Clinical Electrophysiology Review

SECOND EDITION

George J. Klein
Eric N. Prystowsky

Mc Graw Hill Education

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Clinical Electrophysiology Review

Second Edition

George J. Klein, MD
Professor of Medicine
University of Western Ontario
London, Ontario, Canada

Eric N. Prystowsky, MD
Director, Electrophysiology Laboratory
St. Vincent Hospital, Indianapolis
St. Vincent Medical Group
Indianapolis, Indiana
Consulting Professor of Medicine
Duke University Medical Center
Durham, North Carolina

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To my wife and best friend, Klara.

To my son Ben and daughter-in-law Elissa and their children Adam, Leah, and Beth, and to my daughter Anna and son-in-law Mark and their daughter Lucy, who are all my real source of pride and joy.

To the memory of my unselfish and giving parents, Paul and Clara.

— George J. Klein

To my wife Bonnie, who is my constant source of love and support.

To my sons David and Daniel, whose lives have filled me with pride and joy, and to their wives, Malia and Beth, who are the daughters we never had.

And for my grandchildren, Dylan, Laila, Amber, and Noah, in whose presence the sun always shines and life stands still.

And in loving memory of my parents, Drs Rose and Milton Prystowsky, who taught me to care for others and that being a physician is not a gift to be wasted.

— Eric N. Prystowsky

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Preface

Enormous change has occurred in our specialty since the publication of the first edition of *Clinical Electrophysiology Review*. Diverse energy sources are in development or common use with robotic and magnetic guidance systems to pinpoint the ablation target. We use sophisticated mapping systems with anatomically true graphics online and nonfluoroscopic visualization of our catheters. Online intracardiac ultrasound has become a routine tool in many laboratories. The ablation of atrial fibrillation has become commonplace and is the most common ablation procedure in many laboratories. The ablation of VT has become routine and the pericardial space is frequently entered to access the epicardium.

Despite this tremendous progress, the need to preserve and indeed to cultivate our skills in electrocardiographic and electrophysiologic reasoning and diagnosis remains. There is an increasing emphasis on the technical aspects of electrophysiology in our teaching centers, and generations of young electrophysiologists may not acquire the skills to decipher a complex arrhythmia puzzle. These cases, when they appear, must be dissected in great detail and the learning points must be thoroughly examined. It is in this spirit that we have produced the second edition of this book.

While new cases have been added and others refreshed, those familiar with the first edition will not see a profound change in emphasis. Indeed, the electrocardiographic and electrophysiologic problem-solving skills required to master these arrhythmias have not changed for a long time and are unlikely to change substantively in the future. There is no attempt to include every bizarre case that we see or to provide encyclopedic coverage of every issue. Rather, we emphasize an organized approach based on making observations, "framing" the problem, and testing each "hypothesis" to explain the observations. We have added several ECG examples because ultimately the ECG and intracardiac tracings are on a continuum and require similar approaches and skills. We discourage a strict pattern recognition approach to the ECG in favor of an electrophysiologic approach.

We hope that this edition will serve you well.

George J. Klein, MD
Eric N. Prystowsky, MD

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Chapter 1

Analysis of Complex Electrophysiologic Data

A novice in a busy electrophysiology (EP) laboratory will generally learn to recognize the common arrhythmias in a relatively short time. It requires considerably more seasoning to recognize the variants and unusual mechanisms, or to "hit the curve ball." It is hoped that the following commentary assists in providing structure and focus to the EP study and facilitates analysis of the case studies to follow.

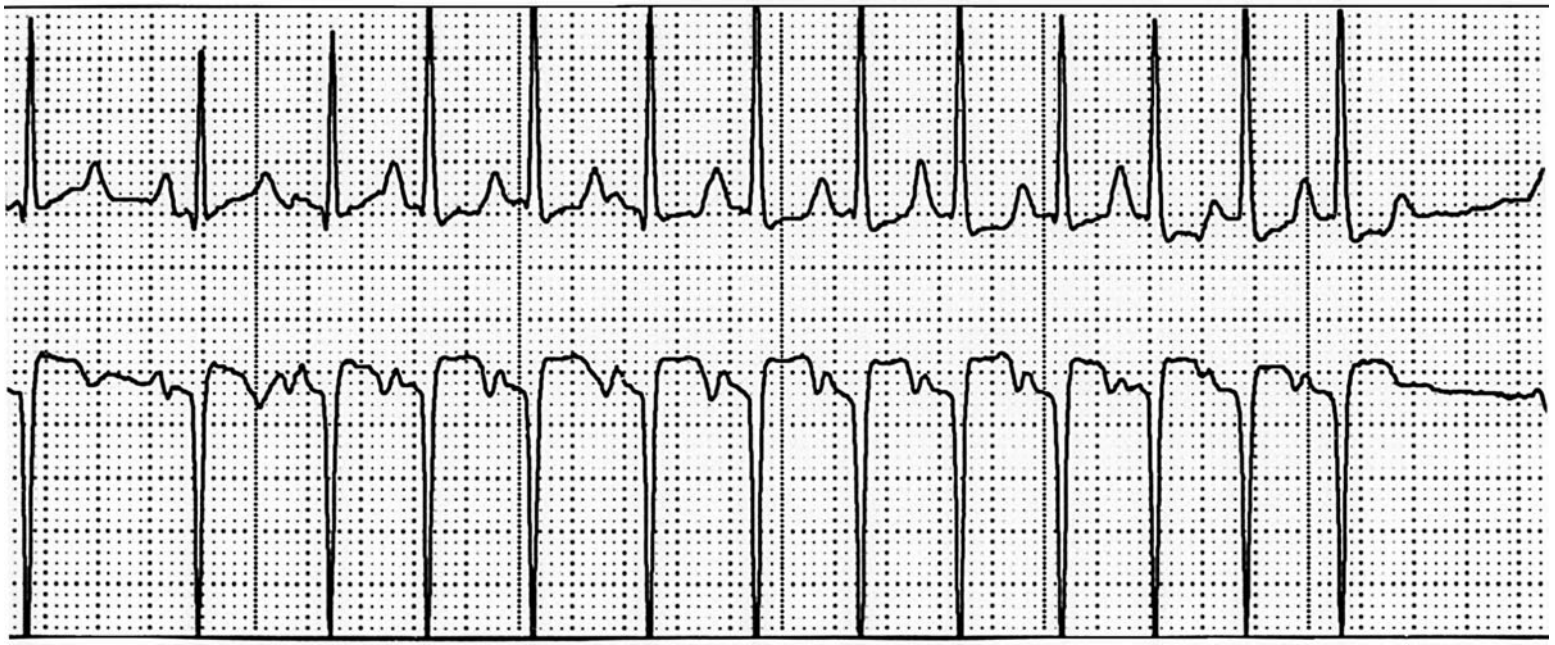
It Begins with the Electrocardiogram

The EP study is an extension of the electrocardiogram (ECG) with the addition of intracardiac recording and programmed electrical stimulation. Insightful interpretation of the ECG allows for prospectively considering additional catheters, stimulation sequences, or maneuvers appropriate for the postulated arrhythmia. This limits the diagnostic possibilities and avoids unnecessary steps (Fig. 1–1). The fundamentals of ECG interpretation of an arrhythmia include identification of P waves, determining the atrioventricular (AV) relationship, and analyzing the QRS morphology (Table 1–1). For confusing problems, it is useful to create a "written" list of all potential hypotheses and to plan for specific interventions that will test them. As data are accumulated during the EP study, the facts supporting or refuting the hypotheses can be tabulated. The hypotheses can be represented by schematic drawings for complicated scenarios. This method is illustrated at the end of this chapter.

Less Is Often Not More

There are those gifted, intuitive individuals who leap to the correct diagnosis and apparently bypass all the rational, systematic steps. Most of us, however, are better served by a consistent, methodical approach that does not cut corners. A sample protocol for an unknown supraventricular tachycardia (SVT) is shown in Table 1–2. When used routinely, such a protocol will usually result in induction of clinically relevant tachycardia and provide an assessment of the pertinent EP of the heart. Determining the functional properties of the atria, ventricles, and AV conduction system in an individual elucidates the potential arrhythmia mechanisms and limits the diagnostic possibilities for the observed arrhythmia. For example, it is difficult to imagine AV reentrant tachycardia occurring in the total absence of retrograde conduction, even realizing the relatively rare occurrence of conduction over an accessory pathway (AP) only in the presence of isoproterenol. It is also important to display the data channels in a consistent sequence to provide an orderly and familiar framework that facilitates analysis. This point is underscored by the fact that even experienced electrophysiologists require a period of adjustment when looking at data from other laboratories. Of course, other nonconventional recording sites can be added to facilitate diagnosis in selected cases. For example, recording from the left bundle branch can be useful when confirming the diagnosis of bundle branch reentrant tachycardia.

A thorough diagnostic study need not be time consuming and pays dividends both intellectually and clinically. The temptation to ablate an obvious AP without study will not be productive if the patient's symptoms are not related to any tachycardia, the patient's tachycardia is not related to the pathway, or the "culprit" AP is a different pathway (Fig. 1–2). The study also provides information regarding other potential rhythm problems that may be unrecognized during the clinical assessment and allows consideration of alternative approaches such as slow pathway ablation in a patient with AV reentrant tachycardia that can only occur with anterograde conduction over the slow AV node pathway. Radio-frequency ablation itself is an important diagnostic



1–1 Two-channel rhythm strip recorded from a patient scheduled for electrophysiology study for palpitations. The onset of the tachycardia occurs after the second QRS and the P wave is noted in the following diastole to be of different morphology than the probable initial sinus P wave. The identification of the P wave during tachycardia is facilitated by comparison with the last QRS in the strip that is not followed by a P wave. Careful measurement with calipers (a critical tool of the electrophysiologist) will illustrate that the onset of the tachycardia does not require PR prolongation. In addition, the cycle length varies and during this, the PR stays constant while the apparent RP varies. These findings are most compatible with an atrial tachycardia. Note that most junctional reentrant tachycardias would require some AV delay at the onset and such variable retrograde conduction would be unusual. This information provides a focused starting point to plan electrophysiology study.

Table 1–1 ECG Rhythm Analysis

- Identify P waves and determine their morphology, if possible
- Determine the atrial rhythm
- Analyze the QRS complex morphology
- Determine the A–V relationship

tool. Cases involving multiple tachycardia mechanisms can be very confusing. In such situations, at least one of the tachycardia mechanisms is frequently obvious and successful ablation of the tachycardia generally simplifies the diagnosis of the remaining mechanism(s).

The Key Is Frequently at a Transition

Fishermen have long appreciated that the majority of the fish are caught in a relatively small area of the lake. Similarly, the correct diagnosis may not be apparent from the copious EP records during stable tachycardia. Although the electrograms may have a certain temporal sequence, there is no indication of cause and effect in the sequence of electrograms. Is the atrium driving the ventricle or vice versa? Is the preexcited QRS an active participant in the tachycardia circuit or merely a bystander camouflaging another mechanism? The "hot spots" that frequently yield the answer are the *zones of transition.* The zones include the *onset* of tachycardia, the *termination* of tachycardia, change to an *alternate QRS morphology, irregularities in cycle length* (CL), and *ectopic cycles* (Table 1–3). The onset reveals the conditions necessary to initiate the tachycardia. Does it require block in an AP, critical prolongation of the atrio-His (AH) interval, or conduction delay in the His–Purkinje system? Does a SVT consistently terminate spontaneously with an atrial electrogram? The latter strongly suggests that the tachycardia mechanism obligates AV node conduction. Does a change from normal QRS to bundle branch block alter any of the conduction intervals or tachycardia CL, suggesting the bundle branch is a critical component of the tachycardia circuit? Careful attention to the zones of transition is often rewarding as is illustrated.

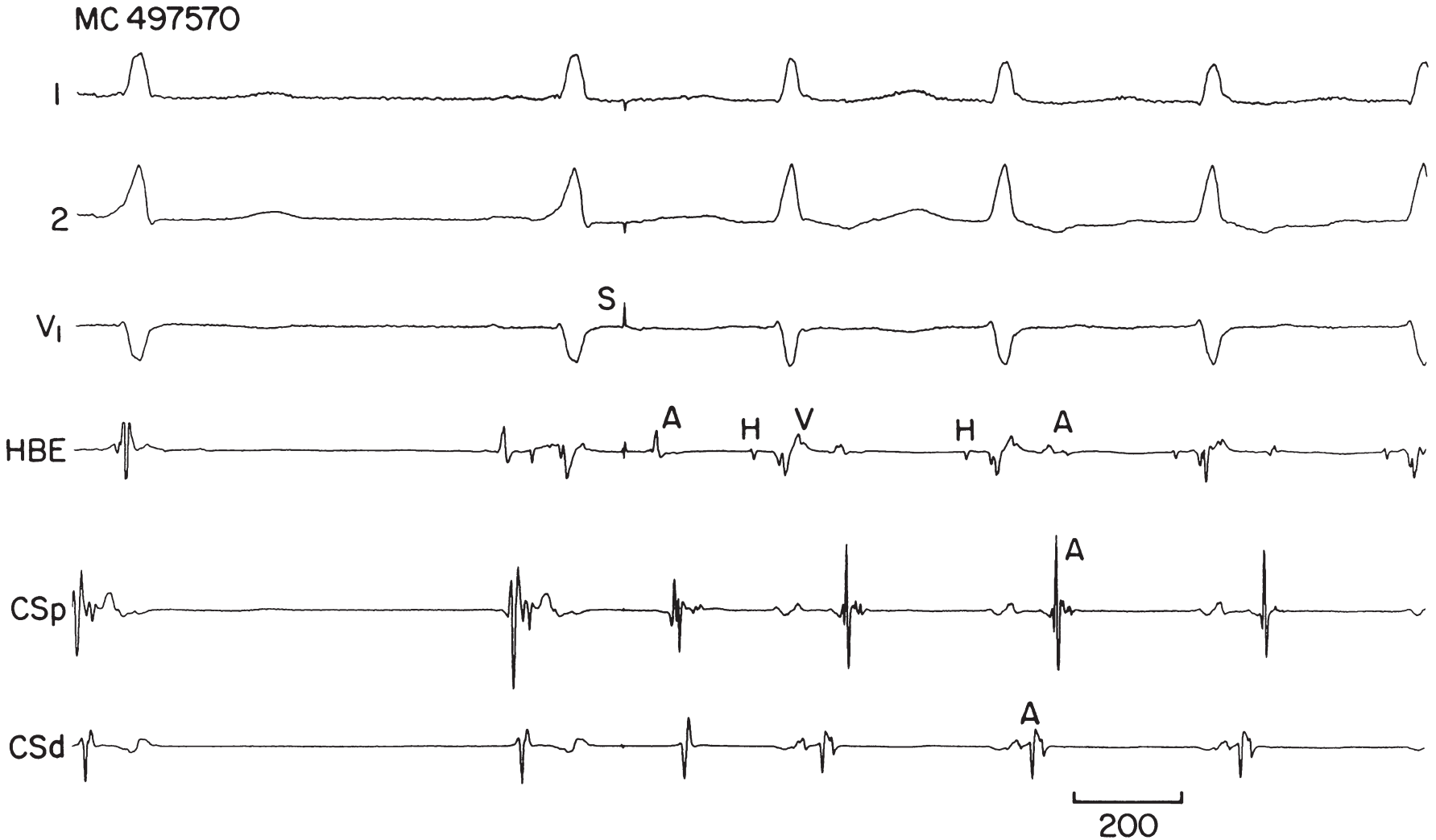
Table 1–2 Sample Protocol for Supraventricular Tachycardia

- Record five surface ECG leads
- Insert four intracardiac catheters to record from the high right atrium (HRA), His bundle (H), right ventricle (RV), and coronary sinus (CS)
- Incremental atrial and ventricular pacing to AV and VA block, respectively
- Atrial and ventricular extrastimulus testing at two or more basic drive cycle lengths
- Use multiple extrastimuli, atrial, and ventricular pacing, pharmaceuticals (e.g., adenosine, isoproterenol, verapamil), as required

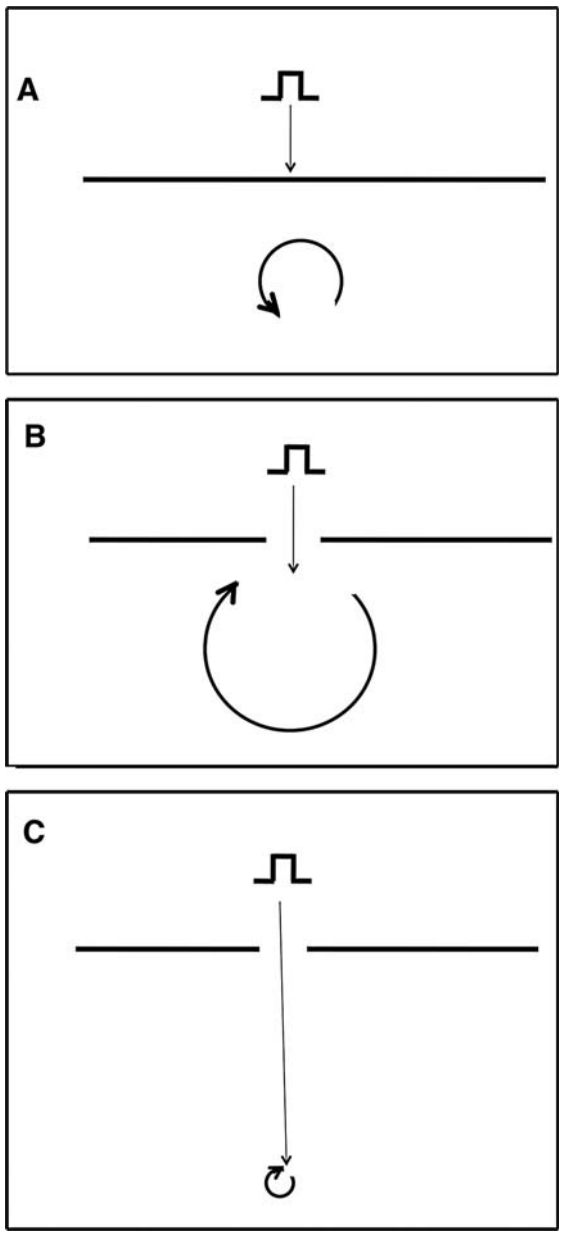
Make Something Happen

The EP study provides an opportunity to disturb an arrhythmia with pacing, extrastimuli, autonomic maneuvers, physical maneuvers, and drugs. Single, or multiple, atrial or ventricular extrastimuli are programmed into the cardiac cycle and made progressively more premature to loss of capture. This invariably provides the *zone of transition* that clarifies the requirement of atrium or ventricle in the mechanism or alters the tachycardia in a manner that clarifies the problem. In other words, if the tachycardia mechanism is not perfectly obvious, overdrive pacing or programming extrastimuli will almost invariably clarify it. A long-standing "inverse rule" may be useful to trainees—initially introduce premature atrial extrasystoles into a wide QRS tachycardia and ventricular extrasystoles into a narrow QRS tachycardia. Changes in posture cause autonomic adjustments and alter cardiac filling. Agents such as adenosine usually affect specific tissues and mechanisms, and can be invaluable. Isoproterenol is useful for mimicking states of catecholamine excess or altering specific EP properties to allow induction of tachycardia.

Although many pacing and extrastimulation maneuvers have been described, it is useful to understand the basic underlying principles by which they function. The overriding principle underpinning most pacing interventions is illustrated schematically in Fig. 1–3. In essence, the ability of pacing to influence or "reset" a tachycardia is dependent on two key variables.



1–2 Tracing from a patient with Wolff–Parkinson–White (WPW) and documented supraventricular tachycardia. The first two cycles are preexcited and a 12-lead ECG suggested a septal pathway conducting anterogradely. Earliest ventricular activation in sinus rhythm is at the proximal coronary sinus electrode (CSp) positioned near the orifice of the coronary sinus. An atrial extrastimulus (S) blocks the pathway and starts supraventricular tachycardia. However, earliest retrograde atrial activity is at the *distal* coronary sinus electrogram. In this patient, complete mapping revealed that the "culprit" accessory pathway was a concealed left lateral pathway and the manifest accessory pathway was of no clinical significance. Ablation of this pathway would have served no purpose. 1, 2, and V1, surface ECG; HBE, His bundle electrogram.



1–3 Schematic illustration of dependence of pacing intervention on distance and access to the tachycardia mechanism. (**A**) The pacing cannot conduct to the tachycardia circuit. An example would be ventricular pacing with an atrial tachycardia in the absence of ventriculoatrial conduction. (**B**) The paced impulse is close to and has excellent access to the tachycardia mechanism. An example might be right ventricular basal pacing in AV reentrant tachycardia over a right accessory pathway. You would expect the tachycardia to be easily reset by pacing at relatively long coupling intervals and you would expect also the VA interval during tachycardia to approximate the stimulus to A interval during pacing. You would also expect the postpacing interval (PPI) to be close to the tachycardia cycle length. (**C**) The pacing site is relatively far from the circuit and the circuit is relatively small. The usual example would be RV pacing during AV node reentrant tachycardia. You would expect that it would be difficult to reset this tachycardia and only with short coupling intervals. In addition, you would expect the VA interval during tachycardia to be considerably longer than the stimulus to A interval during pacing. You would also expect the PPI to be considerably longer than the tachycardia cycle length.

Table 1–3 Zones of Transition: The Key to the Mechanism

- The onset and termination of tachycardia
- Change to an alternate QRS morphology
- Irregularities in cycle length
- Ectopic cycles
- The onset and termination of overdrive pacing

The first is *distance* from the pacing site to the tachycardia mechanism. (Note that this distance may also be "electrophysiologic" as well as geographic in that the pacing site may be "far" from the tachycardia mechanism if there is conduction block adjacent to pacing site requiring a roundabout access to the mechanism.)

The second is *access to the circuit*. A large reentrant circuit with a large excitable gap is more penetrable by pacing than a "focal" arrhythmia source. In essence, preexcitation of the subsequent A by a premature ventricular complex (PVC) during SVT occurs much more readily with a large AV reentrant circuit close to the pacing site rather than the much smaller and most distant circuit of AV node reentry. Similarly, capture of a SVT by ventricular pacing also depends on the same factors. The postpacing interval (PPI) and the difference of ventriculoatrial (VA) during tachycardia compared with VA during pacing clearly depend on these. In fact, the PVC that resets the A during SVT is just capture for one cycle and all the useful postpacing data also apply to the single PVC that resets tachycardia as will become evident in some of the exercises.

Expect the Unexpected

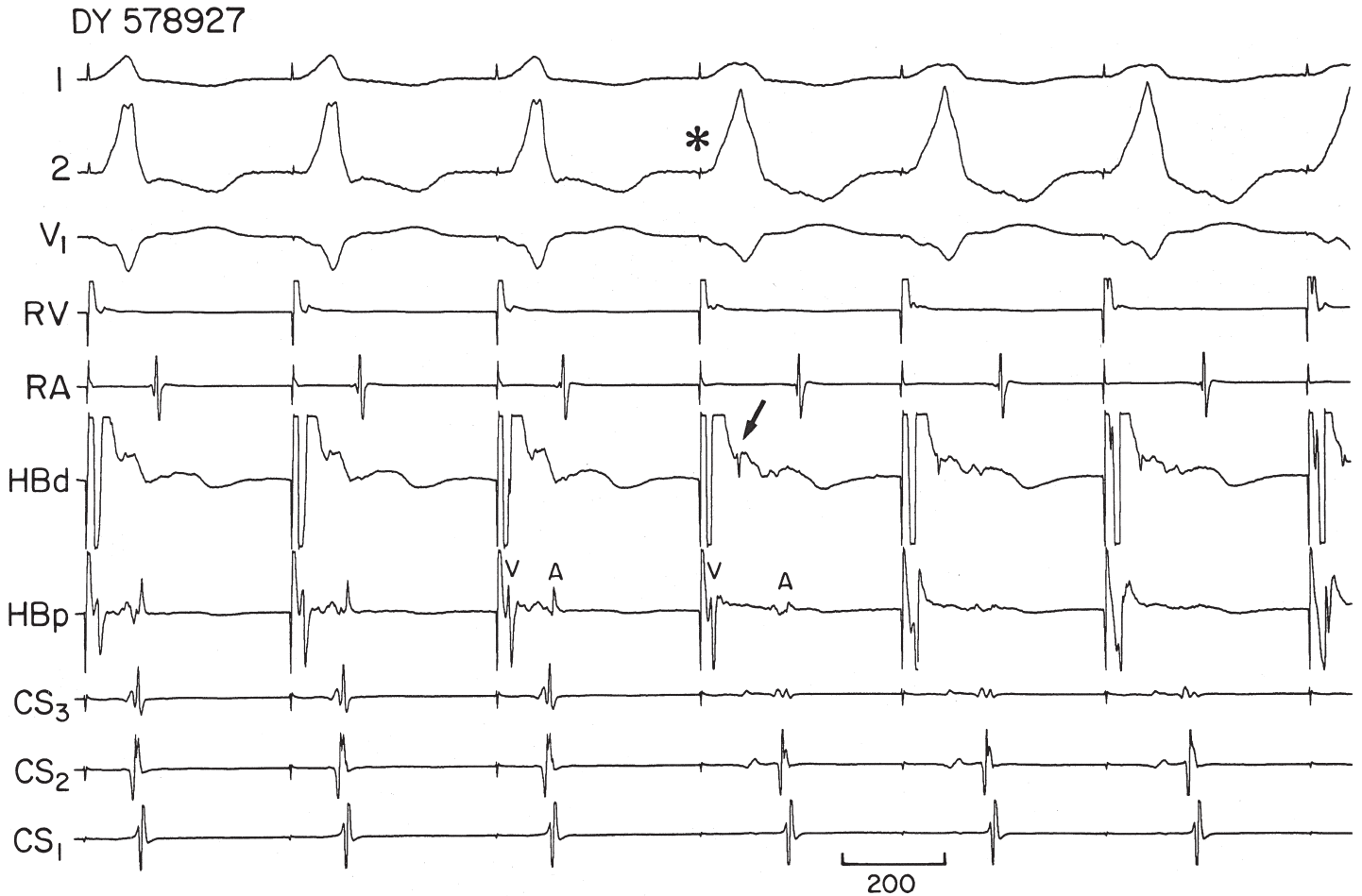
It is important to keep an open mind to all the diagnostic possibilities until the correct one has been clearly established. Prematurely accepting what appears to be obvious may result in the psychological trap of fitting subsequent observations to the expected and may blind a person from performing the required steps. For example, a SVT with simultaneous atrial and ventricular activation immediately suggests AV node reentry but does not rule out atrial tachycardia with a long AV interval or junctional tachycardia with retrograde conduction.

You Must Have the Tools

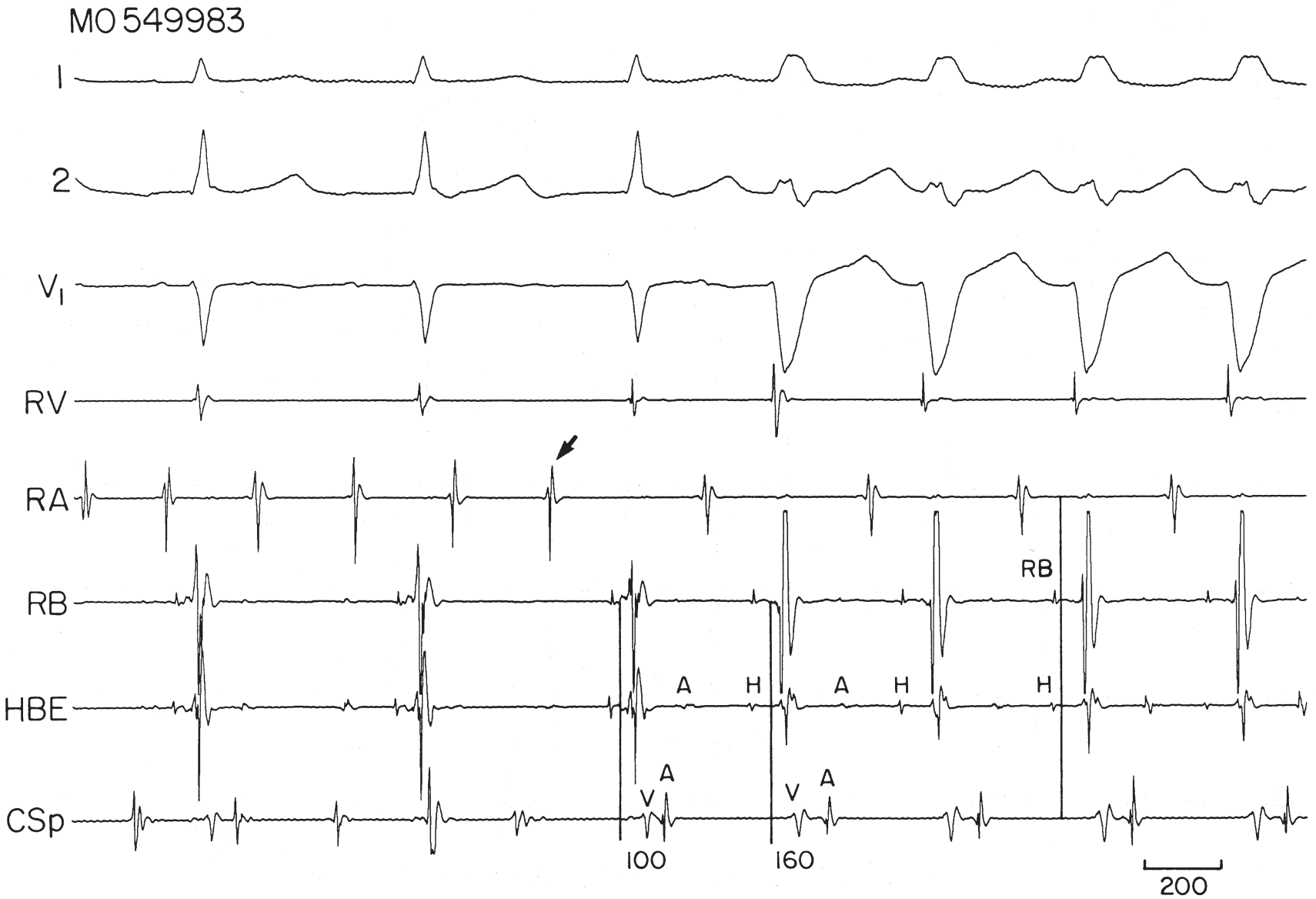
An organized approach and a strategic plan are only useful with a firm knowledge of physiologic principles and mechanisms. Consider an example where ventricular pacing produces a "central" (concentric) retrograde activation sequence with earliest retrograde atrial activation at the His bundle electrogram. Retrograde conduction time is constant (not rate dependent), and there is no suggestion of anterograde preexcitation. Is retrograde conduction proceeding over the AV node, or is it proceeding over a "concealed" septal AP? Block of retrograde conduction with adenosine favors but does not definitively prove AV node conduction since adenosine may affect some APs. A fundamental physiologic principle can be applied. *The VA conduction will be shortest when pacing at the ventricular site closest to the retrograde pathway.* In the case of an anteroseptal AP, pacing the ventricle near the His bundle will provide the shortest VA interval (assuming one does not use sufficient energy to cause His bundle capture). In the case of AV node conduction, pacing near the terminus of the right bundle branch (the RV apex is close to this) will provide the shorter VA interval when pacing at the same rate. This principle is illustrated in Fig. 1–4. Pacing the His bundle directly will result in very early capture of the atrium near the His bundle. Loss of His bundle capture (by lowering current strength) and retaining capture of myocardium in the region will result in no change in VA interval if retrograde conduction was proceeding over an anteroseptal AP but will result in VA prolongation if conduction was proceeding over the AV node or a more distant AP.

As another example, AV node conduction is rate dependent (decremental) with prolongation of the AH interval after progressively more premature atrial extrastimuli, while AP conduction is generally not rate dependent. However, it is important to appreciate that some APs exhibit impressive rate dependence comparable to the AV node, whereas other AV nodes exhibit little or no rate dependence and mimic AP conduction. There is no shortcut to the assimilation of EP principles.

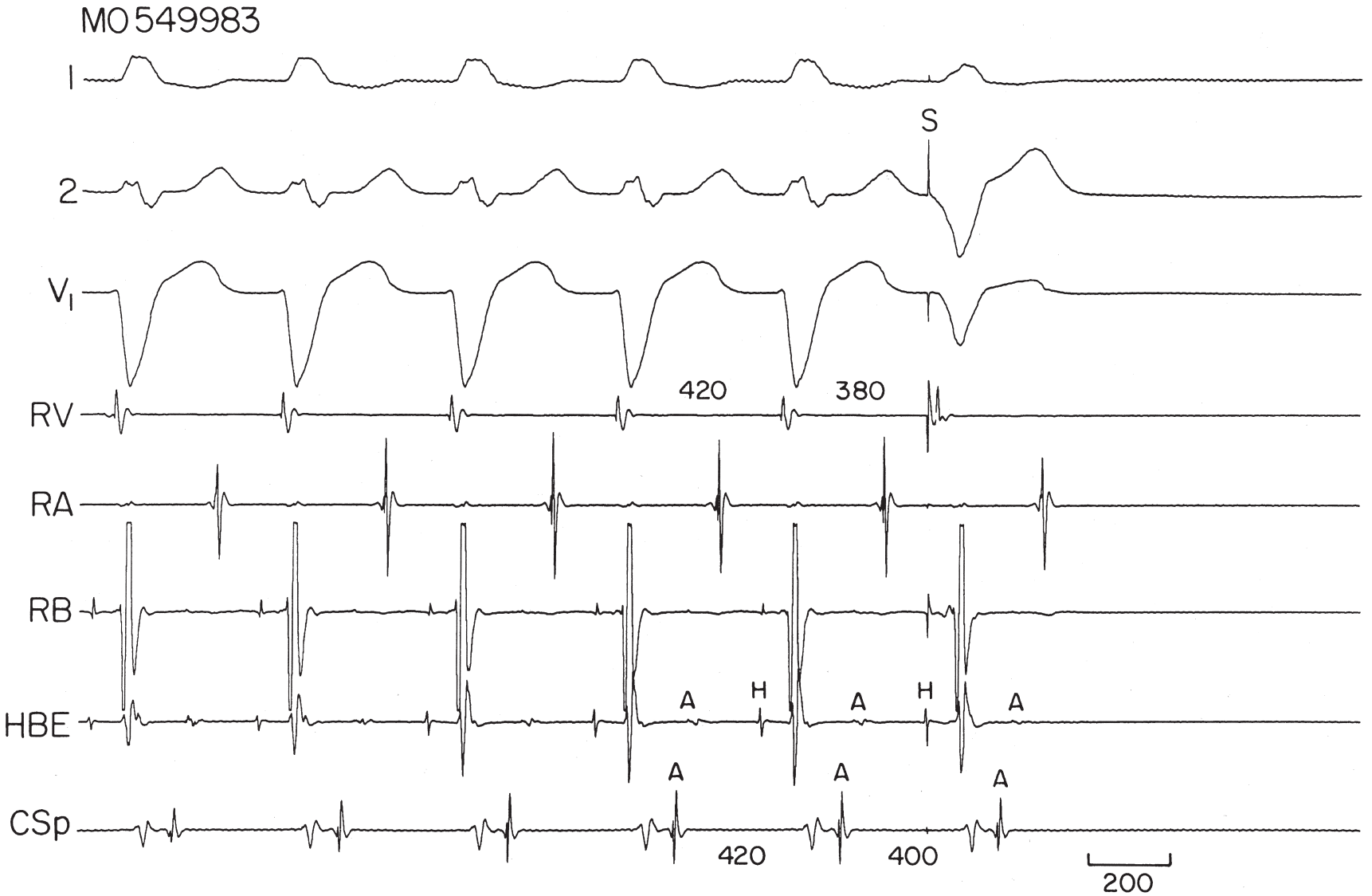
A differential diagnosis of potential entities when a phenomenon is encountered is a fundamental tool to begin the process of hypothesis testing to arrive at the correct diagnosis. Tables 1–4 to 1–12 may be useful in this regard in the analysis of the unknown traces providing the possible mechanisms under each category of observation.



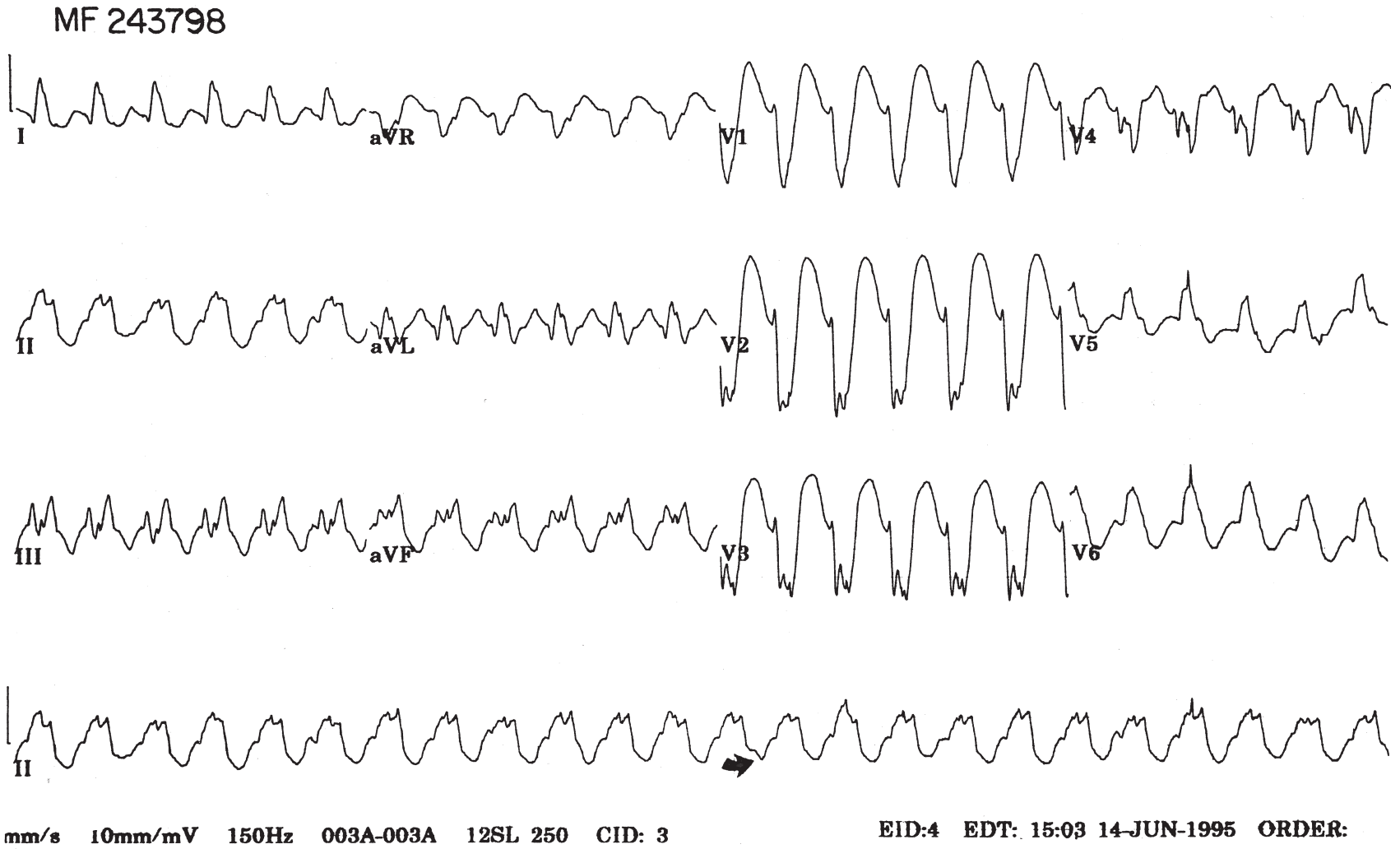
1–4 Evaluation of retrograde conduction during EP testing, "para-Hisian pacing." This patient had concentric retrograde atrial conduction that was rate independent (not "decremental") and it was unclear whether retrograde conduction was proceeding over the normal AV node or over a concealed septal accessory pathway. "Para-Hisian" pacing is started by pacing the distal pole of the His catheter with the electrogram at the site showing a clear His deflection and relatively small A so as not to pace the A inadvertently. *Right ventricular paraseptal pacing* has been achieved because the HBd catheter shows local capture. His bundle pacing is achieved (first three cycles) as suggested by the relatively narrow QRS (in fact, fusion between RV pacing and His pacing). Noticeably, atrial capture is *not* present since the stimulus to A at the atrial septum is longer than you would expect if such were the case. As the current strength is reduced, His bundle capture is lost and the adjacent RV myocardium is still paced. This is indicated by the widening of the QRS (*asterisk*) and the appearance of a retrograde His potential (*arrow*). This clearly indicates that retrograde conduction was proceeding over the AV node since conduction over an anteroseptal AP would not be affected by the loss of His bundle capture. A, atrial electrogram; CS, coronary sinus electrograms from proximal (3) to distal (1), respectively; HB, His bundle electrograms from the distal (d) and proximal (p) poles of the catheter; RA and RV, right atrial and ventricular electrograms, respectively; V, ventricular electrogram.



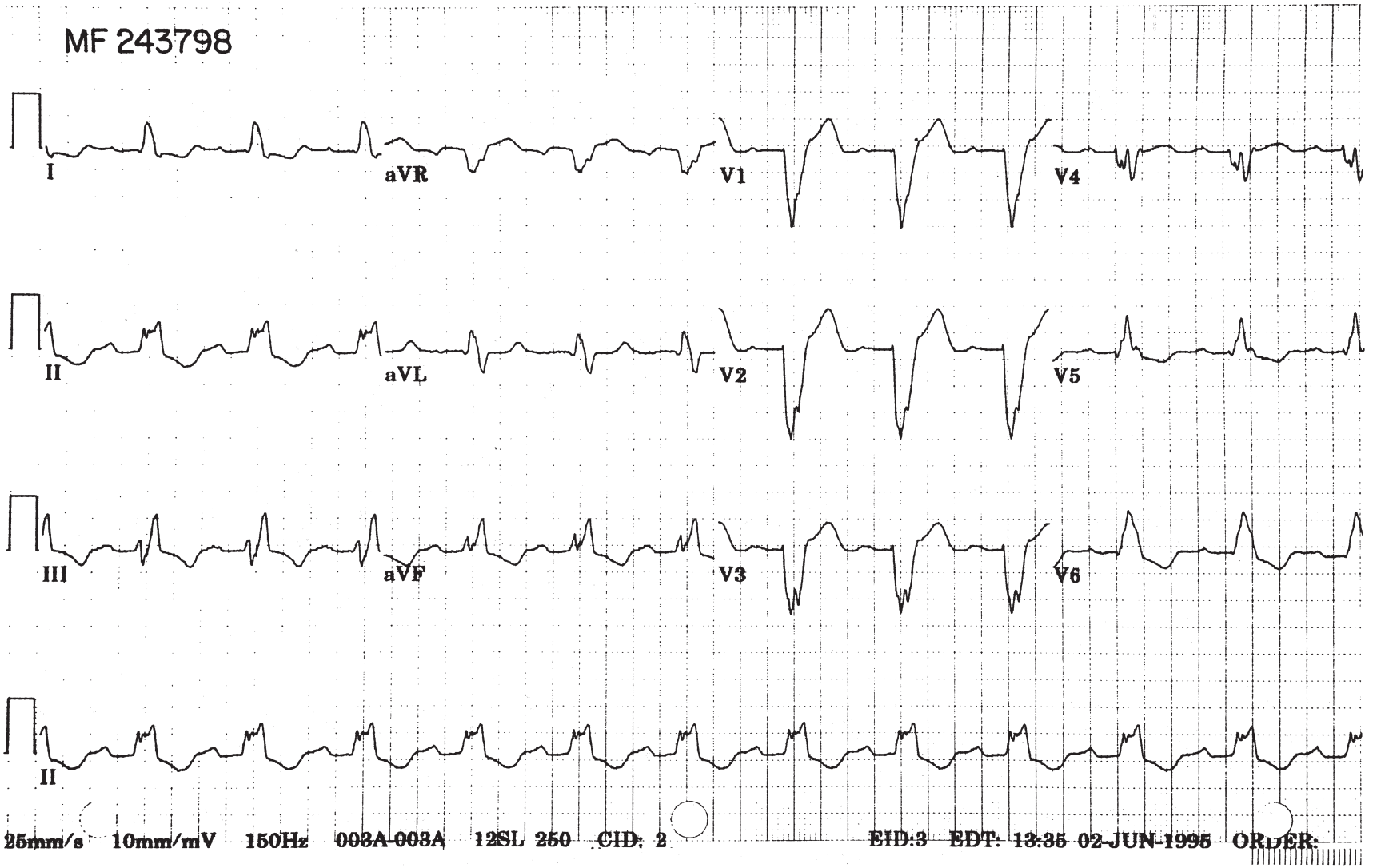
1–5 Tracing from patient described in the section “example A” (see text). H, His bundle electrogram; RB, right bundle branch electrogram.



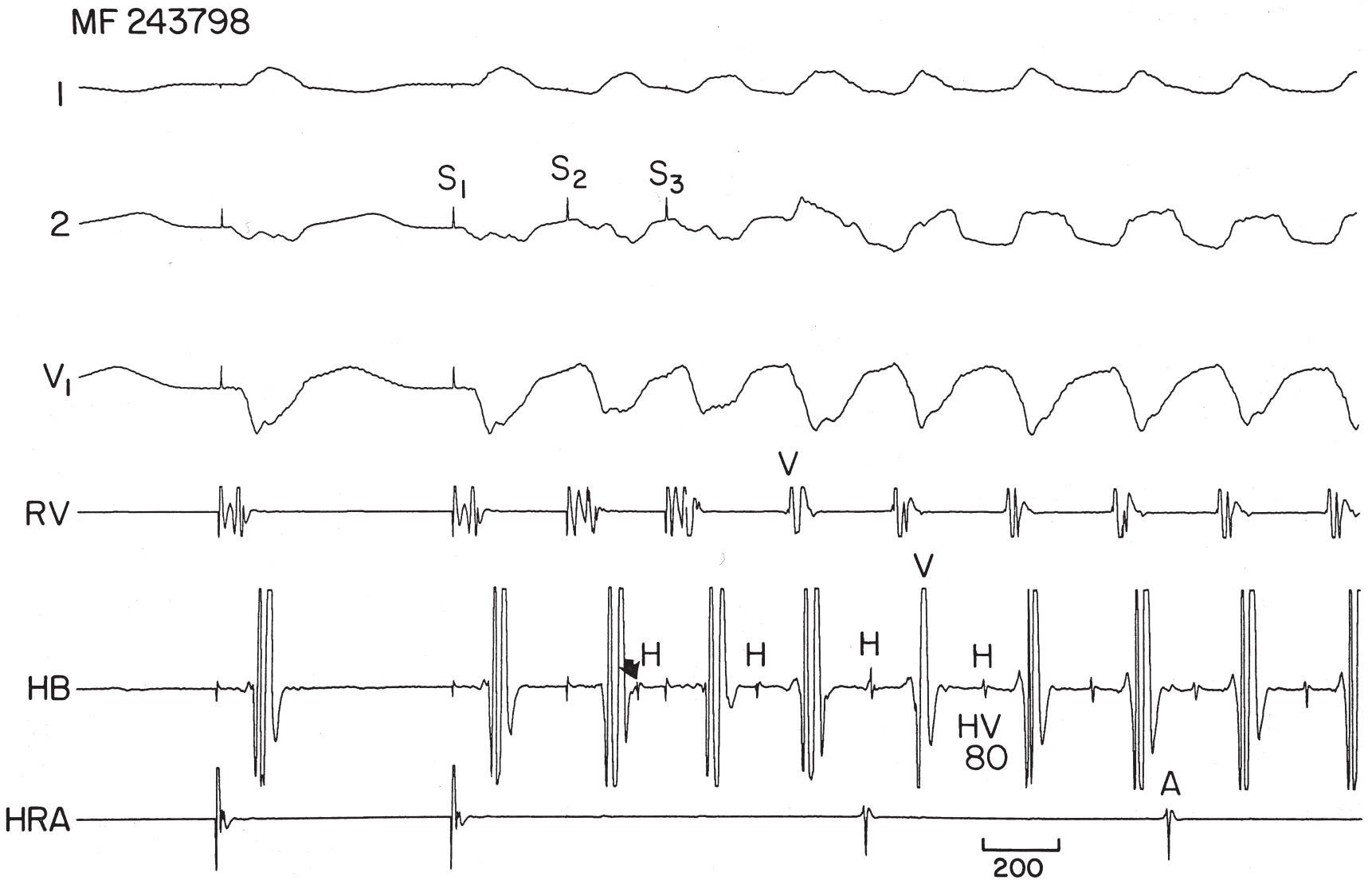
1–6 Second tracing from patient described in the section “example A” (see text).



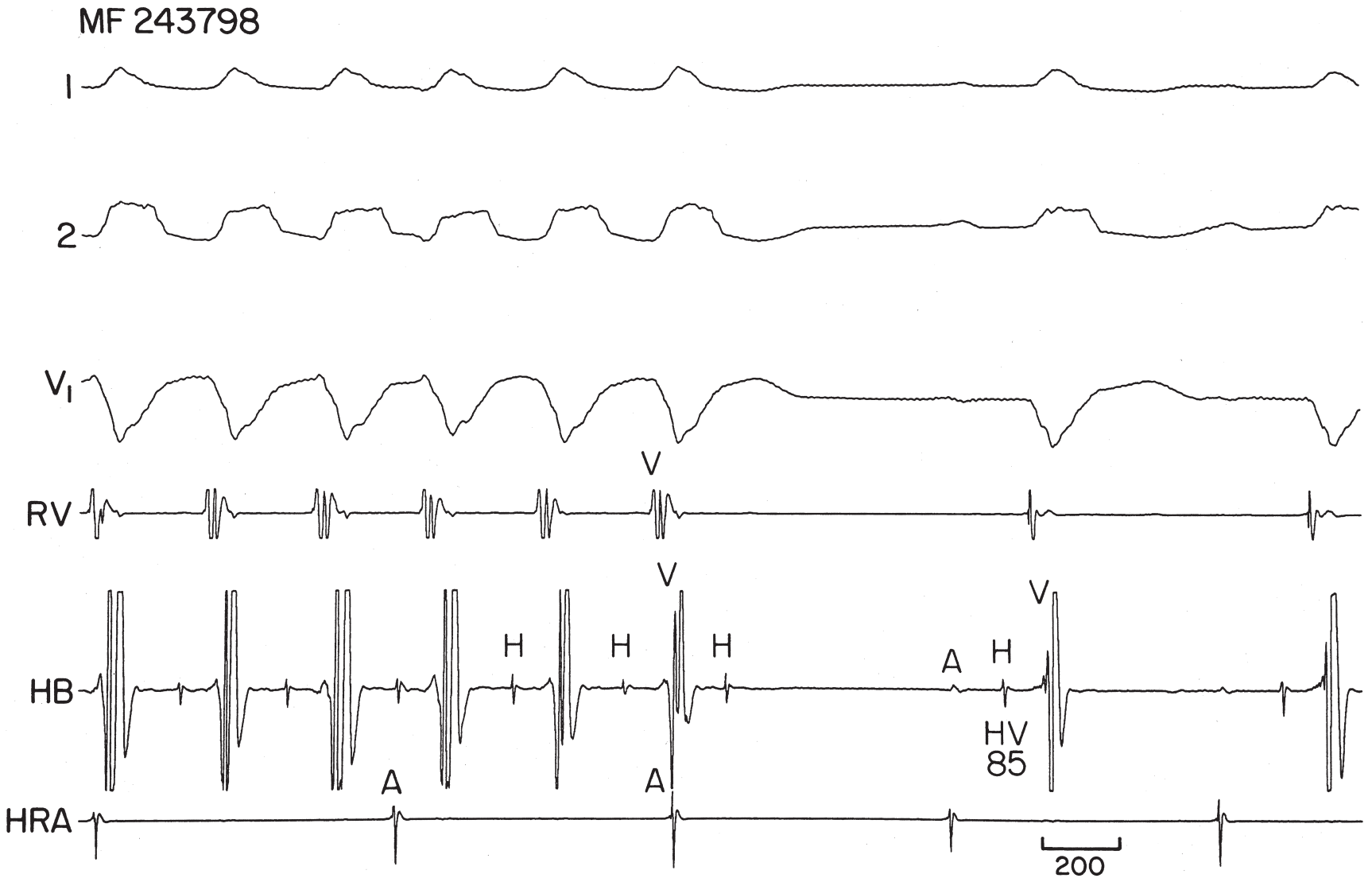
1–7 ECG during tachycardia from patient in the section “example B” (see text).



1–8 ECG during sinus rhythm from patient in the section “example B” (see text).



1–9 Induction of tachycardia in patient from the section "example B." The arrow indicates retrograde activation of the His bundle. HV, His–ventricular interval; S_1, last paced cycle of drive; S_2 and S_3, extrastimuli.



1–10 Spontaneous termination of tachycardia in patient from the section "example B."

Table 1–4 Differential Diagnosis of Wide QRS Tachycardia

- SVT with aberrancy
- VT
- Preexcited tachycardia
- Consider artifact (pseudotachycardia)
- Consider ventricular pacing

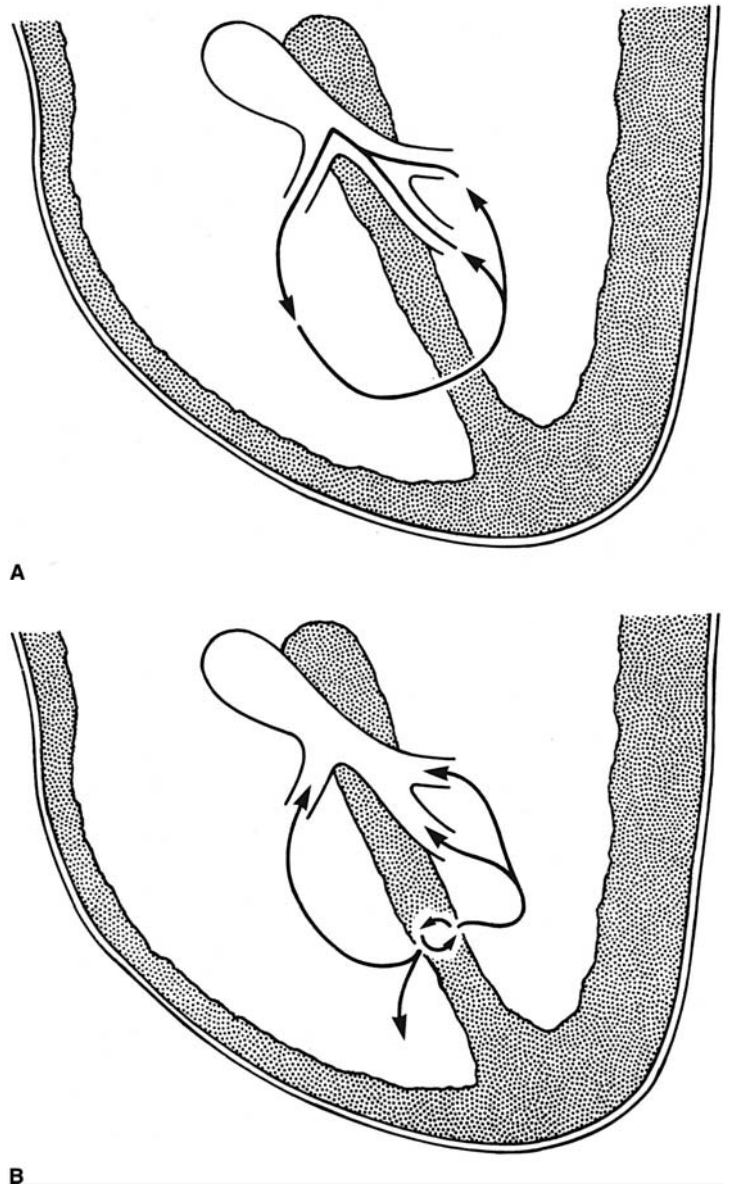
Table 1–5 Differential Diagnosis of Narrow QRS Tachycardia

- Atrial tachycardia
- AV node reentry
- AV reentry
- Junctional tachycardia
- VT (fascicular tachycardia, "septal" VT can mimic SVT)

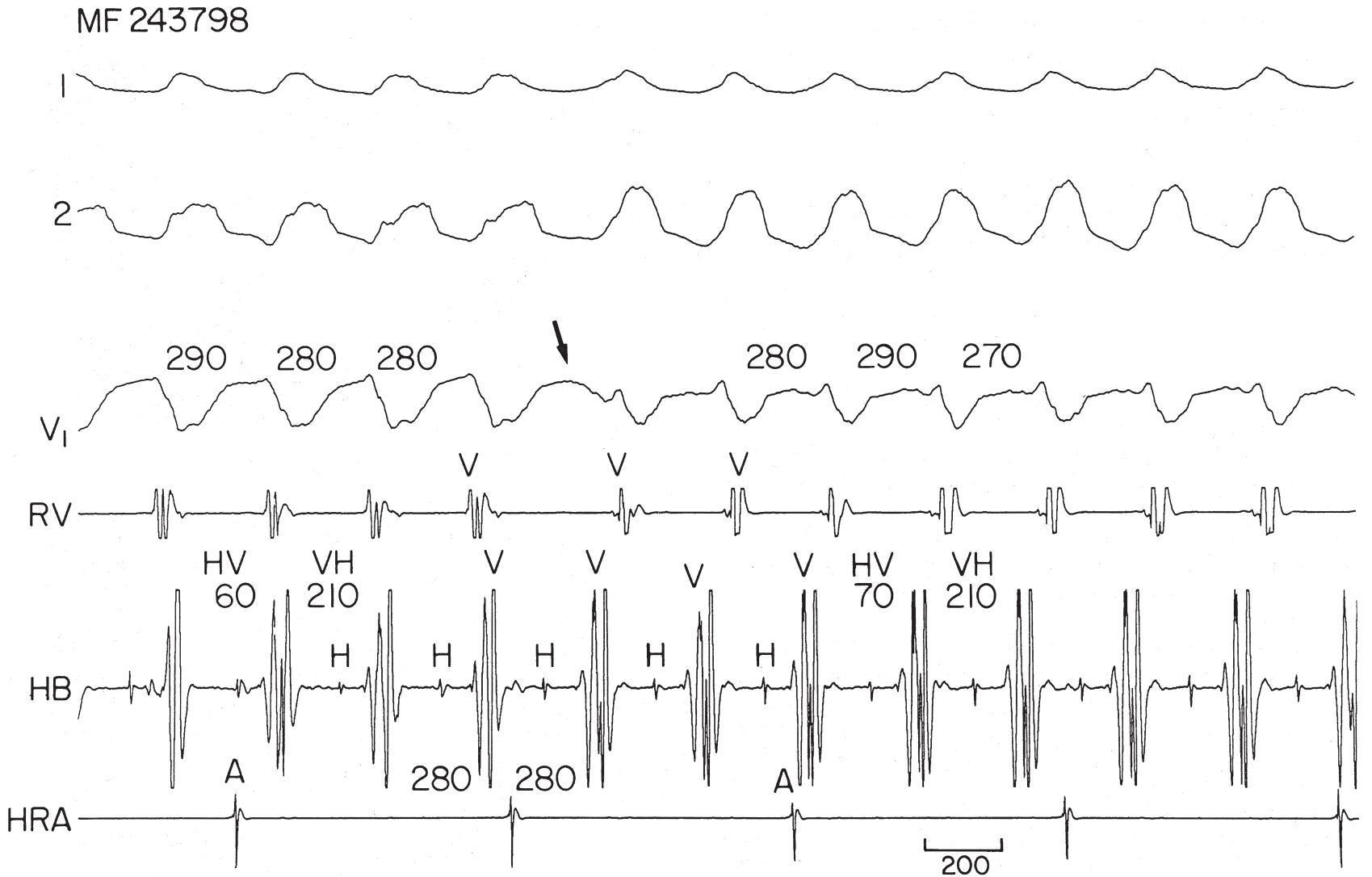
The Electrophysiology Study: An Application of Hypothesis Testing

An orderly EP study is an exercise in establishing a differential diagnosis and systematically gathering evidence to arrive at the correct one. Analysis of an unknown tracing is easier in the context of a real study where one has the advantage of building on information and applying interventions to assist the process. Nonetheless, the exercise of interpreting an unknown trace out of context is an effective learning tool. Review of an unknown EP record is fundamentally the same as reading an ECG with the advantage (and challenge) of the intracardiac recordings. An approach to this analysis is outlined in Table 1–12 and is illustrated in the following examples.

It is useful to "frame the problem" explicitly before detailed analysis. For example, if one decides from the analysis of the tracing that a tachycardia is present with no H recording when the His catheter is appropriately positioned, there are only two possibilities to consider as per Table 1–8. The tables presented provide "frames" that can be used to organize the search for the correct mechanism. During a multiple choice examination, the "frame" is provided by the choices of the examiner.



1–11 (**A**) Schematic representation of bundle branch reentry. (**B**) Intramyocardial reentry with passive activation of bundle branches. See text.



1–12 Spontaneous change in QRS during tachycardia in the same patient. VH, ventricular–His interval; cycle lengths in milliseconds.

Table 1–6 Concentric Atrial Activation Sequence During Tachycardia

- Narrow QRS complex
  - —Atrial tachycardia
  - —AV node reentry
  - —AV reentry
  - —Junctional tachycardia
- Wide QRS complex
  - —SVT with aberrancy
  - —Preexcited tachycardia
  - —VT

Table 1–7 Eccentric Atrial Activation Sequence During Tachycardia

- Narrow QRS complex
  - —Atrial tachycardia
  - —AV reentry
- Wide QRS complex
  - —Nonseptal AT with aberrancy
  - —Nonseptal AT with preexcitation
  - —Any tachycardia mechanism with retrograde activation of the atria over a left- or right-sided accessory AV pathway

Table 1–8 Regular Supraventricular Tachycardia with A–V Dissociation

- Junctional tachycardia
- AV node reentry
- Reentry utilizing normal AV conduction anterogradely, nodoventricular Mahaim pathway retrogradely
- AV reentry is *not possible*

Table 1–9 Absent H or "Short" His–Ventricular (HV) Interval During Tachycardia

- VT
- Preexcited tachycardia
- Inadequate His recording

Table 1–10 Preexcited Tachycardia: Concentric Atrial Activation

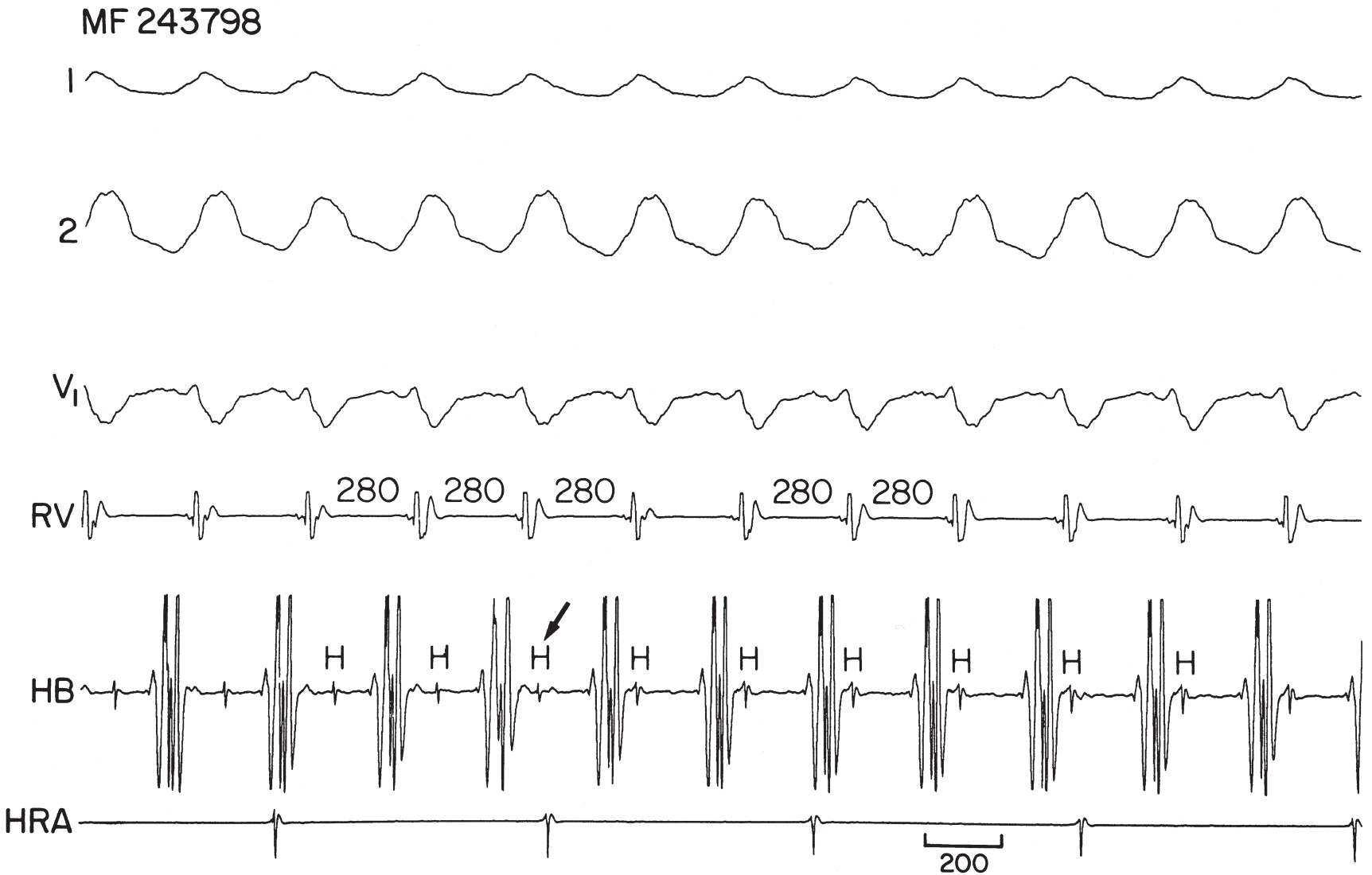
- AP part of tachycardia circuit
  - —Antidromic reentry (including atriofascicular type)
  - —AP-to-AP reentry
  - —Nodoventricular or nodofascicular reentry
- AP *not* part of tachycardia circuit ("bystander" conduction)
  - —Atrial tachycardia
  - —AV node reentry

Table 1–11 Preexcited Tachycardia: Eccentric Atrial Activation

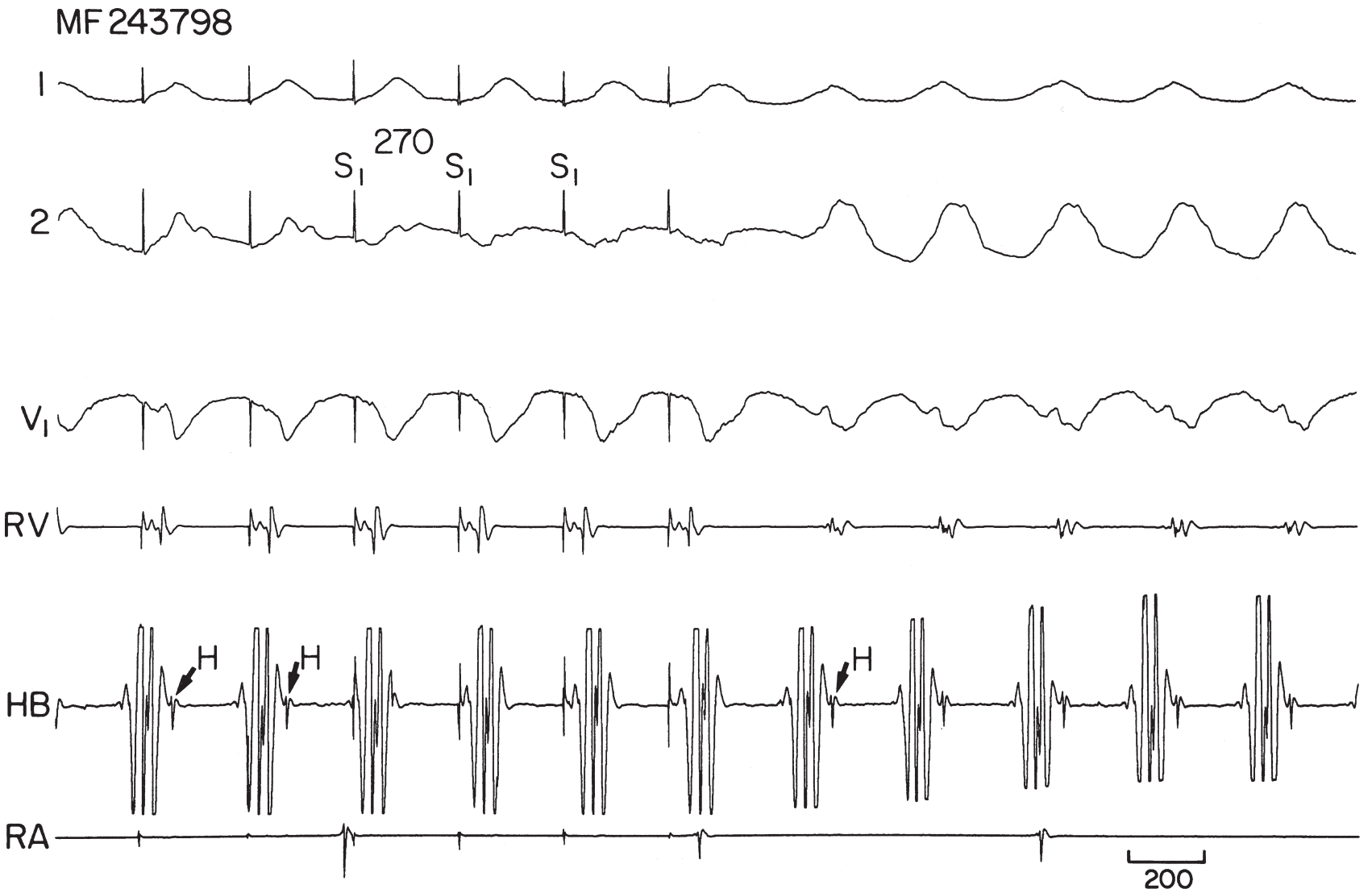
- AP part of tachycardia circuit
  - —AP-to-AP reentry
- AP *not* part of tachycardia circuit ("bystander" conduction)
  - —Right- or left-sided atrial tachycardia

Table 1–12 Approach to Unknown EP Tracing

- General overview
- Analyze the surface ECG
- Analyze the intracardiac records
- What is the *A to V relationship*?
- What is the *atrial activation sequence*?
- What is the *ventricular activation sequence* as determined from available sites?
- Is the His deflection visible and what is its relation to A and V? What are the apparent HV and VA intervals?
- Formulate a *hypothesis* and see if it explains all the observations



1–13 Spontaneous change in timing of His deflection without change in QRS or cycle length in patient from the section "example B."



1–14 Entrainment of tachycardia in patient from the section “example B.” See text.

Example A

The patient whose trace is shown in Fig. 1–5 is a young man with a recent onset of paroxysmal tachycardia.

The surface leads show the onset of a regular wide QRS rhythm with left bundle branch block (LBBB) pattern. The intracardiac records indicate two tachycardias, clearly of different mechanisms because of different rates, QRS morphology, and atrial–ventricular (A–V) relationship. The alternate hypothesis of one tachycardia mechanism with different manifestations is untenable.

The initial part of the trace has more atrial (A) electrograms than ventricular (V) electrograms with a variable A–V relationship and an atrial CL of 250 milliseconds. This can realistically be only atrial flutter.

The *transition zone* is marked by the arrow, the last flutter cycle. This is followed by a normal QRS that heralds the onset of the second tachycardia. The latter has LBBB morphology and an apparent 1:1 A–V relationship. It is not clear whether the atria are driving the ventricles, whether the ventricles are driving the atria, or whether their relationship is reciprocal.

Atrial activation on the available leads begins at the proximal coronary sinus (CS) recording electrodes positioned near the orifice of the CS. This sequence is not discriminating, being compatible with atrial tachycardia, retrograde conduction over a "slow" AV node pathway, and retrograde conduction over an AP (see Table 1–6). The normal His–ventricular (HV) relationship argues against ventricular tachycardia (VT) or preexcited tachycardia (see Table 1–8).

The differential diagnosis at this point includes atrial tachycardia, AV reentry, and AV node reentry. AV reentry is favored over AV node reentry because there is only modest AH prolongation at the onset and the VA interval is too long for the most common type of AV node reentry. More compellingly, the apparent VA interval prolongs by 60 milliseconds with the development of LBBB aberration after the first tachycardia cycle, a situation only compatible with AV reentry utilizing a left lateral AP as part of the circuit (i.e., the LBB is part of the circuit).

The remaining hypothesis of atrial tachycardia is not supported by the mode of onset or the apparent VA intervals, but is not yet ruled out entirely. It is important to remember that the first atrial complex of the second tachycardia could have fortuitously started shortly after the last narrow QRS complex. Introduction of a relatively late-coupled PVC into the cardiac cycle at the time of His bundle refractoriness (Fig. 1–6) advances the next atrial cycle and terminates the tachycardia, verifying the existence of an AP and, for all practical purposes, the diagnosis of AV reentry. Advancement of the A ("reset") with a relatively long coupling interval of 380 milliseconds with tachycardia CL of 420 suggests very easy "access" of the RV apical site to the tachycardia circuit, which would normally not be the case with a left-sided AP. However, with LBBB the right bundle branch is now in the circuit with orthodromic AV reentry over a left lateral pathway as the retrograde limb (see Fig. 1–3).

We do appreciate that the patient could theoretically have a concealed AP near an atrial tachycardia focus, with the PVC preexciting the atrium over the AP and terminating the atrial tachycardia. However, one does not need to look for zebras in a herd of horses!

Example B

The patient is a 74-year-old man with a history of inferior and anterior myocardial infarction (MI) and coronary artery bypass grafting. He experiences the sudden onset of rapid heart beating while watching television, and the ECG recorded subsequently in the emergency room is shown in Fig. 1–7. The arrhythmia stops spontaneously and the ECG recorded in sinus rhythm shows LBBB (Fig. 1–8). Any wide QRS tachycardia in such a patient is presumptively VT, and this is supported by the suggestion of AV dissociation in the rhythm strip (*arrow* Fig. 1–7). However, the QRS during tachycardia is very similar, but not identical, to the QRS in sinus rhythm. VT with a QRS similar to the QRS during sinus rhythm may be seen with bundle branch reentry or VT originating in the His bundle (yes, the His bundle is a ventricular structure). Alternatively, VT originating in the fascicular system or adjacent myocardium could be expected to break out at the same site as the sinus impulse in the presence of bundle branch block. Thus, the EP study is begun with a differential diagnosis of bundle branch reentry or fascicular VT high on the list.

At EP study, tachycardia is induced with two ventricular extrastimuli (Fig. 1–9) and terminates spontaneously (Fig. 1–10). AV dissociation is now clearly evident and the QRS is again similar to that in sinus rhythm 0. The tachycardia begins with prolongation of the

retrograde His bundle (H) and the HV during tachycardia is similar to that in sinus rhythm. This is most compatible with bundle branch reentry as illustrated in Fig. 1–11*A*, although the alternative hypothesis (Fig. 1–11*B*) is not disproved. The diagnosis is further supported by the ventricular activation sequence that shows very early activation of the right ventricular apex (near the terminus of the right bundle branch). Spontaneous termination with a retrograde H is compatible with either hypothesis.

Is there enough evidence to proceed with ablation of the right bundle branch? A further induction of tachycardia is pursued (Fig. 1–12) and a spontaneous transition to another QRS morphology (*arrow*) is observed. In spite of a clear change in the QRS (although still LBBB morphology) and ventricular activation sequence (the RV apex is now very late), the tachycardia rate and the H electrogram are unchanged! Consider the two hypotheses in Fig. 1–11. The evident loss of anterograde right bundle activation (as assumed by the relatively late activation of the right ventricular apical electrogram) was not critical to maintenance of tachycardia and the right bundle was clearly a bystander. Further observation of the tachycardia (Fig. 1–13) illustrates another transition (*arrow*). The ventricular–His (VH) interval shortens to a new steady state and again the tachycardia rate remains unchanged, oblivious to activity in the H. It is now obvious that the H and right bundle are passive bystanders and the tachycardia is best explained by myocardial reentry with passive activation of the bundle branches (Fig. 1–11*B*).

What if we were not fortunate enough to see the phenomena in Figs. 1–12 and 1–13? Entraining VT by pacing the right ventricular apex (Fig. 1–14) at a CL only 20 milliseconds shorter than the VT CL advances the retrograde H by 50 milliseconds or more, dynamically dissociating the H from the tachycardia circuit.

Two other fundamental observations are important. First, careful measurement will reveal that the VT CL is about 310 milliseconds with minimal irregularity. The "PPI" at the RV apical pacing is approximately 420 milliseconds. Since this electrode is near the RBB terminus, one would expect the PPI in bundle branch reentry to approximate the tachycardia CL. In this instance, the PPI is clearly "out" of the circuit making bundle branch very unlikely. Entrainment is exceedingly useful to diagnose or exclude bundle branch reentry when the tachycardia is stable enough to permit it.

A final "subtle" observation: by careful measurement, you will find a small prolongation of the CL of the tachycardia after the first two beats after the cessation of entrainment pacing. You will find that the V to V interval prolongs slightly and the subsequent H to H interval follows it. That is, the ventricular muscle change *precedes* the H to H change suggesting again that the His bundle is reacting *passively* to changes in mechanism elsewhere. It is always useful to watch for oscillations or "wobble" in the CL to decide who is leading and who is following!

The unknown tracings in the following chapters provide an opportunity to practice these principles. A question after each trace is intended to focus attention on the intended point of interest, although the tracings usually provide other lessons. Measuring calipers and a clear right angle for vertical alignment are recommended. Finally, there may well be alternative explanations for phenomena to the ones suggested. Dr Charles Fisch once responded to a contrary student at an ECG course by saying that his explanation was correct because it was his slide. We can say it no better.

Chapter 2

Electrophysiologic Approach to the ECG

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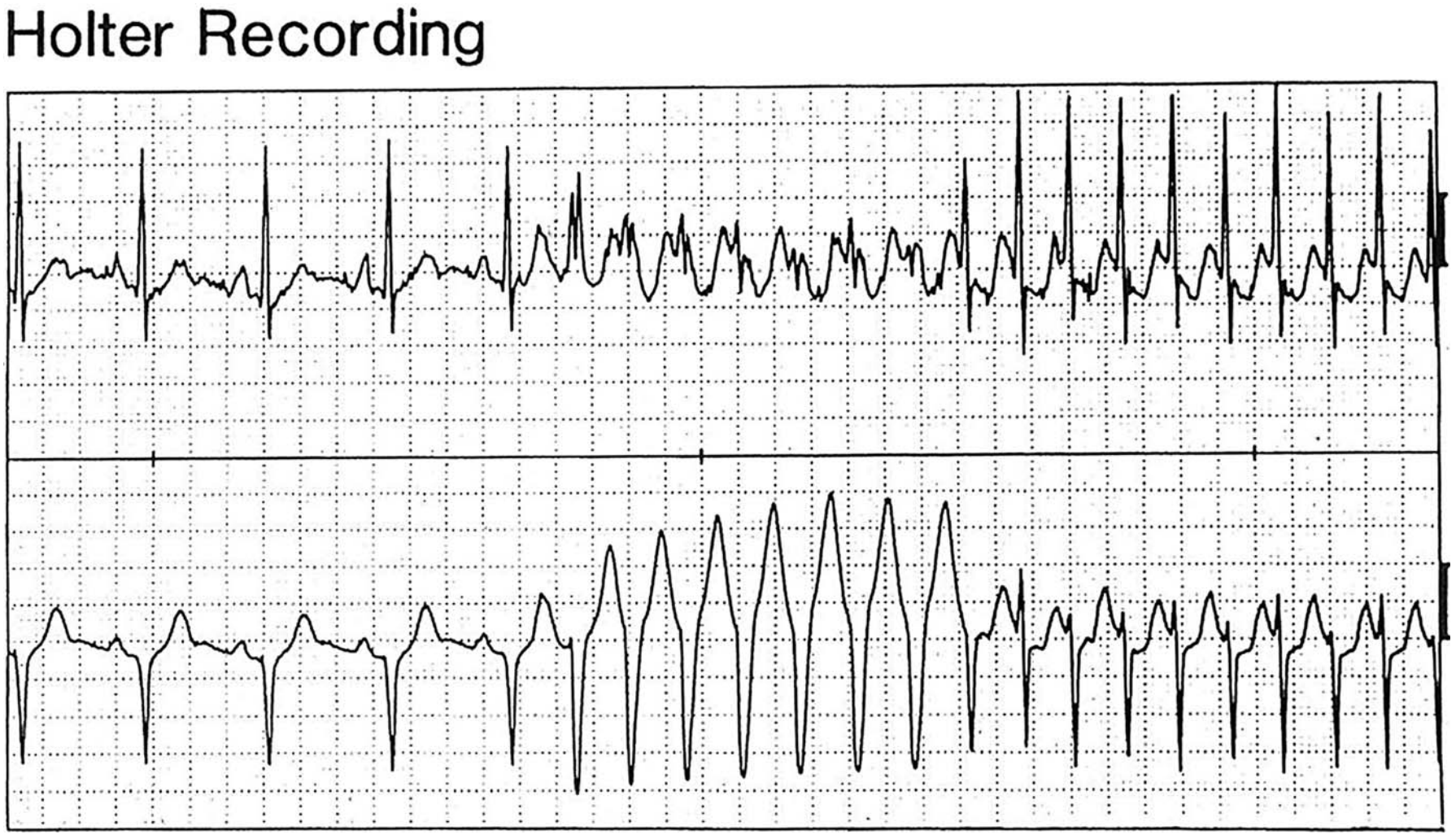
Figure 2–1



This recording was obtained in a 25-year-old woman with a history of palpitations and dizziness. What is the mechanism of tachycardia(s)?

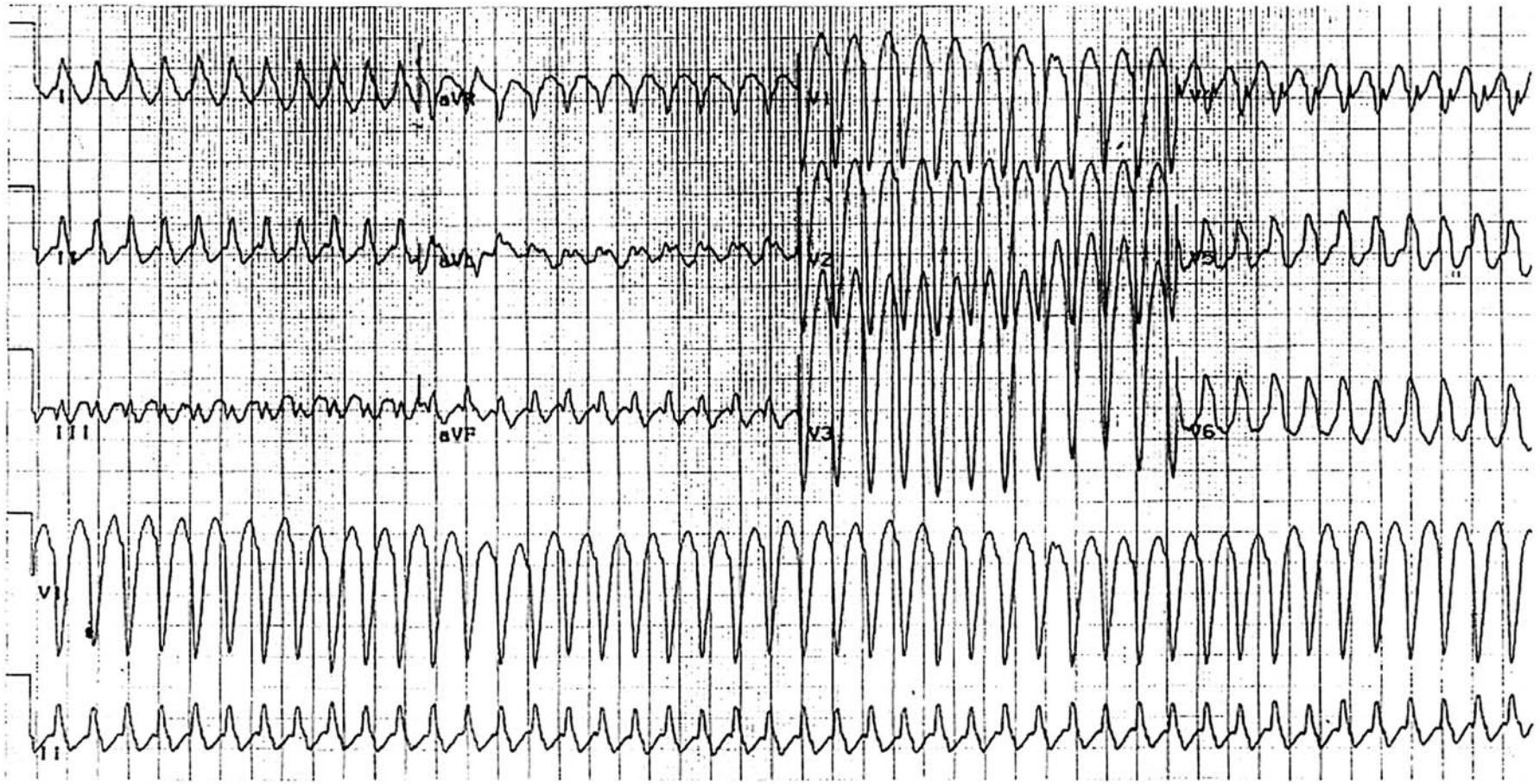
Explanation:

This figure demonstrates the transition from a wide QRS complex tachycardia to a narrow QRS complex tachycardia. The first question is whether the patient has supraventricular tachycardia with aberrancy or ventricular tachycardia with a transition to a supraventricular tachycardia. Typically, one would not expect the tachycardia rate to increase with the disappearance of aberrancy and careful measurement of the wide QRS complex tachycardia shows that it has a longer cycle length than the subsequent narrow QRS tachycardia. Does this mean that the wide complex tachycardia is ventricular tachycardia and it somehow induces a supraventricular tachycardia?

Evidence to support that this is a supraventricular tachycardia can be found in careful analysis of the T wave just preceding the onset of the arrhythmia. Note that this T wave shows a peaked contour compared with the preceding T waves and this strongly suggests that a P wave is inscribed on the T wave. This is most compatible with the onset of a supraventricular tachycardia. If the bundle branch system were used in supraventricular tachycardia, one might indeed anticipate a shorter cycle length with the disappearance of the bundle branch block. This is characteristic of AV reentry (AVRT) utilizing an accessory pathway for retrograde conduction. In such an instance, the cycle length will prolong in approximately 85% of patients who have a bundle branch block occurring on the side of the accessory pathway, in this instance a left-sided accessory pathway with left bundle branch block (LBBB) aberrancy. This is because of an increase in the circuit time reflected in the ventriculoatrial interval due to transseptal conduction time from the right to left ventricle in the presence of LBBB. The disappearance of the LBBB will shorten the reentrant circuit by allowing the left side of the heart to be activated sooner and thus shorten the tachycardia cycle length.

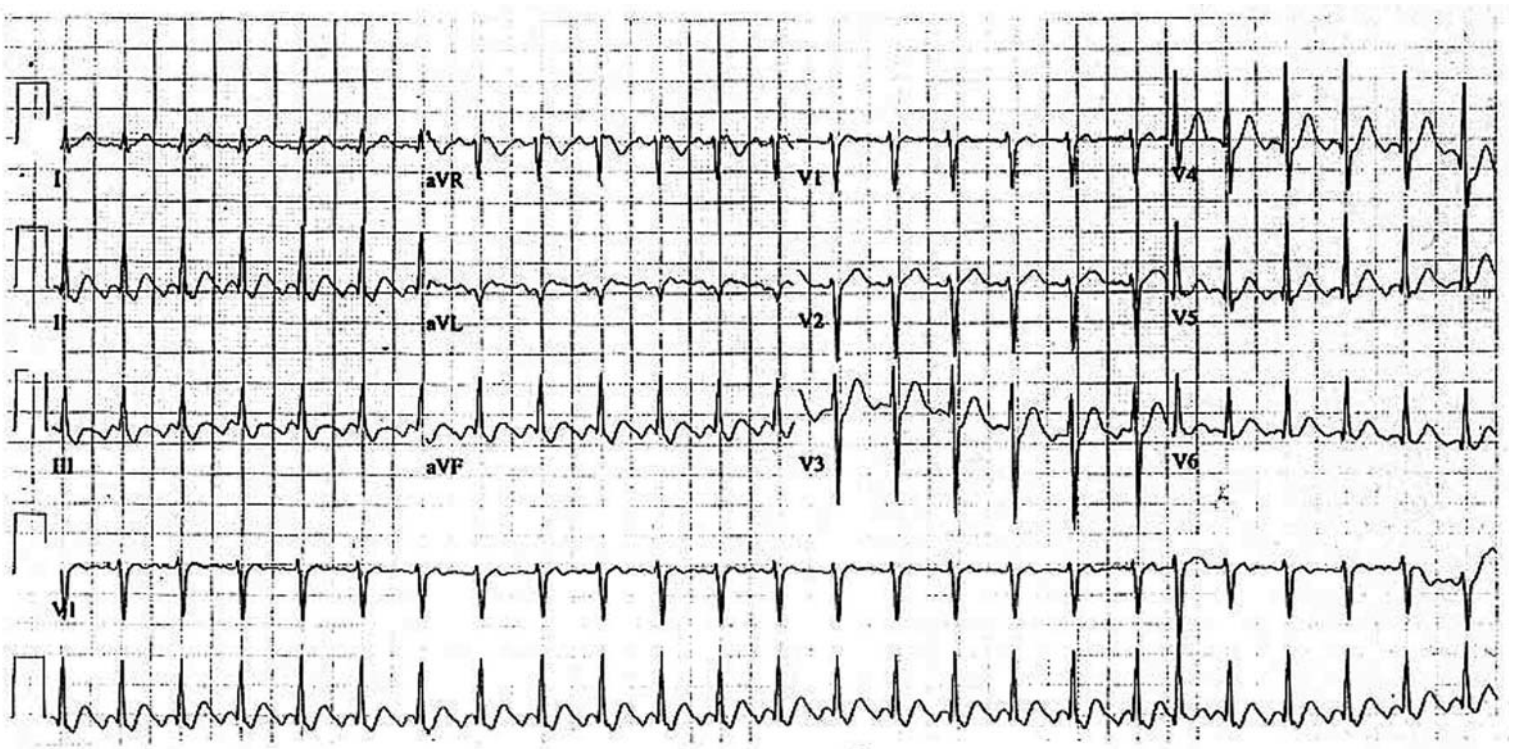
This patient had a concealed left free wall accessory pathway that was used in AVRT that was successfully ablated.

Figure 2–2A



This 68-year-old woman is receiving propafenone to treat atrial fibrillation. She is otherwise well. She developed near syncope and this electrocardiogram was obtained (Fig. 2–2*A*). What is the diagnosis?

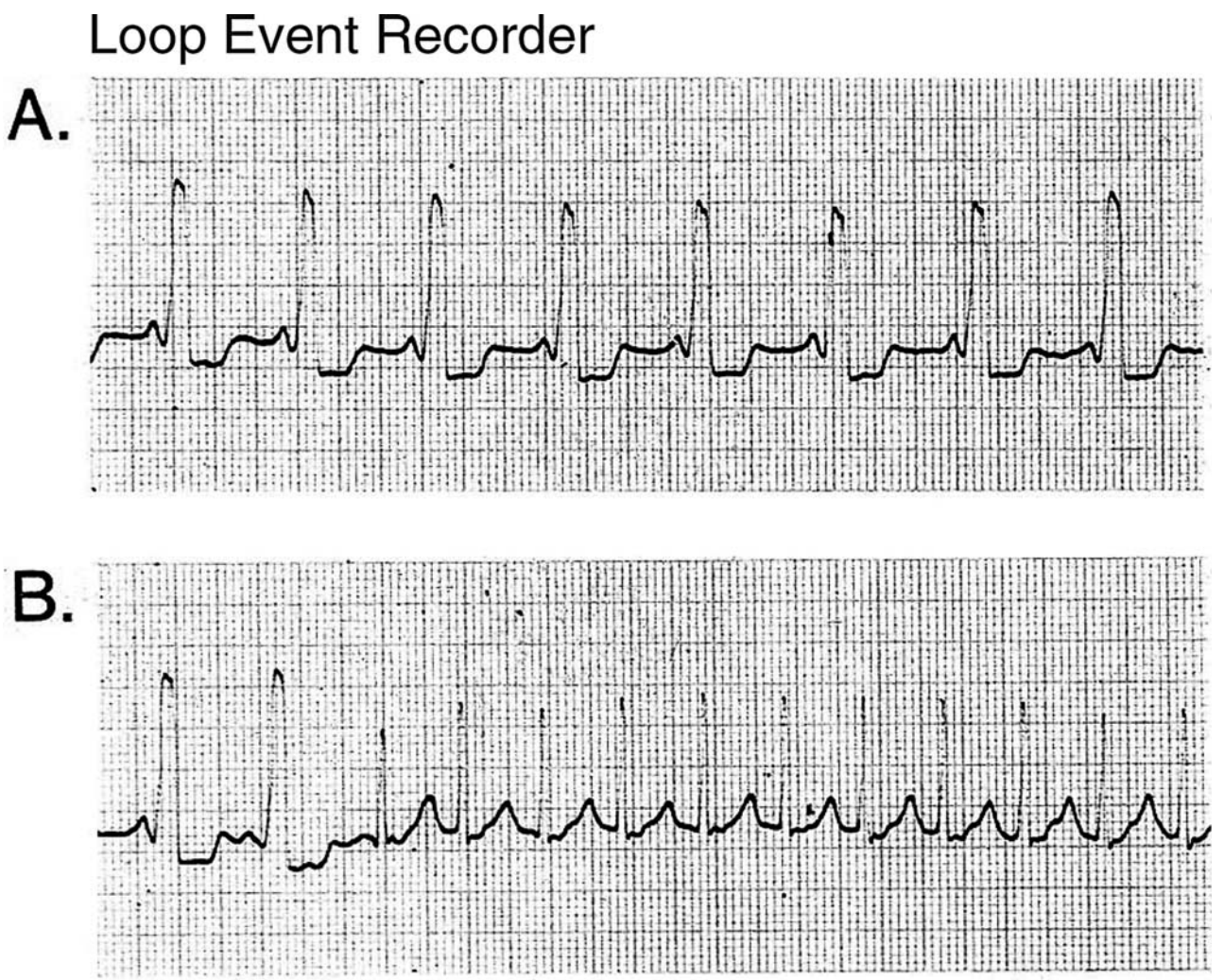
Figure 2–2B



Explanation:

The 12-lead electrocardiogram shows a regular tachycardia with LBBB morphology and a cycle length of approximately 220 milliseconds. While not "classic" for a typical LBBB pattern, the QS complex appears to be more typical than atypical for LBBB. Note the lack of a Q wave in ECG leads 1 and aVL and a rather rapid downstroke of the initial portion of the QRS in the early precordial leads. Figure 2–2*B* demonstrates the mechanism of tachycardia. Because the patient was stable in the emergency room setting, the treating physician administered intravenous verapamil with the assumption that this was a supraventricular tachycardia. As an aside, this should not be done, of course, when VT remains in the differential diagnosis as it surely is in this case. Regardless, the mechanism of tachycardia was revealed when this was performed and the patient has atrial flutter shown well in ECG leads 2, 3, and aVF. Note also that after block the slower tachycardia has half the ventricular rate of the wide QRS tachycardia. Agents such as propafenone and flecainide are well known to allow the emergence of atrial flutter in patients with atrial fibrillation and 1:1 AV node conduction can occur if an AV node blocking agent is not present. This can lead to a cardiac arrest, which fortunately did not occur here.

Figure 2–3

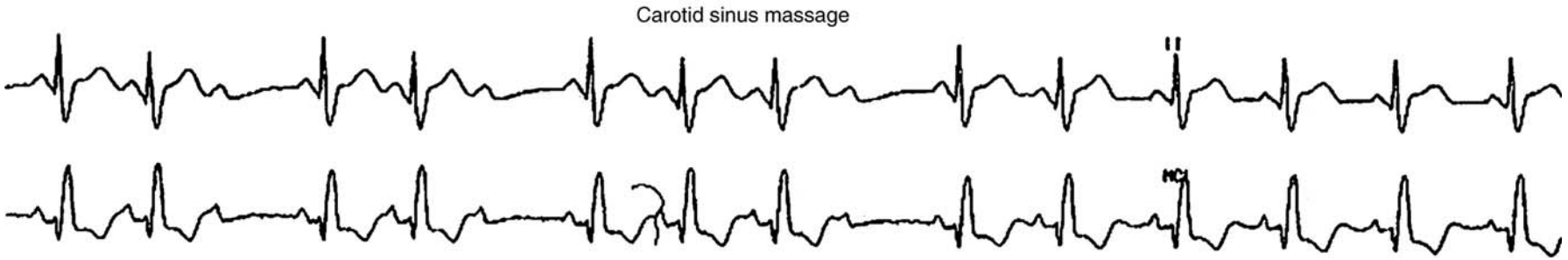


A 48-year-old woman with a history of palpitations for several years was prescribed a loop event recorder to correlate the ECG with her symptoms. What is the mechanism of her tachycardia?

Explanation:

In the ECG rhythm strip designated A, note sinus rhythm with a short PR interval and a broad QRS complex consistent with ventricular preexcitation. The onset of tachycardia in B is shown in the lower rhythm strip. Careful analysis of the ST segment of the second sinus complex demonstrates a deformation that is most likely a P wave that results in a narrow QRS complex and the onset of tachycardia. Note that this premature atrial complex initiates tachycardia not only with loss of preexcitation but also with a markedly prolonged PR interval of approximately 360 milliseconds. This almost surely represents conduction over a slow AV nodal pathway. There is a P wave noted just at the end of the QRS complex during tachycardia. While this could be AV reentry with retrograde conduction over an accessory pathway and anterograde conduction over a slow AV nodal pathway, the V to retrograde P interval is very short (approximately 80–100 milliseconds) and this would be "borderline" for retrograde conduction over an accessory pathway. In this instance, the mechanism identified at EP study and ablation was slow–fast AV node reentry. The accessory pathway was not capable of retrograde conduction, and it was just by chance that a PAC blocked anterograde conduction over the accessory pathway while starting AV node reentry.

Figure 2–4

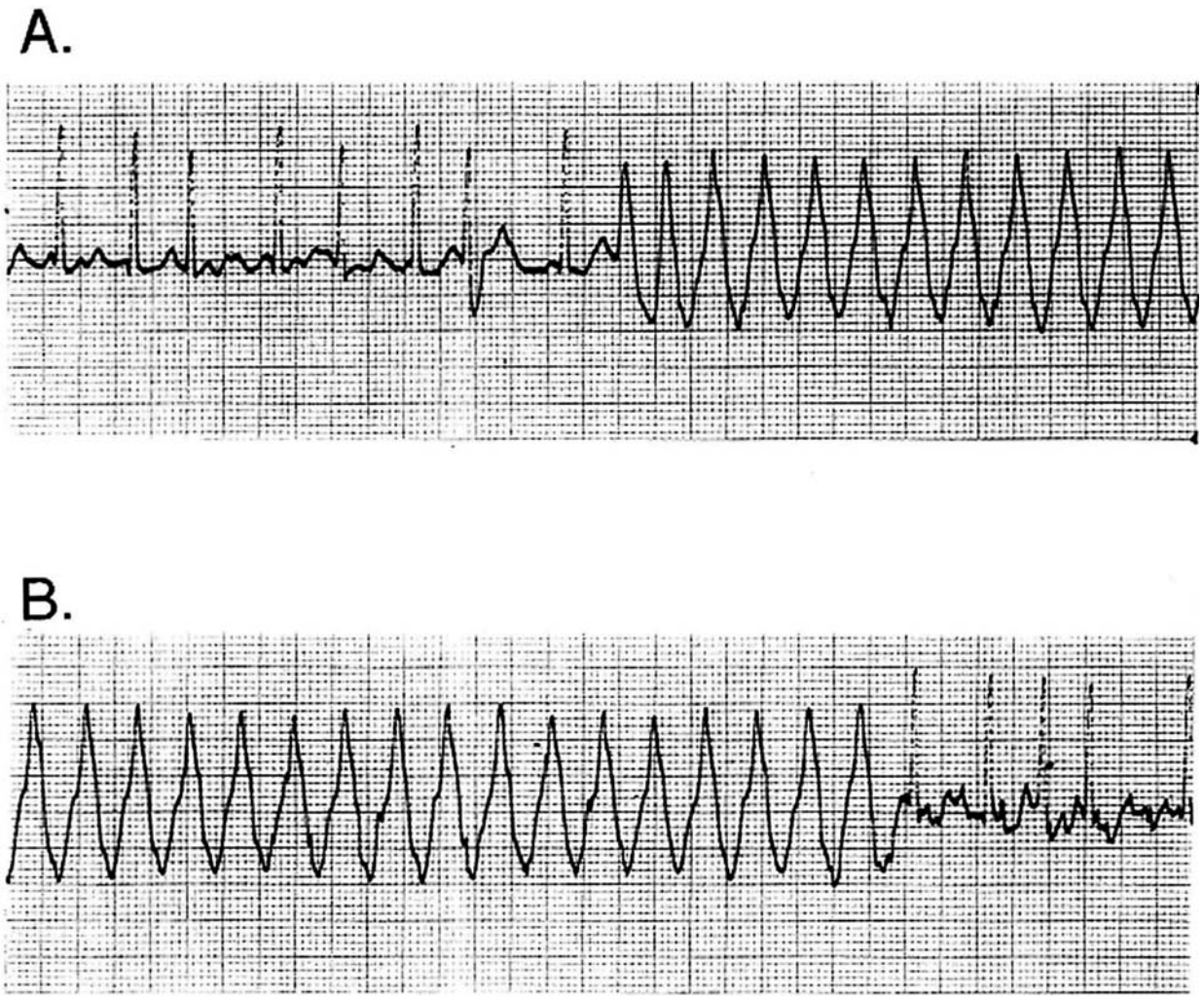


You are consulted on a 69-year-old man who is in the early convalescent phase after aortic valve surgery. He is asymptomatic but the nurse noted intermittent heart block and requested a consultation. What is the most likely mechanism of heart block?

Explanation:

This patient demonstrates sinus rhythm with RBBB QRS morphology and a Mobitz 1 Wenckebach sequence on the left-hand side of the tracing (Fig. 2–4). The PR interval does prolong slightly before the third P wave in the sequence blocks. Carotid sinus massage was performed on the right side of the rhythm strip, and the sinus cycle slows with resumption of 1:1 AV conduction. These findings indicate block in the His–Purkinje system and not in the AV node. The relatively short PR interval at the start of the Wenckebach sequence, a small PR interval increase prior to the nonconducting P wave, and a relatively fast sinus rate are consistent with His–Purkinje pathology and block. Slowing of the rate with carotid massage allows resumption of 1:1 AV conduction, contrary to what one would expect if block were in the AV node. This patient underwent electrophysiology study where infra-Hisian block was confirmed and a pacemaker was implanted.

Figure 2–5



A 55-year-old woman with a recent history of palpitations and near syncope is admitted for observation. During continuous monitoring, the following arrhythmia occurs (Fig. 2–5). What is the diagnosis?

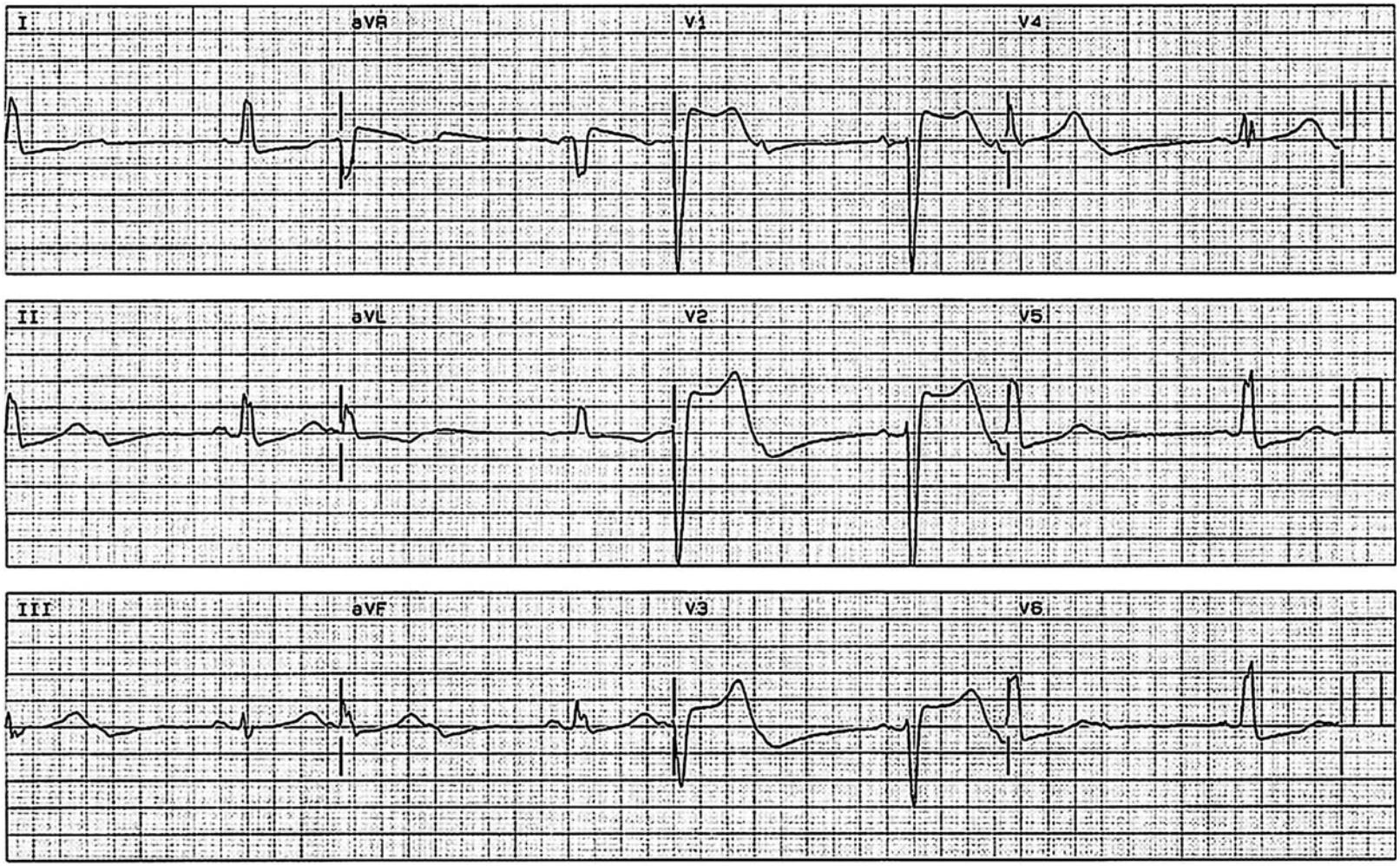
Explanation:

In the upper rhythm strip (A), note that there is an irregular narrow QRS complex rhythm with P waves that are variable. The seventh QRS complex demonstrates an intermediate QRS morphology compared with the wide QRS complexes that are seen on the right-hand portion of ECG strip A as well as the left-hand portion of the bottom strip (B). The differential diagnosis is ventricular tachycardia, a preexcited tachycardia, or supraventricular tachycardia with aberrancy.

Several features strongly suggest aberrancy. First, there is a relatively long–short interval preceding the incomplete BBB of the seventh QRS complex. This is followed by an even longer pause and a short interval, now with a persistent wide QRS complex noted. The cycle length is mostly regular with some slight variability. The RR intervals of the wide complex tachycardia and the P–P intervals noted at the end of the rhythm strip in B are nearly identical. This suggests SVT with aberrancy during 1:1 AV conduction.

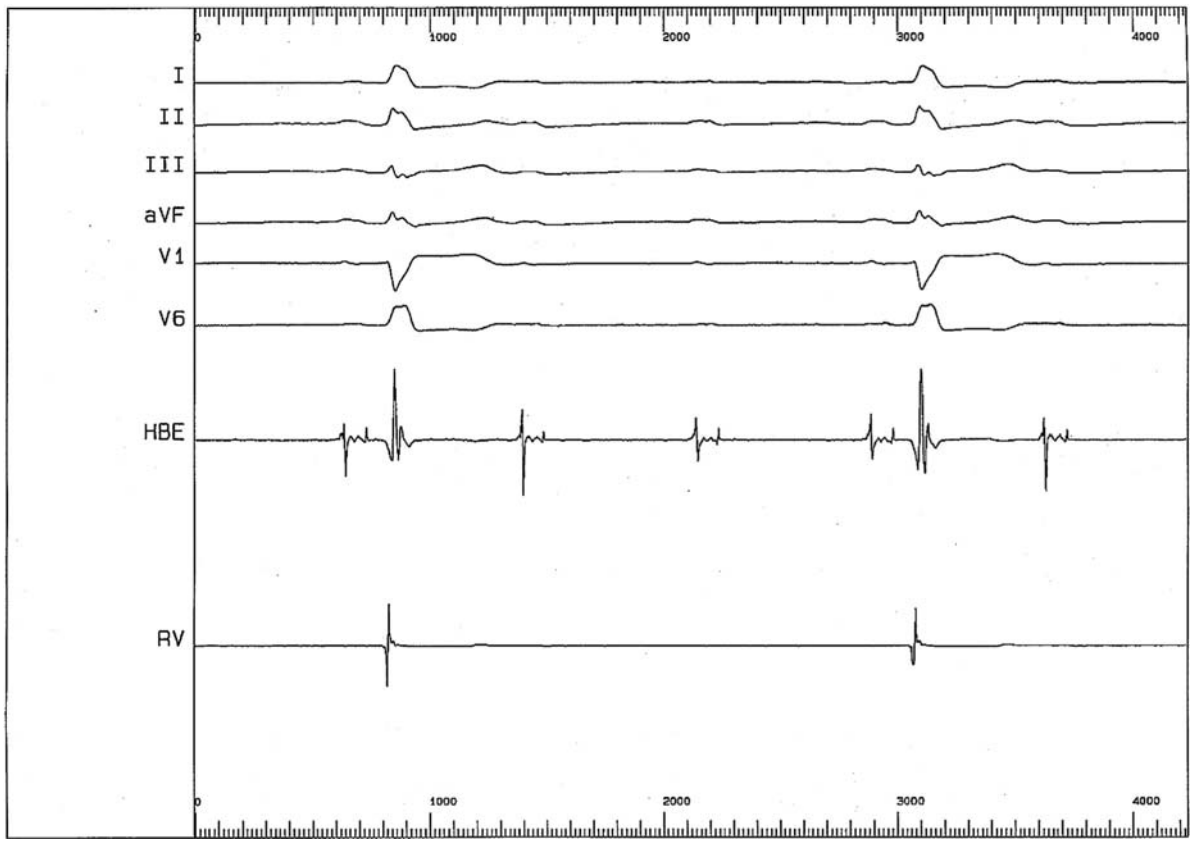
Most compellingly, there is a relatively short interval between the last BBB complex and the first narrow complex seen on the right-hand side of strip B. This interval is approximately 280 milliseconds, and it would be extremely unusual for a supraventricular complex to conduct so quickly after termination of ventricular tachycardia, because ventricular tachycardia produces retrograde concealed conduction into the AV node preventing subsequent rapid supraventricular conduction. The seventh complex might be considered partially preexcited, suggesting preexcited tachycardia, but the initial, not the latter, part of the ECG complex should have reflected this. This patient did have supraventricular tachycardia with aberrancy.

Figure 2–6A



A 75-year-old man with a slow pulse is sent to you for consultation. He is totally asymptomatic and his rhythm is recorded on a 12-lead electrocardiogram in your office, shown here (Fig. 2–6*A*). What is the diagnosis?

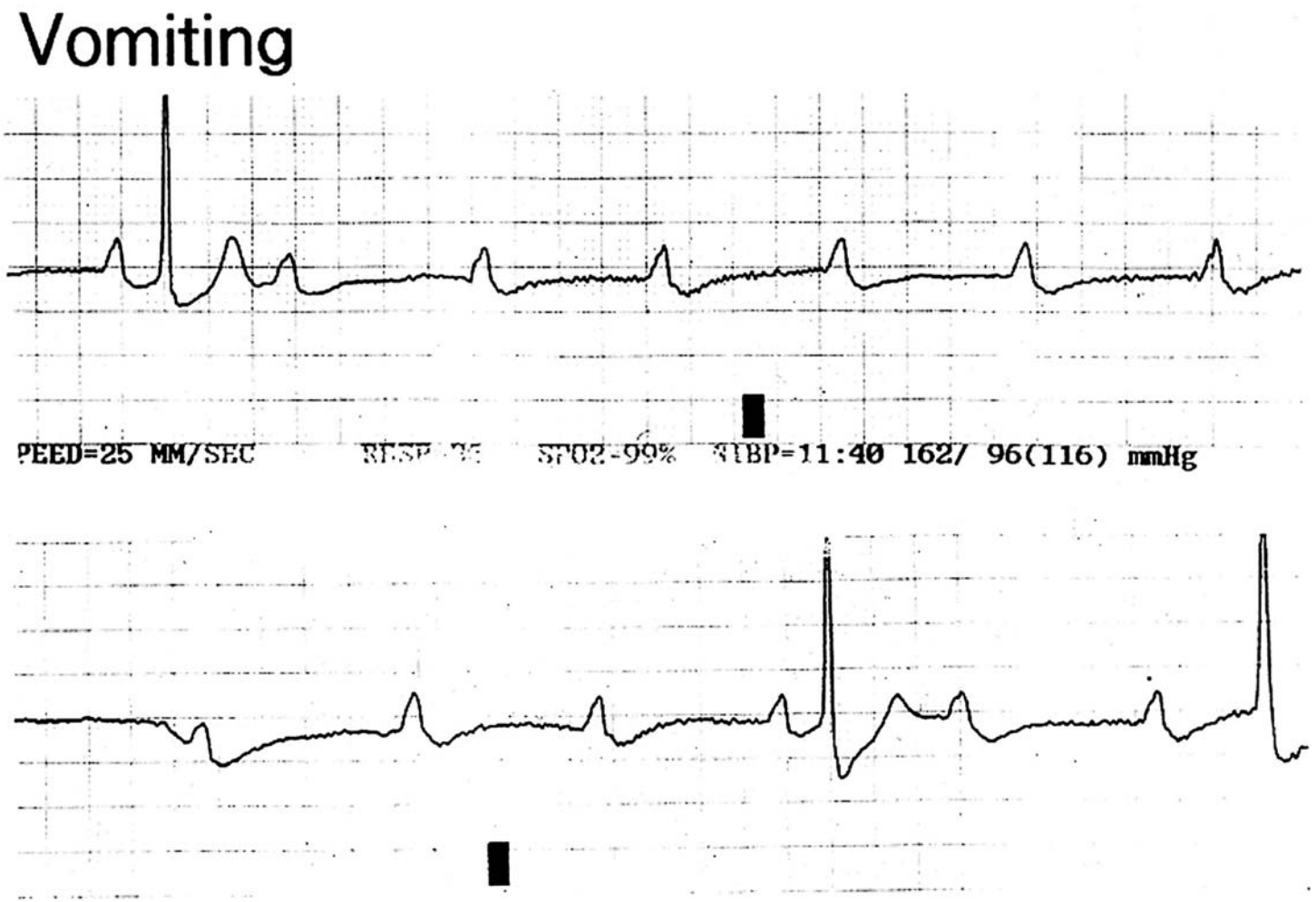
Figure 2–6B



Explanation:

The ECG demonstrates sinus rhythm with apparent Mobitz 2 AV block and is a little confusing due to the lead changes. Nonetheless, the conducted QRS complexes have LBBB morphology with a PR interval just at the upper limit of normal. The site of block can be reasoned by "simply doing the math." Conduction from the sinus node to the ventricle involves the PA interval, which is the time from the sinus node to the AV nodal area, the AH interval, and the HV interval. If we estimate a PA interval of approximately 40 milliseconds and an HV interval of 50 milliseconds, that would leave approximately 100 milliseconds for AV node conduction. It would be rare for normal conduction through the AV node to be associated with block. Thus, the most likely site of block is within the His–Purkinje system. As shown in Fig. 2–6*B*, infra-His block was present at electrophysiologic study and the patient received a pacemaker.

Figure 2–7

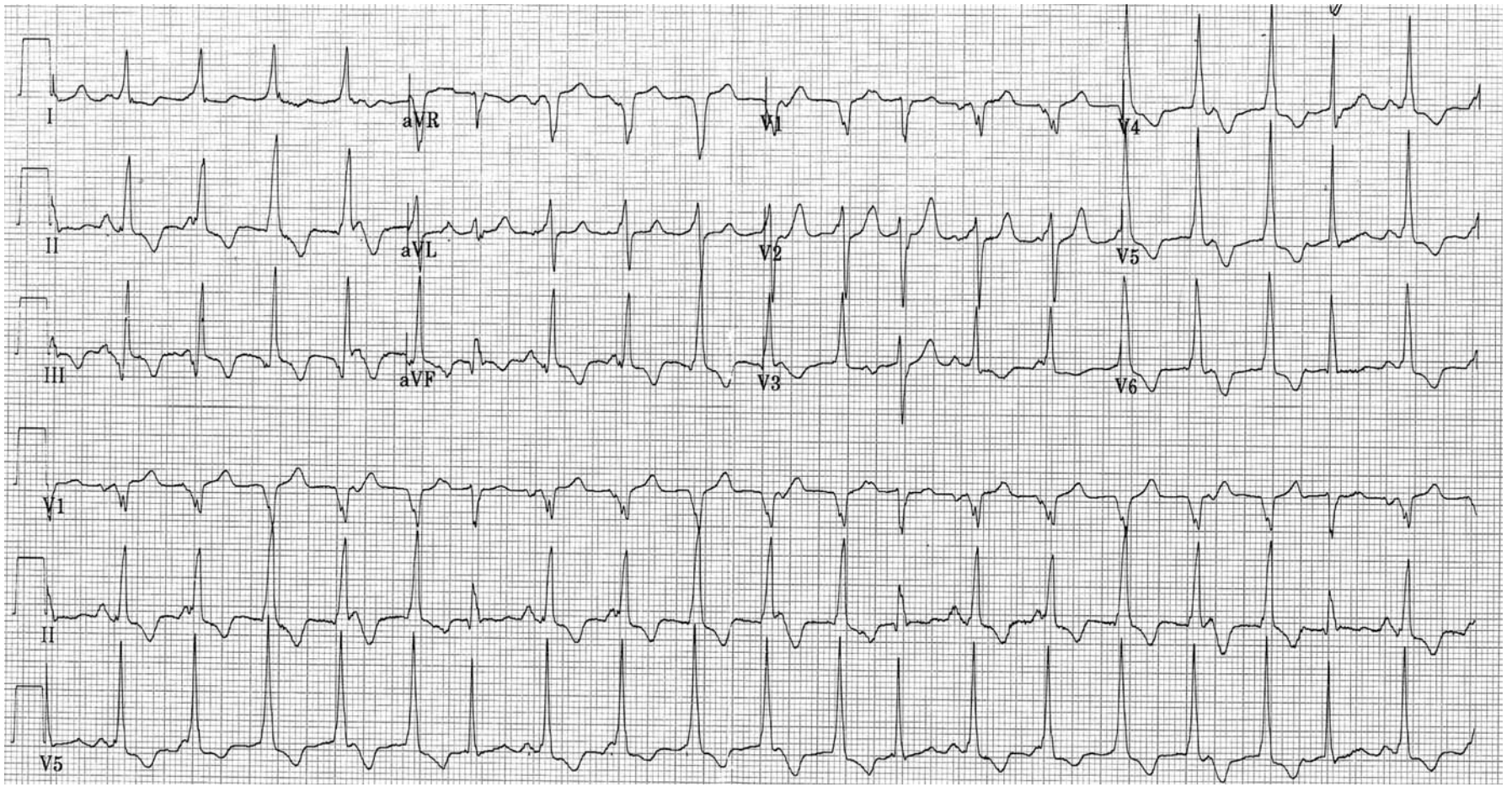


A 52-year-old man had repeated episodes of dizziness and near syncope during vomiting 2 days after orthopedic surgery. The rhythm strip shown was recorded during one such episode (Fig. 2–7). What is the most likely cause of heart block?

Explanation:

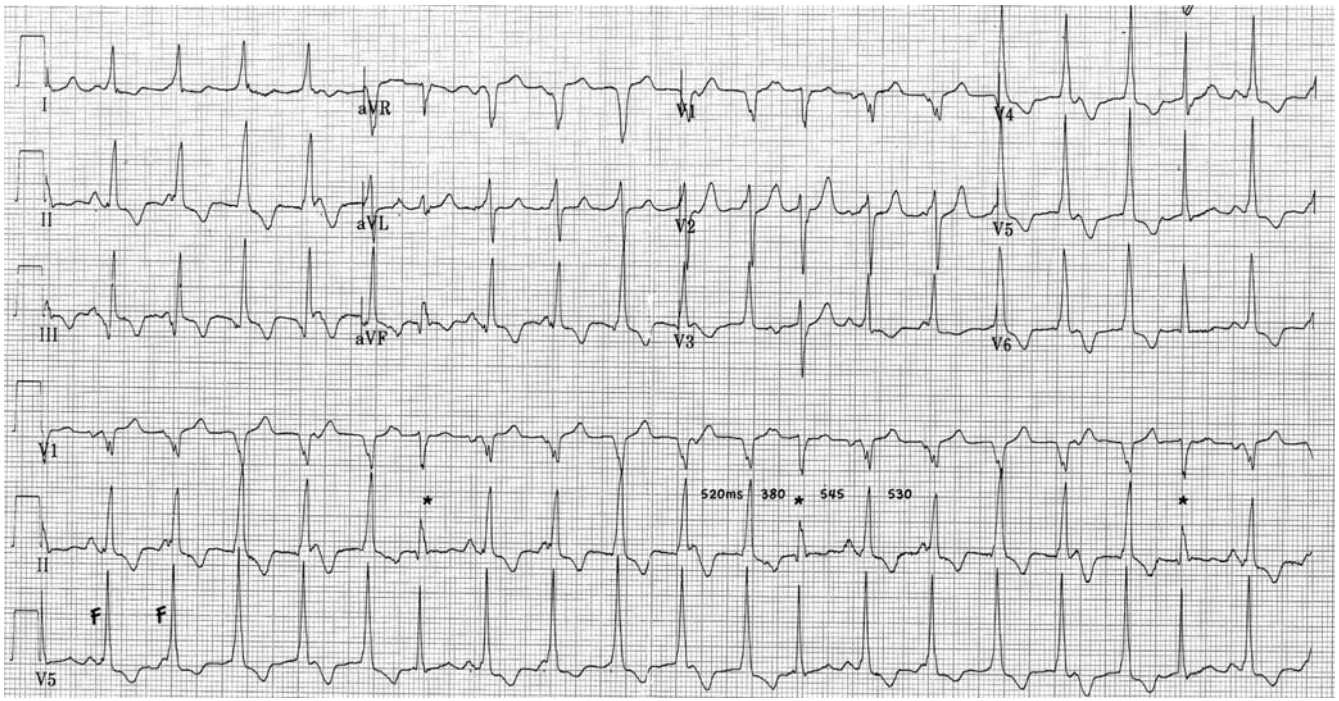
This is a very exaggerated example of a much more common problem, that is, heart block due to increased vagal tone. The first clue comes from the history where vomiting would be expected to be associated with high vagal tone. The second clue can be seen by examining the P–P intervals in the upper strip. Note an initial prolongation of the P–P interval associated with AV block. Slowing of the sinus rate coincident with heart block requires a mechanism simultaneously affecting both the sinus node and the AV node, both relatively far (centimeters) apart. Heightened vagal tone best explains this situation and, while the degree of P–P interval increase may be slight versus the many nonconducted P waves, the mechanism remains the same. The patient was receiving medications after surgery that caused a significant amount of gastrointestinal upset and vomiting and the treatment was to stop his medications, after which both vomiting and AV block stopped.

Figure 2–8A



The electrocardiogram is recorded in a 64-year-old man presenting with palpitations. What is the interpretation?

Figure 2–8B



Explanation:

The heart rate is approximately 120/min. The QRS is relatively narrow with QRS width of 100 milliseconds. Nonetheless, AV dissociation is evident in many leads. The P wave vector is high to low, compatible with sinus rhythm.

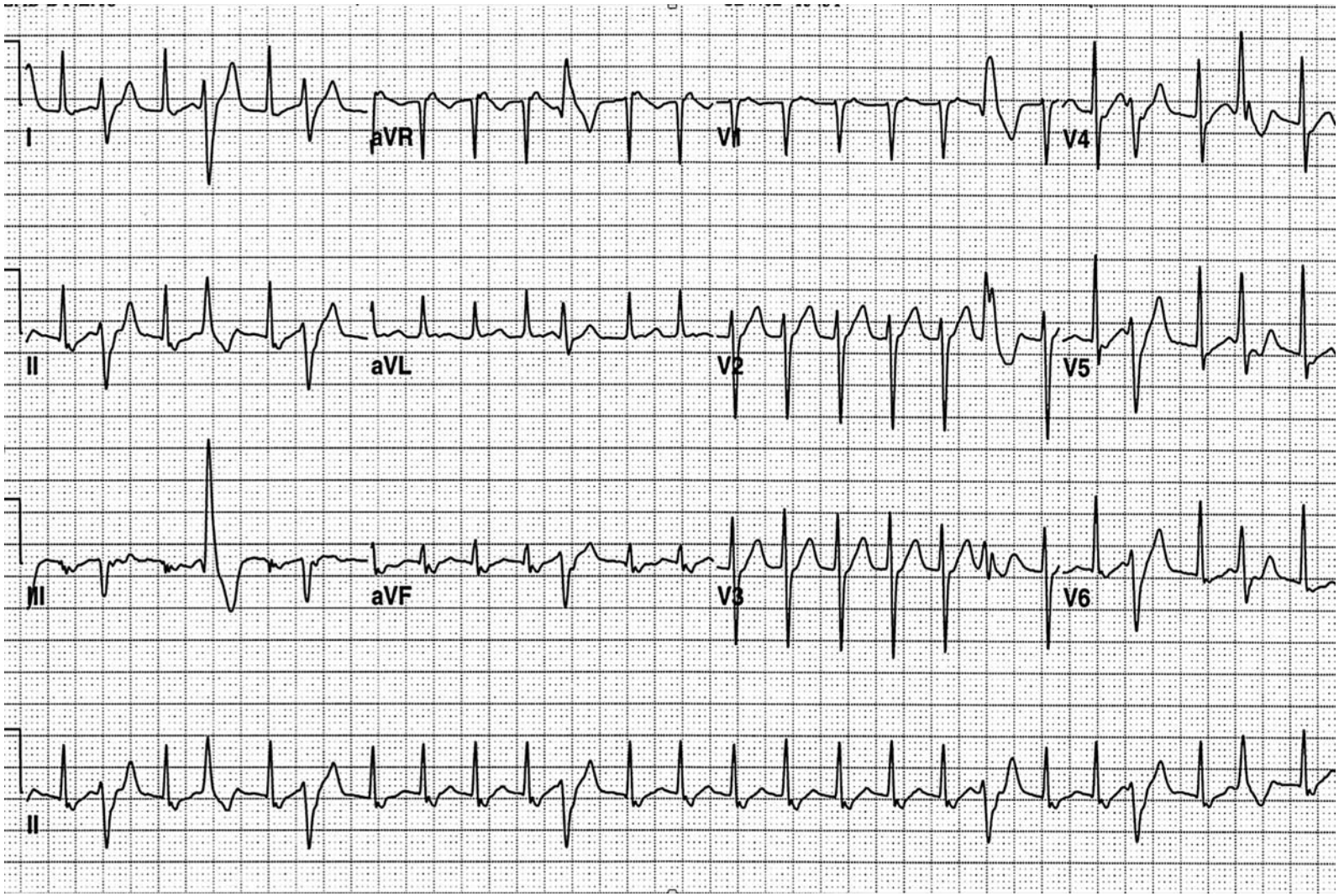
The differential diagnosis of tachycardia with AV dissociation is relatively narrow (Tables 1–4 and 1–7).

Cycles 6, 12, and 18 are preceded by a P wave followed by a narrower QRS. Careful examination also shows "intermediate" QRS morphologies, for example, the first cycle shown. Thus, cycles 6, 12, and 18 (asterisks) are most compatible with capture beats and the first two cycles with fusion beats (F).

This is clearly VT in spite of the relatively narrow QRS during the tachycardia. The tachycardia is relatively slow and virtually the same rate as sinus; hence, it is "isorhythmic" with the sinus rate.

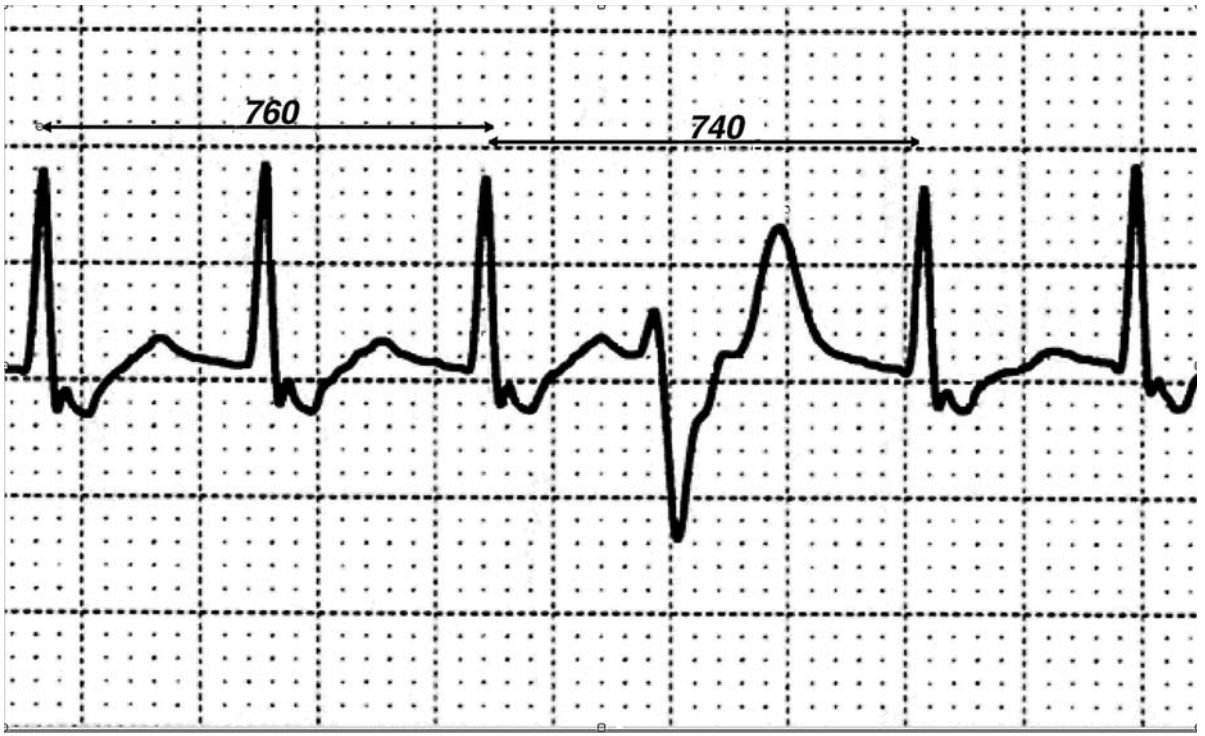
Also note that the capture beat advances or "resets" the ventricular tachycardia. The R–R interval after the capture beat virtually equals the tachycardia cycle length. One can conclude that the sinus beat has very good access or proximity to the VT mechanism (focal or reentrant). This could be the case if VT were arising from the specialized conduction system.

Figure 2–9A



What conclusions can be drawn regarding mechanism of tachycardia as recorded in this 12-lead electrocardiogram?

Figure 2–9B



Explanation:

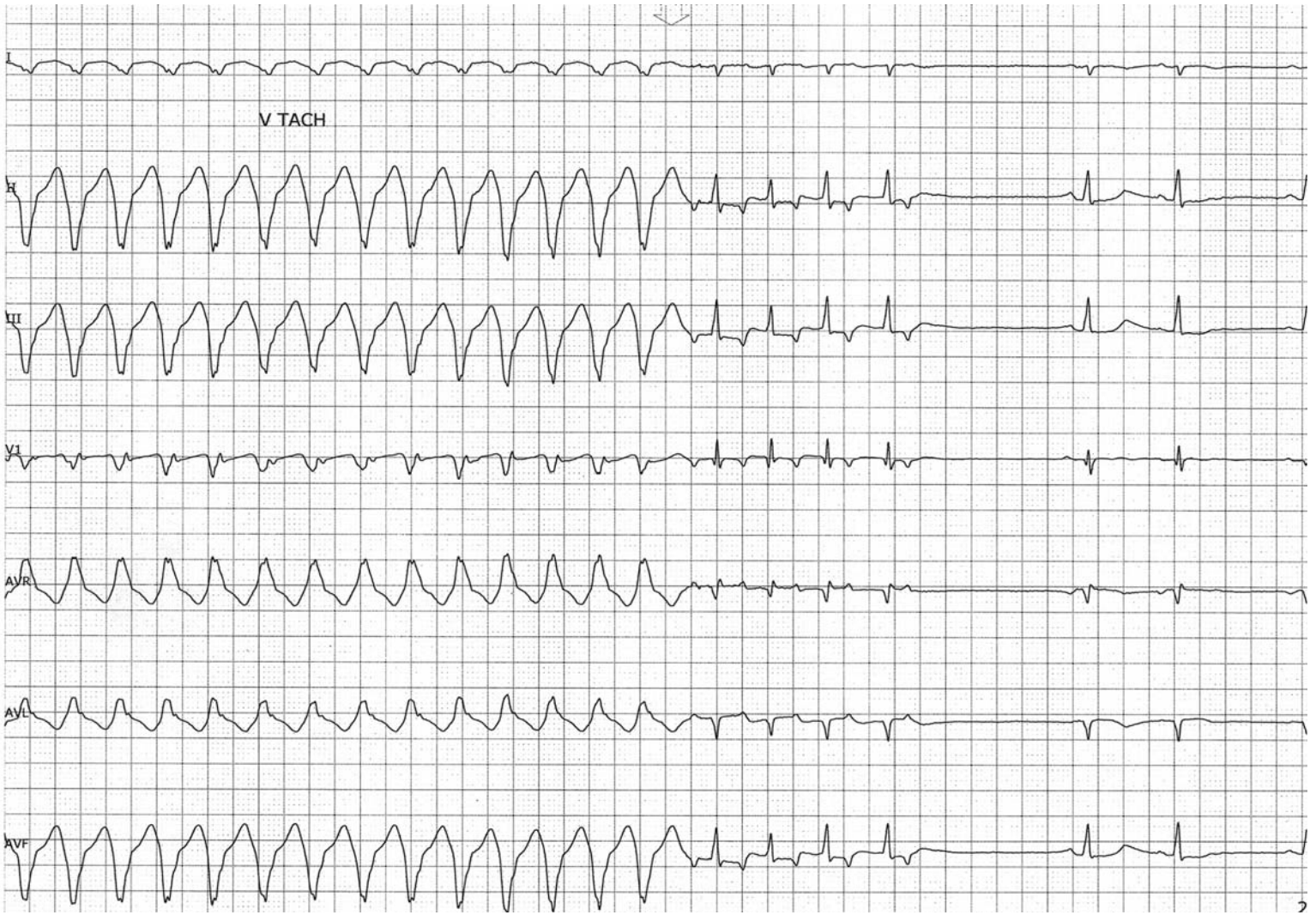
The following provides an exercise in interpreting the ECG as an electrophysiologist rather than a "pattern" reader. The tracing shows a regular supraventricular tachycardia with frequent PVCs, most of right bundle branch block morphology.

There is a P wave in the early ST segment with a low to high activation sequence as seen in inferior leads. This is compatible with atrial tachycardia with long PR, AV reentry over an accessory pathway, or AV node reentry.

Attention can be drawn to the spontaneous PVCs that can serve just as PVCs programmed into the cardiac cycle during EP study. With careful measurement of this very regular tachycardia, there appears to be a subtle reset of the QRS following the PVC that suggests that we are dealing with AVRT. Although we cannot be sure of ventricular fusion and obviously cannot see the His deflection, reset would not be expected in the case of AVNRT or AT with a relatively late-coupled PVC as we have here. The expert ECG reader would have also detected the negative P wave in lead I, which reflects initial left atrial activation, which can be seen in either AVRT or a left-sided AT.

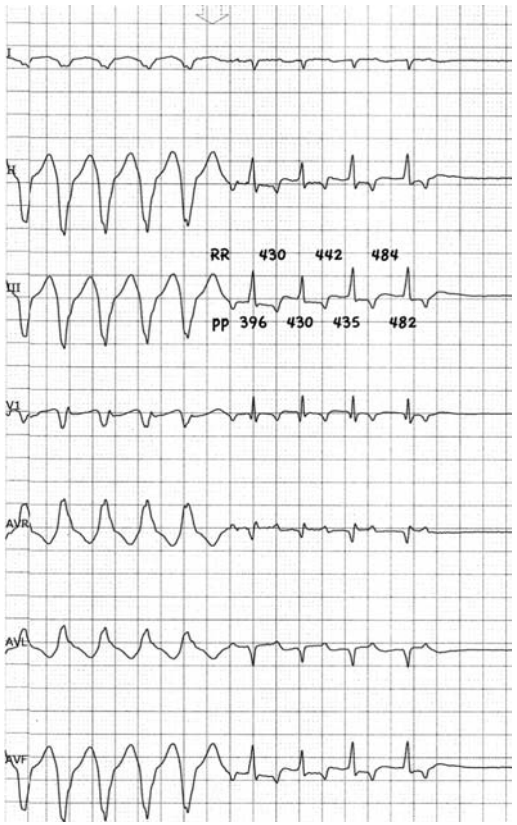
This was, in fact, AV reentry over a posterolateral accessory pathway.

Figure 2–10A



This tracing was recorded from a 74-year-old woman being investigated for chest pain and undocumented palpitations.

Figure 2–10B



Explanation:

It is useful in a complex tracing to divide it into units choosing a section where one can make sufficient observations to start the analysis meaningfully. In this example, one can identify three areas corresponding to two tachycardias and a transition zone between the two.

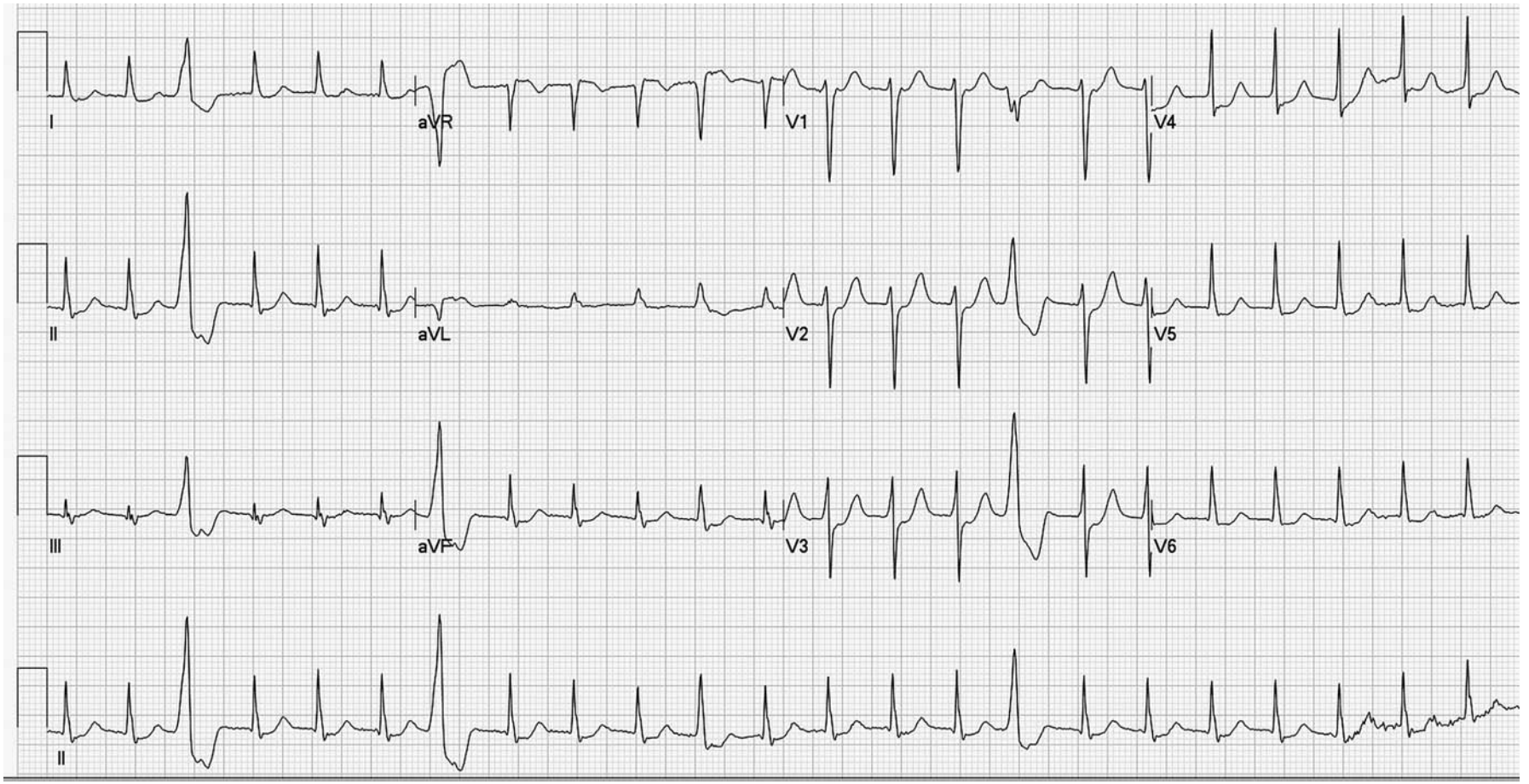
The tracing begins with a regular wide QRS tachycardia. This can only be one of VT, SVT with aberration, preexcited tachycardia, paced rhythm, or artifact. It is obviously not artifact or paced in this case. Preexcitation is unlikely if previously not diagnosed in this 74-year-old individual and the sinus rhythm at the end of the trace is obviously not preexcited, so this possibility can be reasonably discounted at this time. P waves are not apparent during this tachycardia. The QRS morphology here is very helpful. The sinus cycles have a relatively normal-looking narrow QRS and one would thus expect aberration (which is essentially functional bundle branch block) to resemble a fairly typical right bundle branch block or LBBB pattern. In this case, the QRS morphology is very atypical for any bundle branch block pattern with low to high frontal plane and a "northwest" axis making a diagnosis of VT most probable. There is a hint of AV dissociation (see second cycle in V1) but it is difficult to be dogmatic about this.

VT ends with a P wave with low to high activation sequence and several beats of normal QRS tachycardia that is safe to call SVT. This is the *transition zone*. If one assumes that the VT induces the new tachycardia in this way, one has induction of the SVT from the ventricle that is most suggestive of a junctional reentrant tachycardia, either AVRT or AVNRT. A de novo atrial tachycardia starting like this cannot be completely rejected but would be most unusual.

The end of the tracing shows a supraventricular tachycardia with a 1:1 AV relationship. The P wave vector is "low to high" and compatible with a septal pattern of atrial activation. The SVT ends with a P wave, making atrial tachycardia now even more unlikely since one would have to postulate AV block occurring at the same instant as tachycardia termination. In addition, there is cycle length variability ("wobble") with change in both the PR and the RP interval. The change in V-to-V interval precedes the change in P-to-P interval, now making atrial tachycardia as a mechanism untenable. In the experience of the authors, alternation in both PR and RP intervals is almost universally seen in AVN reentry.

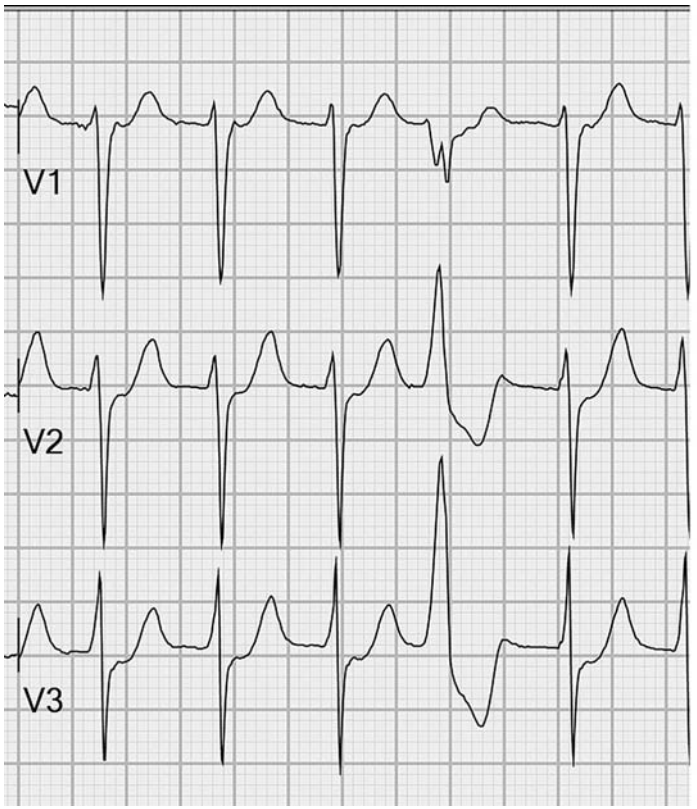
Having looked at all three "segments" of this tracing, one can conclude that VT stops spontaneously; the last VT beat conducts retrogradely over the AV node over a "slow" pathway and this initiates a short run of nonsustained AVNRT.

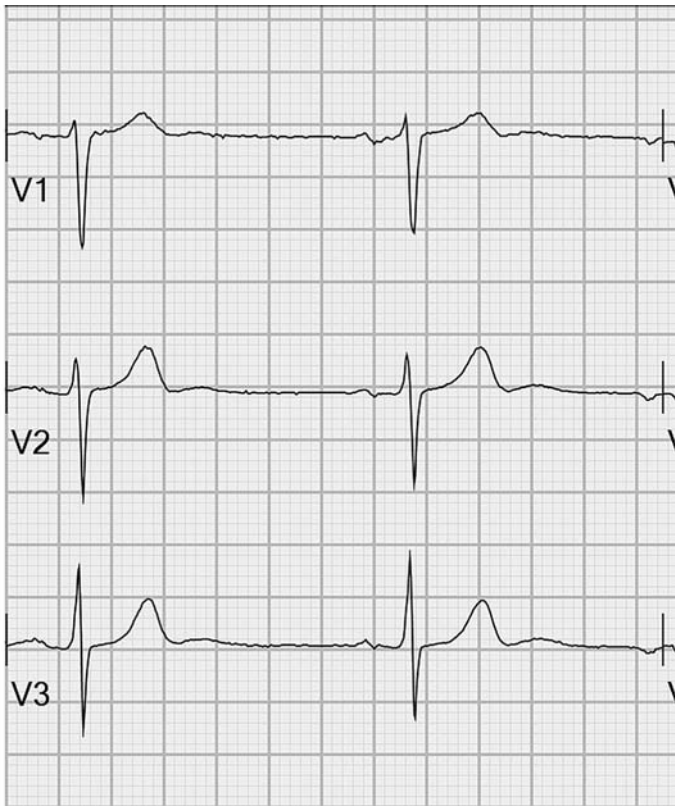
Figure 2–11A



The reading cardiologist's interpretation for this tracing was "supraventricular tachycardia at 142 beats/min." How would the electrophysiologist interpret it?

Figure 2–11B



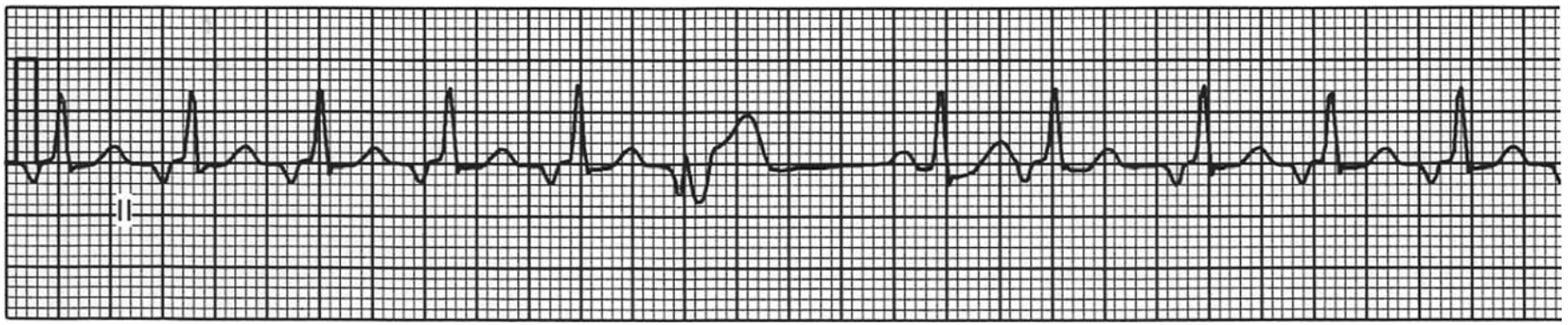


Explanation:

The electrophysiologist would be more explicit about the AV relationship and how it supports a mechanism. There is a terminal distortion of the QRS evident in many leads that has been termed in V1 a "pseudo R prime." This is evident especially when sinus rhythm (right panel) is compared with the tachycardia on the left. The r-prime amplitude is typically small as noted here.

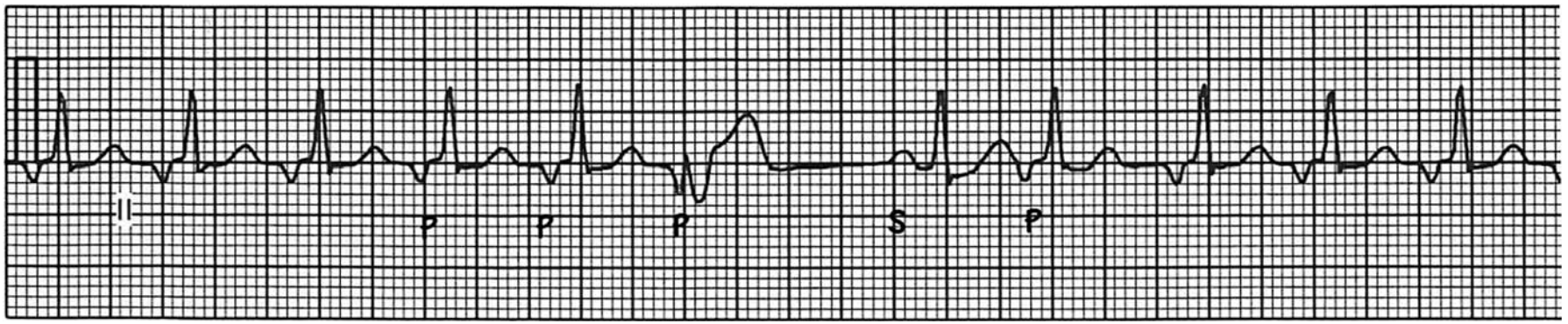
The PVCs provide an opportunity. First, these "open up" the PR segment and it is evident that there is no atrial activity at least within 200 milliseconds of the onset of the QRS complex. Second, there is a full and exact compensatory pause after the PVC so that there are two exact cycles between the two complexes sandwiching the PVC. That is, there is no reset. This favors AVNRT where one would not expect reset since the circuit is relatively small and distant from the PVC source (see Fig. 1–3). The evidence here would justify an interpretation of "probable AVNRT."

Figure 2–12A



ECG from a young man with virtually incessant tachycardia. The observation was reproducible. Can the mechanism of arrhythmia be inferred from this rhythm strip?

Figure 2–12B



Explanation:

A narrow QRS tachycardia with a 1:1 AV relationship is evident on the left of the tracing. The P wave in lead 2 (P) is negative, indicating a "low to high" atrial activation sequence. If we accept the QRS as normal, this can be atrial tachycardia, AV reentry, or AVN reentry, the latter two obviously associated with a long retrograde conduction time.

A PVC interrupts the tachycardia and the following beat has an upright P wave (S) compatible with sinus rhythm before tachycardia resumes.

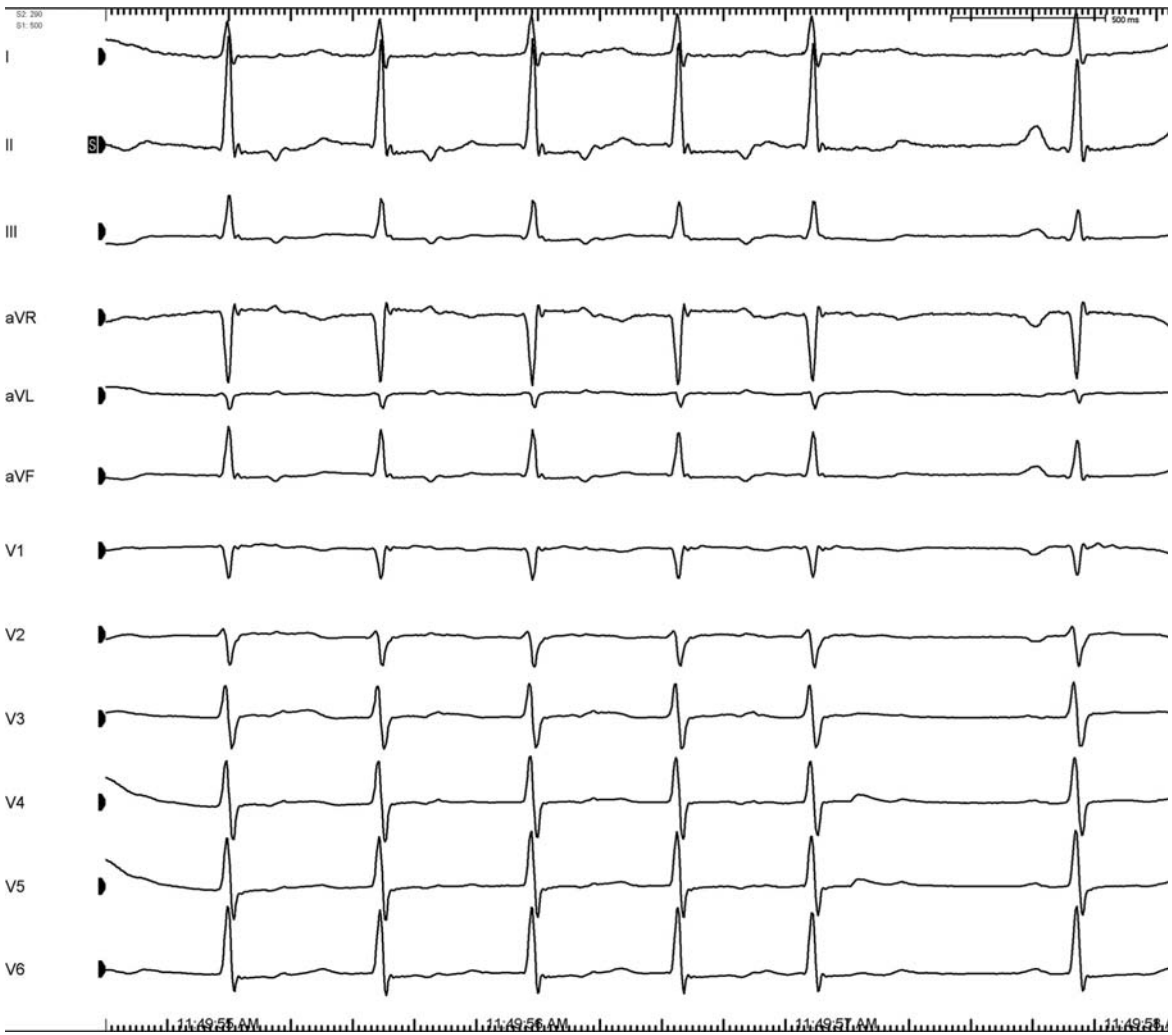
The PVC is late coupled and interrupts the inverted P wave that has already started resulting in merging of the inverted P wave and the PVC. The PVC interrupts the tachycardia without conduction to the atrium, making atrial tachycardia untenable. This PVC is very late coupled but nonetheless is able to penetrate the circuit, thus making AVNRT untenable. AVRT utilizing an accessory pathway with a long VA time could provide good access of the PVC if the PVC were relatively close to the pathway that in this case was an RV PVC with a septal accessory pathway.

Additionally, if one estimates where the His bundle deflection would be on an intracardiac tracing and measures carefully, this PVC would surely find the His refractory and one could then look at it as a "His refractory" PVC that terminates the tachycardia, which would obviously unequivocally prove both presence and participation of an accessory pathway in the circuit.

It is reasonable to mention for completeness that the tracing would also be compatible with the rare possibility of a nodoventricular pathway as the retrograde limb of the circuit.

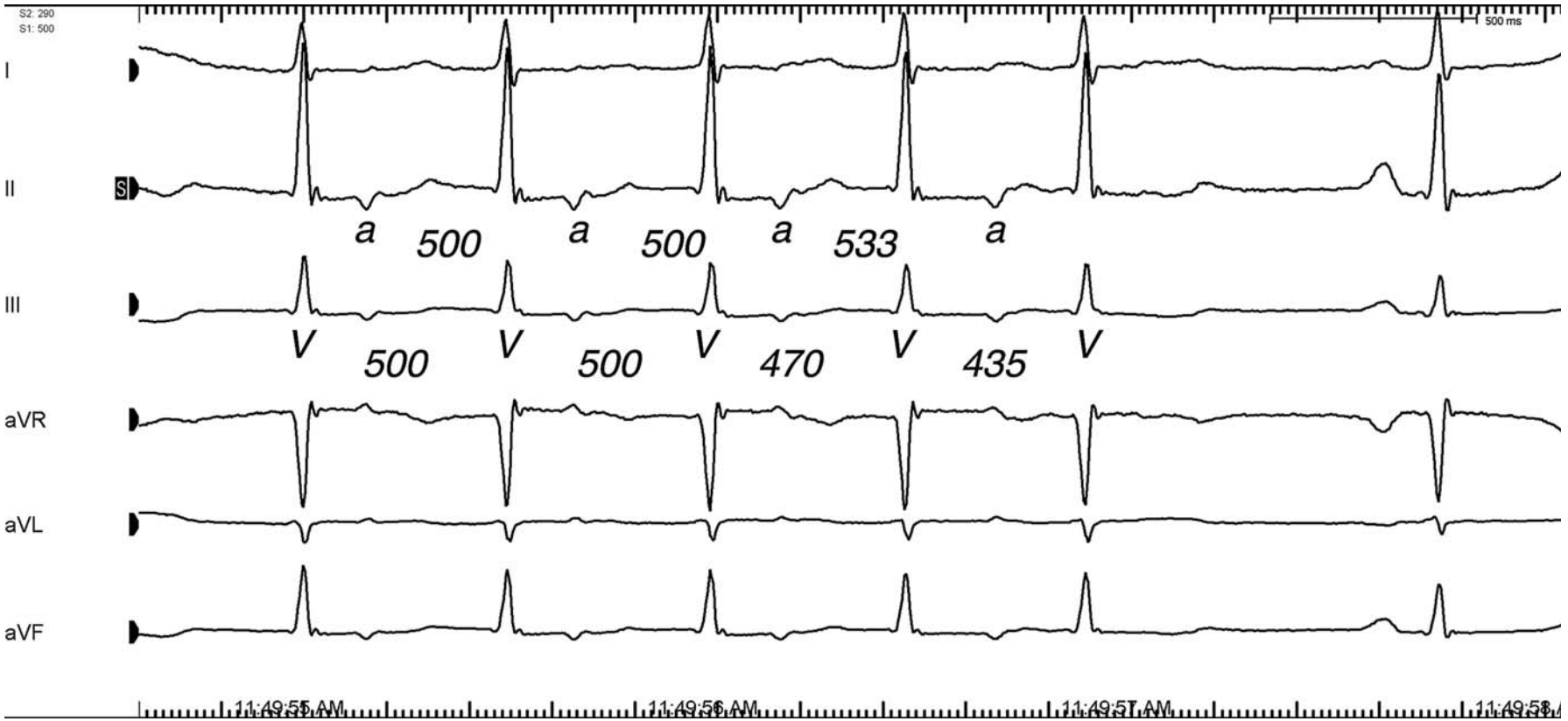
Figure 1–3 and the discussion pertaining to it are relevant to this example.

Figure 2–13A



The patient has a history of sustained tachycardia and a brief run is recorded. What would be the "prime suspect" at electrophysiologic assessment?

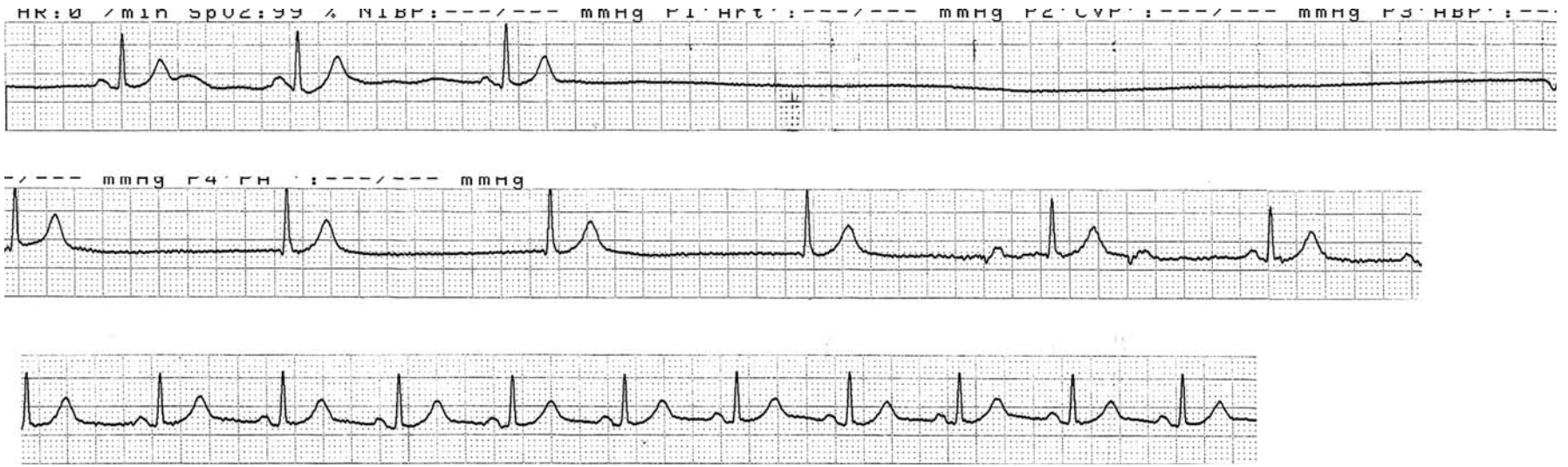
Figure 2–13B



Explanation:

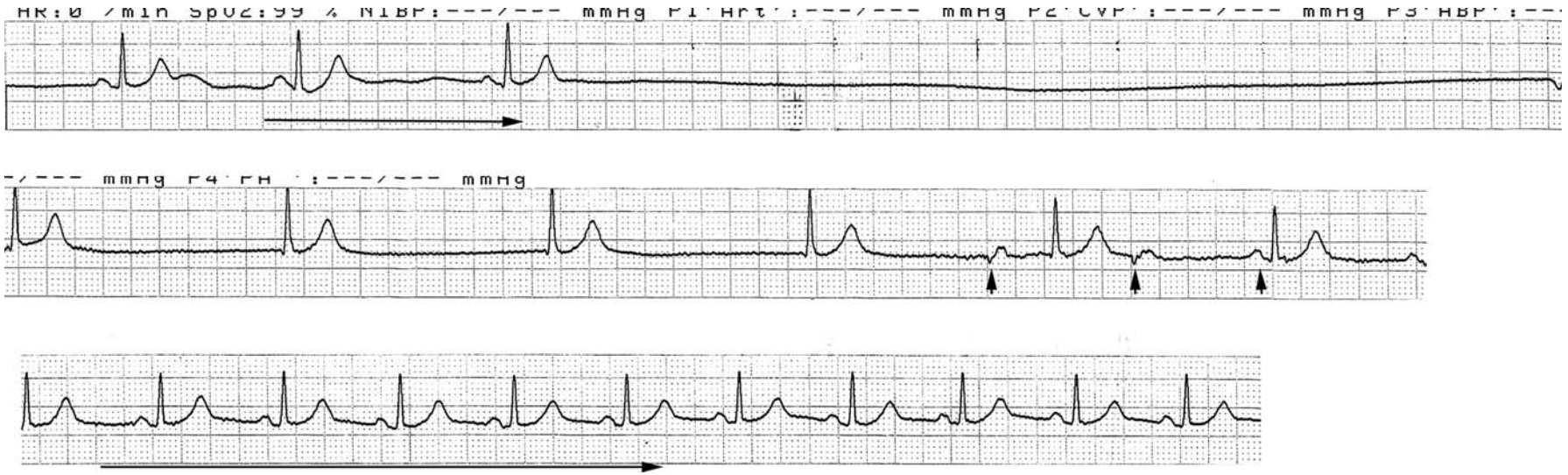
P waves are clearly visible in this self-terminating episode. The P wave vector is low to high. Careful measurement is needed in addition to observation. There is slight irregularity and the change in V-to-V interval *precedes* a change in the subsequent A-to-A interval. The subsequent V-to-V interval is even shorter and there is no subsequent P wave. This is not compatible with atrial tachycardia (i.e., change in VV precedes change in AA) and is most compatible with AVNRT with prolongation of retrograde conduction time and then block as a result of further shortening of anterograde AV conduction time, that is, PR shortening resulting in RP prolongation and then retrograde block. The final diagnosis after EP study was AVNRT.

Figure 2–14A



The patient is an elderly gentleman seen in the emergency department for assessment of light-headed spells. A consultation request was made for consideration of temporary pacing.

Figure 2–14B



Explanation:

The rhythm strip starts with sinus rhythm that slows and a junctional escape emerges. An atrial rhythm resumes but two P waves are not conducted to the ventricles before sinus rhythm and AV conduction resume. The more graded onset and termination along with the coexistence of AV block are strongly suggested of a vagal response due to some unknown provocation and pacing alone does generally not help this.

Chapter 3

Fundamentals of Clinical Electrophysiology

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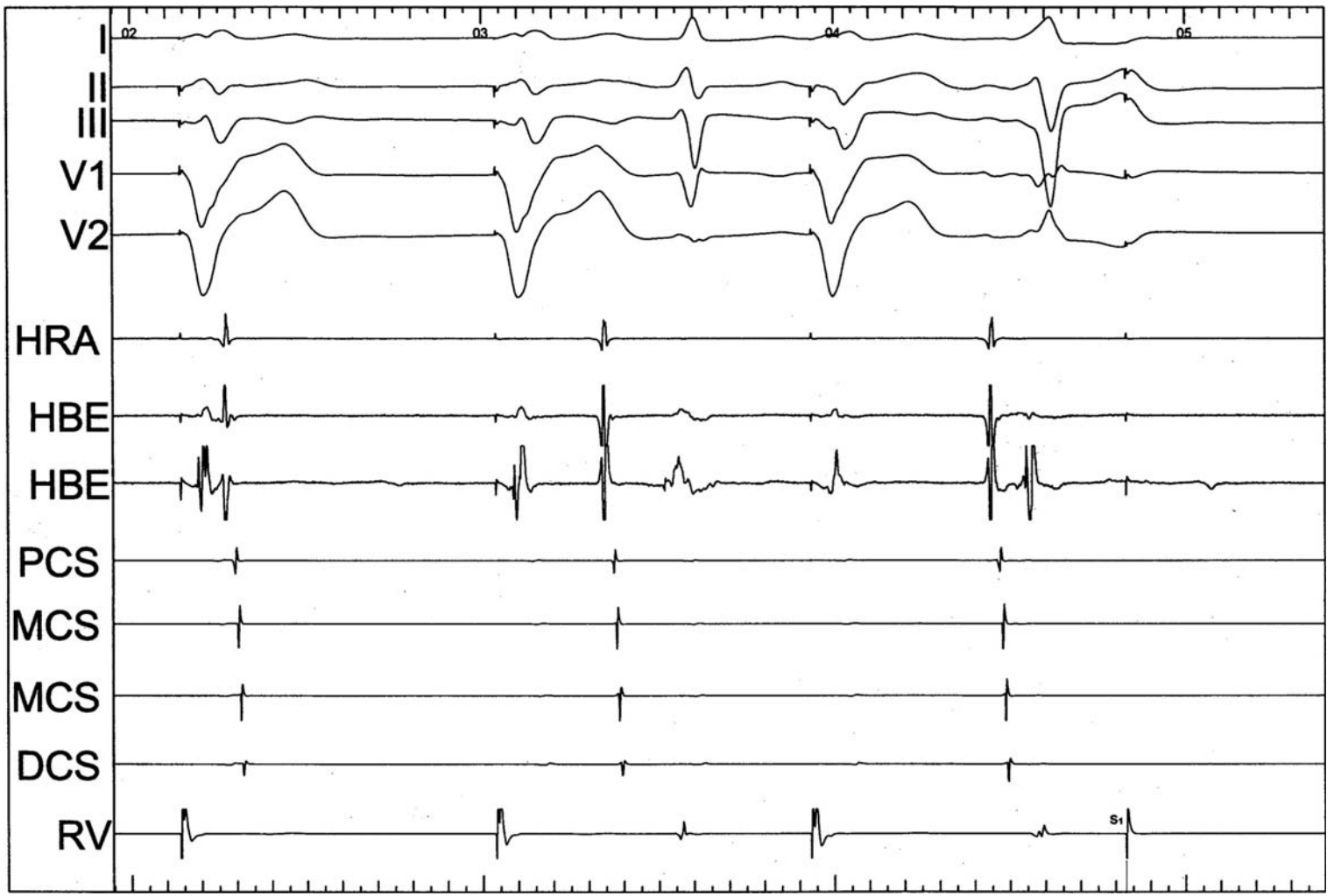
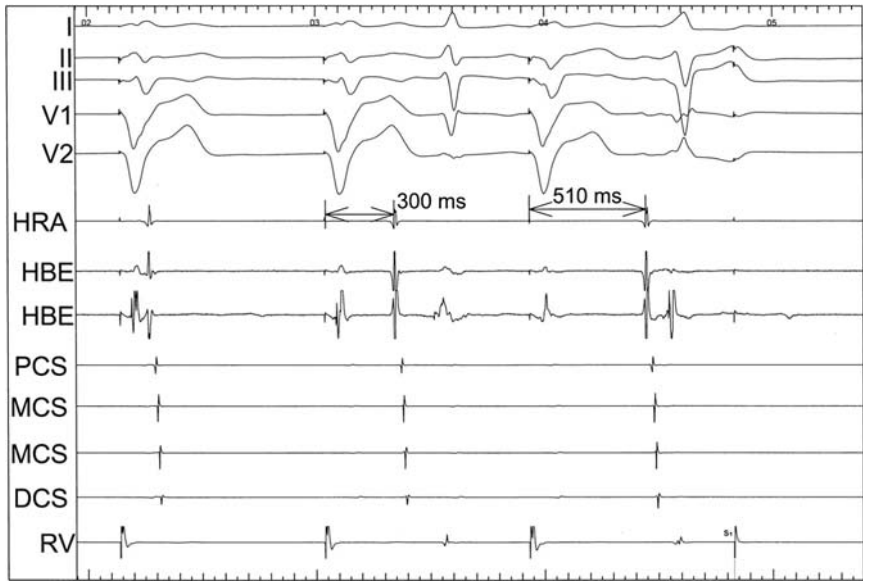
Figure 3–1A



A 30-year-old man with a history of PSVT undergoes electrophysiologic study. During ventricular pacing several electrophysiologic phenomena occur. How many can you name?

Figure 3–1B



Explanation:

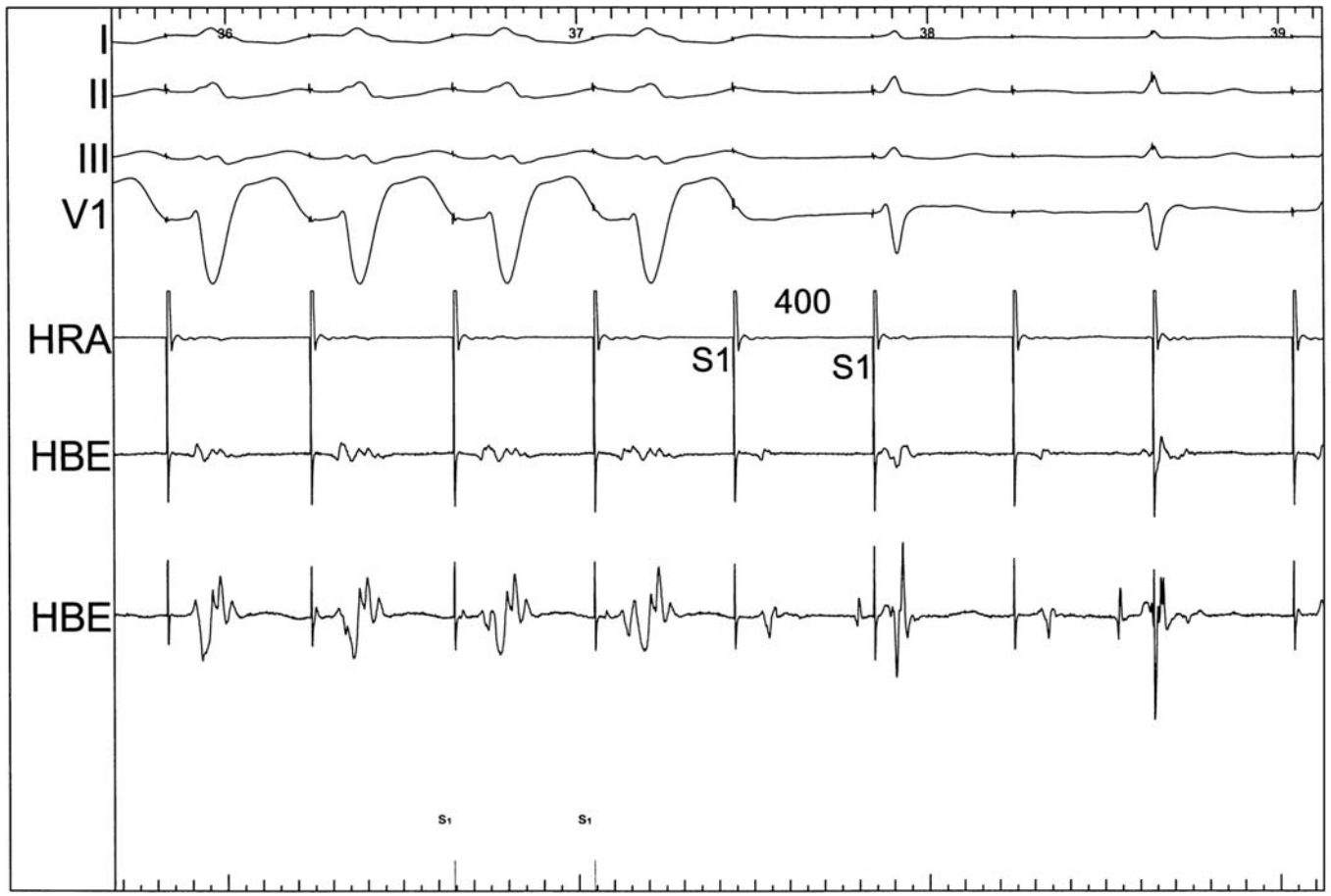
First of all, note that during ventricular pacing there is ventriculoatrial (VA) block. The fourth pacing stimulus does not capture the ventricle because it occurs during ventricular refractoriness that is present shortly after activation from the sinus complex. The third and fifth QRS complexes conduct from the sinus origin to the ventricle. The first sinus complex occurs very soon after the ventricular paced beat and does not conduct through the atrioventricular (AV) node as noted by absence of a His bundle electrogram. Thus, there is concealed conduction into the AV node.

The fifth QRS complex is a result of conduction from the atrium over an AV accessory pathway. It is able to conduct because the time from the previous ventricular paced beat to the sinus complex, 510 milliseconds, is sufficient to allow conduction over the accessory pathway. Since this pathway is not noted on the first sinus complex, either there is also concealed conduction into the accessory pathway preventing AV conduction over the accessory pathway or the ventricle is simply refractory since it comes too early after the initial paced beat.

The third QRS complex is not normal but it does not show conduction over the AV accessory pathway. The paced V to the subsequent atrial interval is 300 milliseconds, and there is substantial prolongation of the AH interval suggesting concealed conduction into the AV node but not to the degree that it causes anterograde block. The HV interval is very short. No matter what the AH interval was in this patient, whenever the AV accessory pathway was blocked there would be a similar short HV interval. This represents conduction over a fasciculoventricular (Mahaim) pathway. Clearly there is concealed conduction into the AV accessory pathway here because the sinus complex can conduct to the ventricle over the normal system as well as the fasciculoventricular pathway. With the longer paced V to A interval of 510 milliseconds, there is now manifest conduction over the AV accessory pathway with a shorter AH interval, but the His is within the QRS complex, characteristic of this type of accessory pathway.

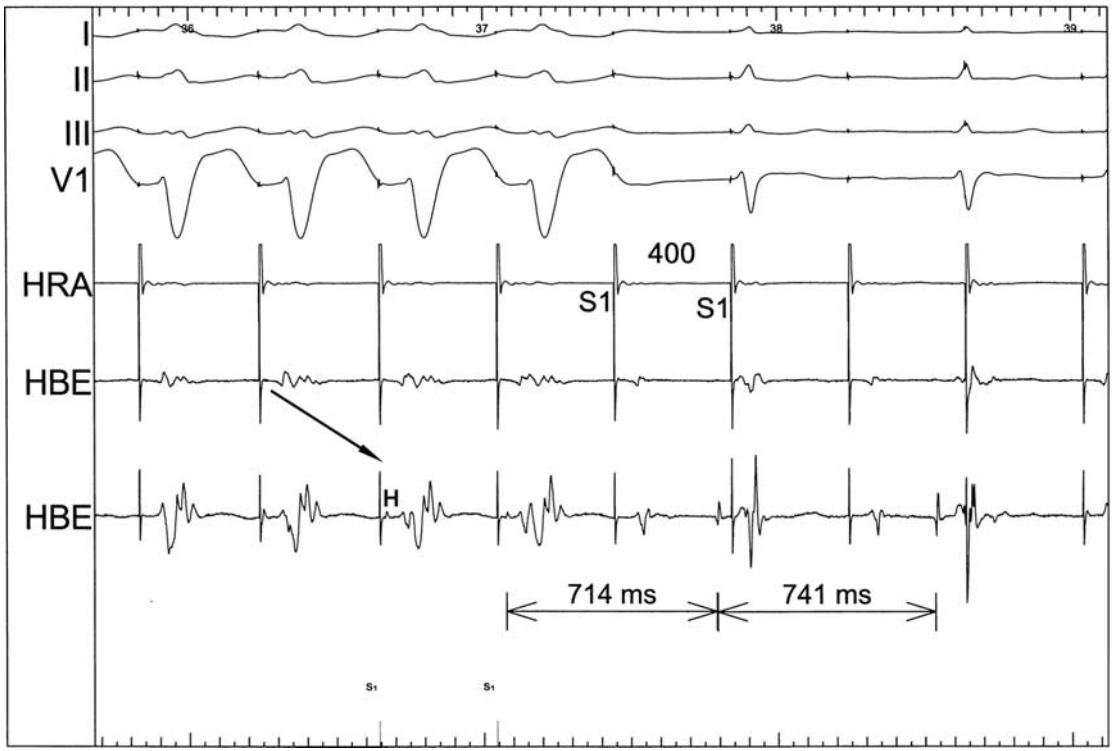
In summary, the electrophysiologic observations in this tracing include concealed conduction into both the accessory AV pathway and the AV node, manifest preexcitation over an AV accessory pathway, and manifest preexcitation over a fasciculoventricular pathway.

Figure 3–2A



At electrophysiologic study, atrial pacing at 400 milliseconds produced the following observation in a 36-year-old woman with history of wide QRS complex tachycardia. Explain the observations.

Figure 3–2B

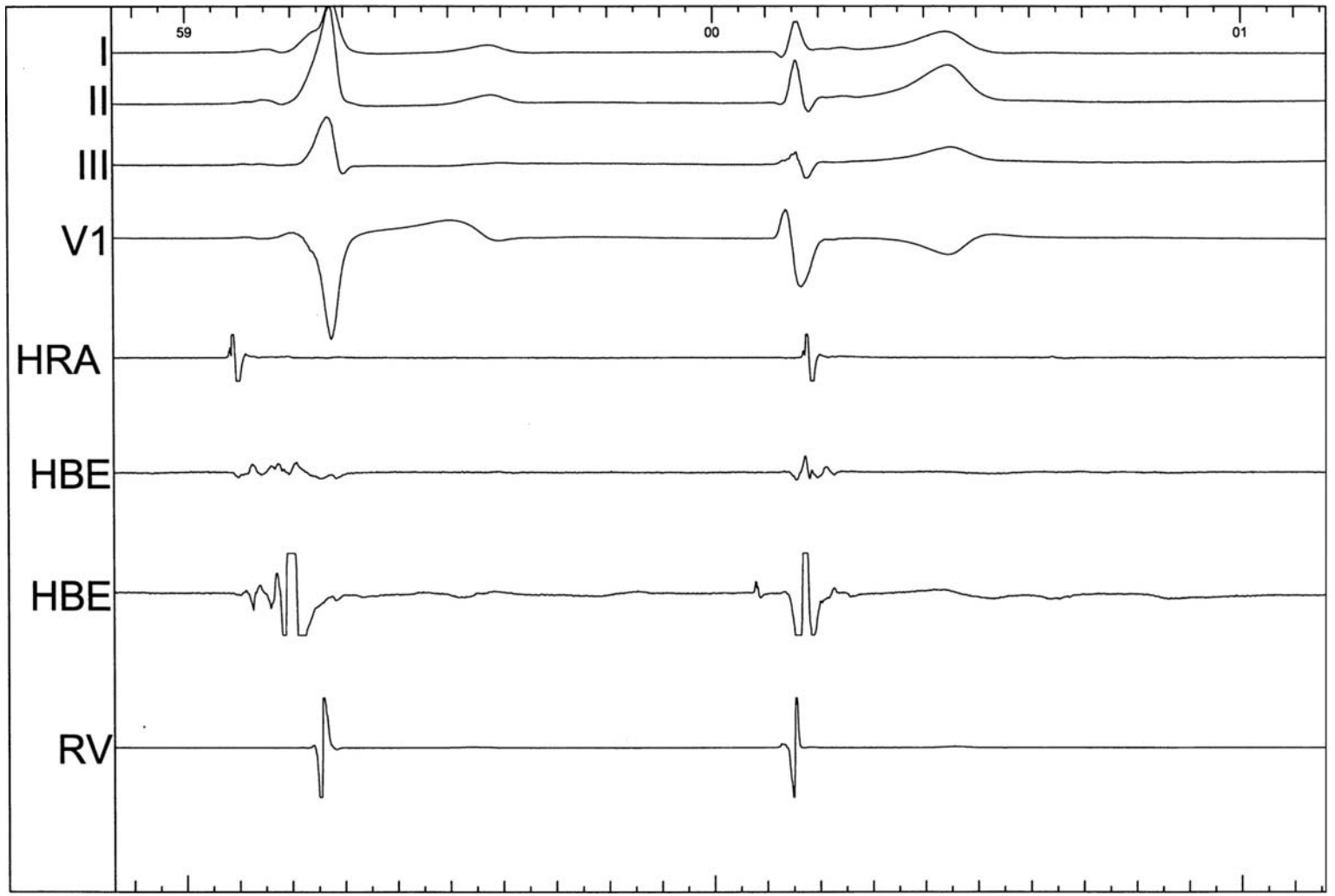


Explanation:

The baseline QRS complex noted on the right side of the figure is normal. The wide QRS on the left side of the figure can only realistically be preexcited or left bundle branch block (LBBB). During incremental atrial pacing a rate-dependent LBBB occurred and the patient also demonstrated a markedly long AH interval. Thus, the paced ventricular responses are essentially "one complex off," that is, they are conducting to the subsequent LBBB QRS complex (*arrow*). The fourth paced atrial complex blocks in the AV node and the next atrial paced complex conducts with a narrow QRS complex and subsequent 2:1 block in the AV node. The LBBB is no longer present because the 2:1 AV block results in an H–H cycle length, 714 and 741 milliseconds, which is outside of the refractory period of the LBB.

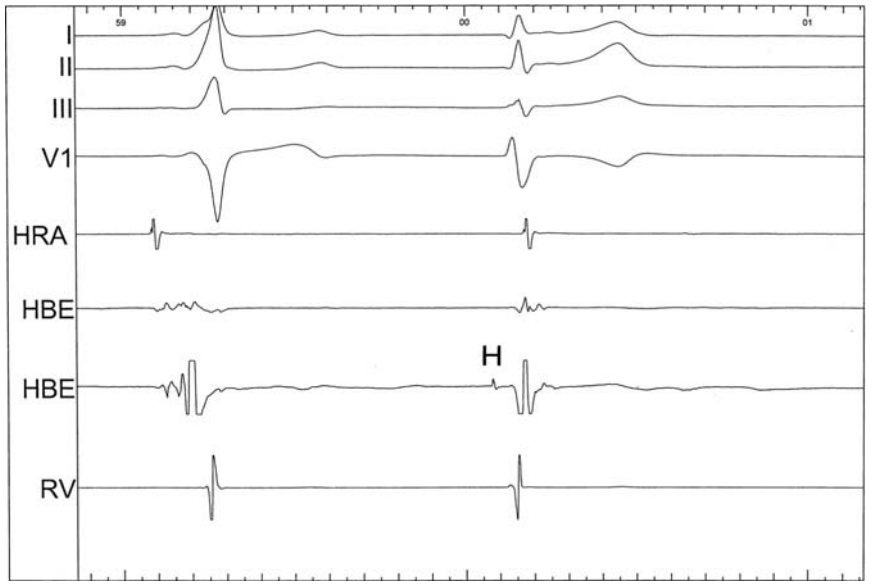
During the LBBB complexes, the His potential occurs at nearly the same time as the pacing stimulus artifact, totally obscuring the His on the first beat, but a close look at the subsequent complexes reveals a His potential well in front of the LBB QRS (H) ruling out preexcitation. Of note, the patient actually had tachycardia secondary to an epicardial ventricular tachycardia site in the posterior area of the heart but otherwise had normal cardiac function.

Figure 3–3A



The preexcitation syndrome involves a variety of accessory pathways. What is the type of accessory pathway noted in the first QRS complex in this tracing?

Figure 3–3B

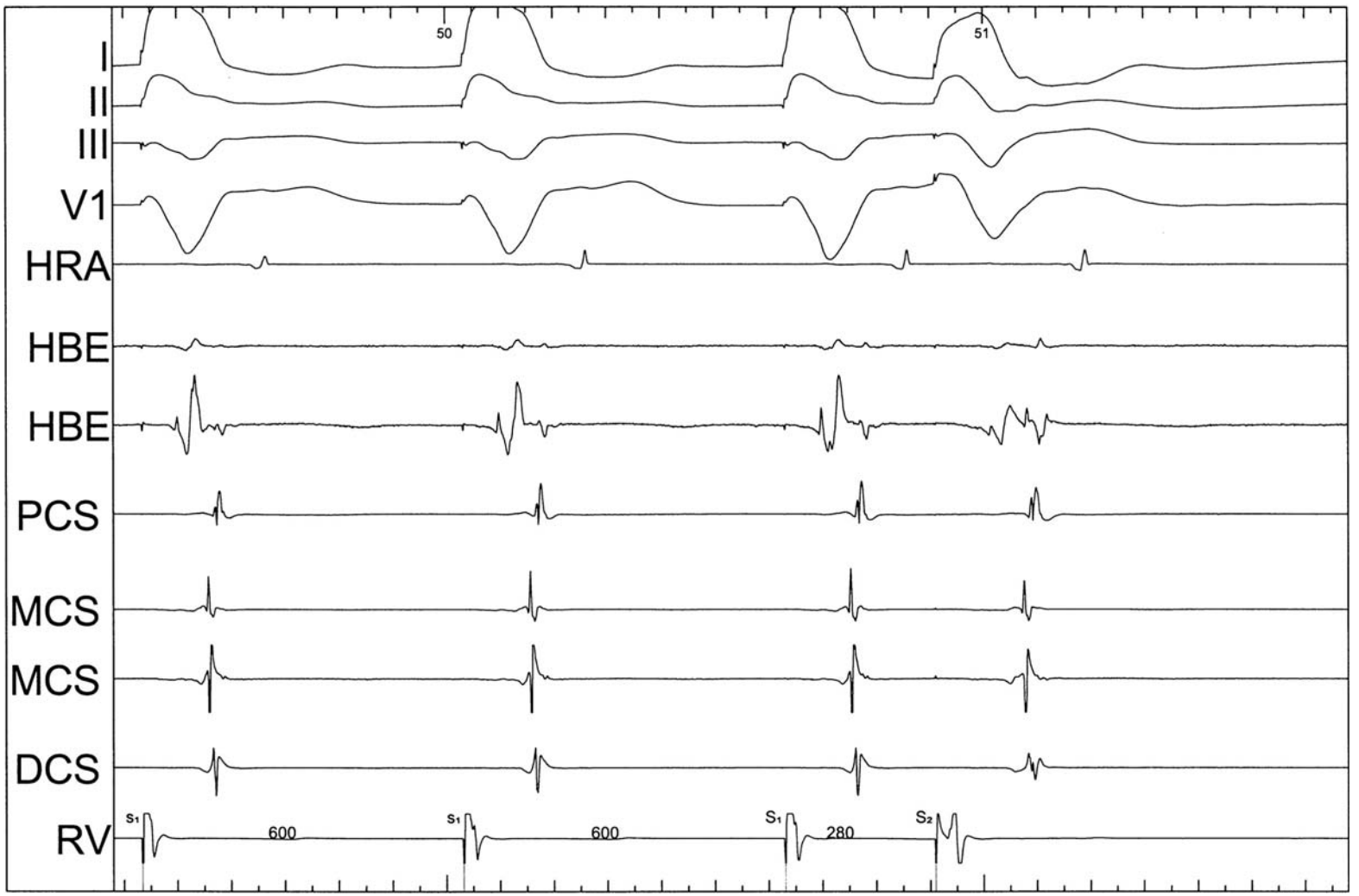


Explanation:

The first QRS complex has the classic preexcited pattern with a short PR interval followed by a delta wave noted most prominently here in ECG lead II. This has sometimes been referred to as an "Eiffel Tower" appearance. The location of this AV accessory pathway was in the atrial septum very close to the normal AV conduction system. The second QRS complex was secondary to a catheter-induced junctional beat. The His electrogram is readily seen in this cycle, suggesting that it is "buried" in the preceding preexcited QRS complex even if not seen.

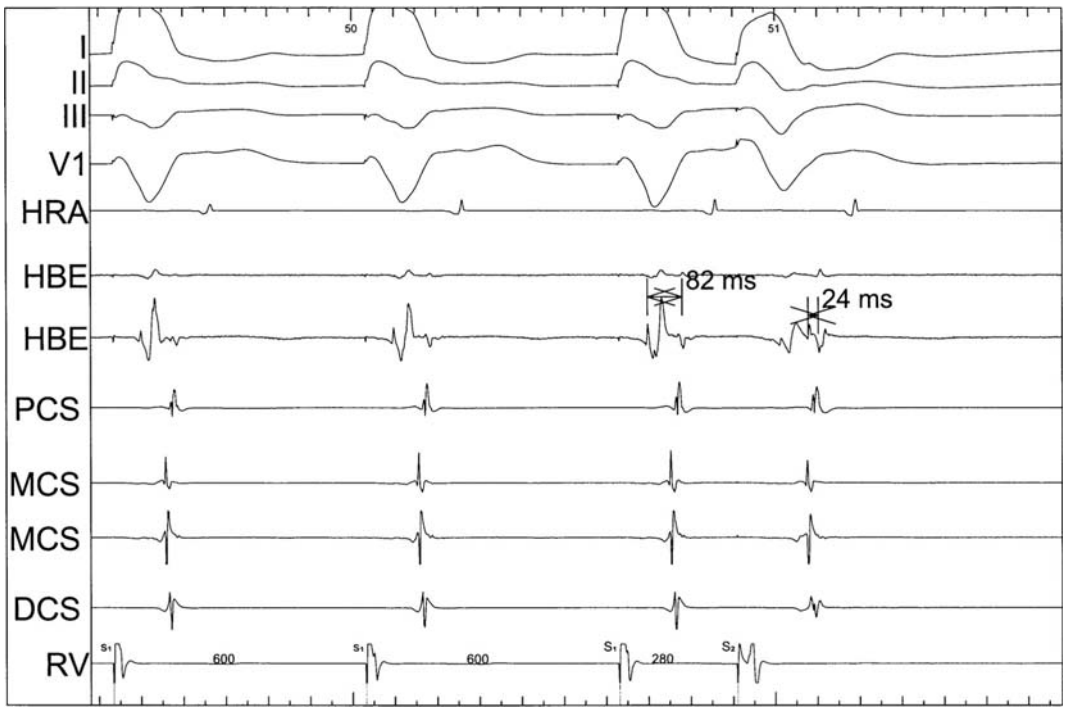
The most common type of accessory pathway is an AV muscle connection, seen here, that originates at the base of the atrium and conducts into the base of the ventricle. This is confirmed when a His extrasystole conducts to the ventricle without preexcitation, clearly demonstrating that the accessory pathway originates above the level of the His bundle. "True" Mahaim fibers as described by Mahaim originate in either the AV node or the His–Purkinje system (HPS), namely, nodoventricular/fascicular or fasciculoventricular pathways, respectively. (The descriptor "Mahaim" has been applied to many atypical pathways that exhibit "rate-dependent" or "decremental" conduction for historical reasons, namely, that all such pathways were thought to involve the AV node as per the pathway described by Mahaim. We believe it is more precise to name the pathways by their origin and insertion sites to be clear since decremental pathways may really be anywhere on the AV ring and the great majority of those (often atriofascicular) seen clinically have nothing to do with the AV junctional connections described by Mahaim.) While one cannot totally rule out a nodoventricular accessory pathway in this instance, they are rare and would not be expected to have such prominent preexcitation with such a short PR interval.

Figure 3–4A



A 24-year-old woman underwent electrophysiologic study for recurrent PSVT. The tracing was obtained during programmed ventricular stimulation. What can be concluded about retrograde conduction?

Figure 3–4B

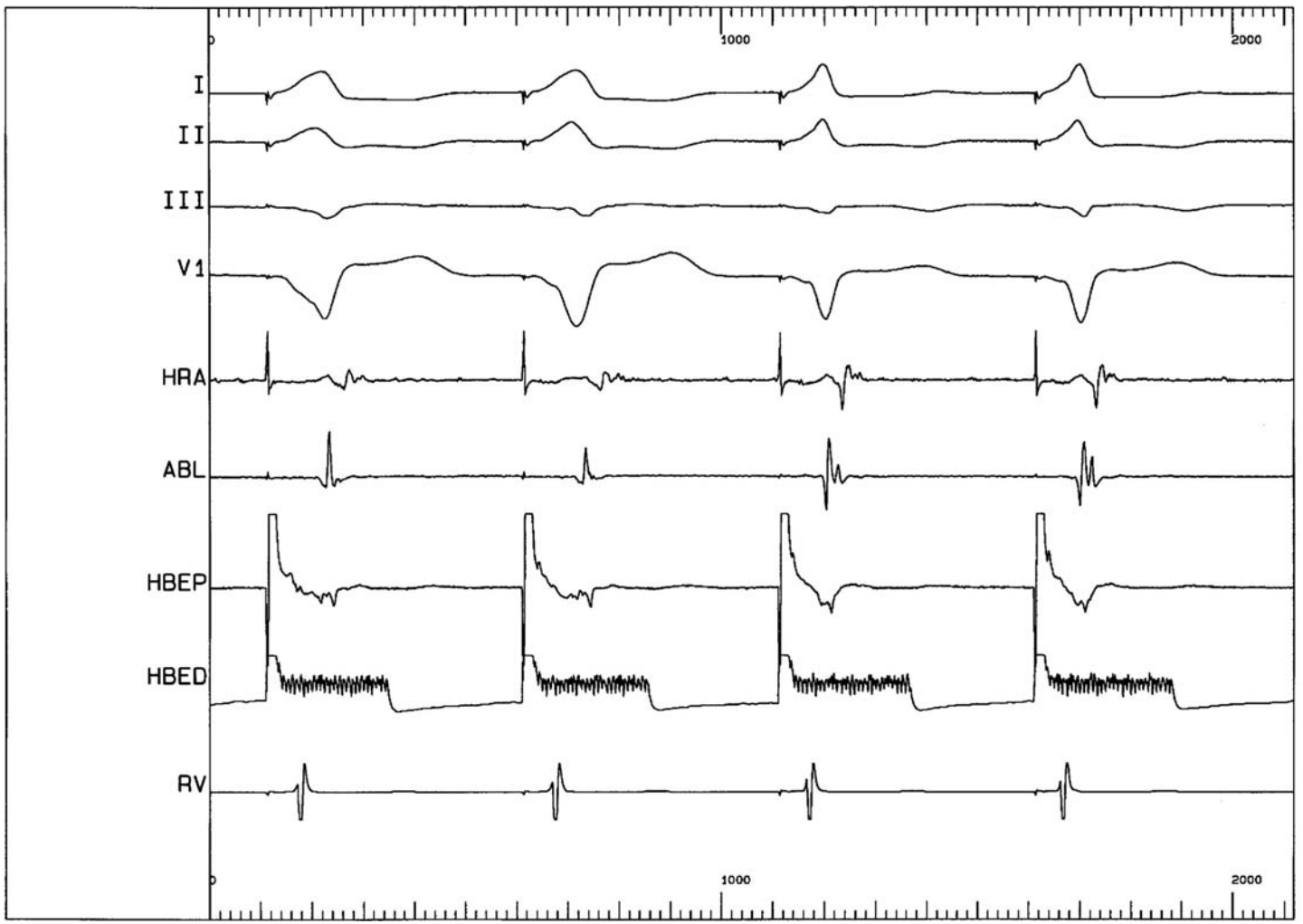


Explanation:

During ventricular pacing, retrograde atrial activation sequence is slightly eccentric with the mid-coronary sinus (MCS) electrogram occurring somewhat earlier than the His bundle atrial electrogram (HBE). On the last beat of the drive train (S_1), the HA interval measured on the HBE tracing is 82 milliseconds. This observation alone cannot differentiate conduction over an accessory pathway or both an accessory pathway and the AV node. However, the premature ventricular stimulus (S_2) causes a sudden prolongation of the VH interval with a concomitant shortening of the HA interval to 24 milliseconds. The sudden lengthening of the VH interval likely results from retrograde block in the RBB with subsequent transseptal ventricular conduction and then conduction to the His bundle over the LBB. Note that the general retrograde atrial activation sequence remains much the same.

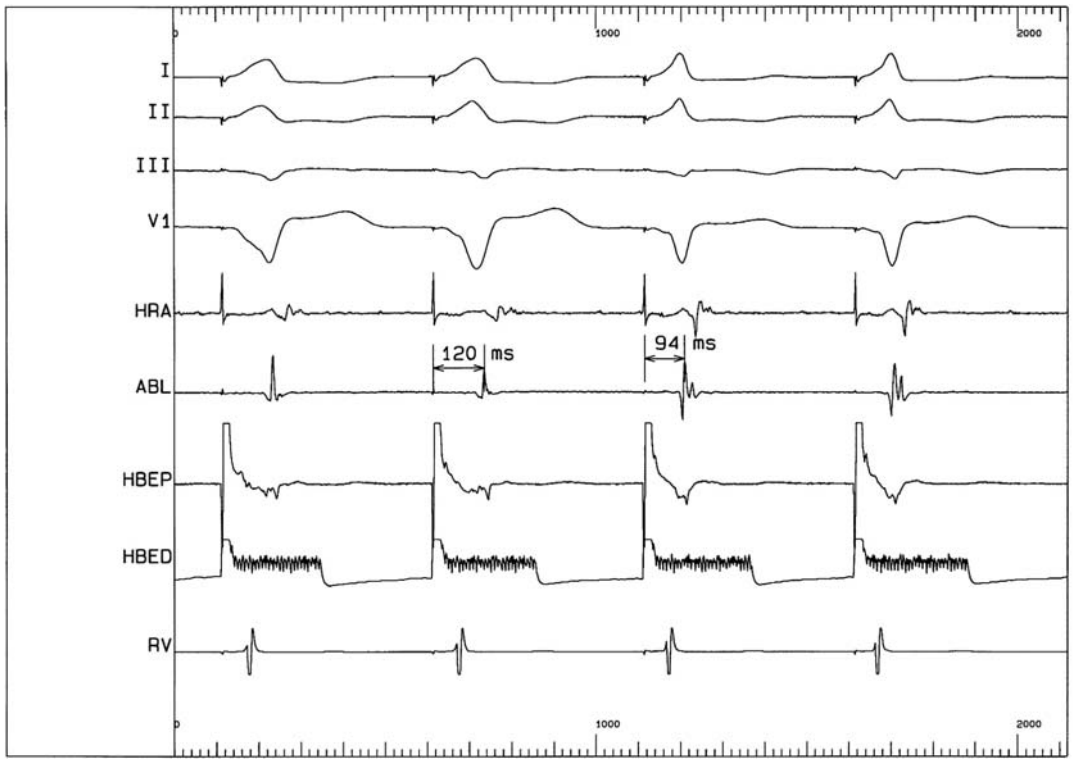
A sudden shortening of the HA interval does not occur with retrograde conduction over the normal AV conduction system. There would be a similar or slightly longer HA interval. It is also noted that atrial activation after S_2 as measured at the MCS electrograms now *precedes* the retrograde His, clearly untenable with retrograde AV node conduction. This observation clearly shows that the HBE atrial electrogram is activated over the accessory pathway and not over the AV node. Of note, this does not mean that conduction over the AV node could not occur at another time during the study, only that it could not be demonstrated in this tracing.

Figure 3–5A



A 25-year-old man with a 2-year history of palpitations and documented PSVT comes for electrophysiologic study. The 12-lead electrocardiogram is normal. The following observation was noted during a pacing maneuver at electrophysiologic study. The ablation catheter, ABL, is positioned in the atrial septum, near the His bundle catheter. Does this indicate the presence of an accessory pathway?

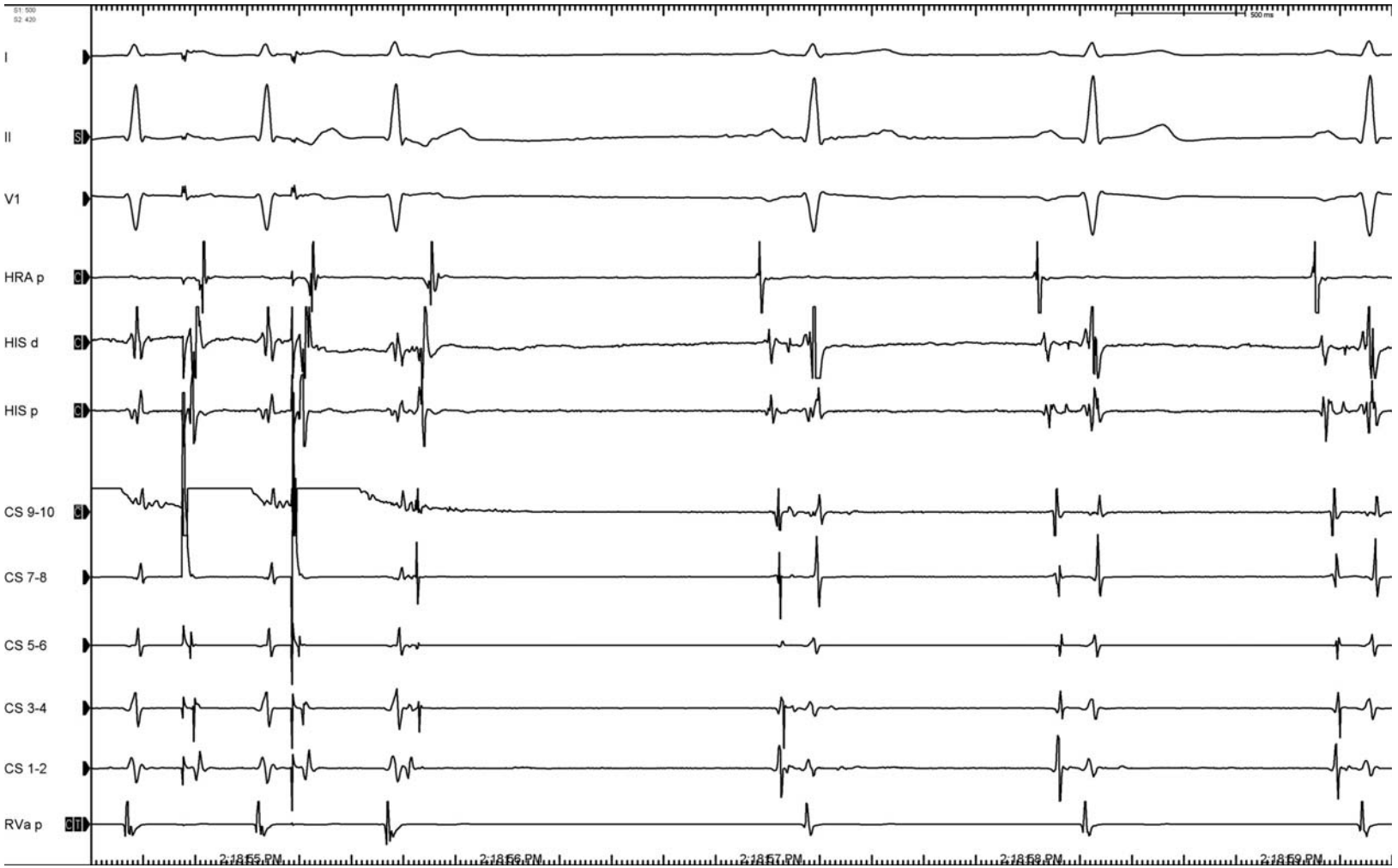
Figure 3–5B



Explanation:

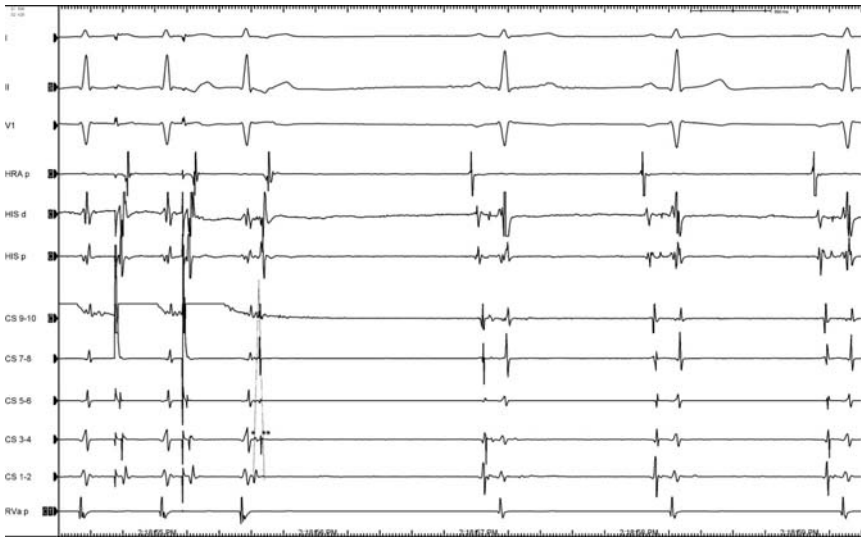
This maneuver is known as para-Hisian pacing. The distal His bundle electrode (HBED) was positioned near the His bundle recording and the pacing energy was incrementally increased to get initial capture only of the ventricle (first two QRS complexes) and subsequently capture of the ventricle and the His bundle (second two, more narrow QRS complexes). Note that the stimulus to atrial electrogram on the ablation catheter (ABL) positioned in the septum is 120 milliseconds without His capture and 94 milliseconds with His capture. This response is characteristic of VA conduction over the AV node and not over a septal accessory pathway. A septal accessory pathway connecting the A and V near this site would typically show a similar VA interval with or without capture of the His bundle during this maneuver. Importantly, while this observation suggests retrograde conduction over the AV node, it does not rule out conduction over an accessory pathway with a long conduction time in which the VA interval might be longer than that noted over the AV node or a nonseptal accessory pathway located farther from the pacing site. This patient did have AV node reentry that was successfully ablated.

Figure 3–6A



This patient presented with a narrow QRS tachycardia and the study began with atrial extrastimulus testing. What will be the likely ablation site?

Figure 3–6B



Explanation:

The atrial extrastimulus results in an echo cycle but the resulting atrial activation is complex with "double" atrial potentials after the QRS. In the distal coronary sinus, there is a large "near-field" atrial electrogram followed by a barely visible "far-field" electrogram. In the adjacent electrode CS 3–4, the first atrial electrogram is far-field (single asterisk) and the second is near-field (double asterisk), as it is for the subsequent two electrode pairs. At the proximal CS (CS 9–10), there is a single electrogram that is relatively early.

It must be remembered that the coronary sinus is often known as the "third" atrial chamber and generally has a muscular coat that is capable of conducting cardiac impulses. This coat is most prominent proximally, although it can extend quite far into the left lateral region. The CS catheter is closer to the CS muscle than the adjacent left atrium so that its potential would generally be expected to be near-field, whereas the LA potential is generally farther and more far-field. This varies somewhat in individuals.

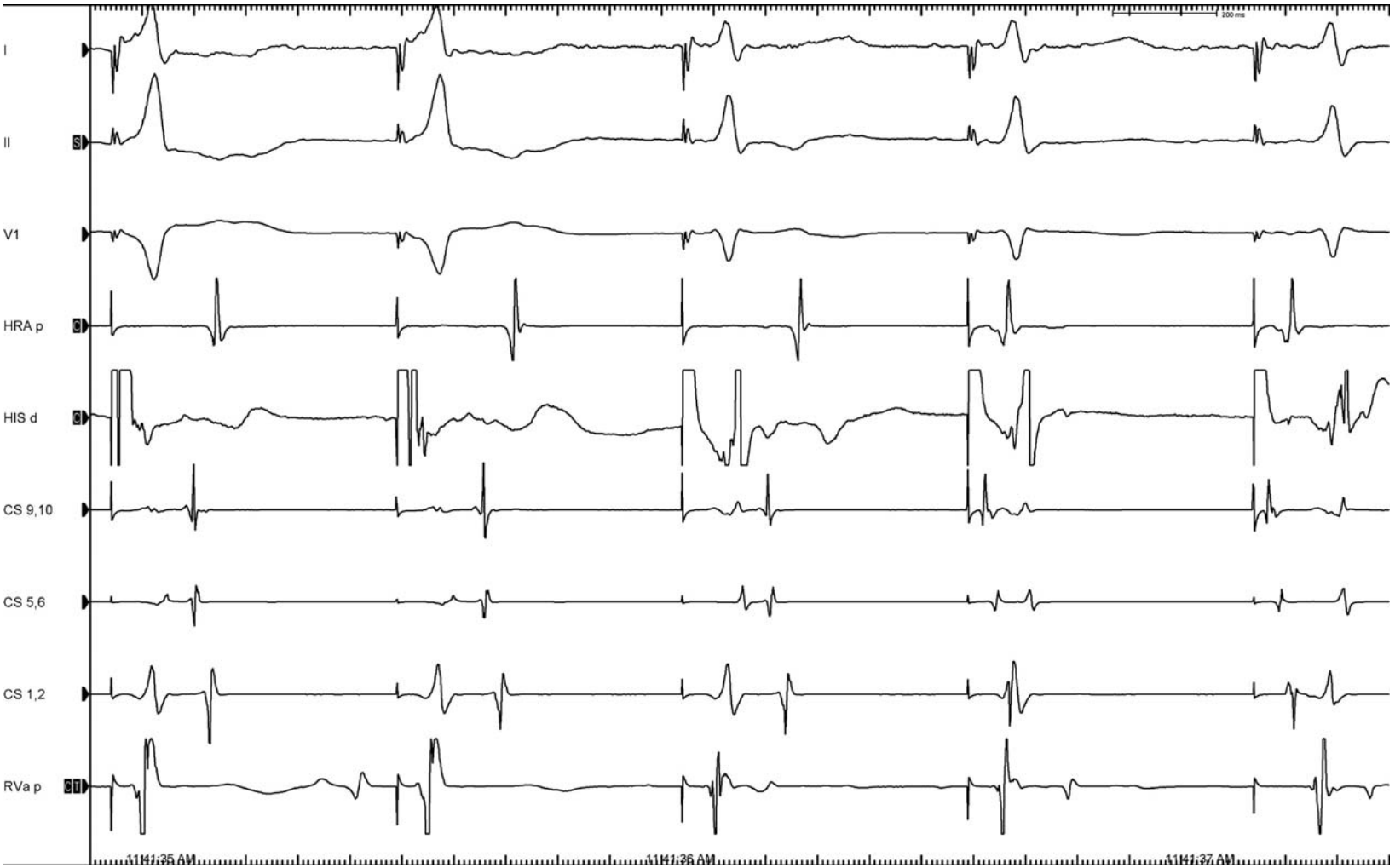
With this background, one can postulate that the atrial extrastimulus conducts to the ventricle and echoes back to the atrium over a left lateral accessory pathway. Conduction then propagates from this left lateral region via the left atrium (not the CS musculature) proximally (first faint line) and then proceeds distally again via the CS musculature (second faint line). There is no apparent communication between the CS muscle and the LA in this individual until the proximal CS region where the two atrial components merge.

The "dual" conduction is hinted at but not readily apparent in sinus rhythm, presumably since conduction is proceeding over pathways providing a fused complex representing both atrial and CS muscle.

It is conceivable that the first of the two atrial components represents an accessory pathway potential but this would then necessitate an extremely long accessory pathway, a little far-fetched. Further, AP potentials generally have a more near-field or "rapid activation" appearance.

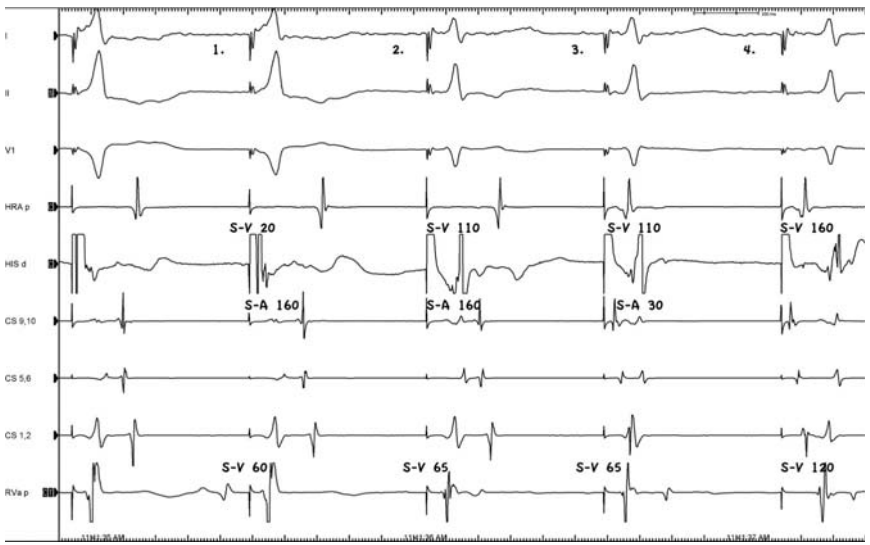
The coronary sinus muscle conduction was considered a bystander in this example and ablation was targeted to the LA endocardium adjacent to CS 1–2, which abolished accessory pathway conduction.

Figure 3–7A



The following was observed during para-Hisian pacing in a patient with supraventricular tachycardia. What can be concluded about retrograde conduction as current strength is diminished with this maneuver?

Figure 3–7B



Explanation:

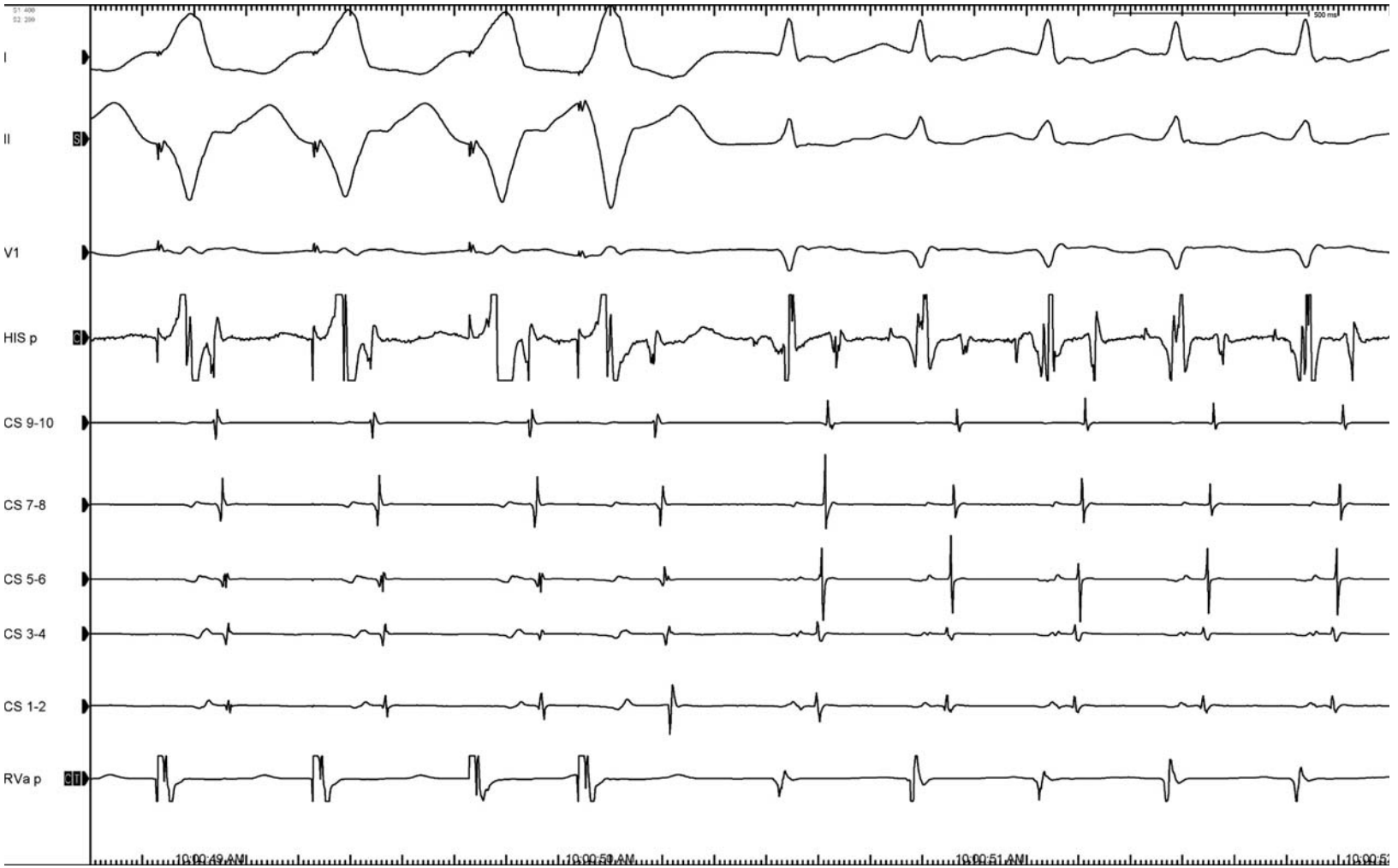
The "checklist" for assessing para-Hisian pacing includes assessing the following:

1. Measure the stimulus to A interval, best at a septal site or orifice of coronary sinus region. A short S–A interval at this site (certainly less than 50 milliseconds) should arouse suspicion of inadvertent atrial capture.
2. Measure the stimulus to V interval at the His bundle electrode (HB) site of pacing. Evidence of capture and effective pacing of the para-Hisian muscle here is indicated by very short S to V interval, in range of 20 milliseconds or less.
3. Measure the stimulus to V interval at the RV apical electrogram, which is generally relatively close to the RBB terminus. This should be relatively shorter with His capture than with only RV para-Hisian muscle capture since the latter has to proceed from the base of the heart to the apex via muscle.

Individual cycles have been labeled 1–4 for clarity of explanation. Cycle 1 by the above criteria shows His and local muscle capture and no direct atrial capture. His capture is also signaled by the relatively narrow QRS. Cycle 2 shows a lengthening of the S–V at the His pacing catheter indicating loss of para-Hisian muscle capture. Pure His capture is indicated by normal QRS morphology and no change in the S–V at the RV apical electrogram. The S–V is about the same as the HV interval in sinus rhythm. Further, the similar S–A interval with pure His capture and His plus direct local muscle capture would be expected with retrograde conduction over the normal AV conduction system. The only change from cycle 2 to 3 is shortening of the S–A interval at the proximal coronary sinus. This signals direct local atrial capture in addition to His capture seen in cycle 2. In cycle 4, the S–A at the proximal coronary sinus again indicates atrial capture but here the S to V becomes later at all sites and this indicates atrial capture only.

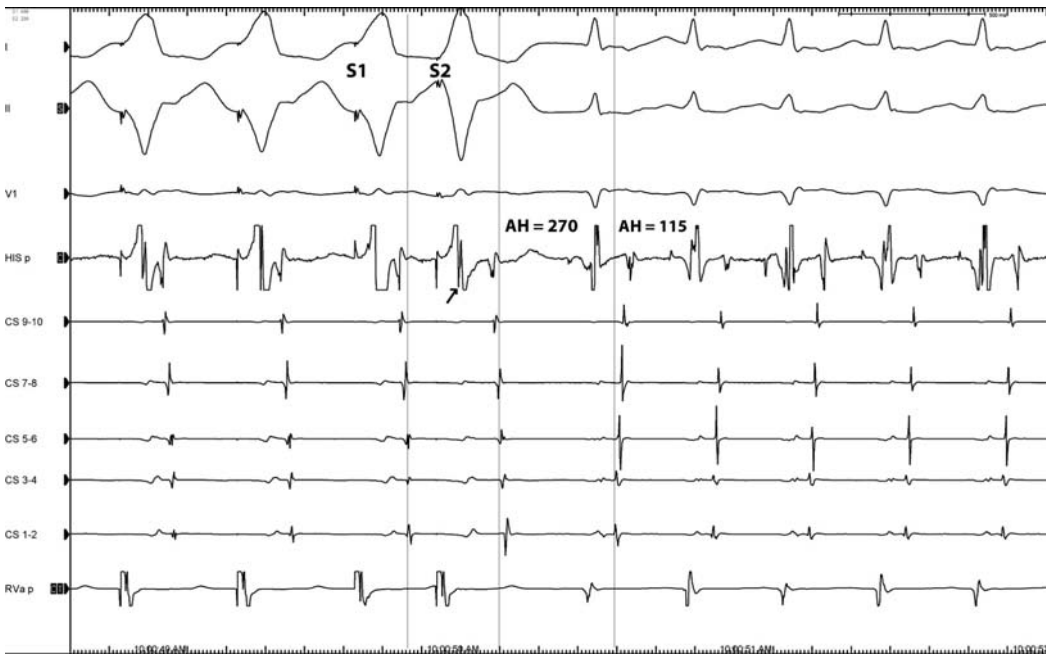
The answer to the original question posed is that this was not helpful in assessing retrograde conduction since His pacing was never lost in the presence of para-Hisian muscle capture.

Figure 3–8A



A narrow QRS tachycardia is induced with a ventricular extrastimulus. What is the mechanism of the tachycardia and how does one explain the induction?

Figure 3–8B

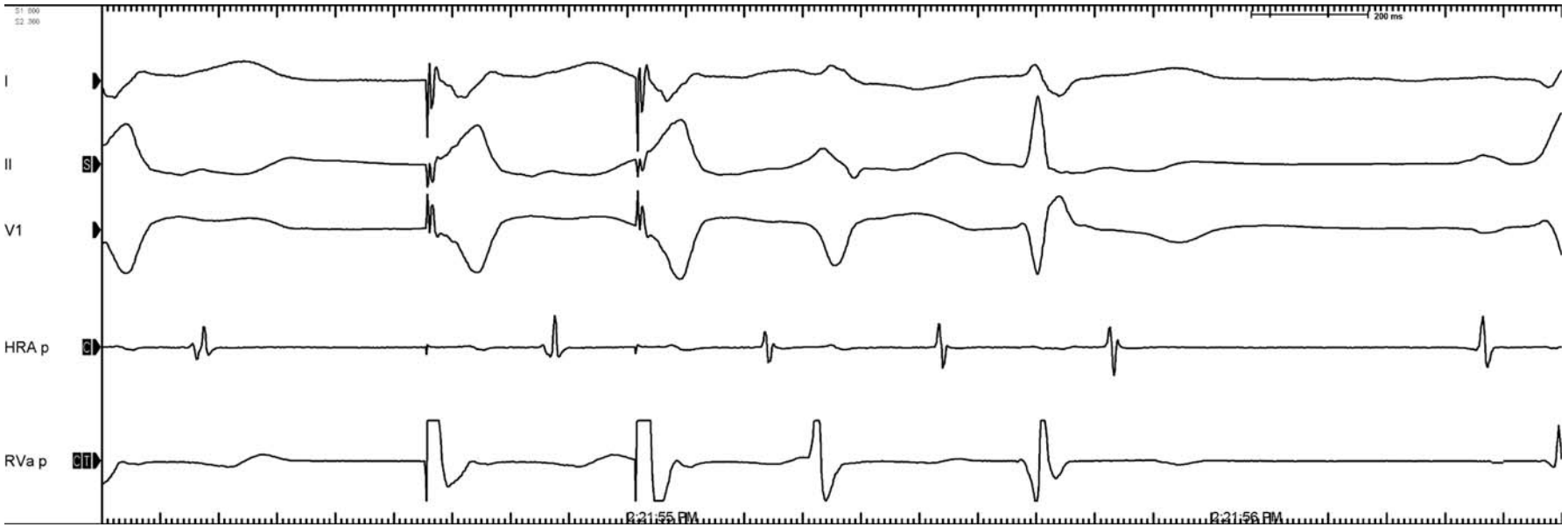


Explanation:

The tachycardia has a normal QRS with normal HV interval and an eccentric atrial activation sequence with earliest atrial activation at the distal coronary sinus. This can only be atrial tachycardia or AVRT over a left lateral accessory pathway. The mode of onset, initiated by a ventricular extrastimulus with the VA interval following the first cycle being the same as subsequent cycles, leaves little doubt that it is AV reentry. This is so even though there is some variability in the initial AH interval. Further, the initiation is with a VAV sequence, characteristic of either AVNRT or AVRT.

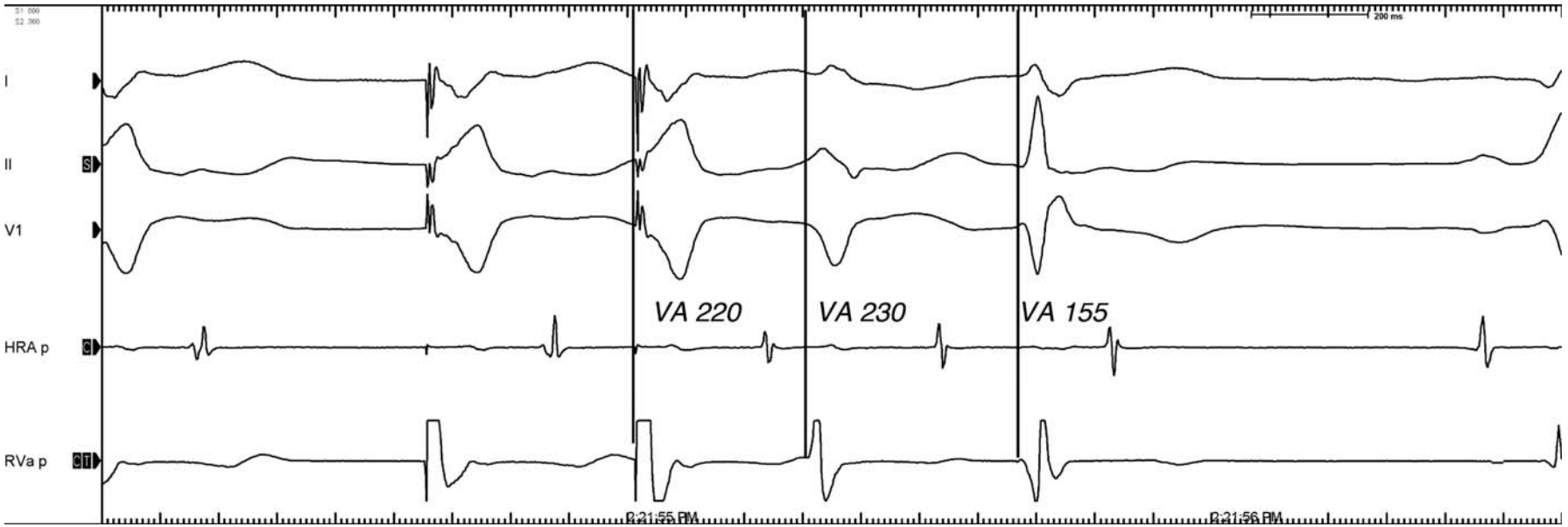
The mode of onset is nonetheless a little unusual. The atrial activation sequence (highlighted by the faint lines) is different for each of the ventricular drive cycle, ventricular extrastimulus, and the tachycardia. During tachycardia, it is clearly eccentric with the distal coronary sinus activating first. The ventricular extrastimulus is clearly "central" with activation first at the HB electrogram. The ventricular drive is very similar but shows activation relatively earlier at the distal coronary sinus. This is most compatible with retrograde fusion over the AV node and the accessory pathway. The ventricular extrastimulus blocks in the accessory pathway and proceeds retrogradely over the AV node with a longer VA interval. A rapid deflection appearing in the middle of the QRS at the His site (*arrow*) may well be a retrograde His deflection, although it is difficult to be sure. Conduction proceeds to "echo" back to the ventricle via a slow AV node pathway (AH = 270 milliseconds) to start AVRT. AVNRT was observed as a separate arrhythmia at other times in the study.

Figure 3–9A



A few extra cycles are observed after a ventricular extrastimulus. What can be inferred from this?

Figure 3–9B



Explanation:

This is an exercise in extracting data from whatever information is available, that is, a "minimalist" approach. The ECG leads suggest that the pacing site is the RV outflow region since lead 1 is negative and V_1 has LBBB pattern.

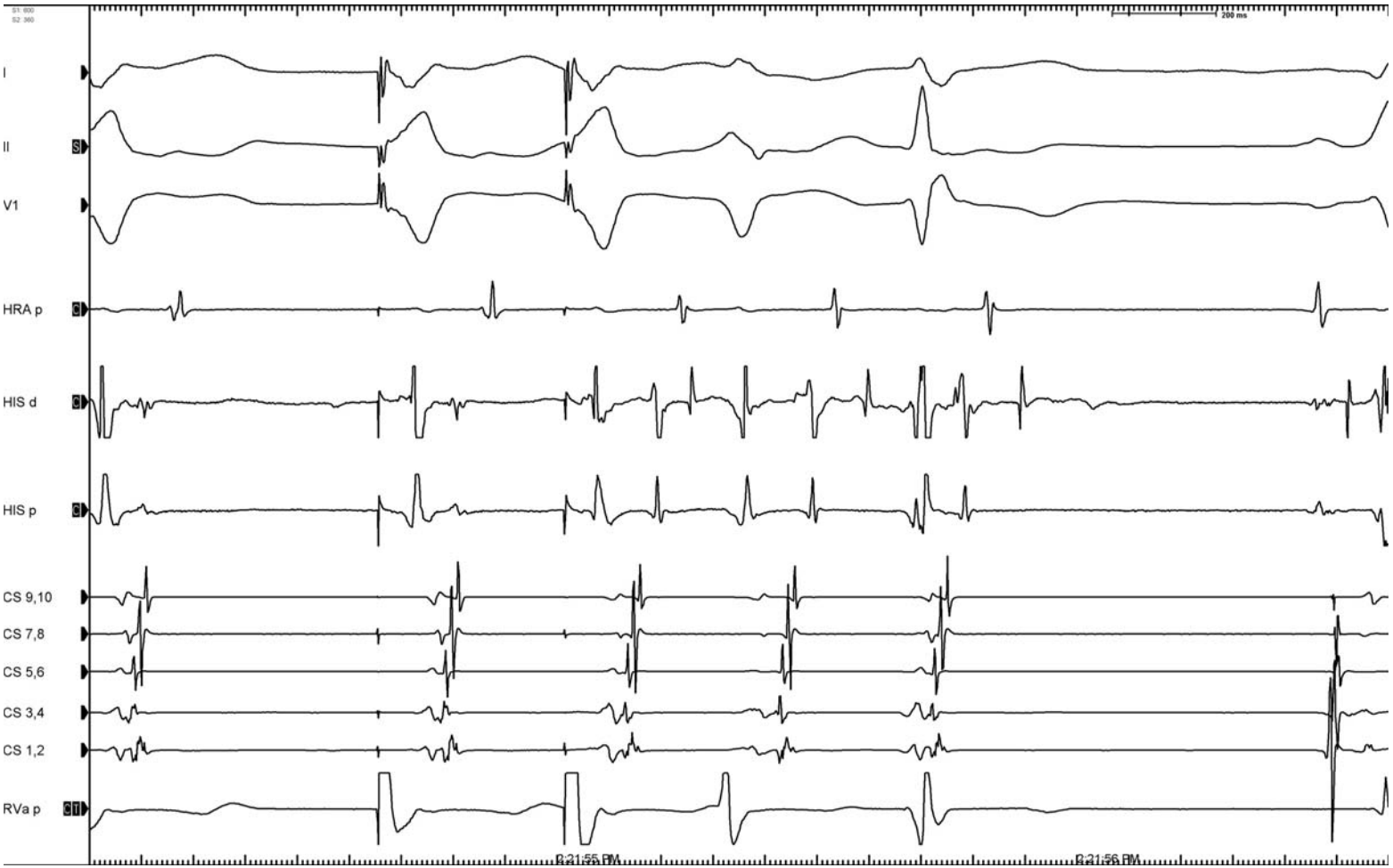
One can consider the two cycles as a nonsustained rhythm. The first cycle may be an echo with LBBB following the A after the ventricular extrastimulus or possibly a repetitive ventricular response (RVR) due to bundle branch reentry. Regardless, the QRS is LBBB type and the very early V in the RV apical electrogram supports this.

The VA interval for this LBBB cycle is 230 milliseconds. The next cycle has right bundle branch block (RBBB) morphology and a VA of 155 milliseconds. This is most compatible with a left lateral accessory pathway since the return cycle to the atrium is much longer (230 milliseconds vs. 155 milliseconds) with the LBBB than with the RBBB cycle.

A prolongation of VA by >30 milliseconds with change to LBBB from normal QRS or RBBB QRS during tachycardia is most compatible with retrograde conduction over a left lateral accessory pathway.

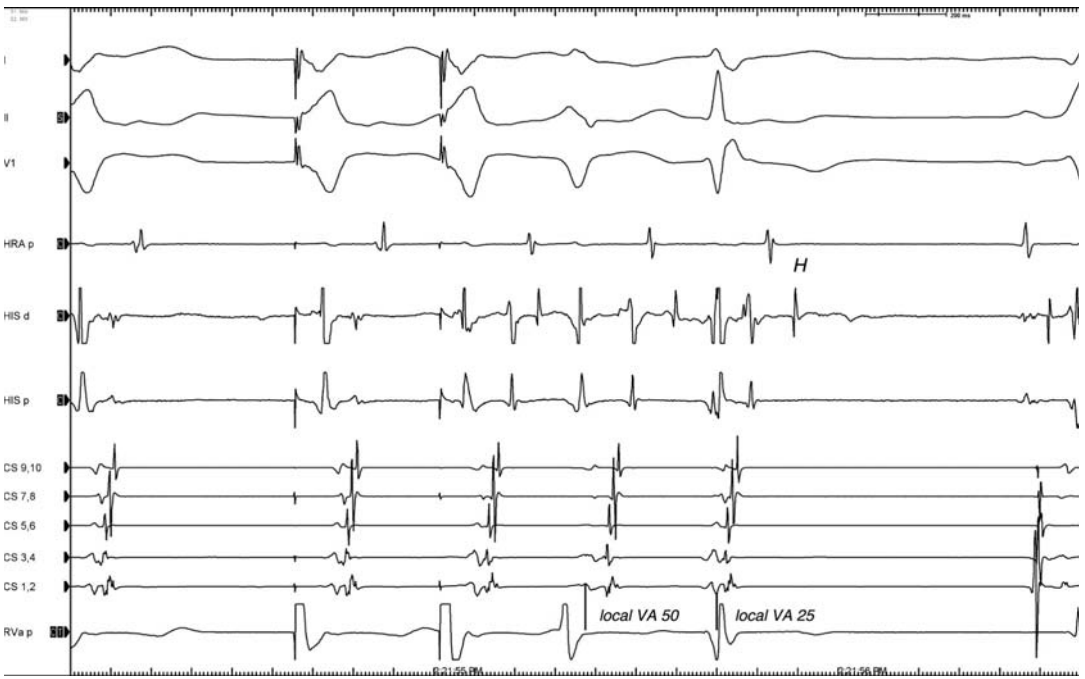
One might also note that termination with an atrial electrogram would not be expected with atrial tachycardia and suggests that anterograde conduction to the ventricle after the atrial cycle is necessary to complete a tachycardia circuit in this individual.

Figure 3–10A



A ventricular extrastimulus V_2 after a drive conducts over a left lateral accessory pathway and two additional cycles are observed, all conducting over the accessory pathway. What is the mechanism of the nonstimulated cycles?

Figure 3–10B



Explanation:

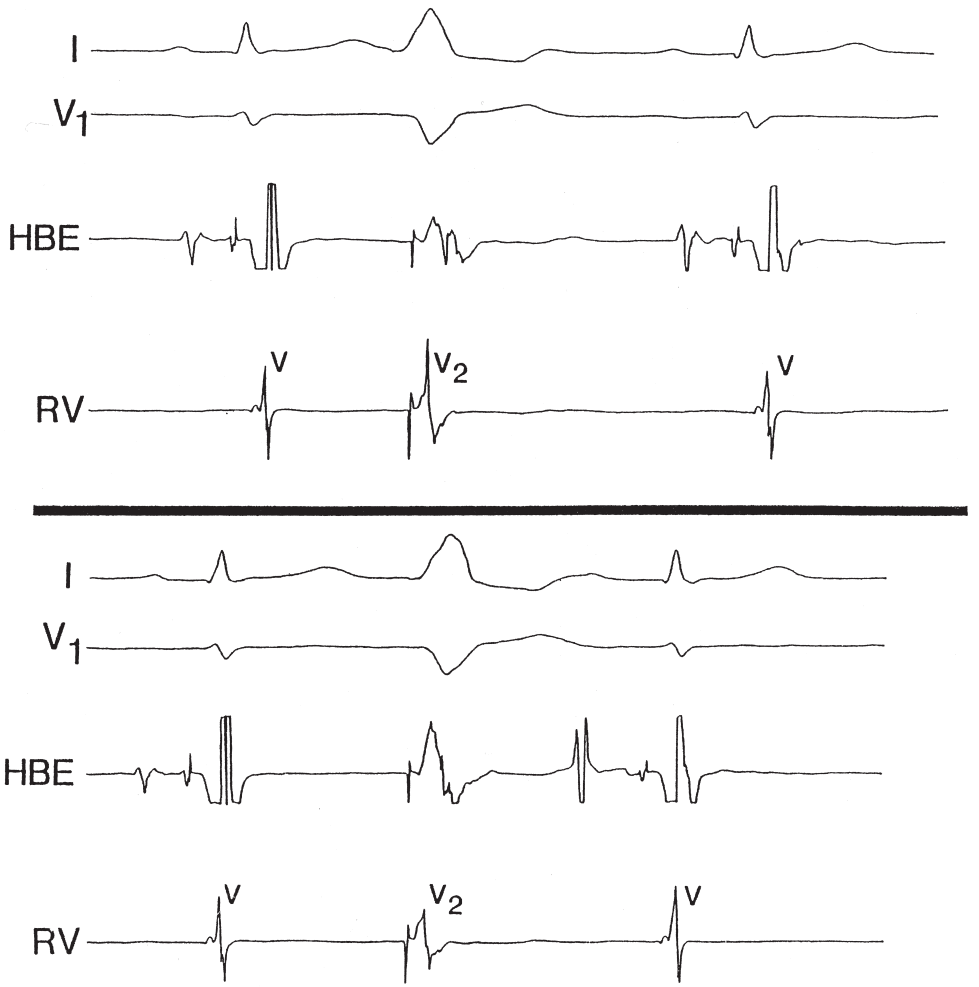
The first cycle has LBBB morphology and the second has RBBB morphology and terminates after a retrograde A. This can be thought of as AVRT that terminates spontaneously. The last event is an anterograde His, very similar to the anterograde His in the sinus cycle at the end of the tracing.

Termination with a His suggests that the His and bundle branch are critical to this short "tachycardia" since block below the His stops it.

The point of interest is indicated by the "local" VA intervals noted at CS 1–2. This is the site of the accessory pathway and corresponds to the site of successful ablation. The "local" VA interval is the interval between the rapid ventricular activation and the rapid atrial activation at the site of the accessory pathway. At the AP site, this represents the conduction time over the AP. *The local VA time in this case lengthens with LBBB, that is, 25–50 milliseconds.* This is best explained by "slanting" of the accessory pathway (i.e., the atrial and ventricular ends are not exactly concordant at the AV ring). If the accessory pathway went directly from ventricle to atrium at the AP site, one would *not* expect the "local" VA interval to change with a change in ventricular activation sequence (i.e., a change in the direction from which ventricular depolarization reaches the accessory pathway).

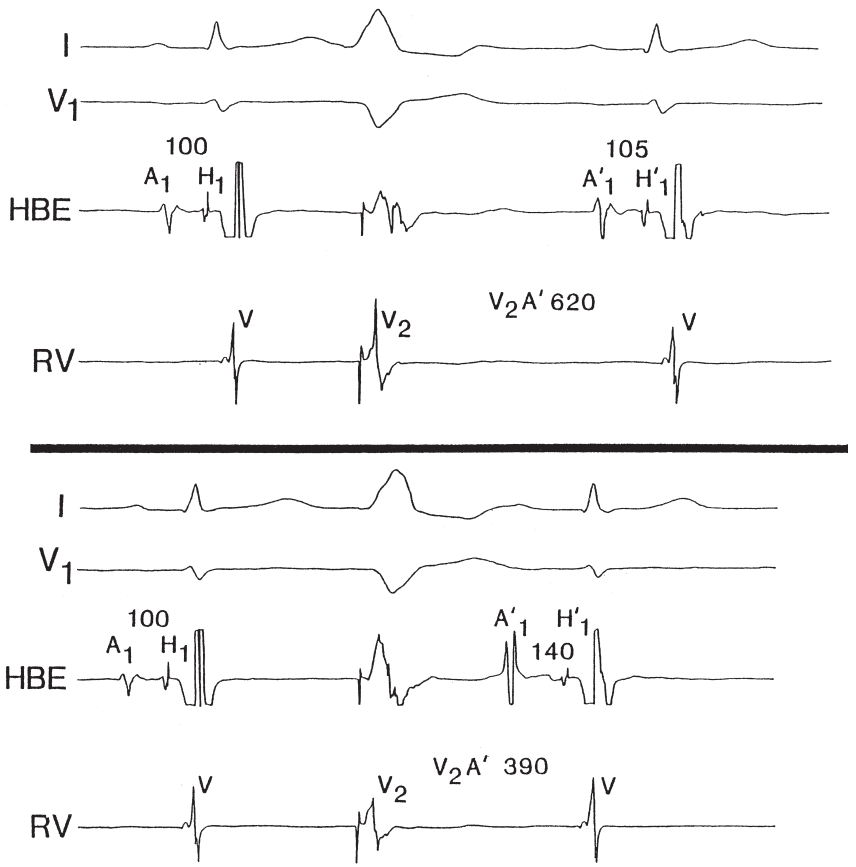
The "local" VA interval is to be distinguished from the (global) VA interval that is from the "onset of ventricular depolarization to the rapid atrial deflection at a designated site."

Figure 3–11A



A series of interpolated PVCs was introduced in this patient during sinus rhythm. What is the effect of the PVC on conduction of the subsequent sinus complex? What is this called? Why is conduction of the sinus complex after the PVC different in the upper and lower panels?

Figure 3–11B



Explanation:

The electrophysiologic phenomenon exhibited is *concealed conduction*. Concealed conduction occurs because the PVC conducts retrogradely into the AV node but not to the atrium, and conduction into the AV node is not visible (concealed). The result is prolongation of AV nodal refractoriness. The sinus complex after the PVC has an increase of the AH interval from 100 to 105 milliseconds (upper panel). This increase in AH interval would likely be missed during analysis of the surface electrocardiogram performed at 25 mm/s paper speed. It is, however, demonstrated during intracardiac measurements performed at faster paper speeds. Conduction of the PVC into the AV node is confirmed because of the subsequent prolongation of AV node conduction.

The effects of concealed conduction are more prominent in the lower panel. Note that the AH interval after the PVC is 140 milliseconds compared with 105 milliseconds in the upper panel. The longer AH interval occurs because the PVC is introduced later in diastole and consequently closer to the next sinus complex. There is less recovery of AV nodal excitability as a consequence. Note that the V_2A' in the upper panel is 620 milliseconds but only 390 milliseconds in the lower panel. This demonstrates the well-known inverse relationship of the effect of PVCs on PR and RP intervals as identified in the electrocardiogram. In other words, as a PVC occurs closer to the next sinus P wave (short RP), there is a more marked effect on subsequent PR prolongation or even block. This relationship is identified in Fig. 3–11*C*. As $V_2A'_1$ shortens, $A'_1H'_1$ progressively lengthens until block of the subsequent sinus complex occurs.

Figure 3–11C

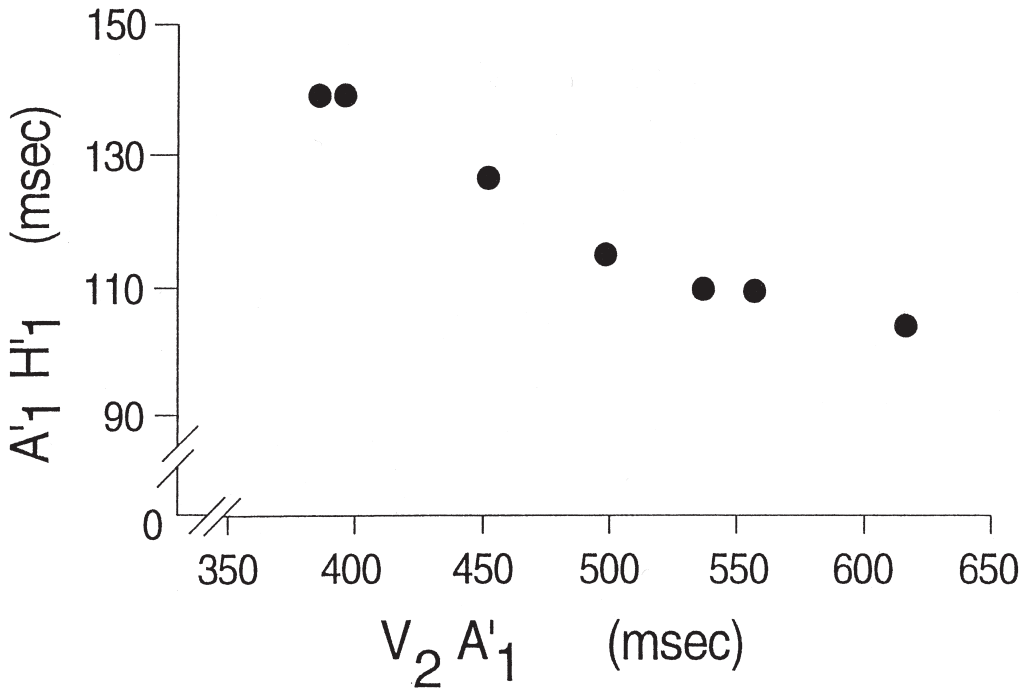


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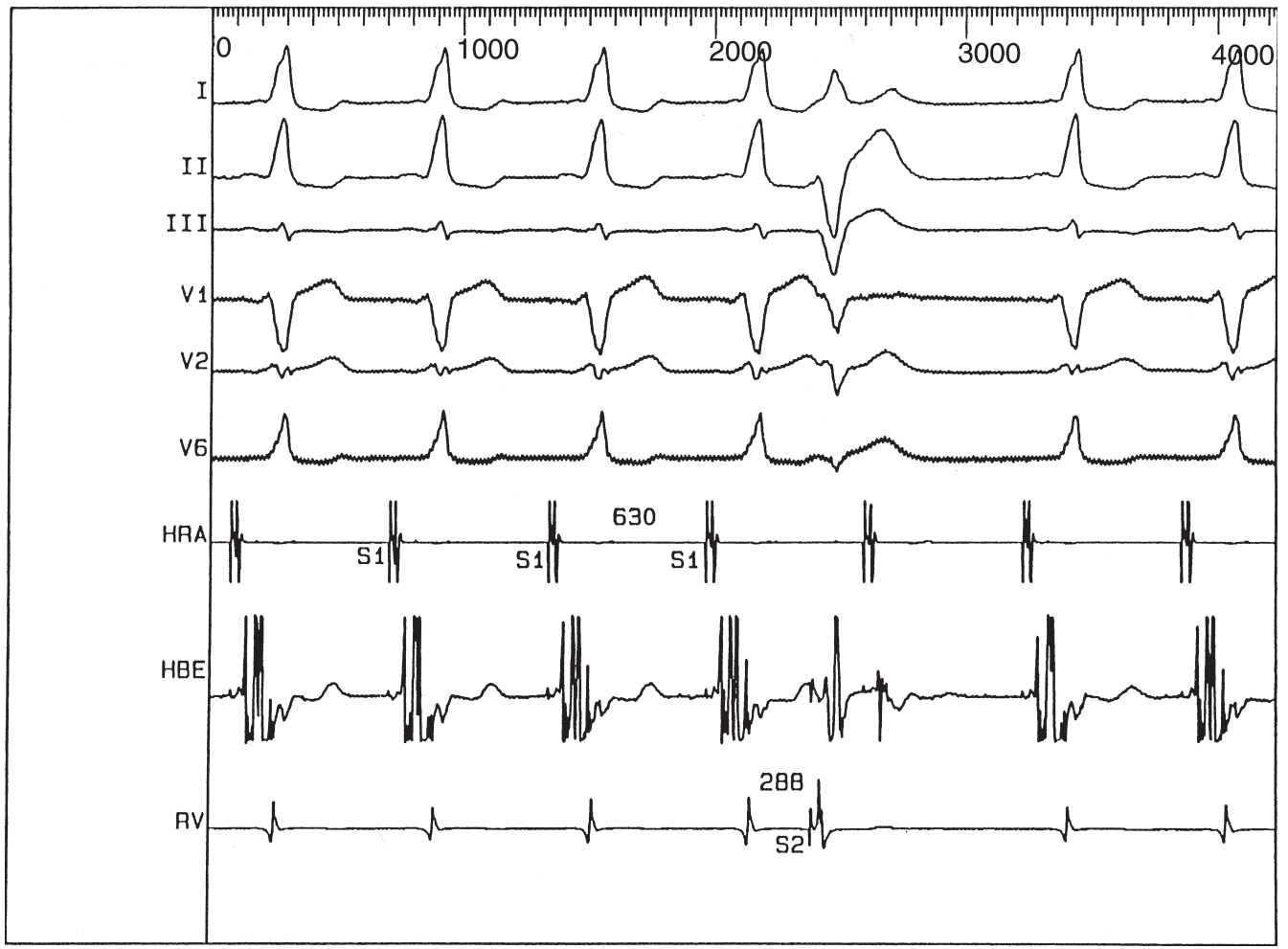
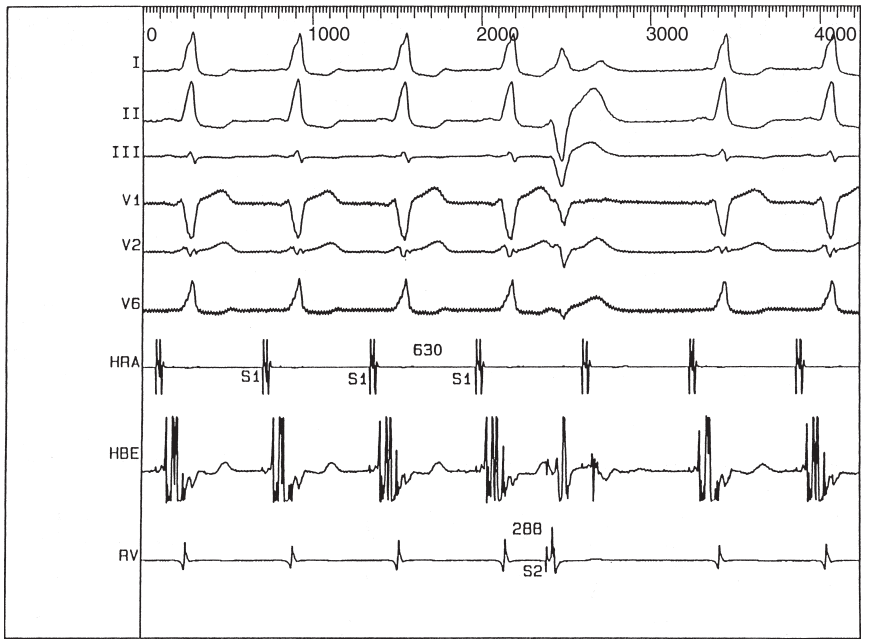
Figure 3–12A



This patient with a history of tachycardia had a PVC introduced during atrial pacing at CL 630 milliseconds. What EP phenomenon occurs after the PVC? What is the most likely cause of this patient's tachycardia?

Figure 3–12B

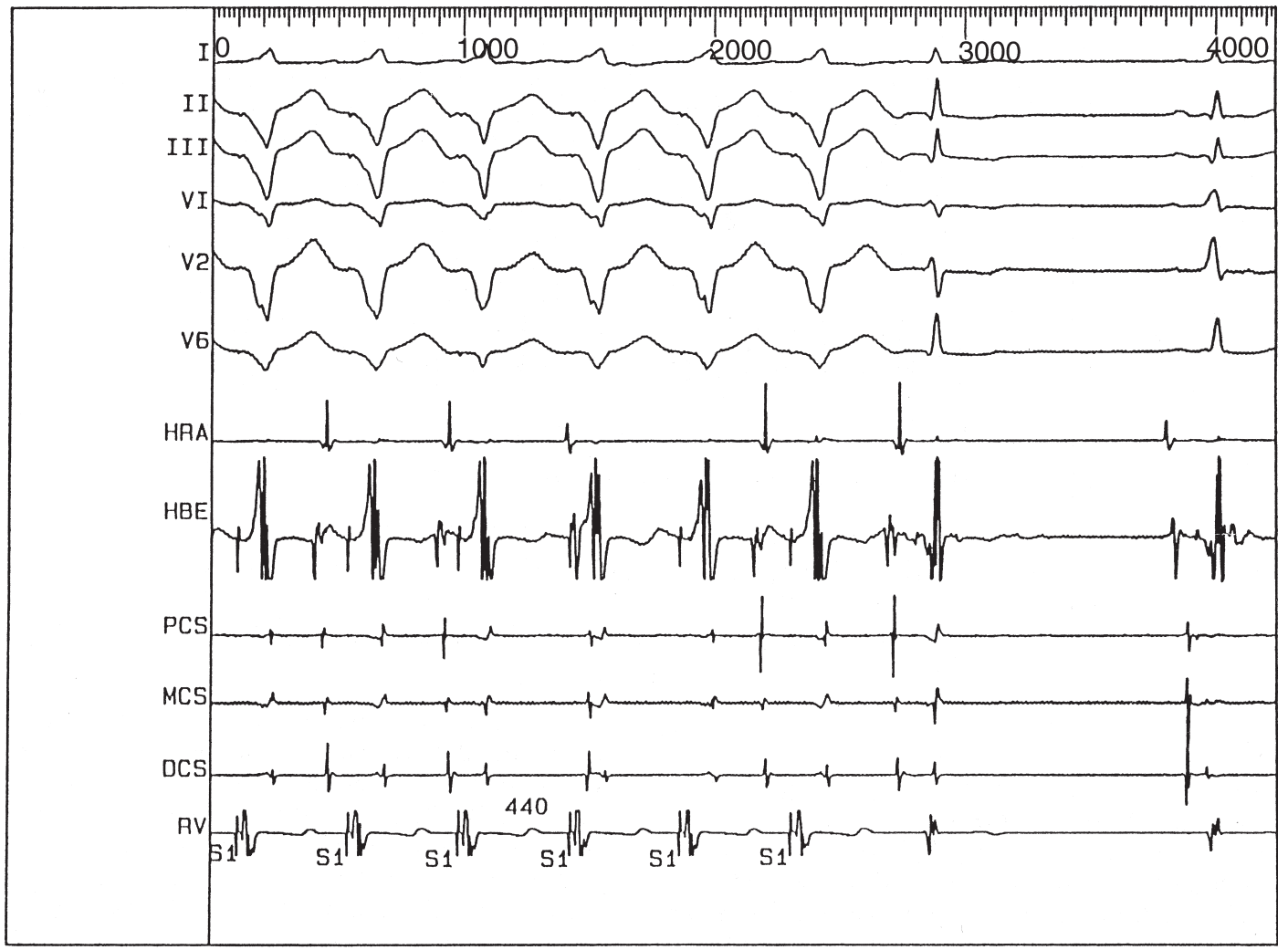


Explanation:

During atrial pacing the ECG leads demonstrate a relatively short PR interval with a wide QRS complex. Analysis of the His bundle electrogram reveals continuous electrical activity with the atrial and ventricular electrograms appearing to merge into one another. No clearly defined His deflection is seen. These two observations strongly imply the wide QRS complex is due to ventricular preexcitation with conduction over an AP. The fact that there is almost continuous electrical activity identified on the His bundle electrogram suggests, but does not prove, the AP is in the vicinity of the His bundle recording site. Statistically, the most likely tachycardia would be AV reentry, although patients with ventricular preexcitation are not immune to other forms of tachyarrhythmias.

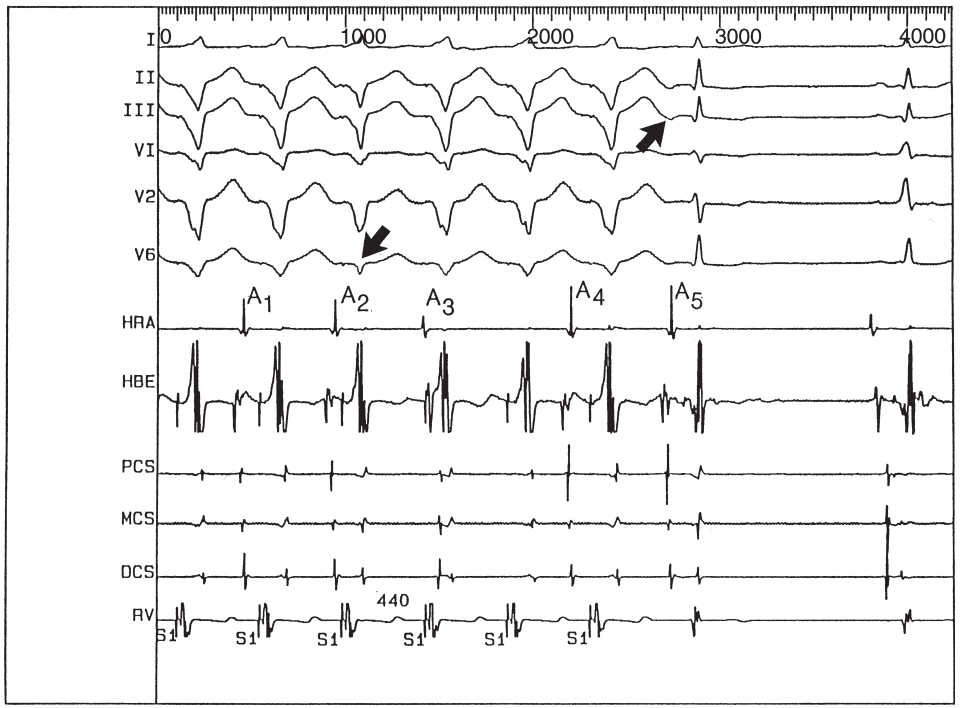
The subsequent paced atrial complex after the PVC (S_2) does not conduct to the ventricle. Thus, retrograde concealed conduction has occurred not only into the normal VA conduction system but also into the AP. Otherwise, one would have expected the atrial paced complex to conduct over the AP, which does not occur. A much less likely but alternative explanation for lack of AP conduction is refractoriness of the ventricle after the PVC. We think this is extremely unlikely since the anticipated ventricular activation over the AP would have occurred more than 300 milliseconds after the PVC was introduced. There should be more than sufficient time for ventricular repolarization to have occurred, and the ventricle should be excitable if the AP is capable of conduction.

Figure 3–13A



Retrograde Wenckebach block occurs during ventricular pacing at 440-milliseconds CL. What is the likely mechanism for block?

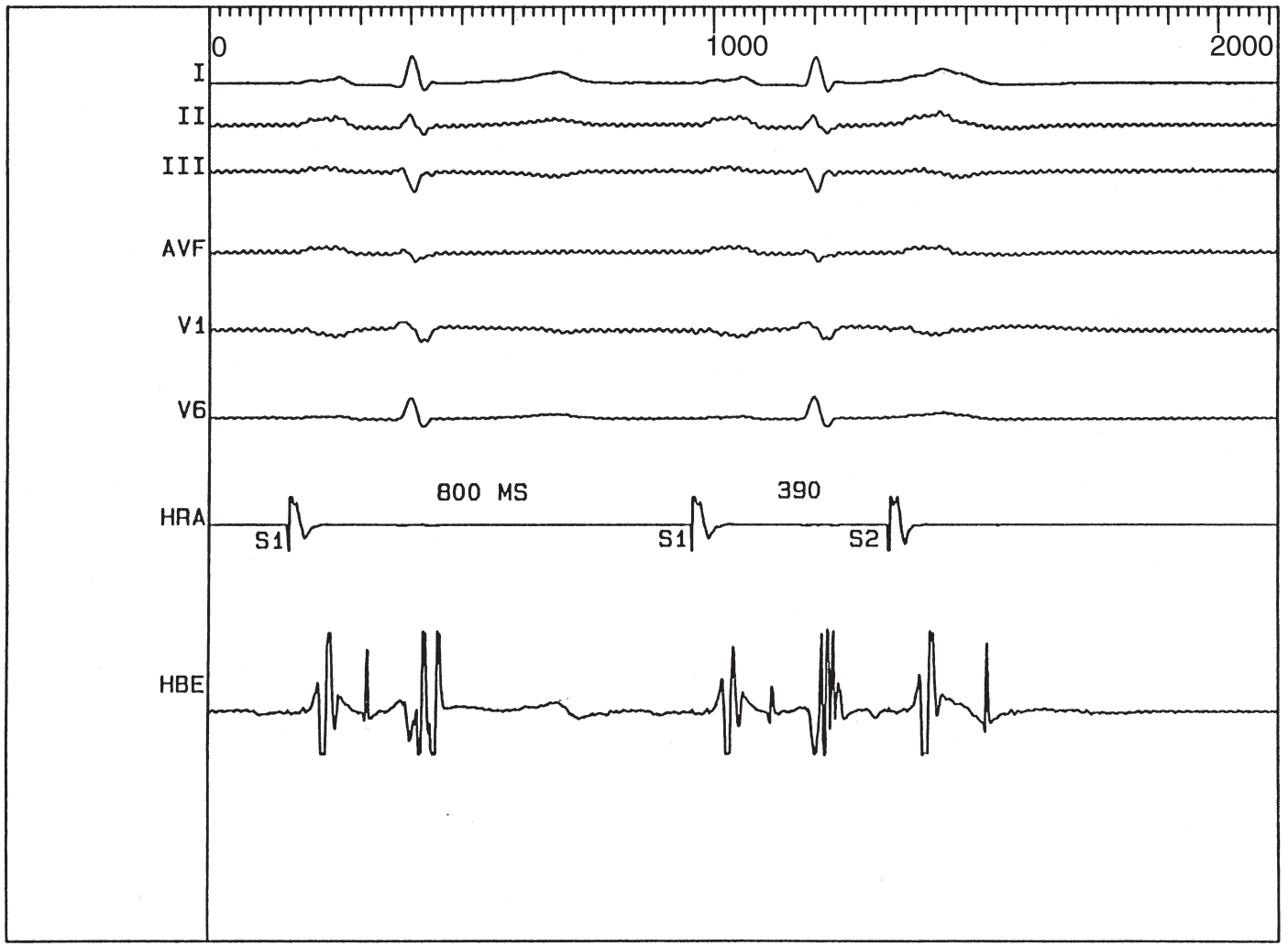
Figure 3–13B



Explanation:

Apparent Wenckebach VA block occurs because of reentry in the AV node. Analysis of the surface electrocardiogram reveals a shortened QRS duration following A_2 (*arrow*). This ventricular fusion occurs because a portion of the ventricle is activated by an electrical wave front differing from that produced during pacing alone. The third paced ventricular complex is also associated with VA block. A_3 has a different intracardiac activation sequence from A_1 and A_2, and the high right atrial electrogram precedes the septal atrial electrogram noted in the His bundle lead. This may be a premature atrial complex or less likely sinus capture. Analysis of A_4 and A_5 reveals the AV nodal echo complex. Note the marked increase in VA conduction time for A_5 with a retrograde P wave (*arrow*) in ECG lead III. Pacing was terminated and the echo became manifest as the ventricle was activated with a narrow QRS complex. Anterograde conduction of this echo complex precludes retrograde conduction over the AV node and therefore block occurs. The fusion complex noted by the arrow in ECG lead V_6 can be explained by ventricular activation over the normal AV node/HPS and the ventricular paced complex. Note that A_2-QRS and A_5-QRS conduction times are very similar.

Figure 3–14A



Figures 3–14*A* and 3–14*B* were recorded during an EP study of a patient with syncope. What EP phenomenon is present? Does this patient need a pacemaker?

Figure 3–14B

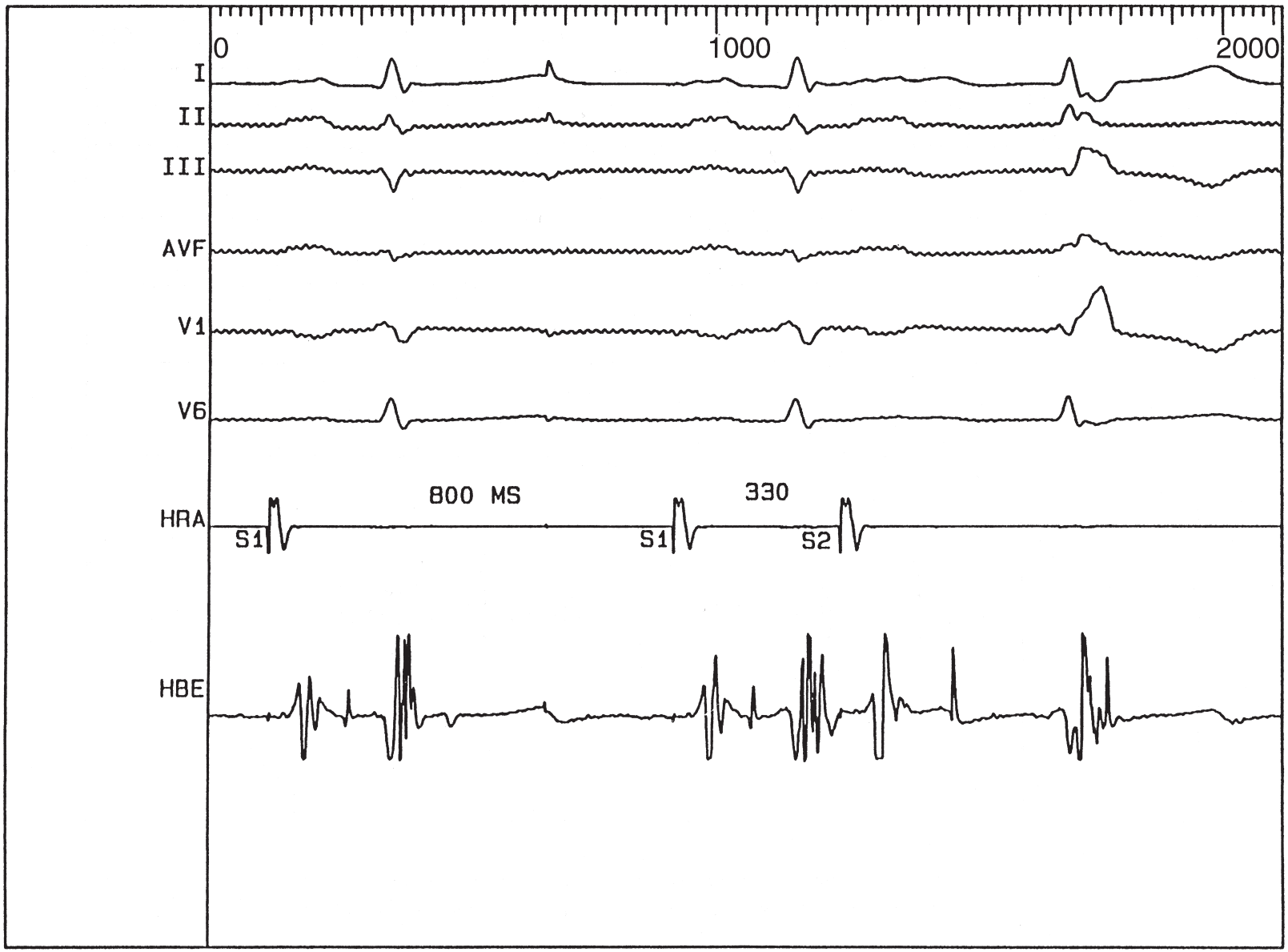


Figure 3–14C

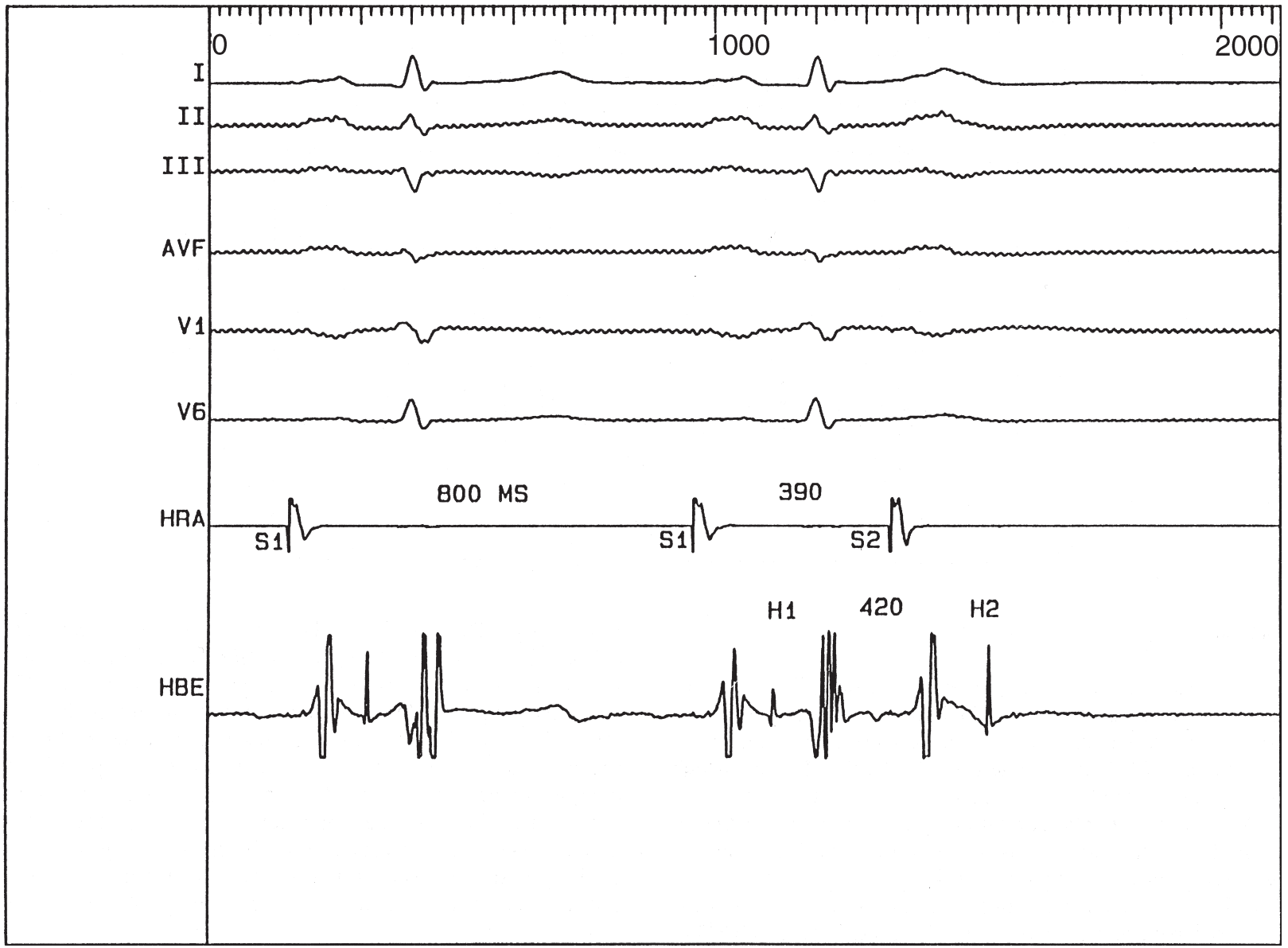
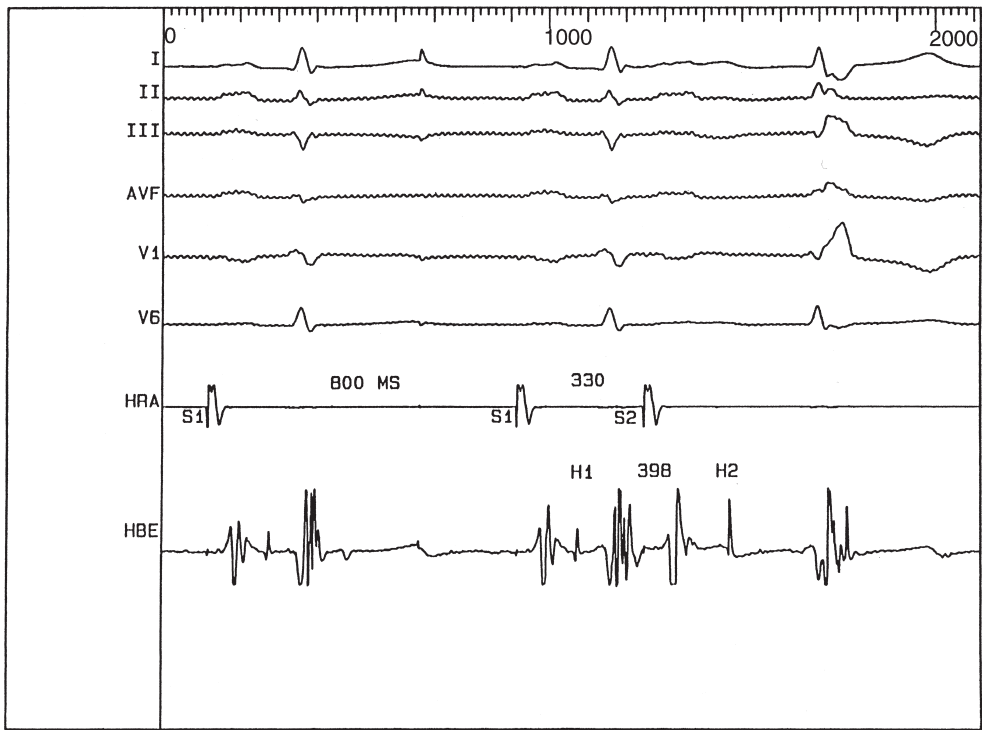


Figure 3–14D

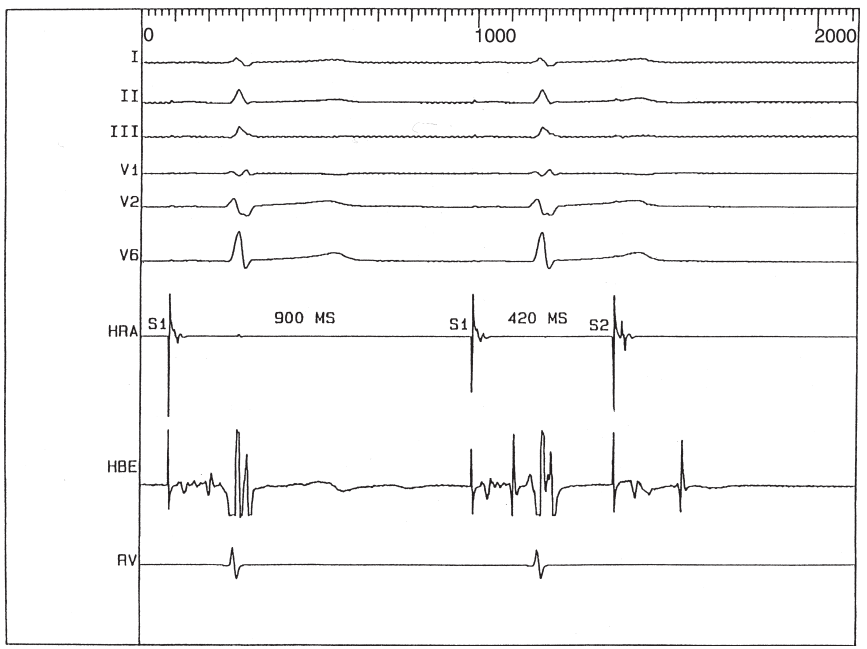


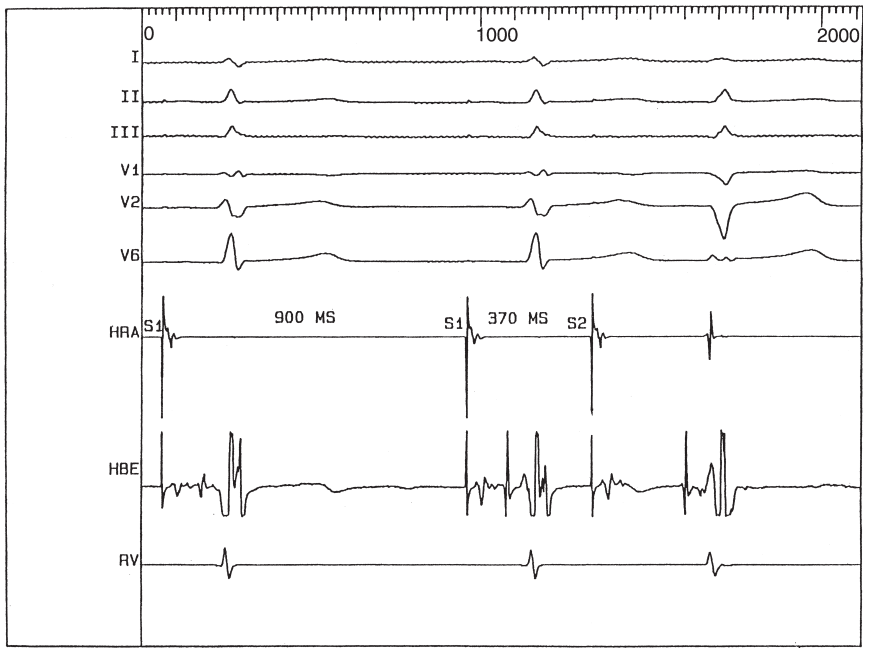
Explanation:

The electrophysiologic phenomenon is conduction GAP. Figure 3–14*C* demonstrates block below the His bundle recording site of a premature atrial complex of 390 milliseconds. A narrow QRS complex occurs during atrial paced CL 800 milliseconds. The H_1H_2 interval with block is 420 milliseconds, which is probably within normal limits at this heart rate. This patient has excellent AV nodal conduction and therefore a relatively short H_1H_2 interval could be achieved. This is not an abnormal finding and is certainly not an indication for a pacemaker.

In Fig. 3–14*D*, a shorter premature atrial complex of 330 milliseconds conducts to the ventricle with RBBB morphology. The H_1H_2 interval is shorter in Fig. 3–14*D* compared with that in Fig. 3–14*C*. Note the marked prolongation of His–Purkinje conduction time (H_2V) in this complex compared with the normal His–Purkinje conduction time of S_1. Conduction at the shorter premature atrial interval and block at the longer premature atrial interval is an example of gap physiology. Gap occurs because the more premature atrial complex exhibits slower conduction proximal to the previous site of block; this allows subsequent recovery of excitability of that area and conduction to the ventricle. In this instance gap occurred in the HPS as noted by block initially after the recorded H_2 potential. Subsequent conduction has marked slowing of infra-His conduction time.

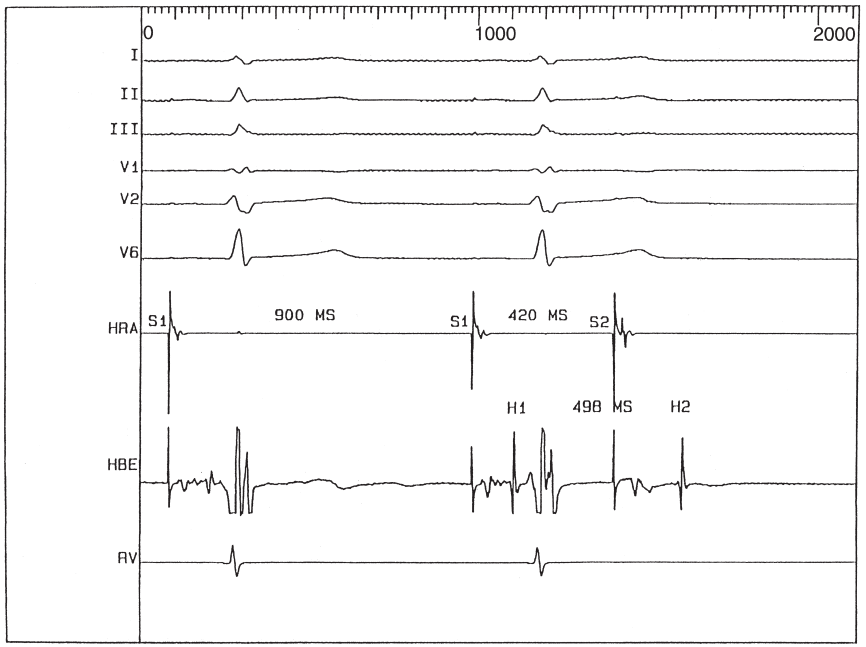
Figures 3–15A and 3–15B

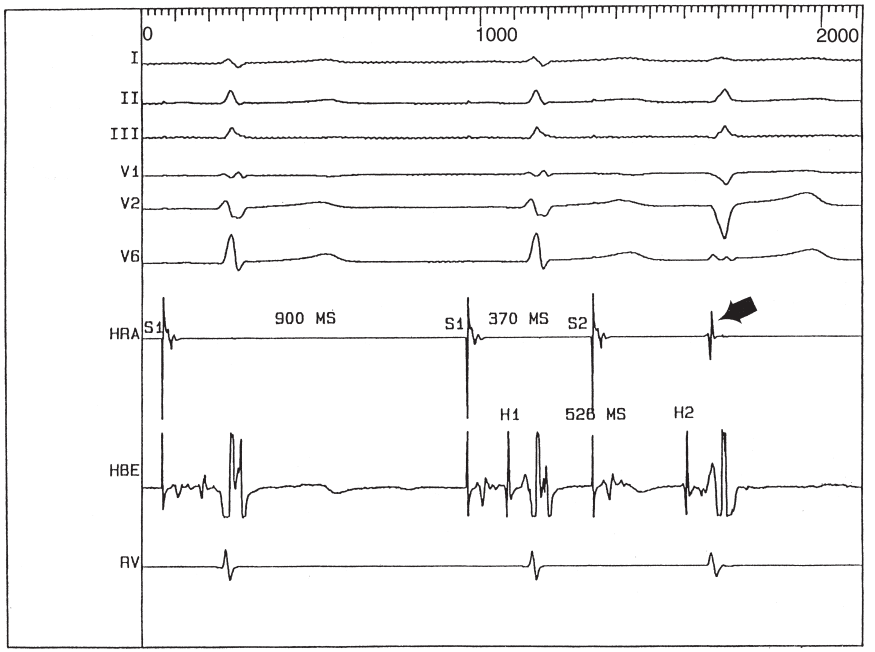




This patient was being studied because of SVT. What EP phenomenon occurs and what is the likely cause of tachycardia?

Figures 3–15C and 3–15D

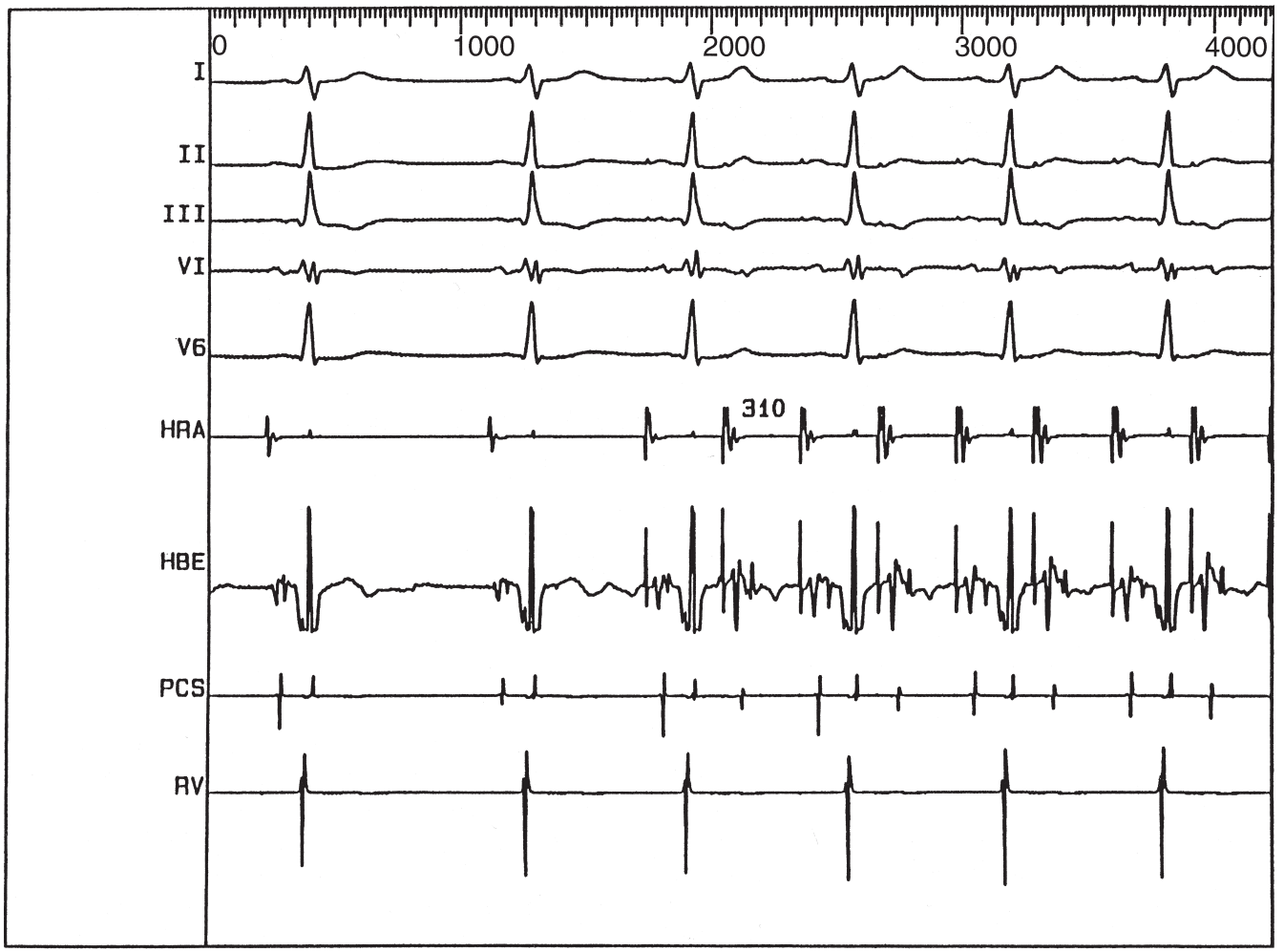




Explanation:

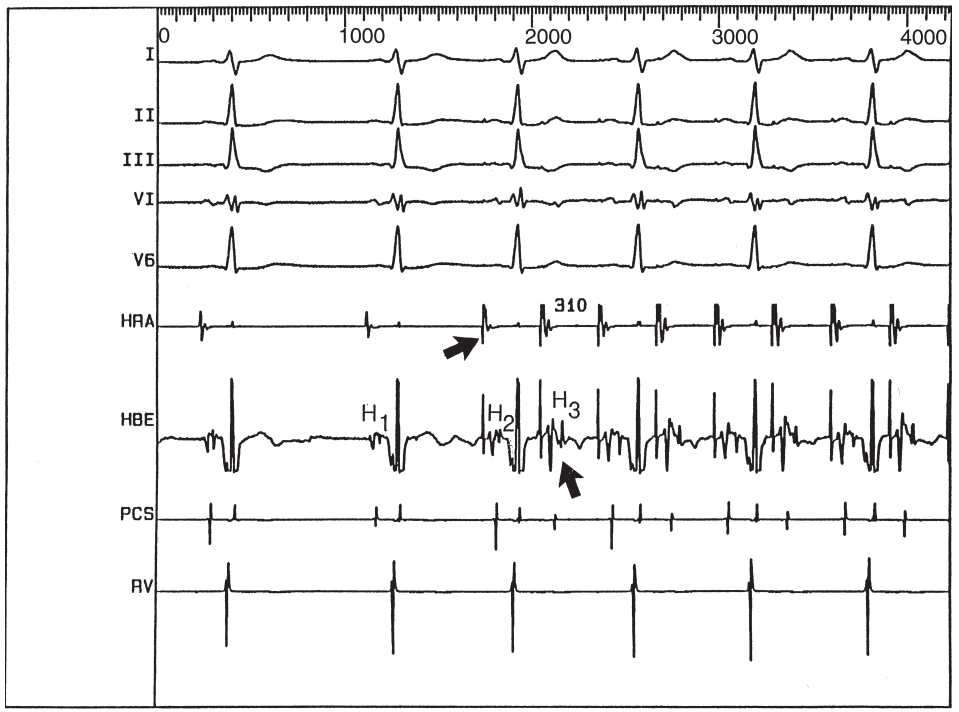
During atrial pacing at a slow rate of 900 milliseconds, a premature atrial complex of 420 milliseconds blocked below the His bundle recording (Fig. 3–15*C*). Note that H_1H_2 is 498 milliseconds, which is relatively long. Regardless, block below the His bundle recording in this situation should not be considered abnormal and does not require pacemaker implantation. In Fig. 3–15*D*, there is resumption of conduction with a shorter premature atrial coupling interval of 370 milliseconds. However, there is a substantial prolongation of H_1H_2 interval to 526 milliseconds and the conducted QRS complex has a relatively normal HV interval with incomplete LBBB. Thus, there is some delay in conduction in the left bundle branch system. This example of gap occurs in the AV node, with resumption of conduction resulting primarily from an increase in AH interval of the S_2. Note also the retrograde atrial complex (*arrow*). This is an AV nodal echo and strongly suggests that the patient has AV node reentry as the cause of tachycardia. This echo complex did not conduct subsequently to the AV node, but AV node reentry was easily initiated during isoproterenol infusion that allowed facilitation of anterograde AV node conduction.

Figure 3–16A



This patient underwent EP study because of syncope. Atrial pacing was initiated at a relatively short CL of 310 milliseconds. Where does block occur? Is this abnormal? Does the patient require pacemaker therapy?

Figure 3–16B

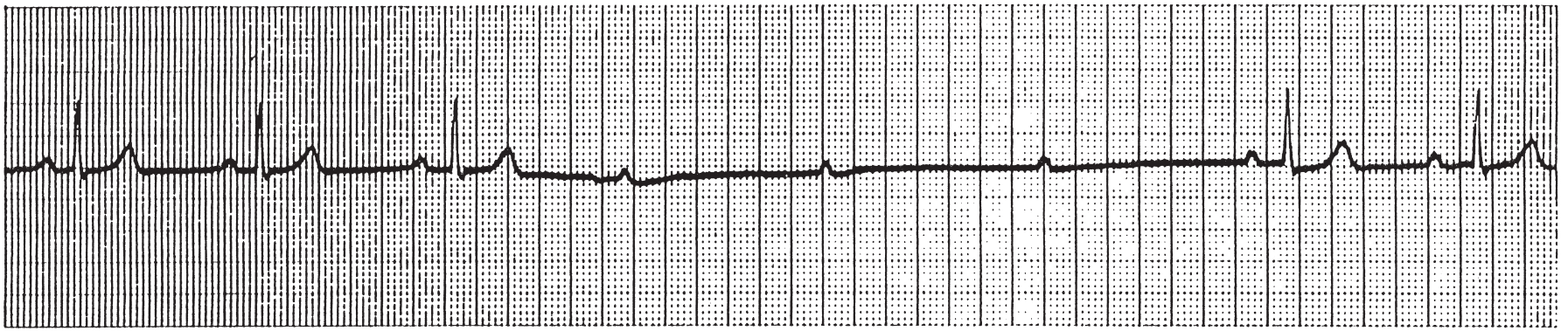


Explanation:

This patient has a relatively slow sinus CL of approximately 900 milliseconds. Atrial pacing is initiated suddenly at a cycle length of 310 milliseconds. The first paced atrial complex (arrow on the HRA lead) conducts to the ventricle. The H_1H_2 interval noted on the His bundle lead is still relatively long and conduction occurs without any problem. The second atrial complex blocks below a recorded His potential (*arrow*; H_3). The H_2H_3 interval is short and follows a relatively long H_1H_2 interval. Then, 2:1 block occurs below the recorded His potential for the rest of this tracing. This is a normal finding that happens because of the preceding slow CL before rapid atrial pacing. The long CL results in prolongation of His–Purkinje refractoriness, and the short H_2H_3 interval occurs within the absolute refractory period of the HPS. Thus, block occurs below the recorded H_3 deflection, a normal finding. A pacemaker is not required. In this same patient, a gradual increase in atrial pacing rate to CL 310 milliseconds produced 1:1 AV conduction. This was due to the progressive shortening of His–Purkinje refractoriness as the rate was incrementally increased, rather than an abrupt increase in heart rate.

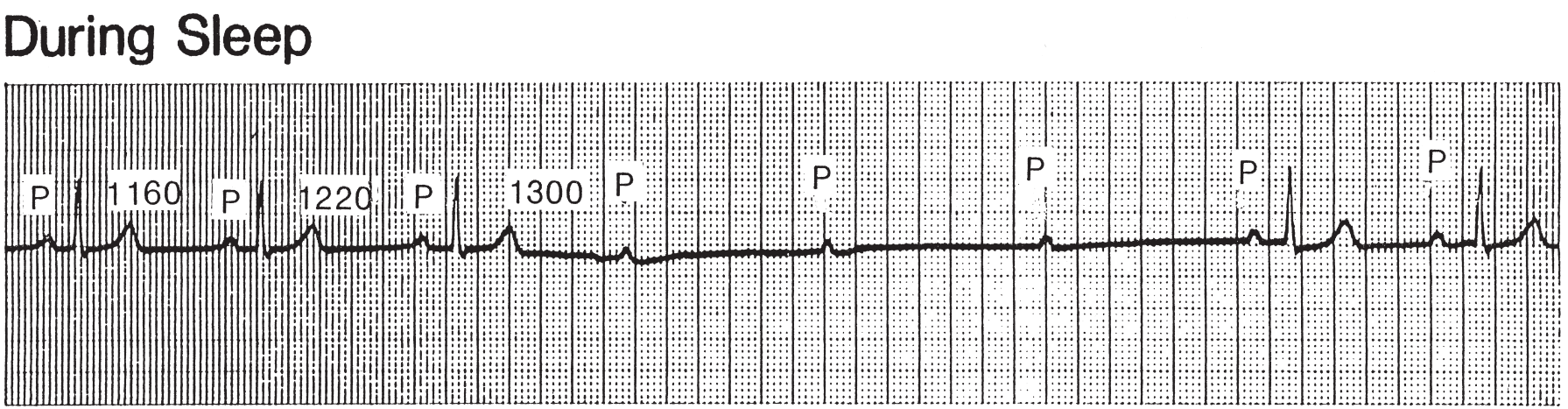
Figure 3–17A

During Sleep



This tracing occurred during in-hospital telemetry monitoring. There is no history of syncope or presyncope. Does this patient require electrophysiologic testing? Should a pacemaker be implanted?

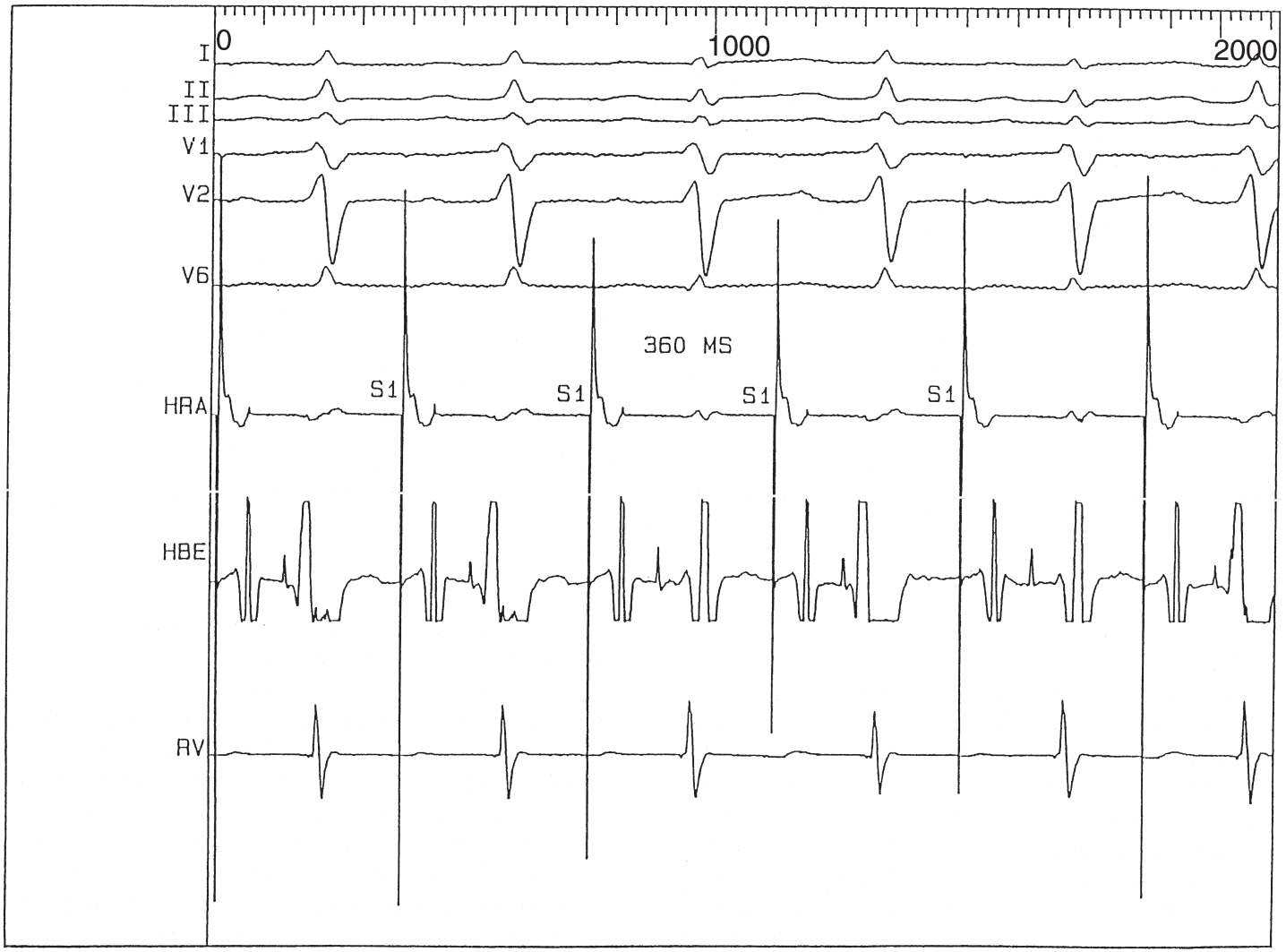
Figure 3–17B



Explanation:

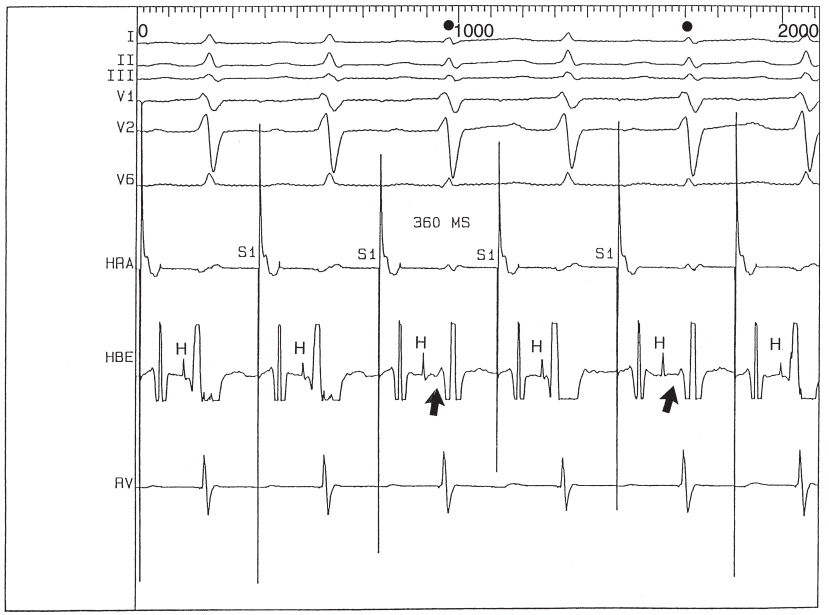
This recording was made while the patient was sleeping. Note that there is progressive increase in the P–P interval until block occurs. In addition, there is the unusual finding of several blocked P waves prior to a junctional escape or conducted complex, a distinction impossible to determine from the data presented. The progressive prolongation of P–P intervals prior to a nonconducted P wave strongly suggests a vagal mechanism as the cause of AV block. The heightened vagal tone could explain both slowing of sinus node automaticity and AV node block. Although it cannot be proven by this tracing, block invariably occurs in the AV node in this situation. This is a relatively common occurrence during Holter recordings or in-hospital telemetry monitoring. Distinctly unusual is the high-grade AV block noted in this patient. However, in the absence of any symptoms of syncope or presyncope, this patient does not require EP testing or an implanted pacemaker. A workup for sleep apnea may be useful.

Figure 3–18A



This tracing was taken during an EP study during atrial pacing at CL 360 milliseconds. What is the diagnosis?

Figure 3–18B

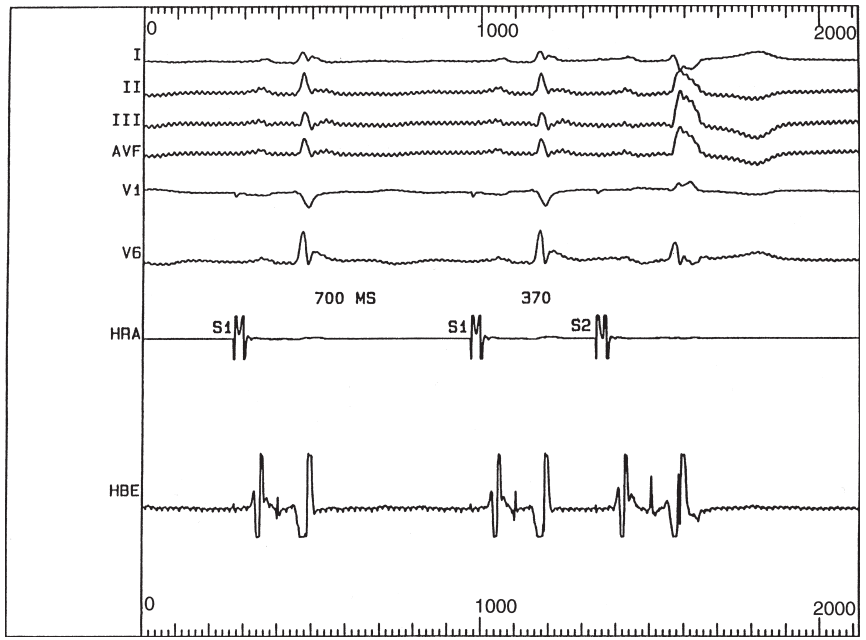


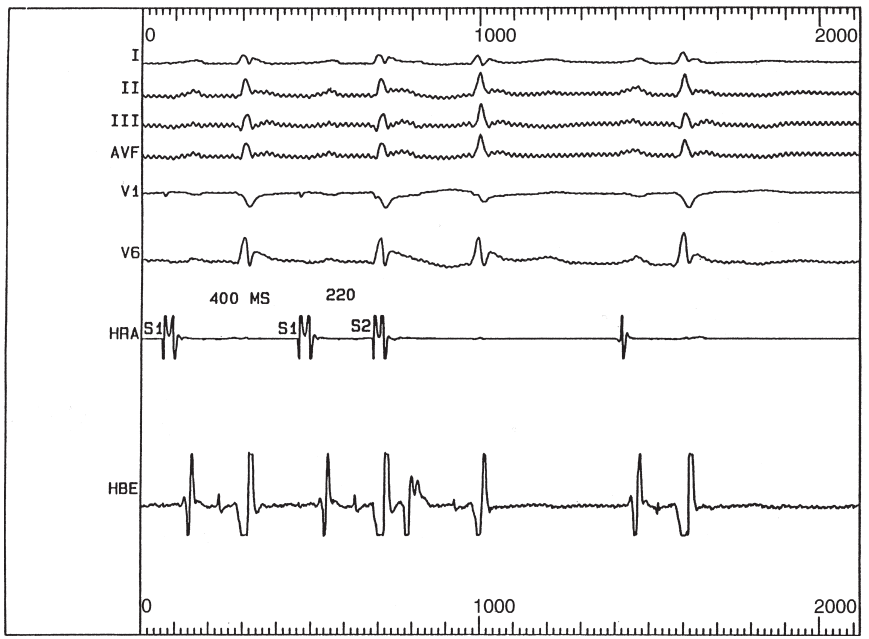
Explanation:

This patient had SVT due to AV reentry using an AP for retrograde conduction. After successful ablation of the AP a repeat EP study was performed. During incremental atrial pacing there was a sudden change in the AV interval as noted in Fig. 3–18*B*. Note that the arrow reveals a sudden lengthening of the HV interval that occurred in a 2:1 conduction pattern for several complexes. In addition, the QRS complexes with a filled dot on top reveal a different morphology from the rest of the QRS complexes. This is a very unusual finding, and the shorter HV intervals without the arrow occur because of conduction over a fasciculoventricular AP. This diagnosis is made by the subtle preexcitation noted in the QRS complexes associated with a shorter than expected HV interval. The degree of preexcitation is minimal since the ventricle is activated over an AP that connects the bundle branch system with the ventricle. Thus, there is minimal time to preexcite the ventricle compared with conduction over the normal AV conduction system.

It would be extremely difficult to diagnose a fasciculoventricular AP unless block occurred revealing the normal HV interval, as happened here. One could argue that the shorter HV intervals are actually not His deflections but right bundle branch deflections, and the true HV interval is noted by the complexes with an arrow. However, a right bundle branch potential typically occurs with a smaller atrial electrogram compared with the atrial electrogram recorded with the His bundle. In the former situation the catheter is situated more distally nearer to the ventricle. In this tracing, there is no significant change in atrial amplitude for any of the paced complexes. In addition, this would not explain the subtle but real change in the QRS complex that occurs with the longer HV interval. These observations are best explained by the presence of a fasciculoventricular AP.

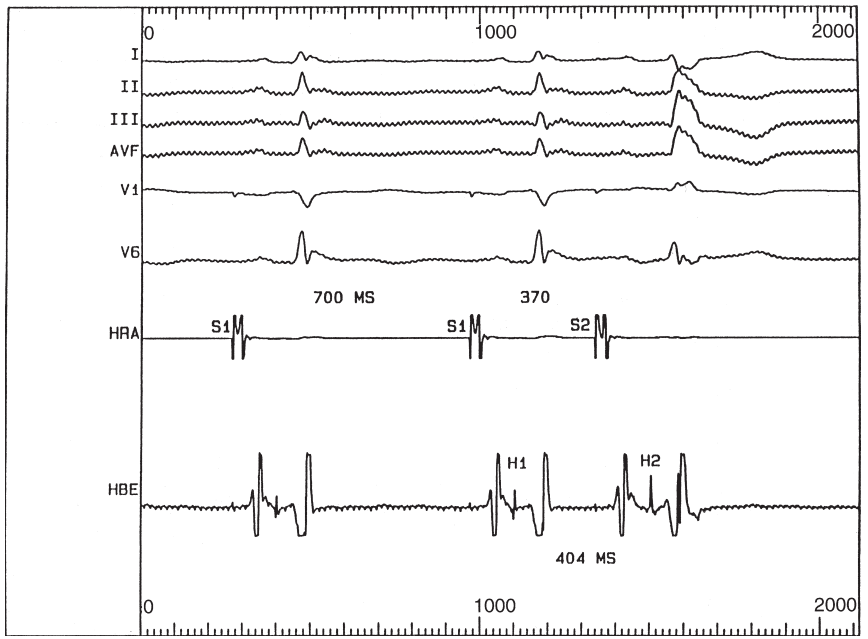
Figures 3–19A and 3–19B

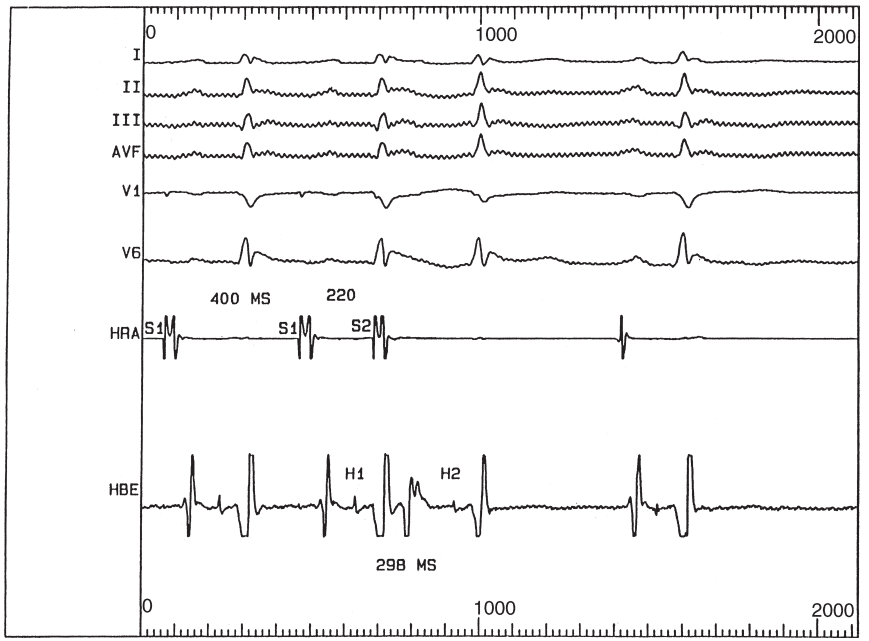




During the EP study in this patient, premature atrial complexes (PACs) were introduced at paced CL 700 milliseconds (Fig. 3–19*A*) and 400 milliseconds (Fig. 3–19*B*). What occurs and why?

Figures 3–19C and 3–19D



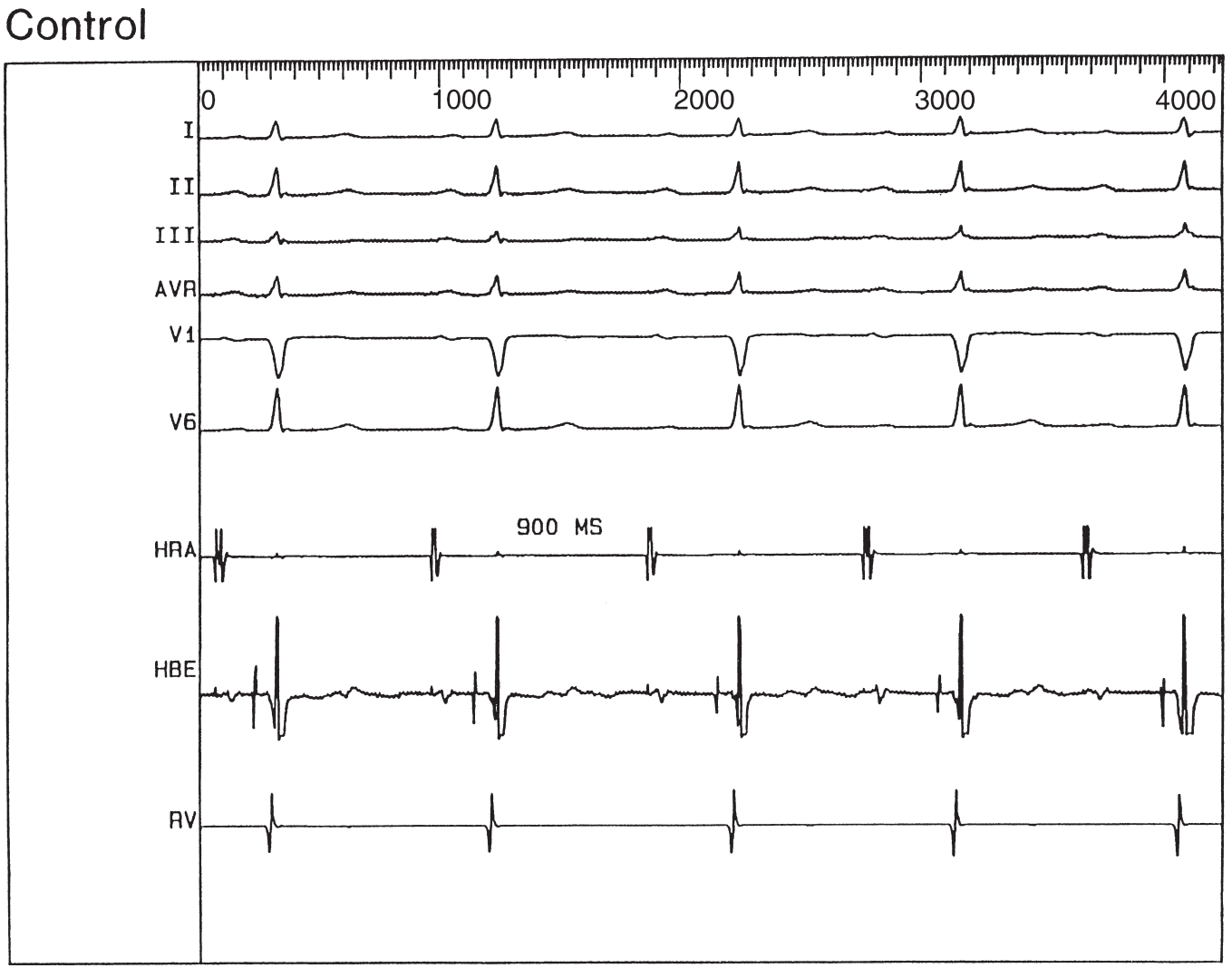


Explanation:

Figure 3–19*C* demonstrates high right atrial pacing at 700 milliseconds with a PAC introduced at 370 milliseconds. This results in an H_1H_2 interval of 404 milliseconds and a RBBB pattern. All longer H_1H_2 intervals were conducted with a normal QRS complex. Thus, the refractory period of the right bundle branch is 404 milliseconds. By electrocardiographic criteria, this is a RBBB pattern. However, actual block in the right bundle branch does not have to occur in this situation. Instead, there could be slowed conduction in the right bundle branch relative to the left bundle branch, allowing delayed activation of the right ventricle with a RBBB pattern. Thus, a RBBB block morphology can represent either marked slowing of conduction or actual block in the right bundle branch.

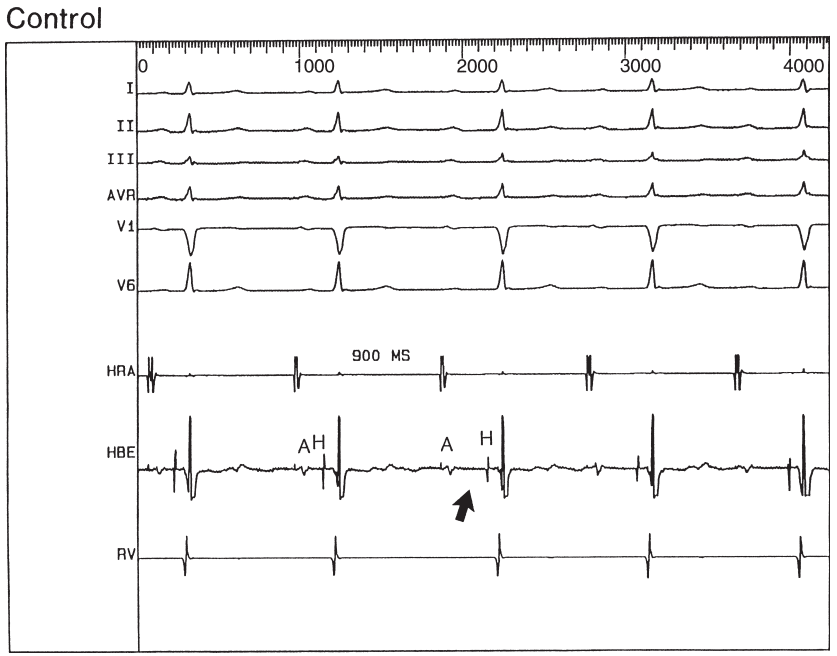
Figure 3–19*D* occurs during high right atrial pacing at CL 400 milliseconds with a markedly shorter premature atrial interval than noted in Fig. 3–19*C*. The resulting H_1H_2 interval is considerably shorter but right bundle branch conduction occurs. This demonstrates the effect of heart rate on bundle branch refractoriness. At slower heart rates the refractoriness of the HPS is relatively longer compared with faster heart rates. In this example, RBBB refractoriness is less than 298 milliseconds at a heart rate of 150 beats/min (Fig. 3–19*D*) but is 404 milliseconds at a heart rate of 86 beats/min (Fig. 3–19*C*). These effects of heart rate on bundle branch refractoriness are commonly manifested during continuous electrocardiographic recordings. Typically, a PAC with aberrancy occurs during slow rates but, when the heart rate is faster, a PAC with a similar coupling interval conducts with a narrow QRS.

Figure 3–20A



This recording was obtained during study of a patient with a history of SVT. What is the most likely cause of tachycardia in this individual?

Figure 3–20B



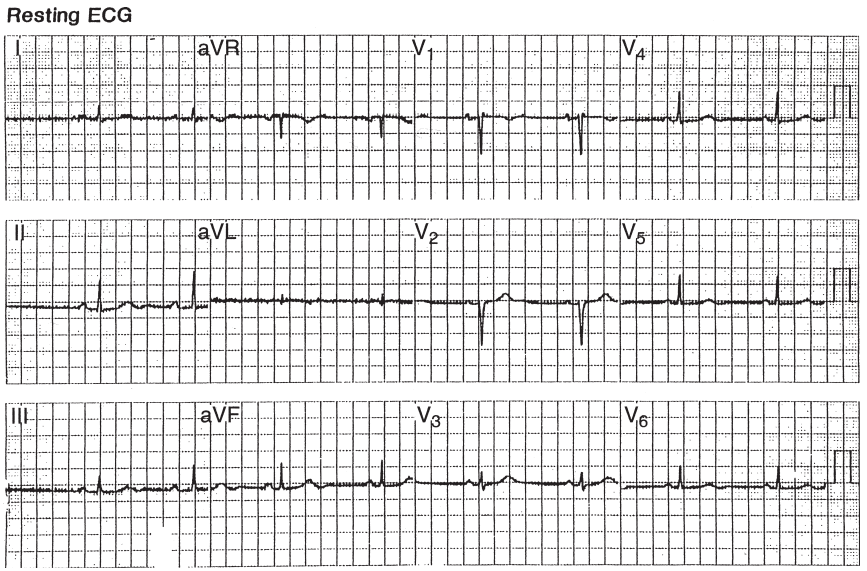
Explanation:

During fixed atrial pacing at CL 900 milliseconds there is a sudden prolongation in the PR interval that stays relatively fixed. The arrow demonstrates the marked sudden prolongation in AH interval compared with the previous shorter AH interval. When a sudden and unexpected prolongation in PR interval, or AH interval, occurs and remains relatively fixed, the likely diagnosis is dual AV nodal physiology. In this example, the first two atrial paced complexes conduct to the ventricle with a much shorter PR and AH interval than the subsequent complexes because conduction proceeds over the fast pathway of the AV node. The third atrial complex has a sudden prolongation of the PR and AH interval because block occurred over the fast AV nodal pathway and conduction now proceeds over the slow AV nodal pathway.

The demonstration of dual AV nodal physiology in a patient with documented paroxysmal SVT strongly suggests AV node reentry is the cause of tachycardia. In fact, with intravenous isoproterenol, AV node reentry was initiated.

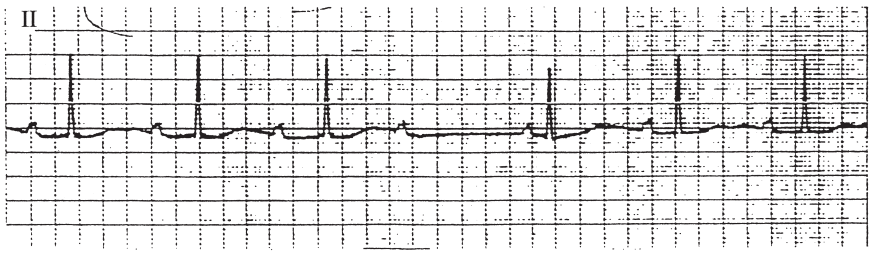
The demonstration of dual AV nodal physiology can be recognized occasionally during routine electrocardiography or continuous electrocardiographic recordings. If there is a sudden, unexpected, constant prolongation of the PR interval that does not progress to block, one should strongly suspect dual AV node physiology. This does not suggest that the patient has or will develop tachycardia since the patient may lack retrograde AV nodal conduction, which typically precludes AV node reentry. Thus, to initiate the slow/fast variety of AV node reentry there must be *both* anterograde block in the fast pathway and subsequent retrograde conduction over this pathway.

Figures 3–21A and 3–21B

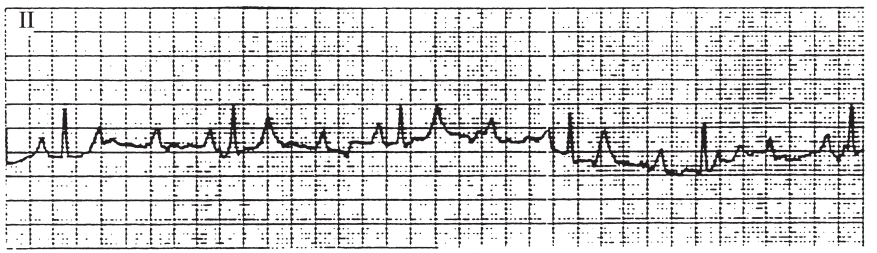


A middle-age woman presented with exercise-induced presyncope. Her resting ECG (left) and ECG lead II rhythm strips during exercise (right) are shown. Is conduction delay and block most likely to occur in the AV node, His bundle, or bundle branch system?

A. Standing



B. Stage 3 TM



C. Recovery

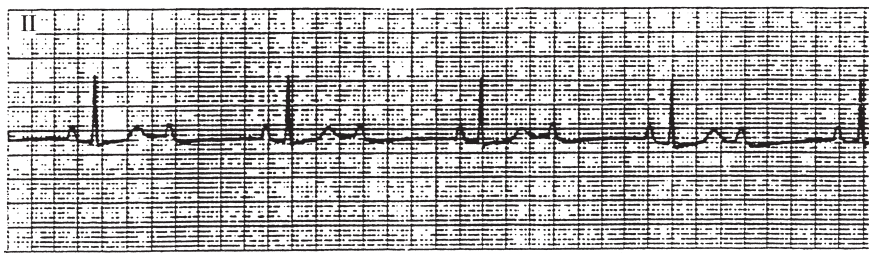
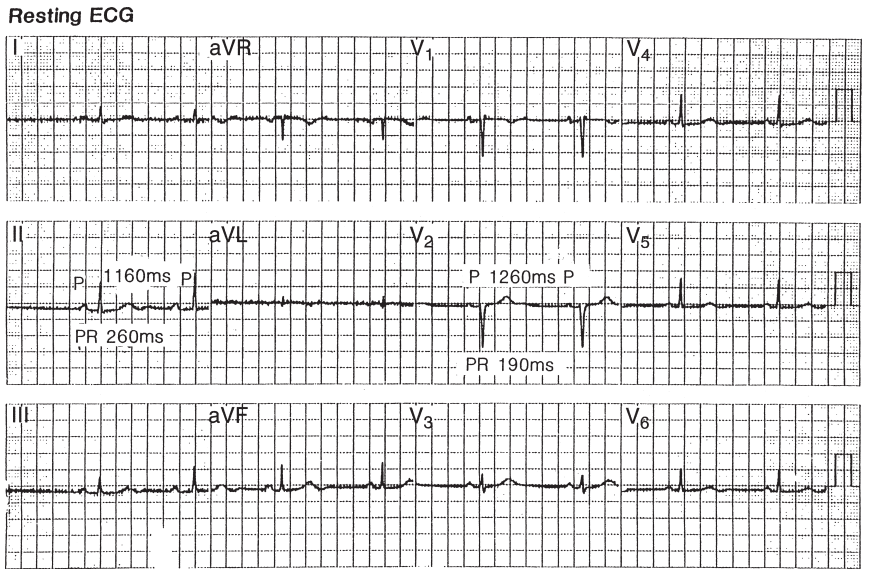


Figure 3–21C

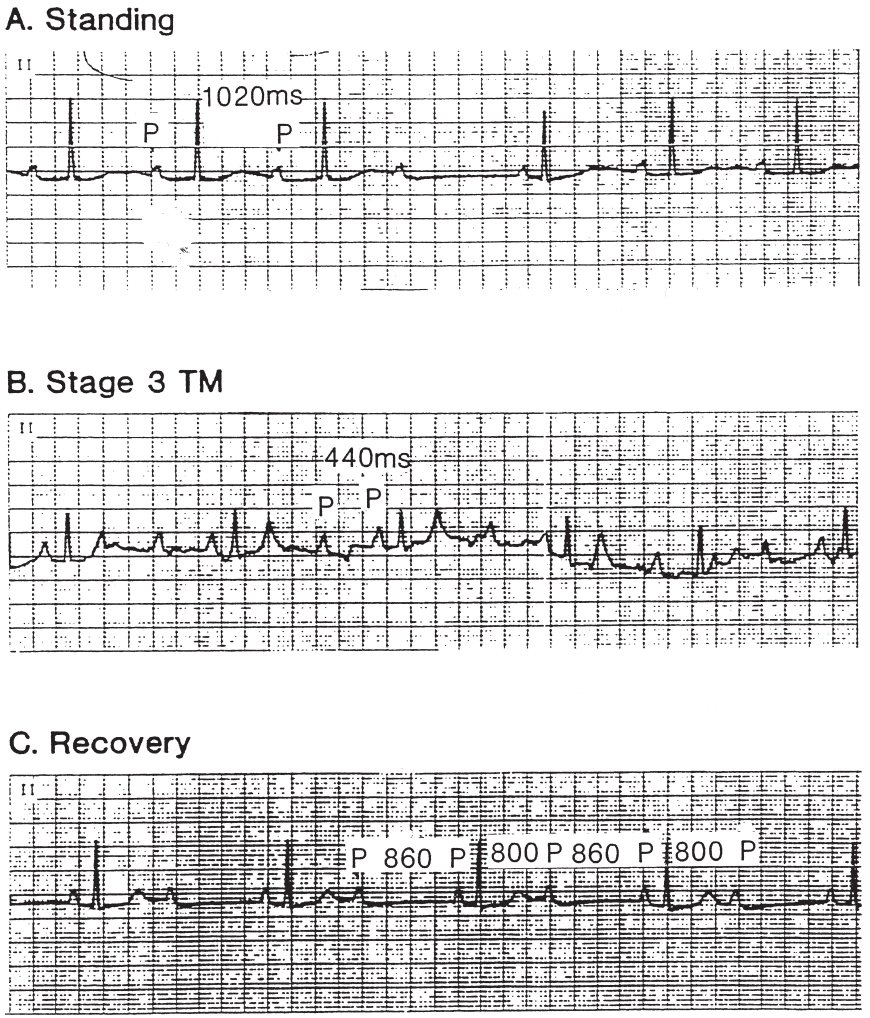


Explanation:

Figure 3–21*C* demonstrates the resting ECG with variable PR intervals. Note that during the beginning of the tracing in lead II with a sinus cycle length (SCL) of 1160 milliseconds the PR interval is 260 milliseconds. Later in the tracing as shown in V_2 the SCL is 1260 milliseconds with a PR interval of 190 milliseconds. Ideally, one should measure changes using the same ECG lead, but the PR differences are obvious. The QRS complex is narrow, suggesting lack of significant disease in the bundle branch system. However, this certainly does not exclude disease within the His bundle that may occur without bundle branch block. From this electrocardiogram alone, it is difficult to determine the site of variable conduction. However, one should suspect disease in the His bundle. Shortening of the SCL is likely due to autonomic changes such as decreased vagal tone or increased sympathetic tone, both of which usually improve conduction through the AV node.

Figure 3–21*D* shows the tracings from the treadmill evaluation and highlights the effect of heart rate on AV conduction. When the patient was standing (panel A), the SCL shortened to 1020 milliseconds and this resulted in type 1 second-degree AV block. As the SCL progressively decreased during the treadmill test, the degree of conduction block increased, for example, 3:1 conduction in stage 3 of the treadmill with a SCL of 440 milliseconds (panel B). During recovery (panel C), the SCL progressively lengthened to 860 milliseconds and 2:1 conduction occurred. At this time the phenomenon of ventriculophasic sinus arrhythmia is noted (see below). The autonomic effects of standing and exercise are parasympathetic withdrawal and increased sympathetic tone. These combined changes decrease SCL. They also facilitate AV node conduction. Thus, the paradoxical effect of decreased SCL producing progressively worse AV conduction strongly implicates the HPS as the site of conduction problems. The narrow QRS complex implies lack of bundle branch disease and the assumption is that conduction abnormalities occur within the His bundle. There was no need to perform

Figure 3–21D



an EP evaluation in this patient since the cause of her symptoms was identified during treadmill testing. In other patients we have correlated intra-His block in similar circumstances. This patient required a dual-chamber pacemaker and symptoms abated after its implantation.

Ventriculophasic Sinus Arrhythmia

During recovery there is 2:1 conduction (Fig. 3–21*D*). There is also an interesting pattern of longer P–P intervals between QRS complexes compared with shorter P–P intervals that surround a QRS complex. The mechanism for this ventriculophasic sinus arrhythmia is in part due to alternations in sinus nodal automaticity secondary to changes in blood pressure. Each QRS complex is associated with an increase in blood pressure, which affects the baroreceptors. One needs to postulate that the baroreceptor-mediated slowing of the sinus rate occurs too late to affect the P wave immediately after the QRS complex, but does prolong the subsequent SCL. Thus, the P–P interval between the two QRS complexes is lengthened. Because the effect of heightened blood pressure is short lived, the P wave immediately after the QRS complex is not delayed, resulting in a shortened P–P interval surrounding the QRS complex.

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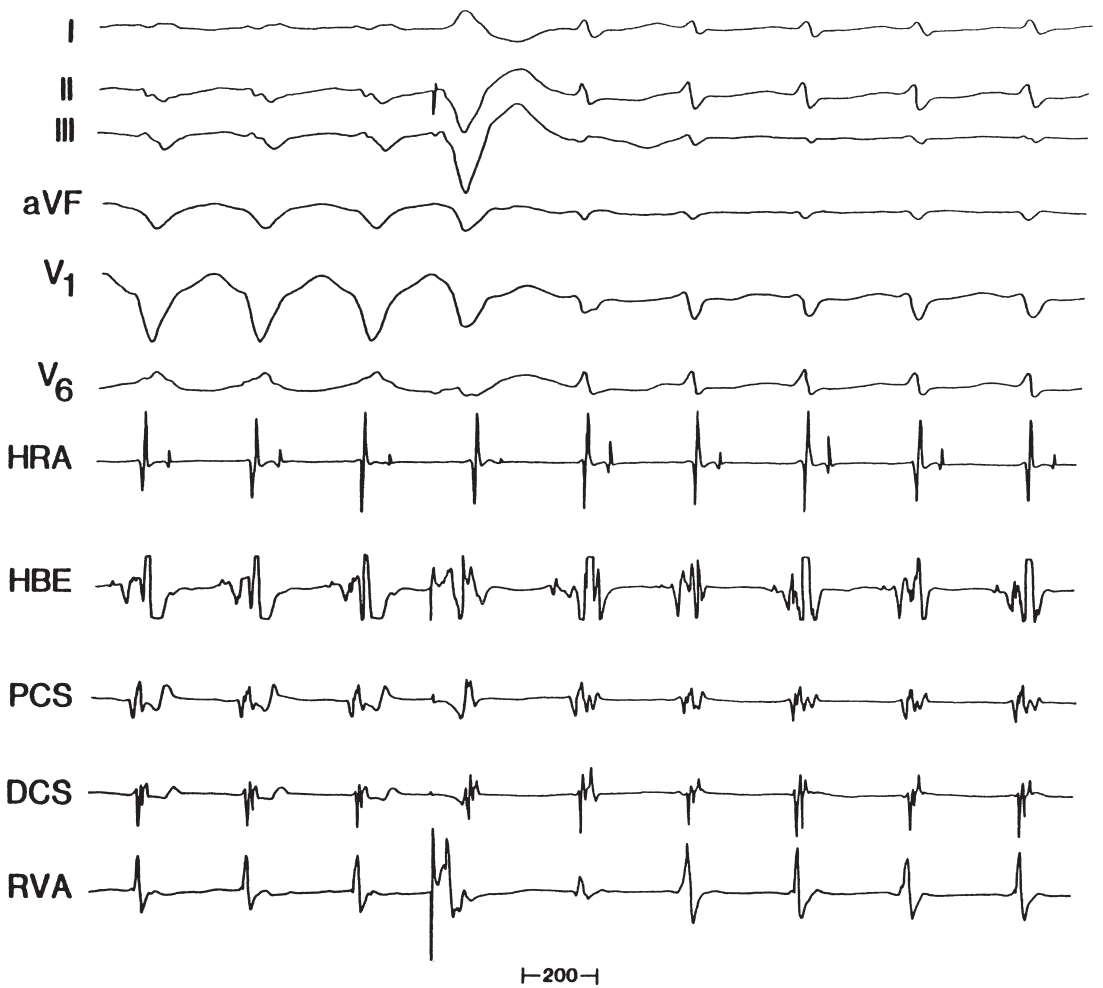
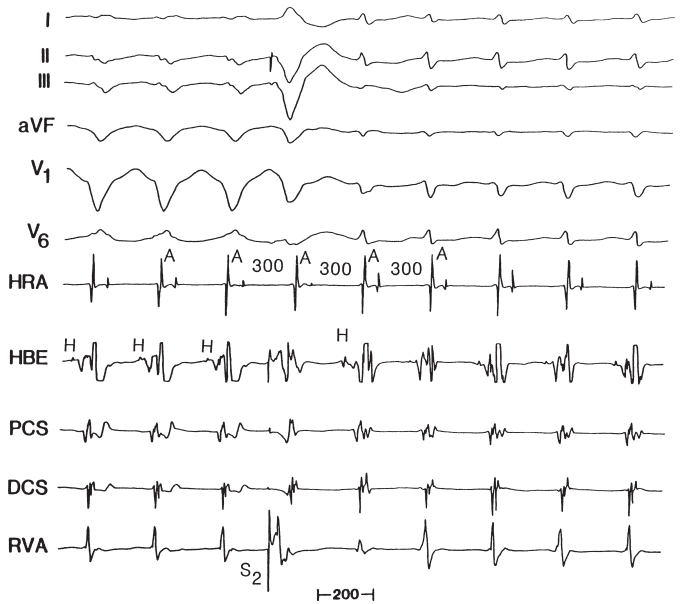
Figure 3–22A



This patient has a history of wide and narrow QRS complex tachycardia. This tracing was taken during an EP study. What is the most likely diagnosis? What is the most likely mechanism for transformation from the wide QRS complex to the narrow QRS complex during tachycardia?

Figure 3–22B



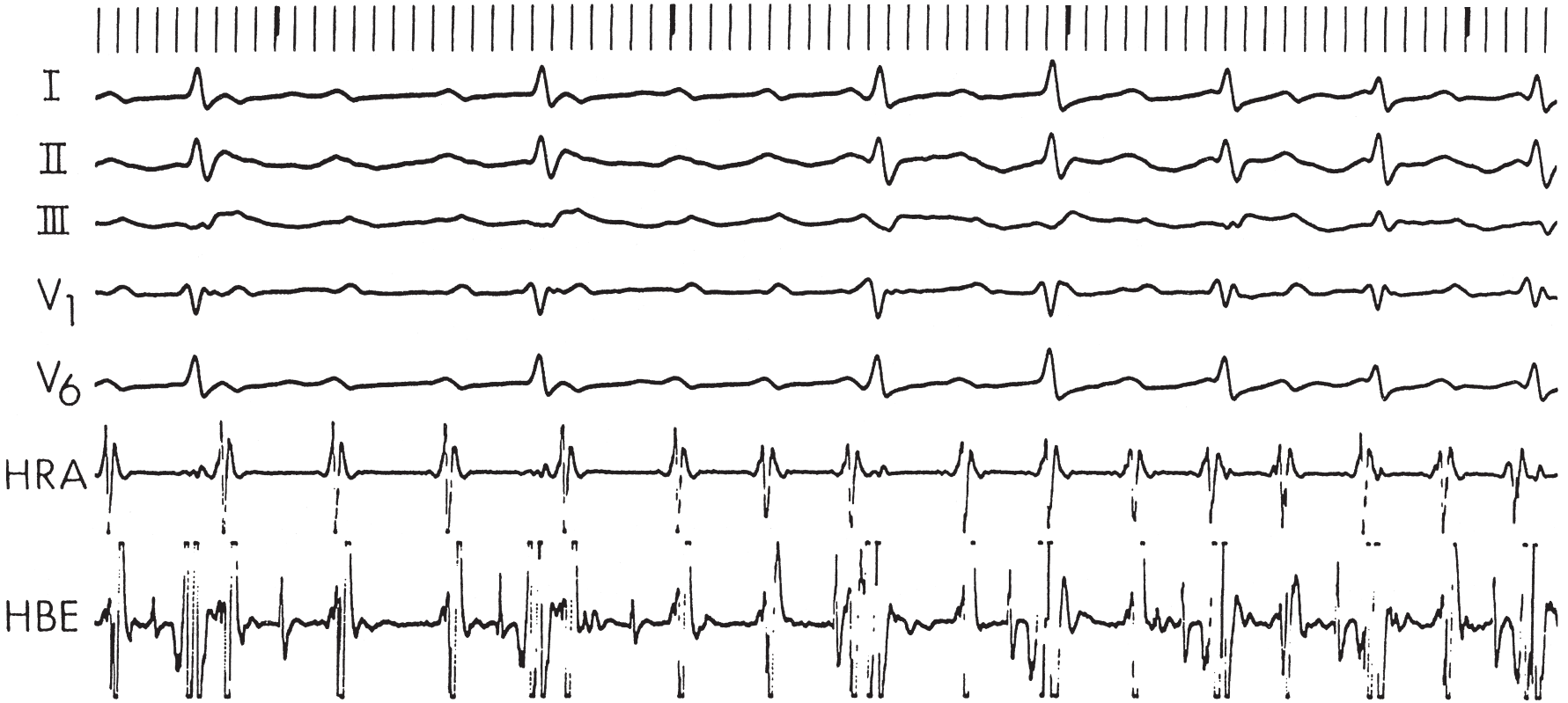
Explanation:

The tachycardia mechanism is similar during bundle branch block aberrancy or normal QRS morphology. The short VA interval, identified as an atrial ECG within the QRS complex, rules out AV reentry. The long AH interval and relatively short HA and VA intervals are consistent with AV node reentry. From this tracing alone, an atrial tachycardia cannot be excluded, but this would be a far less likely diagnosis. In this situation one would have to postulate an atrial tachycardia with conduction over a slow pathway. This patient had AV node reentry.

The first three QRS complexes demonstrate typical LBBB morphology. Note in the His bundle lead that a small His deflection is present and the HV interval is similar to that recorded with the subsequent narrow QRS complexes. Thus, the first three QRS complexes represent LBBB aberrancy. There is minimal to no difference in the tachycardia cycle length between LBBB and narrow QRS complex morphology. It is therefore highly unlikely that LBBB aberrancy is due to an acceleration-dependent mechanism. The persistence of LBBB aberrancy is typical for transseptal concealed conduction. In this situation, a supraventricular complex conducts to the ventricle over the right bundle branch and blocks over the left bundle branch. Then, transseptal concealed conduction (not visible) occurs from the right to left ventricle with retrograde invasion of the impulse into the distal left bundle branch. This relatively late activation of the left bundle branch will prolong its refractoriness compared with the right bundle branch. When the next supraventricular impulse arrives at the left bundle branch, it will be refractory and block occurs. This sequence persists and LBBB aberrancy continues with each tachycardia cycle. A similar scenario can occur with persistent RBBB aberrancy due to retrograde invasion from the left bundle branch into the right bundle branch distally.

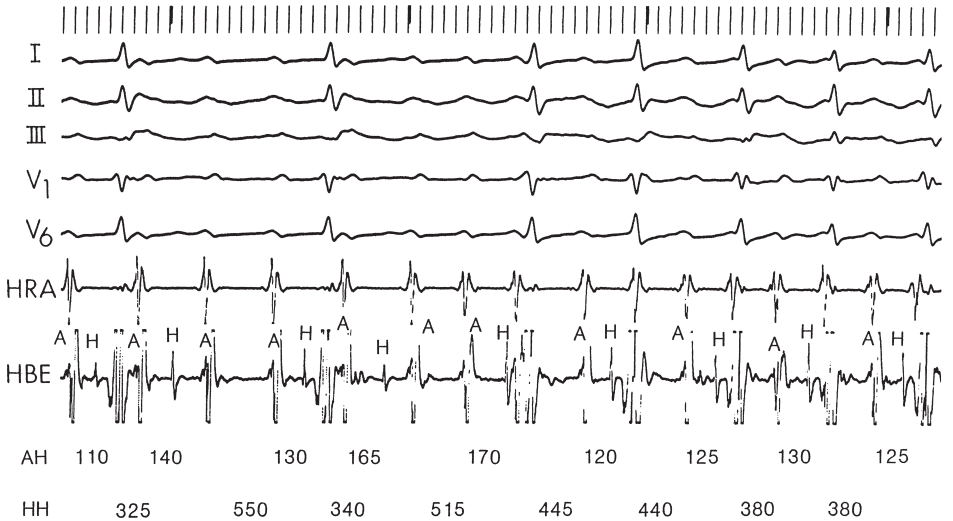
Introduction of a PVC (S_2) during tachycardia at a critical coupling interval can result in termination of the transseptal concealed conduction. The premature ventricular complex "peels" back the refractory period of the left bundle branch by preventing the impulse from the right bundle branch from activating it at its usual later time. In other words, the PVC activates the left bundle branch earlier than would have been anticipated had the right bundle branch caused retrograde activation of this structure. Now, when the next supraventricular impulse reaches the left bundle branch anterogradely, it will conduct without block and this ends the persistence of bundle branch block aberrancy. In summary, this patient has typical AV node reentry with anterograde conduction over the slow pathway and retrograde conduction over the fast pathway. LBBB aberrancy occurred during the initiation of tachycardia and persisted until a PVC was introduced. LBBB aberrancy was caused by transseptal concealed conduction from the right bundle branch into the distal left bundle branch. This sequence was interrupted with the PVC that peeled back refractoriness of the left bundle branch and allowed the next supraventricular impulse to conduct normally over both bundle branches.

Figure 3–23A



This tachycardia was initiated at EP study performed approximately 1 week after surgical ablation of an AP many years ago. Where does AV block occur? Does this patient require permanent pacing? Time lines on top are 50 milliseconds.

Figure 3–23B

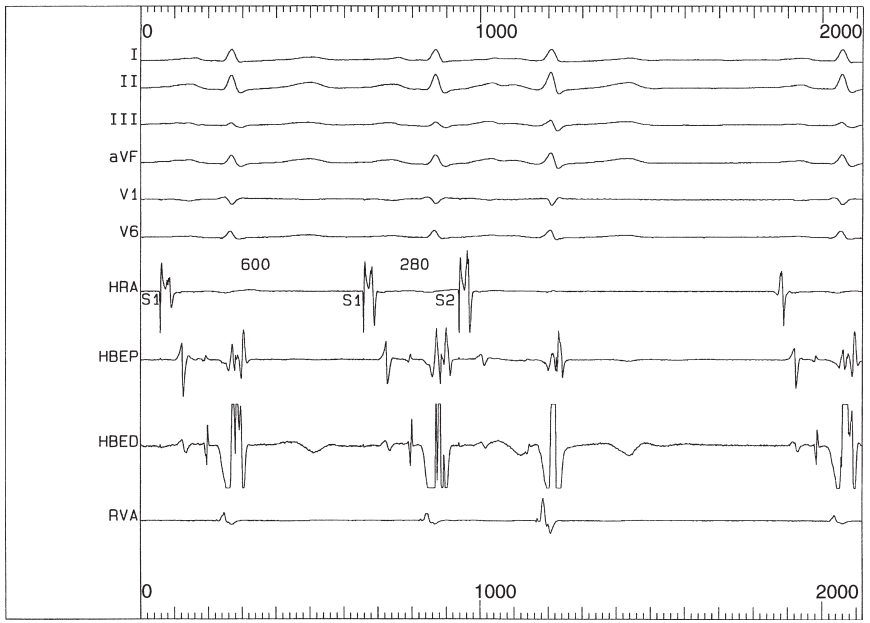


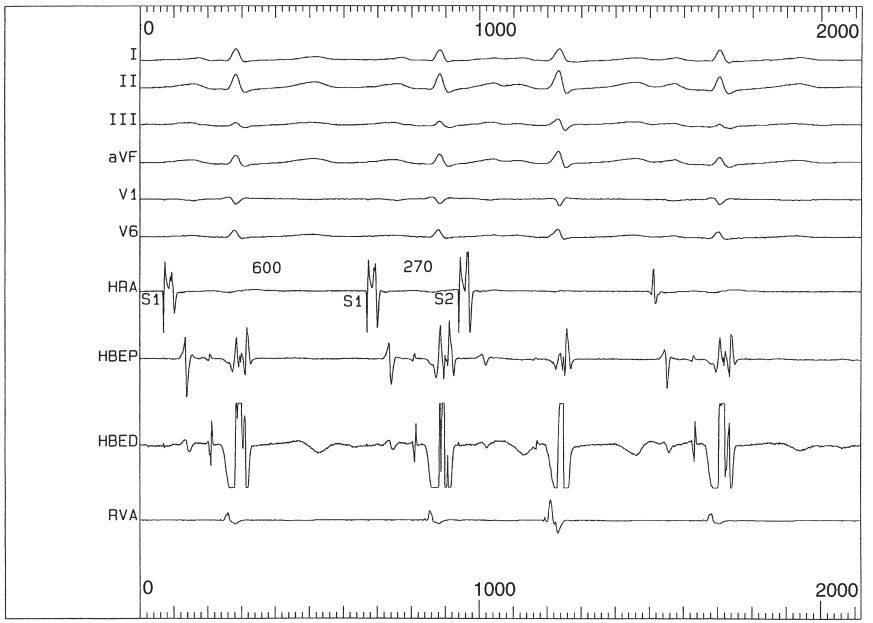
Explanation:

An atrial tachycardia was initiated at EP study, which was not a clinical arrhythmia for this patient. However, an interesting EP phenomenon occurred with AV block in both the AV node and HPS. On the left-hand portion of the figure AV nodal Wenckebach block occurs with an increase in the AH interval from 110 to 140 milliseconds before block. Note the lack of a His bundle deflection after the third atrial electrogram. The fourth atrial electrogram demonstrates an AH interval of 130 milliseconds and another Wenckebach AV nodal conduction pattern occurs. Then, the patient develops 2:1 AV conduction with a change in atrial rate.

The AV nodal Wenckebach conduction pattern produced variable HH intervals, which is physiologic. The changes in HH intervals result in block below the recorded His deflection. Block occurs with the shorter HH cycle immediately after a relatively long HH interval that results in prolongation of His–Purkinje refractoriness. Analysis of the sequence starting with the HH interval of 550 milliseconds reveals that the subsequent HH interval is 340 milliseconds and block occurs below the His deflection. The next HH interval is 515 milliseconds, but now there is 2:1 block. This allows a relatively long HH interval of 445 milliseconds and the subsequent HH intervals are similarly prolonged enough to allow conduction with each His deflection. The second atrial complex demonstrates block below the His bundle deflection and it is presumed that this HH interval of 325 milliseconds was preceded by a relatively long HH interval, although this is not shown in the tracing. Infra-His block that results from a long–short HH sequence is physiologic. No pacemaker is required.

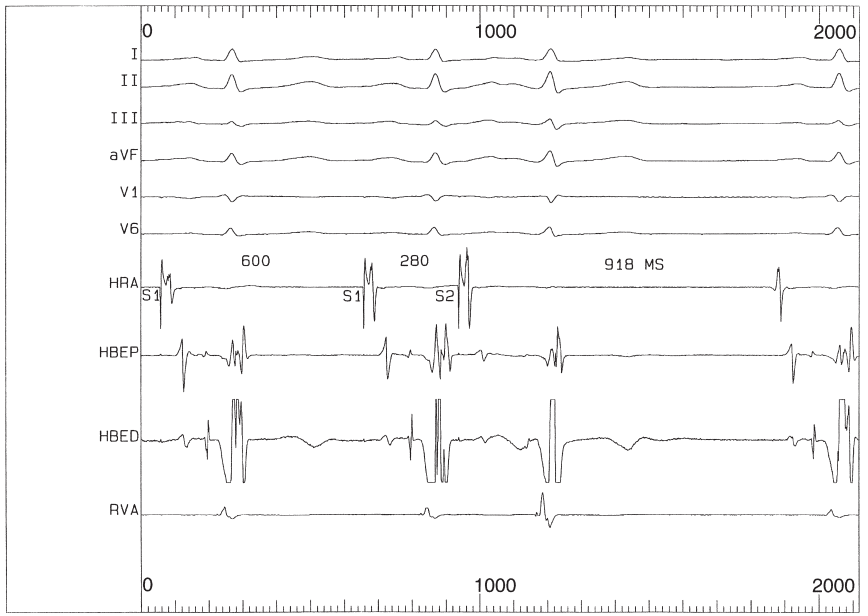
Figures 3–24A and 3–24B

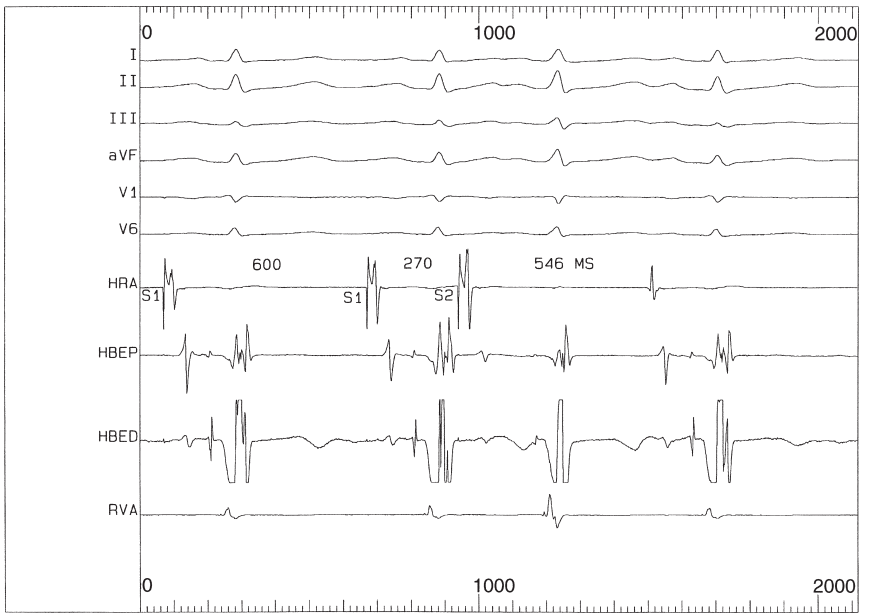




Figures 3–24*A* and 3–24*B* were recorded during right atrial pacing at CL 600 milliseconds with a premature atrial complex of 280 milliseconds (Fig. 3–24*A*) and 270 milliseconds (Fig. 3–24*B*). What is the most likely cause of the difference in sinus return cycle after the premature complex?

Figures 3–24C and 3–24D

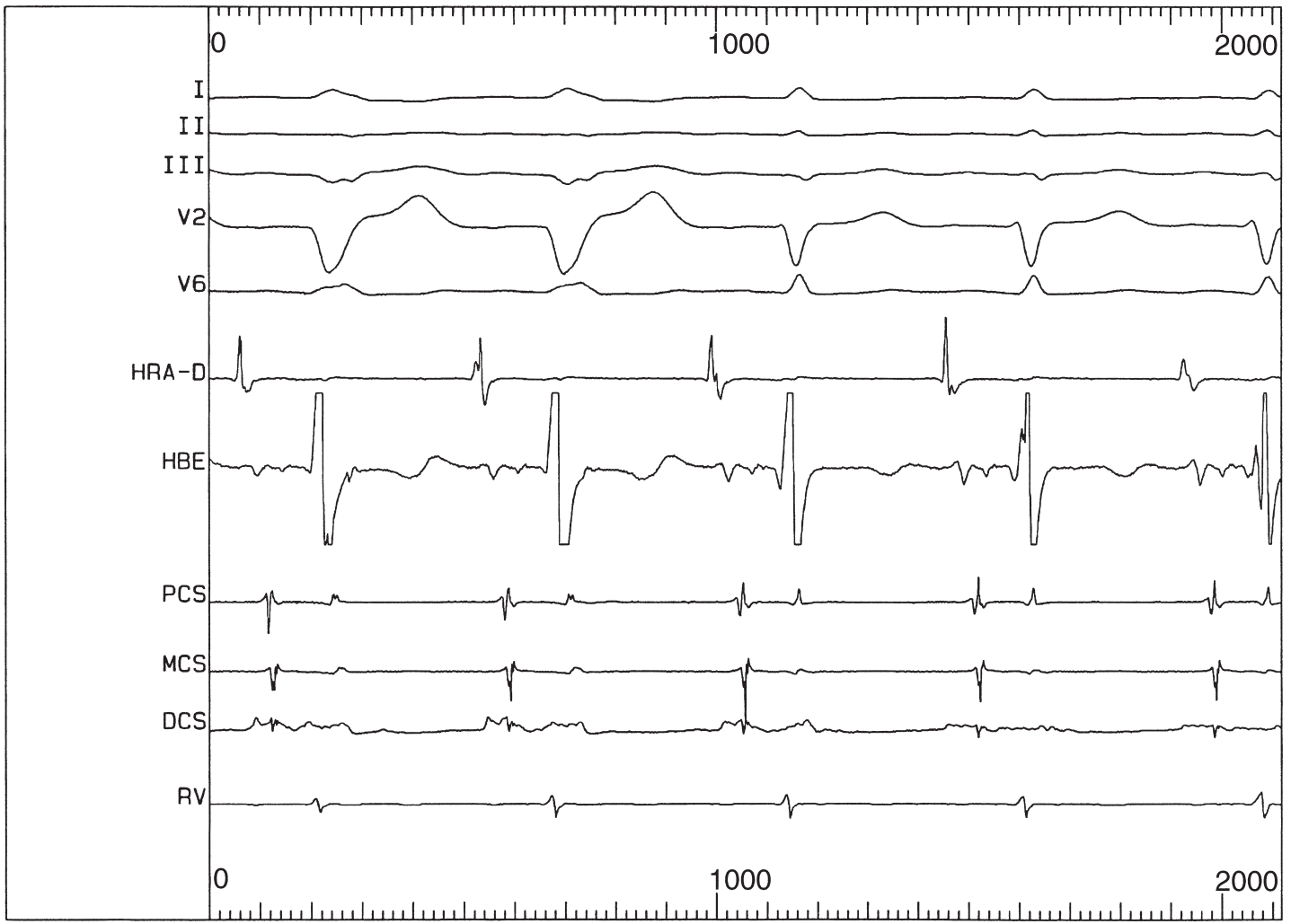




Explanation:

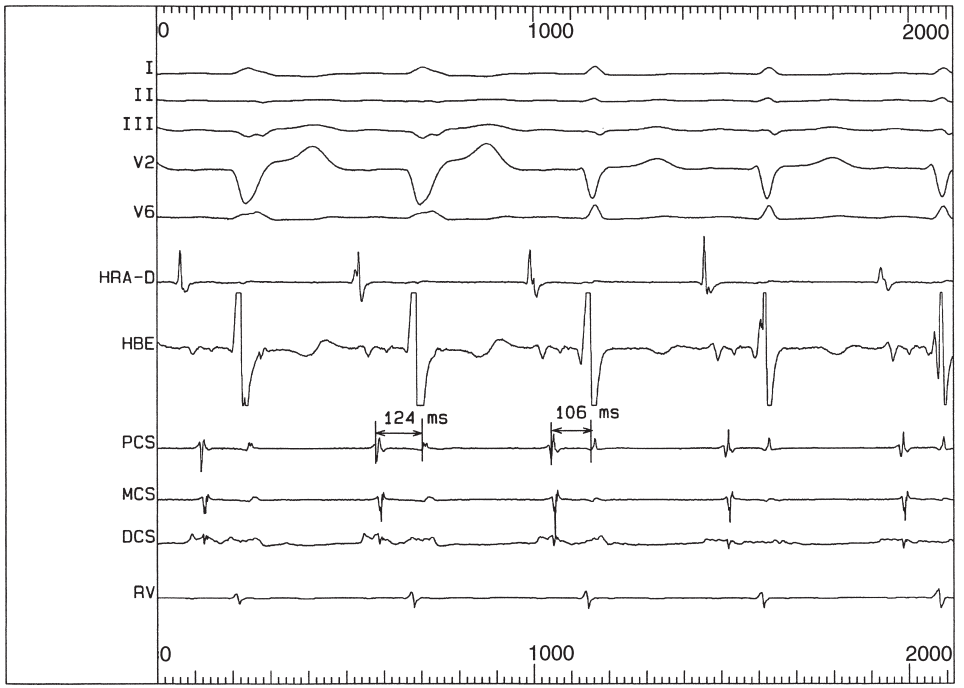
In Fig. 3–24*C*, a PAC conducts to the ventricle and the subsequent sinus complex occurs 918 milliseconds later. In Fig. 3–24*D*, a more premature atrial complex results in a sudden shortening of the return SCL to 546 milliseconds. This may represent a sinus nodal echo or the attainment of the sinus node effective refractory period. In the latter case the PAC presumably did not invade the sinus node. Therefore, it was not reset allowing an earlier sinus complex to occur. This is one measure of sinus nodal function.

Figure 3–25A



This recording was made during an electrophysiologic study in a patient with an AP who just underwent radio-frequency catheter ablation. What EP phenomena are present?

Figure 3–25B

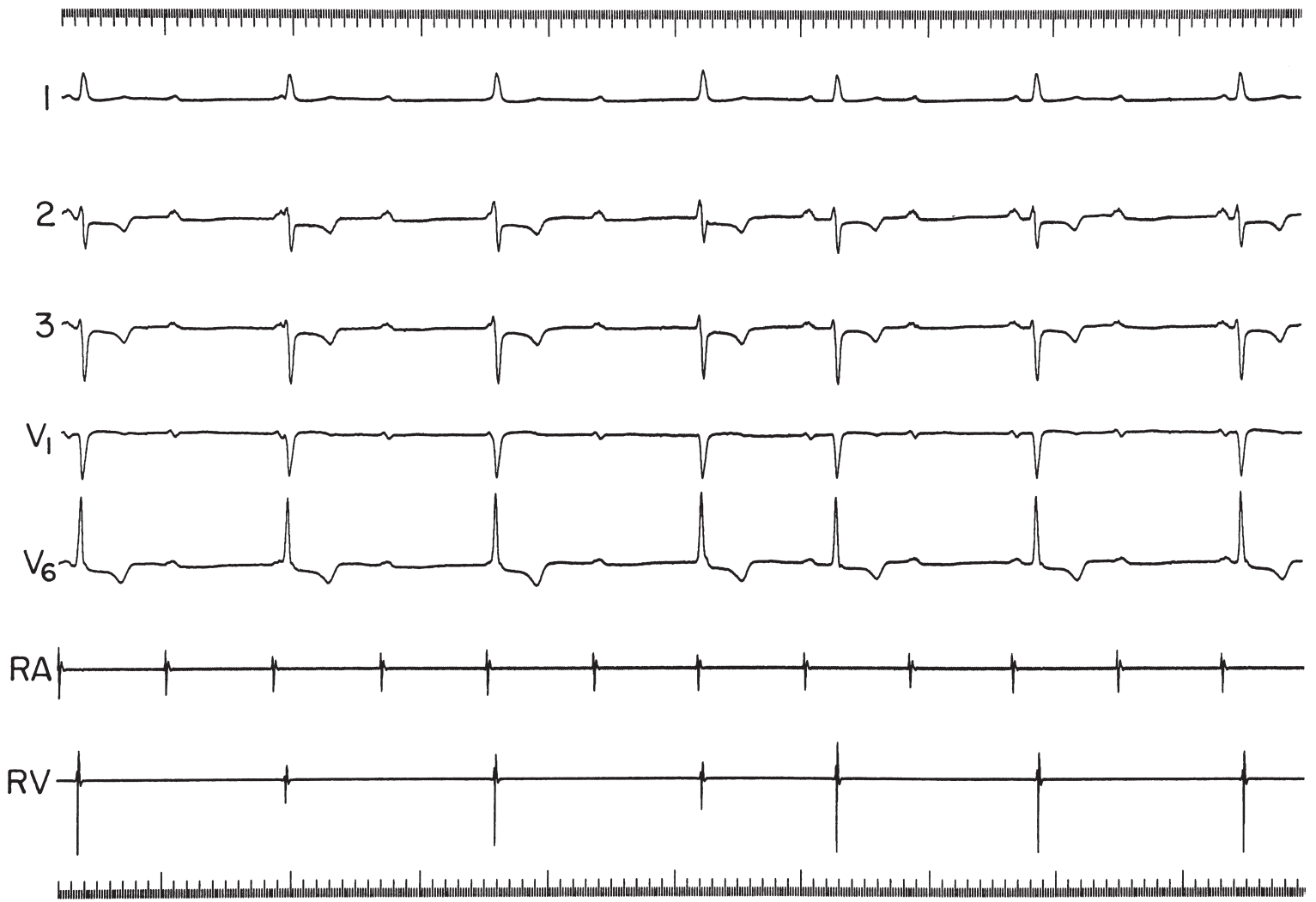


Explanation:

This patient had a posterolateral AP with bidirectional conduction. Radio-frequency energy successfully ablated the AP. Shortly thereafter the patient was noted to have wide QRS complexes followed by narrow QRS complexes during sinus rhythm. The first two complexes represent LBBB aberrancy and not AP conduction. Note that the HV interval is similar for the narrow and wide QRS complexes. It is not clear why this patient had LBBB aberrancy at this part of the study, but he did have LBBB aberrancy during orthodromic AV reentry. Importantly, there is a definite change in the local AV interval recorded on the coronary sinus catheter. Note on the proximal coronary sinus electrogram the local AV interval is 124 milliseconds during LBBB conduction and this becomes shorter (106 milliseconds) during normal conduction. Since the activation time from the sinus node to the local atrial electrogram at the coronary sinus electrode remains constant (HRA-D to PCS), the difference in local conduction time results from delayed activation of the posterior left ventricular wall during LBBB.

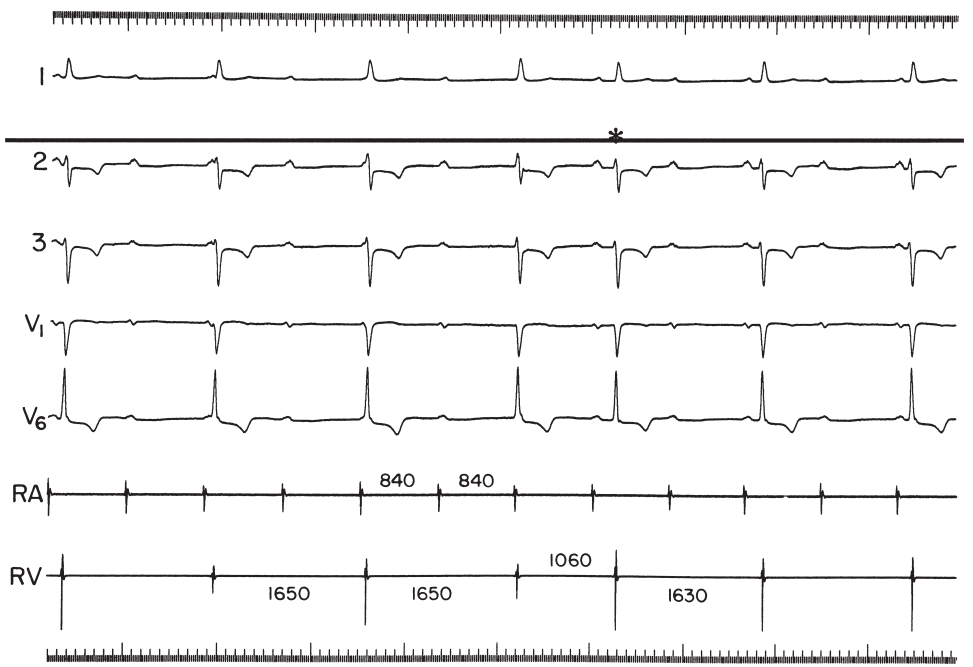
In summary, the two key observations present are LBBB aberrancy and its accompanying effect to delay conduction to the left ventricular posterior wall.

Figure 3–26A



This 62-year-old man underwent radio-frequency ablation of the AV node 30 minutes prior to this record. Is this complete AV block?

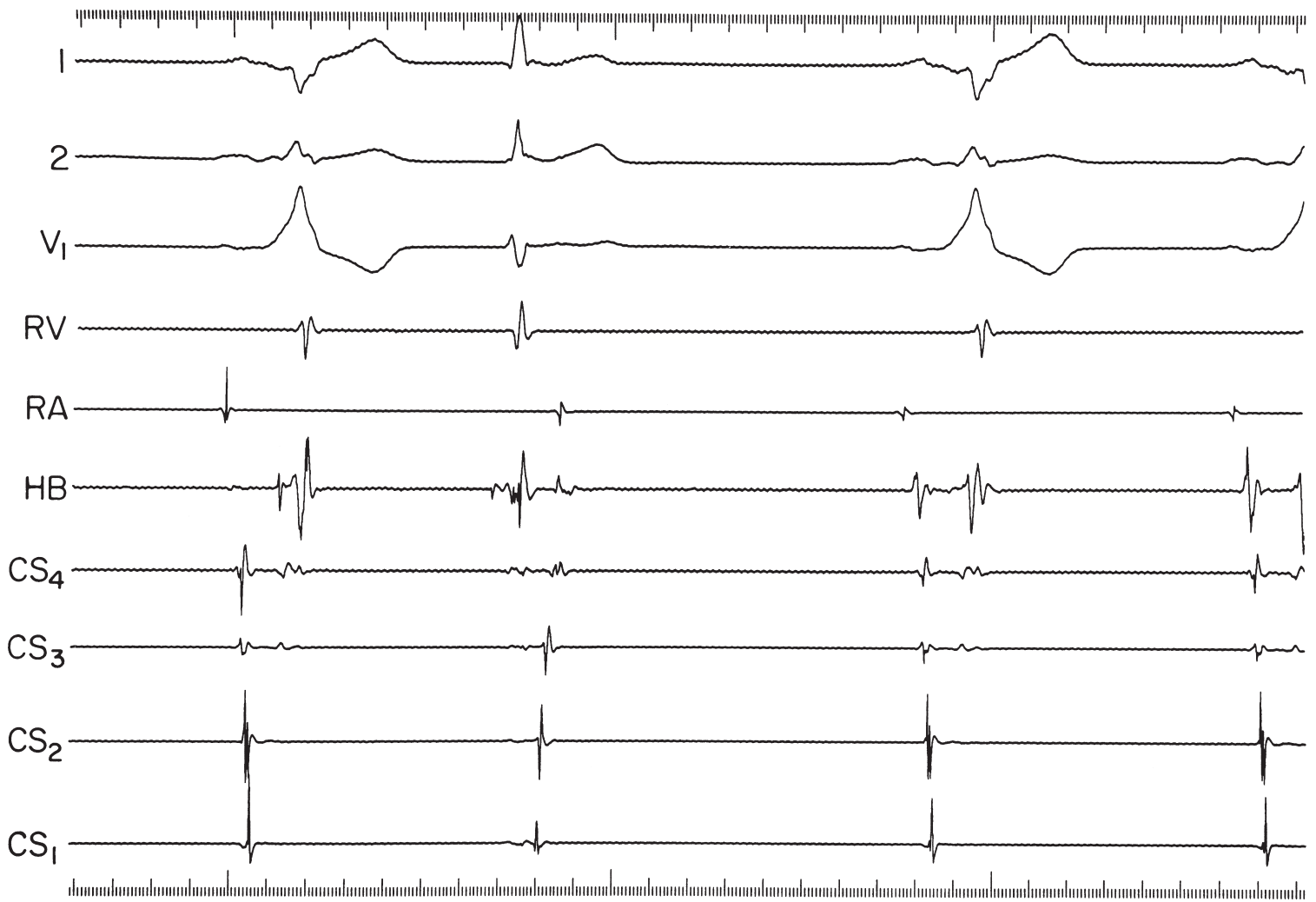
Figure 3–26B



Explanation:

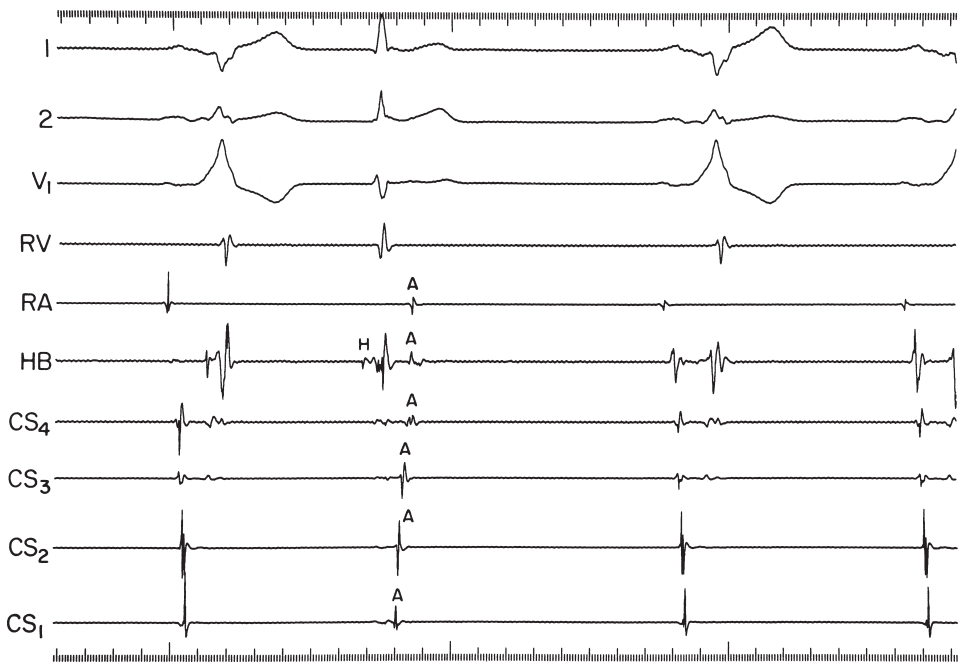
The atrial CL is 840 milliseconds and the ventricular CL is 1650 milliseconds. The QRS has normal supraventricular morphology, compatible with a junctional escape rhythm. AV block appears to be complete except for the occasional ventricular cycle that is premature (*asterisk*). The cycle *after* the premature beat is "on time," indicating that the junctional pacemaker has been reset. This capture beat always occurred if the P wave was coupled to the previous QRS by a critical interval (RP >870 milliseconds will capture) and the pattern observed on the figure was repetitive. AV block is obviously not "complete," although it may be described as high grade. The conducted P wave has a PR interval of 220 milliseconds, the minimal time needed to capture the junctional focus. A P wave occurring less than 220 milliseconds from the subsequent QRS will have no chance to capture the ventricles.

Figure 3–27A



This patient with the WPW pattern presented clinically with a regular, wide QRS tachycardia. Based on the data in this figure, is this likely to be antidromic tachycardia?

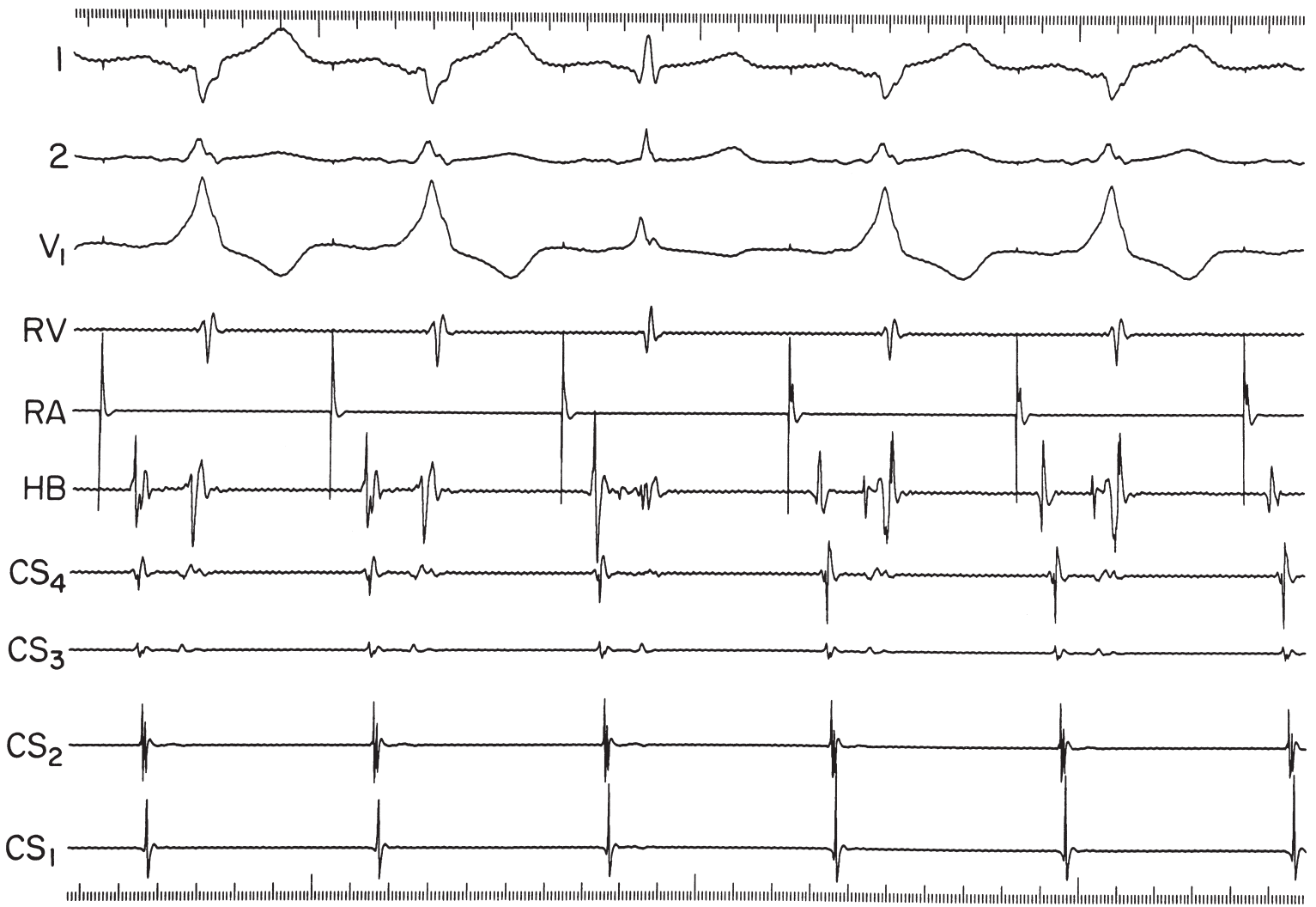
Figure 3–27B



Explanation:

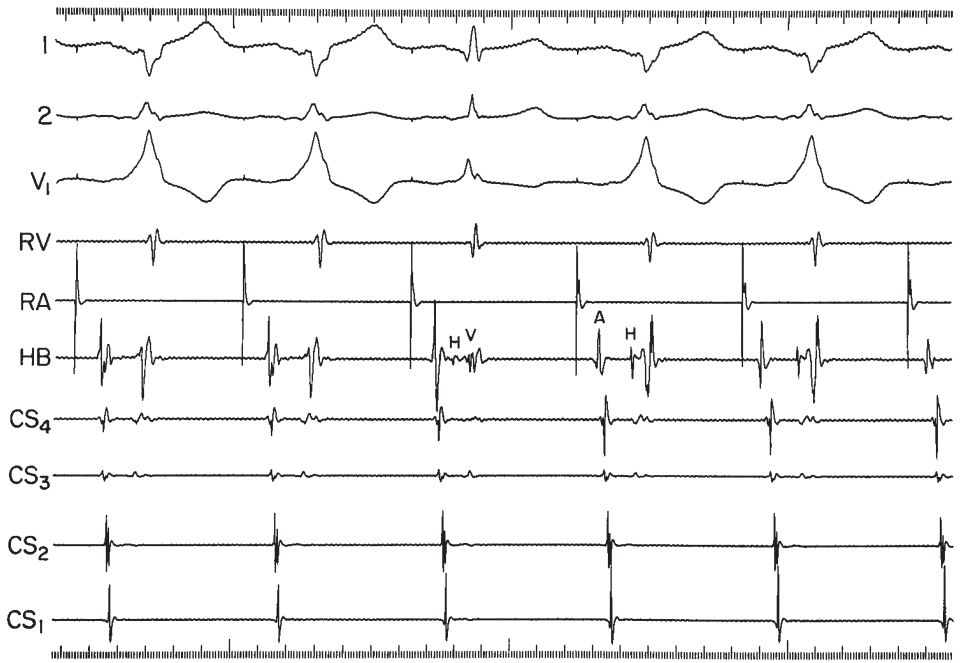
The first cycle is a sinus cycle and is preexcited with onset of QRS preceding the His deflection. The second cycle, a His extrasystole, is preceded by a His deflection and the QRS has normalized. His extrasystoles are usually observed during or shortly after positioning of the His bundle catheter. In this instance, retrograde conduction to the atrium is occurring over a left lateral AP as evidenced by earliest atrial activation at the distal coronary sinus (CS_1). There is no evidence for retrograde conduction over the AV node, a condition that must be possible to enable antidromic tachycardia, a tachycardia circuit with anterograde conduction over the AP, and retrograde conduction over AV node. However, retrograde conduction over the AV node may have a longer conduction time than the AP and could become apparent only after retrograde block over the AP. Furthermore, retrograde conduction over the AV node could be catecholamine dependent. Consequently, antidromic tachycardia as a clinical tachycardia is unlikely but not precluded by this record.

Figure 3–28A



Same patient as in Fig. 3–27. Why has the QRS apparently normalized?

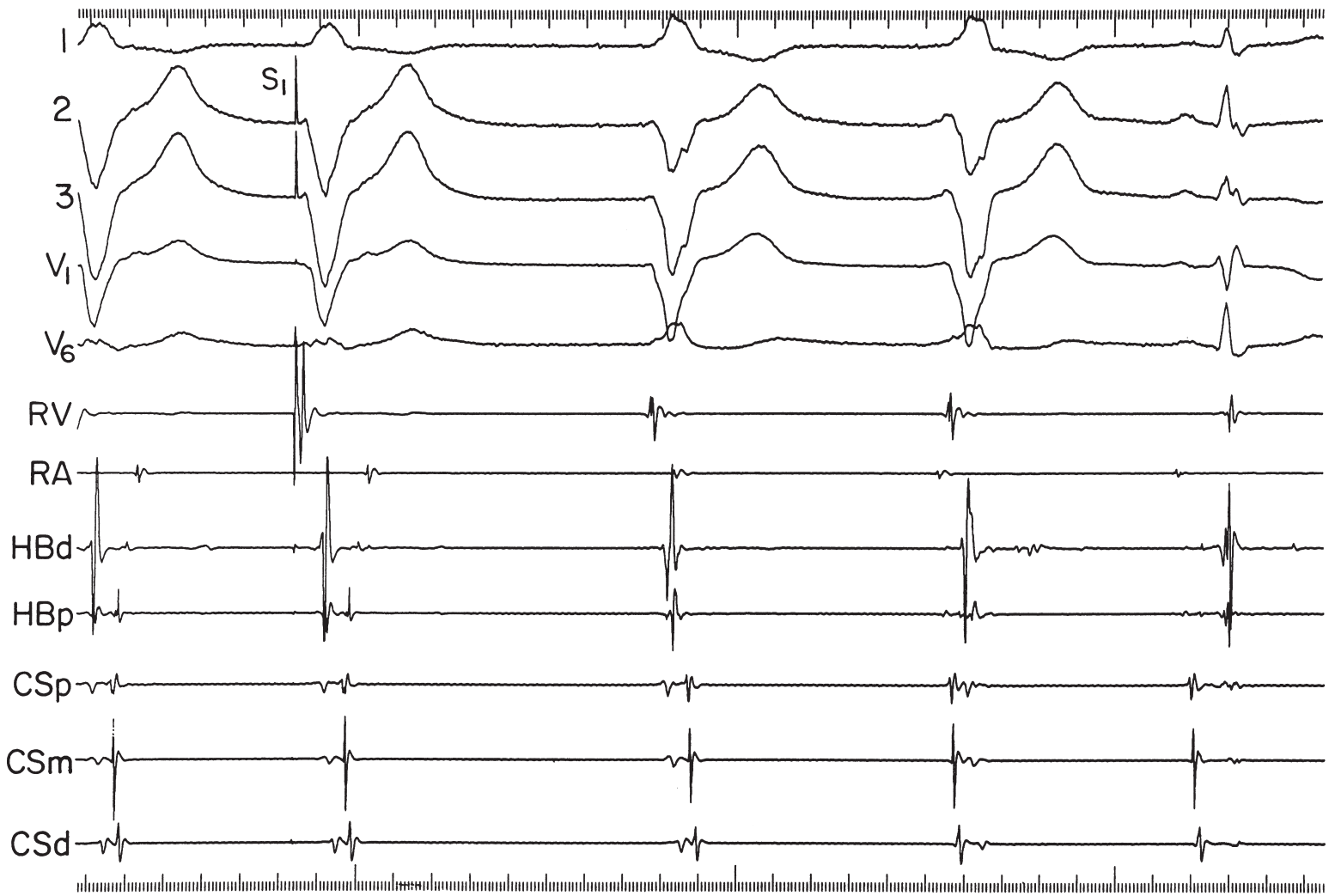
Figure 3–28B



Explanation:

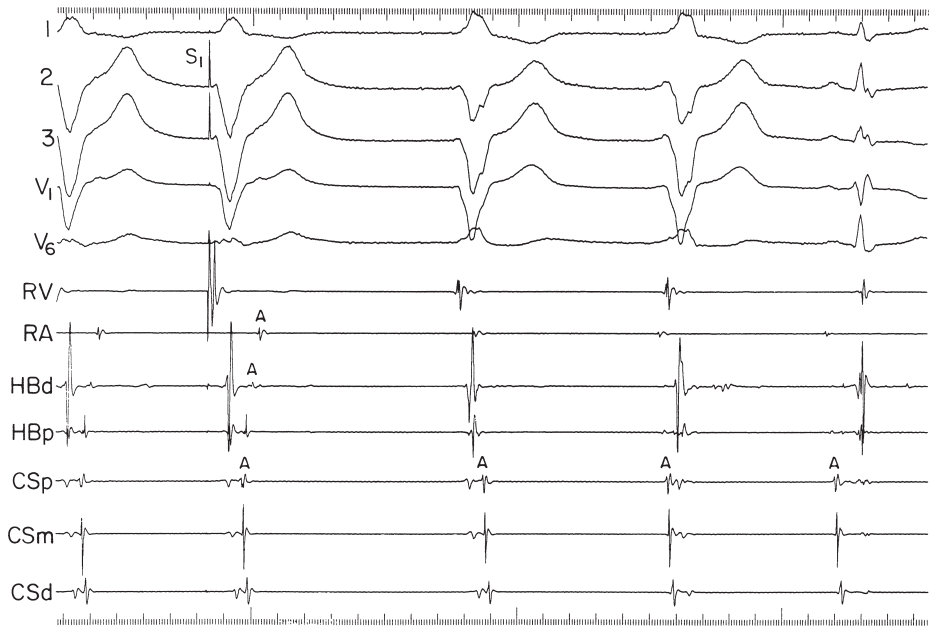
Normalization of the preexcited QRS in this instance could theoretically occur (1) after block over the AP, (2) with the fortuitous occurrence of a PVC after atrial activation (pseudonormalization), or (3) with the fortuitous occurrence of a His extrasystole, resulting in more activation of the ventricle over the normal conduction system than the AP. The continued presence of preexcitation, albeit less marked, and the apparent shortening of the AH interval favors the diagnosis of a fortuitous His extrasystole, resulting in a more normal appearance of the QRS.

Figure 3–29A



This patient had a right AV pathway with a long conduction time. After cessation of pacing, two cycles with QRS identical to the preexcited QRS are observed consistently. How is this explained?

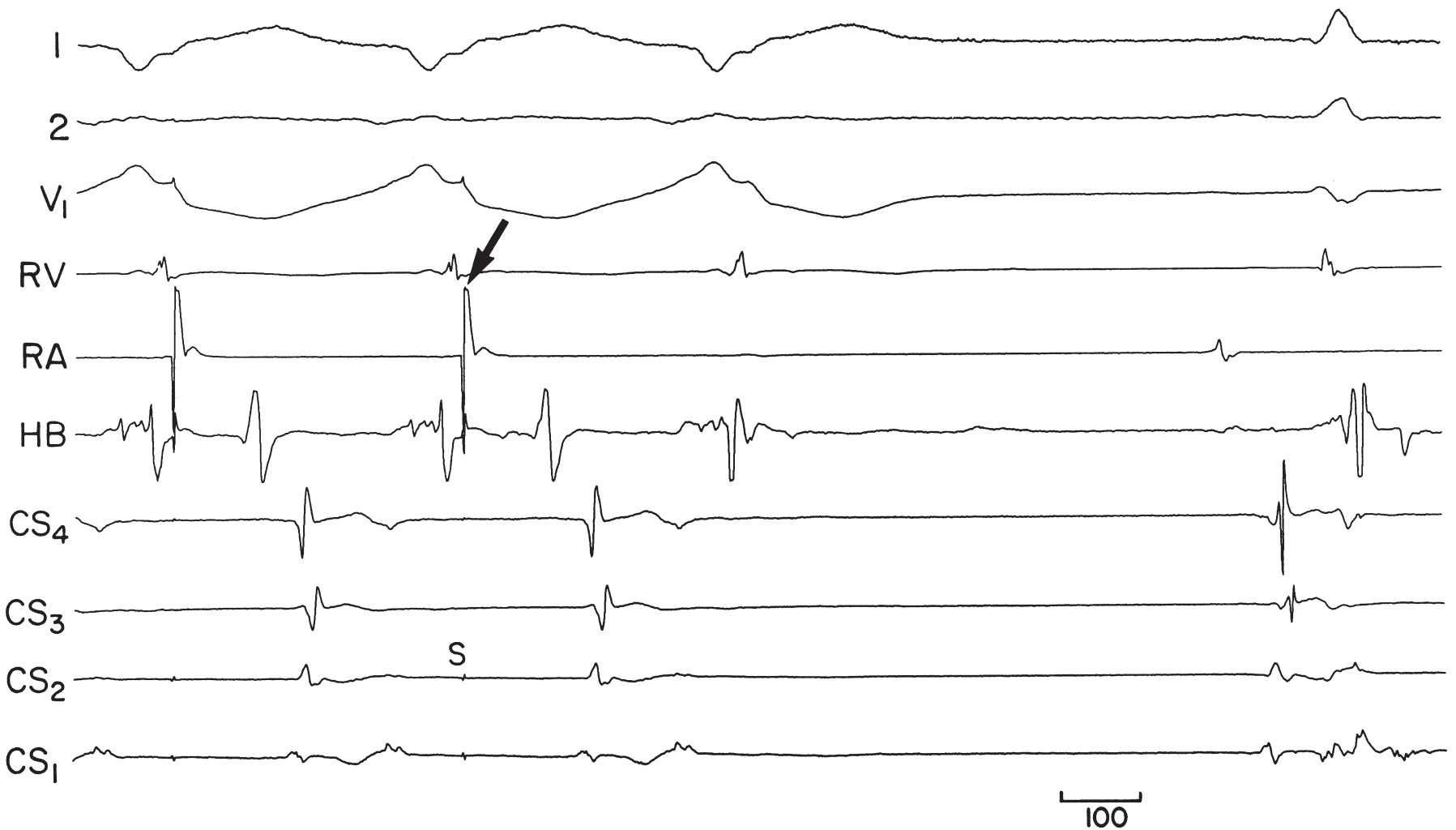
Figure 3–29B



Explanation:

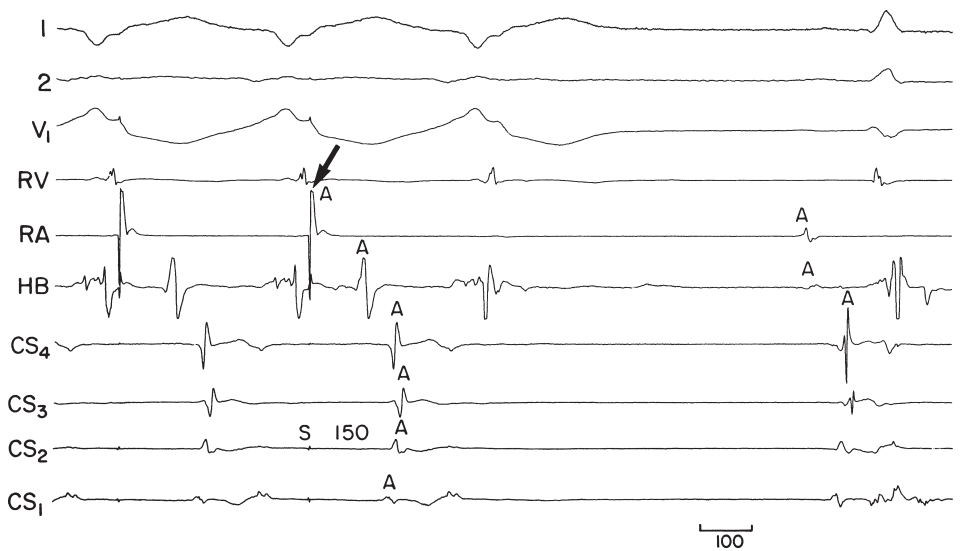
The relative sinus pauses after cessation of pacing allowed the emergence of automaticity in this right-sided pathway. APs may exhibit intrinsic automaticity, especially those with decremental properties and long conduction times. The two cycles could theoretically be ventricular in origin but the QRS morphology identical to the preexcited QRS argues against this.

Figure 3–30A



This patient's preexcitation pattern was not evident on the surface ECG in the absence of atrial pacing at a rapid rate. Why not?

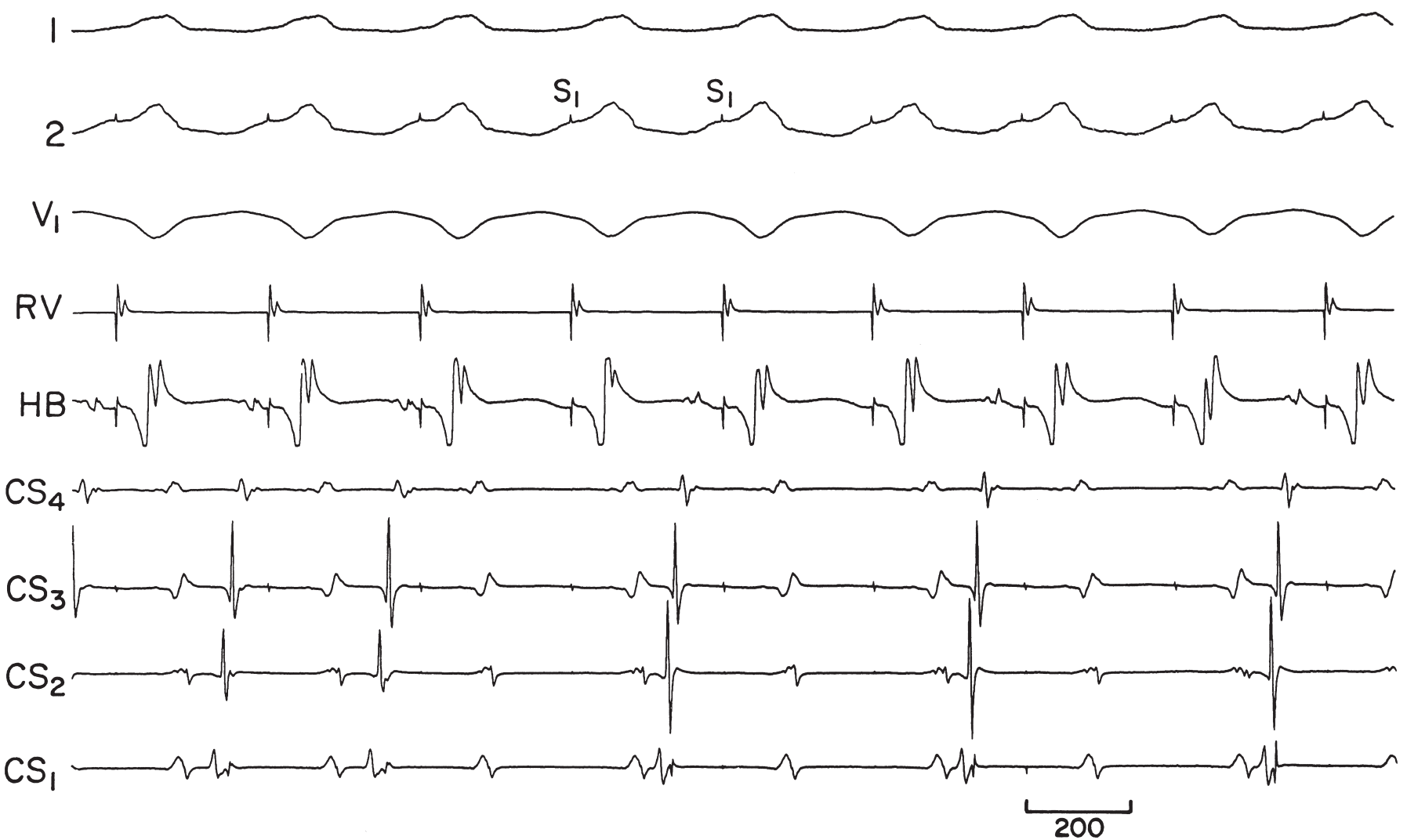
Figure 3–30B



Explanation:

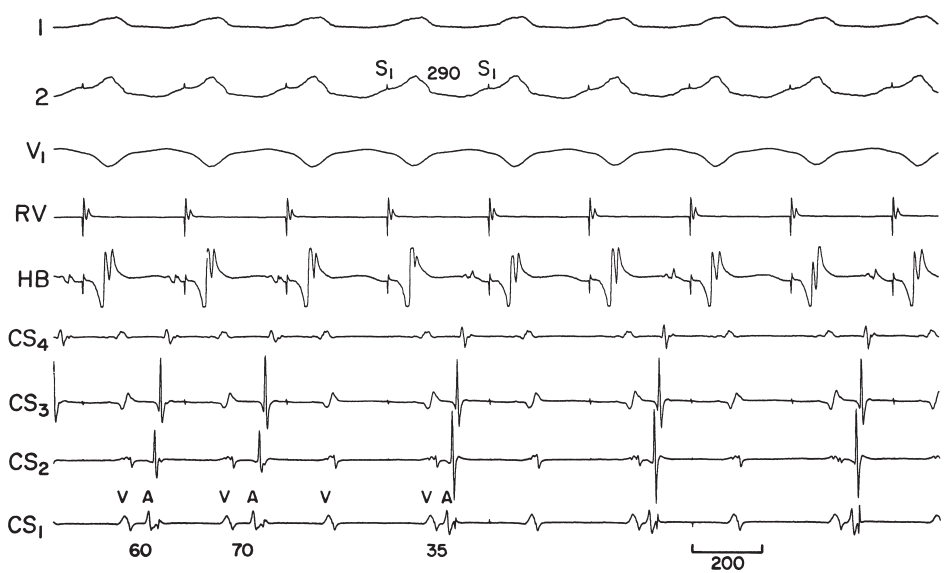
The QRS morphology during pacing clearly shows a left lateral pre-excitation pattern. The record also reveals impressive intra-atrial and interatrial conduction delay during pacing (S to A interval is 150 milliseconds). In sinus rhythm, this delay contributes to the relatively lesser contribution of the left AP to ventricular activation. In sinus rhythm, the atrium at the HB is activated approximately 20 milliseconds after the right atrial site (RA), whereas the atrial electrogram near the AP insertion site in the distal coronary sinus (CS_2) is only activated 60 milliseconds later. The latter allows more direct imput into the AV node than the left AP, causing the balance of fusion to be shifted to the normal conducting system. Importantly, pacing prolongs AV node conduction time but not AP conduction time.

Figure 3–31A



Incremental ventricular pacing in this young man referred for recurrent SVT revealed this phenomenon. How many APs are present?

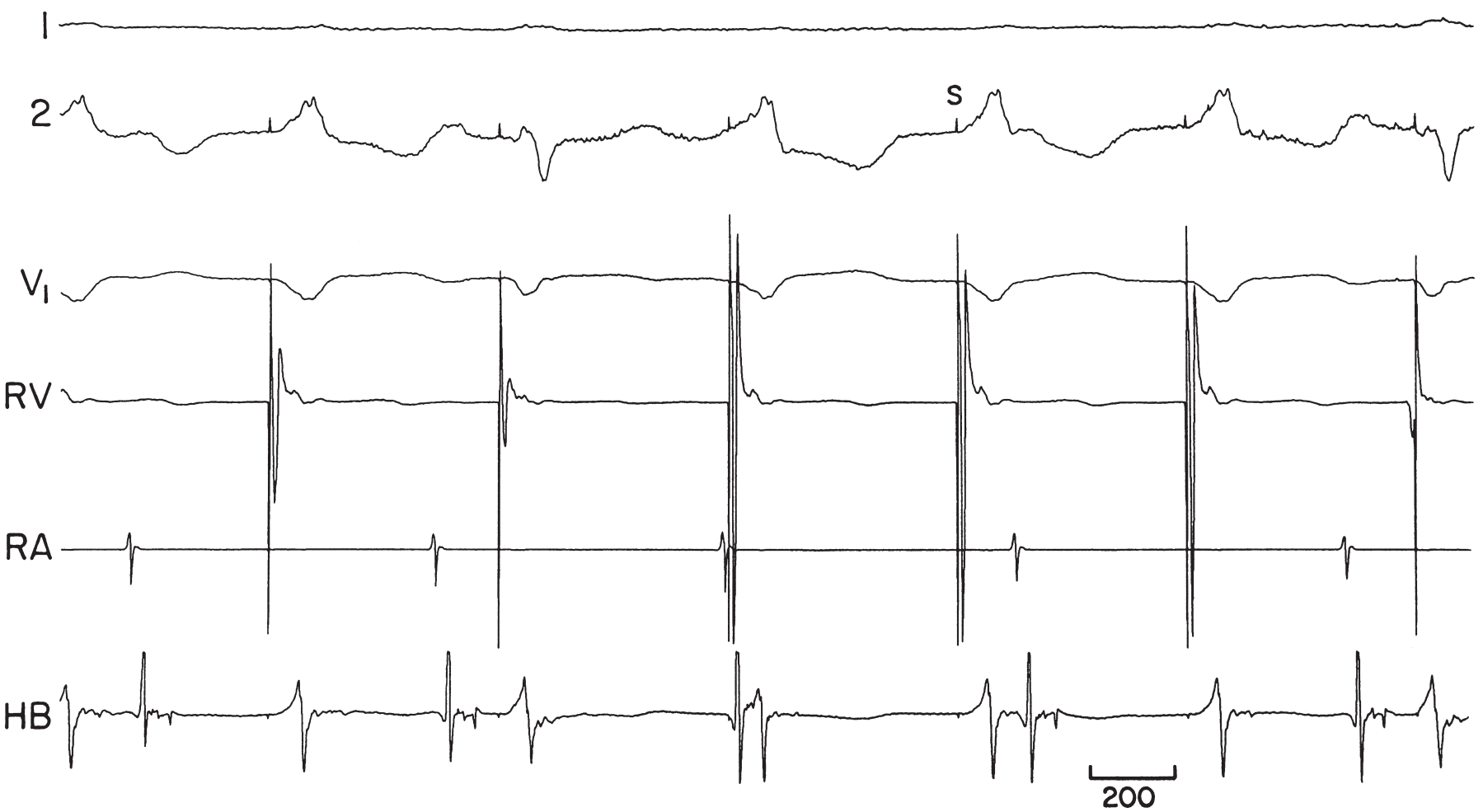
Figure 3–31B



Explanation:

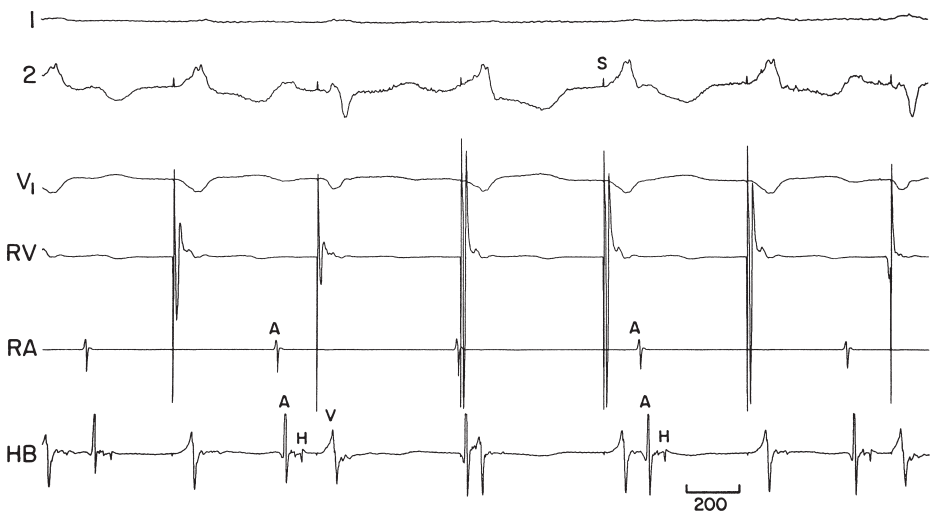
Incremental ventricular pacing has resulted in retrograde block. Earliest retrograde atrial activation is occurring in the distal coronary sinus (CS_1) compatible with conduction over a left lateral AP. The interesting observation is the prolongation of the local VA interval prior to block with subsequent *shortening* of the VA interval in the first ventricular cycle after the blocked one. APs in general conduct in an "all or none" fashion and Wenckebach type block is unusual. *Decremental* conduction such as this, however, is occasionally observed in patients with APs. The retrograde atrial activation sequence appears similar regardless of the VA interval. It is generally believed that this decremental conduction is related to the intrinsic properties of a given AP, although it is conceivable that there are multiple, closely spaced APs with different VA intervals.

Figure 3–32A



The tracing shows ventricular pacing. Does this patient need a pacemaker?

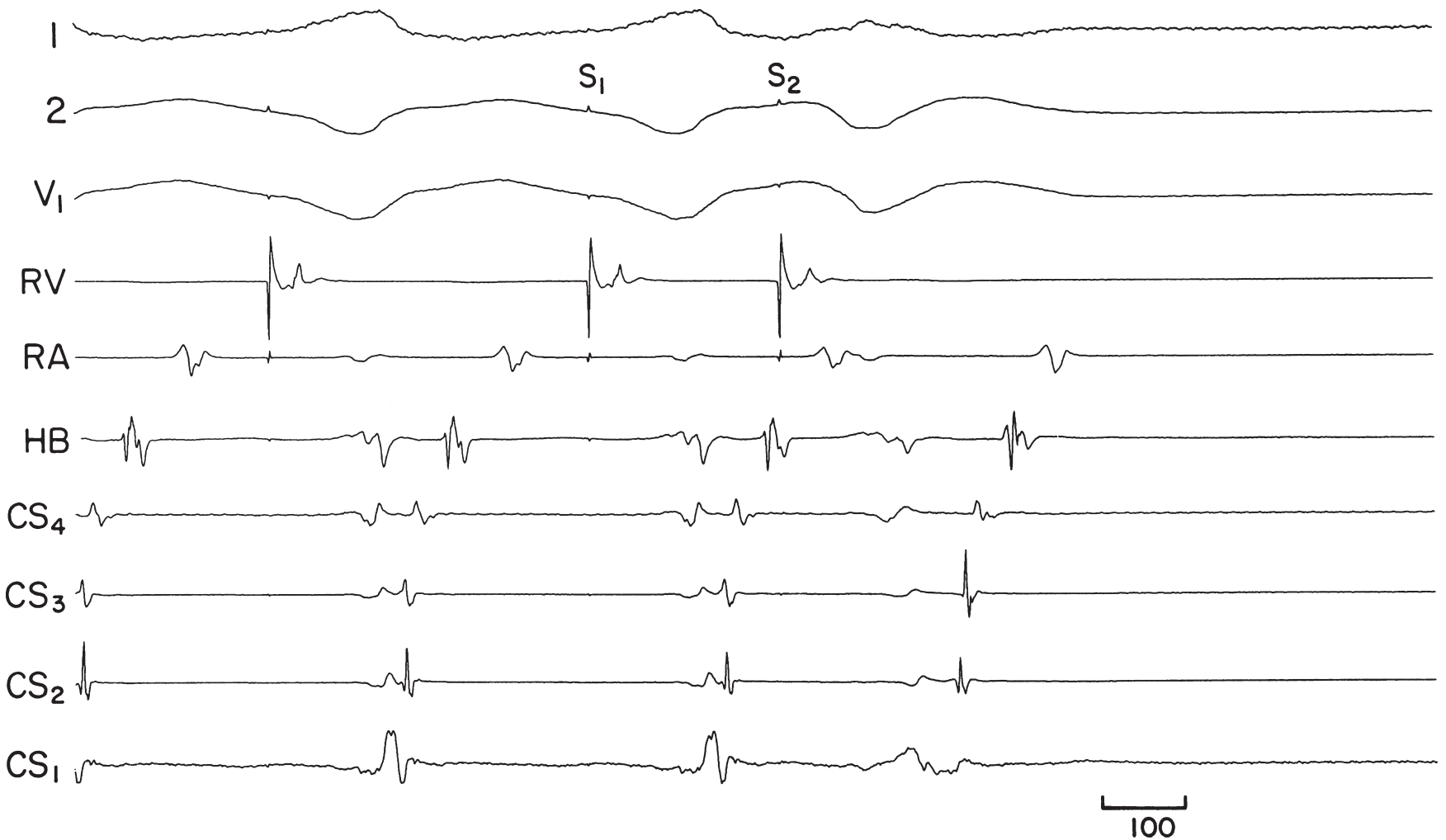
Figure 3–32B



Explanation:

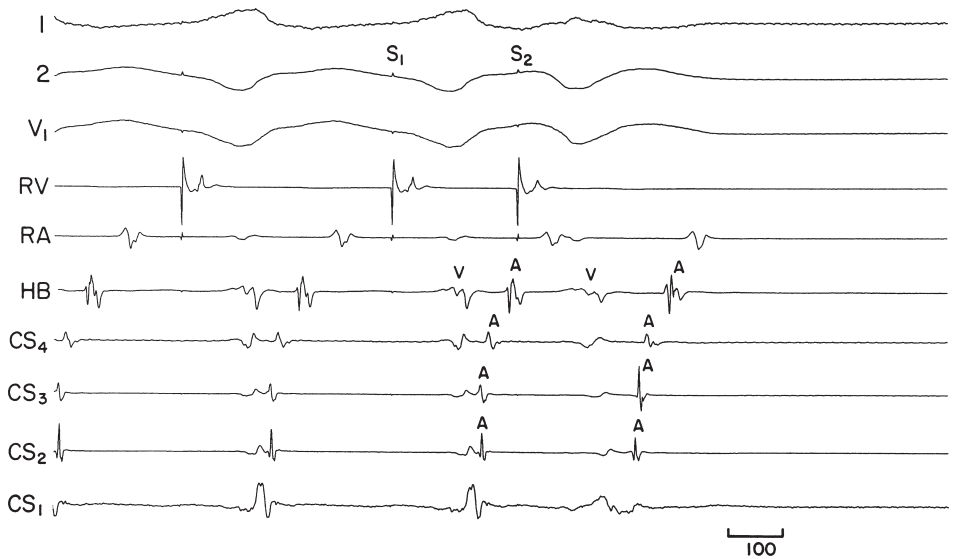
The tracing demonstrates VA dissociation. The atrial activation sequence is high to low, beginning at the right atrial electrogram. The third complex is a *capture–fusion* beat, demonstrating fusion between the ventricular paced cycle and the sinus cycle. The seventh complex appears to be a pure capture beat as the pacing stimulus artifact occurs too late to capture the ventricle. AV block in this situation is physiologic and is best explained by concealed retrograde conduction of the paced ventricular rhythm into the normal AV conduction system. The level of block is below the recorded His bundle. Block below the recorded His is generally pathologic but can be accounted for in this case by concealed retrograde conduction. The patient was undergoing study for suspected tachycardia and was otherwise asymptomatic. He does not need a pacemaker based solely on the EP observations.

Figure 3–33A



This patient was referred for AP ablation. Ventricular extrastimulus testing is displayed with the last two beats of the drive and the extrastimulus shown. CS_4 is at the orifice of the coronary sinus and CS_3 to CS_1 are progressively distal. How many APs are present?

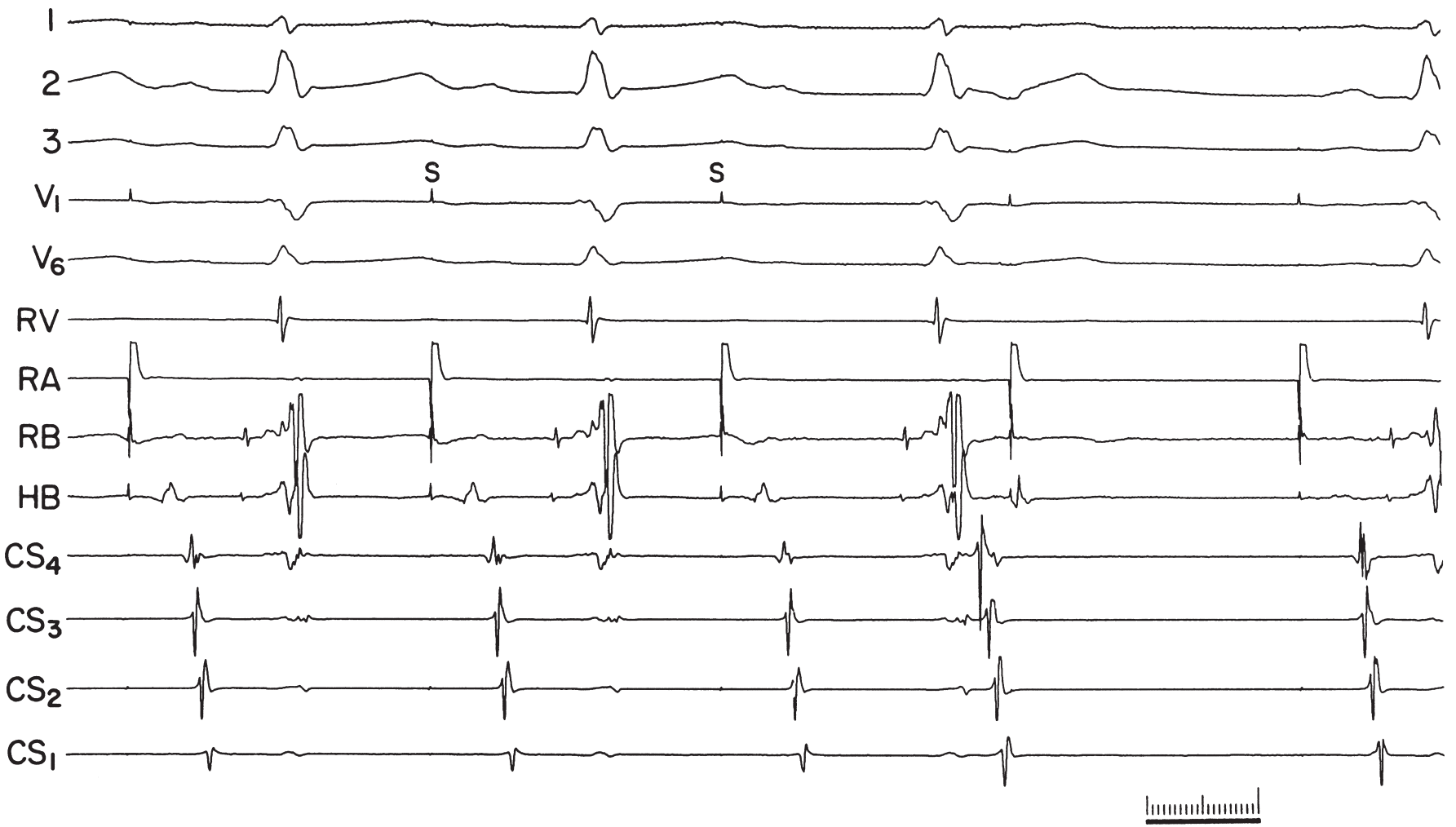
Figure 3–33B



Explanation:

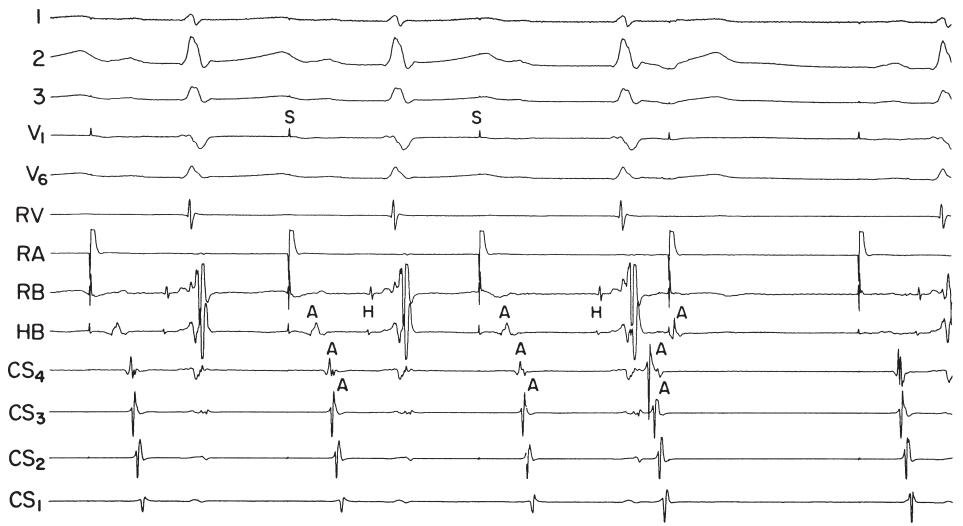
The retrograde atrial activation sequence is eccentric, with earliest activation in the distal coronary sinus clearly indicating a left lateral AP. The VA interval prolongs abruptly with the extrastimulus. This may represent decremental conduction over the AP as illustrated in Fig. 3–31. In this case, however, there is a subtle but distinct change in atrial activation sequence, which in this patient represented a second AP slightly more distal to the first with a longer VA conduction time. This was verified by ablation.

Figure 3–34A



This tracing was observed during atrial pacing in a young patient studied for tachycardia. The interval between stimuli (S) is 520 milliseconds. CS_4 is at the orifice of the coronary sinus and CS_3 to CS_1 are progressively distal. What should be ablated?

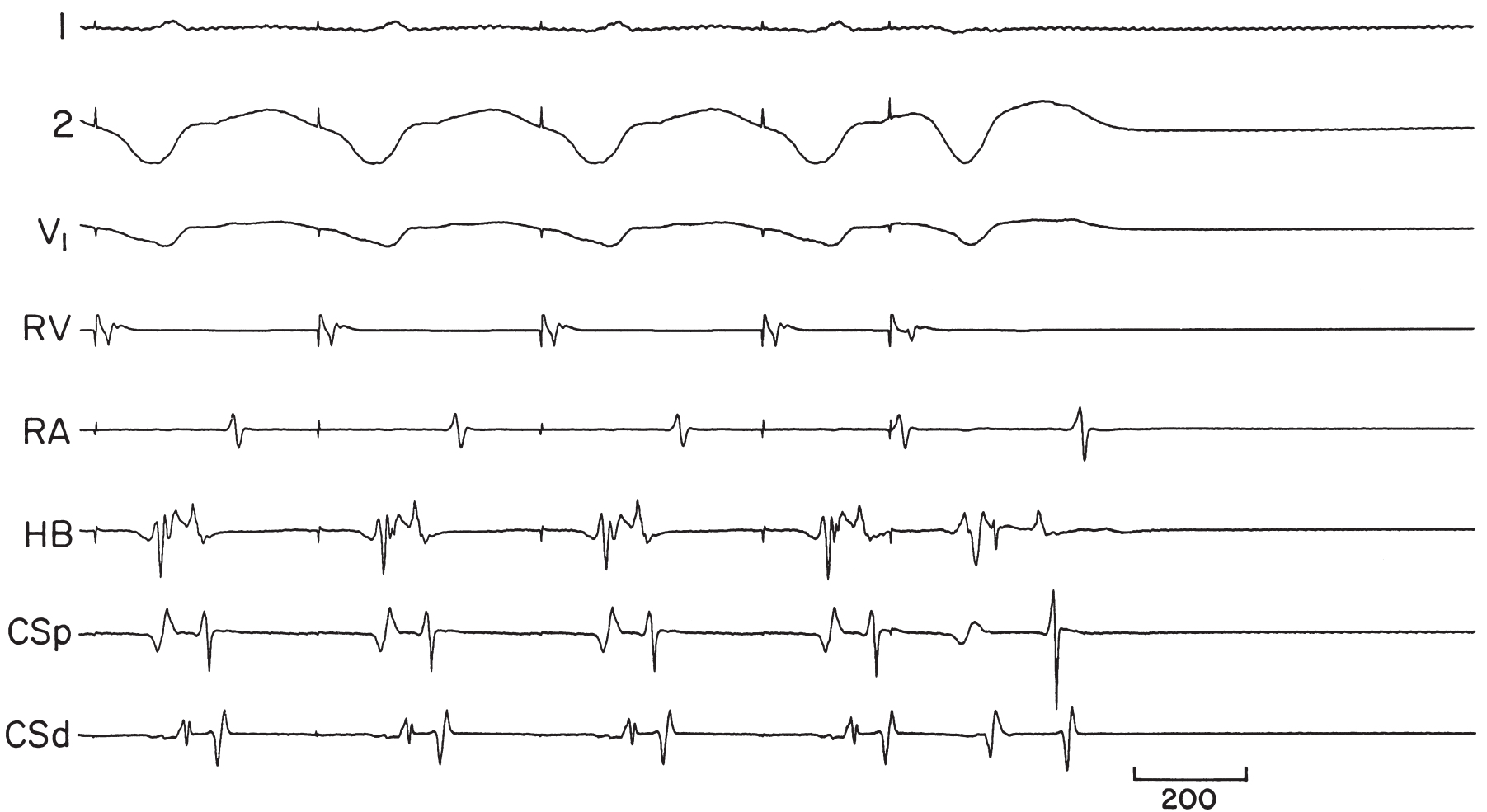
Figure 3–34B



Explanation:

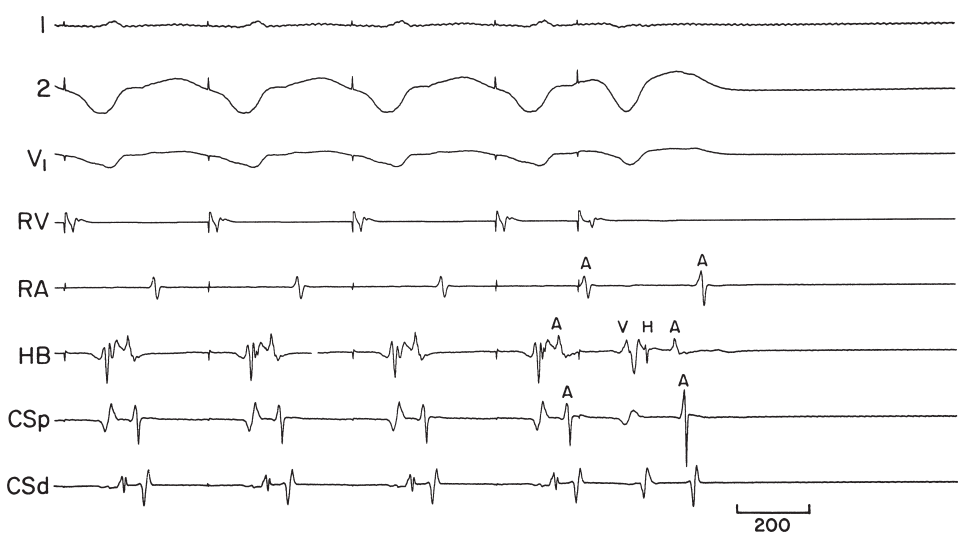
During atrial pacing, there is a sudden prolongation of the AH interval, representing a shift to a slow AV node pathway (third QRS). This is associated with an atrial echo cycle that does not subsequently conduct to the ventricle. The atrial echo largely preempts the stimulus artifact (local atrial capture may be present but clearly does not depolarize most of the atrium), which is inscribed shortly thereafter. The atrial echo shows earliest activation just inside the orifice of the coronary sinus (CS_4). This is compatible with either conduction over a posteroseptal AP or a retrograde AV node pathway. In this particular study, observations during sustained tachycardia observed elsewhere verified the diagnosis of a posteroseptal AP. Atrial reciprocation in this patient required conduction delay, which in this instance was afforded by prolonged conduction over the slow AV node pathway. In this instance, slow pathway ablation could be reasonably contemplated to prevent tachycardia if ablation of the posteroseptal pathway was otherwise problematic.

Figure 3–35A



During progressively more rapid ("incremental") ventricular pacing in this patient, the VA conduction time changed only marginally from the slowest to the fastest paced rate prior to block. With ventricular extrastimulus testing, however, there was sudden prolongation of the VA interval. Why?

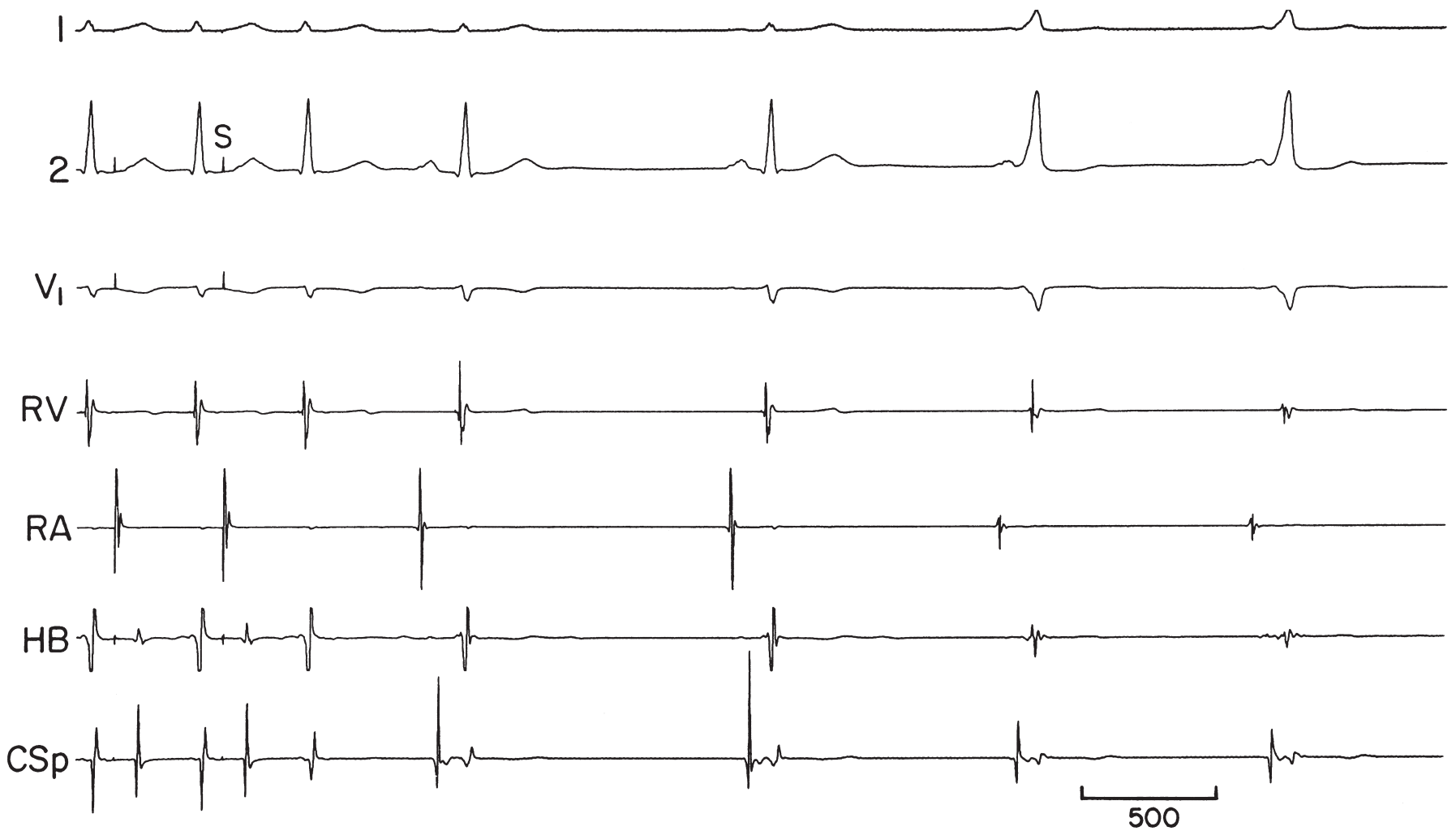
Figure 3–35B



Explanation:

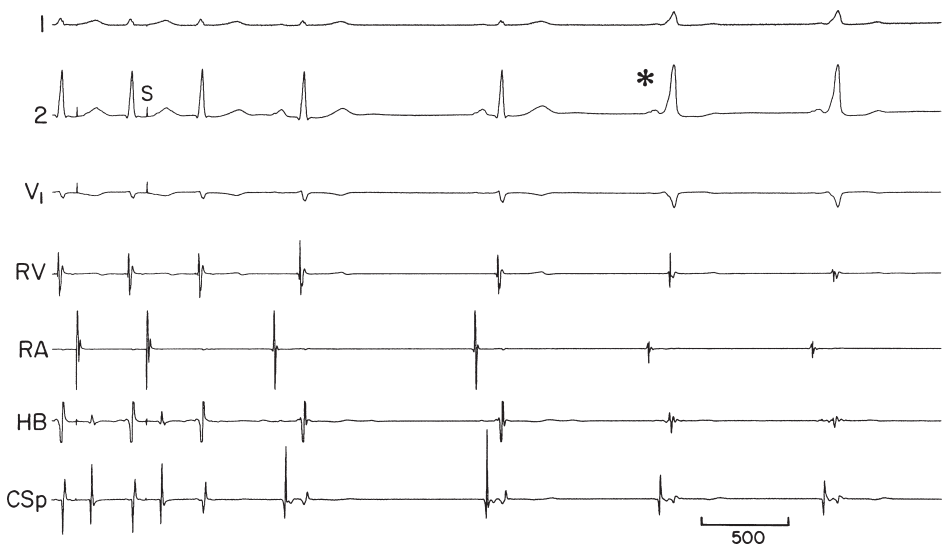
The His deflection is not obvious during the ventricular drive but clearly emerges after ventricular depolarization subsequent to the extrastimulus. The abrupt change in CL results in conduction delay in the HPS, which is usually not observed during incremental ventricular pacing with a more gradual decrement in CL. A sudden "jump" of the VA interval with ventricular extrastimulus testing is usually related to prolongation of the VH interval rather than being representative of dual AV node pathways. It is not unusual to observe only minimal changes in the VA interval during incremental ventricular pacing, an observation that does not necessarily suggest the presence of an AP.

Figure 3–36A



This patient was referred for assessment of the Wolff–Parkinson–White syndrome. Preexcitation was observed during incremental atrial pacing down to a CL of 400 milliseconds at which point the QRS abruptly normalized. The end of this pacing run is illustrated in the figure. What phenomenon is observed?

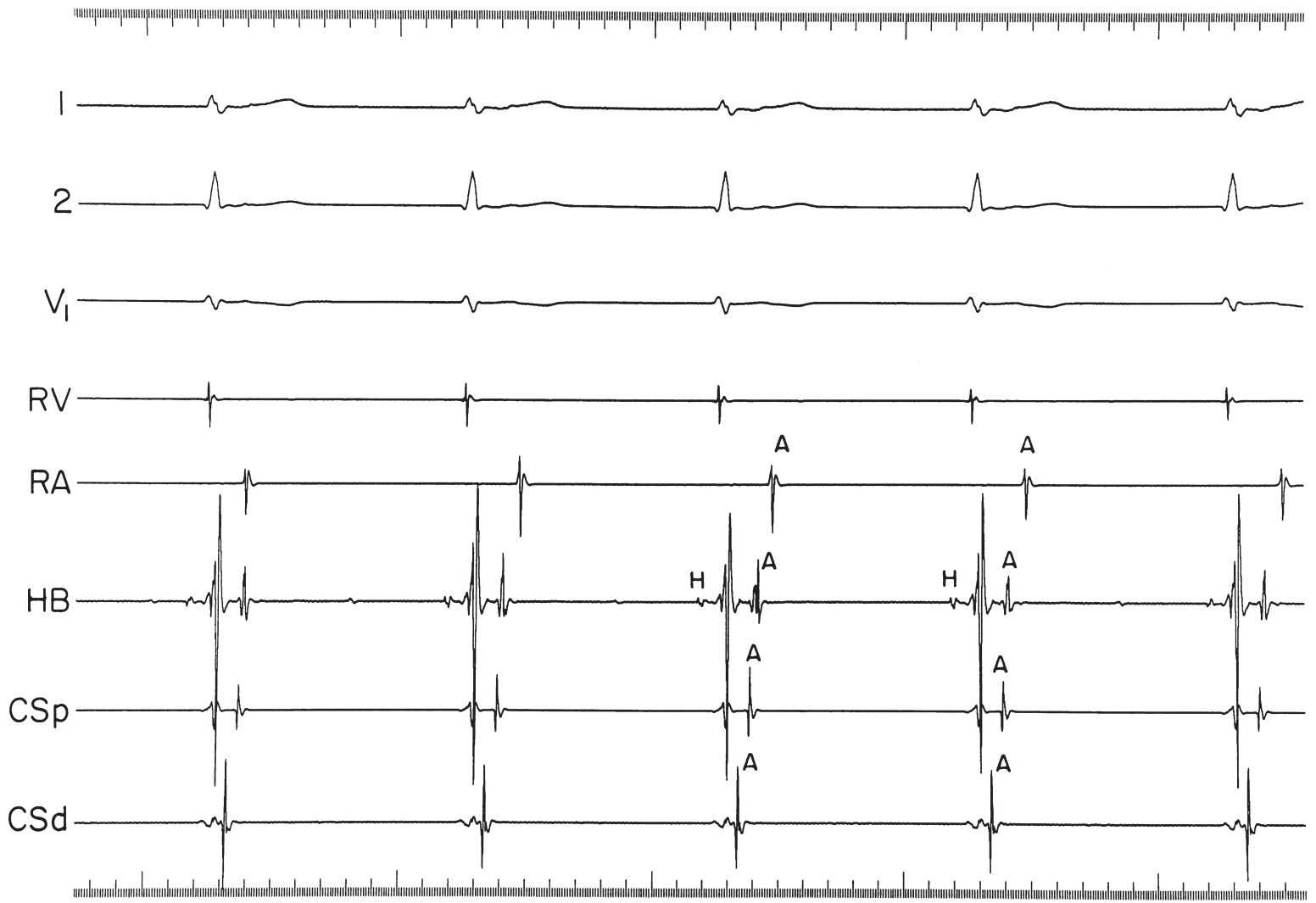
Figure 3–36B



Explanation:

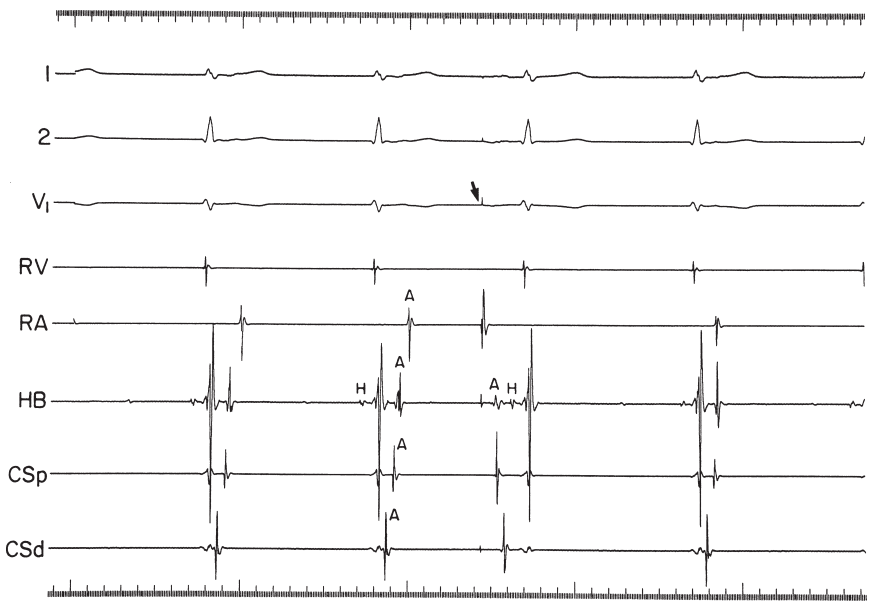
The QRS remains normal for the first two spontaneous sinus cycles after cessation of pacing. It is only the third spontaneous sinus cycle that results in resumption of preexcitation. Knowing that preexcitation persisted until CL 410 milliseconds during incremental pacing would lead one to predict that the first two spontaneous cycles after termination of pacing should be preexcited since sufficient delay was present to permit recovery of excitability over the pathway. "Fatigue" is defined as a transient failure of conduction after repetitive excitation and was observed in this patient after the cessation of rapid pacing or a sufficiently premature extrastimulus. The duration of fatigue was dependent on both the rate and duration of pacing. Repetitive excitation may produce prolonged and persistent depolarization of the diastolic membrane potential, which is unfavorable to impulse propagation. The mechanism of this phenomenon is unknown.

Figure 3–37A



What is the differential diagnosis of this rhythm? How would one prove the diagnosis?

Figure 3–37B



Explanation:

The atrial activation sequence is eccentric (see Table 1–7) suggesting a diagnosis of atrial tachycardia or AV reentry. With either diagnosis, one must postulate the presence of a slow AV node pathway resulting in a very long AH interval. It is also noteworthy that the atrial activation pattern does not support the presence of a retrograde fast AV node pathway. Such a long AH interval (770 milliseconds) would, however, be distinctly unusual for a beat conducted over the AV node.

A third possibility is a junctional rhythm with retrograde conduction over a left lateral AP, the latter not directly involved in the mechanism of the rhythm. This third possibility does not require a slow pathway as part of the explanation. A premature atrial depolarization inserted in middiastole should and did clarify this diagnosis. It conducted with a short AH interval, verifying the diagnosis of junctional rhythm with retrograde conduction over an AP. If the long diastolic interval were occupied by slow pathway conduction, this atrial extrastimulus likely would not have conducted to the His and certainly not with a short AH interval.

Chapter 4

Narrow QRS Tachycardia

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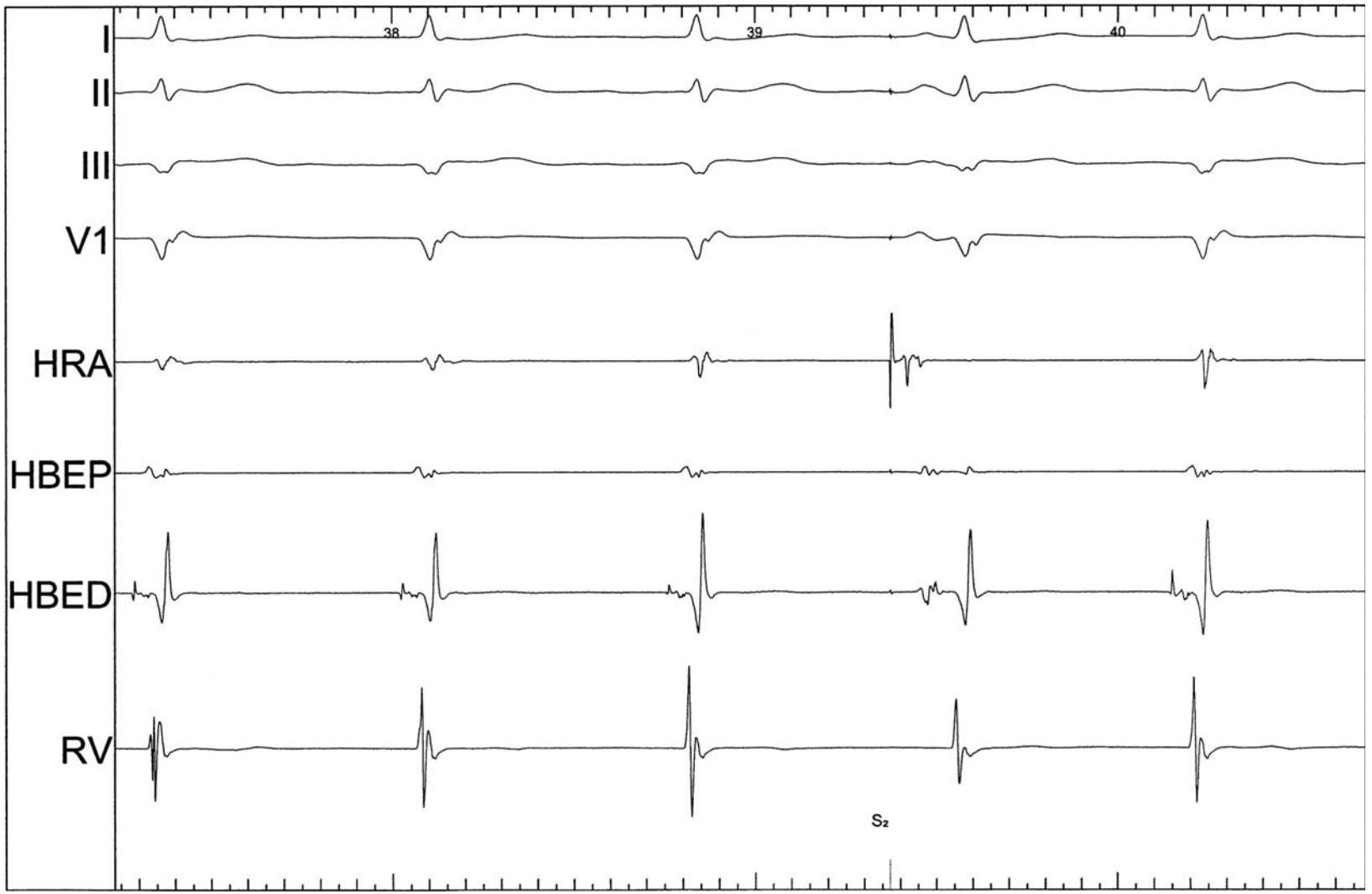
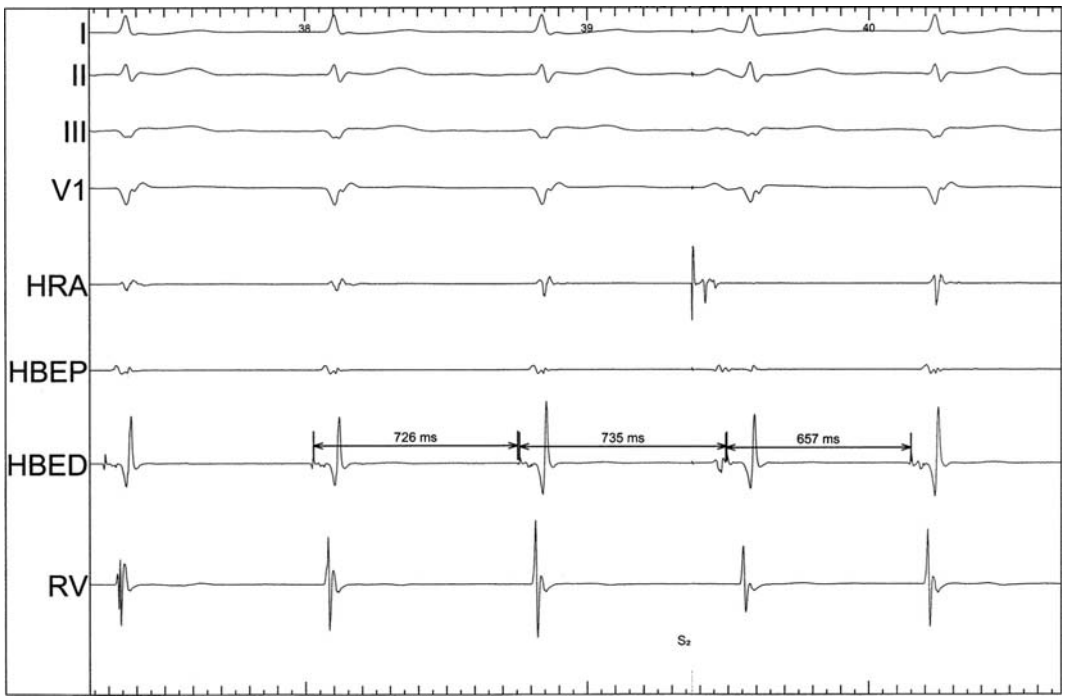
Figure 4–1A



A 35-year-old woman with a history of palpitations and supraventricular tachycardia was noted to be in this rhythm after catheters were placed in her heart. A maneuver was performed to identify the mechanism of tachycardia. Has this helped?

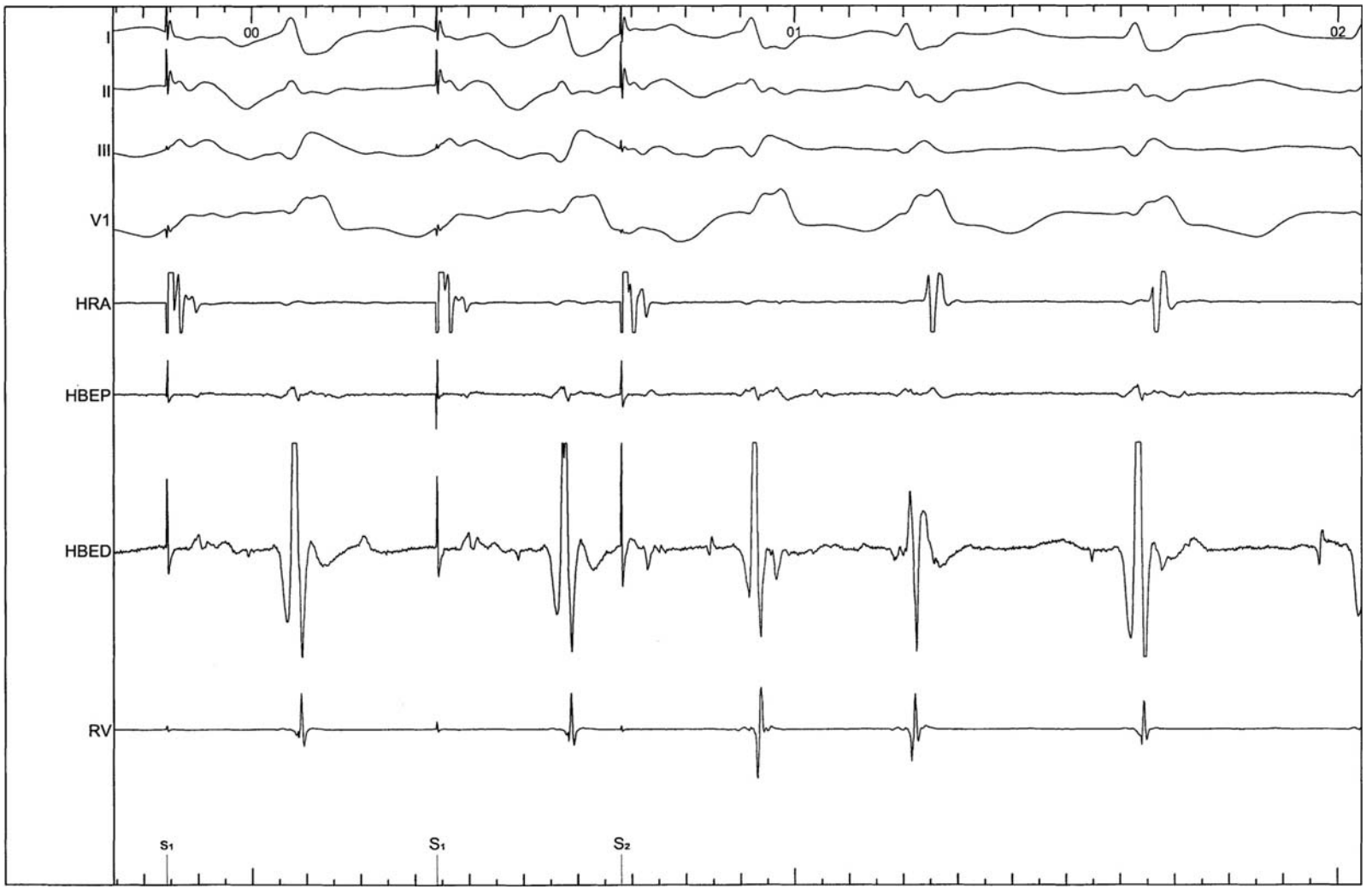
Figure 4–1B



Explanation:

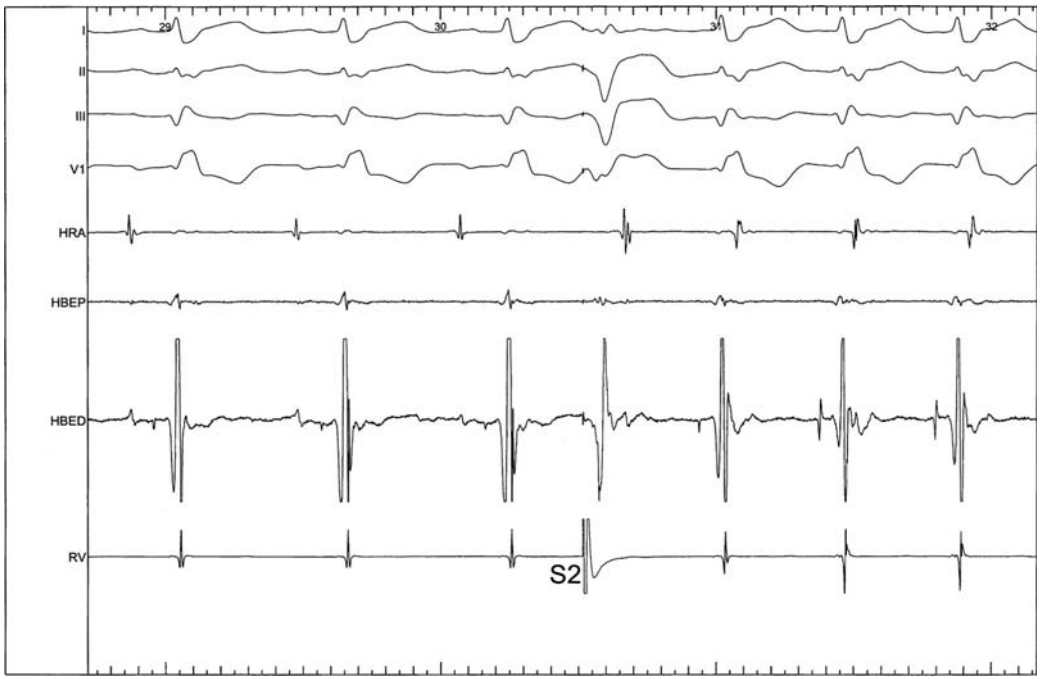
This is a relatively slow supraventricular tachycardia with a His electrogram preceding each QRS complex (HBED lead). The cycle length of tachycardia varied slightly from 726 to 735 milliseconds. The differential diagnosis includes an automatic junctional rhythm, slow–fast AV node reentry, or an atrial tachycardia with anterograde conduction over a slow pathway. The very short VA interval excludes AV reentry. A premature atrial complex is introduced at a time when the His bundle electrogram has already been activated and results in a shortening of the *subsequent* H–H interval to 657 milliseconds. There should be no change in the subsequent H–H interval if the mechanism is an automatic junctional rhythm because the junction would have already discharged and therefore the premature atrial complex could not have affected the subsequent cycle length. The shortening of the H–H interval is consistent with slow–fast AV node reentry in which the premature atrial complex engages a slow pathway earlier than anticipated and affects the next H–H interval. It could have also prolonged the next H–H interval and that would have still supported this diagnosis. Can one totally rule out an atrial tachycardia with conduction over a slow pathway? Note that the His to high rate interval remains constant even though the H–H interval changes, a finding consistent with AV node reentry but not atrial tachycardia. Indeed, this patient had relatively slow AV node reentry that was successfully ablated at a site around the coronary sinus ostium.

Figure 4–2A



A 59-year-old woman underwent electrophysiologic evaluation for recurrent episodes of tachycardia. She had known right bundle branch block. Explain how initiation of tachycardia occurs, the most likely mechanism for it, and the tachycardia diagnosis.

Figure 4–2B

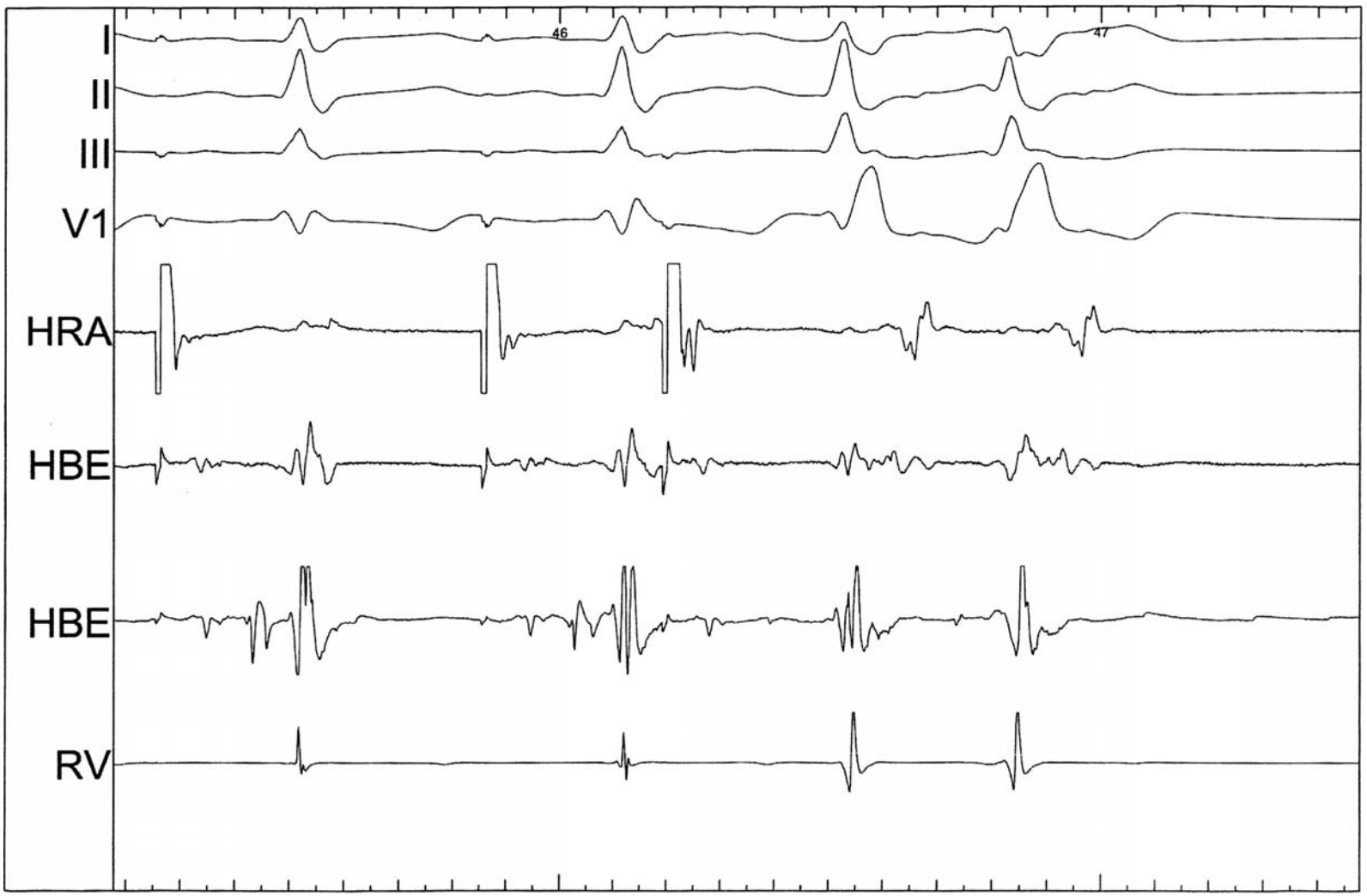


Explanation:

A single atrial premature stimulus is introduced at 340 milliseconds during an atrial paced cycle length of 500 milliseconds. The premature complex conducts over the AV node and induces a short VA interval tachycardia that is most consistent with AV node reentry. This mode of induction is referred to as a two-for-one response and seems to break the laws of initiation for a reentrant circuit. In other words, a single premature complex is conducted over both the fast and slow AV nodal pathways to initiate tachycardia.

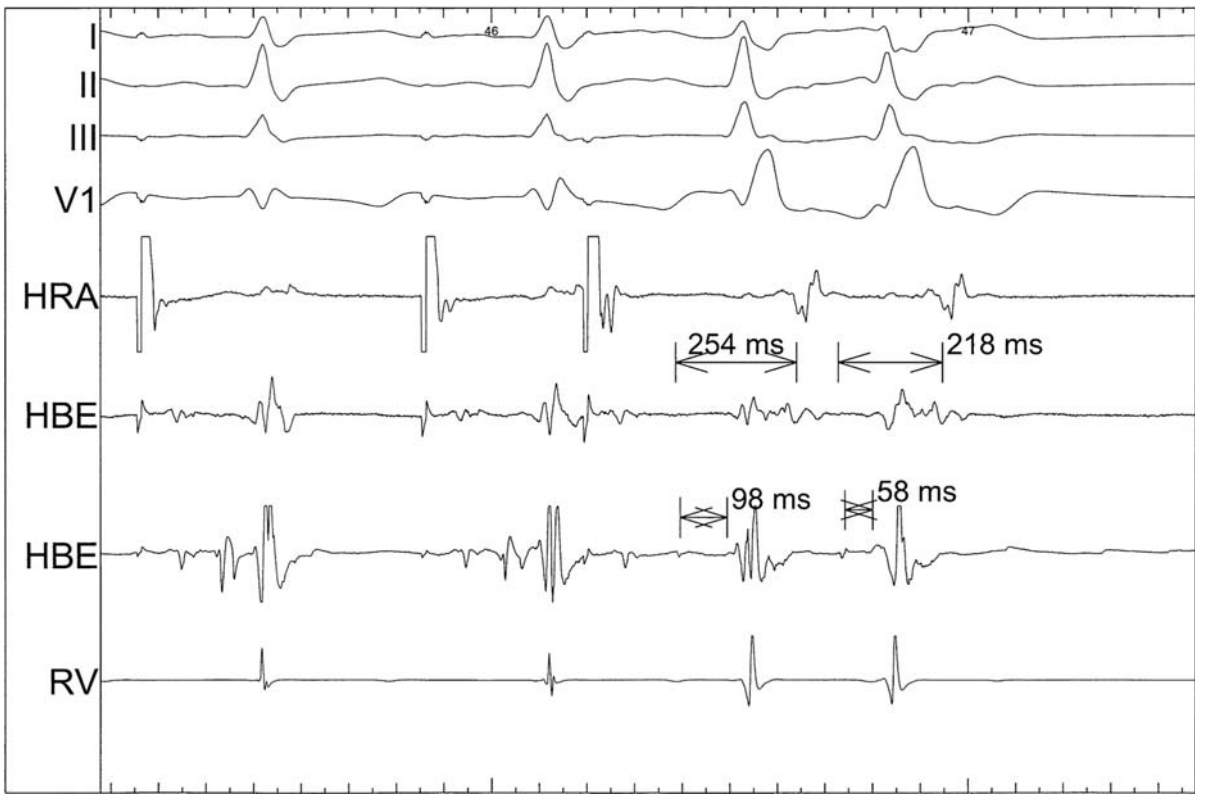
Classical teaching of reentry proposes three requisites to form the circuit: two pathways of conduction, initial block in one pathway, and slowing of conduction in the second pathway to allow reexcitation of the initial blocked pathway and subsequent reentry. In this example, there is no block in either pathway since the premature atrial complex conducts anterogradely over both. This "breaking of the rules" is best explained by the inability of the initial fast pathway conduction to conceal retrogradely into the slow pathway, which thereby allows the slow pathway to conduct in an anterograde manner and start tachycardia. In all cases we have studied like this, slow–fast AV node reentry can always be induced during premature ventricular stimulation or incremental ventricular pacing, consistent with essentially minimal to no retrograde conduction into the slow pathway. This is shown with a premature ventricular complex initiating the same tachycardia in this patient at another point in this study. The successful ablation site was just anterior to the coronary sinus ostium and not substantively different from the usual site one selects for patients with AV node reentry.

Figure 4–3A



A 29-year-old man with a history of paroxysmal SVT (PSVT) undergoes electrophysiologic evaluation. During programmed atrial stimulation in the control state, a premature atrial complex could only induce a few beats of tachycardia. What is the diagnosis?

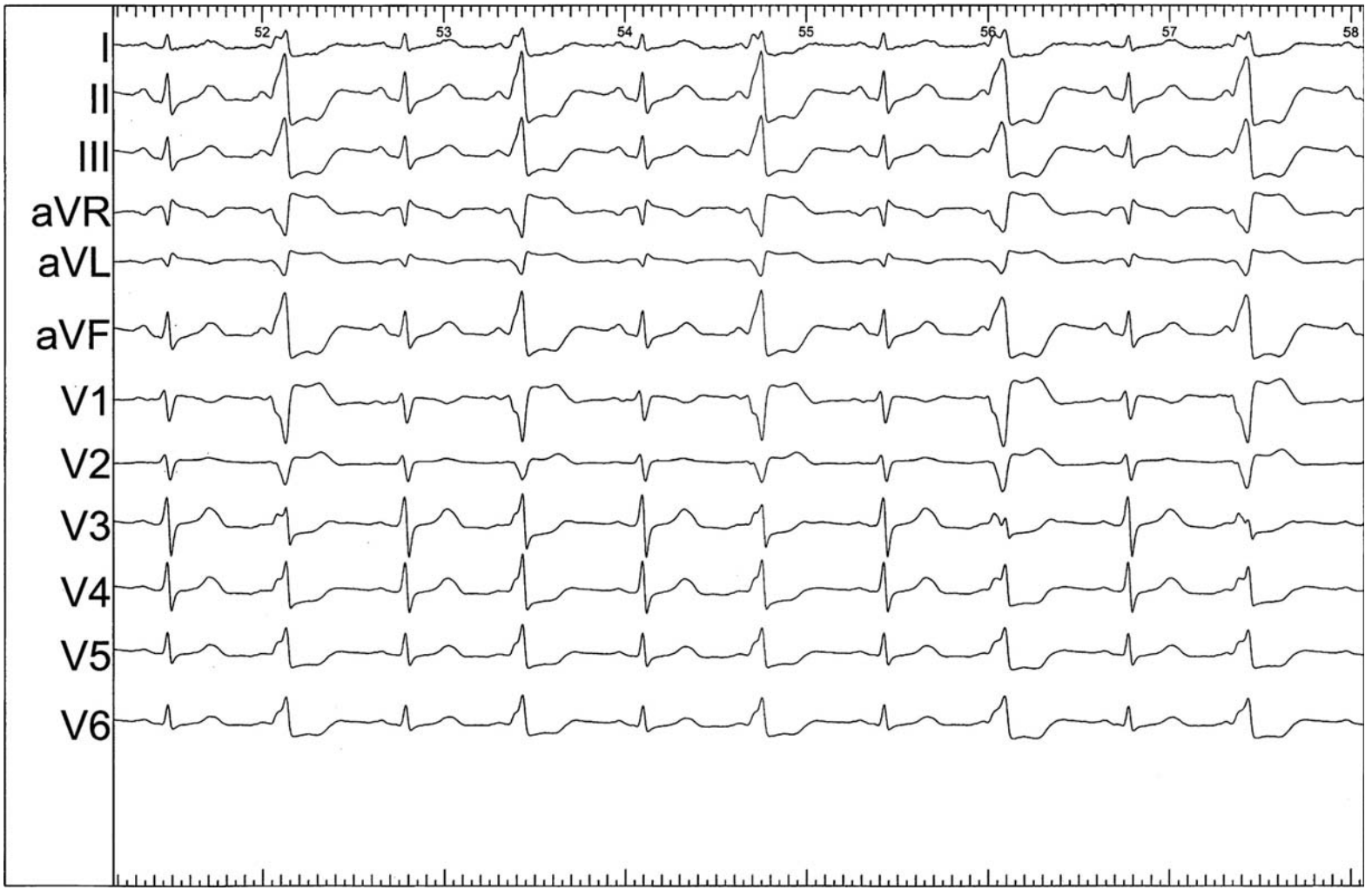
Figure 4–3B



Explanation:

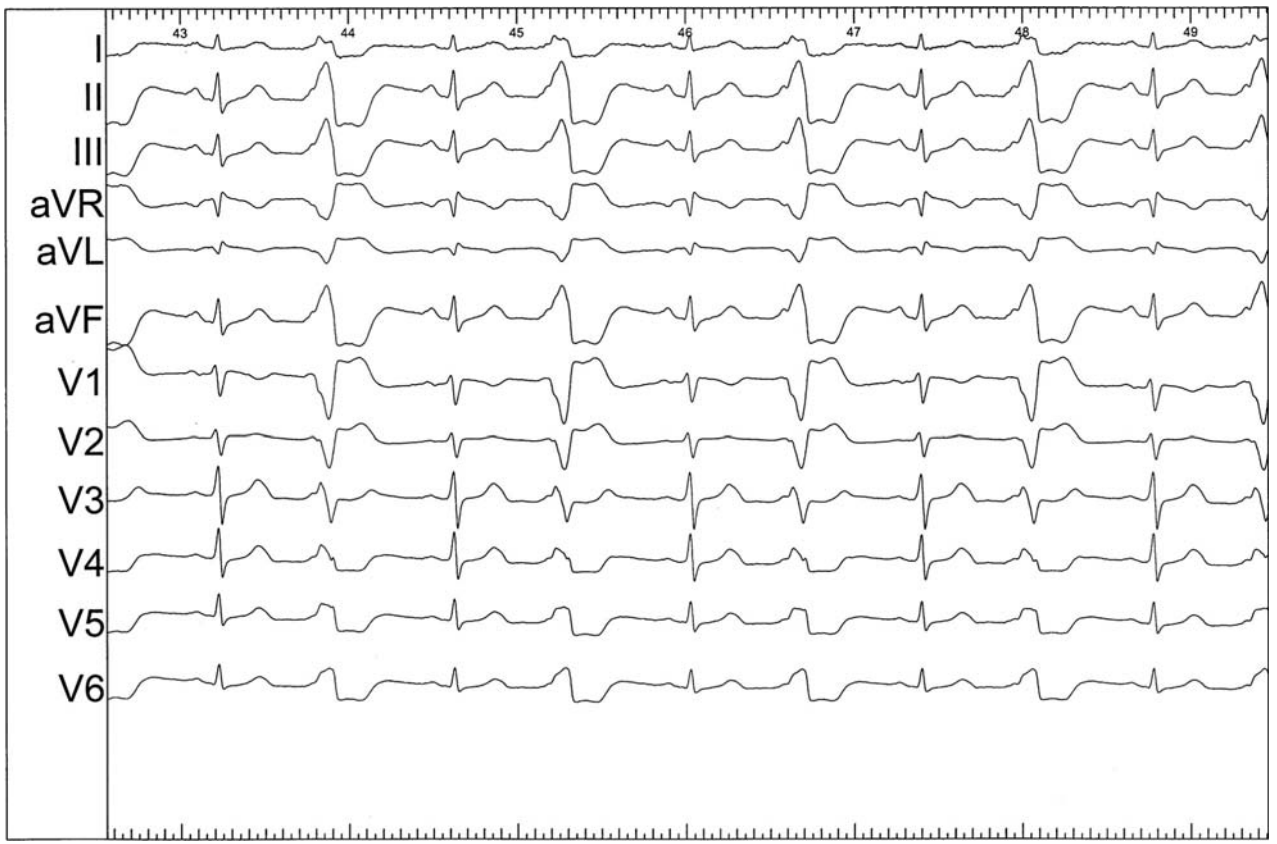
As discussed in Chapter 1, it is always important to look for "wobble" or changes in intervals during tachycardia and these usually occur at the initiation or termination of tachycardia. This patient has some key observations that prove the mechanism of at least these two echo complexes. The accompanying figure shows an initial prolonged HV interval of 98 milliseconds followed by a shortening to 58 milliseconds. The HA interval on the first echo complex is 254 milliseconds and this shortens to 218 milliseconds as the HV interval shortens. The change in HV predicts the change in HA. The only supraventricular tachycardia that utilizes both the His–Purkinje system and ventricle in the circuit is AV reentry, and that is the diagnosis. Also note that termination of these echo complexes occurs with an atrial electrogram without conduction to the His bundle, which was a consistent finding and strongly suggests an AV node–dependent mechanism. Indeed, during isoproterenol infusion tachycardia persisted and ablation was successful.

Figure 4–4A



A 46-year-old woman with a history of recurrent palpitations undergoes an electrophysiologic study. This simultaneous 12-lead electrocardiogram was recorded in the electrophysiology laboratory prior to the insertion of catheters. Do you think this patient will have AV reentry as the cause of her palpitations?

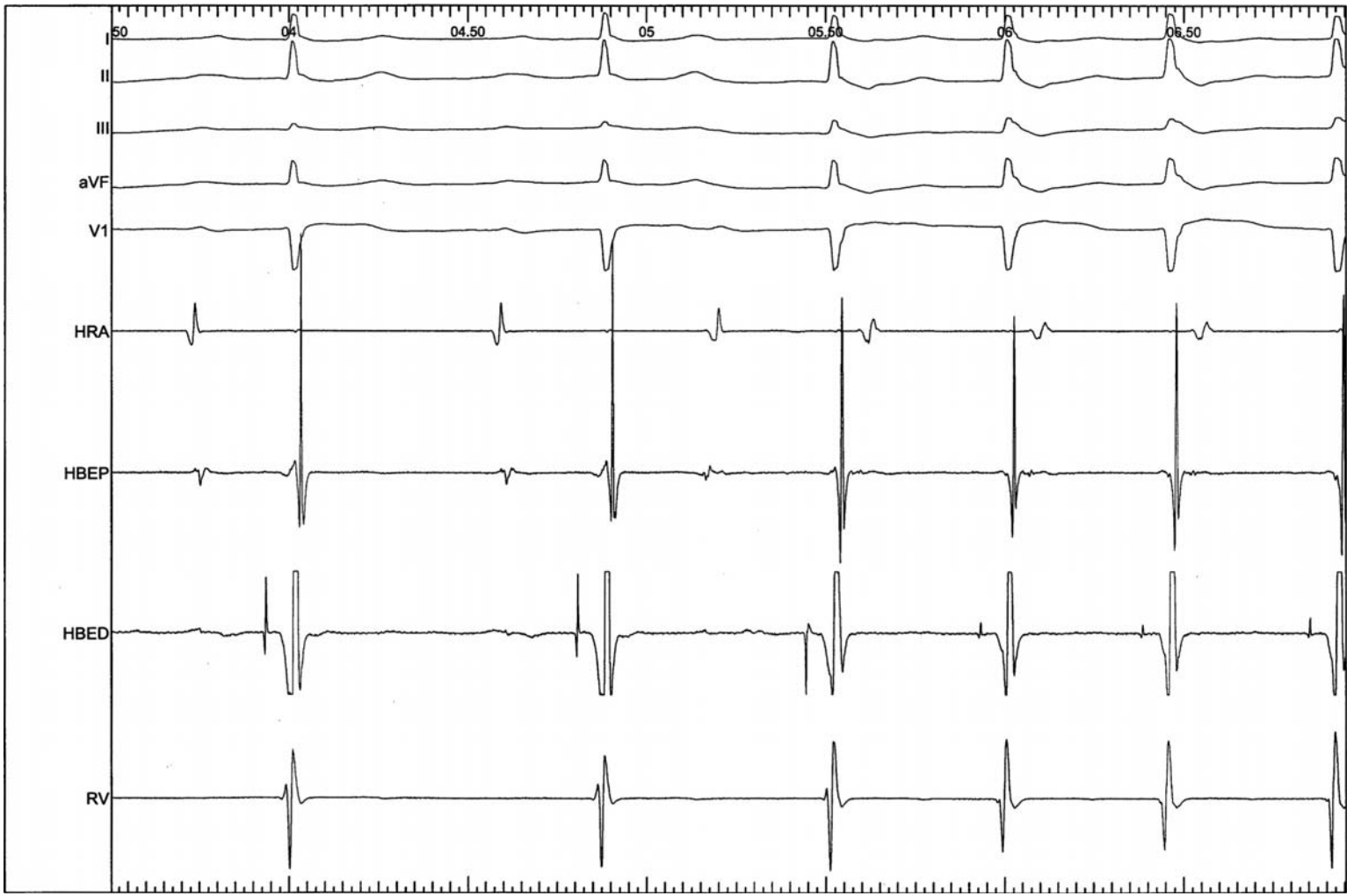
Figure 4–4B



Explanation:

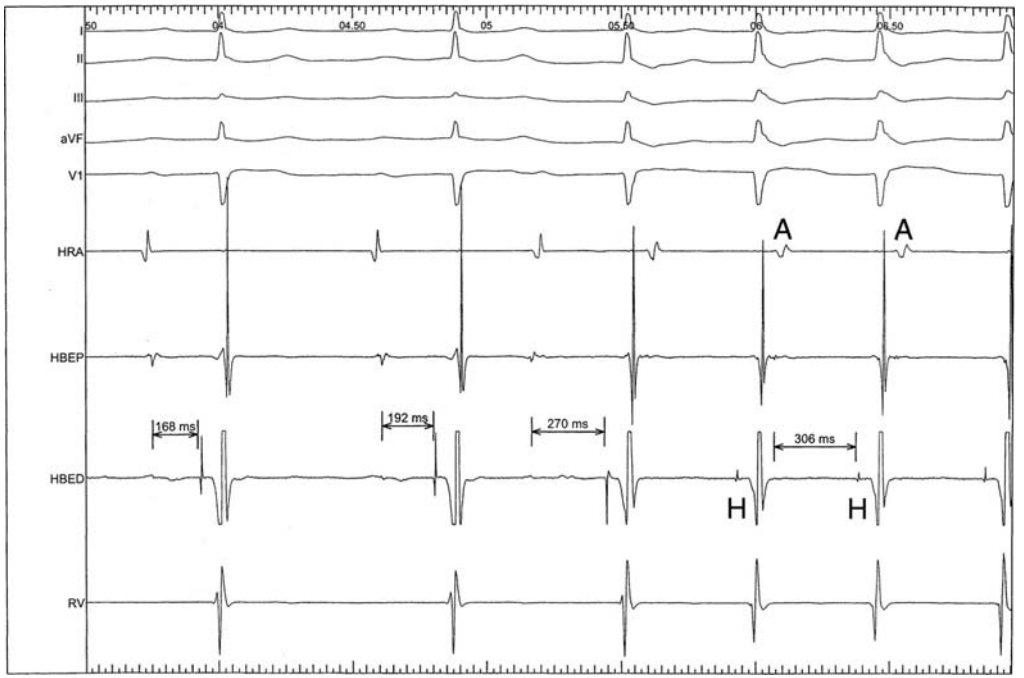
This tracing demands careful measurement before coming to a conclusion. It also reminds us of the famous Shakespearean quote, "all that glitters is not gold." At first glance it appears the patient may have 2:1 conduction over an accessory pathway. One of the hallmarks of an AV pathway, which is the typical accessory pathway, is a short PR interval that remains constant assuming there is no change of the site of origin of the P wave. Note that there is an apparent short PR interval associated with the wide QRS complexes. Importantly, the "PR interval" is not constant and this is clearly seen in the last two wide QRS complexes on this tracing. In essence, this is a "fooler" and is really a series of critically timed premature ventricular complexes that happen to be late in timing, in essence, "R on P waves." The accompanying figure shows that these wide QRS complexes are PVCs. Indeed, this patient had PVCs and nonsustained VT that occurred in the presence of isoproterenol and no evidence of an accessory pathway.

Figure 4–5A



A 72-year-old gentleman has had nearly incessant tachycardia for the past 6 months and the following tracing occurred at electrophysiologic study. What is the diagnosis?

Figure 4–5B

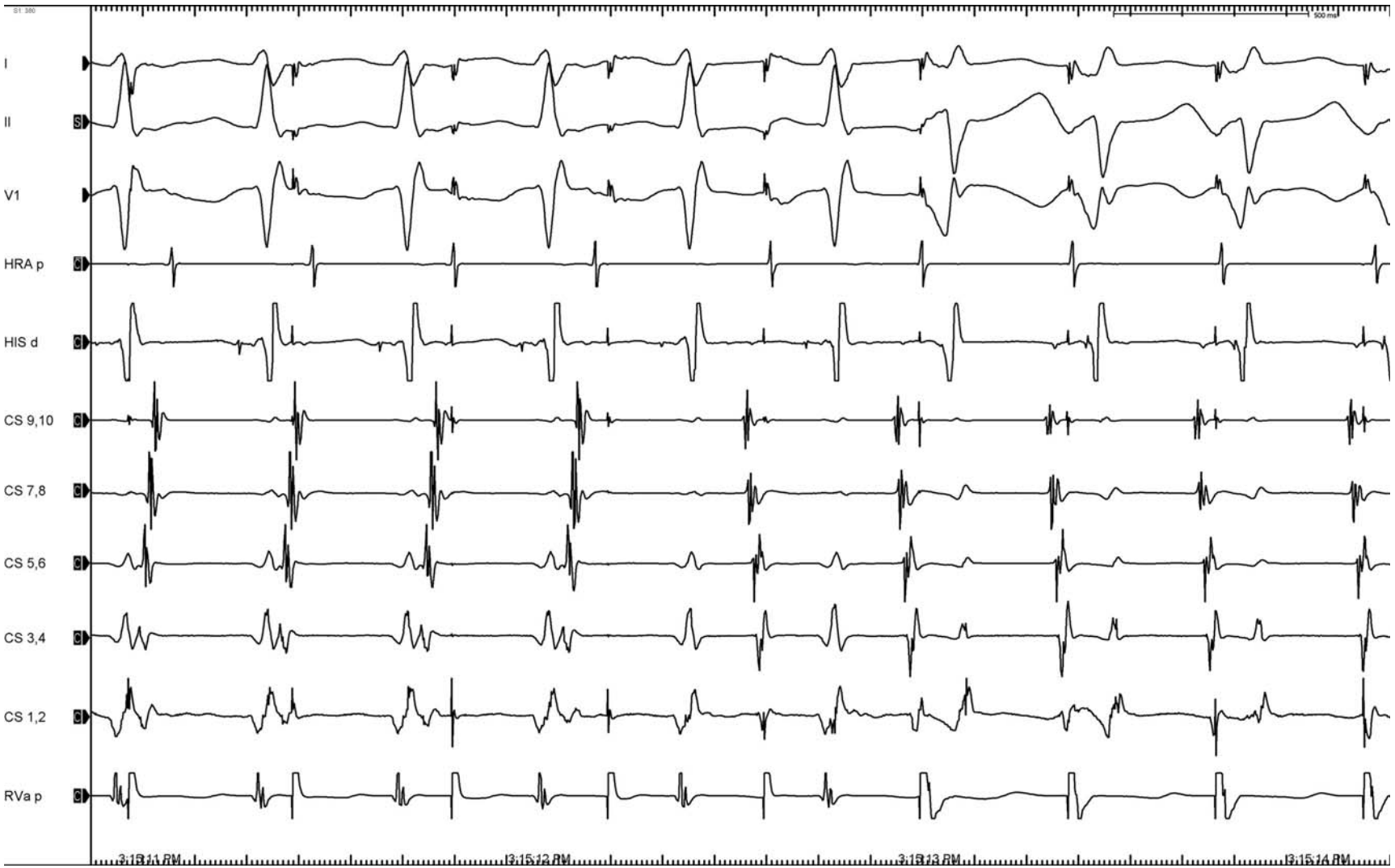


Explanation:

This is an extremely unusual variant of a much more common problem, slow–fast AV node reentry, which is rarely incessant. Typically, tachycardia starts with a premature atrial complex, infrequently with a PVC, and will terminate suddenly and not spontaneously reoccur. This patient typically showed Wenckebach conduction block over the fast AV nodal pathway until a critical AH interval occurred that allowed tachycardia to be initiated. Tachycardia would spontaneously terminate only to restart with another Wenckebach sequence. On occasion, as noted in the accompanying figure, during the Wenckebach sequence a PAC would occur, and here the AH interval increases from 168 to 270 milliseconds at which time reentry occurred with a relatively short HA interval. The tachycardia cycle length is 306 milliseconds and even though there are variable AH intervals at the start of tachycardia, the HA interval remains nearly constant, consistent with AV node dependence, ruling out atrial tachycardia as a mechanism. The VA interval is short but not short enough to eliminate conclusively AV reentry, which was excluded at electrophysiologic study.

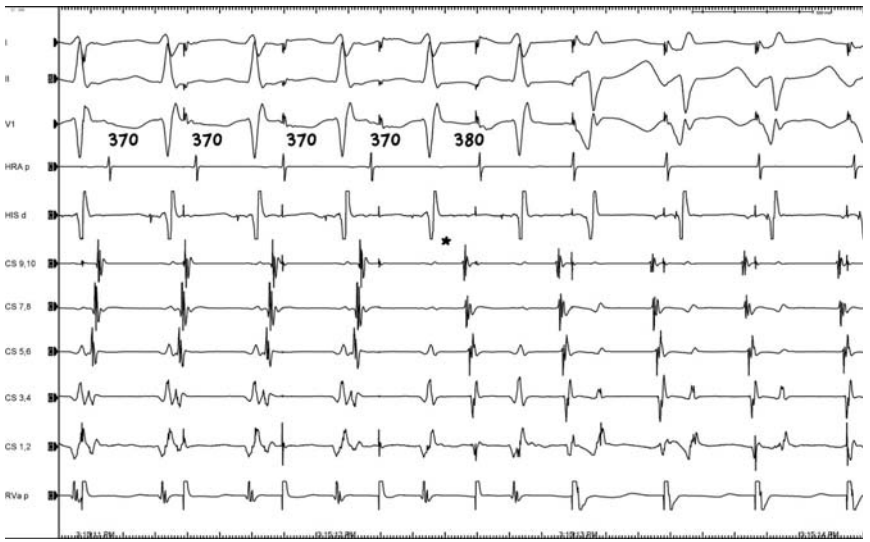
As you prepare to ablate the slow pathway, you might wonder whether the patient will have repetitive sequences of Wenckebach block after elimination of slow pathway conduction. However, in such situations what usually happens is 1:1 conduction over the fast pathway after elimination of slow pathway conduction. The presumption is that electrotonic interaction occurs between the two AV nodal pathways and with elimination of the slow pathway the fast pathway can now maintain 1:1 conduction, which was what occurred in this patient.

Figure 4–6A



The patient has a narrow QRS tachycardia induced and ventricular pacing has begun to entrain the tachycardia. What has happened?

Figure 4–6B



Explanation:

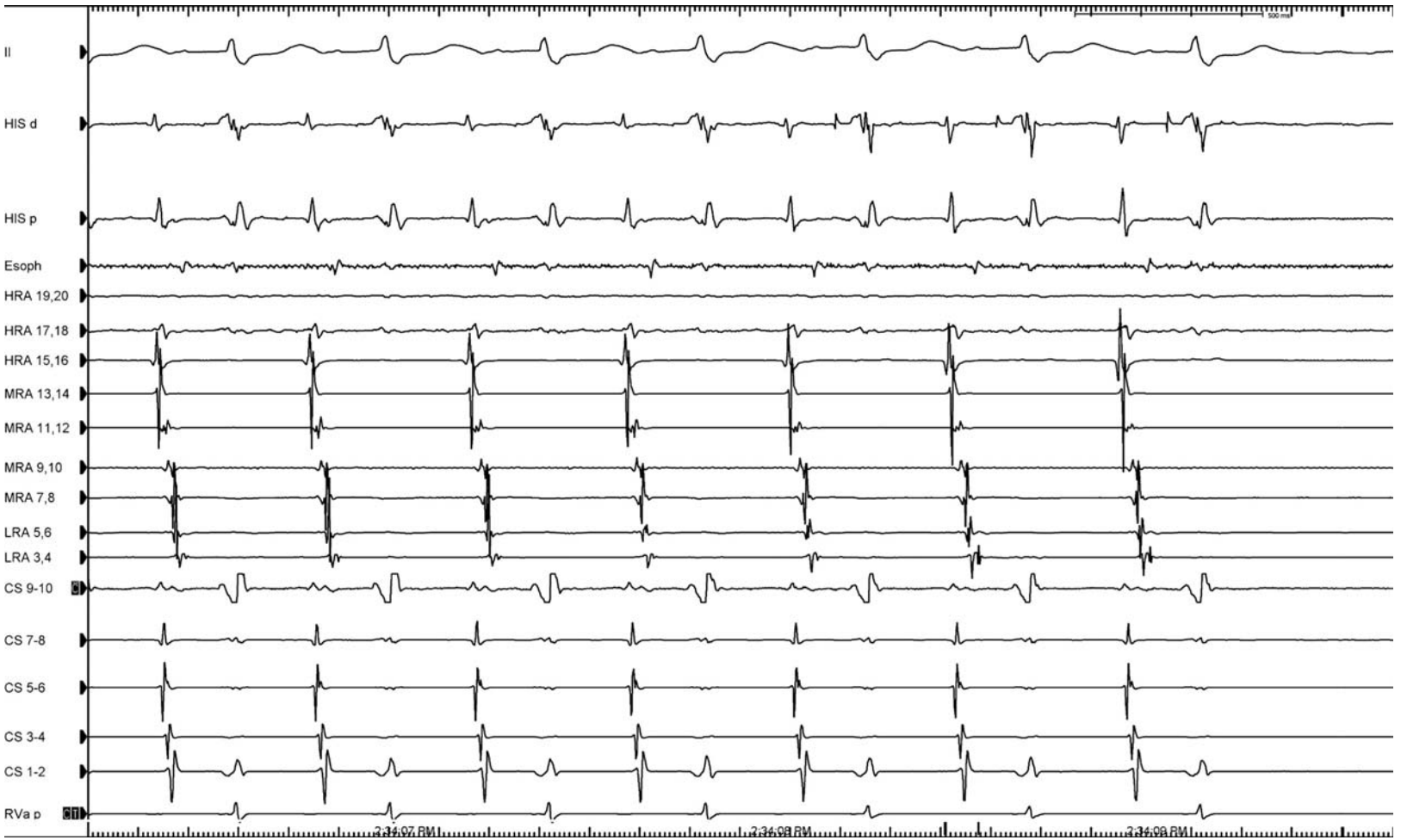
The tracing starts with a narrow QRS tachycardia with a 1:1 AV relationship. The atrial activation is "eccentric," that is, earliest in the distal coronary sinus. This can only be AVRT over a left lateral AP or an atrial tachycardia from the left atrium. The VA relationship stays constant in the few cycles seen, consistent with AVRT but not ruling out an AT, and AVRT was proved elsewhere in the study. A sudden change in atrial activation occurs after the asterisk, with prolongation of the AA interval and a total change in the atrial activation sequence to a central pattern. The subsequent QRS is also reset (CL 380 milliseconds from 370 milliseconds).

The differential diagnosis includes AVRT using a septal AP for retrograde conduction with a longer VA interval, AVNRT, and even AT. AVNRT was subsequently diagnosed. The pacing spikes are distracting but do not capture until the seventh cycle and even then the pacing is too slow to overtake the tachycardia.

It is interesting to speculate how this transition occurred. Why did the retrograde AV node pathway suddenly appear when the AP blocked, to start its own tachycardia? It is possible that the each QRS was resulting in both retrograde AP and retrograde AVN conduction but the AP was always "beating" the AVN to the atrium and the latter was not manifest. With block in the AP, the retrograde AVN pathway could now capture the atrium and drive its own tachycardia. One may think of this as the faster AVRT "entraining" the slightly slower potential AVNRT until block occurred in the AP, that is, analogous to termination of "overdrive pacing" from the atrial insertion of the left lateral AP.

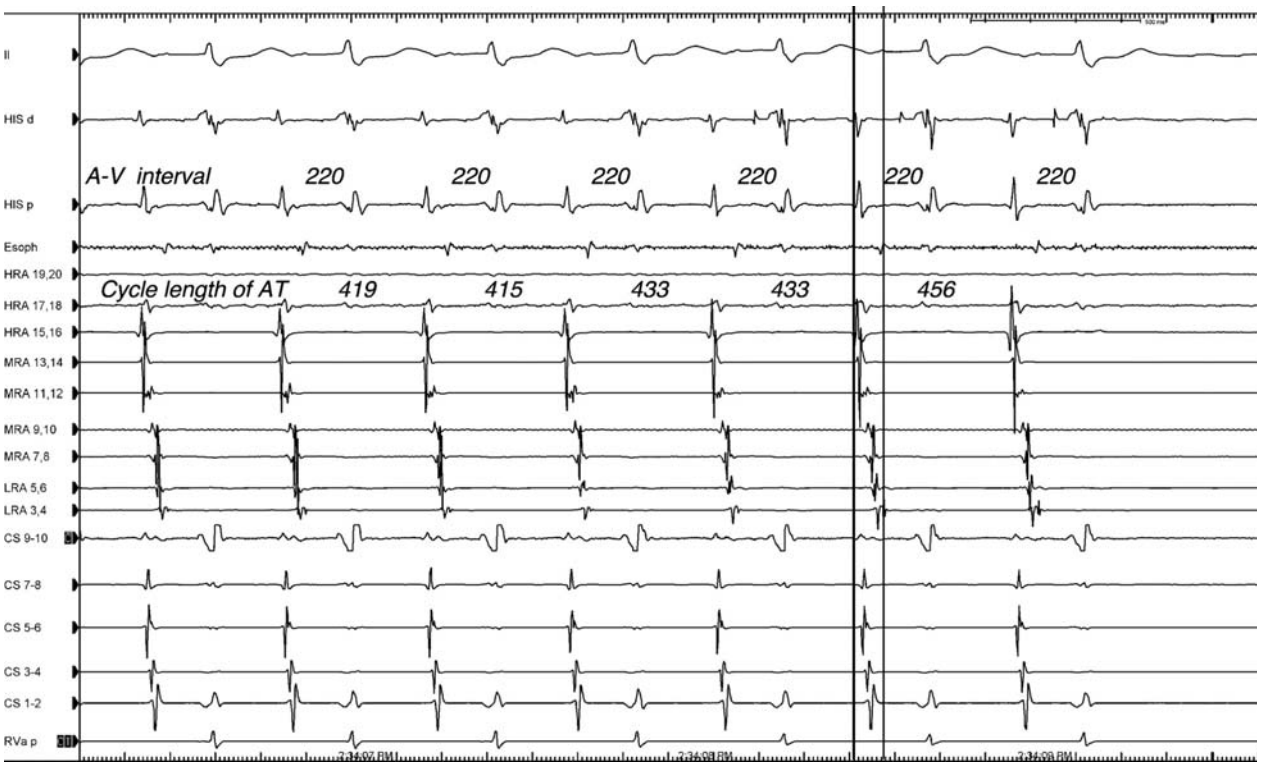
It is also possible that onset of AVNRT was merely coincidental with the cessation of AVRT, although it is somewhat difficult to explain why there is immediate retrograde conduction over the AV node with the next cycle.

Figure 4–7A



SVT is induced during positioning of a multipolar catheter along the crista terminalis in the right atrium. Adenosine is given and termination is observed. What can be concluded about the mechanism of tachycardia?

Figure 4–7B



Explanation:

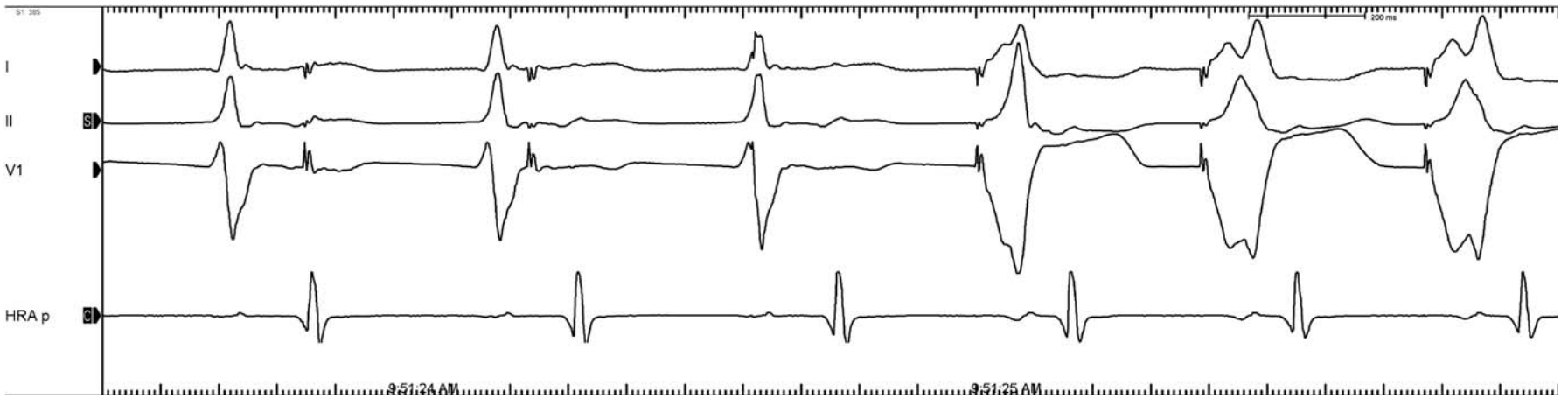
Earliest atrial activation is observed in the right atrium near the HB region. The coronary sinus is activated from proximal to distal.

The multipolar right atrial catheter and the coronary sinus cover a relatively large part of the atria and atrial electrograms cover a rather narrow band of the cardiac cycle (vertical lines). This is most compatible with a focal source rather than macroreentry during which the electrograms would fill more of the cardiac cycle.

The tachycardia terminates with a QRS complex. The last several complexes have a His recorded on the HIS d tracing and there is no preceding AH prolongation, which would usually be expected with termination of a junctional-dependent tachycardia, and slowing of the atrial cycle length precedes termination. Prolongation of conduction in a slowly conducting AV nodal or accessory retrograde pathway cannot be entirely excluded from this tracing alone.

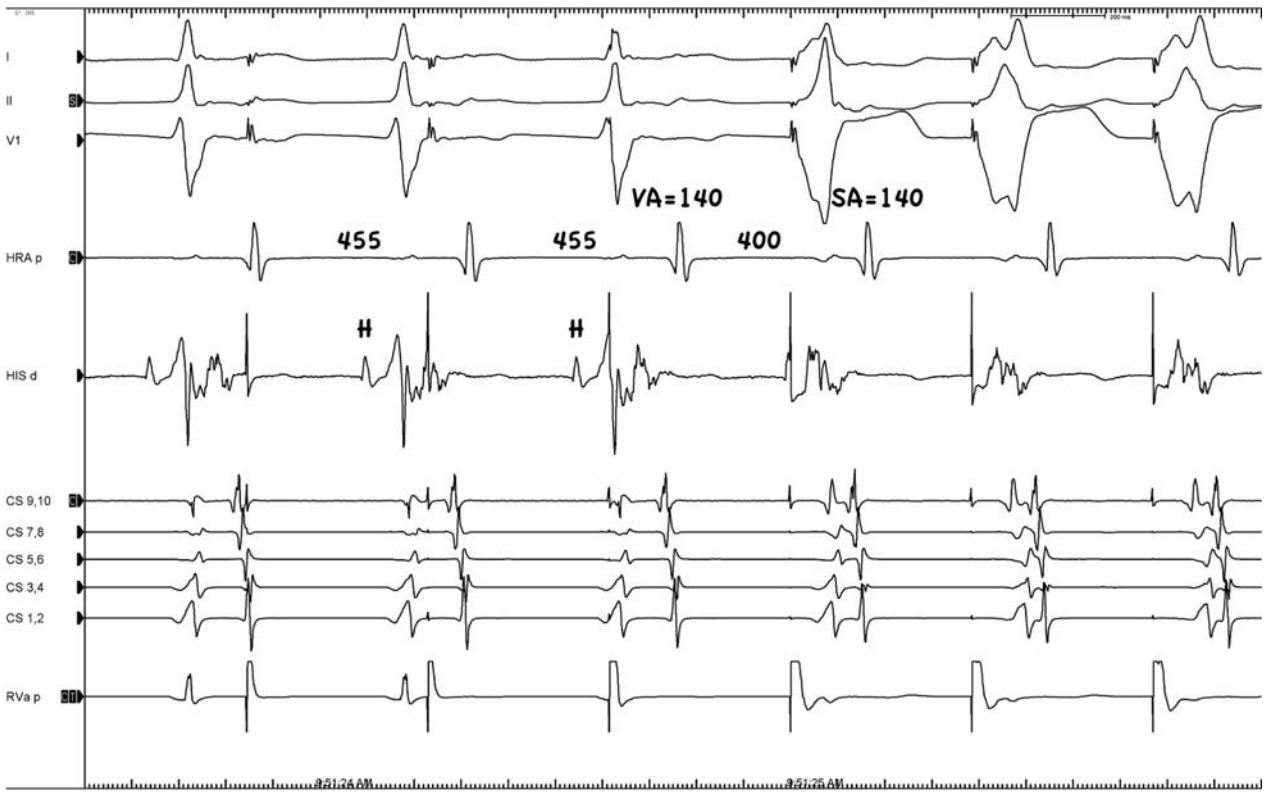
This tracing is most compatible with an adenosine-sensitive atrial focus near the His bundle region and this is where it was ultimately mapped and ablated.

Figure 4–8A



This tachycardia can be described as narrow QRS with a one to one AV relationship. Overdrive ventricular pacing is begun. Can the mechanism of tachycardia be determined from this tracing?

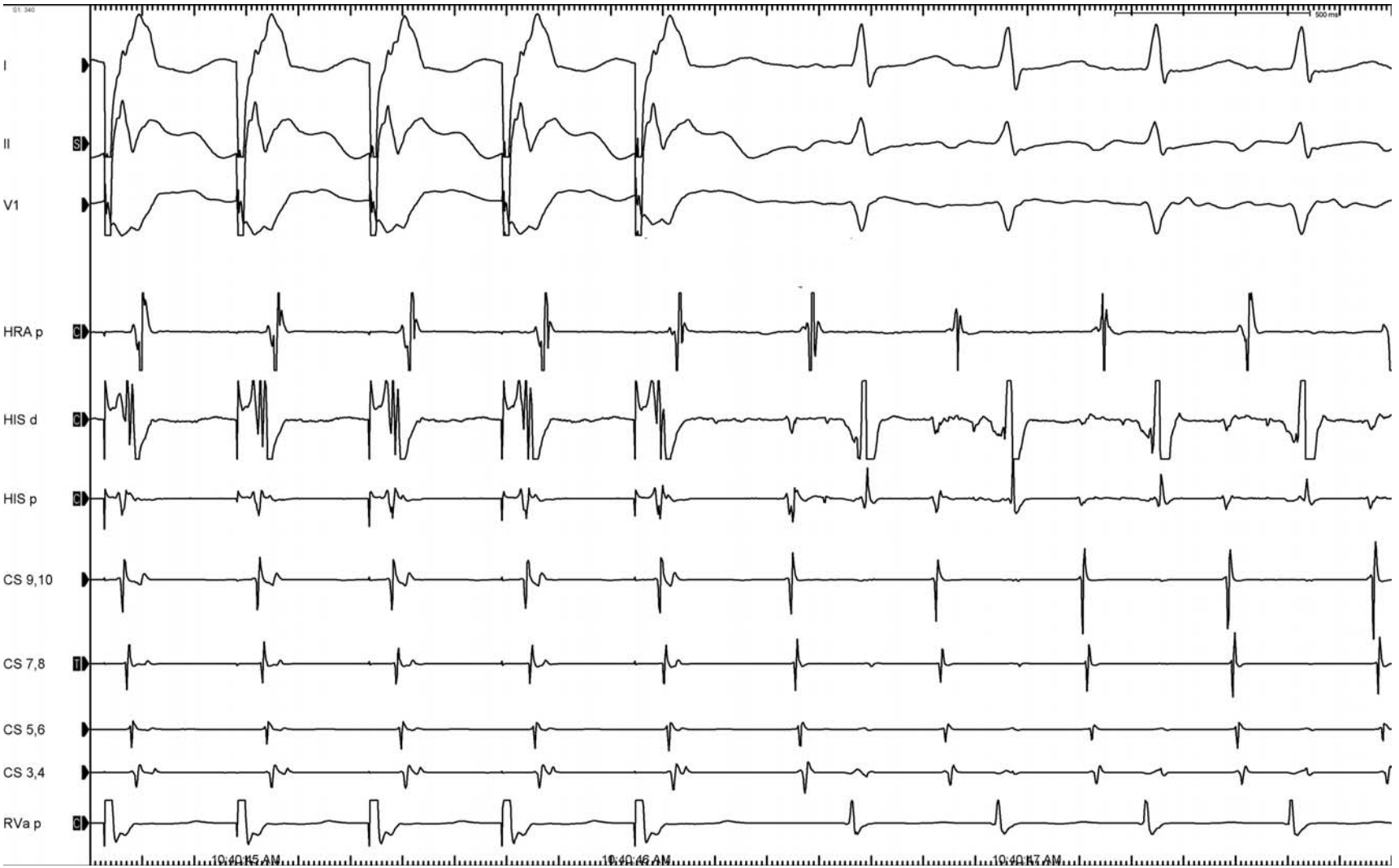
Figure 4–8B



Explanation:

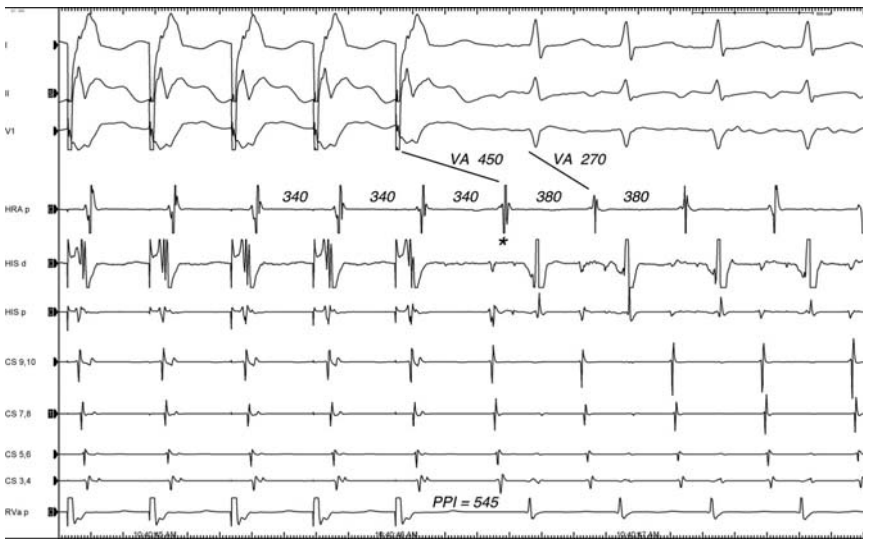
The figure shows onset of ventricular pacing during tachycardia. Ventricular capture is evident only after the fourth spike with the fourth QRS complex. This QRS obviously reflects fusion between the tachycardia QRS and the fully paced QRS. The subsequent atrial activation is advanced. This is the equivalent of the PVC programmed into the tachycardia cycle at a time when the His is refractory, that is, the "His refractory" PVC. It is not necessary to see the His deflection since the fused complex clearly derives in part from anterograde conduction over the His, which then must be refractory. Note also that the stimulus to A interval of the fused beat is almost the same as the VA interval of tachycardia, proving that the pacing stimulus is activating the atrium by the same route as during tachycardia, that is, the pacing catheter is "in" the circuit. This is analogous to comparing these two intervals after cessation of pacing with entrainment. Refer to Fig. 1–3 and consider how all this reflects good access of the pacing site to a sizable excitable gap.

Figure 4–9A



Overdrive ventricular pacing during supraventricular tachycardia. How does one interpret this result?

Figure 4–9B



Explanation:

This is a classical "entrainment" maneuver. The atrial activation sequence during tachycardia is central and might be an atrial tachycardia, AV node reentry, or AV reentry over a septal accessory pathway. At first glance, termination of pacing suggests a "V A A V" response diagnostic of an atrial tachycardia but this is not the case.

The maneuver is best interpreted with a checklist approach. Pacing should be done as close as possible to the tachycardia cycle length to minimize potential decremental conduction, which could confound the interpretation.

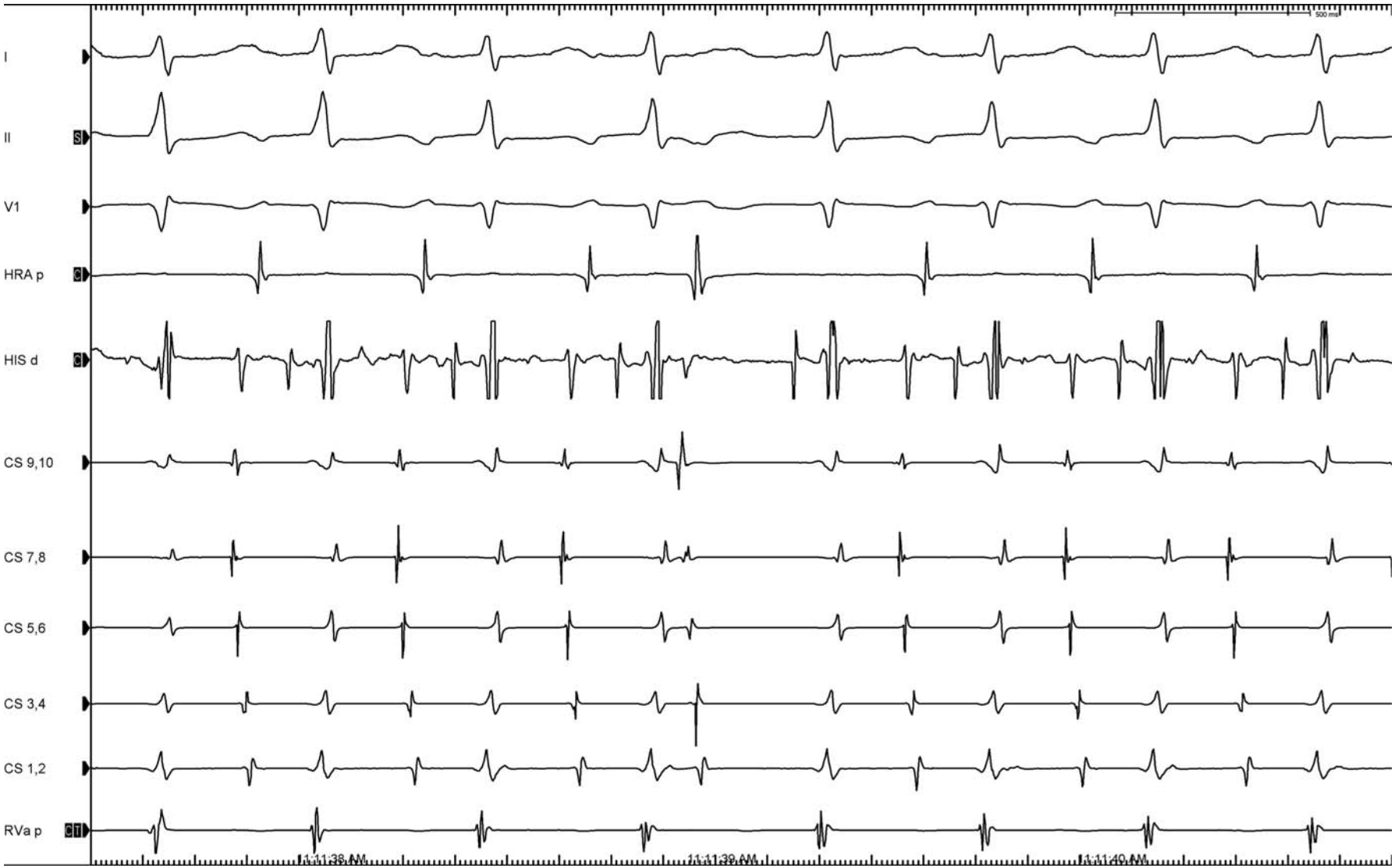
The first priority is to verify that the tachycardia has been accelerated to the pacing cycle length in a stable fashion. This is indeed the case here, that is, 340 milliseconds.

The second step is to identify the last entrained atrial electrogram. In this case it is indicated by the asterisk and it is the last atrial deflection at the paced CL. It is now apparent that the V during pacing does not conduct to the first but to the second atrial electrogram. This is then a "V A V" response. Another clue to relating the V to the correct A is that the VA during pacing should not be shorter than the VA during tachycardia and this alone would eliminate the shorter VA during pacing as the correct one.

At this point, one notes that the postpacing interval (PPI) at the RV apex is 165 milliseconds longer than the tachycardia cycle length and the change in VA between pacing and tachycardia is 180 milliseconds (i.e., 450–270). The usual published maximum PPI to be considered "in" the circuit for AV reentry over a septal AP is 115 milliseconds and the corresponding delta VA is 85 milliseconds; so one can consider that the pacing site is "out" of the circuit.

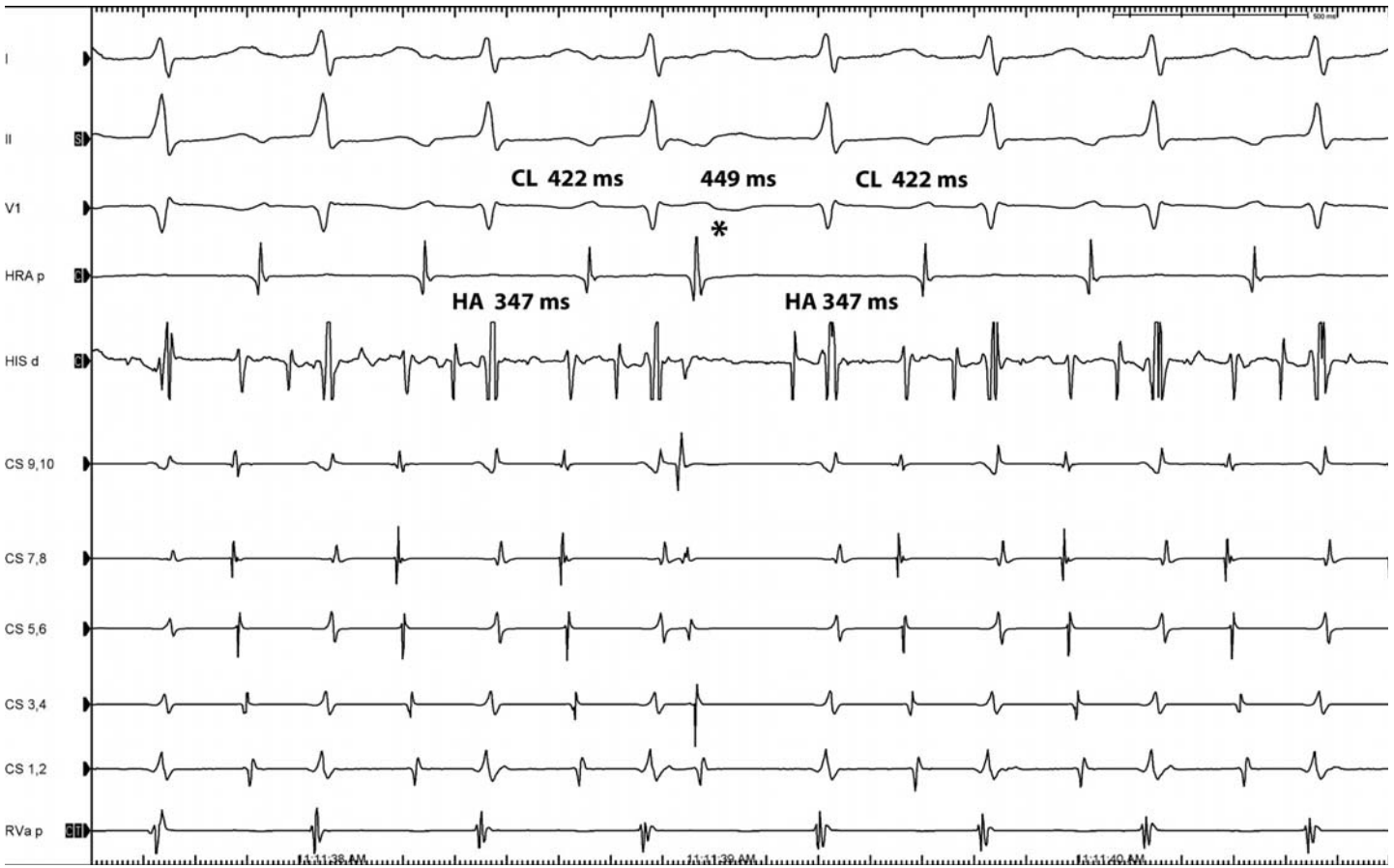
This is often loosely expressed as an "AV nodal" response but this can be misleading since other factors (free wall AP, decremental septal AP) can result in this type of response. It is more accurate to say that one has excluded a "conventional" or nondecremental septal accessory pathway as the retrograde limb of the circuit. In this case it was AVNRT.

Figure 4–10A



What can be said of the mechanism of this tachycardia?

Figure 4–10B

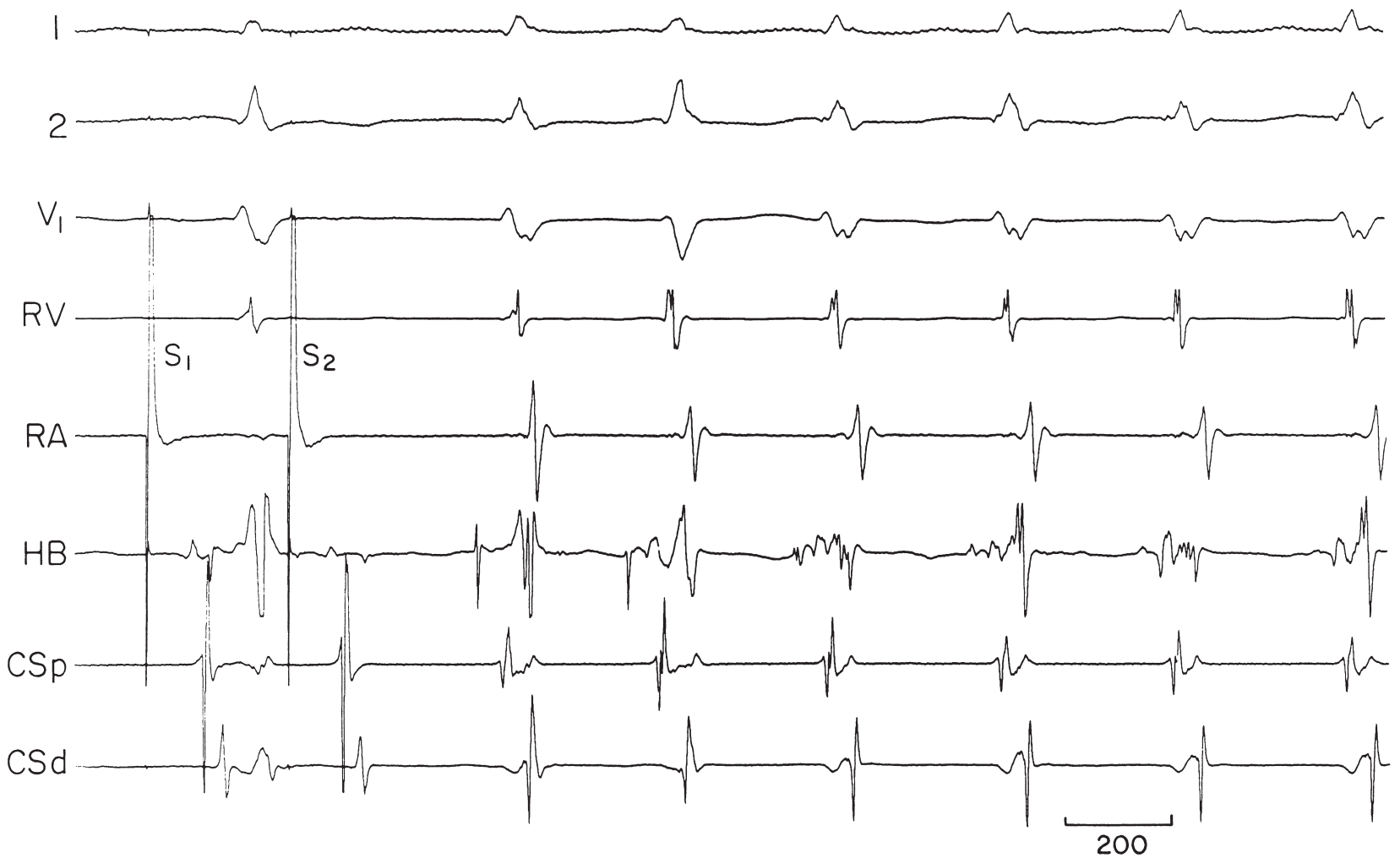


Explanation:

The tachycardia is regular with a one to one AV relationship. The atrial activation shows earliest depolarization at CS 7,8, which is slightly in from the orifice of the CS. This is not an anterior septal pattern and is most likely either atrial tachycardia or atrioventricular reentry. AVNRT is technically possible with this pattern but would be more unusual.

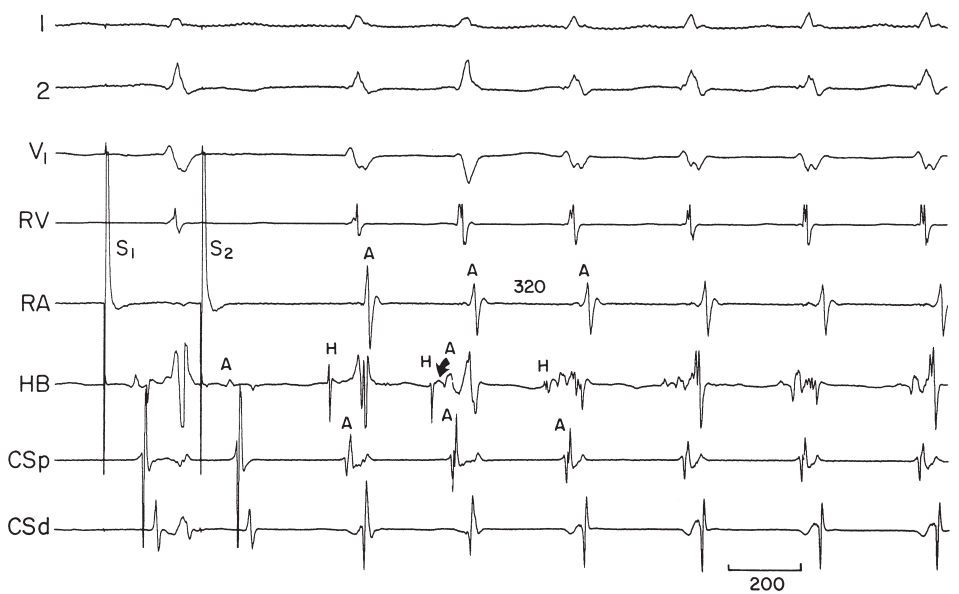
A PAC with a different activation pattern is seen (asterisk) and delays the next cycle. This delayed cycle nonetheless has the same HA interval as the tachycardia (the A appears "linked" to the previous H) and this would be extremely fortuitous with an atrial tachycardia but expected with AVRT, which it was.

Figure 4–11A



The patient is a young woman with paroxysmal tachycardia. Tachycardia is initiated by a critically timed atrial extrastimulus. What is the differential diagnosis and probable mechanism of the tachycardia?

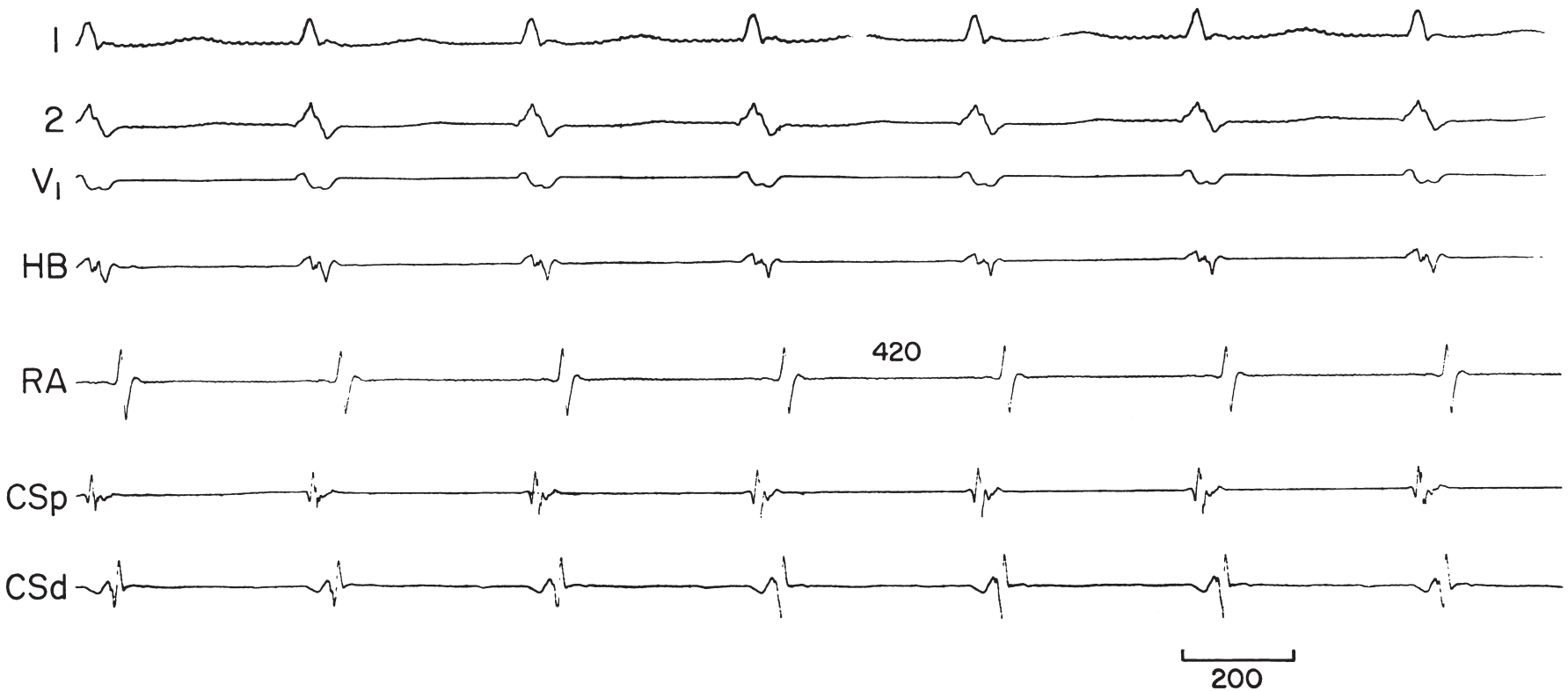
Figure 4–11B



Explanation:

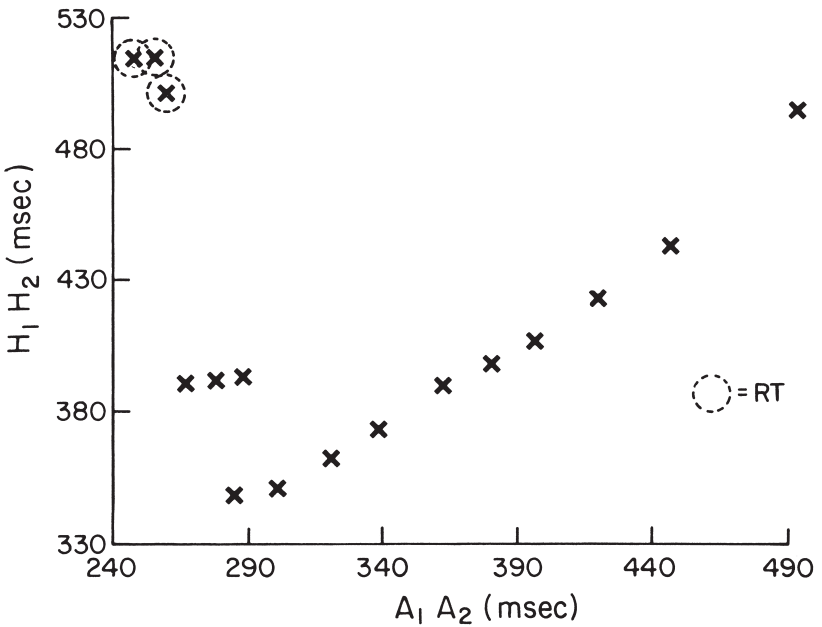
The differential diagnosis of a narrow QRS tachycardia is presented in Table 1–5. The tachycardia has a normal QRS, a cycle length of 320 milliseconds, and a 1:1 AV relationship. The atrial activation is central with earliest activation at the His (*arrow*) where atrial activation precedes ventricular activation. This excludes sinus node reentry. The short VA interval rules out AV reentry. Although atrial tachycardia is not excluded, the apparent requirement of AH prolongation at the onset of tachycardia makes AV node reentry most likely. Maneuvers to assess AV node participation in tachycardia such as carotid sinus massage will confirm the diagnosis.

Figure 4–12A



Same patient as in Fig. 4–11. There has been a sudden increase in the tachycardia cycle length to 420 milliseconds (transition not recorded, His catheter out of position). What is the mechanism?

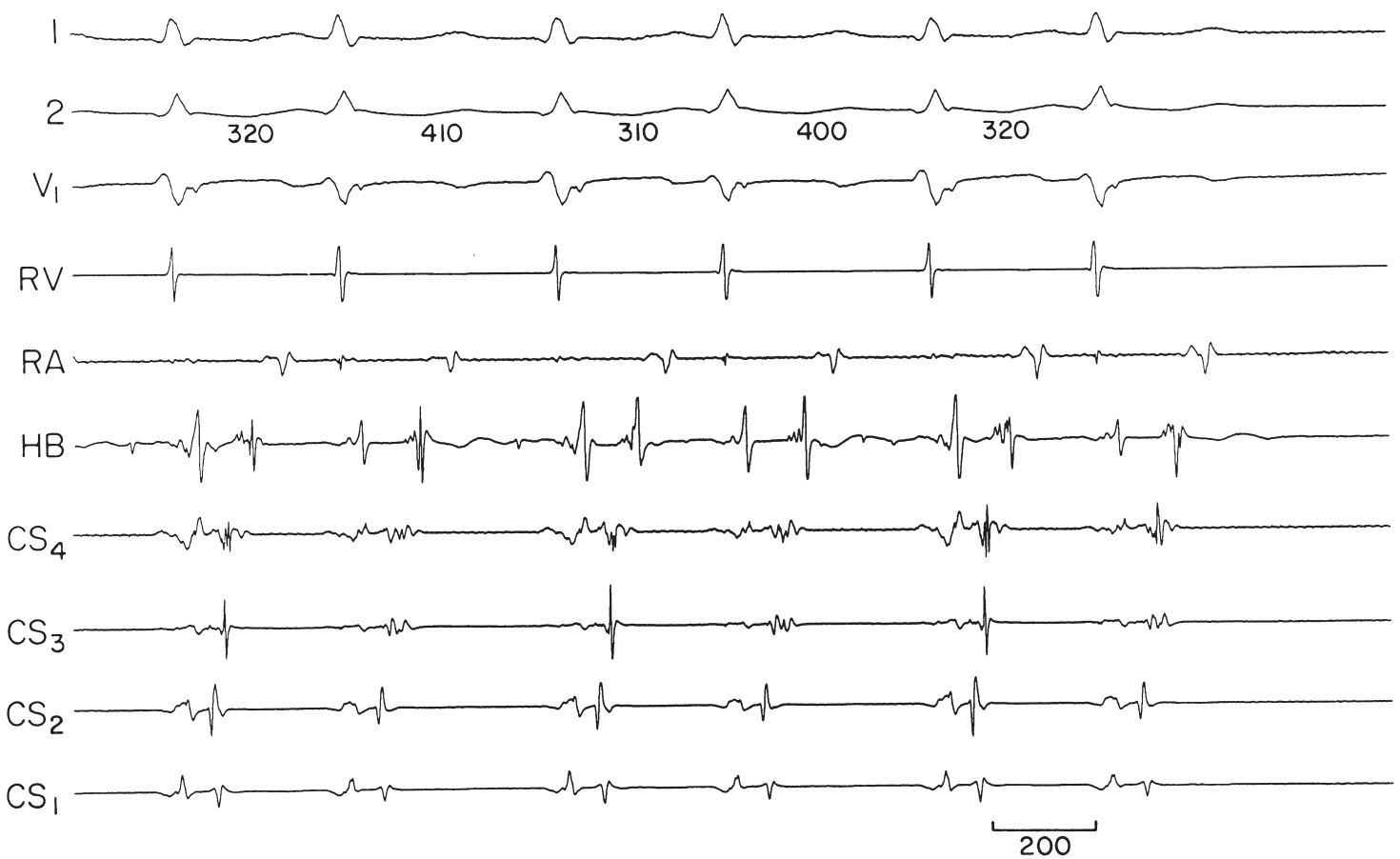
Figure 4–12B



Explanation:

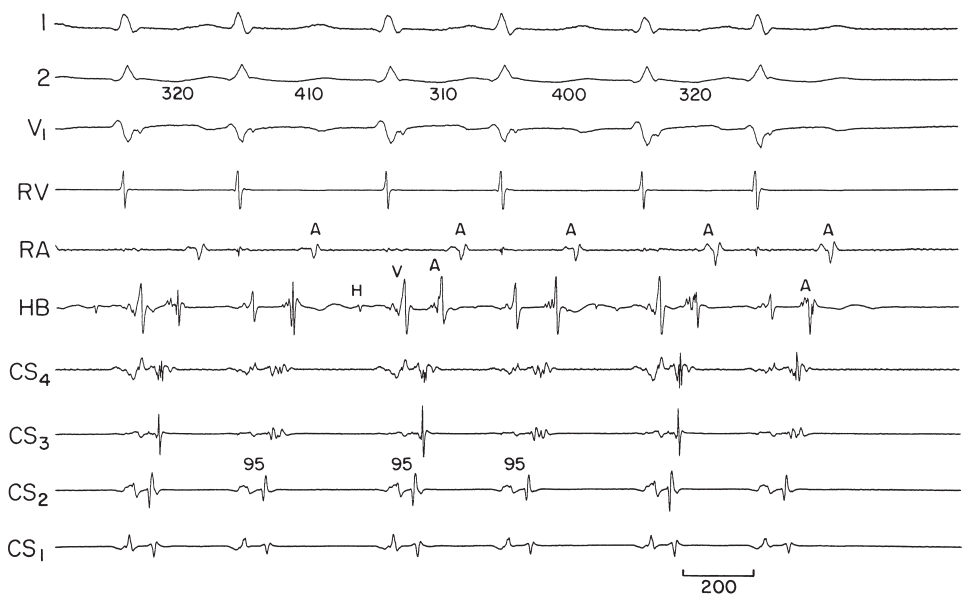
The new tachycardia appears identical to the original with the exception of the cycle length, which is longer entirely due to presumed prolongation of the AH interval. The His catheter is out of position but the absence of bundle branch block makes HV delay very unlikely. The most probable mechanism is AV node reentry using a second "slower" slow AV node pathway. The presence of two tachycardia rates could have been predicted by examination of the curve relating AV node conduction time (H_1H_2) to prematurity of an atrial extrastimulus (A_1A_2). This shows a double discontinuity, suggesting the existence of two slow pathways. RT signifies onset of tachycardia.

Figure 4–13A



The record is from a young man with paroxysmal tachycardia. Tachycardia was never recorded because it always stopped prior to his arrival in the emergency department. The 12-lead ECG was normal. Induction of tachycardia was by atrial extrastimuli and required critical AH prolongation. Why did his tachycardia always stop spontaneously? CS_4 to CS_1 are coronary sinus electrograms from proximal (4) to distal (1), respectively. CS_4 is positioned near the orifice of the coronary sinus.

Figure 4–13B

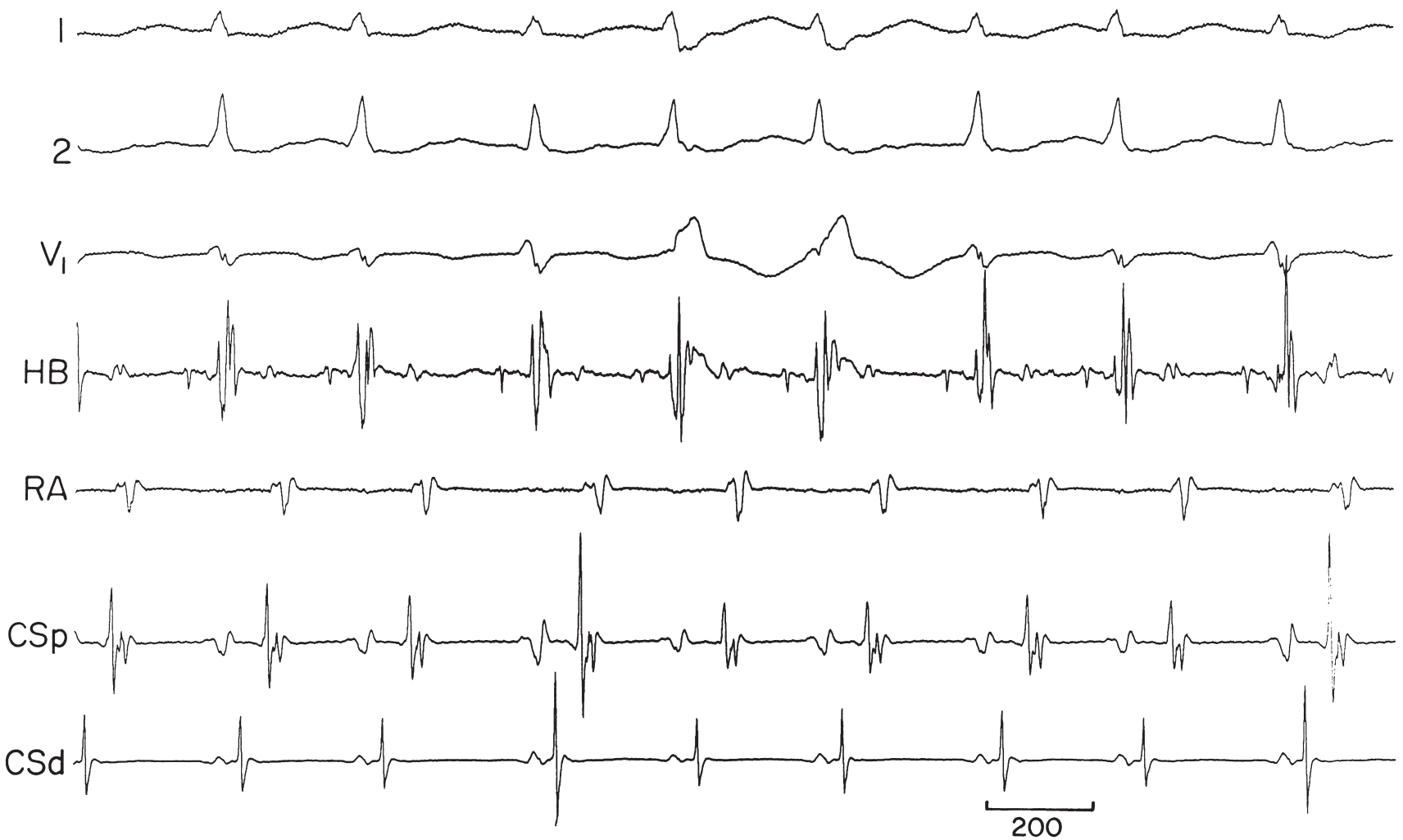


Explanation:

The tachycardia is irregular and the cycle length alternates from approximately 300 to 400 milliseconds due entirely to change in the AH interval. This suggests anterograde conduction over dual AV node pathways and dual pathway physiology was indeed observed during atrial extrastimulus testing. The atrial activation is eccentric (distal CS first), suggesting left atrial tachycardia or AV reentry over a left lateral AP. AV reentry was verified during the study but could have been deduced by two observations. Spontaneous termination occurred with an A, an unlikely event with atrial tachycardia because coincidental block in the AV node at the same time would have to be postulated. During oscillation of the cycle length, the change in AH precedes and predicts subsequent AA intervals, strongly implicating AV node participation. The oscillation in AV node conduction time facilitated spontaneous termination as fast pathway conduction impinged on the refractory period of the slow pathway.

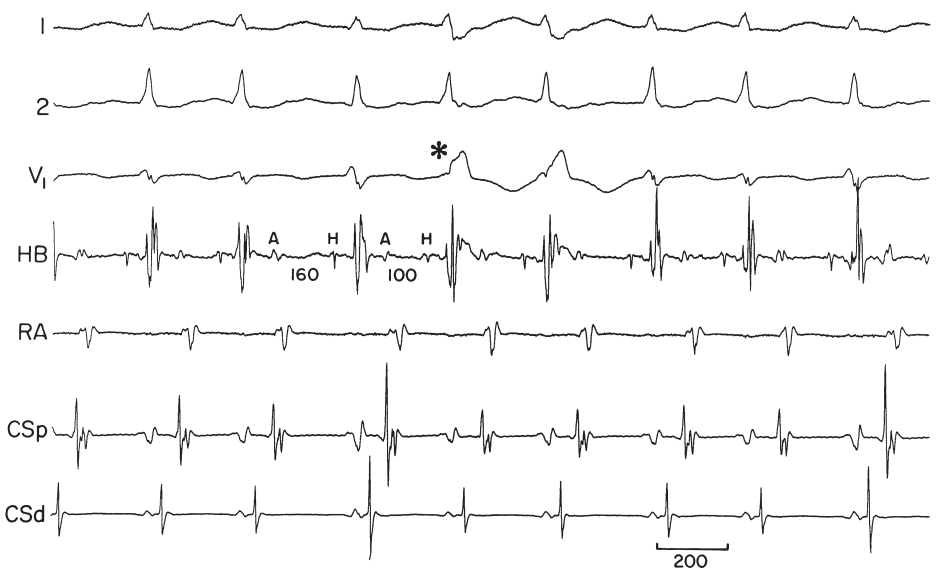
Did this patient also have "typical" AV node reentry? It would not be expected (and was not observed) since slow pathway conduction during tachycardia did not result in retrograde fast AV node pathway conduction that would have preempted retrograde AP conduction.

Figure 4–14A



The record is from a young man otherwise well except for paroxysmal tachycardia. The surface ECG was normal. Tachycardia was induced with critically timed atrial extrastimuli. What is the mechanism, and why is there a change in the QRS morphology?

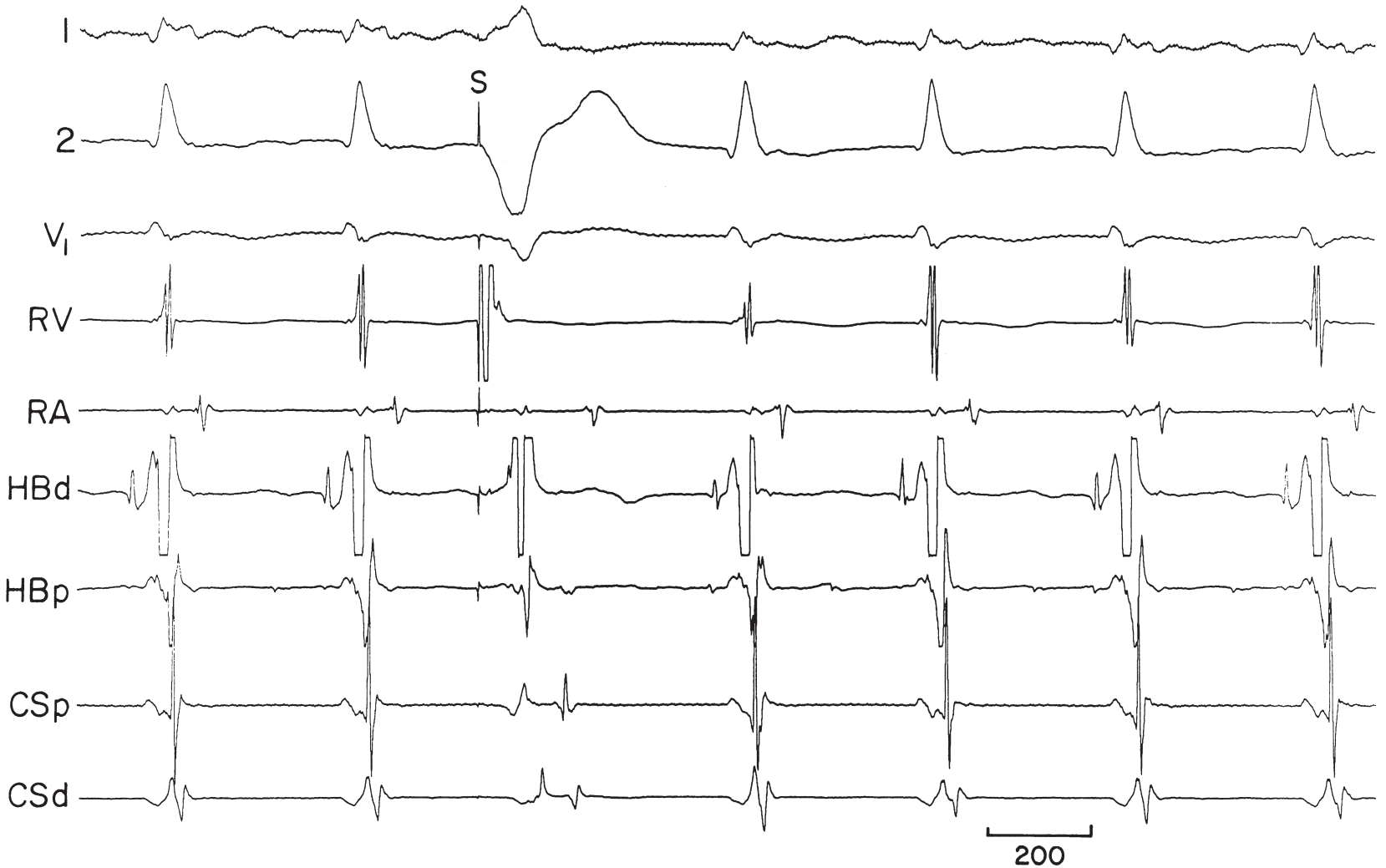
Figure 4–14B



Explanation:

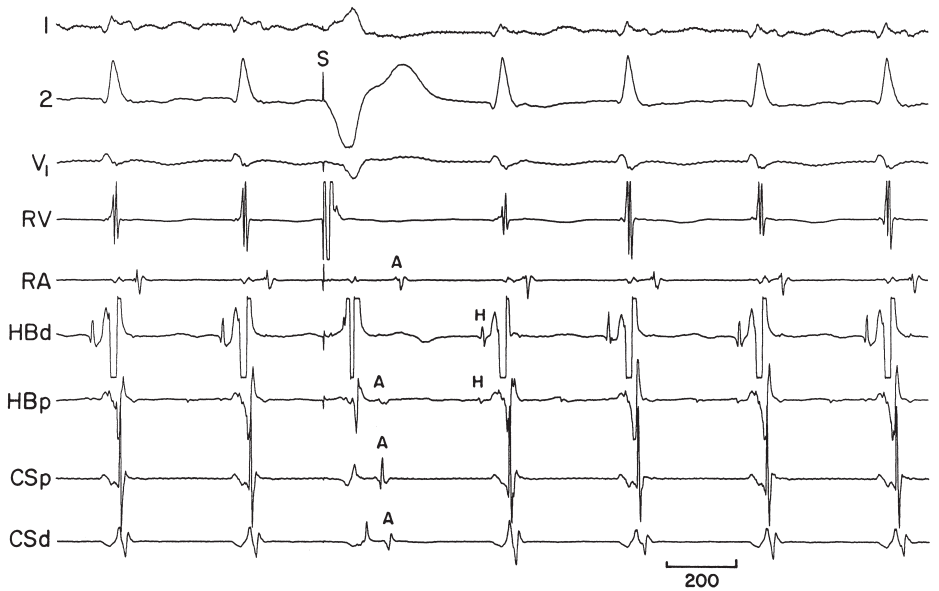
The tachycardia is irregular and this is related to two populations of AH intervals, approximately 100 and 160 milliseconds. The atrial activation sequence is eccentric and the earliest A is recorded in the distal coronary sinus (CS_d). The differential diagnosis includes atrial tachycardia or AV reentry utilizing a left lateral AP. Atrial tachycardia is ruled out by the delay in atrial timing after a long AH, a fact suggesting that atrial activation is dependent on preceding AV conduction time (the VA interval is constant despite rate irregularity). RBBB aberration is observed after a long–short cycle sequence (Ashman phenomenon) related to AH changes.

Figure 4–15A



The PVC (*S*) programmed into the cardiac cycle during this regular tachycardia proves the diagnosis of AV node reentry, does it not?

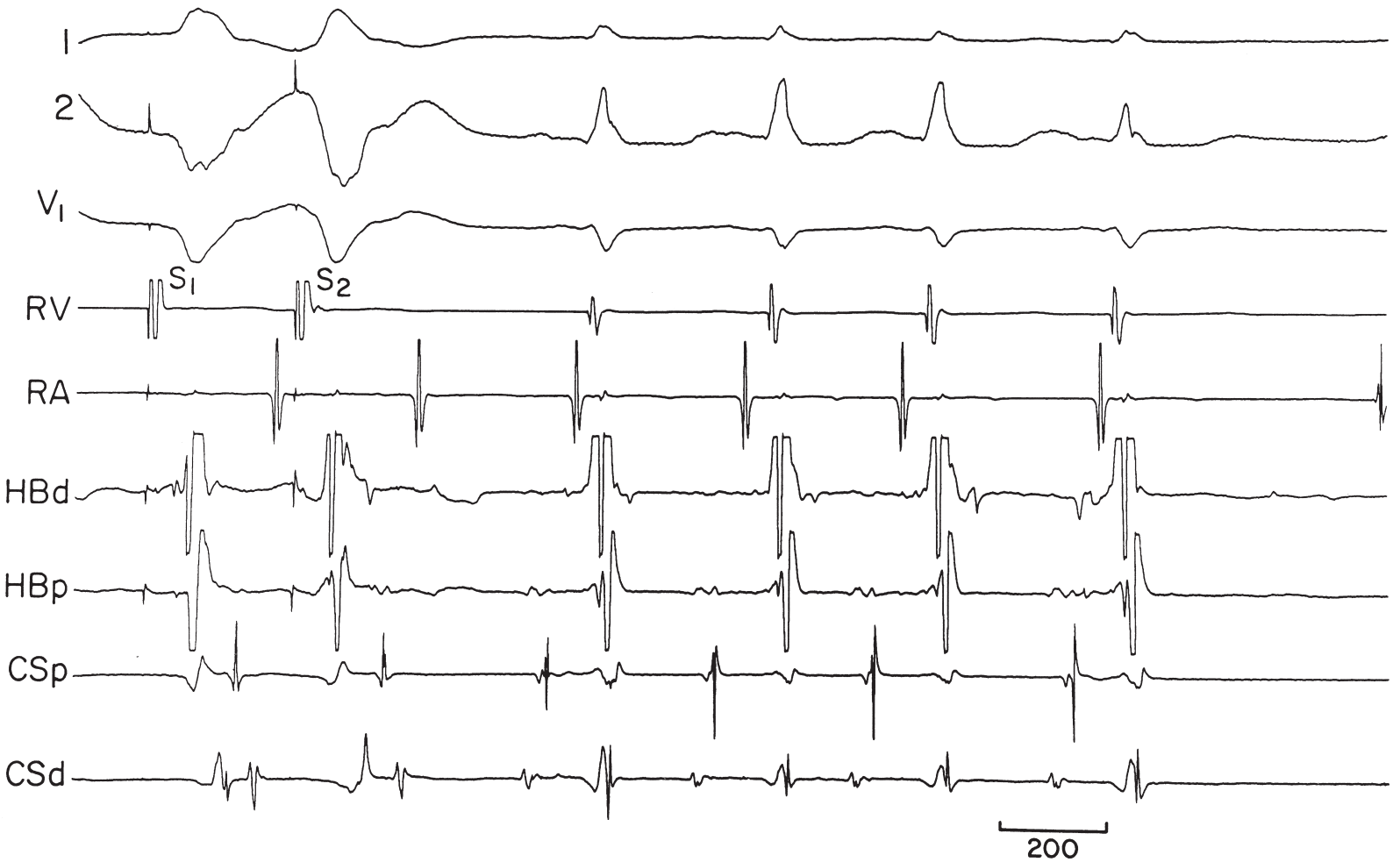
Figure 4–15B



Explanation:

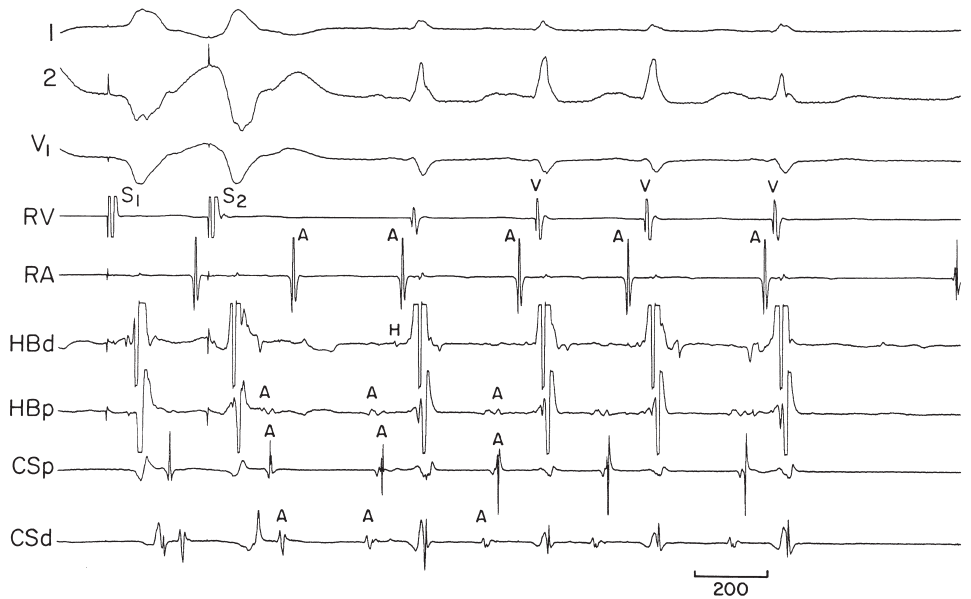
The tachycardia is regular. Both the long AH and the short VA interval support the diagnosis of AV node reentry and the atrial activation sequence is concentric as made clear by the early coupled PVC that advances the V and exposes the atrial electrograms. The PVC does not preexcite the next A and further supports the diagnosis. However, failure to advance the A does *not* rule out atrial tachycardia. Indeed, failure to advance the A does not rule out atrioventricular reentry because the AP may be decremental or far away from a right ventricular extrastimulus (i.e., left lateral). In the present example, AV reentry is ruled out by the coincidental atrial and ventricular activation.

Figure 4–16A



The patient was referred for assessment of supraventricular tachycardia, generally exercise induced. The following tachycardia was consistently induced by ventricular extrastimuli at a critical coupling interval as well as atrial extrastimuli. What is the mechanism of tachycardia?

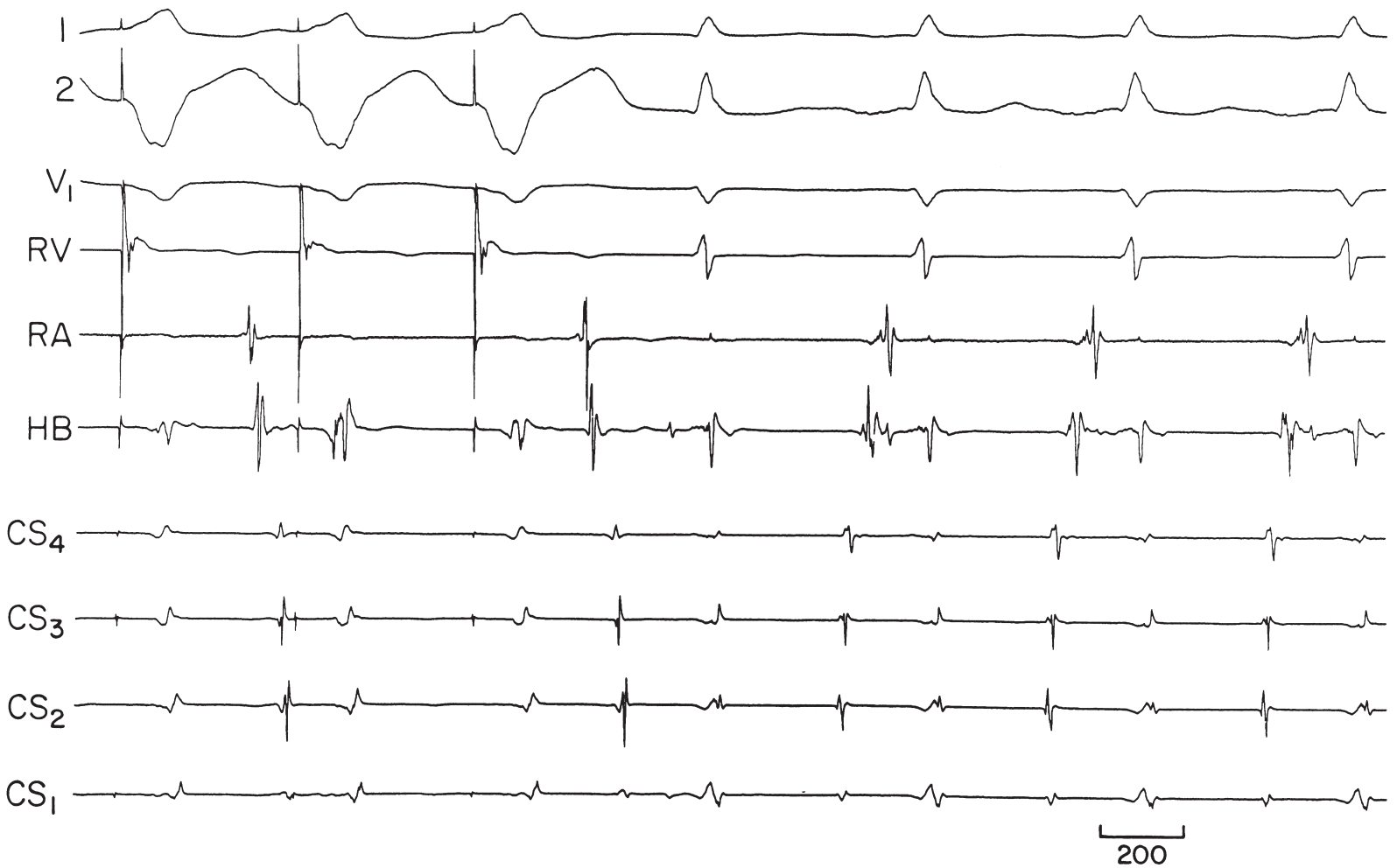
Figure 4–16B



Explanation:

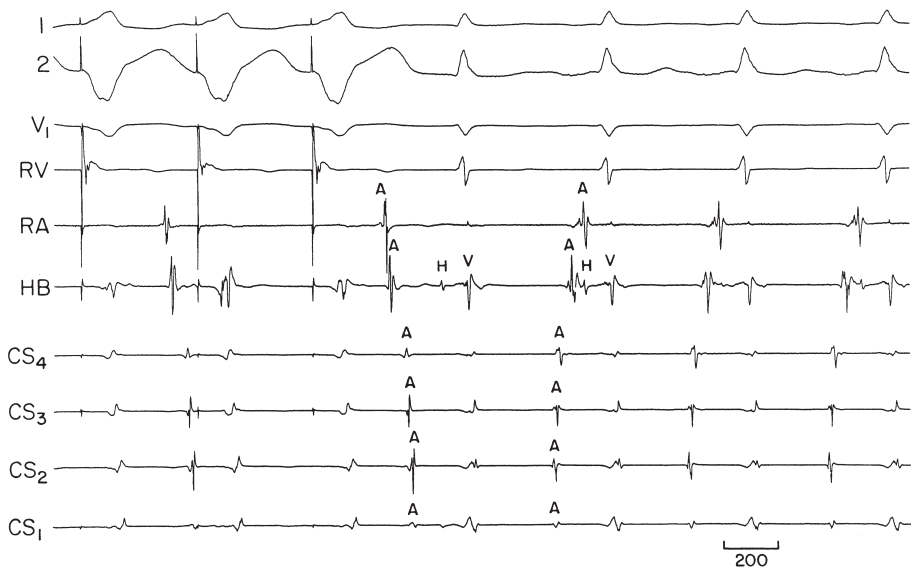
The ventricular extrastimulus conducts with a concentric atrial activation sequence with slight prolongation of the VA interval. This is compatible with conduction over the AV node. The next event is atrial activation with an eccentric atrial activation sequence with earliest atrial activation recorded at the distal coronary sinus electrogram (CS_d). Referring to Table 1–7, it is clear that this must be either an atrial tachycardia or AV reentry. The first spontaneous event in the tachycardia is atrial activation and points to the correct diagnosis of a left atrial tachycardia. A 2:1 phenomenon is remotely possible, that is, ventricular activation resulting from S_2 conducting to the atrium over both the AV node and a slowly conducting left lateral AP. This cannot be entirely ruled out from this record but is unlikely because of the variability of the apparent V to A interval during tachycardia.

Figure 4–17A



This tachycardia was initiated relatively reproducibly by a burst of ventricular pacing, as shown, with no apparent VA conduction during the burst. However, tachycardia only occurred if the first sinus complex after ventricular pacing conducted to the ventricle. What is the mechanism of tachycardia?

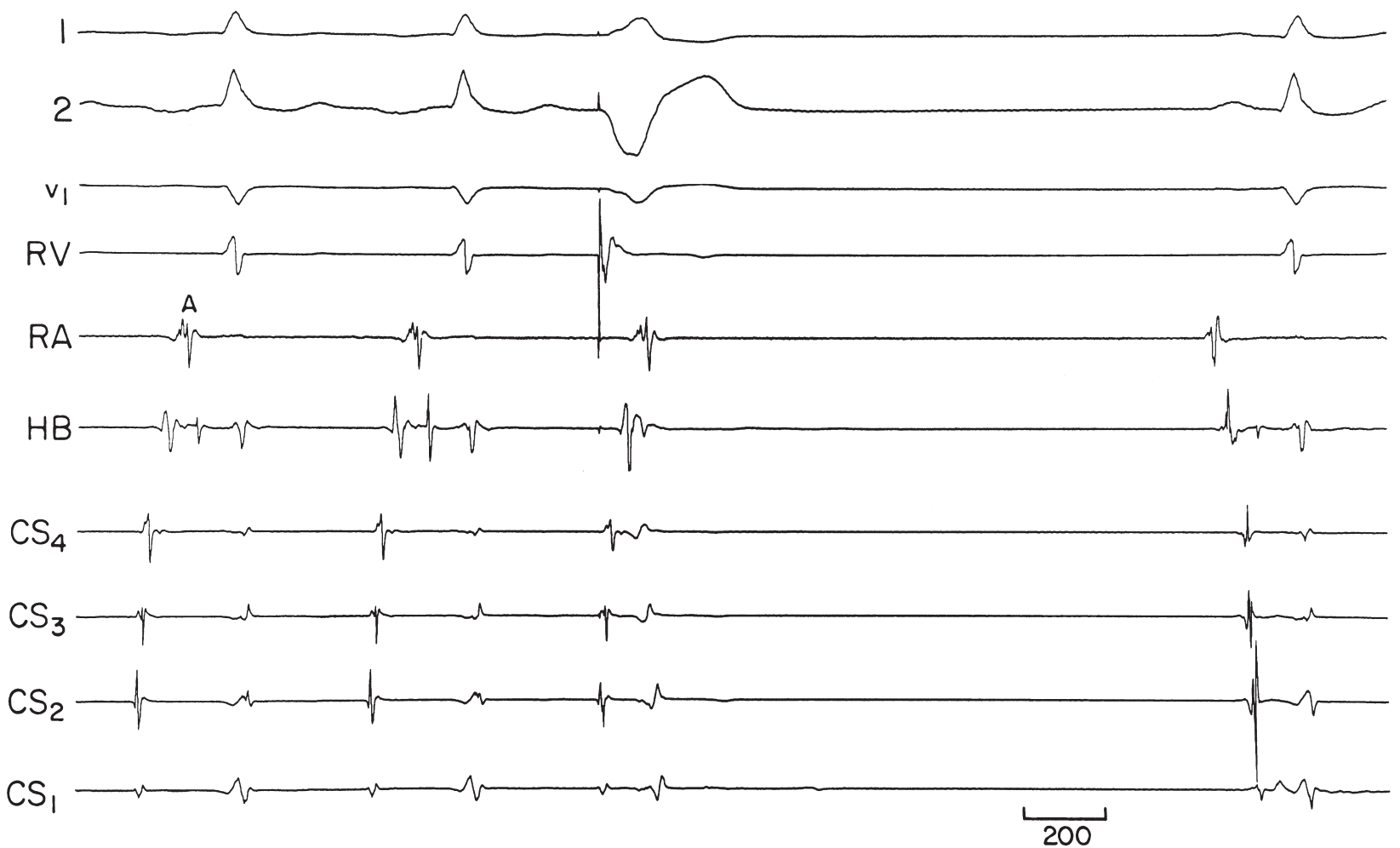
Figure 4–17B



Explanation:

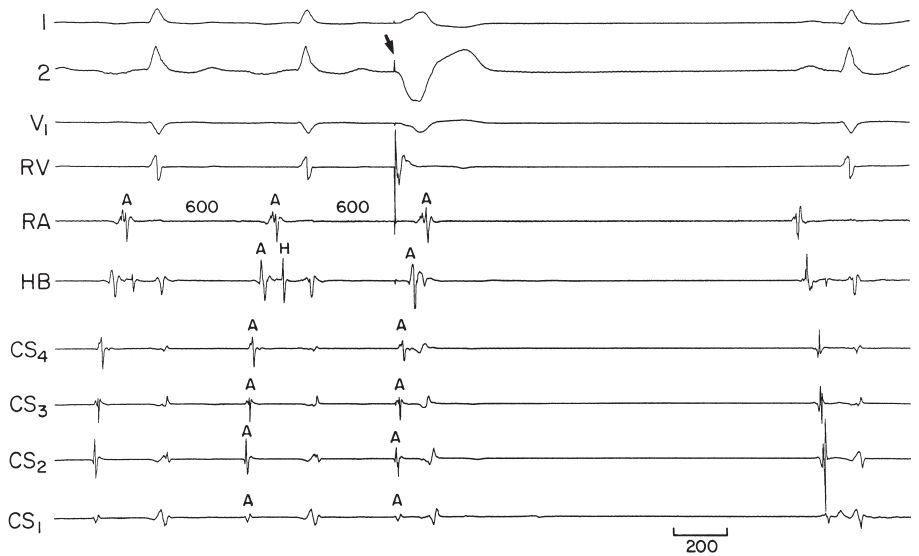
The first atrial cycle after the last ventricular paced cycle has a high to low activation sequence and is sinus. This conducts with a relatively long AH interval, in all likelihood related to concealed retrograde conduction into the AV node by the last paced QRS. The first spontaneous tachycardia event is atrial activation with an eccentric activation sequence earliest at the distal coronary sinus (CS_1). The differential diagnosis then becomes (Table 1–7) atrial tachycardia or AV reentry over a left AP with a long conduction time. The latter is favored by the apparent requirement of previous AH prolongation as was observed during repetitive inductions. Tachycardia only occurred if the first sinus cycle after ventricular pacing conducted with AH prolongation. It was also known that eccentric retrograde atrial activation was observed with ventricular pacing at slower rates. However, atrial tachycardia could not be excluded from this record alone.

Figure 4–18A



Data from the patient described in Fig. 4–17. A PVC programmed into the cardiac cycle at a critical coupling interval terminates tachycardia consistently. Does this clarify the mechanism?

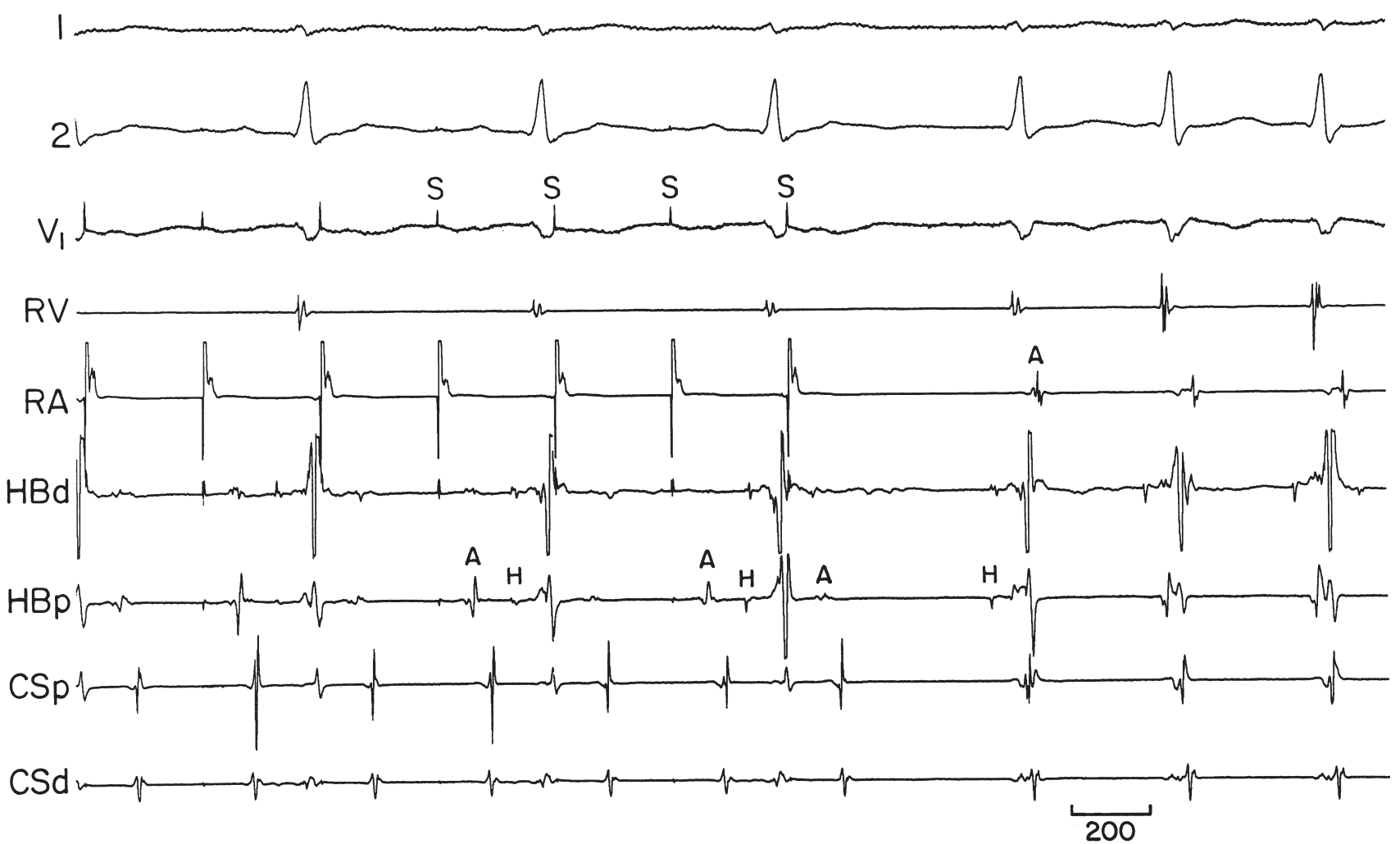
Figure 4–18B



Explanation:

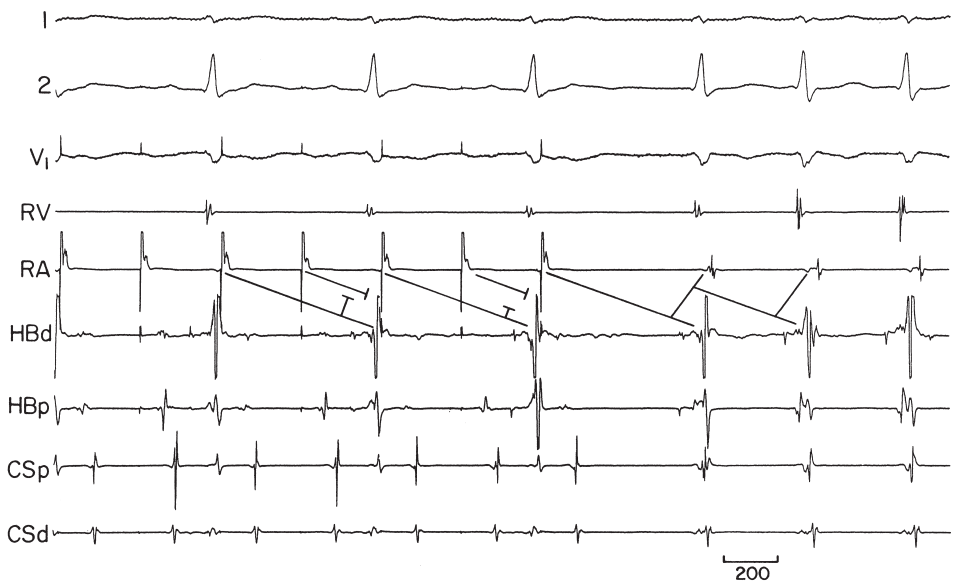
This tachycardia was consistently terminated by a PVC that did not alter atrial activation or timing. This essentially excludes the diagnosis of atrial tachycardia, leaving the diagnosis of AV reentry over a left lateral AP with a long conduction time, as was the case.

Figure 4–19A



This tachycardia onset was recorded after termination of incremental atrial pacing that resulted in 2:1 AV block. What is the tachycardia mechanism and why did tachycardia start after termination of pacing?

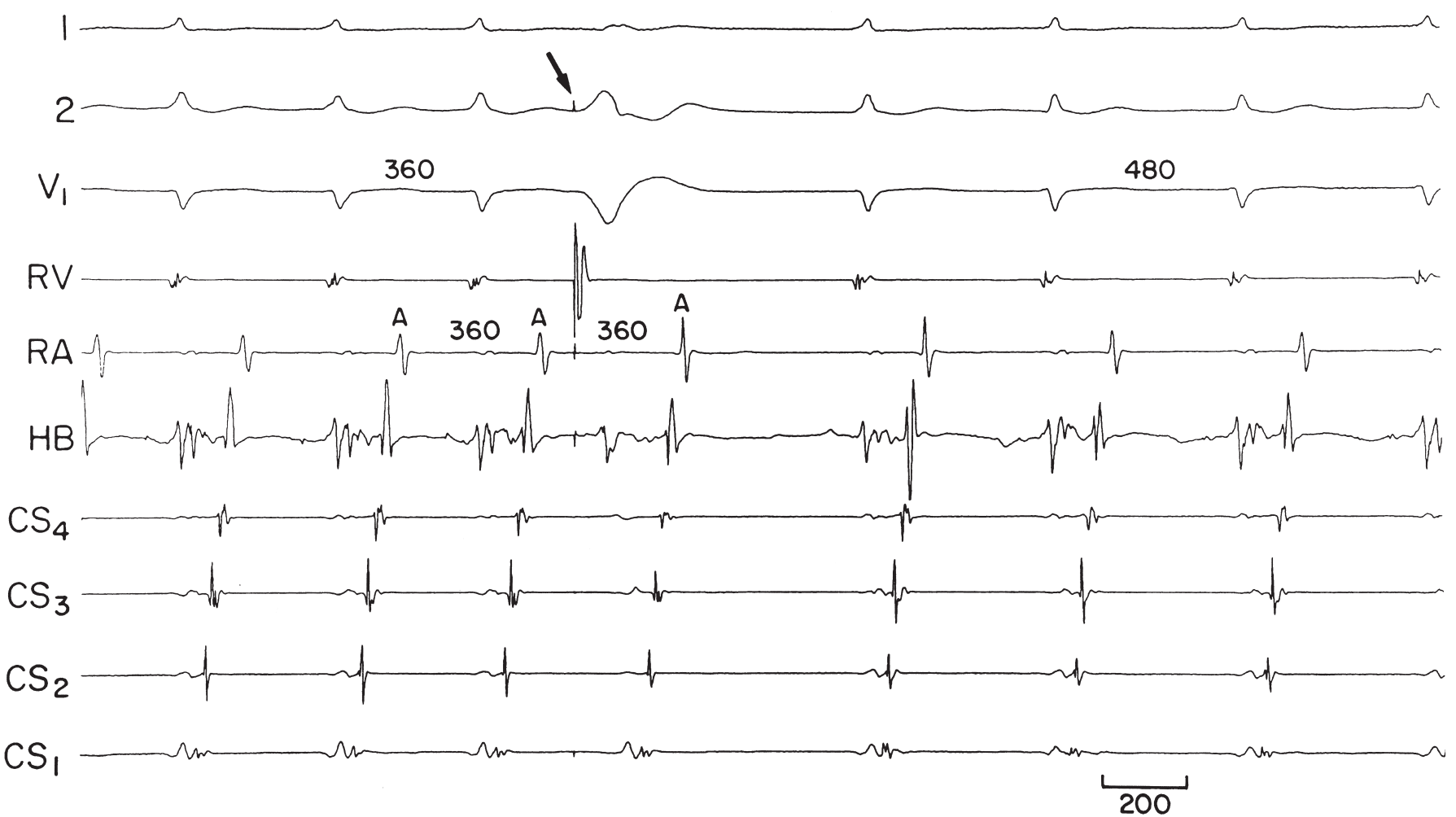
Figure 4–19B



Explanation:

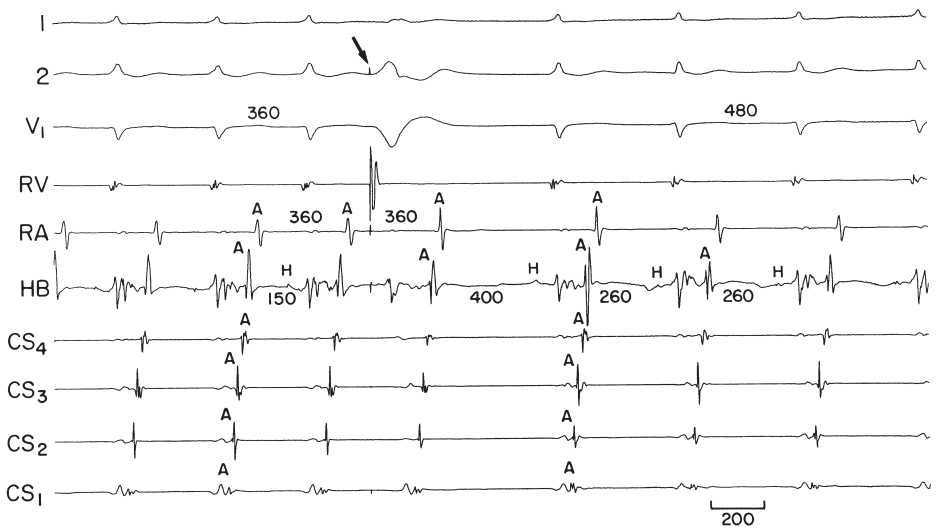
The tachycardia mechanism is AV node reentry, as suggested by the extremely long AH at the onset, the concentric atrial activation sequence, and the simultaneous ventricular and atrial activation. Although atrial tachycardia or a junctional tachycardia (Table 1–6) could not be ruled out from this record, other criteria for AV node reentry were met during the study. During pacing, every second beat was conducted over the slow AV node pathway with manifest reentry aborted by the alternate atrial cycle that failed to conduct to the ventricle. With termination of pacing, slow pathway conduction is manifest and nothing prevents the return atrial cycle from continuing reentry. The excessive PR prolongation after the last paced atrial cycle is probably related to the effects of the previous nonconducted atrial cycle, which penetrated the slow pathway to some degree. The curve relating A_1–A_2 to A_H in this patient demonstrated a single but not a double discontinuity.

Figure 4–20A



A PVC is introduced into this regular tachycardia at a relatively early coupling interval. It does not preexcite the atrium but changes the tachycardia. How does one explain this?

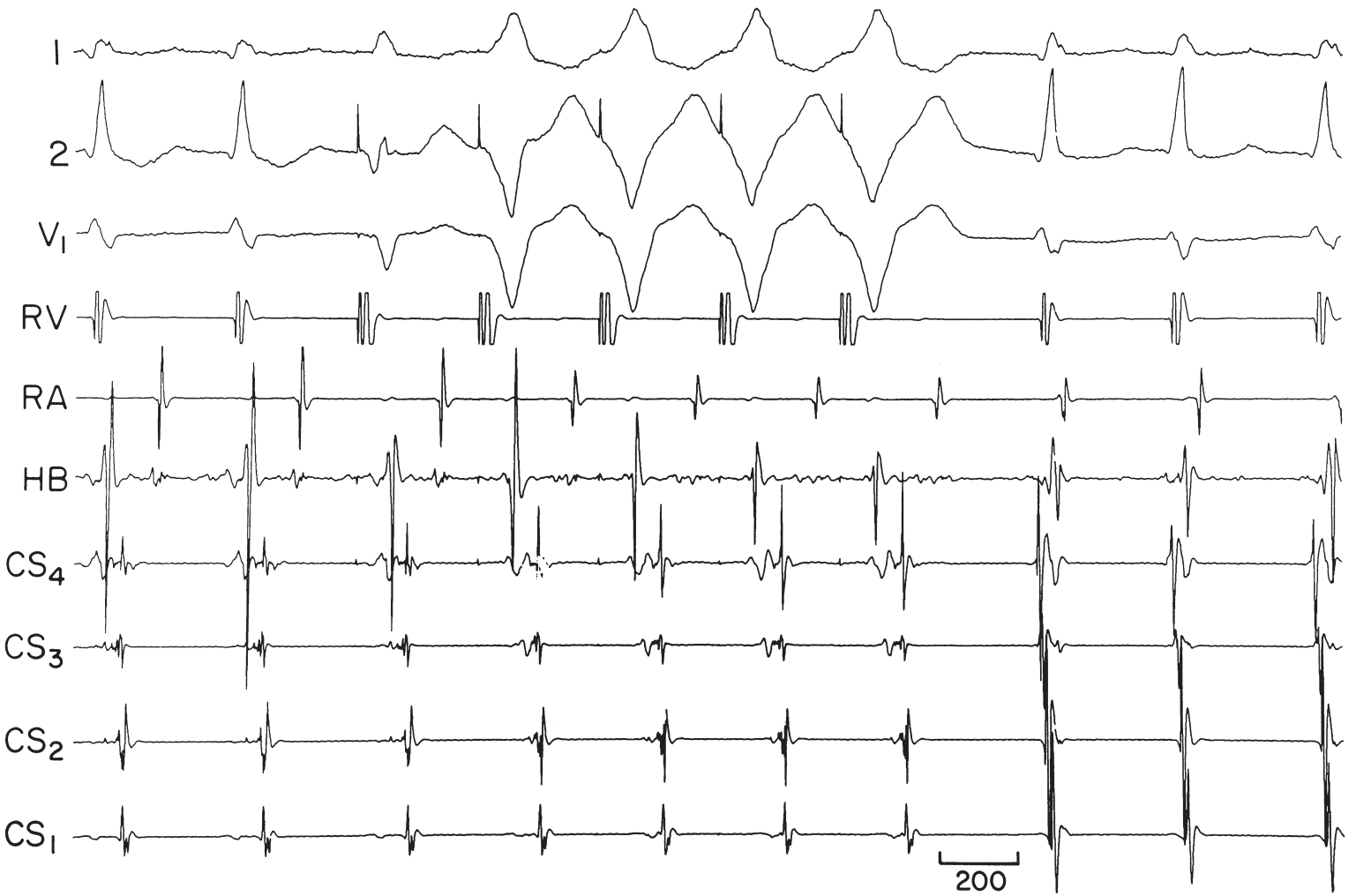
Figure 4–20B



Explanation:

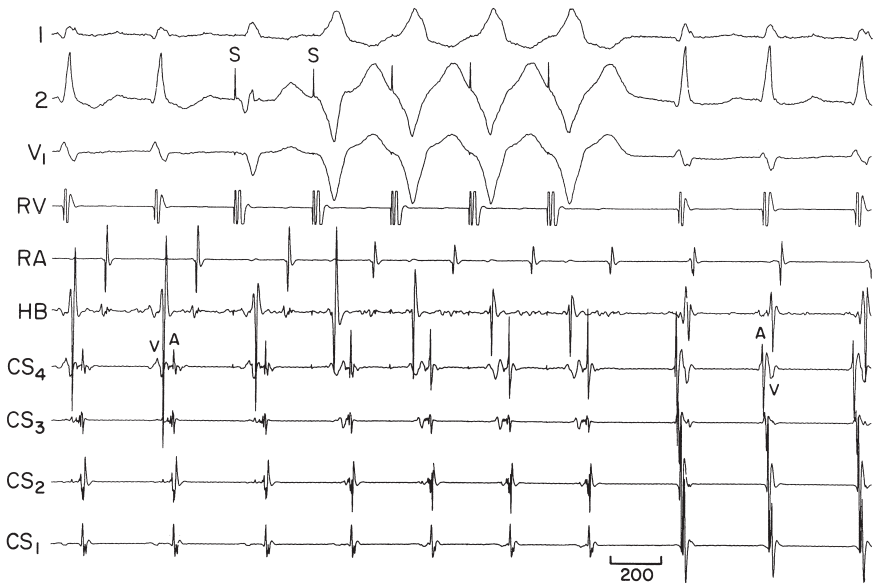
The tachycardia is associated with an eccentric atrial activation sequence, leaving a differential diagnosis of atrial tachycardia or AV reentry over a left-sided AP. The PVC does not preexcite the atrium but prolongs the AH interval and converts the tachycardia to a slower one with a longer AH interval. Importantly, the change in atrial cycle length is linked to preceding change in the AH interval. Such an influence of a programmed PVC would be most unlikely with atrial tachycardia. It is probable that the PVC reached the AV node retrogradely, resulting in subsequent anterograde block of the fast pathway and subsequent conduction over the slow pathway. The first AH is prolonged more than the subsequent AH intervals, suggesting concealed conduction into the slow pathway by the PVC. This patient had AV reentry over a left lateral pathway with two distinct AH intervals consistent with dual AV node pathway physiology demonstrated by the atrial extrastimulus technique. Would one expect to have sustained AV node reentry induced in this patient? The absence of retrograde fast pathway conduction after the slow pathway is initially reached would suggest that the AV node reentrant circuit, at least under baseline conditions, is not operative.

Figure 4–21A



A brief burst of ventricular pacing is provided into this regular supraventricular tachycardia, resulting in change of activation sequence with minimal change in rate. What has happened?

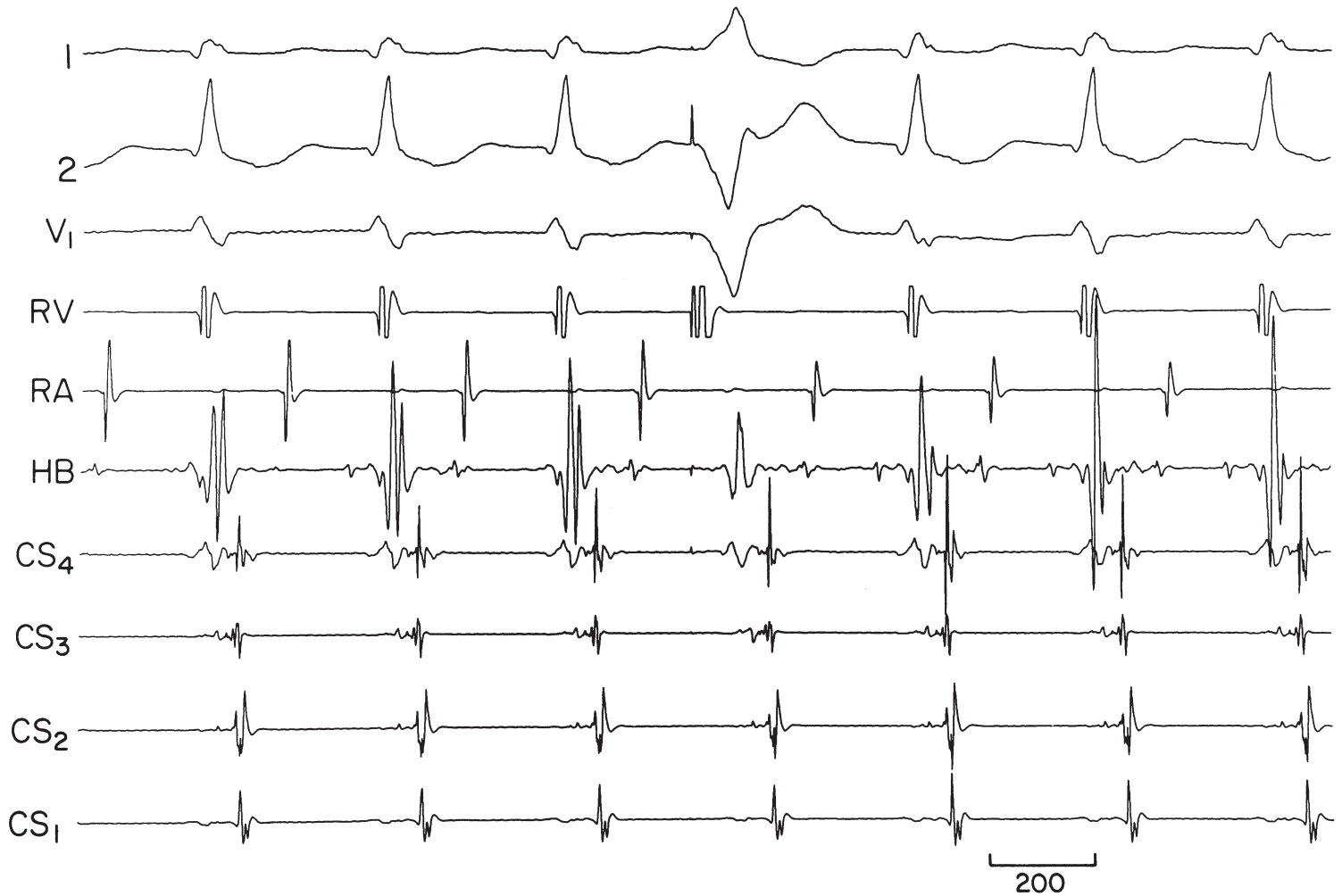
Figure 4–21B



Explanation:

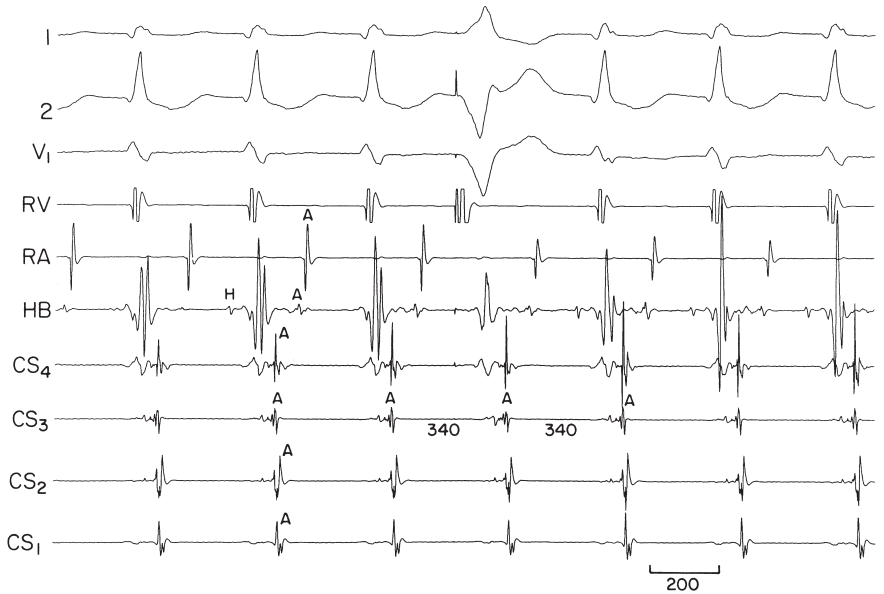
Two cycles of a regular SVT are shown at the beginning of the trace. The earliest atrial activation is in the proximal coronary sinus region (CS_3), approximately 10–20 mm from the orifice of the CS. The differential diagnosis at this point includes atrial tachycardia or AV reentry (Table 1–7). Ventricular pacing results in entrainment of the tachycardia without change in the atrial activation sequence. Note that the second paced beat preexcites the atrium at a pacing interval that would not have been able to conduct retrogradely over the AV node. These two observations essentially confirm the diagnosis of atrioventricular reentry. After termination of pacing, a long AV interval is observed followed by a distinct change of atrial activation sequence. This tachycardia was subsequently confirmed to be AV node reentry. It is probable that entrainment of AV reentry by ventricular pacing resulted in concealed conduction into the AV node with block in the fast pathway after the first atrial cycle after cessation of pacing. Shift to the slow anterograde AV node pathway allowed reentrance up the fast AV nodal pathway with initiation of sustained AV node reentry. Where is the AP at this point? It is probable that there is concealed retrograde conduction into the AP that is preempted from capturing the atrium in whole or in part by retrograde activation over the fast AV node pathway.

Figure 4–22A



A PVC programmed into the cardiac cycle during the tachycardia rules out AV reentry as the mechanism of tachycardia, does it not?

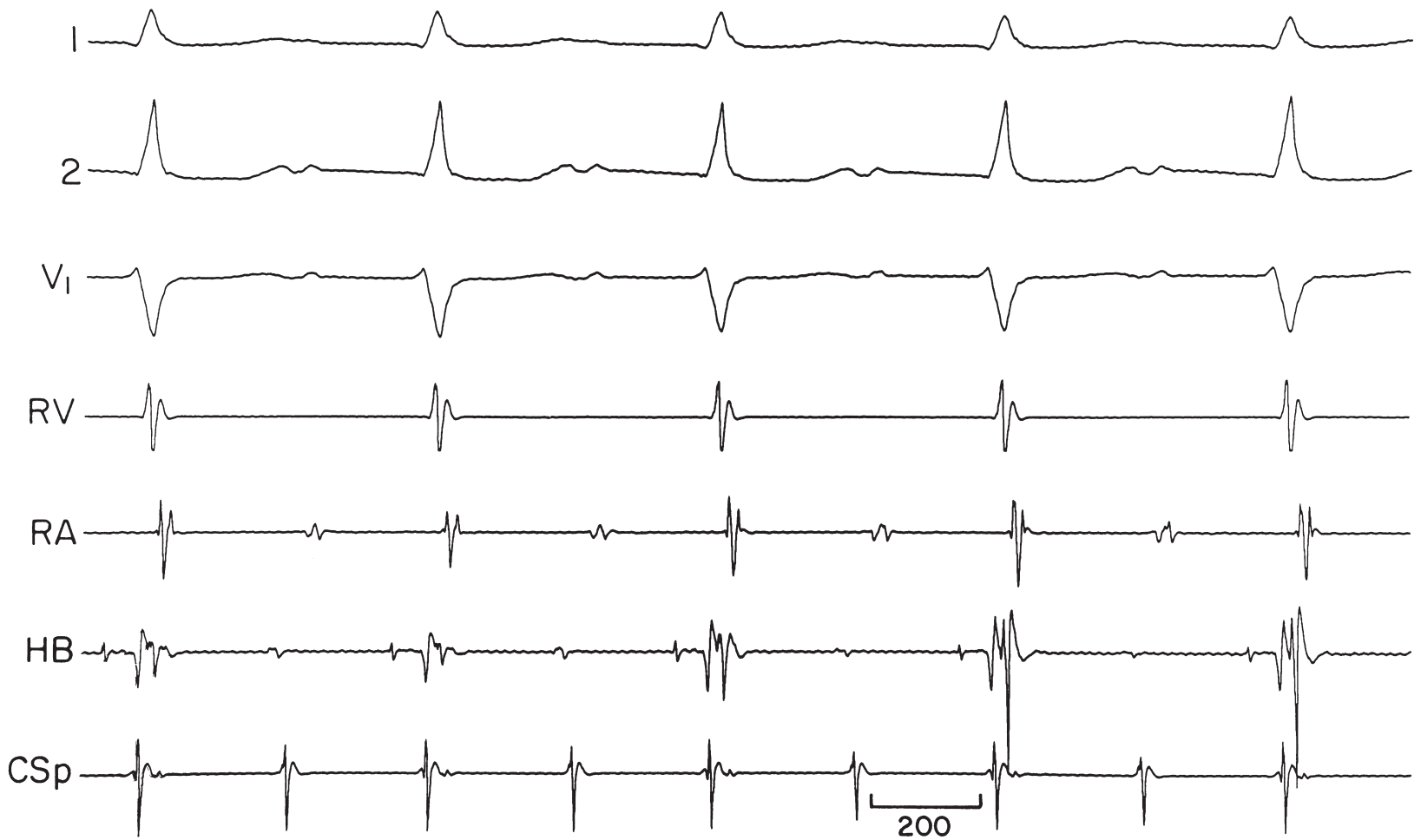
Figure 4–22B



Explanation:

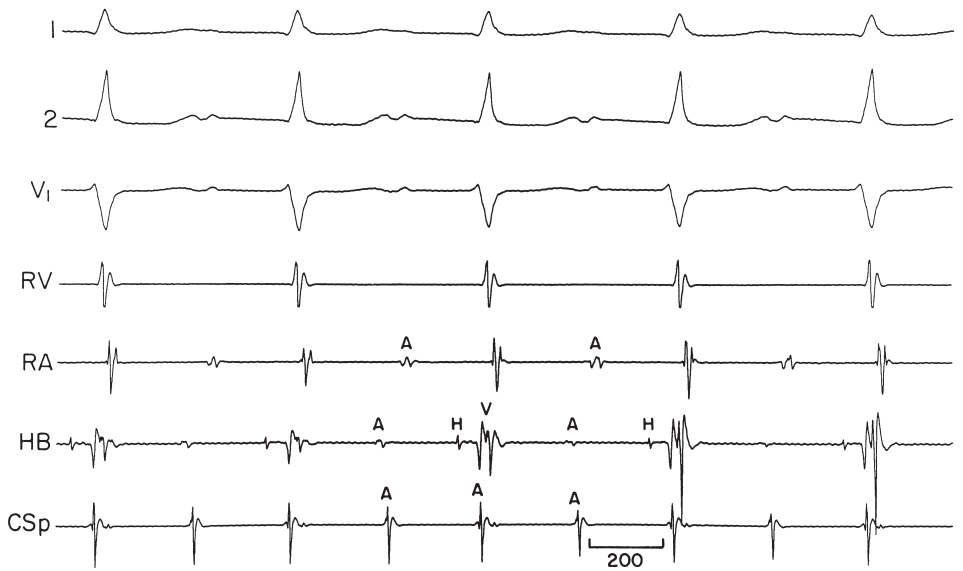
The earliest activation during this tachycardia occurs in the region of the orifice of the coronary sinus and left paraseptal area (CS_4, CS_3). The mechanism of this tachycardia could be AV reentry, atypical AV node reentry, or atrial tachycardia. A PVC delivered during His refractoriness that preexcites the subsequent atrial activation must be conducting over an AP. However, failure to preexcite (as is the case here) is not helpful and leaves the differential diagnosis open. Failure of a PVC to preexcite during AV reentry may be related to distance of the stimulating catheter from the AP and the reentrant circuit. In the example shown, it would be useful to position the pacing catheter at the base of the heart near the site of the AP and repeat the programmed stimulation during tachycardia. This will invariably demonstrate preexcitation in AV reentry unless the AP exhibits "decremental" or rate-dependent conduction, which offsets the prematurity of the extrastimulus. This patient had AV reentry.

Figure 4–23A



At EP testing, this patient with a history of SVT had regular SVT induced by an atrial extrastimulus that achieved a critical AH interval. The tachycardia was compatible with AV node reentry, although the curve relating AH to prematurity of the atrial extrastimulus was continuous. A 2:1 AV block occurred spontaneously during this tachycardia and was recorded. What is the likely diagnosis?

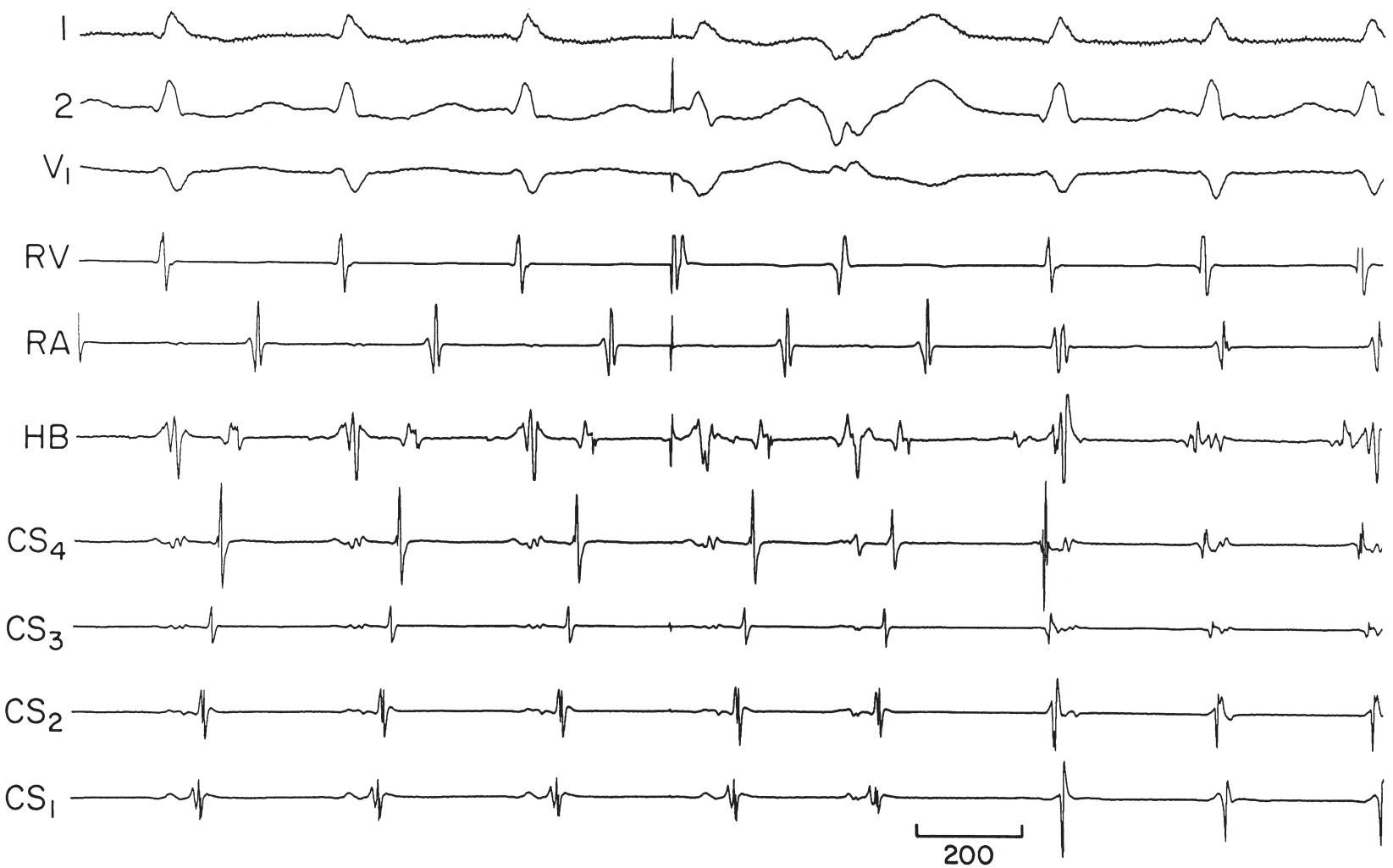
Figure 4–23B



Explanation:

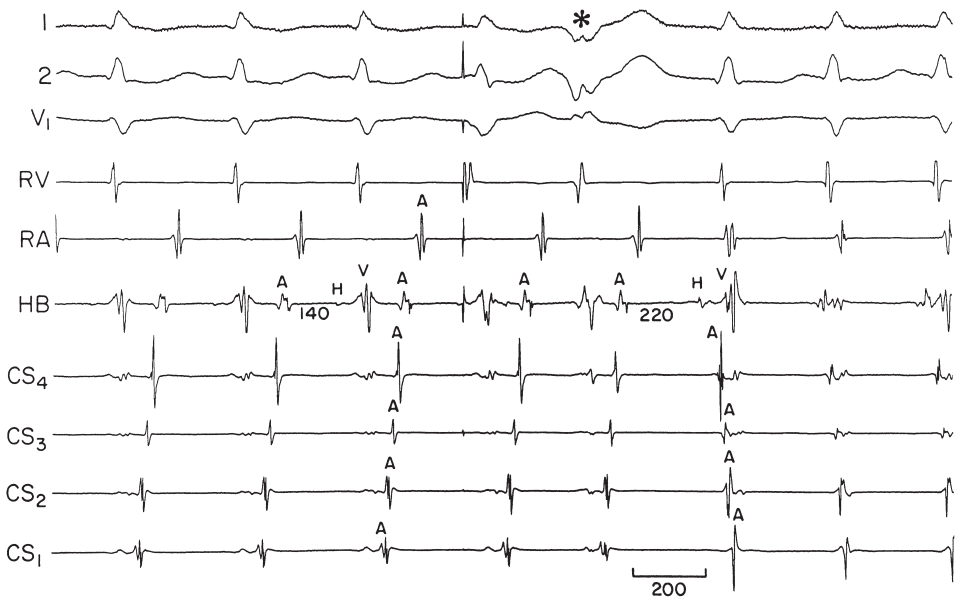
The differential diagnosis is atrial tachycardia or AV node reentry. The distinction cannot be made definitively on examination of this tracing alone. However, note that the atrial electrogram of the blocked cycle is virtually at the midpoint between two QRS complexes, a feature favoring AV node reentry with 2:1 AV block. Another clue to AV node reentry is the characteristic narrow P wave during tachycardia compared with a longer duration during sinus rhythm. If there is AV node reentry with AV block, the block is occurring above the level of the recorded His. This also suggests that the final common pathway is also above the recorded His. However, the recorded His of conducted complexes has a low amplitude and it can be argued that failure to record the His deflection of nonconducted cycles is related to catheter position. Ablation in the slow pathway region eliminated all tachycardia.

Figure 4–24A



A PVC programmed into the cardiac cycle during this tachycardia resulted in a change in tachycardia cycle length. How does one explain the observations? Will ablation at a single site cure this patient?

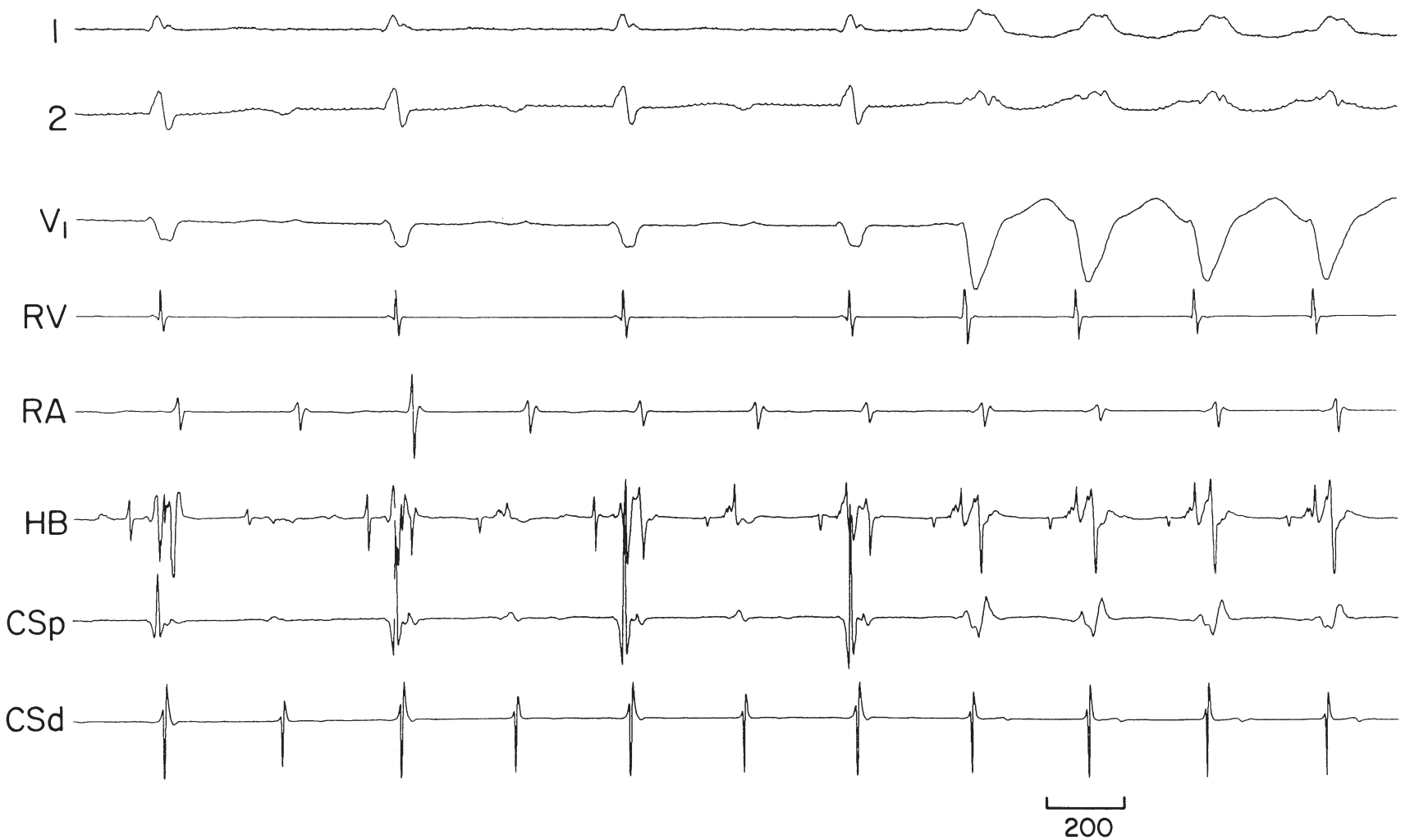
Figure 4–24B



Explanation:

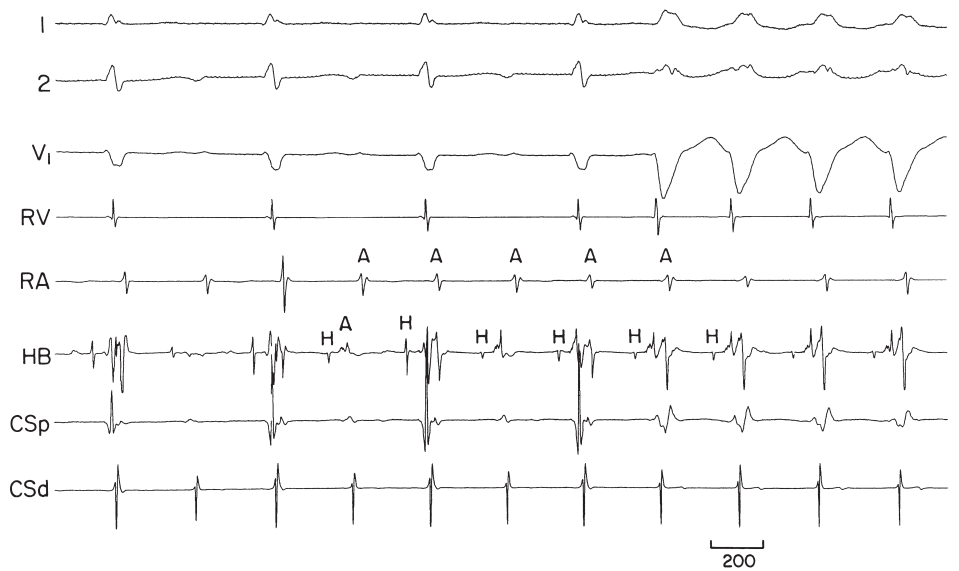
At the onset, the tachycardia is associated with an eccentric atrial activation sequence (distal CS first). The differential diagnosis is atrial tachycardia versus AV reentry utilizing a left lateral AP as the retrograde limb of a circuit. A PVC introduced into the cycle at the time of His bundle refractoriness fails to affect the tachycardia circuit. However, a spontaneous PVC (*) occurs relatively earlier in the cycle and readily preexcites the atrium in the proportion to prematurity of the ventricular ectopic, supporting a diagnosis of AV reentry. The morphology of the spontaneous PVC suggests a left ventricular origin, explaining why the PVC preexcites the atrium over a left lateral pathway more readily than the induced one from the right ventricular apex. With preexcitation of the atrium (essentially a premature atrial complex), the subsequent AH interval prolongs suddenly, indicating block in the fast AV node pathway and shift to a slow AV node pathway. Subsequent retrograde fast AV node pathway activation then perpetuates sustained AV node reentry. This patient requires ablation of both the AP and the slow AV node pathway.

Figure 4–25A



This tachycardia was induced by a critically timed atrial extrastimulus associated with prolongation of the AH interval. What is the mechanism of tachycardia?

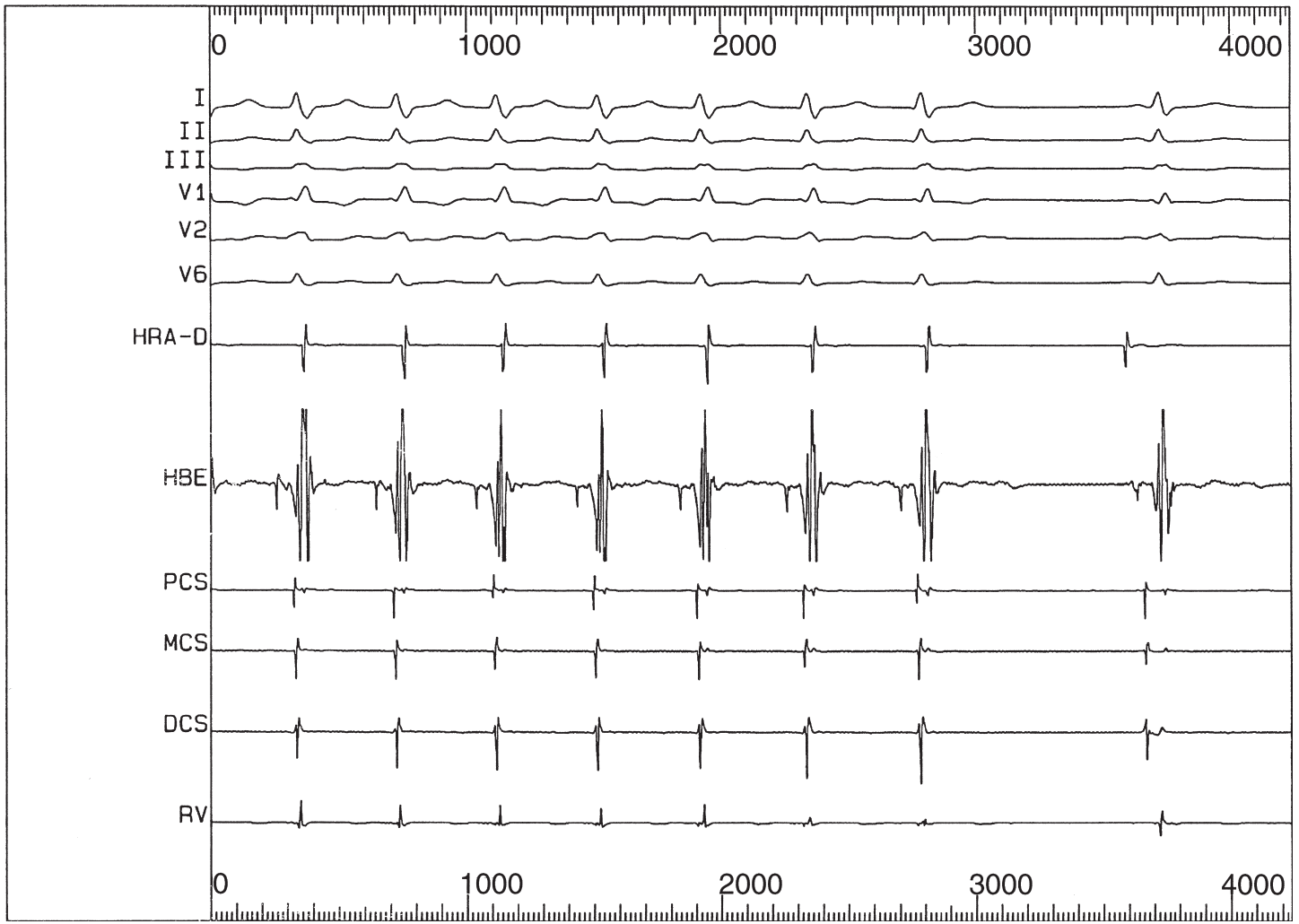
Figure 4–25B



Explanation:

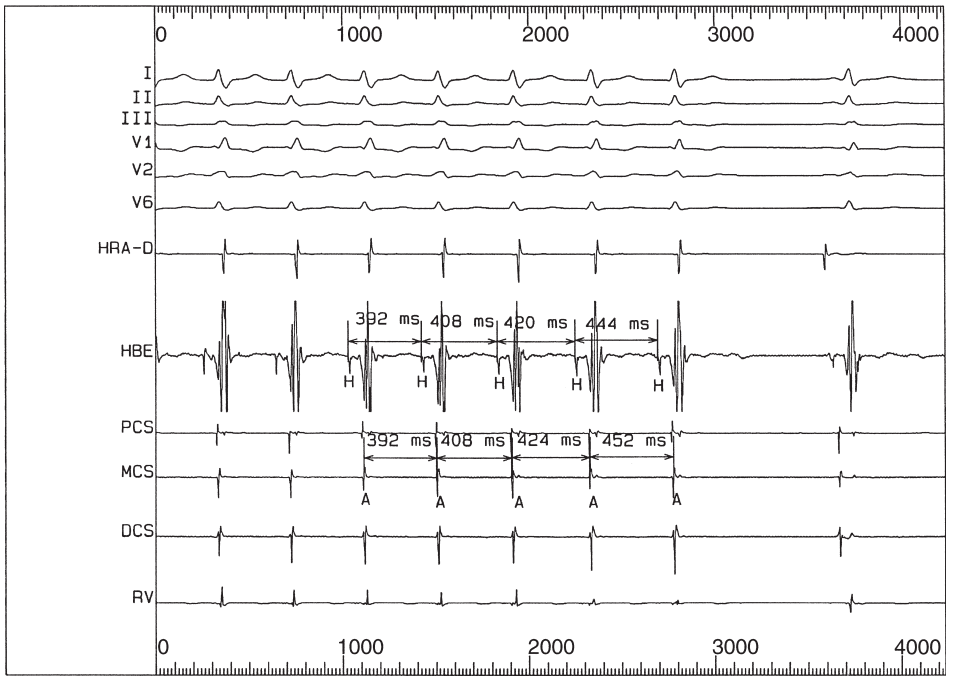
The tracing begins with a supraventricular tachycardia with 2:1 AV block. The activation sequence is central with earliest recorded atrial activation at the His bundle electrogram. There is an abrupt transition to 1:1 AV conduction. Resumption of 1:1 conduction is associated with LBBB aberration, commonly noted in this situation. SVT with AV block immediately suggests atrial tachycardia as the mechanism. However, the apparently nonconducted atrial electrogram is virtually in the center of the diastolic interval between two QRS complexes. This immediately suggests a diagnosis of AV node reentry, in this case with 2:1 block below the level of the recorded His. This was indeed the case in this patient in whom slow pathway ablation eliminated tachycardia. Note, however, that this tracing in isolation does not rule out atrial tachycardia.

Figure 4–26A



This is a 36-year-old woman with a history of palpitations. At EP study the observation shown here was made. What is the mechanism of tachycardia?

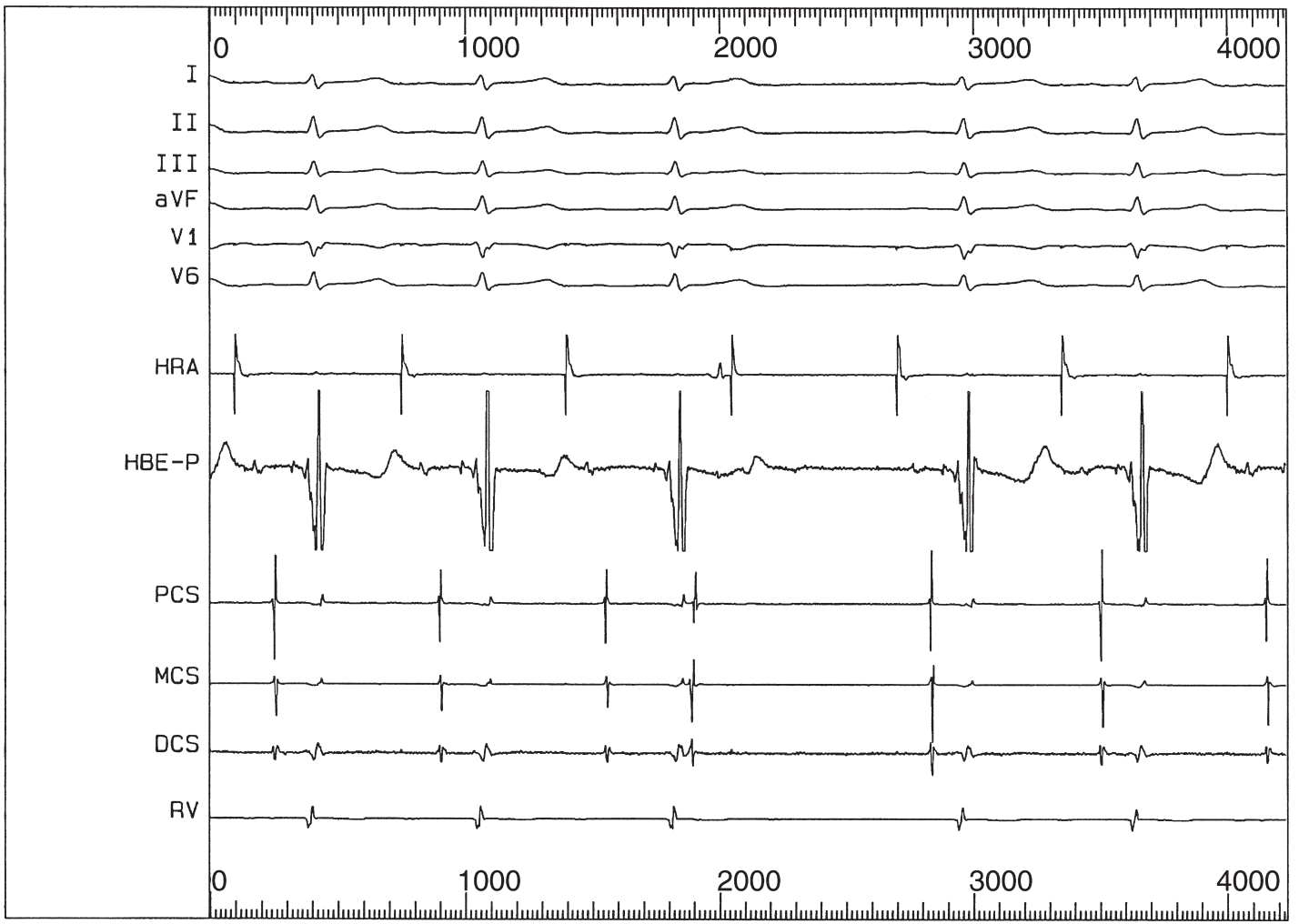
Figure 4–26B



Explanation:

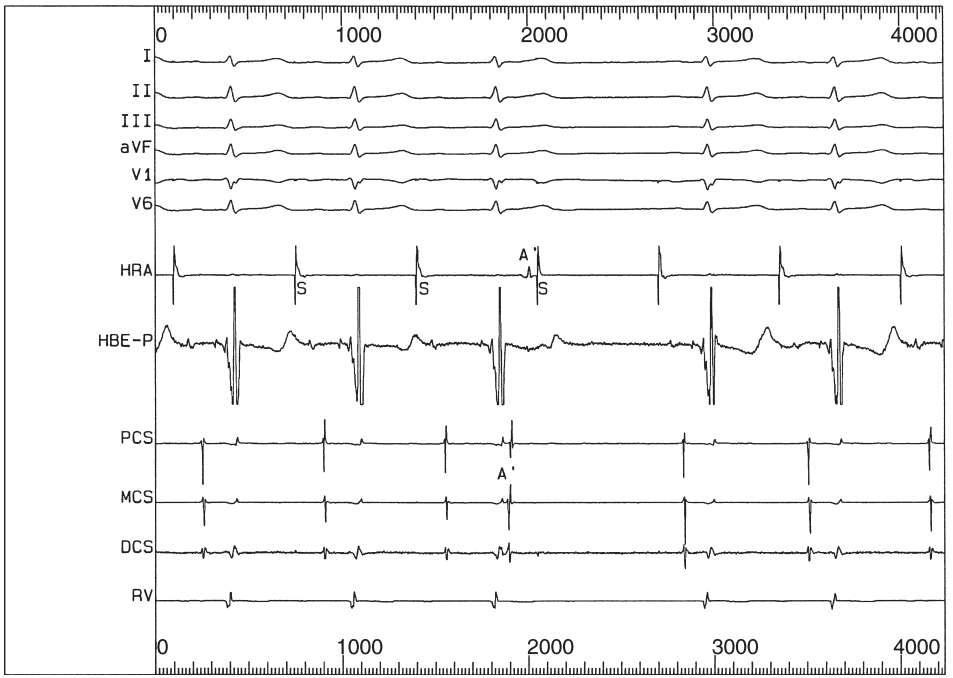
This patient has SVT with a relatively short VA interval. Of note, the activation sequence is low to high atrium. Thus, sinus tachycardia is excluded by the atrial activation sequence, and AV reentry is excluded by the short VA interval. AV nodal reentry and atrial tachycardia remain possibilities. This differential diagnosis often depends on demonstrating that the AV node is part of the tachycardia circuit, or that the atrium is not required. In this instance the AV node is implicated in the circuit. Note the progressive prolongation of tachycardia cycle length as measured by the H–H intervals on the His bundle lead. These precede and predict subsequent changes in atrial cycle length, and the tachycardia terminates with an atrial electrogram that does not conduct to the AV node. This observation implies that the AV node is part of the tachycardia circuit, and the diagnosis is AV node reentry. Changes in AV node conduction have no effect on the rate of atrial tachycardia. One would not expect atrial tachycardia to terminate because of loss of AV node conduction, although this could happen fortuitously. Consistent termination of tachycardia without AV node conduction implicates the AV node as part of the circuit.

Figure 4–27A



This patient has a history of documented PSVT. During atrial pacing the observation shown was noted. What is the likely mechanism of tachycardia? Why did the patient not have tachycardia? What would you do next to try to initiate tachycardia?

Figure 4–27B



Explanation:

During atrial pacing there is a progressive prolongation of the AH interval. After the third paced beat the drive train is interrupted by a premature atrial complex (*A′*). The activation sequence is eccentric, occurring first in the CS. Although this could be a premature atrial complex originating in the left atrium, the more likely diagnosis is an AV reentrant echo utilizing a concealed left-sided AP for retrograde conduction.

Tachycardia does not occur because the atrial echo does not conduct over the AV node. This is not surprising since there was progressive prolongation of the AH interval during pacing, suggesting a Wenckebach sequence, and the AV reentrant echo has an even shorter interval than the drive complexes. Note that retrograde conduction over the AP occurred only after a substantial AV nodal delay on the third paced beat. It is not known whether there is complete anterograde block in the retrograde concealed AP or some degree of penetrance. Regardless, there must be a requisite delay before retrograde conduction can occur over the AP, and this is provided by the critically prolonged AH interval from the third paced beat. To initiate sustained tachycardia, isoproterenol was given and sustained AV reentry was initiated during atrial pacing (Fig. 4–27*C*).

Figure 4–27C

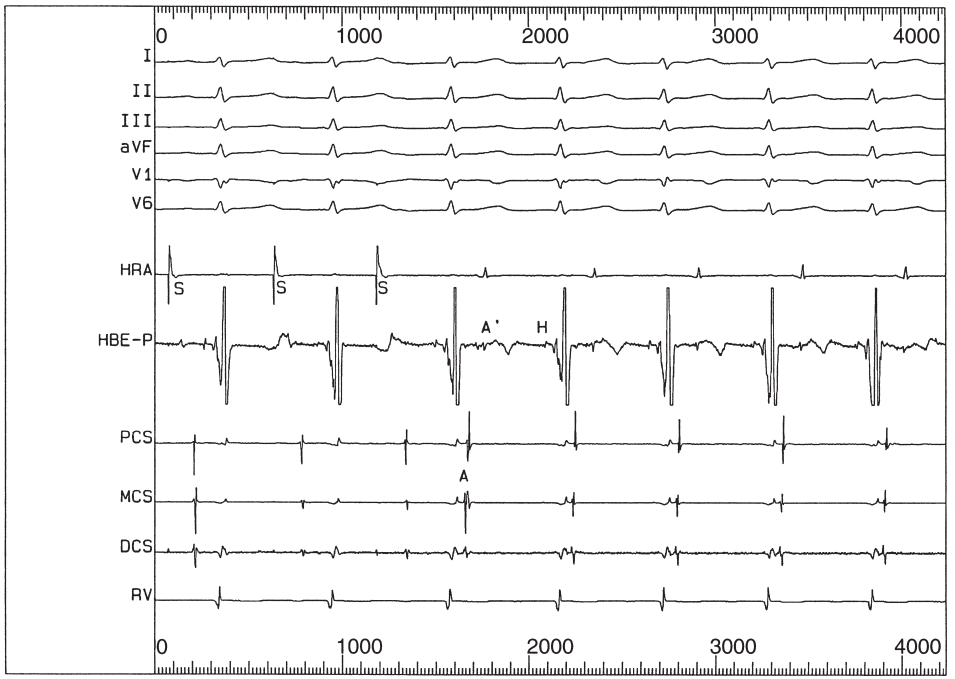

0
1000
2000
3000
4000
I
II
III
aVF
V1
V6
HRA
S
S
S
HBE-P
A'
H
PCS
A
MCS
DCS
RV
0
1000
2000
3000
4000

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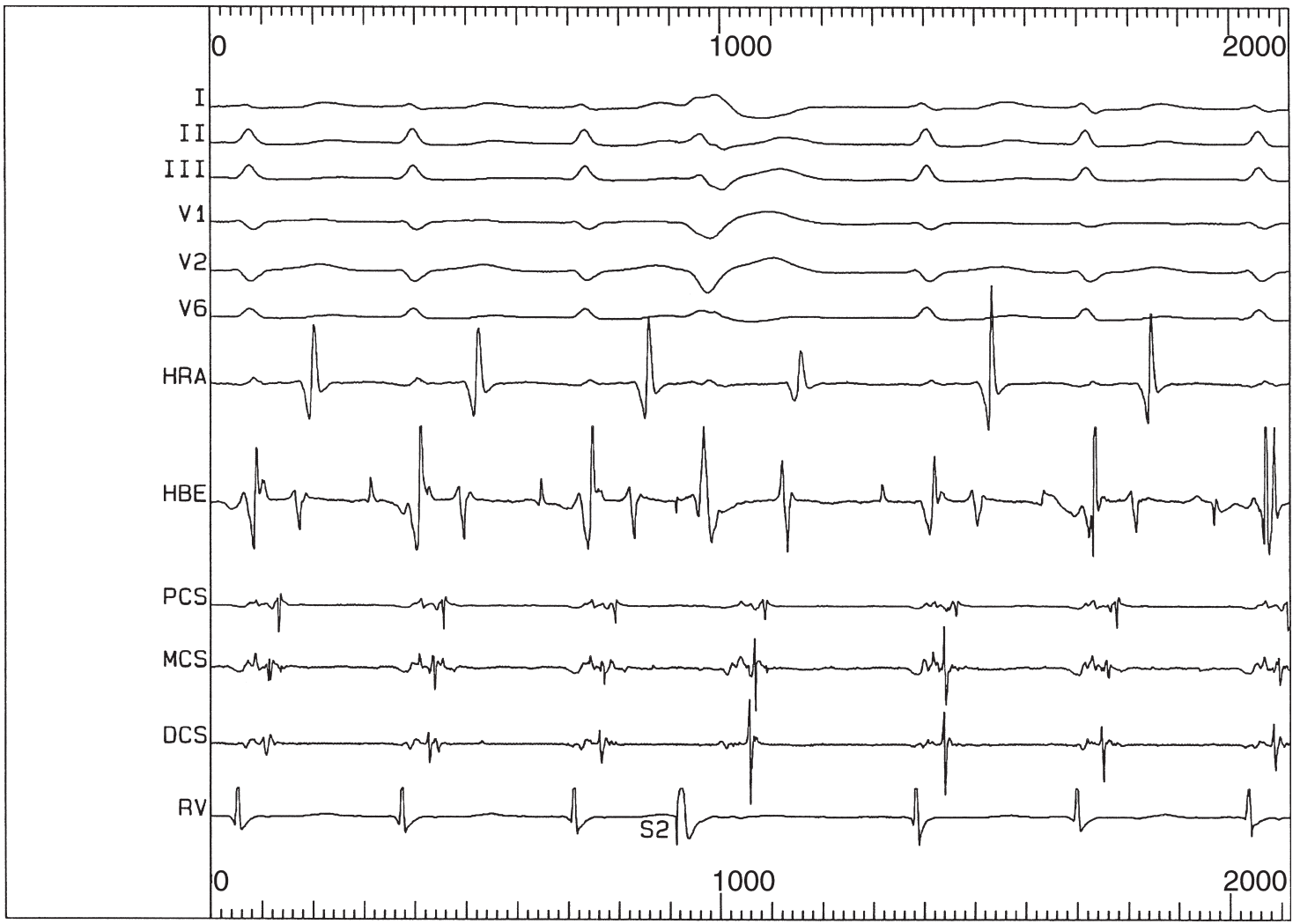
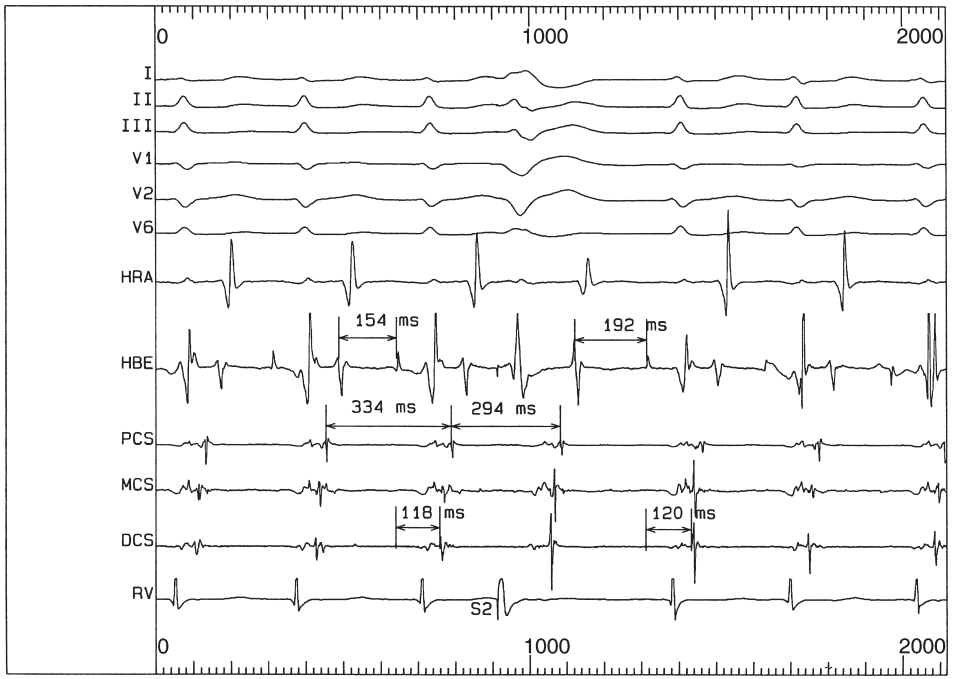
Figure 4–28A



A premature ventricular complex was introduced during tachycardia.
Has the mechanism of tachycardia been identified?

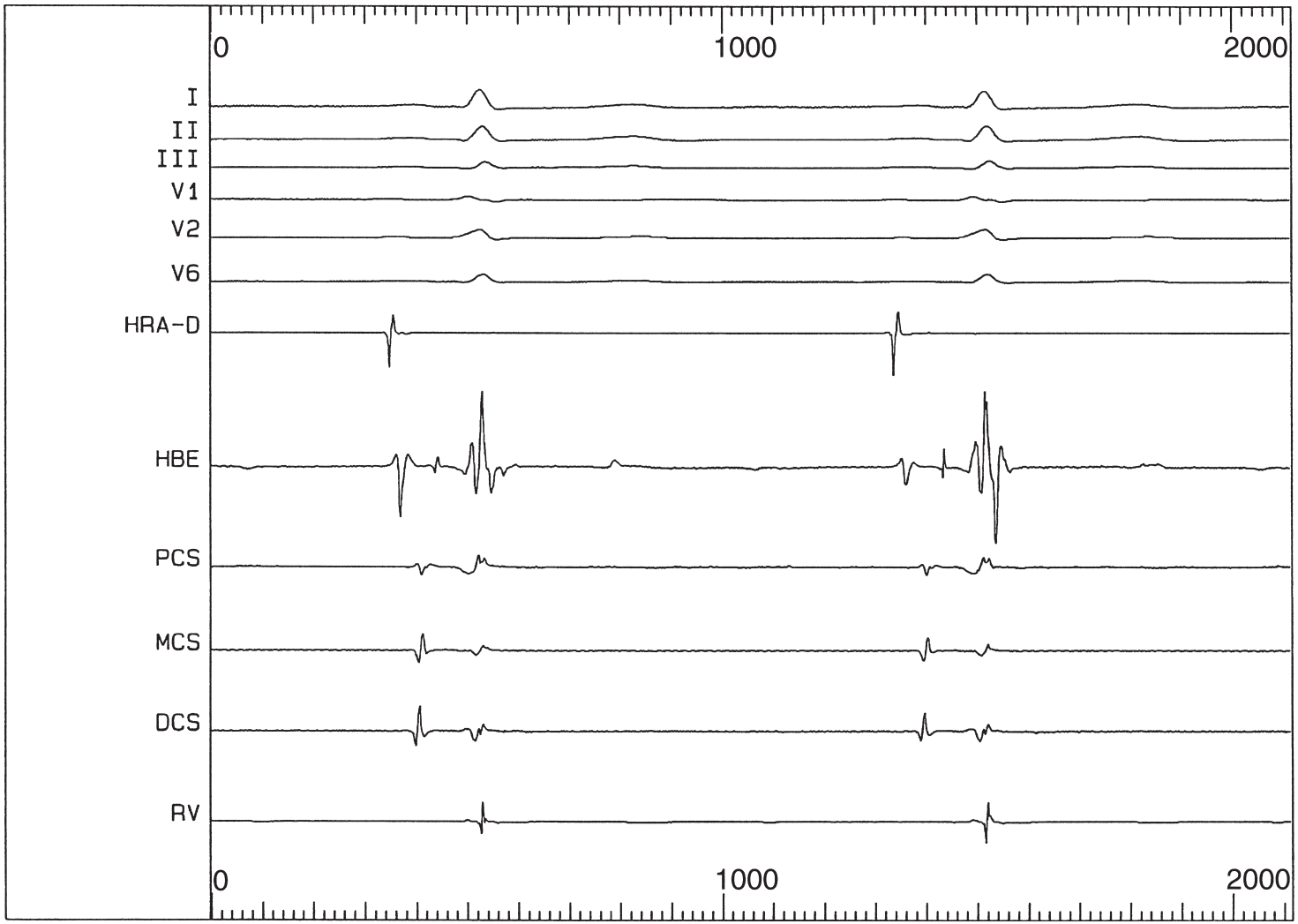
Figure 4–28B



Explanation:

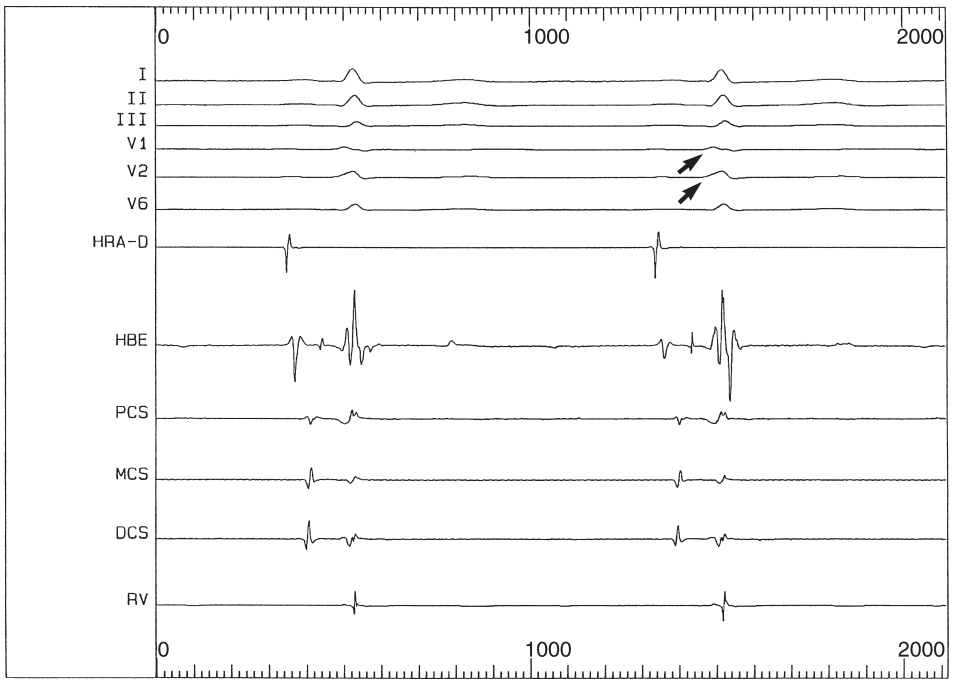
The patient has a regular SVT with an eccentric atrial activation sequence with earliest activation occurring on the distal CS electrode positioned at the left lateral atrium. The differential diagnosis is a left atrial tachycardia versus AV reentry. The PVC preexcites the atrium during tachycardia, identifying the presence of a left-sided AP. Preexcitation of the atrium does not confirm participation of the pathway during tachycardia. This is true even though the activation sequence is similar for the tachycardia and preexcited atrial complexes. One could postulate the very unlikely occurrence of a left-sided atrial tachycardia originating in an area very near the left-sided AP. Additional data in this figure essentially confirm the diagnosis of AV reentry. Note that the premature atrial complex (294 milliseconds) following the premature ventricular complex causes a subsequent prolongation in AV nodal conduction time from 154 to 192 milliseconds. Regardless, the His to local atrial interval is essentially unchanged from 118 to 120 milliseconds. The “marriage” of the local atrial electrogram to the preceding His bundle deflection regardless of AV node conduction time would not be anticipated in an atrial tachycardia. Since fortuitous resetting of the atrial interval during an atrial tachycardia could produce the same result for a single premature complex, multiple intervals should be measured to confirm the diagnosis.

Figure 4–29A



This patient has a history of palpitations and presyncope. Are there any clues to the diagnosis identified in this figure?

Figure 4–29B



Explanation:

Two subtle observations are suggestive of Wolff–Parkinson–White syndrome. First, the initial portion of the QRS complex in V1 and V2 (*arrows*) is abnormal and could represent preexcitation. Second, the HV interval is short, measuring 30 milliseconds. The relatively short AH interval precluded more marked preexcitation during sinus rhythm in this patient. Note the rapid preexcited ventricular response during atrial fibrillation in Fig. 4–29*C*, which was associated with hypotension and dizziness, and likely was the cause of the patient's presyncopal episode.

Figure 4–29C

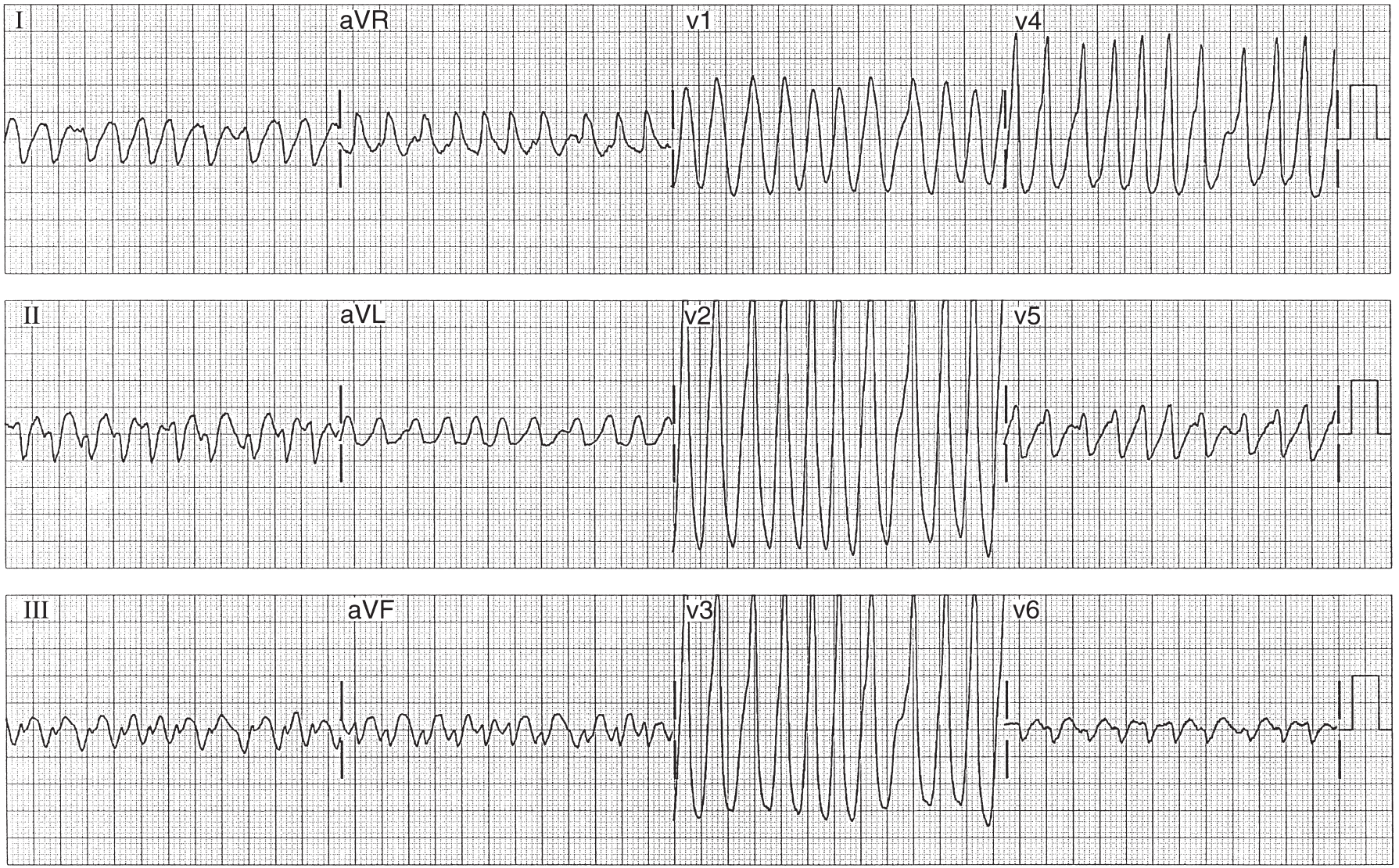


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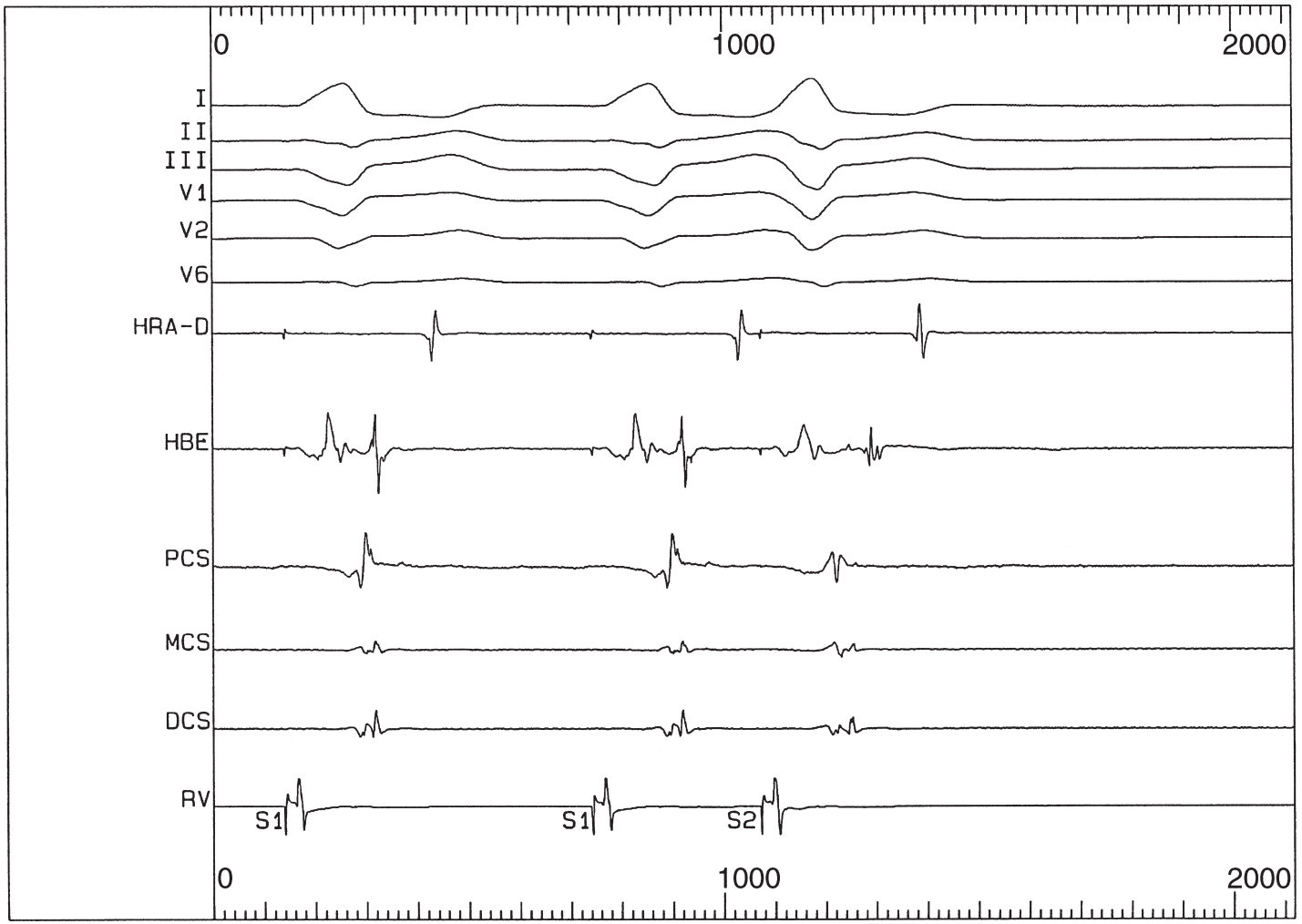
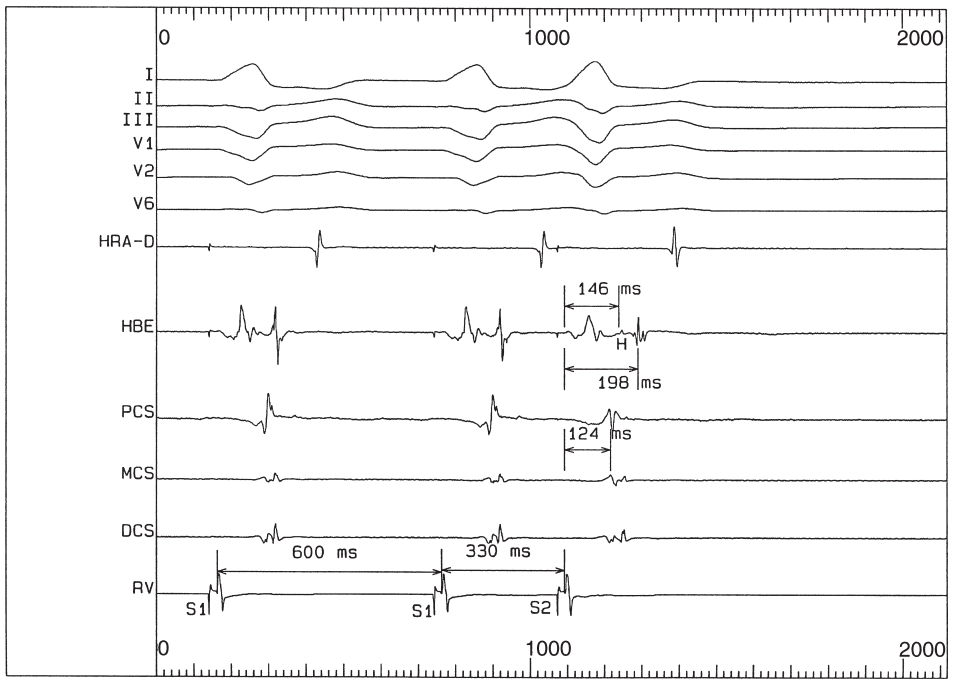
Figure 4–30A



The tracing was recorded during premature ventricular stimulation in a patient with documented SVT. What is the likely mechanism of tachycardia? Why was it not initiated? What pacing maneuvers would help to initiate tachycardia?

Figure 4–30B



Explanation:

The patient has a left free wall AP. This is confirmed during premature ventricular stimulation. Note that activation of the atrial electrogram in the proximal CS lead precedes activation of a retrograde His potential. The CS VA interval of 124 milliseconds is substantially shorter than the 198-millisecond interval recorded in the His bundle electrogram. Tachycardia does not occur because the PVC conducted retrogradely into the AV node, which precluded subsequent anterograde AV nodal conduction.

Initiation of AV reentry during programmed ventricular stimulation requires minimal to no retrograde conduction into the AV node, that is, unidirectional block. The simplest method to achieve this goal is to introduce progressively shorter premature intervals as noted in Fig. 4–30*C*. In this instance tachycardia is initiated with an S1S2 interval of 320 milliseconds, and the VA conduction times on the CS and His bundle leads are similar to those in Fig. 4–30*B*. Note that a retrograde His deflection is not present, suggesting block in the HPS with this PVC. The similar VA conduction times on the His bundle electrogram with both PVCs strongly suggest that they were activated by conduction proceeding from the left atrium to the septum over the AP. Other methods to initiate AV reentry during ventricular pacing include multiple premature complexes, burst ventricular pacing, and pacing at slower cycle lengths. At slower paced cycle lengths, the refractoriness of the HPS often prolongs more than the AP, allowing block in the HPS to occur while conduction can still proceed over the AP.

Figure 4–30C

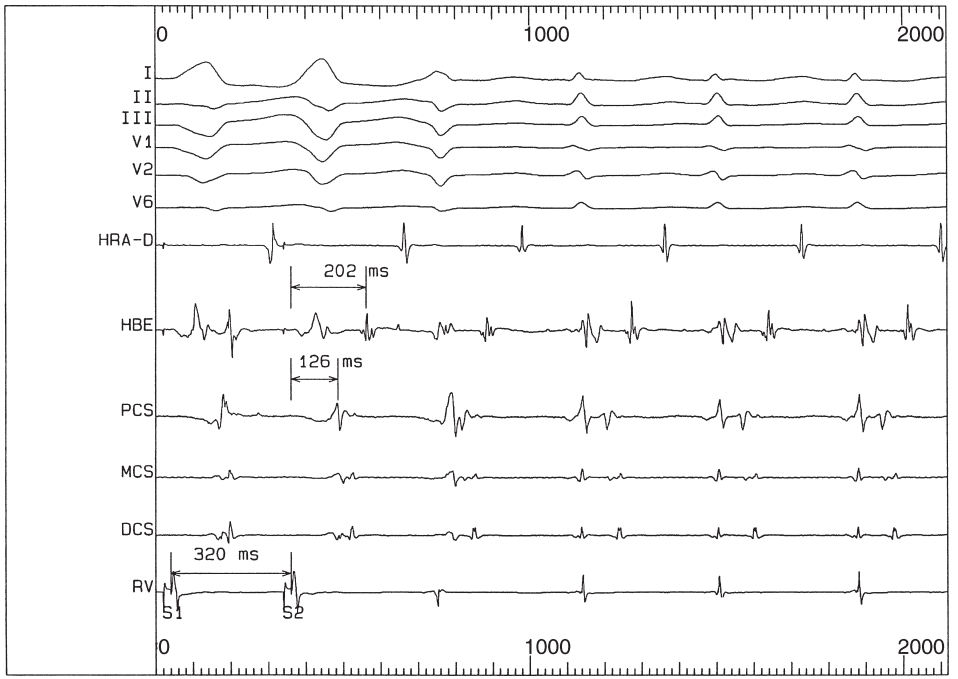

0
1000
2000
I
II
III
V1
V2
V6
HRA-D
202 ms
HBE
126 ms
PCS
MCS
DCS
320 ms
RV
S1
S2
0
1000
2000

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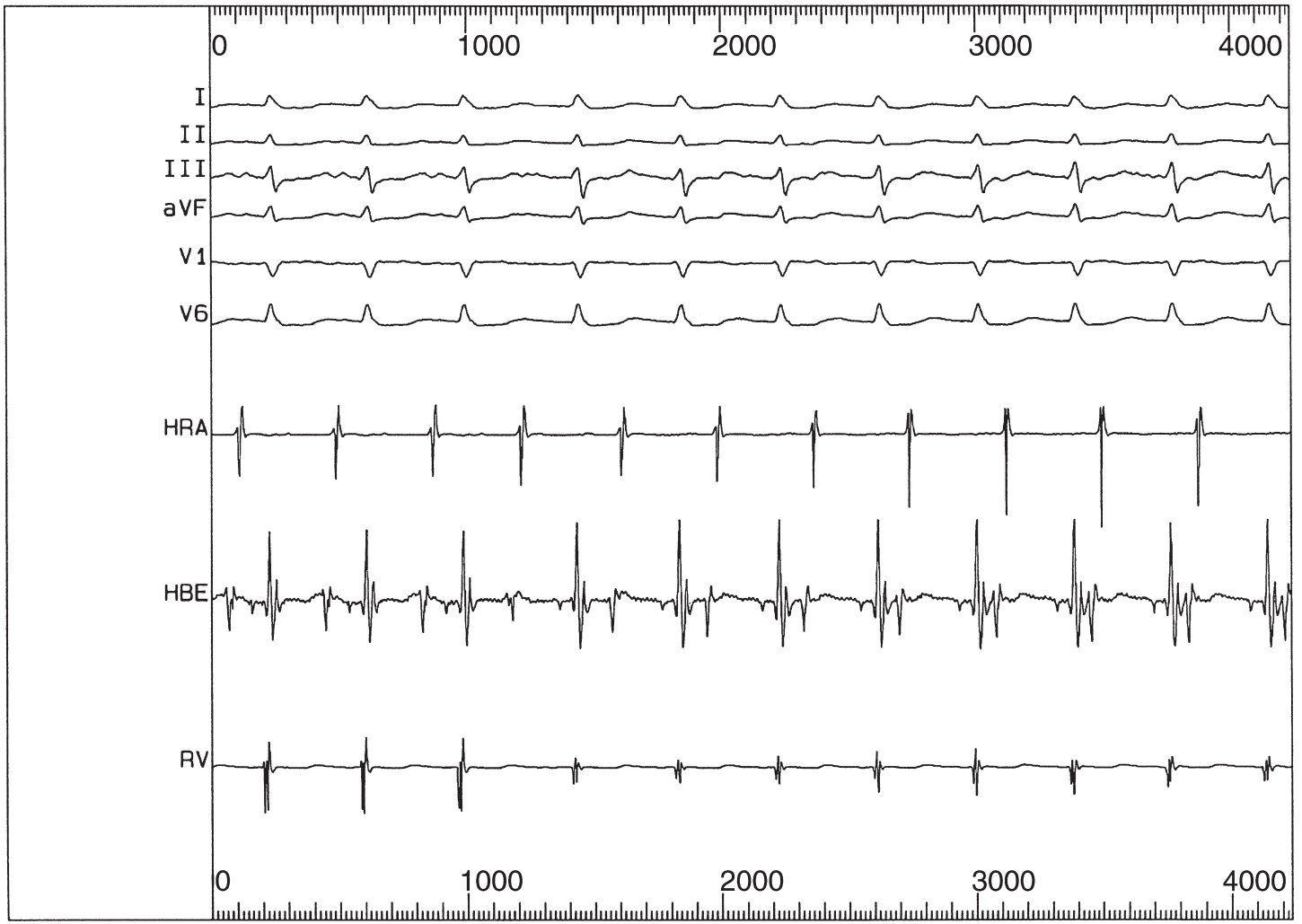
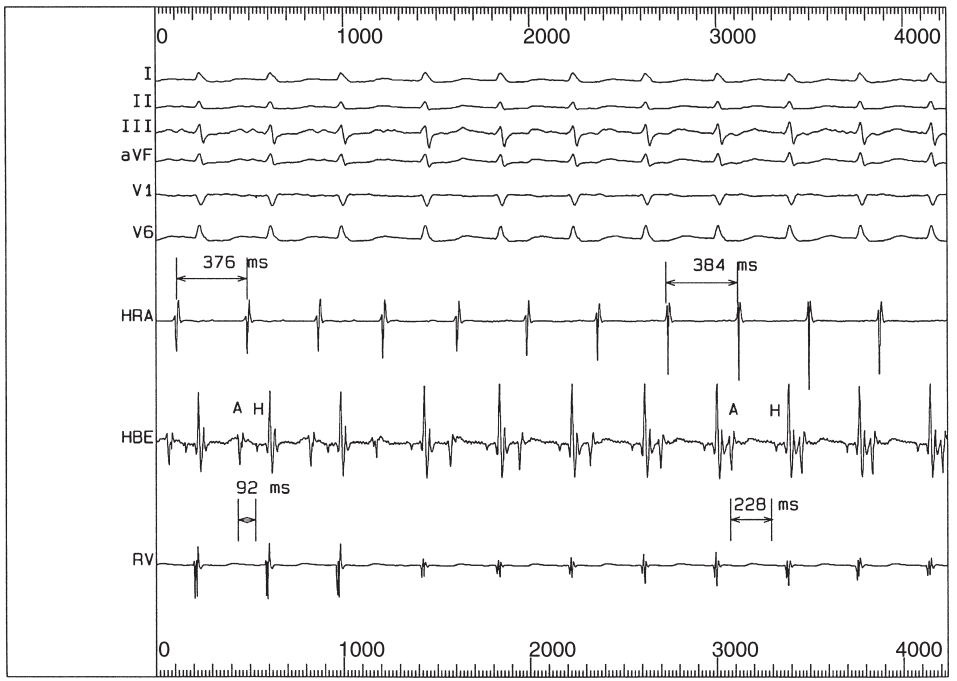
Figure 4–31A



This patient had nearly incessant tachycardia. What is the likely mechanism?

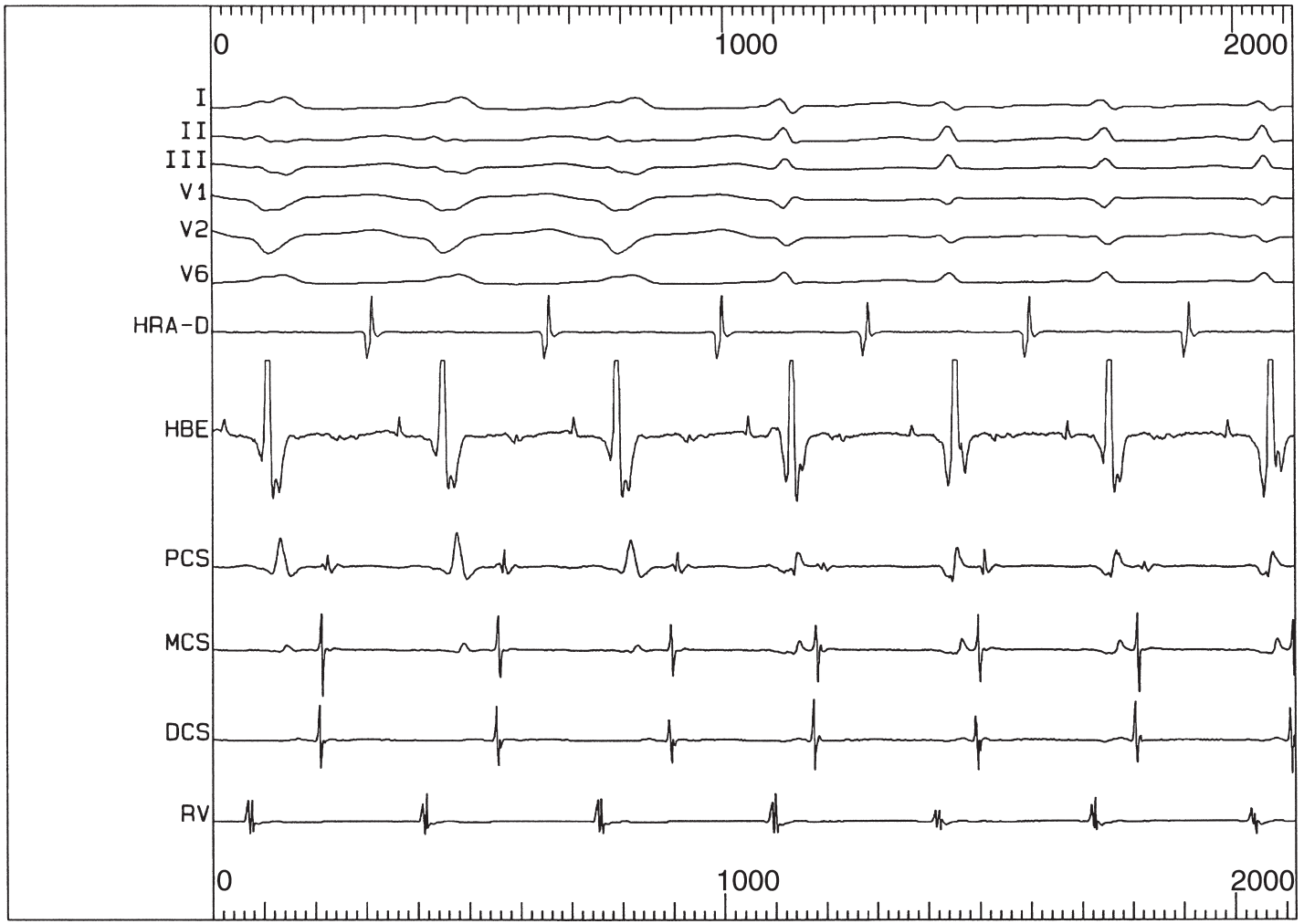
Figure 4–31B



Explanation:

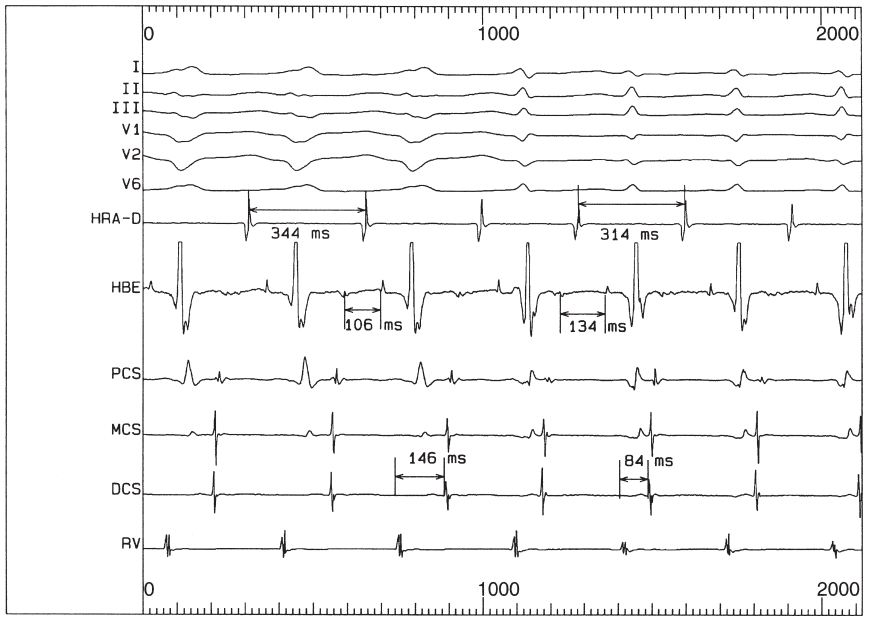
This is a SVT with a low to high atrial activation pattern. There is minimal variability in the atrial cycle length. Differential diagnosis is AV node reentry, AV reentry, and atrial tachycardia. Note that the AH interval progressively lengthens from 92 to 228 milliseconds in this tracing. With lengthening of the AH interval, there is a shortening of the measured VA interval. Yet, there is minimal change in the atrial cycle length. This is typical of atrial tachycardia and essentially excludes AV and AV node reentry. It is conceivable that one could have AV node reentry with the circuit totally proximal to the area of AV nodal conduction delay. If this occurred in the interatrial septum, especially along the tricuspid annulus, it would be impossible to differentiate from a septal tachycardia in this location. There have to be some rules established to differentiate these arrhythmias. In general, when marked changes in the AH interval occur without corresponding changes in atrial cycle length, we exclude the AV node as a significant part of the tachycardia circuit. Programmed stimulation techniques are also useful to support the diagnosis of atrial tachycardia. Initiation with PACs of tachycardia with a similar cycle length but with marked variability in AH interval, or without AV node conduction, suggests atrial tachycardia. Continuation of tachycardia without changes in atrial cycle length when PVCs are introduced and cause marked AH variation also supports the diagnosis of atrial tachycardia.

Figure 4–32A



This tracing contains many interesting electrophysiologic observations. Are there one or two tachycardias present? Is there proof of the tachycardia mechanism? Why is there a change in atrial cycle length, and why is it not larger?

Figure 4–32B

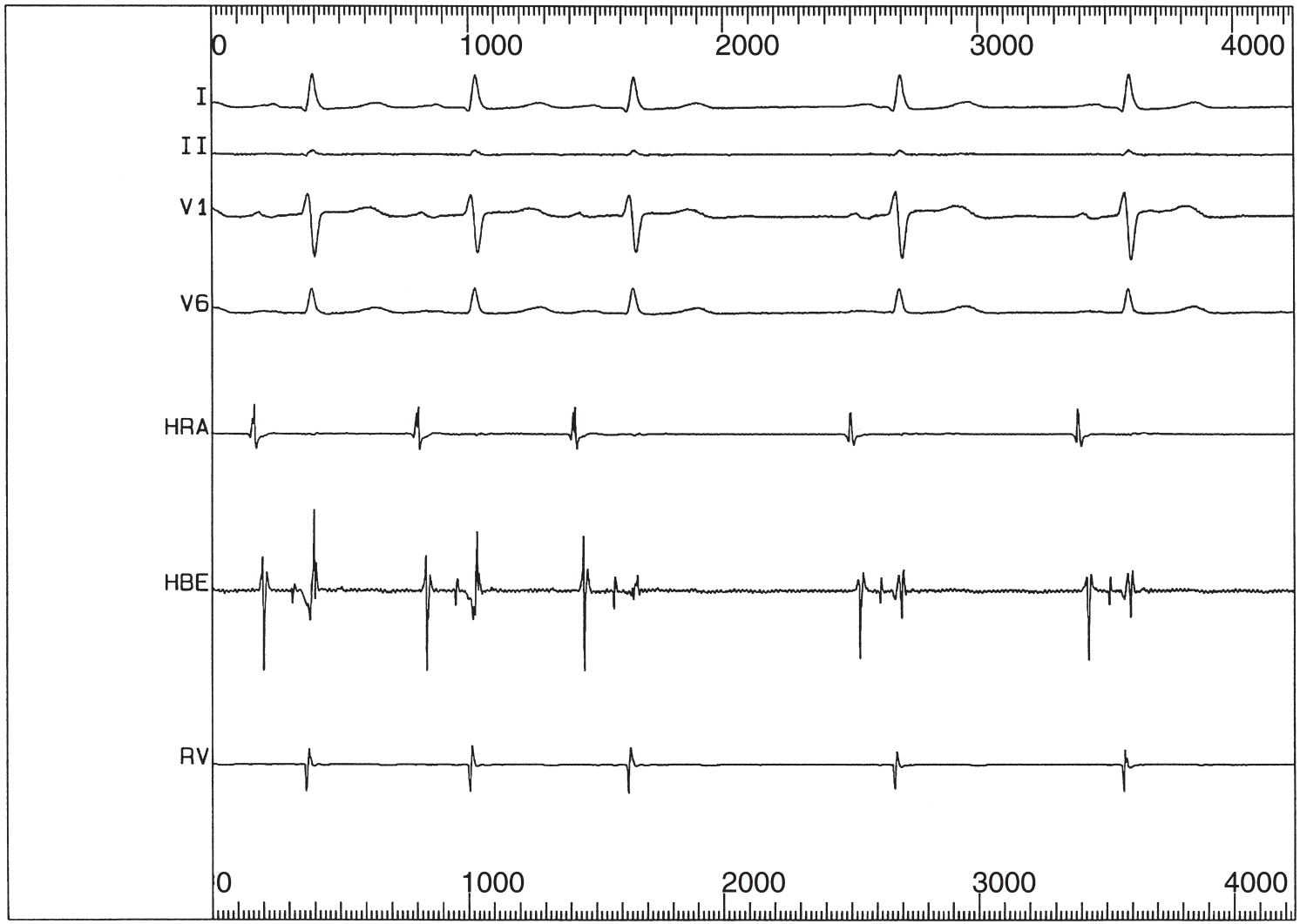


Explanation:

This patient has one tachycardia, AV reentry using a left-sided AP for retrograde conduction. During induction of tachycardia, the patient frequently had a short run of LBBB aberrancy that normalized. This probably represents progressive shortening of the refractory period of the left bundle branch to allow conduction to occur, rather than transseptal concealed conduction, which typically is long lasting. With normalization of the QRS, the VA interval shortens from 146 to 84 milliseconds. This confirms participation of the left-sided AP in the tachycardia circuit. This sudden shortening of the VA interval also results in a shortened AA interval, followed by prolongation of the AH interval from 106 to 134 milliseconds. Thus, although the atrial cycle length is shortened from 344 to 314 milliseconds, the amount of cycle length decrease is mitigated by the concomitant increase in AH interval. In approximately 10–15% of patients, there is an increase in AH interval that approximates the shortening of VA interval and no change in tachycardia cycle length occurs. Thus, the change in VA interval is the critical measurement and not change in cycle length.

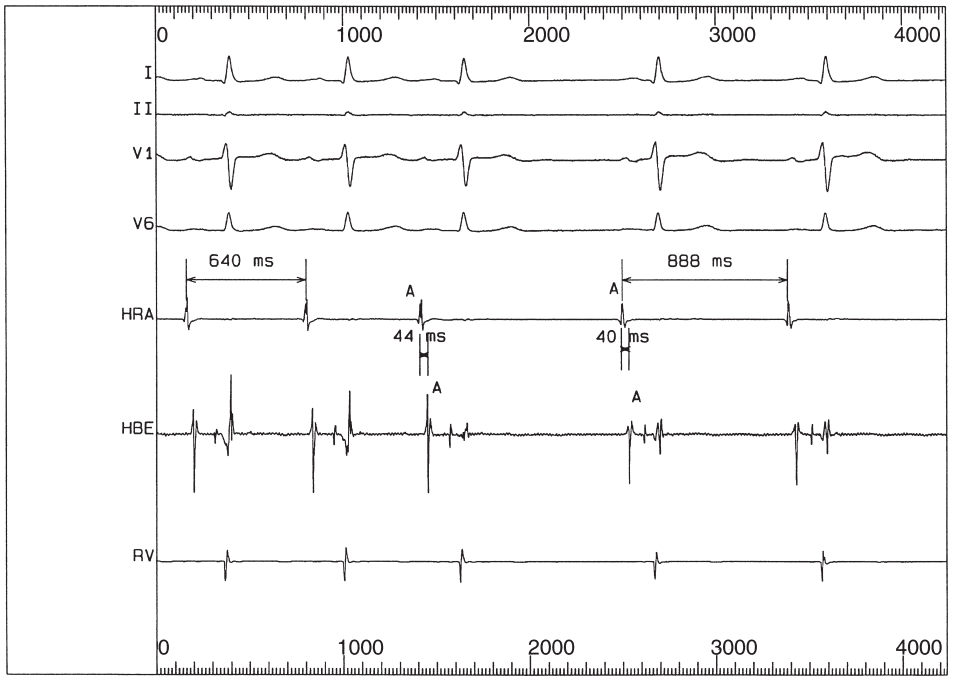
When analyzing electrocardiograms, one does not have the advantage of accurately measuring VA intervals in most cases. Thus, in this situation a difference in tachycardia cycle length between bundle branch block and narrow QRS complex morphology is the critical diagnostic point that implicates an AP as part of the tachycardia circuit. However, constant cycle length in this situation never excludes an AP as part of the tachycardia circuit. When a change in cycle length occurs it is typically stated that the AP is ipsilateral to the blocked bundle branch, yet there is an exception to this "rule." A patient could have marked prolongation of His–Purkinje conduction time with the bundle branch block complex, which shortens substantially with normalization of the QRS complex. In this situation the tachycardia cycle length might decrease because His–Purkinje conduction is part of the tachycardia cycle length. This would occur at any location of the AP. Regardless, this observation does implicate an AP in the tachycardia circuit.

Figure 4–33A



What is the likely mechanism of tachycardia on the left side of this figure?

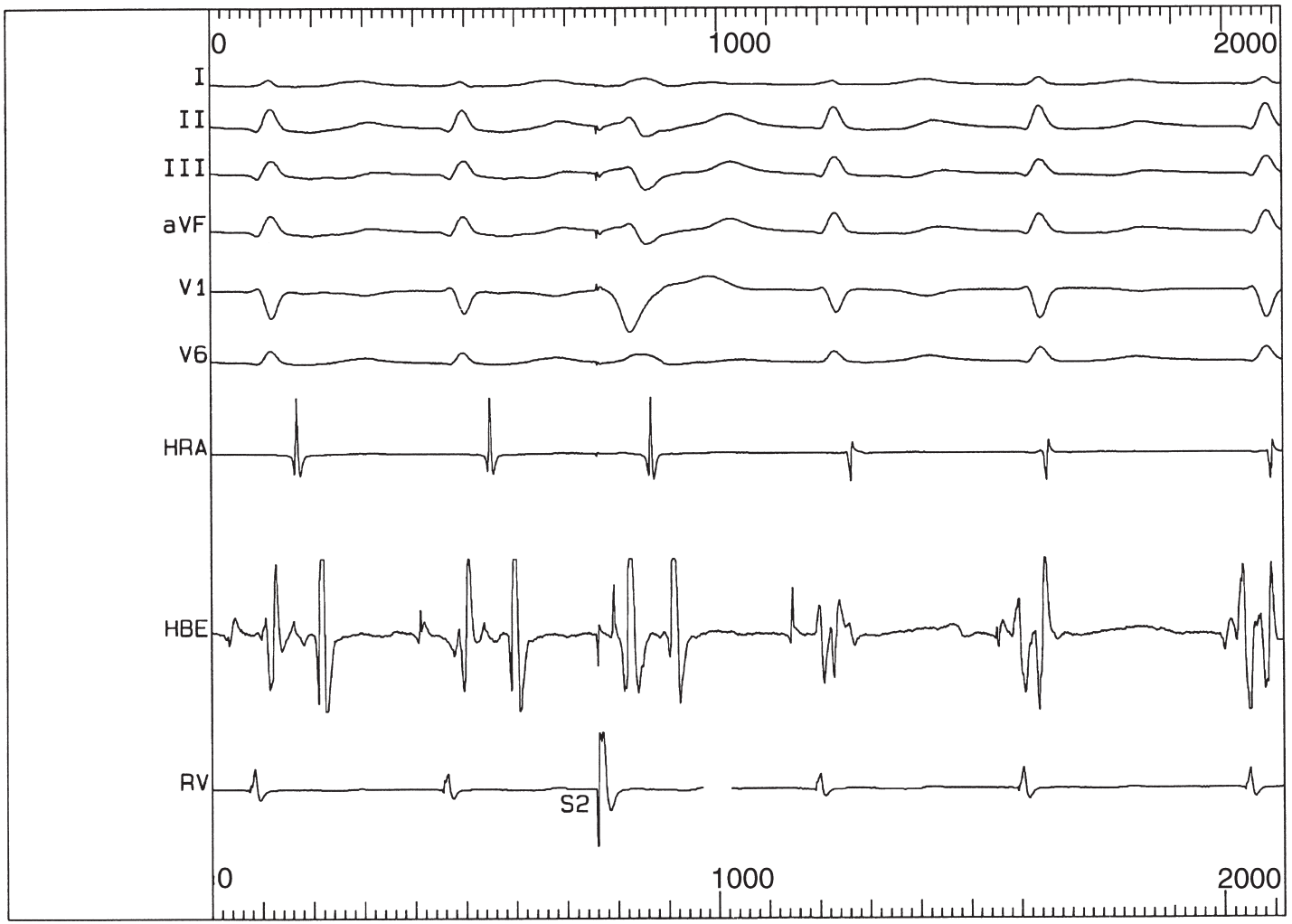
Figure 4–33B



Explanation:

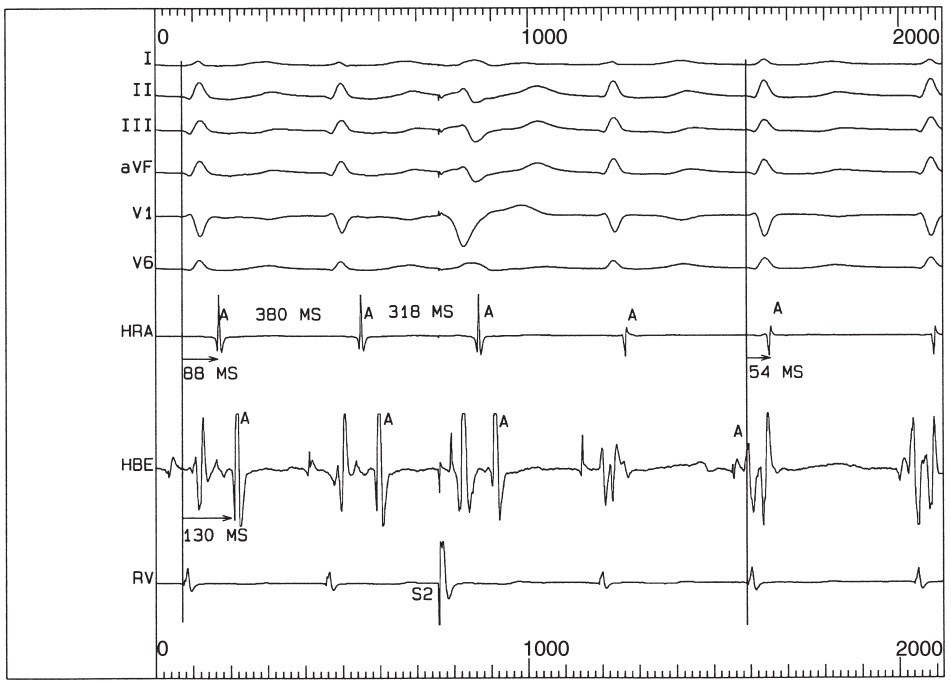
This patient had a more rapid PSVT, but at EP study another form of tachycardia was also induced, and was never sustained. Note that the atrial activation sequence during tachycardia (cycle length of 640 milliseconds) is nearly identical to that in sinus rhythm (cycle length of 888 milliseconds). Although only two intracardiac leads are present, comparison of the P waves on the surface ECG shows that they are nearly identical. This likely represents sinus node reentry, although one can never exclude an atrial tachycardia originating within a few millimeters of the sinus node. Nonsustained sinus node reentry is commonly initiated at EP study. In general, it is not an arrhythmia requiring treatment.

Figure 4–34A



This observation occurred during SVT after introduction of a PVC. Explain the finding.

Figure 4–34B

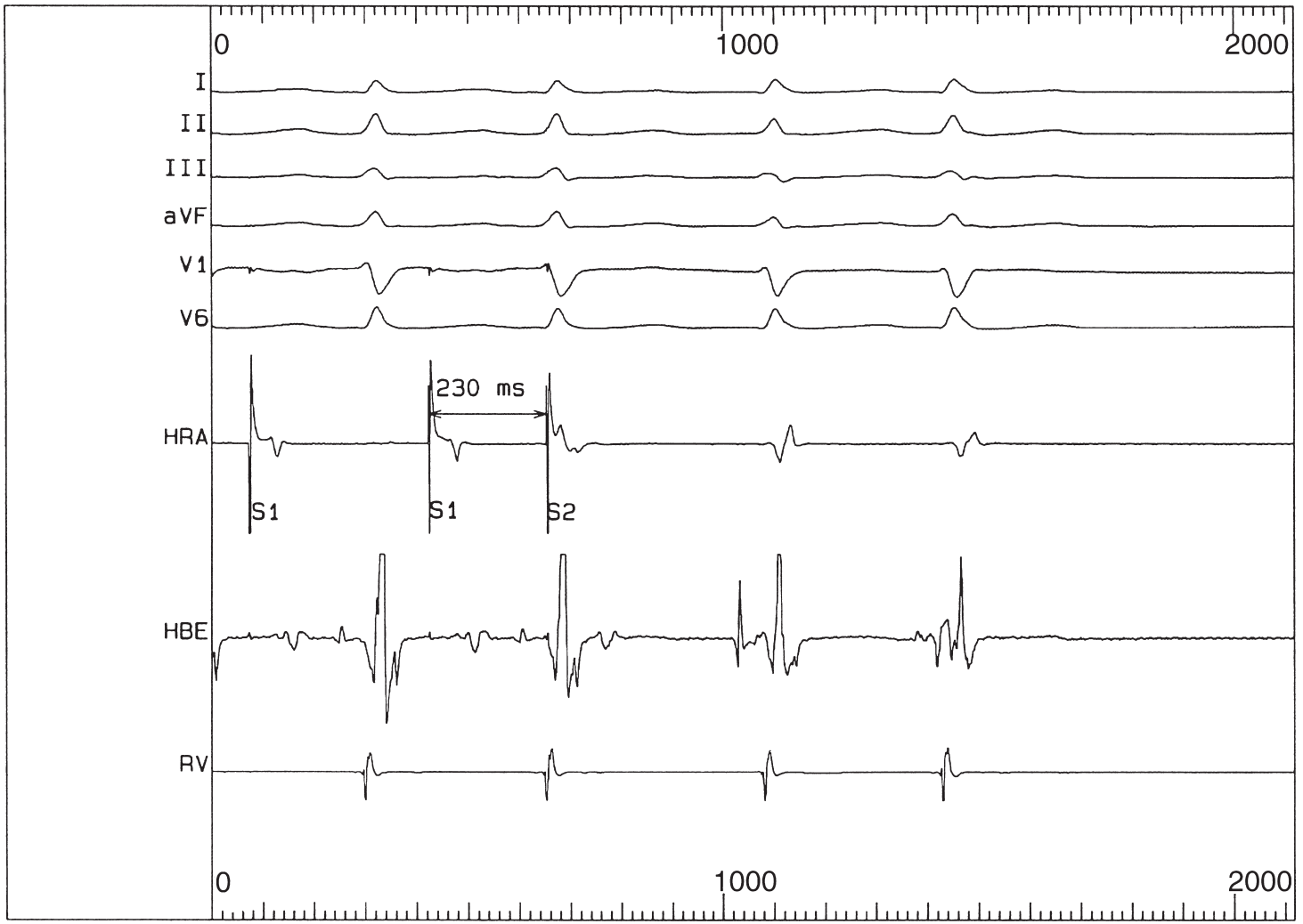


Explanation:

The PVC is introduced prior to activation of the His bundle electrogram, and does not change the timing of the HH interval. The ventricle to high right atrium and His bundle atrium intervals are 88 and 130 milliseconds, respectively. Prior to introduction of the PVC, there is no way to know whether the "VA" interval actually represents conduction from ventricle to atrium or if it is really AV conduction. In fact, there appears to be a high to low atrial activation sequence. One must remember that electrodes positioned in the high lateral right atrial area also record atrial activity from the lateral right atrium. Thus, if retrograde conduction occurs over a right lateral AP, activation on the high right atrial catheter can precede activation of the interatrial septum. This gives the illusion of a high to low atrial activation sequence.

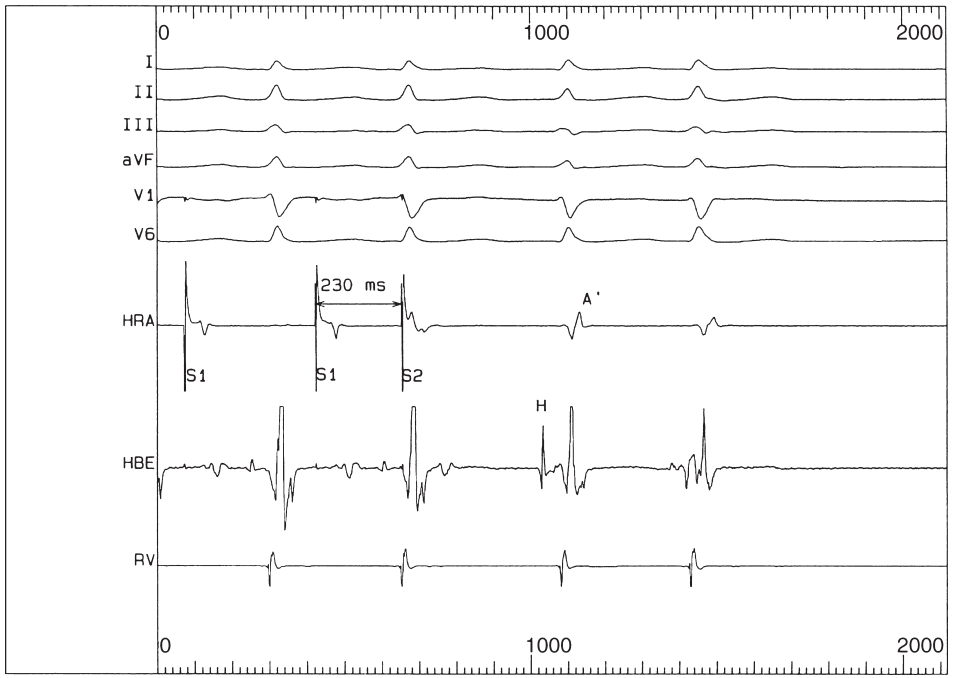
The PVC clearly preexcites the atrium and shortens the cycle length from 380 to 318 milliseconds without a change in the atrial activation sequence. Since the PVC was introduced at a time when the His bundle was refractory, the tachycardia mechanism most likely is AV reentry and the presumption is that a right lateral AP is present. Note that the QRS complex after the PVC is associated with a change in atrial activation sequence. The PVC "creates" a PAC (equivalent to atrial extrastimulus) and there is prolongation of the AH interval. The prolonged AH interval is followed by early atrial activation in the interatrial septum. The ventricle to high right atrium interval shortens to 54 milliseconds, but is preceded by atrial septal activation. This patient now has AV node reentry with a slow anterograde and fast retrograde conduction pattern. Both arrhythmias were sustained and required ablation.

Figure 4–35A



This patient has documented SVT. At EP study in the baseline state, the observation shown in the tracing was made. What is the likely mechanism of tachycardia?

Figure 4–35B



Explanation:

A premature atrial complex of 230 milliseconds conducts with a long AH interval and is followed by two subsequent atrial complexes. The VA interval is too short for AV reentry. Thus, the mechanism is either atrial tachycardia or AV node reentry. The echo complexes never terminated with conduction to the His bundle. This strongly suggests that the AV node is part of the tachycardia circuit, since termination of atrial tachycardia is not coupled to AV node conduction. A useful technique is to try to establish a premature atrial coupling interval at which there is intermittent conduction to the His bundle. This is noted in Figs. 4–35*B* and 4–35*C*. The same coupling interval repeated many times produced atrial echoes only when His bundle activation occurred, strongly suggesting that AV nodal conduction was necessary for the atrial echo, implicating the AV node as part of the circuit. In the presence of isoproterenol, sustained AV node reentry was initiated.

Figure 4–35C

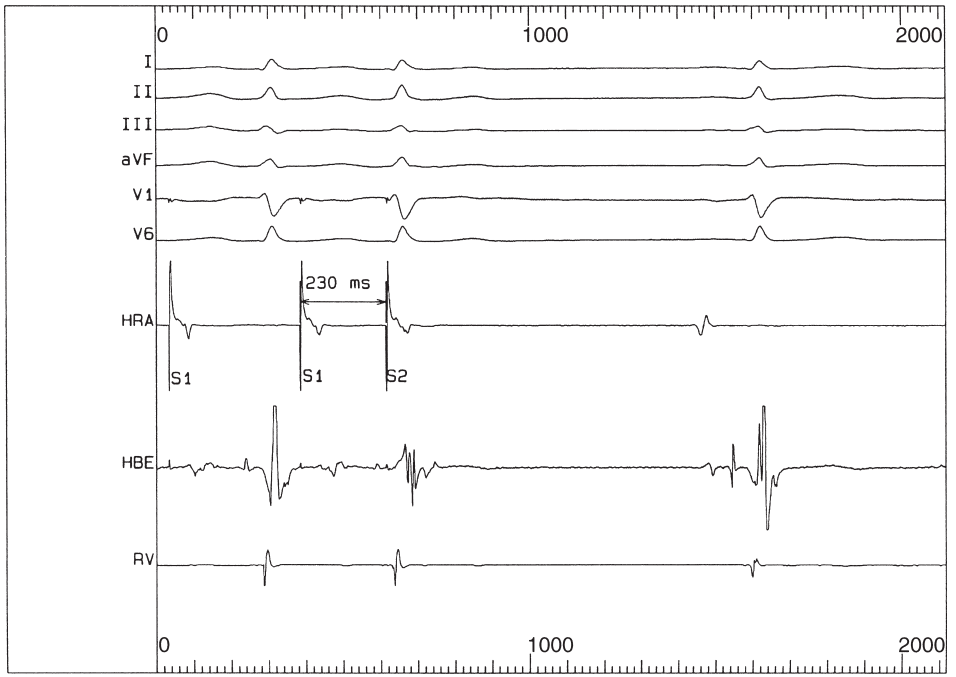

0
1000
2000
I
II
III
aVF
V1
V6
230 ms
HRA
S1
S1
S2
HBE
RV
0
1000
2000

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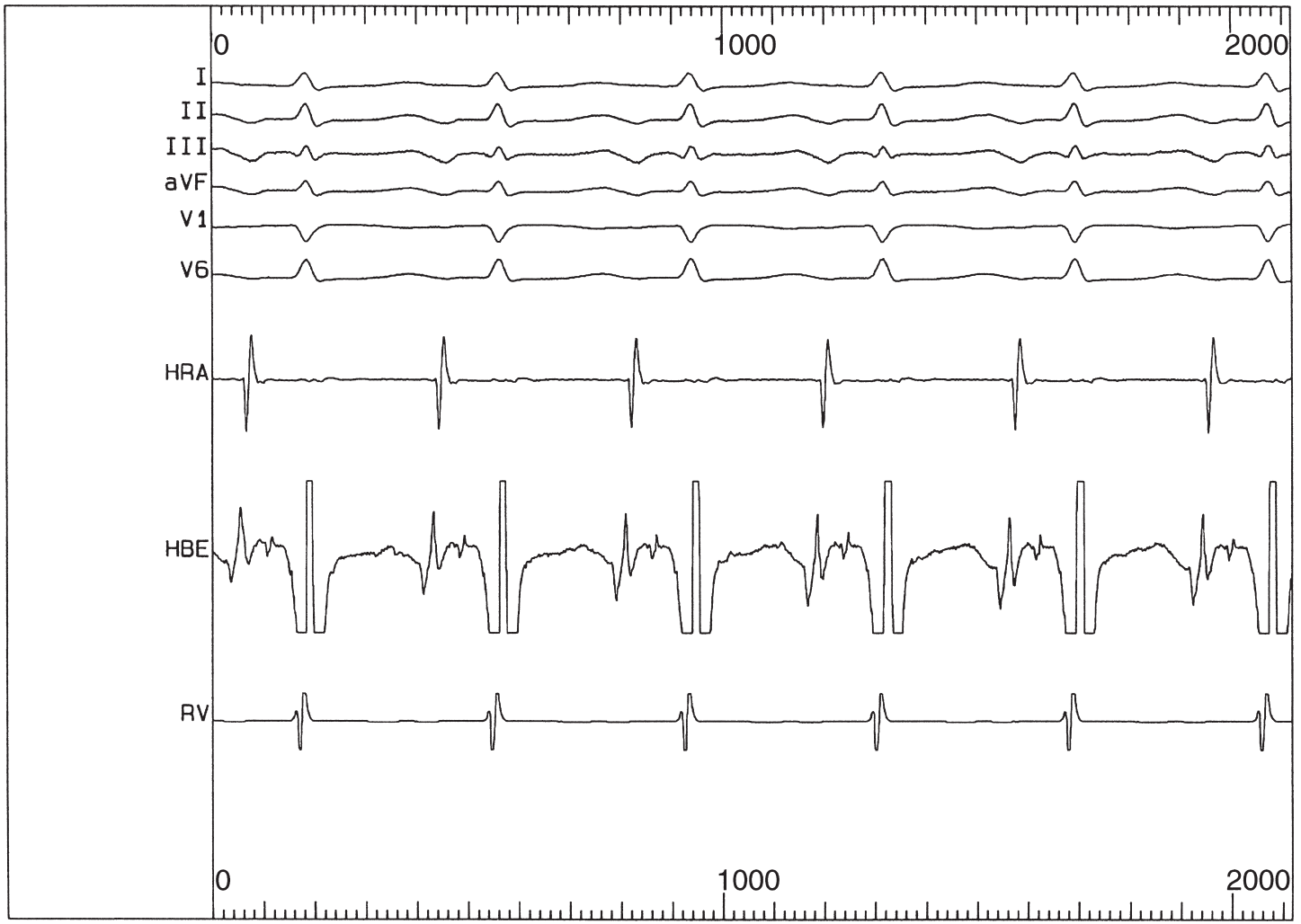
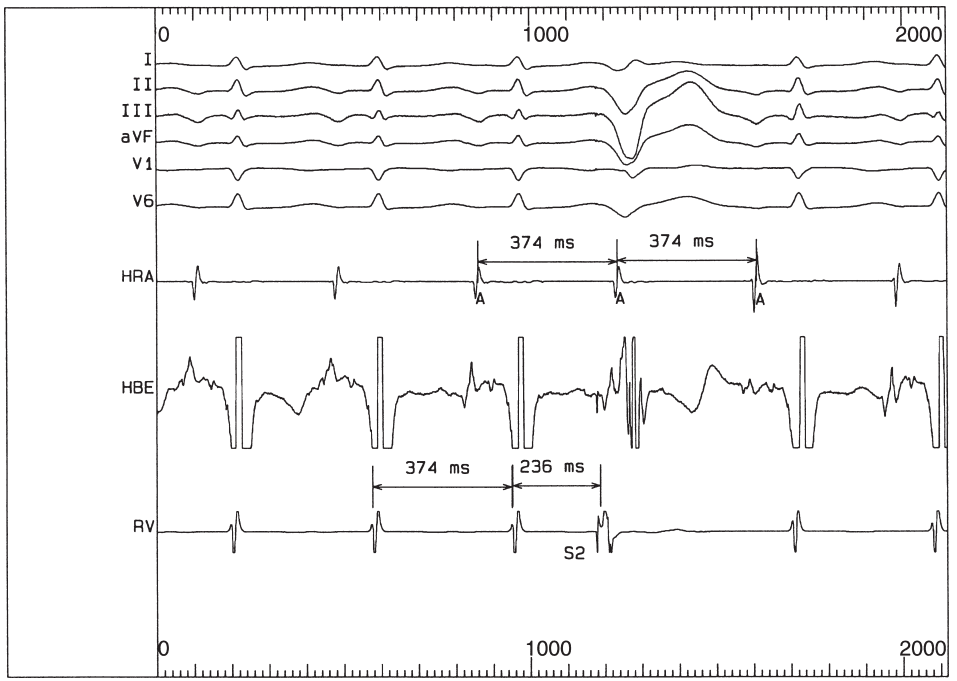
Figure 4–36A



This record was taken during EP study of a patient with a long RP tachycardia. What is the differential diagnosis, and what techniques could you use to confirm the diagnosis without additional catheter placement?

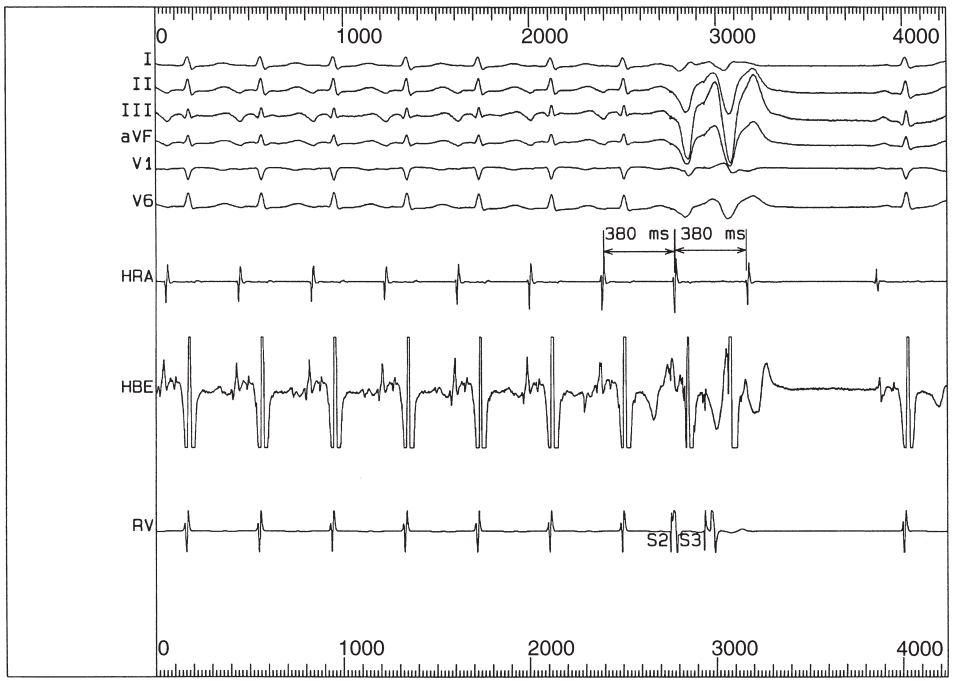
Figure 4–36B



Explanation:

The differential diagnosis in a long RP tachycardia includes atrial tachycardia, AV reentry using an AP with slow retrograde conduction characteristics, and AV node reentry with slow retrograde and fast anterograde AV nodal conduction. Figure 4–36*B* demonstrates introduction of a very close-coupled PVC during tachycardia. No preexcitation occurs and the preexcitation index is greater than 138 milliseconds, inconsistent with conduction over an AP. However, this does not absolutely rule out AV reentry. Lack of atrial preexcitation during programmed right ventricular stimulation near the coronary sinus os area makes a diagnosis of AV reentry even less likely. The likely differential is atrial tachycardia versus AV node reentry. Figure 4–36*C* shows termination of tachycardia with two closely coupled PVCs without any preceding change in the tachycardia cycle length, and without any conduction of the PVCs to the atrium. This was a reproducible finding and rules out atrial tachycardia. The resultant diagnosis is AV node reentry. CS catheterization was performed during this study and confirmed concentric retrograde atrial activation.

Figure 4–36C


0
1000
2000
3000
4000
I
II
III
aVF
V1
V6
380 ms
380 ms
HRA
HBE
RV
S2
S3
0
1000
2000
3000
4000

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Chapter 5

Wide QRS Complex Tachycardia

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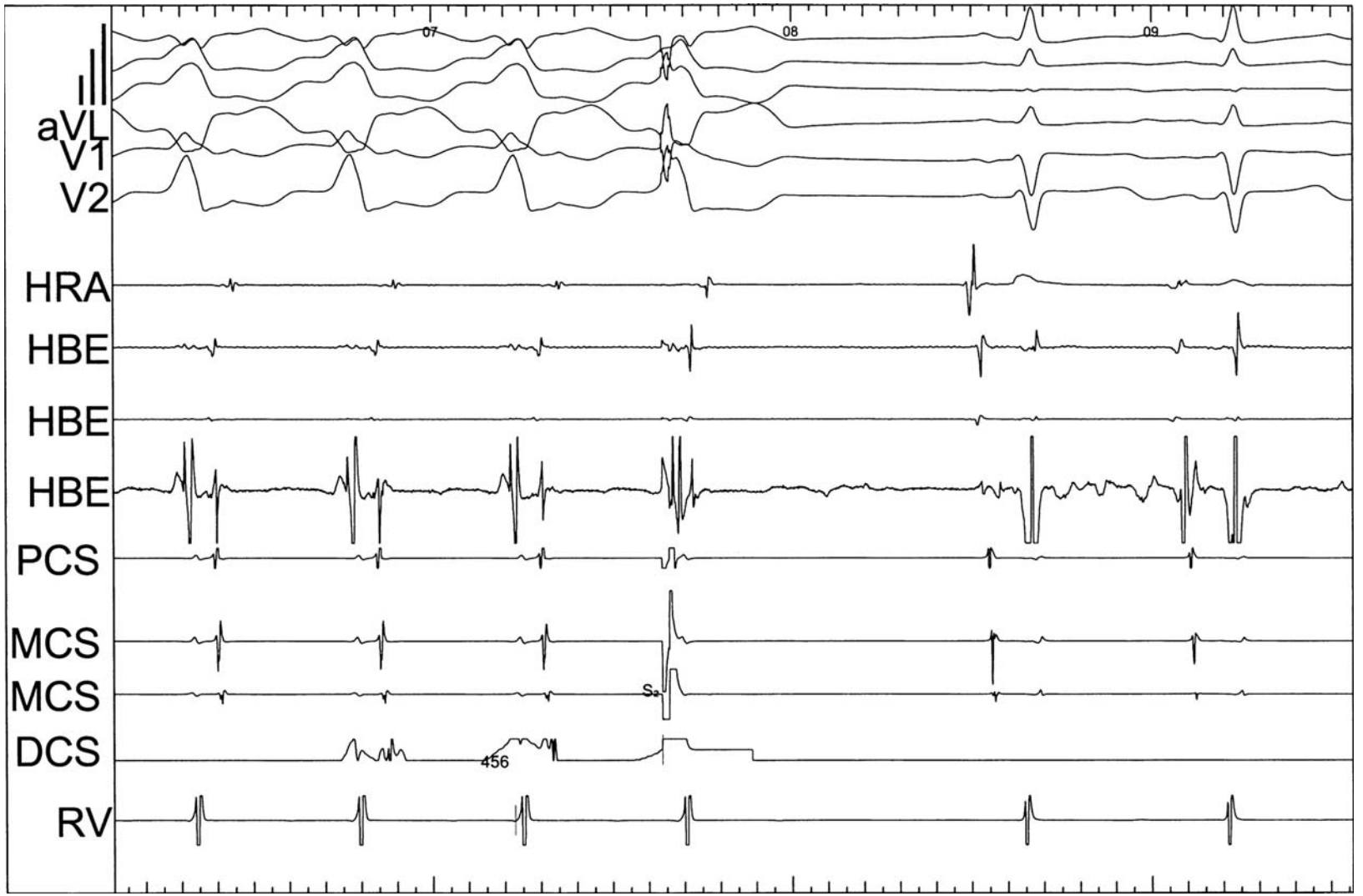
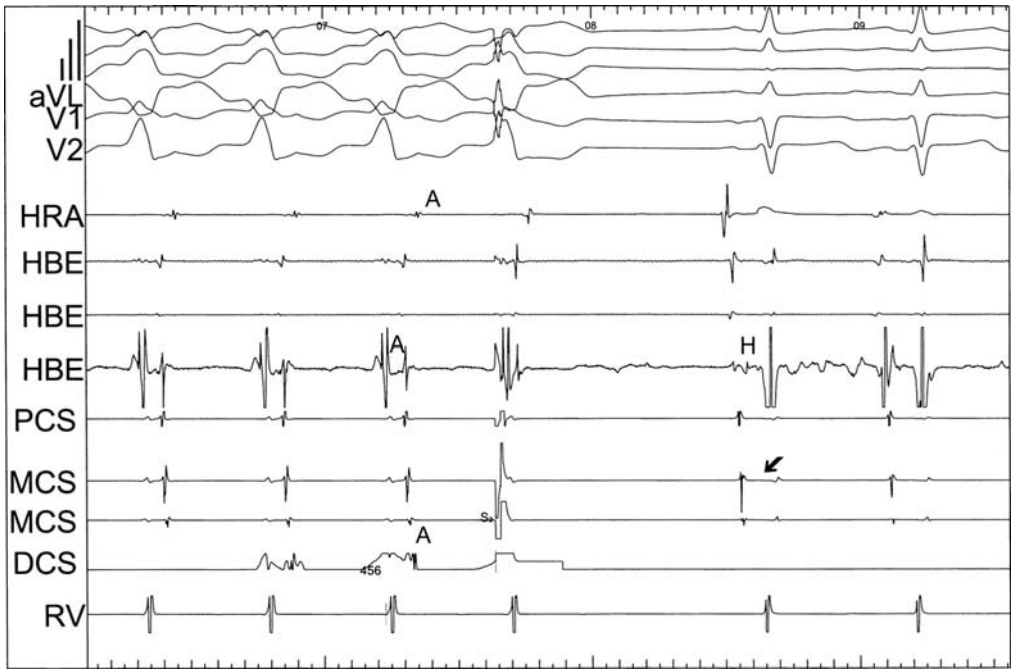
Figure 5–1A



A 33-year-old man with a history of recurrent palpitations and documented wide QRS tachycardia undergoes electrophysiologic evaluation. His 12-lead ECG is normal. The following was observed (figure). What is the most likely mechanism of tachycardia?

Figure 5–1B



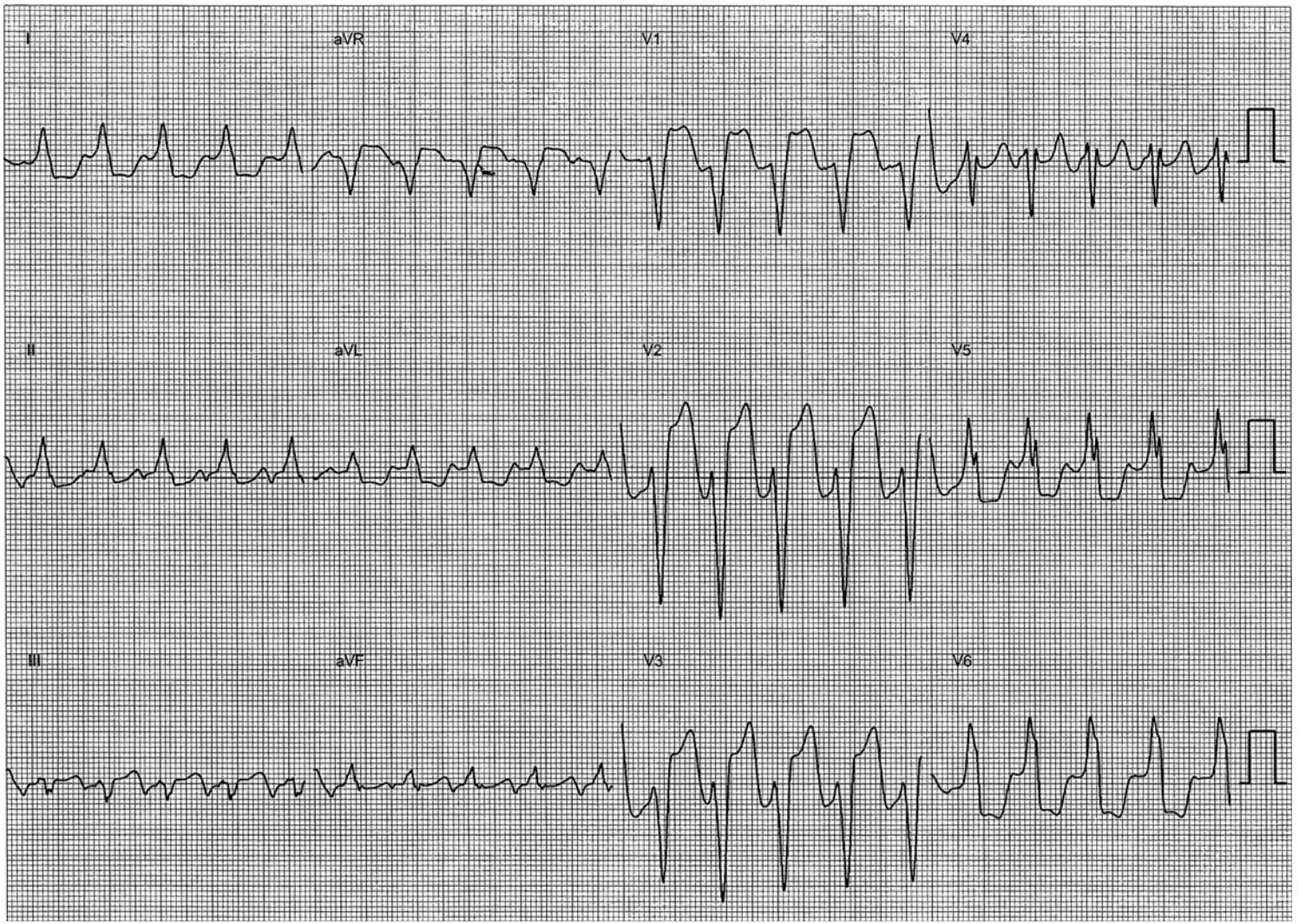
Explanation:

The initial four QRS complexes have an atypical right bundle branch block morphology suggesting either ventricular tachycardia (VT) or a preexcited tachycardia. There is 1:1 AV relationship with the earliest atrial electrogram in the septum. In such a situation, the differential diagnosis includes VT with 1:1 VA conduction, atrial tachycardia or AV node reentry with anterograde conduction over an accessory pathway (AP), and antidromic tachycardia with anterograde conduction over an AP and retrograde conduction over either a second septal AP or the normal VA conduction system.

An atrial stimulus (S2) introduced during tachycardia at the MCS electrode site terminates tachycardia without conduction to the ventricle. This eliminates VT as a diagnosis. Termination with a relatively late-coupled PAC also favors a macroreentrant tachycardia with close access of the PAC to the excitable gap, that is, antidromic reentry in this case. Additional pacing maneuvers during the study proved the mechanism to be antidromic tachycardia with retrograde conduction over the normal VA conduction system.

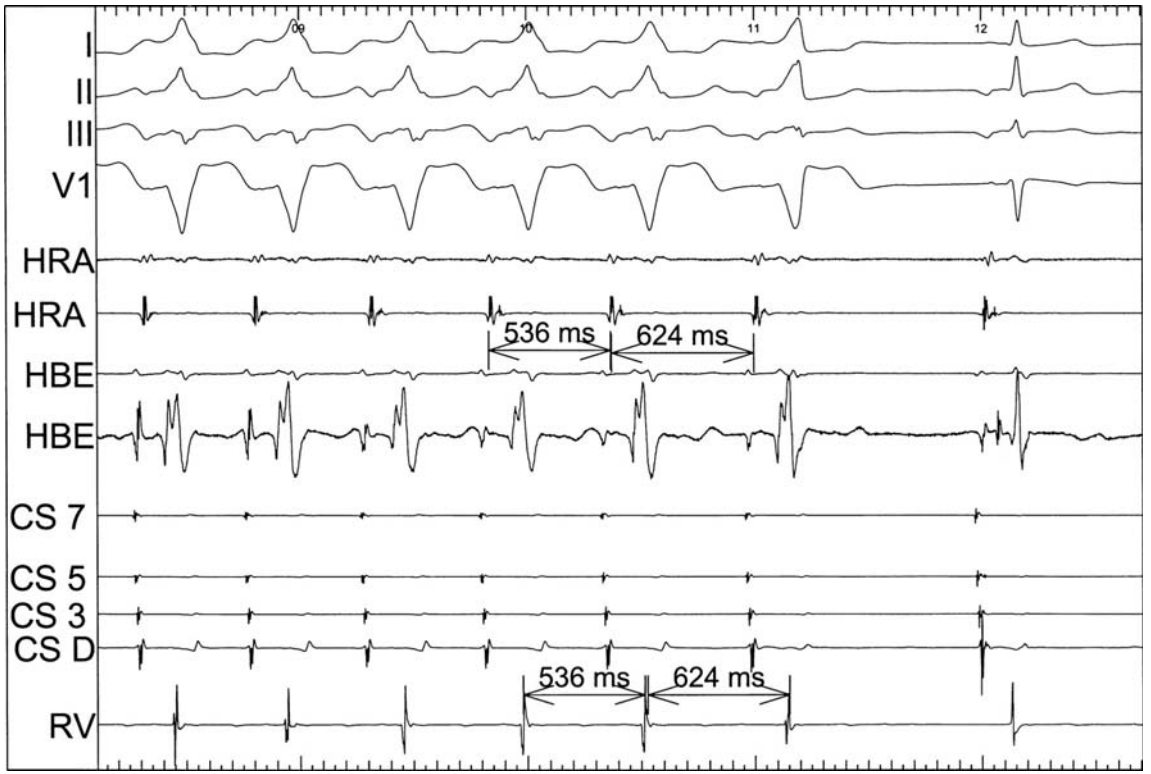
The last two QRS complexes show no evidence of anterograde AP conduction. In fact, there is not even local evidence of preexcitation on the coronary sinus leads as demonstrated by a long conduction time between the local A and V (*arrow*). In a patient with antidromic reciprocating tachycardia (ART) there is typically some evidence of anterograde AP activation during sinus rhythm. This was a very unusual AP that only became manifest with multiple premature atrial extrastimuli that resulted in block over the AV node and a very long conduction time over the AP.

Figure 5–2A



A 46-year-old man with a history of recurrent palpitations and documented wide complex QRS tachycardia undergoes electrophysiologic study. His 12-lead ECG during tachycardia is shown in the figure. What are the diagnostic possibilities?

Figure 5–2B

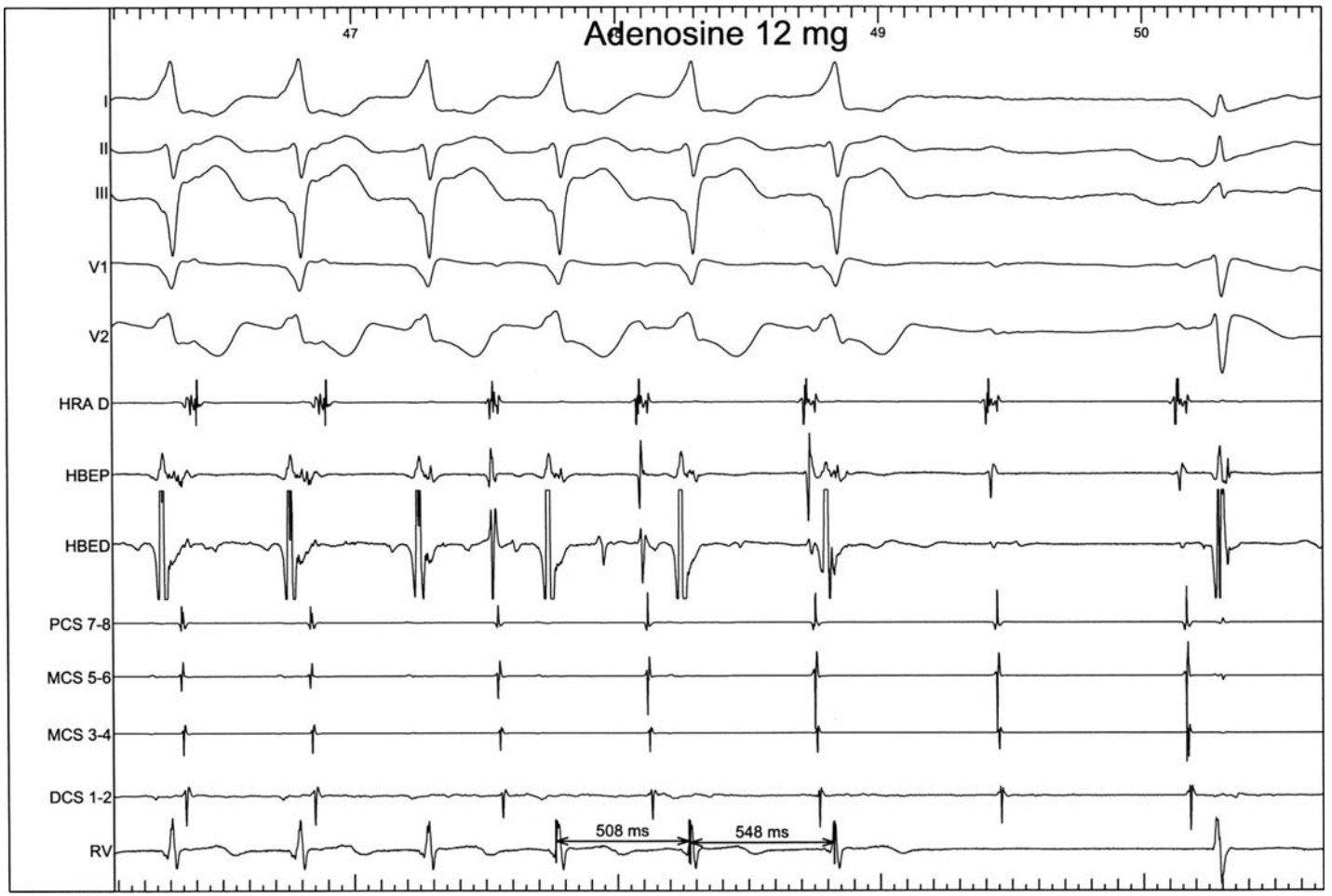


Explanation:

The tachycardia exhibits an atypical left bundle branch block morphology that is less likely aberrancy and more consistent with either VT or preexcited tachycardia. If it is a preexcited tachycardia, it is conducting anterogradely over a right free wall AP. There appears to be a negative P wave before each QRS complex in ECG leads II, III, and aVF. If this is VT, 1:1 retrograde conduction is most likely over a slow AV nodal pathway. A preexcited tachycardia could be atrial tachycardia or AV node reentry with bystander conduction over the AP or ART.

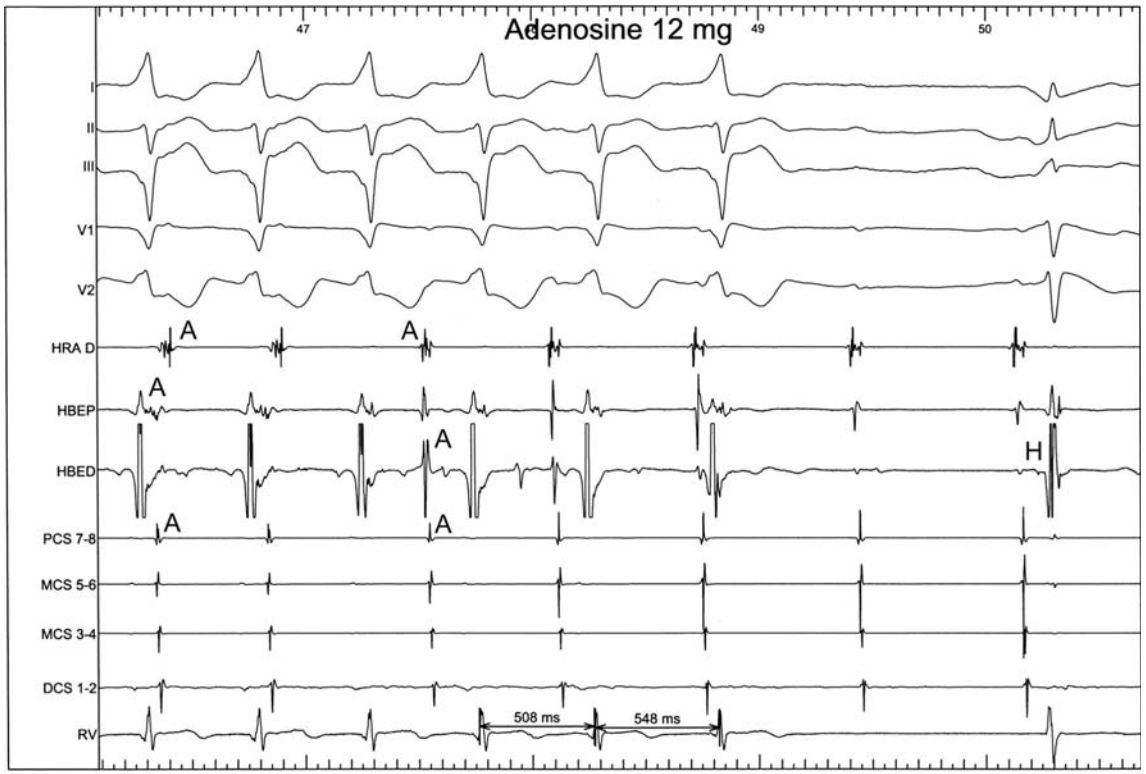
The next figure demonstrates termination of tachycardia. Note the increase in the atrial cycle length from 536 to 624 milliseconds is followed by a similar increase in the ventricular cycle length, or, said another way, the change in A–A interval drives the change in V–V interval. This eliminates VT as the diagnosis. Initial atrial activation is in the septum, or concentric, which does not allow differentiation of septal atrial tachycardia, AV node reentry, and ART. Further maneuvers during the study confirmed the diagnosis of ART with anterograde conduction over a right free wall AP, and retrograde conduction over a slow AV node pathway. The latter was the cause of the long RP interval during tachycardia.

Figure 5–3A



A 41-year-old woman with a history of wide QRS complex tachycardia terminating with adenosine undergoes electrophysiologic evaluation. Her ventricular function is normal. Sustained tachycardia initiated at electrophysiologic study was terminated with 12 mg of IV adenosine (figure). What is the most likely diagnosis?

Figure 5–3B

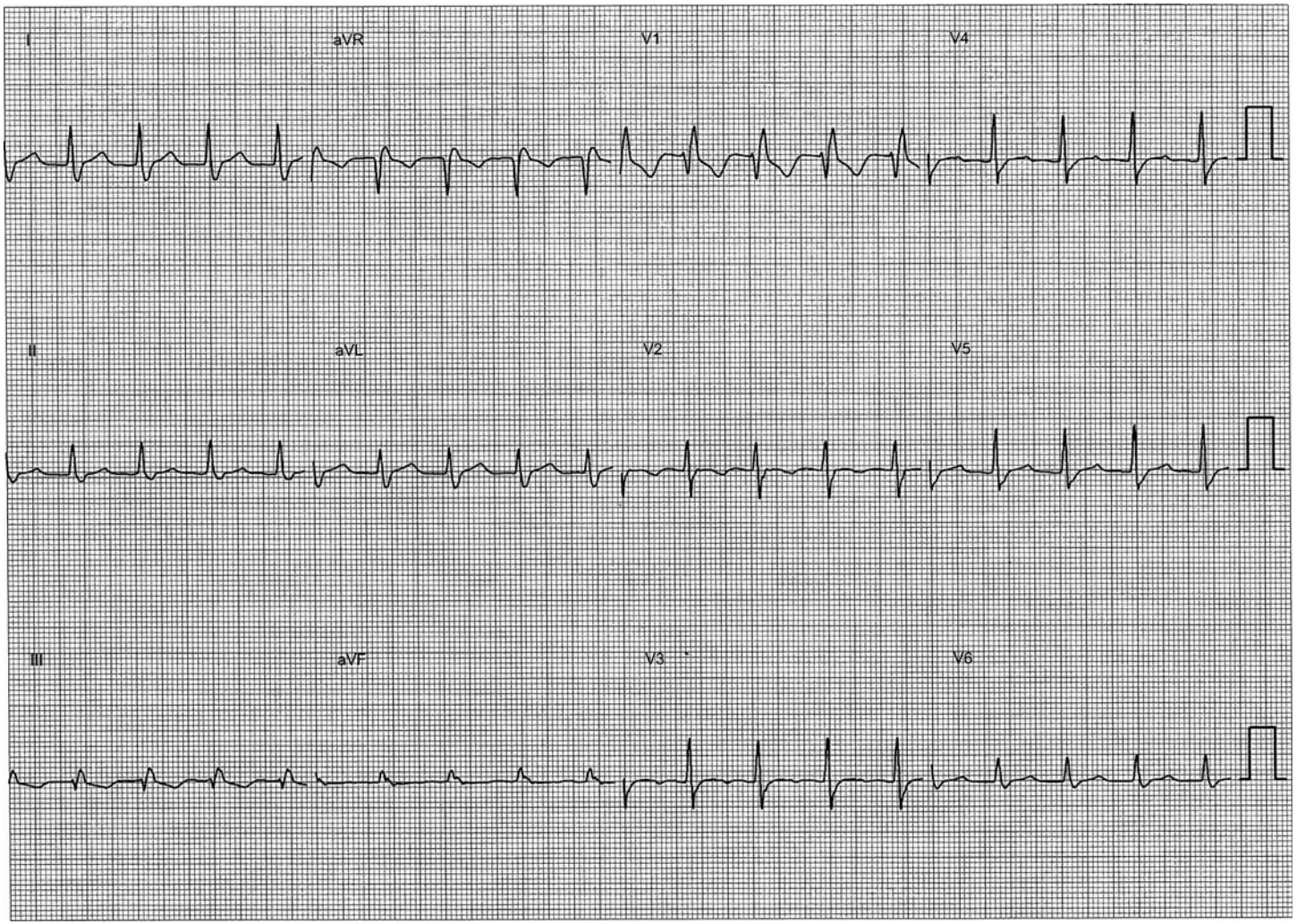


Explanation:

The atypical left bundle branch block pattern during tachycardia is consistent with either preexcitation or VT. The first two QRS complexes demonstrate retrograde VA conduction with a concentric atrial activation sequence. The third QRS complex has VA block and sinus rhythm persists for the rest of the tracing as noted by the change in atrial activation sequence. Regardless, the tachycardia persists for a few more beats with slowing from 508 to 548 milliseconds prior to termination. After termination there is a nonconducted sinus complex followed by conduction over the normal AV conduction system. Continuation of tachycardia with VA dissociation eliminates an AP-mediated tachycardia and this is clearly VT.

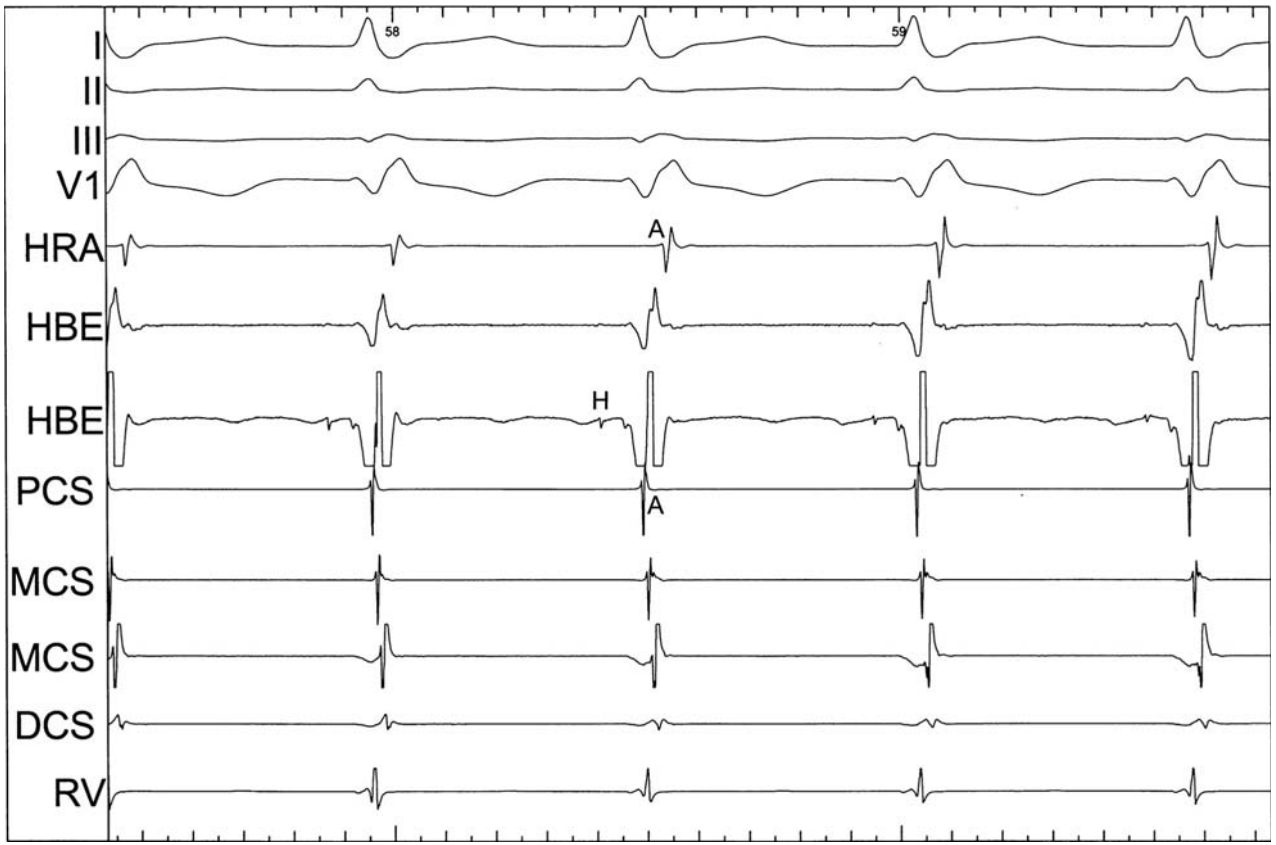
Why did VT terminate with adenosine? Some varieties of VT are sensitive to adenosine, especially those originating in the right and left ventricular outflow tracts. This patient obviously does not have an outflow tract VT. Some epicardial VTs and those around the epicardial veins can also terminate with adenosine. Initial mapping demonstrated early electrograms during VT near the posteroseptal area and the middle coronary vein. The exact site was never determined because the patient refused ablation.

Figure 5–4A



A 68-year-old woman undergoes electrophysiologic study because of recurrent, very symptomatic tachycardia. A 12-lead ECG during tachycardia is shown (figure). What is the differential diagnosis?

Figure 5–4B

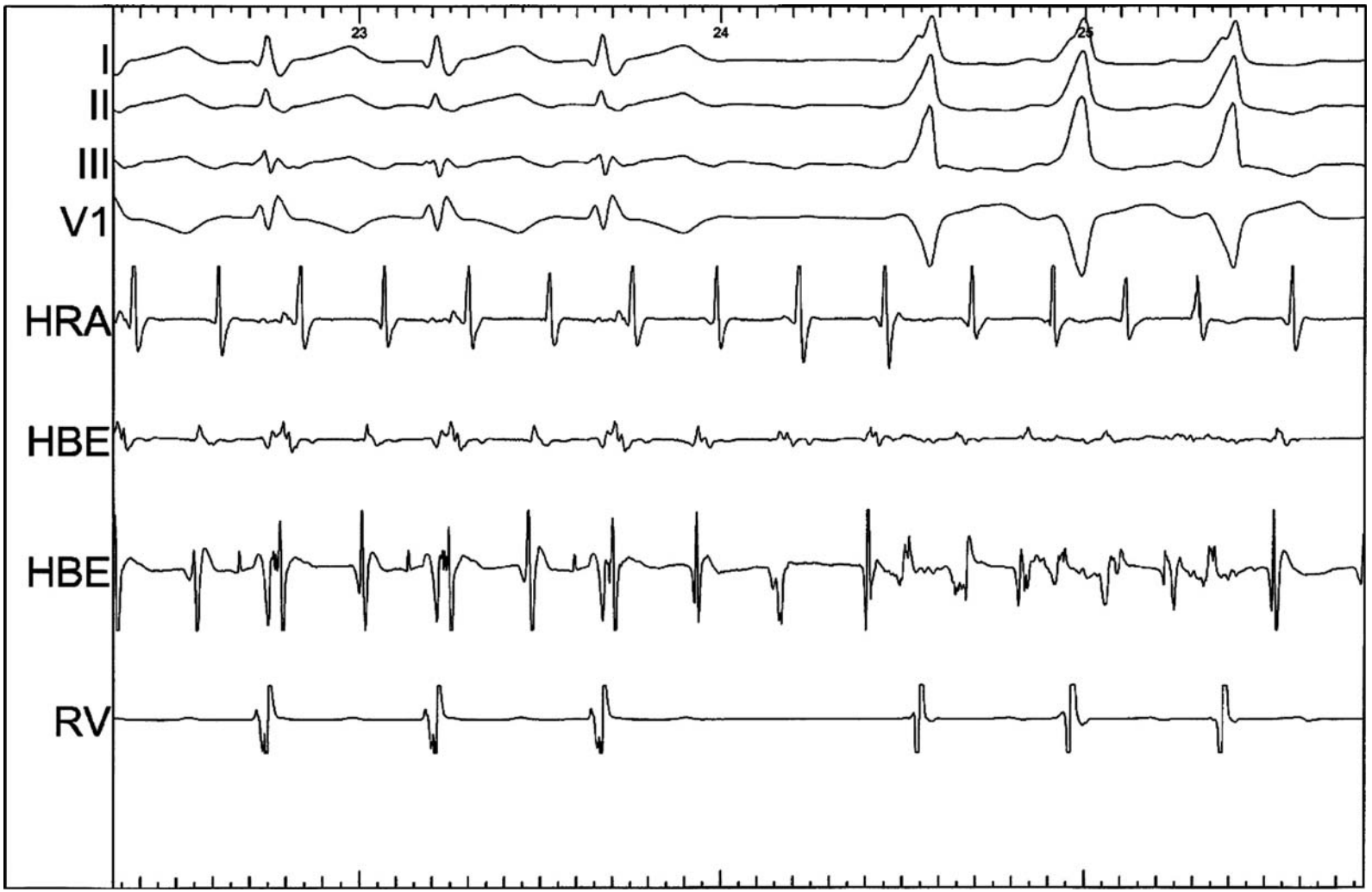


Explanation:

A typical right bundle branch block is present during tachycardia. The tachycardia is regular and no clear P waves are identified. In essence, this could be any tachycardia that activates the ventricles with conduction over the left bundle branch. P waves are typically seen during atrial tachycardia, making this diagnosis much less likely. While a P wave is often discernible in the early ST segment in patients with AV reentry, this is not always the case, especially in the presence of a bundle branch block. Typical AV node reentry is the most likely diagnosis but junctional tachycardia is also a possibility. A fascicular VT originating in the proximal left bundle branch cannot be excluded but is highly unlikely.

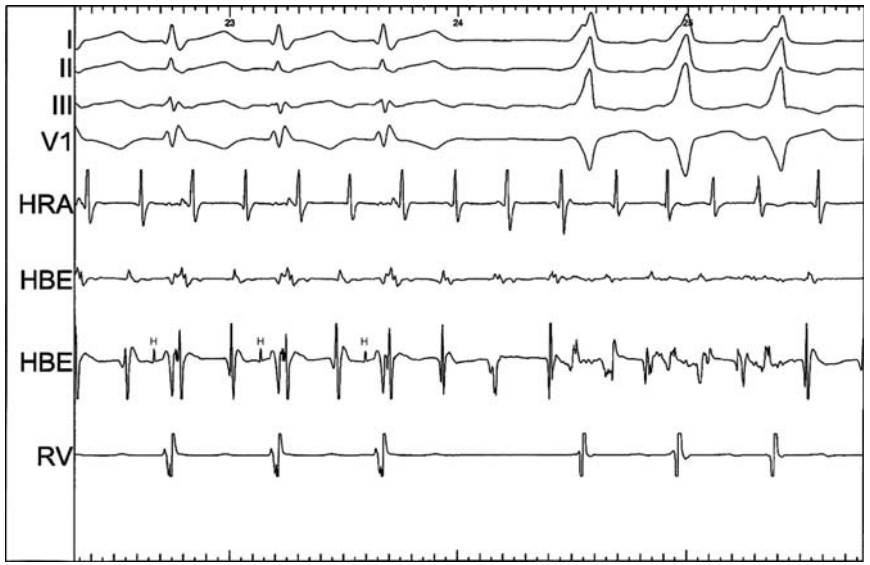
The next figure demonstrates a short VA interval and a normal HV interval during tachycardia, which excludes AV reentry and VT. The diagnosis of AV node reentry was confirmed with pacing maneuvers and the patient underwent successful ablation.

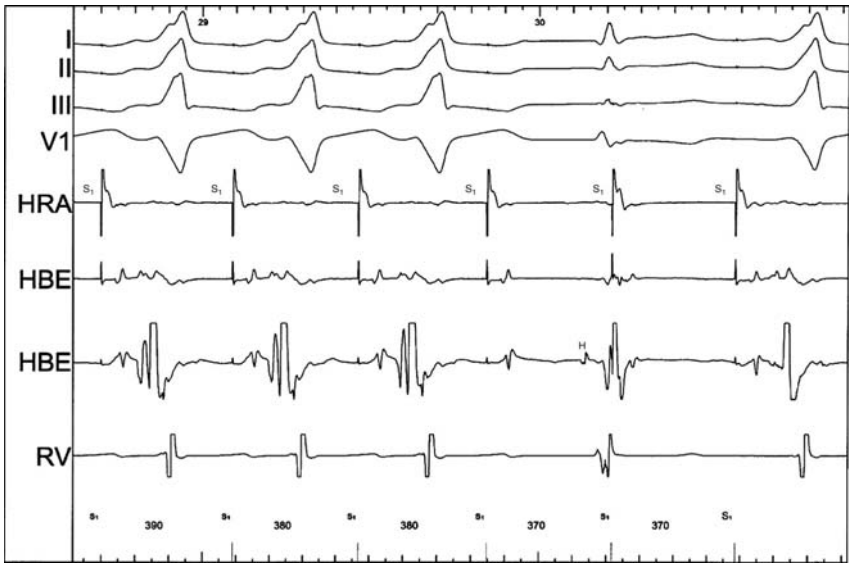
Figure 5–5A



This tracing was obtained during electrophysiology study in a patient with a history of palpitations. What is the mechanism of the wide QRS complexes on the left and right sides of this figure?

Figures 5–5B and 5–5C





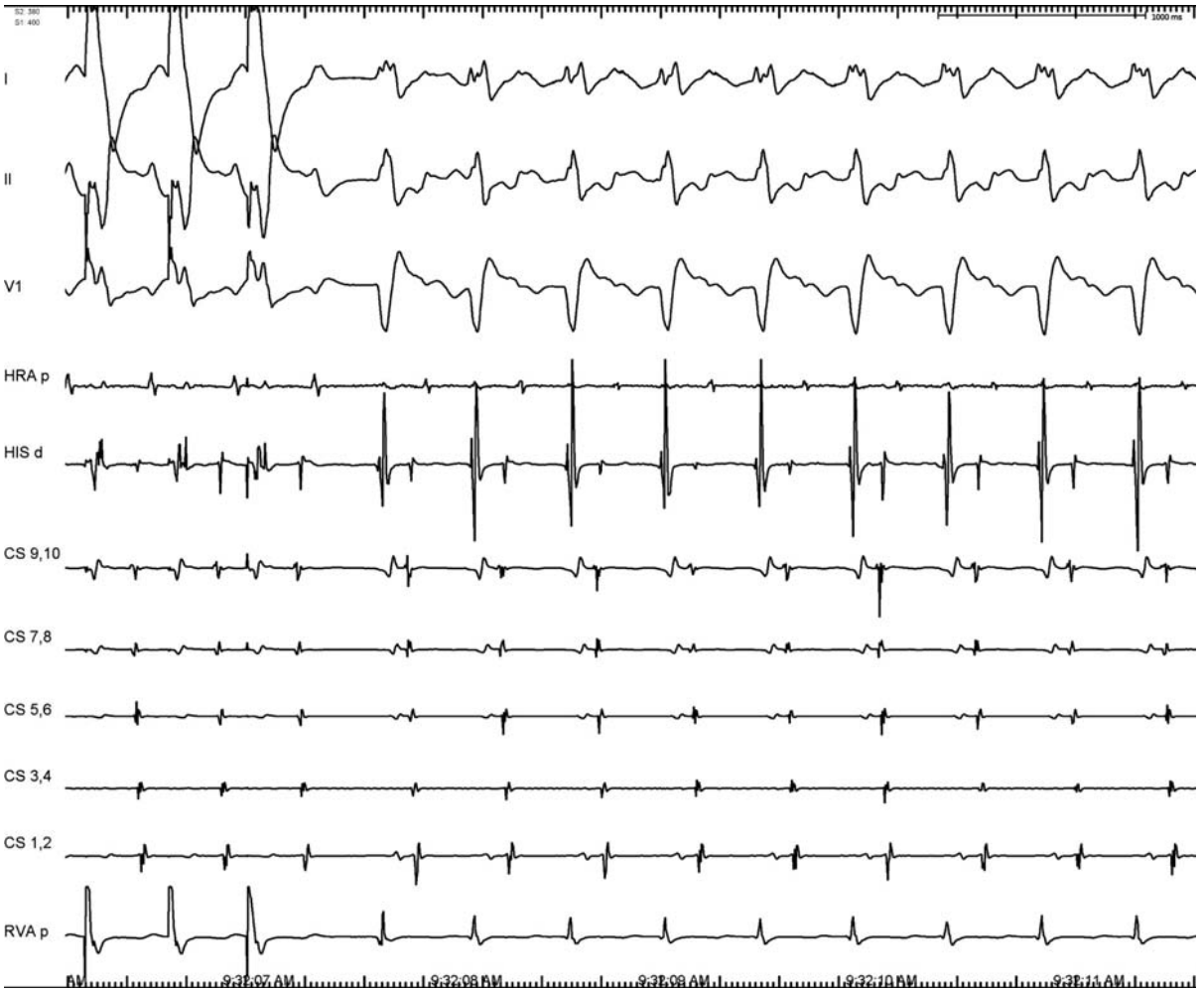
Explanation:

Atrial fibrillation occurred during catheter manipulation at the start of the study. The high right atrial (HRA) tracing demonstrates a more organized pattern that is not infrequently seen in the right atrium during atrial fibrillation. The three QRS complexes on the left have a typical right bundle branch block morphology strongly favoring aberrancy. Ventricular activation during these cycles occurs near the onset of QRS at the HBE site (i.e., near the base of the heart), also consistent with aberrancy. After a pause, the three QRS complexes on the right have atypical left bundle branch block morphology consistent with VT or preexcitation.

The second figure highlights the normal HV interval in the first three complexes confirming aberrancy. However, the His is not visible in the last three beats and the mechanism remains unknown.

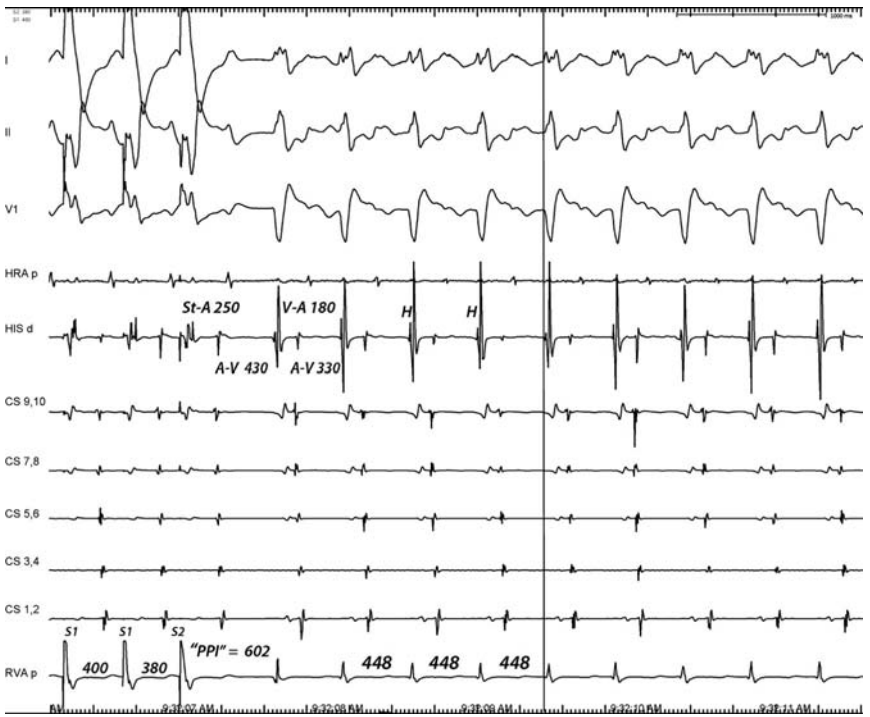
Tachycardia spontaneously terminated and the rest of the electrophysiology study was performed. The third figure shows atrial pacing (S1). The first three complexes conduct with the same morphology as noted on the right-hand portion of the initial figure and the His is not visible. This eliminates VT and establishes that conduction is over a left free wall AP. Note also in this figure that the fourth paced atrial complex blocks over the AP and conducts over the normal system with a normal HV interval. AV reentry was induced later in the study.

Figure 5–6A



The patient is a 24-year-old man with no heart disease. He has both wide and narrow QRS tachycardia clinically. The following tachycardia (Fig. 5–6*A*) was induced during ventricular extrastimulus testing. Does the tracing provide enough data to determine the mechanism?

Figure 5–6B



Explanation:

This complex tracing is best dealt with by breaking it up into components starting with a part that is manageable and that can form the basis for *framing* the problem.

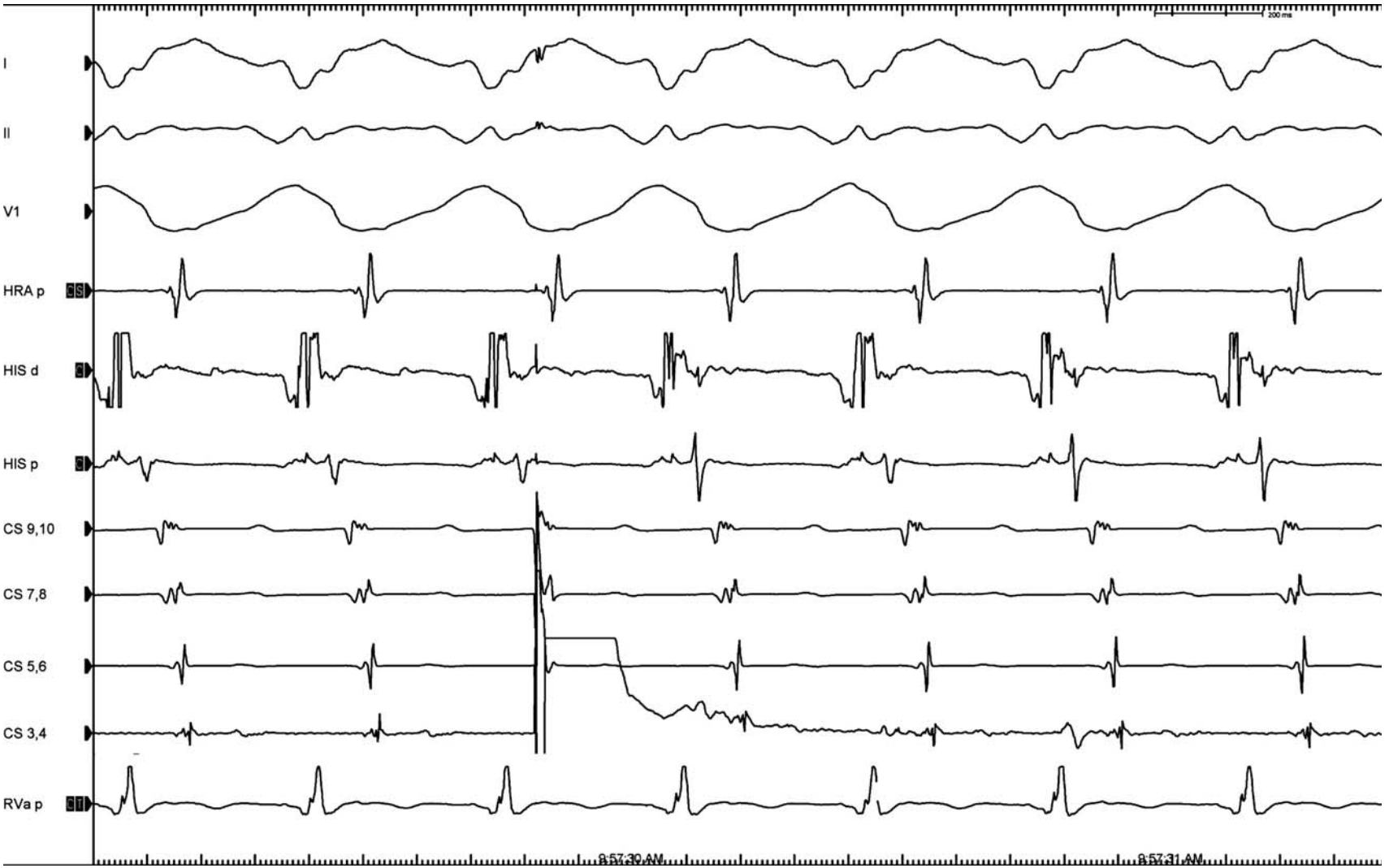
The right part of the tracing shows a wide QRS tachycardia with a 1:1 AV relationship with a septal pattern of atrial activation. A rapid His deflection is evident right at the onset of the V electrogram at the HBE site. *With this HV relationship, the tachycardia can only be VT or preexcited tachycardia.* The RV apical EGM is very near the onset of the QRS, suggesting conduction over an *atriofascicular AP*. Note that the His recorded near the onset of the QRS in such a scenario is a *retrograde* His resulting from ventricular insertion of a decremental AP (i.e., the atriofascicular) into or near the RBB terminus. If we discount VT with the knowledge that atrial pacing reproduces this QRS morphology with a long AV interval, we can make conclusions about the mechanism of this preexcited tachycardia.

We then analyze the onset of tachycardia. The S2 captures the V and conducts to the A, the latter having a septal activation pattern identical to the tachycardia. The first QRS of tachycardia onsets 430 milliseconds after the A measured at the His site.

In essence, *we can use the VES starting tachycardia in a fashion analogous to using ventricular entrainment from the RV apical region*.

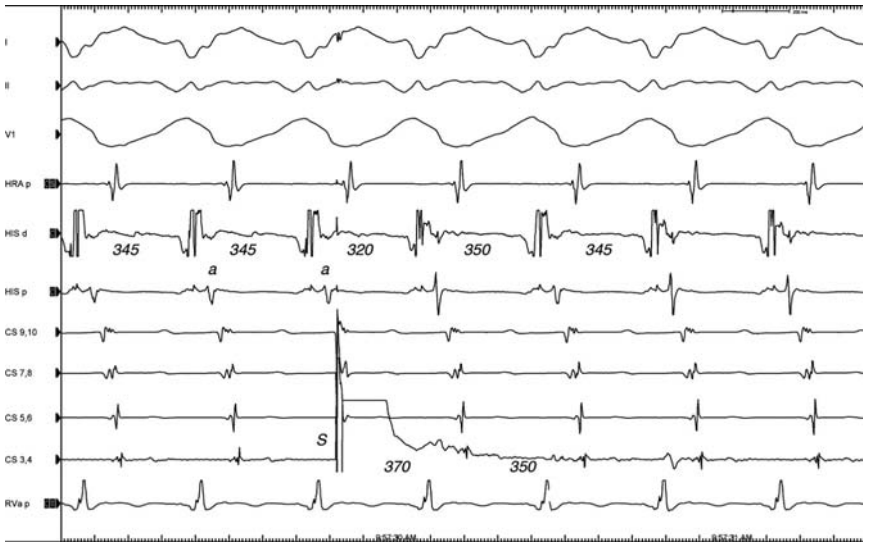
The first V of tachycardia at the pacing site is seen 602 milliseconds after the extrastimulus. We subtract 100 milliseconds from this to correct for the AV delay after the extrastimulus and we get a "corrected postpacing interval (PPI)" of 54 milliseconds (602 minus 448 minus 100). Thus, the "PPI" is "in." Similarly, the St-A of 250 milliseconds minus the V-A of 180 of 70 milliseconds also supports the RV apical pacing site being "in" the circuit and supporting the diagnosis of antidromic reentry with anterograde conduction over an atriofascicular AP and returning via the normal AV conduction system. The results of "conventional" entrainment from the RV apex were identical to this. Note that we have not ruled out the very unlikely possibility of retrograde conduction over another septal AP.

Figure 5–7A



Can a definitive tachycardia diagnosis be established in this young man with no heart disease on the basis of this tracing?

Figure 5–7B



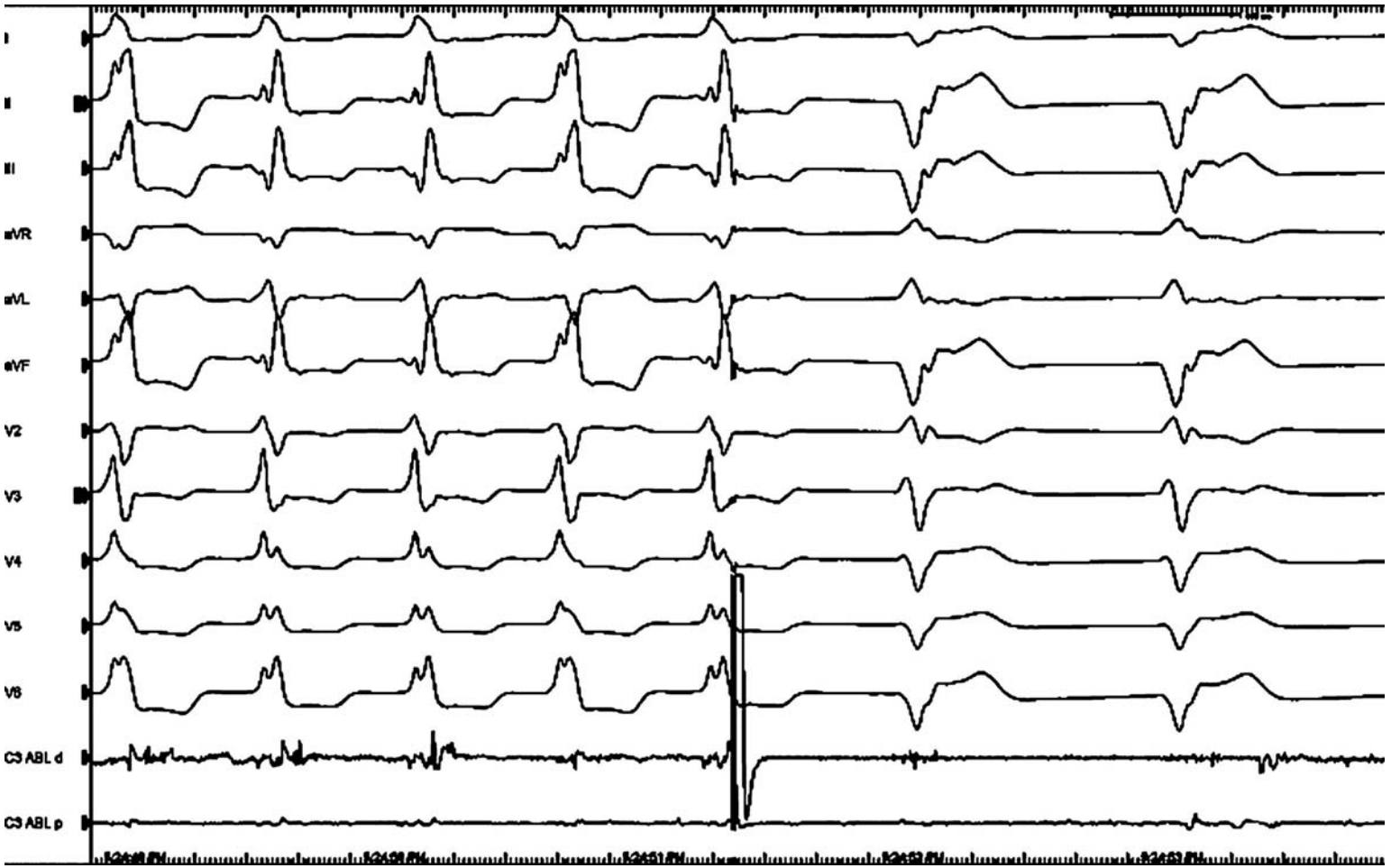
Explanation:

The tracing shows a wide QRS tachycardia with a 1:1 AV relationship. The atrial activation is central with early activation at the His bundle electrogram. The QRS morphology is quite atypical for a bundle branch block pattern and there is no His deflection visible at the His channel where one was clearly visible in sinus rhythm. This leaves us with only two tenable diagnoses, namely, VT and preexcited tachycardia. A PAC is programmed into the cardiac cycle at the distal coronary sinus (S).

The relatively late-coupled PAC introduced at a time when the septal atrial electrogram has been already activated resets the tachycardia without a change in the QRS morphology, that is, advances the next QRS (345–320 milliseconds). This means that the PAC has excellent access to the circuit and makes a diagnosis of VT untenable. The A following the advanced QRS is also advanced and the VA relationship remains essentially the same. Since the preceding septal activation was not altered, advancement of this site cannot be caused by an atrial tachycardia. That is, the PAC results in fusion with "reset" of the subsequent A. In fact, the "PPI" at the distal CS pacing site is only a few milliseconds greater than the tachycardia cycle length (370 milliseconds vs. 350 milliseconds). That is, the distal CS region is "in" the circuit. This eliminates atrial tachycardia as the mechanism. Since the late PAC becomes essentially a late PVC, the fact that the last PVC preexcites the next atrial complex eliminates AVN reentry. Hence, we have a macroreentrant atrioventricular tachycardia with a left lateral AP as the anterograde limb and a retrograde limb that is either the normal VA conduction system (i.e., true antidromic atrioventricular reentry) or a second AP. This in fact was the former, a much more common tachycardia mechanism than the latter.

How would one prove that the retrograde limb is the normal AV conduction system? One might entrain from both the RV base and the RV apex and show that the RV apex is closer to the circuit (i.e., shorter PPI). This is because the RV base is closer to a potential posteroseptal accessory if that were part of the circuit. Alternately, one might just ablate the obvious AP first, which would make it much easier to discern the presence or absence of a septal AP. In fact, in our experience it is relatively rare for the retrograde limb to be a second AP.

Figure 5–8

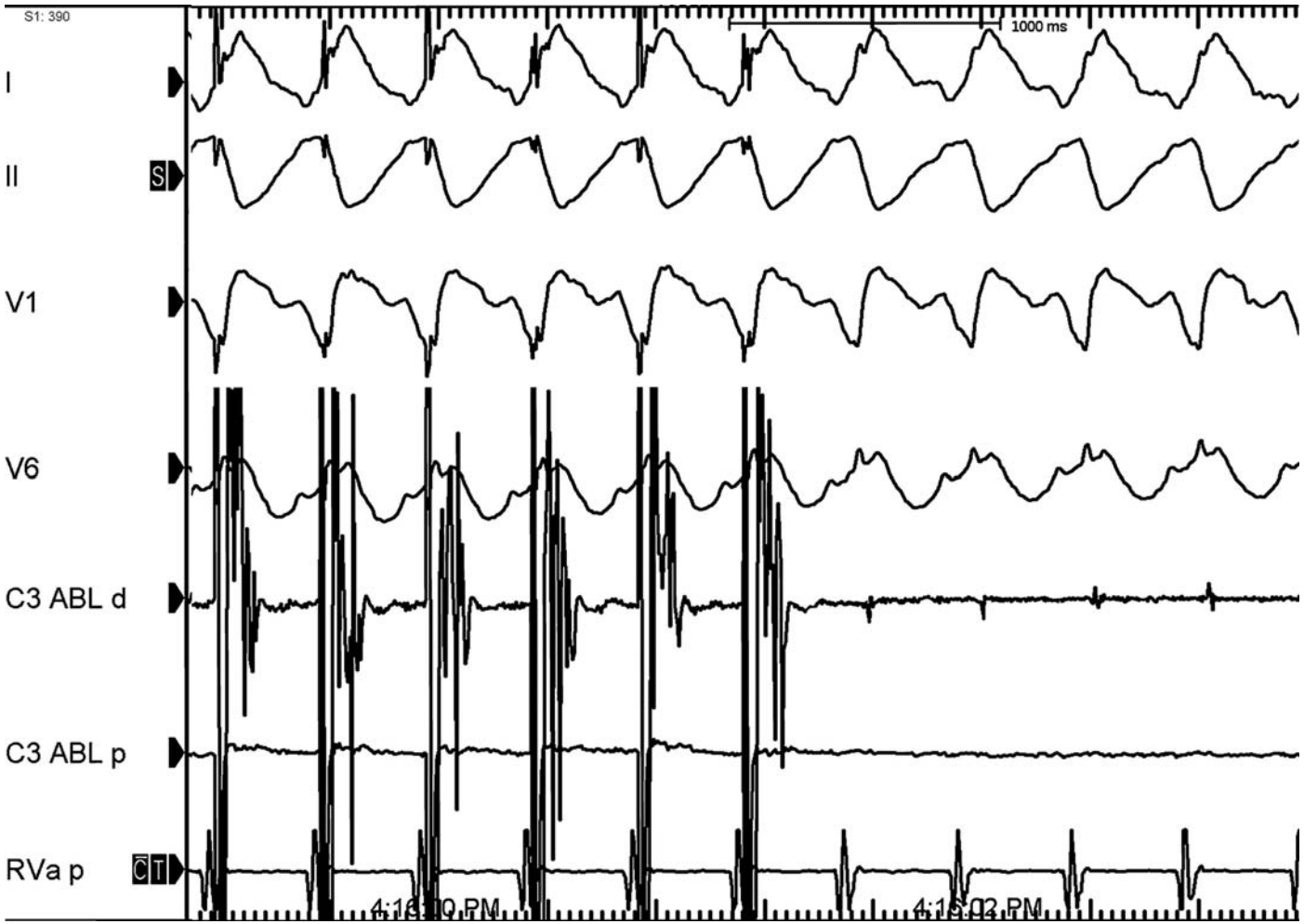


The patient is undergoing ablation for VT associated with multiple ICD discharges. VT of similar cycle length to the clinical VT was readily induced and showed slight QRS variability as shown. A ventricular extrastimulus inserted into the cardiac cycle terminated the tachycardia (figure).

Explanation:

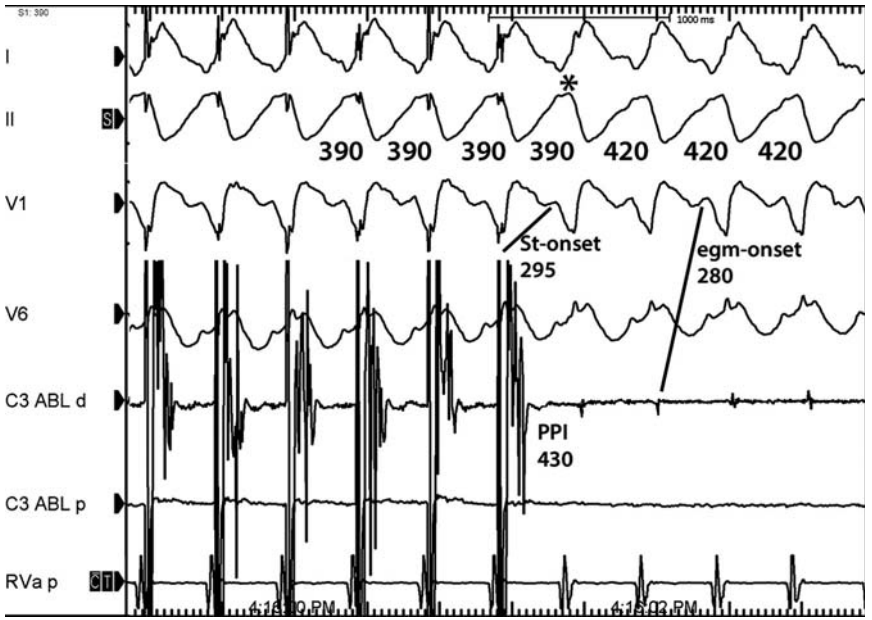
If one assumes that this phenomenon is reproducible, one has an important clue to successful ablation. The VES has terminated the VT without apparent conduction to the ventricle as evidenced by absence of ventricular activation seen on the surface ECG. Since the electrical influence of the stimulating electrode at this site in the absence of generalized myocardial capture is very regional and local, it is probable that the electrode is well positioned near a critical component of a reentrant circuit and ablation is appropriate even in the absence of a visible electrogram suggesting that it is a good site.

Figure 5–9A



The tracing was recorded during an ablation procedure for VT. Entrainment during tachycardia is attempted at an electrogram site during mapping of VT. How would one interpret the suitability of the pacing site for ablation?

Figure 5–9B

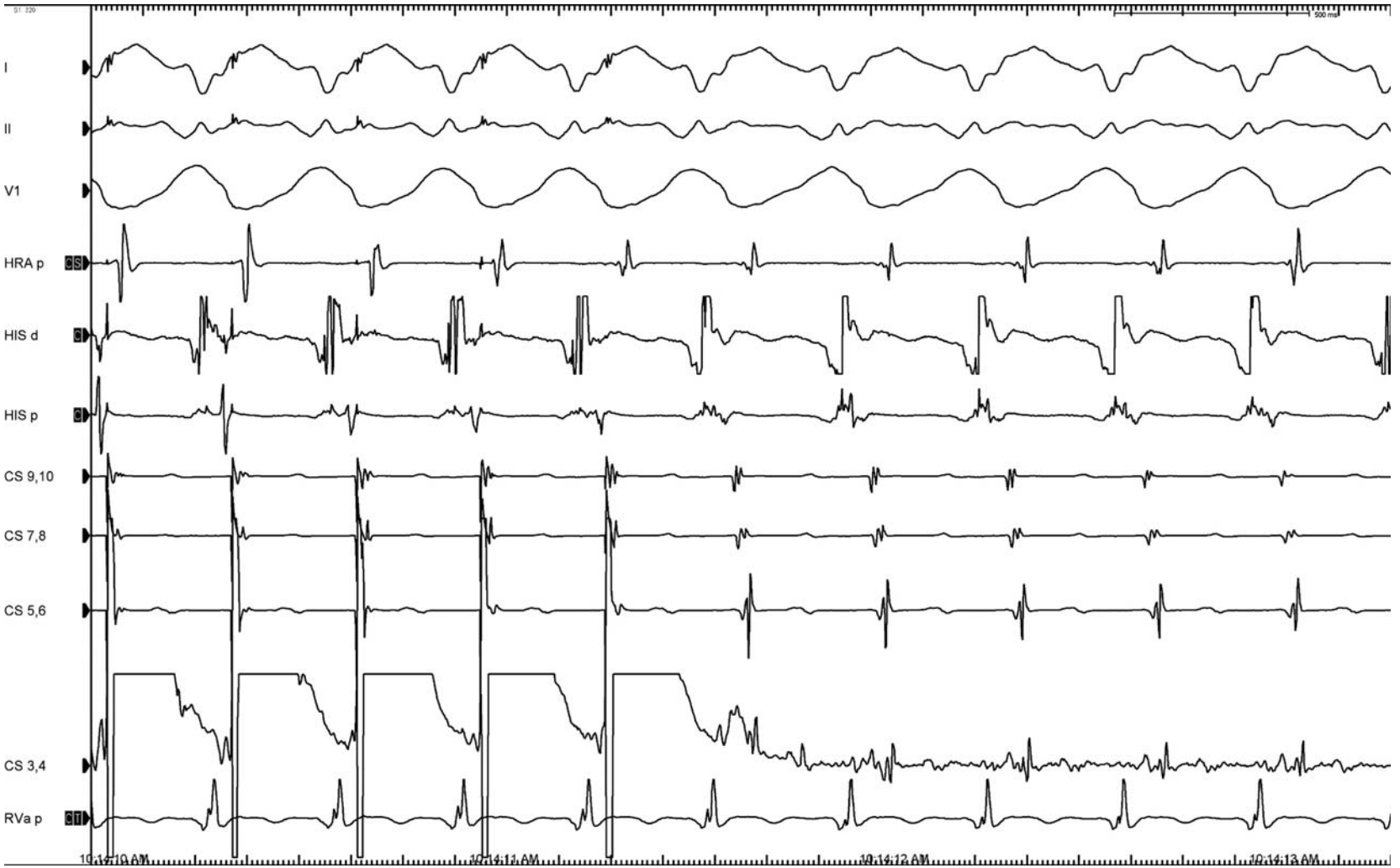


Explanation:

A checklist of observations is useful to evaluate the effects of overdrive pacing and potential entrainment.

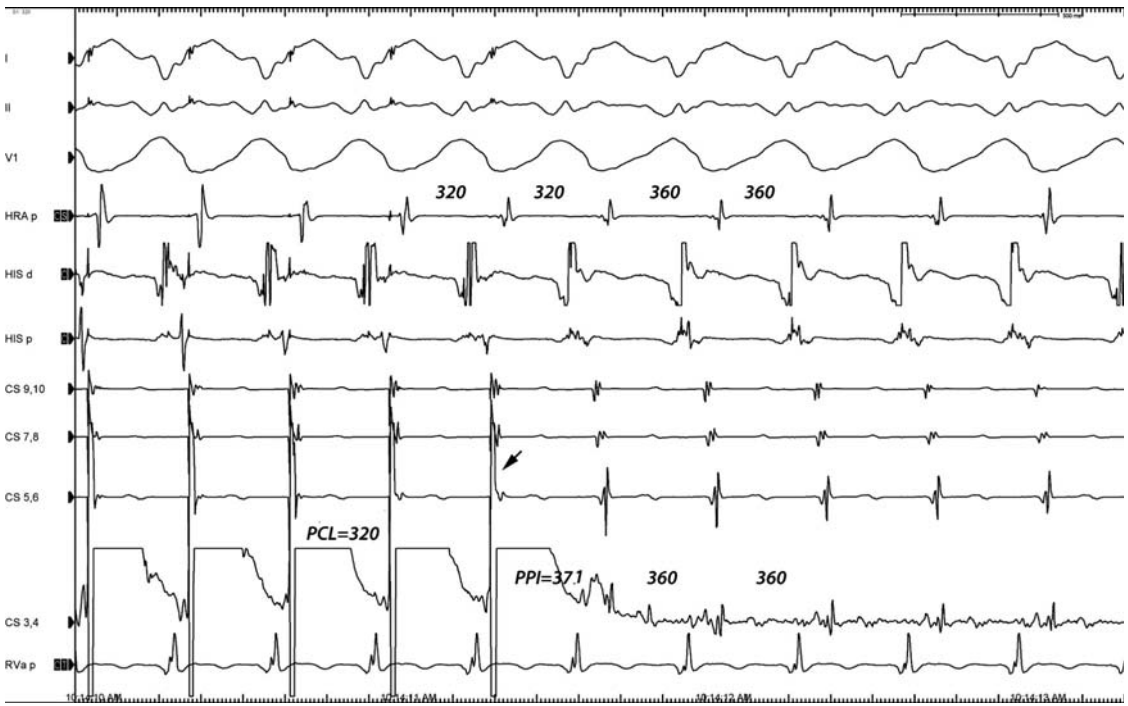
1. The cycle length of the tachycardia has shortened from 420 milliseconds to the pacing cycle length of 390 milliseconds and is in a "steady state" when pacing is stopped.
2. The QRS of the paced complex is identical to the QRS of the tachycardia. Although only 3 leads are shown here, this was verified for all 12 leads. This is termed "concealed" fusion because fusion is not apparent electrocardiographically and suggests that the pacing site is within the slow conduction zone of the circuit. (In this example, it may be "overtly" fused if one counts the RV apical electrogram slightly ahead of the pacing spike but this is a semantic issue.)
3. The last cycle accelerated to the paced rate needs to be identified, that is, the last entrained QRS. This is identified in the figure by an asterisk before the following cycle ensues at the VT cycle length.
4. Since the pacing ultimately drives the last entrained cycle, it is seen that the interval from the last stimulus to the QRS onset is relatively long (295 milliseconds), approximating the time required to get from the stimulus to the breakout point from the delayed conduction zone of the circuit. This is also reflected in the interval from the electrogram to the onset of the QRS (280 milliseconds), which is approximately the same. In general, the longer this interval, the more proximal in the delayed conduction zone it is hypothesized to be. In this case, it might be nearer the "entrance" than the "exit" of the slow zone of conduction. (The importance of step 3 now becomes obvious, that is, to identify which cycle is driven by the stimulus.)
5. Finally, the PPI needs to be measured. The electrogram during pacing is quite noisy and the PPI here is approximated from the stimulus artifact to the return electrogram. It is approximately the cycle length of the tachycardia and is therefore "in" the circuit. It is a reasonable ablation site, although it may not for many reasons be the sole lesion required to "cure" the tachycardia.

Figure 5–10A



The clinical tachycardia is induced in the laboratory and atrial overdrive pacing is attempted and terminated. Can the mechanism of tachycardia be definitively determined?

Figure 5–10B



Explanation:

The tachycardia on the right has a wide QRS with a 1:1 A and V relationship. The QRS morphology is atypical for a bundle branch block pattern. There is no obviously visible His deflection. The atrial activation sequence is "central."

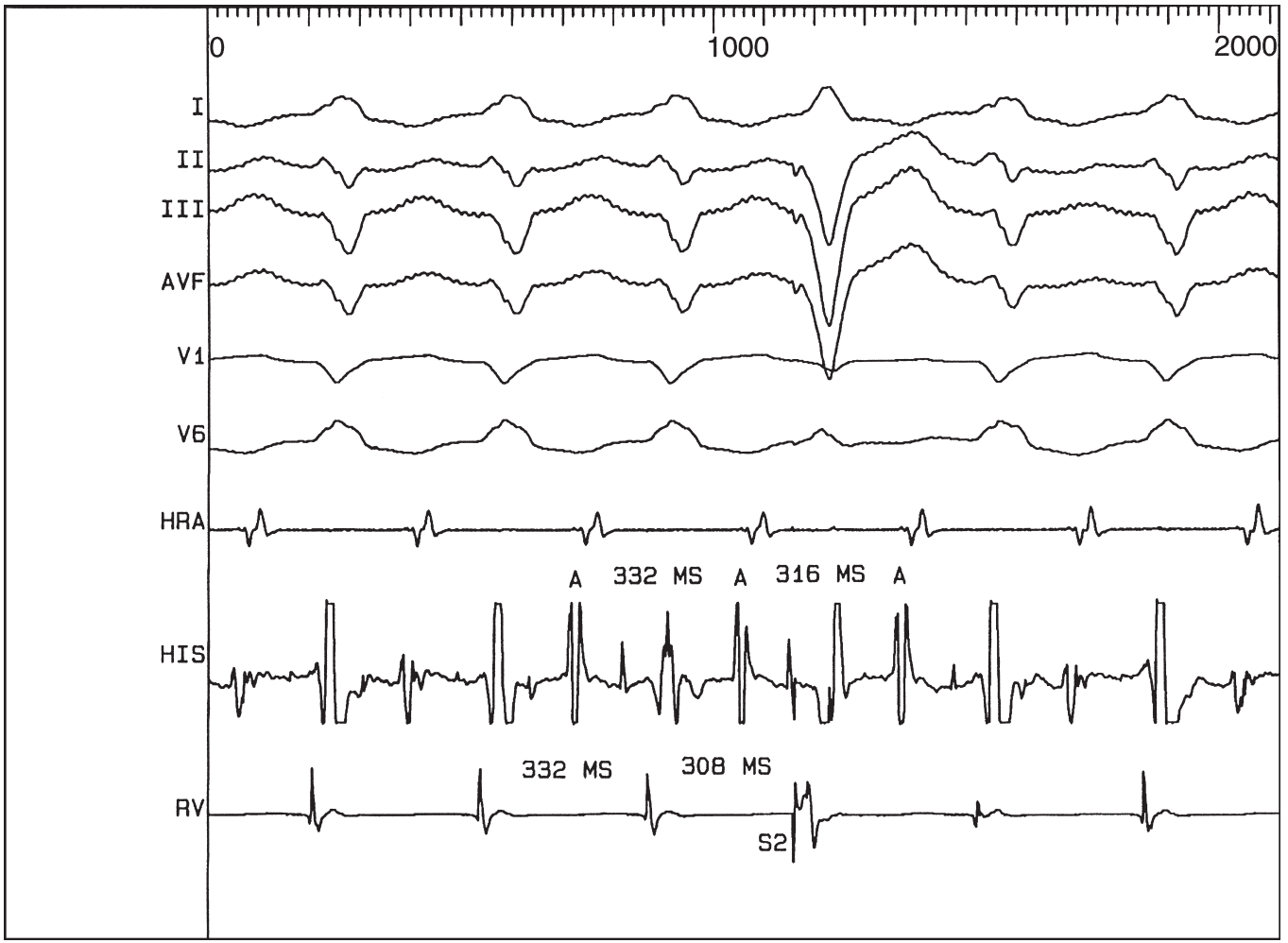
This tachycardia can only be preexcited SVT or VT. Overdrive pacing from the distal coronary sinus atrial electrogram results in entrainment of the tachycardia with identical QRS morphology ruling out VT.

The question is now to determine the mechanism of the preexcited tachycardia, namely, AT, AVNRT with bystander AP, or AVRT (antidromic). Statistically, antidromic tachycardia would be the leading candidate by prevalence alone.

One notes that the PPI at the distal CS is virtually identical to the tachycardia cycle length, that is, it is "in." This clearly rules out AVNRT with a bystander, as the PPI would be considerably longer. The only viable possibilities for this tachycardia now remain atrial tachycardia fortuitously at the pacing site in the presence of an AP at that site or antidromic reentry over an AP at that site. Clearly the former would constitute a very rare happening indeed but the maintenance of a constant VA relation in the cycle immediately after entrainment essentially rules out AT.

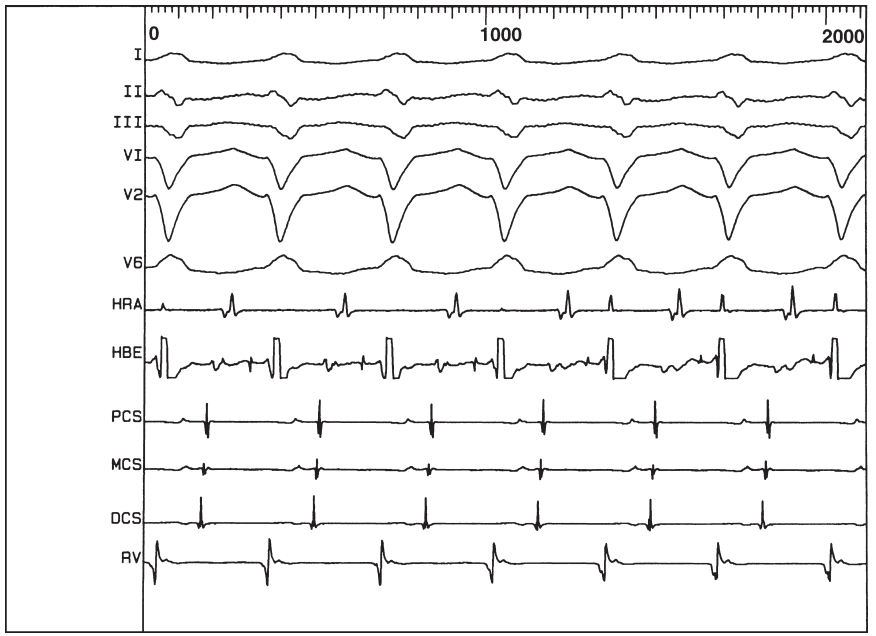
We now have preexcited AVRT with a left lateral AP as the anterograde limb of the circuit. The retrograde limb is in all likelihood the normal AV conducting system. How might one prove that? That is, from where might one entrain?

Figure 5–11A



This 32-year-old man presented with a wide QRS complex tachycardia. At EP study the observation shown was made. What is the probable mechanism of tachycardia? What is the likely tachycardia circuit in this patient?

Figure 5–11B



Explanation:

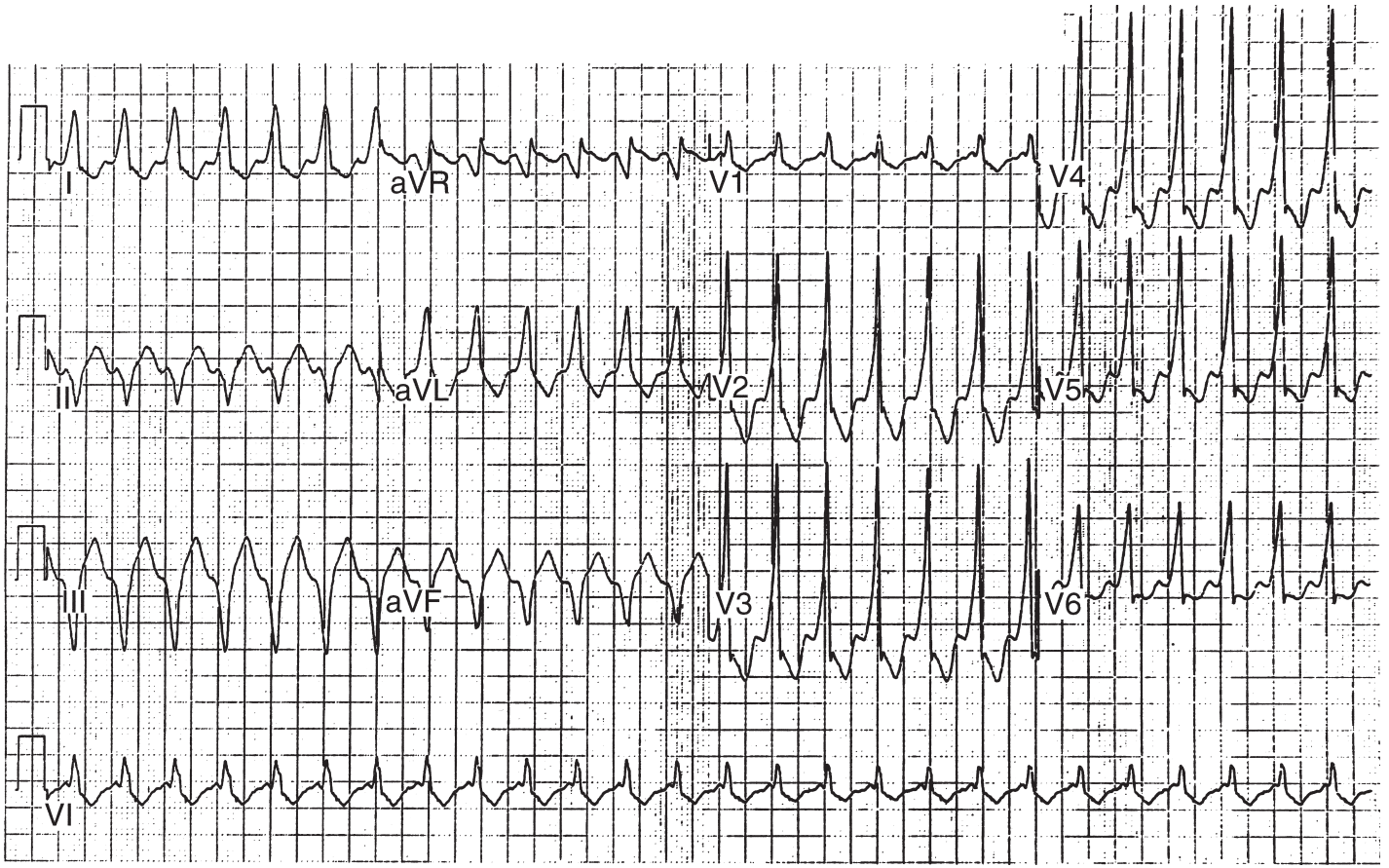
This figure demonstrates tachycardia with a CS lead in place, which was omitted in Fig. 5–11*A*. Note that there is eccentric atrial activation with earliest atrial activation at the distal CS electrode. The HV interval is normal. Thus, this is either a left atrial tachycardia or AV reentry utilizing a left-sided AP (Table 1–7). Analysis of Fig. 5–11*A* demonstrates preexcitation of the atrium with a PVC introduced at a time when the His bundle is refractory. Thus, a left-sided AP is present and in this patient was involved in the tachycardia circuit. This observation alone does not absolutely exclude a coexisting atrial tachycardia.

The preexcitation index (PI) is a helpful adjunct to locate the site of the AP. It is determined by introducing progressively more premature right ventricular extrastimuli during tachycardia. The PI is calculated by subtracting the longest premature interval (V_1V_2) that preexcites the atrium from the tachycardia cycle length (V_1V_1). During a narrow QRS complex tachycardia, a relatively late-coupled V_1V_2 that preexcites the atrium (short PI) is typical for a right-sided or septal AP, and a PI <45 milliseconds essentially excludes a left-sided AP. The PI of slow/fast AV node reentry is almost always >90 milliseconds.

In Fig. 5–11*A*, the PI is 24 milliseconds (332–308 milliseconds). Note that this patient has LBBB aberrancy. In this situation the ventricle is activated anterogradely over the right bundle branch, which exits near the moderator band in the right ventricular apex. The PVC has a morphology consistent with right ventricular apical pacing. Thus, the right ventricular catheter is near the tachycardia circuit, which now incorporates the right ventricular endocardium. During narrow QRS complex tachycardias, the premature ventricular complex conducts transseptally to enter the tachycardia circuit in a patient with a left-sided AP, and the PI is much longer.

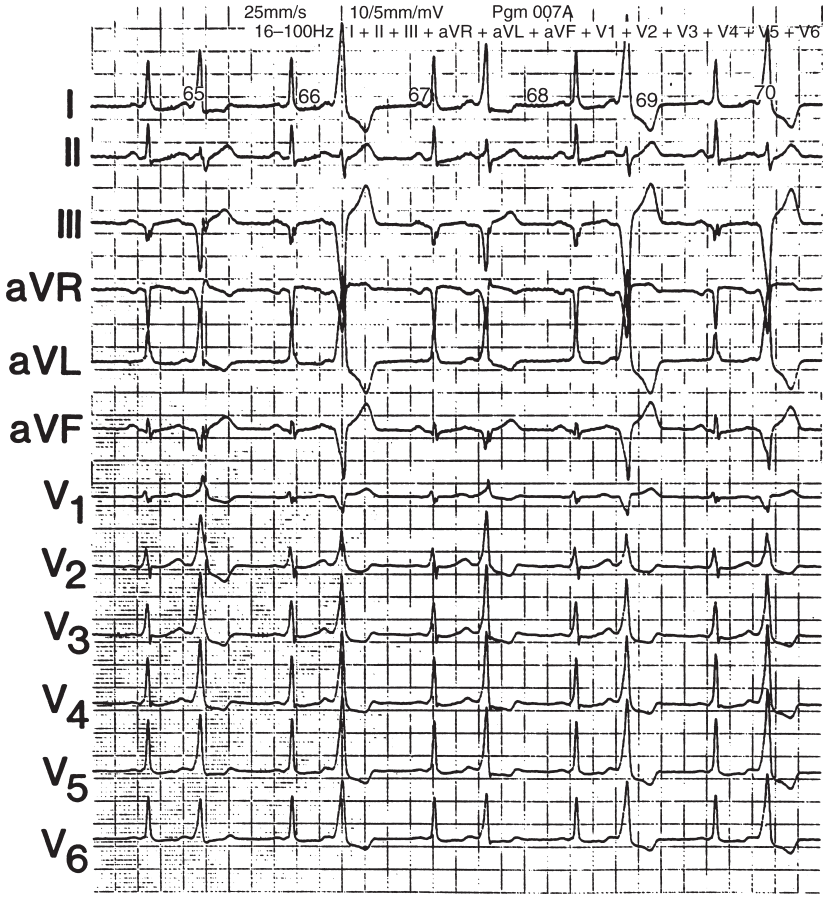
In summary, a diagnosis of AV reentry was reasonable from Fig. 5–11*A*, but the location of the AP could not be determined from just this figure.

Figure 5–12A



A 45-year-old man presents to the emergency room with a history of 2 hours of palpitations. A 12-lead electrocardiogram was recorded and is shown in Fig. 5–12*A*. What is the differential diagnosis? While taking the patient's history, what is probably the most important question you can ask to differentiate the mechanism of tachycardia? Does hemodynamic stability in this patient help to differentiate supraventricular tachycardia from VT?

Figure 5–12B



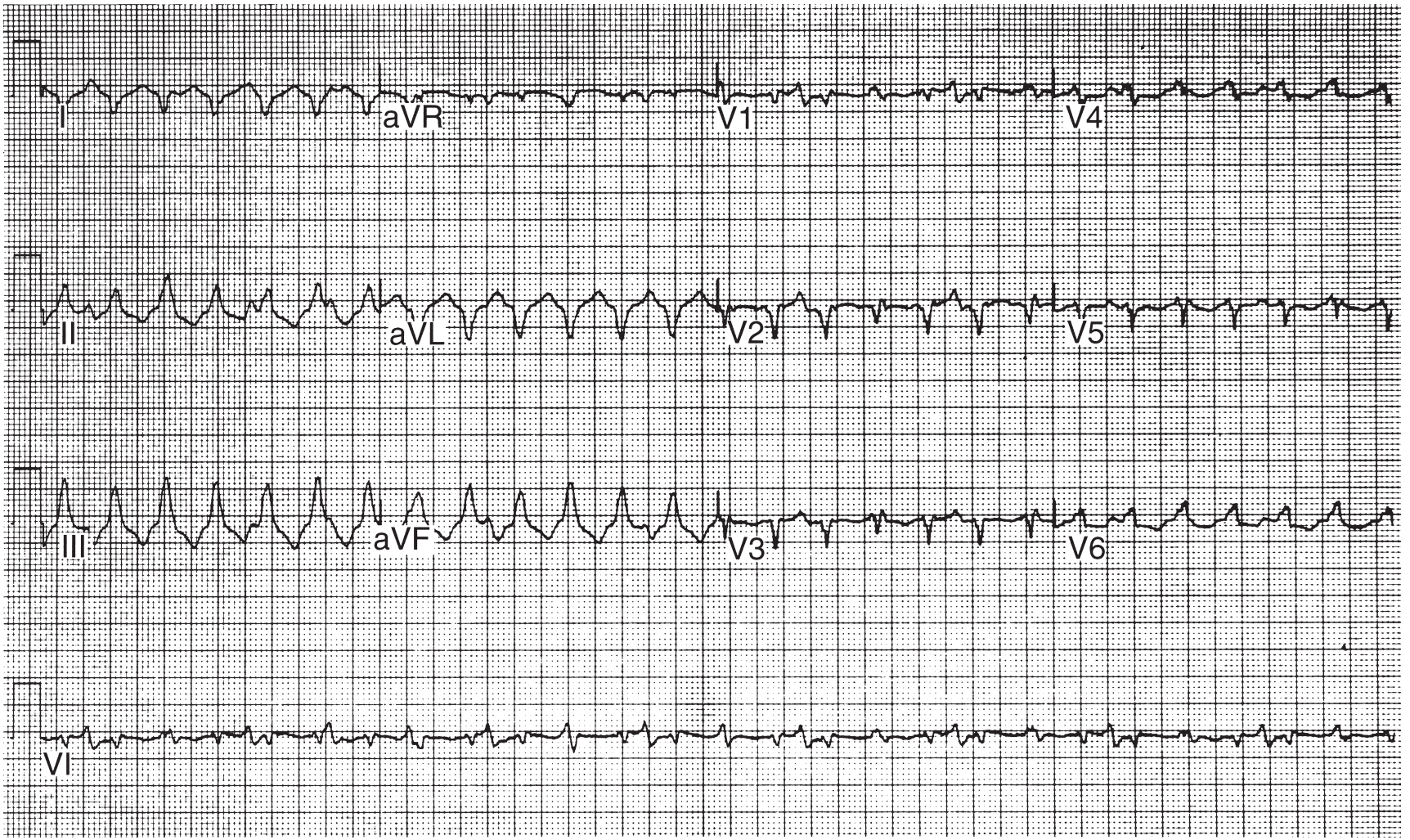
Explanation:

Figure 5–12*A* is a RBBB tachycardia without evidence of VA dissociation. The differential diagnosis is VT, SVT with aberrancy, and a preexcited tachycardia (Table 1–4). The RBBB is "atypical," and less likely to represent aberrancy. The presumptive diagnosis should be VT until proven otherwise. After tachycardia was terminated a simultaneous 12-lead ECG rhythm strip was obtained (Fig. 5–12*B*). Note that this patient has intermittent wide QRS complexes during sinus rhythm, but more than one wide QRS morphology is present. Complexes 2 and 6 are nearly identical to the QRS complexes noted in Fig. 5–12*A*. This strongly suggests that a preexcited tachycardia was present in Fig. 5–12*A* since the PR intervals of the wide complexes are constant. An alternative diagnosis that must be excluded is coupled PVCs giving the illusion of a constant PR interval. The presence of an AP can be confirmed at EP study. The preexcited QRS morphology suggests a left posterior AP location. In contrast, QRS complexes 4, 8, and 10 demonstrate a different wide QRS morphology, and the PR intervals are constant. This likely represents a second preexcited QRS morphology, and the 12-lead ECG is consistent with a posteroseptal AP position. In patients with more than one AP, a common combination is posteroseptal and left free wall. VT is always part of the differential diagnosis.

The most important question you can ask a patient with a wide QRS complex tachycardia is whether he or she has a history of heart disease. In this case the patient had no heart disease and this makes VT a less likely possibility. Further, if the patient has had a history of sustained tachycardia since childhood, the diagnosis of Wolff–Parkinson–White (WPW) syndrome is even greater. The morphology "rules" to differentiate supraventricular tachycardia from VT in the presence of RBBB do not apply in the presence of preexcitation. Even though the QRS morphology is more suggestive of VT in Fig. 5–12*A*, this is not helpful since a preexcited tachycardia is present.

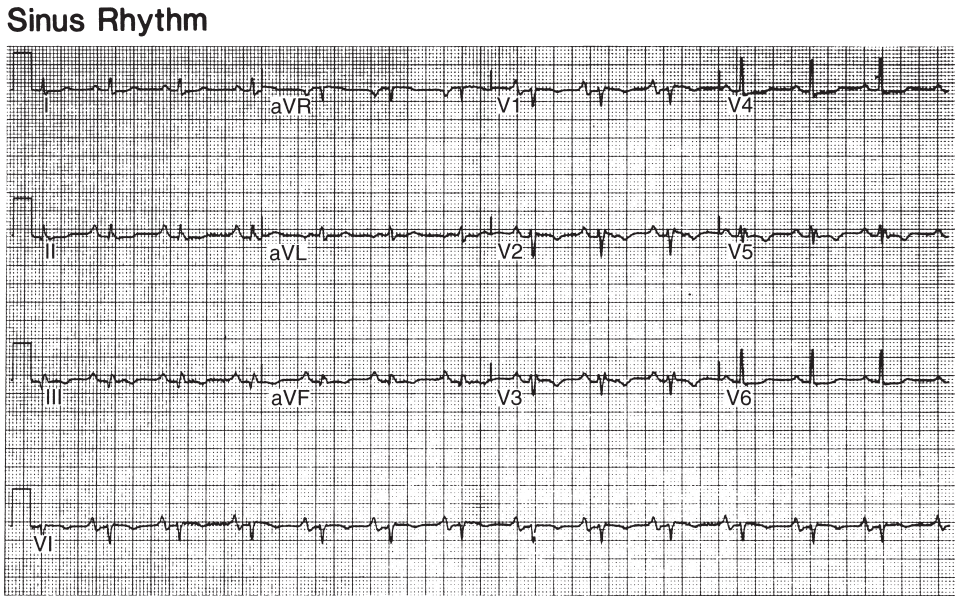
Hemodynamic stability does not differentiate SVT from VT.

Figure 5–13A



You are called to the emergency room to evaluate a patient who presents with palpitations. The 12-lead electrocardiogram in Fig. 5–13*A* was recorded during the patient's symptoms. What is the diagnosis?

Figure 5–13B

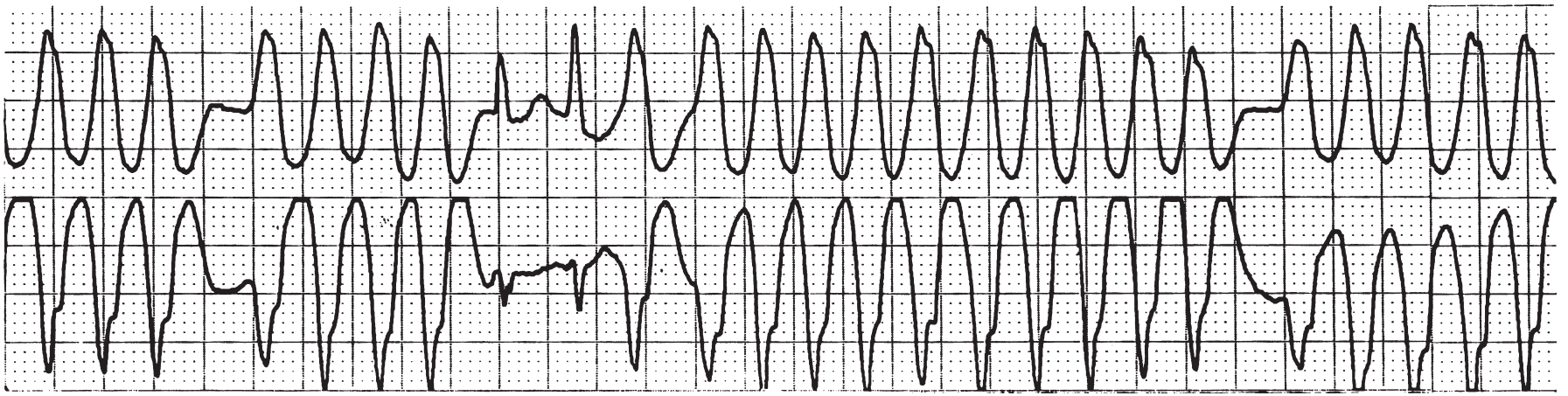


Explanation:

Analysis of the rhythm strip in V_1 of Fig. 5–13*A* demonstrates clear VA dissociation during tachycardia. The somewhat confusing pattern occurs because the P waves are actually larger in amplitude than the QRS complexes in this lead, as noted during sinus rhythm (Fig. 5–13*B*).

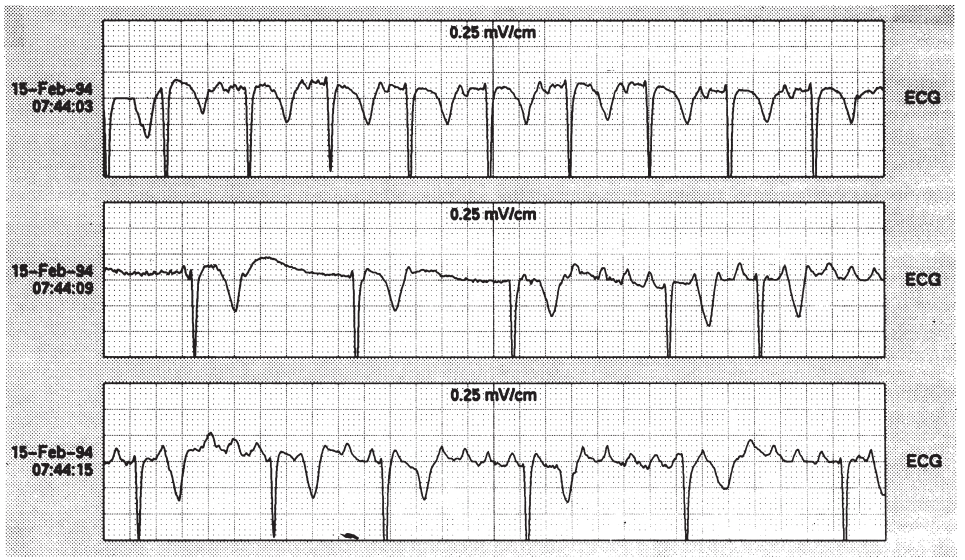
This patient has VT. The diagnosis might have been more difficult if only a rhythm strip of V_1 was available. Further, the QRS width in V_1 is barely 120 milliseconds. This stresses the importance of obtaining a 12-lead electrocardiogram during tachycardia, and reserving judgment until multiple leads are analyzed.

Figure 5–14A



This patient was admitted with a history of syncope. During inhospital monitoring, the rhythm shown was recorded during which the patient was hypotensive. What is the diagnosis?

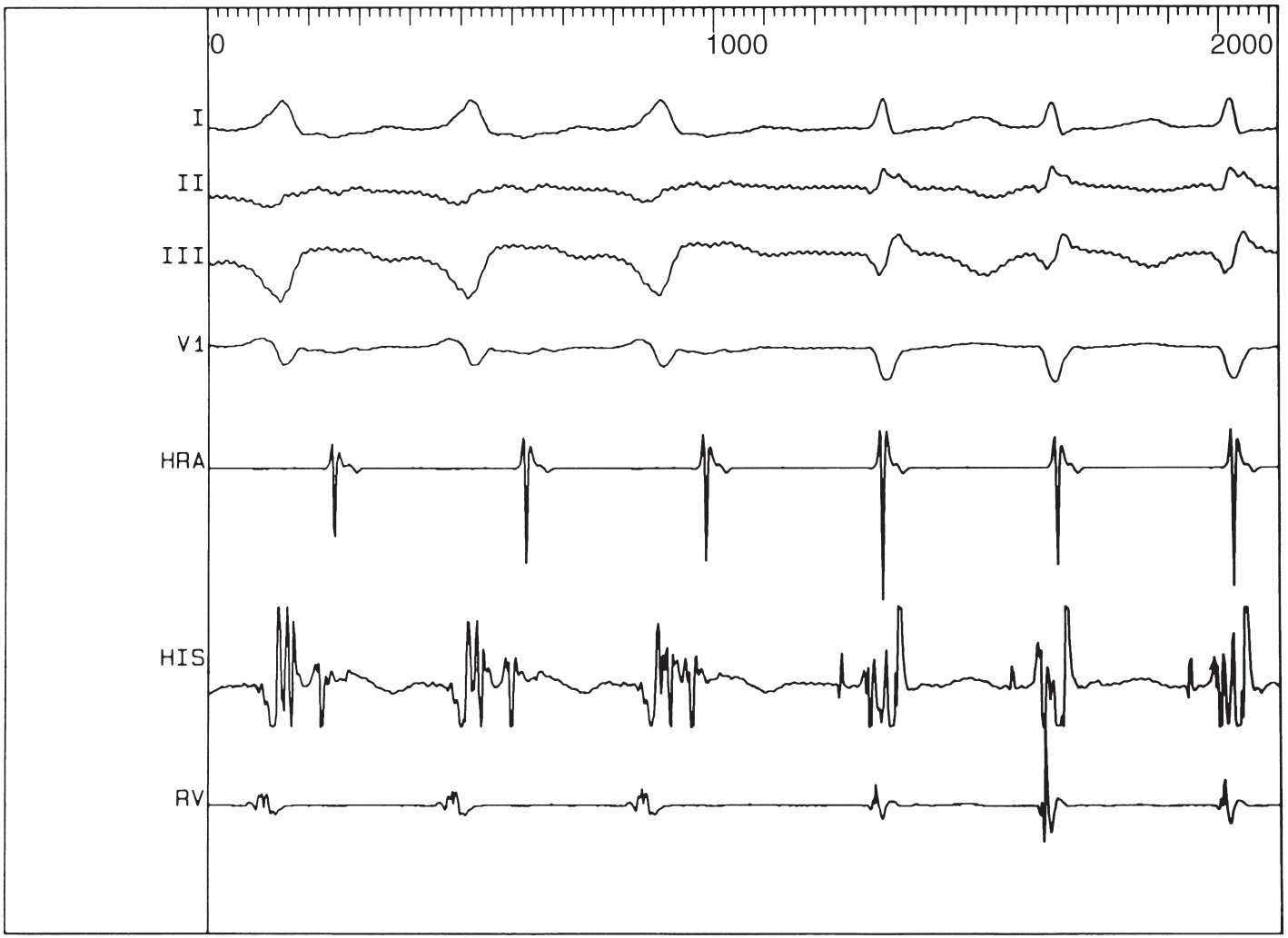
Figure 5–14B



Explanation:

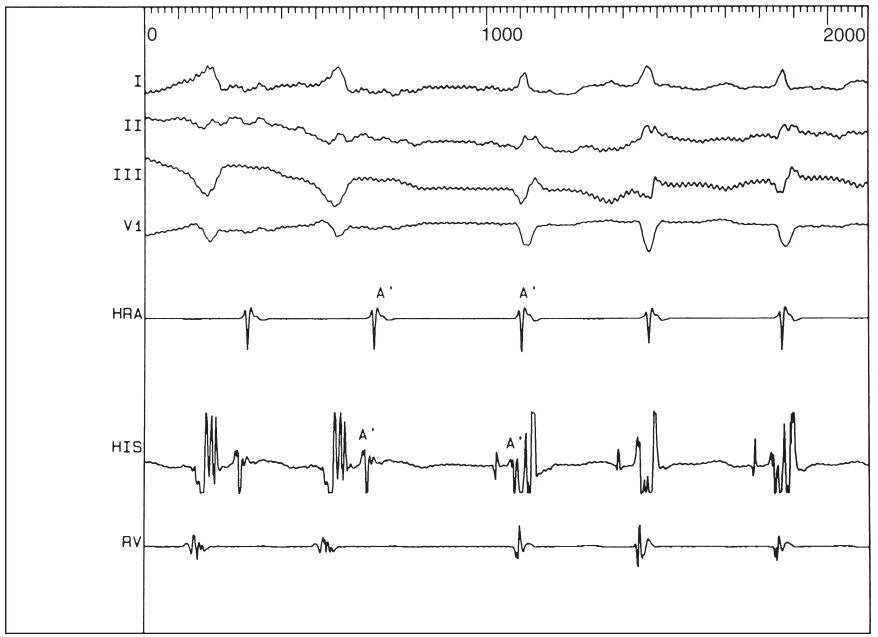
A grossly irregular, rapid wide QRS complex tachycardia is nearly always atrial fibrillation with conduction over an AP. This patient had a posteroseptal AP that was successfully ablated at EP study. In Fig. 5–14*A*, there is a preexcited tachycardia with only two normally conducted QRS complexes. Degeneration of AV reentry to atrial fibrillation is the usual mechanism for AF in patients with WPW, and successful ablation of the AP prevents recurrences of AF in more than 90% of these patients. Clearly this was not the case in this patient as noted in Fig. 5–14*B*. The top rhythm strip reveals sinus rhythm and atrial flutter/fibrillation occurred spontaneously (middle rhythm strip). In patients in whom atrial fibrillation can be demonstrated as a primary arrhythmia, ablation of the AP should not be performed to prevent AF. However, prevention of conduction over the AP after ablation removes a potentially life-threatening situation in patients with rapid preexcited ventricular rates. If a patient demonstrated relatively poor conduction over the AP during atrial fibrillation, and antiarrhythmic drugs were selected to maintain sinus rhythm, ablation of the AP would offer little benefit to the patient.

Figure 5–15A



This middle-aged man has a history of myocardial infarction. What is the diagnosis?

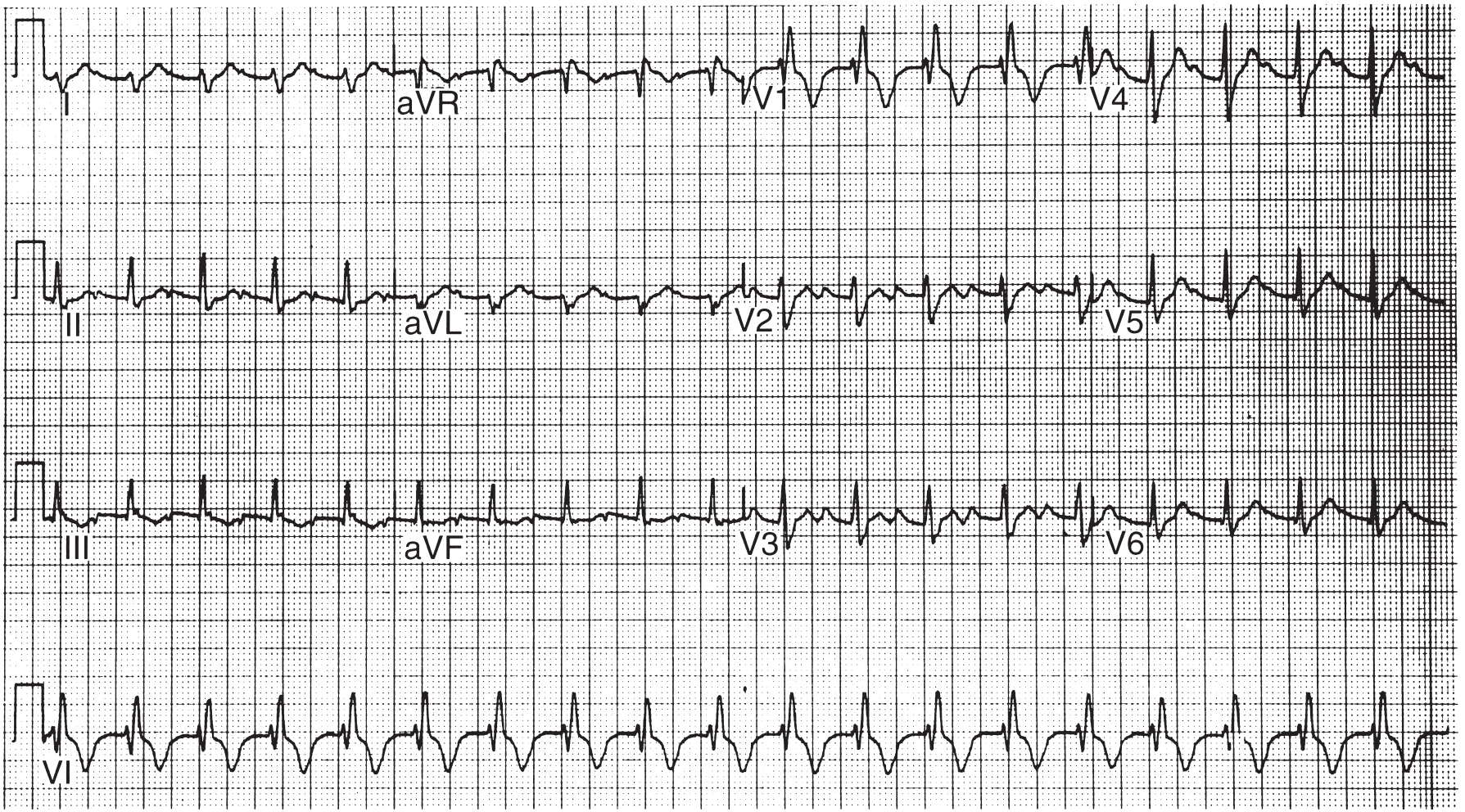
Figure 5–15B



Explanation:

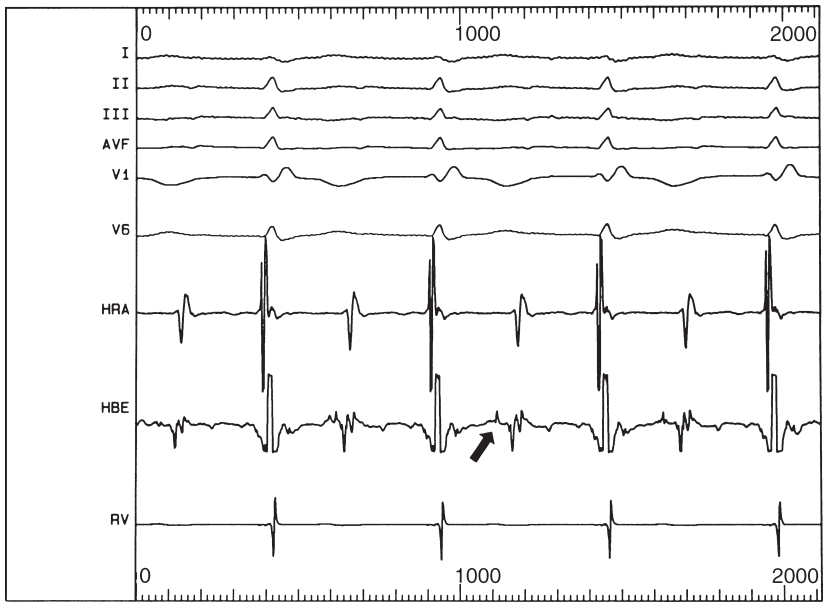
This figure demonstrates a wide QRS tachycardia followed by a narrow QRS tachycardia. There is no His deflection preceding ventricular activation in the wide QRS complexes, and the differential diagnosis is VT versus a preexcited tachycardia. This patient did not have ventricular preexcitation; thus, this is VT with 1:1 VA conduction. Tachycardia spontaneously terminates and a narrow QRS complex tachycardia is induced (tachycardia-induced tachycardia) (Table 1–3). There is a long AH interval followed by a change in retrograde atrial activation sequence. Atrial septal activation precedes HRA activation, and the His bundle atrial electrogram is located between the His bundle and QRS deflections. The VA interval is too short for AV reentry, and the most likely diagnosis is AV node reentry. At EP study both types of tachycardia could be independently induced. In addition, during continuous ECG inhospital monitoring, the patient had spontaneous episodes of tachycardia that demonstrated both a wide and a narrow QRS complex morphology. The rates of the two tachycardias were very similar, causing confusion as to the appropriate diagnosis. These patients should undergo EP testing to confirm the diagnosis. The importance of tachycardia-induced tachycardia cannot be overemphasized. Two particular situations need to be considered. First, it may explain a discrepancy in the clinical presentation. For example, patients may have documented, stable SVT but a history of syncope prior to recording the ECG. If VT initiated SVT in these individuals, it is possible that syncope may have resulted from the VT. Of course, patients may also have syncope with SVT. In someone with a history of heart disease this mechanism must be sought at EP testing. Second, in some individuals one arrhythmia always initiates the other arrhythmia. The most common example is AV reentry producing atrial fibrillation. Elimination of AV reentry can prevent both arrhythmias in this situation, and one does not need to treat the tachycardias as separate entities.

Figure 5–16A



This patient has a history of palpitations and presyncope. What is the diagnosis?

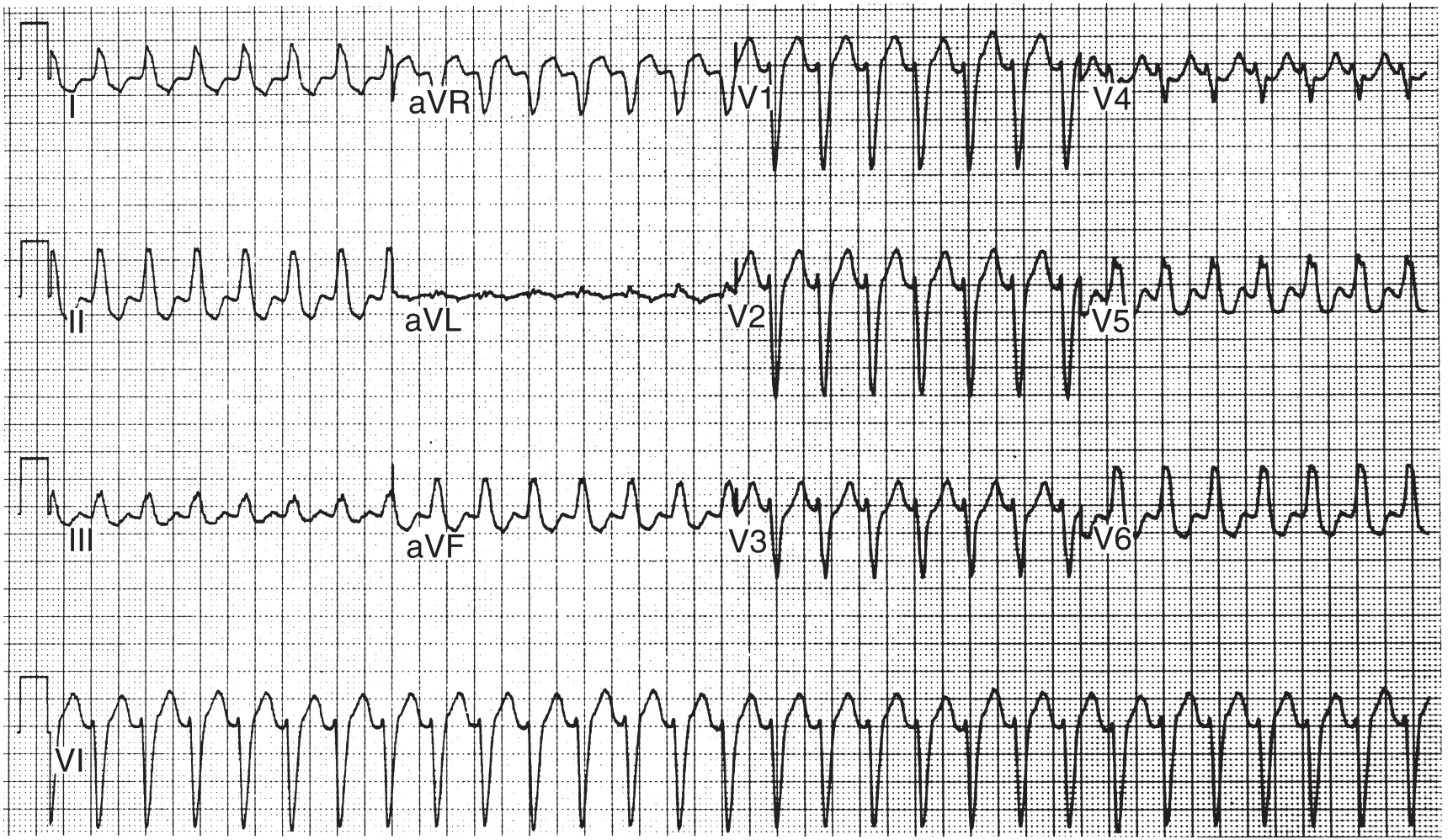
Figure 5–16B



Explanation:

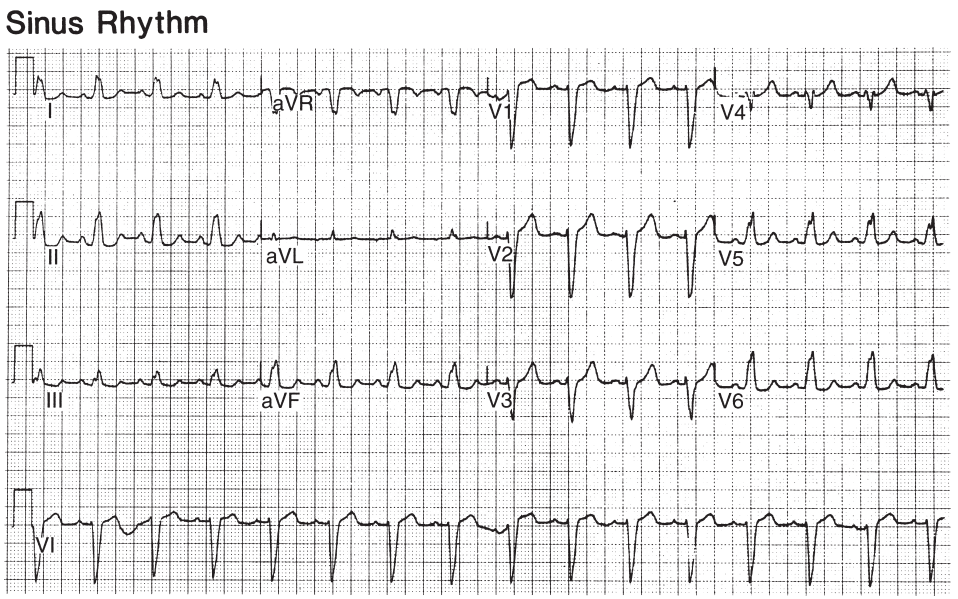
In Fig. 5–16*A*, there is a typical triphasic RBBB tachycardia with atrial activity positioned almost exactly between the two QRS complexes. The QRS pattern suggests a supraventricular mechanism. ECG leads II, III, and aVF demonstrate P-wave activity equidistant between the two QRS complexes. In addition, the P waves suggest caudal–cranial activation and they are narrow. This is typical for atrial activation that originates in the interatrial septum. Thus, analysis of the 12-lead ECG in Fig. 5–16*A* reveals clues to diagnose AV node reentry with 2:1 block (Table 1–6). However, EP study (Fig. 5–16*B*) was necessary for confirmation. The His bundle electrogram reveals 2:1 block below the His bundle deflection (*arrow*). There is a low to high atrial activation sequence, with early septal activation. This is typical for AV node reentry with 2:1 block below the His bundle.

Figure 5–17A



A 26-year-old man presents with a several-year history of intermittent palpitations. There is no history of heart disease. What is the differential diagnosis? Is this VT or supraventricular tachycardia?

Figure 5–17B



Explanation:

Differentiation of LBBB tachycardias begins with careful analysis of the LBBB pattern. Activation of the ventricle over the right bundle yields a "typical" LBBB pattern as noted in this patient during sinus rhythm (Fig. 5–17*B*). The initial r wave in ECG leads V_1 and V_2 is narrow and followed by a rapidly conducted S wave. No Q wave is present in V_6. "Atypical" LBBB patterns can occur with activation of the right ventricle preceding the left ventricle and are quite variable. Examples are VT as well as right-sided APs. If a typical LBBB pattern is present, the usual differential is any SVT with LBBB aberrancy, or unusual varieties of preexcited tachycardia such as atriofascicular and nodofascicular reentry. One exception might be bundle branch block reentry, but these patients typically have large dilated hearts and the QRS duration during tachycardia is often substantially prolonged (>160 milliseconds).

Analysis of Fig. 5–17*A* reveals a typical LBBB pattern suggesting some form of supraventricular arrhythmia in this patient without a history of heart disease. Note the P-wave activity seen at the apex of the T wave in multiple leads, especially I, II, aVL, and aVF. The negative P wave in leads I and aVL suggests initiation of excitation in the left atrium. The differential diagnosis is a left atrial tachycardia with relatively long AV conduction time versus AV reentry using a concealed left-sided AP. The relatively long RP interval in this situation occurs because of the LBBB, which adds conduction time to the circuit since the right ventricle is activated prior to the left ventricle. Therefore, the P wave appears later than expected after the QRS complex.

At electrophysiologic study the tracing of Fig. 5–17*C* was obtained. Atrial activation is eccentric with earliest activity on the distal coronary sinus electrode. Pacing maneuvers confirmed a concealed left lateral AP and the patient had AV reentry.

Figure 5–17C

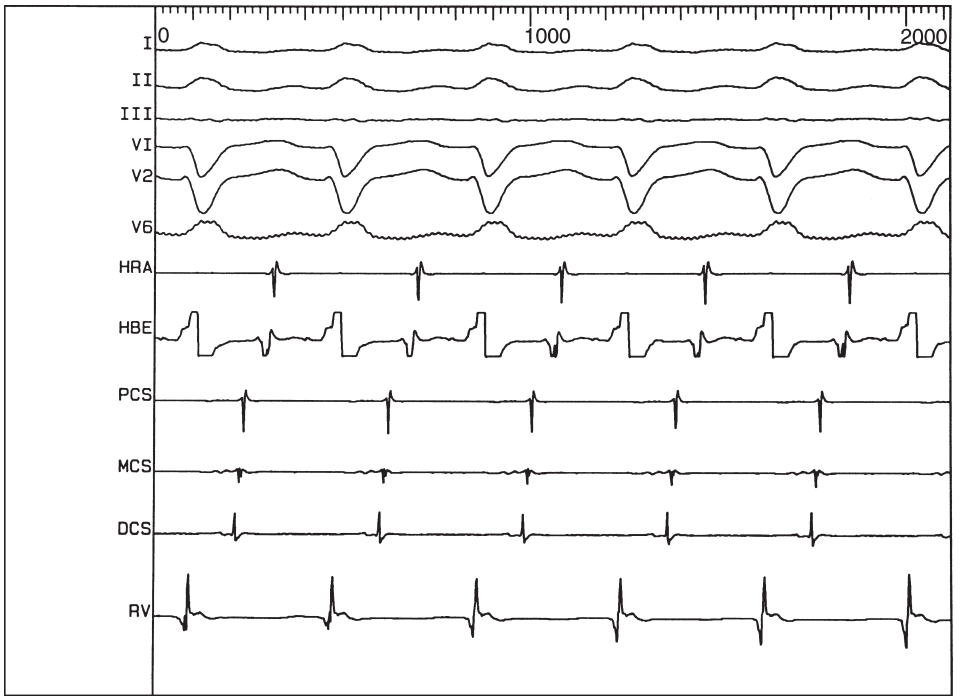


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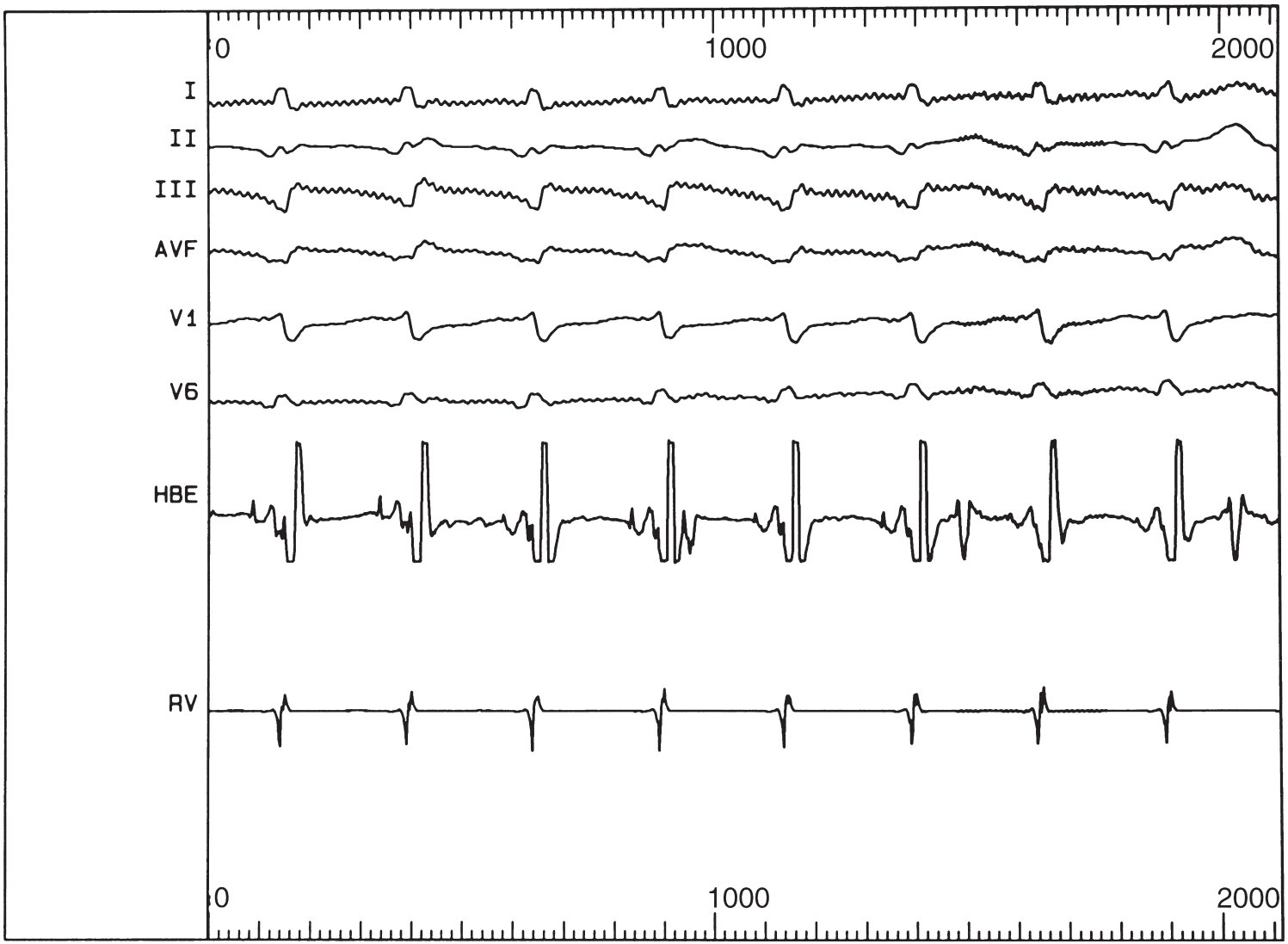
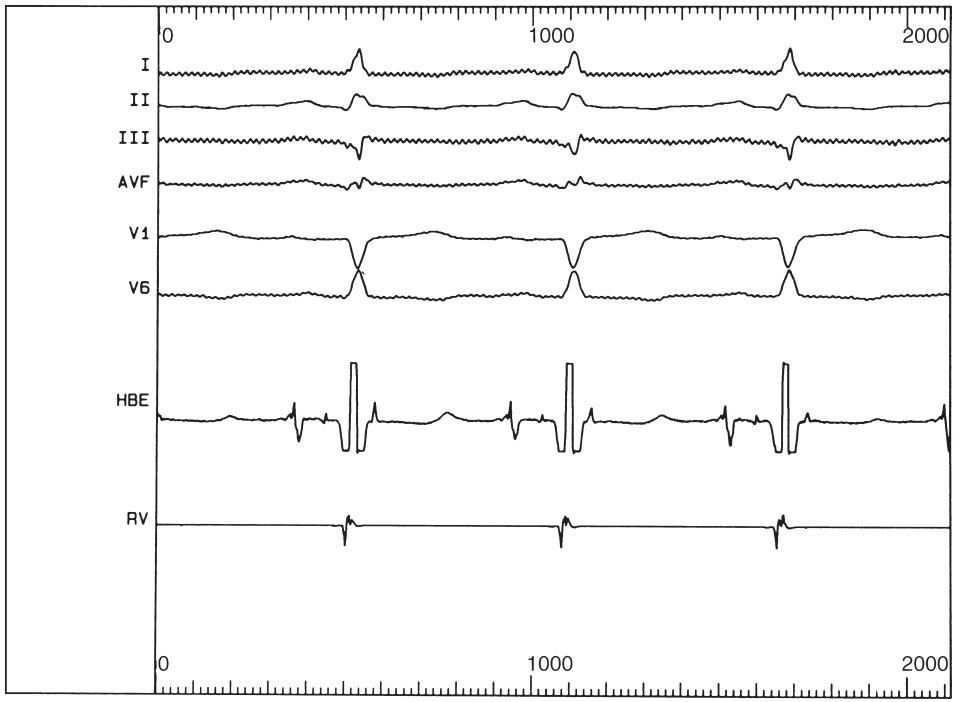
Figure 5–18A



The rhythm shown was recorded at EP study. What is the diagnosis?

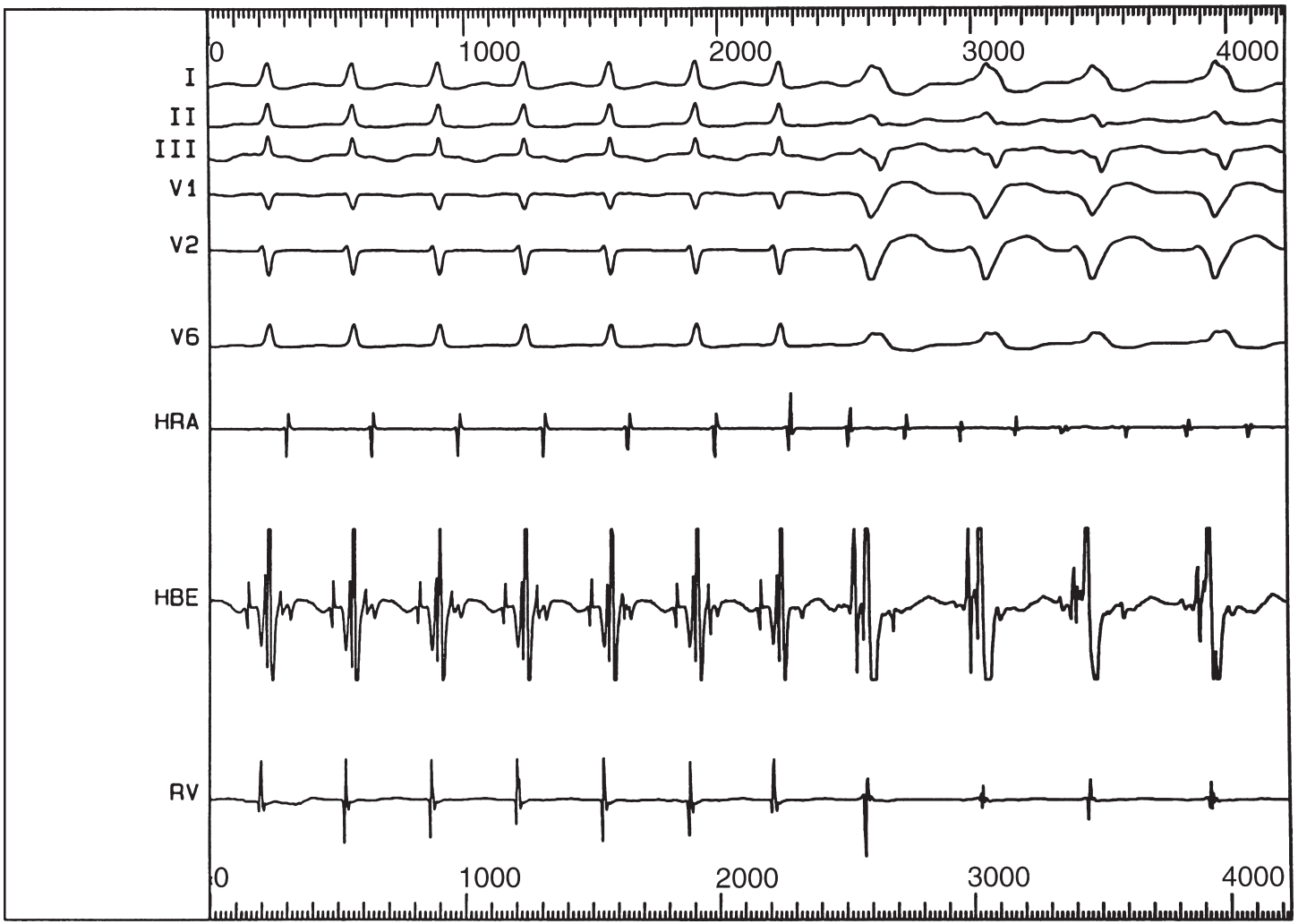
Figure 5–18B



Explanation:

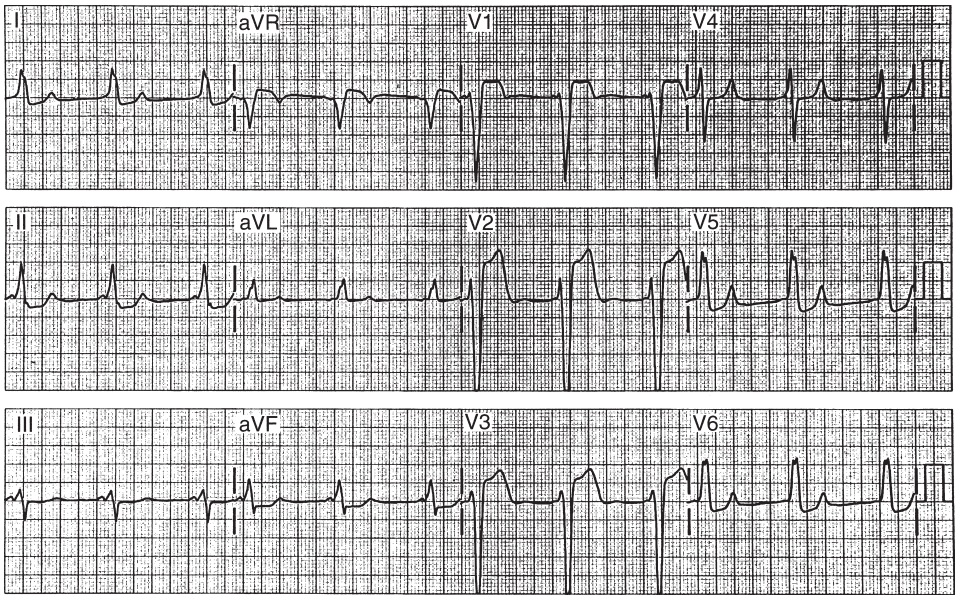
Figure 5–18*A* shows a regular tachycardia with the QRS duration slightly prolonged. A His bundle deflection is recorded before each ventricular activation on the His bundle lead. However, careful analysis reveals that the HV interval is actually short, with His activation occurring nearly simultaneously with the onset of ventricular activation as measured from the surface ECG (Table 1–8). In addition, there is ventriculoatrial dissociation present, which is clearly demonstrated on the His bundle electrogram with the sixth and eighth QRS complexes. The HV interval during sinus rhythm is noted in Fig. 5–18*B*, considerably longer than that recorded during tachycardia. Thus, the mere presence of a His bundle deflection preceding each QRS complex during tachycardia is not diagnostic of a supraventricular arrhythmia. The HV interval must be equal to or greater than the HV recorded without tachycardia, or the patient has a preexcited tachycardia or VT. This patient had VT with retrograde activation of the His bundle.

Figure 5–19A



The tracing shown was observed at EP study in a young man without evidence of heart disease. What is the likely mechanism of tachycardia for both the narrow and wide QRS complex arrhythmias?

Figure 5–19B

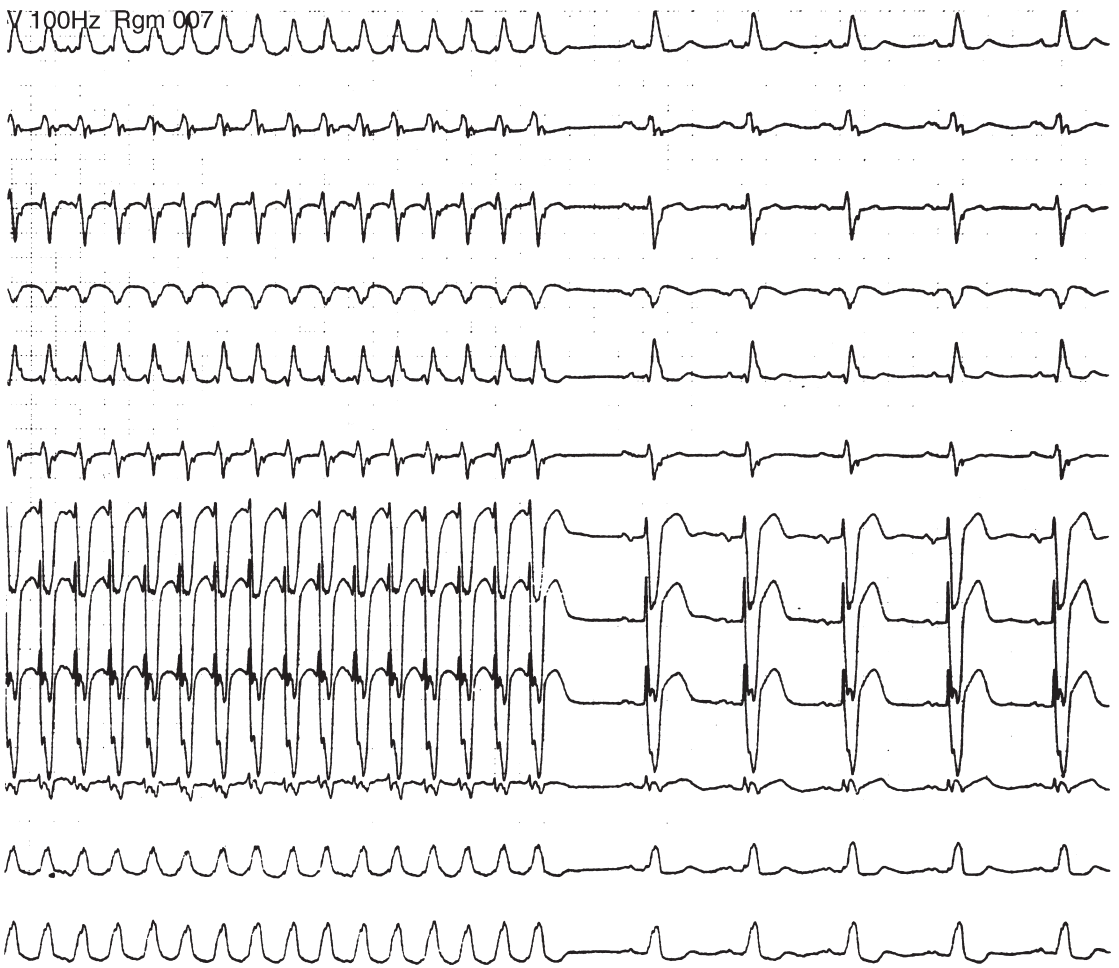


Explanation:

During sinus rhythm this patient exhibited ventricular preexcitation, and the location of the AP is near the right lateral AV groove (Fig. 5–19*B*). The tracing in Fig. 5–19*A* reveals a regular narrow QRS complex tachycardia that turns into an irregular wide QRS complex tachycardia. The narrow QRS tachycardia could be due to several mechanisms, including atrial tachycardia, AV node reentry, and AV reentry. The VA interval is longer than usual for AV node reentry, but certainly does not exclude this diagnosis. The male gender and younger age of the patient is more consistent with AV reentry. Regardless, one cannot make a definitive statement regarding mechanism of tachycardia from this tracing alone.

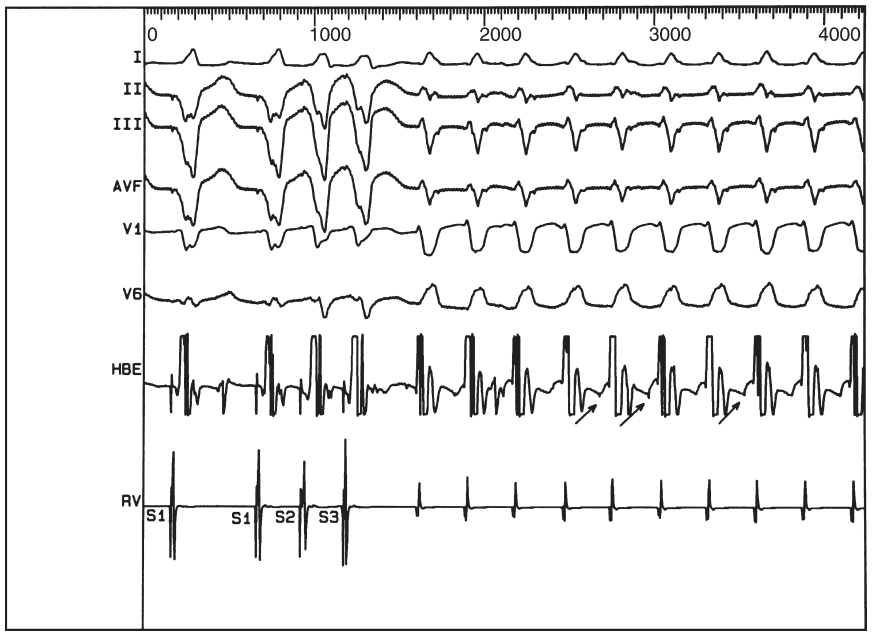
The regular SVT degenerates into atrial fibrillation as noted by the HRA recording. Atrial fibrillation is associated with a wide QRS complex morphology, consistent with a right-sided AP. Further, a distinct His bundle deflection no longer precedes each ventricular depolarization during the wide QRS complex arrhythmia. Thus, this represents either a preexcited tachycardia or VT, and not aberrancy. When one considers all the data, the most likely single diagnosis is AV reentry that degenerates into atrial fibrillation, producing a preexcited tachycardia.

Figure 5–20A



This patient with cardiomyopathy presents to the emergency room with hypotension and a history of syncope. A simultaneous 12-lead electrocardiogram is taken and the leads from top to bottom are I, II, III, aVR, aVL, aVF, and V_1 to V_6. The tachydardia spontaneously terminated. What is the differential diagnosis?

Figure 5–20B



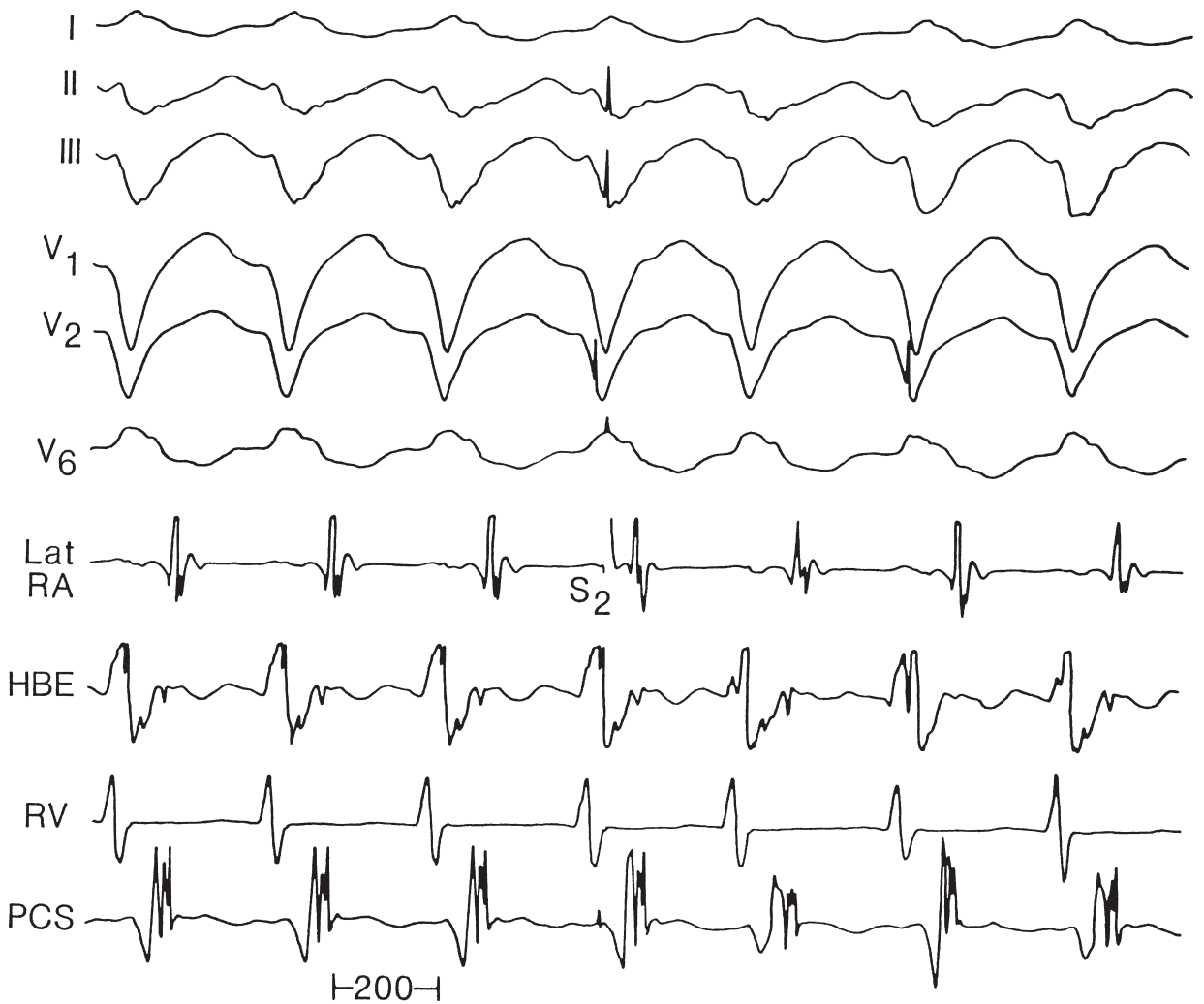
Explanation:

Careful analysis of lead I in Fig. 5–20*A* shows VA dissociation, a subtle finding in this tracing. There is an LBBB pattern during tachycardia that is nearly identical to that seen in sinus rhythm. These observations suggest bundle branch reentrant VT.

Figure 5–20*B* was obtained during EP testing in this patient. During ventricular drive, a second PVC initiated sustained VT with an identical ECG morphology as recorded in Fig. 5–20*A*. Note that there is VA dissociation. More important, His bundle deflections (*arrows*) occur prior to each QRS complex. The HV interval was slightly longer than that recorded during sinus rhythm. This patient had bundle branch reentrant VT. The typical EP findings are inducibility at EP study, sometimes requiring the unusual pacing protocol of a fast paced rate followed by a pause and then introduction of one or more extrastimuli. This theoretically produces divergence in refractoriness in the HPS and ventricle, allowing for retrograde block in the right bundle branch, transseptal conduction to the left bundle branch, and then reexcitation of the right bundle branch for initiation of tachycardia. Each QRS complex is preceded by a His potential with an HV interval typically equal to or greater than the HV measured in sinus rhythm. VA dissociation is the rule. In fact, this arrhythmia often occurs in patients with atrial fibrillation. Importantly, since the ventricle is activated over the right bundle branch, a "typical" LBBB morphology may be seen. This is well illustrated in Fig. 5–20*A* in which the patient has an underlying LBBB pattern that is nearly identical to that occurring during tachycardia on the left-hand portion of the figure.

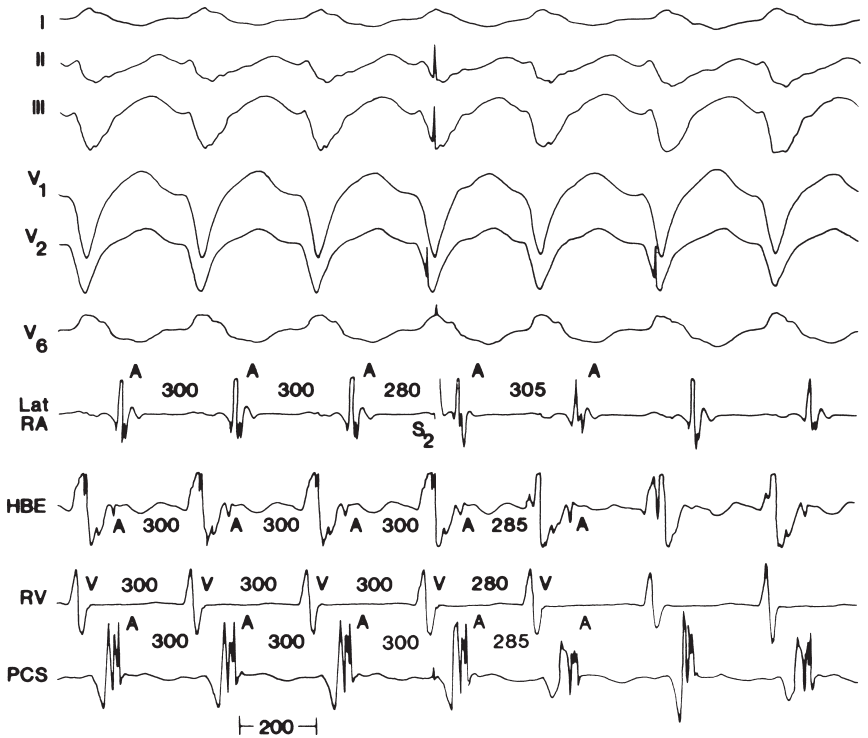
Bundle branch reentry occurs almost exclusively in patients with dilated cardiomyopathies, ischemic or nonischemic, and the QRS duration during tachycardia is often >160 milliseconds. Although ablation of the right bundle branch may prevent tachycardia in some of these patients, these individuals also have poor left ventricular function and may have other sustained ventricular arrhythmias. This should be evaluated very carefully before accepting right bundle branch ablation as a sole therapy for a particular patient.

Figure 5–21A



This tracing was obtained at EP study in a patient with a history of wide QRS complex tachycardia. The proximal CS recording electrode was positioned at the os of the CS. What is the diagnosis?

Figure 5–21B

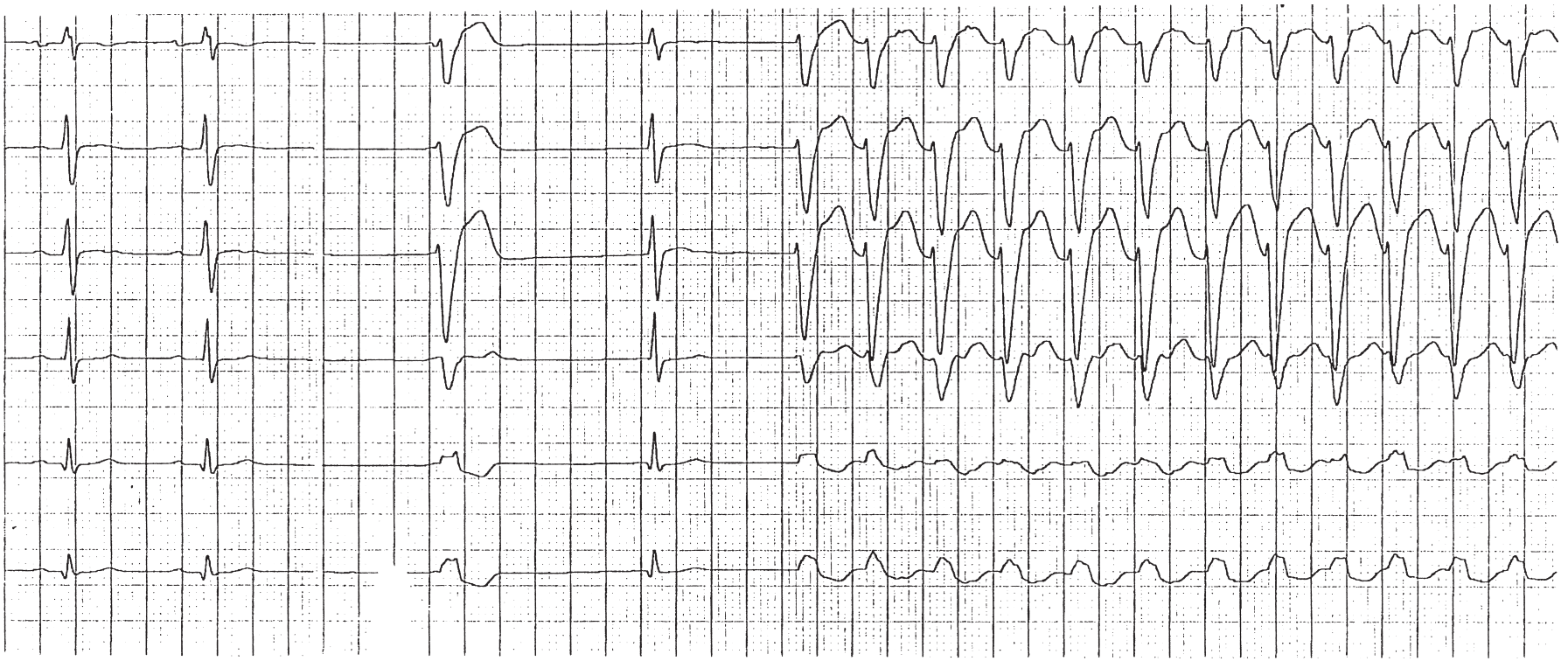


Explanation:

A regular wide QRS complex tachycardia is present with 1:1 VA association. Atrial activation sequence demonstrates earliest activity in the interatrial septum, as recorded on the His bundle lead. There is no obvious His bundle deflection preceding each QRS complex, and the His bundle electrode was recording a good His potential prior to initiation of tachycardia. Thus, the differential diagnosis is VT versus preexcited tachycardia. The QRS morphology demonstrates an atypical LBBB pattern that is consistent with either diagnosis. The tachycardia cycle length is 300 milliseconds, and a 20-millisecond premature atrial stimulus is introduced in the lateral right atrium. This premature complex does not affect septal activation as noted by a lack of change in the AA interval on the His bundle electrogram. However, the PAC shortens the VV interval, with an identical QRS morphology. This confirms AV activation over an AP during tachycardia. Although VT has been eliminated, the mechanism of tachycardia requires further analysis of this tracing.

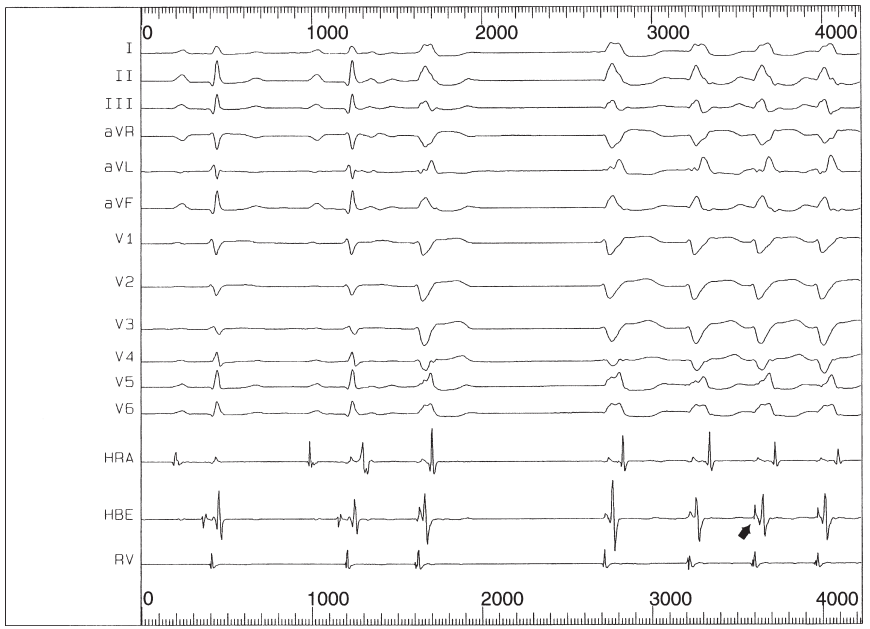
The preexcited tachycardia could be antidromic reentry utilizing a right free wall AP for anterograde conduction and the normal VA conduction system for retrograde activation, AP–AP tachycardia using a septal AP for retrograde conduction, bystander AP conduction during arrhythmias such as AV node reentry, and septal atrial tachycardia, or nodoventricular reentry. Careful measurement of the His bundle AA interval following shortening of the VV interval shows a decrease from 300 to 285 milliseconds. Atrial preexcitation of the ventricle without affecting the septal atrial sequence eliminates nodoventricular reentry. Likewise, "reset" of a septal atrial tachycardia would not occur since the initial atrial activation sequence was unperturbed by the PAC. AV node reentry with bystander participation is also excluded. The preexcited ventricular response of only 20 milliseconds (280 milliseconds) produces a shortening of the subsequent septal AA interval. Although preexcitation of the atrium during AV node reentry can occur, the PI is >90 milliseconds. In this case the PI would be only 20 milliseconds, too short for AV node reentry. The final differential diagnosis is true antidromic tachycardia utilizing the normal VA conduction system for retrograde activation versus retrograde conduction over a second AP located in the septum. This patient had antidromic tachycardia, the most common diagnosis with this presentation.

Figure 5–22A



A 15-year-old student presents with a 12-year history of palpitations. They have been getting progressively worse for the past few years, lasting for several hours. While being examined in the office, the patient began to have paroxysms of tachycardia and a simultaneous six-lead electrocardiogram was obtained. From top to bottom ECG leads are V_1 to V_6. What EP observations can be made, and what is the differential diagnosis?

Figure 5–22B



Explanation:

In Fig. 5–22*A*, the initiating and tachycardia QRS complexes appear almost identical. Since there is no atrial activity preceding the initiating beat, VT was highly suspect. The third QRS complex is similar to the tachycardia morphology and does not originate from the atrium. This also suggested the possibility of VT. However, contrary to this diagnosis is the presence of a typical LBBB morphology during tachycardia, and the presence of normal ventricular function by echocardiography. This essentially excludes bundle branch reentry, but does not exclude idiopathic septal VT. Therefore, one had to consider alternative possibilities, which were uncovered at EP study. At EP study (Fig. 5–22*B*), spontaneous onset of tachycardia is remarkably similar to that noted in Fig. 5–22*A*. The His bundle deflection is easily identified with the first two sinus complexes. The third QRS complex is caused by a PAC and has the morphology of the tachycardia. It appears that a His bundle deflection is present within the early portion of the ventricular electrogram; retrograde atrial activity is noted. The fourth QRS complex has a similar morphology and is also associated with retrograde atrial activity, but no initial atrial activity. This is followed by the tachycardia, and the last two QRS complexes are associated with a His bundle deflection occurring shortly after activation of the ventricle (*arrow*).

This situation is clarified in Fig. 5–22*C*. During tachycardia a PAC was introduced in the high right atrium. The septal atrial electrogram was refractory at this time as noted on the proximal His bundle lead. However, the premature atrial complex preexcited the ventricle (376 milliseconds on RV lead) and did so with an identical QRS complex as noted during tachycardia. This confirmed the presence of anterograde conduction over an AP (see earlier discussion). This observation was made during onset of atrial pacing during tachycardia. At other parts of the study the presence of an atriofascicular pathway was confirmed and the final diagnosis was atriofascicular reentry and not VT. Reanalysis of Fig. 5–22*A* suggests that the onset of tachycardia in this patient may have occurred secondary to automaticity in the atriofascicular tract. These pathways are known to have retrograde block. It is impossible to exclude a ventricular premature complex originating very close to the insertion site of the right bundle branch in this patient.

In summary, because of the extremely unusual initiation of tachycardia and the relative rarity of the phenomenon in this patient, the ECG diagnosis was more difficult than usual. One should always include atriofascicular reentry in the differential diagnosis of a typical LBBB tachycardia in a patient with normal ventricular function. This case emphasizes the need for EP evaluation in such patients.

Figure 5–22C

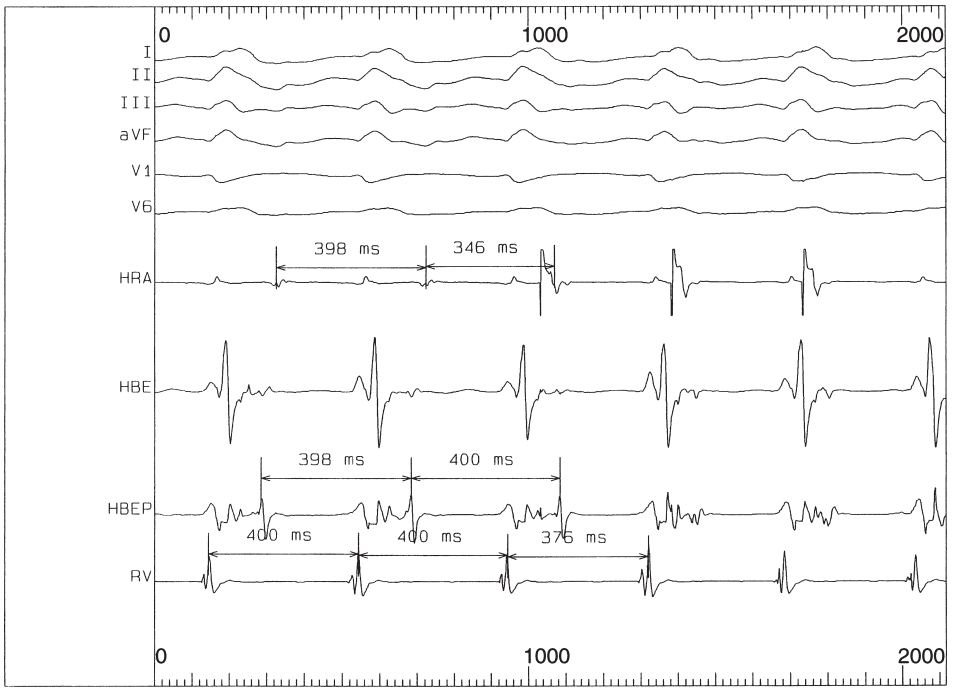

0
1000
2000
I
II
III
aVF
V1
V6
398 ms
346 ms
HRA
HBE
398 ms
400 ms
HBEP
400 ms
400 ms
375 ms
RV
0
1000
2000

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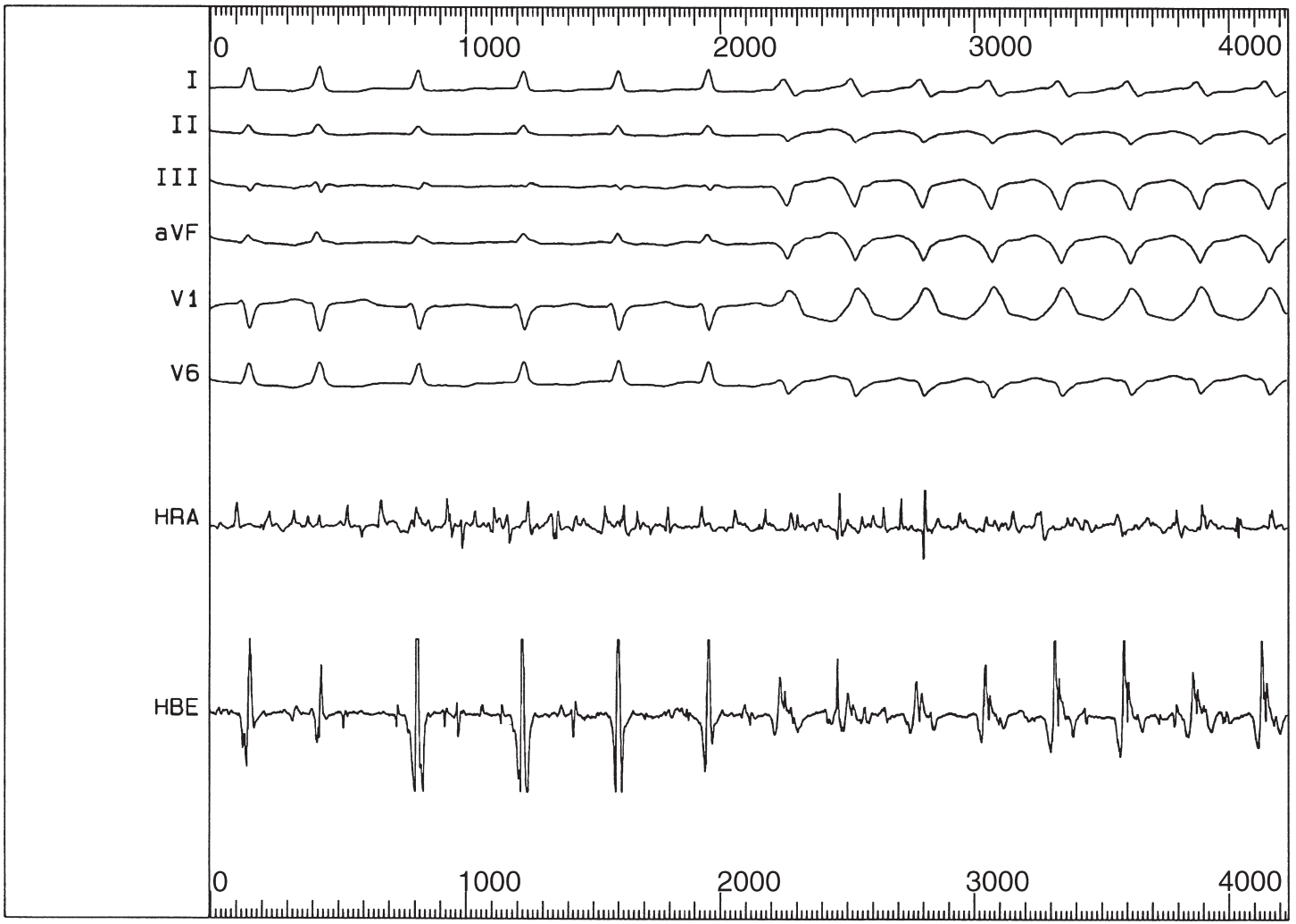
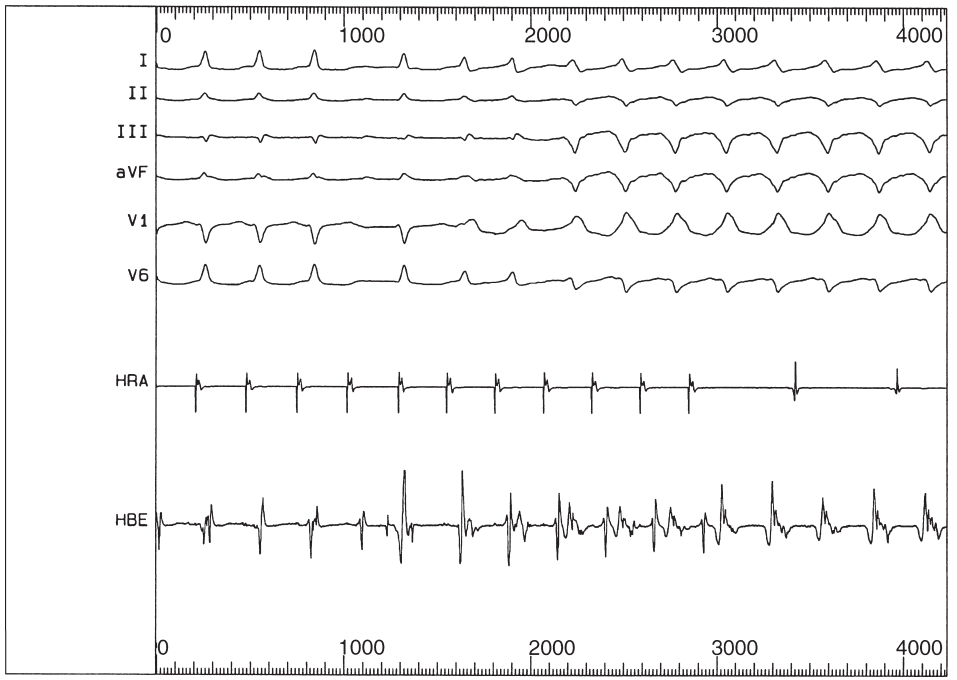
Figure 5–23A



The trace shown occurred at EP study. How many tachycardias are present and what are they?

Figure 5–23B

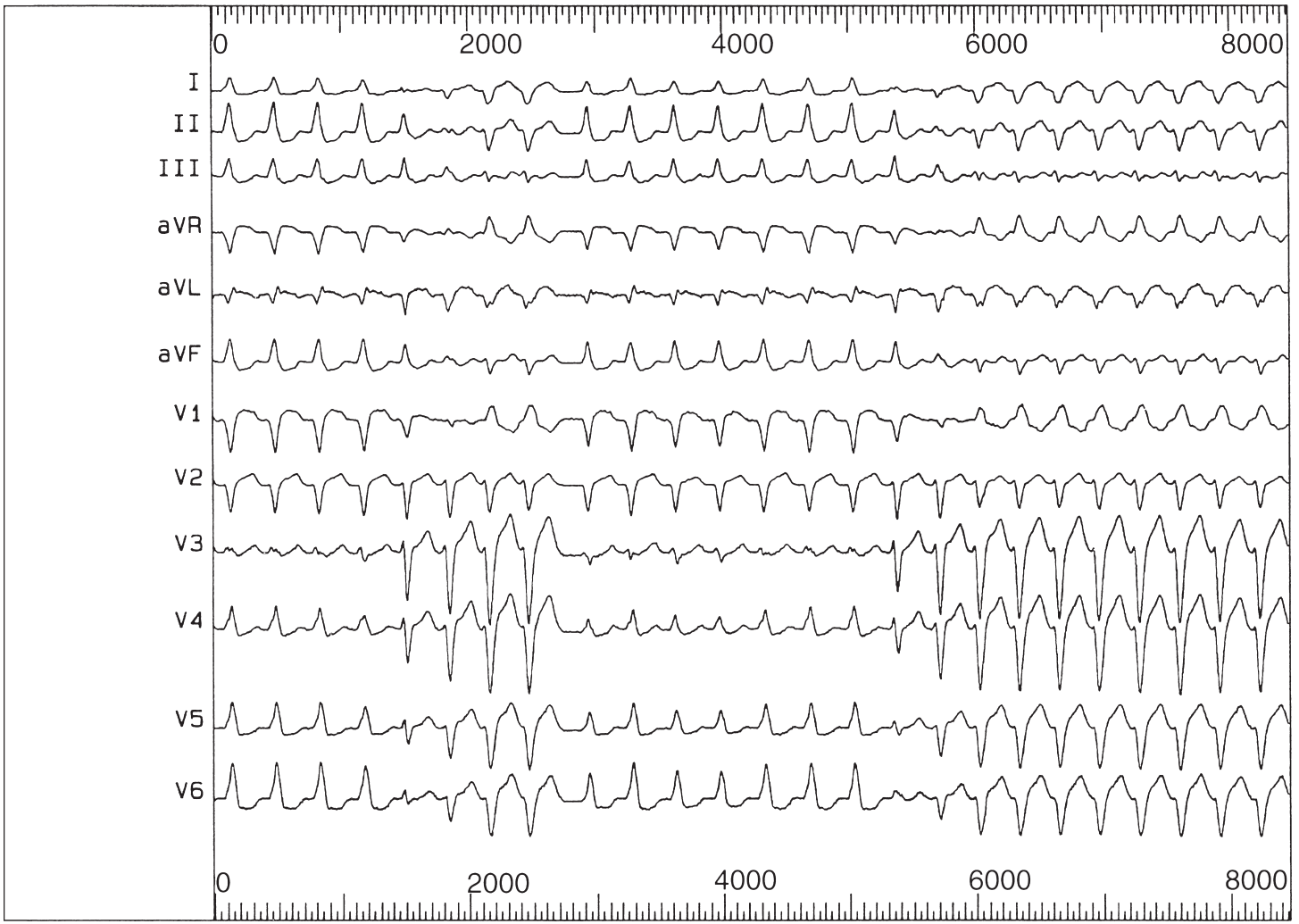


Explanation:

In Fig. 5–23*A*, the underlying rhythm is atrial fibrillation as documented on the HRA recording. There are both narrow and wide QRS complexes. The narrow QRS complexes have an irregularly irregular rhythm consistent with atrial fibrillation. A His bundle electrogram is not well visualized in all complexes, but is present in several beats. The wide QRS complex tachycardia is a regular rhythm and not consistent with atrial fibrillation. Favoring VT is the appearance of the first wide QRS complex without any preceding long–short sequence, which usually occurs with aberrancy.

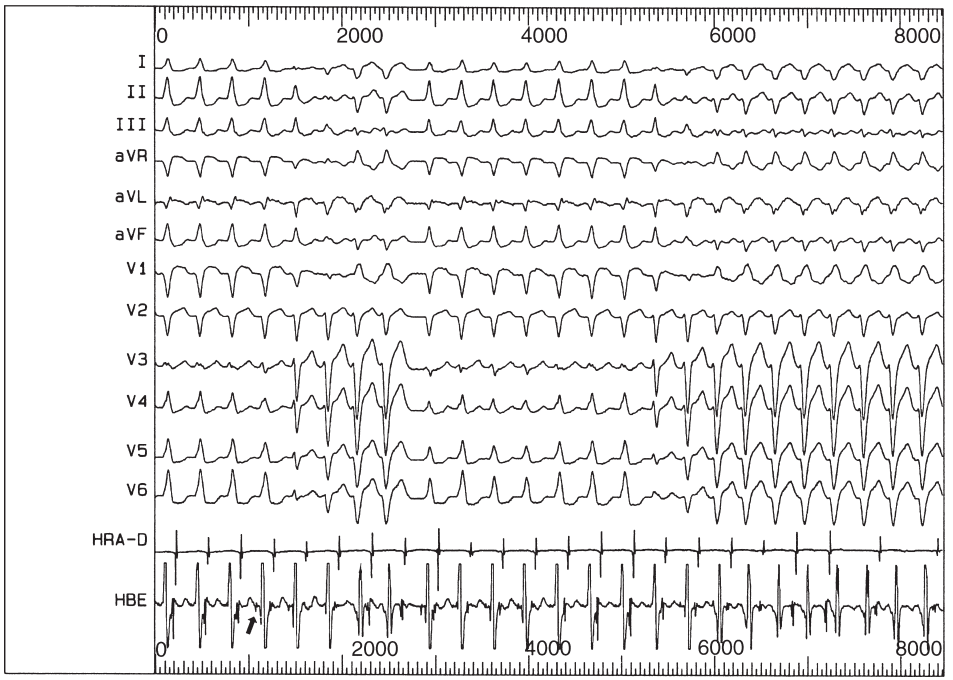
The RBBB morphology in V_1 also suggests VT, although a preexcited QRS morphology cannot be excluded. To diagnose a preexcited tachycardia one would have to suggest the very unlikely possibility of transition of atrial fibrillation to a regular tachycardia, and conduction over an AP only during the regular faster rhythm. Figure 5–23*B* shows initiation of VT with VA dissociation on the right-hand portion of the figure. Atrial pacing very infrequently induces VT. Note that VT occurs when a critical preceding ventricular rate was present, similar to what happened during atrial fibrillation. This is a very unusual variety of tachycardia-induced tachycardia (see discussion of Fig. 5–15).

Figure 5–24A



This elderly woman had a history of nonsustained wide QRS complex tachycardia and underwent EP evaluation. The simultaneous 12-lead ECG shown was recorded at EP study. What is the diagnosis?

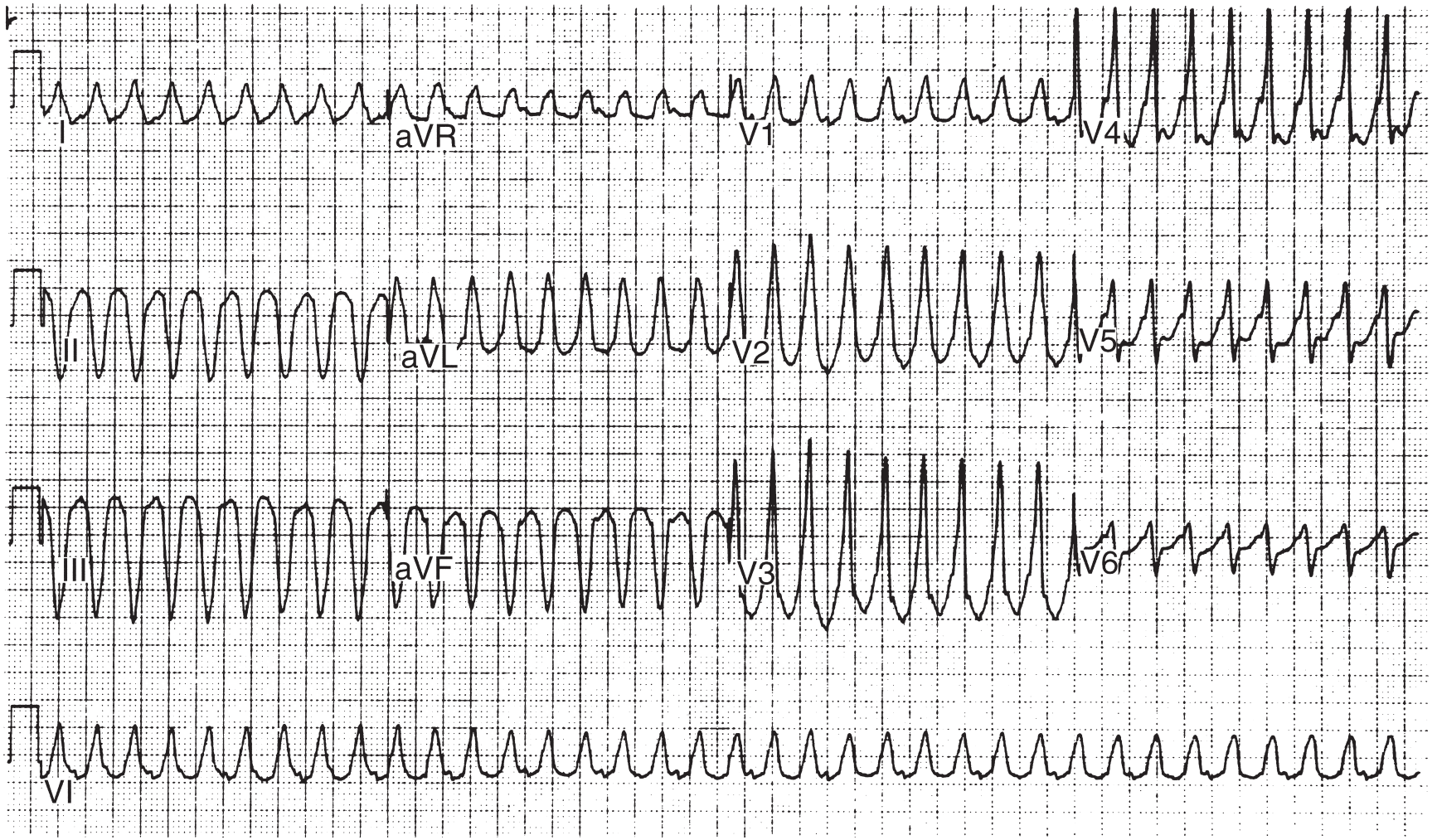
Figure 5–24B



Explanation:

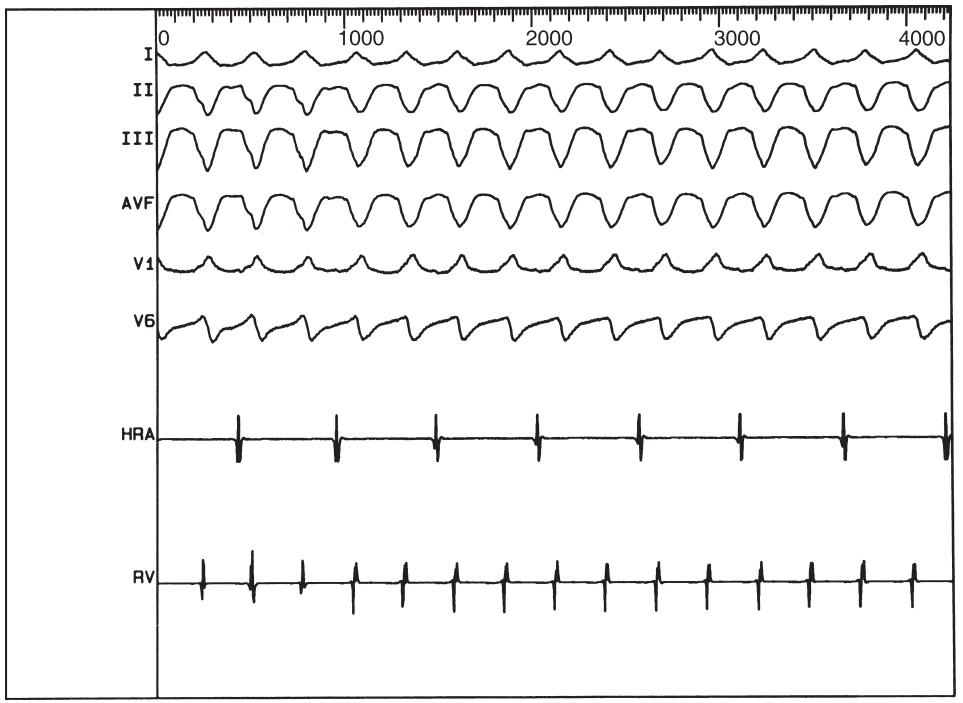
The intracardiac electrograms are included in Fig. 5–24*B*. Narrow and wide QRS complex tachycardias are present. The first four QRS complexes are representative of the narrow QRS tachycardia, and a His bundle deflection is present before ventricular activation (*arrow*). The wide QRS tachycardia has an RBBB morphology as noted in V_1. Note that fusion beats occur during the transition from the narrow to wide QRS tachycardia. A His bundle deflection is not routinely present before ventricular activation in the wide complex tachycardia, which is VT. Further, after VT terminates the SVT, VA block is also present as noted at the end of the tracing. The rates of tachycardia are similar, but the wide QRS tachycardia is clearly faster. EP testing confirmed AV node reentry for the narrow QRS tachycardia. The VT typically occurred during AV node reentry and is an example of tachycardia-induced tachycardia. It usually terminated AV node reentry, but was nonsustained at all times. The VT could also occur without preceding AV node reentry and, therefore, elimination of AV node reentry would not be expected to prevent recurrences of VT in this patient.

Figure 5–25A



This patient presented with a history of syncope. At EP study the 12-lead ECG shown was recorded. What is the most likely diagnosis? Is antidromic tachycardia using a left free wall AP part of the differential diagnosis?

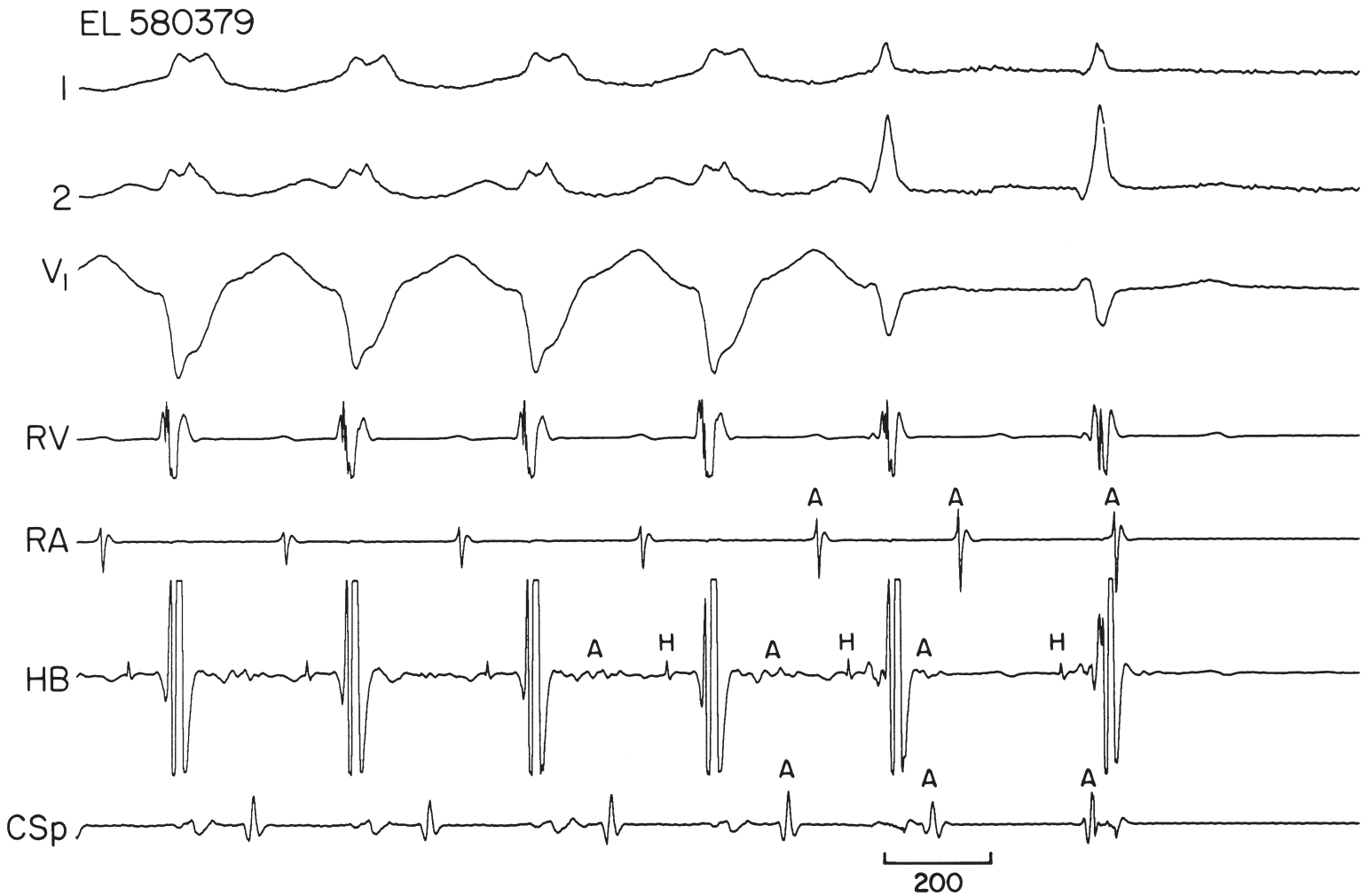
Figure 5–25B



Explanation:

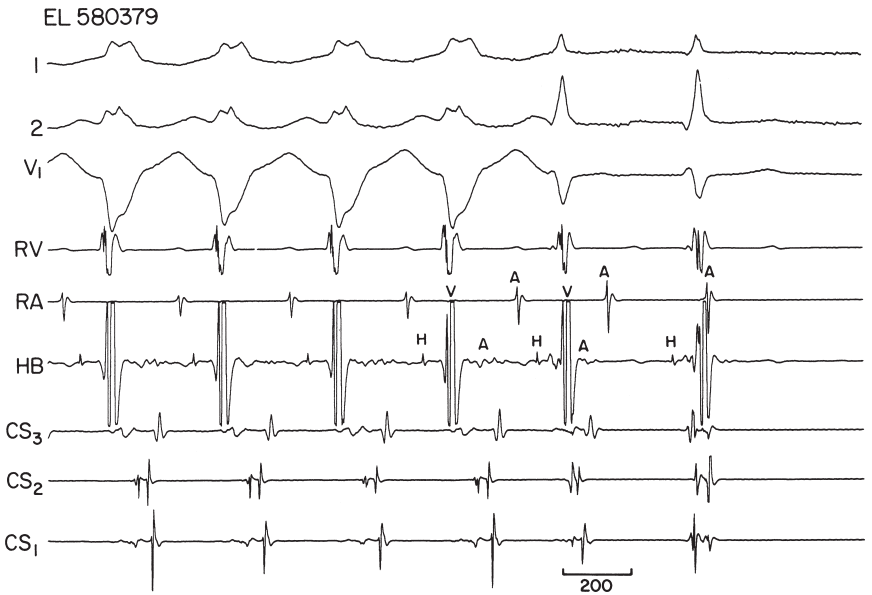
Figure 5–25*B* includes the intracardiac electrograms. Note that the RBBB tachycardia is associated with 2:1 VA conduction. In fact, this is clearly seen during careful inspection of the 12-lead ECG in Fig. 5–25*A* (lead V_1). The most likely diagnosis is VT. Antidromic tachycardia should not be considered since 1:1 VA association must be present in this situation.

Figure 5–26A



The record is from a young patient with recurrent palpitations that always stop spontaneously after a couple of minutes. What is the tachycardia mechanism and why does it terminate spontaneously? Lead CSp is the coronary sinus electrode at the orifice of the CS.

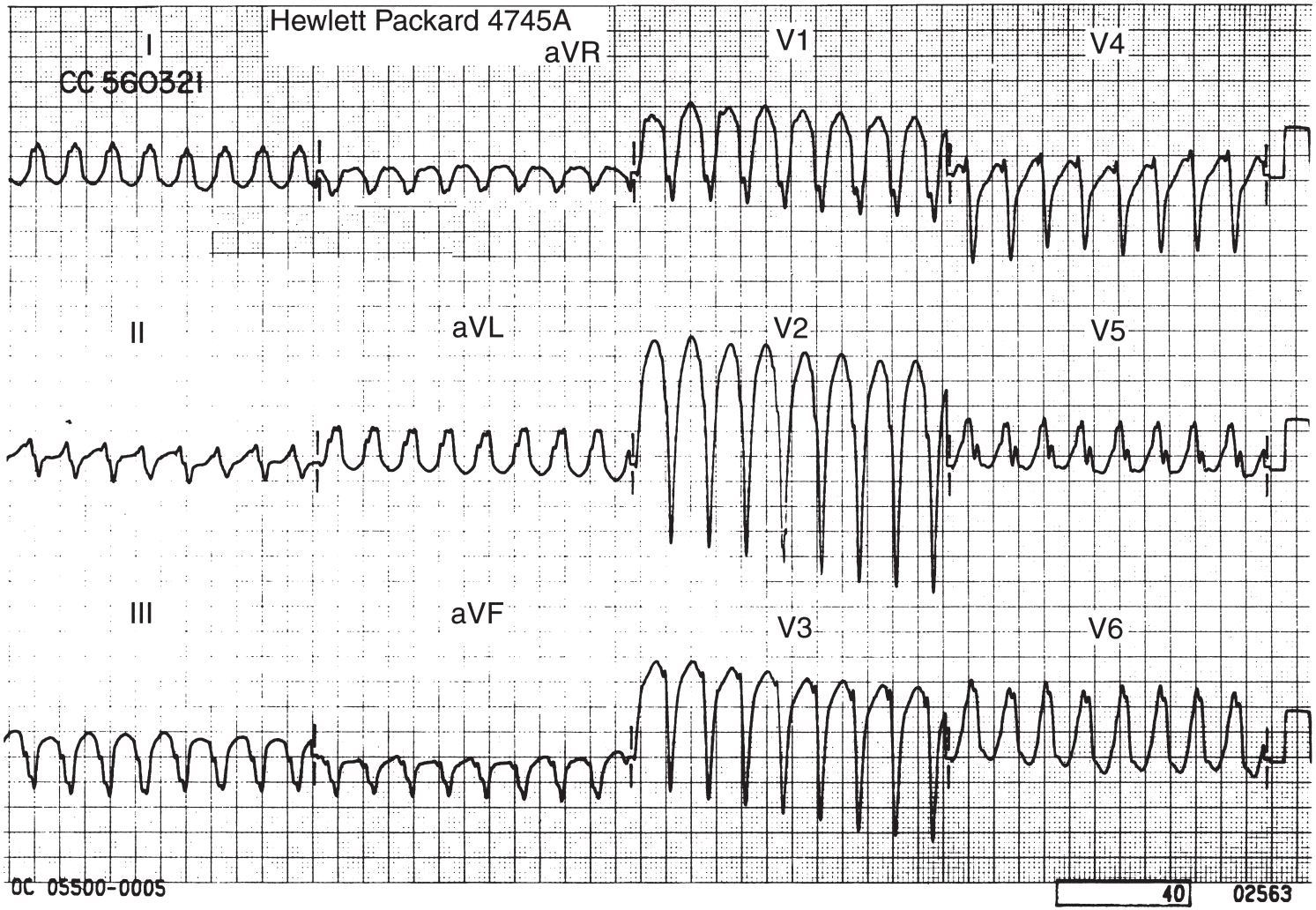
Figure 5–26B



Explanation:

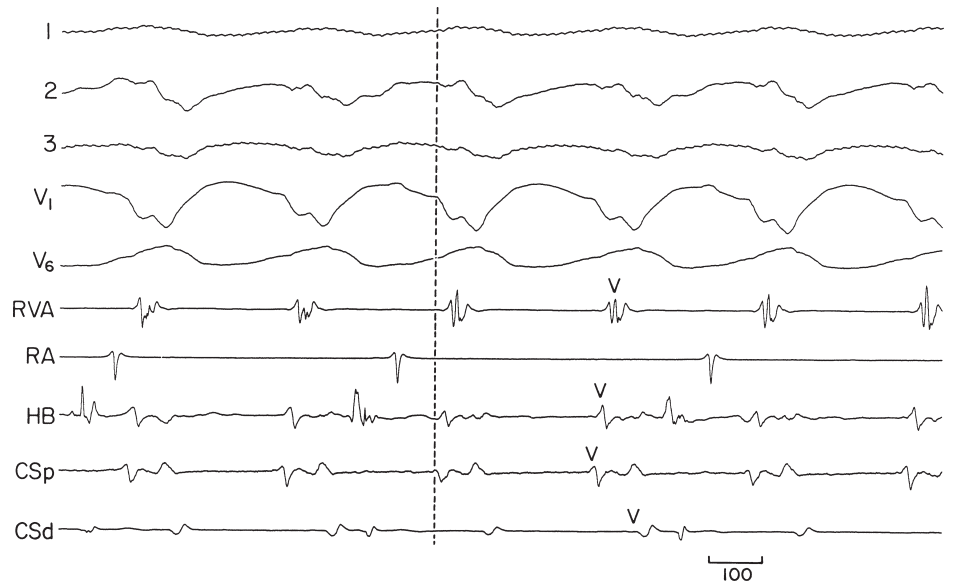
In essence, this wide QRS tachycardia abruptly normalizes with subsequent shortening of the VA interval (same atrial activation sequence) and blocks after the next cycle. Figure 5–26*B* is the same record with the addition of other CS leads with CS_3 at the orifice and CS_2 and CS_1, respectively, more distal. However, the diagnosis should be made readily without the additional leads. The tachycardia starts as a wide QRS tachycardia with an eccentric atrial activation sequence with earliest activation in the mid CS (CS_2). The typical LBBB morphology with the normal HV interval during tachycardia would favor a diagnosis of AV reentry with LBBB aberration (Tables 1–7 and 1–8). The QRS abruptly normalizes with the fifth cycle with resolution of the functional LBBB during the AV reentrant tachycardia. Normalization of the QRS occurs without preceding change in the cycle length, suggesting that the mechanism of normalization is not cycle length dependent. The mechanism is likely either progressive shortening of the left bundle branch refractory period at the newly established faster heart rate during tachycardia or sudden loss of retrograde transseptal concealed conduction into the left bundle branch. Regardless of the mechanism of normalization, it is associated with abrupt shortening of the VA interval, a reflection of the shortened tachycardia circuit resulting from elimination of transseptal conduction time from the RV to the LV. The decreased VA interval produces a shorter subsequent AA interval, which is analogous to an atrial extrastimulus or premature atrial depolarization. The "premature" atrial depolarization blocks over a fast AV node pathway and conducts over a slow AV node pathway. Tachycardia terminates spontaneously after a single AV node echo, which encounters anterograde refractoriness in the slow pathway. This is an interesting example of interplay of tachycardia mechanisms that limits rather than facilitates maintenance of tachycardia.

Figure 5–27A



This electrocardiogram was recorded in a 30-year-old patient with a history of tachycardia of sudden onset with no specific provocation. There was no clinical evidence of heart disease and the echocardiogram was normal. What is the mechanism of tachycardia?

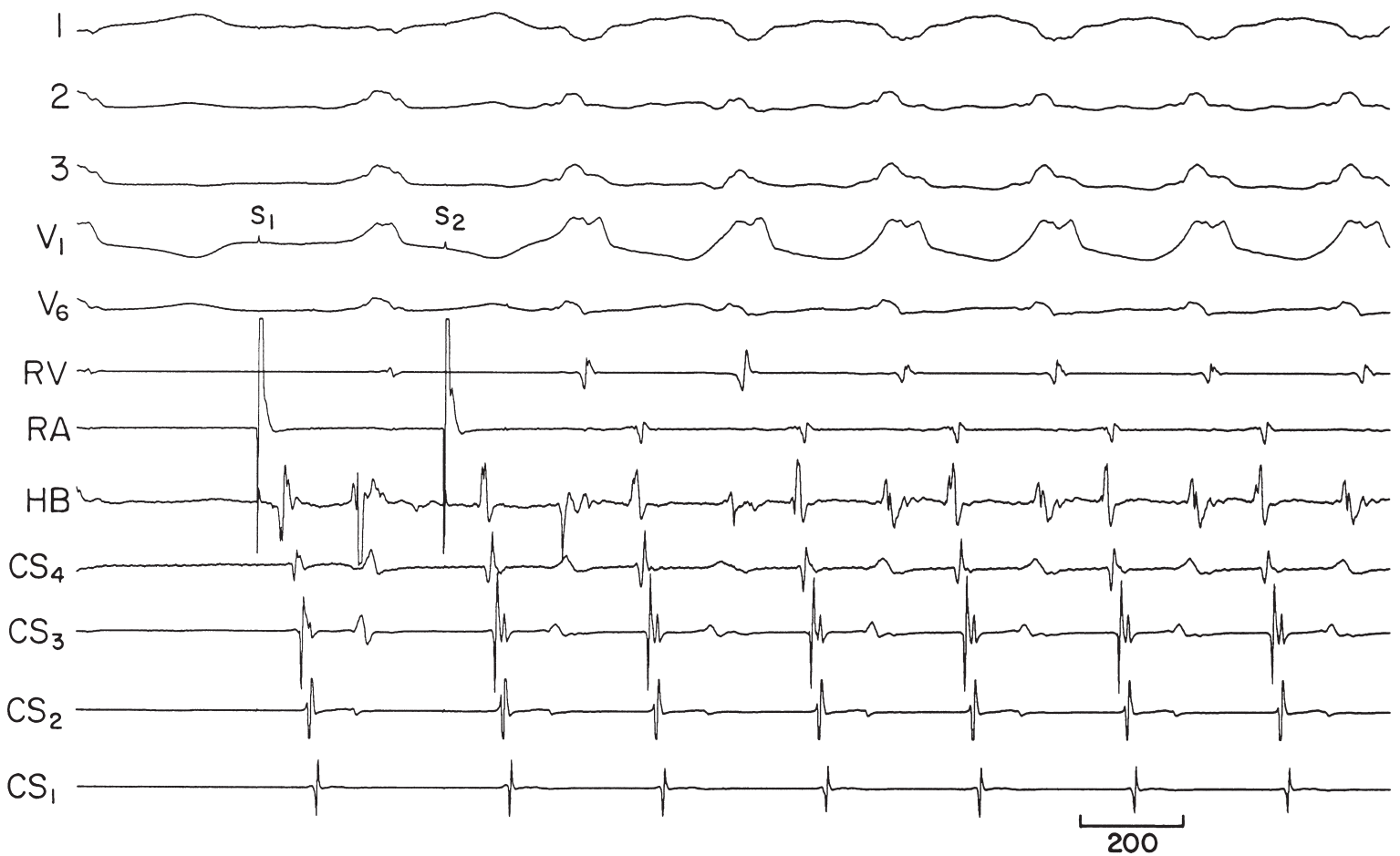
Figure 5–27B



Explanation:

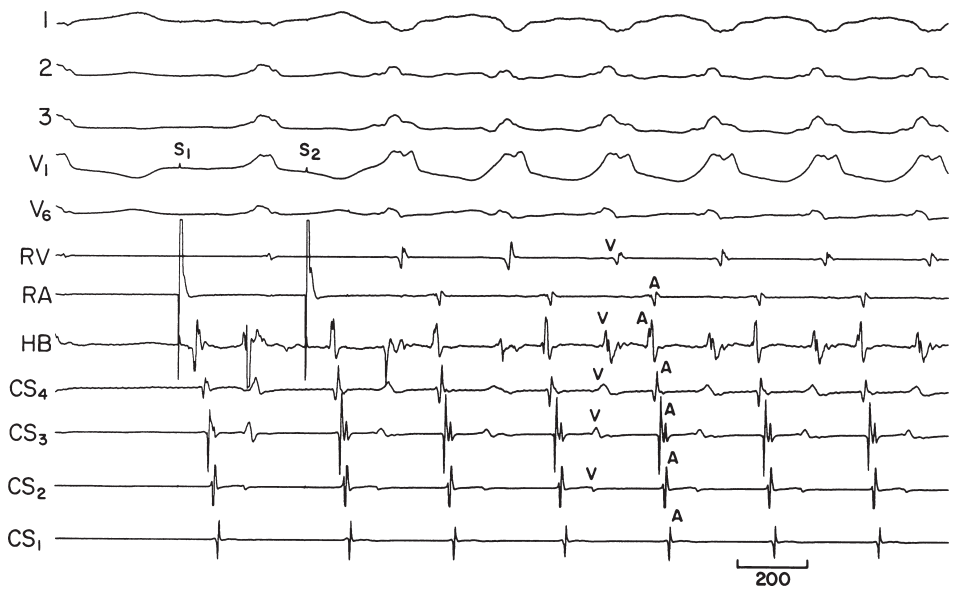
The 12-lead electrocardiogram showed a regular wide QRS tachycardia with LBBB morphology. P waves were not clearly discernible. In a young patient without heart disease, a mechanism of SVT with aberration or preexcited tachycardia should be initially considered. However, this young patient clearly had VT as is evident on the intracardiac records. There was no evidence of cardiac disease in this patient after extensive investigation and this tachycardia would be classified as "idiopathic" VT. The most common type of idiopathic VT originates in the RV outflow region and is associated with LBBB and inferior axis. This morphology is unusual, suggesting an origin of VT in the "posteroseptal" region (earliest ventricular activation in Fig. 5–27*B* is recorded in CSp, the electrode at the orifice of the CS). LBBB morphology, especially related to areas other than the RV outflow region, should raise a suspicion of arrhythmogenic right ventricular dysplasia for which no evidence could be found in this individual.

Figure 5–28A



The record is from a 13-year-old boy with a history of recurrent tachycardia of sudden onset. The 12-lead electrocardiogram in sinus rhythm showed a left lateral preexcitation pattern. CS_4 is in the orifice of the coronary sinus and CS_3 to CS_1 are progressively more distal. S_1 is the last of a drive of eight right atrial paced beats at cycle length 600 milliseconds and S_2 is an atrial extrastimulus. What is the mechanism of tachycardia?

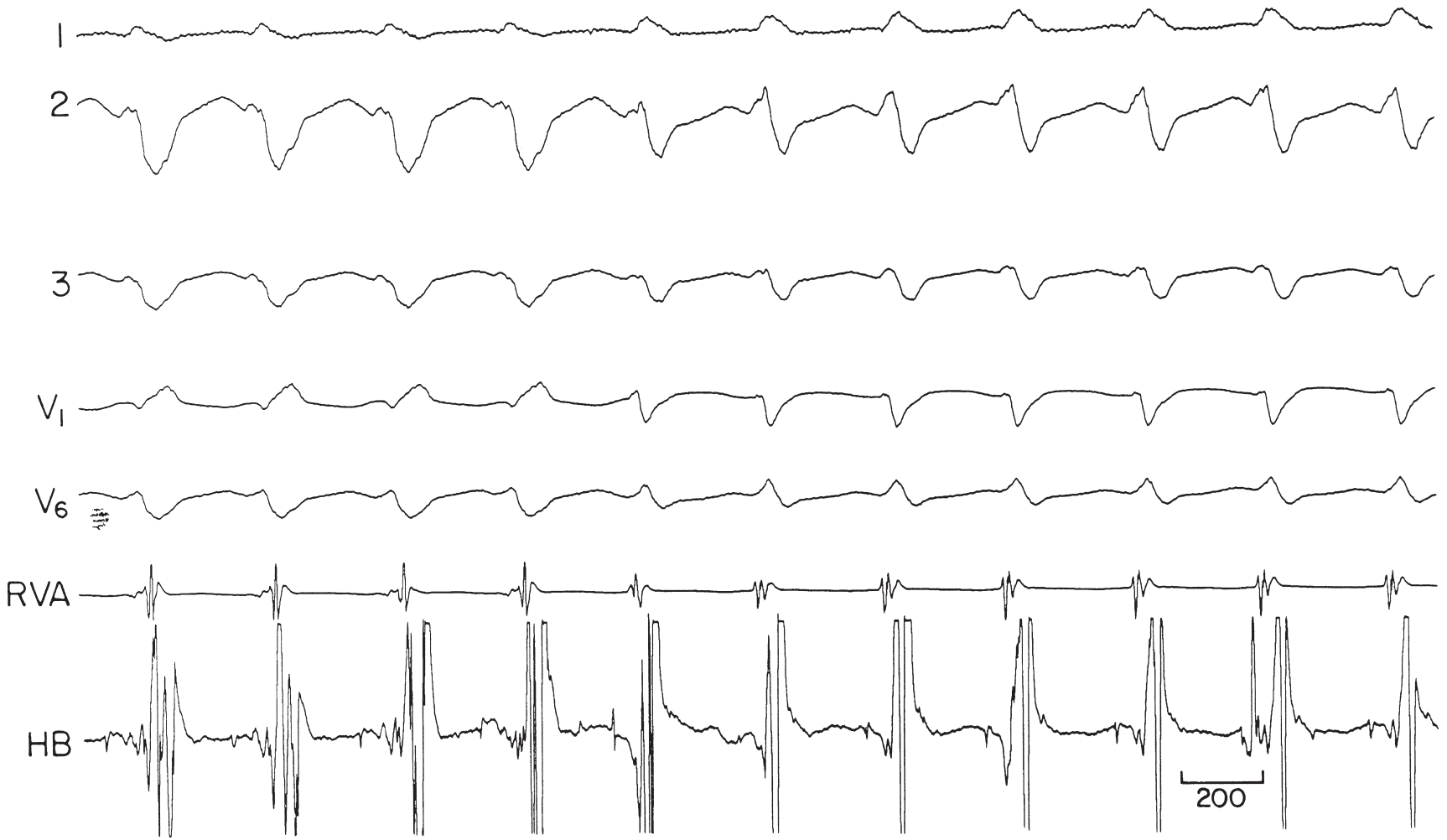
Figure 5–28B



Explanation:

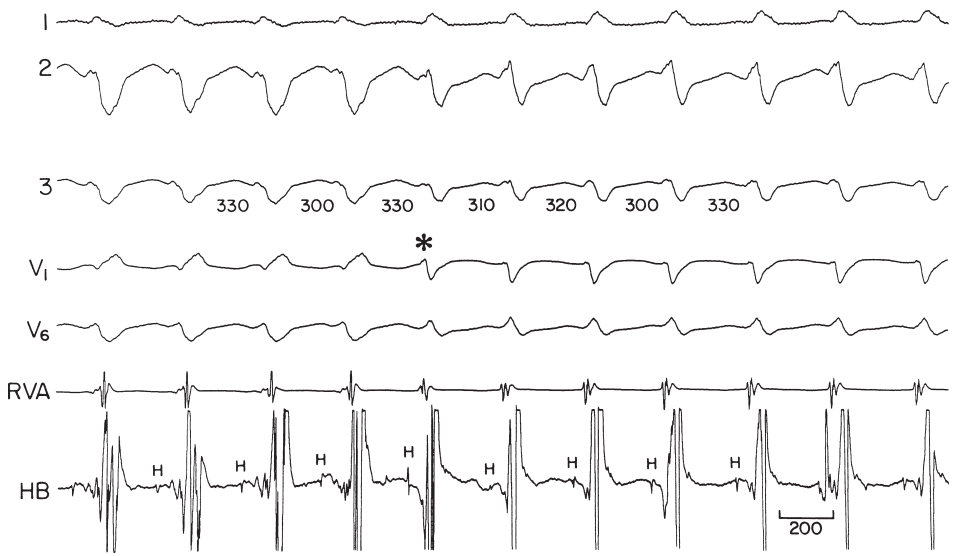
The QRS morphology during established tachycardia is identical to that observed during atrial pacing and reflects conduction over a left lateral AP. Supporting this is earliest ventricular activation on the available leads at the distal CS. Atrial activation sequence begins in the His bundle region and we can therefore classify this tachycardia as a preexcited tachycardia with a concentric atrial activation sequence (Table 1–9). This tracing alone does not establish whether the pathway was part of the tachycardia circuit. Both atrial tachycardia and AV node reentry were ruled out by demonstrating that relatively long coupled atrial premature extrastimuli conducted over the AP and advanced and reset tachycardia. Accepting that the AP was part of the tachycardia circuit, the major dilemma now is to determine whether the retrograde limb is the normal AV conduction system or a second retrogradely conducting septal AP. This must be established by pacing techniques, a useful one relying on the observation that PVCs delivered into tachycardia near the His bundle region will preexcite the atrium at much longer coupling intervals than pacing at the right ventricular apex when an anteroseptal pathway is the retrograde limb, and will require shorter coupling intervals when the AV node is the retrograde limb. This fundamental physiologic observation is merely based on proximity of the pacing site to the entrance of the excitable gap in the circuit. It is thus clear that the diagnostic possibilities in Table 1–9 can only be distinguished in this record for certainty with dynamic observations and not from this record alone.

Figure 5–29A



The record is taken from a 73-year-old man who presented with wide QRS tachycardia several years after uncomplicated anterior MI. The tachycardia was consistently induced by two extrastimuli that resulted in prolongation of the VH interval after the extrastimulus. The HV during sinus rhythm was 55 milliseconds and was approximately 40 milliseconds during tachycardia. Will ablation of the right bundle branch cure this tachycardia?

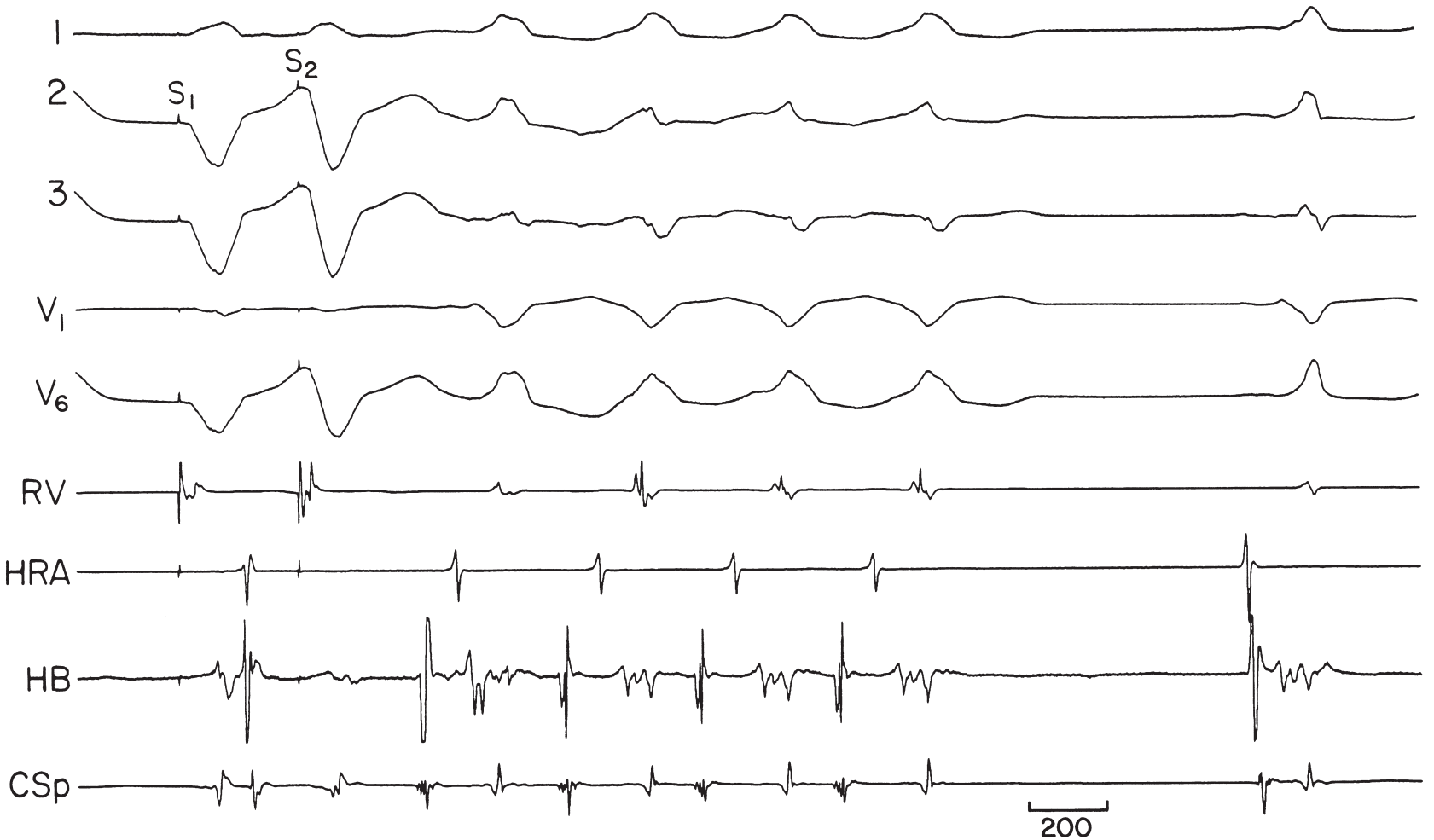
Figure 5–29B



Explanation:

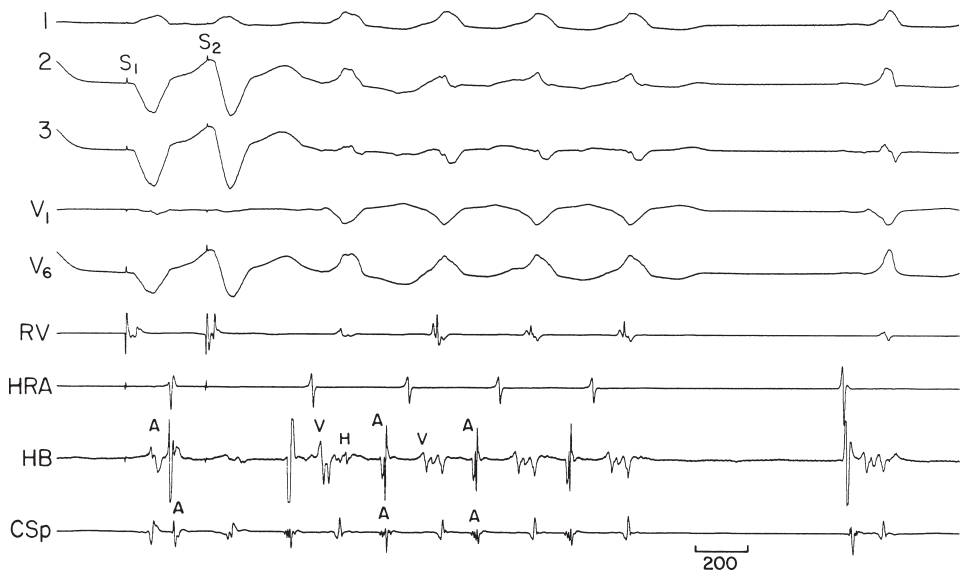
The therapeutic decision is dependent on the mechanism of tachycardia. The diagnosis of VT is supported by the clinical setting and the AV dissociation evident on the His bundle channel (atrial electrogram clearly preceding next to last V). It is tempting to diagnose bundle branch reentry (see Fig. 5–20). However, a distinct change in morphology of this tachycardia from right bundle type to left bundle type is observed at the asterisk without any change in cycle length of the tachycardia or the apparent HV interval. This transition (Table 1–3) is critical to mechanism as transition from right bundle to left bundle in a single cycle without change in tachycardia rate is virtually inconceivable for bundle branch reentry. Intramyocardial or distal fascicular reentry with secondary penetration of the His bundle is a much more tenable mechanism to explain this observation. This must always be considered in a situation that appears to be bundle branch reentry. Ablation of the right bundle branch will not cure this tachycardia.

Figure 5–30A



The record is taken from a young woman with a WPW pattern on the 12-lead electrocardiogram compatible with a right anteroseptal AP. S_1 is the last of a drive of eight ventricular cycles from the right ventricular apex at cycle length 600 milliseconds and S_2 is a ventricular extrastimulus. What is the mechanism of tachycardia?

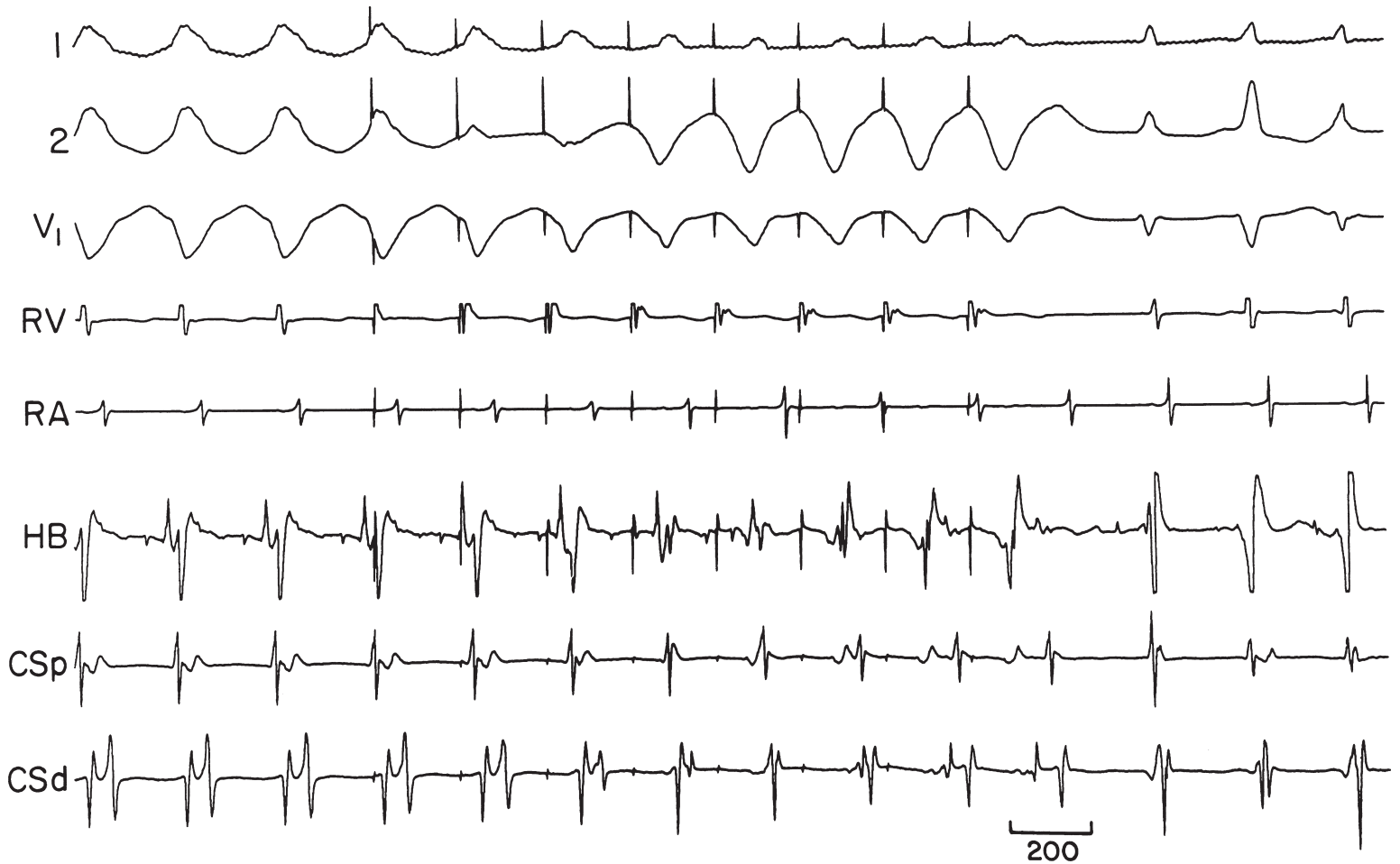
Figure 5–30B



Explanation:

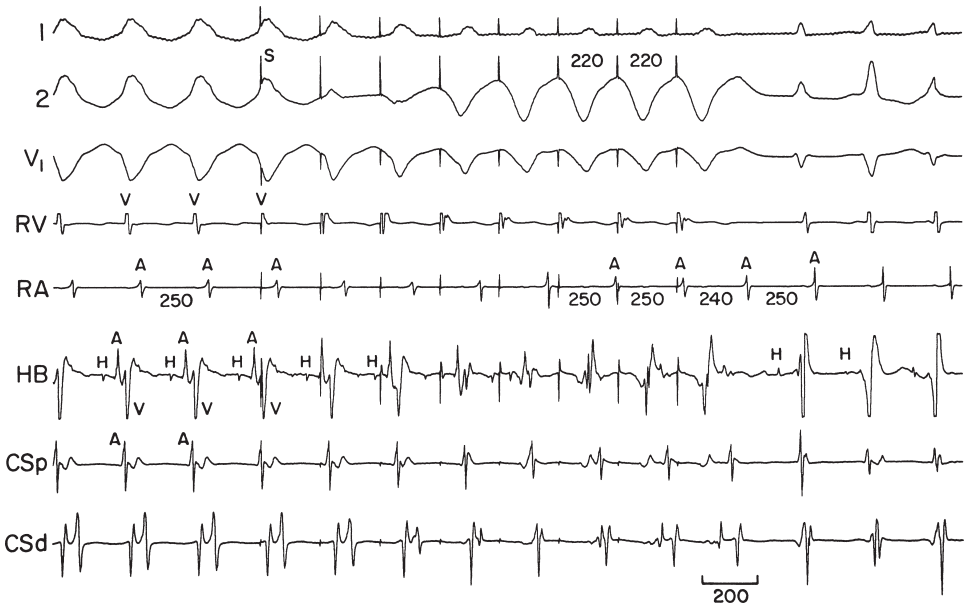
This can be considered a preexcited tachycardia since there is no H preceding the QRS and the QRS morphology is compatible with the preexcitation pattern seen in sinus rhythm. The issues related to this tachycardia were discussed in the explanation section of Fig. 5–28*B*. In this instance, a diagnosis of nonsustained antidromic tachycardia utilizing the normal AV conduction system as the retrograde limb can be made with reasonable certainty. S_1 conducts over the AP with a short VA interval and S_2 fails to conduct over the AP. Note that a retrograde His deflection is clearly observed after the first tachycardia cycle. The His merges into the V with a second cycle (His not seen) with shortening of the VA interval and proportional advancement of the next preexcited QRS. That is, V to His conduction is part of the circuit. Resolution of retrograde conduction delay in the HPS, here probably the right bundle branch, after the first cycle or two is not unusual at the onset of antidromic tachycardia.

Figure 5–31A



The record is from a 43-year-old woman with a history of paroxysmal tachycardia. Tachycardia of LBBB morphology was reproducibly induced by atrial extrastimuli attaining a critical AH delay. What happened after a burst of ventricular pacing was introduced into this tachycardia? CSp is the coronary sinus electrode at the orifice of the CS, and CSd is in the distal CS.

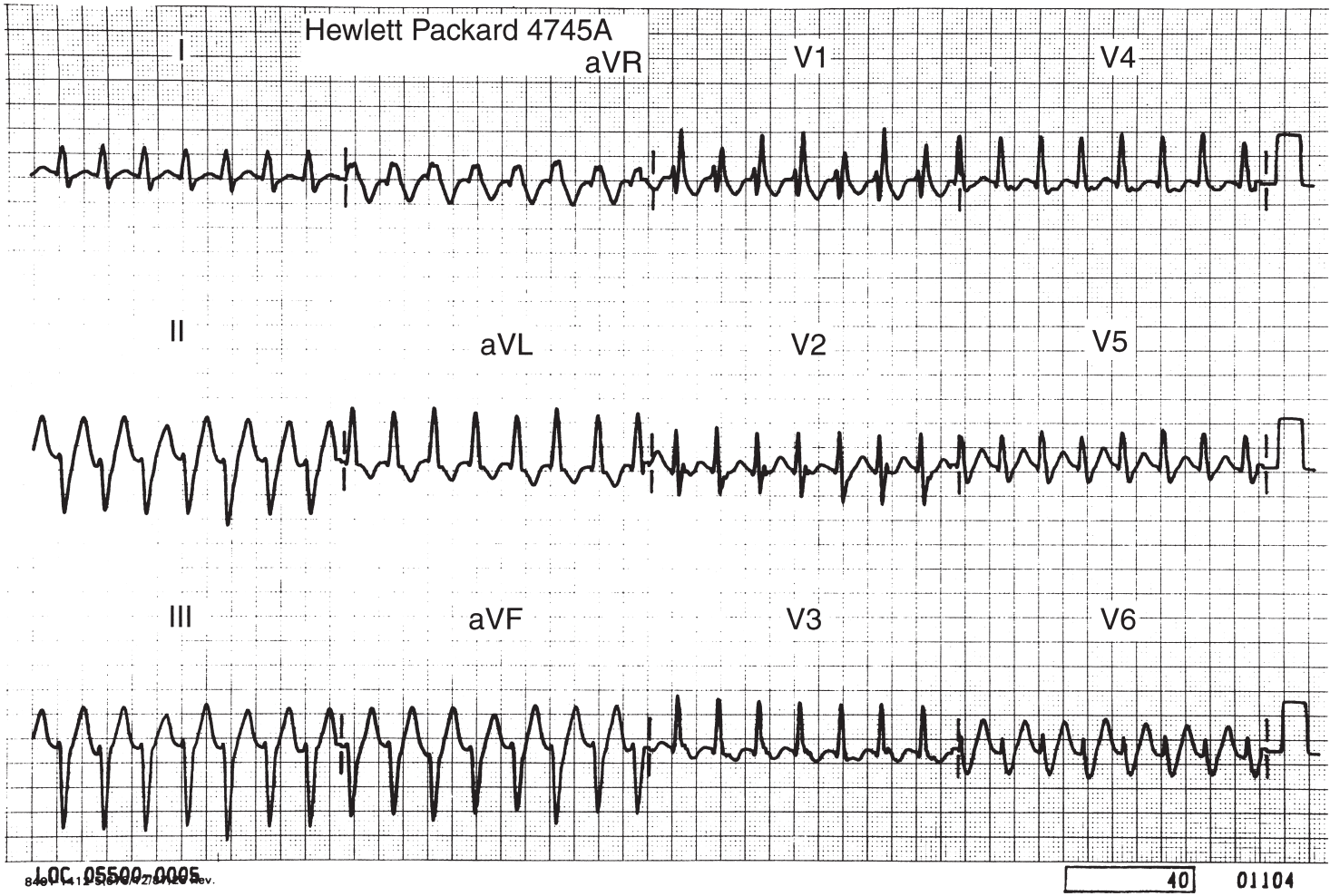
Figure 5–31B



Explanation:

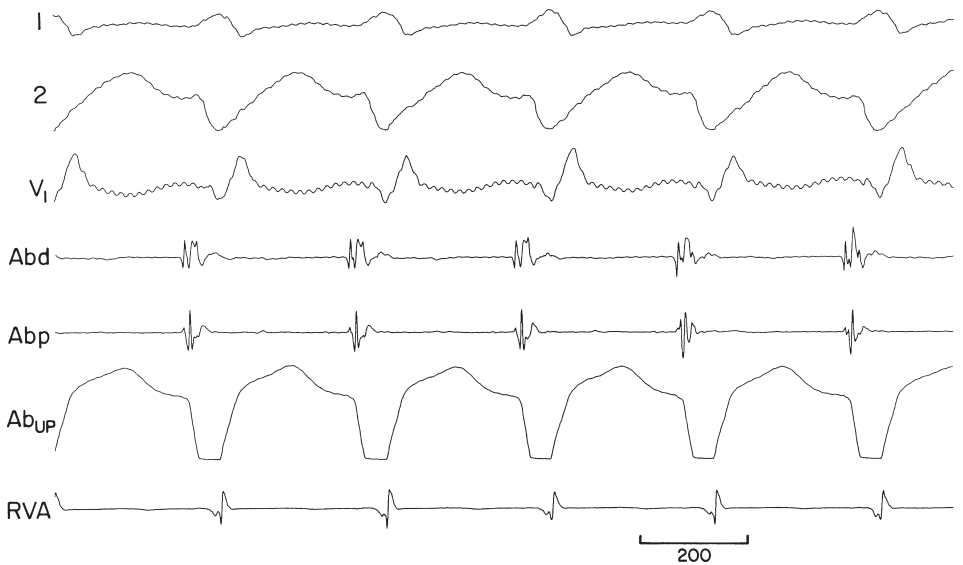
The clinical and EP presentation in this patient is typical for AV node reentry. Tachycardia has a relatively short cycle length of 250 milliseconds and it would not be unusual for acceleration-dependent LBBB to be observed in this instance. A burst of ventricular pacing at a slightly shorter cycle length (220 milliseconds) does not perceptibly alter the tachycardia mechanism, which continues with the identical cycle length but with a normal QRS after the burst. This would suggest that the mechanism of LBBB during this tachycardia is not simply acceleration dependent but related to concealed retrograde transseptal conduction that was altered by the ventricular pacing burst.

Figure 5–32A



The 12-lead electrocardiogram is recorded from a patient with a long-standing history of paroxysmal, well-tolerated tachycardia frequently converted in the emergency room with intravenous verapamil. There is no associated cardiac disease. What is the differential diagnosis of this tachycardia?

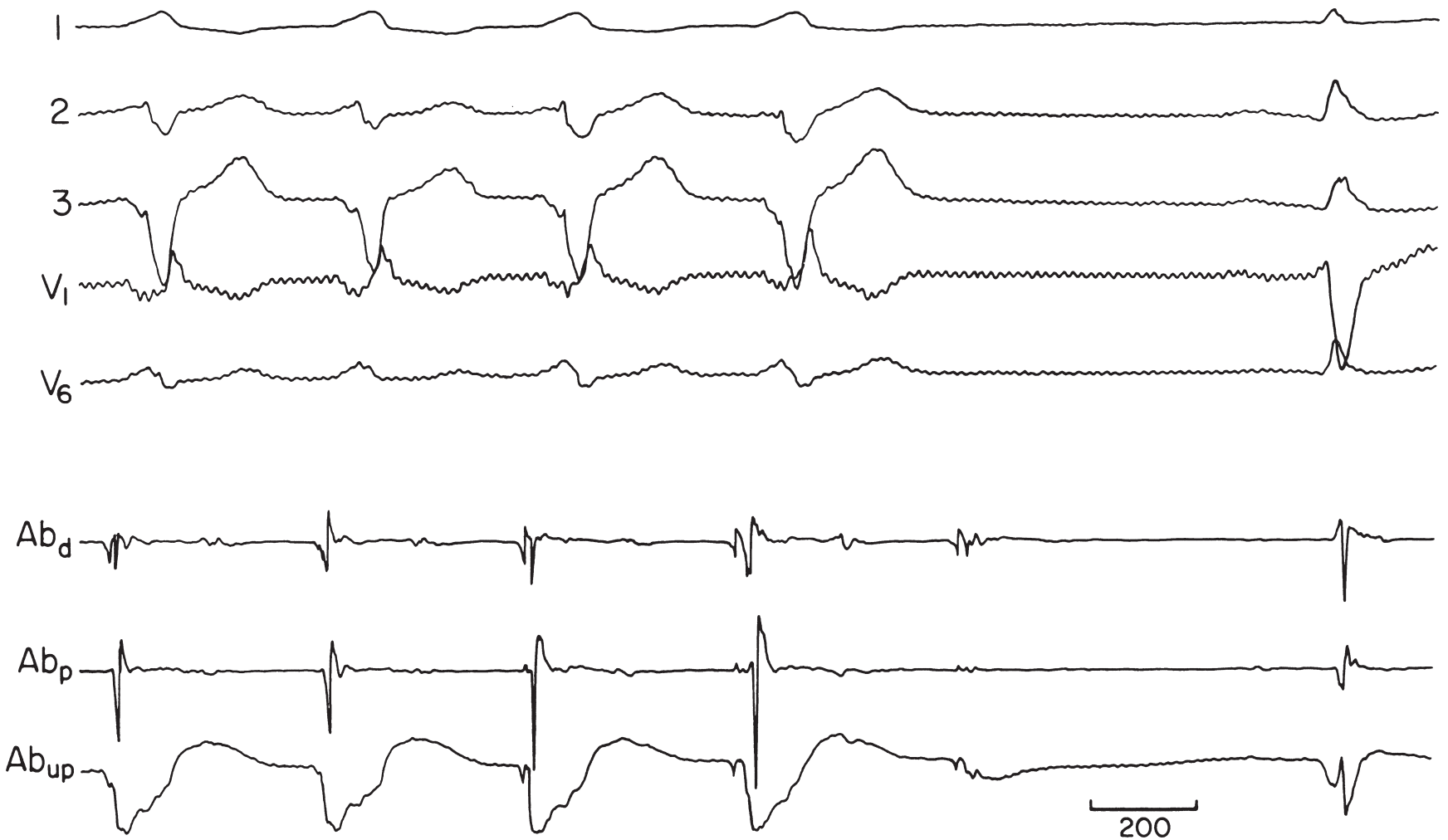
Figure 5–32B



Explanation:

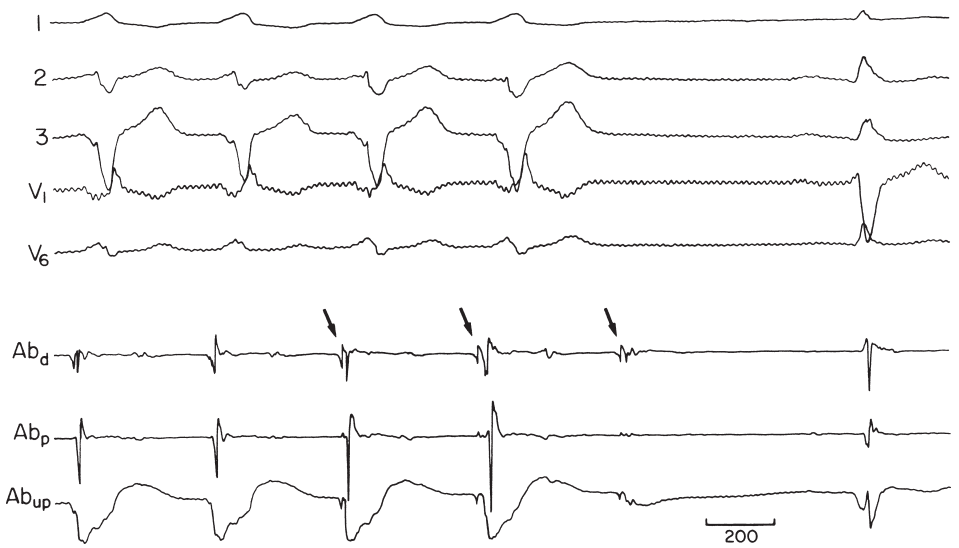
The clinical history and electrocardiogram would immediately suggest a diagnosis of paroxysmal SVT with aberrant conduction. There are, however, electrocardiographic features that do not support this diagnosis. First, the RBBB pattern is slightly atypical with a deep S wave in V_6. Second, there is striking left axis deviation in addition to the RBBB aberration, a pattern of bifascicular block that is distinctly unusual in paroxysmal SVT. Finally, there is a suggestion of AV dissociation best appreciated in lead V_3. This is a very typical electrocardiogram for patients with the most common type of idiopathic left VT (verapamil-sensitive VT). This tachycardia is believed to originate from reentry in the distal Purkinje system in the left ventricular apical septal region. The intracardiac record in Fig. 5–32*B* demonstrates a catheter (Abd) at the site of earliest ventricular activation during tachycardia recording a typical Purkinje-type spike preceding the QRS. The recording was made prior to successful ablation of tachycardia at this site. Abd, Abp, and Ab_{up} represent the distal, proximal, and unipolar distal poles of the ablation catheter, respectively. RVA is the right ventricular apex.

Figure 5–33A



This intracardiac record is recorded from the patient in Fig. 5–32 with mapping in the region of interest described in Fig. 5–32*B*. What has happened?

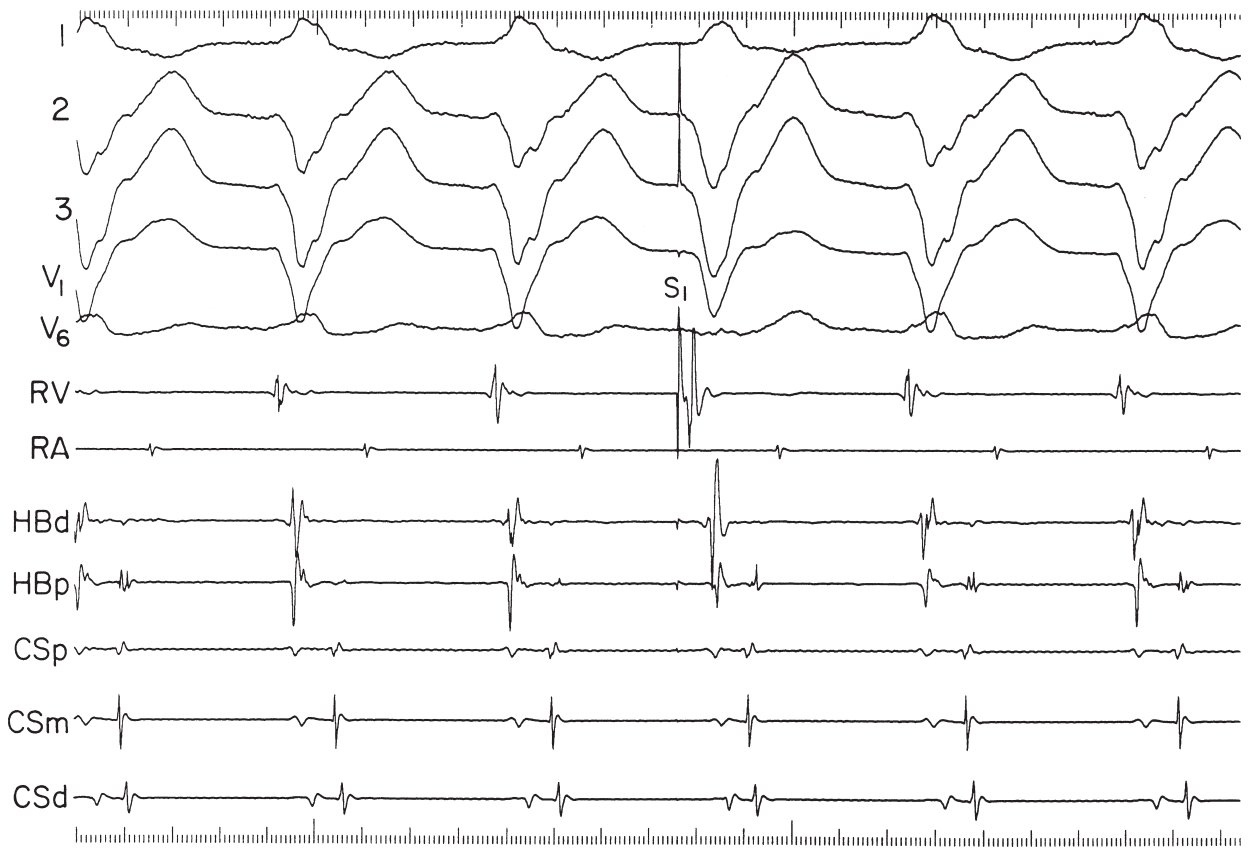
Figure 5–33B



Explanation:

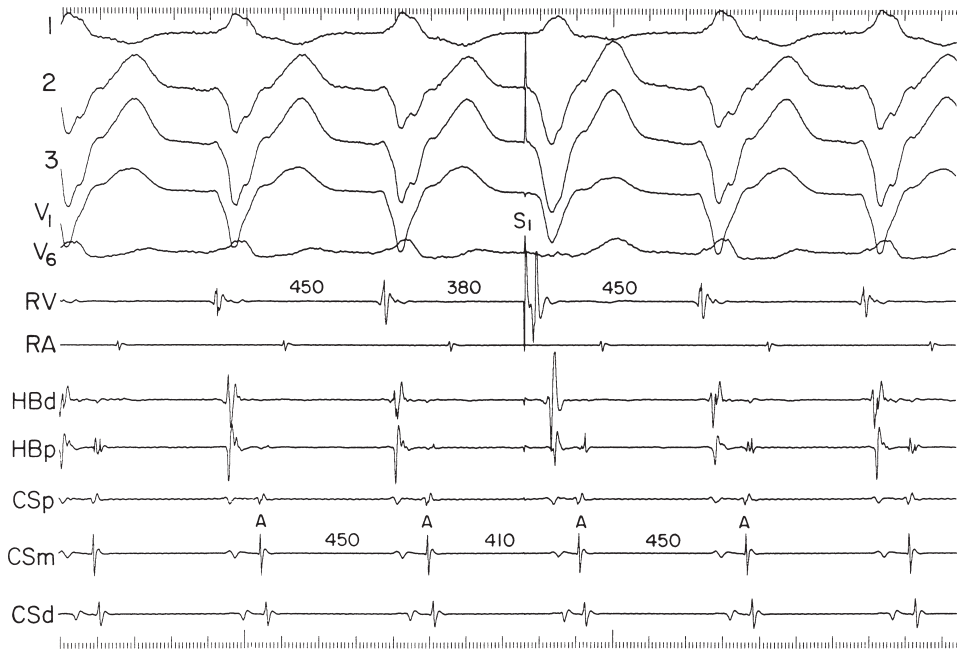
This tachycardia terminated during catheter manipulation in the region recording earliest ventricular activation during tachycardia preceded by the Purkinje spike (*arrows*). This tachycardia terminated spontaneously on several occasions during such maneuvering, probably due to catheter pressure in a region critical for sustaining tachycardia ("bump mapping"). The record would suggest that termination of tachycardia was related to conduction block between the Purkinje spike and the QRS.

Figure 5–34A



The intracardiac records are from a young woman without heart disease and a history of paroxysmal tachycardia. EP testing prior to tachycardia induction revealed a decremental, right free wall accessory AV pathway with the maximal preexcited morphology observed during atrial pacing identical to that observed in the recorded tachycardia in the figure. What is learned from the ventricular extrastimulus (S_1) delivered from the right ventricular apical region? HBd and HBp are His bundle records from the distal and proximal recording electrodes, respectively. CSp, CSm, and CSd represent coronary sinus recordings from the proximal to distal coronary sinus regions, respectively, with CSp near the orifice.

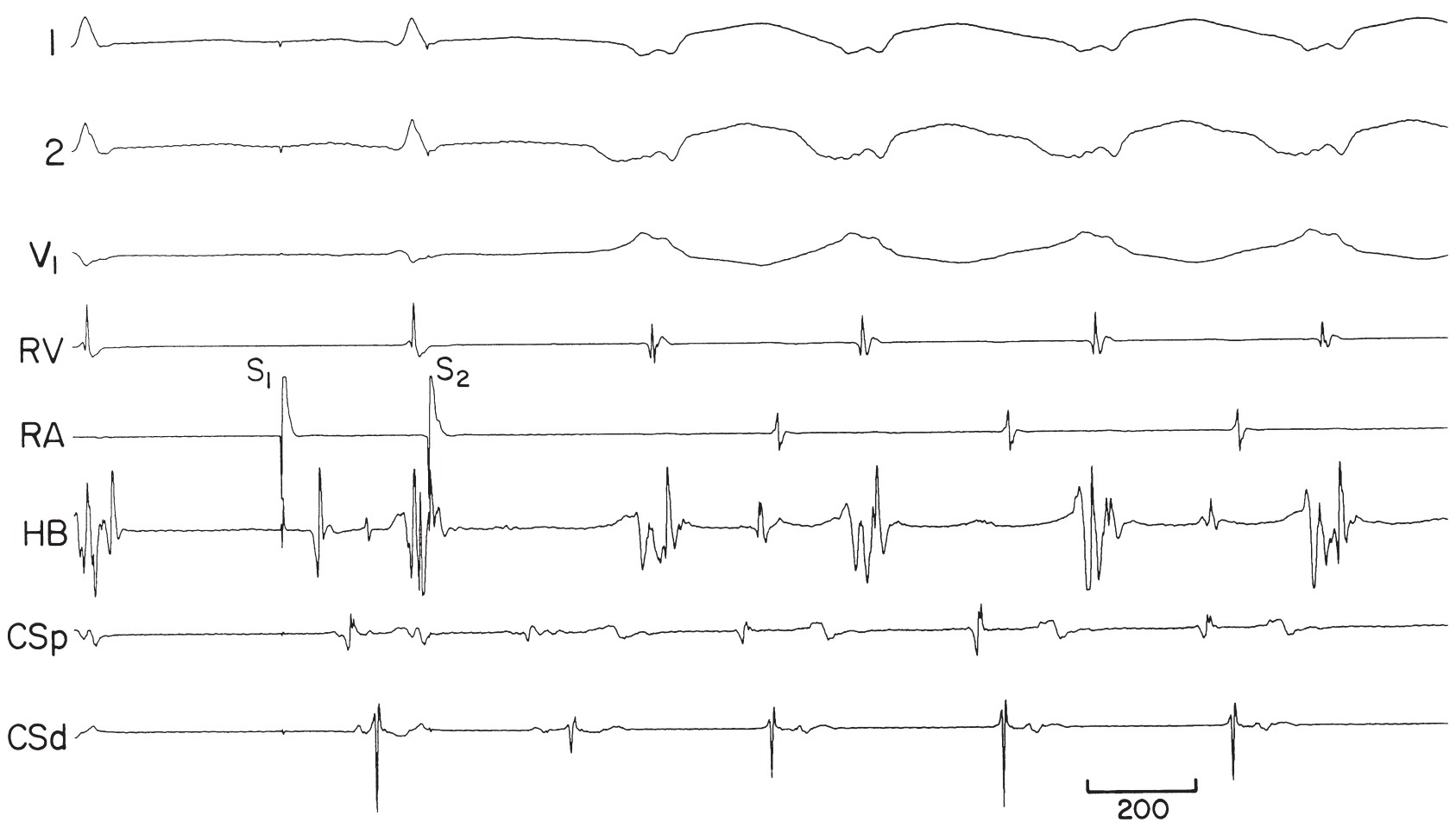
Figure 5–34B



Explanation:

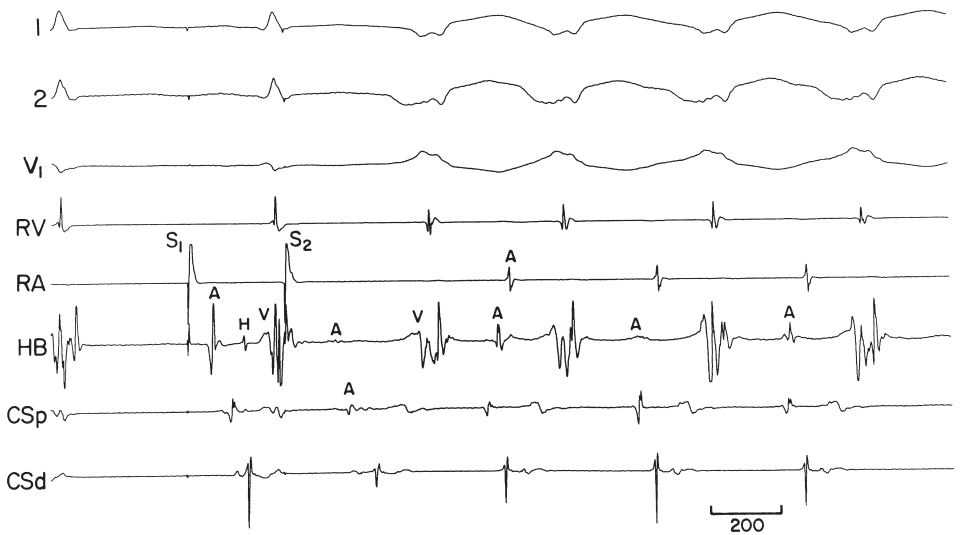
Since the tachycardia has the preexcited morphology observed during atrial pacing and no preceding His deflection, it can be considered to be a preexcited tachycardia. The atrial activation sequence is concentric (Tables 1–6 and 1–9). A relatively late-coupled PVC preexcites the atrium and resets the tachycardia. This virtually proves that the AP is part of the tachycardia circuit (Table 1–9), leaving us to determine if the retrograde atrial activation is related to the normal AV conduction system (typical antidromic tachycardia) or to a second AP in the septal region. Preexcitation from the right ventricular apical region at such a long coupling interval demonstrates excellent access to the excitable gap of the reentrant circuit, which would be expected if the retrograde limb were the right bundle branch and the normal AV conduction system as was the case in this example. When the latency time after the extrastimulus is accounted for, the RV apical electrogram is advanced by only 40 milliseconds and advances the subsequent A by 40 milliseconds. This suggests that the RV apical electrogram is right in the circuit, which would not be the case if the retrograde limb were a second septal AP. A more definitive distinction could be made with reasonable certainty by comparing PVCs inserted into the tachycardia circuit at a right ventricular basal site near the septum versus a right ventricular apical site to determine which has easier access to the excitable gap. Theoretically, the tracing could also be explained by retrograde conduction over a nodofascicular pathway with retrograde conduction, probably a very rare entity.

Figure 5–35A



The patient is a young man with paroxysmal tachycardia in the absence of heart disease. The 12-lead electrocardiogram was normal. A wide QRS tachycardia was induced after a critically timed atrial extrastimulus as illustrated in this figure. At longer coupling intervals, the QRS was normal. What are the components of this tachycardia circuit? S_1 is the last of a drive of eight atrial depolarizations delivered at the high right atrium and S_2 is the atrial extrastimulus.

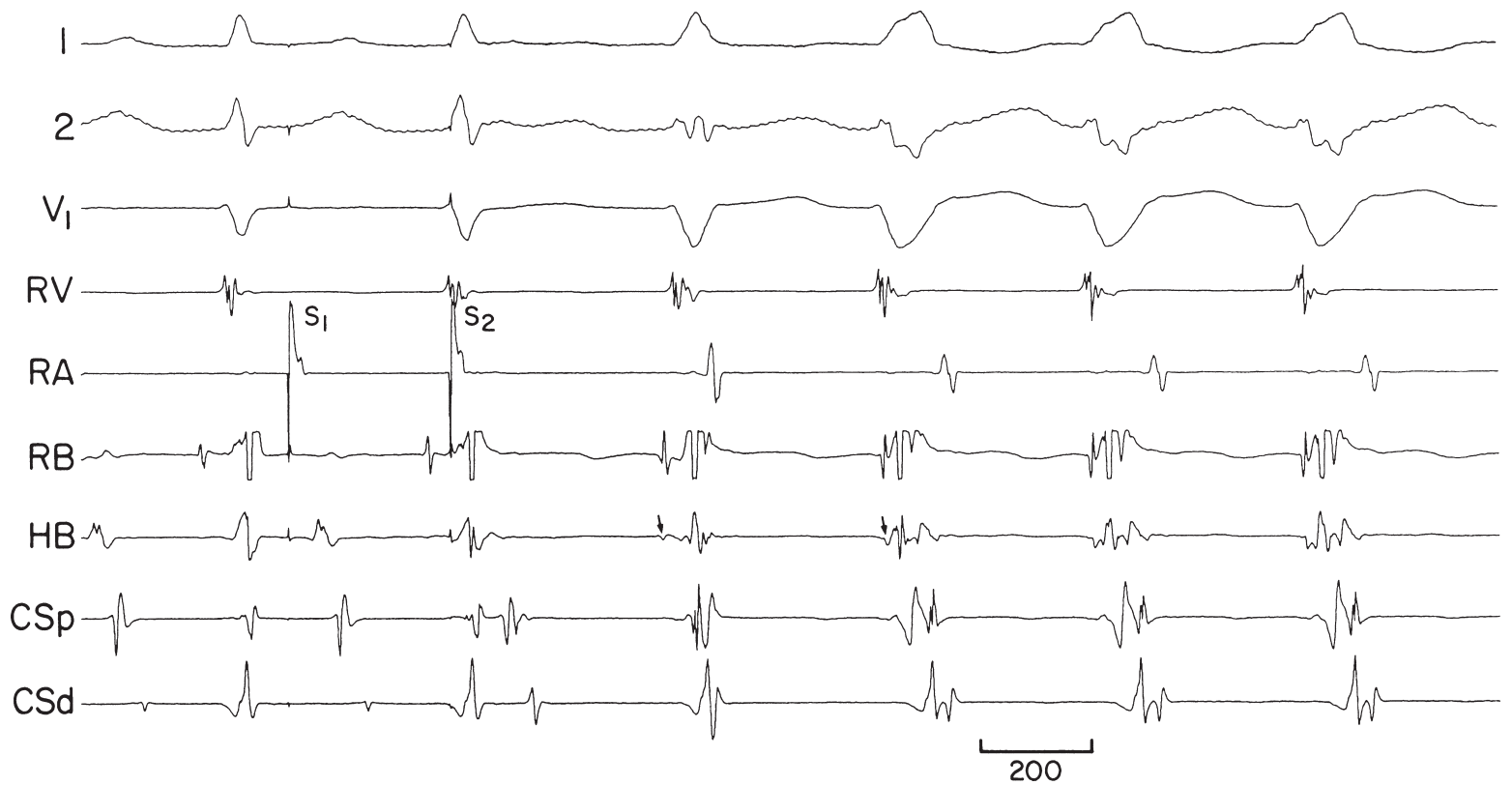
Figure 5–35B



Explanation:

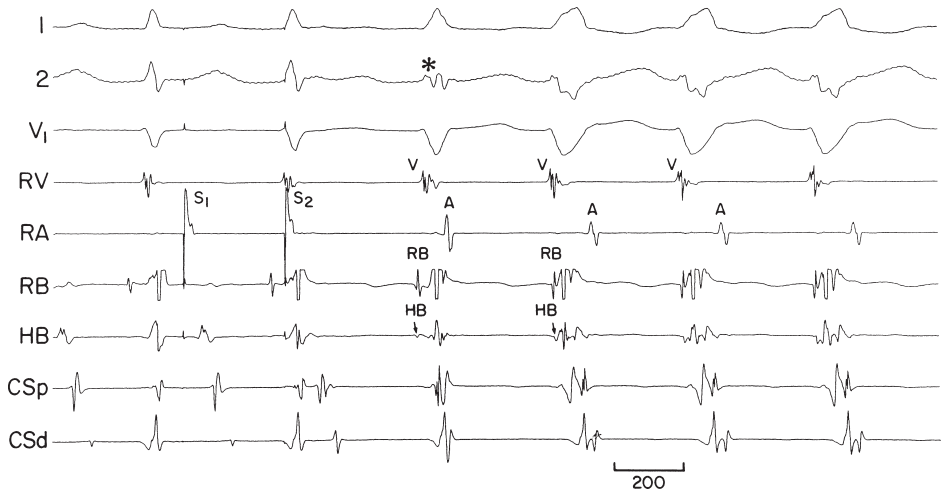
This method of induction was highly reproducible with a stable His recording site. With no H preceding the QRS (Table 1–8), this can be considered to be a preexcited tachycardia. The QRS morphology could be reproduced by atrial pacing at rapid rates. VT induction by atrial extrastimuli has been described, especially in idiopathic LV VT, but one would not expect reproduction of the tachycardia QRS morphology by rapid atrial pacing in such a circumstance. Further, the S_2 would have to reach the ventricle before starting VT. The preexcited morphology suggests a left lateral or posterolateral AP as is supported by early ventricular activation at the distal CS electrogram (CSd). The AP has a long conduction time, a phenomenon usually observed with posteroseptal APs but also observed elsewhere. The retrograde limb of this tachycardia has a concentric atrial activation sequence with atrial activation occurring first at the orifice of the coronary sinus (CSp) slightly ahead of the His bundle electrogram. As discussed previously (Table 1–9 and Fig. 5–34) we must distinguish retrograde conduction occurring over the AV node versus over a septal AP. In this particular instance, retrograde conduction proceeded over a slow AV node pathway, as could be demonstrated by inserting PVCs into the cardiac cycle during tachycardia. Preexcitation from the right ventricular apical region would preexcite the atria at a relatively longer coupling interval than PVCs from the base of the heart near the AP, demonstrating better access to the excitable gap of the reentrant circuit, which would be expected if the retrograde limb were the right bundle branch and the normal AV conduction system (see Fig. 5–34). This antidromic tachycardia is unusual in that the AP has a long anterograde conduction time and retrograde conduction was proceeding over a slow AV node pathway.

Figure 5–36A



The patient is a 15-year-old boy with a recent history of paroxysmal tachycardia, no heart disease, and a normal 12-lead electrocardiogram. At EP testing, incremental atrial pacing from the lateral right atrium resulted in a preexcited QRS morphology that was not observed during similar atrial pacing from the proximal CS. The A to preexcited QRS conduction time was long. Wide QRS tachycardia with identical morphology to the preexcited QRS was initiated with a critically timed atrial extrastimulus as shown in the record. What are the observations? CSp and CSd are recorded from the proximal and distal coronary sinus, respectively, with the proximal recording near the orifice of the CS. HB and RB are at right bundle branch and His bundle recording sites.

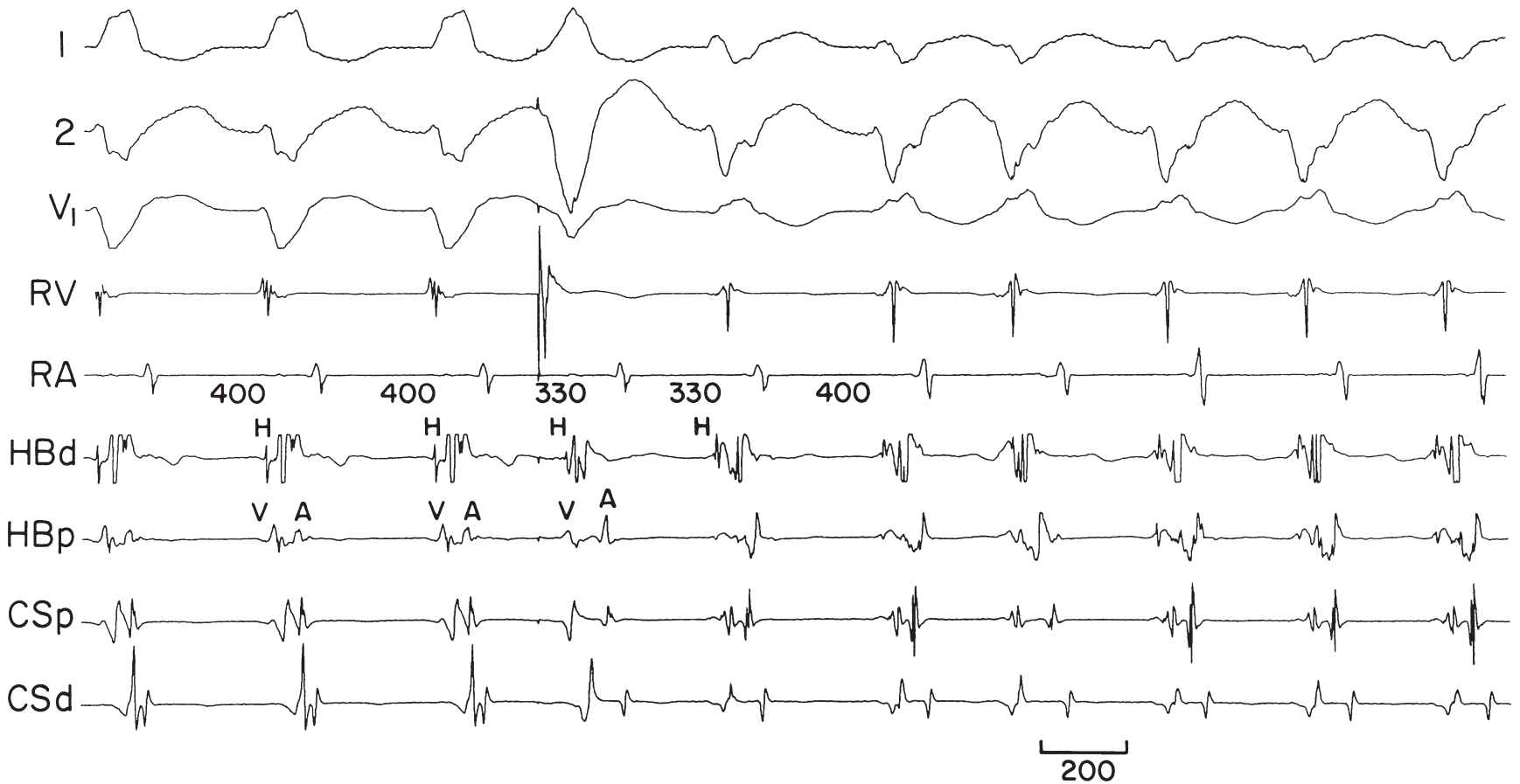
Figure 5–36B



Explanation:

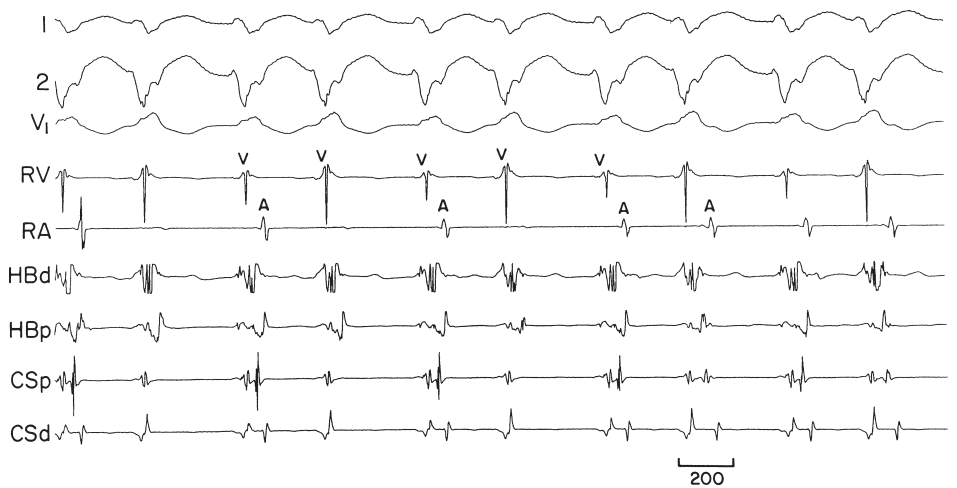
The preceding description and the QRS morphology during the preexcited tachycardia suggest a right-sided AP with a long conduction time ("decremental"). The rapid component of the right ventricular electrogram at the right ventricular apex is approximately at the onset of the QRS, an observation compatible with an atriofascicular AP that activates the right ventricle near the right bundle branch terminus, as was the case in this patient. Note also that the right bundle electrogram during tachycardia is also at the onset of the QRS and precedes activation of the His bundle (less well seen) by 5–10 milliseconds. We are then left with a preexcited tachycardia (over a right atriofascicular pathway) with retrograde activation of the right bundle branch and, subsequently, the His bundle. The atrial activation sequence is concentric and is probably proceeding over the normal AV conduction system. However, this can only be determined with certainty by other measures such as the relative ability of PVCs from the right ventricular apical region and the base of the heart near the His bundle to preexcite the atrium during tachycardia. In this differential diagnosis, remember that the most common regular preexcited tachycardia (i.e., excluding atrial fibrillation) is true antidromic tachycardia with conduction over the AP and a return circuit via the normal AV conduction system. An astute observer will also notice that the first tachycardia cycle (asterisk) is a little different from the subsequent cycles with the QRS being a reasonable fusion between the normal QRS and the totally preexcited QRS. Furthermore, the His bundle electrogram (low amplitude) *precedes* the right bundle by a few milliseconds, supporting the view that the tachycardia has not yet started at this point since the AV node has been activated anterogradely. In fact, antegrade conduction is proceeding over a slow AV node pathway that is associated with an AV node echo cycle over a retrograde fast AV node pathway, which subsequently results in a fully preexcited QRS and the beginning of antidromic reentry. Although antidromic tachycardia is the most common tachycardia with atriofascicular APs, there is an association with AV node reentry and, indeed, other APs.

Figure 5–37A



The record is from the patient described in Fig. 5–36. A PVC is introduced into the cardiac cycle during the antidromic tachycardia described in the previous case. What observations can be made from this "zone of transition"? HBd and HBp are recordings from the proximal and distal electrodes of a standard His bundle catheter (bipolar recordings, four poles, 10-mm separation). CSp and CSd are proximal and distal coronary sinus electrodes, respectively, with the proximal electrode being near the orifice of the CS.

Figure 5–37B



Explanation:

The right ventricular apical extrastimulus readily preexcites both His and atrial activation without changing atrial activation sequence, an observation compatible with antidromic reentry with a relatively large excitable gap accessible to the right ventricular apical catheter by virtue of its proximity to the distal right bundle branch. This is not unexpected. However, there is a change in QRS morphology after the extrastimulus (now right bundle, leftward axis) and the tachycardia becomes somewhat irregular. The retrograde atrial activation sequence has not changed appreciably. The His bundle electrogram cannot be discerned with certainty but is probably within the QRS and certainly past the onset of the QRS. Since the His recording site was stable, this can only be VT or a preexcited tachycardia from a second AP. The diagnosis becomes more evident in Fig. 5–37*B* when the tachycardia continues in the presence of VA block, a situation not compatible with AV reentry although theoretically compatible with nodoventricular or nodofascicular reentry. In this instance, this patient also had inducible idiopathic LV VT, a tachycardia never previously documented clinically in this patient. Although common things are common, it is important to be methodical and continue to have an open mind and "expect the unexpected."

Chapter 6

Catheter Ablation

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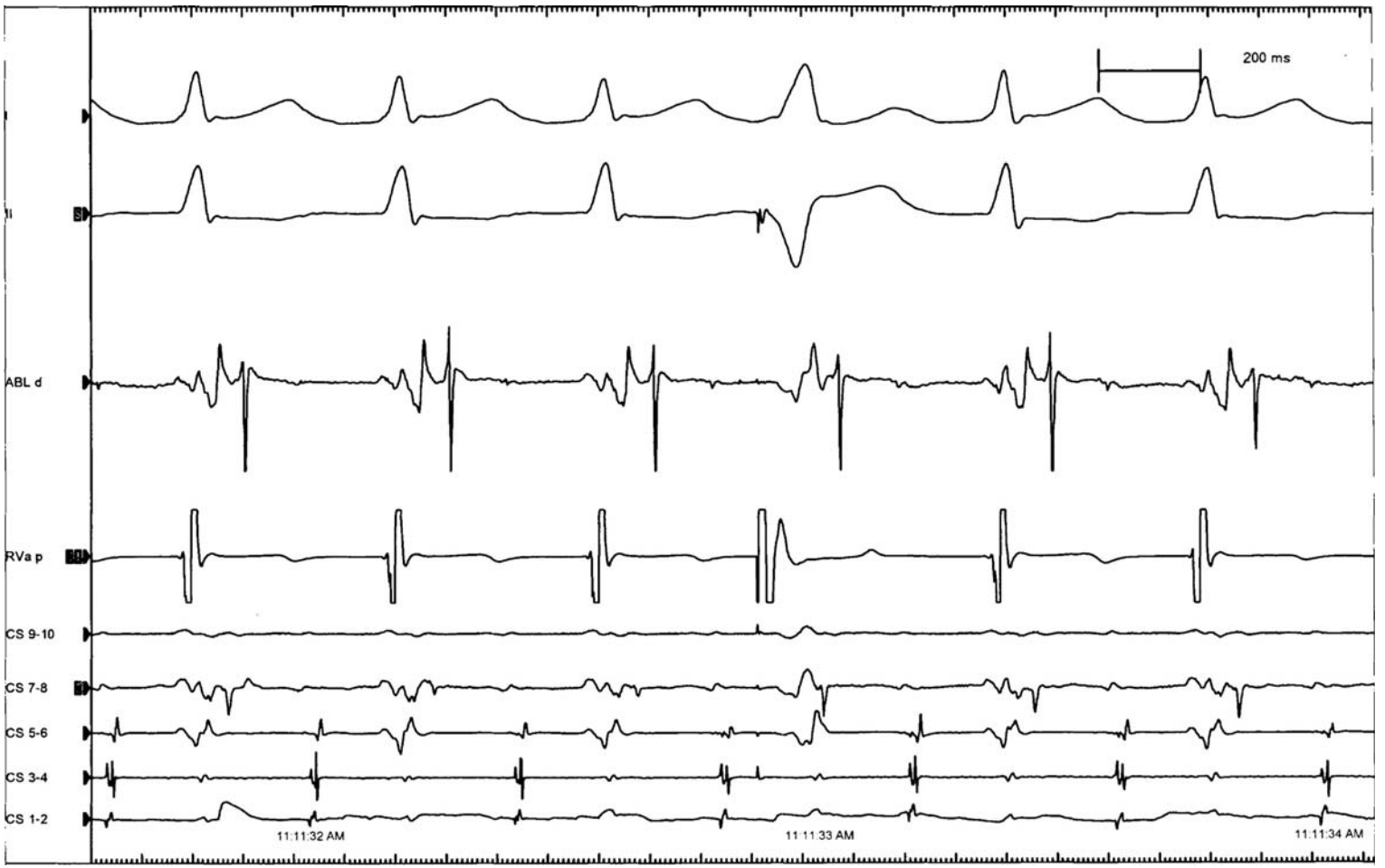
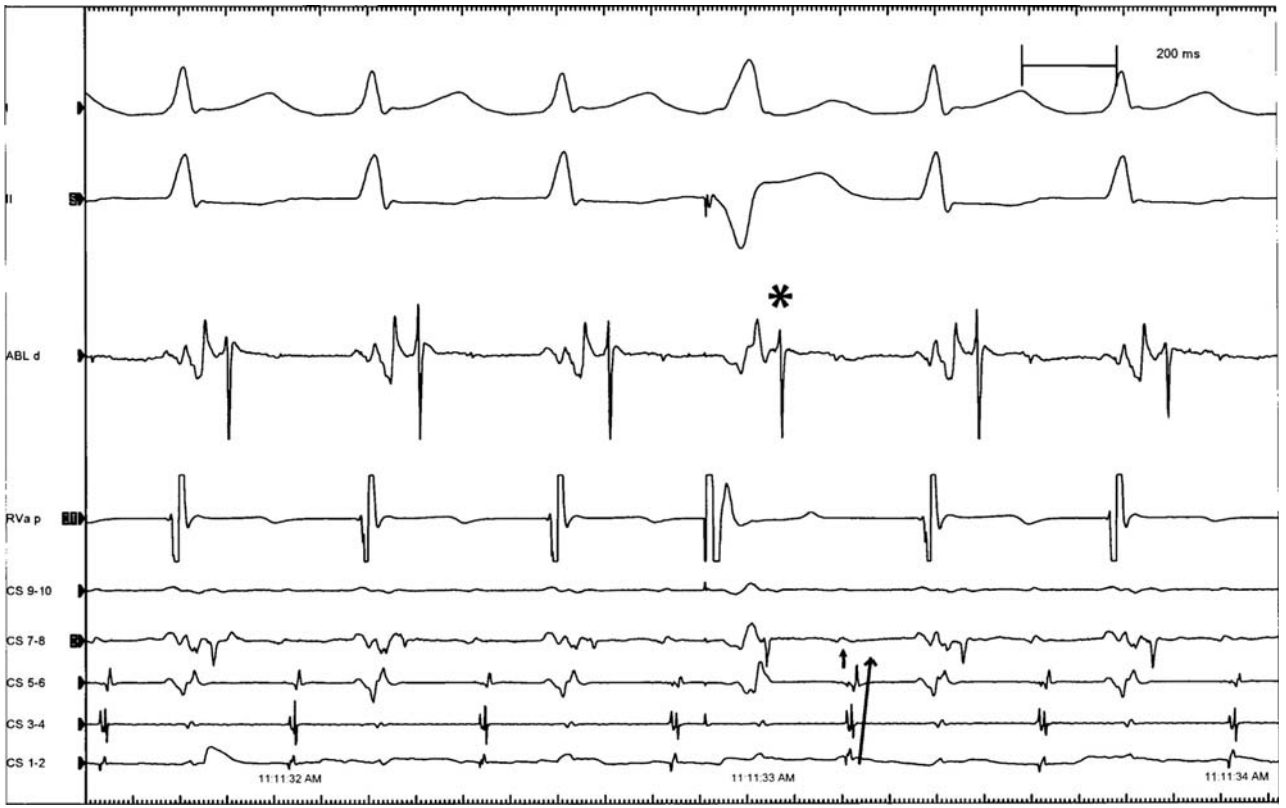
Figure 6–1A



The patient had electrophysiologic assessment for WPW and recurrent SVT. AVRT over a posteroseptal AP was found with earliest atrial activation during tachycardia at the proximal coronary sinus (PCS) electrode. Ablation adjacent to the CS os was performed and posteroseptal preexcitation was abolished with no inducible tachycardia. Fifteen minutes later, SVT was again induced and a PVC was introduced into the cycle as in the figure. The ablation catheter is near the previous site and the coronary sinus catheter has not moved. Where would one ablate now?

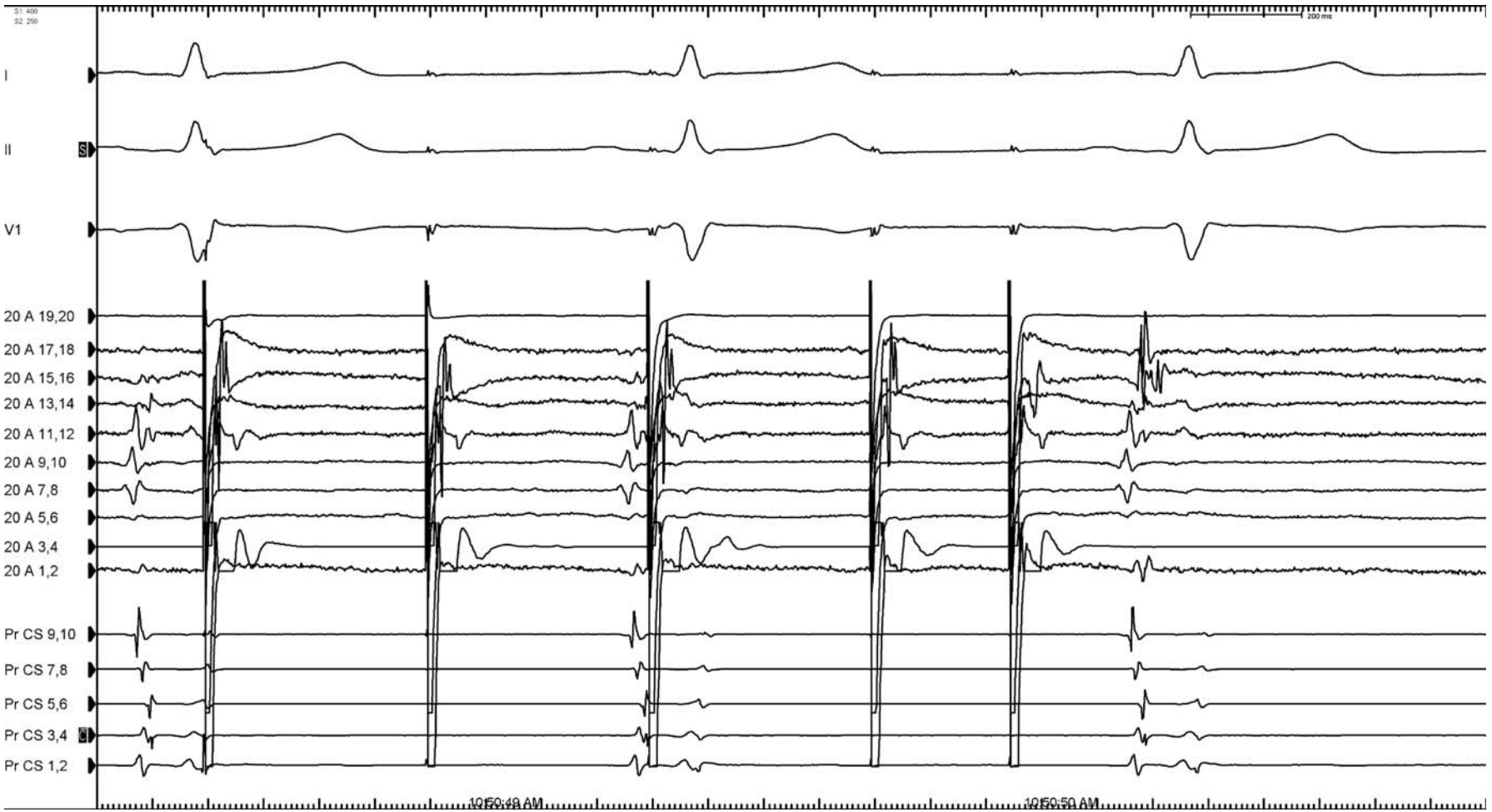
Figure 6–1B



Explanation:

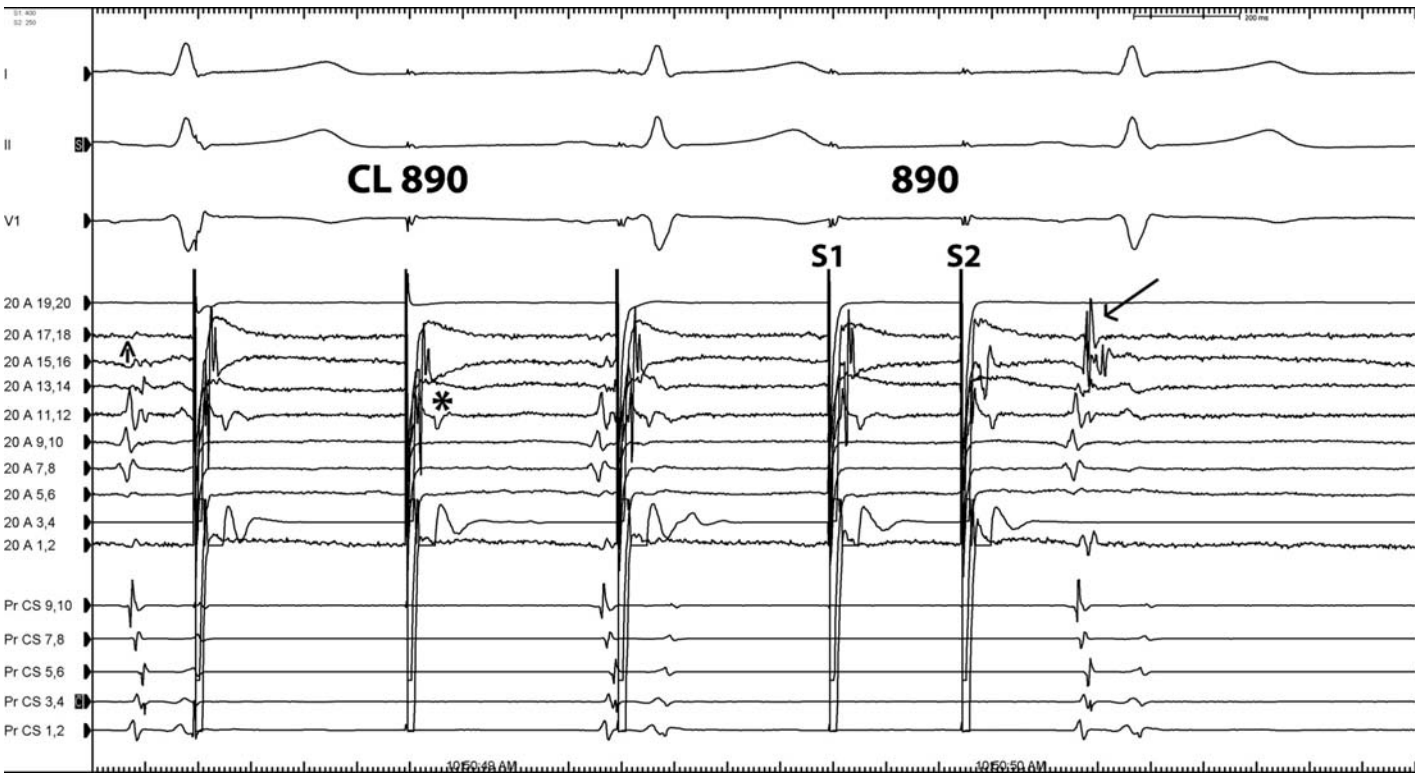
The PVC programmed into the cardiac cycle is relatively late coupled with minimal fusion and yet preexcites the next atrial cycle, so it is clearly AVRT. Earliest atrial activation is still near the CS os as identified on the ABL atrial electrogram, but now the atrial activation in the coronary sinus has shifted (*long arrow*) from distal to proximal, although the far-field PCS EGM is relatively earlier (*short arrow*). One may speculate that there is a now a left pathway but the atrial activation at the CS os (ABL catheter) is still very early. Ablation at this site terminated tachycardia and this was no longer inducible. The initial ablation at the CS os region likely resulted in interatrial block over the CS interatrial connection. Although AVRT was still occurring over a posteroseptal accessory pathway, there was a change in atrial activation of the LA resulting in a distal to proximal CS activation in spite of the origin of preexcitation near the CS os. This is infrequently seen with minimal ablation in the CS os region but is an interpretative issue that has consequences for successful ablation.

Figure 6–2A



The patient was undergoing PVI ablation and the left upper PV had just been completed. The CS electrograms are shown in lower part and the circular mapping catheter electrodes are shown in the middle of the tracing. Pacing is attempted from the circular mapping catheter within the left pulmonary vein. How would one interpret the observations?

Figure 6–2B

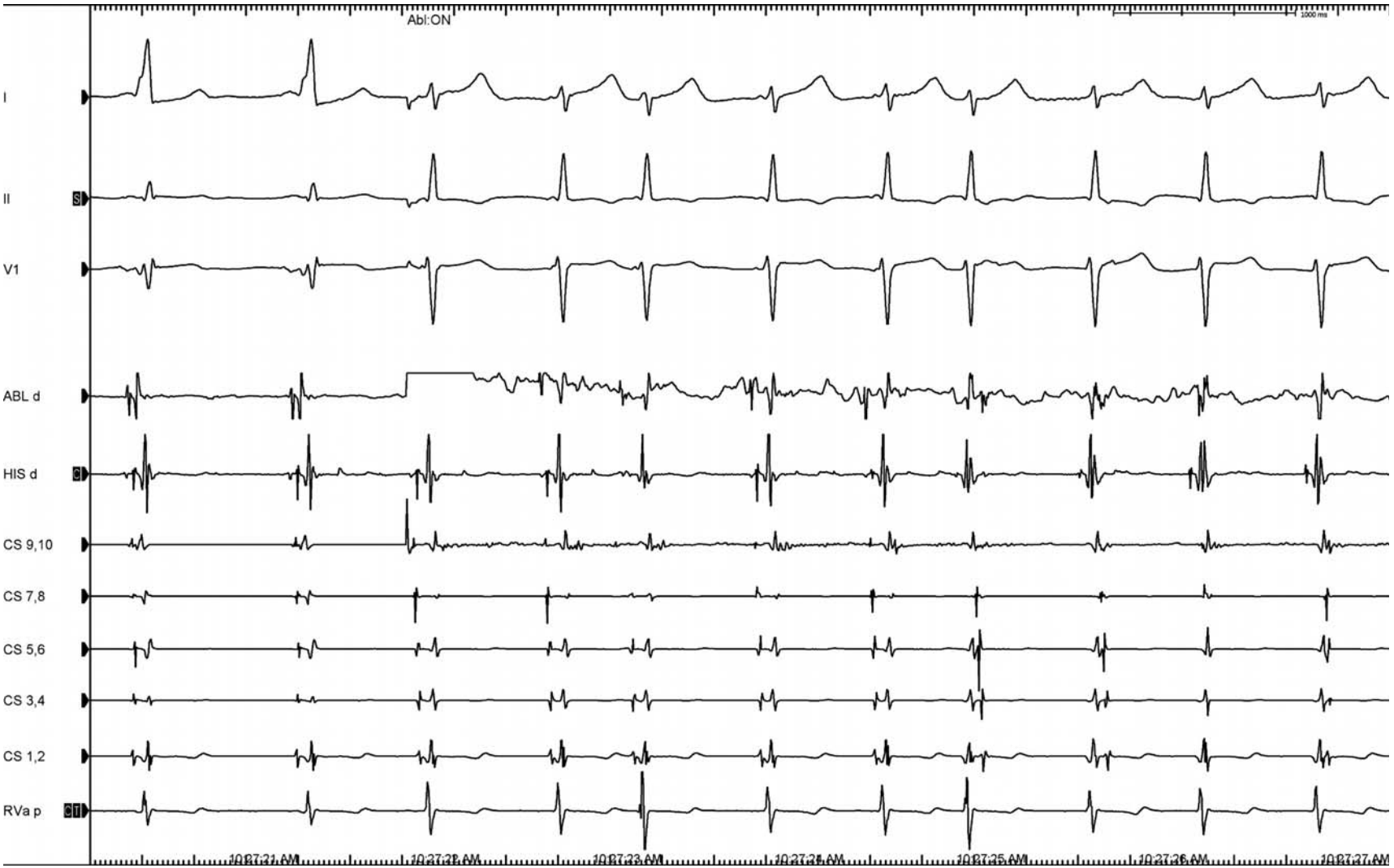


Explanation:

The first observation is that there is some local activation after each pacing spike (asterisk) so that the spike is capturing the PV. The second is that the underlying rhythm is sinus (CL 890 milliseconds) and is dissociated from the paced activity inside the PV. The potentials in the pulmonary vein are "far field" coming from the left atrial appendage and this characteristically shows the biggest far-field potentials in the anterior part of the circumferential mapping catheter (i.e., 13–14, 11–12, 9–10, 7–8). Thus, "exit block" has been achieved. Entrance block is presumably present since sinus activity does not perturb the activity in the pulmonary vein but this is best assessed by noting activity inside the PV without the confounding effect of pacing within the vein.

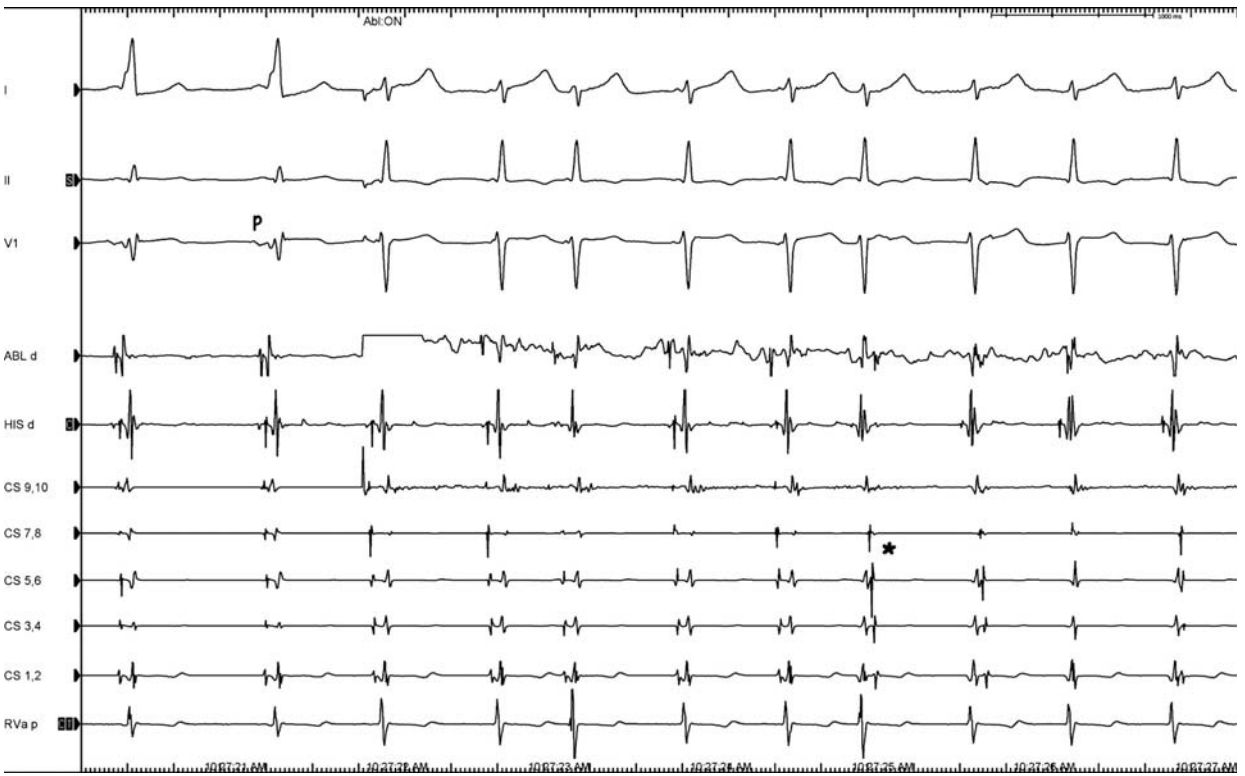
There are interesting observations after the atrial extrastimulus (S2). In addition to the local activity right after the spike (asterisk), there is now near-field activity following this (*long arrow*) that is clearly not present without an extrastimulus (see first complex, *short arrow*). This is best explained by conduction delay within the pulmonary vein causing a double potential, although other explanations are conceivable. The observation does not impact on decisions for further ablation since there is still exit block and the activity is confined to the vein.

Figure 6–3A



Ablation has just begun near the orifice of the coronary sinus in this patient with WPW. How long should ablation be continued?

Figure 6–3B

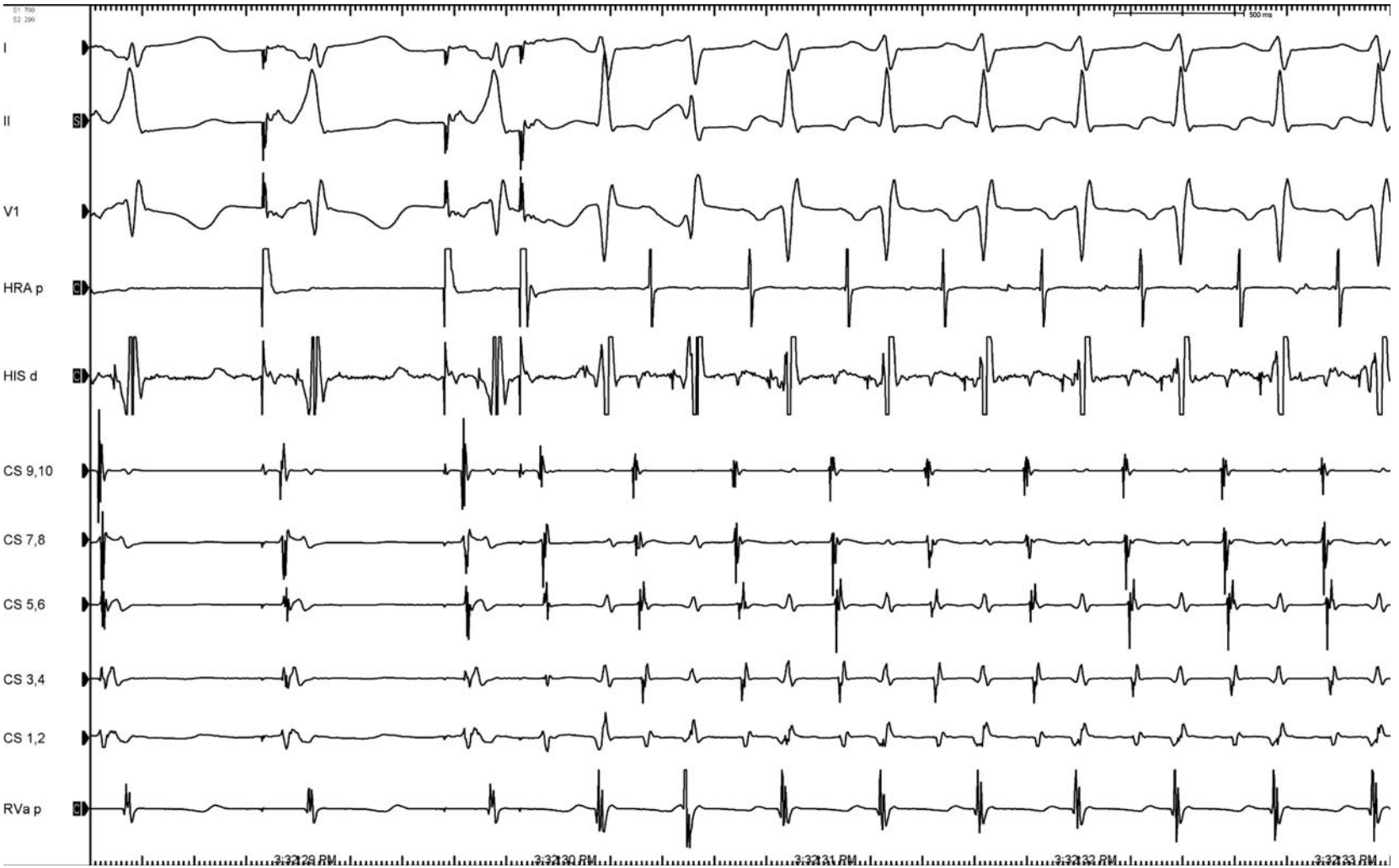


Explanation:

This is a posteroseptal pathway as evidenced by the early ventricular electrogram at the orifice of the coronary sinus. Although preexcitation disappears immediately with the onset of current, suggesting close proximity to the accessory pathway, there is a problem. Note that the P wave, which is upright in leads 1 and 2 during sinus rhythm (p), changes to "low to high" immediately with onset of ablation with a very short PR interval most evident on the surface ecg. This is an accelerated junctional rhythm (JT) and the loss of preexcitation does *not* reflect accessory pathway ablation. After a few more cycles of JT, the atrial component follows the ventricular component, making the diagnosis of JT even more apparent (asterisk).

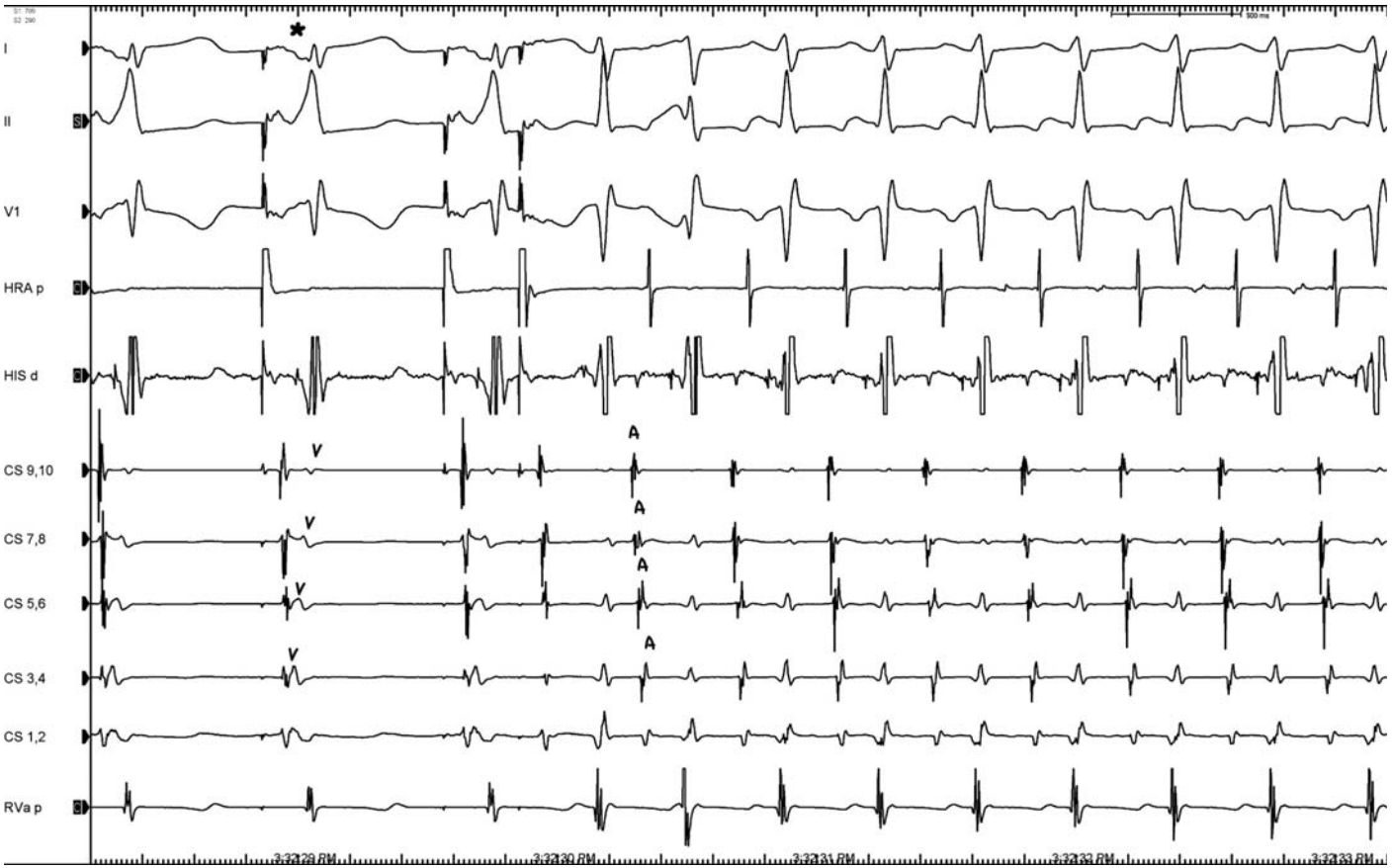
Current was discontinued immediately with return of sinus rhythm and preexcitation and the catheter was repositioned. Continuation of current in such a case may result in JT with retrograde conduction over the intact accessory pathway with a false sense that the fast AV nodal pathway is not at risk. Inadvertent AV block in this young individual with WPW would have been very unfortunate and great vigilance is required to diagnose apparent loss of preexcitation related to accelerated junctional rhythm.

Figure 6–4A



This arrhythmia was induced in a young man referred for tachycardia associated with the WPW syndrome. What is the mechanism of tachycardia?

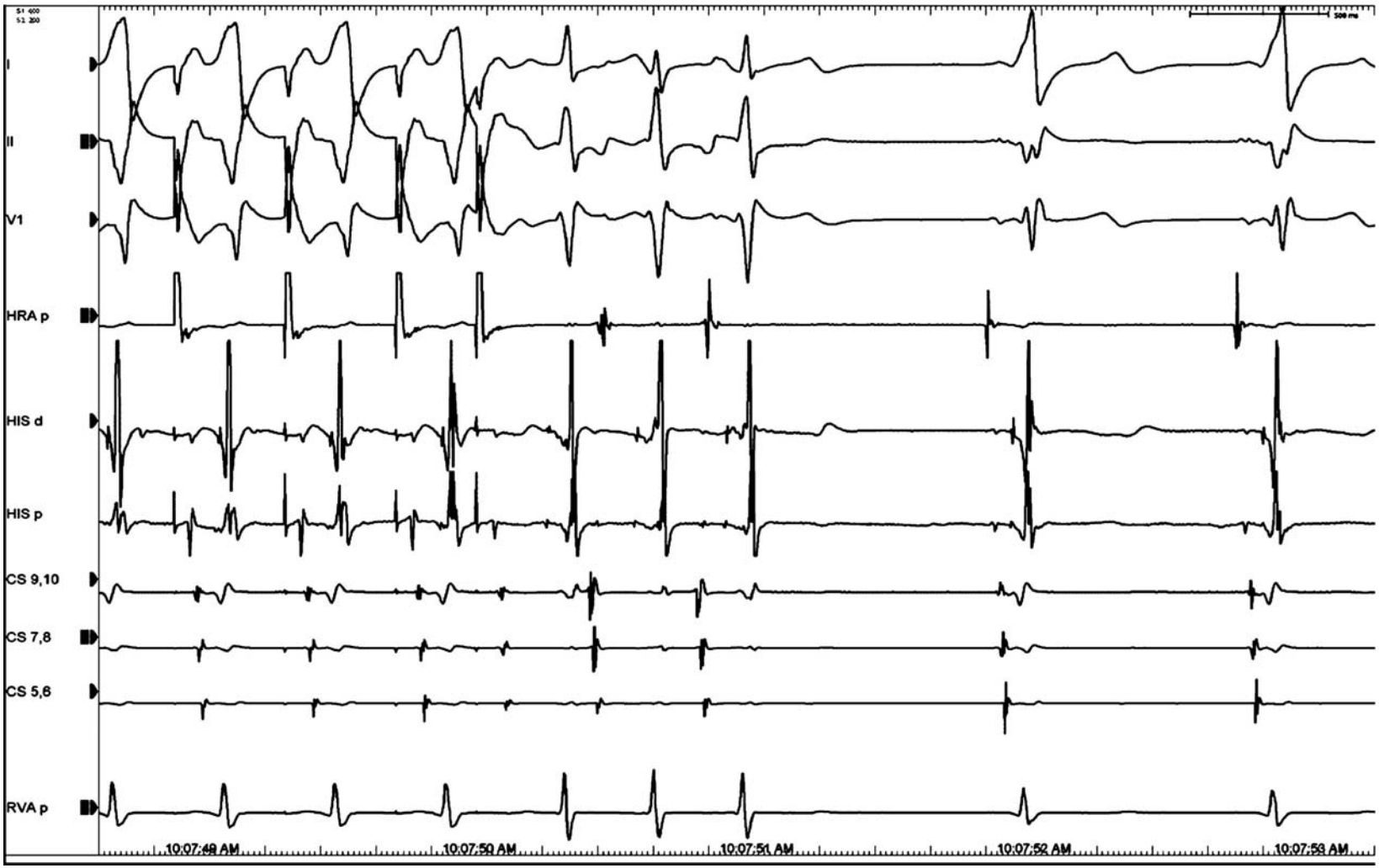
Figure 6–4B



Explanation:

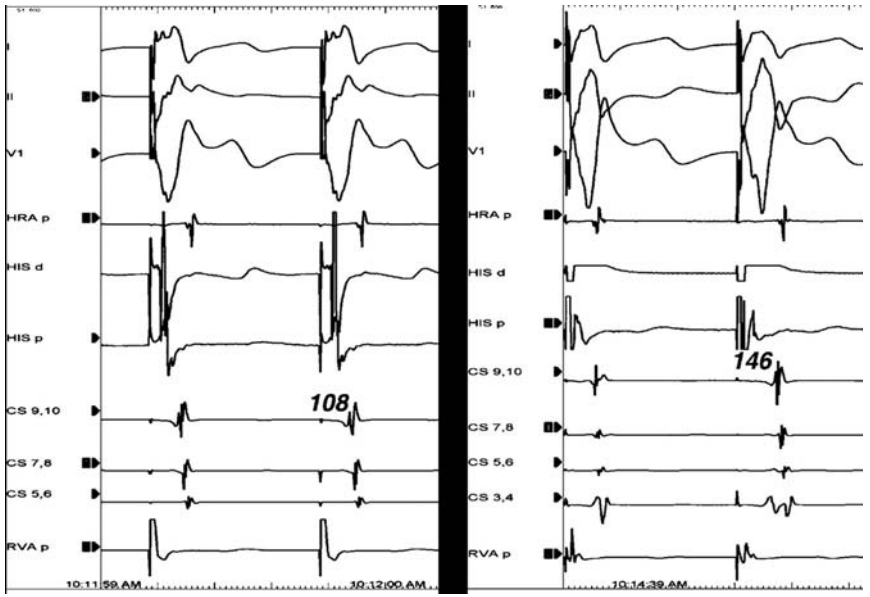
The QRS morphology during atrial pacing reflects a left lateral accessory pathway (asterisk). Note also that the earliest ventricular (V) activation on the intracardiac electrograms occurs in the distal coronary sinus (DCS; CS 3–4). Supraventricular tachycardia was induced with an atrial extrastimulus that blocked anterograde over the accessory pathway. However, the atrial activation sequence is septal with earliest atrial activation in the orifice (CS 9–10) of the coronary sinus. The retrograde limb of this tachycardia is clearly not over a left lateral accessory pathway and the tachycardia mechanism was proven to be AV node reentry with a relatively long VA interval. An expedited strategy of just ablating the obvious left lateral accessory pathway would not have prevented tachycardia in this patient, and would have been incorrect to do.

Figure 6–5A



The tracing is recorded from a patient undergoing EP study for electrocardiographically undocumented palpitations. Is there any clue as to clinical tachycardia?

Figure 6–5B



Explanation:

The sinus cycles at the right of the tracing are preexcited with earliest ventricular activation at the CS orifice (CS 9–10) of the available electrodes for review. Preexcitation is more marked during atrial pacing and the atrial extrastimulus blocks in the accessory pathway. Several nonstimulated cycles follow and this sequence was reproducible. The atrial activation is septal with earliest activation at the proximal CS and His electrograms. The possibilities for the repetitive atrial responses are atrial ectopy, AVRT echoes, or AV nodal echoes.

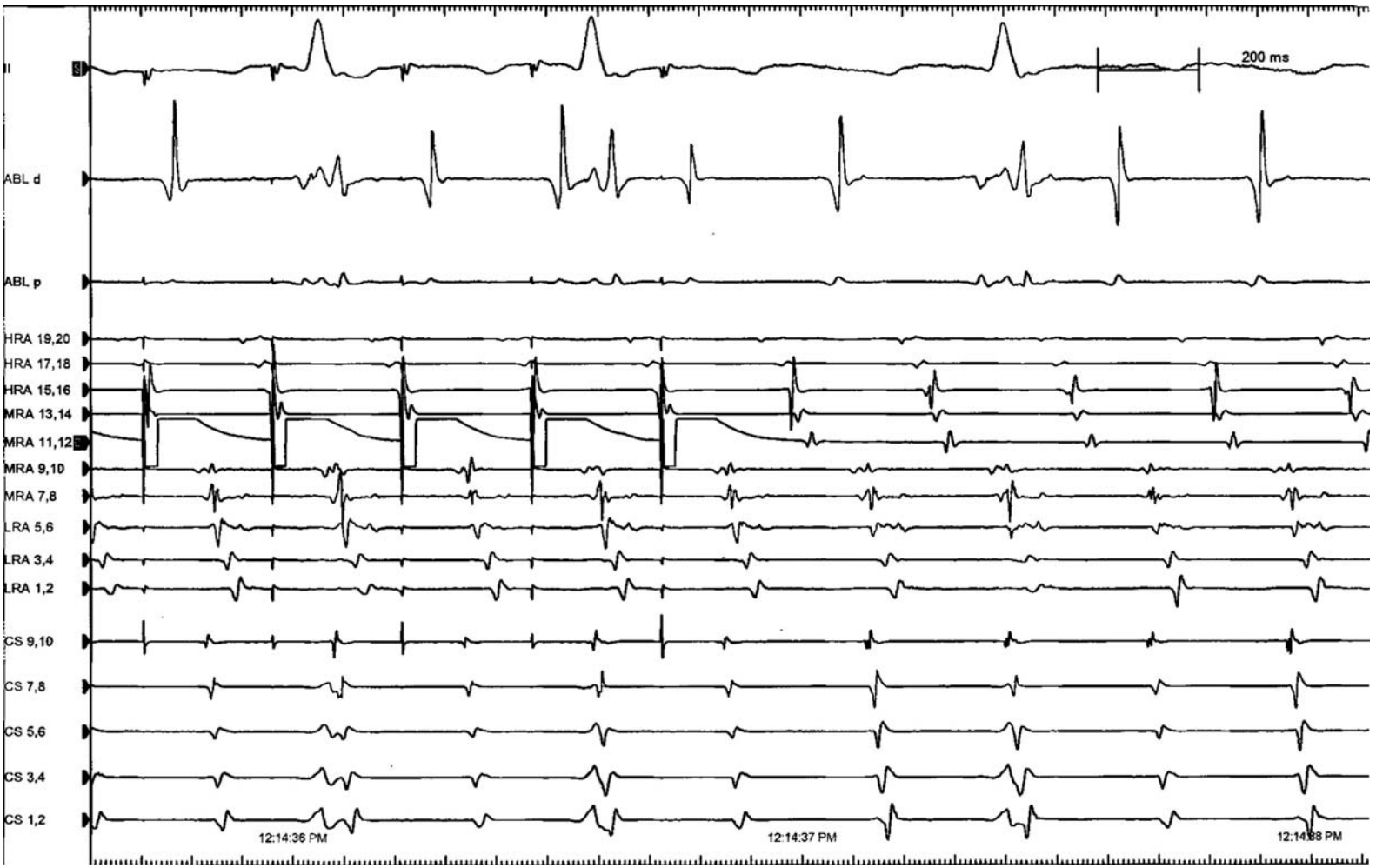
In general, repetitive atrial responses are more closely coupled to the atrial extrastimulus and have an inconsistent relationship to the stimulated QRS.

If we consider atrial activation as retrograde, we are left with possible retrograde conduction over the AV node versus a septal or parasseptal accessory pathway. The retrograde Wenckebach sequence favors AV nodal conduction but the presence of an anterograde septal accessory pathway raises the possibility of an accessory pathway with decremental retrograde conduction. In this case, anterograde conduction over the accessory pathway was *not* decremental, making the latter less likely. Pacing maneuvers during tachycardia are problematic if sustained tachycardia is not induced.

In this instance, it is useful to determine if baseline retrograde conduction is occurring over the AV node or not. The next figure shows the atrial response to ventricular pacing from two sites, the right ventricle (RV) apex on the left and the RV posterobasal region on the right. Since the accessory pathway is closer to the RV base, the VA time here should be shorter than during pacing of the RV apex if a septal accessory pathway were present. The opposite holds true for AV nodal conduction since the RV apex is closer to the distal right bundle branch (RBB) where the normal AV conduction system activates the ventricles. This rationale is similar to that of the para-Hisian pacing technique. In this case, retrograde conduction is clearly over the AV node.

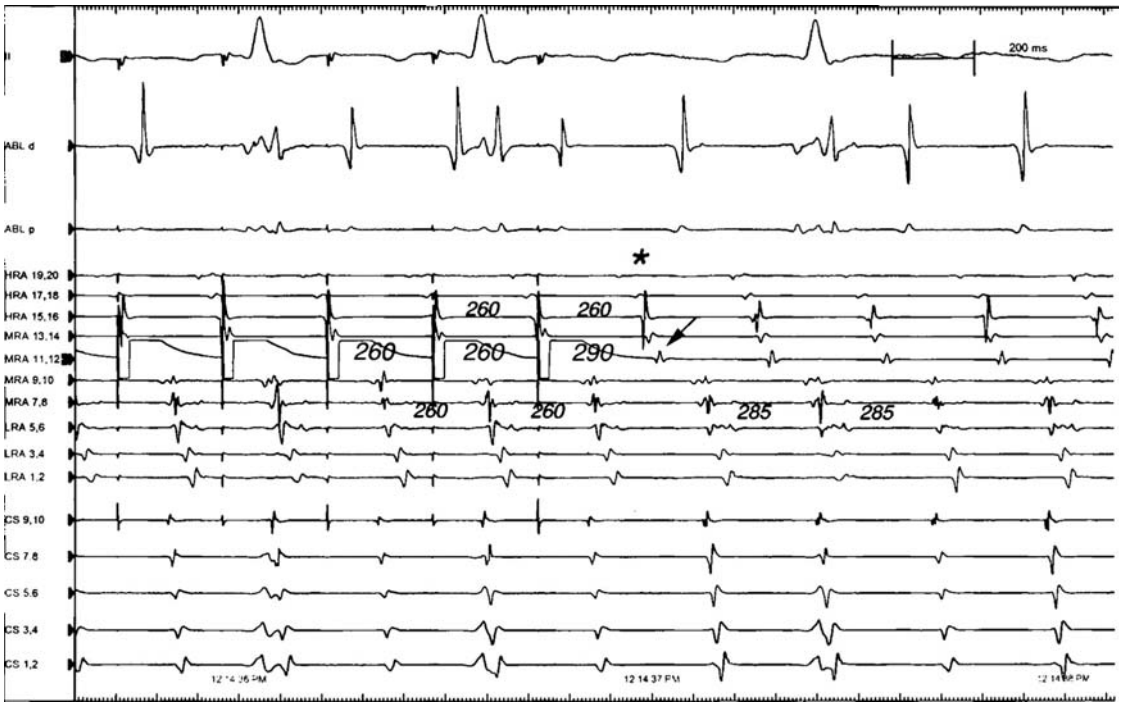
There is a caveat since the maneuver does not tell us what is happening during tachycardia, which is conceivably different. Repeating this differential pacing at multiple cycle lengths with similar results increases the confidence that the observation is relevant to the observed tachycardia. In this case, conduction over the accessory pathway was unidirectional (anterograde only). It would obviously be a mistake to map during ventricular pacing in this instance to ablate the accessory pathway since retrograde conduction is via the AV node.

Figure 6–6A



The patient presented with atrial flutter 10 years after ASD repair. A multipolar catheter with 10 bipoles (halo) was positioned in the right atrium and oriented in the "usual" way with the proximal poles against the septum, the catheter circling at the top of the right atrium and descending the lateral wall with the distal poles pointing to the cavotricuspid isthmus. The coronary sinus catheter is positioned so that the proximal pair is 10–20 mm inside the CS os. Entrainment from the lateral RA 11–12 is shown. What is the probable mechanism of the flutter?

Figure 6–6B



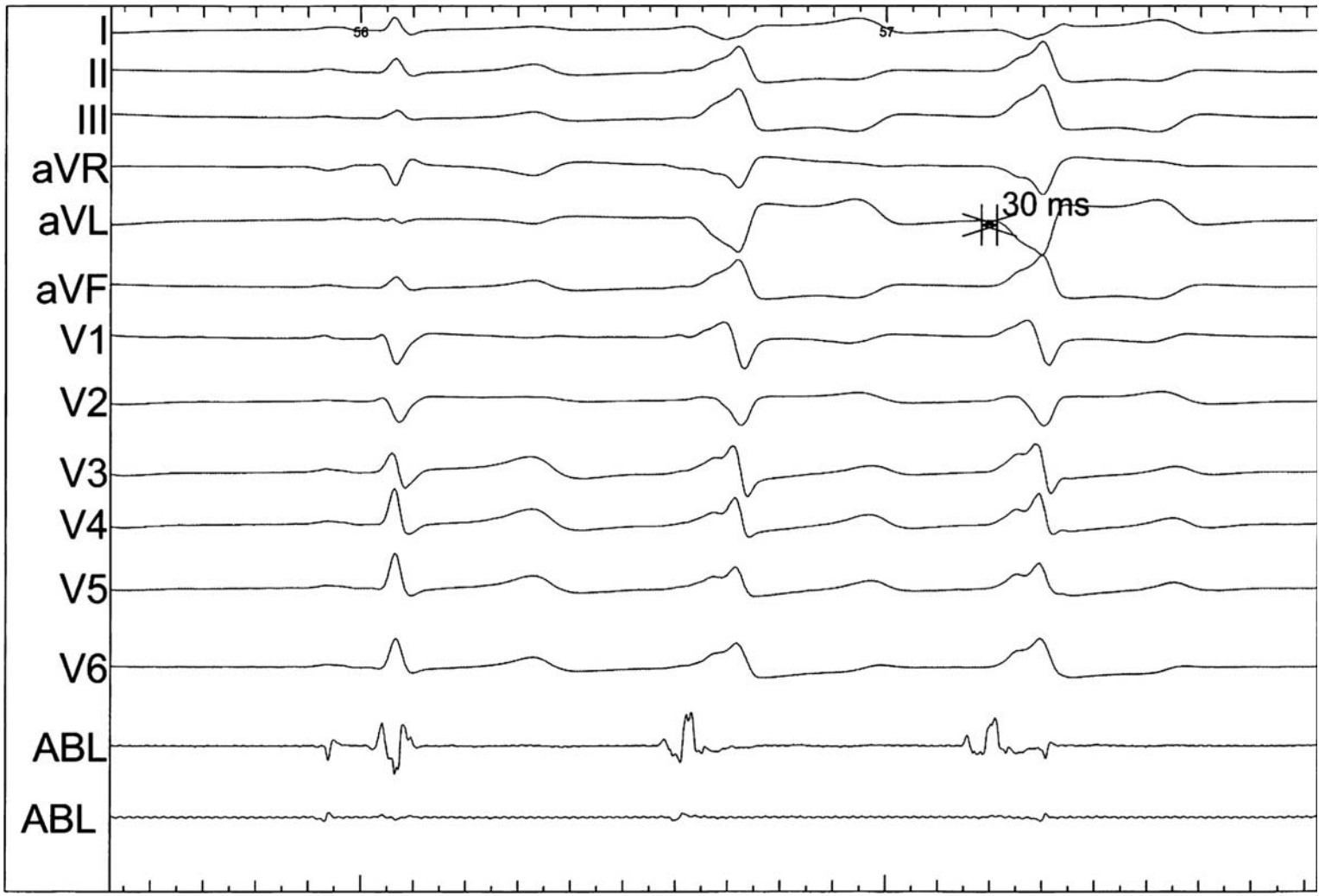
Explanation:

One can begin with the tachycardia at the right of the tracing. The first observation is that the halo catheter in the right atrium has electrograms that span virtually the whole cardiac cycle. This is strongly supportive of right atrial macroreentry. The direction of activation is up the septum (starting from 19–20) and down the lateral wall (to 1–2), consistent with "counterclockwise" flutter. There is a relatively long "gap" between poles 11–12 and poles 9–10 of the halo catheter.

Poles 11–12 are paced slightly faster than the tachycardia and pacing is stopped. The tachycardia has been entrained or accelerated to the pacing cycle length with constant fusion. One identifies the last entrained electrograms (i.e., accelerated to 260 milliseconds) and notes the atrial cycle "entrained but not fused" indicated by the asterisk. The postpacing interval at MRA 11–12 is essentially equal to the tachycardia cycle length and this site is thus "in" the circuit.

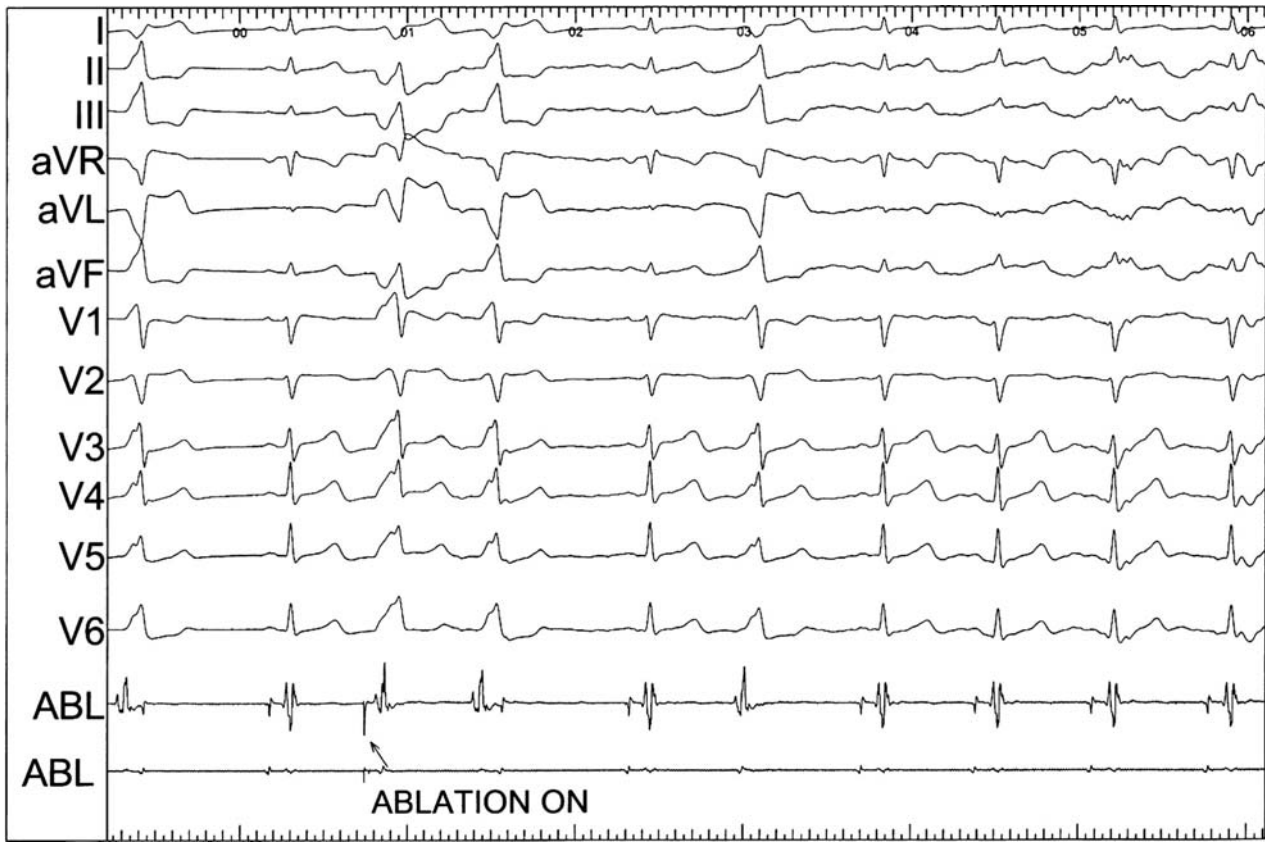
Isthmus-dependent right atrial macroreentry is in fact the most common mechanism even in presence of atriotomy scar. In this instance, the isthmus was also "in" with entrainment, confirming the mechanism. Ablation here resulted in bidirectional block and failure to induce further tachycardia. The relative delay between electrode pair 11–12 and 9–10 was probably related to conduction delay around or through atriotomy scar.

Figure 6–7A



A 19-year-old woman has a history of palpitations due to PVCs and nonsustained VT, with over 30,000 PVCs daily and normal ventricular function. She opted for ablation and the following was noted. Frequent spontaneous PVCs similar in morphology to the patient's PVCs prior to catheter insertion were observed and the tracing shows the ablation catheter situated in the left coronary cusp. Would you ablate here?

Figure 6–7B

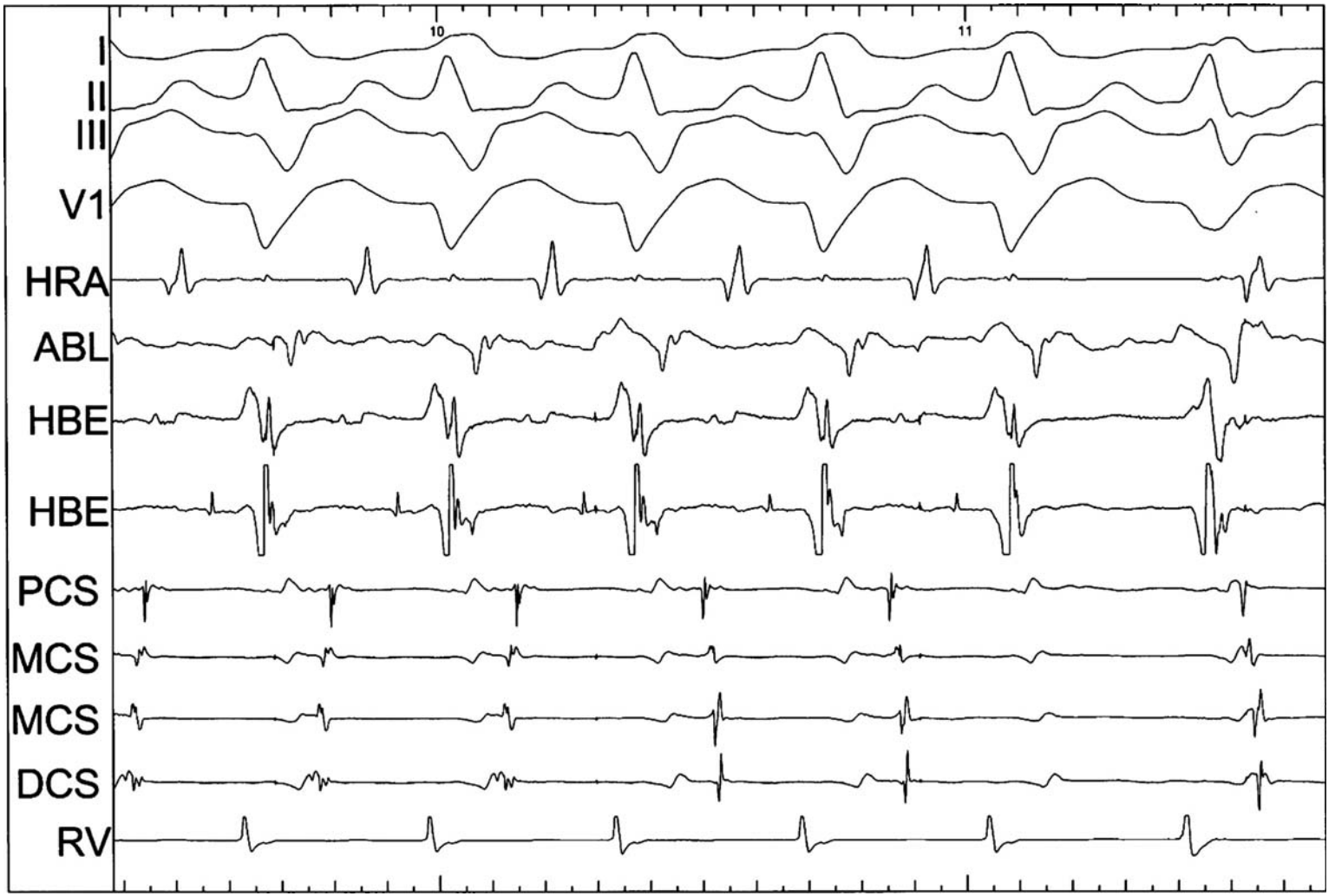


Explanation:

The local ventricular electrogram on the ablation catheter during PVCs was complex with near-field activity noted 30 milliseconds prior to the onset of the QRS at the distal electrode of the catheter. This was the earliest that was obtainable after careful mapping of the region and injection of contrast showed that it was relatively distant from the orifice of the left coronary artery. Onset of energy (maximum 30 W) resulted in loss of ventricular ectopy within 10 seconds and no PVCs were seen during 1-hour waiting period with and without isoproterenol infusion.

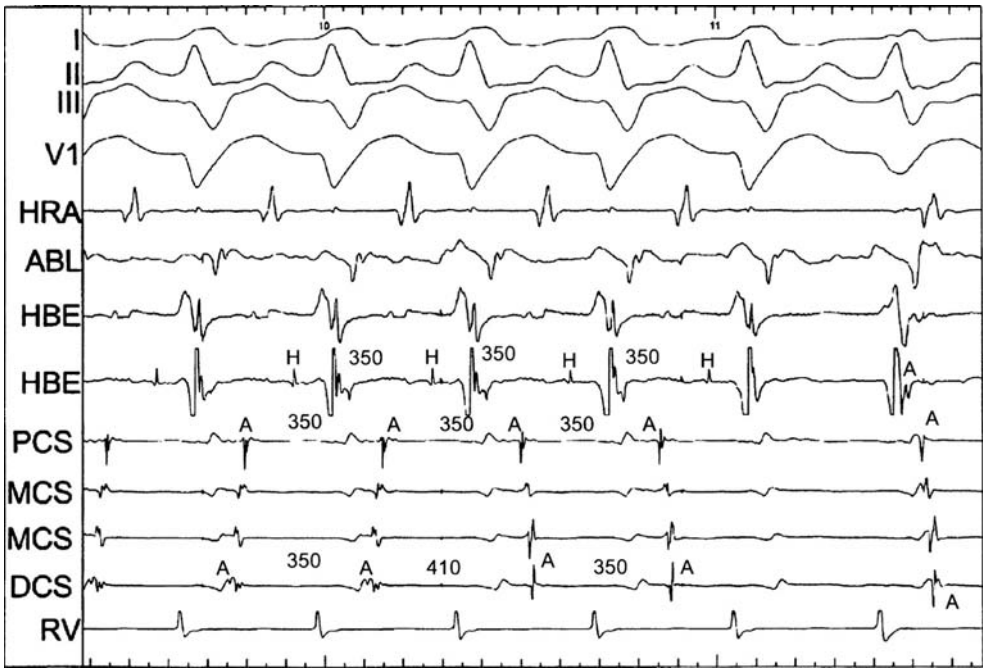
The "optimal" site to ablate is generally relative and practically speaking is the best that can be obtained after careful mapping in the region of interest. In this case, the electrogram characteristics were reasonable but ectopy recurred 2 weeks later. At subsequent ablation, a site a few millimeters away from the initial ablation site resulted in a long-term cure.

Figure 6–8A



During ablation of a left-sided accessory pathway the following was noted and the energy was continued. The retrograde activation on the last QRS complex was present for the rest of the study with and without isoproterenol. How do you explain the changes in retrograde activation sequences?

Figure 6–8B

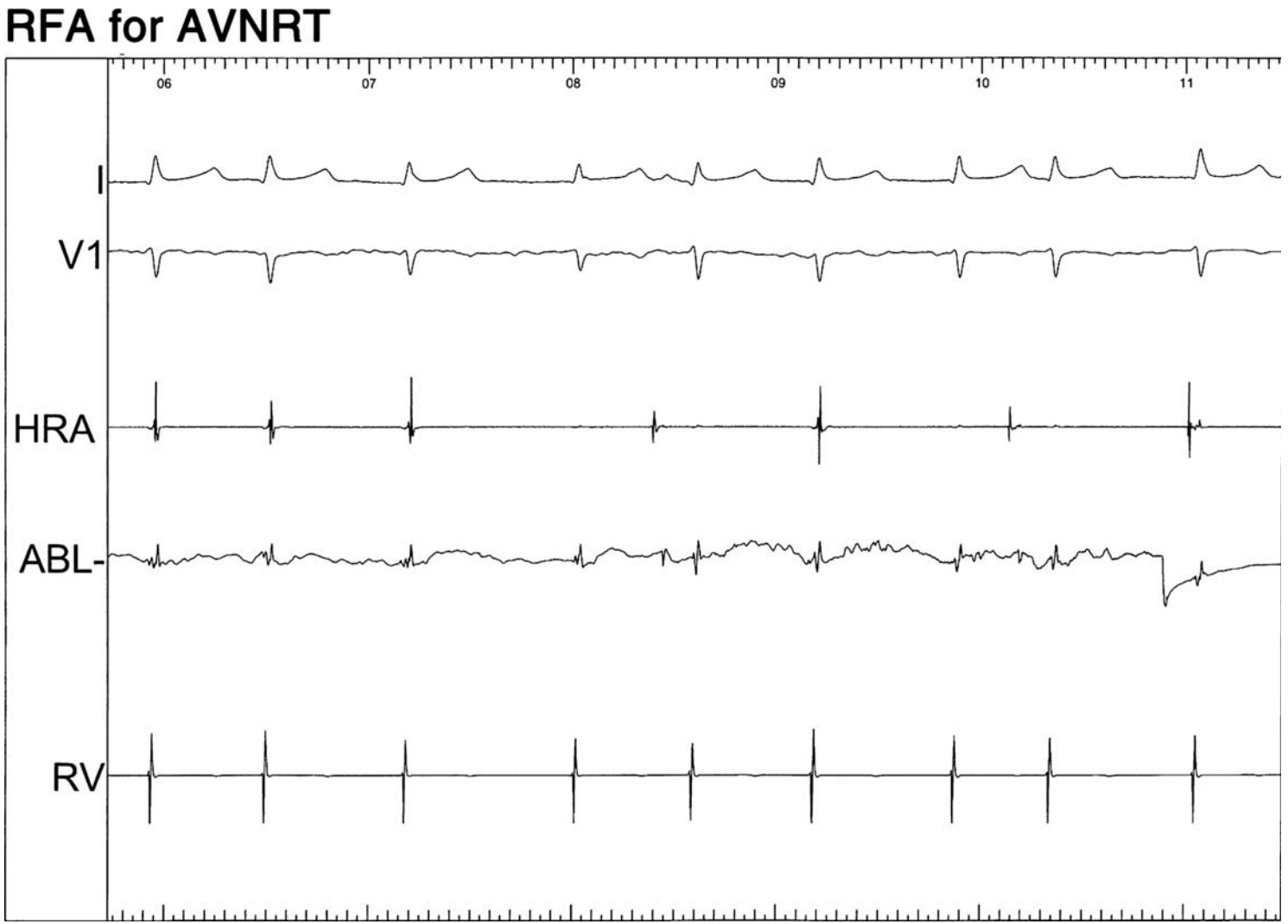


Explanation:

The first three atrial activation sequences are the same with the earliest activation on the DCS electrode. The fourth and fifth atrial activation sequence patterns change with earliest activation on the PCS electrode. However, note that there is no change in the H–H interval or in the atrial interval on the PCS electrode. This would be the case if the main AV circuit was over a second more septal pathway with the left lateral pathway providing another circuit simultaneously so that disruption of the left lateral pathway allows seamless continuity of AVRT over the more septal pathway. In other words, the atrial activation sequence over the first three cycles is a fusion of atrial activation over a more septal and left lateral accessory pathway. In this scenario, the more proximal pathway would have to branch off near the ablation site and slant toward the septum so that both branches would be ablated eventually as the lesion expands. Alternatively, block of lateral to medial conduction in the CS musculature with preserved conduction over the adjacent LA could also result in a change to proximal to distal CS activation times initially until the lesion expands to also incorporate the actual accessory pathway to LA muscle connection. If such were the case, one might expect "far-field" left atrial potentials going from distal to proximal to get to the septum via the LA prior to block to the A and such were not seen in the CS.

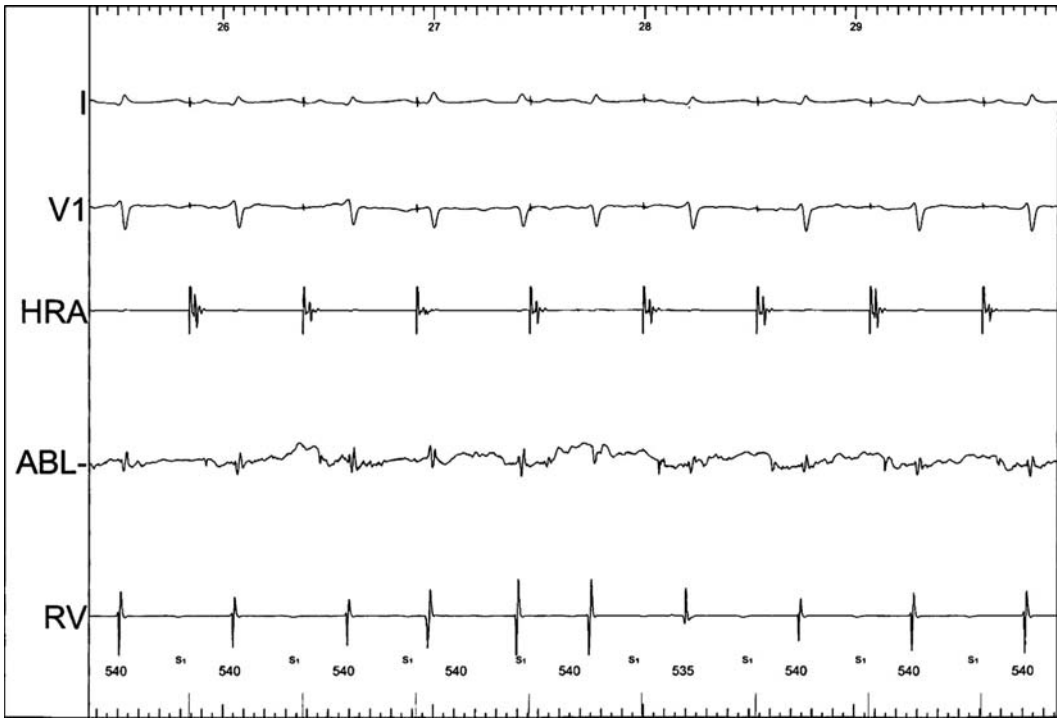
Tachycardia terminates without conduction to the atrium, and a PVC occurs on the last beat with normal retrograde septal activation sequence. There was no evidence of accessory pathway activation for the rest of the study and the patient has been arrhythmia free during follow-up.

Figure 6–9A



Radio-frequency energy is delivered through the distal electrode of an ablation catheter positioned near the os of the coronary sinus in a patient with AV node reentry. Energy was immediately discontinued when the following occurred. What would you do next?

Figure 6–9B

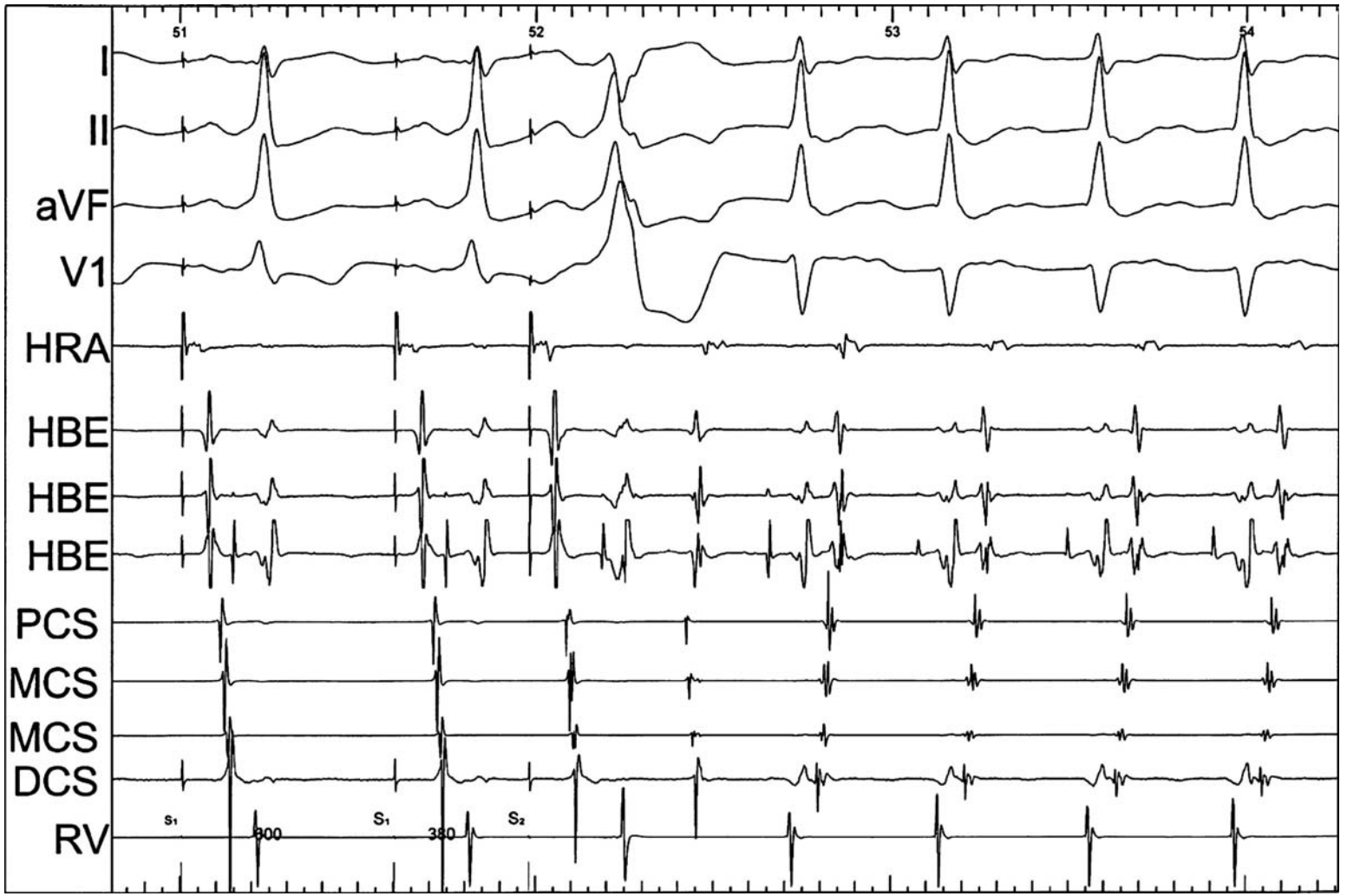


Explanation:

This patient had relatively poor retrograde conduction during the diagnostic part of the study and required small doses of isoproterenol for induction of sustained AVN reentry. Regardless, excellent junctional runs with 1:1 VA conduction during RF energy delivery often occur even in the absence of isoproterenol in these patients. Typically, we start with no more than 15 W of energy and ramp up as needed with careful observation of retrograde conduction. As the energy was increased to 20 W retrograde block occurred.

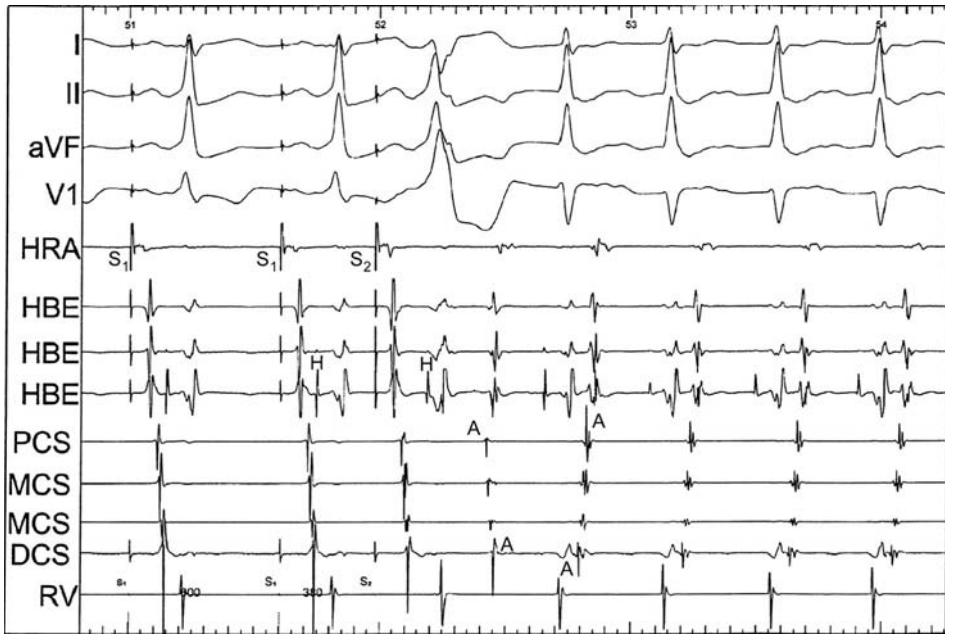
There are several methods to troubleshoot this situation. The easiest may be to move the catheter to a more posterior position, but the resultant site may not yield any junctional beats at all. We prefer to pace the atrium during energy delivery and observe anterograde AV conduction, as is shown on the second tracing. Junctional beats commonly interrupt the drive train and that is fine as long as the subsequent atrial beats capture the AVN and conduct to the ventricle. Commonly, the atrial paced rate needs to increase during the ablation if more junctional complexes interrupt the drive train, and this maneuver requires the operator to keep a very close vigil on the paced AV or PR interval. Other strategies may be tried. Ablation during isoproterenol to allow better retrograde conduction may work but there is often more catheter movement during isoproterenol. Cryoablation with careful monitoring of AV conduction is also reasonable.

Figure 6–10A



During programmed atrial stimulation, the premature beat initiated supraventricular tachycardia. Where do you think you will need to ablate to cure this patient?

Figure 6–10B



Explanation:

The best way to approach this complex tracing is to state what you know and then what maneuvers are needed to determine the rest. There is clear anterograde preexcitation over a left lateral AP during atrial pacing, and the premature stimulus (S2) causes prolongation of the AH interval with the His moving into the more preexcited QRS complex, characteristic physiology of an AV muscle connection. This is followed by an atrial complex with the initial activation at the PCS electrode, which was situated near the CS ostium. The differential for this beat is an atrial ectopic complex or retrograde activation over the AV node or a septal AP. More information is needed to determine this. The next atrial complex has a different activation pattern and the earliest activation is now on the DCS lead that is near the lateral margin of the mitral ring. This could be a left atrial tachycardia, but its location is consistent with the anterograde AP location, and, more importantly, there is slight wobble or irregularity in the AH intervals with constant VA intervals, confirming AV node involvement in the tachycardia. Another way of looking at this is that the V–V interval change precedes the A–A interval change during this cycle length irregularity and this is not compatible with atrial tachycardia. This is AV reentry and one ablation site will be at the left-sided AP.

What about the septal activation pattern? At another part of the study we were able to get sustained tachycardia with this activation pattern. Note on the next figure that a PVC introduced at the time of anterograde His activation terminates tachycardia without conducting to the atria. This confirms AV reentry with AP involvement in the circuit. This patient also had a concealed posteroseptal AP that required ablation, and this was done after the left-sided AP was ablated.

Figure 6–10C

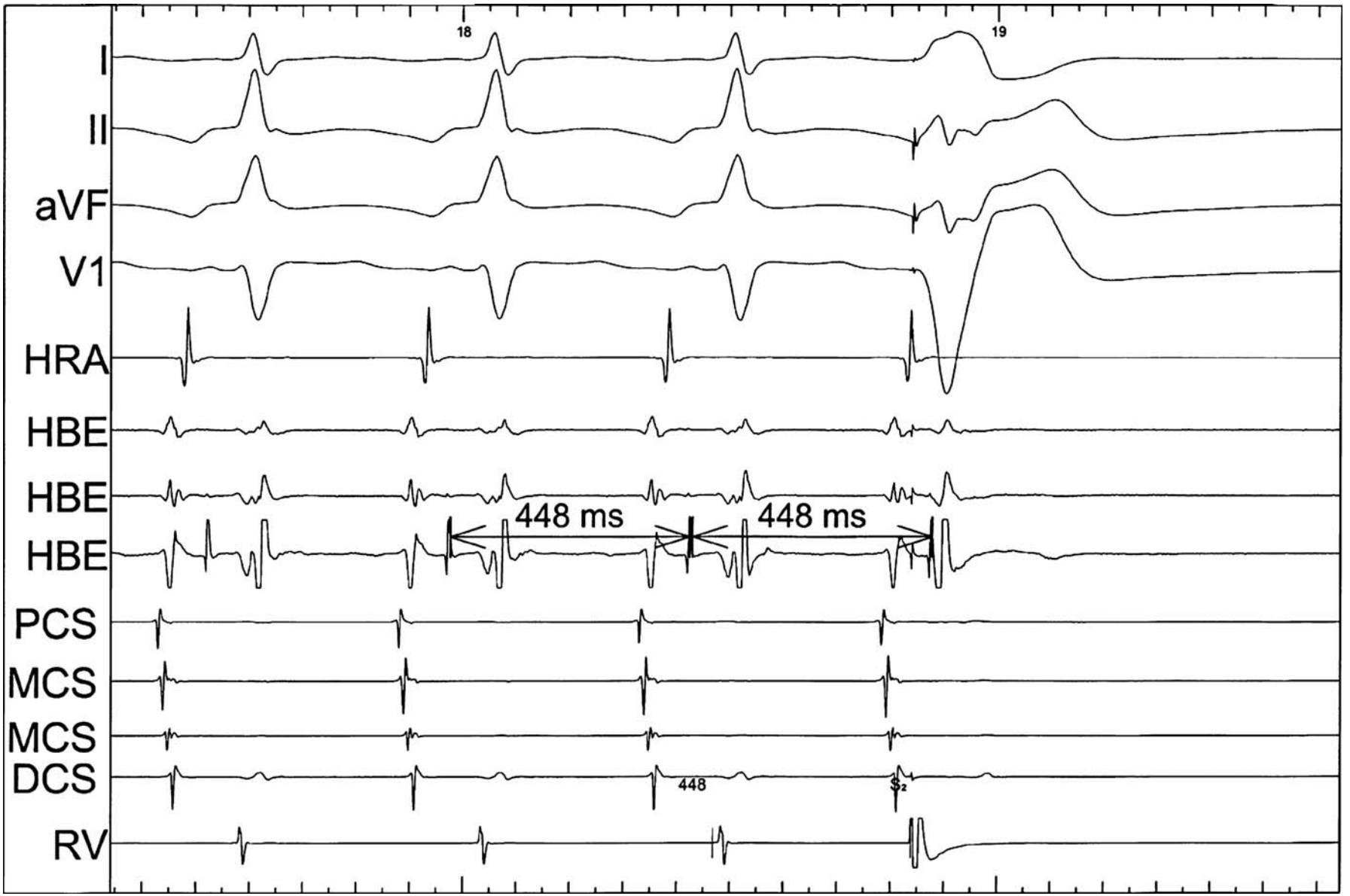

18
19
I
II
aVF
V1
HRA
HBE
HBE
HBE
448 ms
448 ms
PCS
MCS
MCS
DCS
448
S2
RV

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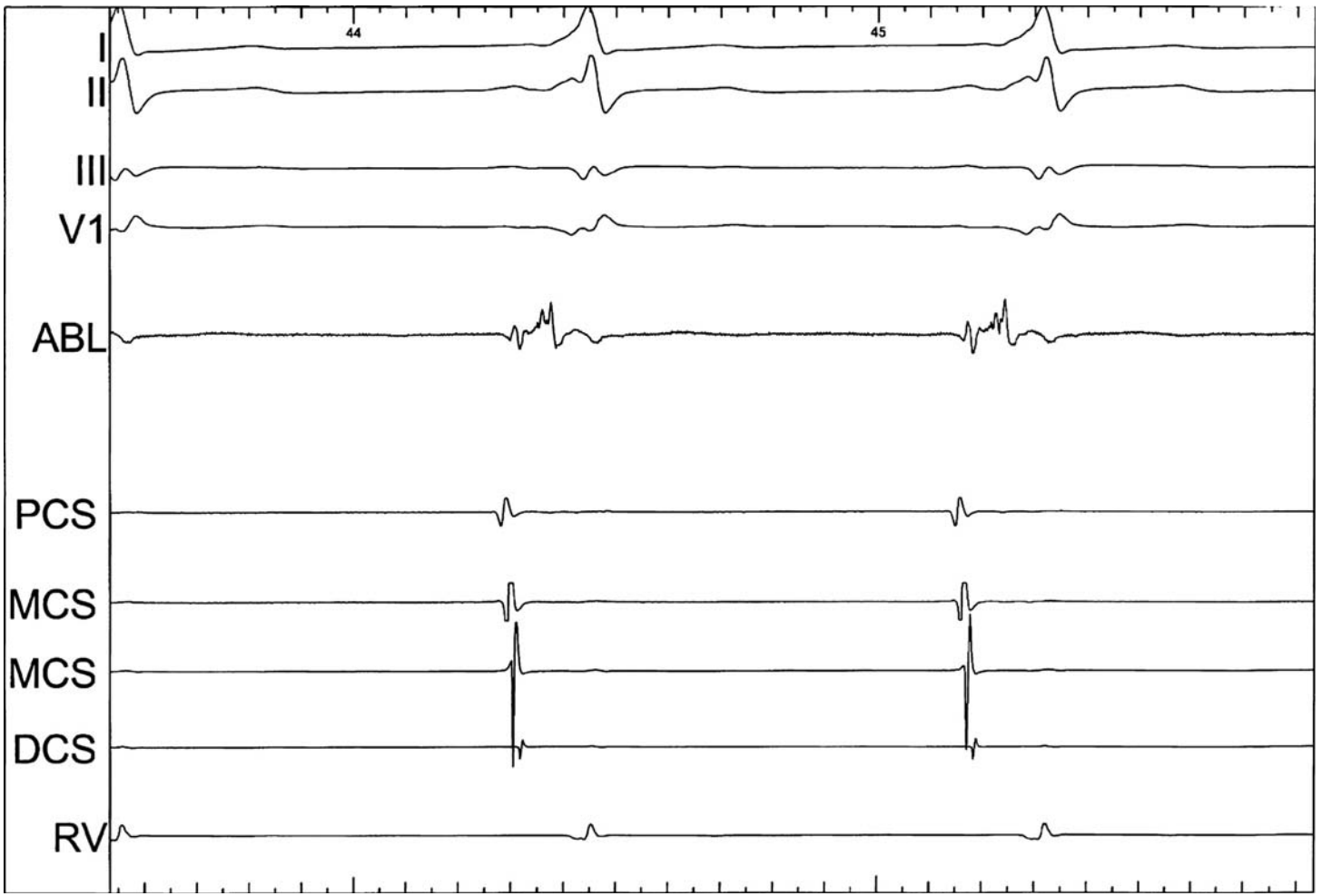
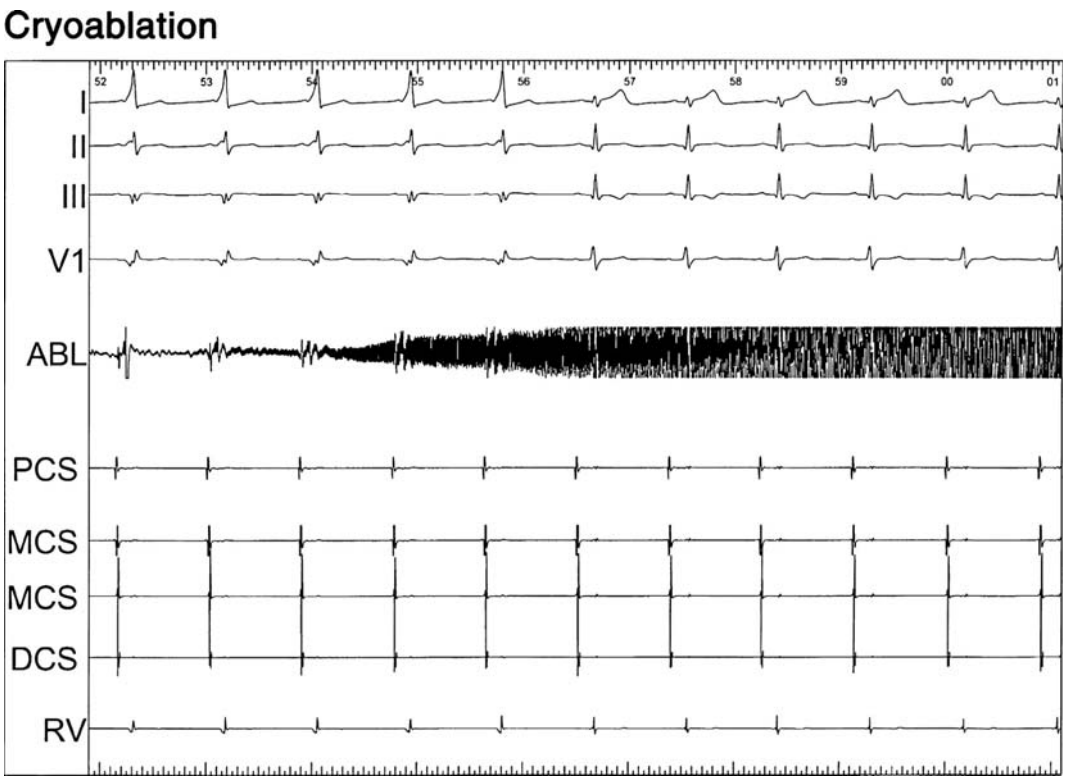
Figure 6–11A



A 19-year-old highly ranked athlete has WPW syndrome and documented PSVT and comes for study and ablation. The AP is located in the area of the AV node, and during tachycardia the earliest retrograde atrial electrogram is on the His catheter that also has a relatively large His deflection. The site with the optimum local electrogram on the ablation (ABL) catheter is shown, and there is continuous electrical activity from the A to the V with a probable AP potential present. What technique would you use to ablate this pathway, or would you not do it at all?

Figure 6–11B

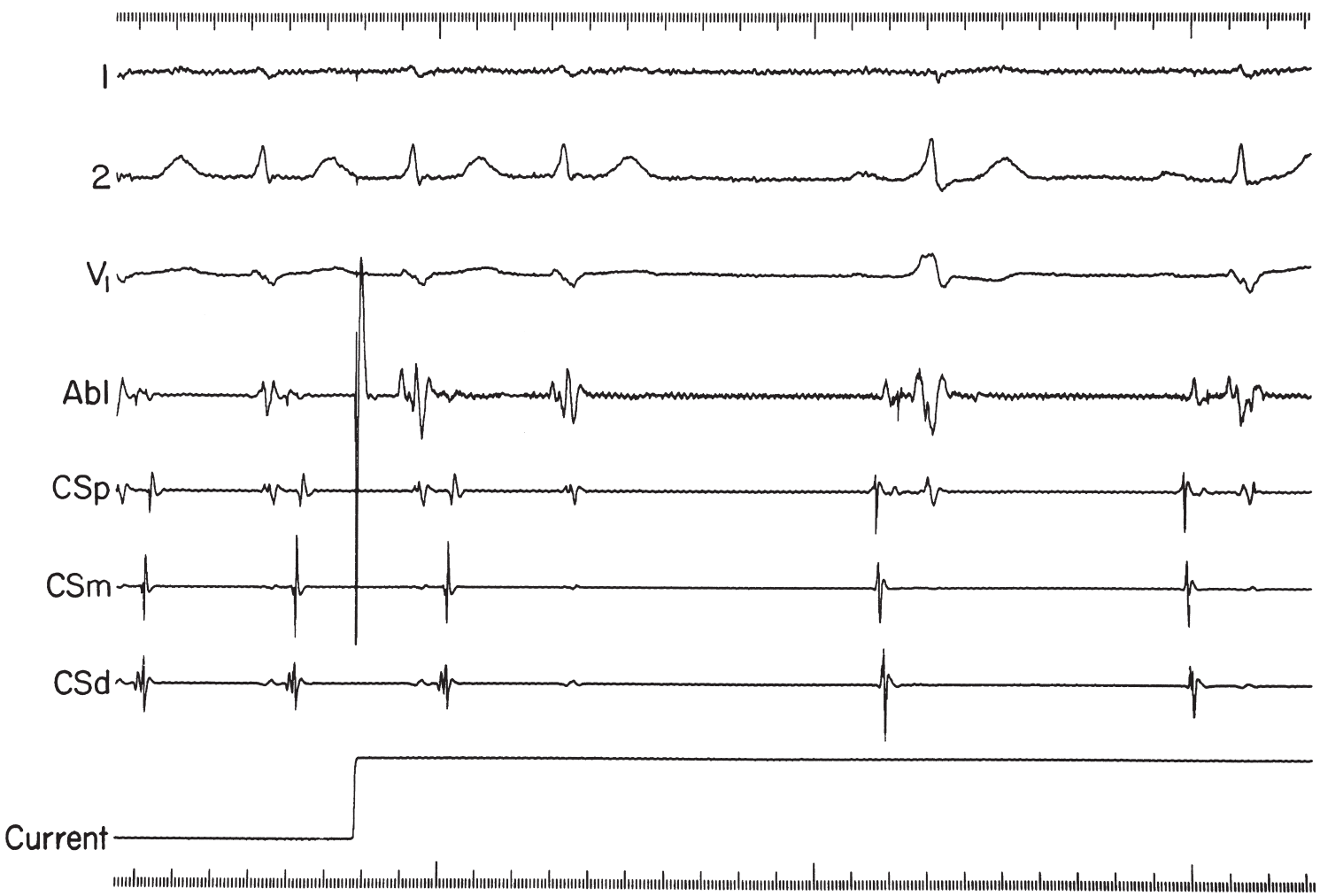


Explanation:

The key is to preserve the normal conduction system while trying to get a cure. If radio-frequency energy is used, you can start with very low energy levels, for example, 10 W, and go up very slowly with the hope that preexcitation will disappear before you increase the energy to levels that could result in damage to the AV node, typically more than 15 W. Alternatively, AV reentry can be induced and energy delivered during atrial pacing that has captured the circuit, and this will allow observation of the AV conduction during ablation and also prevent catheter movement after conduction over the pathway is gone. Another method is to pace the atrium until preexcitation disappears, if possible, and then introduce energy. The latter two techniques will allow careful observation of AV conduction during ablation, but the effect of the energy on AP conduction can only be determined after the energy delivery is done.

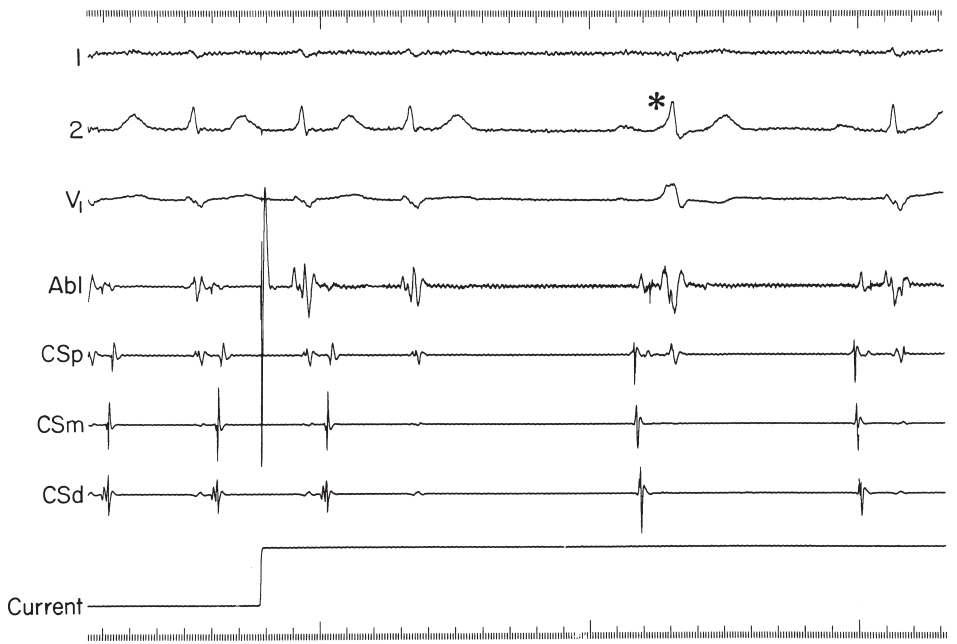
Initial ablation was with radio-frequency energy but very frequent junctional complexes interfered with the ablation attempt. Thus, cryoablation was employed as shown in the next figure and, fortunately, loss of preexcitation occurred without any AV conduction damage. This in fact is a superb location to use cryoablation.

Figure 6–12A



The patient is undergoing ablation of a left AP during ongoing AV reentrant tachycardia. How many accessory pathways are involved? CSp, CSm, and Csd represent coronary sinus electrodes from CS os to distal CS positions, respectively.

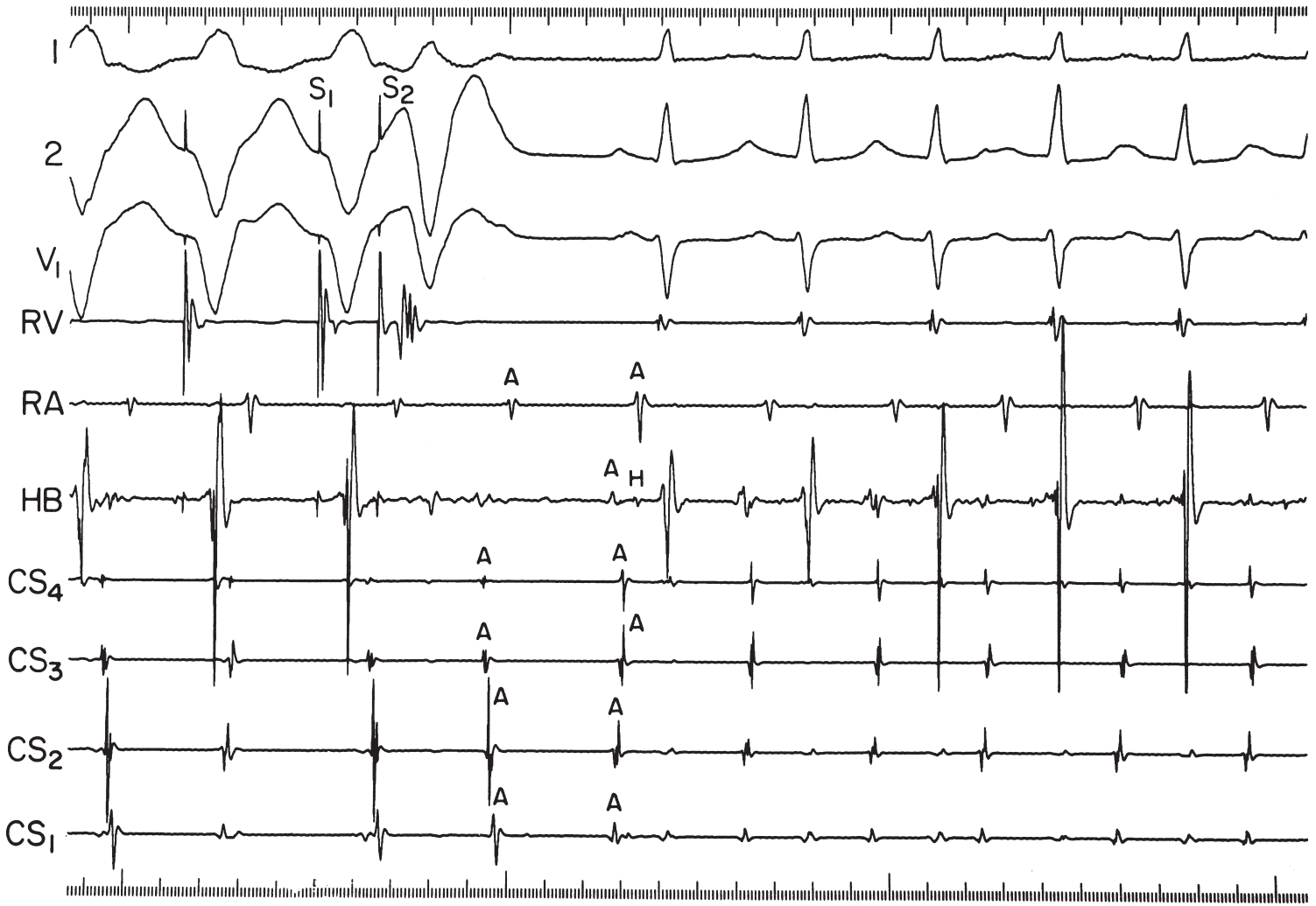
Figure 6–12B



Explanation:

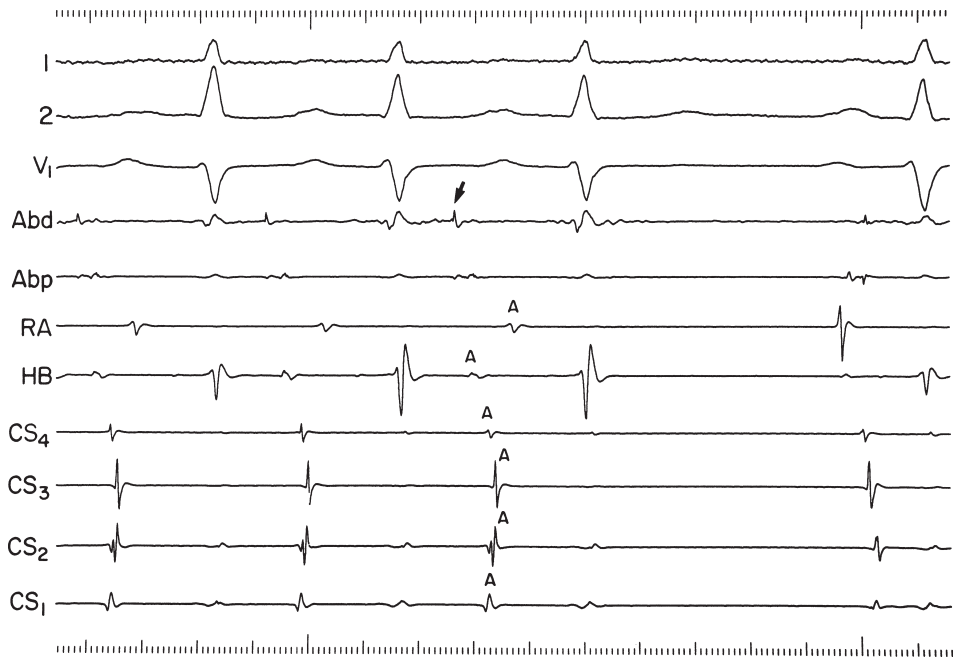
Tachycardia stops within two cycles after current is applied with block occurring in the AP. However, the first sinus escape cycle is associated with a preexcited QRS (denoted by the asterisk) with a left lateral preexcitation pattern. Retrograde conduction has been apparently ablated prior to anterograde conduction. There are several potential explanations for this. It is indeed possible that there were two closely spaced APs with initial ablation of the one responsible for retrograde conduction and the second for anterograde conduction. Alternatively, there may be longitudinal dissociation in a single fiber with the ablation injury originally sustained by the portion responsible for retrograde conduction. It may just be that the injury prolonged retrograde refractoriness to a point permitting termination of tachycardia with complete destruction of the pathway requiring a little more time. Finally, one cannot rule out a catheter-induced PVC occurring with the relative pause after termination of tachycardia.

Figure 6–13A



Where would one ablate this tachycardia recorded from a young individual with recurrent, paroxysmal tachycardia in the absence of preexcitation?

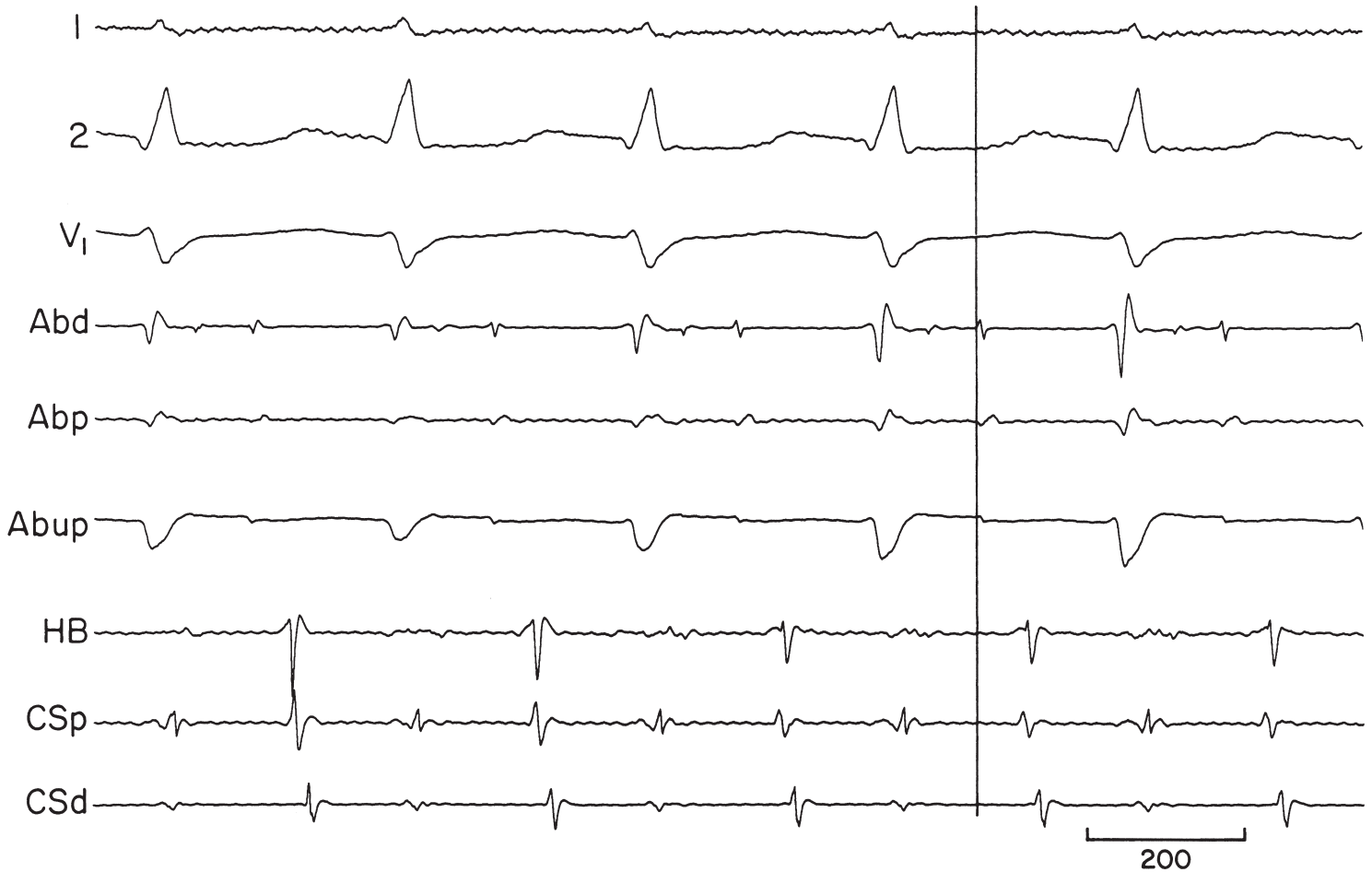
Figure 6–13B



Explanation:

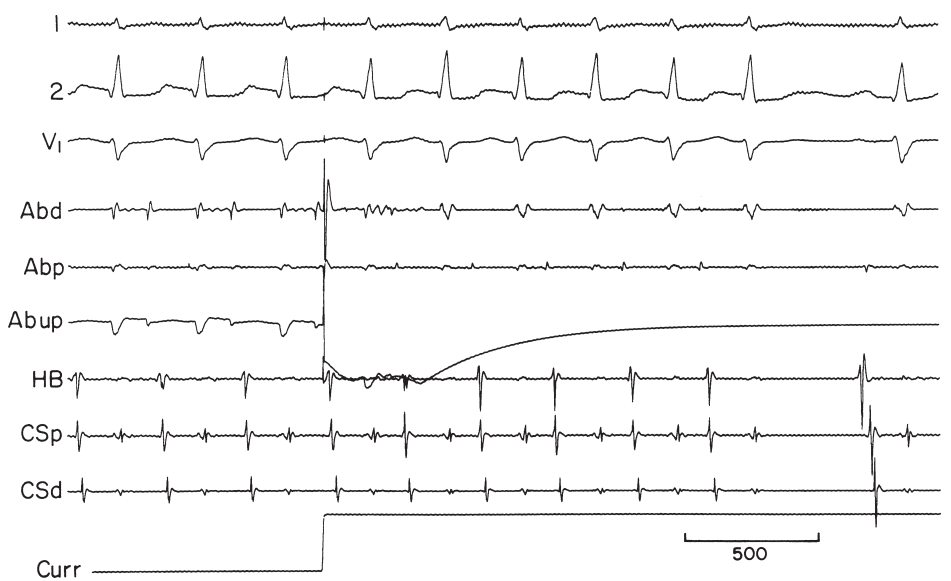
The record is most consistent with an atrial tachycardia (Table 1–7). The atrial activation sequence is abnormal and the first atrial activation related to tachycardia is not preceded by ventricular activation. In addition, the "apparent" VA conduction time is variable, whereas the AV conduction time is relatively constant. The atrial activation times at all the CS sites and the His bundle site are relatively close together and certainly not compatible with a single focal origin at the AV ring. In fact, the similarity of activation times at all recorded CS sites and the His bundle site would suggest that the focus is relatively equidistant from all of these sites, as indeed it was, in the roof of the left atrium. Ablation directed at the electrogram indicated (*arrow*) was successful.

Figure 6–14A



The record was from an individual otherwise well except for paroxysmal tachycardia. The tachycardia could be induced by rapid atrial pacing and extrastimuli, although induction at any given rate or coupling interval was poorly reproducible. Would the site indicated be acceptable for ablation? Abd, distal electrode pair of ablation catheter; Abp, proximal pair; Abup, distal unipolar electrode.

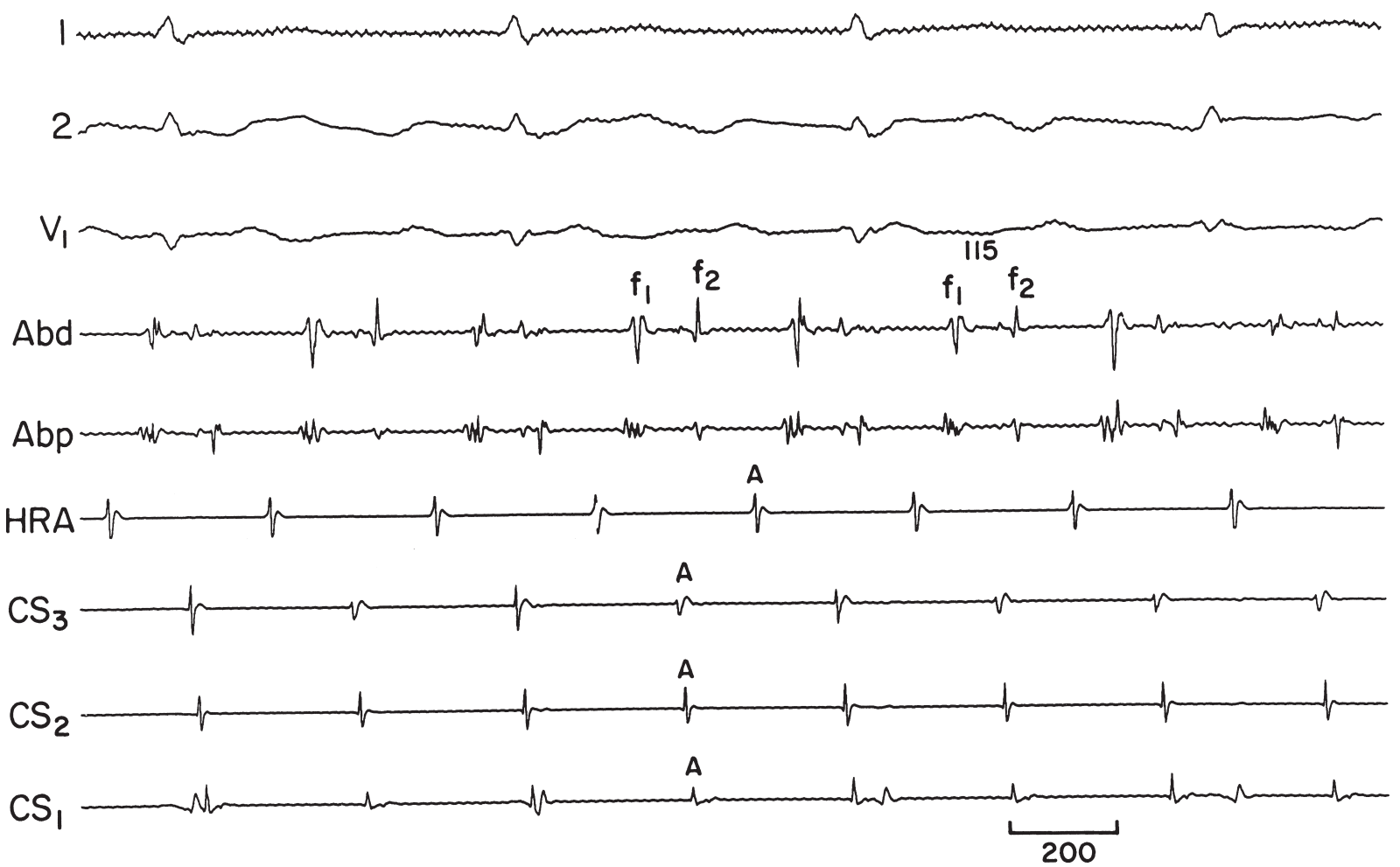
Figure 6–14B



Explanation:

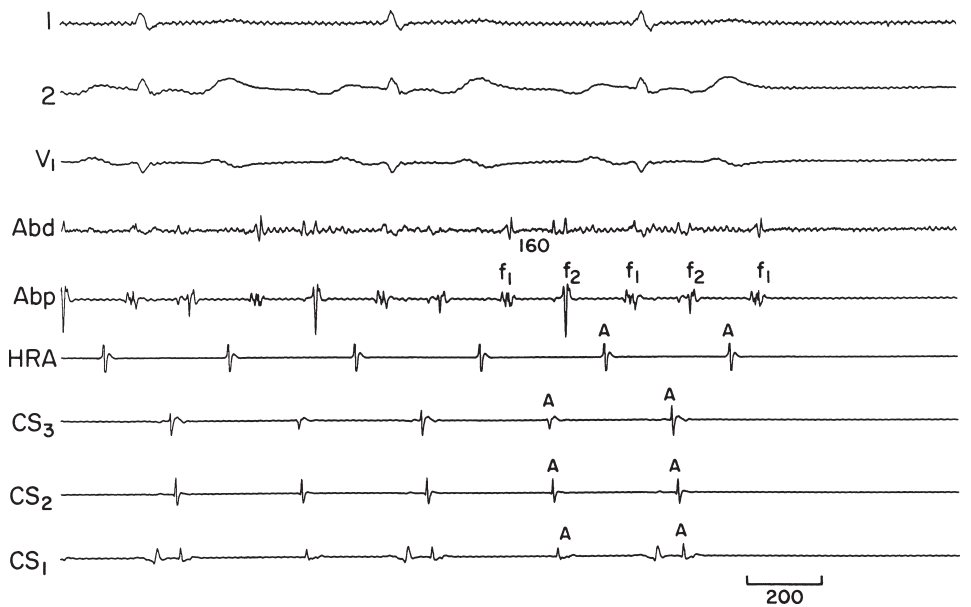
In the absence of a global atrial activation map, it is difficult to know exactly how early the earliest atrial activation during tachycardia should be. The surface P wave does give some guide if clearly seen. One can usually program PVCs into the cardiac cycle during tachycardia with the intention of advancing the QRS and exposing atrial activity without altering the atrial activation sequence. The unipolar electrogram may be helpful because this records both far-field and near-field activity. A rapid, "sharp" QS deflection at the unipolar electrogram indicates that activation is proceeding from that site and is a favorable observation. In this instance, there is no positive initial deflection in the unipolar electrogram (Abup) but there does appear to be a short isoelectric component. Nonetheless, a clear spike almost midway between ventricular and atrial activation at the ablation catheter prompted an attempt at ablation at this left atrial appendage site and was successful.

Figure 6–15A



The tracing was recorded from a middle-aged patient undergoing ablation for "typical" atrial flutter. The ablation catheter is located inferior to the CS os, near the isthmus between the tricuspid valve and the inferior vena cava. What is the significance of the double activation (f_1, f_2) recorded from the ablation catheter?

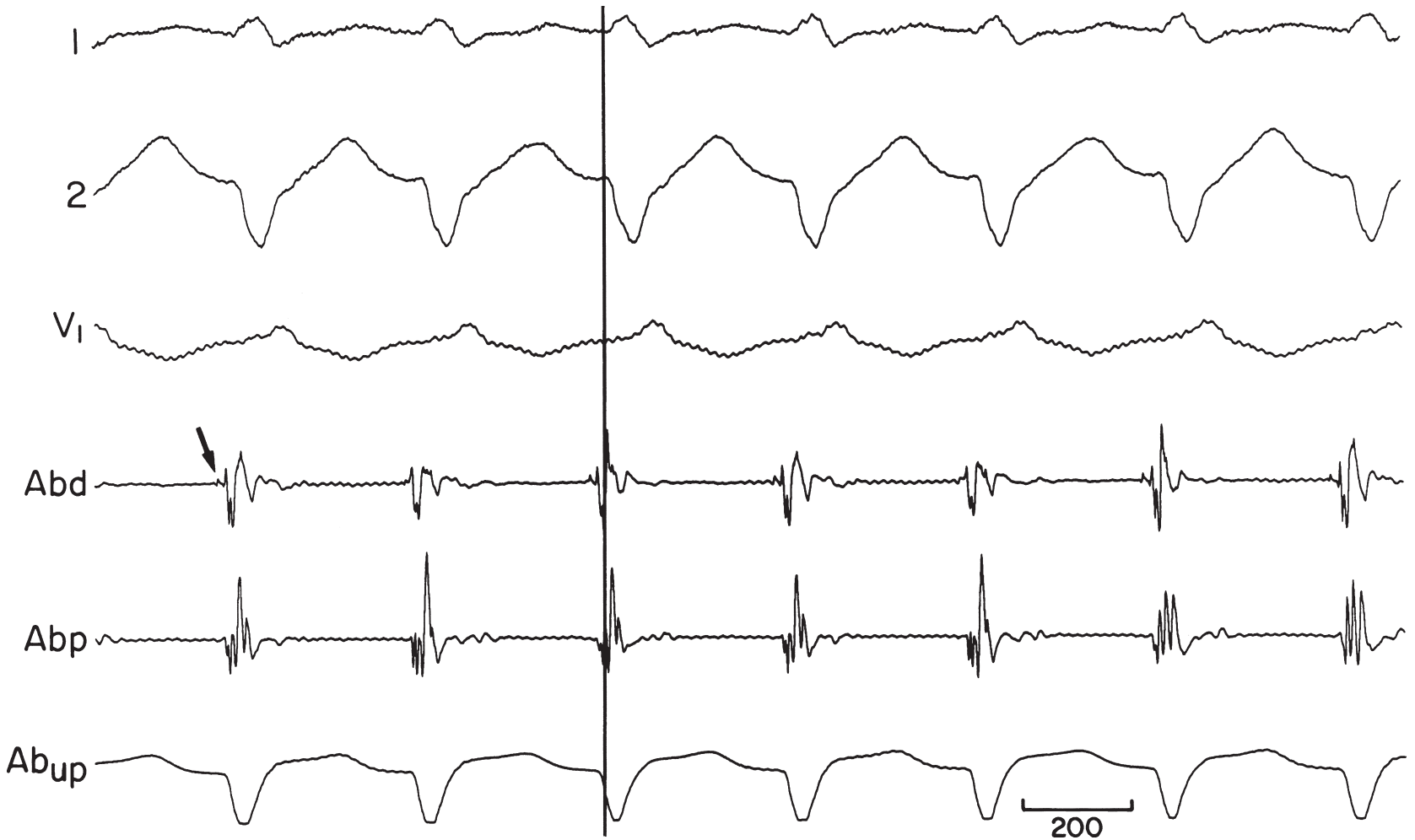
Figure 6–15B



Explanation:

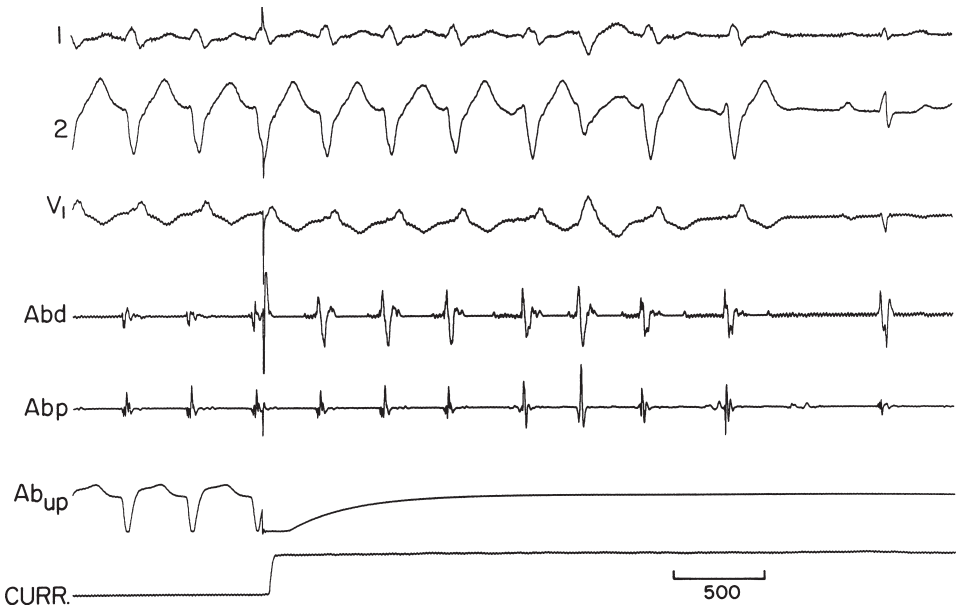
These two very distinct potentials (f_1, f_2) are separated by 115 milliseconds and suggest that the catheter is within a zone of slow conduction with the two activations representing entry (f_1) and exit (f_2) from the zone of slow conduction. Alternatively, the catheter could be straddling a line of block and recording activation on each side of the line of block but not necessarily located in a zone critical to perpetuation of reentry. In this instance, application of current at that site resulted in prolongation of the interval between f_1 and f_2 with termination of tachycardia after f_1. This would suggest, in retrospect, that the site was a critical zone of slow conduction with f_1 proximal to it and f_2 distal to it. It is, of course, not necessary to demonstrate this phenomenon for successful ablation.

Figure 6–16A



The record was taken during an ablation session in a patient with idiopathic left ventricular tachycardia (Belhassen type) with the ablation catheter located in the apical, septal region. Is this a good ablation site?

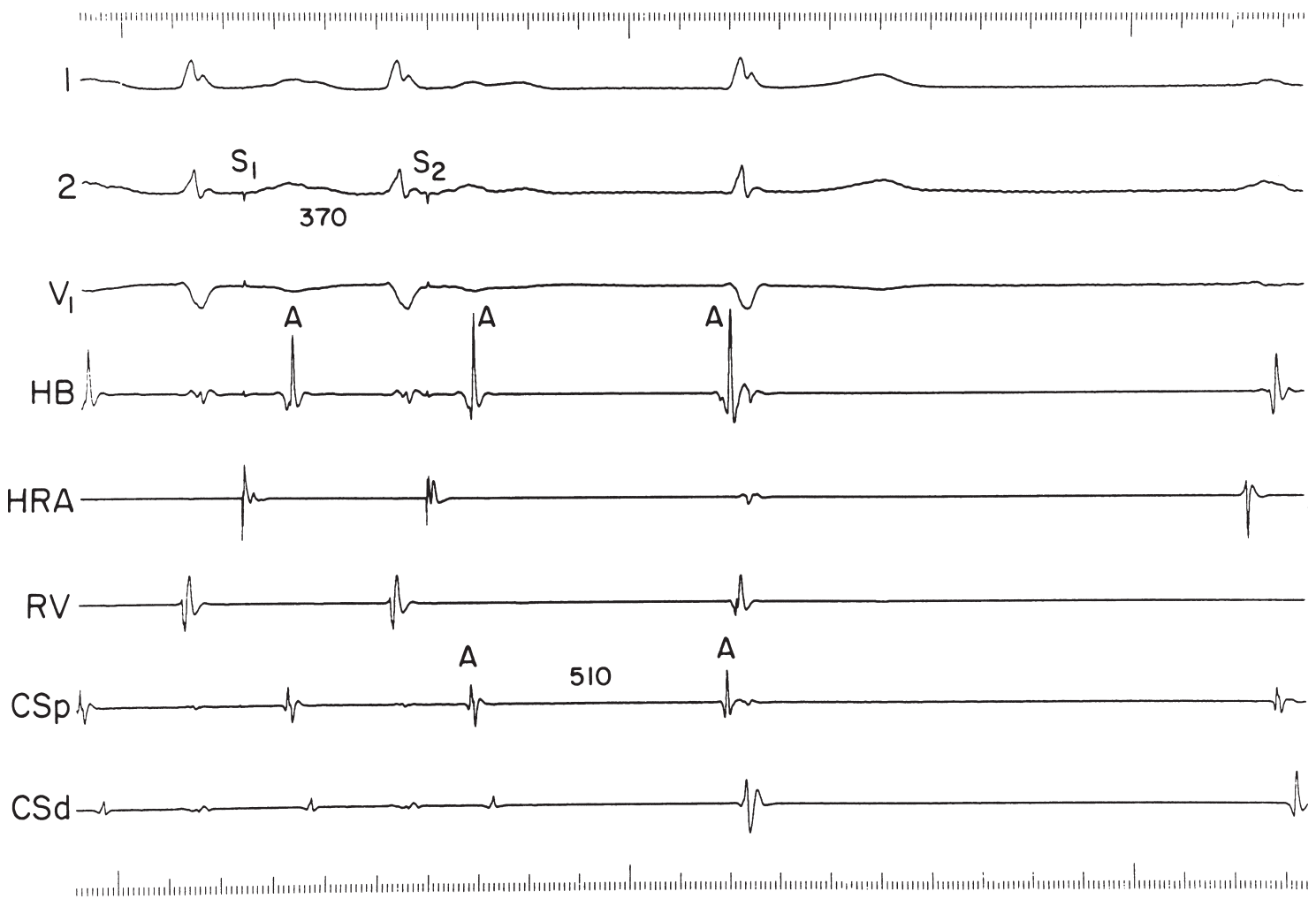
Figure 6–16B



Explanation:

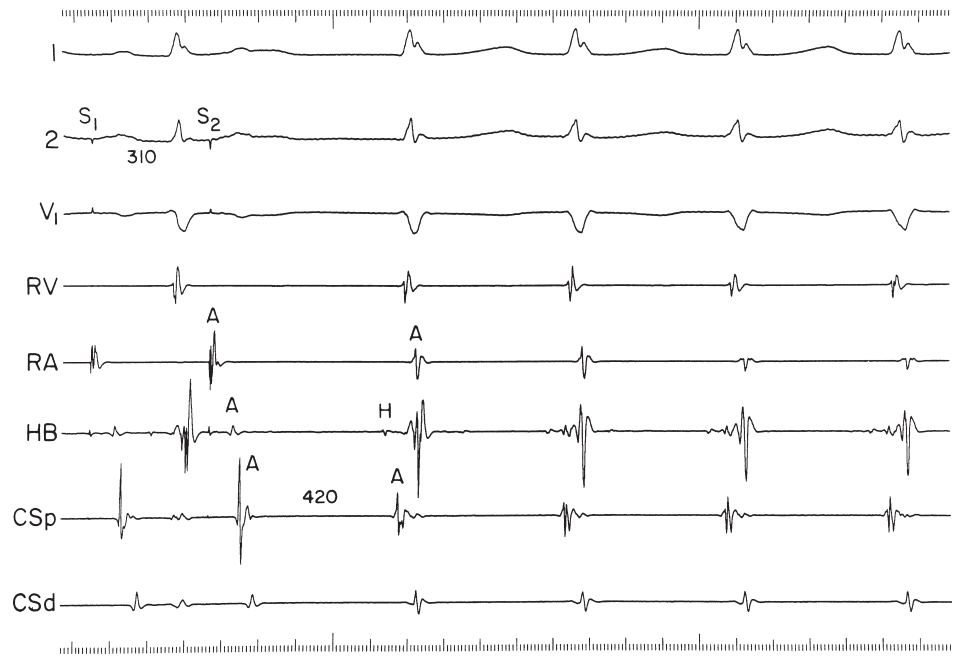
Although the mechanism is not entirely resolved, this type of tachycardia appears to be reentrant and the mechanism incorporates the distal Purkinje system. Ventricular activation at the successful site should precede the onset of ventricular activation during tachycardia on the surface QRS by at least 10 milliseconds. In addition; a rapid Purkinje spike can frequently be recorded prior to ventricular activation (see *arrow,* Fig. 6–16*A*). The unipolar component of the ventricular electrogram at the ablation electrode should have a rapid and downsloping QS deflection without initial positivity near the breakout point reflecting that activation is spreading from that site. Ablation at this site (30 W) was followed immediately by a slight slowing and irregularity of tachycardia prior to termination at 5 seconds.

Figure 6–17A



This patient had readily inducible AV node reentry (typical) prior to ablation in the "slow pathway zone" at the tricuspid annulus anterior to the CS os with this phenomenon observed after ablation. What is its significance?

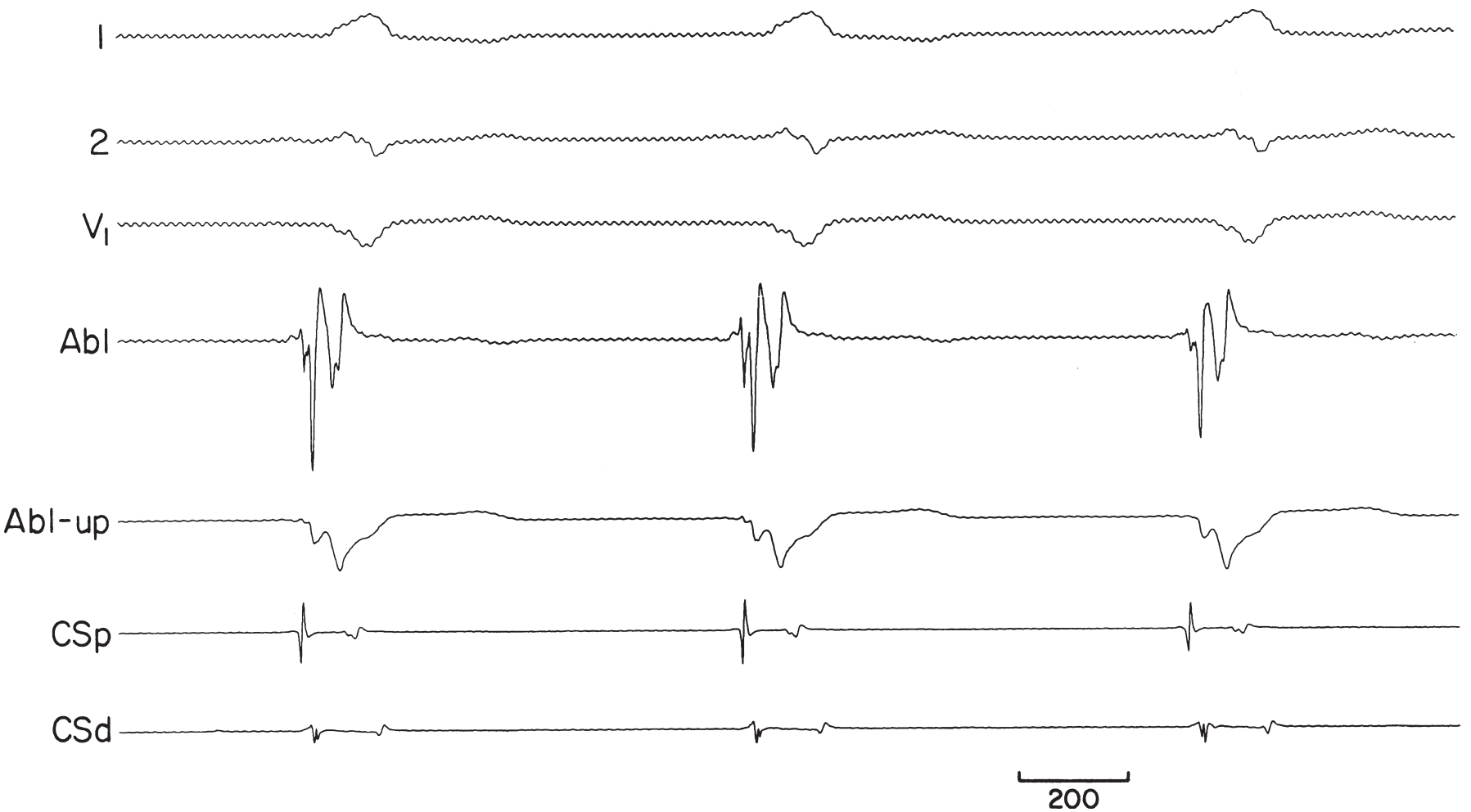
Figure 6–17B



Explanation:

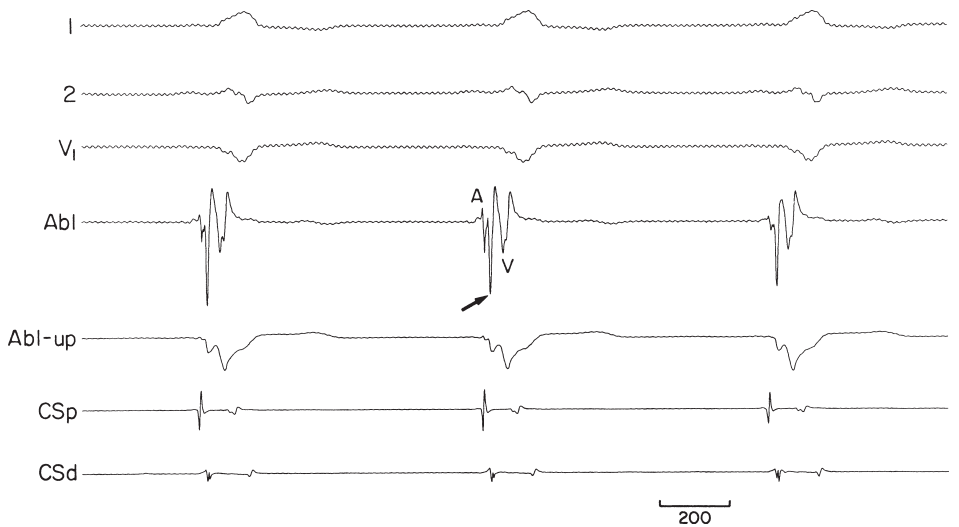
The phenomenon in the record was reproducible and indicates residual slow pathway conduction associated with an AV node echo. Although elimination of slow pathway conduction is a clear and useful end point, it is not required for a successful clinical result. The addition of isoproterenol in such an example might be useful to test the integrity of the total circuit and see if sustained tachycardia can be induced. Examination of the *preablation* induction (see Fig. 6–17*B*) shows that the clinically documented tachycardia was induced by an extrastimulus that resulted in an interatrial interval of 420 milliseconds measured at the proximal CS and this was the cycle length of tachycardia. After ablation, however, this interval prolonged to 510 milliseconds. This would suggest that conduction now occurs over a second, slower pathway that may be incapable of sustained reentry or, alternatively, sufficient damage to the previous slow pathway has occurred to achieve the same result. This residual slow pathway conduction may be clinically irrelevant.

Figure 6–18A



The tracing was recorded from a patient with a posteroseptal preexcitation pattern who was undergoing ablation. The ablation catheter is recording from the origin of the middle cardiac vein. What does the electrogram demonstrate?

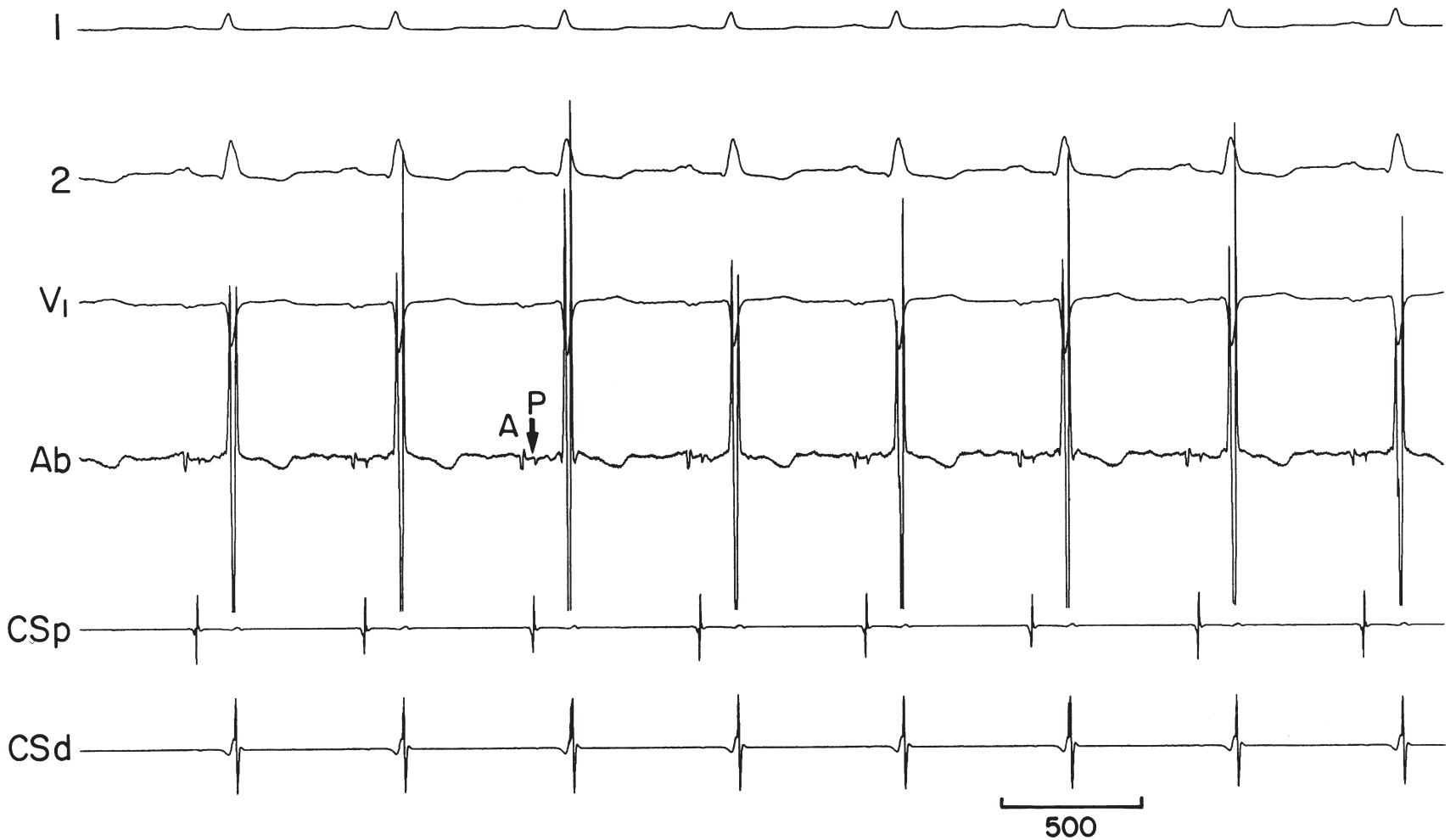
Figure 6–18B



Explanation:

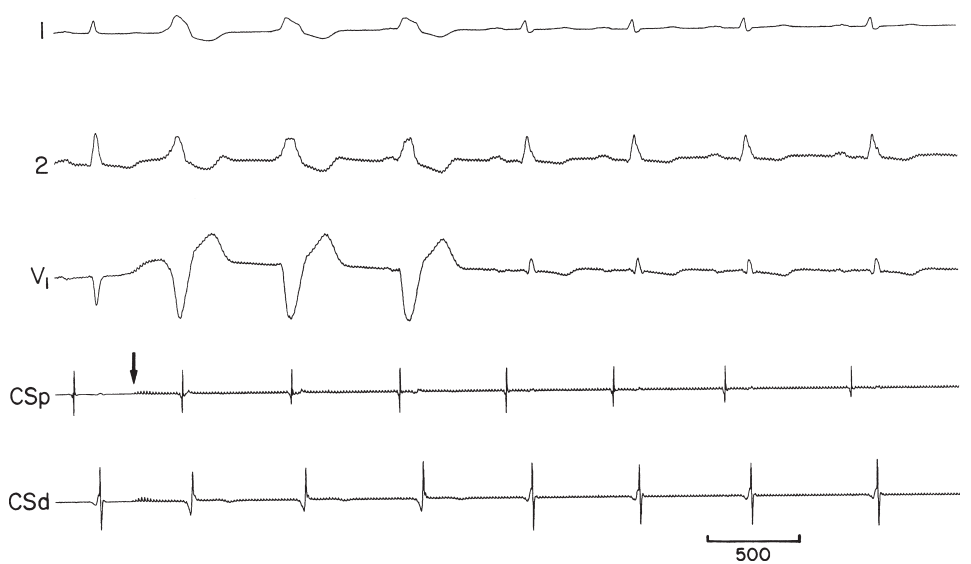
The electrogram is triphasic with the largest component being the middle component (*arrow*), which we interpreted as an AP potential. Electrograms in this region (within the coronary venous system) not infrequently have accessory pathway potentials larger than either the atrial or ventricular components. This could be verified by the extrastimulus technique, demonstrating that the AP potential does not belong to either the A or the V. For example, consider an atrial extrastimulus that results in block over the AP. Loss of the putative AP potential without change in the atrial electrogram clearly indicates that the potential in question was not part of the atrial electrogram. This was not done in this instance but the site was a successful one.

Figure 6–19A



The electrogram at the ablation catheter (AB) was recorded on the tricuspid AV ring (subvalvular approach) at approximately 9 o'clock as viewed in the left anterior oblique (LAO) projection. The patient presented with antidromic tachycardia related to an atriofascicular pathway. Is this the best "target" for ablation?

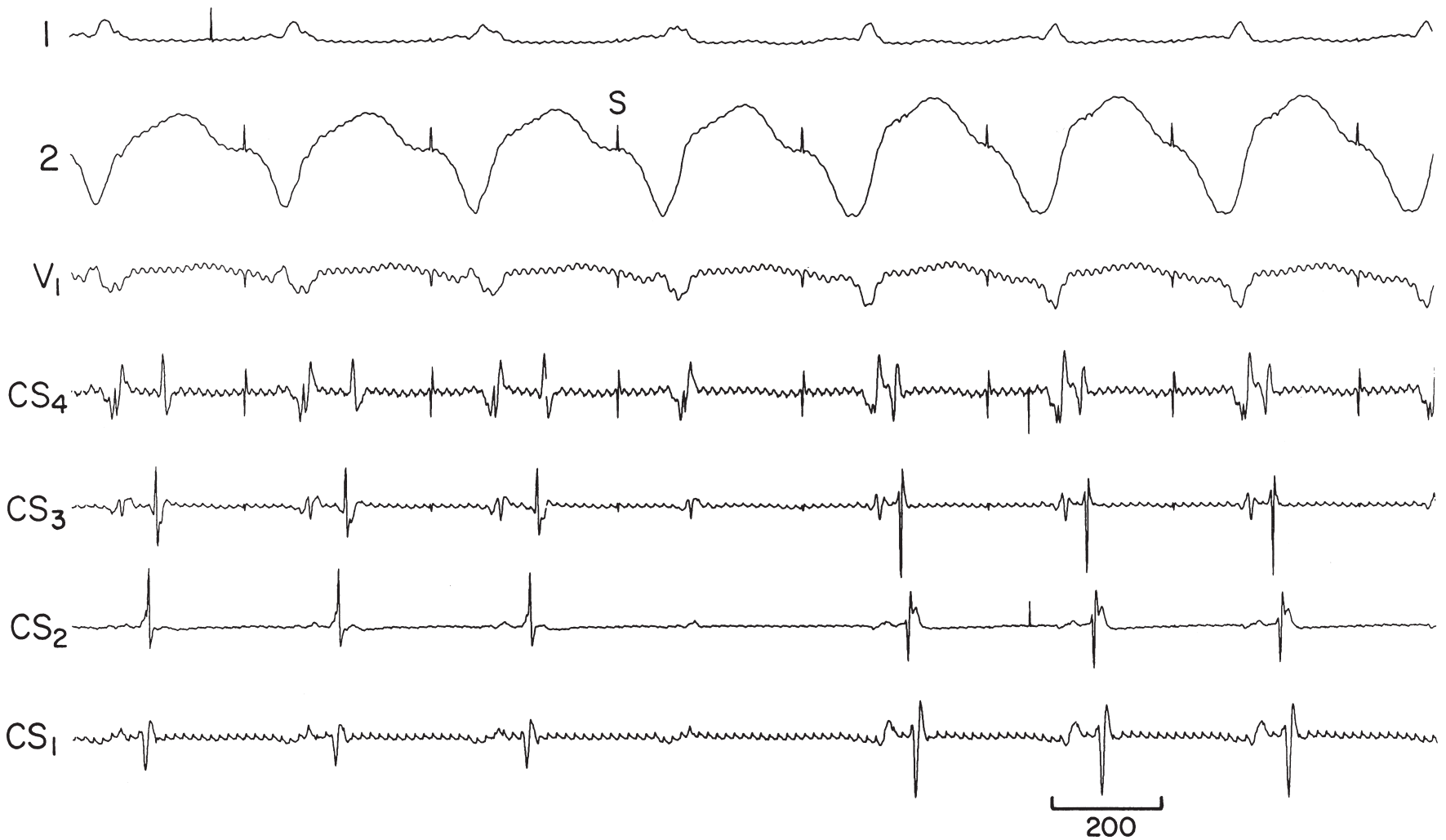
Figure 6–19B



Explanation:

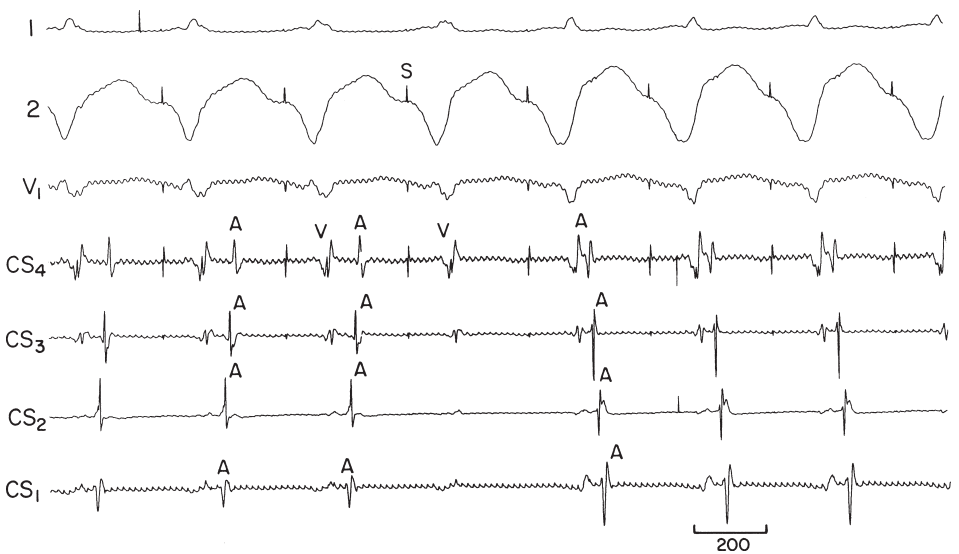
The optimum target site for ablation of atriofascicular pathways can be problematic. Mapping of earliest ventricular activation at the AV ring is not useful since the ventricular insertion site is well within the RV, near the RBB terminus. Mapping retrograde atrial activation is not useful since such pathways rarely (never?) exhibit retrograde conduction. Pacing the AV ring to find the site of pacing that gives the shortest AV conduction time has been used but is problematic in its resolution and the fact that the pathway is decremental with variability in AV conduction time. Thus, location of the pathway potential in this particular entity at the AV ring is by far the most useful means of obtaining an appropriate target for ablation. This potential can actually be tracked through the RV to its insertion site but this is technically difficult. At this successful site, turning on current (*arrow*) immediately results in accelerated rhythm with the patient's preexcited morphology that undoubtedly represents the response of this pathway to injury. This is reminiscent of the junctional tachycardia that is seen during ablation for AV node reentry in the region of Koch's triangle and is consistent with the speculation that these are AV node-like structures.

Figure 6–20A



The patient was undergoing ablation of a left lateral pathway with unidirectional retrograde conduction. The decision was made to ablate during entrainment of AV reentrant tachycardia. How does one explain the sequence of events?

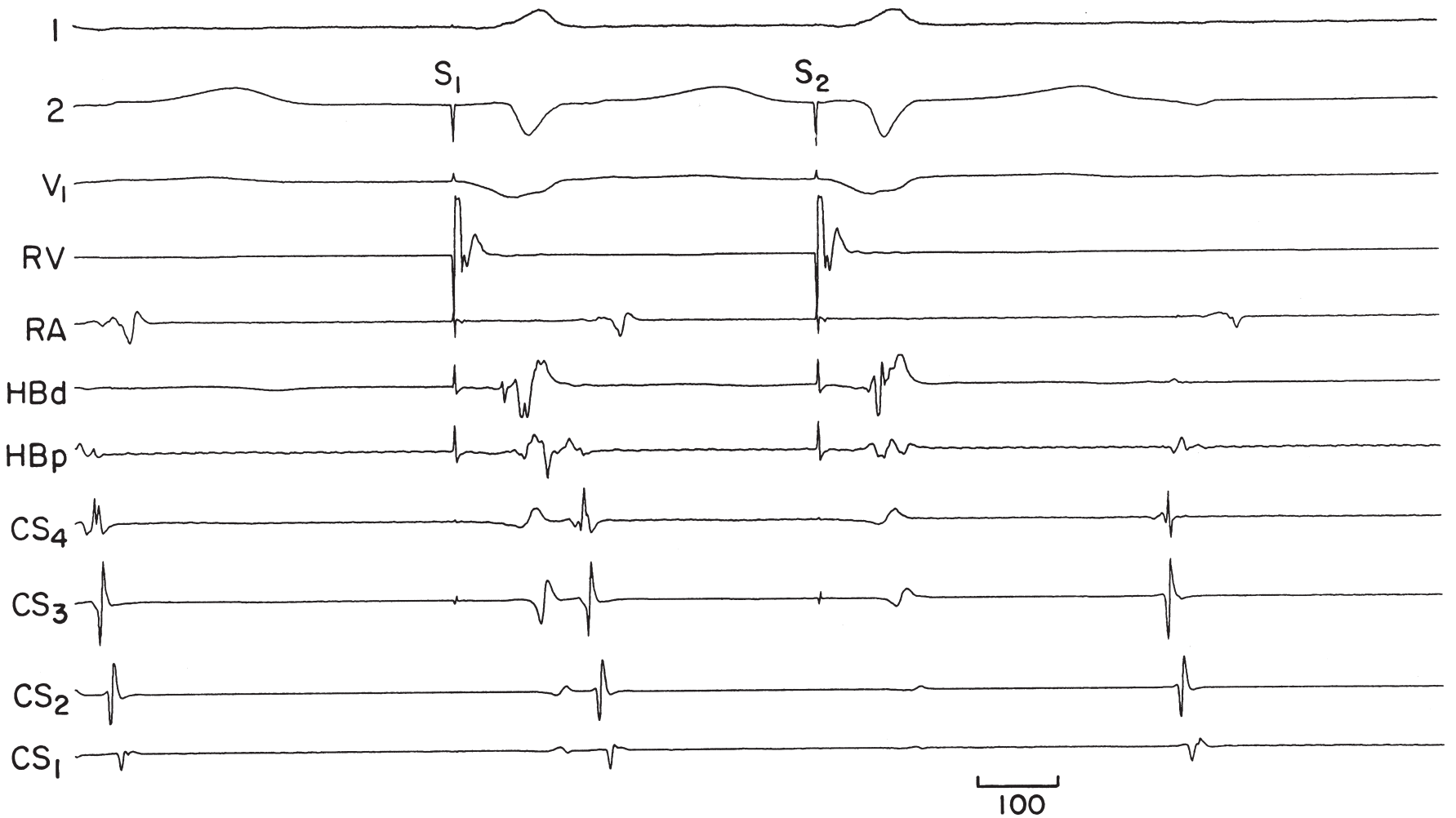
Figure 6–20B



Explanation:

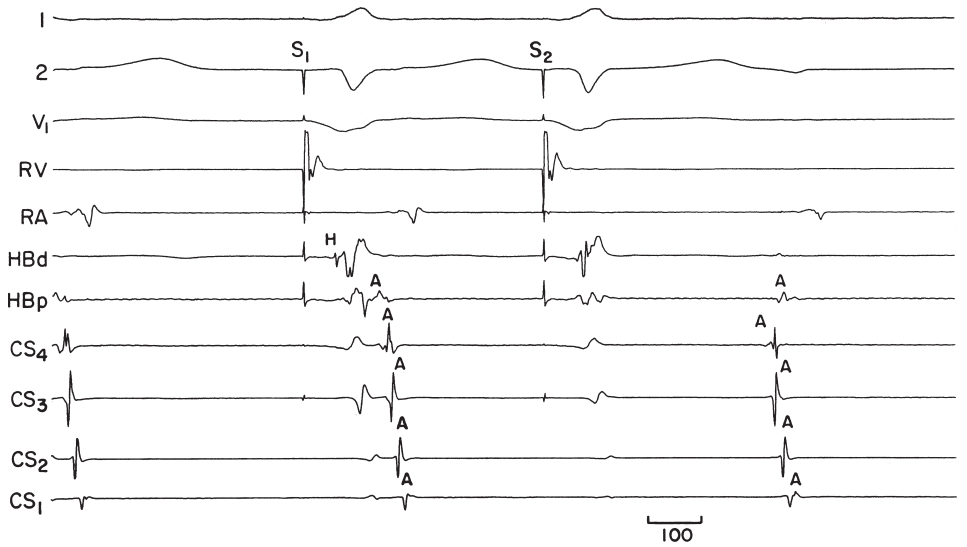
Ablation during tachycardia can be problematic because sudden cessation of tachycardia with conduction block over a pathway can result in catheter movement. At times, ablation during tachycardia can be very useful such as in the present example where there was no anterograde conduction and analysis of retrograde conduction was obscured by excellent AV node conduction. During entrainment at a rate slightly faster than the intrinsic rate of tachycardia, the AP is activated orthodromically to the circuit and the AV node is activated both antidromically (retrograde AV node conduction) and orthodromically via the advanced atrial activation over the AP. This is verified during ablation when loss of AP conduction resulted in complete VA block for one cycle. Absence of retrograde AV node conduction after this cycle is best explained by concealed anterograde conduction in the AV node by the previous atrial activation over the AP. The ventricular cycle following the totally blocked one proceeds retrogradely over the normal AV conduction system. Continuation of pacing after block over the AP allows for a stable catheter position throughout the delivery of current.

Figure 6–21A



The record was obtained from a patient immediately after apparently successful ablation of a left lateral AP. S_1 is the last drive beat and S_2 is a ventricular extrastimulus. What is the observation and its significance?

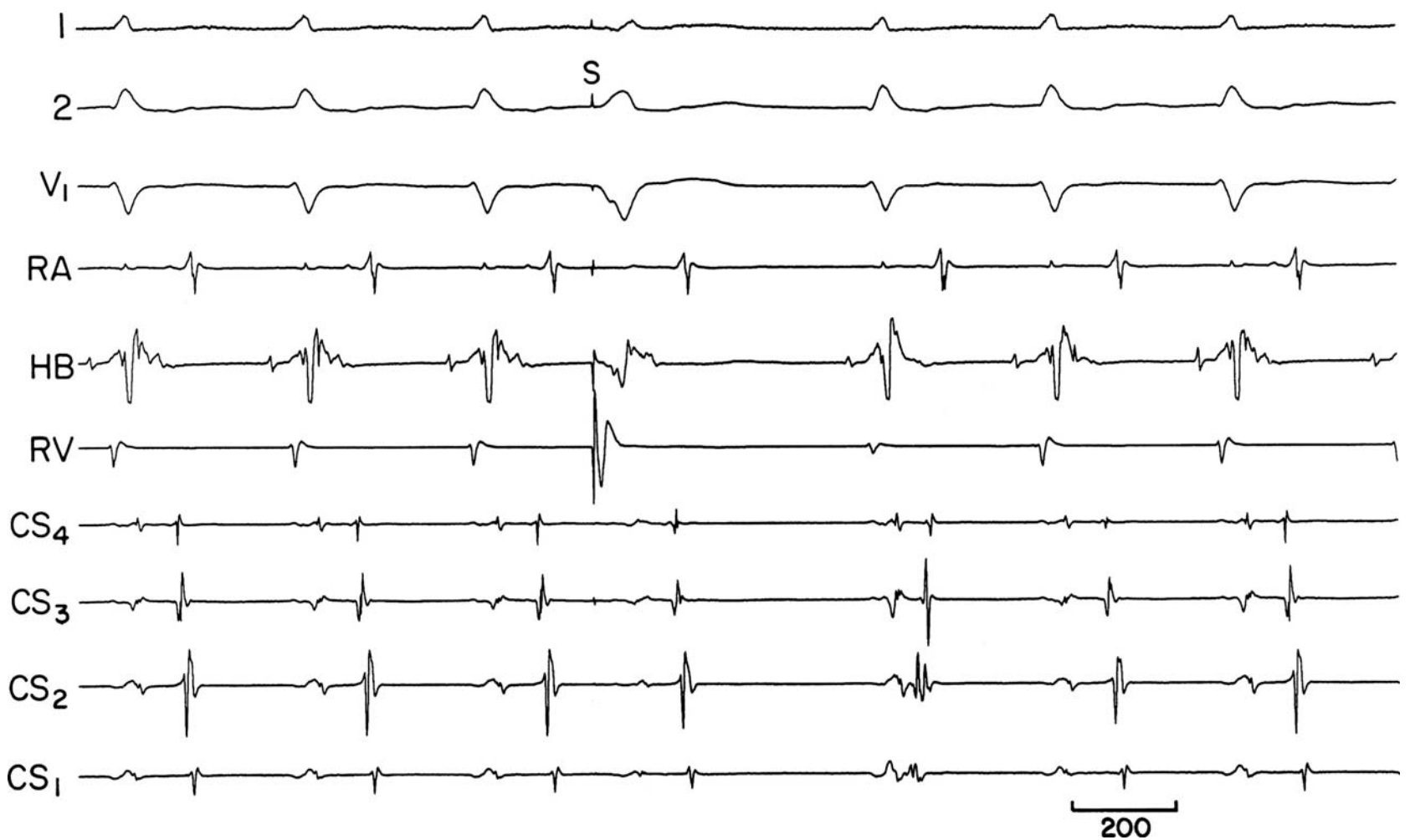
Figure 6–21B



Explanation:

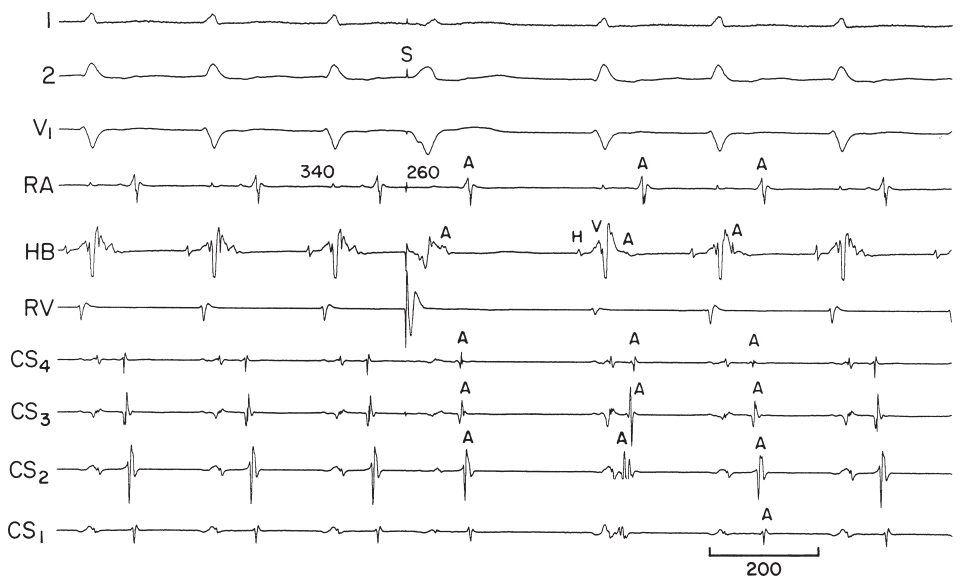
Atrial activation sequence during the drive cycle is compatible with conduction over the normal AV conduction system with central atrial activation sequence clearly preceded by a His deflection. With the extrastimulus, the His appears to be displaced into the QRS, the VA conduction time prolongs considerably, and the atrial activation sequence is altered with earliest activation now in the PCS near the orifice (CS_4, CS_3 early). This in all probability represents conduction over a slow, posterior AV node pathway, although a decremental, unidirectional posteroseptal pathway could not be definitively ruled out from this tracing. Dual AV node pathway physiology commonly coexists with the Wolff–Parkinson–White syndrome as well as the general population but ablation is only indicated if it can be related to a clinically relevant tachycardia.

Figure 6–22A



This patient has a right anteroseptal preexcitation pattern during sinus rhythm. A PVC introduced into ongoing supraventricular tachycardia yielded the phenomena shown. What needs to be ablated?

Figure 6–22B

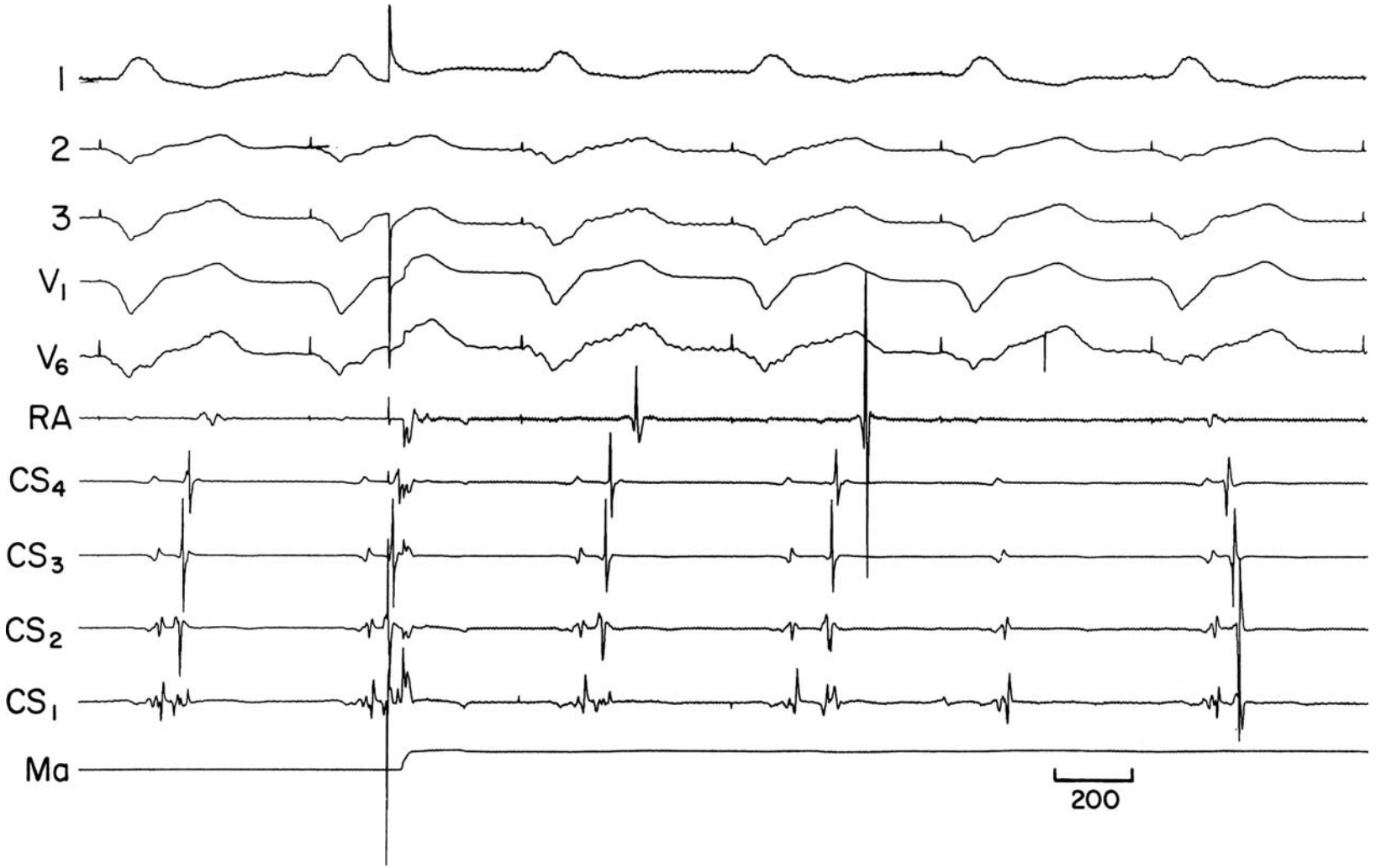


Explanation:

The first three cycles of tachycardia represent tachycardia as was repeatedly induced in this individual and demonstrated to be AV reentry utilizing a right anteroseptal pathway as the retrograde limb. Note that earliest atrial activation is at the His site. A PVC with the stimulus artifact inscribed approximately 50 milliseconds in front of the destined His (not seen) preexcited the atrium with no change in the activation sequence. The subsequent AH is very long and the first cycle after the PVC is associated with a different atrial preexcitation pattern, suggestive of a left lateral AP. The subsequent AH shortened and the former retrograde atrial activation pattern over the right anteroseptal pathway resumed. This patient clearly has a second left lateral AP. However, this pathway has a relatively long refractory period and is only seen after a very long AH interval. Sustained tachycardia over this pathway could not be demonstrated and, arguably, this pathway would not require ablation.

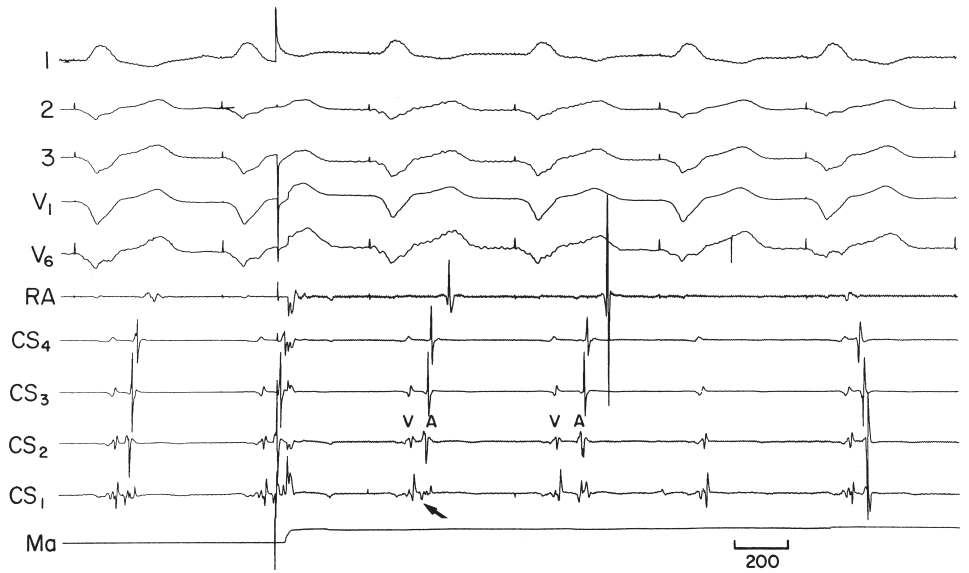
This patient also has dual AV node pathway physiology (the AV node curve relating AH to prematurity of an atrial extrastimulus was discontinuous after ablation) as evidenced by the long AH observed after the PVC during tachycardia. The slow pathway would not require ablation in its own right unless it could be demonstrated that it was capable of participating in AV node reentry. A clue that the patient does not have sustained AV node reentry is the absence of an AV node echo after the long AH. The right anteroseptal pathway is the "culprit" pathway and is the only one requiring ablation in this patient.

Figure 6–23A



The tracing was taken from a patient undergoing ablation of a left lateral AP that was done during ventricular pacing. How many APs are involved?

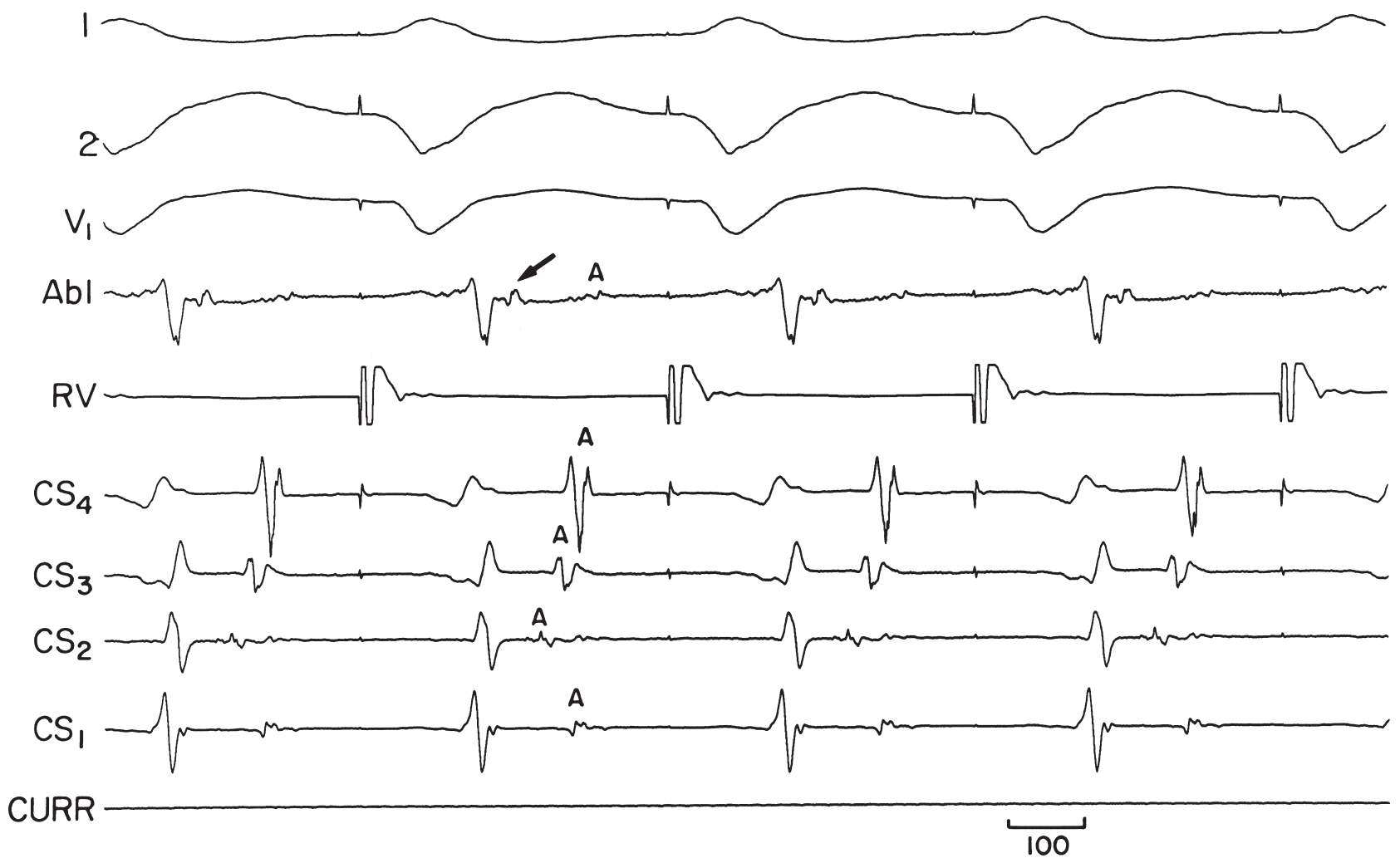
Figure 6–23B



Explanation:

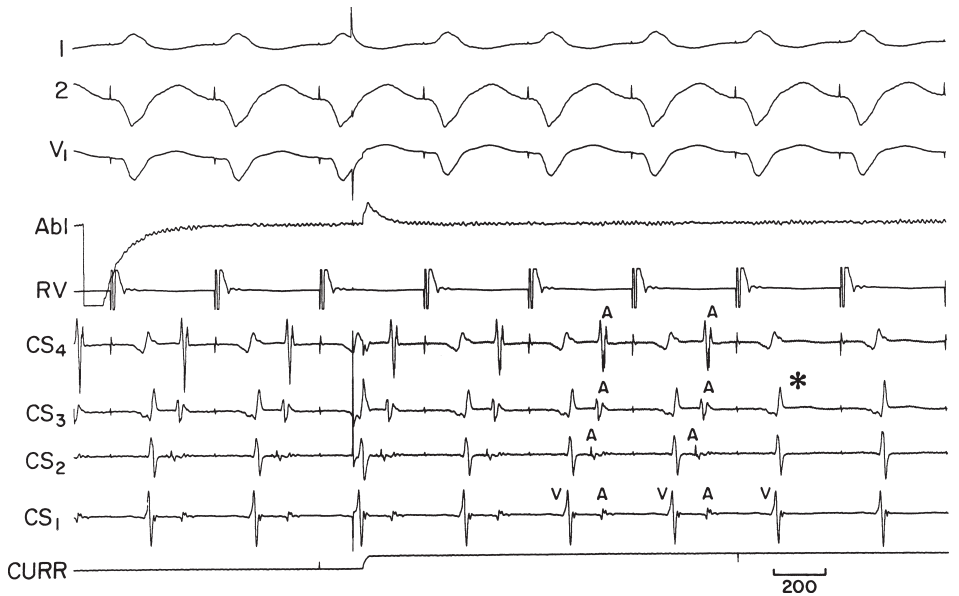
Ablation was targeted by using the distal CS electrode (ablation catheter not shown). A potential (*arrow*), assumed to be an AP potential, was recorded. The retrograde atrial activation sequence shifted abruptly with the second paced cycle after the onset of current. The VA conduction time is now considerably longer with a subtle shift in activation sequence and electrogram morphology. The third cycle blocked completely and the fourth cycle was probably a sinus escape cycle (high right atrial electrogram first). It is our view that the sequence of events is best explained by two very closely spaced pathways that are both ablated at one site in the temporal sequence observed. This is suggested by the slight change in atrial activation that would not be expected to change if the injury was merely prolonging atrial conduction time. The two closely spaced pathways could be entirely distinct or interconnected (branching).

Figure 6–24A



The record was taken from a patient undergoing ablation of a left lateral AP (subvalvular approach). Is this a reasonable ablation site?

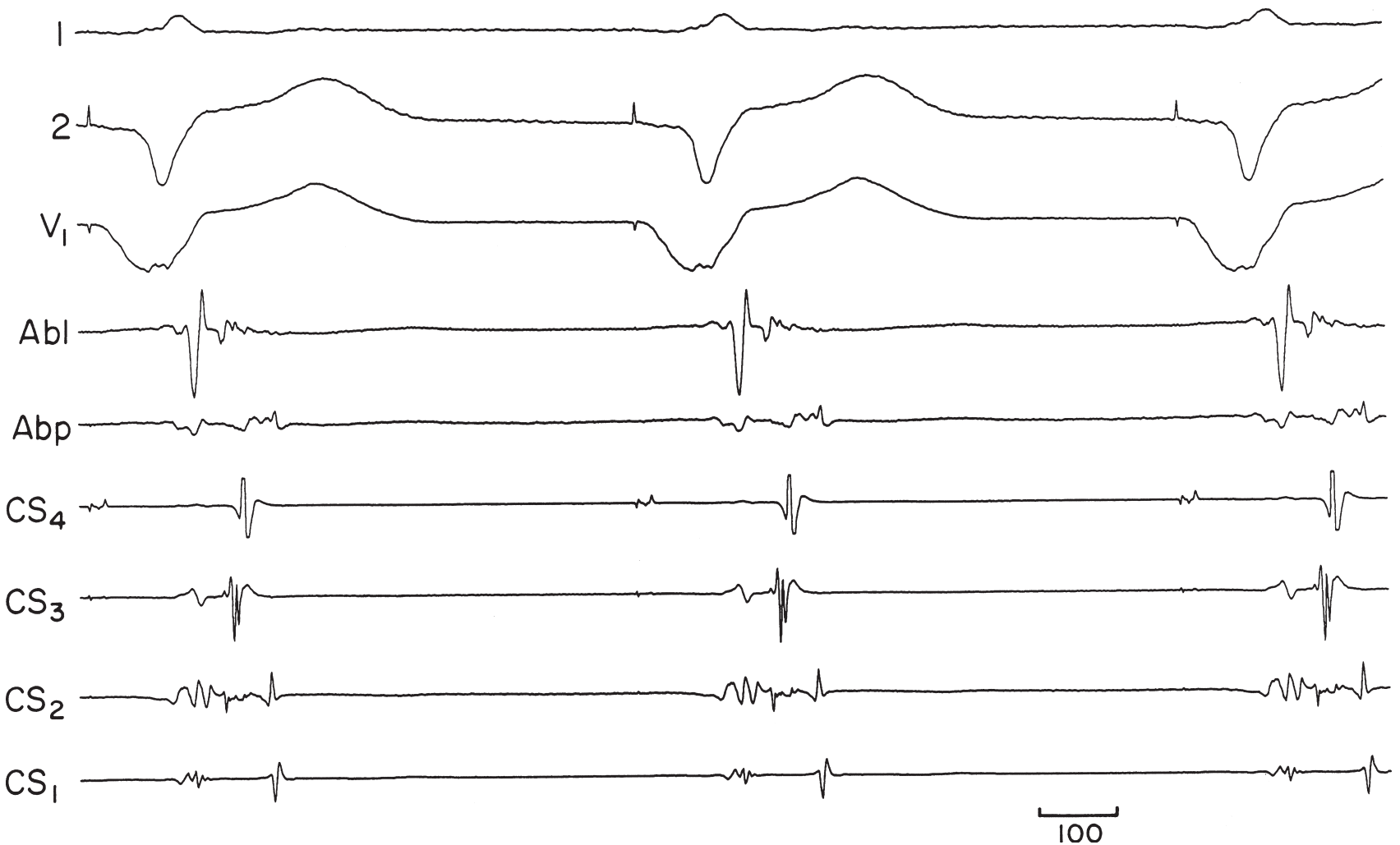
Figure 6–24B



Explanation:

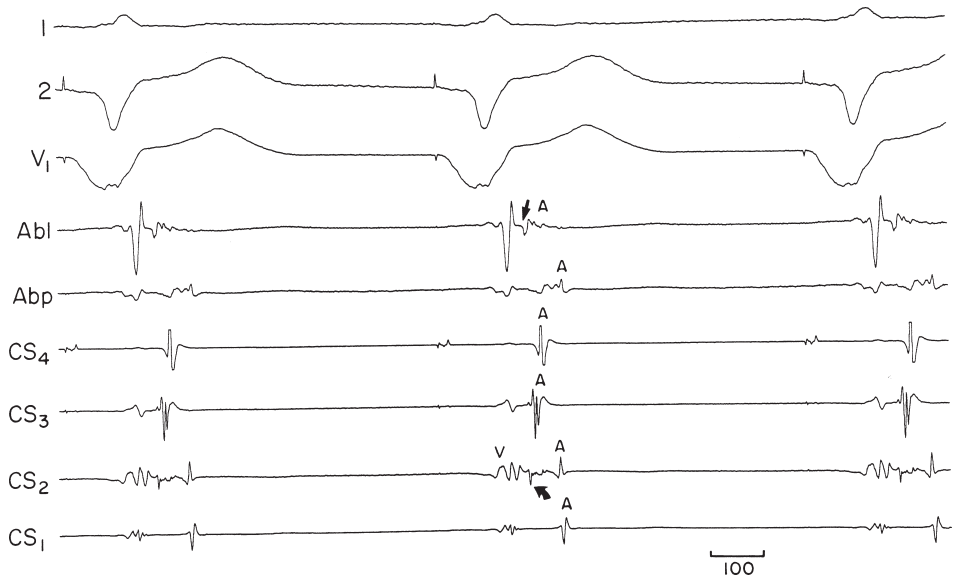
The ablation catheter in this instance was directed toward the DCS (CS_2) at the site of earliest atrial activation. At this site, an adequate atrial electrogram could not be recorded and the late deflection labeled as A at the ablation site was of low amplitude and arguably not even an atrial deflection. Nonetheless, a potential felt to be an AP potential (*arrow*) was observed though not validated. In this instance, the pathway blocked after the fourth cycle after onset of current (denoted by an asterisk). The general teaching is that ablation of an AP at the AV ring requires both adequate atrial and ventricular deflections to indicate that it is truly at the ring. Nonetheless, the recording of a potential will allow ablation of the pathway at its ventricular insertion site even in the absence of a believable atrial electrogram.

Figure 6–25A



This patient was undergoing ablation of the left lateral AP. What would be a better site for ablation, CS_3 or CS_2?

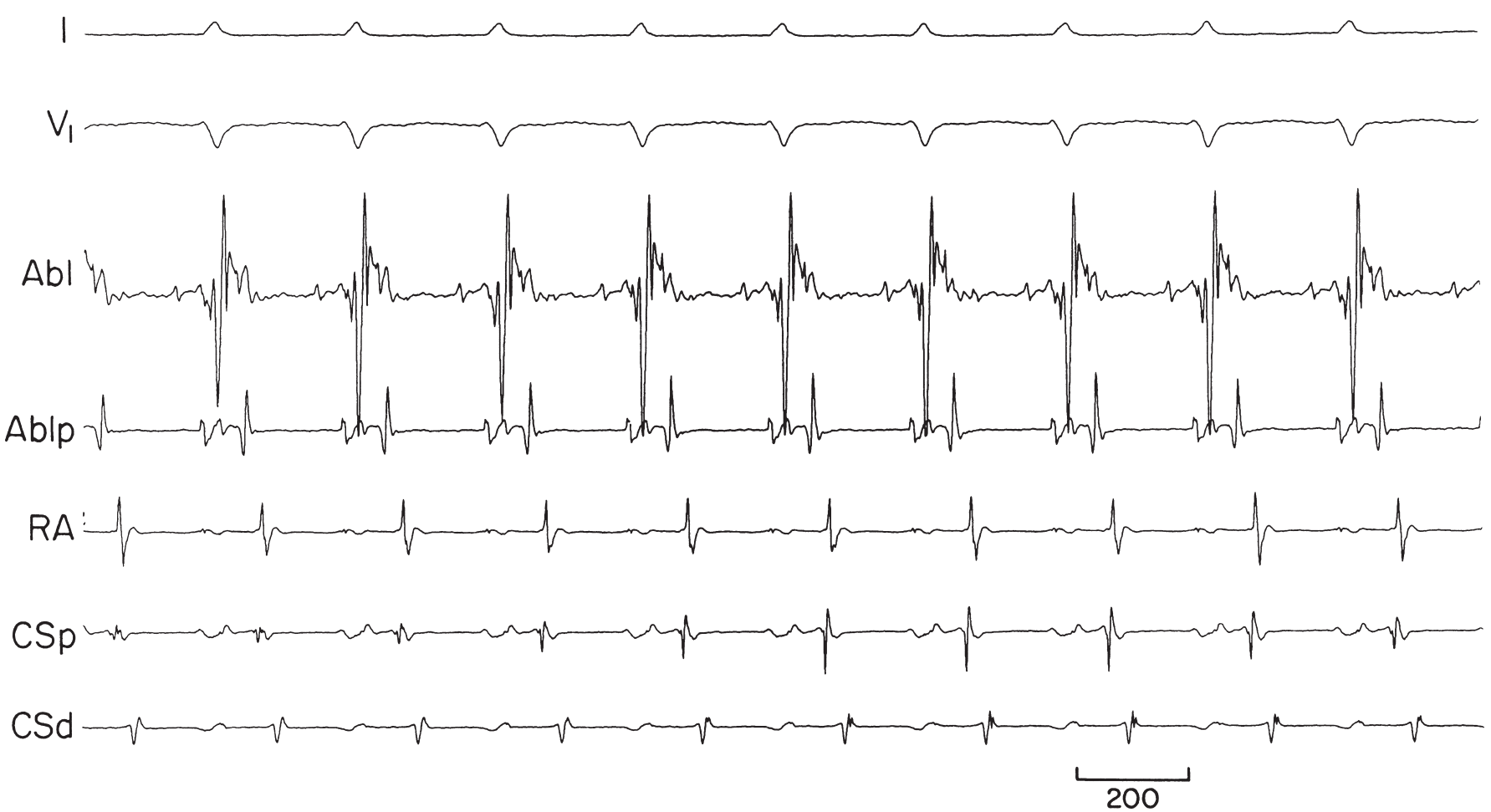
Figure 6–25B



Explanation:

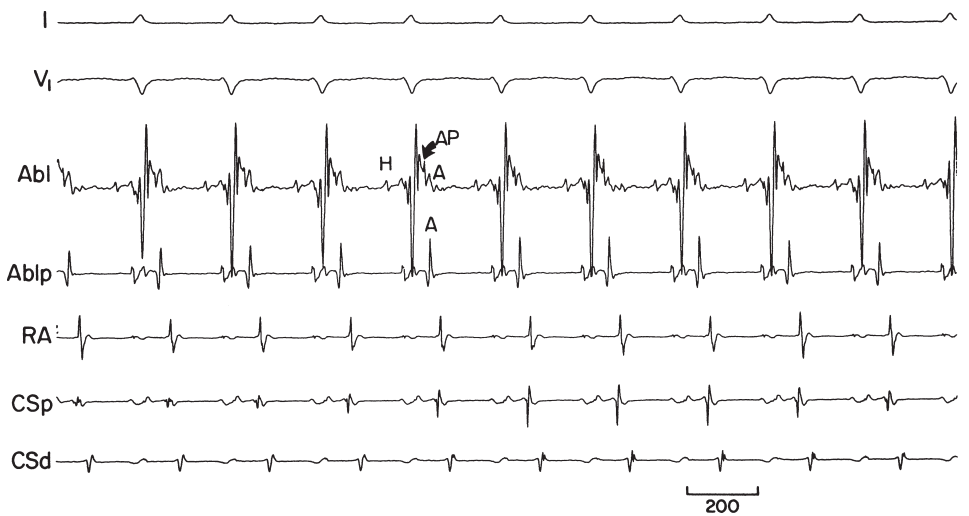
Earliest retrograde atrial activation during ventricular pacing was observed posterolaterally (CS_3) approximately 20 mm from the CS os. However, a distinct AP potential (*lower arrow*) was observed at CS_2, 10 mm more distal than CS_3. The A deflection at CS_2 is much later than the earliest A. This is compatible with the ventricular insertion site recorded more distally along the CS with a pathway slanting more medially to provide atrial activation earliest at CS_3. The ablation catheter was positioned subvalvularly in an attempt to approximate the electrode at CS_2, and a similar recording showing a putative pathway potential (*upper arrow*) was seen. Ablation at this site was successful, again illustrating the value of the pathway potential in guiding ablation.

Figure 6–26A



This patient had a right anteroseptal preexcitation pattern (same patient as Fig. 6–22) and supraventricular tachycardia using a right anteroseptal pathway as the retrograde pathway. Is this a suitable ablation site?

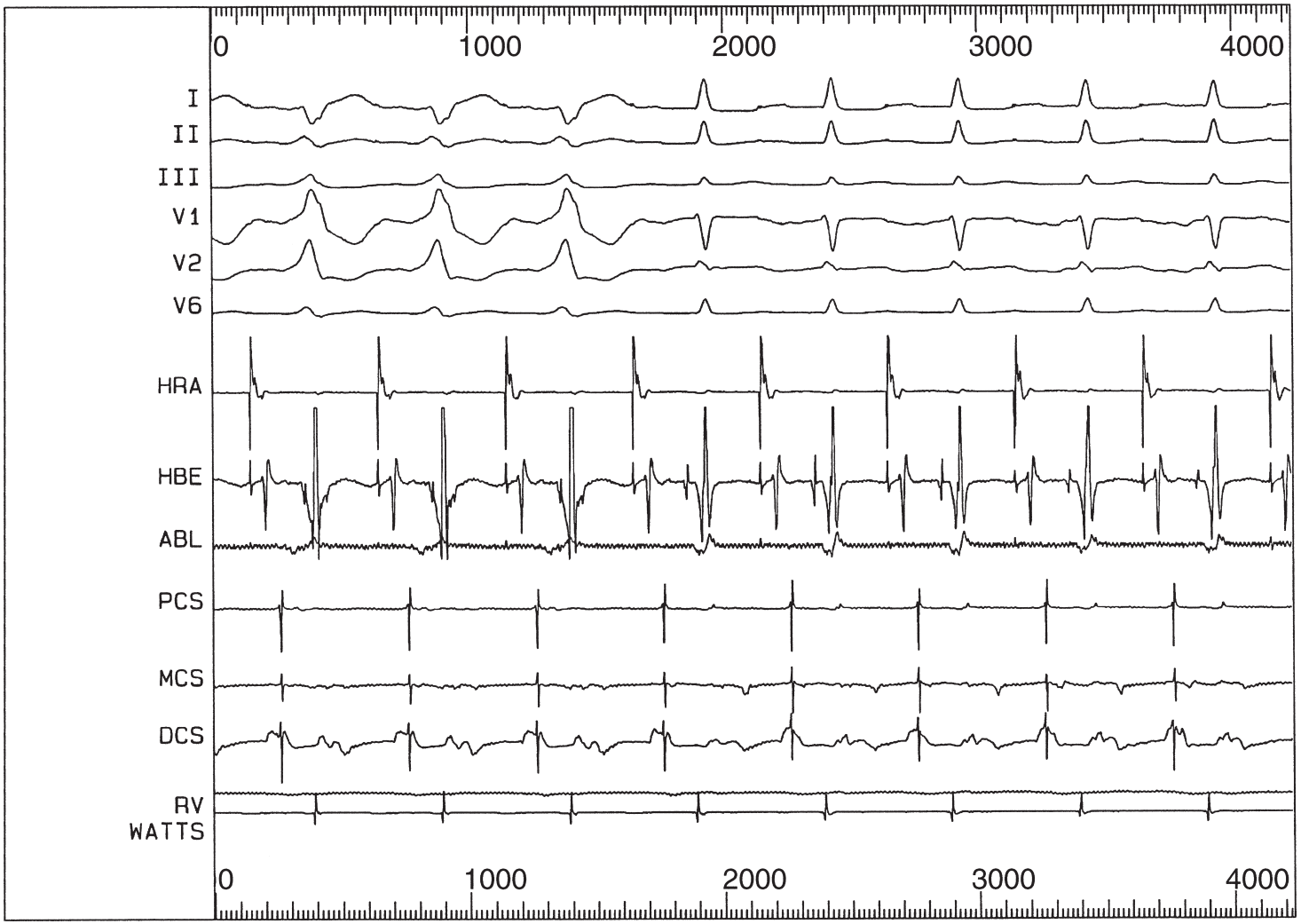
Figure 6–26B



Explanation:

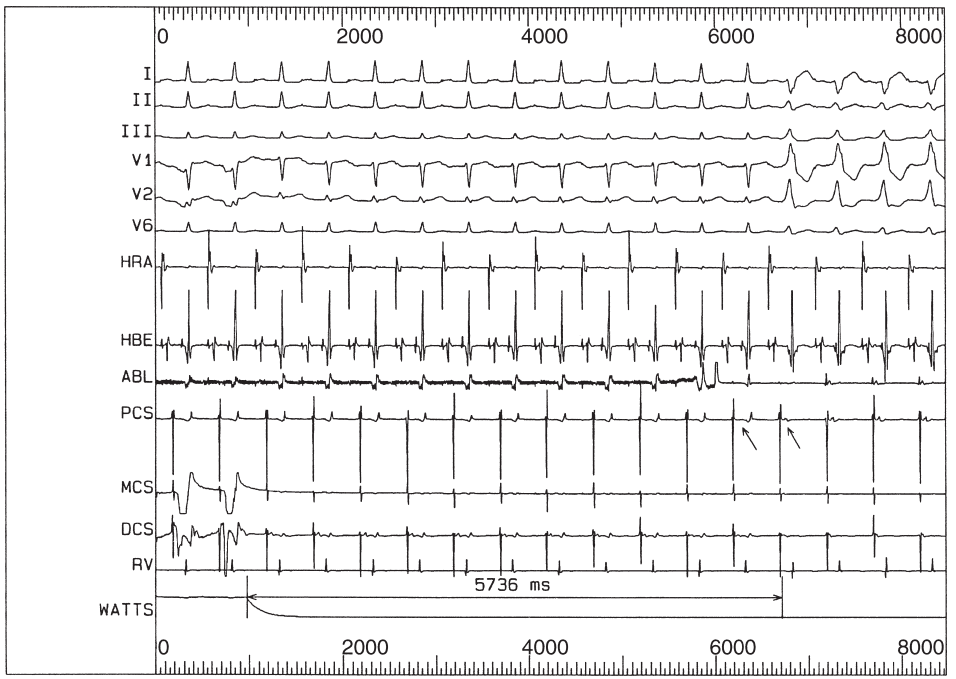
By definition, the right anteroseptal AP is located in proximity to the penetrating His bundle, and both a His spike and AP potential (*arrow*) can be recorded at the same site. Ideally, the His deflection should be lower in amplitude and less rapid (more far field), whereas the pathway potential should be maximized in amplitude and slope. This ideal ratio should be carefully sought. In this example, the labeled pathway potential has a larger amplitude and is "sharper" than the His potential. Furthermore, the atrial deflection is relatively small, supporting a more distal catheter position that would be less likely to result in AV block. Ablation during tachycardia in this instance will also allow one to monitor the AV node during ablation.

Figure 6–27A



This patient was undergoing ablation of a left lateral AP. Approximately 7 seconds after onset of radio-frequency energy the observation shown in Fig. 6–27*A* was made. Is this a successful ablation?

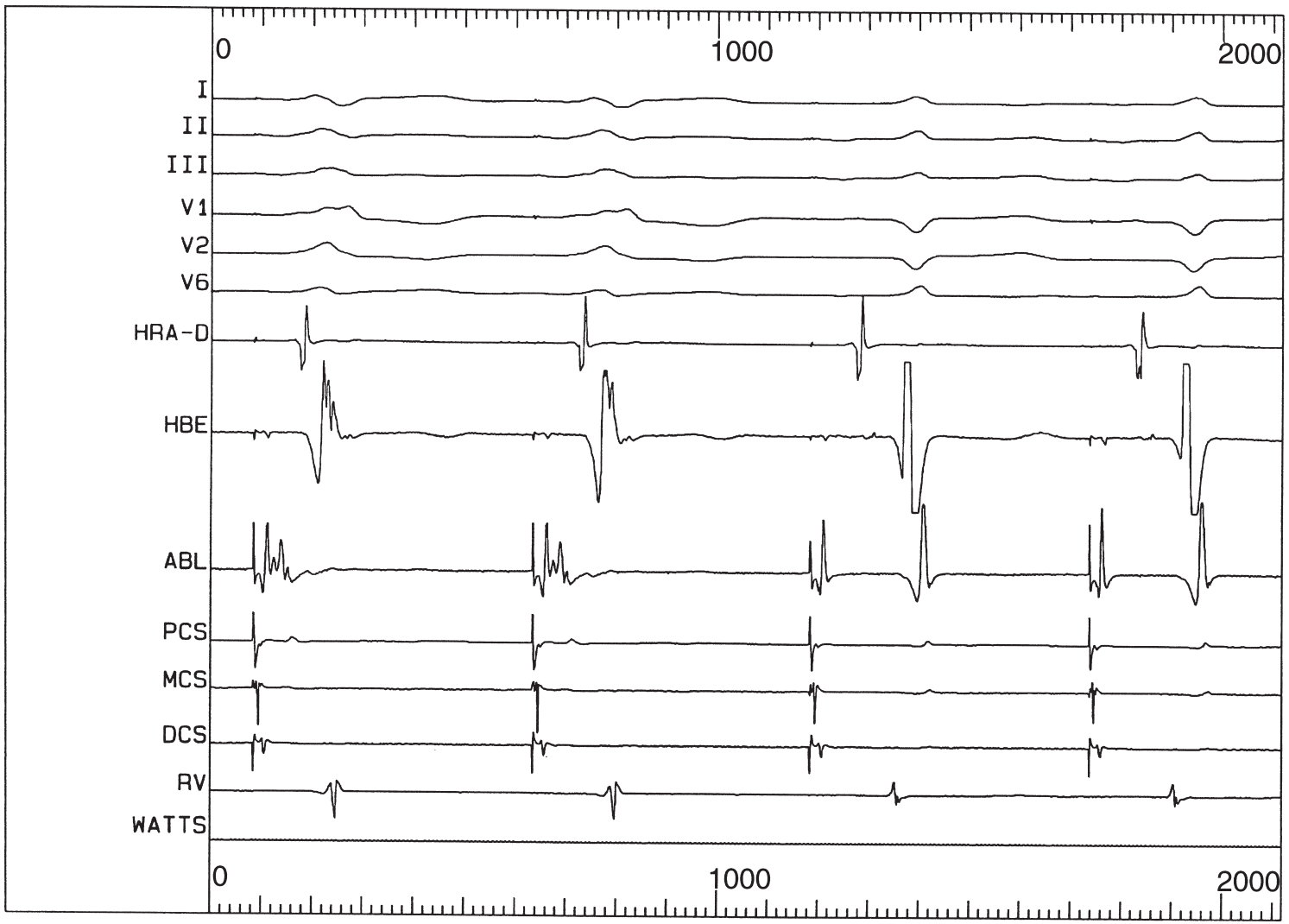
Figure 6–27B



Explanation:

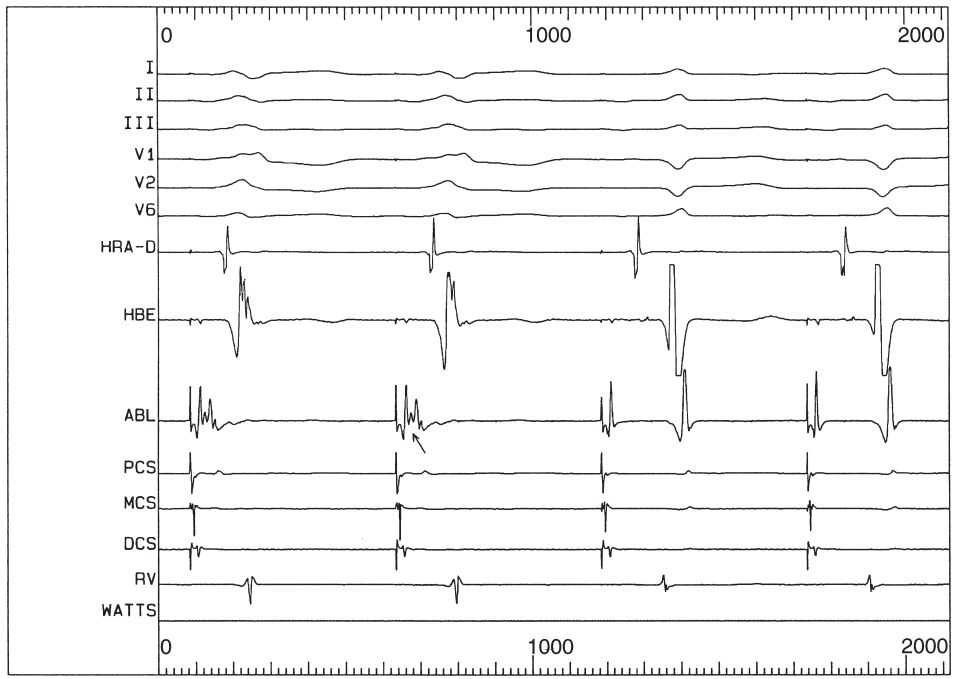
This figure demonstrates that AP conduction returned approximately 6 seconds after radio-frequency energy was discontinued. Simultaneous tracings were recorded from the high right atrium (HRA), His bundle area (HBE), ablation catheter (ABL), PCS, mid coronary sinus (MCS), and DCS electrodes, and the RV. The arrows point to the local AV interval on the proximal CS electrode. The last normal QRS complex has a widely spaced A and V electrogram, but when preexcitation returns the AV interval shortens due to local activation of the ventricle over the AP. Although the initial observation in Fig. 6–27*A* was encouraging, this merely represented the effects of increased heat to prevent AP conduction, and not destruction of the AP complex. The AP was successfully ablated with slight movement of the catheter to an adjacent area. When loss of AP conduction occurs during energy delivery, the catheter is close to the AP because the heat is transmitted only for a short distance from the catheter tip. Clearly, loss of AP conduction during energy delivery does not always represent permanent destruction of the pathway.

Figure 6–28A



Mapping was performed prior to energy delivery in this patient with a left free wall AP. Would you anticipate successful catheter ablation of the AP at this site?

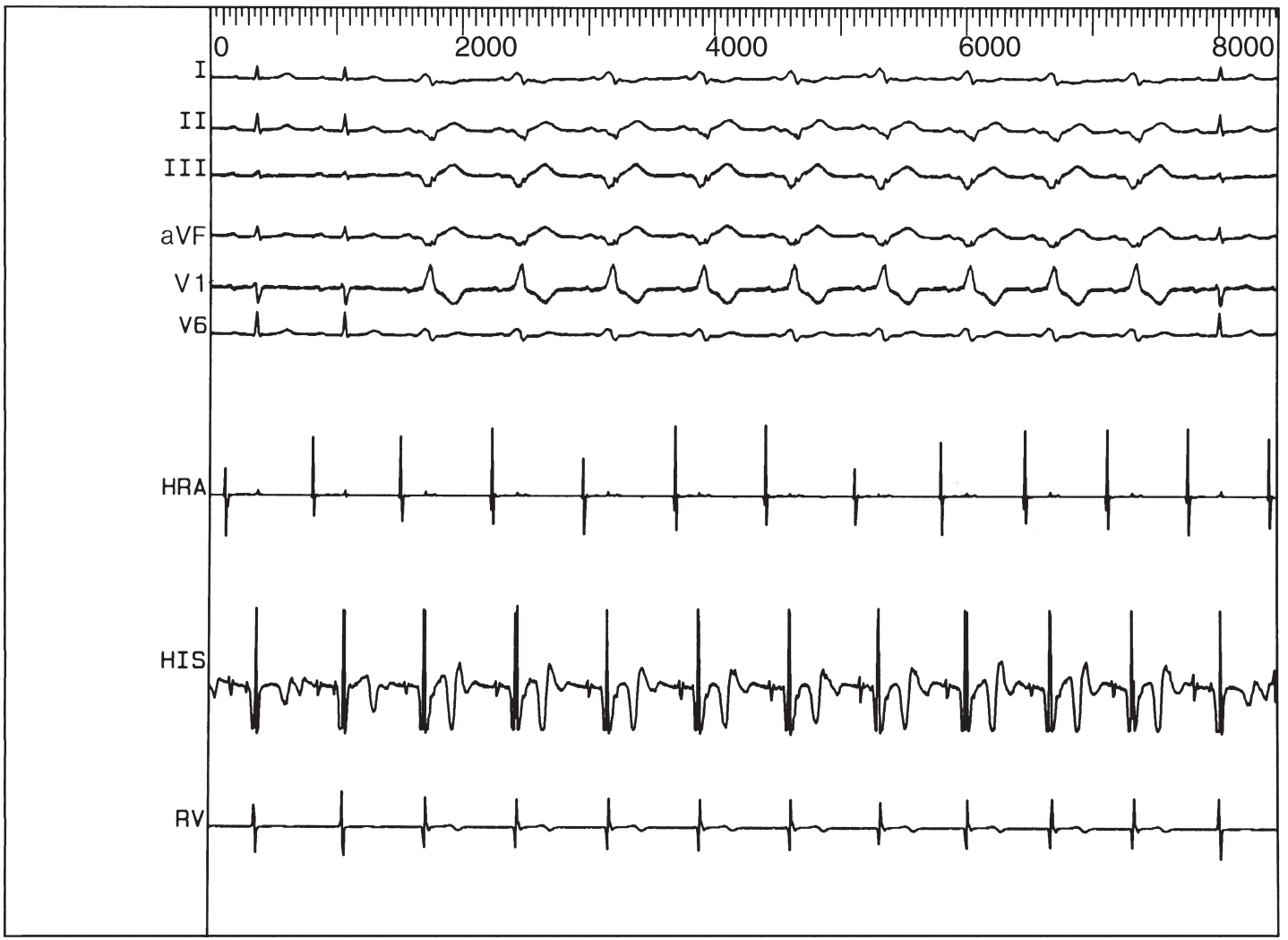
Figure 6–28B



Explanation:

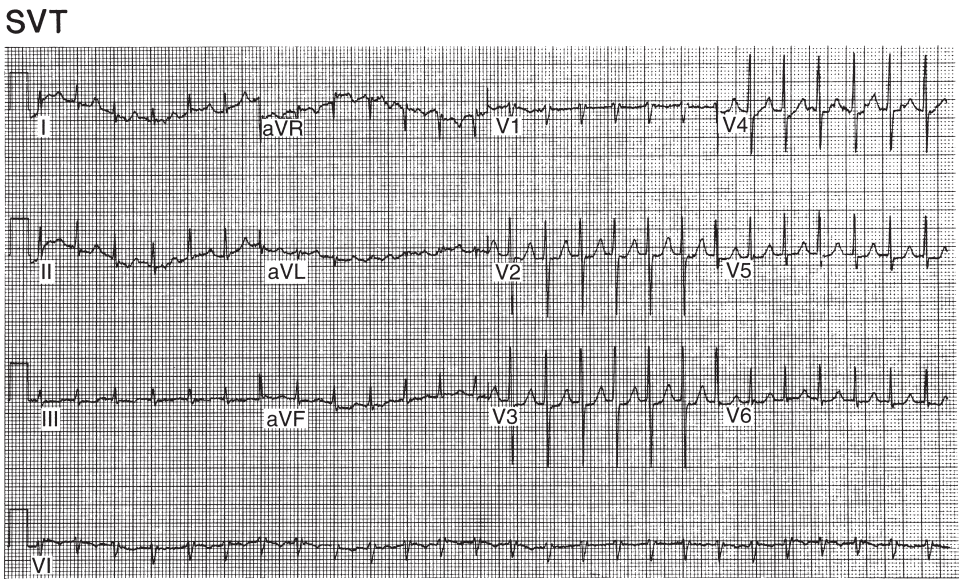
Energy delivery at this site has an extremely high chance to ablate successfully AP conduction. Note the arrow pointing to a triphasic local electrogram at the ablation site. The atrial electrogram is large, and the arrow points to a probable AP potential between the atrial and ventricular local electrograms. More important is the effect of local trauma ("bump map") to the AP, as demonstrated in the last two complexes. Loss of preexcitation occurs due to catheter pressure and the local AV interval markedly separates without the AP potential visible any longer. Ablation at this site, which was performed quickly after this observation occurred, produced permanent block over the AP. Although demonstration of an AP potential at the site of ablation is usually predictive for subsequent successful destruction of the AP with energy delivery, local trauma to the AP with catheter pressure is an even better predictor of success.

Figure 6–29A



This patient has a history of documented paroxysmal SVT. No prior electrophysiologic study has been performed. Would a single catheter approach to ablate the AP likely cure this patient?

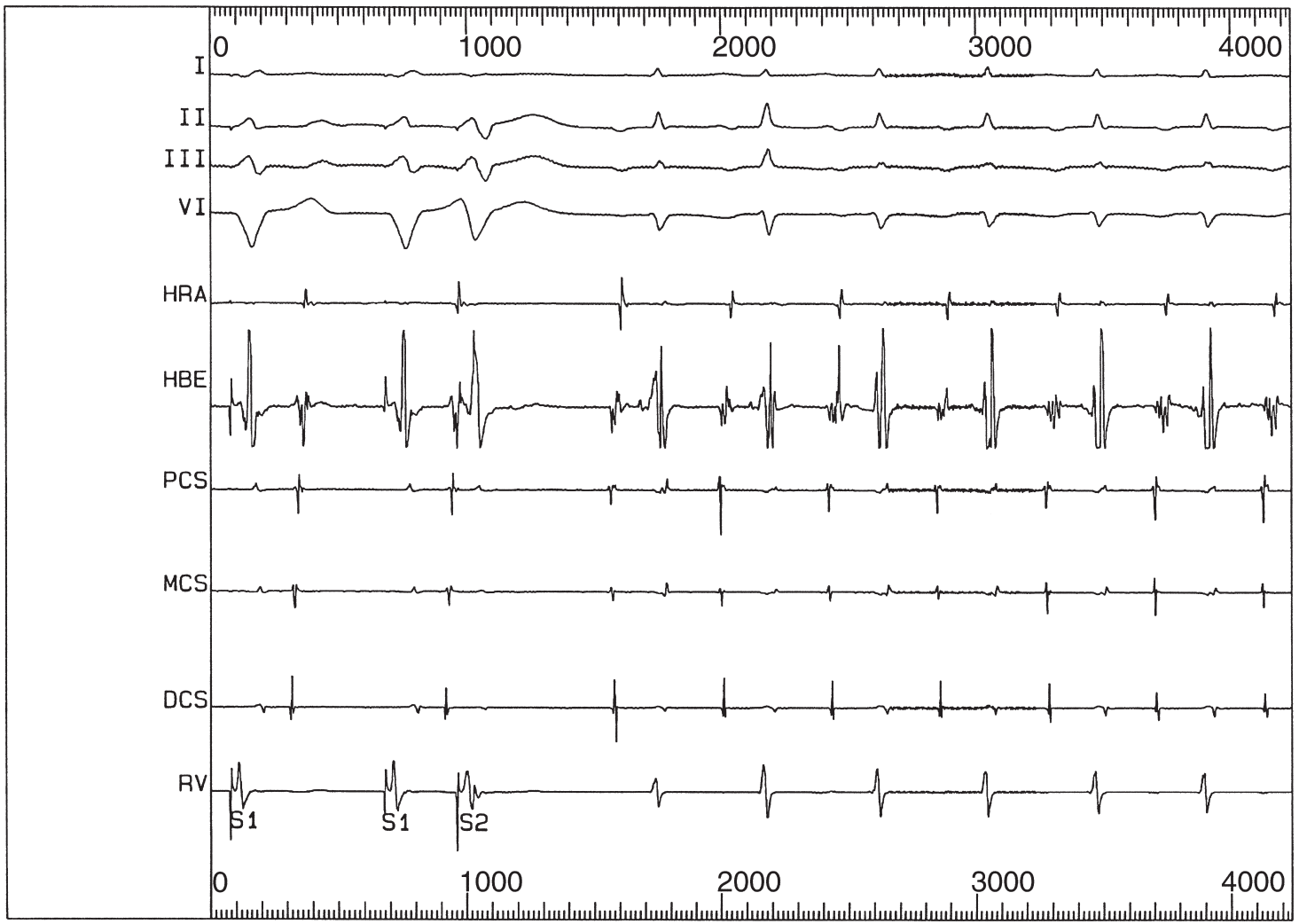
Figure 6–29B



Explanation:

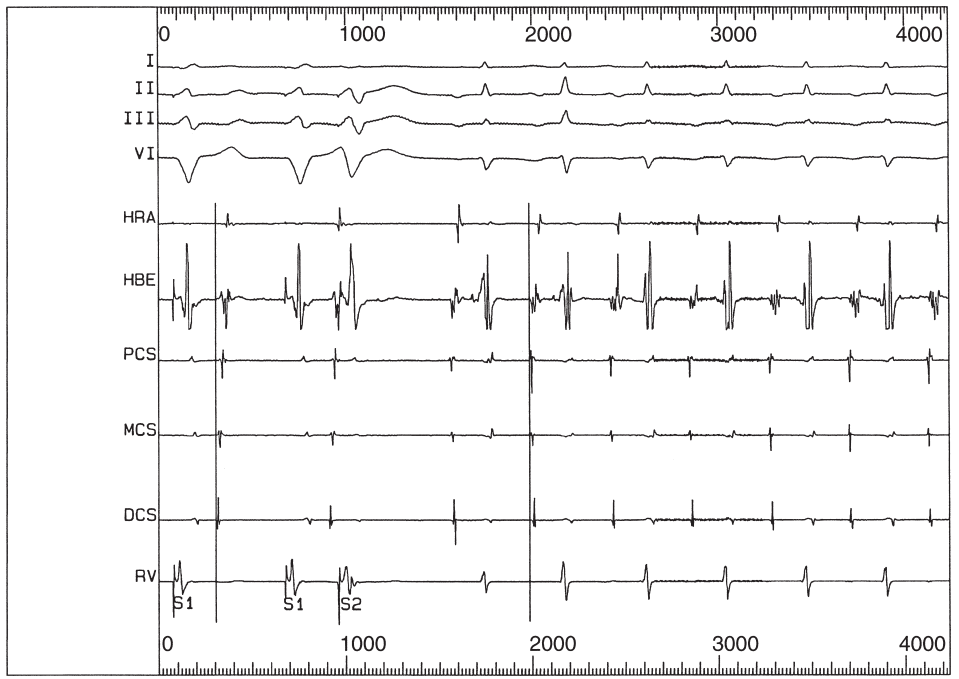
This patient had an AP with intermittent anterograde conduction, but no retrograde conduction. His clinical arrhythmia was AV node reentry as seen here in the 12-lead electrocardiogram. Note the typical small *r´* in VI during tachycardia. Use of a single catheter ablation approach to destroy the AP would have resulted in a cosmetic alteration of the electrocardiogram without cure of this patient's tachycardia. In our opinion, it is important to document the cause of tachycardia and formulate an ablation plan based on physiologic data. Playing the odds will usually work, but we have seen a variety of arrhythmias, including VT, occur in patients with ventricular preexcitation in whom the AP does not participate in tachycardia.

Figure 6–30A



This tracing was recorded during an EP study prior to attempts at radio-frequency catheter ablation. Will ablation at a single site cure this patient?

Figure 6–30B

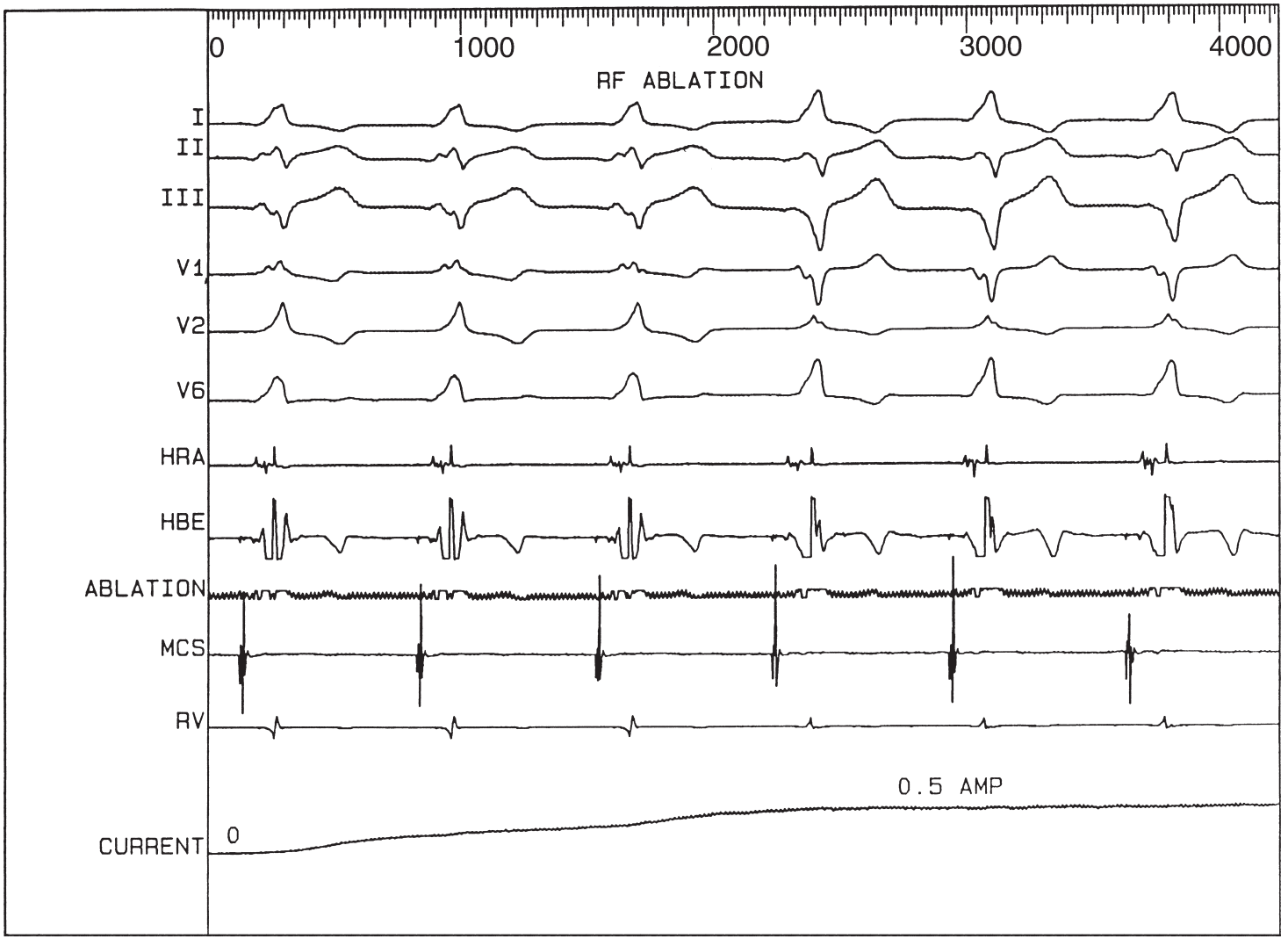


Explanation:

The arrhythmia induced in this tracing is a long RP tachycardia with initial atrial activation occurring near the CS os in the posteroseptal area of the right atrium. The differential diagnosis is fast/slow AV node reentry, AV reentry utilizing a slowly conducting retrograde pathway, and atrial tachycardia. Subsequent EP pacing maneuvers diagnosed AV node reentry. However, careful inspection of the initial S1 paced beat reveals a different retrograde atrial activation sequence.

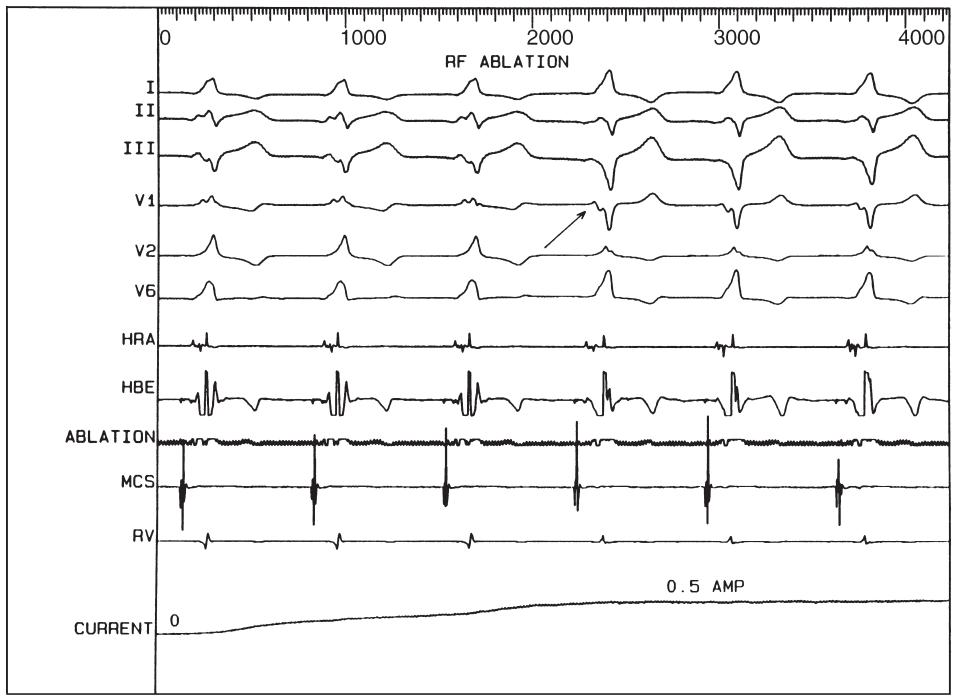
This patient also had a concealed left lateral AP that could be used in AV reentry. The CS catheter was initially positioned with the proximal electrode close to the CS os, and when the catheter was advanced more laterally the eccentric atrial activation sequence became more obvious. Thus, a single catheter ablation site did not cure this patient.

Figure 6–31A



This patient was undergoing ablation of a left posterior AP. Should current application be continued?

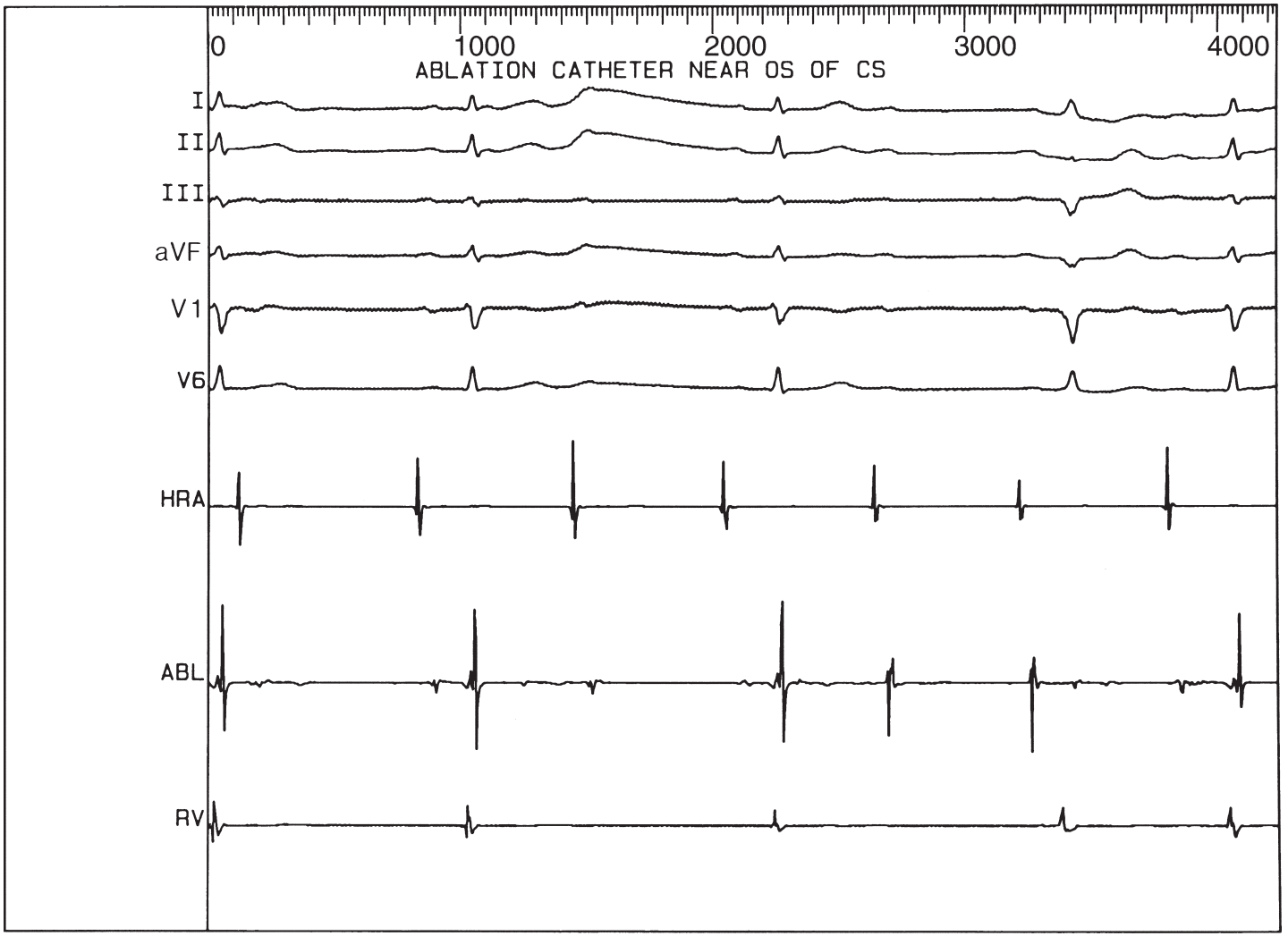
Figure 6–31B



Explanation:

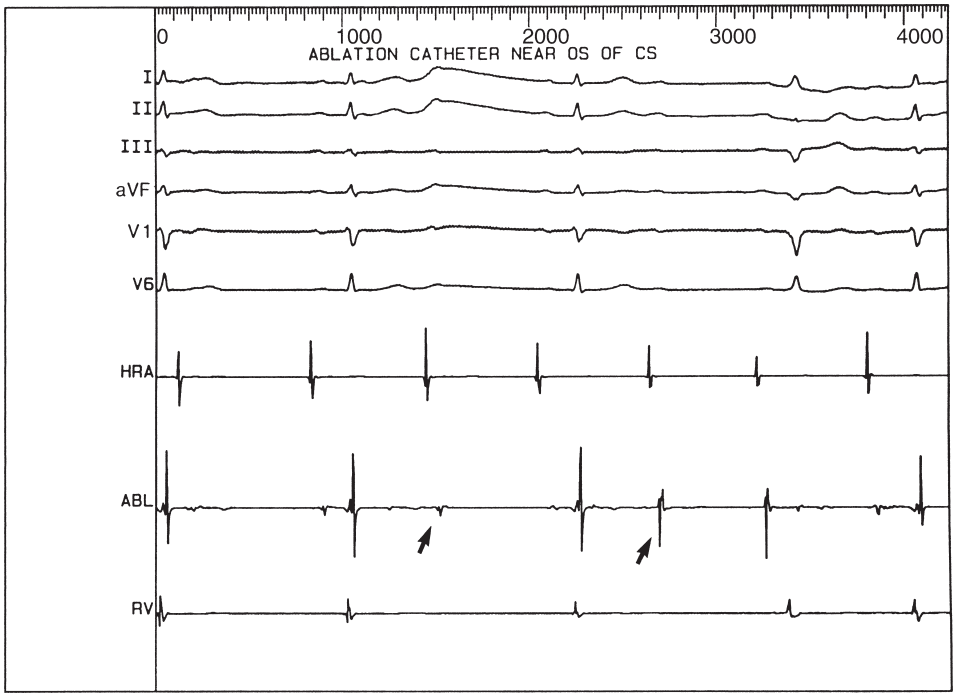
An excellent site was selected and radio-frequency energy was initiated. Note that the fourth QRS complex has a change in morphology (*arrow*). Although preexcitation is still present, the preexcited QRS pattern has changed. The first three complexes demonstrate an upright delta wave in V1 that becomes negative in the fourth and subsequent complexes. This patient did have successful ablation of the left posterior AP, but a second AP became apparent and it was located in the right posteroseptal area. Another ablation attempt in the right posteroseptal area produced block over the second AP. Even then this patient was not cured. Tachycardia could be reinduced and complete mapping revealed a left lateral AP that had not been evident prior to ablation of the first two APs. A third ablation had to be performed, after which the patient was cured permanently.

Figure 6–32A



This patient had typical slow/fast AV node reentry. The ablation catheter was positioned in the area of the CS os. Is this a good ablation site to initiate radio-frequency energy?

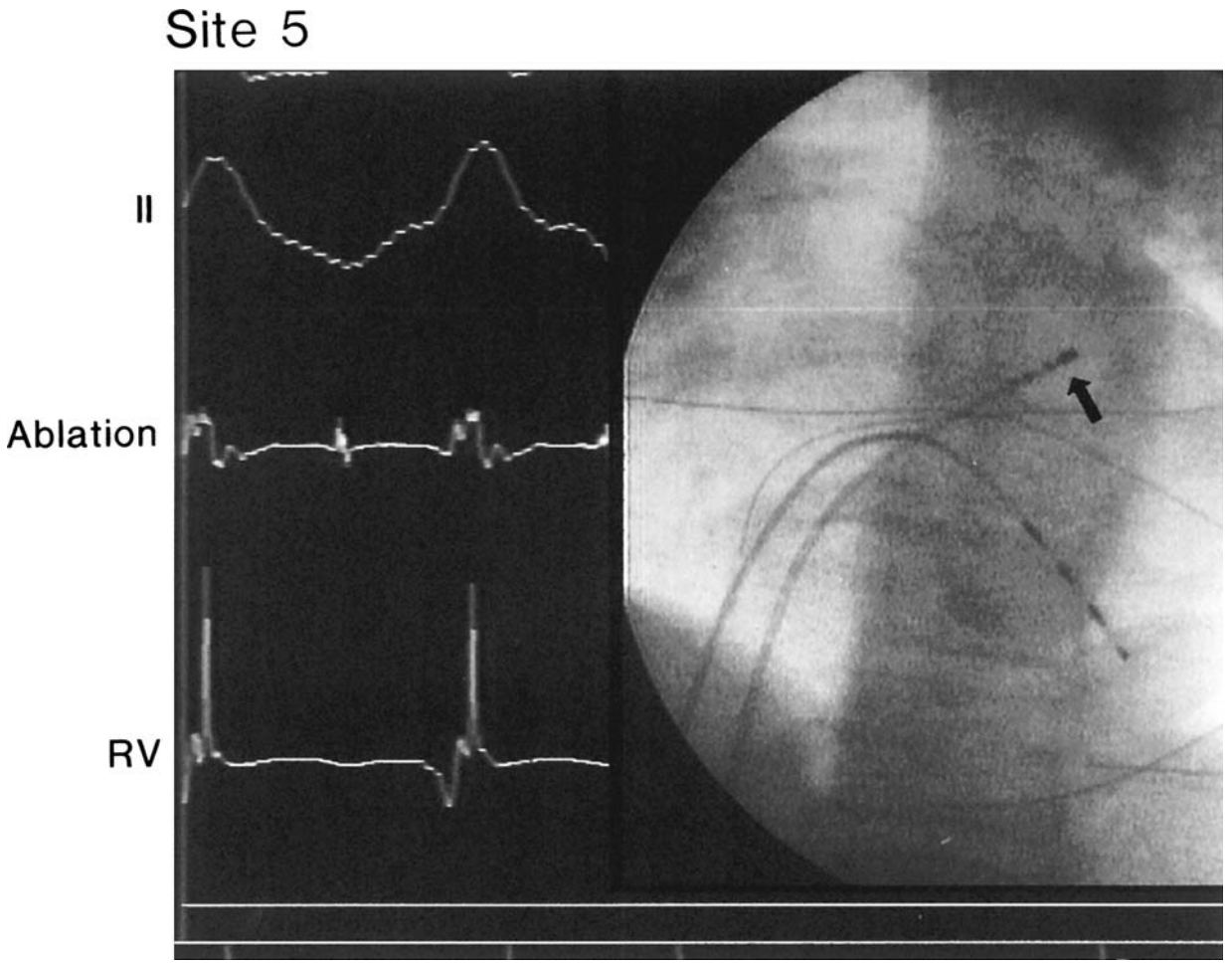
Figure 6–32B



Explanation:

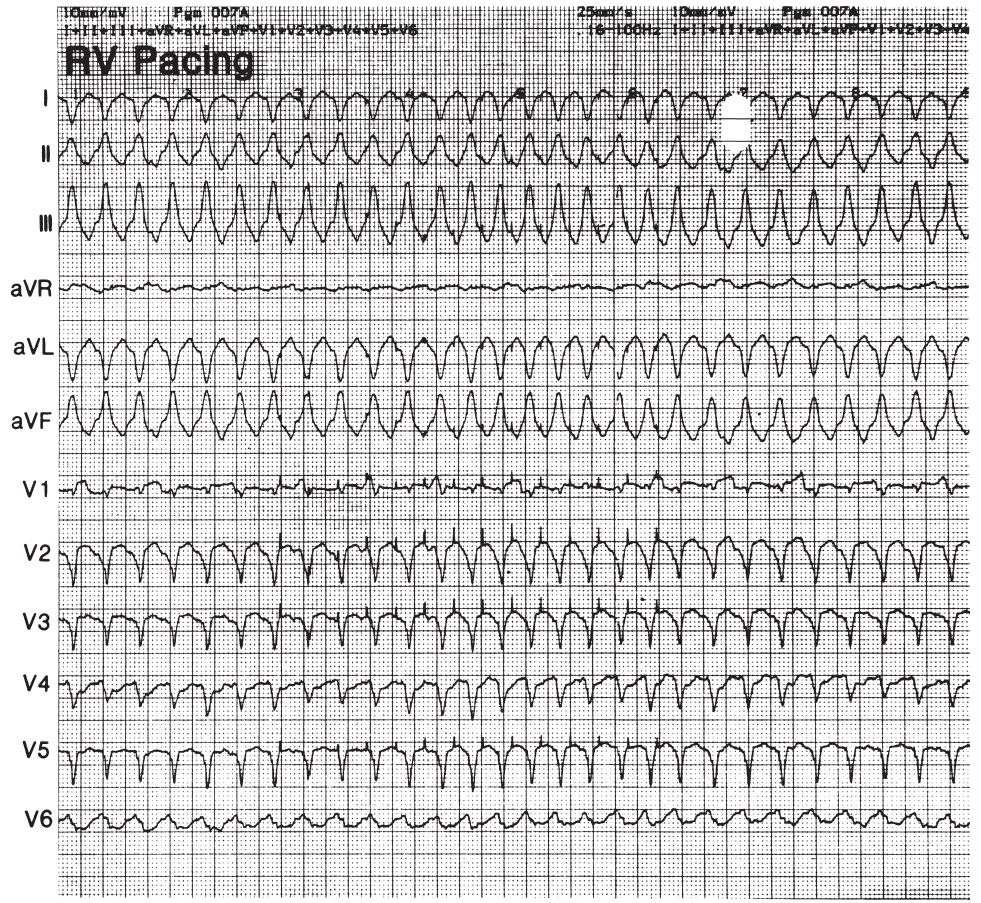
It would not be wise to introduce energy at this catheter ablation site. Note that intermittent heart block occurs (*arrows*). This suggests that catheter trauma of AV nodal conduction is occurring, and another ablation site should be chosen to avoid permanent heart block with introduction of energy. There can be considerable variation in the position of the AV node in relation to the CS os. Placement of the ablation catheter near the os of the coronary sinus does not imply that one is always a "safe" distance from the AV node. When doubt exists, one can move the catheter more posteriorly. Whenever there is concern about the anatomical relationship between the ablation catheter position and AV node, energy should be introduced at relatively low levels and progressively titrated upward.

Figure 6–33A



This patient had cardiomyopathy and a ventricular tachycardia occurring in the right ventricular outflow tract area. The radiograph on the right side of this figure shows the position of the ablation catheter (*arrow*) in the right ventricular outflow tract. The electrogram from the distal electrode pair is demonstrated on the left side of this figure. Is this a good site to introduce radio-frequency energy?

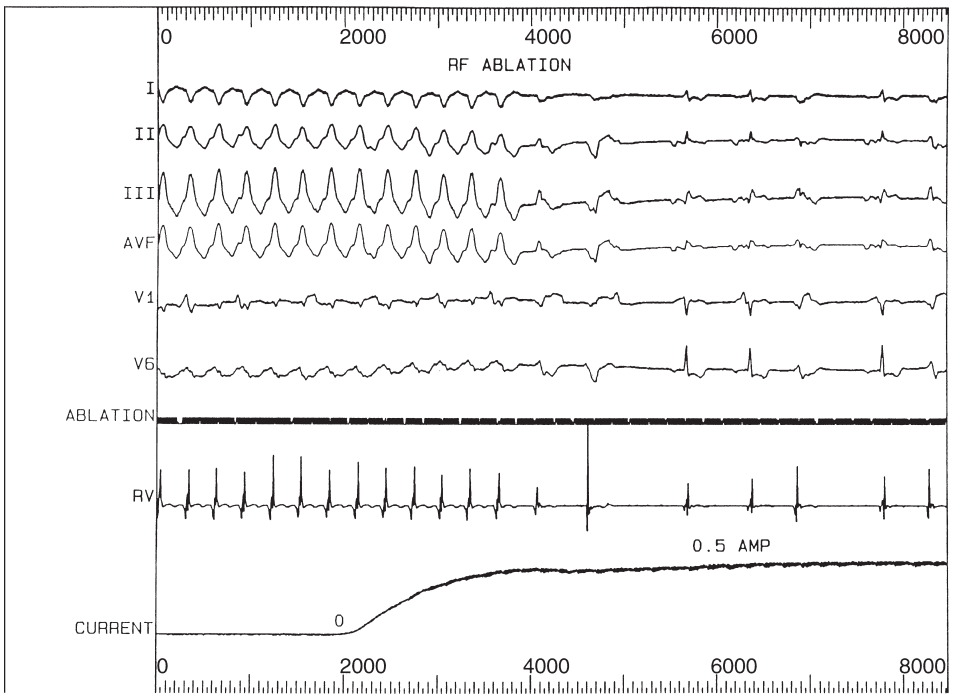
Figure 6–33B



Explanation:

The electrogram noted in Fig. 6–33*A* reveals a middiastolic potential during ventricular tachycardia. This is usually an excellent site for ablation in patients with structural heart disease. In patients with right ventricular outflow tract tachycardias and no structural heart disease, middiastolic potentials are not typically recorded. Figure 6–33*B* demonstrates paced mapping from this site. Note the excellent concordant 12-lead electrocardiogram during VT and local RV pacing. Introduction of radio-frequency energy is shown in Fig. 6–33*C*, and tachycardia is terminated even before full power is reached.

Figure 6–33C


0
2000
4000
6000
8000
RF ABLATION
I
II
III
AVF
V1
V6
ABLATION
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CURRENT
0
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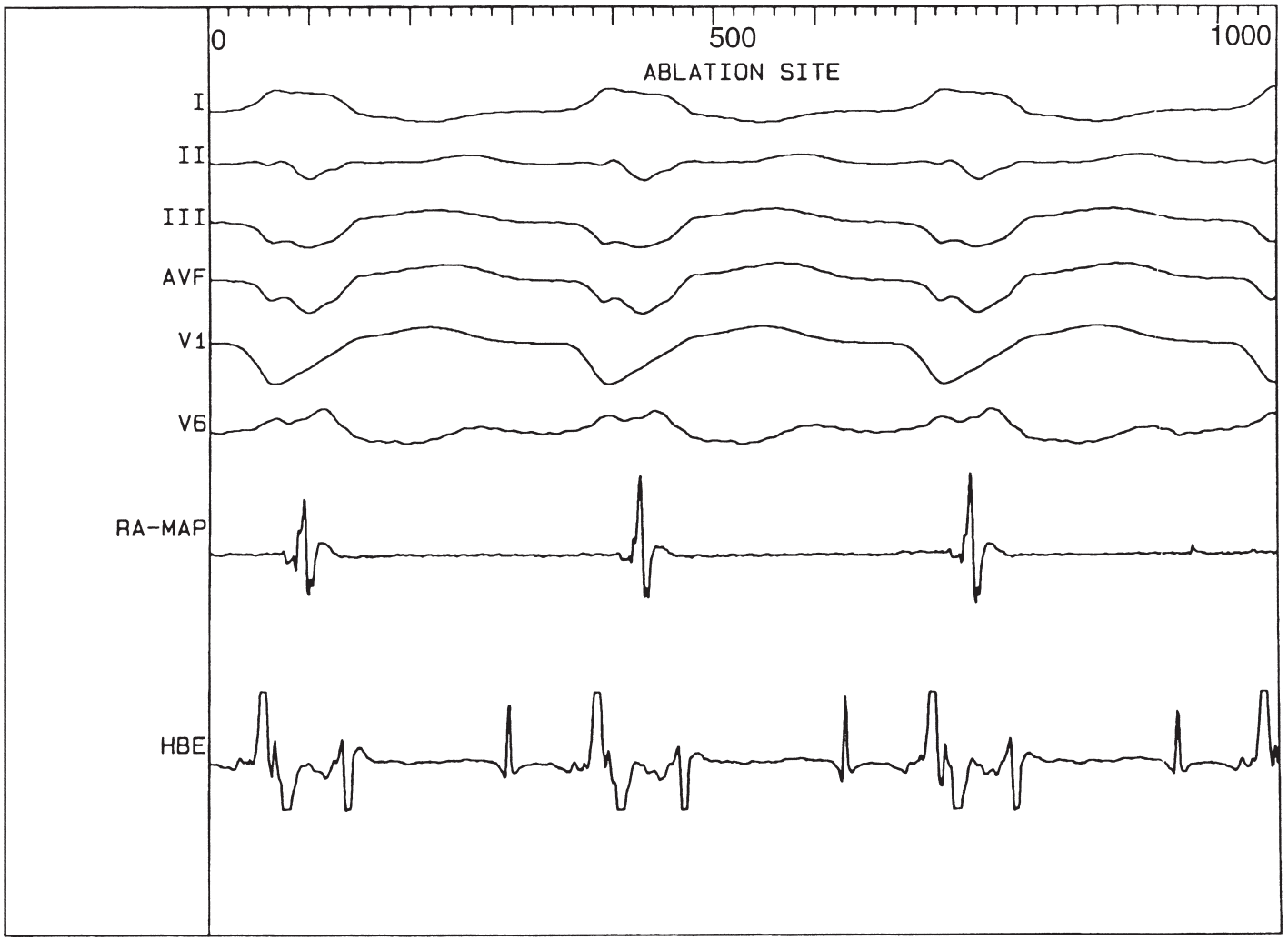
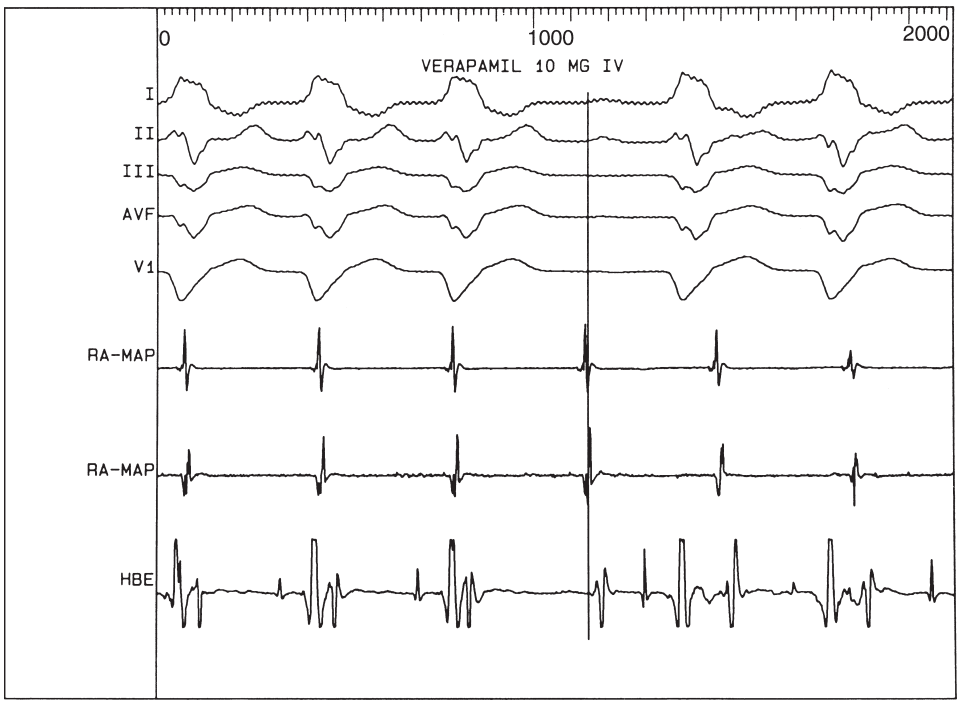
Figure 6–34A



This patient had atrial tachycardia and was undergoing mapping prior to radio-frequency ablation. Left bundle branch block aberrancy is present. Is this a good ablation site?

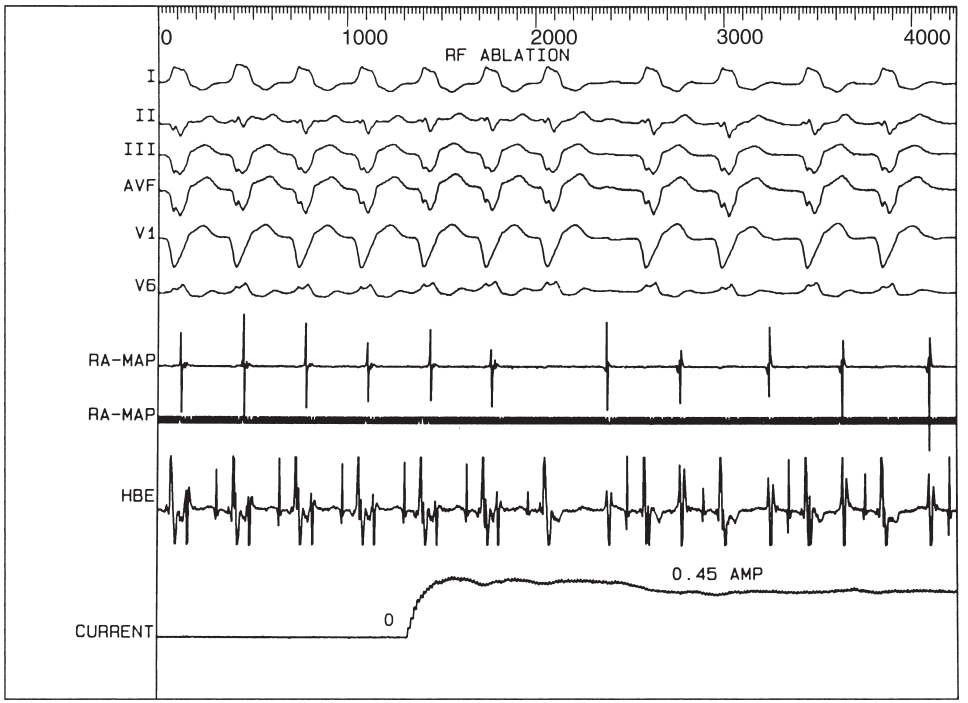
Figure 6–34B



Explanation:

It is very difficult to know if the recorded atrial electrogram at the ablation site in Fig. 6–34*A* is early in relation to the surface P wave. Thus, one would try to identify the P wave prior to onset of energy delivery. There are several methods to do this. In this case verapamil was given as demonstrated in Fig. 6–34*B*. Atrial tachycardia continues undisturbed during AV nodal block. With block, there is a clearly defined P wave and the local atrial electrogram clearly precedes the P wave by a significant interval. An alternative method is introduction of one or more premature ventricular complexes during tachycardia to produce retrograde concealed conduction in the AV node. In this instance the P wave may become visible for one complex, and the relationship of the local atrial electrogram to the P wave can be ascertained. This patient had successful ablation of the atrial tachycardia as shown in Fig. 6–34*C*.

Figure 6–34C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RF ABLATION
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AVF
V1
V6
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RA-MAP
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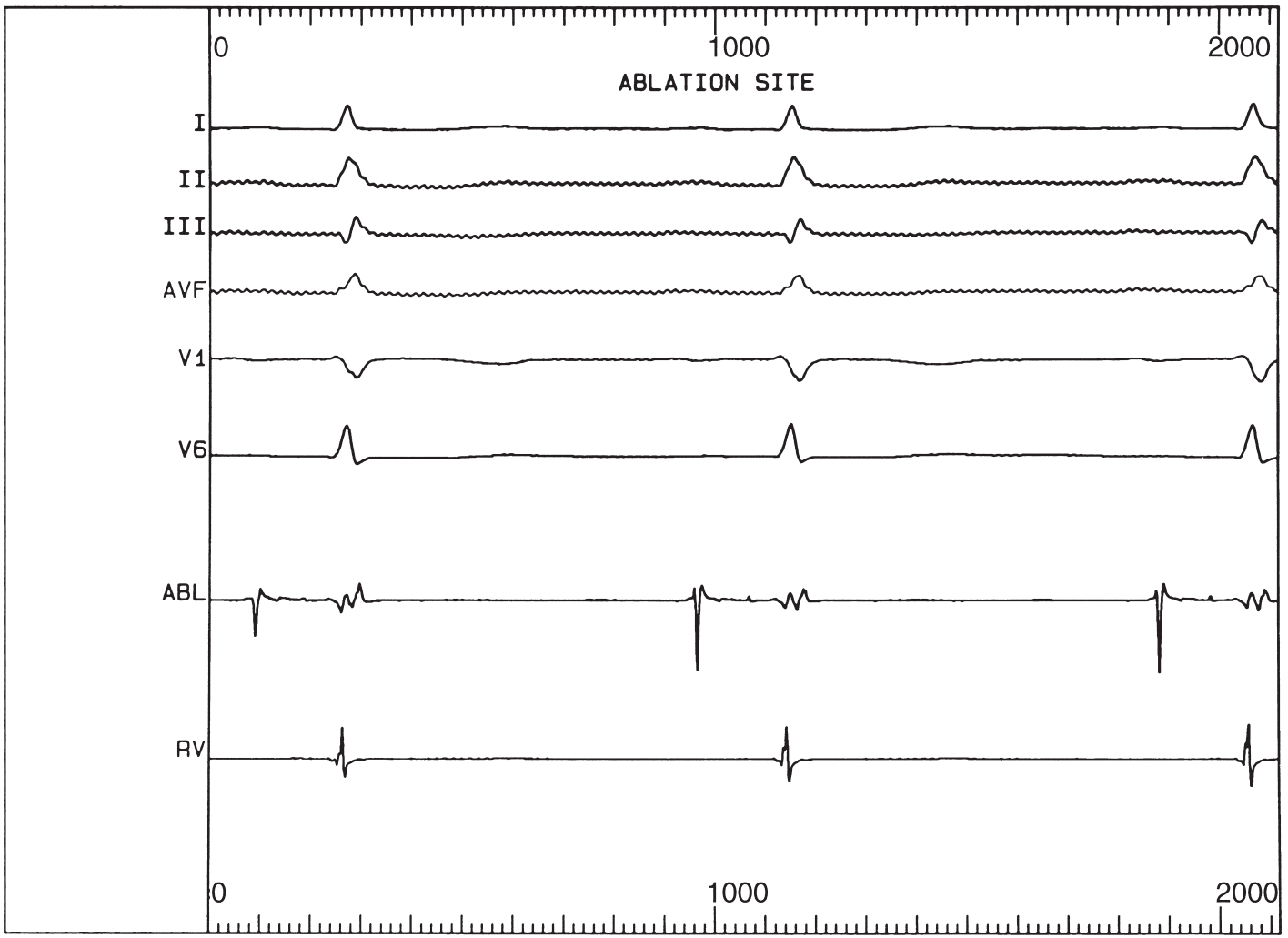
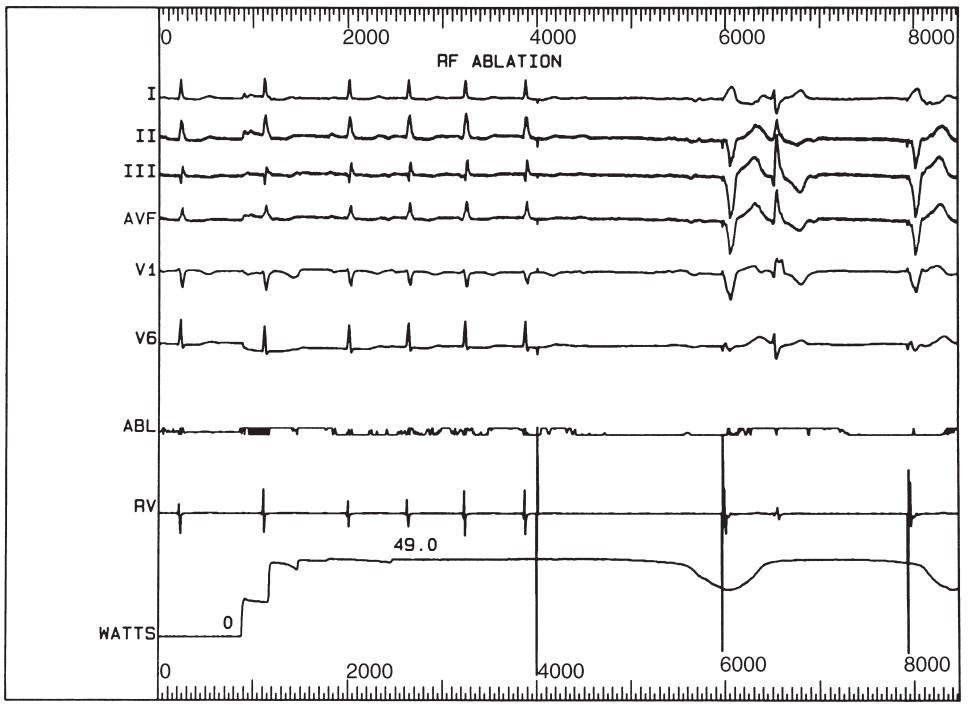
Figure 6–35A



This patient had recurrent atrial fibrillation uncontrolled with multiple antiarrhythmic agents. Radio-frequency ablation of the AV junction to create complete heart block was performed. Is this a good ablation site?

Figure 6–35B



Explanation:

Onset of radio-frequency energy at this site produced almost instantaneous rapid junctional tachycardia and complete heart block shortly thereafter. After discontinuation of energy, an electrocardiogram was recorded and is shown in Fig. 6–35*C*. Note that this patient has a narrow QRS escape rhythm that was stable. The ablation site in Fig. 6–35*A* had excellent local electrogram characteristics for AV junctional ablation. Note the large A wave with a smaller His bundle deflection. This suggests a more proximal site and after ablation the patient typically has a reasonably stable junctional escape rhythm.

A permanent pacemaker is necessary, but it is somewhat reassuring to have a backup junctional escape rhythm. Many use the term *His bundle ablation*. The His bundle is encased in the central fibrous body and is very difficult to destroy with usual radio-frequency energy catheter systems. It is much more likely that the AV node is damaged in most patients. However, the more distal the ablation site, the less likely a stable narrow QRS junctional rhythm will be present after ablation.

After AVJ Ablation

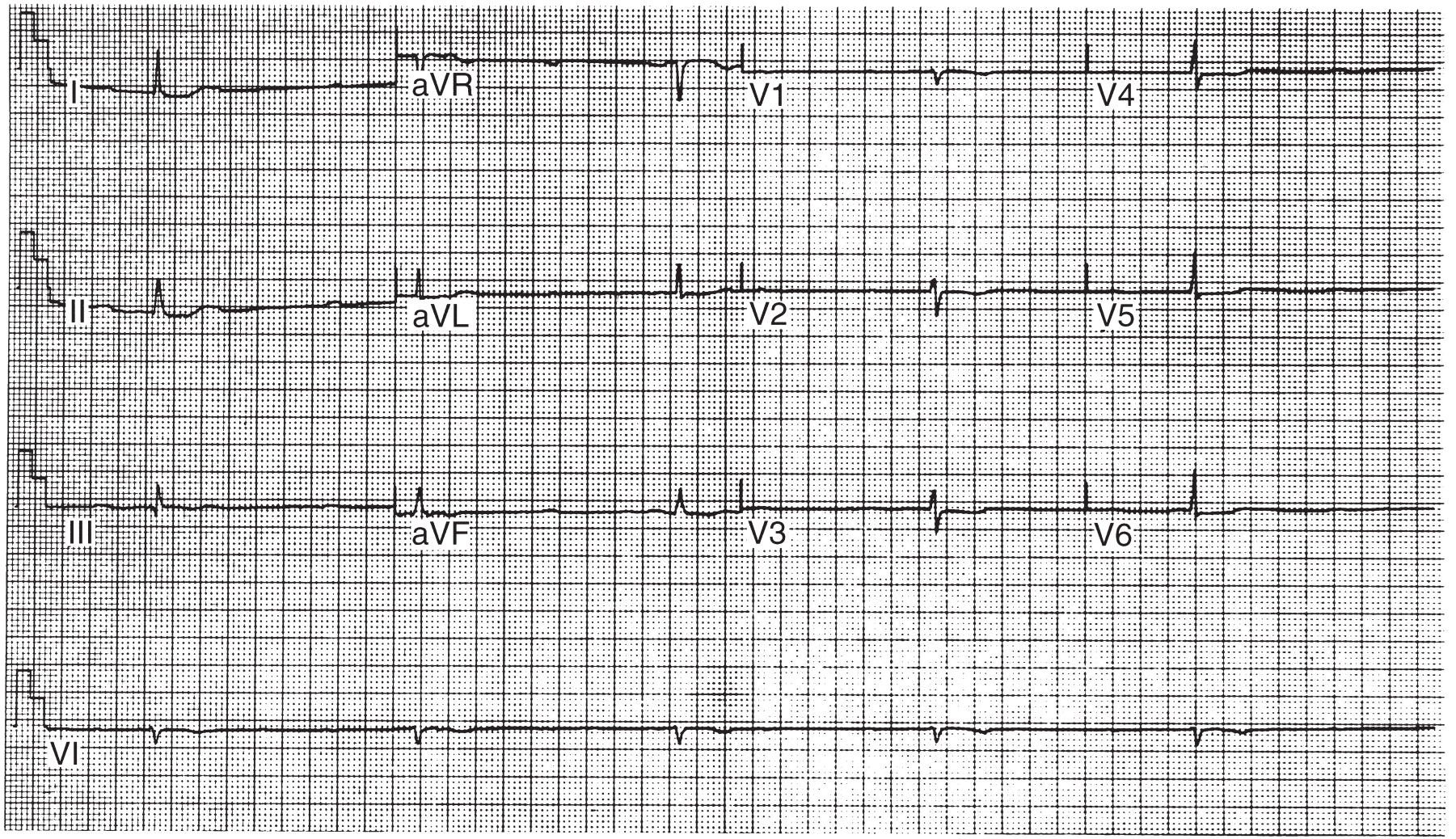


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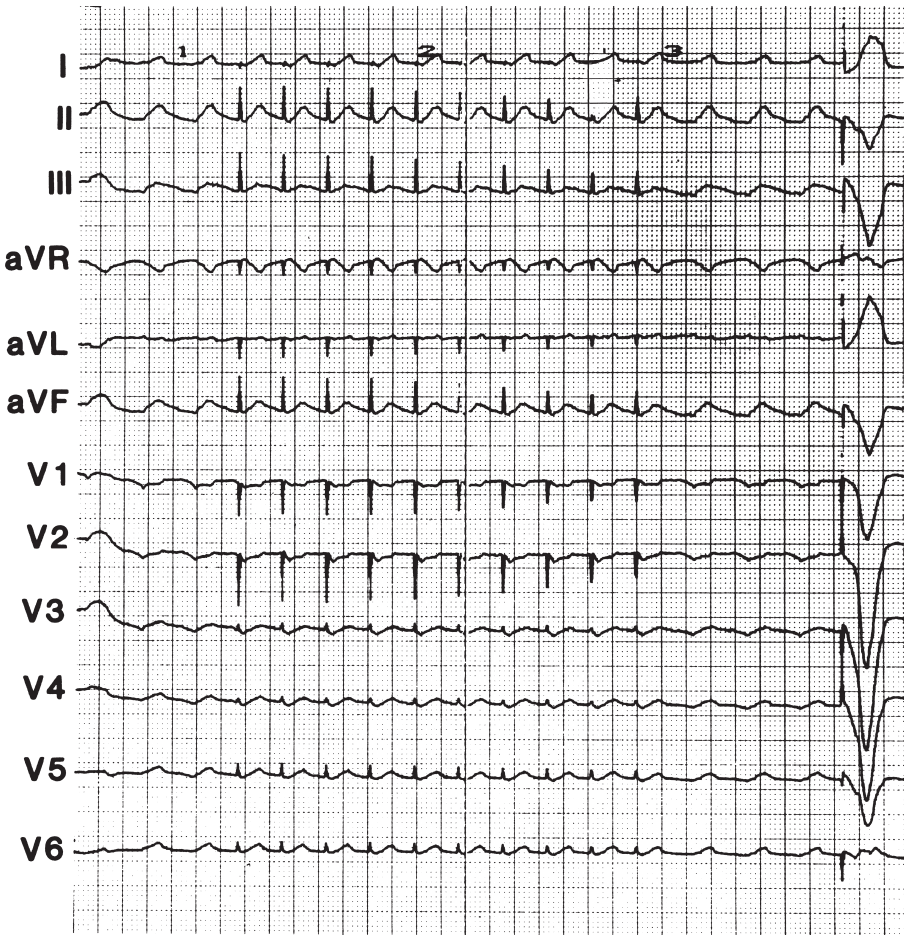
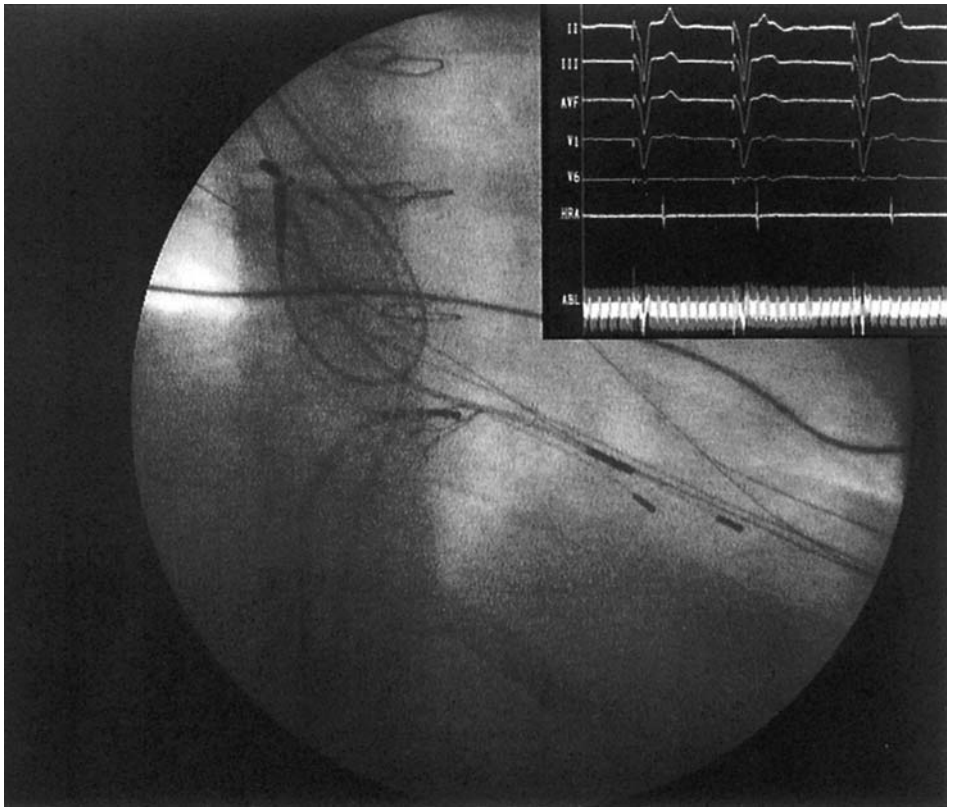
Figure 6–36A



This patient had persistent atrial flutter unresponsive to multiple antiarrhythmic drugs. Radio-frequency ablation was performed to cure the atrial flutter. The patient also had extremely poor AV node conduction and had a permanent dual chamber pacemaker in place. Prior to introduction of radio-frequency energy, pacing was performed at the ablation site. Does this appear to be a good site for ablation?

Figure 6–36B

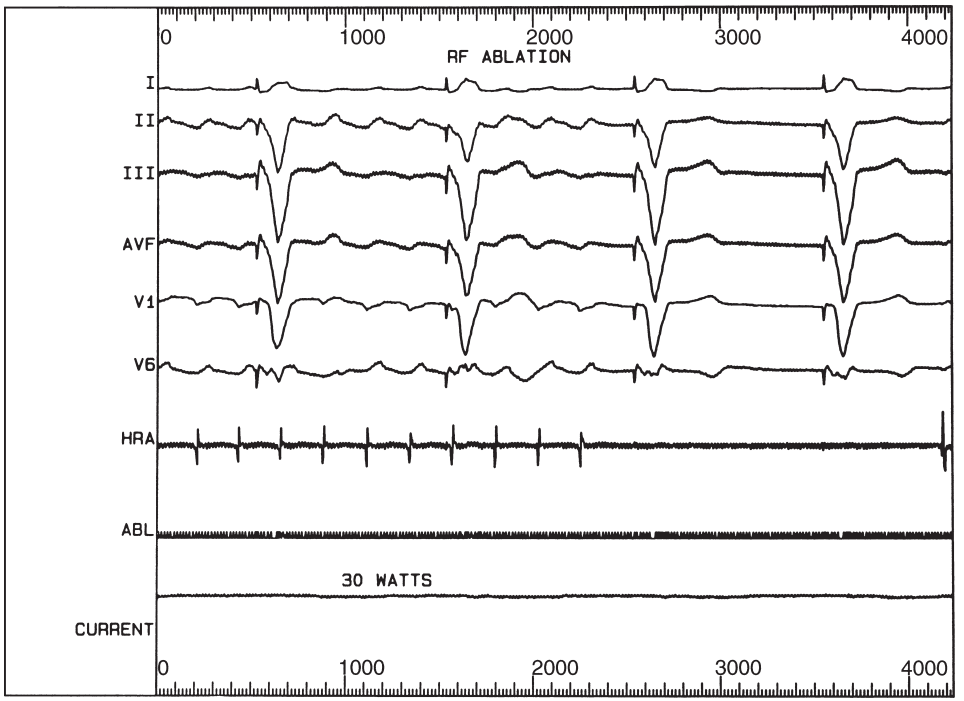


Explanation:

The ablation catheter was positioned in an area posterior to the CS os in the AV groove as noted in Fig. 6–36*B*. In Fig. 6–36*A*, pacing entrains the P waves during atrial flutter and the morphology of the paced and spontaneous P waves is nearly identical. Thus, the catheter must be very close to or within the tachycardia circuit. The stimulus to P wave is rather short, suggesting the catheter position is near the exit point in the isthmus of slow conduction in the circuit. Onset of radio-frequency energy at this site resulted in termination of atrial flutter Fig. 6–36*C*, and flutter has not recurred in this patient during long-term follow-up.

It is important to verify conduction block in the isthmus after RF ablation. This can be done by analyzing the conduction pattern and time from pacing sites near the CS os and the low lateral right atrium.

Figure 6–36C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HRA
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