in an effort to maintain resynchronization in the setting of ventricular ectopy or conducted supraventricular rhythms. These features attempt to maintain a semblance of resynchronization by delivering an LV pacing pulse when RV sensed events occur. The efficacy may be limited, however, since much of the ventricle may already be activated by the PVC by the time the event is sensed in the RV. Thus, fusion or pseudofusion results, and for LV PVCs, RV sensing may be a particularly late event, minimizing the benefit of LV pacing at that time.

Other features promote delivery of resynchronization therapy during atrial arrhythmia episodes by increasing the pacing rate as the patient's ventricular response rates increase. While resynchronization may be better promoted, the rapid rates themselves may be counterproductive, mitigating any CRT benefit.

PVCs may be detrimental to effective resynchronization in other ways as well.

Figure 60.3 is from a patient with incessant bigeminal ventricular ectopy. When this condition is frequent, the ectopy itself may produce a type of tachycardia-related cardiomyopathy. The effect on AV synchrony should also not be underestimated. There may be retrograde conduction to the atrium from the ventricular beats, and based on when the PVC is sensed, ventricular pacing may be delayed, safety pacing may occur, or ventricular pacing may occur quite late (circled).

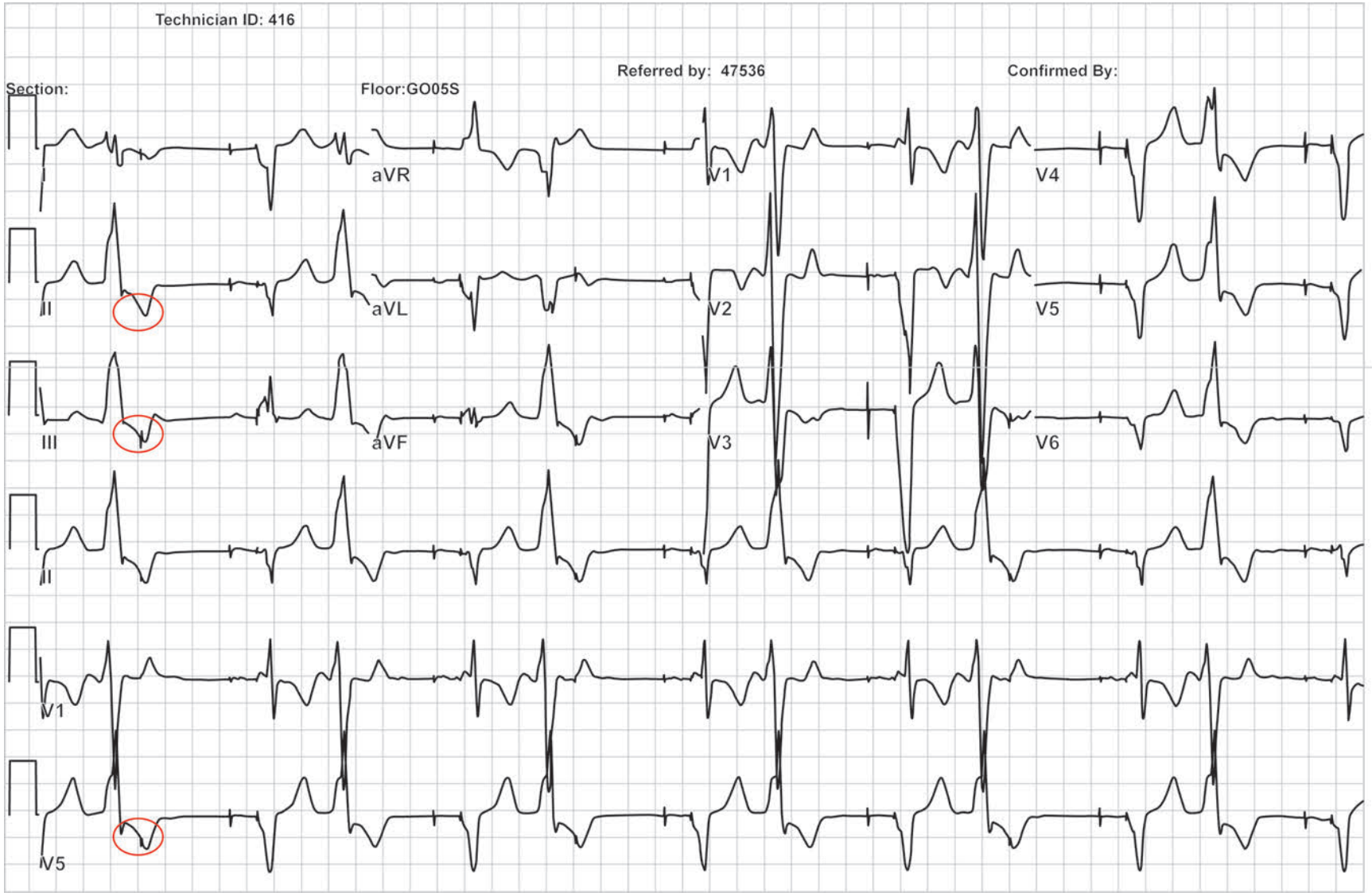

Figure 60.3 Patient showing incessant bigeminal ventricular ectopy.

60

The relationship with atrial pacing is also variable, and nonphysiologically short AV conduction times occur as a result of atrial pacing—PVC and inhibited ventricular pacing (Figure 60.4, circled). In this example, there is alternation between AP and VP (which is delivered as biventricular pacing), and AP and safety pacing (note the double down marker with VS, which indicates safety pacing). Safety pacing is occurring due to PVCs that immediately follow the atrial pacing event. Since the device cannot be certain whether this is crosstalk (sensing of atrial output on the ventricular channel) or a ventricular event, a ventricular pacing pulse is delivered with a shortened AV interval—typically 110 ms. In CRT devices, safety pacing is delivered only via the RV lead. Thus, in this example, every other complex is resynchronized (AP and VP) and the alternating complexes are PVCs with likely ineffective RV pacing (AP followed by VS with double marker). Note that the small but visible far-field R wave in the atrial EGM indicates the different morphology QRS for each of the pacing types. The clearly visible and nonsaturated ventricular EGM favors the presence of PVCs as opposed to crosstalk. Additionally, since ventricular sensitivity increases over time in defibrillators following each paced beat, the fact that the safety pacing occurs following shorter intervals (VP to VS interval) rather than longer ones

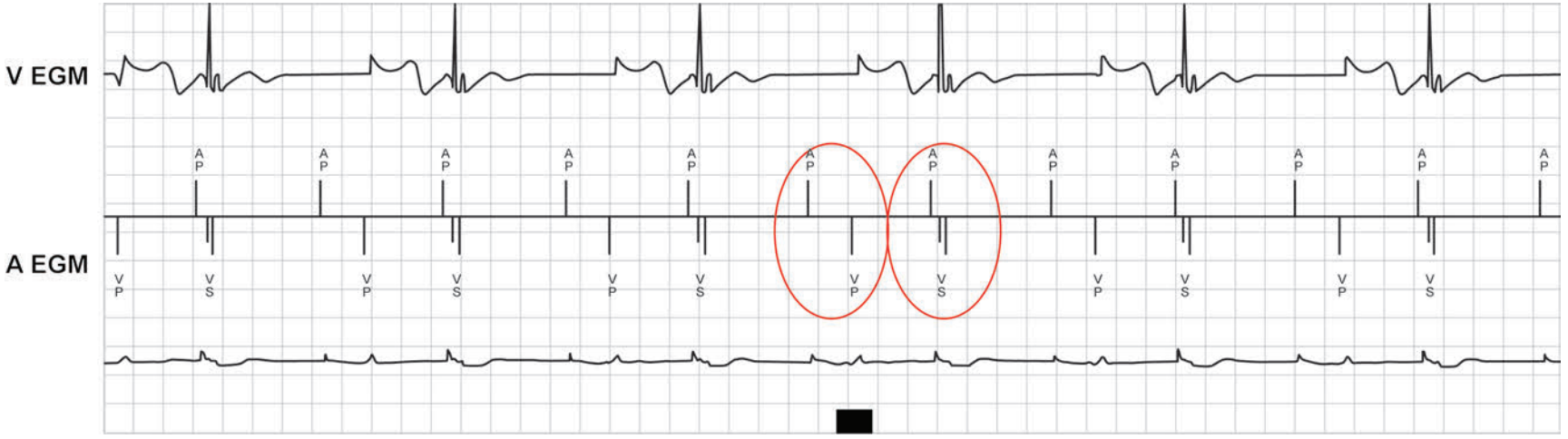

Figure 60.4 PVC and inhibited ventricular pacing.

argues against crosstalk and favors these events as being PVCs that trigger safety pacing.

A single PVC may result not only in inhibition of one resynchronized paced beat but in continuous promotion of intrinsic conduction and continued suppression of biventricular pacing. This results from functional undersensing in the atrium as illustrated in Figure 60.5. With most devices, following a PVC, the PVARP is extended. Either retrograde conduction from the PVC or the next sinus beat may fall in this extended PVARP and will not be tracked. Atrial pacing may then occur, but since the atrium is refractory, it will not capture, and if antegrade conduction through the AV node is present, then the sinus beats falling in the PVARP will conduct to the ventricle (Figure 60.6), and this, in turn, will result in persistent loss of biventricular pacing and continued antegrade conduction of sinus rhythm.

In some cardiac devices (Medtronic—atrial tracking recovery), features are designed to promote AV synchrony even if temporarily lost during a PVC or rapid atrial rhythm by temporarily shortening the PVARP to regain atrial tracking.

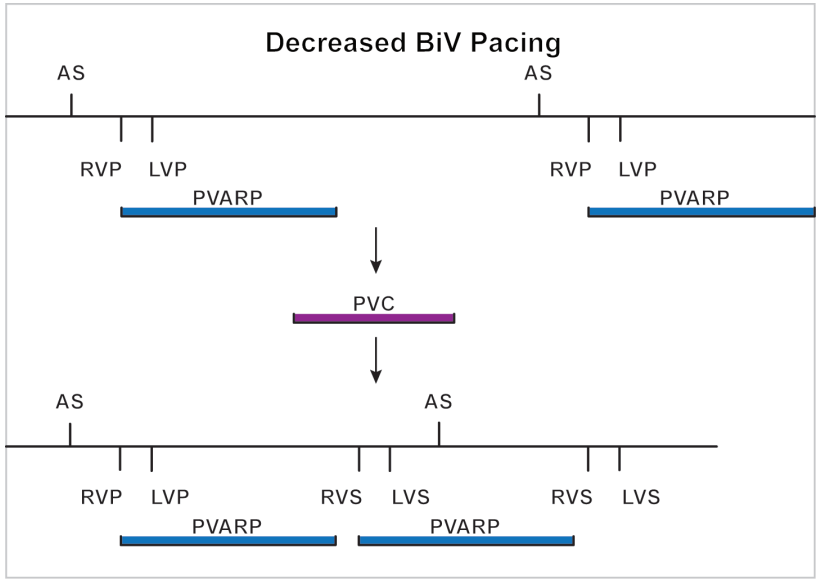

Figure 60.5 Decreased biventricular pacing.

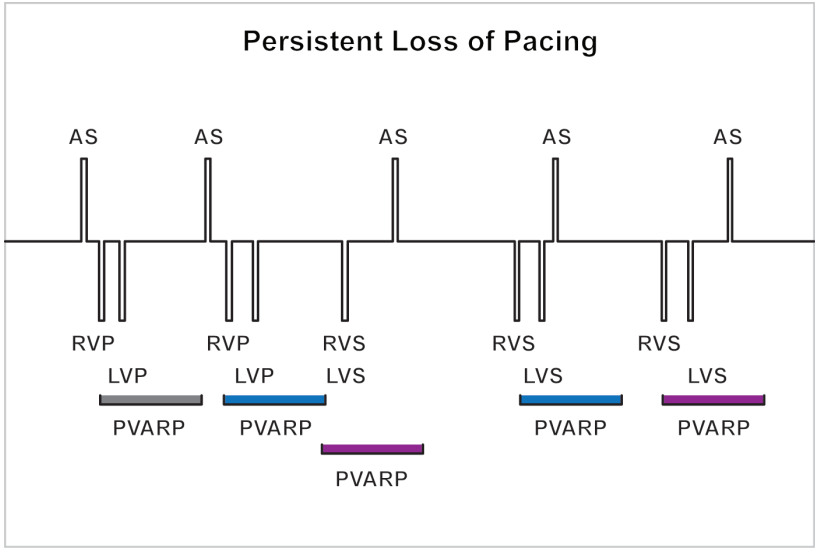

Figure 60.6 Persistent loss of pacing.

60

The ECG in Figure 60.7 shows frequent atrial ectopy. Although atrial ectopy is not generally as symptomatic as PVCs, they also result in several features similarly detrimental to resynchronization. AV synchrony is not maintained in a consistent fashion, as shown in the figure. A premature atrial beat or increase in the sinus rate may also give rise to functional undersensing with antegrade conduction and potential perpetuation of inhibition of biventricular pacing (not shown).

Higher atrial rates, frequent premature atrial and ventricular beats, and the presence of antegrade delayed AV conduction (a long PR interval) all promote this phenomenon and may prevent biventricular

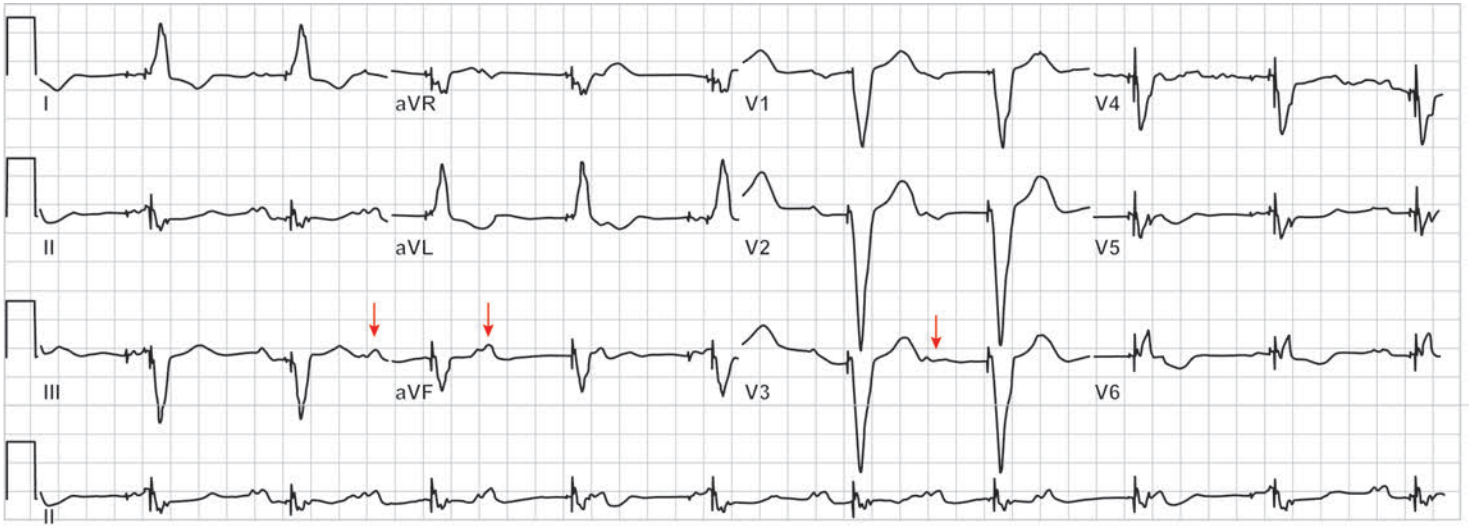

Figure 60.7 ECG showing frequent atrial ectopy.

stimulation, thus lowering the total "dose" of resynchronization (Figure 60.8). The key interval to keep in mind when troubleshooting insufficient biventricular pacing is that the total atrial refractory period (TARP) is the sum of the sensed AV interval and the PVARP. By preventing rapid rates in the atrium (beta-blockers), PVCs and PACs (antiarrhythmic drugs, ablation), and shortening the PVARP when possible (atrial tracking, recovery, turning off PVC PVARP extension) the frequency of biventricular pacing is increased. Conversely, algorithms to terminate pacemaker-mediated tachycardia (PMT) may interrupt CRT delivery by promoting intrinsic rhythms.

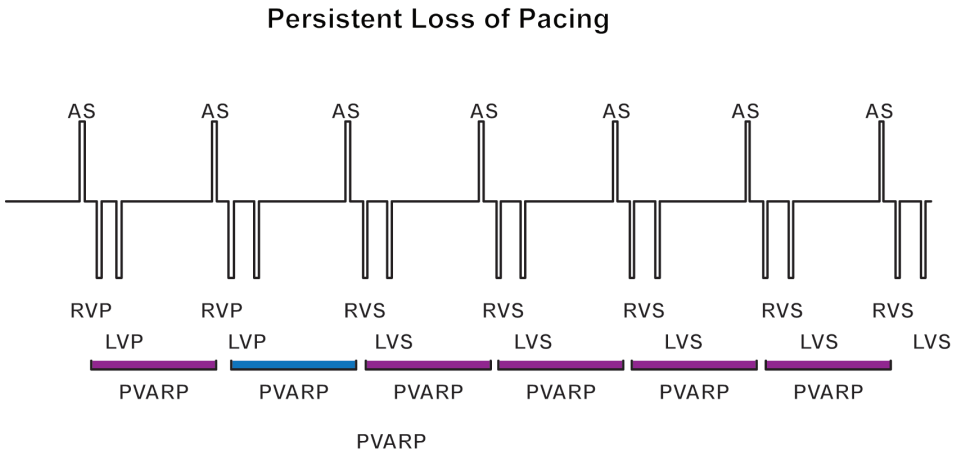

Figure 60.8 High atrial rate: persistent loss of pacing.

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Figure 60.9 is an ECG from a patient with a CRT device placed 1 year ago in sinus rhythm.

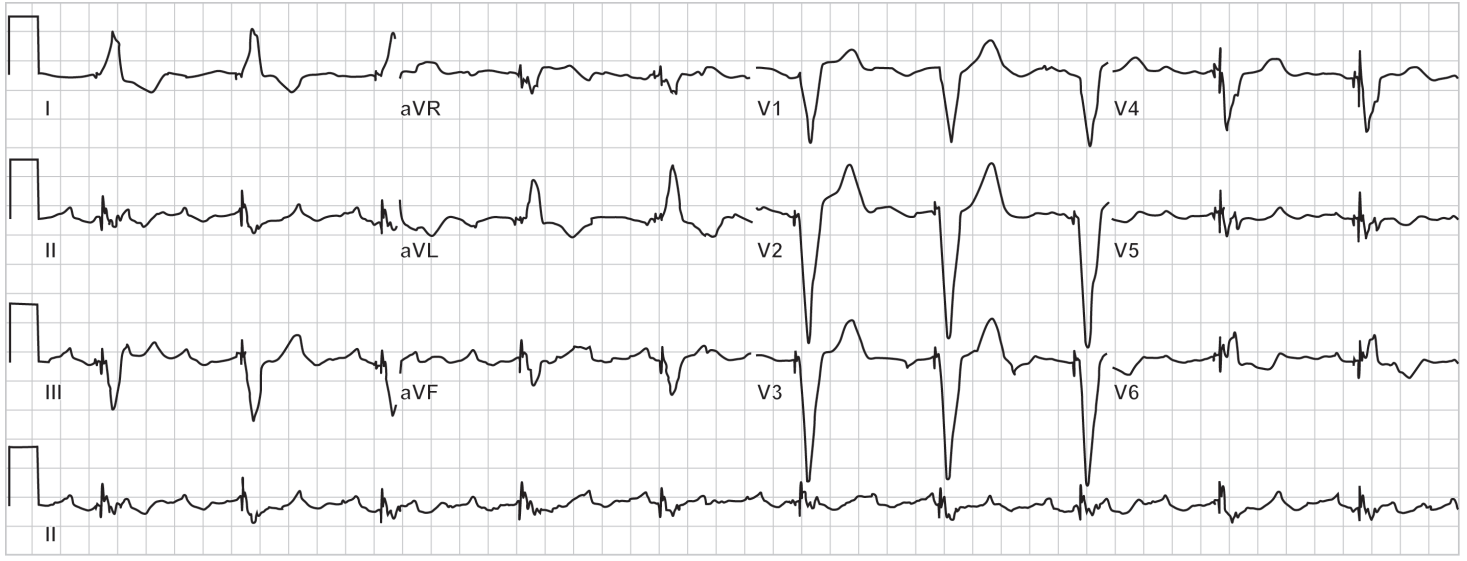

Figure 60.9 Patient's ECG.

60

Q:

In Figure 60.9, what is the most likely cause of abrupt clinical deterioration?

1. Functional undersensing
2. Failure to capture
3. Atrial flutter
4. Frequent PVCs
5. None of the above

60

3. Atrial flutter

Regular atrial flutter waves are shown by arrows in Figure 60.10. The patient's rhythm has changed, and often temporal correlation with clinical deterioration will be evident. Although the same syndrome may occur with AF, in atrial flutter, because of continued organized atrial activity, symptoms are often more pronounced as AV dyssynchrony is caused by poorly timed flutter contractions, as opposed to the complete loss of AV synchrony associated with AF due to the lack of meaningful atrial activity. Thus, with flutter, symptoms may be more pronounced even when ventricular rates are well controlled and CRT therapy is otherwise delivered. This patient had a marked improvement in symptoms with cardioversion and was subsequently treated with radiofrequency ablation. Atrial ATP may also be appropriate in some cases, but at present CRT devices with atrial ATP therapy are not available. In selected patients, use of an ATP device with Y-adapted RV and LV leads may be tried to get both the benefits of maintaining sinus rhythm and CRT when radiofrequency ablation and antiarrhythmic drug therapy fails. However, adapting RV and LV leads in defibrillators is generally avoided due to the significant risk of R-wave double-counting and inappropriate shock.

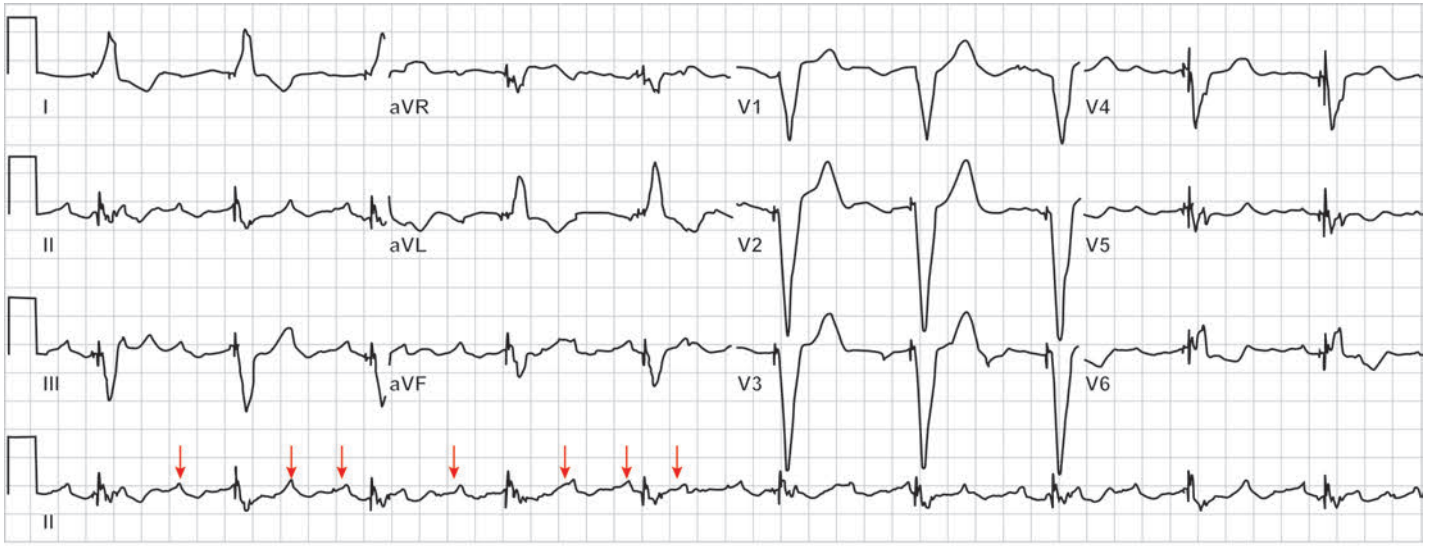

Figure 60.10 Regular atrial flutter waves.