ECG Masters' Collection

Favorite ECGs from Master Teachers Around the World

VOLUME 2

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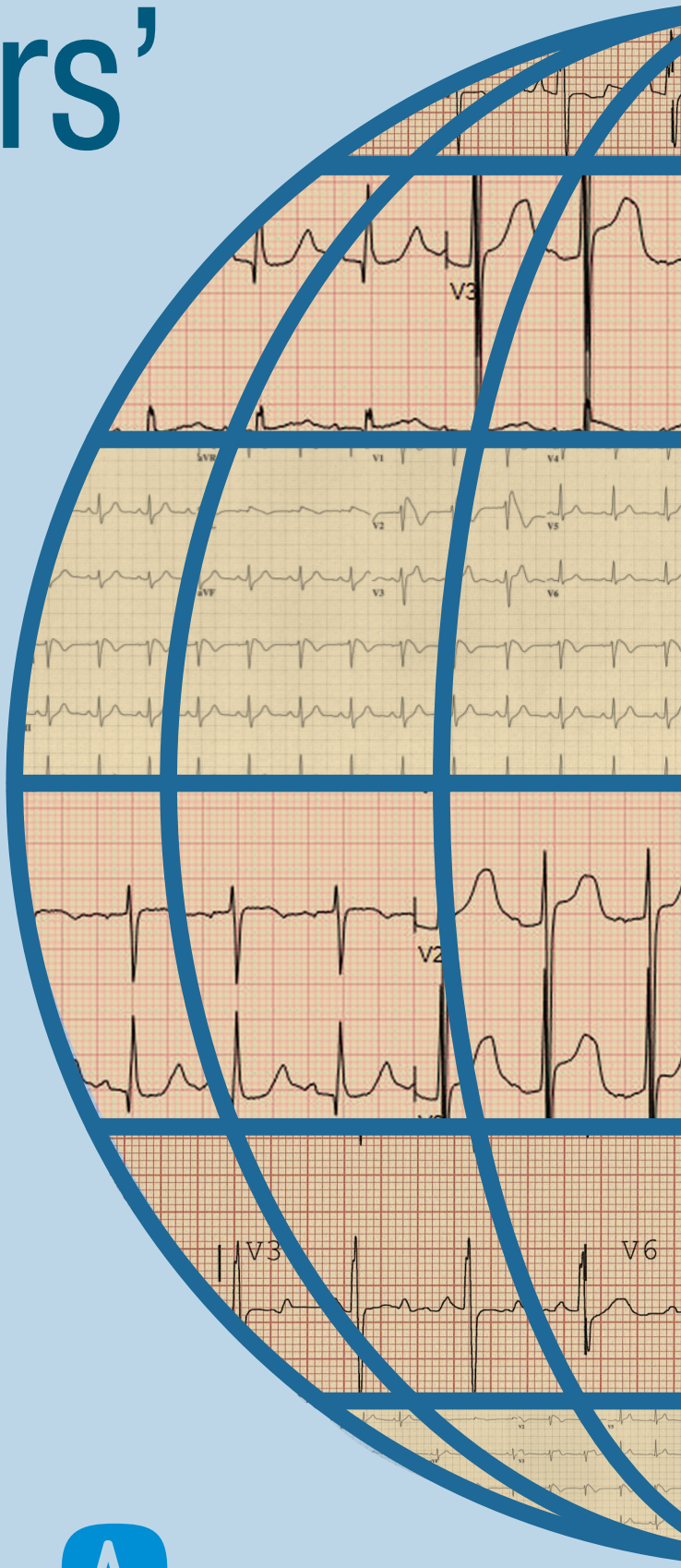
Forewords by
Francis E. Marchlinski and Samuel Lévy



cardiotext

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Dedication

We dedicate this book in memory of Mark E. Josephson, MD (January 27, 1943 – January 11, 2017), who inspired and mentored many of us. He gave the electrogram a new look: "ECG as a mapping tool." He is also the senior editor on this book.

Contents

* Included only in Volume 1.

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Foreword

It is exciting to see the publication of these case-based collections of ECGs from "Masters" in electrocardiography and electrophysiology. It is also a pleasure for me to contribute the foreword to these important volumes. This collection should be read by both early trainees and experienced electrocardiographers. There are pearls littered throughout and the explanations and interpretations grounded in physiology and fundamental vector analysis. The clinical relevance is made obvious by the case format. The outstanding group of editors has done a superb job with the organization of the text in dividing it into focused sections to maximize ease of review and educational value. The ECG recordings are characteristically the best from the experts' collections. The tracings show the incredible value of this simple yet elegant tool for diagnosing and localizing arrhythmias and recognizing signature ECG patterns associated with unique genetically determined and acquired arrhythmogenic syndromes.

It is important for me to also pay a special tip of the hat to one of the Co-Editors, Dr. Mark Josephson. Not only is he the father of modern cardiac electrophysiology, but Mark has also been inspirational in his love of the 12-lead ECG and his desire to maximize its full potential. For more than 40 years, he has mentored a long collection of trainees on the correct interpretation of the 12-lead ECG. Such phrases as "burn it in your brain" for a unique ECG pattern that was critical to recognize within a second of display are always remembered with a smile throughout one's career. I was a lucky trainee who has many critical ECG images "burned in my brain." His participation in this important text adds to the glow of the other stellar editors and ECG aficionados and provides the 'Grand Cru' stamp to this effort.

These books should serve as important references, and I guarantee they will be pulled from the shelf for decades to come. They are gems and should be enjoyed by even those with only a modest interest in the 12-lead ECG and the care of patients with cardiac arrhythmias. The true fans of the ECG will be awed by the experience.

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Foreword

The father of electrocardiography is Willem Einthoven (1860–1927), who first recorded the first human ECG in 1902 at the University of Leiden, the Netherlands, where he used to teach. He received the Nobel Prize in 1924 for his major invention. Since then, the ECG has fascinated a number of cardiologists by the amount of information that can be derived. Some of these famous electrocardiographers such as Alfred Pick and Richard Langendorf have described a number of phenomena such as "concealed conduction" or "tachycardia/bradycardia-dependent bundle branch block," which were found later on to be correct using invasive electrophysiology. My generation is fortunate to have met some of them and to learn from them. It is refreshing that Mohammad Shenasa, Mark E. Josephson, N.A. Mark Estes III, Ezra A. Amsterdam, and Melvin Scheinman, the editors of this ECG Masters' collection, have emphasized that the ECG remains an invaluable tool for clinicians despite the advances made in the field of arrhythmias. This contribution, with the participation of experts from around the world, will be extremely useful to clinicians, fellows in cardiology, and all those who are involved in the management of cardiac arrhythmia patients.

These books are not a simple collection of ECGs. They are, in fact, reports of clinical situations in which the ECG guides the diagnosis, signals the choice of the appropriate tests, and leads to the appropriate management. The cases presented are not rare or unusual. They represent clinical settings that cardiologists and clinicians will encounter in their daily practice — and this, in my view, adds to the educational value of these books. I found it very interesting and enjoyable to read the ECG tracings put into their clinical context.

The field of cardiac arrhythmias has been enriched by the major advances made in the last four decades in better understanding tachycardia mechanisms due to the advent of intracardiac recordings, invasive clinical electrophysiology, ablation, and new mapping techniques. The authors of the cases refer to these techniques to support their interpretation and document the concepts used in their interpretation, adding, when necessary, references and suggested reading.

I have no doubt that the authors have succeeded in providing the reader with an interesting, enjoyable, and useful collection of clinical situations in which the correct ECG interpretation has played a major role.

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Preface

Each year, several new books or new editions are published on electrocardiography. Since the invention of the electrocardiogram (ECG) by Willem Einthoven almost 110 years ago, the ECG has become the most commonly used test worldwide, and its use continues to increase. The medical community has subsequently gained a wealth of knowledge from the ECG for the diagnosis of many cardiac and non-cardiac conditions, ranging from acute ischemia and infarction on the one hand to arrhythmias on the other. Furthermore, the ECG is the first step in evaluating patients arriving at the emergency department, as the results are immediately available. Likewise, the ECG has been used as a screening test for athletes and is also used to identify patients at a high-risk of arrhythmias and sudden cardiac death.

Despite the emergence of other imaging modalities, the ECG remains a benchmark diagnostic test and is an integral part of a risk stratification algorithm in almost all guidelines of all disciplines of medicine.

Since the success of our 2015 book, *The ECG Handbook of Contemporary Challenges*, many of our colleagues and friends encouraged us to provide a case-based collection of ECGs. Thus, we have invited the most renowned physicians from around the world who read and interpret ECGs (i.e., electrocardiographers) to provide their most insightful examples. We also asked them to include their interpretation of the ECG findings with appropriate, up-to-date references. All tracings are well annotated and described. In addition, we suggested providing questions for the readers relating to the ECGs and a discussion that makes these books useful for trainees at all levels.

Although the main theme of these two volumes is electrocardiography, other imaging techniques are discussed to validate the authors' interpretations. We are extremely grateful that all of our colleagues have unanimously accepted our invitation and provided their best cases.

The cases are arranged according to topics in electrocardiography and arrhythmias. Areas of focus include ECGs of inherited arrhythmia syndromes, athletic ECGs, and ECGs in congenital heart disease. There is also discussion of new ECG criteria/markers and syndromes related to recently discovered channelopathies such as Brugada syndrome, early repolarization syndrome, and the like. We are confident that this collection of ECGs from masters of electrocardiography from around the world will prove useful and of great educational value to clinicians in many areas of medicine. We believe this unique collection is similar to receiving a master art collection from the Louvre or the Metropolitan Museum of Art that should be on everyone's shelf as an ECG museum.

Finally, we wish to thank the Cardiotext staff, namely Mike Crouchet and Carol Syverson, for their professionalism and for providing the text and figures in a high-quality format.

The Editors

Abbreviations

ACS	acute coronary syndrome
AF	atrial fibrillation
AP	accessory pathway
APC	atrial premature contraction
APD	action potential duration
ARVC/D	arrhythmogenic right ventricular cardiomyopathy/dysplasia
AT	atrial tachycardia
AVNRT	atrioventricular nodal reentrant tachycardia
AVRT	atrioventricular reentrant tachycardia
BB	bundle branch
BBB	bundle branch block
BS	Brugada syndrome
CAD	coronary artery disease
CHF	congestive heart failure
CL	cycle length
DDD	dual-chamber pacing
EADs	early afterdepolarizations
ECG	electrocardiogram
EF	ejection fraction
HCM	hypertrophic cardiomyopathy
HPS	His-Purkinje system
HR	heart rate
ICD	implantable cardioverter-defibrillator
LA	left atrial
LAD	left axis deviation
LAFB	left anterior fascicular block
LBB	left bundle branch
LBBB	left bundle branch block
LQTS	long QT syndrome

LV	left ventricle
LVEF	left ventricular ejection fraction
LVH	left ventricular hypertrophy
LVOT	left ventricular outflow tract
MI	myocardial infarction
MRI	magnetic resonance imaging
OD	once daily
ORT	orthodromic reciprocating tachycardia
PABs	premature atrial beats
PAC	premature atrial contraction
PJRT	permanent form of junctional reciprocating tachycardia
PV	pulmonary vein
PVC	premature ventricular contraction
PVI	pulmonary vein isolation
RA	right atrial
RBB	right bundle branch
RBBB	right bundle branch block
RV	right ventricle
RVA	right ventricular apex
RVH	right ventricular hypertrophy
RVOT	right ventricular outflow tract
SCD	sudden cardiac death
SQTS	short QT syndrome
SR	sinus rhythm
SVT	supraventricular tachycardia
TdP	torsades de pointes
VF	ventricular fibrillation
VT	ventricular tachycardia
WCT	wide complex tachycardia
WPW	Wolff–Parkinson–White syndrome

SECTION 1

Introduction to the Interpretation of the Electrocardiogram

CASE
1.1

Mohammad Shenasa, MD

The first and most important step in ECG interpretation is the differentiation between "normal" and "abnormal."

The second step consists of differentiation between the various abnormal ECG patterns and their correlation with known pathologic conditions. In particular, the recent discoveries with small, subtle, significant markers for adverse events such as early repolarization, Brugada-type ECGs, and other channelopathies.

Information about the ECG in disease is much more complex than knowledge of normal variation. Yet, it is in the differentiation between normal and abnormal that difficulties in ECG interpretation frequently arise.

Below are two examples of normal ECGs.

Heart rate: 64 bpm
PR interval: 154 ms
QRS duration: 98 ms
QT/QTc: 406/415 ms
Normal ST-T wave patterns

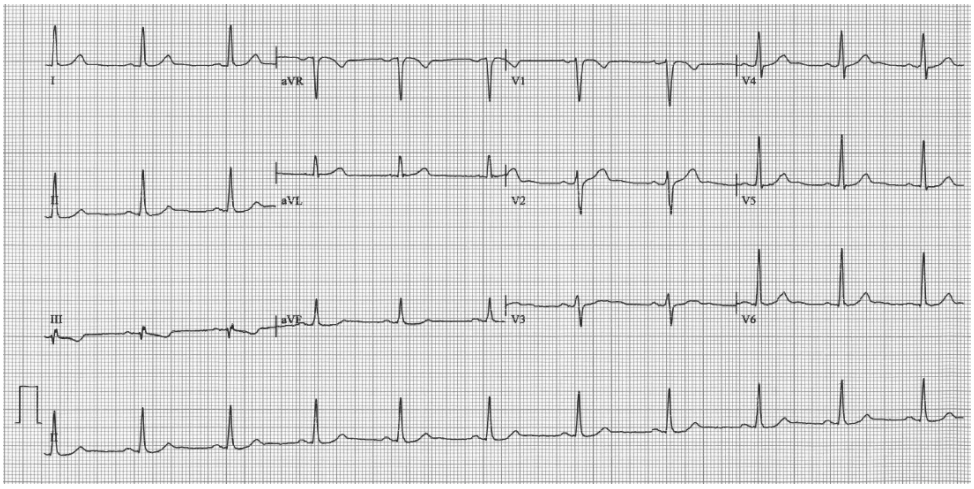

Figure 1.1.1

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Heart rate: 80 bpm
PR interval: 148 ms
QRS duration: 92 ms
QT/QTc: 364/420 ms

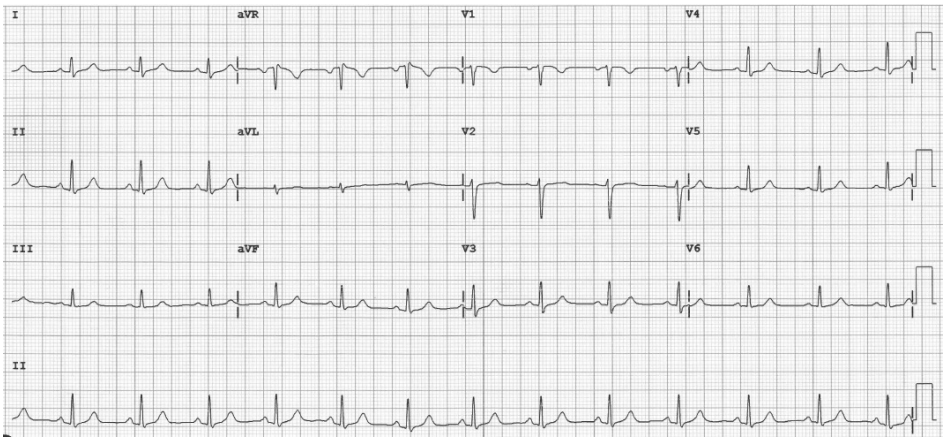

Figure 1.1.2

It is important to have a systematic approach when analyzing and interpreting of ECGs.

1. Baseline findings in sinus rhythm.
2. Observations during tachycardias.
3. Analysis of the changes of the cardiographic morphologies (transient changes).
4. Mode of spontaneous initiation and termination.
5. Maneuvers during tachycardias.

In a stepwise approach to ECG or rhythm analysis, one should determine the rate of the tachycardia (fast or slow), the QRS duration (wide or narrow) and morphology, and the relationship of the P wave to the QRS, whether it is before, during, or after, and if there is a one-to-one relationship between the P wave and the QRS.

Other important points regarding interpretation of the ECG.

1. Determine the origin and initiation of cardiac arrhythmias.
2. Look for myocardial ischemia and infarction.
3. Evidence of electrolyte imbalance and reversible causes.
4. Systemic and myocardial disorders.
5. Measure; do not eyeball the intervals.
6. Focus on the zone of transition.

References

1. Wellens HJ, Gorgels AP. The electrocardiogram 102 years after Einthoven. *Circulation*. 2004;109(5):562–564.
2. Yong CM, Froelicher V, Wagner G. The electrocardiogram at a crossroads. *Circulation*. 2013;128(1):79–82.
3. Stern S. Electrocardiogram: Still the cardiologist's best friend. *Circulation*. 2006;113(19):e753–e756.

SECTION 2

Conduction Disturbances: Sinus Node Disease/Sick Sinus Syndrome, AV Conduction Disturbances, AV Blocks, Bundle Branch Blocks, and Fascicular Blocks

Mohammad-Ali Jazayeri, MD
Mohammad-Reza Jazayeri, MD

CASE
2.1

Patient History

Two cases are shown in **Figure 2.1.1**. The first is from a 68-year-old female with severe aortic stenosis who underwent aortic replacement three days prior to the date that the rhythm strip (Panel A) was obtained. Her surgery was uneventful, and her native valve was replaced with a 21-mm Medtronic Mosaic tissue valve. The second case is an 84-year-old male with a 10-day history of recurrent syncope who was admitted to the hospital.

The rhythm strip (Panel B) was obtained from the patient one day after admission.

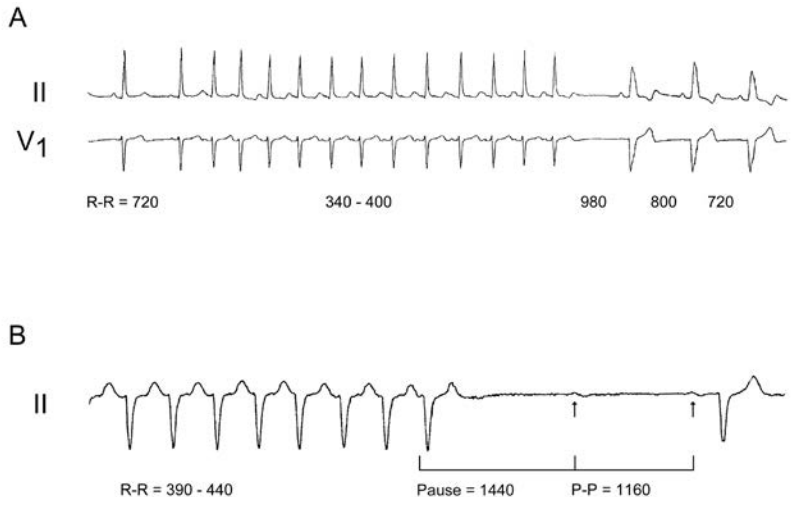

Figure 2.1.1 The occurrence of block after termination of supraventricular tachycardia (SVT). Panel A shows a non-sustained run of narrow QRS complex atrial tachycardia (AT) followed by sinus beats exhibiting left bundle branch block (LBBB). Panel B depicts termination of a wide QRS complex AT followed by a 1440-ms pause and then a blocked sinus beat. The subsequent sinus beat is conducted with first-degree atrioventricular (AV) block (PR interval of 300 ms) and a QRS complex similar to that during AT. All of the intervals are in ms. The paper speed is different in these two panels.

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Question

What is the probable mechanism of block in **Figure 2.1.1A** and **B**?

1. Pause-dependent block
2. His-Purkinje system (HPS) fatigue phenomenon
3. Functional block in the HPS
4. Potent vagal stimulation

Answer

The correct answer is A. The common theme in these two cases is the termination of a run of supraventricular tachycardia (SVT), followed by a pause before the arrival of the next sinus (P wave) beat. Subsequently, the P wave is conducted with LBBB in panel A and bilateral BBB in panel B. In other words, the occurrence of block is preceded by a sudden short-to-long input to the AV conduction system. It should be mentioned that the SVT, in both cases, is most likely AT with narrow QRS complex in Case 1; and with RBBB and left anterior fascicular block (LAFB) (bifascicular block) in Case 2 (**Figure 2.1.2**). Case 2 suddenly developed third-degree AV block later during the same hospital course (**Figure 2.1.3**).

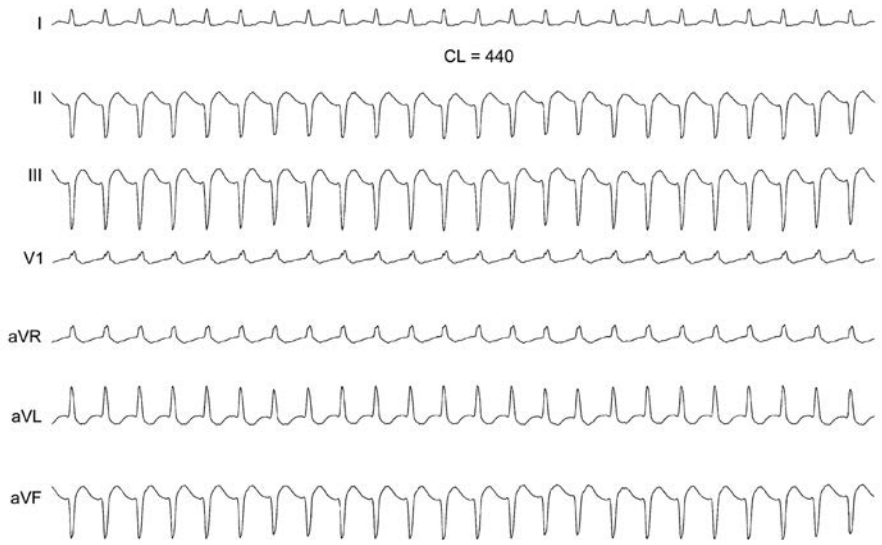

Figure 2.1.2 Sustained AT. Seven-lead ECG of a sustained episode of AT with a cycle length (CL) of 440 ms is shown. The QRS complex is 120 ms with RBBB and LAFB present.

Discussion

Over the past 65 years, a number of terms have been offered for this intriguing HPS block. These include bradycardia-dependent block, Phase 4 block, (heart-rate) deceleration block, and pause-dependent block. Spontaneous diastolic depolarization (SDD) is a normal electrophysiology property of the specialized automatic cells including the HPS, which is normally responsible for the impulse formation. Although the exact pathophysiologic mechanism of this type of block is still unknown, preexisting HPS conduction disease is almost always the case even if the QRS complex appears narrow. Therefore, this is a pathologic response that is usually not expected to occur in the normal HPS. Among several mechanisms that have been offered, we discuss the two prevailing hypotheses as follows.

From a historical point of view, bradycardia-dependent BBB was recognized in the early years of the 20th century, shortly after the invention of the ECG recorder.[1] Bradycardia was attributed to a

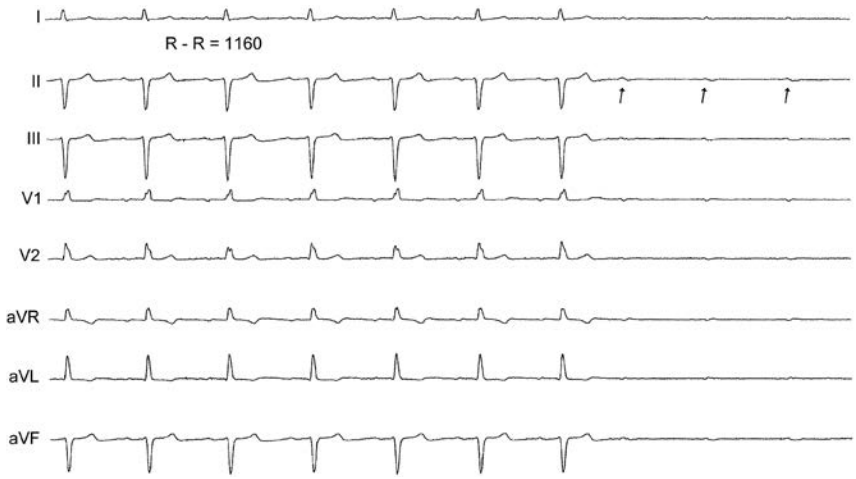

Figure 2.1.3 Spontaneous third-degree AV block. The 8-lead ECG tracing starts with sinus bradycardia (cycle length of 1160 ms) and 1:1 AV conduction. There is first-degree AV block and RBBB plus LAFB present during sinus rhythm, the same as during the AT shown in **Figure 2.1.2**. The last three sinus beats (arrows) block spontaneously without any obvious provocation.

heightened vagal tone in those patients. The first indisputable case of BBB during heart-rate deceleration was reported in 1959.[2] Subsequently, in 1967, enhanced SDD during phase 4 (**Figure 2.1.4**) in the HPS automatic cells was put forth as a putative mechanism of a broad spectrum of conduction disturbances ranging from simple slowing to complete block.[3] The same investigators also proposed two other concomitant alterations in the EP characteristics of the

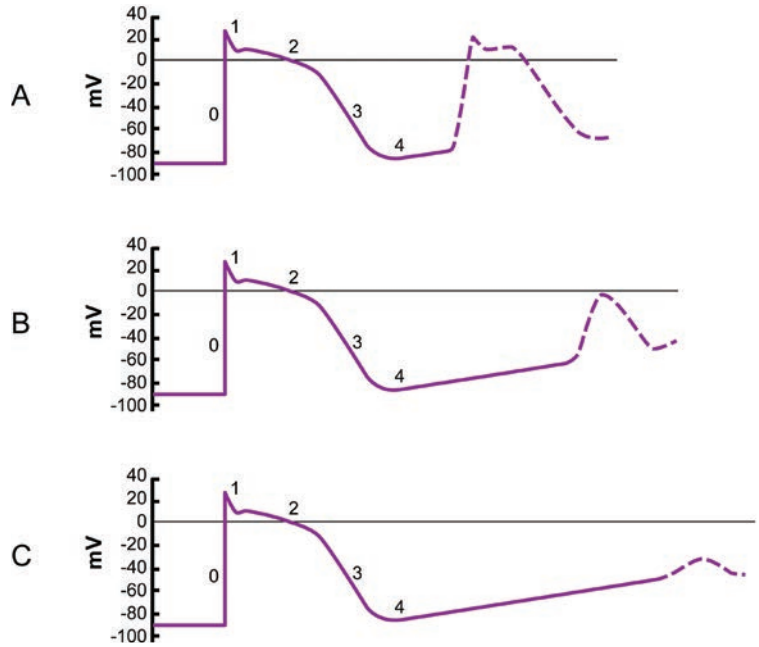

Figure 2.1.4 Spontaneous phase 4 depolarization. This is a schematic representation of transmembrane action potentials (AP) of the automatic Purkinje cells shown during phase 4 depolarization. The AP on the left side of each panel shows the first impulse with normal depolarization (phase 0) and repolarization phases 1, 2, and 3. Spontaneous depolarization of phase 4 is presumably in response to a decrease in the frequency of stimulating impulses. The AP on the right side of each panel (dashed line) represents the second impulse arriving at different times during diastole. In panel A, the second impulse (SI) provokes a near normal response because the membrane potential (MP) is –80 mV, slightly less negative than the normal resting MP. In panel B, the SI is introduced at the time the MP is –60 mV and therefore, the AP amplitude and its dv/dt_{max} is markedly diminished. In panel C, the SI arrives later during diastole at a time when the MP is –40 mV. This results in a subthreshold response. (Used with permission from Singer DS et al., *Circ Res* 1967;21:537-558.)

transmembrane action potential (TAP) in order for SDD to exert its maximal deleterious effect on the conduction disturbances in a diseased HPS. These alterations were: (1) generalized diastolic depolarization (hypopolarization),[3-5] which raise the resting potential to the less negative values (>–70), giving rise to TAPs with diminished phase-0 amplitude, and (2) a shift of threshold potential towards zero.[3] In 1968, development of "bradycardia-dependent" BBB in patients was linked to enhanced SDD during phase-4 of TAP for the first time.[6] The majority of ensuing reports also favored the same mechanism as the most plausible explanation for development of "bradycardia-dependent" HPS block and the term "phase-4 block" emerged in the literature and quickly became popular.[7-13]

The second hypothesis appeared in the literature in 1983.[14,15] By using the sucrose gap model, these investigators studied the frequency-dependent alterations of conduction in Purkinje fibers. They assessed the time-dependent changes of two determining factors for conduction across a zone of block: (1) the magnitude of the electrotonic current flow and (2) the efficacy of the electrotonic inputs in exciting the distal site. Based on these studies, successful propagation from proximal to distal sites required a delicate balance between these two factors. Otherwise, conduction delay or block developed due to a source-to-sink mismatch. The resultant time-course conduction disturbances, by appearing in early and late diastole (**Figure 2.1.5**), exhibited a biphasic pattern (an early block followed by full conduction, followed by a late block). This biphasic behavior could be

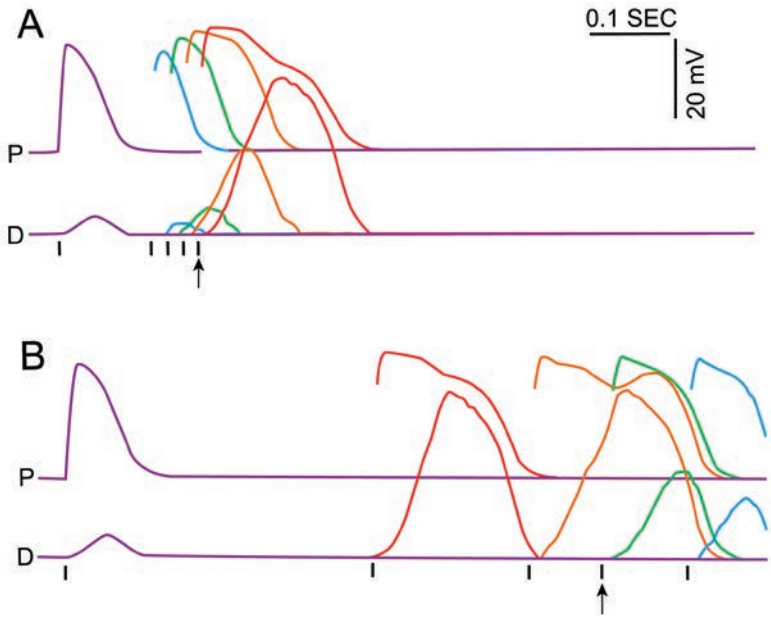

Figure 2.1.5 Time-dependent biphasic ("phase 3" and "phase 4") responses in Purkinje fiber-sucrose gap preparation. Tracings are schematic representations of the superimposed APs across a Purkinje fiber from proximal (P) to distal (D) sites, 12 mm apart, in response to bipolar stimulating electrodes. Ten-beat trains of stimuli were applied at a basic cycle length of 500 ms, and then the diastolic intervals between the trains were scanned with test stimuli at progressively longer intervals. Note that the initial stimuli during basic drive (purple) generate APs with short duration in the P sites and subthreshold responses in the D sites. Subsequent test stimuli (panel A) continue to generate short-duration APs in the P site and subthreshold responses in the D site. This early diastolic P-to-D block may correspond to what is also known as short-cycle ("phase 3") block. At a critical interval, the stimulus evoking a full-fledged AP (arrow) generates a response at the D site, which reaches threshold. All the ensuing stimuli (panel B) follow suit for the next 300 ms until the stimulus again becomes unable to elicit fully developed AP (arrow) at the D sites. This late diastolic P-to-D block may correspond to what is also known as long-cycle ("phase 4") block. (Used with permission from Jalife J et al., *Circulation* 1983;67:912-922.)

interpreted as the equivalent of what had been designated previously as "phase 3" and "phase 4" block, respectively. Furthermore, these investigators did not dismiss the SDD contribution to the development of deceleration-dependent block, but they refuted its necessity as a prerequisite. Although both studies have been extremely helpful in understanding the underlying mechanism of the phenomenon observed in experimental animal models, it seems reasonable to address this type of block as "deceleration-dependent," "pause-dependent," or "long-cycle" until the exact mechanism(s) of this arrhythmia is (are) determined in humans. However, most of the current literature accepts the phrase "pause-dependent."

Deceleration-dependent HPS block is occasionally preceded by SVT termination as shown in both cases presented here. More commonly, however, such a block is preceded by premature impulses, either supraventricular (**Figure 2.1.6**) or ventricular (**Figure 2.1.7**) in origin. Occasionally, the onset of block is sudden and without any visible premature beat as a trigger. In such cases, the possibility of premature ventricular or junctional impulses with bidirectional block (i.e., non-propagated impulses)[16] cannot be excluded (**Figure 2.1.8**). Once the block occurs, it perpetuates itself until the area of block is fully repolarized, allowing conduction to resume. Termination of persistent HPS block may be facilitated (or mediated) by acceleration of the impulses approaching the site of block or by premature impulses depolarizing that site (**Figure 2.1.8**).

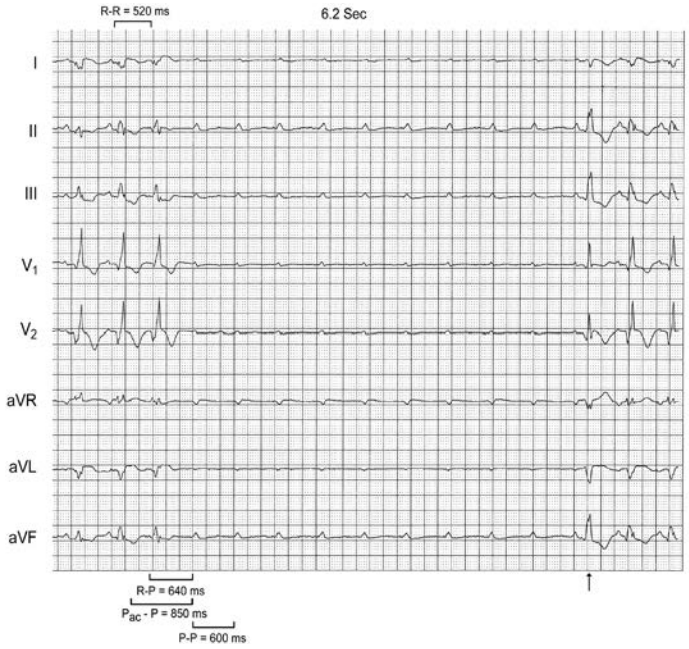

Figure 2.1.6 Sudden onset of third-degree AV block preceded by a premature atrial complex (PAC). This is an 8-lead ECG taken from an 84-year-old woman with a history of prior anteroseptal myocardial infarction (MI), hypertension, and hyperlipidemia admitted with persistent chest pain and elevated troponin consistent with an acute non-STEMI who underwent a percutaneous coronary intervention (PCI). This ECG was obtained on the subsequent day after PCI showing two sinus beats at a rate of 100 bpm with RBBB with a right axis deviation, most likely due to left posterior fascicular block. The presence of abnormal Q waves is compatible with a previous inferior and anteroseptal infarction. The third beat is a conducted PAC. The subsequent sinus P wave arrives 850 ms after the PAC and blocks completely due to heart-rate deceleration, which persist for the next 6.2 seconds. Worth noting is that the sinus PP interval remains 600 ms in duration throughout. The first QRS complex (arrow) after the pause appear slightly different than the other complexes, which may be due to incomplete recovery of the HPS at the time of sinus impulse arrival. Alternatively, this QRS complex could be an extrasystolic beat arising from the HPS.

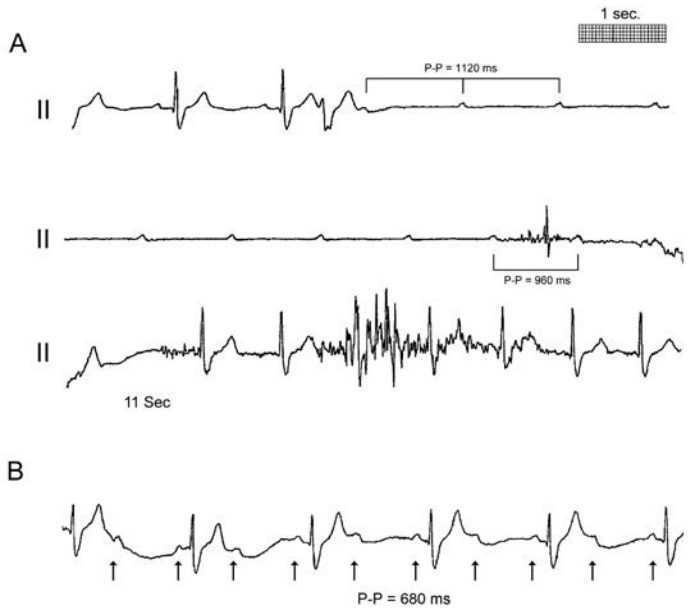

Figure 2.1.7 Sudden onset of third-degree AV block preceded by a premature ventricular complex (PVC). This lead II rhythm strip was obtained from an 80-year-old man who presented with a recent-onset recurrent syncope. A 12-lead ECG showed sinus rhythm with RBBB. The rhythm strip shows sinus bradycardia with a rate of 50 bpm. A PVC is followed by an 11-second period of third-degree AV block and ventricular asystole. The first blocked sinus beat is most likely an encountered block in the AV node due to retrograde concealment of the PVC. However, the subsequent sinus beat is blocked due to rate deceleration of the impulse arriving at the His-Purkinje system (HPS), approximately 1600 ms after the PVC (i.e., prior to HPS activation). Note that the sinus cycle length shortens to 960 ms, shortly before resumption of the AV conduction. The rhythm strip is continuous in panel A. The noise (third tracing) is probably due to the patient's involuntary activity during a brief episode of loss of consciousness. Panel B shows an episode of 2:1 AV block, which had occurred approximately 2 hours before the episode shown in panel A.

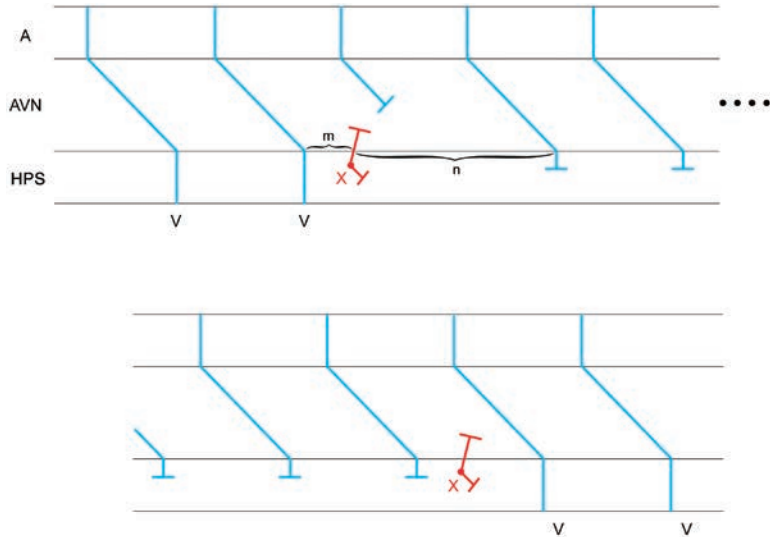

Figure 2.1.8 Possible mechanisms of initiation and termination of deceleration-dependent block. The laddergram depicts sinus beats and non-propagated premature ventricular beats (X). The first two sinus beats are conducted normally. The first X impulse is non-propagated because it is blocked retrogradely in the AV node and antegradely in the HPS. The subsequent (third) sinus beat is blocked in the AV node because of the retrograde concealment of the X impulse. From the time of the retrograde HPS concealment to the subsequently occurring antegrade HPS activation during the fourth sinus beat, a pause (n) is created, which is longer than the prior HPS activation (m) CL. This short-to-long CL variation sets the stage for deceleration-dependent AV block in the HPS. The second X impulse, by depolarizing the HPS, resets the electrophysiological parameter(s) crucial for the persistence of the block and therefore allows resumption of HPS conduction. The asterisks signify a lapse of time between the upper and lower laddergrams.

HPS fatigue (answer B) is a rare phenomenon, in which HPS block occurs after either cessation of rapid ventricular burst pacing or termination of ventricular tachycardia. Like deceleration-dependent block, the HPS fatigue phenomenon is also a pathological behavior of the diseased HPS.[17-20] Both situations share a common feature, and that is a pause preceding the onset of the HPS block. Thus, it is conceivable that they also share the same underlying mechanism. Alternatively, it has been shown in experimental animals that overdrive suppression of conduction may be due to transient block at the Purkinje muscle junction.[21] Depending upon whether or not the supraventricular impulses can penetrate the conduction system and reach the depressed HPS as effectively as their ventricular counterparts, rapid atrial pacing may or may not provoke suppression of conduction and HPS block. Functional HPS block (answer C), unlike this entity, occurs physiologically when the heart rate accelerates beyond a critical point or the HPS input is preceded by a long-to-short cycle length variation. Potent vagal stimulation (answer D) may also cause AV block, but its potential mechanism(s), ECG manifestations, diagnosis, and treatment are entirely different.[22]

References

1. Wilson FN. A case in which the vagus influenced the form of the ventricular complex of the electrocardiogram. *Arch Intern Med.* 1915;16:1008–1027.
2. Dressler W. Transient bundle branch block occurring during slowing of the heartbeat and following gagging. *Am Heart J*. 1959;58:760–764.
3. Singer DH, Lazzara R, Hoffman BF. Interrelationship between automaticity and conduction in Purkinje fibers. *Circ Res*. 1967;21:537–558.
4. Rosenbaum MB, Elizari MV, Lázzari JO, et al. The mechanism of intermittent bundle branch block: Relationship to prolonged recovery, hypopolarization and spontaneous diastolic depolarization. *Chest*. 1973;63:666–677.
5. Rosenbaum MB, Elizari MV, Levi RJ, et al. Paroxysmal atrioventricular block related to hypopolarization and spontaneous diastolic depolarization. *Chest*. 1973;63:678–688.
6. Massumi RA. Bradycardia-dependent bundle-branch block. A critique and proposed criteria. *Circulation*. 1968;38:1066–1073.
7. El-Sherif N. Tachycardia-dependent versus bradycardia-dependent intermittent bundle-branch block. *Br Heart J*. 1972;34(2):167–176.
8. Elizari MV, Nau GJ, Levi RJ, et al. Experimental production of rate-dependent bundle branch block in the canine heart. *Circ Res.* 1974;34(5):730–742.
9. Rosenbaum MB, Elizari MV, Chiale P, et al. Relationships between increased automaticity and depressed conduction in the main intraventricular conducting fascicles of the human and canine heart. *Circulation*. 1974;49:818–828.
10. Barold SS, Ong LS, Young JA. Electrocardiographic observations in bradycardia and tachycardia-dependent atrioventricular block. Relationship to supernormal phase of intraventricular conduction. *Chest*. 1975;67:450–745.
11. MB Rosenbaum, JO Lazzari, MV Elizari. The role of phase 3 and phase 4 block in clinical electrocardiography. In: *The Conduction System of the Heart.* Wellens HJJ, et al., Eds. Philadelphia, PA: Lea & Febiger. 1976:126–144.
12. Cohen HC, D'Cruz I, Arbel ER, et al. Tachycardia and bradycardia-dependent bundle branch block alternans: Clinical observations. *Circulation*. 1977;55:242–246.
13. Wu D, Deedwania P, Dhingra RC, et al. Electrophysiologic observations in a patient with bradycardia-dependent atrioventricular block. *Am J Cardiol*. 1978;42:506–512.
14. Antzelevitch C, Jalife J, Moe GK. Frequency-dependent alterations of conduction in Purkinje fibers: A model of phase 4 facilitation and block. In: *Frontiers of Cardiac Electrophysiology*. Rosenbaum MB, Elizari MV, Eds. Amsterdam: Martinus Nijhoff. 1983:397–415.
15. Jalife J, Antzelevitch C, Lamanna V, et al. Rate-dependent changes in excitability of depressed cardiac Purkinje fibers as a mechanism of intermittent bundle branch block. *Circulation*. 1983;67:912–922.
16. Rosen KM, Rahimtoola SH, Gunnar RM. Pseudo A-V block secondary to premature nonpropagated His bundle depolarizations: Documentation by His bundle electrocardiography. *Circulation*. 1970;42:367–373.

17. Narula DS and Runge M. Accommodation of AV nodal conduction and fatigue phenomenon in the His-Purkinje system. In: *The Conduction System of the Heart.* Wellens HJJ, et al., Eds. Philadelphia, PA: Lea & Febiger. 1976:529–544.
18. Fisch C. Bundle branch block after ventricular tachycardia: A manifestation of "fatigue" or "overdrive suppression." *J Am Coll Cardiol*. 1984;3:1562–1564.
19. DiLorenzo DR, Sellers TD. Fatigue of the His-Purkinje system during routine electrophysiologic studies. *Pacing Clin Electrophysiol*. 1988;11:263–270.
20. Barold SS, Barold HS. Demonstration of a His-Purkinje fatigue phenomenon with programmed stimulation of the right ventricular outflow tract. *J Interv Card. Electrophysiol.* 2000;4:489–491.
21. Gilmour RF, Jr, Davis JR, Zipes DP. Overdrive suppression of conduction at the canine Purkinje-muscle junction. *Circulation*. 1987;76:1388–1396.
22. Lee S, Wellens HJ, Josephson ME. Paroxysmal atrioventricular block. *Heart Rhythm*. 2009;6:1229–1234.

Balaji Krishnan, MD
David G. Benditt, MD

CASE 2.2

Patient History

These tracings were obtained in a 21-year-old female college-level rower who had been in good health and was on no prescription or over-the-counter medications. She presented with complaints of intermittent palpitations but denied dizziness or loss of consciousness. Laboratory tests including an echocardiogram and cardiac MRI were normal. An exercise stress test was well tolerated and induced no arrhythmias.

Heart rate: 56 bpm
QRS duration: 80 ms
QT: 405 ms

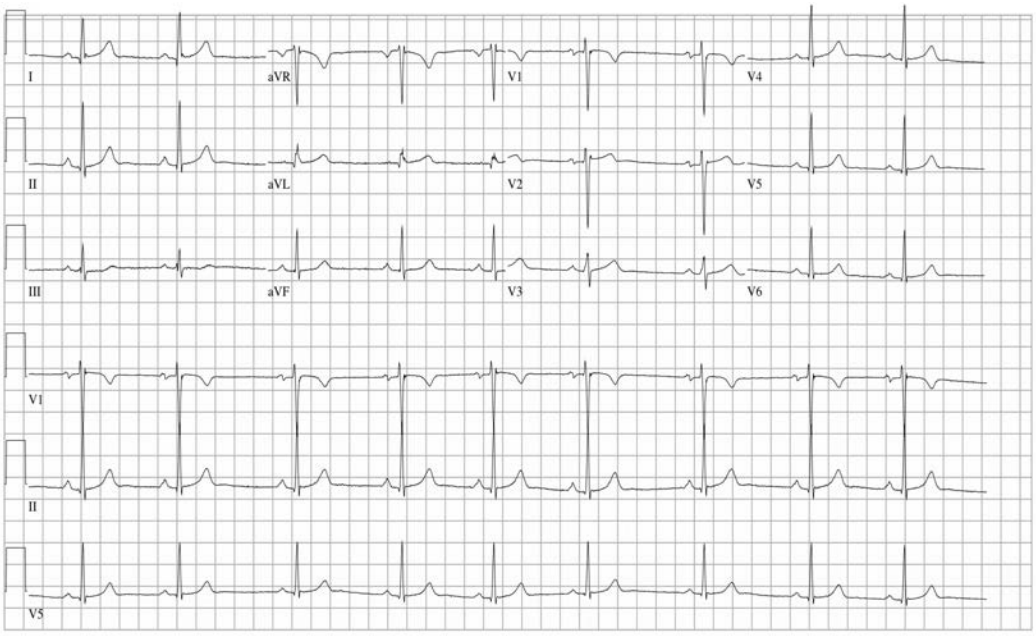

Figure 2.2.1

The 12-lead ECG showed sinus bradycardia with marked sinus arrhythmia (average heart rate 56 bpm). An ambulatory ECG monitor revealed sinus arrhythmia, which correlated with her "palpitations," and episodes of paroxysmal complete atrioventricular (AV) block ranging from 4 to 13.6 seconds mainly at night (see **Figure 2.2.2**). The pauses were not associated symptoms and there was no past medical history to suggest sleep apnea.

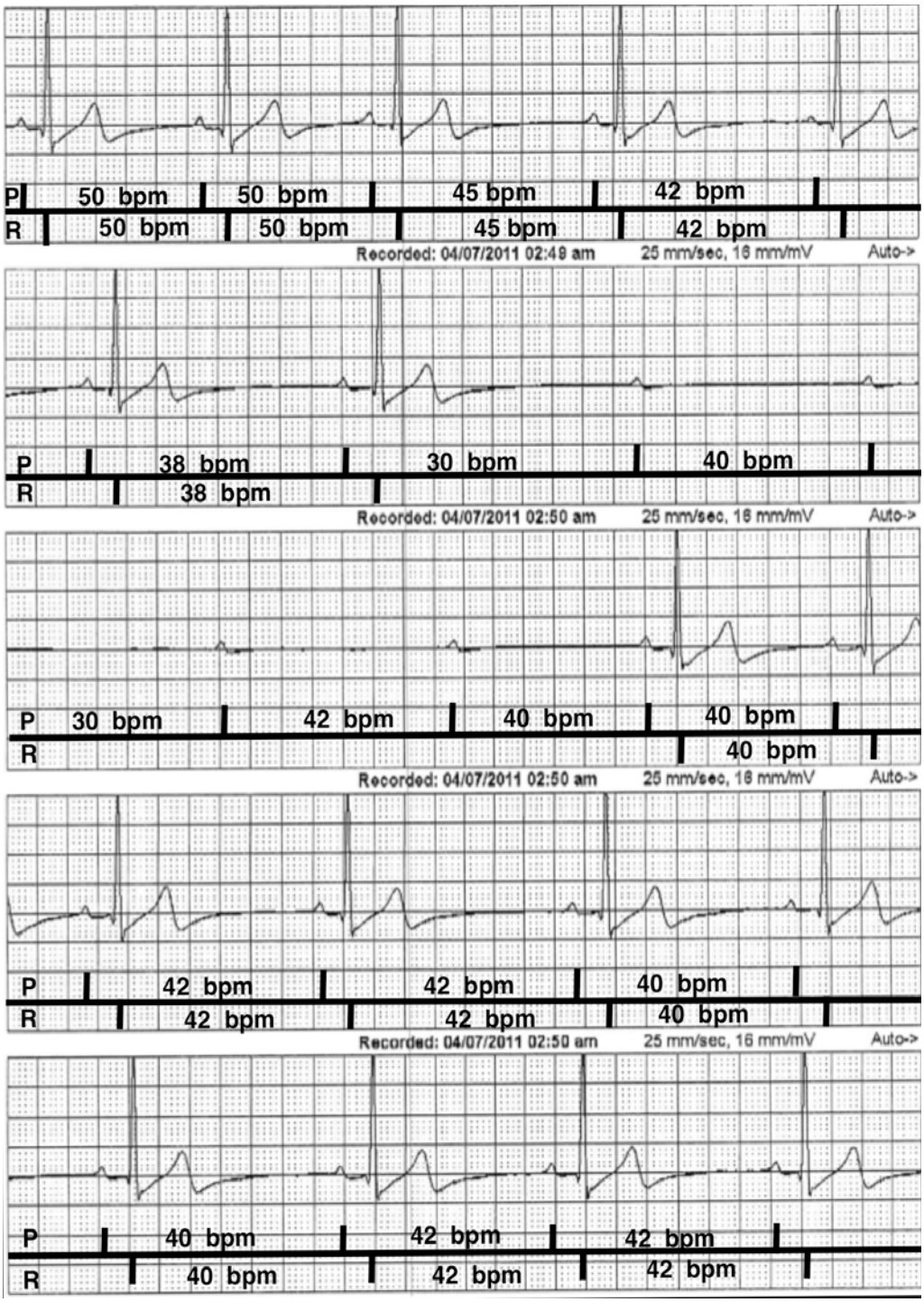

Figure 2.2.2 Ambulatory ECG monitoring strip at the time of symptoms. Note the recording shows a gradually slowing heart rate before there is complete AV block, resulting in a pause of 7.8 seconds. During AV block, the atrial rate remains relatively slow, strongly favoring an autonomic etiology for the AV block.

Discussion

Due to lack of symptoms during daytime activity, including sports, and absence of guidelines to the contrary, a pacemaker was not deemed indicated and there was no reason to restrict her activities.

In 5 years of annual follow-up, the patient has continued vigorous competitive rowing without problems.

Figure 2.2.2 showed a gradually decreasing heart rate prior to transient complete AV block. During the block the atrial rate remains slow, favoring an autonomic basis for the disturbance.[1–3] In AV block due to conduction system disease, one would expect the atrial rate to increase in an attempt to "compensate" for the hemodynamic stress (assuming absence of drug effects, such as from beta-adrenergic blockers that may impair this response). In any case, an increased sinus rate would not provide effective compensation if the AV block persists. In AV block due to autonomic disturbance, as in this case, both the AV node and the sinus node are affected by presumed excess parasympathetic influence.[1,3]

Disclosure

This work was supported in part by a grant from the Dr. Earl E. Bakken Family in support of Heart-Brain research.

References

1. Massie B, Scheinman MM, Peters R, et al. Clinical and electrophysiologic findings in patients with paroxysmal slowing of the sinus rate and apparent Mobitz II atrioventricular block. *Circulation.* 1978;58:305–314.
2. Strasberg B, Lam W, Swiryn S, et al. Symptomatic spontaneous paroxysmal AV nodal block due to localized hyperresponsiveness of the AV node to vagotonic reflexes. *Am Heart J.* 1982;103:304–314.
3. Alboni P, Holz A, Brignole M. Vagally mediated atrioventricular block: Pathophysiology and diagnosis. *Heart.* 2013;99(13):904–908.

CASE 2.3

Balaji Krishnan, MD
David G. Benditt, MD

Patient History

A 67-year-old female presented to the emergency department (ED) after an abrupt collapse with a minor laceration on her scalp. She reported having a similar event about 1 year earlier. She had no known cardiac disease, no previous illnesses, and no pertinent family history. She was not taking any cardioactive medications. A witness reported the fall to have been without any apparent warning. The patient was neither pale nor clammy and recovered promptly in less than 30 seconds.

Findings in the ED were unremarkable. A 12-lead ECG was reported to be normal with a narrow QRS and normal QT interval (unavailable). An echocardiogram in the ED was reported as normal.

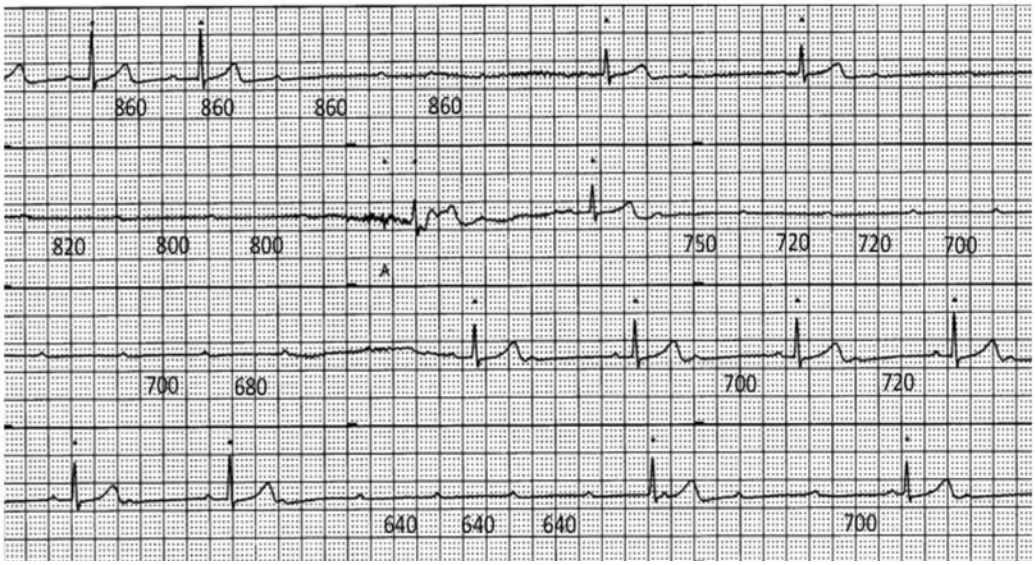

Figure 2.3.1 The tracing shows sinus rhythm with a narrow QRS and an initial PP interval of 860 ms. During subsequent high-grade AV block the PP intervals (indicated in ms) progressively shorten. As the block begins to resolve toward the end of the tracing, the PP interval begins to return toward baseline value.

Discussion

While being monitored in the ED, the recording shown (**Figure 2.3.1**) was obtained. The patient felt lightheaded. The tracing shows sinus rhythm with a narrow QRS, which abruptly transitions to high-grade atrioventricular (AV) block. Note that there is progressive shortening of the PP interval (i.e., speeding of atrial rate) in response to the presumed hemodynamic stress of the conduction disturbance. The latter observation tends to exclude an autonomically-mediated hyper-parasympathetic block.[1,2] A diagnosis of conduction disease was made, despite the normal QRS duration. A pacemaker was implanted and the patient remained symptom free after 4 years of follow-up.

Disclosure

This work was supported in part by a grant from the Dr. Earl E. Bakken Family in support of Heart-Brain research.

References

1. Zysko D, Gajek J, Kozluk E, et al. Electrocardiographic characteristics of atrioventricular block induced by tilt testing. *Europace*. 2009;11(2):225–230.
2. Barold SS, Hayes DL. Second degree atrioventricular block—A reappraisal. *Mayo Clin Proc*. 2001;76(1):44–57.

CASE 2.4

Balaji Krishnan, MD
David G. Benditt, MD

Patient History

A 76-year-old male had been complaining of palpitations and intermittent "dizzy" spells for 6–8 months. On the day that this recording was obtained, he had collapsed while shopping. He suffered a clavicular fracture and was admitted to the emergency department (ED) Observation Unit. He had no prior cardiac evaluation and did not have a previous ECG on record.

During the course of the evening, he complained of his heart beating fast. His wife noticed that he briefly slumped to the side with his eyes open. He recovered promptly. The ECG tracing (**Figure 2.4.1**) was recorded at the time of this in-hospital event.

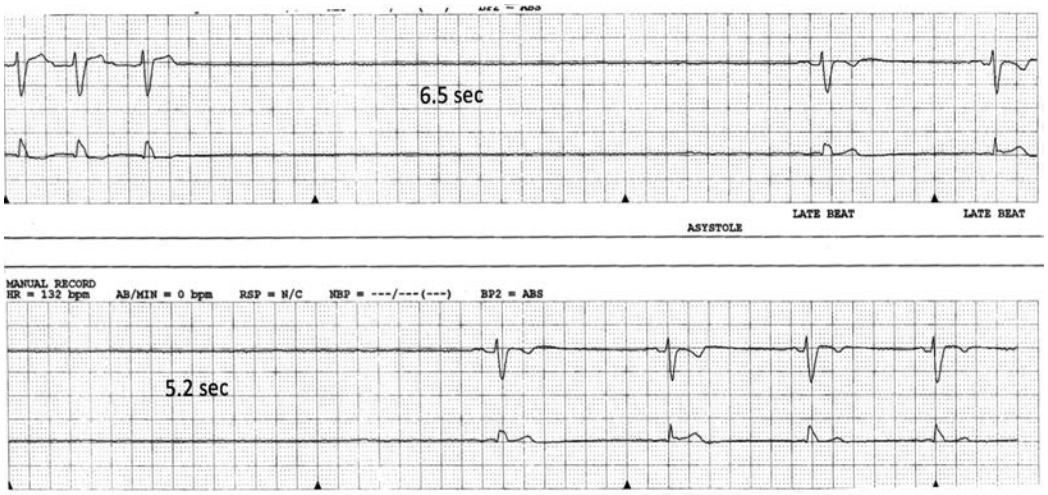

Figure 2.4.1 The 2-channel rhythm strip begins with a relatively slow atrial tachycardia (approx. 95/min), which terminates abruptly, resulting in two sequential cardiac asystolic pauses of 6.5 and 5.2 seconds, respectively. It was in association with these pauses that the patient was symptomatic in the ED. The tracing concludes with sinus bradycardia at approximately 30–50 bpm.

Electrophysiology was consulted and a pacemaker was placed the following morning. Ablation of his atrial tachycardia was discussed, but the patient declined. Sotalol therapy was initiated, and the patient has been without symptomatic rapid heart rate or near-syncope for 3 years.

Discussion

Sinus node dysfunction comprises a range of clinical scenarios. The most common ECG manifestations are sinus bradycardia, sinus pauses, and chronotropic incompetence. In this patient, the combination of atrial tachycardia termination with delayed recovery of sinus node activity resulted in asystolic pauses with sufficient drop in cerebral blood flow to cause near-syncope. Presumably, had the patient been upright as he was while shopping, a frank syncope may have occurred.

Disclosure

This work was supported in part by a grant from the Dr. Earl E. Bakken Family in support of Heart-Brain research.

References

1. Short DS. The syndrome of alternating bradycardia and tachycardia. *Br Heart J*. 1954;16:208–214.
2. Benditt DG, Milstein S, Goldstein MA, et al. Sinus node dysfunction: Pathophysiology, clinical features, evaluation and treatment. In *Cardiac Electrophysiology: From Cell to Bedside*. 2nd ed. Zipes DP, Jalife J, Eds. Philadelphia, PA: WB Saunders Company. 1990:1215–1247.

CASE 2.5

Alan Cheng, MD
Jane E. Crosson, MD

Patient History

A 64-year-old male presented with new-onset exertional dyspnea. The baseline ECG is shown (**Figure 2.5.1**). During stage II of the Bruce protocol, his heart rate increased to 113 bpm (**Figure 2.5.2**) but suddenly dropped to 75 bpm (**Figure 2.5.3**) while exercising.

Question

What is the reason for this change in heart rate?

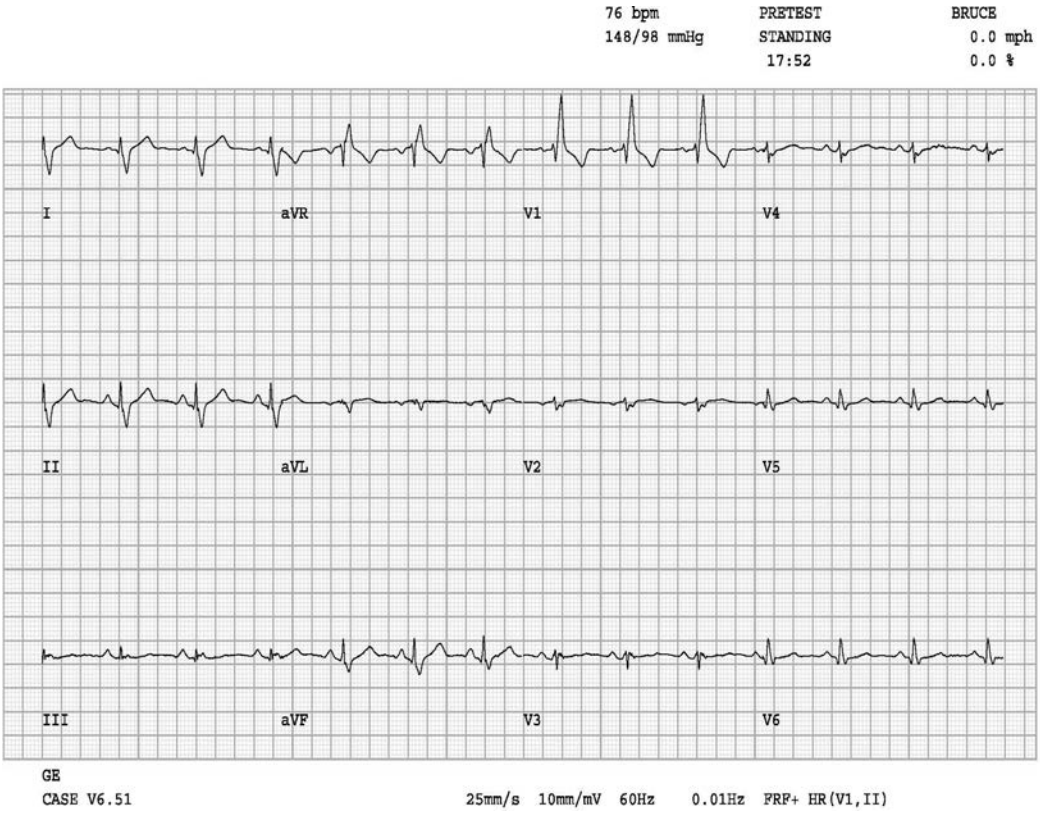

Figure 2.5.1

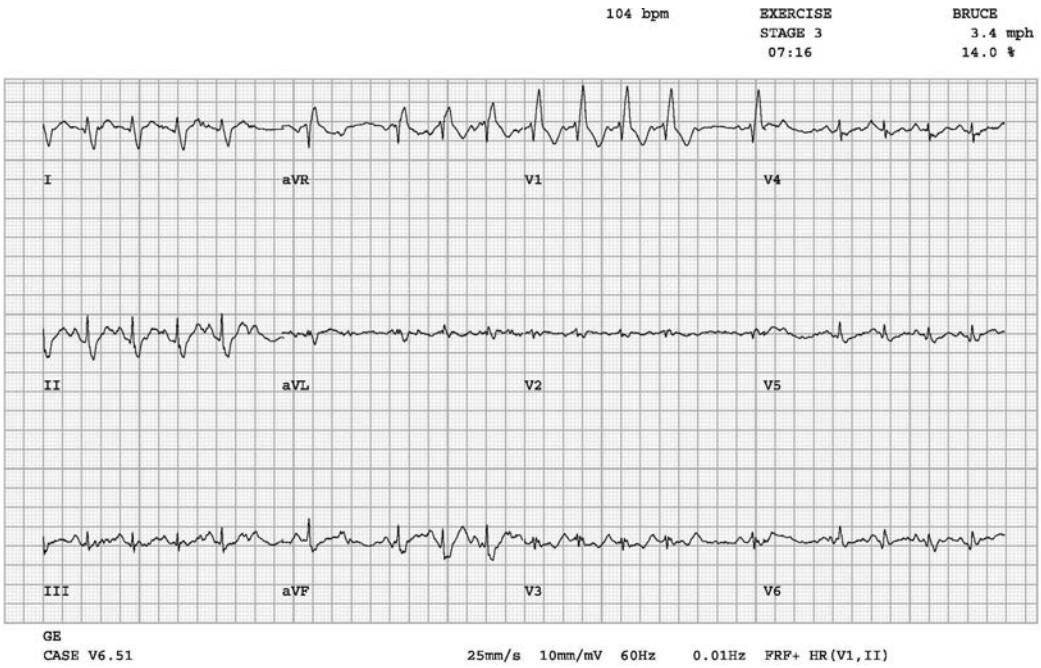

Figure 2.5.2

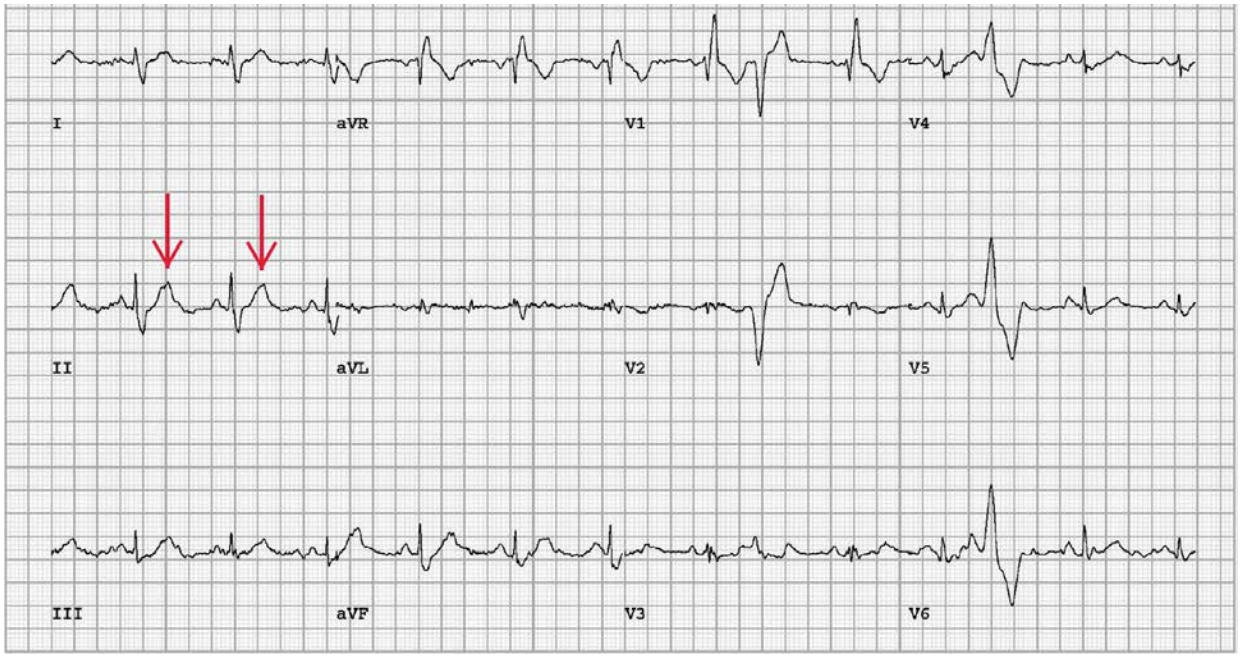

Figure 2.5.3

Discussion

This a case of exercise-induced heart block. In **Figure 2.5.1** at rest, the patient exhibits sinus rhythm with right bundle branch conduction delay/block. As he exercises, there is appropriate augmentation of his sinus rate and atrioventricular (AV) conduction. However, as the heart rate increases with adrenergic stimulation, a 2:1 block (red arrows denoting P waves buried in T waves)

is observed. Exercise-induced heart block suggests disease that is below the AV node, which has a higher risk for progression to complete heart block. With AV nodal disease, AV conduction is expected to improve with catecholamine stimulation and vagal withdrawal in response to exercise. Given the worsening AV conduction exhibited in the setting of increased adrenergic stimulation, this case illustrates an individual who has progressive infranodal conduction system disease and would likely benefit from pacemaker implantation.[1]

Reference

1. Hemann BA, Jezior MR, Atwood JE. Exercise-induced atrioventricular block: a report of 2 cases and review of the literature. *J Cardiopulm Rehabil.* 2006;26(5):314–318.

Sanoj Chacko, MD, PhD
Benedict M. Glover, MD
Adrian Baranchuk, MD

CASE 2.6

Patient History

A 76-year-old male patient with a history of hypertension lasting 10 years was admitted due to a syncopal episode associated with bradycardia. No history of ischemic heart disease. The patient was not receiving any atrioventricular node (AVN) blocking agents. His initial ECG rhythm strip obtained at a different center is shown in **Figure 2.6.1**.

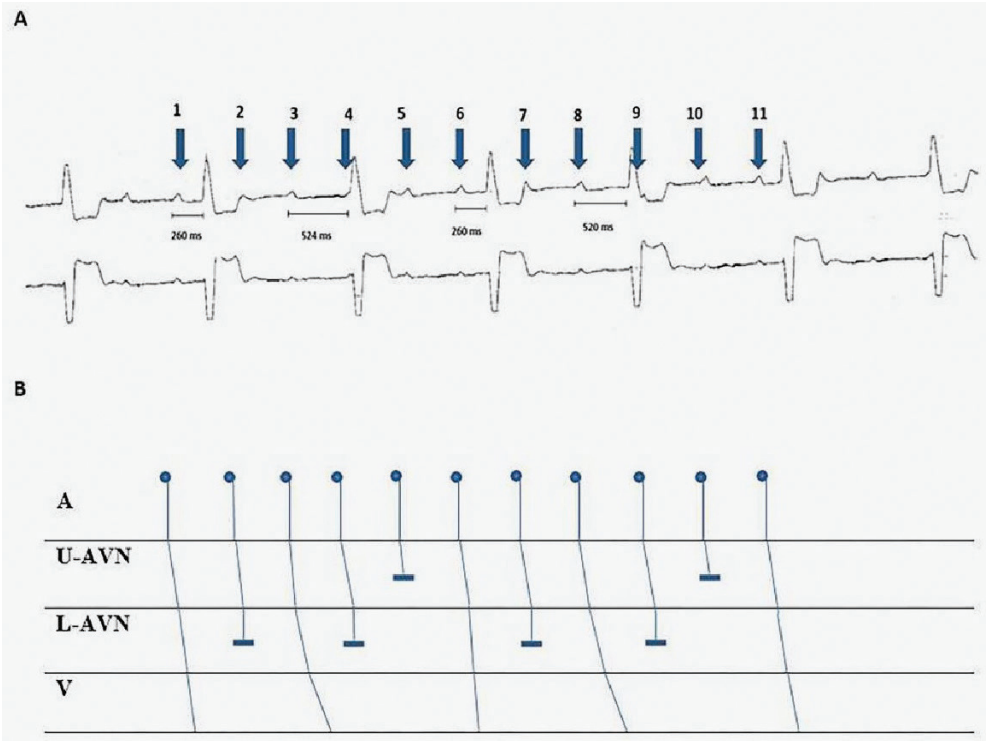

Figure 2.6.1 A. ECG strip showing Wenckebach periods of alternate beats. There is a special relationship between P waves and R waves. The arrows are pointing to the P waves to facilitate analysis. The first P wave conducts with a PR interval of 260 ms, the second P wave is blocked in the lower AV node; the third P wave conducts with a PR interval of 524 ms. The fourth P wave is blocked in the lower AV node (L-AVN) and the fifth P wave is also blocked but in the upper AV node (U-AVN), completing a Wenckebach series. The sixth P wave conducts with a PR interval of 260 ms reinitiating the sequence. See text for details. B. Ladder diagram of the rhythm strip showing the conduction abnormalities and the level of block. A, atrium; U-AVN, Upper atrioventricular node; L-AVN, Lower atrioventricular node; V, Ventricle.

Question

What is the mechanism?

1. Complete heart block
2. Wenckebach heart block
3. Mobitz II AV block
4. Wenckebach periods of alternate beats

Discussion, Interpretation, and Answer

The ECG findings are consistent with two levels of block in the AVN with an atrial rate of 115 bpm and a ventricular rate of 35–40 bpm. The superior aspect of the AVN conducts with Wenckebach phenomenon and the inferior aspect with a Mobitz type-II AVN block. We have inserted arrows and numbered the P waves (**Figure 2.6.1A**) and added a ladder diagram (**Figure 2.6.1B**), to facilitate the analysis. The first P wave conducted to the ventricle with a PR interval of 260 ms (possibly in the superior aspect of the AVN), the second P wave is blocked (possibly in the inferior aspect of the AVN); the third P wave conducted again with a PR interval of 524 ms depicting Wenckebach phenomenon (superior aspect of AVN). The fourth P wave is blocked (inferior aspect of the AVN) and the fifth P wave is blocked in the superior aspect of the AVN completing the Wenckebach series (3:2 in the superior aspect of the AVN). The sixth P wave conducted at a PR interval of 260 ms re-initiating the same pattern. This ECG demonstrates 5:2 AV block with Wenckebach Periods of Alternate Beats (WPAB), a phenomenon that was described as associated with transversal dissociation of the AV node-His. However, as the QRS morphology is wide, Wenckebach in the infrahisian region cannot be completely ruled out. Transverse dissociation of the AVN-His bundle into two horizontal levels "connected in series" usually manifests with 2:1 (or higher) block in the "distal" level and Wenckebach periods in the "proximal" level.[1]

Compared to the classical AVN Wenckebach which shows gradual prolongation of the PR interval followed by a single dropped P wave, WPAB ends with or begins from two consecutive blocked P waves and invariably involves infranodal conduction system.[2] WPAB may be a consequence of a single lesion or two independent lesions, one of which causes classical Wenckebach while the other causes Mobitz II block. In a series of five clinical cases of WPAB, marked prolongation of refractoriness within the intraventricular conduction system was found leading to four of them progressing into complete heart block.[2] One more recent case report of a patient with an acute coronary syndrome presented with WPAB evolving into complete heart block.[3]

WPAB is an uncommon electrocardiographic phenomenon representing advanced nodal and distal conduction disease. These cases usually require permanent pacing. Careful interpretation of the ECG provides information about the underlying electrophysiological mechanism.

References

1. Elencwajg B, Zaman L, Rozanski JJ, Myerburg RJ, Castellanos A. Transverse dissociation of the human His bundle. *Pacing Clin Electrophysiol.* 1982; 5(3):323–328.
2. Halpern MS, Nau GJ, Levi RJ, Elizari MV, Rosenbaum M. Wenckebach periods of alternate beats clinical and experimental observations. *Circulation*. 1973;48:41–49.
3. Sclarovsky S, García-Niebla J. Current role of electrocardiography in acute coronary syndrome. Is it an outdated technique? *Rev Esp Cardiol*. 2009;62:451–463.

Pieter Koopman, MD
Hein Heidbuchel, MD, PhD

CASE 2.7

Patient History

An 80-year-old female is admitted to the emergency department (ED) in a state of severe confusion. She suffered a mild myocardial infarction 10 years before, for which she underwent successful percutaneous coronary intervention. She presents with an asymptomatic irregular heartbeat, despite already being treated with beta-blockers.

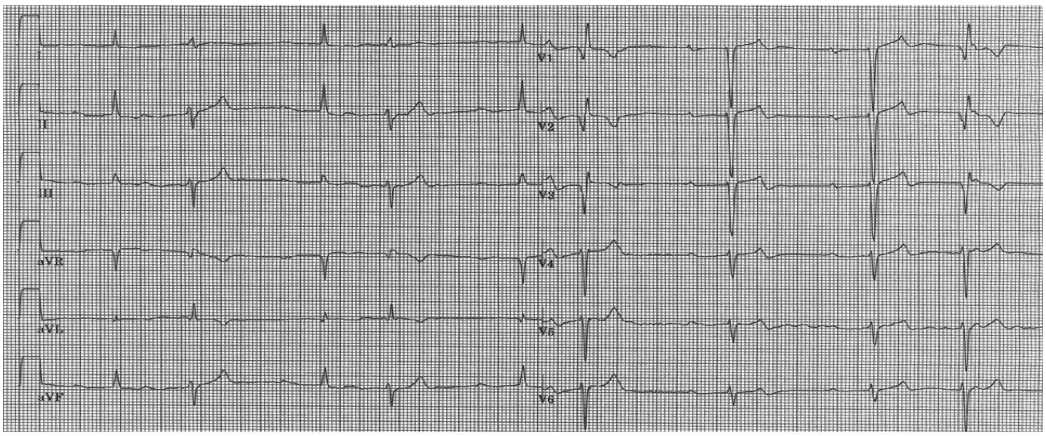

Figure 2.7.1

Questions

1. What is the explanation for the wide QRS complexes (**Figure 2.7.1**)?
2. How should this patient be treated?

Discussion

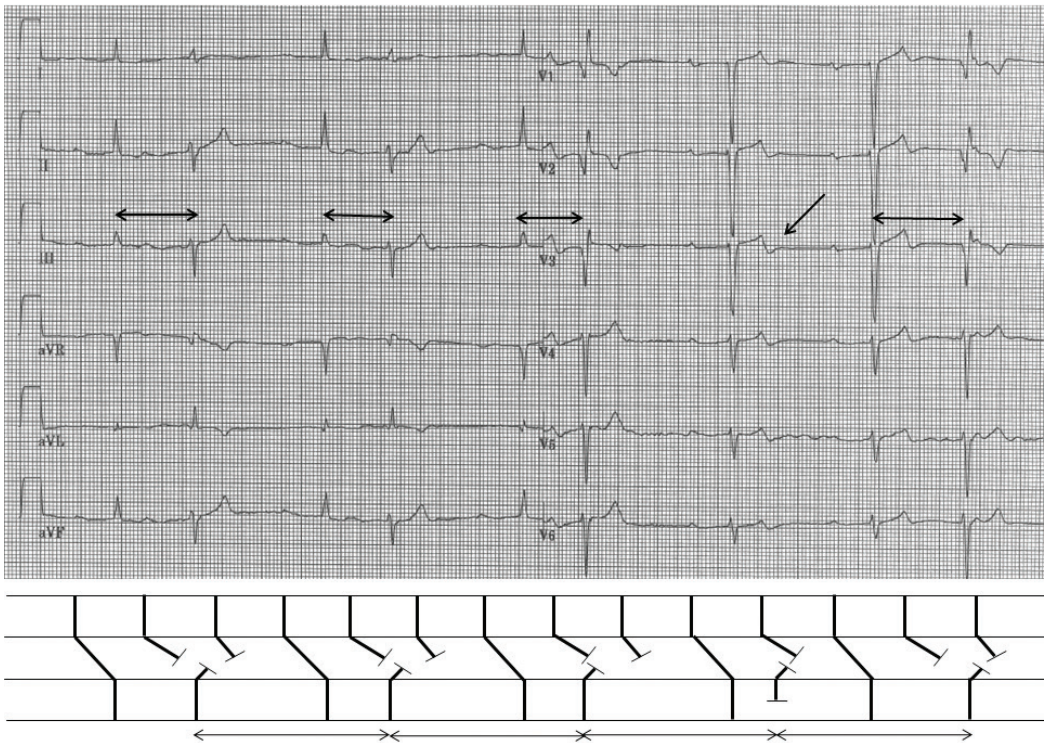

Figure 2.7.2

As the ladder diagram illustrates (**Figure 2.7.2**), the ECG shows a sinus rhythm with second-degree atrioventricular (AV) block. The PR interval is prolonged for the conducting beats, so the AV block is most likely supranodal. Every second P wave is blocked (2:1 A-V conduction), which is evident on the far right side of the tracing. With the exception of the P wave following the black single arrow in the tracing, every third P wave is also blocked. That block is due to ventricular refractoriness following forgoing ventricular extrasystoles. Moreover, the premature beats are not extremely broad, and have a right bundle branch block morphology and left axis, pointing to an origin in the left posterior fascicle. The coupling intervals of these ectopic beats are variable (double arrows), but intervals in between ectopic beats are similar. This behavior is typical for ventricular parasystole due to an automatic focus with entrance block from the ventricle. The parasystolic focus itself was not able to induce a ventricular complex at the timing of the single black arrow, because of ventricular refractoriness due to the prior sinus beat.

Parasystole is caused by a secondary pacemaker site in the heart, firing at a fixed rate independently from the sinus discharge rate. Normally, subsidiary pacemakers are prematurely discharged by impulses from the sinus node, but in the case of parasystole, this independent pacemaker site is "protected" by the presence of "entrance block" (sinus impulses fail to depolarize the latent pacemaker secondary to block in the surrounding tissue). A block must be unidirectional, so impulses from the parasystolic focus can exit. Therefore, parasystolic ectopic beats will wander through the sinus beats with varying coupling intervals, but with fixed inter-ectopic intervals (or sometimes multiples of a common denominator). Features of abnormal automaticity and unidirectional block can be found in Purkinje fibers that survive in regions of transmural

myocardial infarction. Parasystole is more commonly found in cardiac patients than in healthy individuals, but the arrhythmia itself is benign. Parasystole is often refractory to various antiarrhythmic agents and is therefore difficult to treat. As this patient also exhibited a symptomatic second-degree AV block, pacemaker implantation should be considered, especially if the AV block persisted after cessation of beta-blocker therapy.

Reference

1. Courtemanche M, Glass L, Rosengarten MD. Modeling ventricular parasystole. *Ann N Y Acad Sci.* 1990;591:178–189.

CASE 2.8

Nishant Verma, MD, MPH
Bradley P. Knight, MD

Patient History

This ECG (**Figure 2.8.1**) was recorded on a 26-year-old female with Marfan syndrome, complicated by severe aortic insufficiency, dilated cardiomyopathy, and New York Heart Association Class IV heart failure. She had previously undergone orthotopic heart transplantation (OHT). There is a concern for an atrial arrhythmia and need for cardioversion.

Question

What is the diagnosis?

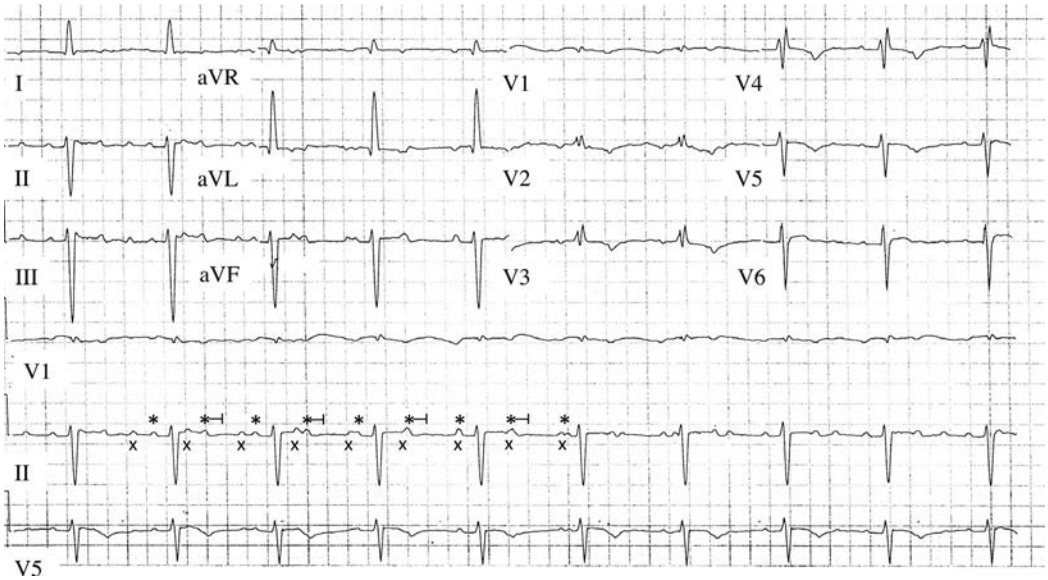

Figure 2.8.1 Rhythm strip in a patient with a recent OHT. For explanation, see discussion.

Discussion

During OHT, the three methods to anastomose the donor and recipient tissue are the classic biatrial (Shumway-Lower) technique,[1] bicaval approach,[2] and total heart transplantation.[3] Currently, the bicaval anastomosis is the most favored method due to anatomic and hemodynamic favorability.[4] Total heart transplantation is technically more difficult,[5] and the biatrial technique has been associated with an increased rate of bradycardia and need for permanent pacemaker.[6] In the bicaval approach, the vena cavae are connected to the donor heart and no native (recipient) right atrial tissue remains. However, with biatrial anastomosis, a significant portion of the native right atrial tissue remains, including the native sinus node. This may often lead to interesting and confusing ECG patterns, especially if the surgical technique that was used is not known during rhythm analysis. Although the bicaval approach is used in most cases, there are still surgeons who perform the classic biatrial approach in some situations.

In this case, the patient underwent biatrial OHT, and the electrophysiology service was consulted due to concern for atrial arrhythmia and need for cardioversion. In fact, two independent atrial rhythms are seen, one from the native heart and the other from the donor heart. The ECG (**Figure 2.8.1**) shows sinus rhythm with 2:1 atrioventricular (AV) block and atrial parasystole mimicking an atrial tachycardia (AT). The donor heart rhythm was determined to be sinus tachycardia at a rate of approximately 115 bpm with 2:1 AV block. The native sinus rhythm had a rate similar to the donor heart; however, it remained dissociated from the rest of the rhythm. Due to persistent AV block, the patient required treatment for her bradycardia rather than a tachycardia, and underwent implantation of a dual-chamber permanent pacemaker.

This patient underwent an OHT via a classic biatrial anastomosis; therefore the native and recipient sinus nodes remained in place. Confusion regarding the ECG rhythm arose due to the existence of two independent atrial rhythms, one from the donor and another from the recipient. The donor atrial rhythm (*) has a rate of approximately 115 bpm and shows 2:1 AV block (|–* denotes blocked beats). The native atrial rhythm (x) is dissociated and continues independently at a similar rate.

References

1. Lower RR and Shumway NE. Studies on orthotopic homotransplantation of the canine heart. *Surg Forum*. 1960;11:18–19.
2. Sarsam MA, Campbell CS, Yonan NA, Deiraniya AK, Rahman AN. An alternative surgical technique in orthotopic cardiac transplantation. *J Card Surg*. 1993;8(3):344–349.
3. Dreyfus G, Jebara V, Mihaileanu S, Carpentier AF. Total orthotopic heart transplantation: An alternative to the standard technique. *Ann Thorac Surg*. 1991;52(5):1181–1184.
4. Jacob S, Sellke F. Is bicaval orthotopic heart transplantation superior to the biatrial technique? *Interact Cardiovasc Thorac Surg*. 2009;9(2):333–342.
5. Schnoor M, Schäfer T, Lühmann D, Sievers HH. Bicaval versus standard technique in orthotopic heart transplantation: A systematic review and meta-analysis. *J Thorac Cardiovasc Surg*. 2007;134(5):1322–1331.
6. Deleuze PH, Benvenuti C, Mazzucotelli JP, et al. Orthotopic cardiac transplantation with direct caval anastomosis: Is it the optimal procedure? *J Thorac Cardiovasc Surg*. 1995;109(4):731–737.

CASE 2.9

Christian Steinberg, MD
Andrew D. Krahn, MD

Patient History

A 65-year-old male patient was evaluated for suspicion of unstable angina. His past medical history was unremarkable and he was on no medication prior to his admission. His initial ECG on arrival showed normal sinus rhythm with a narrow QRS with no significant ST-T changes. While awaiting his coronary angiogram, another ECG with intermittent wide QRS complex tachycardia was recorded (**Figure 2.9.1**). The patient was asymptomatic during the ECG recording.

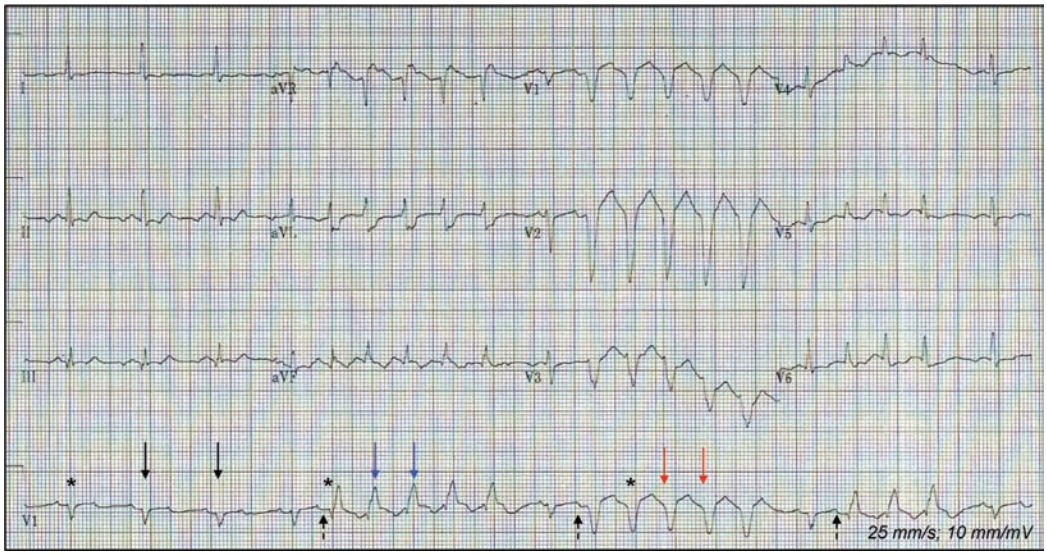

Figure 2.9.1 Atrial tachycardia with alternating bundle-branch block aberrancy. The ECG shows three different types of QRS morphology (*) best seen in the rhythm strip of lead V_1. The baseline QRS complex is narrow and regular (black arrows) at a rate of 81 bpm. There are intermittent regular wide QRS complexes with an alternating typical right bundle branch block (RBBB; blue arrows) and left bundle branch block (LBBB; red arrows) pattern. The rate of the RBBB complexes is 158 bpm, whereas the rate of the LBBB is 150 bpm. At the atrial level there are regular monomorphic P waves at a cycle length of 340 ms (heart rate of 176 bpm). The PR relationship is 2:1 for most of the recording, but there is intermittent 1:1 AV conduction that is associated with an alternating RBBB/LBBB pattern (dashed arrows). The PR relationship and atrial activation pattern suggest an underlying atrial tachycardia or atypical atrial flutter. There is alternating AV-conduction from 2:1 to 1:1 as a result of Wenckebach conduction in the AV node. The wide QRS complexes are the result of alternating bundle branch block aberrancy during 1:1 AV conduction, which explains the typical RBBB and LBBB morphology. The likely mechanisms for the RBBB and LBBB aberrancy include phase 3 block and linking phenomenon (retrograde concealed penetration into the bundle branches).

Interpretation

Atrial tachycardia (AT) with predominant 2:1 AV conduction is present. Intermittent 1:1 AV conduction is associated with alternating bundle branch block (right and left bundle branch block aberrancy). The likely mechanisms of the aberrant conduction include phase 3 block and linking phenomenon.

The P-wave morphology suggests an origin from the superolateral left atrium, possibly from the left superior pulmonary vein.

Discussion

Aberrant conduction is characterized by prolongation of the QRS duration in response to premature supraventricular impulse propagation with functional delay or block through parts of the His-Purkinje system (HPS). A unique feature of the HPS is the variation of the refractory period in response to the preceding cycle length,[1] which is also the basis for aberrant conduction. Four different mechanisms of aberrancy have been described: (1) phase 3 block, (2) acceleration dependent aberrancy, (3) phase 4 block, and (4) aberrancy due to retrograde concealed penetration of an antegrade blocked bundle branch (linking phenomenon).[2–7] Aberrant conduction caused by phase 3 block is also referred to as Ashman's phenomenon and was first described in atrial fibrillation.[2] It is typically observed after abrupt cycle length changes with a long–short pattern.[1,2] In the present case, aberrant conduction is repetitively induced during an intermittent change from 2:1 to 1:1 AV conduction of the underlying atrial tachycardia. Perpetuation of aberrancy and alternating functional bundle branch blocks are typically explained by the linking phenomenon.[3–5]

The understanding of aberrancy is of clinical importance because aberrancy is typically a benign phenomenon that must be differentiated from ventricular arrhythmia. Aberrant conduction can occur in individuals with a healthy HPS and do not reflect the need for a pacemaker.[4]

References

1. Denker S, Shenasa M, Gilbert CJ, et al. Effects of abrupt changes in cycle length on refractoriness of the His-Purkinje system in man. *Circulation.* 1983;67(1):60–68.
2. Gouaux JL, Ashman R. Auricular fibrillation with aberration simulating ventricular paroxysmal tachycardia. *Am Heart J.* 1947;34(3):366–373.
3. Wellens HJ, Durrer D. Supraventricular tachycardia with left aberrant conduction due to retrograde invasion into the left bundle branch. *Circulation.* 1968;38(3):474–479.
4. Stark S, Farshidi A. Mechanism of alternating bundle branch aberrancy with atrial bigeminy: Electrocardiographic-electrophysiologic correlates. *J Am Coll Cardiol.* 1985;5(6):1491–1495.
5. Luzza F, Oreto G, Donato A, et al. Supernormal conduction in the left bundle branch unmasked by the linking phenomenon. *Pacing Clin Electrophysiol.* 1992;15(9):1248–1252.
6. Oreto G, Smeets JL, Rodriguez LM, et al. Supernormal conduction in the left bundle branch. *J Cardiovasc Electrophysiol.* 1994;5(4):345–349.
7. Luzza F, Consolo A, Oreto G. Bundle branch block in alternate beats: The role of supernormal and concealed bundle branch conduction. *Heart Lung.* 1995;24(4):312–314.

CASE 2.10

Yuji Nakazato, MD, PhD

Patient History

A 72-year-old female was admitted with presyncope. She had no specific heart disease except hypertension. She had been taking enalapril (5 mg/day) for 10 years.

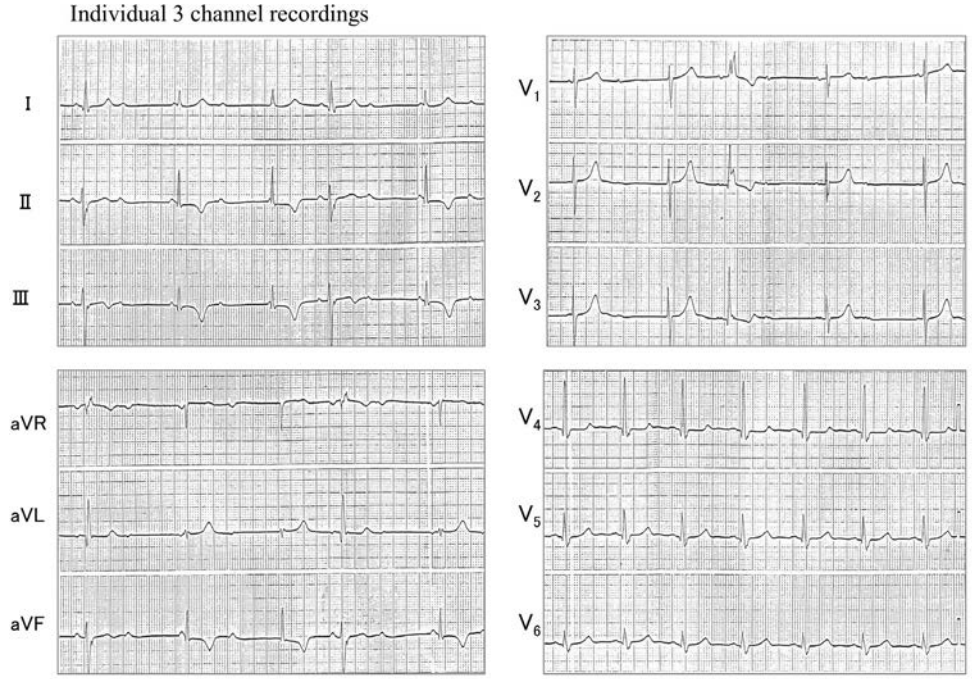

Figure 2.10.1

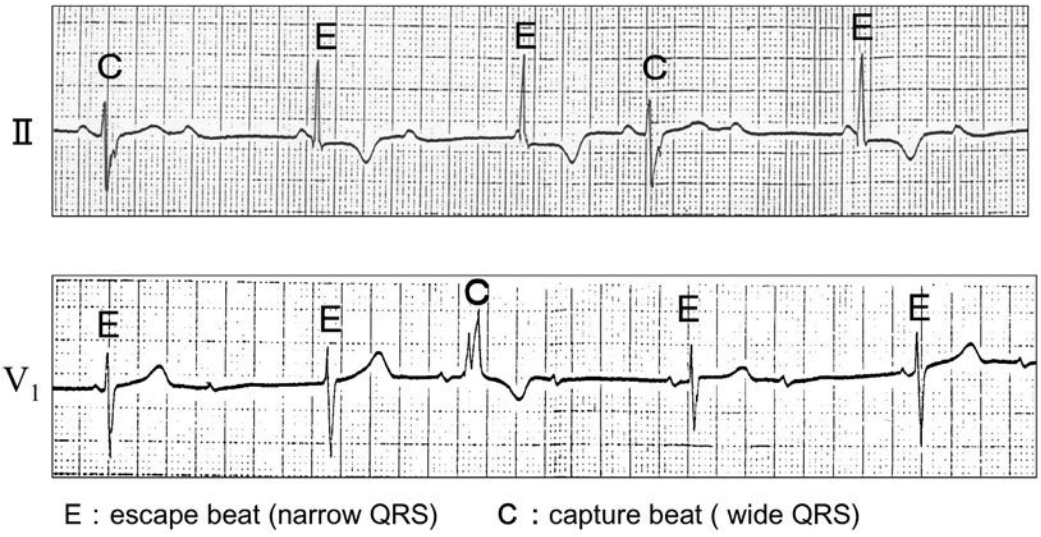

Figure 2.10.2

Questions

1. What is the diagnosis of this ECG?
2. What is the reason for the right bundle branch block (RBBB)?

Discussion

In **Figure 2.10.1**, 3 individual channel ECG strips are displayed. **Figure 2.10.2** clearly demonstrates narrow QRS escape beats (E) and wide QRS (RBBB) capture beats (C). An escape beat is usually generated from just below the blocked site. Therefore, if the blocked site was infrahisian bundle lesion, it should be a wide QRS. However, in this case, the escape beats are narrow QRS. Nevertheless, the captured beats show RBBB.

Question

What is the mechanism of this phenomenon?

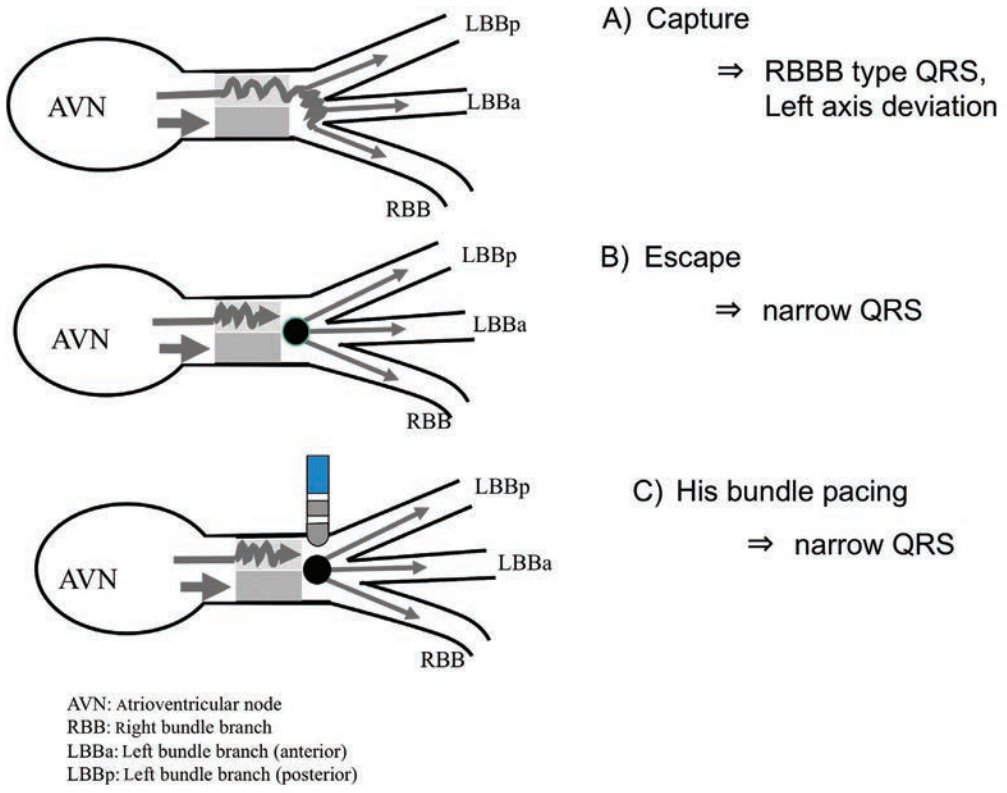

Figure 2.10.3

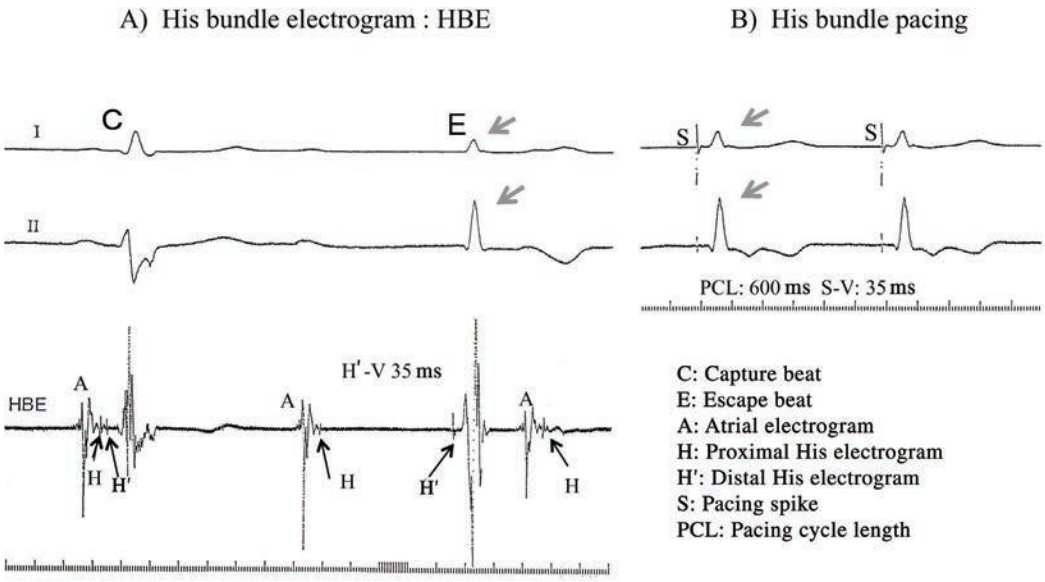

Figure 2.10.4

Discussion

This phenomenon is a longitudinal dissociation of His bundle inducing RBBB. These unusual mechanisms are demonstrated in **Figure 2.10.3**. If there were eccentrically complete conduction disturbances in the His bundle, cardiac impulse would conduct through the incompletely damaged fiber with delay. In addition, there is usually a depressed transverse conduction of the His bundle. Therefore, it causes delayed RBB excitation and results in RBBB pattern QRS configuration on captured beats (**Figure 2.10.3A**). On the other hand, escape beats are narrow QRS (**Figure 2.10.3B**). Thus, bundle branch block on the ECG does not always mean the existence of true bundle branch conduction disturbance. To confirm this phenomenon, His bundle recording was done. Split His potential (H, H′) revealed intra His block and H-V interval (35 ms). The escape beat was identical with spike-V interval (35 ms) by His bundle pacing (**Figures 2.10.3C** and **2.10.4**). It could also prove the RBB conduction is intact.

References

1. El-Sherif N, Amay-Y-Leon F, Schonfield C, et al. Normalization of bundle branch block patterns by distal His bundle pacing. Clinical and experimental evidence of longitudinal dissociation in the pathologic His bundle. *Circulation*. 1978;57:473–483.
2. Narula OS. Longitudinal dissociation in the His bundle. Bundle branch block due to asynchronous conduction within the His bundle in man. *Circulation*. 1977;56:996–1006.

SECTION 3

Miscellaneous Phenomena: Concealed Conduction, Superabnormalities, Aberrancy Conduction, Premature Atrial and Ventricular Contractions (PACs and PVCs)

CASE
3.1

David J. Callans, MD

Patient History

A 46-year-old male with no structural heart disease with palpitations secondary to frequent premature ventricular complexes (PVCs) as shown in **Figure 3.1.1**. An ablation attempt at an outside hospital was unsuccessful and resulted in right bundle branch block (RBBB).

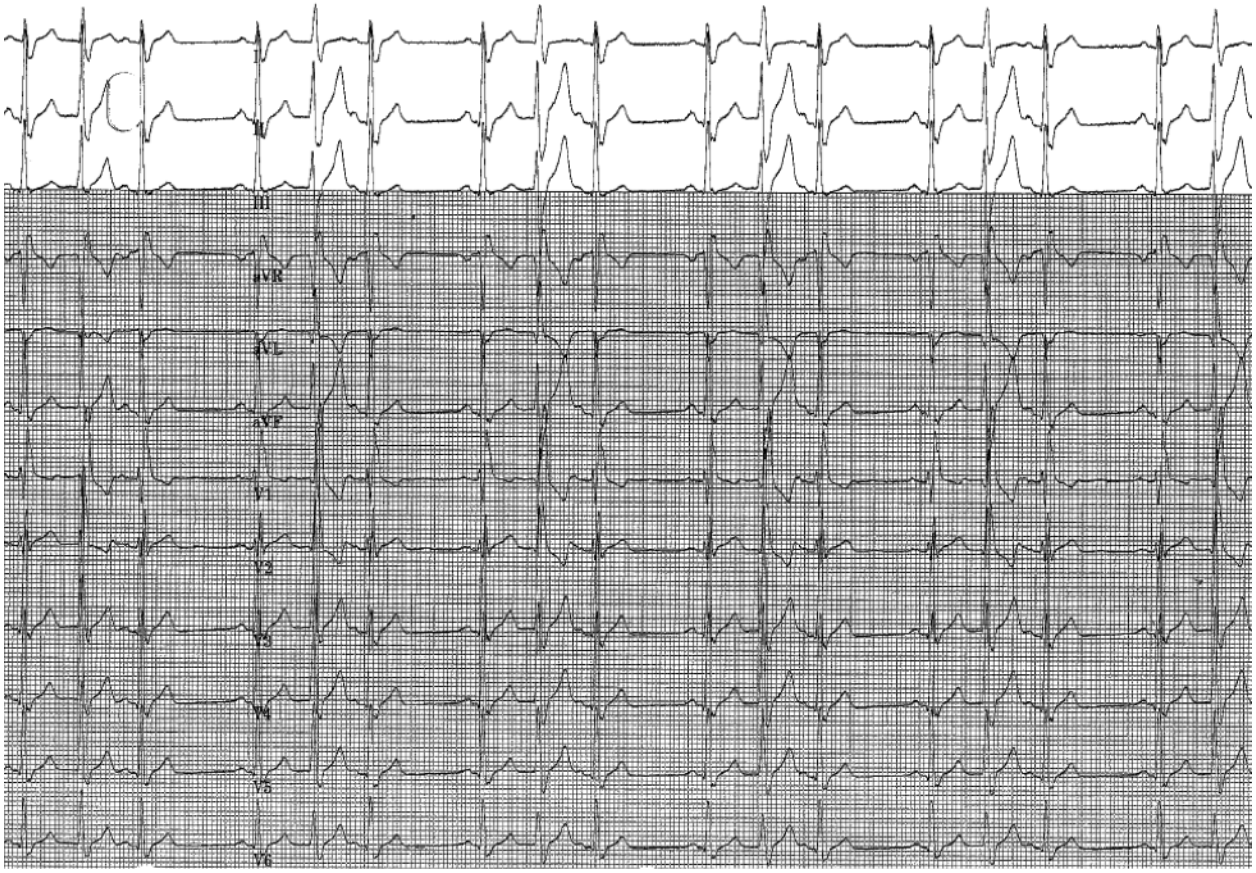

Figure 3.1.1

ECG Masters' Collection: Favorite ECGs from Master Teachers Around the World, Vol. 2 © 2018 Mohammad Shenasa, Mark E. Josephson, N.A. Mark Estes III, Ezra A. Amsterdam, Melvin Scheinman. Cardiotext Publishing, ISBN: 978-1-942909-20-0.

Question

Which of the following sites is the most likely origin of the PVCs?

A. Posterior medial papillary muscle
B. Parahisian location approached from the right ventricle
C. Parahisian location approached from the left ventricle
D. Anterior interventricular vein/great cardiac vein junction

Discussion

The PVC morphology is quite similar to the QRS in conducted sinus rhythm, including being similarly narrow. Despite the RBBB morphology, the PVC was ablated just proximal to the His electrogram recorded in the right ventricle. The parahisian location of the PVC focus conducted with a RBB morphology because of the destruction of the right bundle with the prior ablation attempt.

Reference

1. Ban JE, Chen YL, Park HC, et al. Idiopathic ventricular arrhythmia originating from the para-Hisian area: Prevalence, electrocardiographic and electrophysiological characteristics. *J Arrhythm.* 2014;30:48–54.

Alan Cheng, MD
Jane E. Crosson, MD

CASE 3.2

Patient History

A 69-year-old male presenting for an annual physical was noted to have skipped beats on auscultation. An ECG was performed as shown in **Figure 3.2.1**.

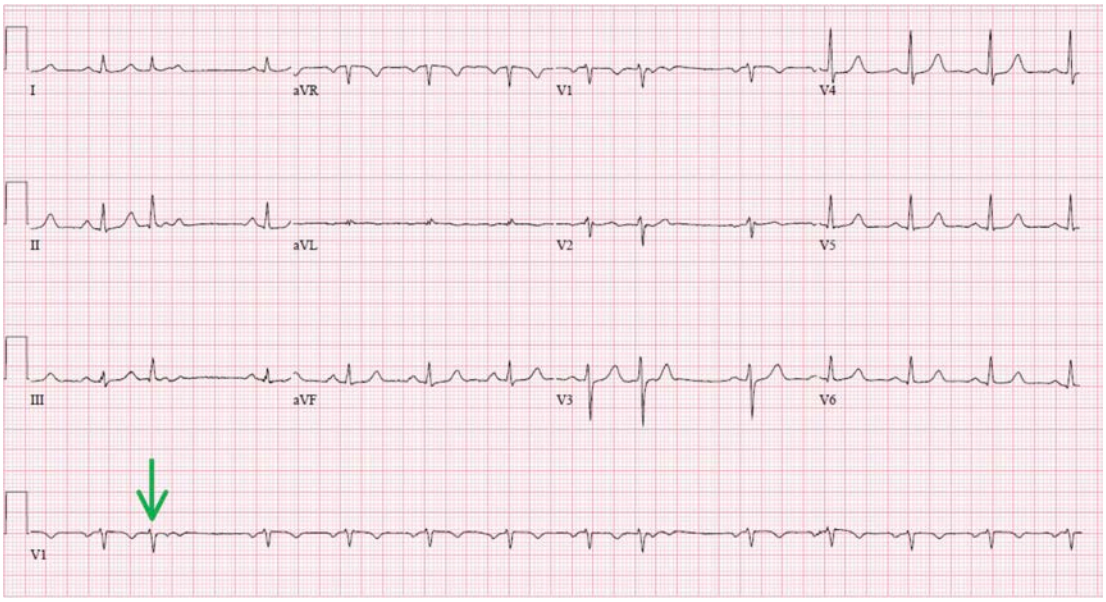

Figure 3.2.1

Discussion

This ECG reveals occasional narrow complex beats that at a first glance appear to be secondary to conducted premature atrial contractions with a compensatory pause. Upon closer inspection, these extra beats are not premature atrial complexes, but rather His extrasystoles.[1] Looking closely at the rhythm strip for lead V_1 at the bottom of the ECG, one can clearly see the P waves preceding most of the QRS complexes. The extra beat (green arrow) does not appear to be preceded by a premature atrial event as the T-wave morphology is identical to other beats that are not followed by a premature QRS. Additionally, the sinus cadence is unperturbed, suggesting no premature atrial event present to reset the sinus cadence.

Reference

1. Marriot HJ, Nizet PM. Main-stem extrasystoles with aberrant ventricular conduction mimicking ventricular extrasystoles. *Am J Cardiol*. 1967;19:755–757.

CASE 3.3

Marc Dubuc, MD
Jason Andrade, BSc, MD

Patient History

A 53-year-old male presented for evaluation after experiencing a long history of intermittent palpitations.

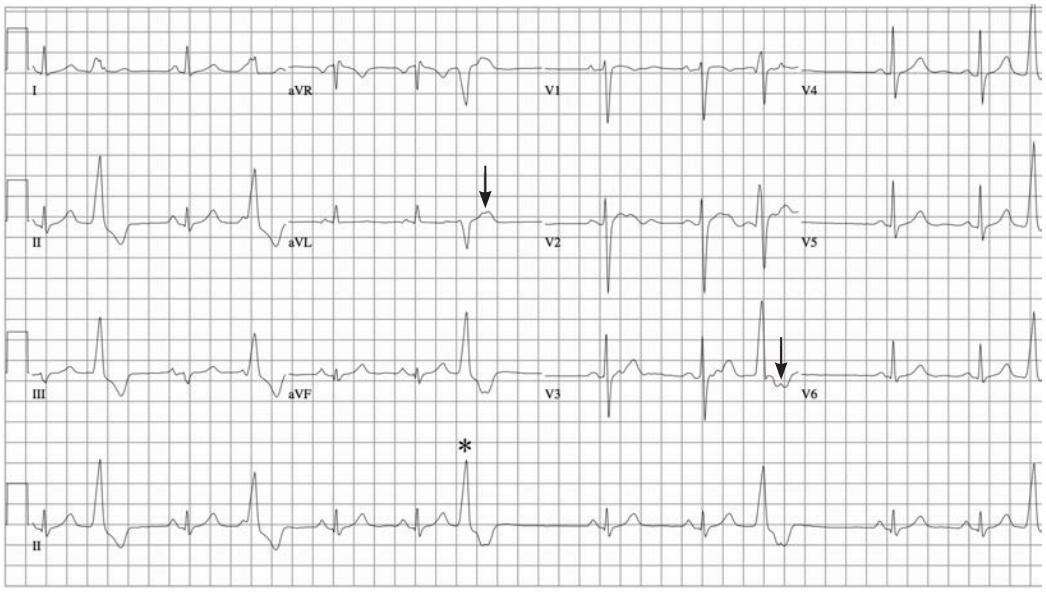

Figure 3.3.1

Question

What abnormality is demonstrated in the figure, and where does this abnormality originate?

Discusssion

This ECG (**Figure 3.3.1**) demonstrates sinus rhythm with frequent monomorphic premature ventricular complexes (PVCs) (*). The PVCs are initially occurring in a bigeminal pattern, and later as ventricular trigeminy. The PVCs are followed by a compensatory pause (retrograde atrial conduction via the atrioventricular node can be seen within the T wave—arrow). Morphologic analysis localizes the PVC to the left ventricular outflow tract region (high to low axis with R wave duration in leads V_1 and V_2 is > 50% of the QRS duration, the V_2 R:S ratio is ≥1, and the PVC QRS transition is earlier than in sinus rhythm).

References

1. Bazan V, Gerstenfeld EP, Garcia FC, et al. Site-specific twelve-lead ECG features to identify an epicardial origin for left ventricular tachycardia in the absence of myocardial infarction. *Heart Rhythm*. 2007;4:1403–1410.
2. Hachiya H, Aonuma K, Yamauchi Y, et al. How to diagnose, locate, and ablate coronary cusp ventricular tachycardia. *J Cardiovasc Electrophysiol*. 2002;13(6):551–556.

N.A. Mark Estes, III, MD

CASE 3.4

Patient History

Figure 3.4.1 shows an ECG of a 40-year-old female with cardiac arrest with sustained monomorphic left bundle branch block (LBBB) morphology and ventricular tachycardia while running.

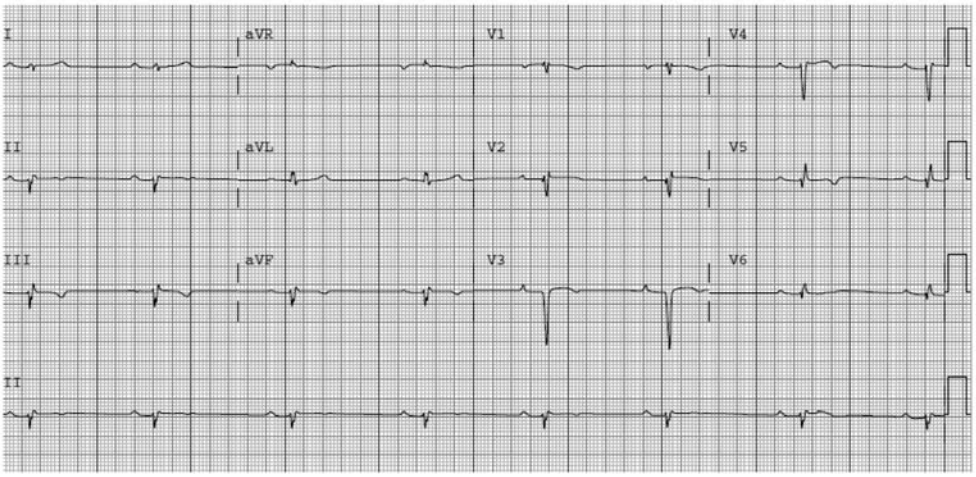

Figure 3.4.1

Question

What is the ECG abnormality?

Discussion

The ECG demonstrates q waves in the inferior and anterior leads with a first-degree atrioventricular block. Cardiac echocardiogram and magnetic resonance imaging with gadolinium were normal, as was an evaluation for pulmonary or systemic sarcoidosis. Despite the extremely abnormal ECG and cardiac arrest, no underlying structural heart disease has been identified. Genetic testing for hypertrophic and dilated cardiomyopathy was negative.

CASE 3.5

Massimo Santini, MD

Patient History

A 55-year-old female patient with medically managed hypertension for 5 years presented to the emergency department (ED) with atypical chest pain for 3 hours. Her ECG is shown in **Figure 3.5.1**.

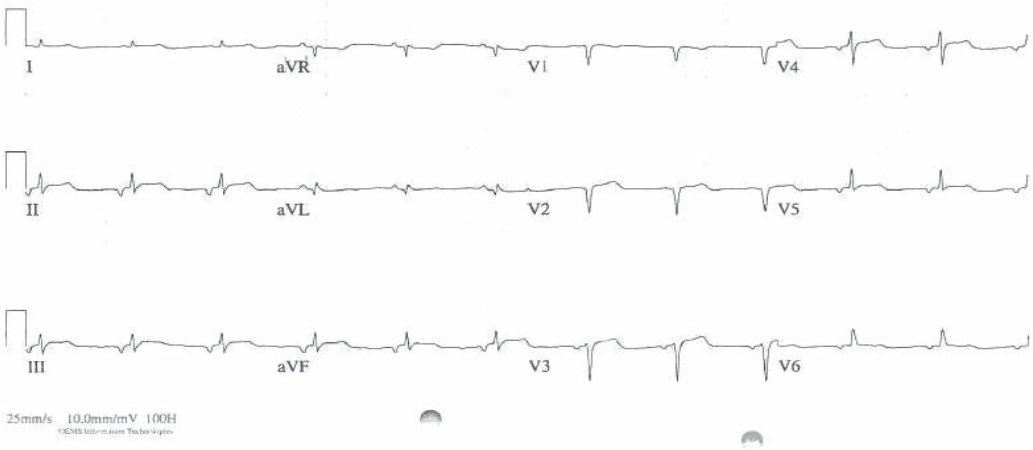

Figure 3.5.1

Question

Is it possible to diagnose the site of ectopic atrial rhythm by ECG analysis?

Discussion, Interpretation, and Answer

The origin of this rhythm is clearly not from the sinus node as the P waves have a different axis from that of the sinus wave; therefore, it appears to be an ectopic atrial rhythm. However, ectopic atrial rhythms may arise from different loci in both atria. The P waves have a different morphology depending on where the ectopic focus is. Here, the PR interval is normal, but the P waves are negative in leads aVF, II, and III as well as leads V_3 to V_6. Moreover, the P wave in lead V_1 is slightly positive and clearly positive in aVL. In this case, the ectopic atrial rhythm is usually called a coronary sinus rhythm or low atrial rhythm since the impulse is assumed to originate from the ostium of the coronary sinus.

SECTION 4

Preexcitation Syndromes

CASE 4.1

Bernard Belhassen, MD

Patient History

Electrophysiologic study was performed in a 40-year-old woman with a history of palpitations. The patient developed a short-lasting, irregular tachyarrhythmia with left bundle branch block (LBBB) complexes during manipulation of a diagnostic electrode catheter in the right atrium (**Figure 4.1.1**).

Question

What is the explanation for the LBBB complexes?

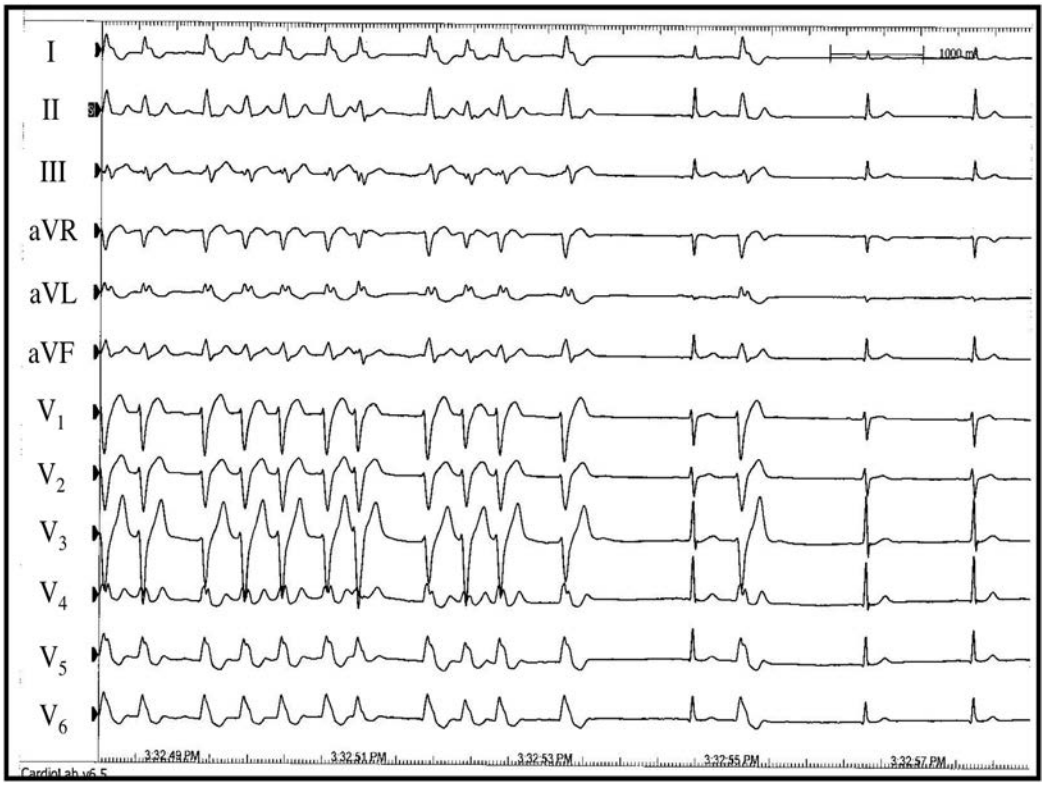

Figure 4.1.1

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Discussion

The tachyarrhythmia is due to an atrial tachyarrhythmia (fibrillation or flutter). All QRS complexes during the tachyarrhythmia have a LBBB pattern. However, various morphologic types of LBBB are present. Taking into account that none of the LBBB complexes have a ventricular origin (such as catheter-induced for example), there are three possible diagnoses for explaining the LBBB complexes: (1) classical LBBB; (2) right ventricular preexcitation due to an anterolateral atrioventricular accessory pathway; or (3) antegrade conduction over an atriofascicular or nodo-ventricular pathway. The actual diagnosis ascertained by the electrophysiology study was antegrade conduction over a right anterolateral atriofascicular Mahaim fiber that was subsequently successfully ablated with radiofrequency at the tricuspid annulus. The various morphologic patterns of LBBB present during tachyarrhythmia suggest the existence of an antegrade preexcitation, rather than classical LBBB. However, the prolonged PR interval associated with the lack of preexcitation during sinus rhythm after termination of the AF makes the possibility of antegrade conduction over an anterolateral atrioventricular accessory pathway very unlikely.

Alan Cheng, MD
Jane E. Crosson, MD

CASE
4.2

Patient History

A 22-year-old man with double inlet left ventricle, intermittent episodes of ventricular preexcitation, and complaints of palpitations underwent 24-hour Holter monitoring.

A baseline ECG (**Figure 4.2.1**) revealed sinus rhythm with first-degree atrioventricular (AV) delay. This tracing was recorded while the patient was asleep. Sinus rhythm was present with first-degree AV conduction delay and no evidence of ventricular preexcitation. By the fourth sinus beat (green arrow), there is evidence of slowing and subsequent conduction block to the ventricle, presumably through a transient increase in vagal tone. AV conduction resumes with the next sinus beat (red arrow), but conduction to the ventricle is now seen to occur predominantly through an accessory pathway.

Question

What is accounting for this change in AV conduction?

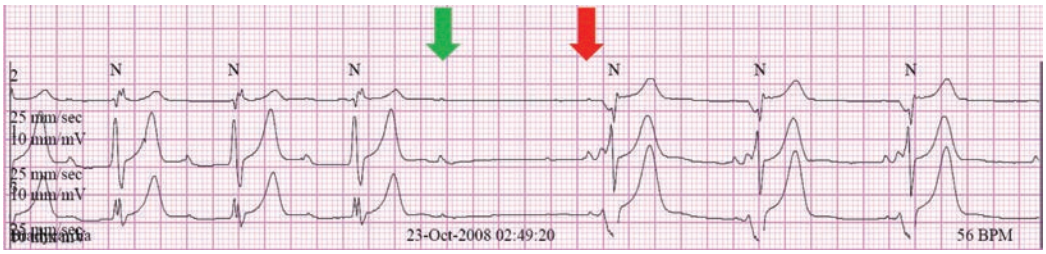

Figure 4.2.1

Discussion

Double inlet left ventricle is one of the most common forms of univentricular heart syndromes, the others being double inlet right ventricle, single ventricle heterotaxy, and unbalanced common atrioventricular canal defects.[1] These patients have a high predilection for heart block[2] and development of sustained atrial arrhythmias, the latter most commonly after palliative surgery.

Figure 4.2.1 illustrates a classic example of the *linking* phenomenon. During the initial portion of the tracing, sinus conduction occurs across the AV node. As the ventricle is depolarized, ventricular depolarization results in retrograde penetration of the accessory pathway, thereby resulting in antegrade block for the next sinus beat. Antegrade conduction across the accessory pathway is blocked and this is perpetuated with subsequent sinus beats. With increasing vagal tone and block across the AV node (green arrow), there is no subsequent retrograde penetration of the accessory pathway. Hence, by the time the next sinus beat is delivered (red arrow), the accessory

pathway has recovered and is now able to support antegrade conduction. As antegrade conduction across the accessory pathway continues, retrograde penetration of the AV node occurs and perpetuates AV node conduction block and persistence of ventricular preexcitation (i.e., linking).

References

1. Khairy P, Poirier N, Mercier LA. Congenital heart disease for the adult cardiologist. *Circulation*. 2007;115:800–812.
2. Davachi F, Moller JH. The electrocardiogram and vectorcardiogram in single ventricle: Anatomic correlations. *Am J Cardiol*. 2969;23:19–31.

CASE
4.3

Robert Frank, MD

Patient History

A 30-year-old female was hospitalized after an episode of abrupt syncope, followed by a long post-syncopal period of dizziness. She had no significant past medical history, a normal clinical examination and cardiac echocardiography, but a wide QRS (**Figure 4.3.1**).

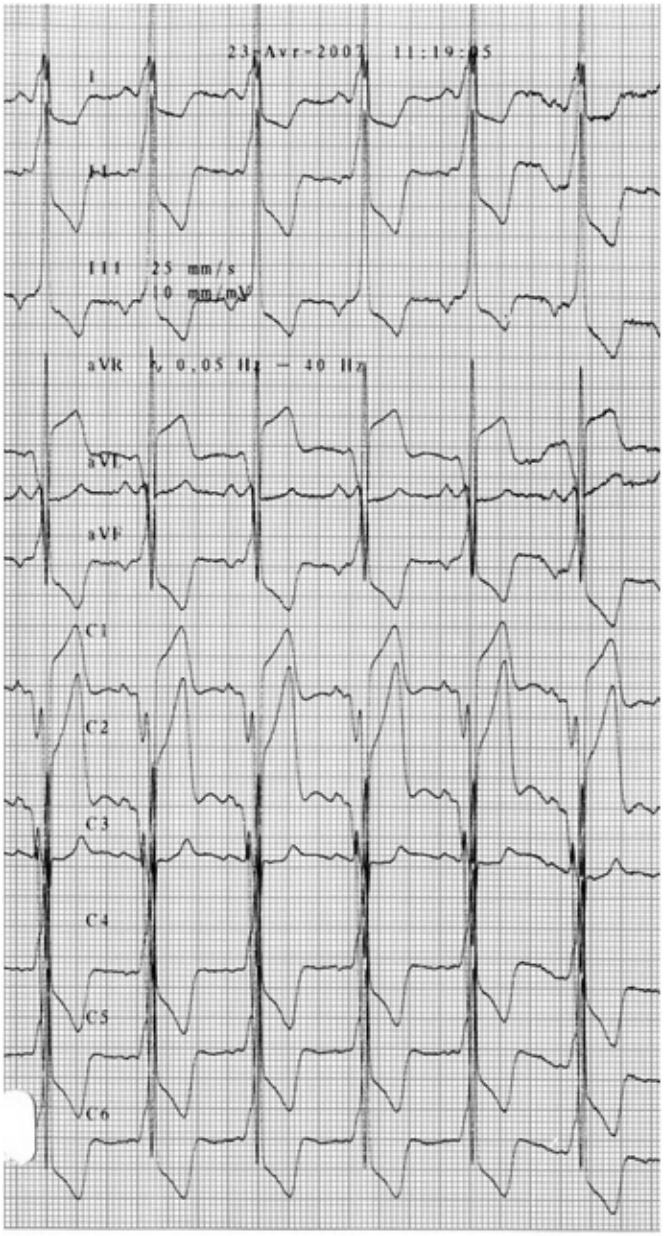

Figure 4.3.1 First ECG in sinus rhythm. C_1 to C_6 is the same as V_1 to V_6.

Question

What is the diagnosis?

Answer

A short PR interval with wide QRS with a slow onset (called delta wave) is characteristic of Wolff–Parkinson–White (WPW) syndrome (ventricular preexcitation).

Question

Where is the site of preexcitation?

Answer

A wide, preexcited negative QRS in leads V_1 and V_2 is always right sided, and an inferior frontal axis ≥60° reflects an anteroseptal localization. A preexcited QRS positive in leads V_1 and V_2 is left sided, and a preexcited QRS negative in lead V_1 and positive in lead V_2 is in a septal location.

A transesophageal incremental atrial pacing study was performed and the shortest 1/1 conducted atrioventricular cycle was of 300 ms (**Figure 4.3.2**).

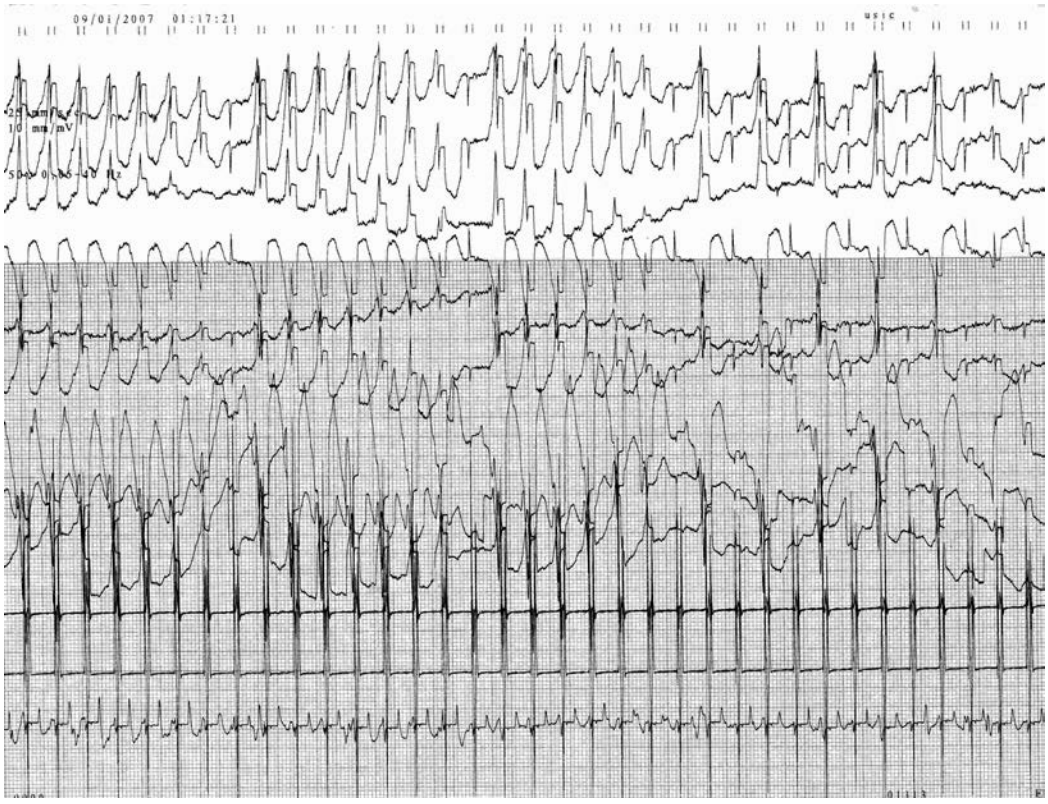

Figure 4.3.2 Incremental transoesophageal atrial pacing just above the shortest 300 ms 1/1 conducted cycle.

Question

What is the most likely cause of syncope in this case?

Answer

Tachyarryhmias with very short ventricular cycles are the most probable mechanism in syncopal WPW patients. However, this is encountered with ventricular cycles below 250 ms (mostly below 200 ms). Therefore, the potential mechanism of tachyarrhythmias with short ventricular cycles did not apply to this case as the accessory pathway was blocked below 300 ms. An electroencephalogram suggested some kind of epilepsy. However, she had a new syncopal episode, which was recorded. The surprise was a complete atrioventricular (AV) block, and a pacemaker was implanted (**Figure 4.3.3**).

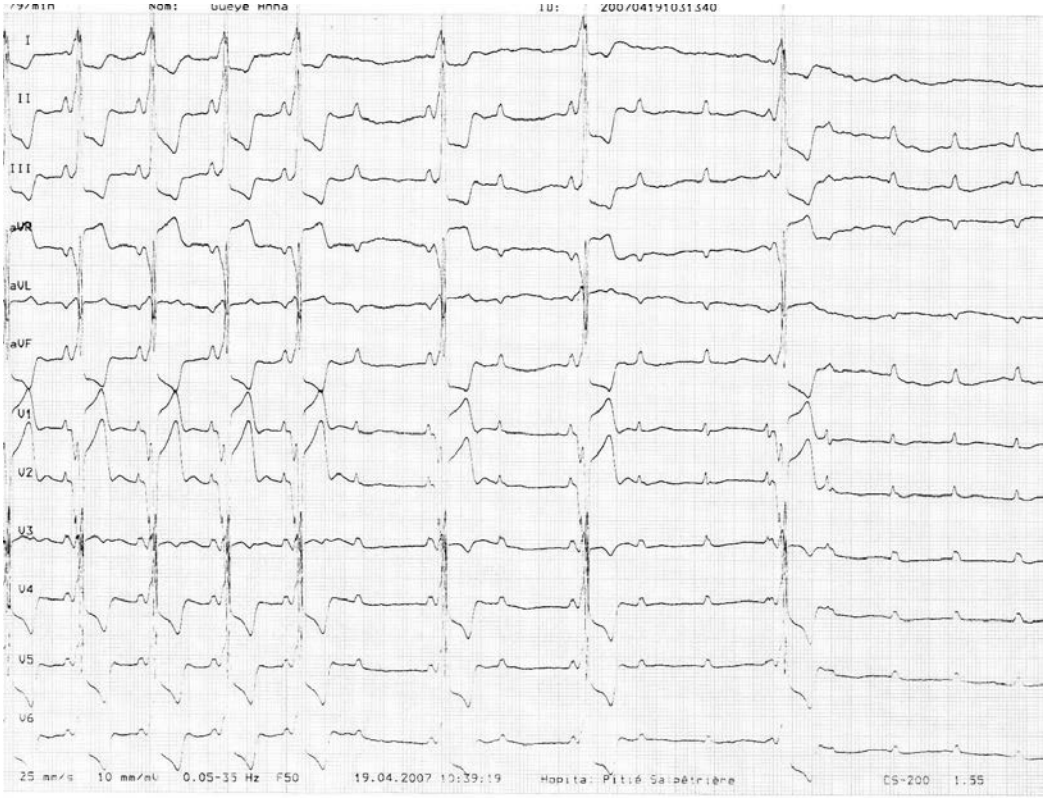

Figure 4.3.3 A few weeks later after the most recent syncopal episode with sudden Mobitz type 2, third-degree AV block.

Discussion

In some patients with preexcited ECGs, the Kent Bundle may be the only conduction pathway. The risk of inducing a complete AV block must be taken in account in patients before the decision to ablate an accessory pathway in a patient without reentrant tachycardia and no conduction through the AV node when the accessory pathway is blocked. Occurrence of high grade AV block in a young person brings up the possibility of an inherited genetic disorder. In this case, a PRAKG2 mutation must be excluded.

References

1. Seipel L, Both A, Breithardt G, et al. His bundle recordings in a case of complete atrioventricular block combined with pre-excitation syndrome. *Am Heart J*. 1976;92(5):623–629.
2. Dinckal MH, Davutoglu V, Bayata S, et al. Masked complete atrioventricular block in a patient with ventricular preexcitation. *J Interv Card Electrophysiol*. 2004;11(1):33–35.

CASE 4.4

Henry H. Hsia, MD

Patient History

A 34-year-old female with paroxysmal symptomatic palpitations. An echocardiogram demonstrated apical displacement of the tricuspid valve with tethering of the septal leaflet, suggestive of Ebstein's anomaly. No evidence of structural heart disease or other valvular pathology was noted. Despite antiarrhythmic therapy, her symptoms persisted with severe dizziness and near syncope.

Question

What is the diagnosis?

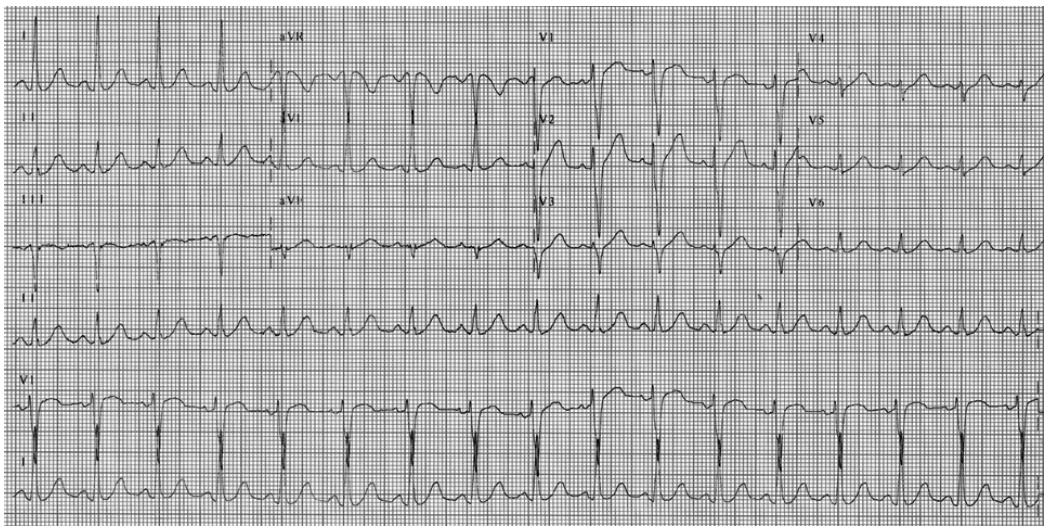

Figure 4.4.1

The 12-lead ECG showed a sinus rhythm with normal intervals and a leftward axis (**Figure 4.4.1**). There is no evidence of ventricular preexcitation, premature atrial or ventricular ectopy, or repolarization abnormalities. A 12-lead ECG obtained during symptomatic palpitations showed a wide QRS complex tachycardia at ~185 bpm (**Figure 4.4.2**).

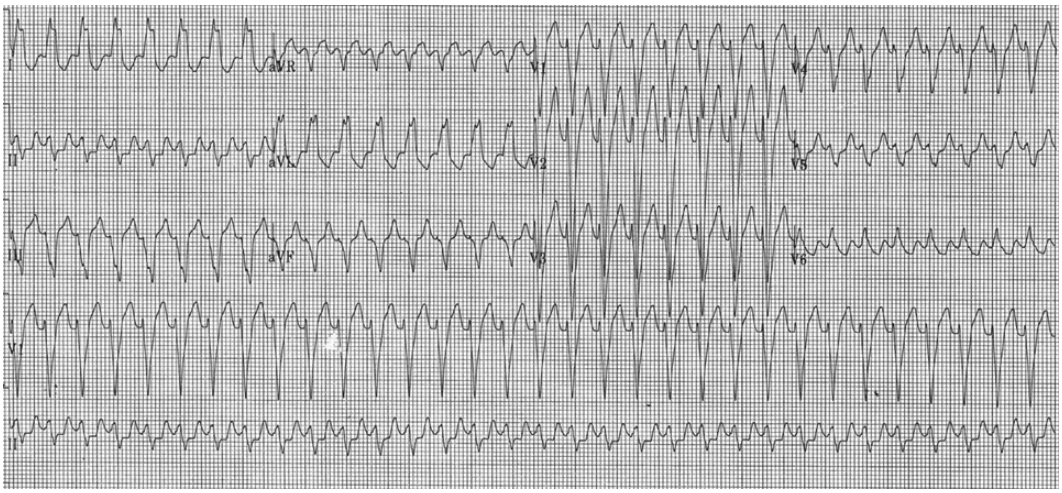

Figure 4.4.2

Discussion and Interpretation

The wide complex tachycardia had a left bundle branch block (LBBB)-like QRS pattern, with a left-superior frontal axis and a late precordial transition (between leads V_5 and V_6) (**Figure 4.4.2**). The presence of a sharp septal R wave with a relatively rapid QRS onset suggests rapid ventricular activations, perhaps utilizing the His-Purkinje system. This raises the possibility of a supraventricular tachycardia (SVT) with aberrant conduction, in the absence of scar-based myocardial substrate.

Although there was no evidence of ventricular preexcitation during baseline sinus rhythm with normal intervals, a characteristic "rS" pattern was noted at lead III (**Figure 4.4.1**). This raises the suspicion of an atriofascicular tract with decremental antegrade conduction property. Sternick, et al found an incidence of "rS" pattern in lead III in 60% of their cohorts with atriofascicular tract and tachycardia, compared to only 6% in matched controls.

The QRS pattern during the wide complex tachycardia is also consistent with the diagnosis of an atriofascicular tachycardia with a LBBB/left superior QRS axis and poor R-wave progression in the precordial leads. The electrocardiographic clues suggesting an antidromic tachycardia using a atriofascicular tract include (1) LBBB QRS morphology tachycardia, (2) QRS axis of 0° to –75° (left axis deviation), (3) QRS duration less than 150 ms, and (4) late precordial transition after lead V_4.

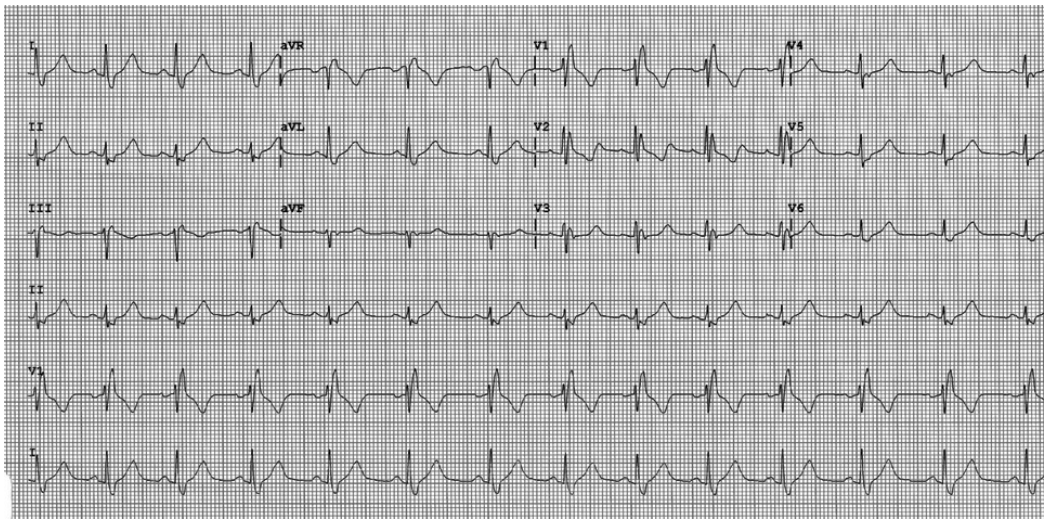

Figure 4.4.3. Post-ablation ECG.

The patient underwent a successful catheter ablation of a right atriofascicular tract. The post-ablation ECG showed a right bundle branch block (RBBB) pattern in sinus rhythm (**Figure 4.4.3**). The presence of RBBB is consistent with the patient's underlying diagnosis of Ebstein's anomaly and right ventricular conduction delay. The presence of a right-side antegrade conducting accessory pathway (atriofascicular) preexcited the right ventricle and masked the underlying RBBB.

References

1. Sternick EB, Timmermans C, Sosa E, et. al. The electrocardiogram during sinus rhythm and tachycardia in patients with Mahaim fibers: The importance of an "rS" pattern in lead III. *J Am Coll Cardiol.* 2004;44:1626–1635.
2. Bardy GH, Fedor JM, German LD, Packer DL, Gallagher JJ. Surface electrocardiographic clues suggesting presence of a nodofascicular Mahaim fiber. *J Am Coll Cardiol*. 1984;3:1161–1168.

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CASE 4.5

Patient History

A 64-year-old woman admitted to the emergency department (ED) with palpitations and dizziness for 2 hours. Her ECG on admittance is shown below (**Figure 4.5.1**).

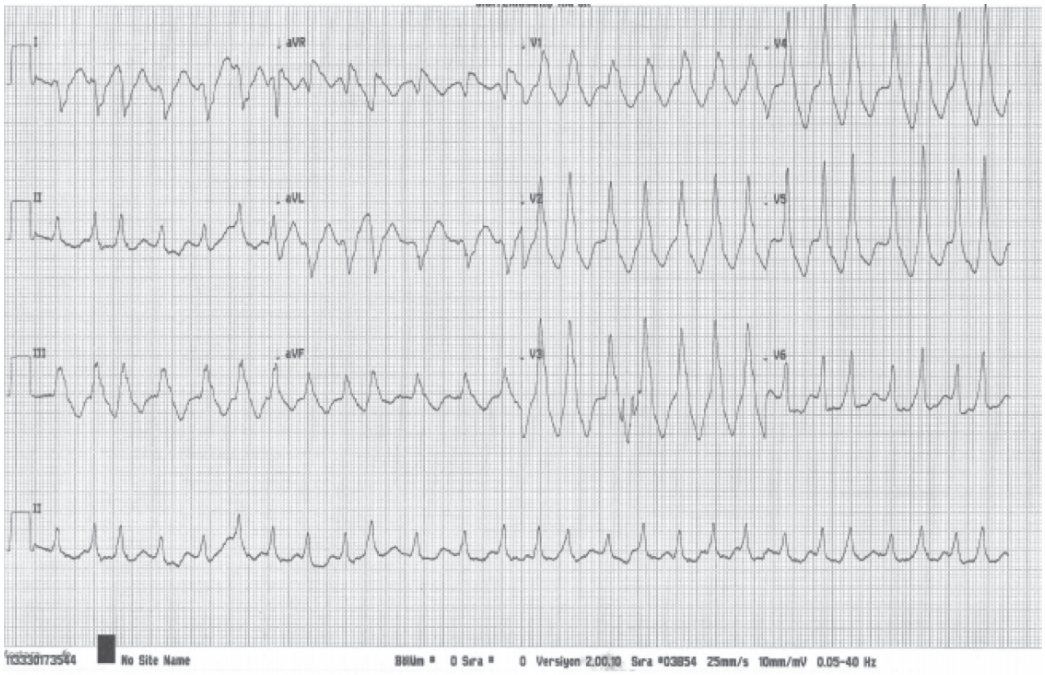

Figure 4.5.1

Question

What is the diagnosis of this ECG?

Discussion

Despite antiarrhythmic medication, sinus rhythm could not be achieved. She was converted to sinus rhythm by DC cardioversion. There was short PR interval and (+) delta waves on inferior leads (**Figure 4.5.2**, arrows). An electrophysiologic study revealed left lateral accessory pathway and tachycardia was diagnosed as preexcited atrial fibrillation. ECGs (**Figure 4.5.2**) before (A) and after ablation (B).

Features of preexcited atrial fibrillation include tachycardia (usually greater than 200 bpm), an irregular rhythm, absence of P waves and a wide QRS due to abnormal ventricular depolarization via accessory pathway.[1]

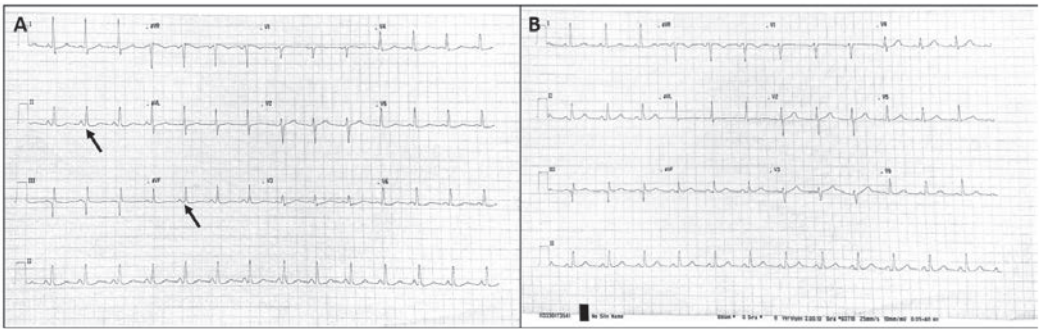

Figure 4.5.2

Reference

1. Page RL, Joglar JA, Caldwell MA, et al. 2015 ACC/AHA/HRS Guideline for the Management of Adult Patients With Supraventricular Tachycardia: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. *J Am Coll Cardiol*. 2016;67(13):e27–e115.

Philip Podrid, MD

CASE 4.6

Patient History

A 35-year-old male with a history of Wolff–Parkinson–White (WPW) undergoes an ablation because of documented atrioventricular reentrant tachycardia (AVRT). His preablation ECG is shown (**Figure 4.6.1**). Two years after the ablation he complains of recurrent palpitations. An ECG is obtained (**Figure 4.6.2**). Several weeks later, he presents to the emergency department (ED) because of an episode of palpitations (**Figure 4.6.3**).

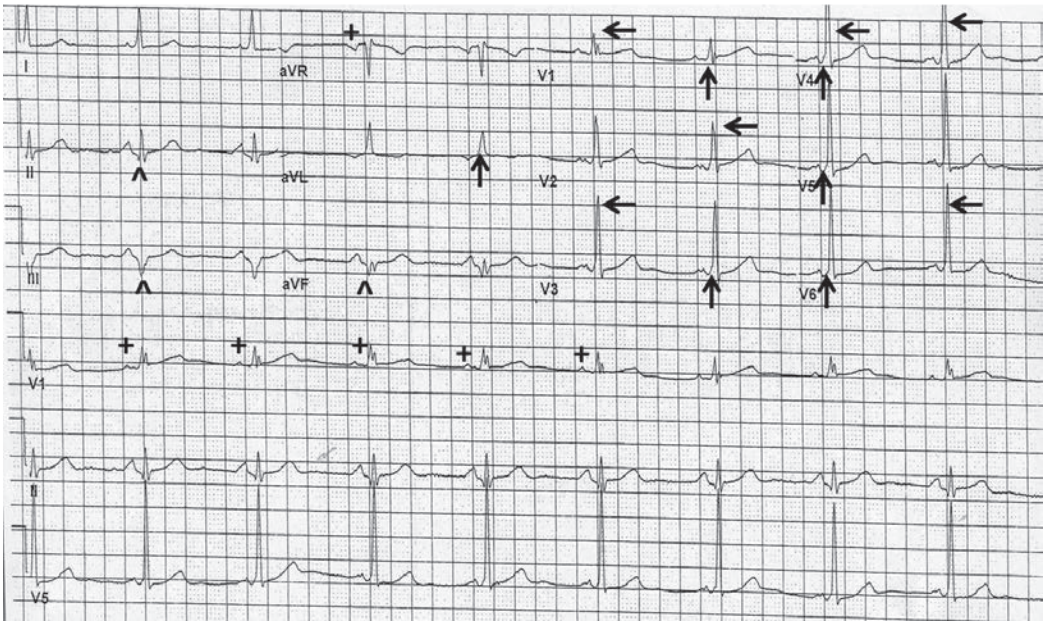

Figure 4.6.1

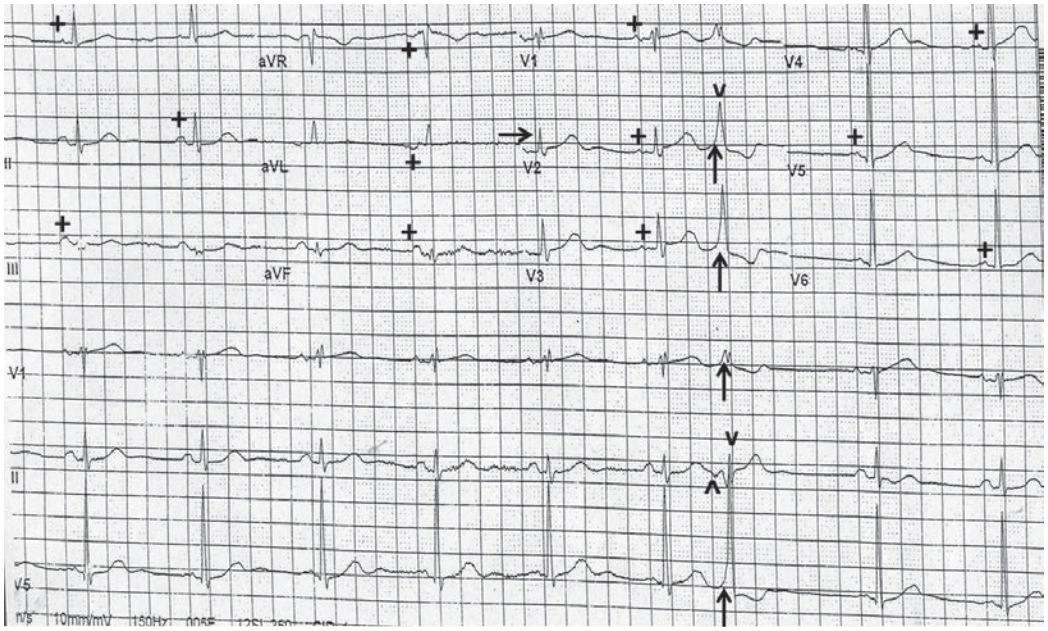

Figure 4.6.2

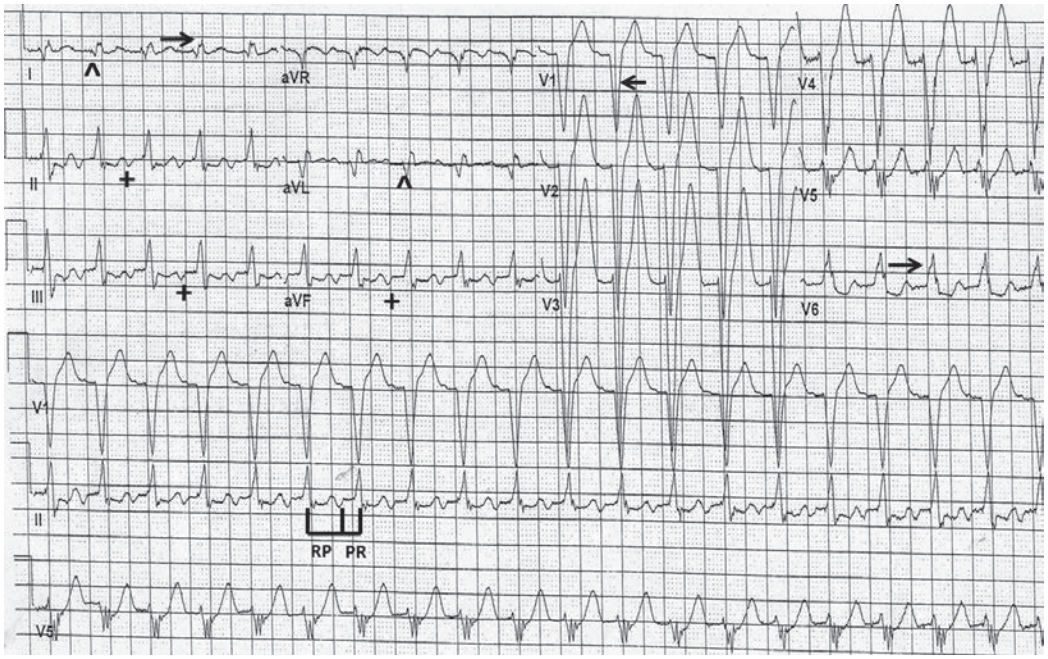

Figure 4.6.3

Questions

1. **Figure 4.6.1** shows a WPW pattern. What is the location of the accessory pathway?
2. What does **Figure 4.6.2** indicate?
3. What is the etiology for the tachycardia in **Figure 4.6.3**?

Diagnosis

Figure 4.6.1 shows sinus bradycardia, left atrial abnormality, WPW pattern (left-sided posteroseptal bypass tract).

Figure 4.6.2 shows sinus bradycardia, left atrial abnormality, premature atrial complex with preexcitation.

Figure 4.6.3 shows long RP tachycardia, orthodromic AVRT with rate-related intraventricular conduction delay.

Discussion

In **Figure 4.6.1**, there is a regular rhythm with a rate of 52 bpm. There is a P wave before each QRS complex (+) with a constant PR interval (0.16 seconds). The P wave is positive in leads I, II, aVF, and V_4–V_6. This is a sinus bradycardia. The P wave is broad and prominently notched in several leads, consistent with a left atrial abnormality or left atrial hypertrophy. While the PR interval is normal, there is no PR segment. As the PR interval includes the P wave as well as the PR segment, the normal PR interval in this patient is due to the broad P wave. The short/absent PR interval means that there is enhanced AV conduction. The QRS complex duration at the base is prolonged (0.12 seconds) as a result of a slurred upstroke (↑); this is a delta wave. The peak of the QRS complex is narrow. The presence of a short PR segment and a delta wave is diagnostic of a WPW pattern. The WPW complex is the result of fusion, with early and direct myocardial activation via the accessory pathway (i.e., preexcitation), which then fuses with impulse conduction via the normal AV node and His-Purkinje system. This accounts for the wide base of the QRS complex and the narrow peak of the QRS complex. The delta wave is positive in lead V_1, meaning that the initial impulse originates in the left ventricle and is directed toward the right ventricle. There are Q waves in leads II, III, and aVF (^), which is consistent with a posteroseptal bypass tract. The Q waves are termed a pseudo inferior wall myocardial infarction. Since there is initial direct myocardial activation, abnormalities affecting the ventricles cannot be reliably diagnosed, including infarction patterns. There is positive concordance (tall R waves) across the precordium (←), which is also consistent with a WPW pattern. This is a pattern seen when there is direct myocardial activation, such as what occurs with WPW pattern or a ventricular complex. The axis is extremely leftward between –30° and –90° (positive QRS complex in lead I and negative in leads II and aVF). The two etiologies for an extreme left axis include a left anterior fascicular block (rS QRS morphology in leads II and aVF), or an inferior wall myocardial infarction (deep Q waves in leads II and aVF). In this case there is a pseudo inferior wall infarction pattern. This is the etiology for the left axis. The QT/QTc intervals are normal (440/410 ms and 420/390 ms when corrected for the prolonged QRS duration).

In **Figure 4.6.2**, there is a regular rhythm at a rate of 52 bpm. There is a P wave before each QRS complex (+), and the P wave is positive in leads I, II, aVF, and V_4–V_6. The PR interval is constant (0.16 seconds) and there is a prominent PR segment. This is a sinus bradycardia. The P-wave morphology is the same as in **Figure 4.6.1**, and there is evidence for a left atrial abnormality or hypertrophy. The QRS complex duration is normal (0.08 seconds) and there is a normal axis between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (420/390 ms). The sinus complexes do not have evidence for preexcitation. However, there is one premature complex seen (v). It is preceded by a P wave (^) that is best seen in lead II where it is negative. This is a premature atrial complex. The QRS complex is wide (0.14 seconds), and it has a

morphology similar to the preexcited complexes seen in **Figure 4.6.1**, although it is wider. There is a slurred upstroke due to a delta wave (↑). This premature atrial complex manifests preexcitation, indicating that the accessory pathway is still active despite the previous ablation. The premature atrial complex is maximally preexcited for one of two reasons:

1. Since it is premature, the AV node is still refractory and hence the impulse is conducted to the ventricle via the accessory pathway.
2. The premature complex originates from a focus close to the accessory pathway.

In **Figure 4.6.3**, there is a regular wide complex tachycardia at a rate of 130 bpm. The QRS complex duration is 0.16 seconds. While the morphology resembles a left bundle branch block (LBBB) with a deep QS complex in lead V_1 (←) and a broad R wave in leads I and V_6 (→), it is not a typical pattern as there appears to be a Q wave in lead aVL and possibly lead I (^). Therefore, this is most likely an intraventricular conduction delay. The axis is normal between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are prolonged (340/500 ms), but are normal when the increased QRS complex duration is considered (280/410 ms). There are P waves seen in leads II, III, and aVF (+) and they are inverted. The RP interval is longer than the PR interval and this is a long RP tachycardia. Etiologies include:

1. Sinus tachycardia, which is not the diagnosis as the P waves are negative in the inferior leads.
2. Atrial flutter with 2:1 block which is not likely as a second atrial waveform is not seen.
3. Ectopic junctional tachycardia.
4. Atrial tachycardia.
5. Atypical atrioventricular nodal reentrant tachycardia.
6. AVRT.

Given the fact that there is still an active accessory pathway, the most likely diagnosis is AVRT (which was indeed confirmed with an electrophysiologic study). Although AVRT is usually associated with a short RP interval, this patient did have a previous ablation of the accessory pathway and while it is still functioning, its properties have been altered. Therefore, it is likely unable to conduct as rapidly as prior to the ablation. Although the QRS complex is wide, this is an orthodromic AVRT with a rate-related intraventricular conduction delay. It is not an antidromic AVRT as the QRS complex morphology during the tachycardia is much different that the morphology of the preexcited complexes during sinus rhythm (**Figure 4.6.1**) and the maximally preexcited premature atrial complex (**Figure 4.6.2**).

SECTION 5

Early Repolarization (ECG Pattern and the Syndrome)

Jonathan Bui, MD
Femi Philip, MD
Ezra A. Amsterdam, MD

CASE 5.1

Patient History

A 51-year-old African American male was seen for a routine evaluation. He was asymptomatic and had no history of heart disease. His ECG is shown in **Figure 5.1.1**. The ECG is notable for ST elevation in a majority of leads. Based on this patient's asymptomatic status, his ethnicity, and the characteristics of the ST segments, the ECG diagnosis was considered to be "benign early repolarization" (BER). It is important for the clinician to recognize this ECG pattern since it overlaps in several respects with two important clinical conditions: acute ST-segment elevation MI (STEMI) and pericarditis.

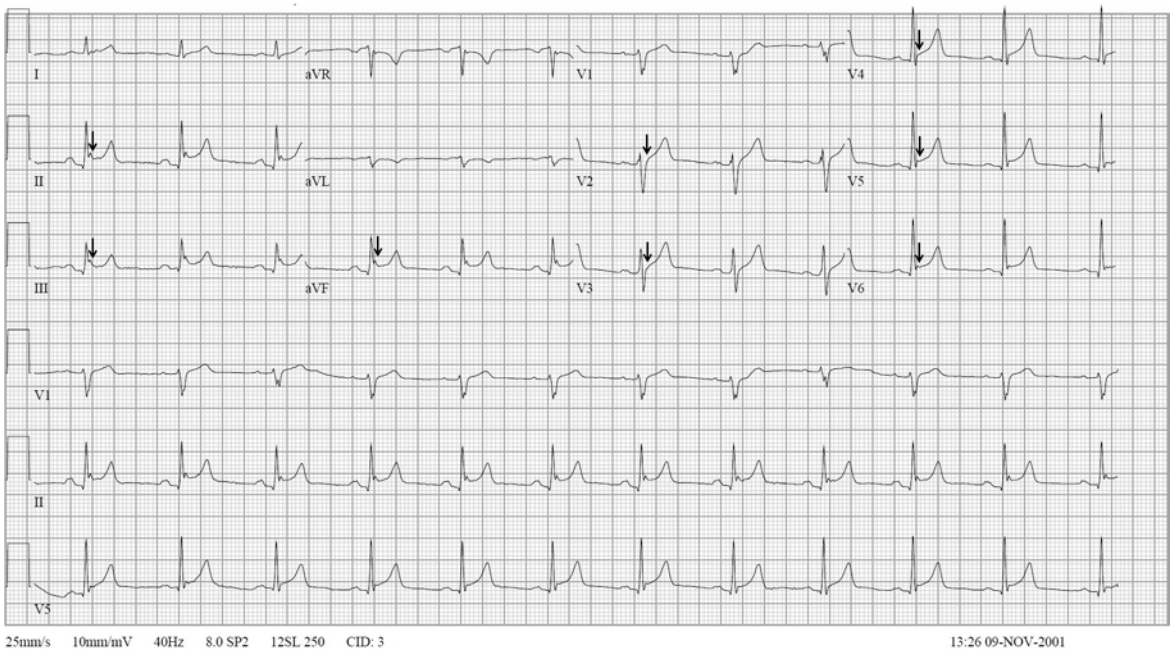

Figure 5.1.1

Question

Should BER always be considered benign?

ECG Masters' Collection: Favorite ECGs from Master Teachers Around the World, Vol. 2 © 2018 Mohammad Shenasa, Mark E. Josephson, N.A. Mark Estes III, Ezra A. Amsterdam, Melvin Scheinman. Cardiotext Publishing, ISBN: 978-1-942909-20-0.

Answer

Although long considered a benign ECG variant,[1–4] recent retrospective epidemiologic studies have suggested an association between early repolarization and serious arrhythmias, including cardiac arrest.[4] In this regard, detailed analysis of arrhythmia risk related to ST segment morphology has implied that an upward sloping ST segment and an upright T wave associated with J point notching (or slurring) is benign. By contrast, early repolarization with a horizontal or downsloping ST segment may be potentially more serious. However, it is advised that in the absence of syncope or a family history of premature sudden cardiac death, further evaluation is currently not indicated in subjects with early repolarization. These findings may introduce a new era in the understanding of "benign" early repolarization.[4]

Discussion

ECG characteristics of BER include: widespread, concave ST elevation, typically most prominent in the precordial leads; ST segments are usually higher in the precordial than limb leads; a small notch at the J point, usually best seen in the precordial leads (**Figure 5.1.1**, leads II, III, aVF, V_5, and V_6); prominent T waves in the precordial leads; and a shortened QT interval.[1] Most of these features are seen in **Figure 5.1.1**. Among these characteristics, the most distinctive evidence of BER is the small notch at the J point, which may not always be present. Also note that in this ECG, the most prominent ST elevations are in the inferior leads. BER may be associated with T-wave inversion in those leads with ST elevation and thus simulate the ECG of a STEMI.[2] However, ST-T changes in the latter condition are regional and usually associated with reciprocal ST changes. The challenge of differentiating the ECGs of BER and STEMI in some instances has been previously demonstrated.[1] This could lead to inappropriate administration of fibrinolytic therapy or urgent coronary angiography in patients with BER, with symptoms suggesting an acute coronary syndrome. It has been suggested that individuals with BER carry a copy of their ECG with them in order to obviate mistaken diagnosis of STEMI should they visit an emergency department (ED) with chest pain of noncardiac origin.

There is also considerable overlap in the ECGs of BER and pericarditis, including widespread ST elevation, concave ST segment morphology and prominent precordial T waves. The J-point notch can be very helpful as evidence of BER, and PR depression is suggestive of pericarditis. Most helpful is the overall presentation of the patient, including sex (male) and ethnicity (African American), although BER is not limited to this population.[2]

References

1. Turnipseed SD, Bair AE, Kirk JD, et al. Electrocardiogram differentiation of benign early repolarization versus acute myocardial infarction by emergency physicians and cardiologists. *Acad Emerg Med.* 2006;13:961–967.
2. Wang K, Asinger RW, Marriott HJL. ST-segment elevation in conditions other than acute myocardial infarction. *N Engl J Med*. 2003;349:2128–2135.
3. Klatsky AL, Oehm R, Cooper RA, Udaltsova N, Armstrong MA. The early repolarization normal variant electrocardiogram: Correlates and consequences. *Am J Med*. 2003;115:171–177.
4. MacFarlane PW, Antzelevitch C, Haissaguerre M, et al. The early repolarization pattern. *J Am Coll Cardiol.* 2015;66:470–477.

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Maichel Sobhy Naguib Botros, MD, PhD

CASE 5.2

Patient History

A 17-year-old female admitted due to repeated syncope post-cardiac arrest. An electroencephalography revealed an active epileptic focus. She was given anti-epileptic treatment, but syncope persisted. When admitted to the intensive care unit post-cardiac arrest, she had repeated seizures with no arrhythmia observed on the monitor. Her basic ECG is shown in **Figure 5.2.1**. She was put on a loop recorder that showed the following (**Figures 5.2.2–5.2.4**).

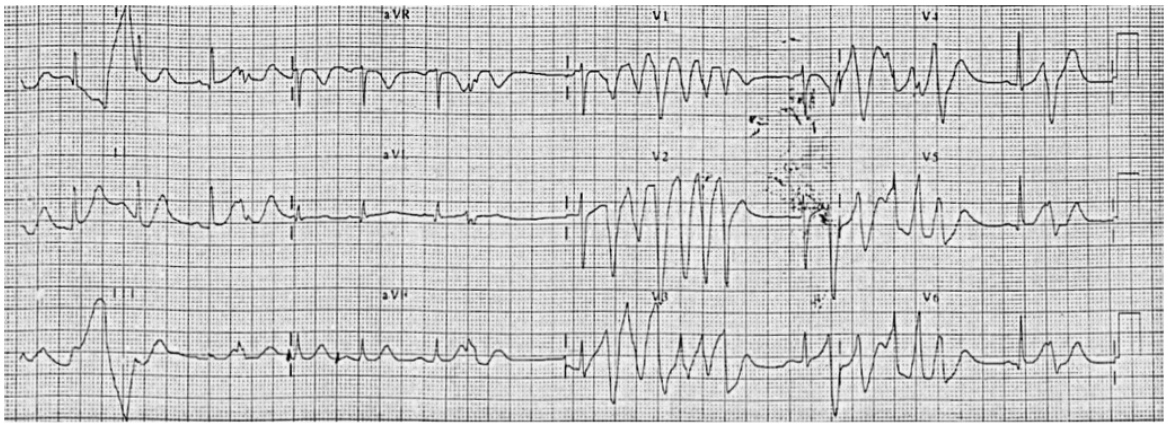

Figure 5.2.1 12-lead ECG of the patient after the syncope attack. ECG shows signs of an early repolarization pattern, as evident in leads I, aVL, and inferior and lateral leads. There are early post-repolarization PVCs, followed by runs of polymorphic ventricular tachycardia (VT).

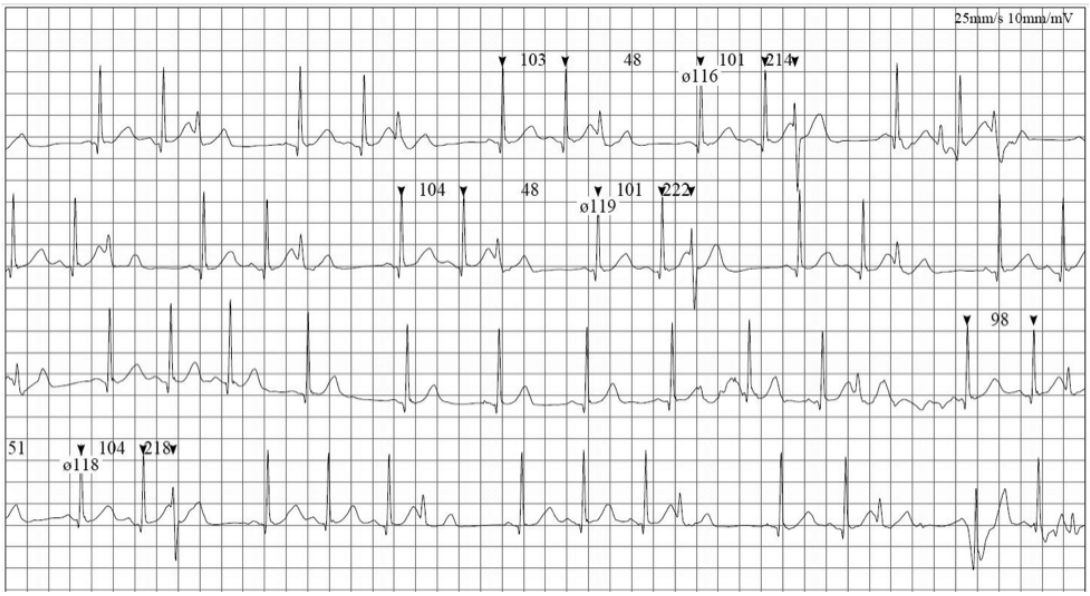

Figure 5.2.2 Loop recording shows early PVCs with short coupling intervals 210–230 bpm, seen in trigeminy.

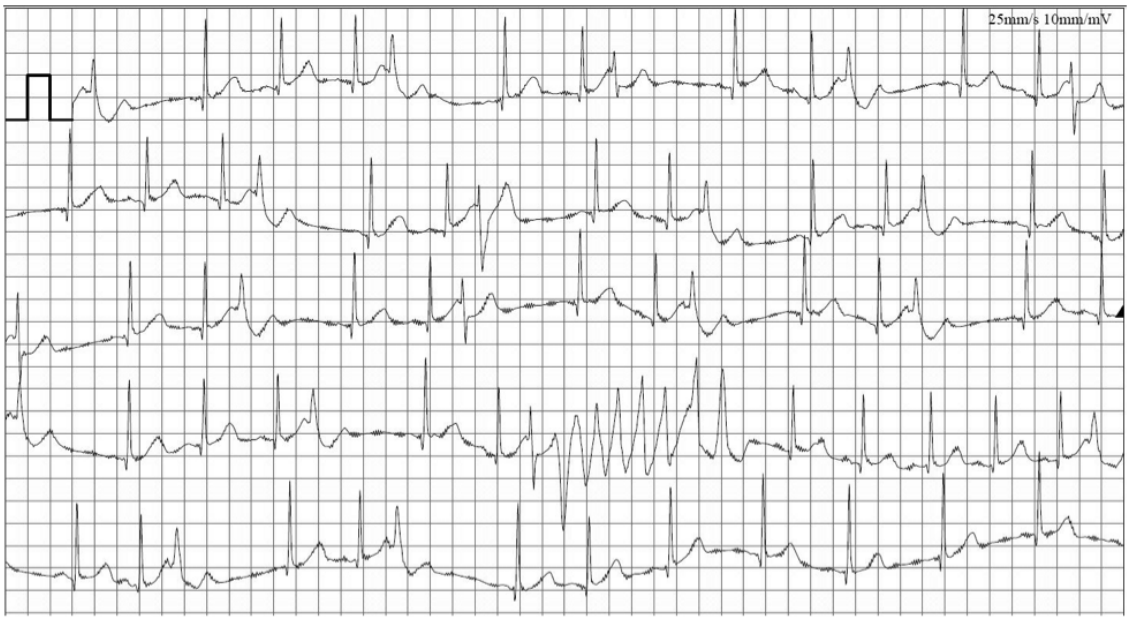

Figure 5.2.3 Loop recording of asymptomatic polymorphic VT, showing frequent PVCs.

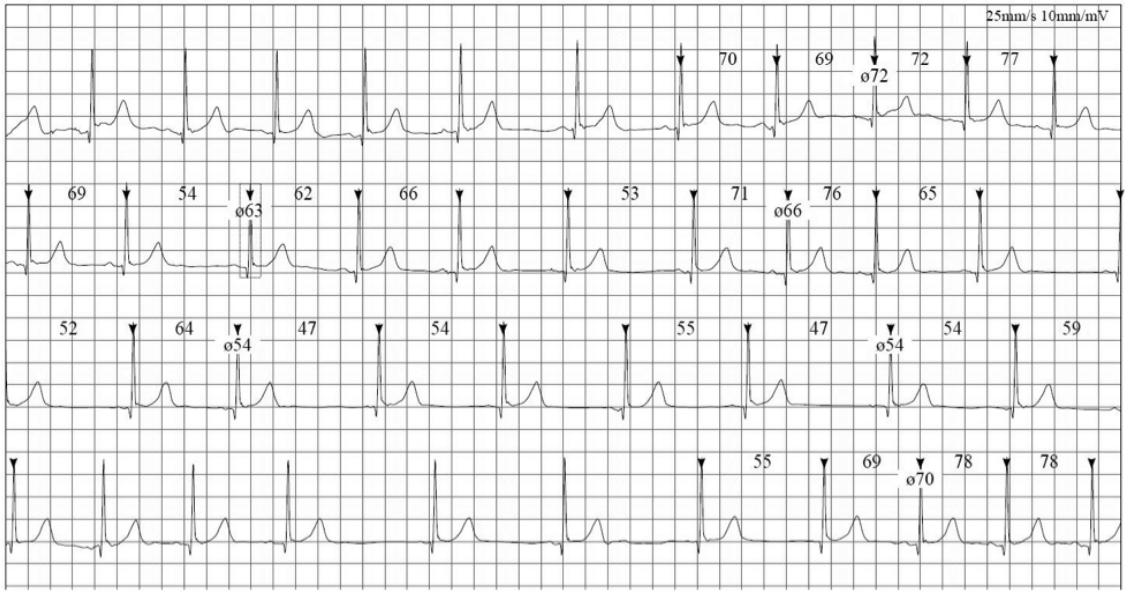

Figure 5.2.4 Loop recording after treatment. Recording showing respiratory sinus arrhythmia. Therapy has been given with a decrease in the frequency of PVCs, though early repolarization seems to be still evident.

Questions

1. What is the possible mechanism of arrhythmia in this patient?
2. What is the optimal management for this patient and her family?

Discussion, Interpretation, and Answers

According to the HRS/ESC/APHRS Expert Consensus Statement in patients with Inherited Primary Arrhythmia Syndromes, J-point and ST-segment elevation in two or more contiguous inferior and/or lateral leads in a standard 12-lead ECG in a patient with documented polymorphic ventricular fibrillation (VF) warrants diagnosis of early repolarization (ER) syndrome. Uneven distribution with resultant gradient of transient outward current (Ito) between the epicardium and endocardium results in J-point elevation.[2] Development of high ionic potassium gradient between the layers could either lead to consequent development of slow conduction forming a substrate for reentry in phase 2 or create boundary currents analogous to "injury currents" in acute myocardial infarction that raise ER cells to threshold and induce spontaneous activity with subsequent arrhythmic events.

Given that the patient was discovered to have epilepsy, association of ER and epilepsy with sodium and potassium channel mutations as well as autonomic dystonia are among the postulated underlying linking mechanisms.[3] Hence, the possibility that cardiac channelopathy has a role in sudden, unexplained death in epileptic patients has been raised.

Being a survivor of sudden cardiac death, ICD implantation is recommended for this patient (Class I). The acute use of isoproterenol for suppression of recurrent VF and quinidine for long-term suppression is also useful (Class IIa). Given that inheritance is not clearly monogenic in most cases and the genetic substrate is not clearly defined in this case, no recommendations can be given to screen the families of individuals with asymptomatic ER pattern. Also, there are no established provocative tests to diagnose concealed ER in family members of ER syndrome patients.[1]

References

1. Priori SG, Wilde AA, Horie M, et al. HRS/EHRA/APHRS expert consensus statement on the diagnosis and management of patients with inherited primary arrhythmia syndromes. *Heart Rhythm*. 2013;10(12):1932–1963.
2. Watanabe H, Nogami A, et al. Electrocardiographic characteristics and SCN5A mutations in idiopathic ventricular fibrillation associated with early repolarization. *Circ Arrhythm Electrophysiol*. 2011;4:874–881.
3. Lamberts RJ, Blom MT, Novy J, et al. Increased prevalence of ECG markers for sudden cardiac arrest in refractory epilepsy. *J Neurol Neurosurg Psychiatry*. 2015 Nov. doi: 10.1136/jnnp-2014-307772.

SECTION 6A

Long QT Syndrome

Li Zhang, MD
Peter R. Kowey, MD

CASE
6A.1

Patient History

A 17-year-old Chinese girl was admitted for seizure-like episodes that occurred at night. Family history revealed that her brother died suddenly at age 10.

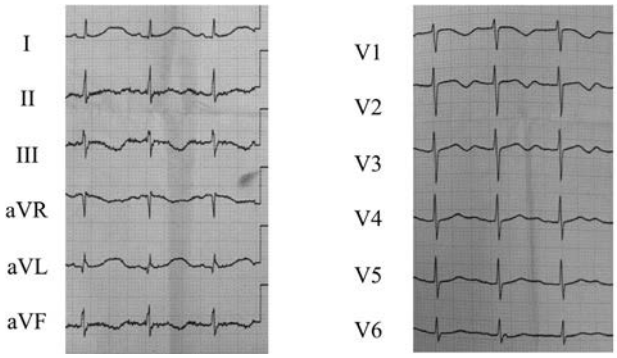

Figure 6A.1.1 Baseline 12-lead ECG shows a markedly prolonged QTc (657 ms) with T-wave pattern typical for type 2 long QT syndrome (LQT2).[1] Genetic testing has confirmed the ECG prediction that this patient indeed carries a *KCNH2* mutation.

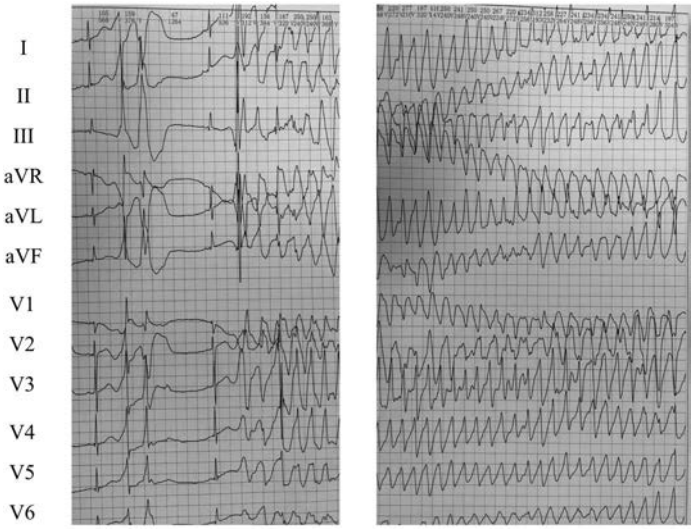

Figure 6A.1.2 Holter monitor revealed polymorphic premature ventricular contractions (PVCs) and pause-dependent torsades de pointes (TdP) (250 bpm) at night. TdP lasted about 8–10 seconds. Ectopic beats originated from the left ventricle.

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Questions

1. Is the frequency of cardiac events affected by age, gender, and genotype in LQTS?
2. Which beta-blocker is most effective in treating patients with LQTS?

Discussion

LQTS-related syncope is caused by TdP in most cases. Adolescents have the highest event rate, especially those with longer QT intervals (QTc ≥ 530 ms).[2] Adult females have a higher event rate than males. Among the common genotypes, the highest cumulative probability of cardiac events is seen in LQT2.[3] Emotional stress and startle are the common precipitators of cardiac events in LQT2. TdP can also occur during sleep in both LQT2 and LQT3.

Beta-blockers can reduce the event rate by 60%–64% in LQTS.[2,3] Propranolol is more effective than atenolol.[4] In this case, she (the proband) was first prescribed atenolol, but she continued to experience syncope. After switching to propranolol, she has been event-free. For high-risk patients, an implantable cardioverter-defibrillator (ICD) is the best protection.[3] ICD therapy was recommended, but the family declined.

References

1. Zhang L, Timothy KW, Vincent GM, et al. Spectrum of ST-T-wave patterns and repolarization parameters in congenital long-QT syndrome: ECG findings identify genotypes. *Circulation.* 2000;102(23):2849–2855.
2. Hobbs JB, Peterson DR, Moss AJ, et al. Risk of aborted cardiac arrest or sudden cardiac death during adolescence in the long-QT syndrome. *JAMA.* 2006;296(10):1249–1254.
3. Sauer AJ, Moss AJ, McNitt S, et al. Long QT syndrome in adults. *J Am Coll Cardiol.* 2007;49(3):329–337.
4. Vincent GM, Schwartz PJ, Denjoy I, et al. High efficacy of beta-blockers in long-QT syndrome type 1: Contribution of noncompliance and QT-prolonging drugs to the occurrence of beta-blocker treatment "failures." *Circulation.* 2009;119(2):215–221.

SECTION 6C

Torsades de Pointes

Li Zhang, MD
Peter R. Kowey, MD

CASE
6C.1

Patient History

A 25-year-old Caucasian female was clinically diagnosed with long QT syndrome (LQTS). Her T-wave pattern was atypical for any of the known genotypes (**Figure 6C.1.1**).

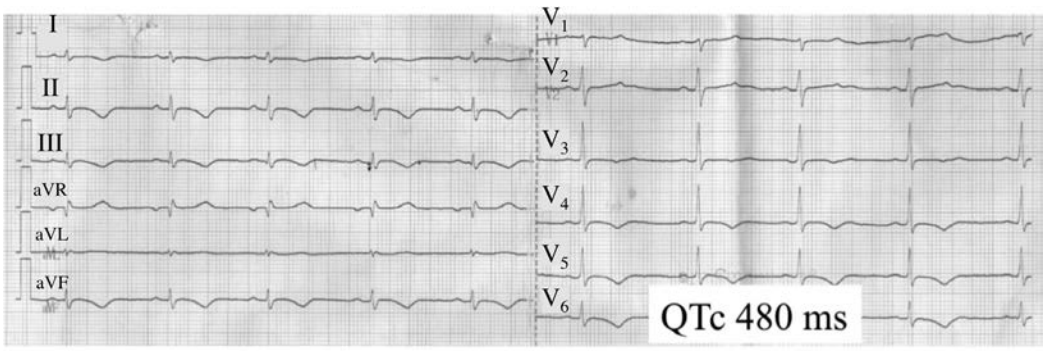

Figure 6C.1.1

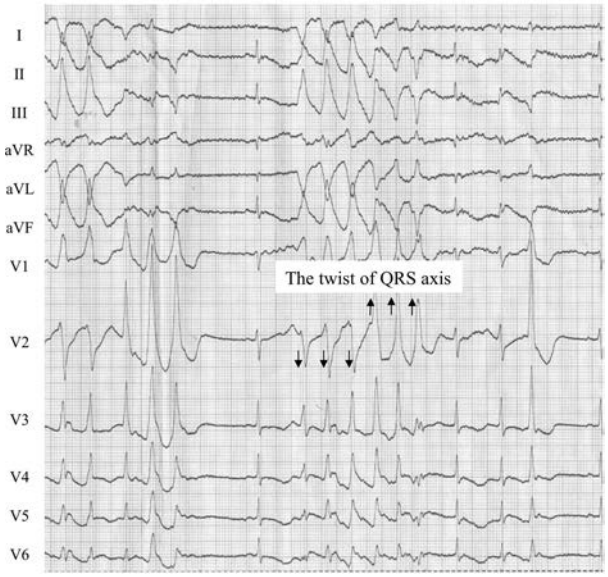

Figure 6C.1.2

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Pause-dependent torsades de pointes (TdP) (rates 167–207 bpm, lasting for 5–6 beats) were captured when she experienced near-syncope. Premature ventricular contractions originated from the left ventricle.

The direction of QRS axis twisting is the same in leads II, III, and aVF, and similar between aVL and V_2. The ectopic QRS is mostly upright in V_1 and V_3–V_5 (**Figure 6C.1.2**).

Because of recurrent syncope and documented TdP, this patient was treated with beta-blockers and an implantable cardioverter-defibrillator (ICD).

Question

Can a single-lead ECG capture TdP?

Discussion

TdP has variable morphologies, and the characteristic QRS morphology may not be shown in all 12 leads. Single lead ambulatory ECG may not capture the typical features of TdP.

The reason patients experience near-syncope or syncope is because TdP is self-terminating in most cases. However, since there is a possibility that TdP can degenerate into ventricular fibrillation, an ICD is recommended for high-risk patients.

SECTION 7

Brugada Syndrome

Marc Dubuc, MD
Jason Andrade, BSc, MD

CASE
7.1

Patient History

A 47-year-old healthy male presented for evaluation of a recent isolated syncopal episode that occurred while jogging. The baseline ECG is presented below (**Figure 7.1.1**).

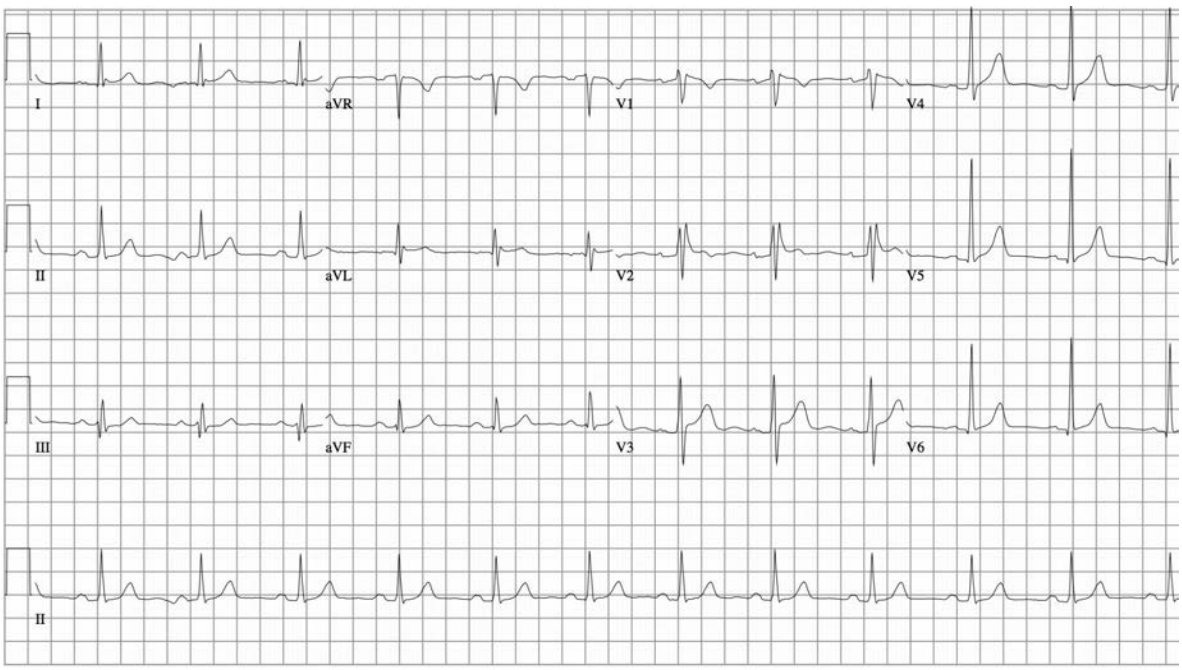

Figure 7.1.1

As part of the evaluation of his syncope, a procainamide infusion was performed. Precordial leads from the ECG performed at 10 minutes (**Figure 7.1.2**, Panel A, 500 mg) and 20 minutes (**Figure 7.1.2**, Panel B, 1 g) are presented.

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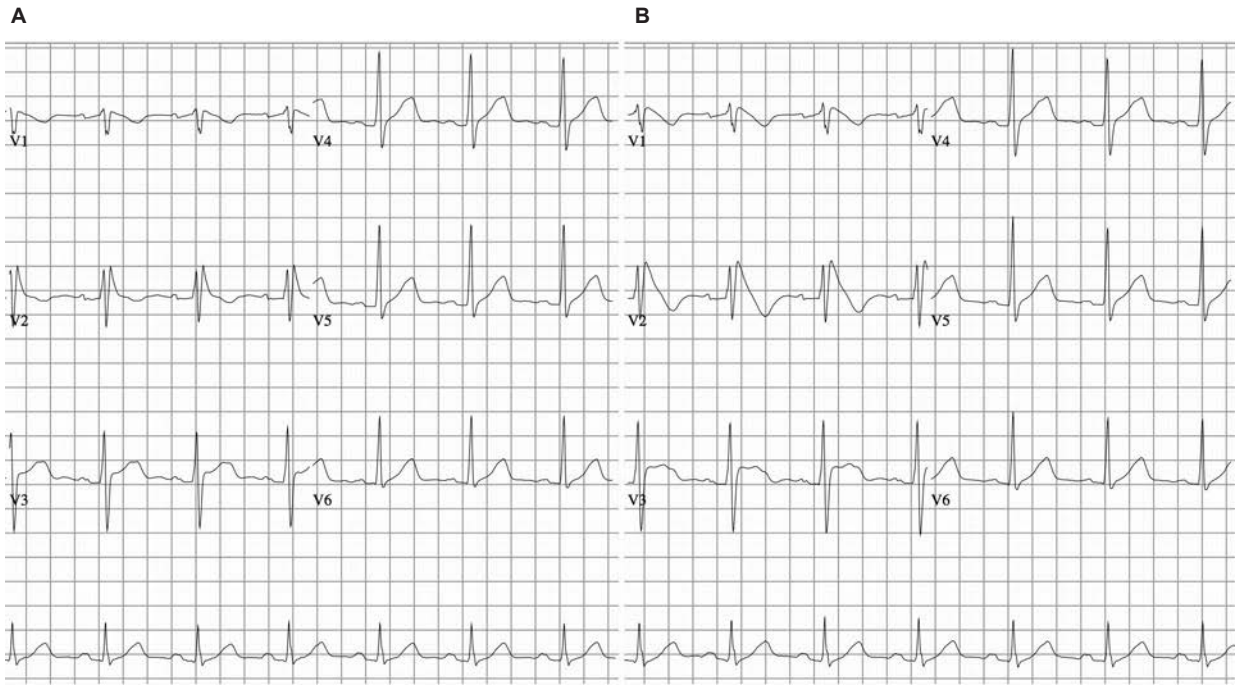

Figure 7.1.2

Question

What findings are evident in the procainamide challenge ECGs at 10 minutes and 20 minutes?

Discussion

At baseline there is an atypical right bundle branch block (RBBB) with minimal ST elevation demonstrated in V_1 and V_2. As these findings did not meet criteria for Brugada pattern, provocative testing using sodium channel blockers was undertaken (**Figure 7.1.2**). During procainamide infusion a typical type 1 pattern emerges (RBBB pattern with coved morphology ST-segment elevation of at least 2 mm followed by a negative T wave in V_1 and V_2).

These ECGs demonstrate the utility of procainamide infusion for the evaluation of suspected Brugada syndrome. Currently provocative testing is useful in symptomatic patients showing only the non–type 1 Brugada ECG pattern. In this population the provocative testing provides relevant information on the diagnosis, prognosis, screening, and therapy. Drug challenge generally is not performed in asymptomatic patients, or those displaying the type 1 ECG at baseline as the additional diagnostic and prognostic value are not clear. Procainamide challenge as well as recording the early precordial leads I and II interspaces higher may also detect the pattern.

Reference

1. Somani R, Krahn AD, Healey JS, et al. Procainamide infusion in the evaluation of unexplained cardiac arrest: from the Cardiac Arrest Survivors with Preserved Ejection Fraction Registry (CASPER). *Heart Rhythm*. 2014;11(6): 1047–1054.

Dan Blendea, MD, PhD
Moussa Mansour, MD

CASE 7.2

Patient History

A 50-year-old male with history of alcohol and cocaine abuse was evaluated given repolarization abnormalities seen in **Figure 7.2.1** during cocaine intoxication, and **Figure 7.2.2** (baseline ECG).

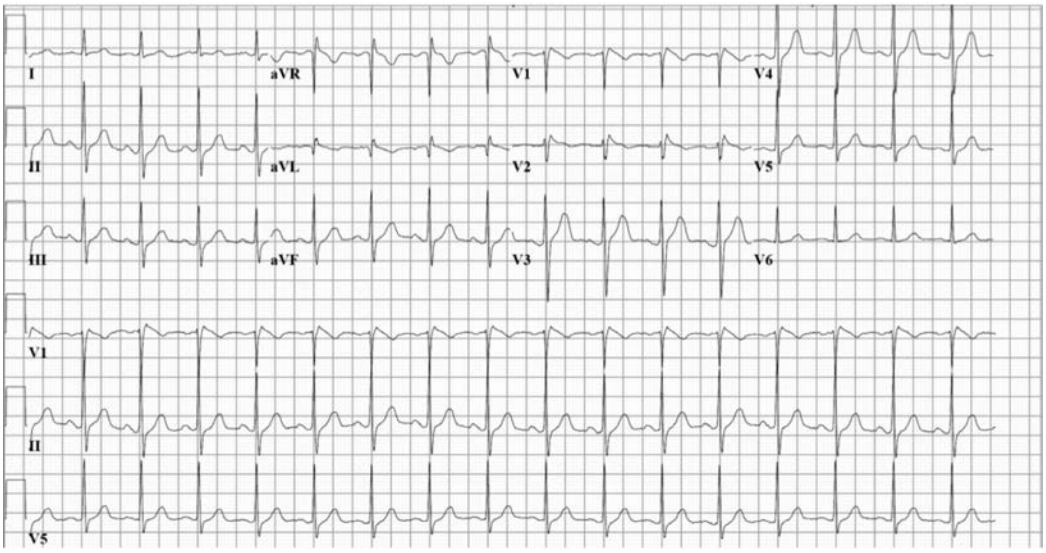

Figure 7.2.1 12-lead electrocardiogram during cocaine intoxication revealing a type 1 Brugada pattern with coved type ST elevation in leads V_1 and V_2.

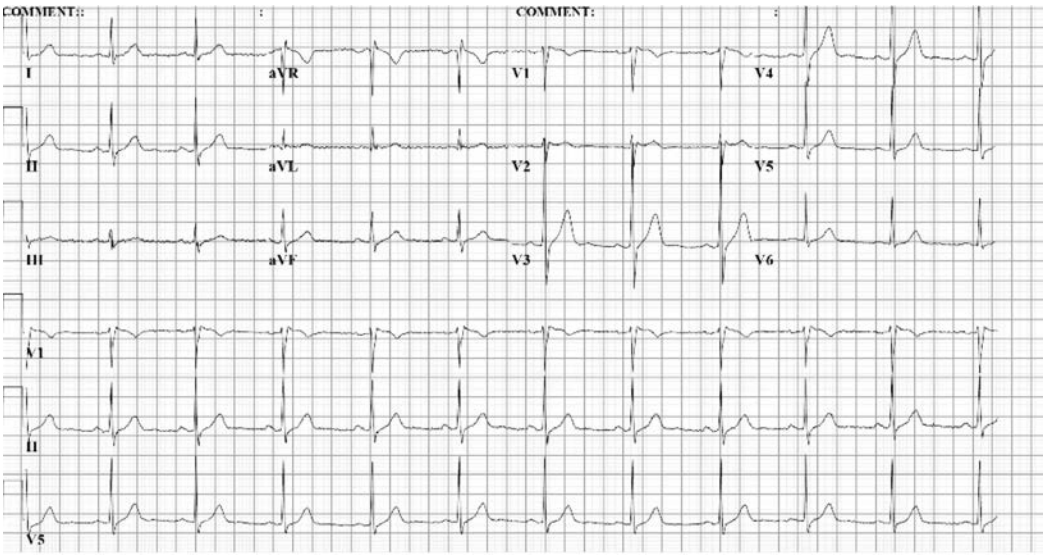

Figure 7.2.2 12-lead electrocardiogram at baseline (after the cocaine intoxication resolved) showing mild, nonspecific repolarization abnormalities in leads V_1–V_3.

Questions

1. What is the abnormality seen on the ECG?
2. Is there any relationship between cocaine abuse and this abnormality?

Answers

1. Brugada type 1 ECG pattern with coved ST elevations seen in leads V_1 and V_2 during cocaine intoxication (**Figure 7.2.3**).
2. Cocaine can induce a Brugada ECG pattern. The significance of this abnormality is unknown.

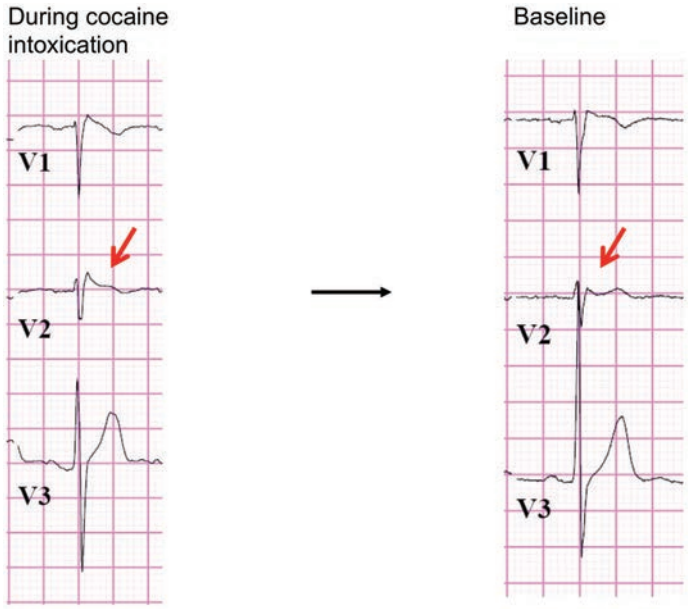

Figure 7.2.3 Comparison between the electrocardiographic pattern of repolarization in leads V_1–V_3 during cocaine intoxication and at baseline.

Discussion and Interpretation

The ECG in **Figure 7.2.1** reveals a type 1 Brugada pattern. The baseline ECG shows mild, nonspecific repolarization abnormalities. The patient underwent procainamide challenge test, which was negative (no Brugada pattern seen). The clinical importance of this cocaine-induced ECG abnormality is currently unknown.

The precise mechanism is not known. Cocaine may unmask the underlying genetic myocardial defect blocking sodium channels and replicate the Brugada ECG pattern, or induce the Brugada pattern by another mechanism.[1] In addition to the Brugada pattern, cocaine intoxication can be associated with accelerated junctional rhythm, right bundle branch block, QRS widening, QT prolongation.[1]

Reference

1. Ortega-Carnicer J, Bertos-Polo J, Gutierrez-Tirado C. Aborted sudden death, transient Brugada pattern, and wide QRS dysrrhythmias after massive cocaine ingestion. *J Electrocardiol.* 2001;34(4):345–349.

CASE
7.3

Robert J. Myerburg, MD

Patient History

This 60-year-old female was referred because of a family history of Brugada syndrome. One year before her visit, her 62-year-old sister, with a prior history of syncope, was diagnosed with Brugada syndrome during a febrile illness. A routine ECG while febrile showed a type 1 Brugada pattern. Prior ECGs, reviewed in retrospect, had shown previously unrecognized Brugada type 2 and type 3 patterns. Genetic studies revealed a Class 1 mutation in SCN5A (Arg225Trp). A subcutaneous implantable cardioverter-defibrillator (ICD) was implanted.

The patient was positive for the same genetic variant found in her sister and reported one episode of syncope about two years prior. The event occurred after donating blood, and she had transient nausea and weakness. Her ECG is shown below.

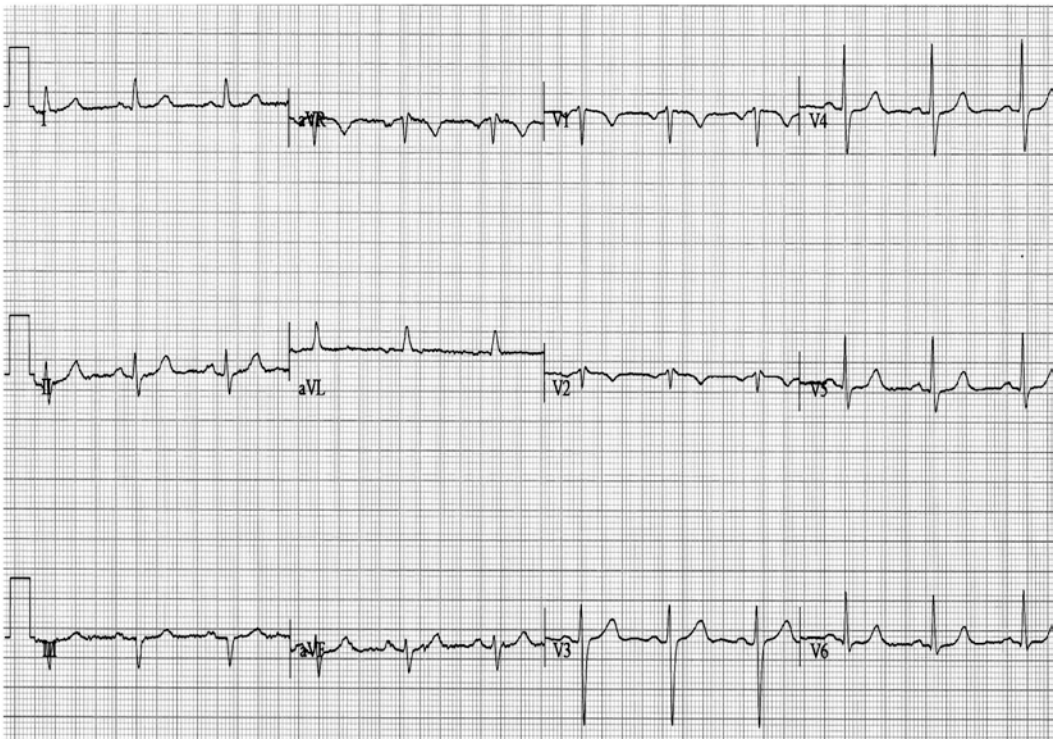

Figure 7.3.1

As a routine ECG during unselected readings, this ECG would be appropriately read as a minimal incomplete RBBB pattern and otherwise within normal limits. Taken in the context of the family history in a clinic setting, however, it would be appropriate to consider the possibility of a Brugada-associated pattern, albeit a type 3. Based upon the family history, including the genetic variant she shared in common with her sister, a flecainide study was done. A single 400 mg oral

dose of flecainide resulted in the widening of her QRS complexes to >120 ms in all leads, but no changes in her repolarization pattern. Her genetic profile also revealed the SCN5A H558R polymorphism, reported as a modifier of expression of Brugada-associated SCN5A variants.

Question

In this patient, an ICD is a reasonable management strategy based upon:

A. The patient's history of syncope and the presence of the SCN5A Arg225Trp genetic variant.
B. The presence of the SCN5A variant in the presence of the H558R polymorphism.
C. Her response to the flecainide challenge, in the presence of SCN5A, Arg225Trp variant.
D. Her family history, plus her response to the flecainide challenge.
E. None of the above.

Discussion, Interpretation, and Answer

Based upon the total available information, this patient is not a candidate for an ICD, and therefore the correct answer is E. This conclusion can only be reached by careful integration of all of the available information.

While it can be concluded that a patient with a history of syncope and a genetic variant associated with Brugada syndrome is a candidate for an ICD, there are complexities in this case that modify this conclusion. First, the syncopal episode she described is typical of neurocardiac syncope, rather than an arrhythmia-related event. While this alone may not be sufficient to preclude an ICD, since neurocardiac syncope may coexist (and perhaps be more common) in patients with Brugada syndrome, the fact that she carries the modifier polymorphism H558R, which has been reported to correct the trafficking defect associated with the Brugada genetic variant, the Brugada-associated risk is significantly reduced, if not eliminated. It is also noteworthy that if she only had site-specific testing for the SCN5A, Arg225Trp variant carried by her sister, who was negative for the H558R modifier polymorphism, this patient's modifier gene status would have been missed.

Another consideration is the response to flecainide; namely, widening of the QRS complex without repolarization changes, which is a pharmacologic effect of the drug, not to be construed as a Brugada-related positive test. Thus, the combination of the baseline ECG pattern, an effectively negative flecainide challenge, the presence of a modifier gene that corrects the Brugada defect, and the absence of symptoms suggesting arrhythmias, all mitigate against the notion that she is an ICD candidate at this time.

References

1. Poelzing S, Forleo C, Samodell M, et al. SCN5A polymorphism restores trafficking of a Brugada syndrome mutation on a separate gene. *Circulation*. 2006;114:368–376.
2. Lizotte E, Junttila MJ, Dube MP, et al. Genetic modulation of Brugada syndrome by a common polymorphism. *J Cardiovasc Electrophysiol*. 2009; 20:1137–1141.

Begüm Yetiş Sayın, MD
Sercan Okutucu, MD
Ali Oto, MD

CASE 7.4

Patient History

A 45-year-old male presented with syncopal attacks. He had no known cardiac disease. There was a family history of Brugada syndrome and sudden cardiac death. The first ECG (**Figure 7.4.1**) was taken in the emergency department (ED). Initial ECG revealed sinus bradycardia (heart rate: 50 bpm, QT: 430 ms, QTc: 393 ms). Please note the appearance of borderline right bundle branch block (RBBB) pattern with slight ST elevation in V_1.

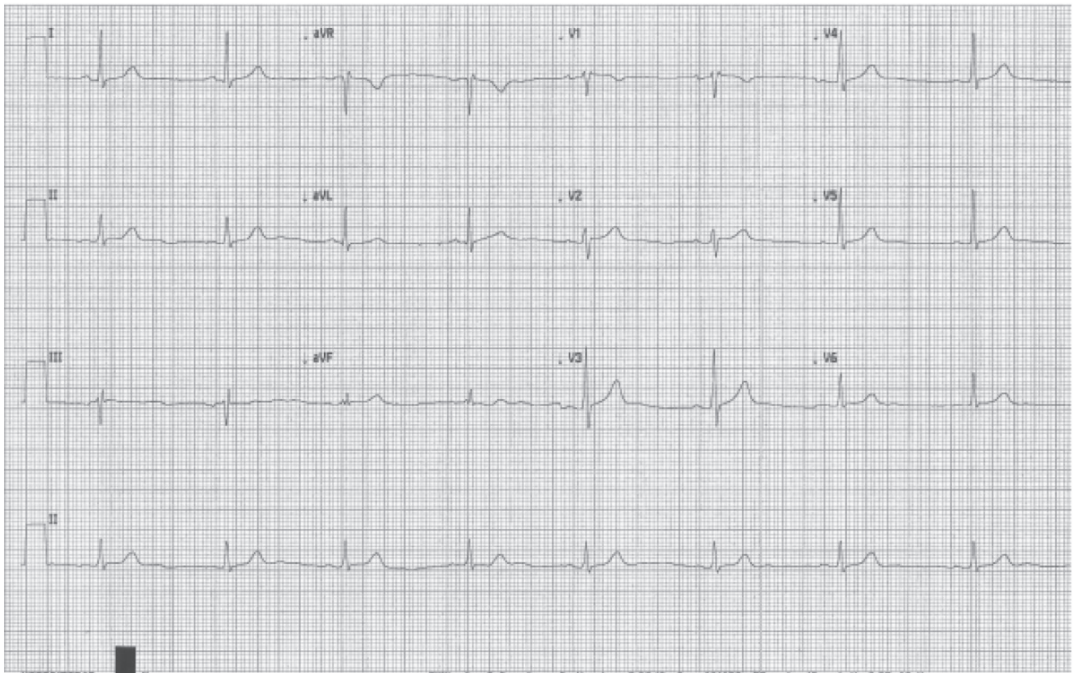

Figure 7.4.1

Question

Which provocative agent is commonly used as a challenge test in this situation?

Discussion

The diagnostic ECG pattern in Brugada syndrome can transiently normalize and may be unmasked by sodium channel blockers. Ajmaline challenge is the most commonly used sodium channel blocker for the diagnosis of Brugada syndrome.[1,2] After ajmaline challenge, typical cove-type ST-T changes were observed on V_1 and V_2 that support Brugada ECG pattern (arrows) (**Figure 7.4.2**).

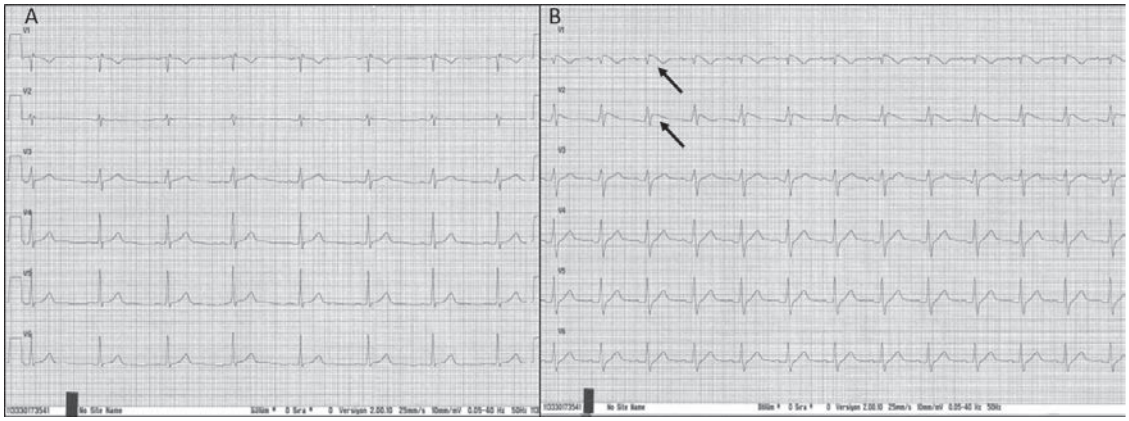

Figure 7.4.2

References

1. Brugada J, Brugada P, Brugada R. The ajmaline challenge in Brugada syndrome: a useful tool or misleading information? *Eur Heart J.* 2003;24(12):1085–1086.
2. Arnalsteen-Dassonvalle E, Hermida JS, Kubala M, et al. Ajmaline challenge for the diagnosis of Brugada syndrome: which protocol? *Arch Cardiovasc Dis.* 2010;103(11-12):570–578.

Carlos Alberto Pastore, MD, PhD
Nelson Samesima, MD, PhD

CASE 7.5

Patient History

A 29-year-old male of white ethnicity; married, laboratory assistant analyst; lives in a medium-size town in the interior of São Paulo, Brazil.

The patient notes three episodes of syncope, the first one at 25 years. Syncopal episodes occurred after physical effort and were preceded by a feeling of dizziness and visual blurring, lasting approximately one minute. He denied any other symptoms. Patient smokes and had a car accident two years before, with a right arm fracture. His father died of sudden death at 25 years.

Physical Examination

The patient is in good general health with no remarkable findings: normal abdomen, upper/lower limbs, and lungs.

Cardiovascular Examination

Regular heart rhythm, pulses full and symmetrical, nonpalpable pictus cordis.

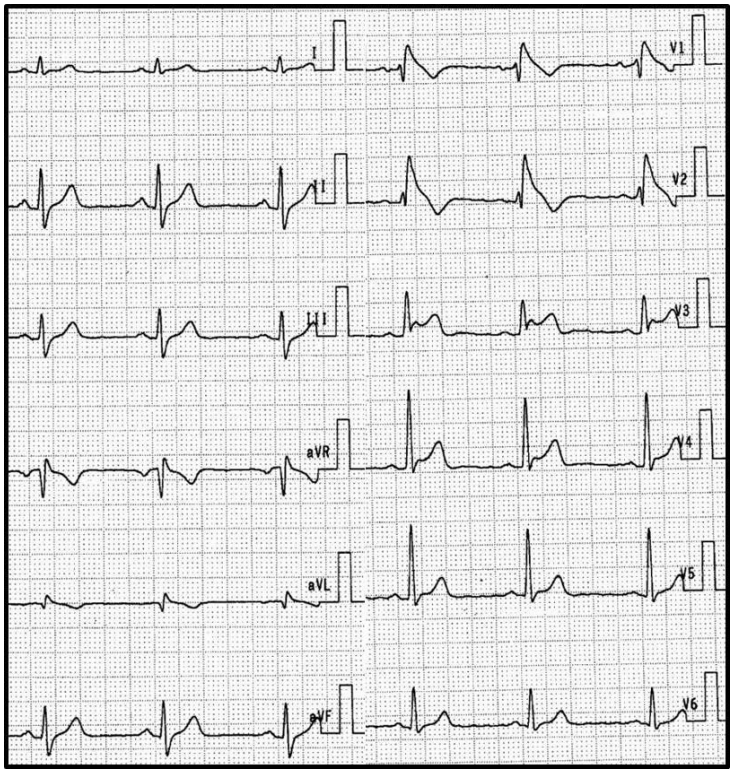

Figure 7.5.1

Diagnosis

Brugada syndrome

Discussion

Patient underwent an electrophysiological study (EPS), which showed: AH: 90 ms, HV: 62 ms, QRS: 120 ms; no induction of arrhythmias (**Figure 7.5.1**). Considering the presence of electrocardiographic abnormalities consistent with type 1 Brugada syndrome, the syncope and positive familial history of sudden death, this patient was considered high risk for sudden arrhythmic death, and was referred for implantation of a ventricular cardio-defibrillator.

Diagnosis of Brugada syndrome is made with the findings of typical electrocardiographic abnormalities and the occurrence of the following events: documented ventricular fibrillation, polymorphic ventricular tachycardia (VT), family history of sudden death in individuals below 45 years of age, syncope, characteristic ECG in relatives, and the induction of VT during EPS.[1]

Brugada syndrome has two typical electrocardiographic patterns: type 1 (**Figure 7.5.2A**) is the only one which definitely establishes the diagnosis, and is characterized by the presence of ST segment elevation ≥ 2 mm (0.2 mV) in right precordial leads (V_1, V_2 and, on occasions, V_3), with convex morphology, followed by a negative or flattened T wave.

Type 2 (saddleback morphology) (**Figure 7.5.2B**) leads to suspicion of Brugada, but it does not establish a definite diagnosis. Its typical alterations are: an initial ST segment elevation ≥ 2 mm, followed by concave elevation of ST segment ≥ 1 mm, and then a positive or biphasic T wave.

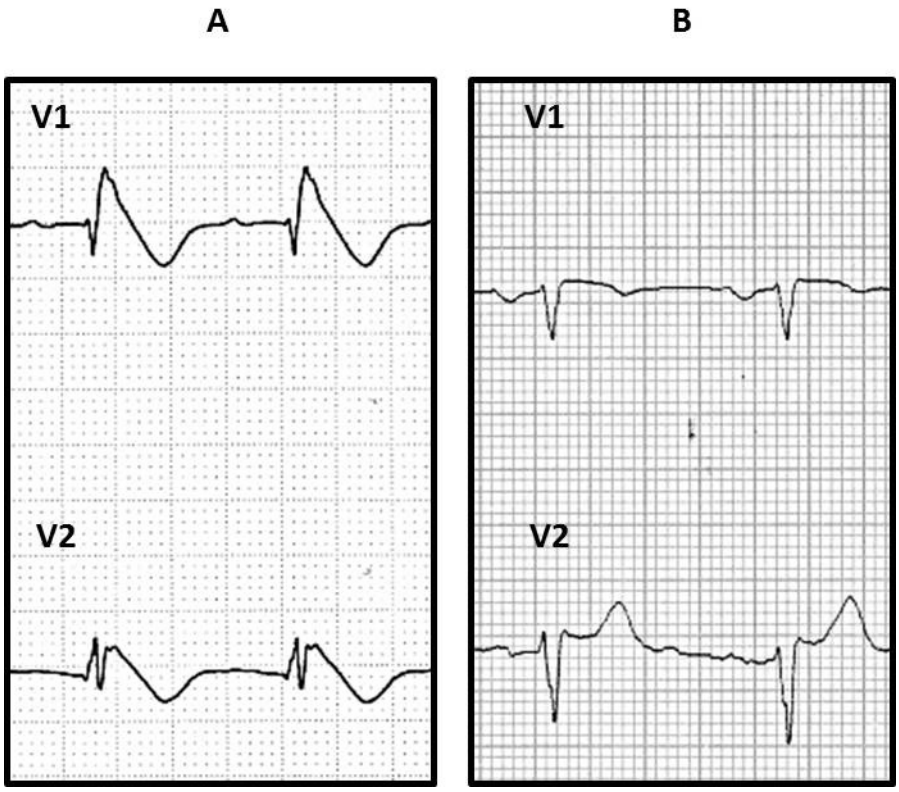

Figure 7.5.2 **A**. Characteristic Brugada type 1 ECG pattern. **B.** Brugada type 2 ECG pattern (saddleback morphology).

The ECG manifestations of Brugada syndrome are dynamic and, occasionally, they are not seen on the ECG. Sodium channel blockers (e.g., procainamide), hyperthermia, and vagotonic agents can unmask these alterations.

Importance of the Diagnosis

Brugada syndrome is a genetically determined arrhythmogenic disease, which usually targets male individuals in their third and fourth decade of life. Cardiac events occur during rest and sleep, with no definite explanation.[3] It is really important that a electrocardiographic diagnosis is made, since this ion channel defect can unleash malignant ventricular arrhythmias.

References

1. Eckardt L, Probst V, Smits JP, et al. Long-term prognosis of individuals with right precordial ST-segment-elevation Brugada syndrome. *Circulation*. 2005;111:257–263.
2. Brugada P, Brugada R, Brugada J. Should patients with an asymptomatic Brugada electrocardiogram undergo pharmacological and electrophysiological testing? *Circulation*. 2005;112:279–292.
3. Antzelevitch C, Brugada P, Borggrefe M, et al. Brugada Syndrome: Report of the second consensus conference. *Circulation*. 2005;111:659–670.

CASE 7.6

Philip Podrid, MD

Patient History

A 53-year-old female presents to the emergency department (ED) with fever, chills, diaphoresis, and shortness of breath. Physical examination of the chest demonstrates rales and rhonchi over the left lower base, and a chest x-ray reveals pneumonia of the left lower lobe. She is begun on antibiotics. A 12-lead ECG is obtained prior to admission to the floor (**Figure 7.6.1**). As a result, the catheterization laboratory is activated. Coronary arteries are normal. Several hours later, after her temperature returns to normal, an ECG is repeated (**Figure 7.6.2**).

Questions

1. What is the etiology for the apparent ST segment elevation in **Figure 7.6.1**?
2. What accounts for the change in the ST segments in **Figure 7.6.2**?

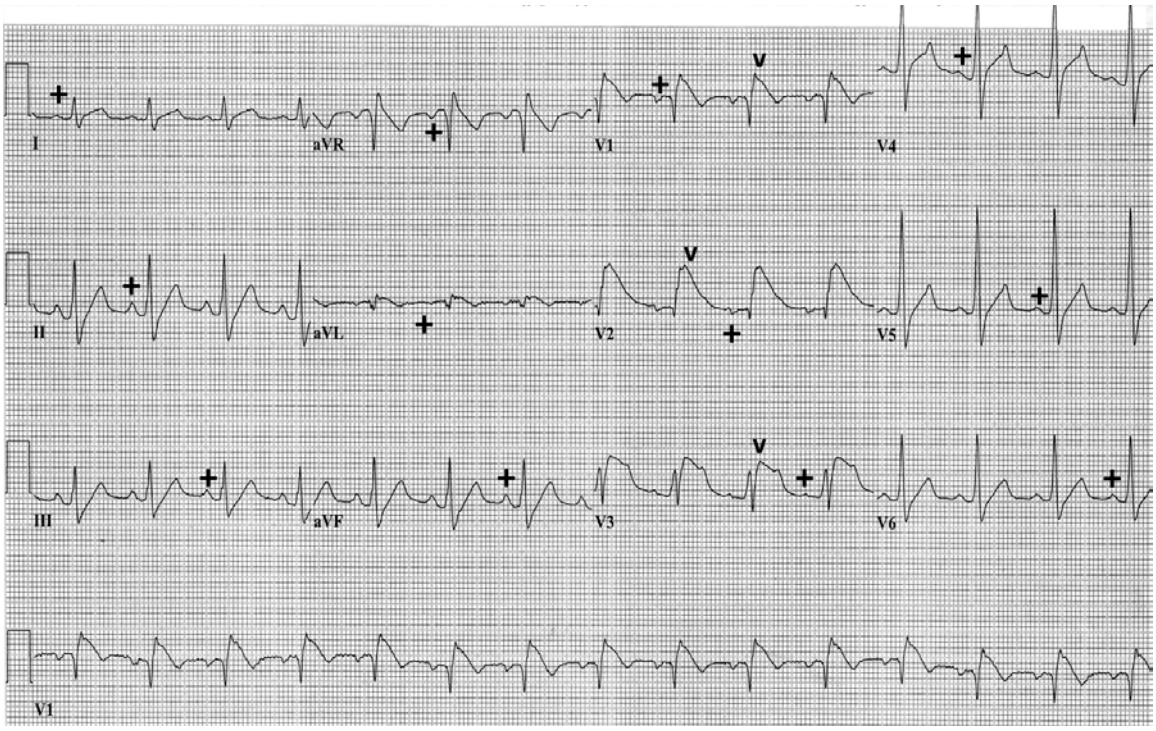

Figure 7.6.1

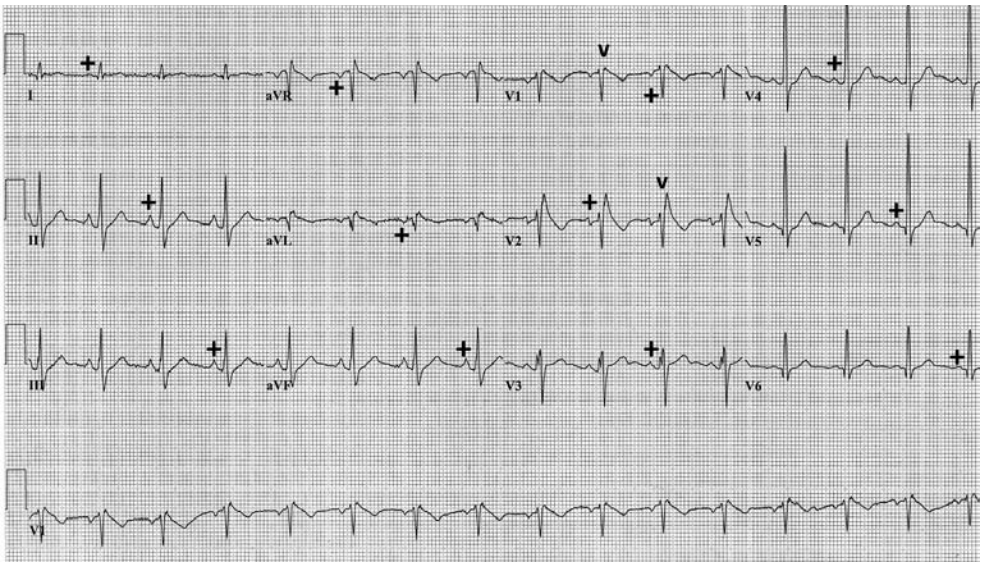

Figure 7.6.2

Diagnosis

Brugada pattern

Discussion

Figure 7.6.1 shows a regular rhythm at a rate of 76 bpm. There is a P wave (+) before each QRS complex with a stable PR interval of 0.16 seconds. The P wave is positive in leads I, II, aVF, and V_4–V_6. Therefore, this is a normal sinus rhythm. The QRS complex duration is normal (0.10 seconds) and has a normal morphology and axis between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (400/450 ms). There appears to be significant J point and ST segment elevation in leads V_1–V_3 (v), consistent with an anteroseptal ST segment elevation myocardial infarction (STEMI). However, it appears that the J point is elevated, but the ST segment slopes downward in leads V_1–V_3. The ST segment slopes down to a negative T wave in lead V_1. In addition, there are no reciprocal changes (i.e., ST segment depression in other leads) and the T waves are still normal (i.e., they are asymmetric). Therefore, this is not a typical pattern for an acute STEMI. Indeed, the cardiac catheterization demonstrated normal coronary arteries. The QRS complex and ST-T wave abnormalities are more consistent with a Brugada pattern, which may be exposed by or made more apparent by an increase in temperature.

Figure 7.6.2 shows a regular rhythm at a rate of 90 bpm. There is a P wave in front of each QRS complex (+) and the P-wave morphology and PR interval is the same as seen in **Figure 7.6.1**. The QRS complex duration, morphology and axis as well as the QT/QTc intervals are the same as seen in **Figure E.7.6.1**. ST segment elevation is no longer seen, but there is an elevated J point and slowly descending ST segment to a negative T wave seen in leads V_1 and V_2 (v), which is characteristic of a Brugada pattern (in this case a type 1 Brugada pattern).

Reference

1. Bayés de Luna A, Brugada J, Baranchuk A, et al. Current electrocardiographic criteria for diagnosis of Brugada pattern: a consensus report. *J Electrocardiol*. 2012;45:433–442.

SECTION 8C

Atrial Tachycardia/Atrial Flutter

CASE
8C.1

Tamer S. Fahmy, MD, PhD

Patient History

A 67-year-old female patient complains of recurrent, rapid palpitations. She is known to have bronchial asthma and maintained on bronchodilators and inhaled steroids. Her resting ECG and rhythm strip are shown in **Figures 8C.1.1** and **8C.1.2**.

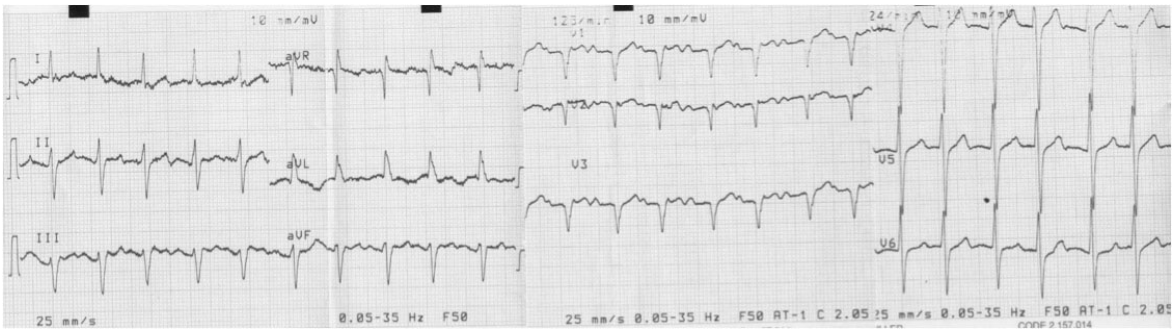

Figure 8C.1.1 Baseline 12-lead ECG: Surface ECG performed at rest, with no complaints. Note that the patient is already on bronchodilators.

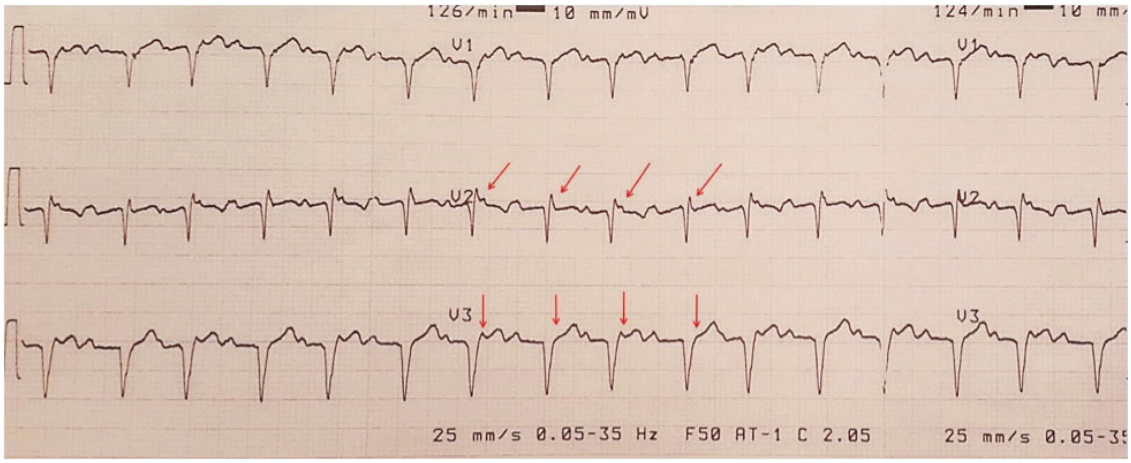

Figure 8C.1.2 Rhythm strip from the same patient. Note the appearance of the second P wave at the end of the QRS on alternate beats (arrows). See text for explanation.

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Questions

1. What is the ECG rhythm?
2. Why are there variations in the QRS morphology in the alternate beats?

Discussion, Interpretation, and Answers

This patient, though not complaining at the time of the ECG, is being maintained on bronchodilators and is prone to different forms of atrial arrhythmias. The ECG shows atrial tachycardia with 2:1 atrioventricular (AV) block. There are variable RR intervals. The appearance of variation of the QRS morphology at alternate beats is due to the visibility of the P wave at the end of the QRS at alternate beats. This is evidently due to variation of the AV nodal conduction, with resultant delay in the QRS, resulting in variable PR and RR intervals. The beats with shorter PR and earlier QRS results in the appearance of the P wave at the end of the QRS due to equal PP intervals.

References

1. Khorfan FM, Smith P, Watt S, Barber KR. Effects of nebulized bronchodilator therapy on heart rate and arrhythmias in critically ill adult patients. *Chest.* 2011;140(6):1466–1472.
2. Lee CH, Choi S, Jang EJ, et al. Inhaled bronchodilators and the risk of tachyarrhythmias. *Int J Cardiol*. 2015;190:133–139.

Jonathan Kalman, MBBS, PhD

CASE 8C.2

Patient History

A 69-year-old female with past history of paroxysmal atrial fibrillation and no structural heart disease presented hypotensive (systolic BP 80) and diaphoretic with this left bundle branch block (LBBB) wide complex tachycardia. The cycle length is 290 ms (**Figure 8C.2.1**).

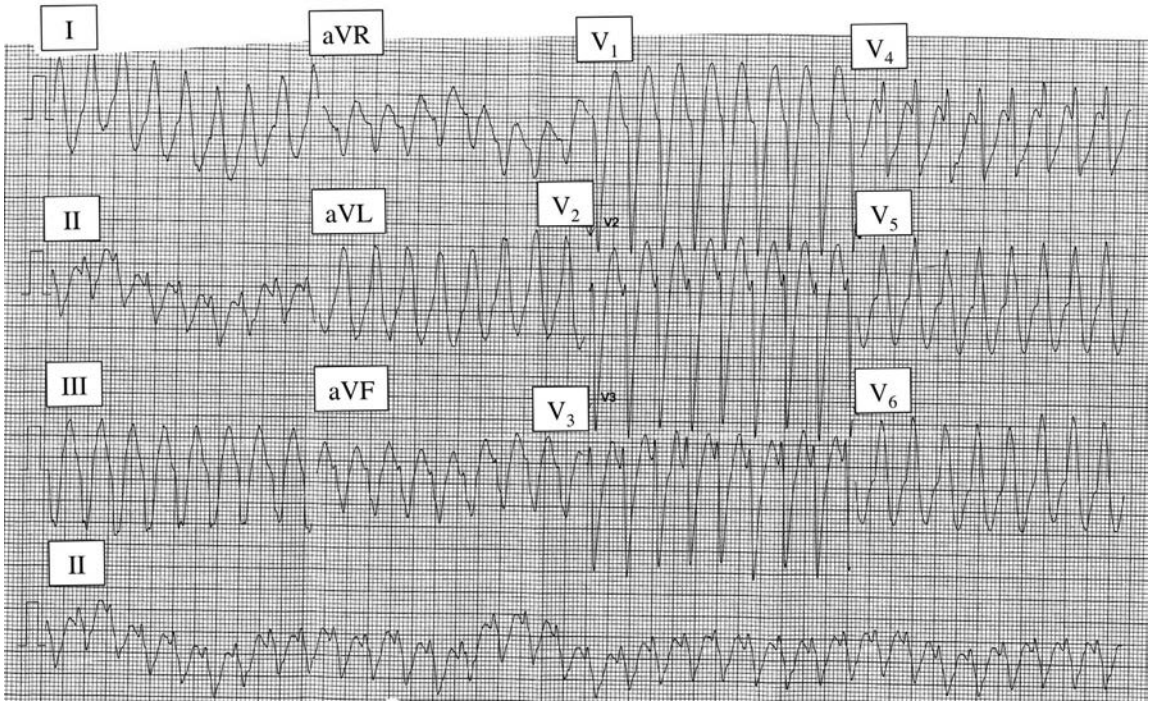

Figure 8C.2.1

Discussion

Shortly after, **Figure 8C.2.2** was recorded. The QRS cycle length is 580 ms and flutter waves can be clearly seen. This is 2:1 flutter with a flutter rate identical to the prior ECG, indicating that this was 1:1 flutter. The patient had been treated with flecainide for paroxysmal atrial fibrillation, which resulted in atrial fibrillation organizing too long of a cycle length flutter (due to conduction slowing in the atrium). The very wide complexes on the preceding trace were due to the effect of flecainide conduction slowing at that rate in the ventricle. At the longer ventricular cycle length when flutter was 2:1, ventricular conduction recovered. This patient had been treated with flecainide without an atrioventricular nodal blocking drug, which might have prevented 1:1 flutter.

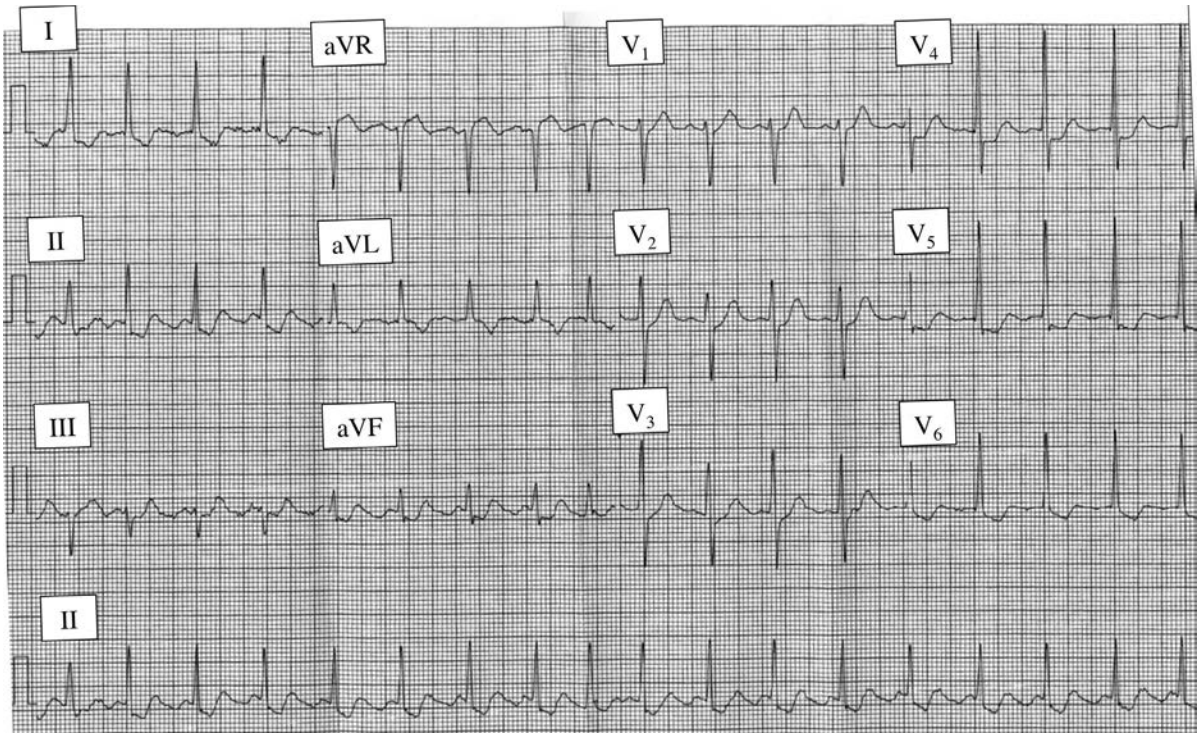

Figure 8C.2.2

Robert Lemery, MD

CASE 8C.3

Patient History

A 63-year-old female was seen in consultation for complete heart block on the sixth day post-cardiac surgery. She had undergone mitral valve repair for severe mitral regurgitation and had a history of severe pulmonary hypertension. During surgery, she had closure of an atrial septal defect and underwent a biatrial cryomaze surgical procedure for atrial fibrillation (AF). On the 2D echocardiogram, the left atrial volume was 232.5 mL, and the right atrium was also severely enlarged; there was normal left ventricular function. Review of the preoperative ECG (**Figure 8C.3.1**) shows marked and prominent atrial activity in V_1, making the QRS and P wave difficult to distinguish. Although a fibrillatory component cannot be excluded, the ECG predominantly shows atrial flutter. Lead V_1, often seen as the antenna of the left atrium,[1] reveals the largest P-wave amplitude seen in this patient.

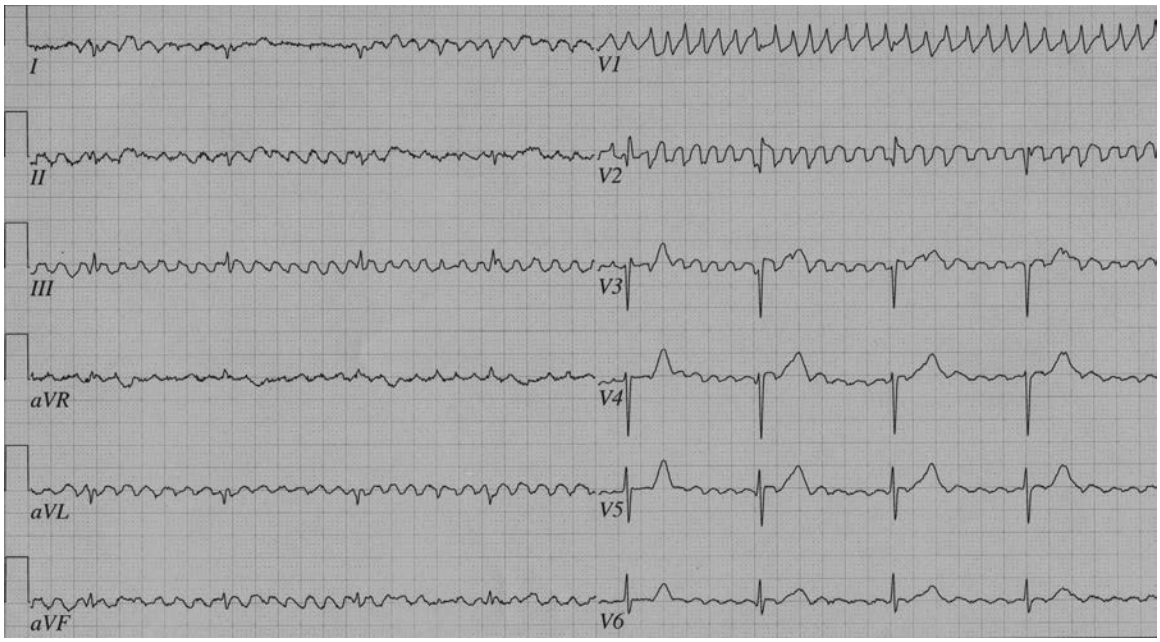

Figure 8C.3.1

Question

What does the narrow complex rhythm in this ECG suggest?

Discussion

The ECG (**Figure 8C.3.1**) shows a regular narrow complex rhythm, suggesting the possibility that heart block was already present prior to surgery. Another preoperative ECG shows AF (**Figure 8C.3.2**),

again with a regular narrow complex rhythm, also suggesting heart block. Following surgery, there is restoration of sinus rhythm with a cycle length of approximately 600 ms, showing complete heart block with a right bundle branch block morphology (**Figure 8C.3.3**).

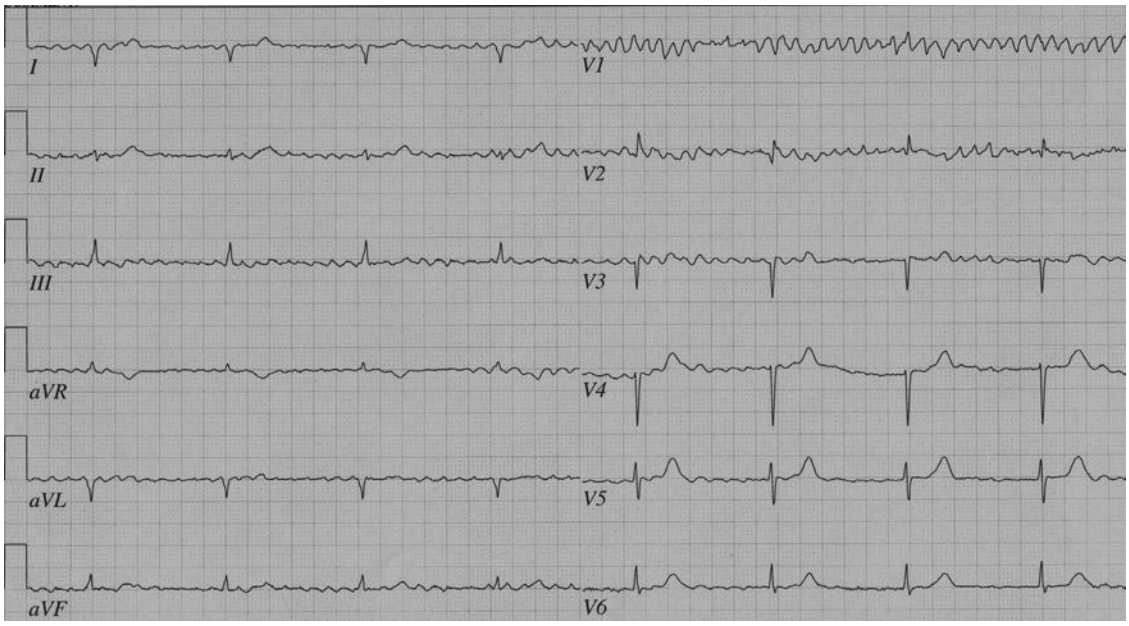

Figure 8C.3.2

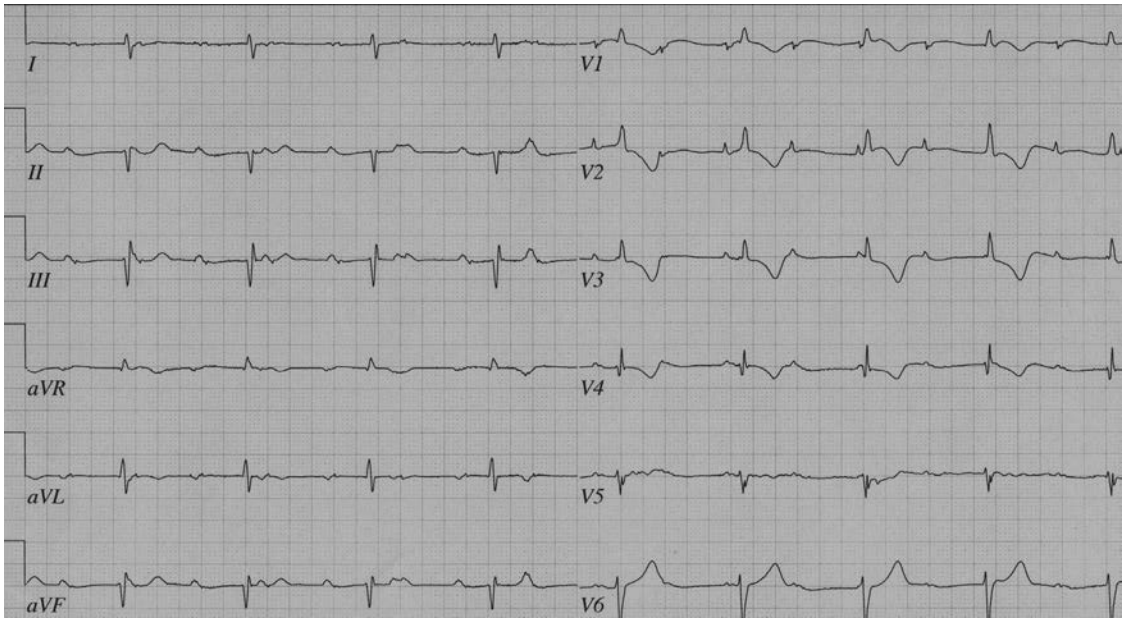

Figure 8C.3.3

The patient then develops atrial flutter, showing negative flutter waves inferiorly, with ventricular pacing (**Figure 8C.3.4**). Subsequently, the ECG showed atrial tachycardia-flutter with positive P waves inferiorly, negative in AVL, and biphasic in V_1, with ventricular pacing.

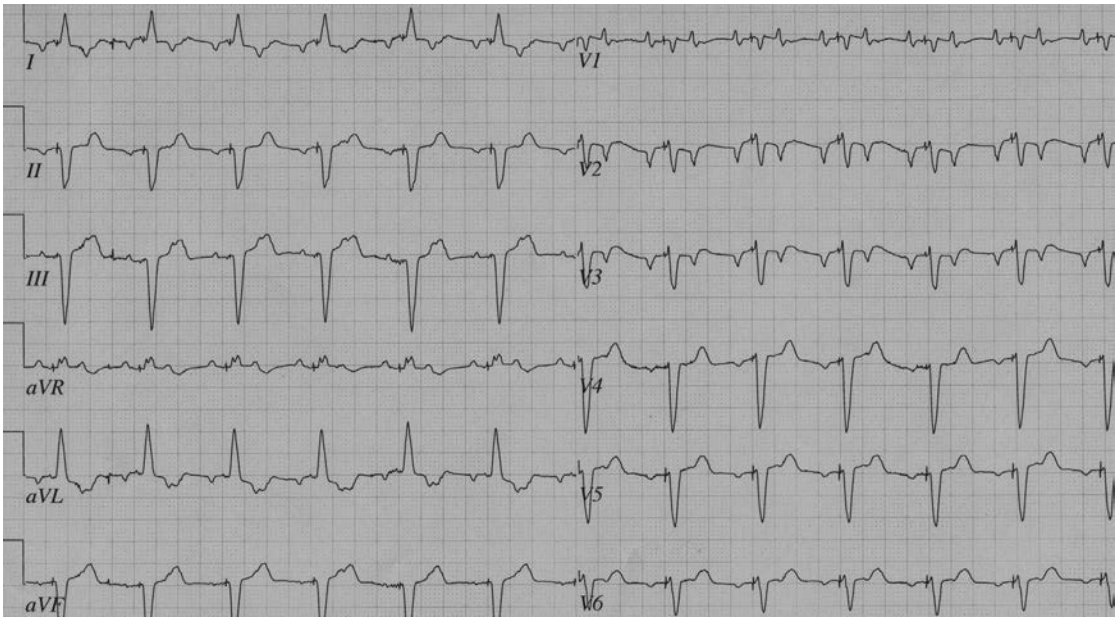

Figure 8C.3.4

References

1. Nault I, Lellouche N, Matsuo S, et al. Clinical value of fibrillatory wave amplitude on surface ECG in patients with persistent atrial fibrillation. *J Interv Card Electrophysiol.* 2009;26:11–19.
2. Magnano AR, Argenziano M, Dizon JM, et al. Mechanisms of atrial tachyarrhythmias following surgical atrial fibrillation ablation. *J Cardiovasc Electrophysiol.* 2006;17:366–373.

CASE 8C.4

Mohamed Magdy, MSc, L'AFSA, PhD, MD

Patient History

A 14-year-old boy complained of palpitations for several years. The left ventricular ejection fraction on his echocardiogram was 45%.

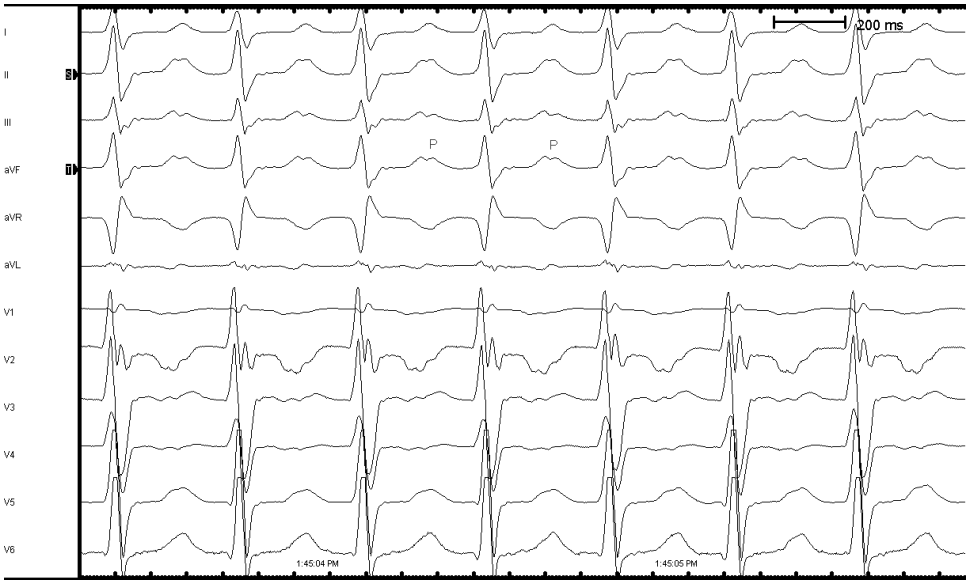

Figure 8C.4.1 Note the long PR interval during the tachycardia, which is almost a terminal T wave. The P-wave morphology suggests right atrial tachycardia. The P wave is positive in lead DI, negative in aVR, and positive in inferior leads. Negative P waves are in leads V_1 and V_2.

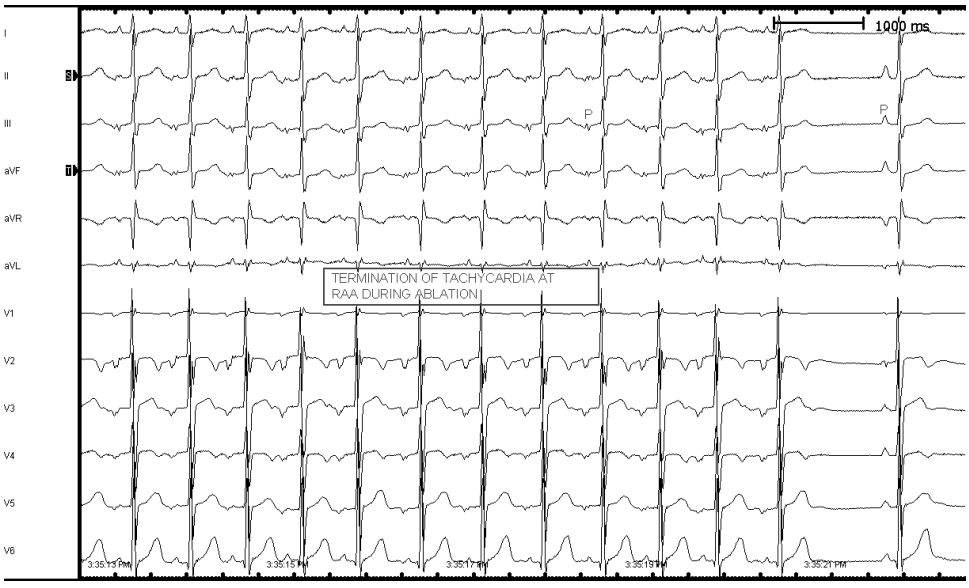

Figure 8C.4.2 Normal sinus rhythm during ablation of the right atrial appendage focus.

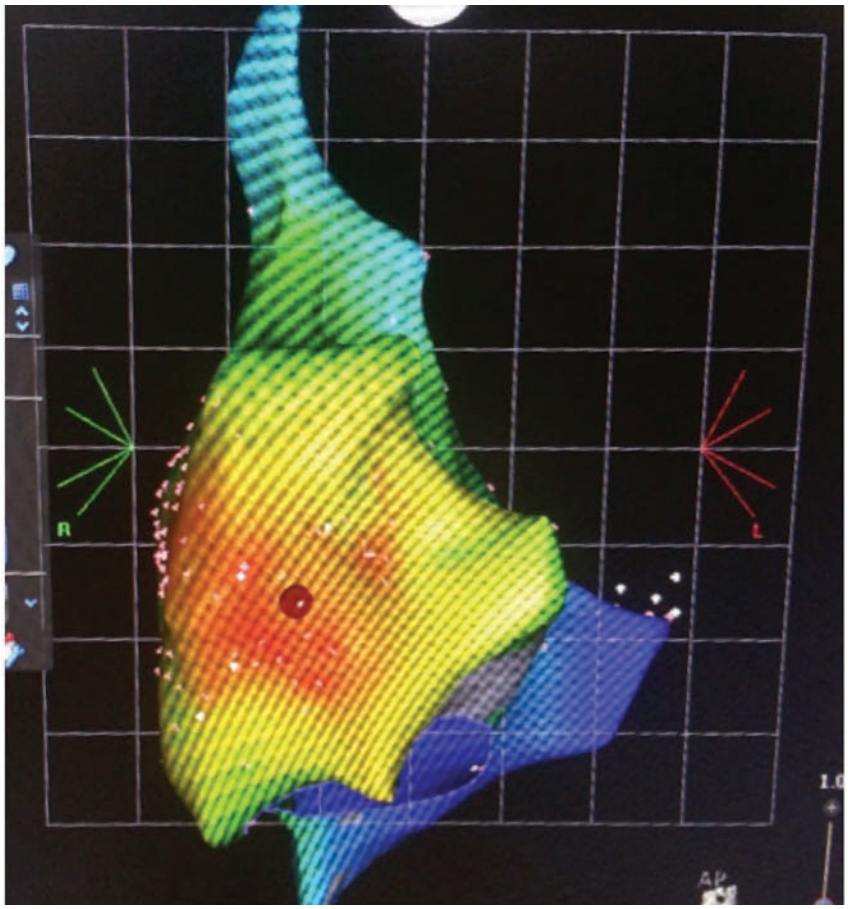

Figure 8C.4.3 Electroanatomical map of the right atrium, showing earliest activation (red dot) corresponding to the location of the right atrial appendage. The activation of red to blue shows that depolarization during tachycardia starts at the right atrial appendage and then spreads to the right.

Discussion

Right atrial appendage tachycardia is relatively rare. Intracardiac studies revealed atrial tachycardia originating from the right atrial appendage (not shown here). Right atrial appendage tachycardia appears most frequently in young male patients. This type of atrial tachycardia is frequently incessant, producing dyspnea and left ventricular systolic dysfunction secondary to tachymyocardiopathy. A highly sensitive and specific ECG pattern can be used to identify it. Radiofrequency ablation is effective for the improvement of all symptoms and the recurrence rate is low.

CASE 8C.5

Magdi M. Saba, MD
David E. Ward, MD

Patient History

A 35-year-old female presented to the emergency department (ED) with palpitations. The left ventricle was dilated and systolic function was moderately depressed.

Question

What are the possible mechanisms of the tachycardia?

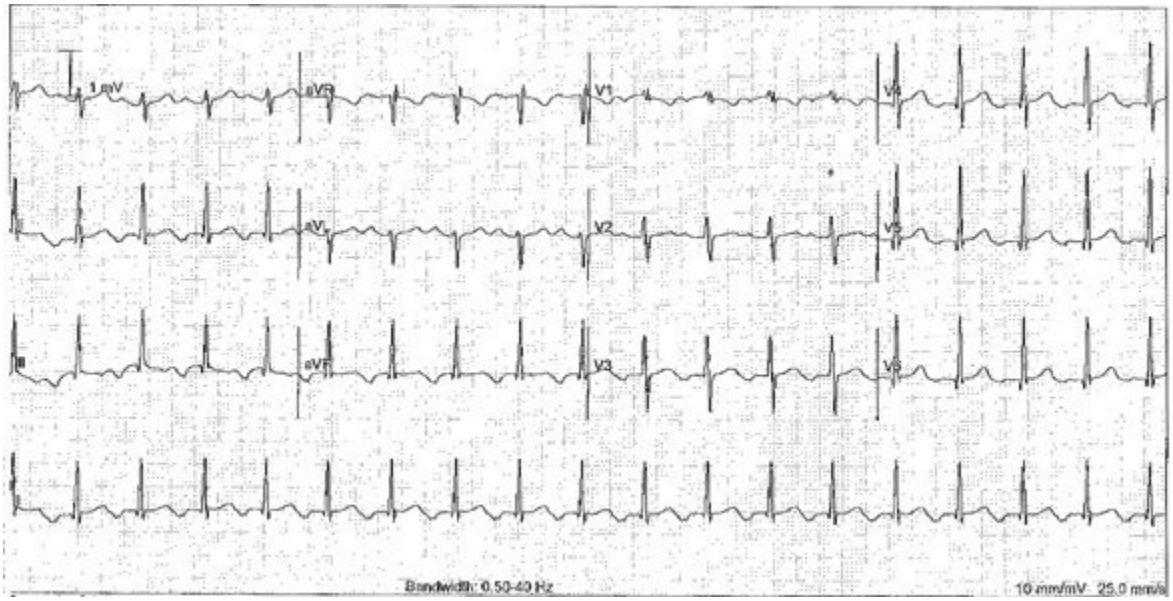

Figure 8C.5.1

The ECG (**Figure 8C.5.1)** shows a long RP tachycardia with negative P waves in the inferior leads, suggesting atypical atrioventricular nodal reentrant tachycardia (AVNRT), orthodromic reciprocating tachycardia utilizing a slow-conducting accessory pathway, or an atrial tachycardia.

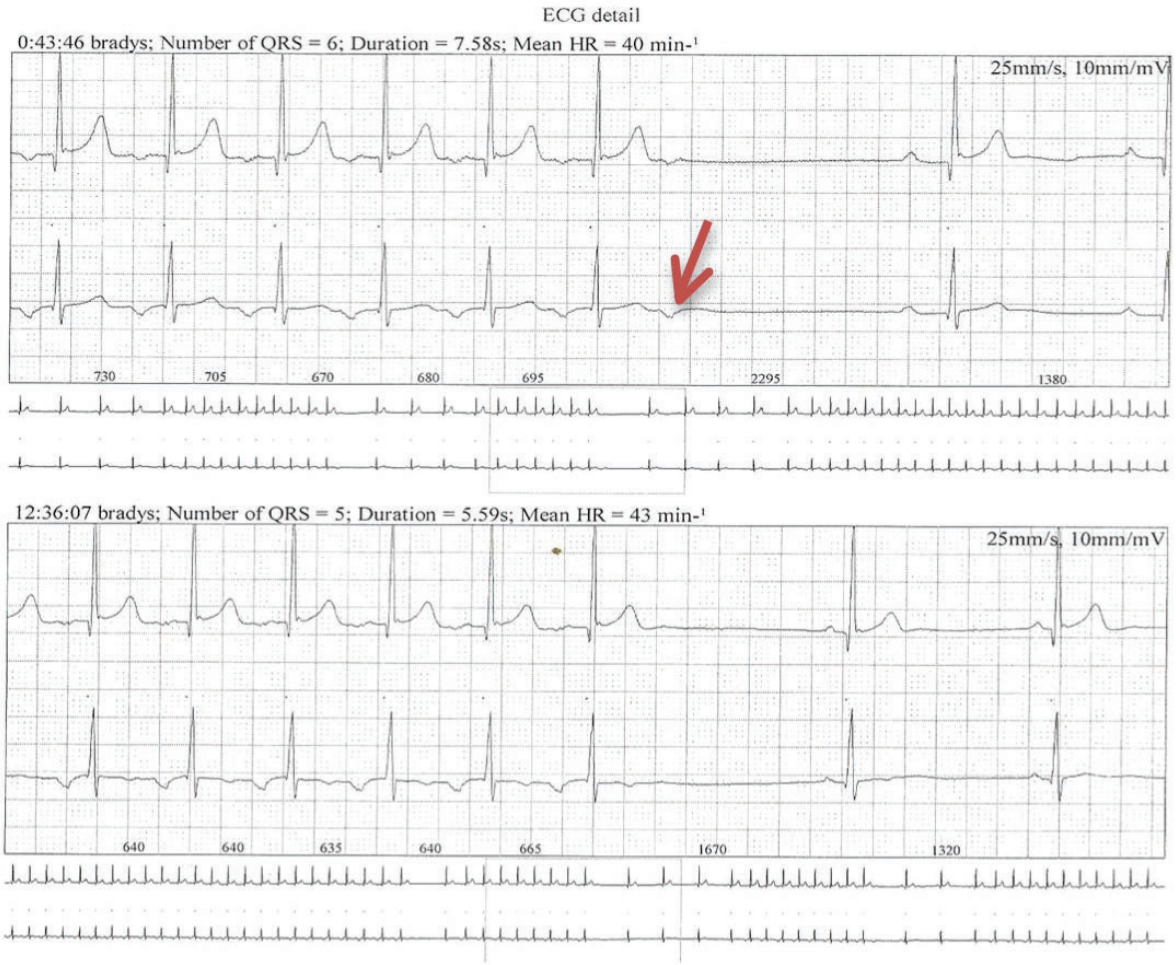

Figure 8C.5.2

Discussion

A 24-hour Holter monitor (**Figure 8C.5.2**) showed repetitive runs of long RP supraventricular tachycardia (SVT). The findings on the Holter monitor, showing SVT termination with (top tracing, arrow) or without (bottom tracing) a P wave, strongly suggest a reentrant mechanism involving the atrioventricular junction. However, at the electrophysiology study, the mechanism was diagnosed as an automatic atrial tachycardia arising from the region of the inferior crista terminalis. This was successfully ablated and the left ventricular size and function normalized within 6 months.

SECTION 8D

Atrioventricular Nodal Reentrant Tachycardia

CASE
8D.1

Bernard Belhassen, MD

Patient History

A 45-year-old woman with a long history of supraventricular tachycardia terminated with intravenous adenosine. During electrophysiologic study, a relatively slow (90/min) supraventricular tachycardia is induced with rapid atrial pacing. **Figure 8D.1.1** shows the intracardiac tracings during tachycardia (paper speed 100 mm/sec). **Figure 8D.1.2** shows 12-lead ECG during the same tachycardia (paper speed 25 mm/sec).

Question

What is the most likely diagnosis of this tachycardia?

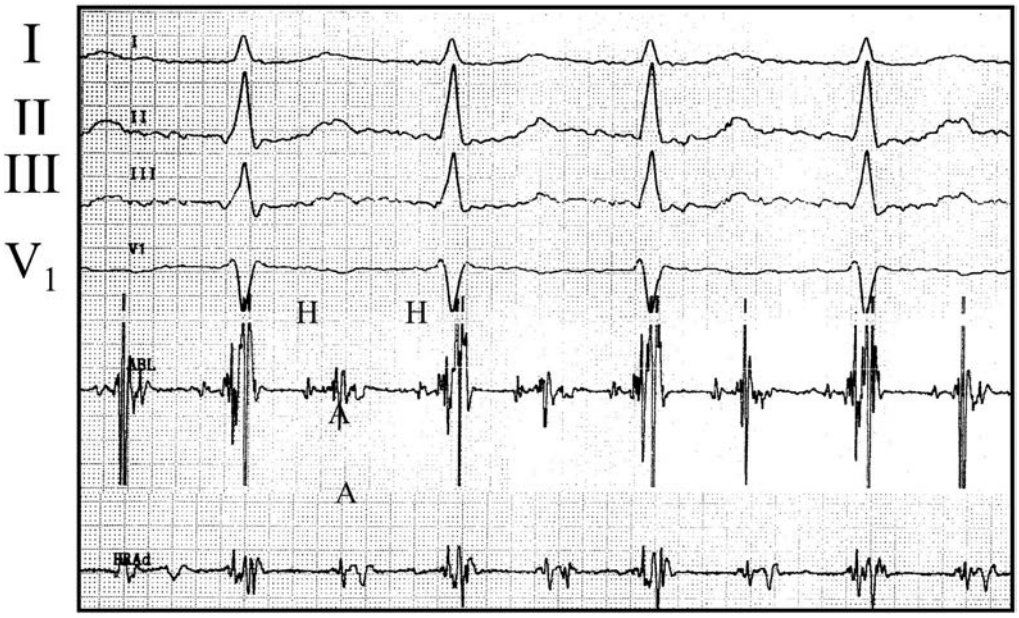

Figure 8D.1.1

ECG Masters' Collection: Favorite ECGs from Master Teachers Around the World, Vol. 2 © 2018 Mohammad Shenasa, Mark E. Josephson, N.A. Mark Estes III, Ezra A. Amsterdam, Melvin Scheinman. Cardiotext Publishing, ISBN: 978-1-942909-20-0.

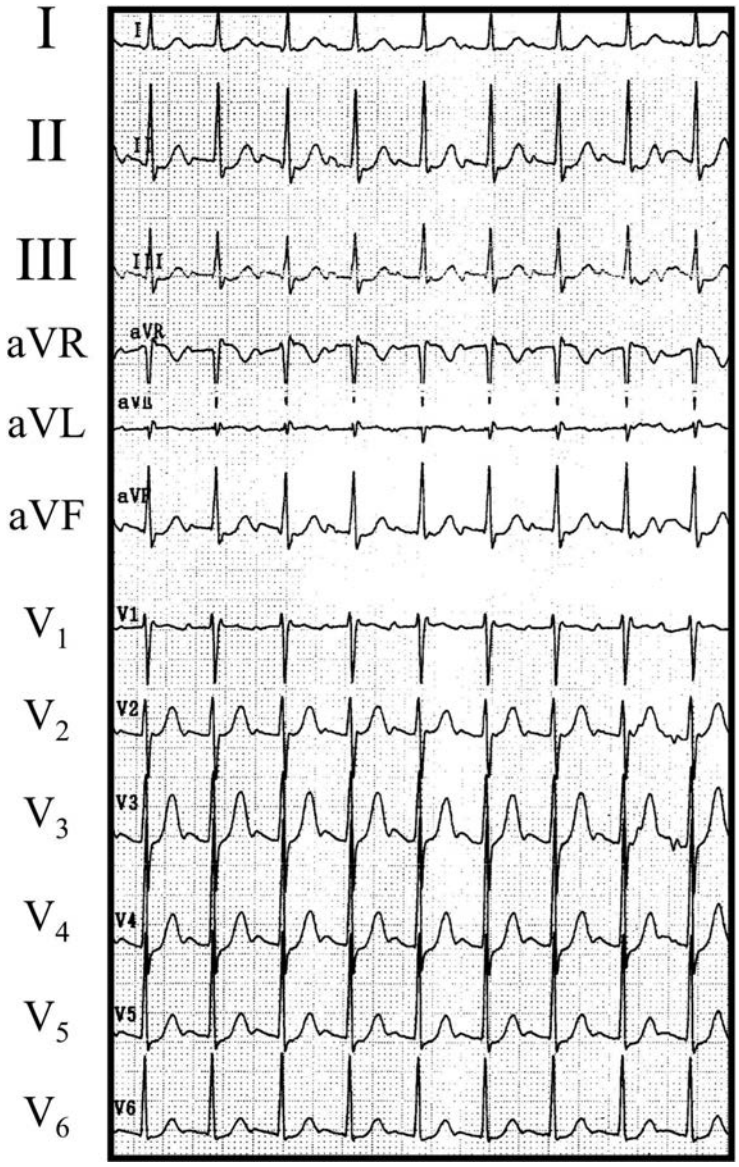

Figure 8D.1.2

Discussion

Figure 8D.1.1 is highly compatible with typical slow/fast atrioventricular nodal reentrant tachycardia (AVNRT) (180/min) associated with 2:1 infranodal block. This type of block is very frequently observed in this type of patient at the onset of induced tachycardias, just for spontaneously disappearing after a few seconds allowing for the resumption of 1:1 AV conduction. **Figure 8D.1.2** mimics sinus tachycardia (90/min) on standard ECG but actually represents the same tachycardia showed in **Figure 8D.1.1**.

CASE
8D.2

Henry H. Hsia, MD

Patient History

A 45-year-old male with intermittent palpitations presented with symptomatic tachycardia of sudden onset, with near syncope. The echocardiogram showed no evidence of structural heart disease.

A 12-lead ECG was obtained in the emergency department (ED) (**Figure 8D.2.1**). The patient later developed sudden onset of rapid palpitations and a tachycardia was documented on the telemetry monitor (**Figure 8D.2.2**).

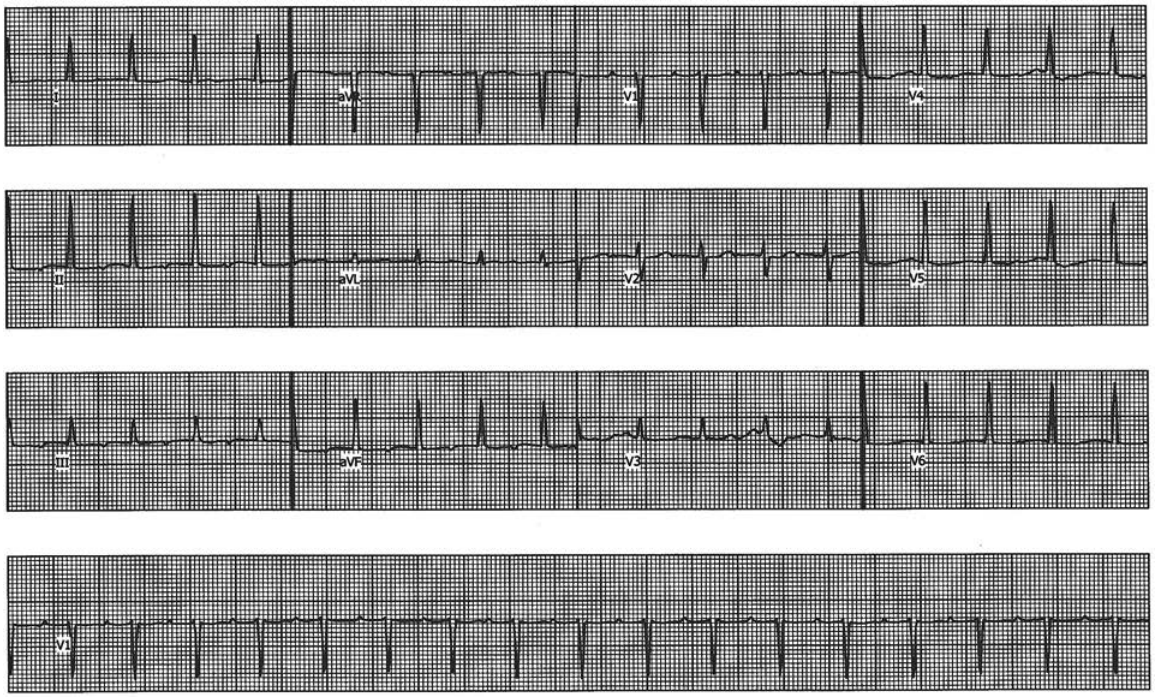

Figure 8D.2.1

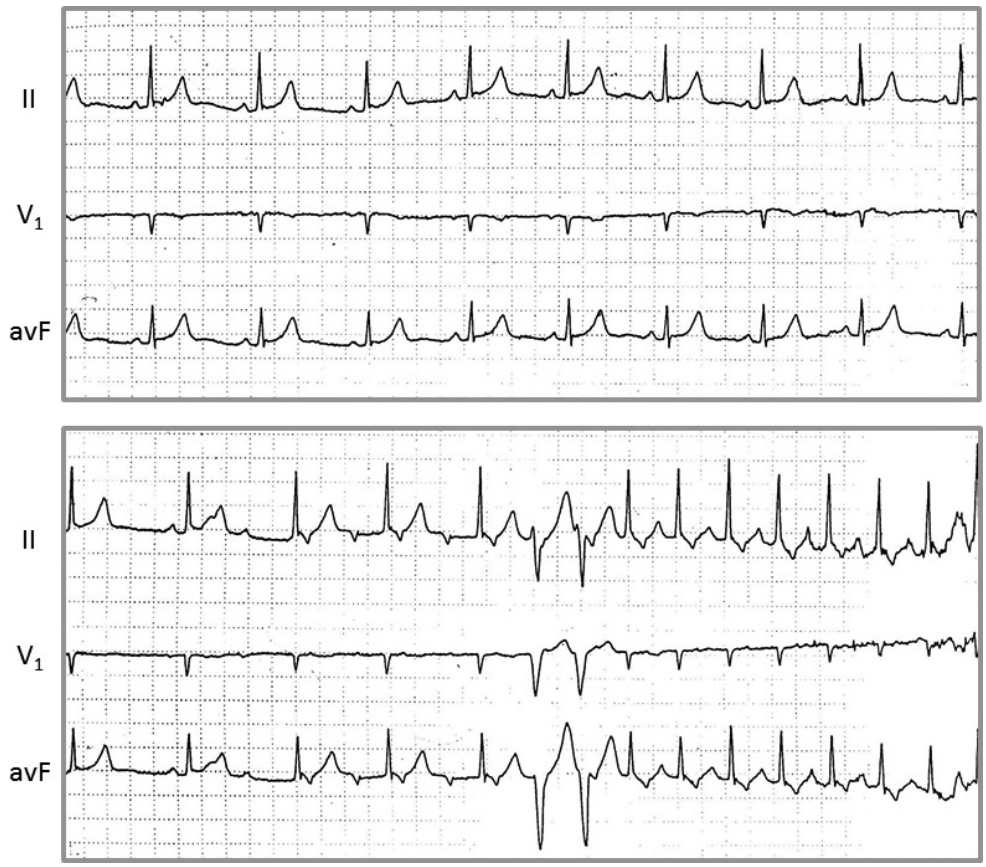

Figure 8D.2.2

Question

What is the diagnosis?

Discussion and Interpretation

Figure 8D.2.1 showed a regular, narrow QRS complex tachycardia at ~110 bpm. The supraventricular tachycardia (SVT) has a normal QRS axis with a nonspecific T-wave abnormality. Possible small narrow P waves were present between the R-R complexes (a "mid-RP" tachycardia) with a long PR interval, best seen in lead V_1.

Figure 8D.2.3 was diagnostic. Top panel of the 3-lead rhythm strip showed a normal sinus rhythm with normal QRS and PR intervals. The lower panel showed the onset of SVT, initiated by two premature atrial depolarizations (PACs) with upright P waves (star). The onset of the SVT was associated with a sudden PR prolongation, followed by what appeared to be narrow retrograde, negative P waves (arrows), with a 2-to-1 RP relationship. The SVT suddenly accelerated and converted to a 1-to-1 RP relationship with a rate at ~165 bpm. Such rate acceleration was accompanied by aberrant conduction, suggestive of "long-short" activation at the His-Purkinje system, and the level of atrioventricular (AV) block was above the His. The mechanism of the SVT was therefore above the His bundle level and at the AV node. These findings were most consistent with a diagnosis of AV nodal reentrant tachycardia (AVNRT), requiring PAC that blocked in the "fast" and conducted down the "slow" AV nodal pathway (sudden PR prolongation) at SVT initiation.

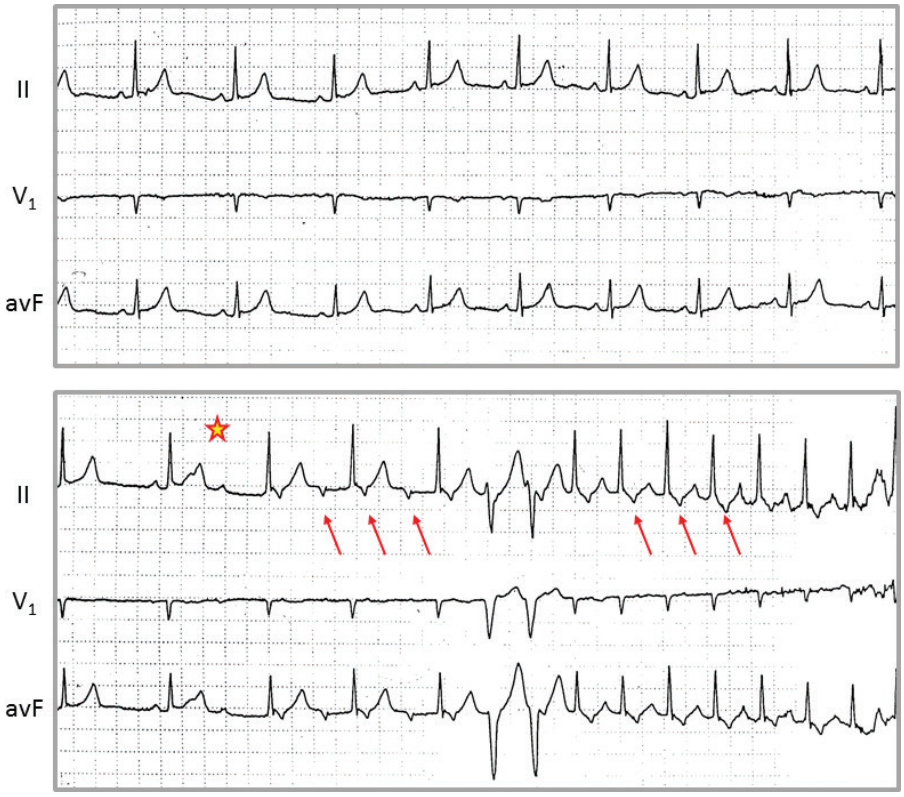

Figure 8D.2.3

The diagnosis was confirmed during the electrophysiology study that demonstrated a "slow–fast" AVNRT with "lower common pathway" block and 2-to-1 RP relationship (arrows) (**Figure 8D.2.4**). The P-P rate was exactly twice of the R-R rate.

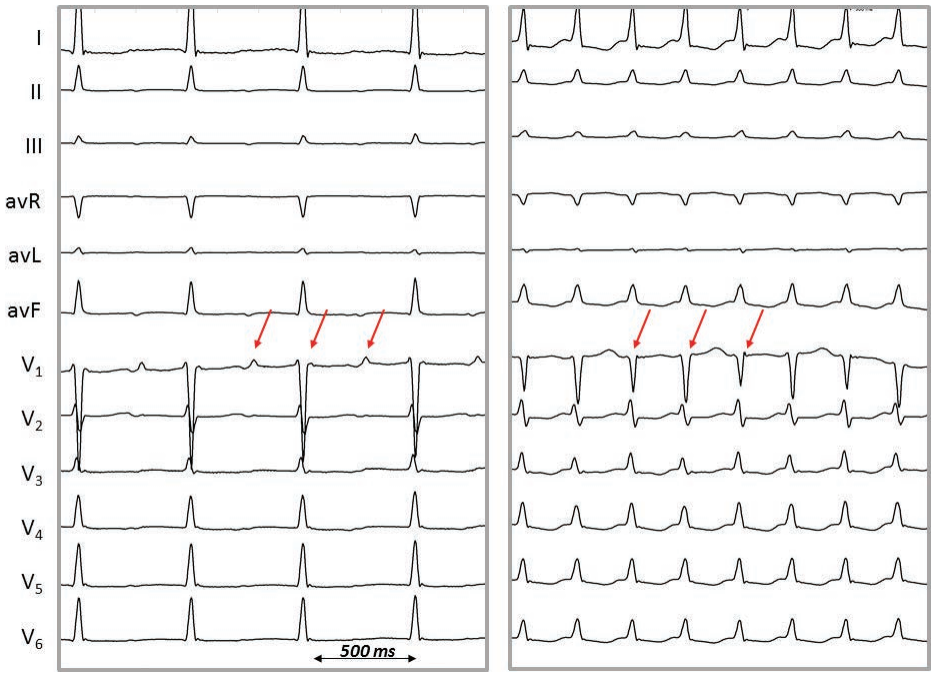

Figure 8D.2.4

CASE 8D.3

Henry H. Hsia, MD

Patient History

A 48-year-old male with coronary artery disease underwent a coronary artery bypass graft surgery. He developed a symptomatic irregular rhythm postoperatively and a diagnosis of atrial fibrillation was made. Anticoagulation was recommended.

The telemetry monitor showed a rhythm strip with the accompanied blood pressure tracings, demonstrating an irregular narrow complex rhythm (**Figure 8D.3.1**).

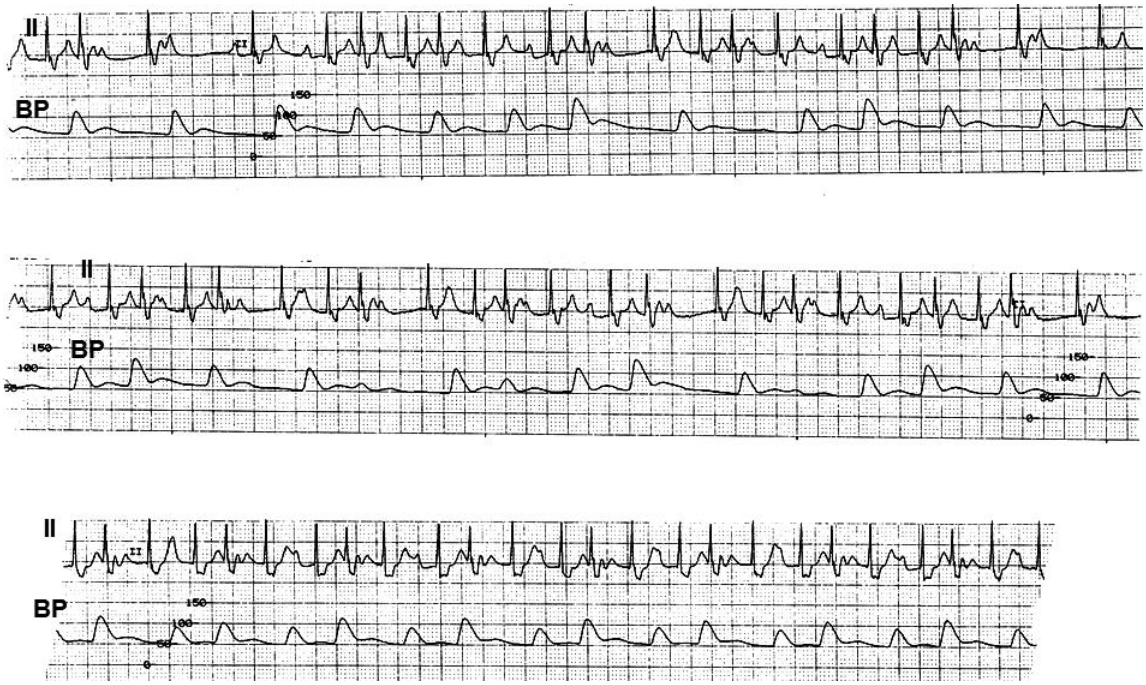

Figure 8D.3.1

Question

Is this atrial fibrillation and how do you confirm the diagnosis?

Discussion and Interpretation

Careful inspection of **Figure 8D.3.1** showed a "regularly irregular" rhythm with what appeared to be discrete P waves; therefore, the diagnosis of atrial fibrillation was excluded. There were more QRSs than P waves with what appeared to be a stable P-to-P interval (arrows). Variable but non-random PR intervals were observed ("fast atrioventricular nodal [AVN] pathway"—red; "slow AVN pathway"—blue) (**Figure 8D.3.2**). A diagnosis of sinus rhythm with 1-to-2 atrioventricular (AV) conduction and multiple AV nodal pathways was made.

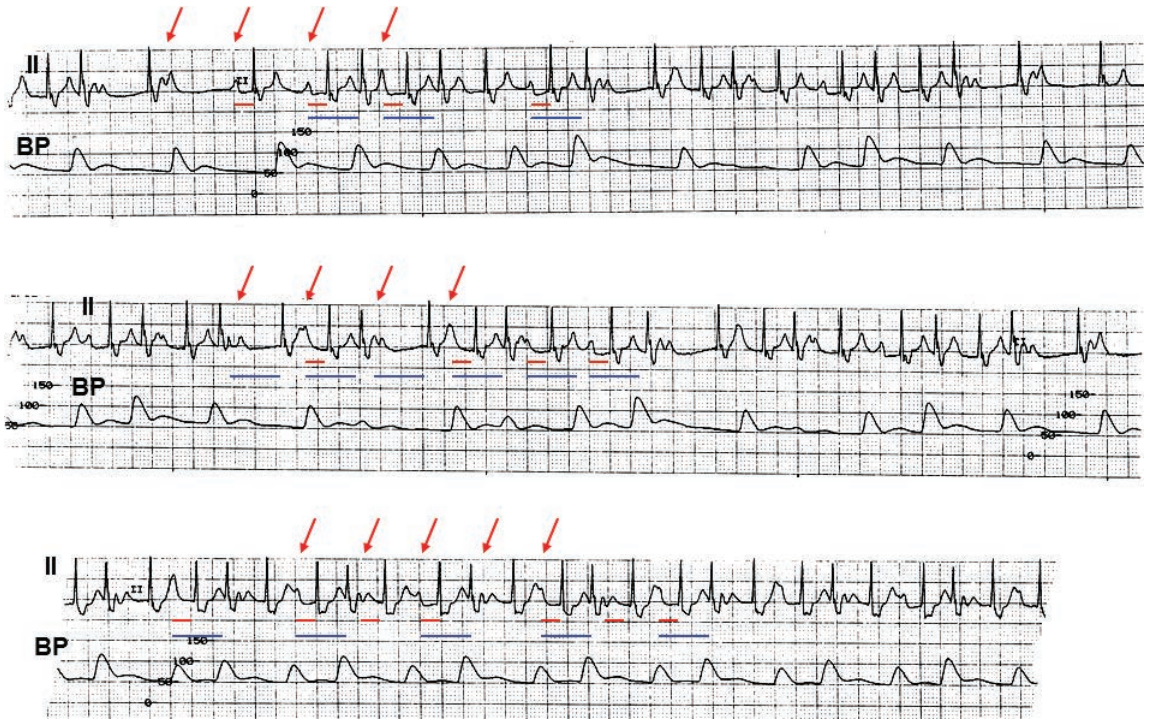

Figure 8D.3.2

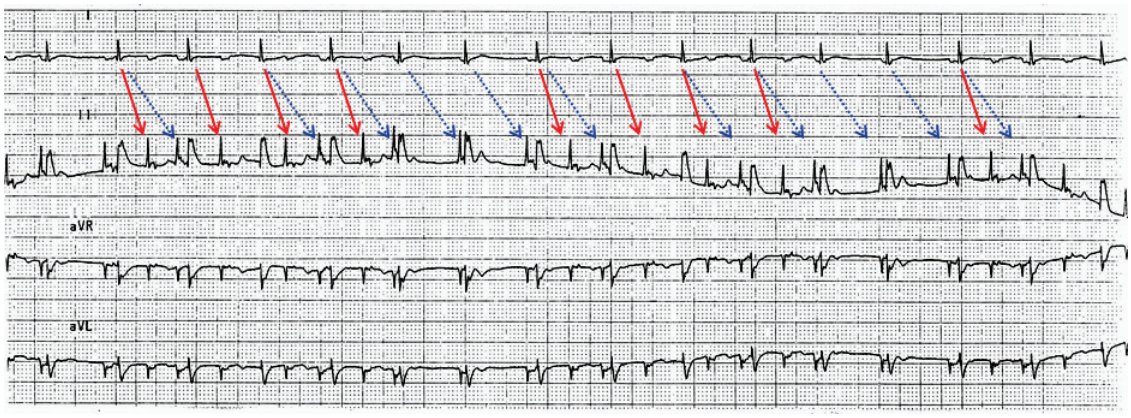

Figure 8D.3.3

In order to confirm the diagnosis, an epicardial atrial electrogram recording was performed by connecting the right arm and left arm ECG electrodes to the two epicardial atrial wires (**Figure 8D.3.3**). Lead I (top tracing: right arm to left arm) depicted the bipolar atrial signals that was regular at ~80 bpm. Leads II, avR, and avL represented hybrid signals recorded between the epicardial atrial wires and body surface. The ventricular signals retained the normal QRS configuration, whereas atrial signals reflected "far-field" recordings. These findings demonstrated a regular sinus rhythm with multiple AV nodal pathways and alternating 1-to-2 AV conduction via the fast (red) and slow (blue) pathways. The patient was not in atrial fibrillation and anticoagulation was discontinued.

CASE 8D.4

Mohammad-Ali Jazayeri, MD
Mohammad-Reza Jazayeri, MD

Patient History

Two patients comprise this scenario. Case 1 is a 62-year-old female who underwent catheter ablation for atrial flutter. She also had a Medtronic LINQ monitor implanted at that time. Two months later, she was noted to have episodes of symptomatic atrial fibrillation recorded on the implantable monitor, and she was started on propafenone at that time. She continued to have arrhythmias while on antiarrhythmic therapy. She underwent pulmonary vein isolation (PVI) and during her postoperative electrophysiologic study (EPS) she developed an episode of arrhythmia shown in **Figure 8D.4.1A**.

Case 2 is a 24-year-old male patient who underwent EPS for recurrent palpitations and an episode of arrhythmia was induced (**Figure 8D.4.1B**).

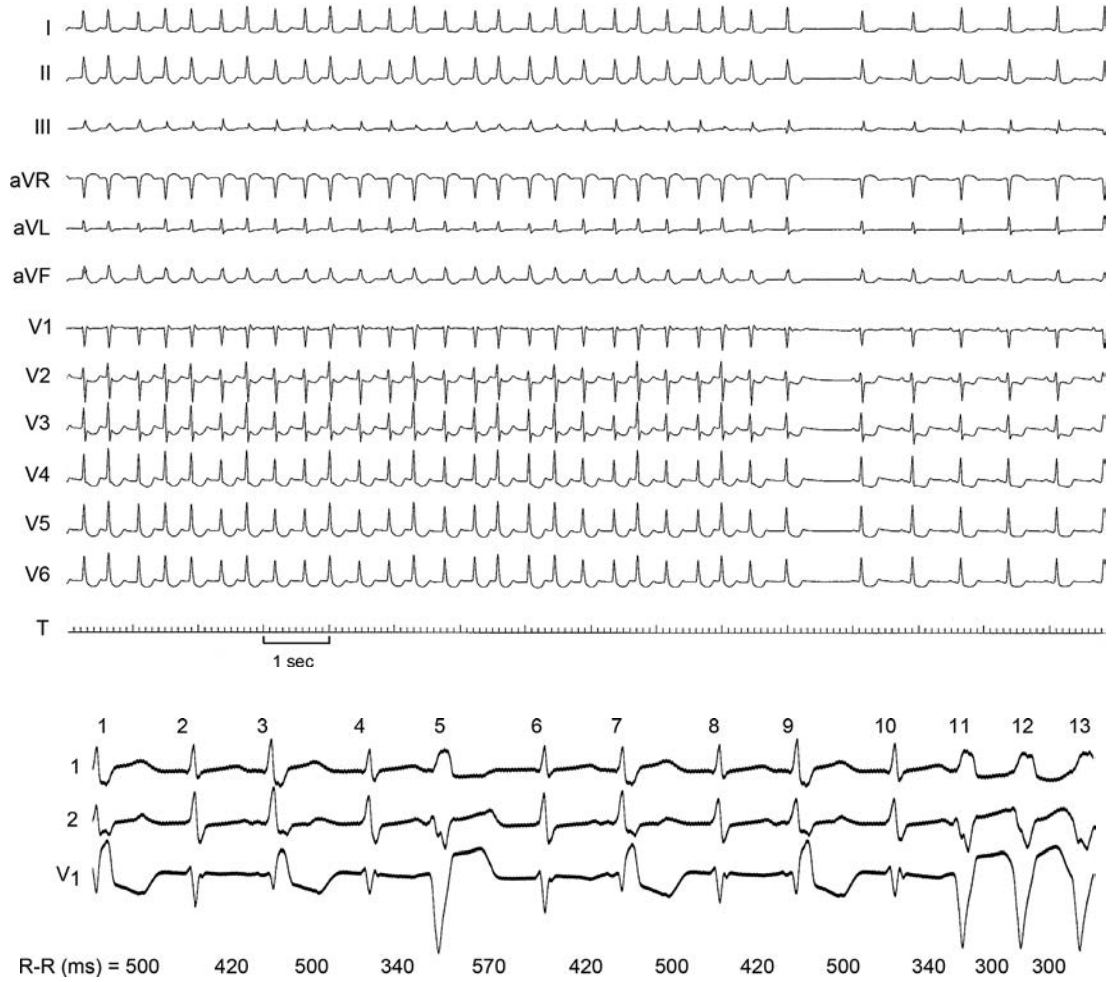

Figure 8D.4.1 Irregular supraventricular arrhythmia. **A.** The last part of a narrow QRS irregular arrhythmia before spontaneous conversion to sinus rhythm. **B.** A segment of irregular supraventricular arrhythmia with frequently occurring aberrant conduction.

Question

What are the most likely arrhythmias shown in **Figure 8D.4.1**?

A. Both are atrial fibrillation (AF)

B. Both are supraventricular tachycardia (SVT)

C. Panel A is AF and panel B is SVT

D. Panel A is SVT and panel B is AF

Answer

Answer B is correct. Case 1 developed typical AV nodal reentrant tachycardia (AVNRT) with a variable cycle length (CL) of 395–480 ms. She appeared to have multiple AV nodal slow pathways and required ablation of a relatively wide area to render her AVNRT non-inducible. **Figure 8D.4.2** is a segment of her clinical arrhythmia, prior to PVI, recorded by her heart monitor showing AF with a CL of 300–480 ms.

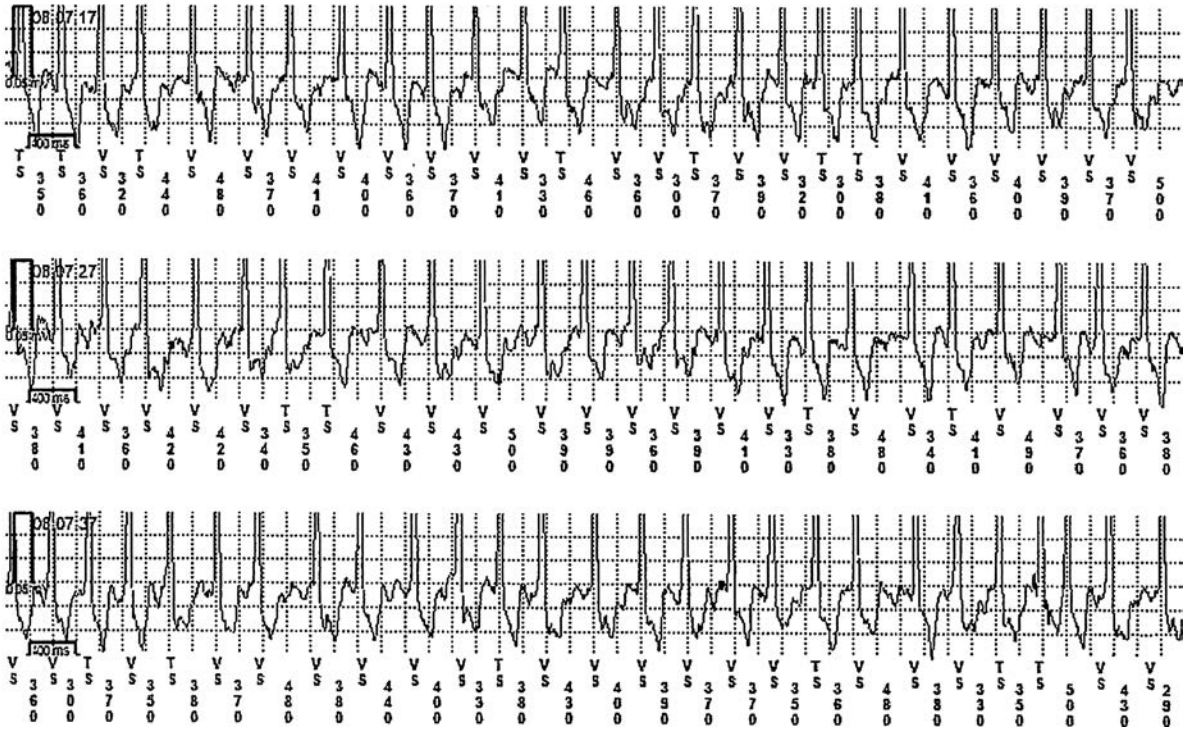

Figure 8D.4.2 Clinical arrhythmia recorded prior to ablation. A snapshot of a bout of arrhythmia recorded by an implantable loop recorder (Medtronic LINQ) showing atrial fibrillation with irregularly irregular ventricular response varying from 300 to 500 ms in cycle length.

Case 2 also had typical AVNRT induced with variable CL from 300 to 500 ms. However, the CL variability in this case was due to the conduction variation below the lower turn-around point (**Figure 8D.4.3**). As seen in **Figure 8D.4.3**, the reason for the variability was functional delay and block in the His-Purkinje system exhibiting a pattern similar to that of Wenckebach periodicity.

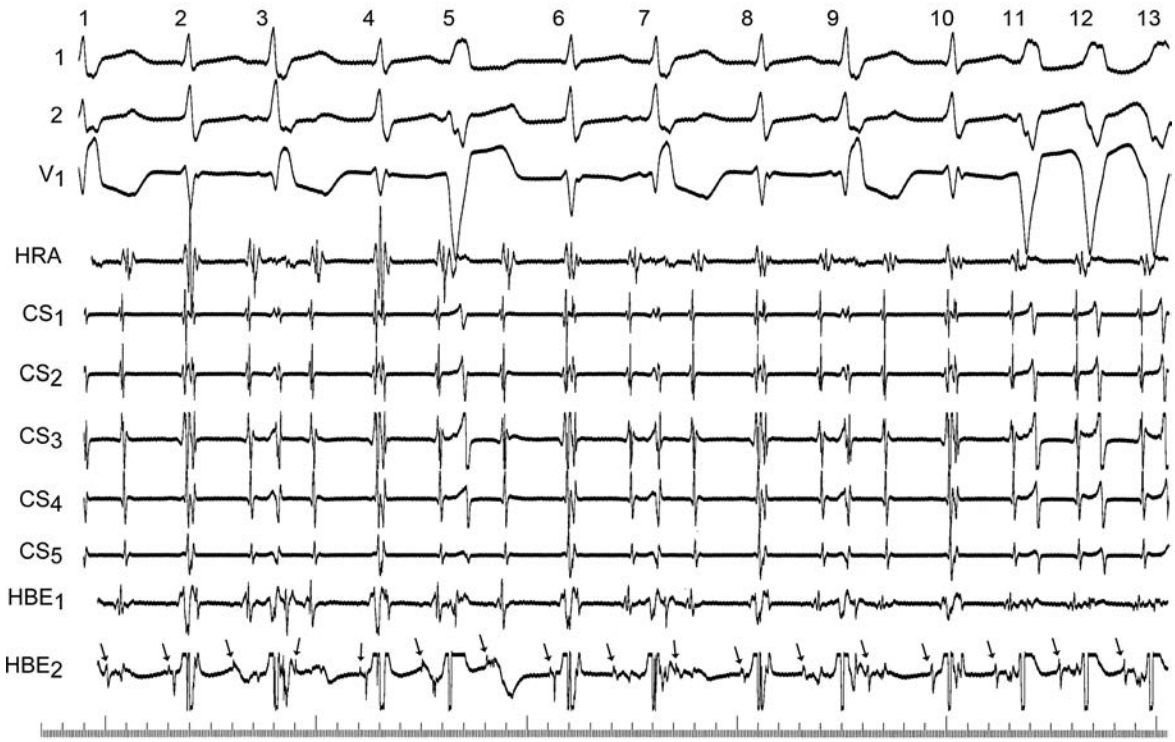

Figure 8D.4.3 AV nodal reentrant tachycardia (AVNRT) with irregular ventricular response. This is the same segment of typical AVNRT episode shown in **Figure 8D.4.1B** but with the advantage of having intracardiac electrograms. The tracings from the top are the ECG leads I, II, and V1 followed by the recordings obtained from high right atrial (HRA), coronary sinus (CS), and His bundle (HB) from proximal to distal sites. Timelines are at the bottom of the panel. Irregular ventricular rate and frequent occurrence of aberrant conduction (Ashman phenomenon) are noteworthy. Note that the atrial cycle length is constant at 300 ms and the irregularity of the ventricular response is primarily due to conduction delay or block in the lower AV nodal tissue, below the turnaround point, or the His-Purkinje system (HPS). This variability of the HPS input sets the stage for the genesis of the aberrant conduction. QRS complexes #1, 3, 7, and 9 show right bundle branch block and complexes #5, 11–13 exhibit left bundle branch block (LBBB). The remaining complexes (#2, 4, 6, 8, and 10) are normally conducted with narrow QRS morphology. The arrows point towards the HB potentials.

Discussion

The ECG hallmark of AF is irregularly irregular ventricular responses. However, the other SVTs can mimic the same pattern and may be misdiagnosed as AF. In a cohort of patients referred for AF ablation, 4.3% were found to have inducible AVNRT.[1] Although irregular ventricular rate during AVNRT or any type of SVT could lead to AF misdiagnosis, and potentially inappropriate AF ablation, our case clearly had both arrhythmias during the most recent EPS and she also had typical atrial flutter that had been previously ablated. It seems reasonable to make an effort and specifically search for inducible SVTs at the beginning of the AF ablation procedure whenever SVT is suspected as a coexistent arrhythmia or a triggering mechanism for AF, particularly in patients less than 40 years old.

Reference

1. Sauer WH, Alonso C, Zado E, et al. Atrioventricular nodal reentrant tachycardia in patients referred for atrial fibrillation ablation: Response to ablation that incorporates slow-pathway modification. *Circulation*. 2006;114:191–195.

Nishant Verma, MD, MPH
Bradley P. Knight, MD

CASE 8D.5

Patient History

This telemetry strip (**Figure 8D.5.1**) was captured from a patient admitted with recurrent palpitations. The patient had multiple, similar events captured on telemetry.

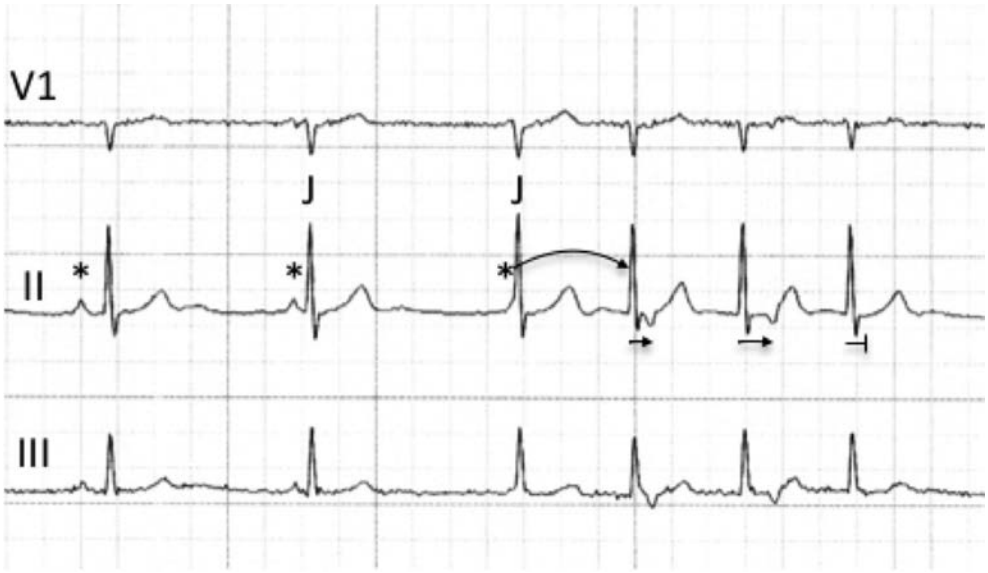

Figure 8D.5.1 Onset and Termination of Nonsustained Paroxysmal Supraventricular Tachycardia. The telemetry strip shows sinus rhythm (*) with two junctional ectopic beats (J). The second junctional beat causes concealed retrograde conduction into the atrioventricular nodal fast pathway. The last sinus beat conducts down the slow pathway (curved arrow) and initiates atrioventricular nodal reentrant tachycardia (AVNRT). There is ventriculoatrial (VA) prolongation (straight arrows) followed by VA block (⊣) and termination of the tachycardia. A decremental, retrogradely conducting pathway cannot be excluded on the basis of this tracing; however, AVNRT was confirmed during an electrophysiology study.

Question

What is the most likely mechanism of the tachycardia and what is the mechanism of initiation?

Discussion

The telemetry strip shows a single beat of normal sinus rhythm. The second beat has a similar P-wave morphology; however, the PR interval is much shorter, likely due to sinus bradycardia with a junctional escape beat. The third beat again shows a junctional escape beat; however, the initial QRS deflection is distorted by a sinus P wave, which appears on time. This is followed by three beats of a narrow complex tachycardia at 100 bpm. A retrograde P wave is seen with the first two beats of tachycardia with ventriculoatrial (VA) prolongation; however, the third beat shows no retrograde atrial activity and the tachycardia terminates. These findings were repeatedly seen during monitoring.

The differential diagnosis for the tachycardia included junctional ectopic tachycardia, atrioventricular nodal reentrant tachycardia (AVNRT), and orthodromic atrioventricular reentrant tachycardia (AVRT). Atrial tachycardia was ruled out since the tachycardia initiates with a junctional ectopic beat. Junctional tachycardia is equally unlikely since the tachycardia reliably terminated with VA block. A decremental, retrogradely conducting pathway cannot be excluded on the basis of this telemetry strip alone. However, the RP interval on the first beat of tachycardia is very short, and typical AVNRT was diagnosed in the electrophysiology laboratory. The patient underwent a successful slow pathway modification with elimination of the tachycardia.

The tachycardia initiation in this case is unusual for AVNRT. Typical AVNRT is most commonly initiated by a premature atrial beat that blocks antegrade in the fast pathway and conducts over the slow pathway, finding the retrograde fast pathway recovered and initiating reentry. In this example, however, a junctional beat initiates the tachycardia. The junctional ectopic beat retrogradely penetrates the fast pathway and results in the coincident sinus beat conducting over the antegrade slow pathway and initiating tachycardia. This mechanism of initiation was more common in the past when digoxin was commonly used for treatment of supraventricular tachycardias. At high doses, digoxin can accelerate the junctional rate while slowing the sinus rate.

SECTION 8G

Junctional Rhythms

CASE
8G.1

N.A. Mark Estes III, MD

Patient History

Figure 8G.1.1 shows the ECG of a 51-year-old female with exercise intolerance, neck pulsations, and headaches.

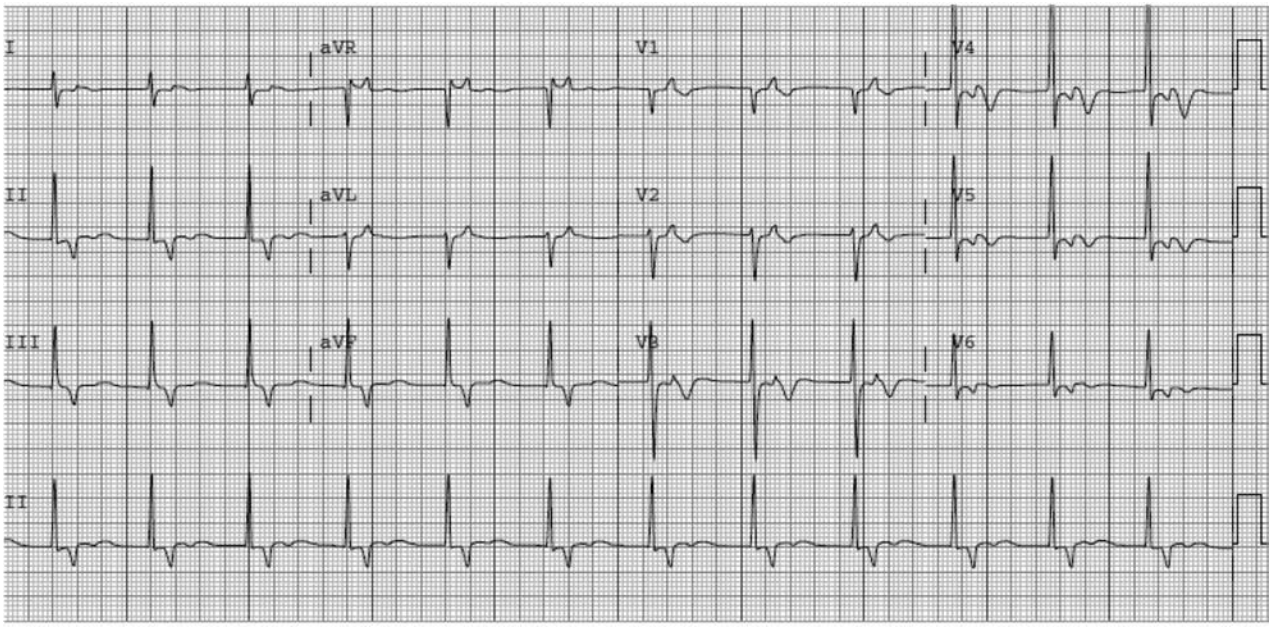

Figure 8G.1.1

Question

What is the ECG abnormality?

Discussion

The ECG demonstrates an accelerated junctional rhythm with retrograde P waves, which are inverted in leads II, III, and aVF and are upright in leads I and aVL. Holter monitoring demonstrated persistent junctional rhythm with rates of 75–140 bpm. Atrial contraction immediately after ventricular contraction resulted in adverse hemodynamic effects and marked "cannon a waves" in the internal jugular pulse on physical examination.

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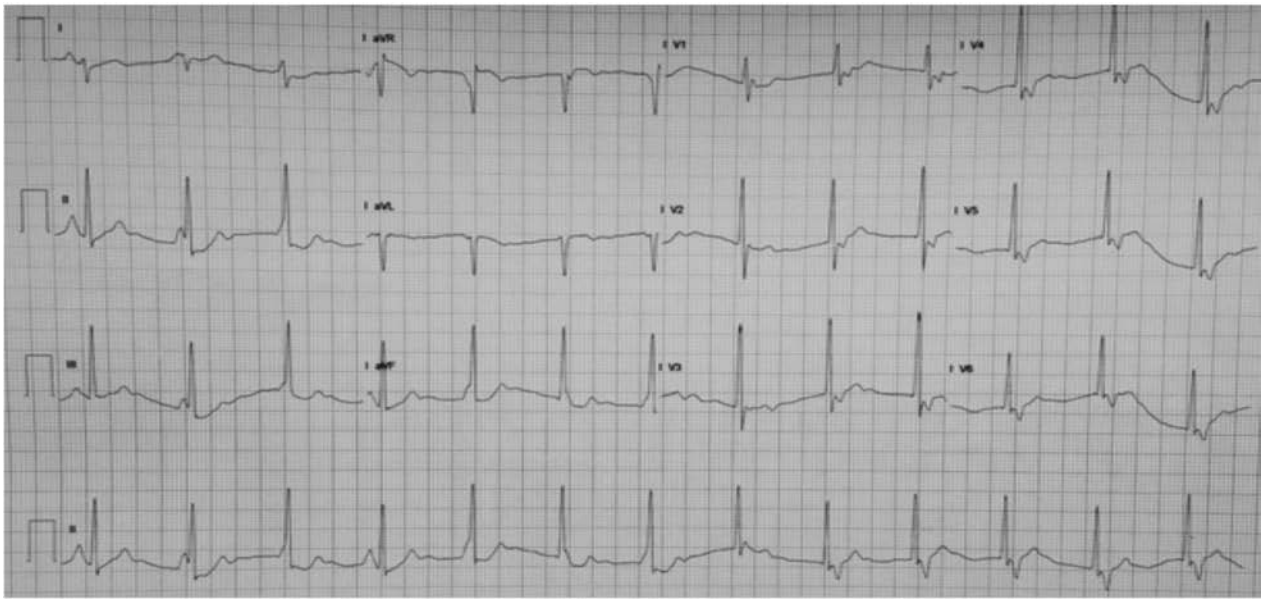

Figure 8G.1.2 ECG of a 51-year-old female shown in **Figure 8G.1.1** during recovery from exercise.

Question

What is the rhythm on this ECG (**Figure 8G.1.2**)?

Discussion

The ECG shows sinus rhythm initially, followed by fusion of sinus and junctional rhythm, and finally junctional rhythm with retrograde P waves. Concealed anterograde conduction of the sinus beats prevents retrograde conduction of the junctional rhythm to the atrium in the mid portion of the tracing. Cryoablation of the junctional rhythm resulted in normal sinus rhythm, improved exercise tolerance, and improvement in all symptoms.

SECTION 9A

Ventricular Tachycardia/Fibrillation

CASE
9A.1

Robert Frank, MD

Patient History

A 25-year-old female presents with a long history of nonsustained tachycardia with sudden onset and sudden cessation, often triggered by exercise or stress. She has a normal physical exam, echocardiogram, and rest ECG. An electrophysiology (EP) study failed to detect any abnormalities or inducible arrhythmia. A spontaneous ECG was recorded in **Figures 9A.1.1** and **9A.1.2**.

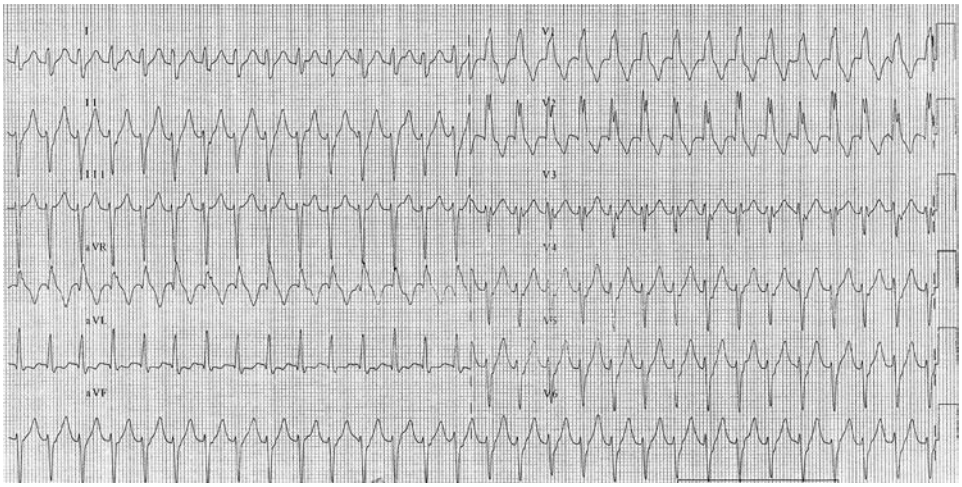

Figure 9A.1.1

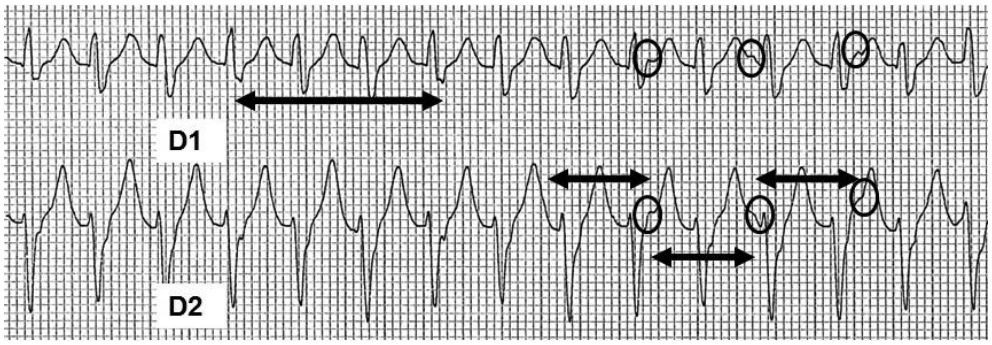

Figure 9A.1.2 Circles are around the dissociated P waves. The large arrow points to the small changes in morphology. The small arrows indicate the P waves. P waves were not apparent in the preceeding cycles, probably masked by a retrograde ventriculoatrial conduction.

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Questions

1. What is this tachycardia?
2. Where does the tachycardia come from?

Discussion, Interpretation, and Answers

This tachycardia with wide, 120-ms QRS has a special morphology of a right bundle branch block (RBBB) with a left-axis deviation (left anterior hemiblock). There is also a small shift in axis every three beats, with a notch in the ascending part of the negative wave in D2.

P waves cannot be seen during the first cycles of the tracing; however, they appear with the last complexes, dissociated from the QRS (**Figure 9A.1.2**), which points towards a diagnosis of ventricular tachycardia.

The QRS morphology with a right bundle branch block (RBBB) with a left anterior hemiblock is characteristic of a tachycardia originating in the Purkinje system, and more specifically a fascicular tachycardia. It occurs between the left hemibranches, usually descending through the left posterior one. This tachycardia is sometimes also called Belhassen's tachycardia, who recognized its mechanism and showed it to be verapamil sensitive. In this case, the small changes in morphology may be due to alternating circuits in two ramifications of the posterior fascicle to explain the slight QRS alternation.

After ablation in the left posterior region on a Purkinje potential, a shift appeared in the QRS frontal plane, suggesting a left posterior hemiblock (**Figure 9A.1.3**).

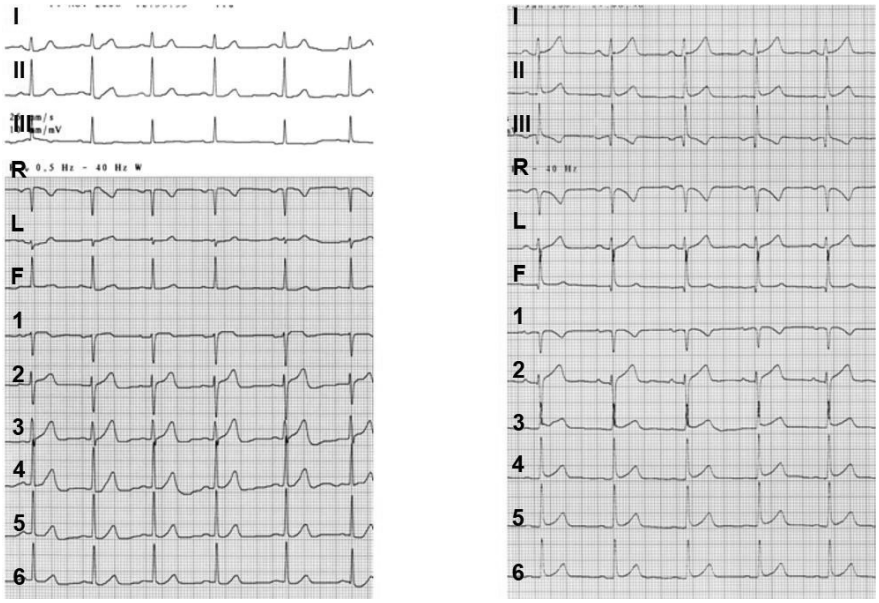

Figure 9A.1.3 Frontal ECG before and after ablation.

References

1. Belhassen B, Rotmensch HH, Laniado S. Response of recurrent sustained ventricular tachycardia to verapamil. *Br Heart J.* 1981;46(6):679–682.
2. Nogami A. Purkinje-related arrhythmias part I: Monomorphic ventricular tachycardias. *Pacing Clin Electrophysiol.* 2011;34(5):624–650.
3. Katritsis D, Heald S, Ahsan A, et al. Catheter ablation for successful management of.posterior fascicular tachycardia: An approach guided by recording of left fascicular potentials. *Heart.* 1996;75:384–388.

CASE
9A.2

Robert Frank, MD

Patient History

A 28-year-old patient had a cardiac arrest while running and was fortunately resuscitated. A right ventricular dysplasia was found. The patient was treated with 5 mg of bisoprolol and received an implantable cardioverter-defibrillator (ICD). One year later, an appropriate shock occurred, again while running. The ICD tracing showed an atrial fibrillation with very short RR intervals and a short-coupled premature ventricular contraction (PVC)-induced ventricular tachycardia (VT) (**Figure 9A.2.1**).

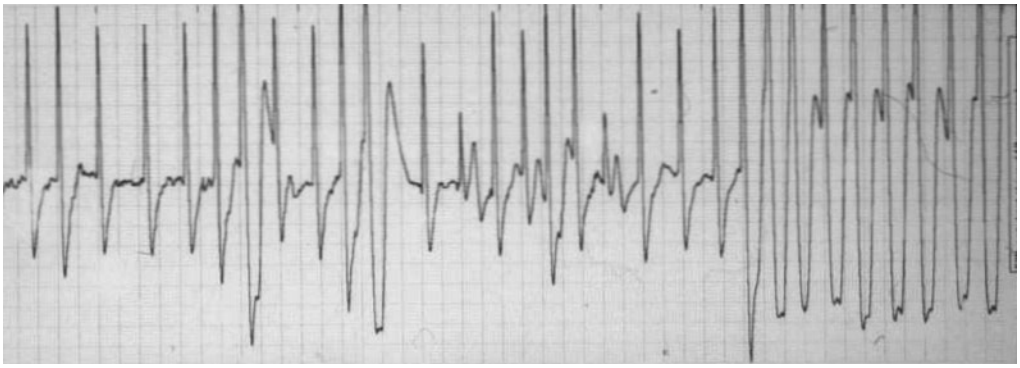

Figure 9A.2.1

The patient was then monitored, and VT occurred at rest (**Figure 9A.2.2**).

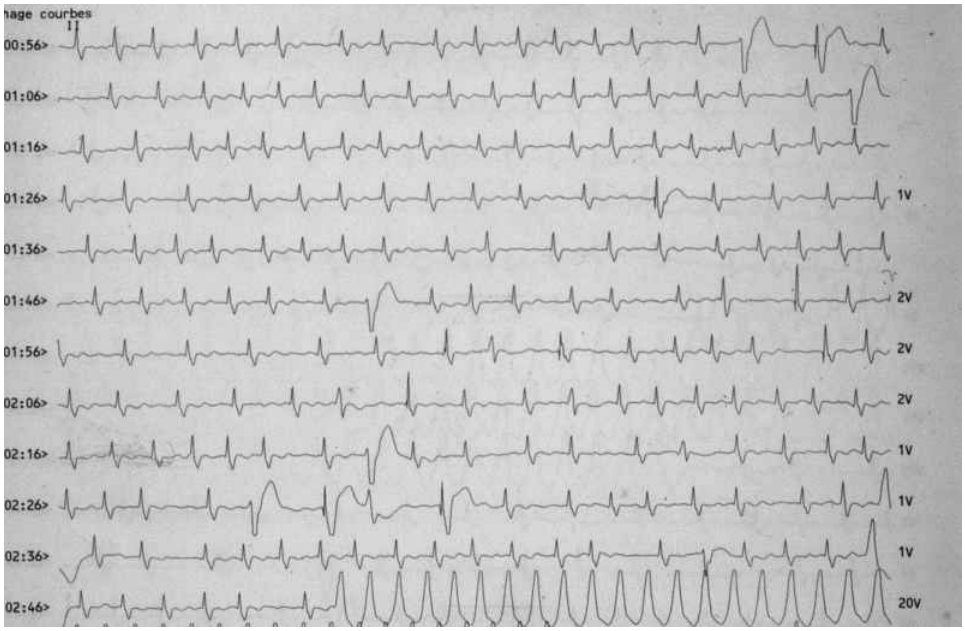

Figure 9A.2.2

Continuous tracing shows atrial fibrillation and PVCs.

Question

What are the conditions for VT induction?

Discussion

This one lead monitor tracing shows PVCs with two morphologies. The first ones are negative and do not induce VT. A second morphology (positive) appears at the end of the tracing, and induces a VT with the same morphology, but only at its third occurrence. This can be explained by the sequence of the ventricular AF cycles, a short one, followed by long, and then by a short-coupled second morphology PVC. Myocardial and Purkinje refractory periods change on abrupt cycle length changes, allowing conditions for local reentry. Several conditions are needed for VT induction in this case: the appropriate PVC, and a short coupling interval on a previous long cycle.

Different types of VT onset can be observed in the same patient, one catecholaminergic, while running with a fast supraventricular rate, and the other at rest, related to the irregular atrial fibrillation cycles.

References

1. Denker S, Lehmann M, Mahmud R, et al. Divergence between refractoriness of His-Purkinje system and ventricular muscle with abrupt changes in cycle length. *Circulation*. 1983;68:1212–1221.
2. Sweeney MO, Ruetz LL, Belk P, et al. Bradycardia pacing-induced short-long-short sequences at the onset of ventricular tachyarrhythmias: A possible mechanism of proarrhythmia? *J Am Coll Cardiol.* 2007;50(7):614–622.

Mohammad-Ali Jazayeri, MD
Mohammad-Reza Jazayeri, MD

CASE 9A.3

Patient History

Two cases are included in this scenario. Case 1 is a 31-year-old female who presented with recurrent palpitations and documented narrow QRS tachycardia. Administration of adenosine and verapamil intravenously on two separate occasions terminated her tachycardia. She had no history of heart disease or syncope and had a normal echocardiogram. She underwent a complex electrophysiology (EP) study. **Figure 9A.3.1A** shows a 12-lead ECG obtained during one of the induced episodes. **Figure 9A.3.1B** is her baseline ECG obtained during EP study. **Figure 9A.3.2** is from a 59-year-old male with a history of alcohol abuse who presented with recurrent palpitations. An echocardiogram showed mild left ventricular hypertrophy with some diastolic dysfunction and normal systolic function. **Figure 9A.3.2** is an episode of spontaneous tachycardia while the patient was monitored on telemetry. **Figure 9A.3.3** was obtained during EP study while he was on isoproterenol infusion at 2 mcg/min.

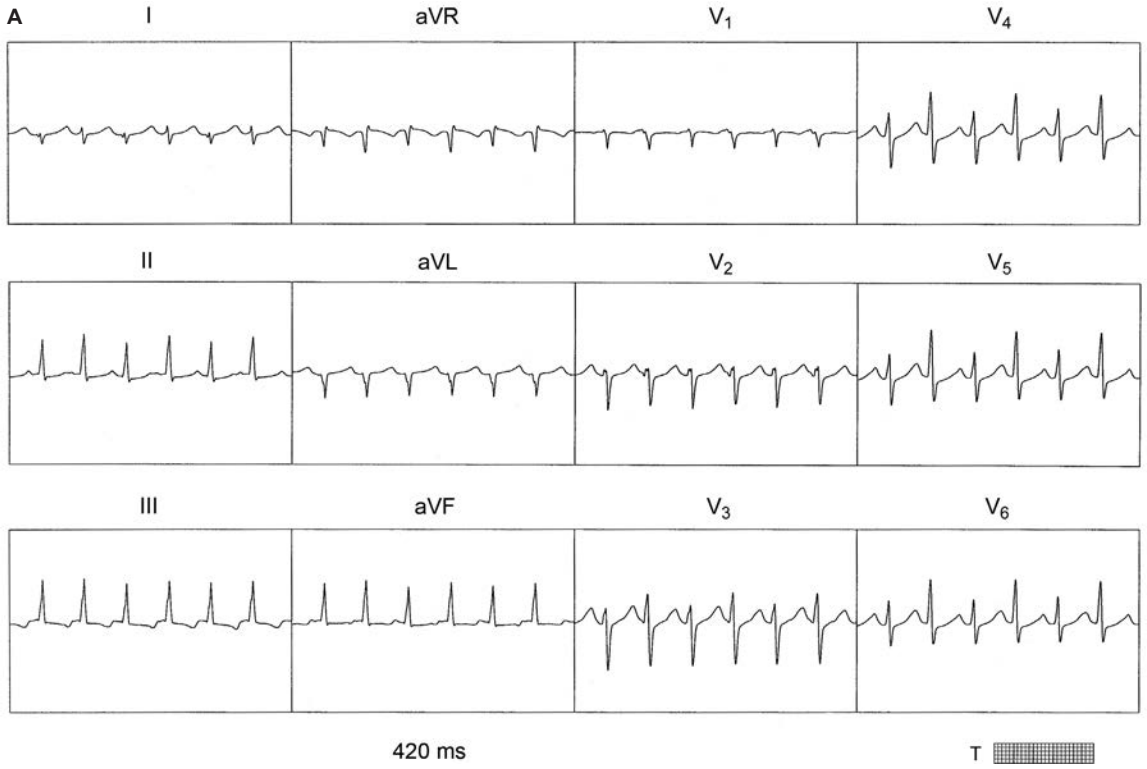

Figure 9A.3.1 Narrow QRS tachycardia (NQRST) with a shift of QRS axis. **A.** 12-lead ECG of NQRST with a cycle length (CL) of 420 ms and no visible P waves. The QRS complex in lead I is predominantly negative, which denotes a right axis deviation, probably due to the presence of left posterior fascicular block. Furthermore, QRS alternans are present in leads I, II, aVR, and V_2–V_6.

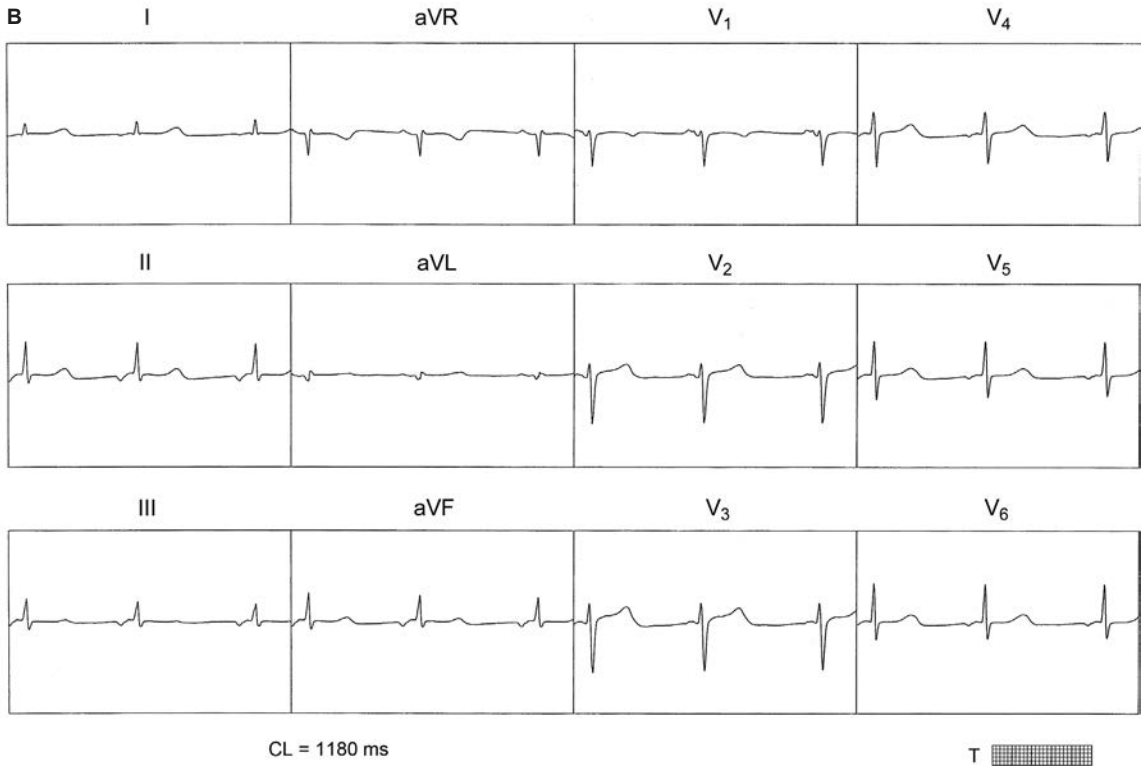

Figure 9A.3.1 **B.** An ectopic atrial bradycardia with a CL of 1180 ms. The QRS axis and morphology appear normal. Comparison of the two tracings show slight differences in the QRS morphology in leads I, III, aVL, aVF, V_1, and V_2. T: time scale.

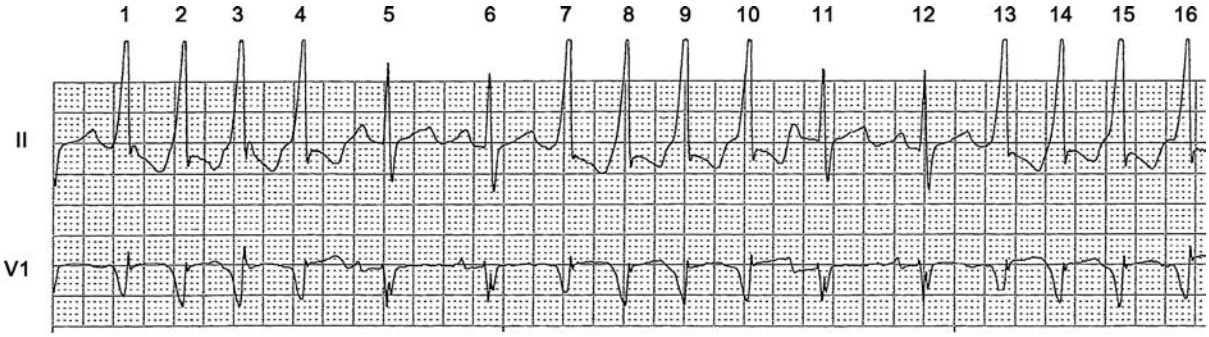

Figure 9A.3.2 Four-beat runs of wide QRS tachycardia (WQRST). A rhythm strip capturing leads II and V_1 shows nonsustained runs of WQRST with an average cycle length of 400 ms, separated by two sinus beats. Some of the wide QRS complexes (#2, 3, 8–10, 14, and 15) have a slurred deflection at their beginning. Also note that the PR interval of the sinus beats #5 and 11 are slightly longer than the other sinus beats (#6 and 12). See the text for further explanation.

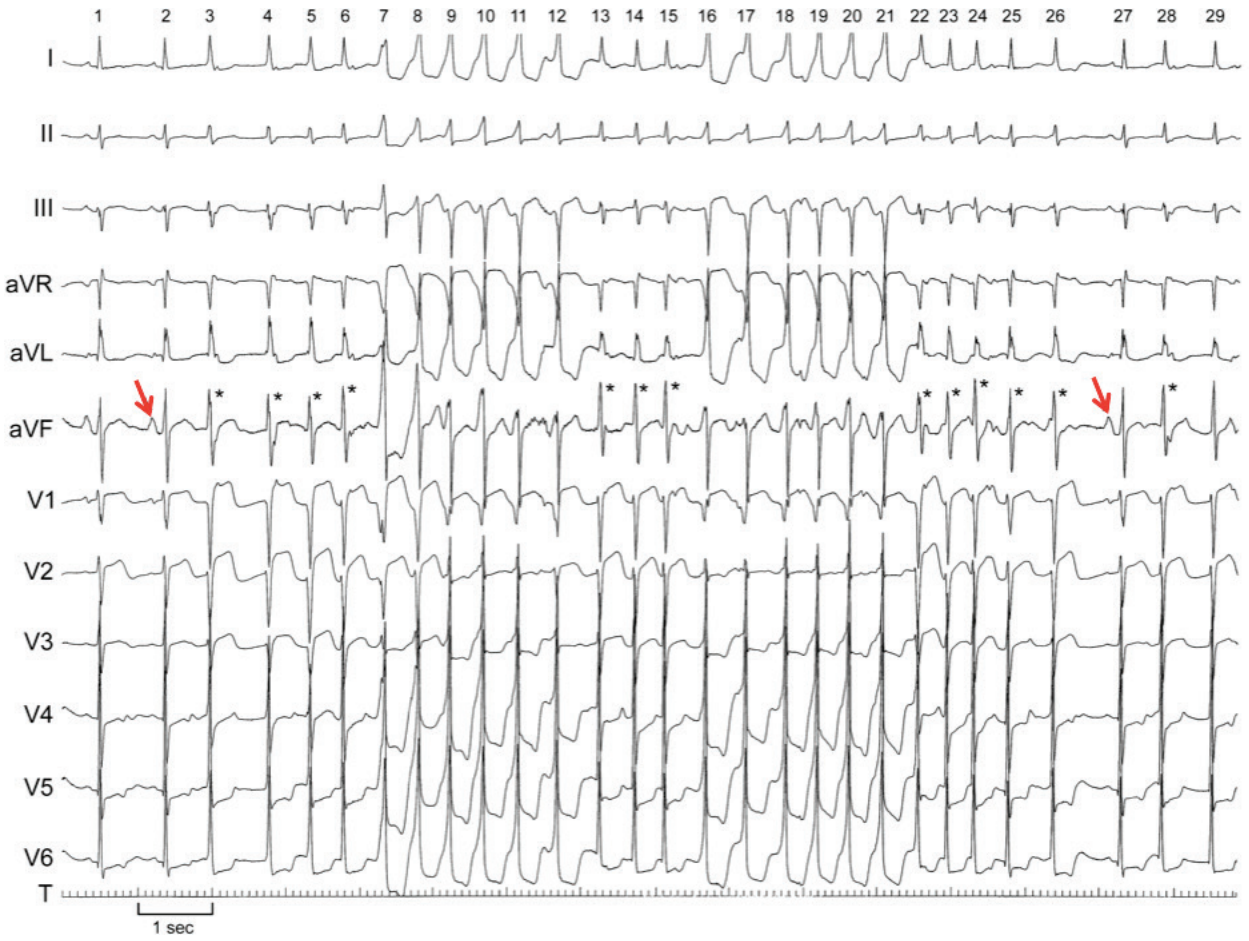

Figure 9A.3.3 A run of tachycardia showing wide and narrow QRS complexes. A 12-lead ECG of a run of tachycardia between two sinus beats (arrows) is shown. Of note, the presence of both wide QRS complexes and narrow QRS complexes (asterisks) are intermingled with each other.

Question

Which of the following best explains the arrhythmias shared in these two patients?

A. Atrioventricular nodal reentrant tachycardia (AVNRT)

B. Accessory pathway-related arrhythmias (APRA)

C. Ventricular tachycardia (VT)

D. AV nodal nonreentrant tachycardia (AVNNRT)

Discussion, Interpretation, and Answer

Answer C is correct. It should be pointed out that both panels in **Figure 9A.3.1** were obtained during an EP study without any change in the lead placement. The following observations are worthy of mention: (1) The QRS duration is narrow (approximately 80 ms) in both panels; (2) The QRS complex morphology is slightly different in leads I, III, aVL, V_1, and V_2; (3) The QRS axis is shifted to the right during tachycardia; and (4) QRS alternans are present in panel A. Except for the last point, the others favor VT as the mechanism of tachycardia in panel A. Persistent isolated axis deviation due to sustained functional fascicular block is extremely rare during supraventricular

tachycardia (SVT). Such an axis shift may be explained by close proximity of the VT exit site to the left anterior fascicle (LAF). If such proximity is to the extent that the impulses enter the right bundle branch (RBB) before they complete their course along the LAF, a nearly simultaneous activation of both BBs gives rise to a narrow QRS complex. Therefore, just by a careful analysis of the ECG during tachycardia and comparing its QRS morphology with that during baseline supraventricular rhythm, one may arrive at the correct diagnosis. The EP study also confirmed that this was a VT originating within or in the vicinity of the proximal LAF,[1] where the LAF potentials could be identified (**Figure 9A.3.4**). As shown in this figure, VT was accompanied by atrioventricular dissociation. Of note, atrial as well as ventricular burst pacing reproducibly induced VT, which was in accordance with its reentrant nature.

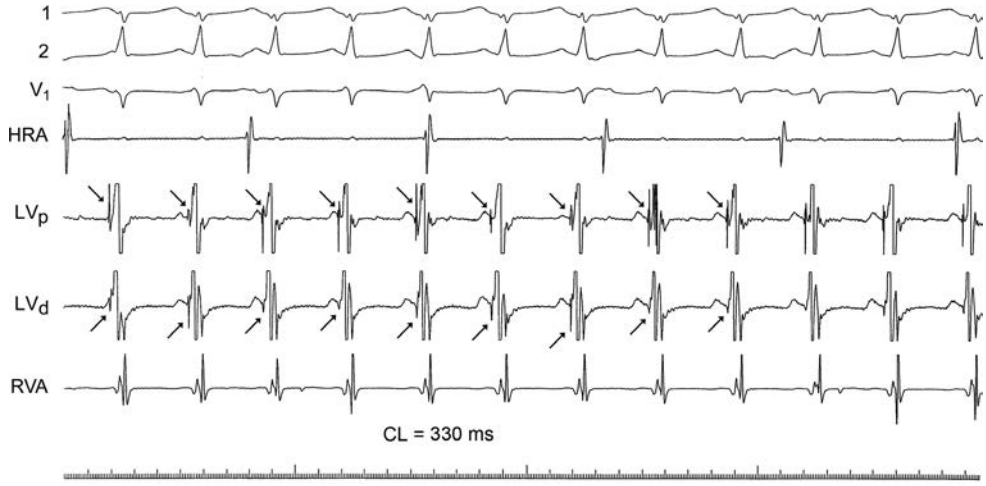

Figure 9A.3.4 Narrow QRS fascicular tachycardia. Tracings from top to bottom are ECG leads I, II, and V_1, followed by high right atrial (HRA), proximal (p) and distal (d) left ventricular (LV), right ventricular apical (RVA) electrograms, and time lines. An episode of narrow QRS complex tachycardia with a cycle length (CL) of 330 ms is shown. Note the presence of AV dissociation lends support to the diagnosis of ventricular tachycardia. Left anterior fascicular potentials (arrows) are inscribed 30 ms earlier than the beginning of the ventricular activation.

In the second case, **Figure 9A.3.2** shows grouped beating, sinus beats separated by 4-beat runs of wide QRS tachycardia (WQRST). The initial portion of some wide complexes is slightly slurred, suggestive of a "delta wave." However, the following clues argue against ventricular preexcitation and APRA (answer B) in this tracing: (1) The PR interval is normal during sinus beats; (2) the PP interval of two consecutive sinus beats can be measured out, as they remain uninterrupted despite a burst of WQRST; and (3) the PR interval of each sinus beat following the last beat of WQRST is slightly prolonged, which is most likely due to the retrograde concealment of the last beat into the AV node. Thus, the most likely diagnosis is nonsustained VT. **Figure 9A.3.3** shows the same nonsustained runs of VT provoked by isoproterenol in the EP laboratory. Additionally, there are several runs of nonsustained narrow QRS complex tachycardia (NQRST) intermingled with the WQRST runs. It is impossible to ascertain the origin of these NQRST runs without having intracardiac ECG recordings. **Figure 9A.3.5** depicts the mechanism of these beats as being AVNNRT (answer D). This entity is due to sequential conduction of at least some of the sinus beats over two (or more) AV nodal pathways.[2] Although AVNNRT in this case is independent of VT, the retrograde concealment of the ventricular complexes during VT may be a facilitating factor at times for the development of the former.

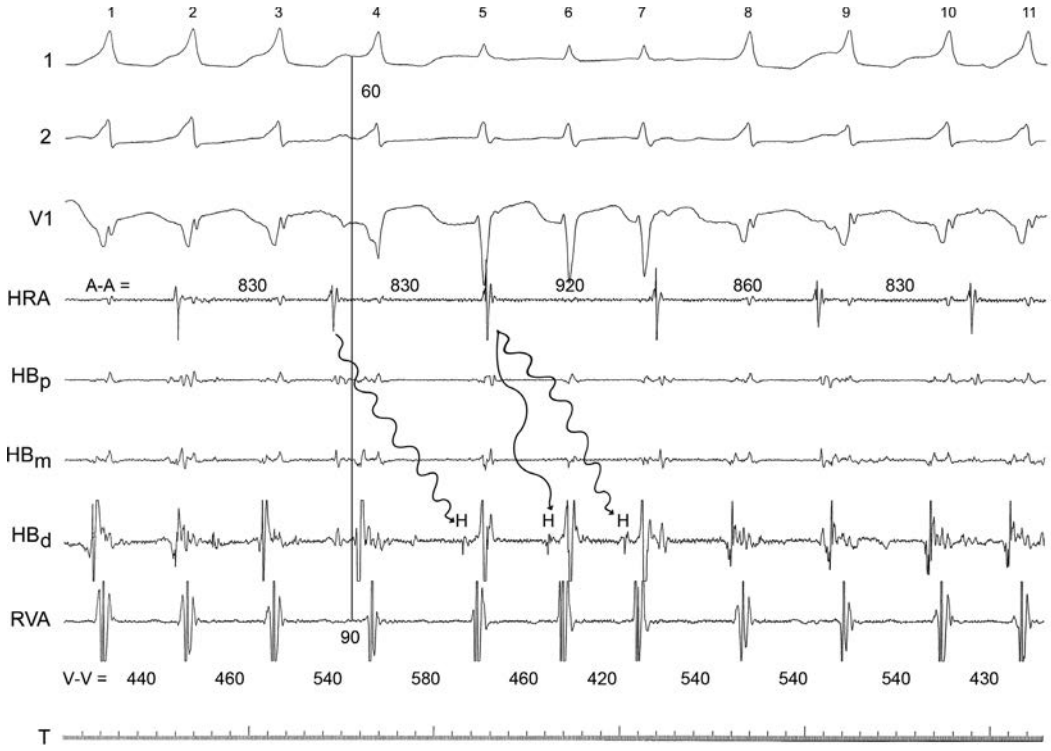

Figure 9A.3.5 Further analysis of wide and narrow QRS tachycardia by intracardiac electrograms. Tracings from the top are surface ECG leads I, II, and V_1 followed by intracardiac electrograms obtained from high right atrial (HRA), proximal (p), medial (m), and distal (d) His bundle (HB), and right ventricular apex (RVA) positions. T: timelines. Wide QRS complexes (#1–4 and 8–11) with cycle length (CL) of 430–540 ms are separated with a three-beat run of narrow QRS complex tachycardia (#5–7). Ventricular activation in the latter group is preceded by an HV interval of 40 ms, supporting a supraventricular origin for these impulses, most likely originating from sinus beats with single and double (sequential) responses over dual AV nodal pathways. The former group, however, by lacking AV association and HB potentials, is most likely ventricular in origin. Note also that the ventricular deflection in the HB recording is 60 ms earlier than the onset of the wide QRS complex, implicating the interventricular septum as being in close proximity to the exit site of these impulses.

References

1. Nogami A, Naito S, Tada H, et al. Verapamil-sensitive left anterior fascicular ventricular tachycardia: Results of radiofrequency ablation in six patients. *J Cardiovasc Electrophysiol.* 1998;9:1269–1278.
2. Nogami A, Naito S, Tada H, et al. Demonstration of diastolic and presystolic Purkinje potentials as critical potentials in a macroreentry circuit of verapamil-sensitive idiopathic left ventricular tachycardia. *J Am Coll Cardiol.* 2000;36:811–823.

CASE 9A.4

Paolo China, MD
Sakis Themistoclakis, MD

Patient History

A 63-year-old male with hypertension and a previous history of anteroapical myocardial infarction (MI) with moderate left ventricular systolic dysfunction was admitted to the emergency department (ED) due to palpitations. The patient reported no other symptoms. Ten years prior to this event, the patient also suffered atrioventricular nodal reentrant tachycardia (AVNRT) and was successfully treated with ablation of the slow pathway. He was on treatment with aspirin 100 mg once daily (OD), carvedilol 12.5 mg twice daily, ramipril 5 mg OD, potassium kanreonate 25 mg OD, and rosuvastatin 20 mg OD. At admission the arrhythmia was well tolerated, and his blood pressure was 110/60 mmHg. The first ECG obtained showed a wide QRS-complex tachycardia (WCT) (**Figure 9A.4.1**). The ED physician treated the patient with external direct current shock, in order to avoid harm in absence of a clear diagnosis, which restored sinus rhythm (**Figure 9A.4.2**).

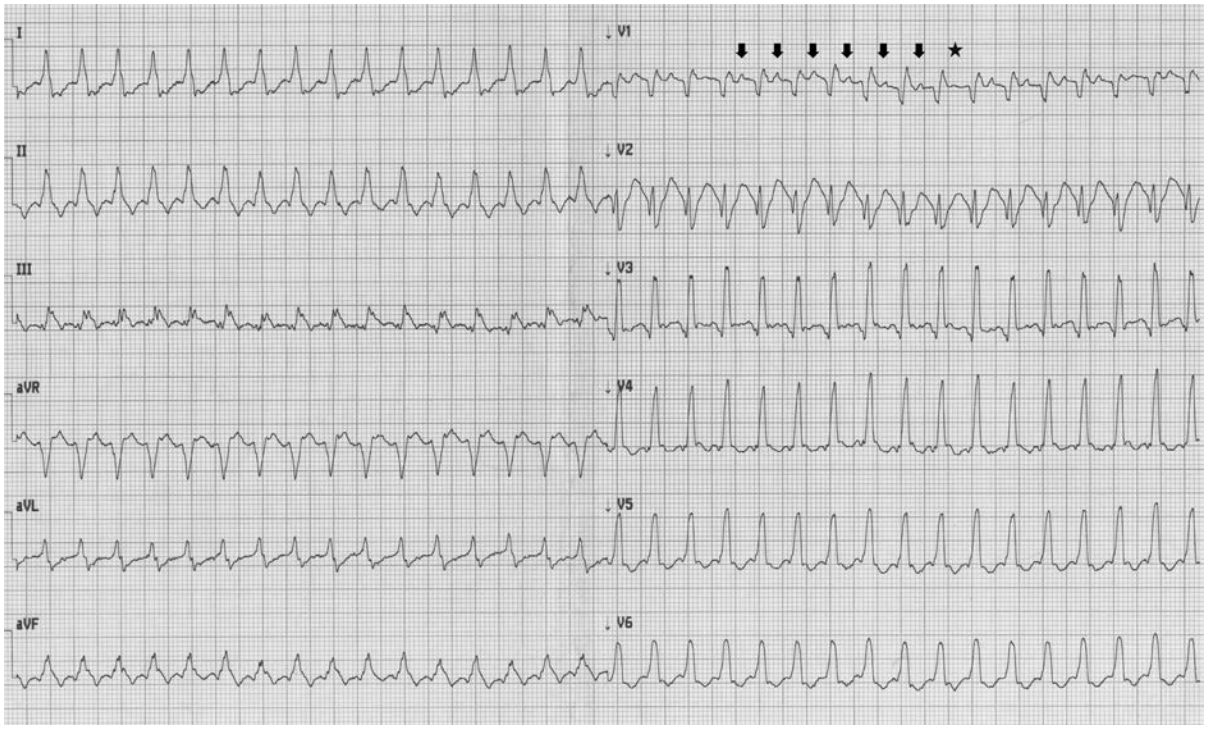

Figure 9A.4.1 12-lead ECG at admission in the ED (paper speed 25 mm/s).

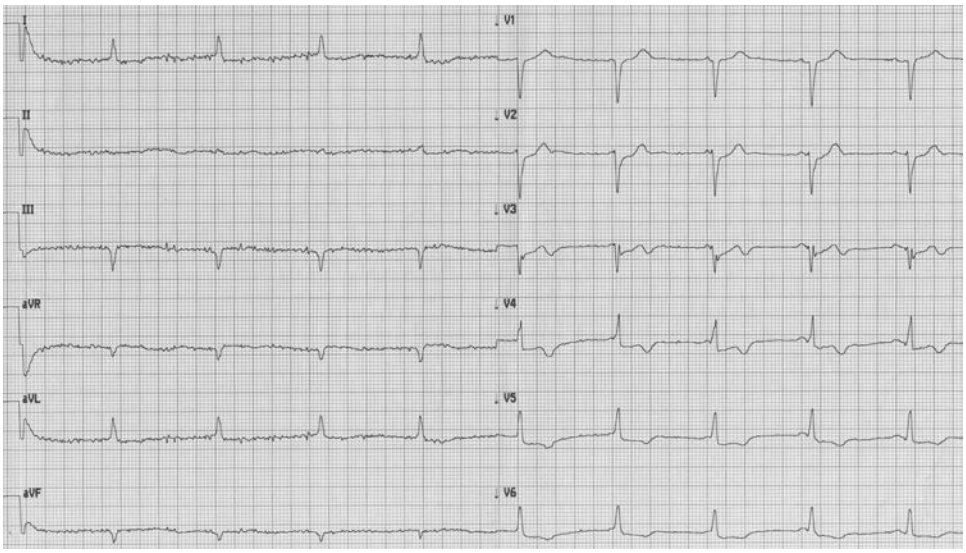

Figure 9A.4.2 12-lead ECG obtained immediately after cardioversion (paper speed 25 mm/s).

Question

What is the diagnosis?

ECG Discussion

The ECG in **Figure 9A.4.1** shows a wide QRS complex and regular tachycardia at about 200 bpm. In the frontal plane, the QRS wave has an inferior axis and duration of 130 ms. The QRS morphology shows a deep QR morphology in leads V_1–V_3, and an rS wave is also observed in aVR. The positive tall P wave in leads V_1 and aVR as well as the negative in the inferior leads with a fixed ventriculoatrial (VA) interval are not constant. P waves superimposed in the preceding T waves are indicated as arrows; in the seventh beat, marked with a star, there is clearly the absence of the expected small P wave.

The 12-lead ECG in **Figure 9A.4.2** shows junctional and sinus rhythm with transient isorhythmic atrioventricular (AV) dissociation, inferior and anterior necrosis, and diphasic T wave in the anterolateral leads that disappeared 48 hours after admission (**Figure 9A.4.3**).

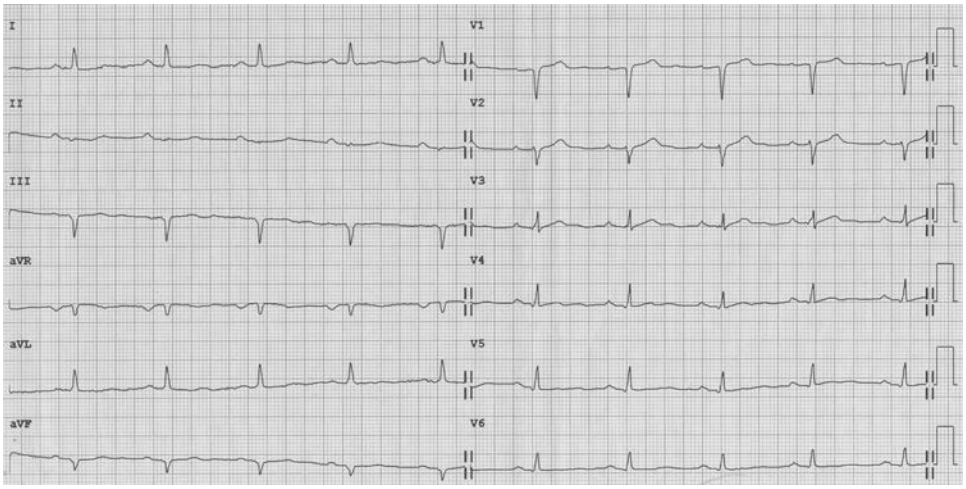

Figure 9A.4.3 12-lead ECG obtained about 48 hours after cardioversion (paper speed 25 mm/s).

Discussion, Interpretation, and Answer

The history of prior MI and previous ablated AVNRT complicate the differential diagnosis of this WCT. Various criteria for the diagnosis of WCT have been proposed and many authors suggest different algorithms to put together ECG findings into a unique diagnostic flow chart.

The AV dissociation and fusion beats, two of the most important criteria to distinguish between supraventricular tachycardia (SVT) with aberrancy and ventricular tachycardia (VT),[1,2] are not evident here. P waves have a fixed VA interval but are not always present and when they suddenly disappear there are no changes in the VV interval. These findings are suggestive of a VA conduction. Morphology criteria in this particular case is also helpful.

The QRS axis in the frontal plane is confined between -60°and +120°. The absence of extreme left axis deviation does not allow the exclusion of an SVT and cannot be useful for the differential diagnosis. This WCT can be classified as a right bundle branch block (RBBB)-like pattern for the QRS morphology in leads V_1–V_3. Patients with RBBB with a QRS duration longer than 140 ms is predictive of VT. However, VT can be associated with a QRS duration of less than 140 ms depending on the exit site; therefore, a QRS duration shorter than 140 ms, such as in this case, is not helpful for excluding VT. The RBB usually gives a minor contribution to the initial part of the "normal" QRS; thus, when blocked in SVT with aberrancy, the first part of the QRS is substantially unchanged. On the contrary, a monophasic R, Rsr′, biphasic qR complex, or broad R (more than 40 ms) in lead V_1 favors VT. Marriott et al[3] first described the RBBB-shaped tachycardia, the presence of a qR or wide R complex in lead V_1 that strongly argued for a ventricular origin of the tachycardia. Steurer et al[4] confirmed the positive predictive value of qR sign in one or more precordial leads from V_2 to V_6. Moreover, a small amount of normal right ventricular depolarization is directed away from V_6 determining a R:S ratio >1. Therefore, in this case the R:S ratio >1 does not exclude the possibility that the arrhythmia could be an aberrancy. Brugada et al[5] defined the absence of RS in any precordial lead as a second, very specific sign (100% specificity in 554 WCTs) for VT and overcomes other morphologic criteria. The more recent algorithm suggested by Vereckei and Coll[6] focused only on aVR morphology and is also helpful. In this case, an initial R wave (Rs complex) in aVR suggestive of VT is not present. Moreover, a small r in aVR with a duration equal or less than 40 ms is suggestive of VT. However, the following criteria for VT are satisfied: (1) a small notching on the downstroke of QRS and (2) a ventricular activation during the initial 40 ms (V_i), inferior than during the terminal 40 ms (V_t) of the QRS (V_i/V_t ratio ≤1).

In **Figure 9A.4.2**, sinus bradycardia with junctional rhythm and repolarization abnormalities can be due to the high heart rate of the previous sustained tachycardia and is common after sustained high heart rate in hearts with a low coronary flow reserve.

In conclusion, this WCT was considered a ventricular tachycardia. To confirm this hypothesis, an electrophysiological study (EPS) was carried out confirming the ventricular nature of this arrhythmia with inconstant VA retroconduction (**Figure 9A.4.4**). A coronary angiography was also done with the relief of critical lesions at the left anterior descending artery treated with percutaneous revascularization.

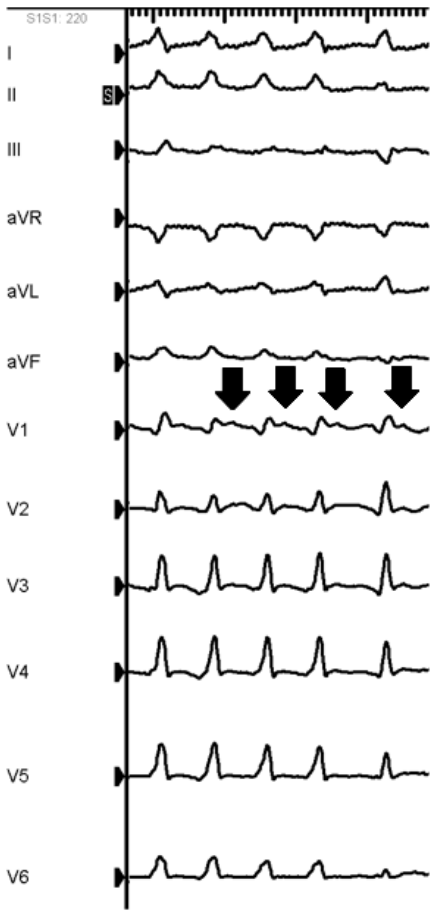

Figure 9A.4.4 12-lead ECG obtained during the electrophysiological study. The tachycardia morphology is the same as **Figure 9A.4.1** (paper speed 25 mm/s).

References

1. Wellens HJ, Bar FWHM, Lie KI. The value of the electrocardiogram in the differential diagnosis of a tachycardia with a widened QRS complex. *Am J Med.* 1978;64:27–33.
2. Wellens HJ. Electrophysiology: Ventricular tachycardia: Diagnosis of broad QRS complex tachycardia. *Heart.* 2001;86:579–585.
3. Marriott HJL. Differential diagnosis of supraventricular and ventricular tachycardia diagnosis. *Geriatrics.* 1970;25:91–101.
4. Steurer G, Gursoy S, Frey B, et al. The differential diagnosis on the electrocardiogram between ventricular tachycardia and preexcited tachycardia. *Clin Cardiol.* 1994;17:306–308.
5. Brugada P, Brugada J, Mont L, et al. A new approach to the differential diagnosis of a regular tachycardia with a wide QRS complex. *Circulation.* 1991;83:1649–1659.
6. Vereckei A, Duray G, Szenàsi G, et al. New algorithm using only lead aVR for differential diagnosis of wide QRS complex tachycardia. *Heart Rhythm*. 2008;5:89–98.

CASE 9A.5

Vassil Traykov, MD

Patient History

A 44-year-old female with recurrent hypertensive crises was admitted to the cardiology clinic for diagnostic workup. During one of the hypertensive episodes, the patient reported palpitations. The wide complex tachycardia shown below was recorded.

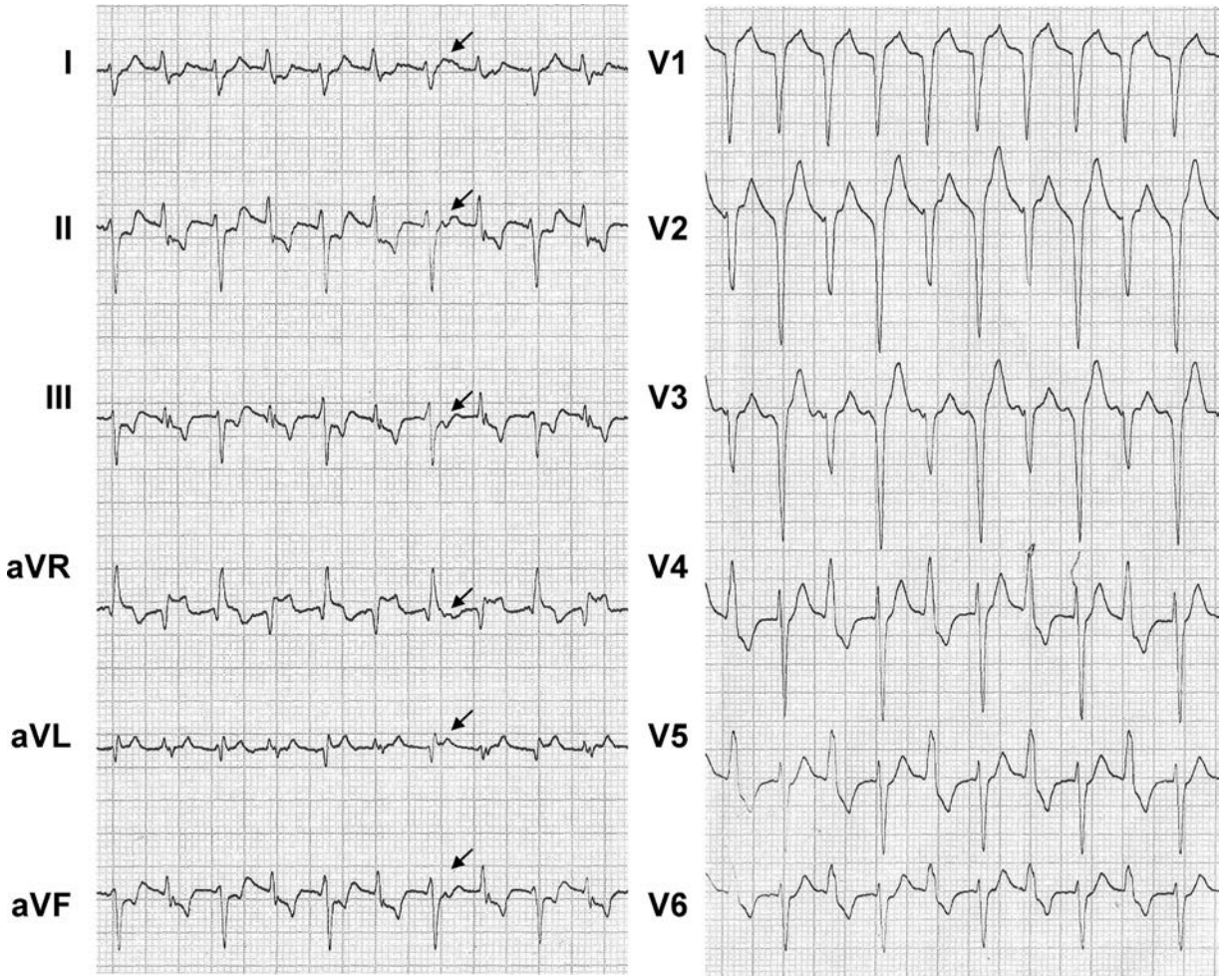

Figure 9A.5.1 Reproduced from[1] with permission from BMJ Publishing Group Ltd.

Questions

1. What type of arrhythmia would be the best explanation for this ECG recording?
2. What could be the underlying condition most likely associated with this tachycardia?

Discussion

The recording shows wide QRS tachycardia with a left bundle branch block (LBBB) morphology and alternating QRS frontal plane axis. Ventricular complex duration is wider than that in sinus rhythm (not shown). The QRS axis alternates between LBBB pattern with late precordial transition and northwest axis and LBBB pattern with earlier precordial transition (between V_3 and V_4) and normal QRS axis. Ventriculoatrial dissociation is also visible (arrows) suggesting ventricular tachycardia (VT). Due to QRS alternans, none of the criteria for differential diagnosis of wide complex tachycardia could be applied. The relatively narrow QRS (120 ms) with LBBB morphology suggests that the tachycardia is likely of fascicular origin arising from the right ventricle. The changing QRS axis suggests a basal exit alternating with a more apical exit site. Based on all the findings described above, the tachycardia was interpreted as bidirectional VT.

Bidirectional VT is a rare arrhythmia occurring in the setting of intracellular calcium overload. One of the proposed explanations for its occurrence is delayed after-depolarizations occurring in different zones of the conduction system.[2] The typical morphology of bidirectional tachycardia is right bundle branch block with alternating left anterior and left posterior fascicular block pattern.[3] However, this ECG recording represents LBB pattern that suggests involvement of the right bundle branch only. Further diagnostic workup in this patient led to the diagnosis of pheochromocytoma based on imaging data showing the presence of a right adrenal mass and elevated urine catecholamine levels. Catecholamine excess during hypertensive episodes in these cases could result in excess intracellular calcium, which would serve to explain the presence of bidirectional ventricular tachycardia. Following treatment with alpha and beta-blockers the patient was referred for right adrenalectomy, which led to complete symptom resolution.

References

1. Traykov VB, Kotirkov KI, Petrov IS. Pheochromocytoma presenting with bidirectional ventricular tachycardia. *Heart*. 2013;99(7):509.
2. Baher AA, Uy M, Xie F, et al. Bidirectional ventricular tachycardia: ping pong in the His-Purkinje system. *Heart Rhythm*. 2011;8:599–605.
3. Leenhardt A, Lucet V, Denjoy I, et al. Catecholaminergic polymorphic ventricular tachycardia in children. A 7-year follow-up of 21 patients. *Circulation.* 1995;91:1512–1519.

CASE 9A.6

Vassil Traykov, MD

Patient History

An 82-year-old female with a history of single-vessel coronary artery disease (CAD), prior myocardial infarction with left ventricular (LV) aneurysm and LV systolic dysfunction presented to the emergency department (ED) with palpitations, hypotension, and worsening heart failure. The ECG shown in **Figure 9A.6.1** was recorded upon presentation. A previously recorded 12-lead resting ECG is shown in **Figure 9A.6.2**. Patient had undergone percutaneous coronary intervention of left anterior descending artery (LAD) 5 years before.

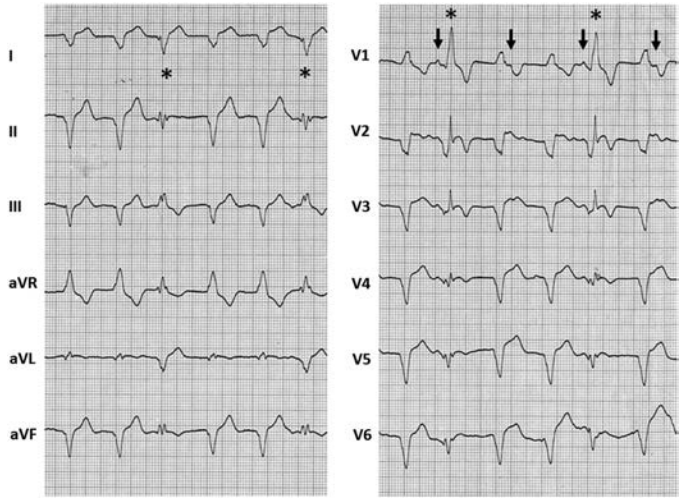

Figure 9A.6.1

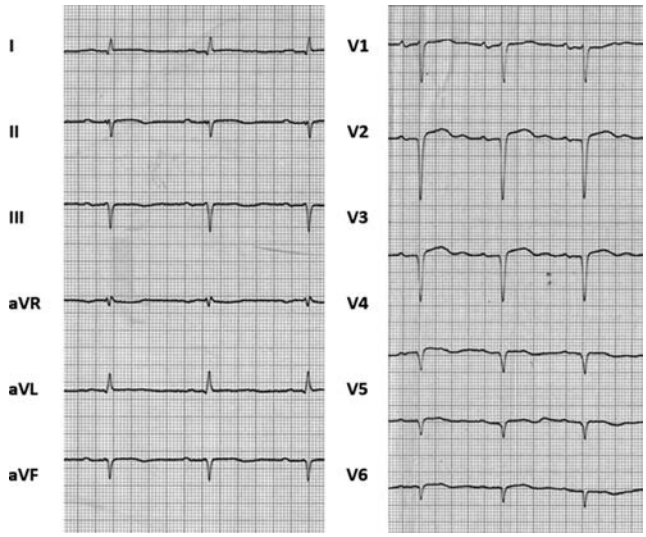

Figure 9A.6.2

Questions

1. How is the rhythm shown on Fig 9A.6.1 best described, and what is the most likely cause of this rhythm?
2. What is the origin of the two beats denoted by asterisks?

Discussion

The complaints of the patient were associated with the wide QRS rhythm—right bundle branch block (RBBB) with northwest axis deviation shown in **Figure 9A.6.1**. Brugada algorithm is able to identify this rhythm as monomorphic ventricular tachycardia (or monomorphic ventricular rhythm) at the first step since there are no RS forms in the precordial leads.[1] Ventriculoatrial dissociation was also clearly visible. The arrows on the recording in **Figure 9A.8.1** denote the P waves demonstrating atrial rate slower than ventricular rate. Based on the history of myocardial infarction, this rhythm was thought to be scar-related reentry.

Occasionally the beats marked by the asterisks were also seen. These beats are premature and demonstrate RBBB pattern with rightward axis deviation. They were always preceded by a P wave with a short PR interval and they reset the ventricular rhythm. These beats might be interpreted as fusion beats due to conducted P waves. However, sinus rhythm recording showed narrow QRS complex with a slightly prolonged PR interval, left axis deviation and QS pattern in all the precordial leads (**Figure 9A.6.2**). It is unlikely that a fusion beat that is so premature would have a PR interval much shorter than that in sinus rhythm. In case of conducted P waves causing fusion, the QRS in lead III would be predominantly negative based on the QRS morphology during sinus rhythm and during VT. The same holds true for QRS morphology in lead II. An alternative explanation of this ECG finding could be monomorphic premature ventricular beats resetting the tachycardia. In that case, P waves preceding them would be occurring coincidentally. This explanation is supported by the slight but measurable variation of PR interval preceding each of the two beats denoted by asterisks.

Despite amiodarone therapy, this rhythm recurred on follow-up so the patient was referred for catheter ablation. Left ventricular voltage maps revealed a large low-voltage zone occupying the apex and apical septum corresponding to a scar from a previous myocardial infarction. The clinical rhythm was inducible and its exit site was mapped endocardially to the lateral border zone of the scar. Scar homogenization was carried out that rendered tachycardias non-inducible.

Reference

1. Brugada P, Brugada J, Mont L, Smeets J. Andries EW. A new approach to the differential diagnosis of a regular tachycardia with a wide QRS complex. *Circulation*. 1991;83(5):1649–1659.

SECTION 9C

Preexcited Tachycardia

CASE
9C.1

Jonathan Kalman, MBBS, PhD

Patient History

A 57-year-old male with recurrent bursts of wide complex tachycardia.

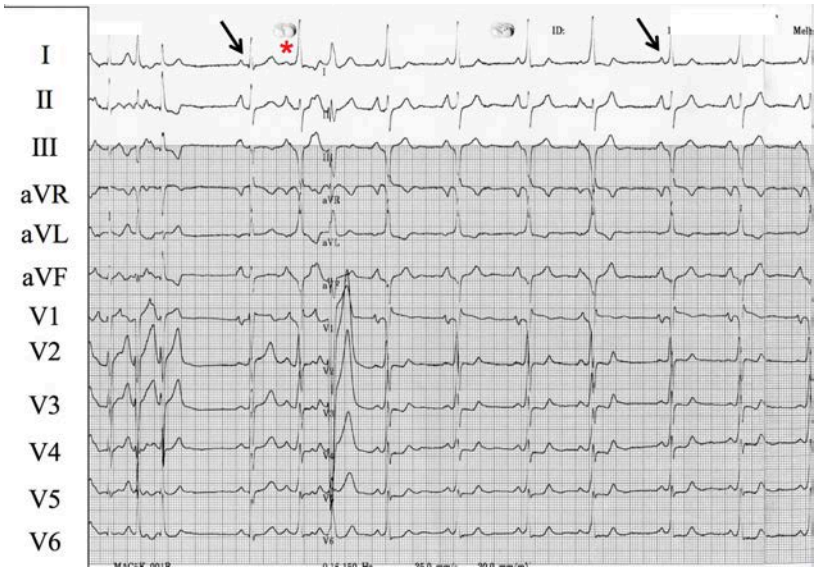

Figure 9C.1.1

Discussion

On the left (**Figure 9C.1.1**) is the termination of a burst of atrial tachycardia followed by a pause. The beat after the pause is a sinus beat with minimal preexcitation, possibly due to more rapid atrioventricular (AV) nodal conduction. Preexcitation also diminishes on the second beat (arrow) later in the trace due to a relatively longer sinus interval.

The first pause is followed by an atrial couplet (asterisk). The P-wave morphology of the first beat can be seen (asterisk) and is upright in lead V_1 and across the precordium. It is also upright and notched in the inferior leads. This focus was mapped to the os (ostium) of the left superior pulmonary vein. The first beat is preexcited and the second beat shows increased preexcitation. There is delta wave transition between leads V_1 and V_2 and negative delta waves in the inferior leads. The accessory pathway was ablated in the right posteroseptal region.

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SECTION 10

Ischemia and Infarction

Miquel Fiol, MD, PhD
Antoni Bayés de Luna, MD, PhD

CASE 10.1

Patient History

A 49-year-old female with a history of hypertension and dyslipemia consulting for anginal pain and dizziness. Cardiogenic shock was detected and an ECG (**Figure 10.1.1**) showed a transmural ischemia pattern in leads I, aVL, and V_2–V_6 with a mirror image in leads II, III, and aVF depicting left anterior fascicular block (LAFB) and right bundle branch block (RBBB). She was transferred to the hospital for an emergency coronary angiography.

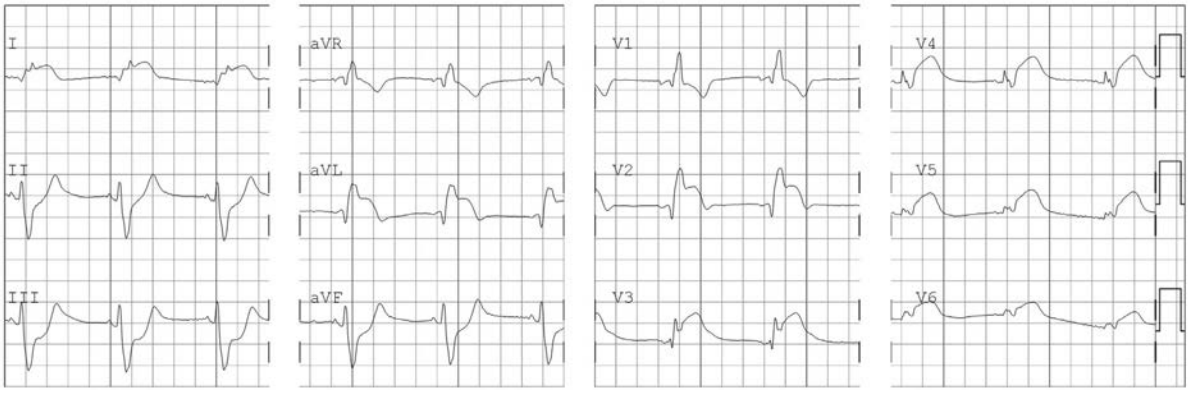

Figure 10.1.1

Question

What is the culprit artery?

A. Left anterior descending (LAD) artery total occlusion

B. LAD plus right coronary artery total occlusion

C. Left main trunk (LMT) total occlusion

D. LMT sub-total occlusion plus long sub-occlusion in the marginal artery

Discussion, Interpretation, and Answer

Figure 10.1.2 confirmed complete occlusion of the LMT of the coronary arteries.
The correct answer is C.

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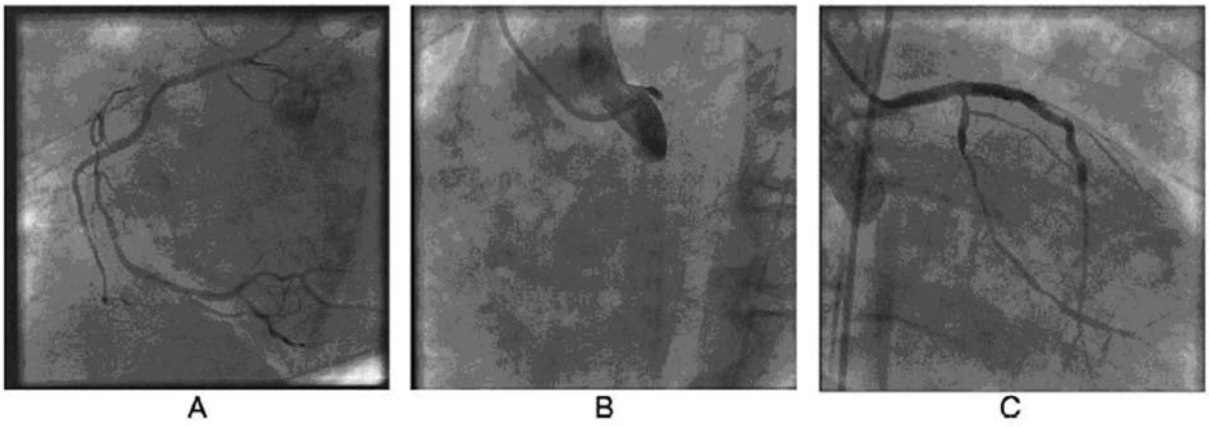

Figure 10.1.2 **A.** Right coronary artery without significant lesions. **B.** Total occlusion of the LMT. **C.** Partial reperfusion after percutaneous coronary intervention.

A stent in the left main coronary artery was implanted, thus improving the clinical status and hemodynamics.

The LMT is the culprit artery in acute coronary syndrome (ACS) in the majority of sub-total occlusion cases when the ECG pattern is of global sub-endocardial ischemia with ST descent in seven or more leads, and ST ascent in leads VR and V_1 (VR>V_1) (**Figure 10.1.3**).[1–3]

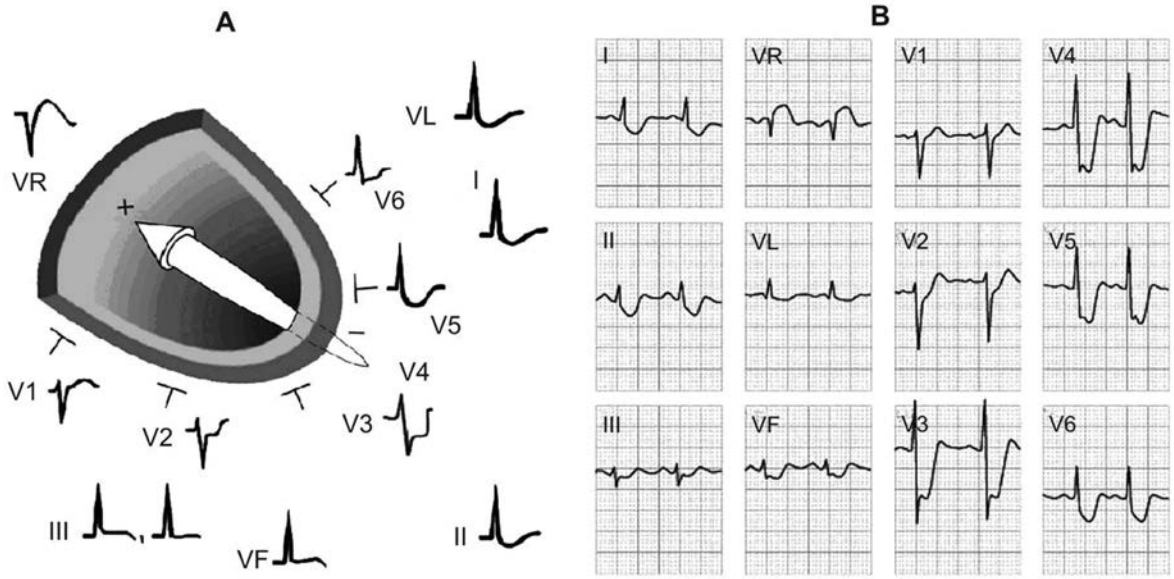

Figure 10.1.3 **A.** In the case of diffuse sub-endocardial global ischemia due to tight sub-occlusion of the LMT in a heart with previous important sub-endocardial ischemia, the ischemic vector that points to the circumferential sub-endocardial area is directed from the apex towards the base, from forward to backwards and from left to right. This explains the typical morphology of ST segment depression in all leads except VR and V_1, with maximal ST segment depression in leads V_3–V_5. As the ischemic vector faces more VR than V_1 the ST segment elevation in VR > V_1. **B.** Typical ECG of LMT critical sub-occlusion. The ST segment depression is higher than 6 mm in leads V_3–V_5 and there is no final positive T wave in leads V_4 and V_5.

Acute complete occlusion of the LMT rapidly triggers cardiogenic shock and ventricular fibrillation (VF), and the patient usually dies before reaching the emergency department (ED). However, new treatments and logistic systems of care have enabled prompt arrival to the ED, and

consequently more patients with complete occlusion of the LMT are seen in clinical practice. Nevertheless, in-hospital mortality remains very high.

There are few papers describing the ECG in cases of total occlusion of the LMT.[4–6] We have published a series of nine cases describing LMT occlusion (TIMI flow=0) with a STEMI pattern.[7] The most important characteristic of this ECG pattern is that it may be similar to that of proximal occlusion (before the first septal branch) of an LAD, with ST elevation in the precordial leads from V_2 to V_4–V_6 and in leads I and aVL, as well as ST depression in the inferior leads, and often with right bundle branch block (RBBB) and LAFB. However, in complete occlusion of the LMT there is also involvement of the LCX, which generates ST depression in leads V_1 and aVR, and consequently attenuates ST elevation especially in V_1. This causes the lack of ST changes in V_1 and often aVR that are frequently found in complete proximal long LAD occlusion.[8]

However, the following exceptions may happen:

A) ECG of ST elevation from V_2 to V_6 that is not explained by LMT total occlusion. This occurs in cases of proximal LAD complete occlusion if there is a large conal branch of the right coronary artery[9] that perfuses the high septum together with the first septal branch. This double perfusion of the high septum provides protection from ischemia. Therefore, this is a case of STEMI with ST elevation from leads V_2 to V_5 but without elevation in lead V_1 that is explained by total proximal LAD occlusion, not by complete LMT total occlusion.

B) The left circumflex coronary artery arises not from LMT but directly from the right coronary artery (**Figure 10.1.4A**).

C) Finally, if there is a great collateral circulation, the ECG in cases of LMT total occlusion (**Figure 10.1.4B**) may present an ECG pattern of circumferential sub-endocardial, LMT sub-occlusion similar to **Figure 10.1.3**.

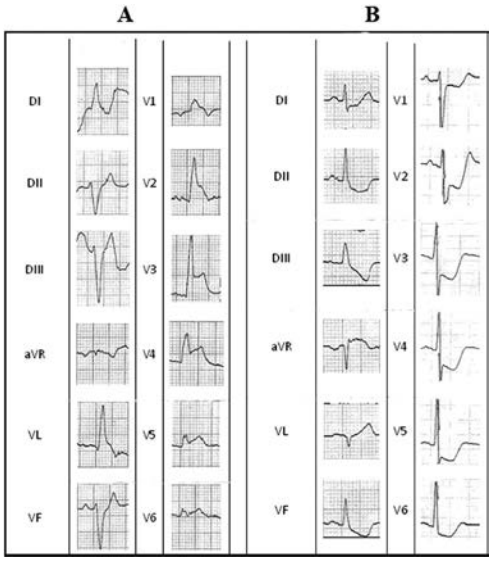

Figure 10.1.4 **A.** The LCX arising from right coronary artery. In this circumstance the LMT occlusion is equivalent to proximal LAD occlusion and there is ST elevation in V_1. **B.** The ECG of total LMT occlusion is due to good collateral circulation and is equivalent to LMT sub-occlusion.

Patients with LMT occlusion who survive usually present dominant right coronary artery when compared to those with LAD proximal occlusion.

Conclusion

LMT occlusion with transmural involvement should be suspected in the following situations:

1. Clinically cardiogenic shock and/or cardiac arrest at admission (70%).
2. ECG:
   - STEMI pattern of LAD occlusion proximal to the first septal and diagonal but without ST-segment elevation in leads V_1 and aVR. This pattern is present in more than 90% of cases. Exceptions are: A) LCX arises directly from the aorta, so there is ST elevation in V_1 as seen in LAD proximal occlusion (**Figure 10.1.4A**); B) In some cases of LMT total occlusion, the ECG may be of LMT sub-total occlusion (**Figure 10.1.4B**) if a great collateral circulation exists; and C) Finally, an ECG with ST elevation in V_2–V_6 not due to complete LMT occlusion may be seen when there is a conal artery that perfuses the high part of the septum. Due to this double perfusion, even in cases of LAD proximal occlusion, there is no ST elevation in V_1.
   - Frequent intraventricular RBBB-type conduction disturbances with LAH (60%).
   - Despite the severe clinical state of these patients at admission, 40% of our series survived.[7]

References

1. Bayés de Luna A, Fiol-Sala M. *Electrocardiography in Ischemic Heart Disease*. Oxford, UK: Blackwell/Futura. 2008:234–237.
2. Bayés de Luna A. *Clinical Electrocardiography*. Sussex, UK: Wiley-Blackwell. 2012:103.
3. Yamaji H, Iwasaki K, Kusachi S, et al. Prediction of acute left main coronary artery obstruction by 12-lead electrocardiography. *J Am Coll Cardiol*. 2001;38:1348–1354.
4. Aygul N, Slamov E, Dogan U, et al. Acute occlusion of the left main trunk presenting as ST-elevation acute coronary syndrome. *J Electrocardiol*. 2010;43:76–78.
5. Widimsky P, Rohác F, Stásek J, et al. Primary angioplasty in acute myocardial infarction with right bundle branch block: Should new onset right bundle branch block be added to future guidelines as an indication for reperfusion therapy? *Eur Heart J*. 2012;33:86–95.
6. Nikus KC. Acute total occlusion of the left main coronary artery with emphasis on electrocardiographic manifestations. *Timely Top Med Cardiovasc Dis*. 2007;1(11):E22.
7. Fiol M, Carrillo A, Rodriguez A, et al. Electrocardiographic changes of ST-elevation myocardial infarction in patients with complete occlusion of left main trunk without collateral circulation: Differential diagnosis and clinical considerations. *J Electrocardiol*. 2012;45:487–490.
8. Fiol M, Carrillo A, Cygankiewicz I, et al. A new electrocardiographic algorithm to locate the occlusion in left anterior descending coronary artery. *Clin Cardiol*. 2009;32:E1–E6.
9. Ben-Gal T, Herz I, Solodky A, et al. Acute anterior wall myocardial infarction entailing ST elevation in V1: Electrocardiographic and angiographic correlations. *Clin Cardiol.* 1998;21:399–404.

SECTION 12

Paced Rhythms and Device Troubleshooting

Andrei G. Dan, MD, PhD
Catalin A. Buzea MD, PhD

CASE
12.1

Patient History

A 48-year-old patient with history of heart failure and atrial fibrillation presents to the emergency department for palpitations. He is under treatment for heart failure, including enalapril, furosemide and metoprolol succinate. His left ventricular ejection fraction is 25%. A resynchronization therapy was applied 3 month before.

Questions

1. What is the underlying rhythm?
2. What is the mechanism of the QRS polymorphism?

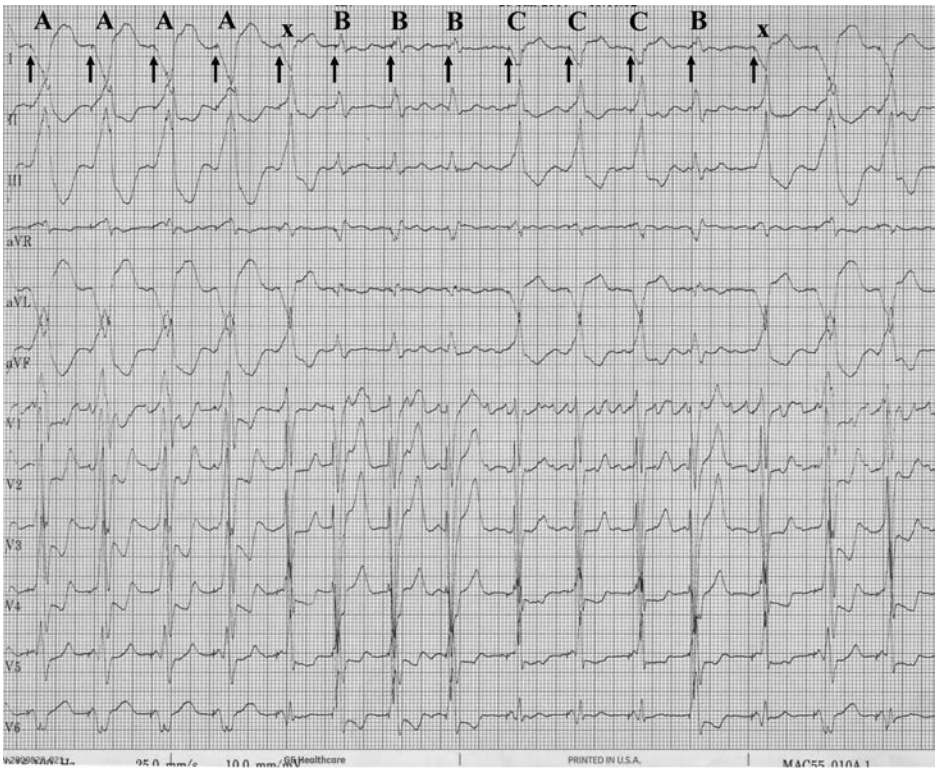

Figure 12.1.1 ECG of a patient with atrial fibrillation and CRT-P showing polymorphism of ventricular complexes.

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Discussion, Interpretation, and Answers

Atrial rhythm is atrial fibrillation; f" waves are well visible in V_1 (coarse atrial fibrillation). Ventricular rhythm is regular at 93 bpm. There are three main different QRS complexes (A, B, and C) showing different intraventricular conduction delay. Each QRS complex is preceded by a small spike (arrow) indicating that they are paced. The first four complexes have a right bundle branch block (RBBB) appearance, indicating a left ventricle paced rhythm by the cardiac resynchronization therapy device. All ventricular complexes are in fact paced from the same site and show different degrees of fusion giving the aspect of a polymorphic ventricular rhythm. This is possible because the atrial rhythm is atrial fibrillation and the ventricle is depolarized both by pacing rhythm and conduction of atrial impulses. As the last beats are irregular, the degree of ventricular penetration is different explaining various types of fusion beats. The beats noted 'x' are intermediate configuration beats.

Santosh K. Padala, MD
Gautham Kalahasty, MD
Kenneth A. Ellenbogen, MD

CASE 12.2

Patient History

A 61-year-old male with a history of sick sinus syndrome underwent implantation of a dual chamber permanent pacemaker (Guidant, Pulsar Max 1270 Pacemaker). Three months later, he was involved in a motor vehicle accident and was admitted to a local hospital. His admission 12-lead ECG showed sinus tachycardia at 145 bpm (**Figure 12.2.1**). While being monitored on telemetry, he was noted to have the following rhythm strip (**Figure 12.2.2**). Pacemaker malfunction was suspected. Device interrogation is shown below.

Mode = DDDR	AP = 30%, VP = 92%
Lower Rate Limit = 60 bpm Maximum Tracking Rate = 130 bpm	RA Lead Sensing = 4.4 mV Amplitude = 3.0 V @ 0.4 ms Impedance = 350 ohms
Maximum Sensor Rate = 130 bpm Dynamic AV Delay = on Maximum Delay = 200 ms Minimum Delay = 80 ms	RV Lead Sensing = 20.4 mV Amplitude = 1.5 V @ 0.4 ms Impedance = 470 ohms
Dynamic PVARP = on Maximum PVARP = 310 ms Minimum PVARP = 270 ms PVARP After PVC = 400 ms	Programmed Settings Sensing RA Lead= 1.0 mV RV Lead= 2.5 mV Threshold RA Lead = 3.5 V @ 0.4 ms RV Lead = 3.5 V @ 0.4 ms

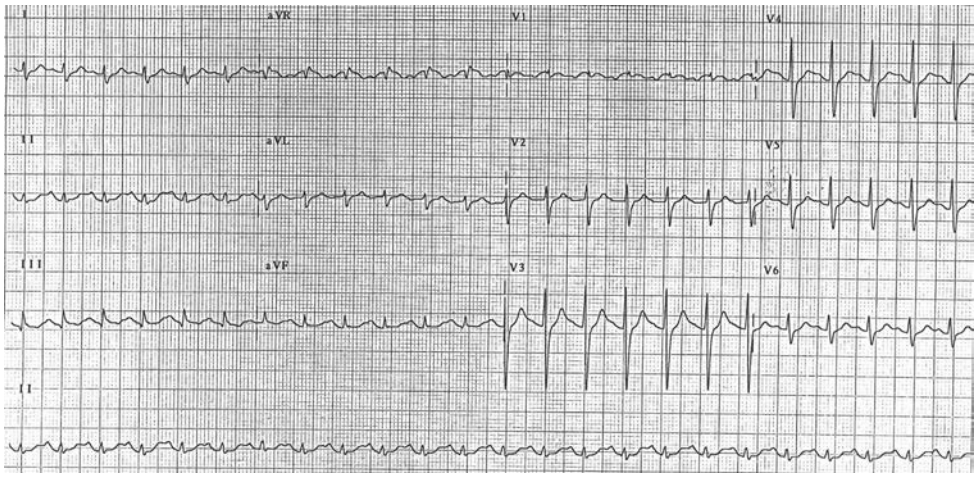


Figure 12.2.1 Baseline ECG showing sinus tachycardia.

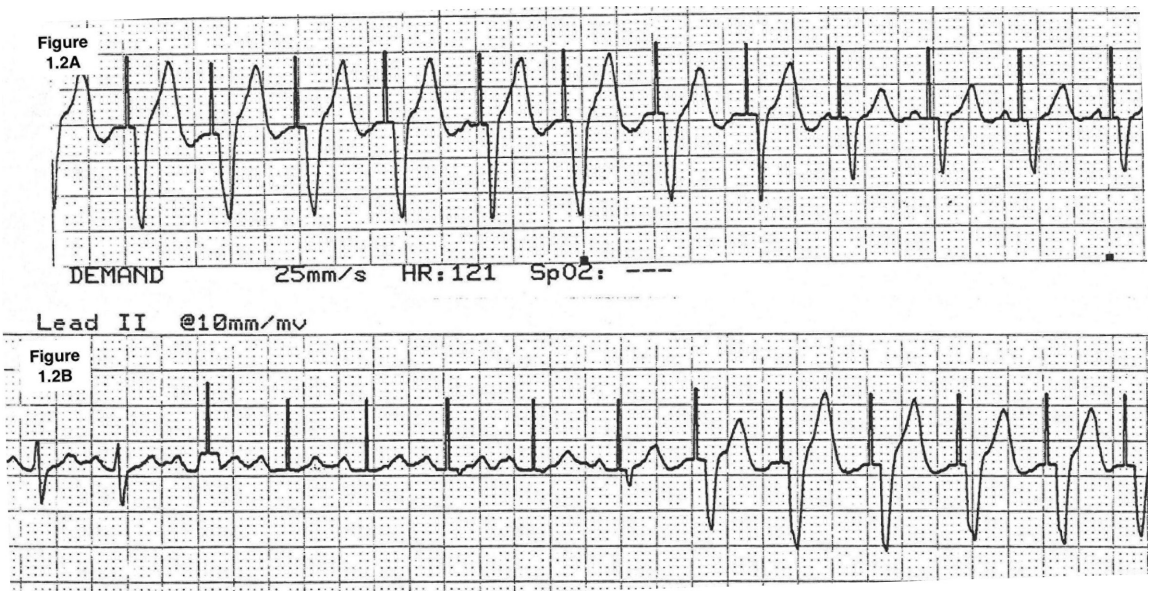


Figure 12.2.2 Rhythm strip.

Questions

1. Pacemaker malfunction was the cause of the motor vehicle accident.

1. True
2. False

2. What is the cause of the findings noted on the first half of the rhythm strip in **Figure 12.2.2B**?

1. Undersensing
2. Oversensing
3. Intermittent failure to capture
4. Normal pacemaker function

Answers

1. False

2. D

Discussion

The first rhythm strip (**Figure 12.2.3A**) demonstrates normal atrial tracking—trial sensing and ventricular pacing (AS-VP). The first half of the rhythm strip shows fully paced ventricular complexes, while the second half of the rhythm strip shows fused ventricular complexes. In the second rhythm strip (**Figure 12.2.3B**), the first two beats are AS-ventricular sensed (VS) beats. In the next five beats, a AS event is followed by a ventricular spike, but no obvious QRS complex is identified on the surface ECG giving a false impression of pacemaker malfunction. This finding does not necessarily imply loss of ventricular capture as depolarization is inferred by the presence of a T wave following each ventricular pacing spike (dashed arrows). The QRS complex is not seen

because the resultant fusion complex between the native QRS and the paced beat is such that it is isoelectric in the monitored lead (lead II here), thus canceling the QRS vector.

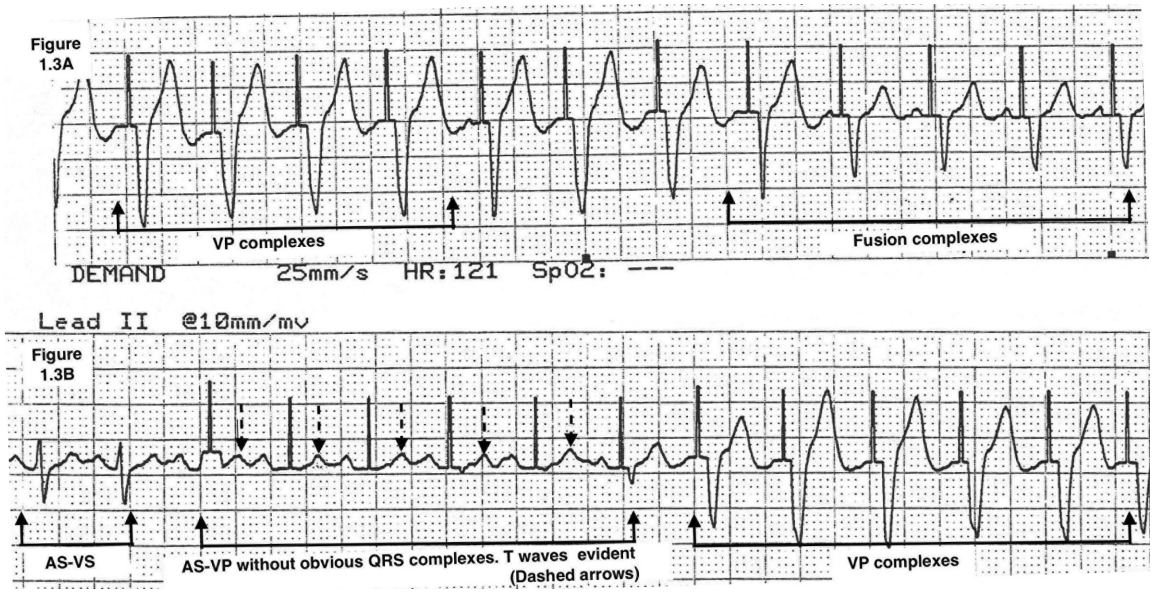


Figure 12.2.3 Annotated rhythm strip.

CASE 12.3

Santosh K. Padala, MD
Gautham Kalahasty, MD
Kenneth A. Ellenbogen, MD

Patient History

A 67-year-old female underwent a dual chamber permanent pacemaker implant for symptomatic bradycardia due to sick sinus syndrome. A 12-lead ECG (**Figure 12.3.1**) performed the day after implantation showed alternating atrial pacing (AP)-ventricular sensed (VS) beat followed by AP-ventricular paced (VP) beat.

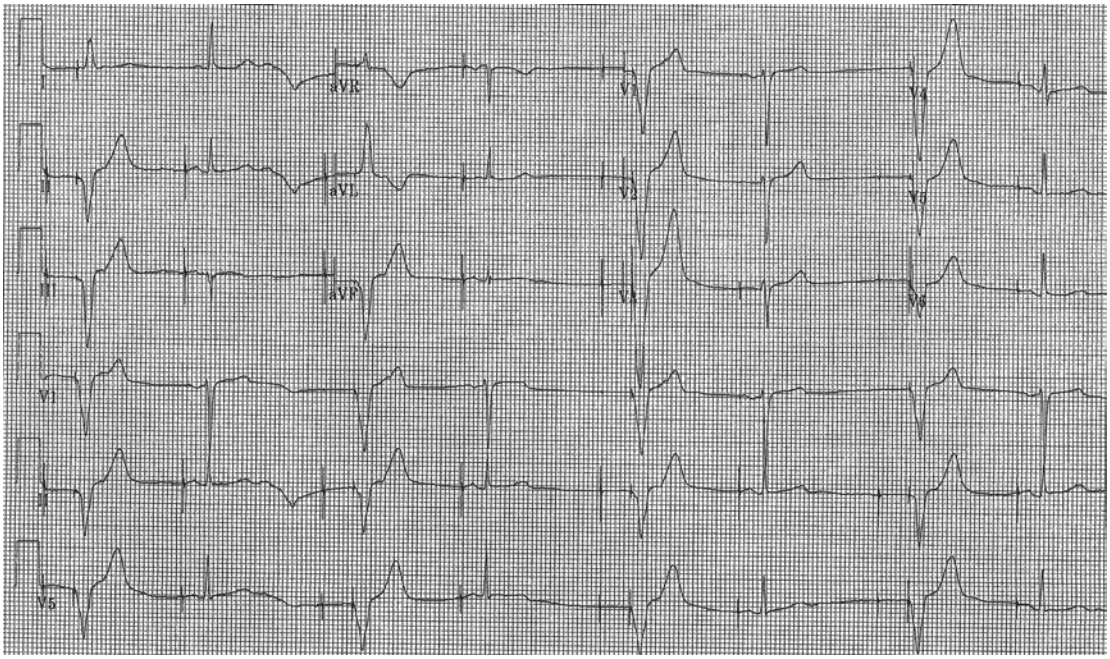


Figure 12.3.1 Baseline ECG.

Question

What is the likely cause of the VP beats?

1. Normal pacemaker function
2. Intermittent AV block
3. Alternating loss of atrial capture
4. Pacemaker malfunction
5. Both 3 and 4

Answer

The correct answer is E.

Discussion

The ECG (**Figure 12.3.2**) shows alternating AP-VP beat followed by AP-VS beat. In lead II of the rhythm strip, the second beat (dashed arrow) shows AP with atrial capture (upright P wave). The impulse traverses down the AV node and depolarizes the ventricle before the paced AV delay times out, resulting in a VS event at 160 ms, suggesting normal AV conduction. This is followed by an AP spike but without atrial capture (no P wave, bold arrow). As a result, there is no conduction down the AV node to depolarize the ventricle. After the paced AV delay times out at 240 ms, a VP spike is seen with ventricular capture. The alternating loss of atrial capture in the present case was due to inadequate contact between the tined atrial lead and the right atrial myocardium.

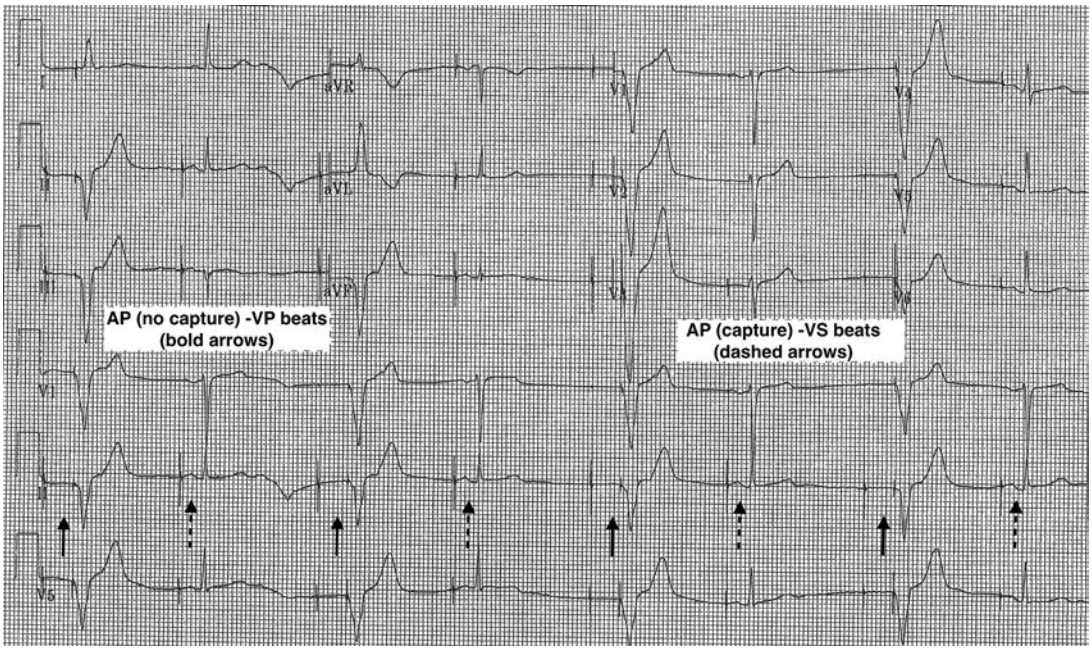


Figure 12.3.2 Annotated ECG demonstrating alternating loss of atrial capture.

CASE 12.4

Tamer S. Fahmy, MD, PhD
Ahmed Abdel Aziz, MD, PhD

Patient History

A 43-year-old female complaining of palpitations after pacemaker implantation for intermittent heart block and asystole of more than 3 seconds. Tracings from the Holter monitor are shown in **Figures 12.4.1–12.4.3**. Lead testing was acceptable and an x-ray showed both leads in the correct position. Lead testing and programming are shown in **Figures 12.4.4** and **12.4.5**.

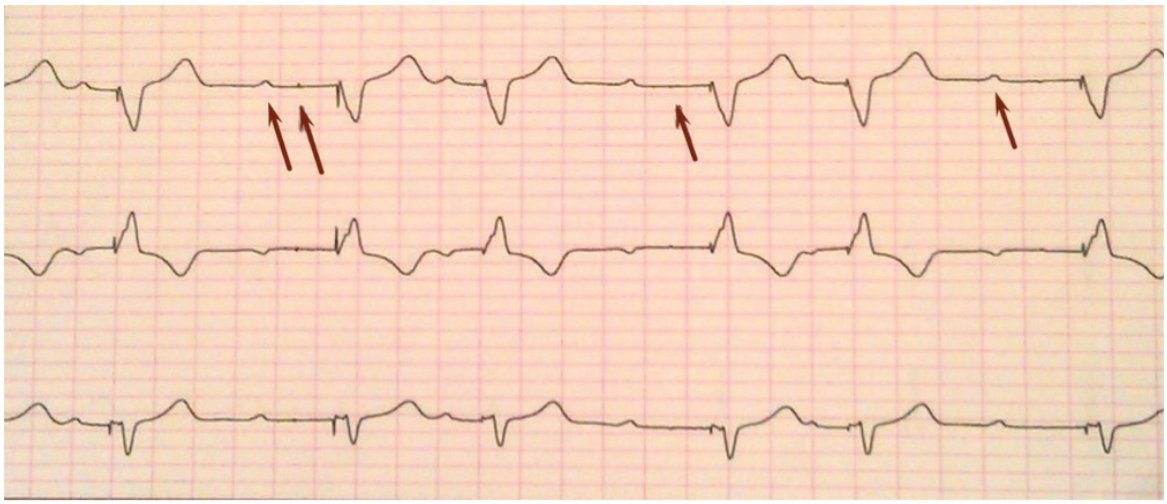


Figure 12.4.1 Sensing and capture abnormalities. As shown by the first arrow, there is malsensing of the P wave. The second arrow indicates lost capture but atrial tracking. The same phenomenon is repeated in the following two arrows.

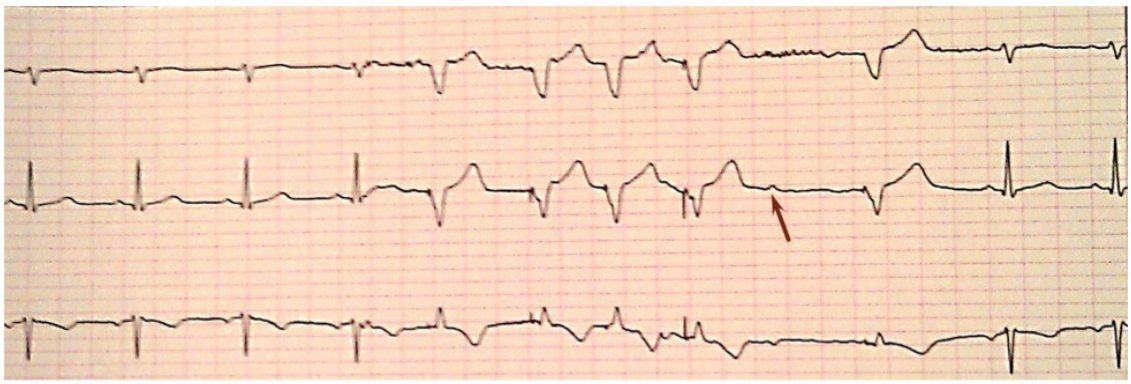


Figure 12.4.2 Atrial sensing and capture abnormalities (oversensing). Rhythm strip shows that the first four beats are conducted sinus beats. The fifth beat is an atrial tracking of a normal P wave. The following three paced beats are tracking atrial ectopics that the patient has with variable atrioventricular (AV) delay. The arrow shows an unsensed P wave for which the pacemaker made a mode switch to VVI.

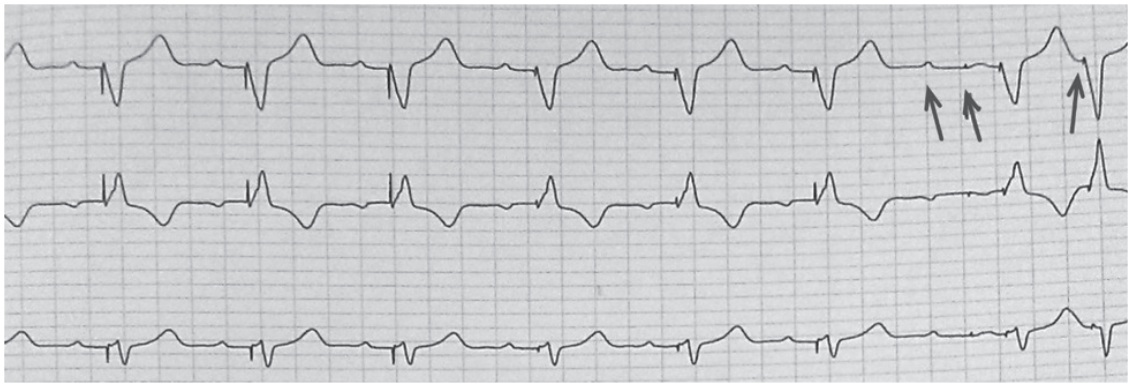


Figure 12.4.3 Atrial sensing and capture abnormalities (under and oversensing). Paced rhythm, with atrial tracking with an AV delay of 240 ms. The first arrow shows an unsensed P wave which was followed by atrial (second arrow) and ventricular pacing. This was followed by sensing of a PAC, or a retrograde P wave. Alternatively, there might have been oversensing of the T wave for which tracking occurred.

ATRIUM	
Pulse amplitude	2.4 V
Pulse width	0.40 ms
Sensitivity	0.5 mV
Refractory period	450 ms
Min. PVARP	215 ms
ARP extension	50 ms
Far-field blanking	56 ms
Pacing polarity	BIPL
Sensing polarity	BIPL
Lead check	ON
VENTRICLE	
Pulse amplitude	1.1 V
Pulse width	0.40 ms
Active Capture Control	On
ACC status	OK
Mode	DDD
Basic/Night rate...	55/50 bpm
Rate hysteresis...	OFF bpm
Repetitive	-----
Scan	-----
Night program	50 bpm
Night begins	01:00
Night ends	05:00
Sensor	
Max. activity rate	120
Sensor gain	8
Auto-gain	ON
Sensor threshold	Medium
Rate increase	2 bpm/cyc
Rate decrease	0.5 bpm/cyc
Upper rate...	130 bpm
	WKB
Max. sync. rate	130/133 bpm
PMT protection	ON
VA criterion	350 ms
Mode switching...	DDIR/140 bpm
Intervention rate	140 bpm
Switch to	DDIR
Onset criterion	7
Resolution criterion	7
AV DELAYS	
Dynamic AV delay...	225 ms
after pace:	
All rates	225 ms
Sense compens.	-45 ms
AV safety window	100 ms
IRSplus...	ON
AV hysteresis	IRSplus
AV repetitive	5
AV scan	5

Figure 12.4.4 Pacemaker set parameters.

Measured values:		
Batt. voltage		2.77 V
Batt. current		18 μA
Batt. imped.		0.6 kΩ
	Atr.	Ven.
Pulse voltage	2.2	1.0 V
Pulse current	3.5	1.7 mA
Pulse energy	2.9	0.6 μJ
Pulse charge	1.4	0.7 μC
Lead impedance	646	610 Ω

Figure 12.4.5 Pacemaker testing.

Questions

1. How was the pacemaker programmed to suit **Figures 12.4.2** and **12.4.3**, specifically?
2. How should the pacemaker be further programmed?

Discussion, Interpretation, and Answers

Having intermittent atrial malsensing with loss of atrial sensing and capture is well perceived; however, the problem arises when there is loss of sensing and over-sensing atrial ectopics, retrograde P, or far-field T wave at the same time. Knowing that the atrial leads are in the appropriate x-ray position with acceptable lead testing, the solution lies in programming. For such reasons, the atrial refractoriness has been set at 450 ms with PVARP set at 215, and far-field blanking at 56 ms. Despite these changes, the patient still has the same phenomenon. The second solution would lie in changing the sensing to unipolar, with decrease in sensitivity than 0.5. Another solution would be to decrease the atrial refractoriness, and further increase the PVARP.

CASE
12.5

Philip Podrid, MD

Patient History

A 76-year-old male with a known dilated cardiomyopathy and a left ventricular ejection fraction (LVEF) of 30% presents to his cardiologist for a post-hospitalization visit. During the hospitalization, he had a pacemaker inserted, although he is not certain why. Physical examination is unremarkable. A 12-lead ECG is obtained (**Figure 12.5.1**).

Questions

1. What type of pacemaker does the patient have?
2. What is the mode of the pacemaker function?
3. What accounts for the pauses and the irregular rhythm?

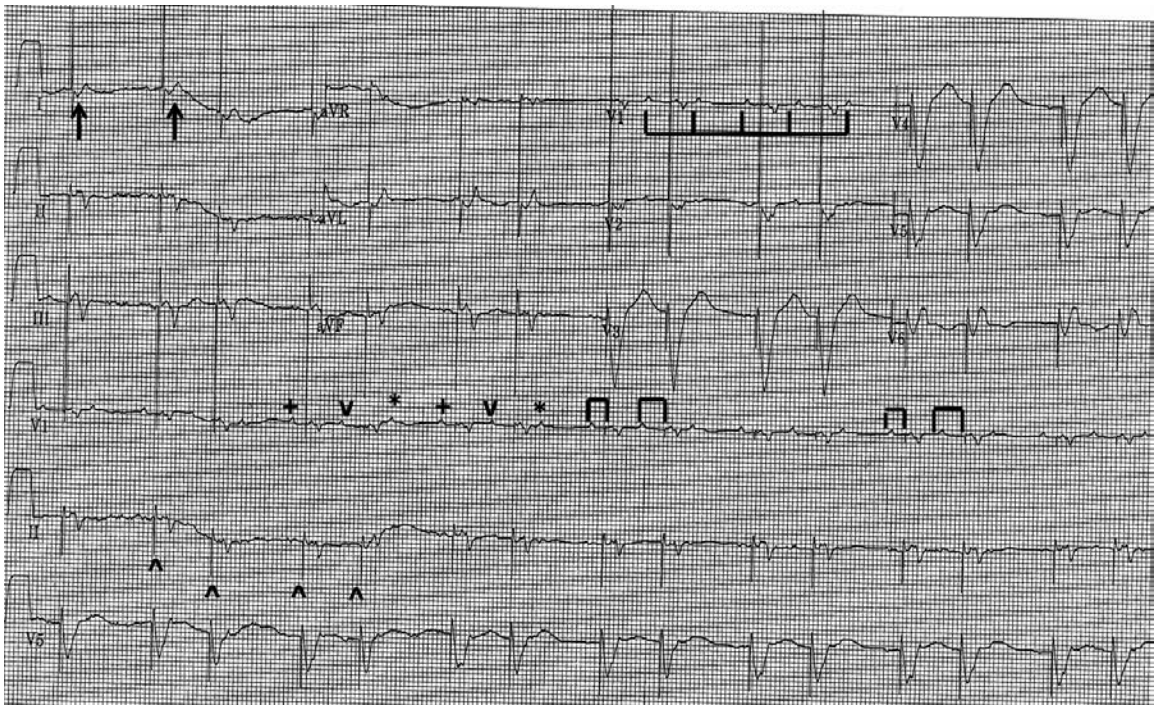


Figure 12.5.1

Diagnosis

Sinus tachycardia vs. atrial tachycardia, biventricular pacemaker, atrial sensed (AS)-ventricular paced (VP) (P-wave synchronous ventricular pacing), pseudo (pacemaker mediated) Wenckebach (3:2).

Discussion

The rhythm is irregular (lead V_1), but there is group beating and all the short intervals are the same and the long intervals are the same. The average rate is 90 bpm. Therefore, the rhythm is regularly irregular. There are pacemaker stimuli seen (^) before each QRS complex (lead II). Prior to each pacemaker stimulus there is a P wave (+,v), best seen in lead V_1. Therefore the mode of the pacemaker is AS-VP or P-wave synchronous ventricular pacing. The QRS complexes do not have a morphology seen with a right ventricular pacing lead, i.e., a left bundle branch block (LBBB). Importantly there is an initial Q wave in lead I (↑). This is a feature associated with a biventricular pacemaker as the initial impulse is from a left ventricular lead. As lead I is a right-left bipolar lead, an impulse originating from the left and going to the right generates an initial Q wave or a QS complex in this lead, while an impulse originating from the right going to the left (i.e., right ventricular pacing lead) generates an R wave in this lead.

P waves are obvious in lead V_1 and the PP interval is constant (⌊⌋) at a rate of 130 bpm. P waves are not clearly seen in other leads. Therefore, it is not certain if the rhythm is a sinus or atrial tachycardia. Although there is a P wave before each pacing stimulus (+), every third P wave (*) is not followed by a pacemaker stimulus, accounting for the long RR interval. This is due to the fact that it is not sensed. Additionally, the atrioventricular (AV) delay, i.e., time between the P wave and ventricular stimulus, is not constant (⌐¬). Following the long RR interval, the AV delay associated with the first P wave (+) is 0.12 seconds while after the second P wave (v) the AV delay is 0.20 seconds. The third P wave (*) is not followed by a pacemaker stimulus, i.e., it is nonconducted. This is a pattern of a second-degree AV block, Mobitz type I or Wenckebach. This is termed pacemaker-mediated or pseudo Wenckebach. The occurrence of this pattern is related to two programmable features, i.e., the post-ventricular atrial refractory period (P-VARP), which determines the upper rate limit of the pacemaker (the fastest atrial rate sensed or tracked by the pacemaker resulting in a ventricular stimulus), and the AV delay of the pacemaker. These two parameters define the total atrial refractory period (TARP). If there is an underlying sinus or atrial rate that is close to the upper rate limit of the pacemaker (as is the case here as two of the P waves are sensed), the atrial impulse will be sensed by the atrial channel (as the atrial impulse occurs after the P-VARP). If the atrial impulse is sensed, the pacemaker is committed to deliver a ventricular stimulus (unless it is inhibited by a spontaneous ventricular complex, i.e., a premature ventricular complex or QRS complex resulting from AV conduction). However, if based on the AV delay (which may be short), delivery of a ventricular stimulus would violate the upper rate limit of the ventricular channel, the pacemaker waits until the upper rate limit is achieved before the ventricular stimulus is delivered. After the ventricular stimulus is delivered, the P-VARP starts again. If there is a stable atrial rate, the next atrial impulse is now closer to the P-VARP, and therefore there is an even longer time before the ventricular stimulus can be delivered, accounting for a lengthening of the AV delay. This continues until the atrial impulse coincides with or is within the P-VARP and is no longer sensed; hence there is no ventricular stimulus, i.e., there is a nonconducted P wave. This is most frequently seen with biventricular pacing since the programmable AV delay is often short, insuring that the pacemaker always captures the ventricles.

Mohammad Shenasa, MD
Shahriar Heidary, MD
Mariah Smith
Hossein Shenasa, MD, MS

CASE 12.6

Patient History

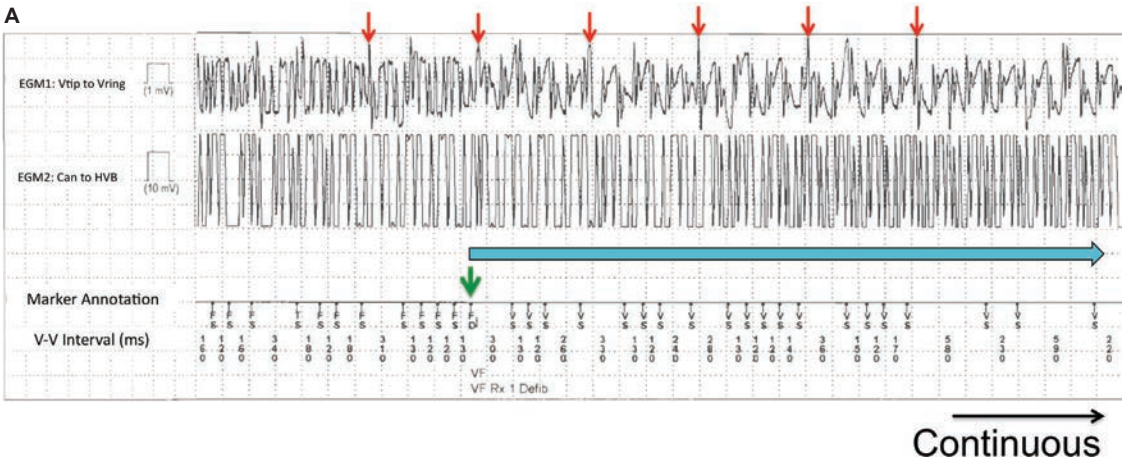
A 72-year-old white male with a history of rapid nonsustained ventricular tachycardia, hypertrophic cardiomyopathy, and intermittent Brugada-type ECG received a single-lead implantable cardioverter defibrillator (ICD) for primary prevention of sudden cardiac death.

His device specification and programming include:

- Single lead Medtronic ICD
- Model number: Maximo VR 7232 (PRN122082H)
- Right ventricular lead: Medtronic model 6936 (TAV203923R)
- Ventricular fibrillation detection rate: 188–500 bpm.
  - Ventricular fibrillation therapies programmed to 35 joules times 6.
- Ventricular tachycardia detection rate: 171–188 bpm.
  - Ventricular tachycardia therapies programmed to burst pacing times 3, ramp pacing times 3, and 35 joules times 4.
- Bradycardia therapies were programmed to VVI mode at 40 bpm.

The patient used his hot tub at home and shortly after turning on the jets he received an ICD shock. He did not loose consciousness and was able to step out of the hot tub.

Figure 12.6.1 shows ICD interrogation and analysis of intracardiac EGMs and markers, which revealed inappropriate ICD shocks due to electrical interference of the hot tub (60-cycle electrical artifact erroneously detected by the device) (**Figure 12.6.1A**). The device then perceived this as ventricular fibrillation and delivered a 34.8 joules shock while the patient was actually in sinus rhythm (**Figure 12.6.1B**, large black arrow). Once he stepped out of the hot tub, the electrical interference dissipated and the patient remained in sinus rhythm at the cycle length of 900–720 ms (**Figure 12.6.1C**). Fortunately, this inappropriate ICD shock did not induce true ventricular fibrillation.



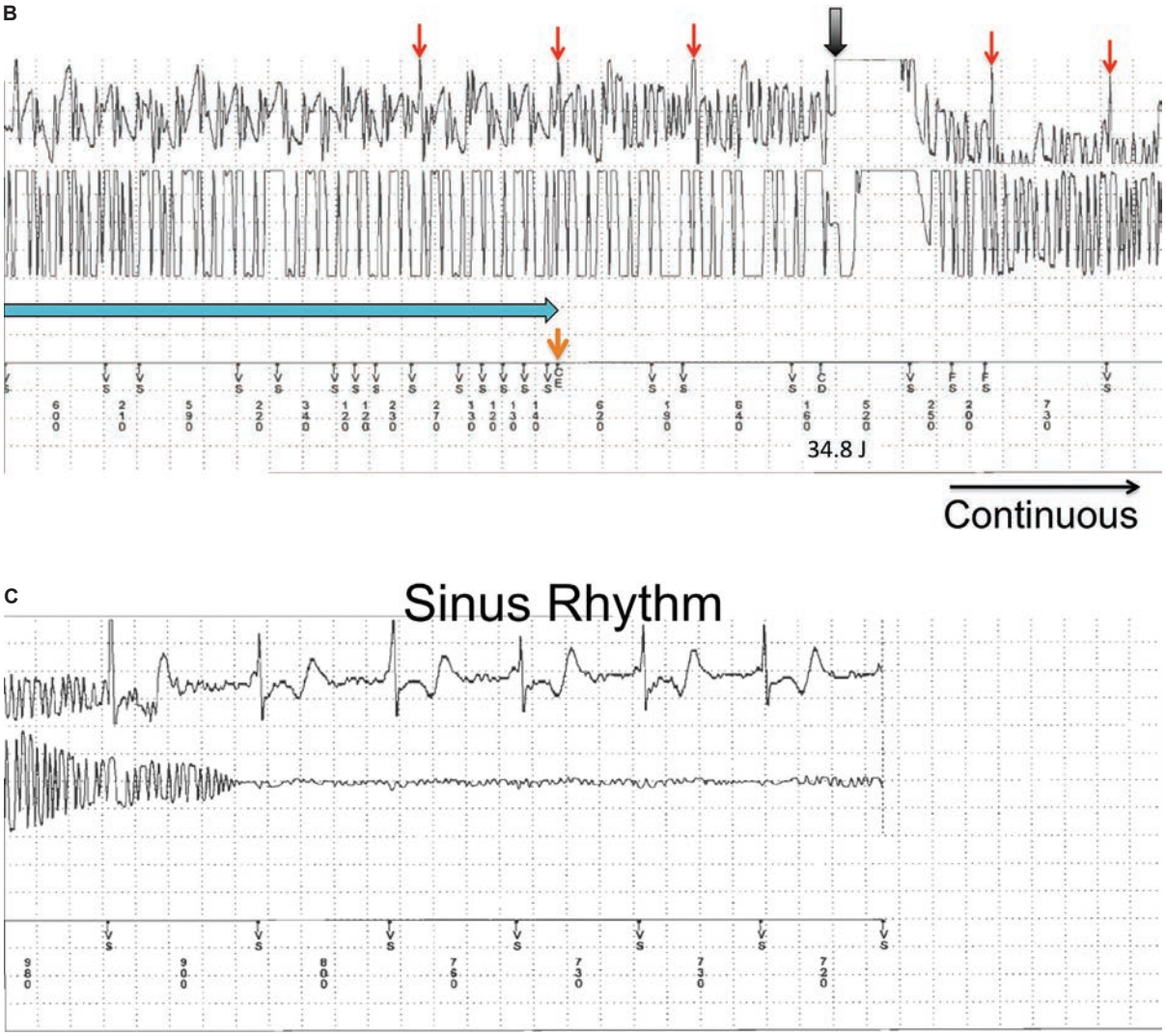


Figure 12.6.1 The tracings are arranged as intracardiac electrograms, (EGM) 1 and 2. The patient's native R waves are denoted by the red arrows. Marker annotations include ventricular intervals (V-V intervals). Note that during this electrical interference-induced artifact, the device erroneously read this as ventricular fibrillation, (FS, fibrillation sense; TS, tachycardia sense, according to zone programmed note). FD denotes fibrillation detected, and the number of intervals satisfied (panel A, green arrow). At this point, the device is charging (blue horizontal arrow). There is occasional undetected fibrillation sensing, where the interval reads 260–330 ms. CE denotes the charge ending (panel B, orange arrow). At this point, the device is committed to deliver the shock, which was delivered at 34.8 joules (panel B, large black arrow). Interference continued while the patient was still in the hot tub. However, once the patient stepped out of the hot tub (panel C), sinus rhythm was visible.

Question

What is the best way to prevent inappropriate ICD therapies?

1. ICD interrogation and careful analysis of intracardiac EGMs and event markers
2. Reprogramming, if appropriate
3. Change the detection rate and therapies, if needed
4. Patient education to avoid any possible sources of electromagnetic interference

Discussion

With the increasing number of ICD recipients, and despite advanced ICD programming and algorithms to differentiate supraventricular from ventricular arrhythmias to avoid inappropriate ICD discharges, these events do still happen.

Other causes of inappropriate ICD discharges include:

1. Lead malfunction
2. Inappropriate programming
3. Atrial high rates, which mostly happen during AF, with rapid ventricular response
4. Oversensing or undersensing of the EGMs
5. Electromagnetic interference from devices such as:
   a. Transcutaneous electrical nerve stimulation devices
   b. Magnets
   c. Electric power tools and devices (drills, leaf blower, electric lawn mower, tablets, e-readers, cell phones, video game wireless controller, speakers, radio)
   d. Anti-theft systems (electronic article surveillance)
   e. Metal detectors
   f. Headphones

Upon further investigation of the hot tub after this event, maintenance noted that the light housing, which was built into the floor of the hot tub, was leaking some electrical voltage. Therefore, careful steps to avoid inappropriate ICD shock related to this case include patient education on the effects of external electromagnetic interference or implantable devices such as ICDs and pacemakers. The patient should also report this event to his/her physician. Obviously, reprogramming of the device for detection rate and therapies would not have been useful in this case.

To the best of our knowledge this is the first case report of hot tub-induced inappropriate ICD shock.

Reference

1. van Rees JB, Borleffs CJ, de Bie MK, et al. Inappropriate implantable cardioverter-defibrillator shocks: incidence, predictors, and impact on mortality. *J Am Coll Cardiol*. 2011;57(5):556–562.

CASE 12.7

Paolo China, MD
Sakis Themistoclakis, MD

Patient History

An 86-year-old male with a long history of severe ischemic cardiomyopathy with heart failure, diabetes, chronic obstructive pulmonary disease, end-stage renal insufficiency, and a pacemaker implantation was admitted at the emergency department (ED) for shortness of breath and swelling of the legs. In the ED, medical history, blood test, and a 12-lead ECG were performed and evaluated. The diagnosis was acute heart failure and patient started appropriate medical therapy. The first ECG (**Figure 12.7.1**) was suspected for pacemaker malfunction and an electrophysiologist consult was requested. When the cardiologist arrived to the ED, a second ECG (**Figure 12.7.2**) was performed and spontaneous sinus rhythm with first-degree AV block and frequent premature beats were observed without spikes or paced beats. Chest radiography was also performed (**Figure 12.7.3**).

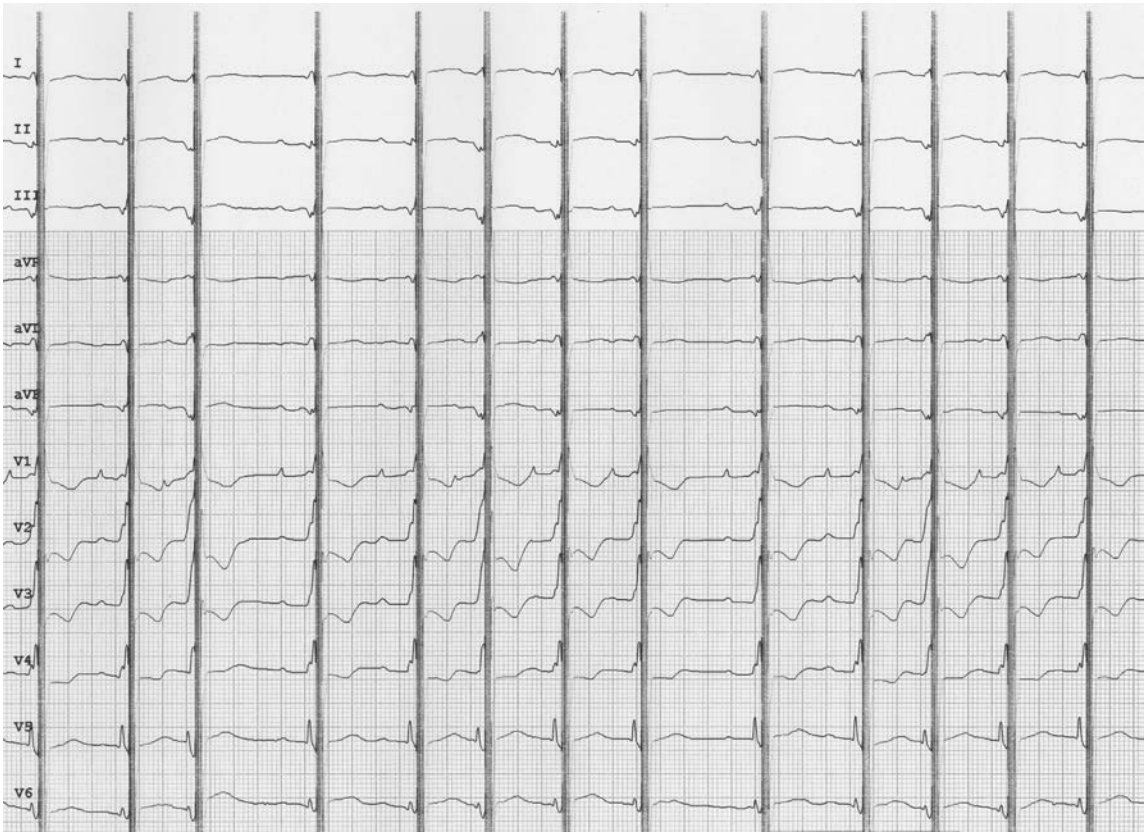


Figure 12.7.1 First 12-lead resting ECG obtained at patient admission (paper speed 25 mm/s).

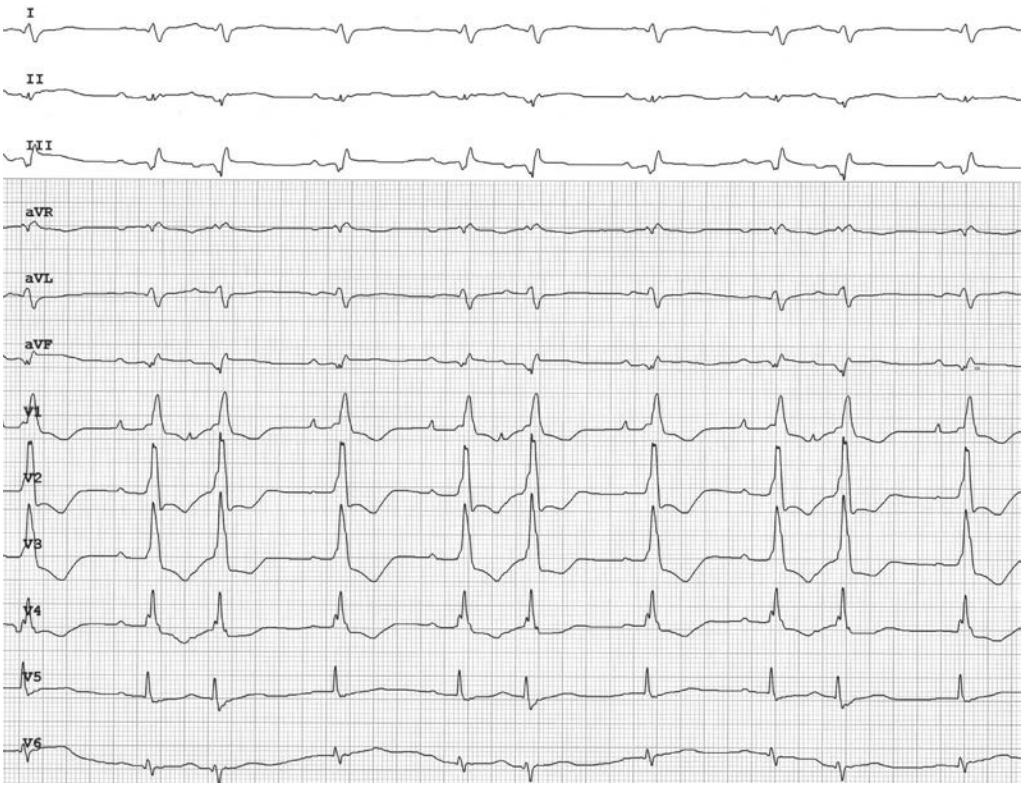


Figure 12.7.2 Second 12-lead ECG obtained about two hours after patient admission (paper speed 25 mm/s).

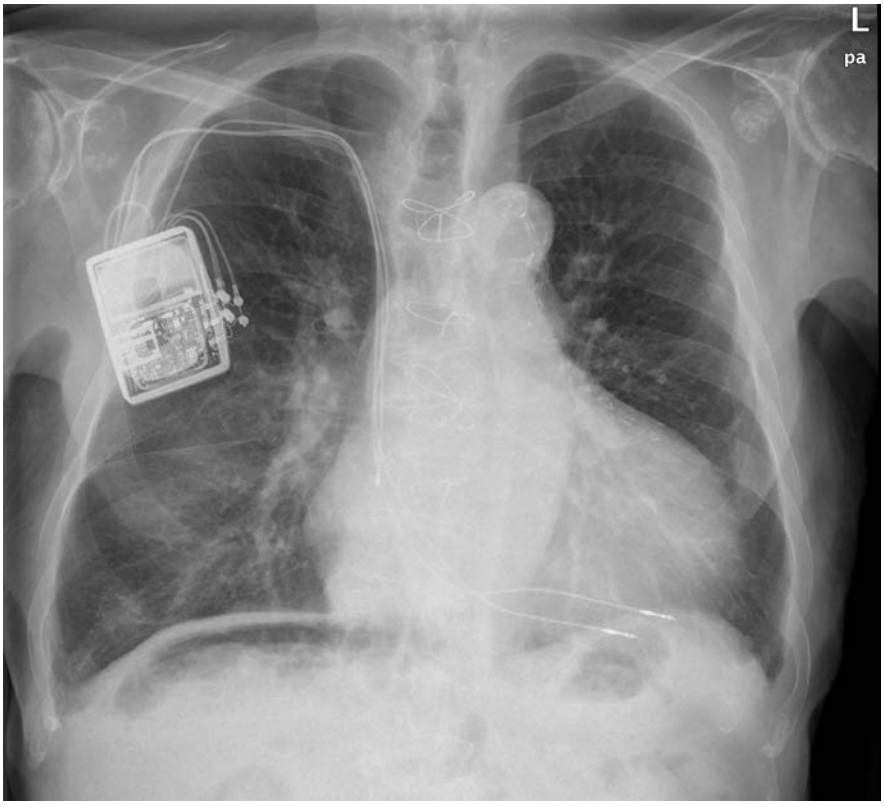


Figure 12.7.3 Posterior-anterior chest radiography.

Question

Is the pacemaker working well?

ECG Discussion

The first ECG (**Figure 12.7.1**) showed sinus rhythm at 95 bpm with a right atrial enlargement, a first-degree AV block and complete right bundle branch block with QRS duration of 150 ms. Q waves in the inferior leads were also visible, indicating an old inferior wall myocardial infarction. Interestingly, there are wide spike signals occurring after the beginning of every QRS with a long AVp interval. In the second ECG (**Figure 12.7.2**), these spikes completely disappear with the remaining findings unchanged. In particular, the QRS morphology was identical.

Discussion, Interpretation, and Answer

In **Figure 12.7.1**, spike signals were late after the beginning of the QRS and QRS morphology was not suggestive of right ventricular paced beat. Pacing stimulus occurred coincidently or after the intrinsic ventricular depolarization and cannot capture the ventricular myocardium because it was already depolarized. This phenomenon is called "pseudofusion." Therefore, the ECG in **Figure 12.7.1** could suggest a useless right ventricular pacing with pseudofusion in a patient with a simple dual chamber pacemaker. Spikes in **Figure 12.7.2** disappeared, which could be interpreted as a consequence of a pacing inhibition subsequent to the activation of a pacemaker algorithm to minimize right ventricular pacing that further prolonged the programmed AVp interval and detected a spontaneous QRS. However, at a more careful analysis, the presence of spikes that were wider and taller than usual were atypical for a standard dual chamber pacemaker. These ECGs could also be interpreted as an incorrect resynchronization therapy in a patient with a biventricular pacemaker with a delayed and high-voltage left ventricular pacing threshold. This second hypothesis was also unlikely for the long AVp interval and QRS morphology, which was different from that expected for left ventricular or biventricular pacing (rs in aVL and DI, tall R in V_1). This hypothesis was proved wrong after evidence of a similar spontaneous QRS in the second ECG. The chest radiography (**Figure 12.7.3**) showed the presence of a pacemaker implanted in the right subclavian region with three pacing leads, one positioned in the right atrium and two on the right ventricular septum. This unconventional pacing mode was explained by the type of device that was implanted. Indeed, this patient was treated with a Cardiac Contractility Modulator (CCM) (Impulse Dynamics Optimizer III) device instead of a biventricular pacemaker/defibrillator. This decision was made based on age, comorbidities, the presence of incomplete right bundle block (at that time QRS duration was 118 ms) and a large inferolateral akinesis on the echocardiogram. After device implantation, he had a significant increase in the left ventricular ejection fraction with reduction of symptoms and NYHA class improvement from III to I.

CCM gives a form of electrical therapy that enhances ventricular contractile strength independently of the synchrony of myocardial contraction. The stimulus is delivered 30–40 ms after detection of local myocardial activation during the absolute refractory period and consists of two biphasic ±7 V pulses spanning a total duration of 20 ms. Although it delivered about 50–100 times the amount of energy during a CCM than during a standard pacemaker impulse, it is a nonexcitatory therapy. These impulses can enhance contractile performance acutely and during long-term follow up with several possible mechanisms including improvement in gene expression,

protein phosphorylation, and reverse remodeling.[1,2] The first ECG (**Figure 12.7.1**) is perfectly compatible with the correct function of this particular device; the energy is delivered after the beginning of the QRS. Spike artifacts can disappear like in the second ECG (**Figure 12.7.2**) because CCM therapy is intermittently delivered for several hours per day.

References

1. Kuck KH, Bordachar P, Borggrefe M, et al. New devices in heart failure: An European Heart Rhythm Association report. *Europace.* 2014;16:109–128.
2. Borggrefe M, Burkhoff D. Clinical effects of cardiac contractility modulation (CCM) as a treatment for chronic heart failure. *Eur J Heart Fail*. 2012;14:703–712.

SECTION 13A

Arrhythmogenic Right Ventricular Dysplasia/Cardiomyopathy (ARVD/C)

CASE
13A.1

N.A. Mark Estes III, MD

Patient History

Figure 13A.1.1 is the ECG of a 20-year-old female presenting with hemodynamic compromise due to sustained monomorphic ventricular tachycardia (VT) with left bundle branch block (LBBB) morphology requiring cardioversion.

Questions

1. What is the ECG abnormality?
2. What cardiovascular condition is likely to cause the ECG abnormality and cardiac arrest?

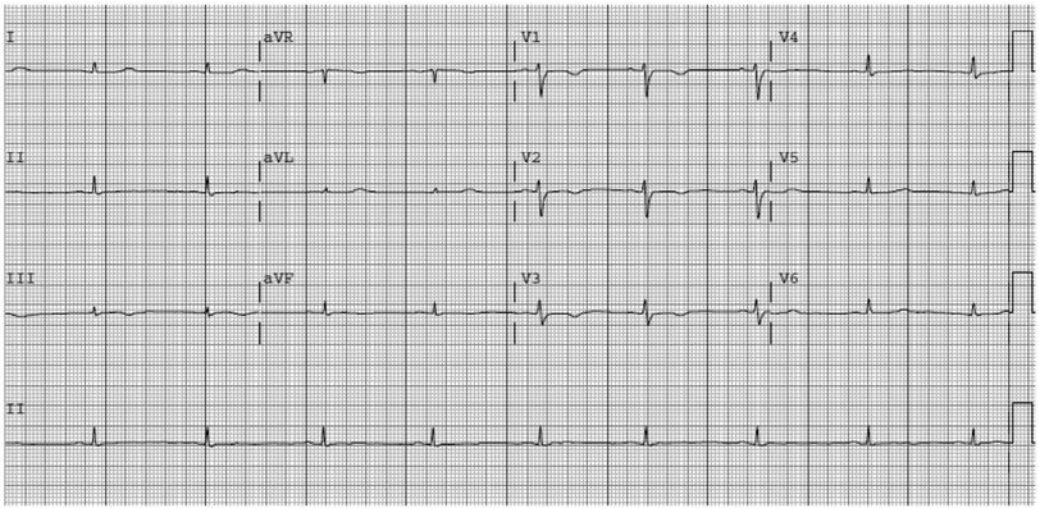


Figure 13A.1.1

Discussion

The ECG demonstrated low voltage with T-wave inversions in leads V_1–V_4. Arrhythmogenic right ventricular dysplasia/cardiomyopathy (ARVD/C) would most commonly be associated with these ECG findings, as well as cardiac arrest due to sustained VT with left bundle morphology. Echocardiogram and magnetic resonance imaging confirmed fibrofatty infiltration of both the right and left ventricle, diagnostic of ARVD/C with left ventricular involvement.

ECG Masters' Collection: Favorite ECGs from Master Teachers Around the World, Vol. 2 © 2018 Mohammad Shenasa, Mark E. Josephson, N.A. Mark Estes III, Ezra A. Amsterdam, Melvin Scheinman. Cardiotext Publishing, ISBN: 978-1-942909-20-0.

CASE 13A.2

Gilles Lascault, MD
Olivier Paziaud, MD

Patient History

A 59-year-old male, known to have an extensive arrhythmogenic right ventricular dysplasia (ARVD) with right ventricular dilatation. He had experienced several episodes of symptomatic sustained monomorphic ventricular tachycardia (VT), which led to the implantation of a dual chamber defibrillator in 2008.

Figure 13A.2.1 shows atrial paced beats with long AV intervals and spontaneous QRS complexes with a right bundle branch block (RBBB) morphology. Marked T-wave inversion is seen from leads V_1 to V_5. Note the presence of a positive notch at the end of S′ in leads V_1 and V_2 which is interpreted as an epsilon wave (red arrow).

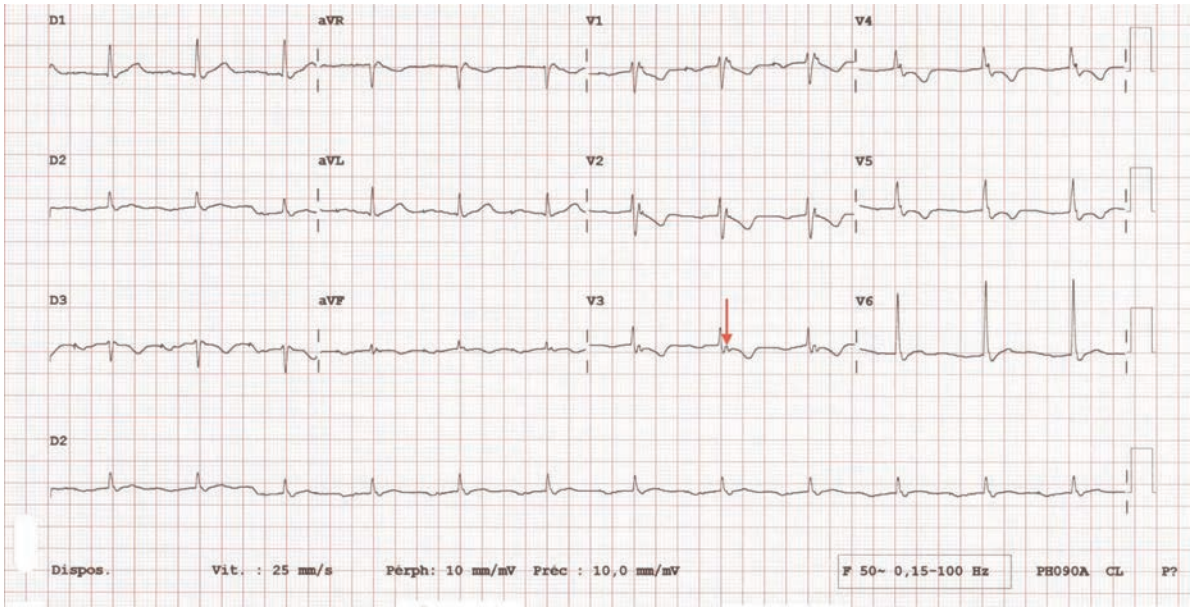


Figure 13A.2.1

Signal-averaged ECG indicates the presence of late potentials: nonfiltered QRS duration: 141 ms; filtered QRS duration: 167 ms; low-amplitude signal duration: 66 ms; terminal QRS root means square voltage: 10 mV. Therefore, late potentials are positive for the three criteria.

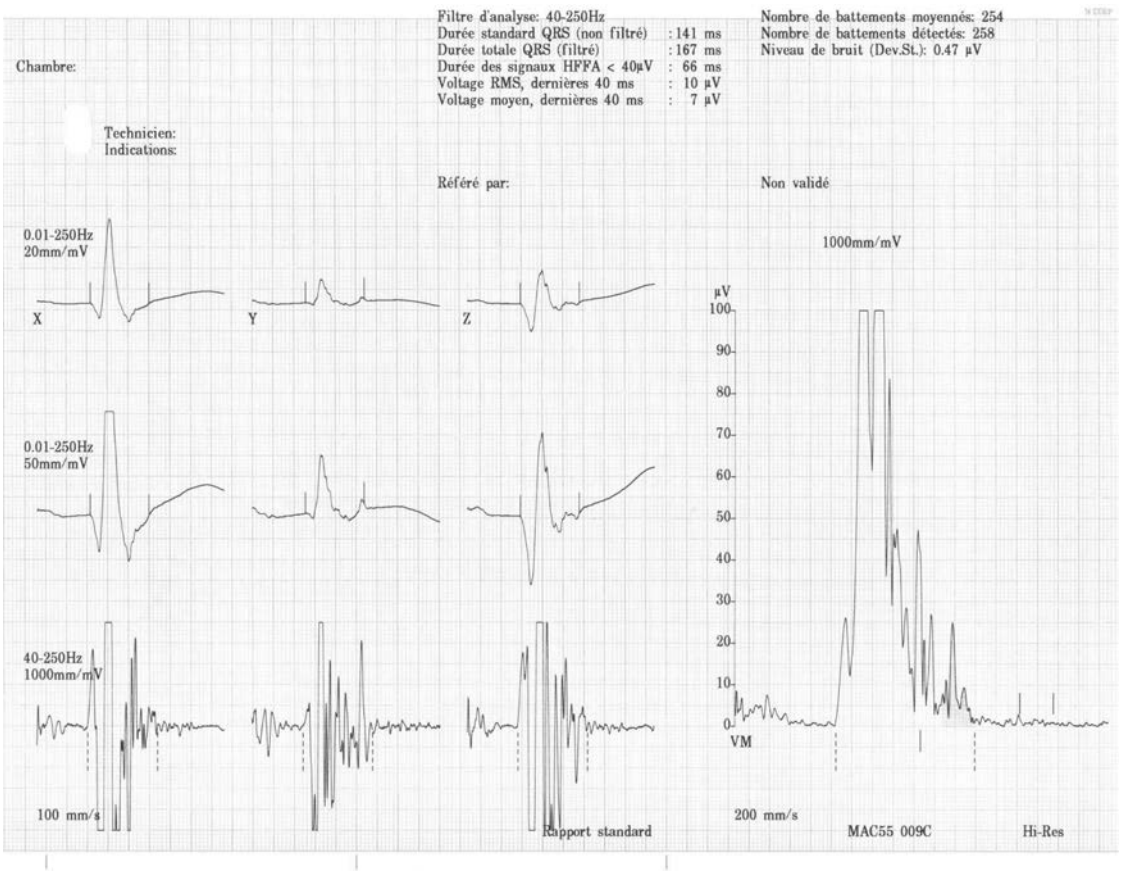


Figure 13A.2.2

Discussion

In ARVD, the prevalence of late potentials is approximately 70%; however, the presence of epsilon waves on the standard ECG is less frequent (30% of cases) and is only observed in advanced cases of ARVD with diffuse conduction disturbances and large areas of slow and delayed conduction in the right ventricle. The epsilon wave is a reflection of "mega" late potentials, which are present in this patient, and strongly positive for the three criteria (3/3). Epsilon wave is a major diagnostic criterion for ARVD. It must not be mistaken for early repolarization, which occurs at the end of the QRS complex as well but is predominant in leads V_4–V_6 and can also be seen in leads I, II, and III in the "major forms."

Reference

1. Zhang L, Liu L, Kowey PR, Fontaine GH. The electrocardiographic manifestations of arrhythmogenic right ventricular dysplasia. *Curr Cardiol Rev*. 2014;10(3):237–245.

CASE 13A.3

Yuji Nakazato, MD, PhD

Patient History

A 57-year-old male arrived to the hospital with recurrent palpitations. His brother had died suddenly during athletic activity at the age of 26.

Question

What kind of electrocardiographic finding does the arrow suggest?

Discussion

In this case, a prominent epsilon wave (arrow) is clearly shown on precordial leads V_1 and V_2, which are both in sinus rhythm with slow atrial flutter (★) during palpitation (**Figures 13A.3.1** and **13A.3.2**). The epsilon wave is observed in approximately 30% of arrhythmogenic right ventricular dysplasia (ARVD) cases.[1] It represents the delay in depolarization of the right ventricle free wall and outflow tract. Signal-averaged ECG showed positive diagnosis of ventricular late potential (filtered QRS 197 ms, RMS40 11.7μV; **Figure 13A.3.3**). If the epsilon wave is too large, it might be misunderstood as a nonconducted premature atrial contraction.

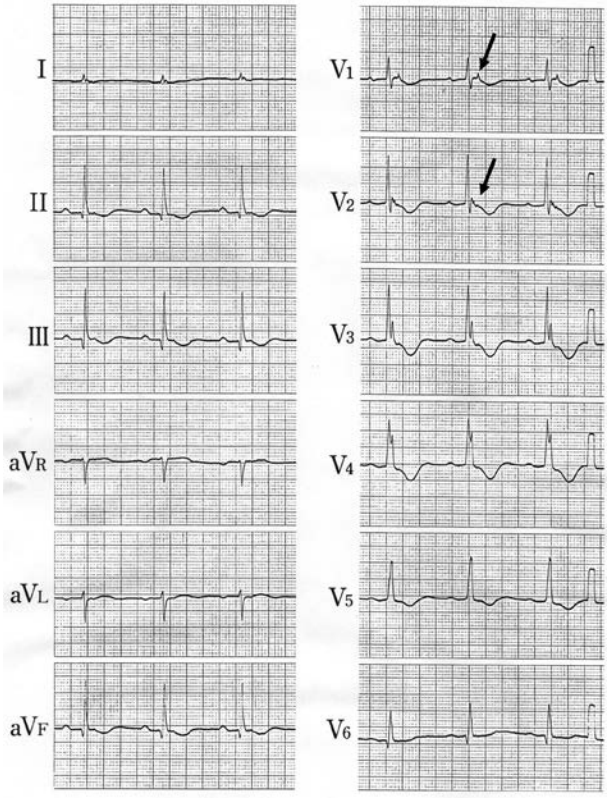


Figure 13A.3.1

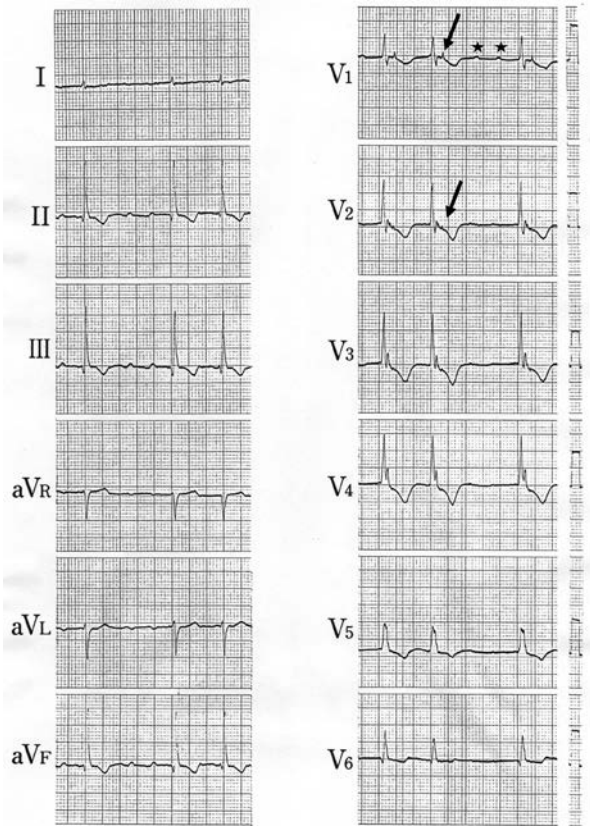


Figure 13A.3.2

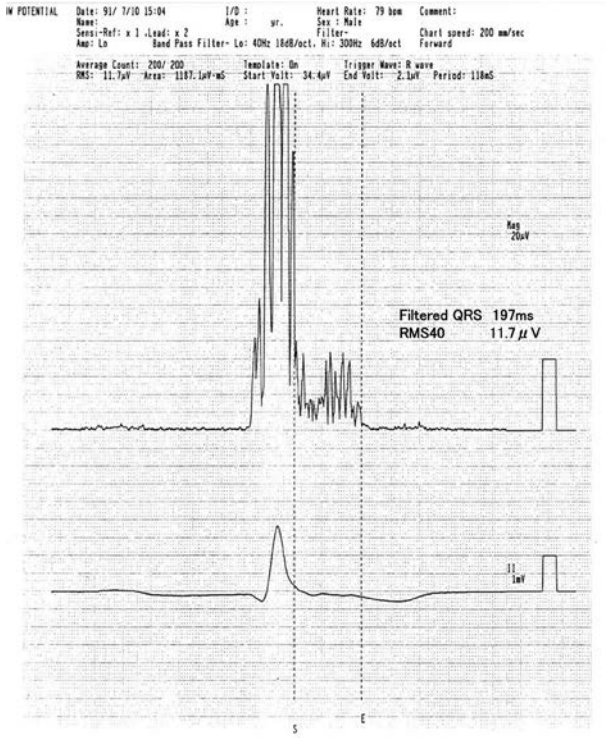


Figure 13A.3.3

Although reentrant ventricular tachycardia commonly appears in ARVD, supraventricular tachycardia occurred in this case even during antiarrhythmic drug therapy.[2] Therefore, the atrial flutter observed in this case is not rare.

References

1. Marcus FI, Fontain G, Guiraudon G, et al. Right ventricular dysplasia: A report of 24 adult cases. *Circulation.* 1982;65:384–398.
2. Tonet JL, Miranda RC, Iwa T, et al. Frequency of supraventricular tachyarrhythmias in arrhythmogenic right ventricular dysplasia. *Am J Cardiol.* 1991;67:1153.

Paolo China, MD
Sakis Themistoclakis, MD

CASE 13A.4

Patient History

A 45-year-old male without any personal or family history of heart disease traveling by cruise ship had a sudden onset of abdominal pain, fatigue, and dyspnea. The physician on board initially suspected gastrointestinal disease The patient had yellow sclera, laboratory results showed slight increase in troponin serum level, normal myoglobin and modest increase of unconjugated bilirubin and d-dimer serum concentration. A 12-lead ECG was acquired (**Figure 13A.4.1**). Suspecting an acute coronary syndrome, aspirin, clopidogrel, heparin, and metoprolol were given. The ship course was immediately diverted to the nearest port to admit the patient to the closest hospital. During the subsequent hours, the patient felt palpitations and a second ECG documented a well-tolerated wide QRS-complex tachycardia (WCT) (**Figures 13A.4.2A** and **13A.4.2B**). It was treated successfully with IV amiodarone. The patient started treatment with amiodarone and metoprolol but in the following days other recurrences of WCT with a heart rate of 100–110 bpm were observed (**Figures 13A.4.3** and **13A.4.4**).

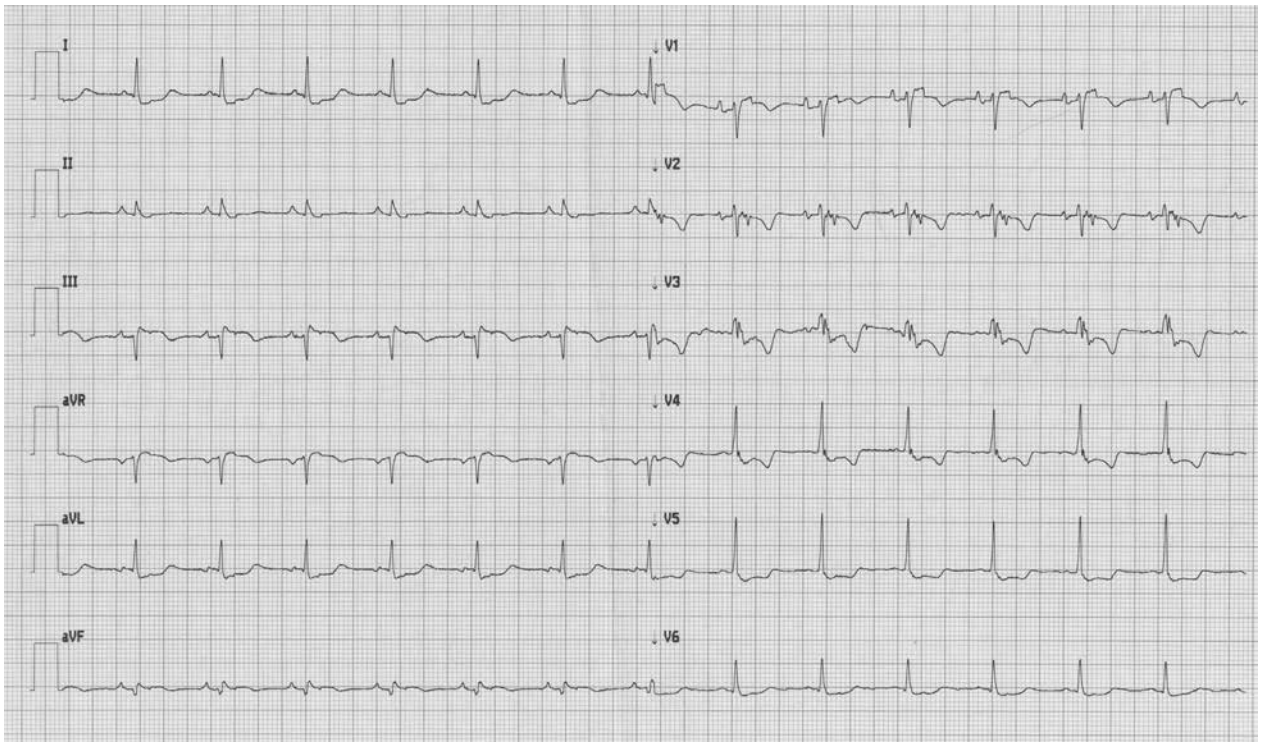
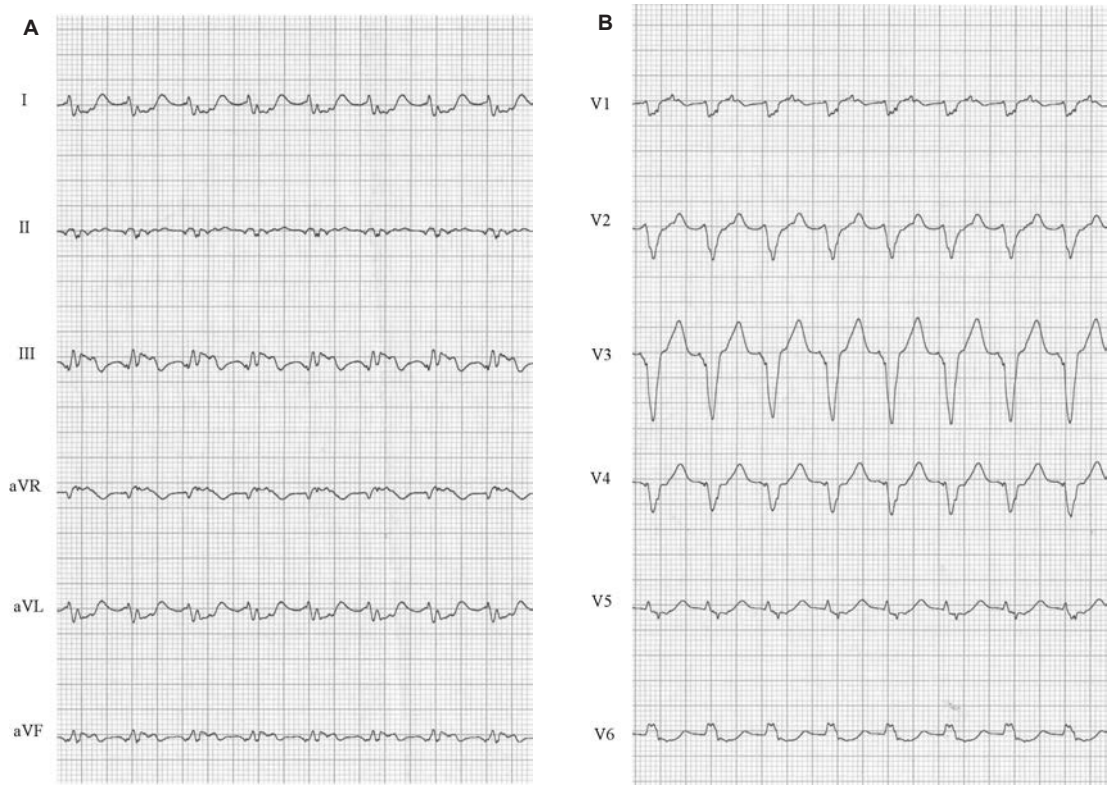


Figure 13A.4.1 12-lead resting ECG (paper speed 25 mm/s).



Figures 13A.4.2A and B 12-lead ECG during the first tachycardia (paper speed 25 mm/s).

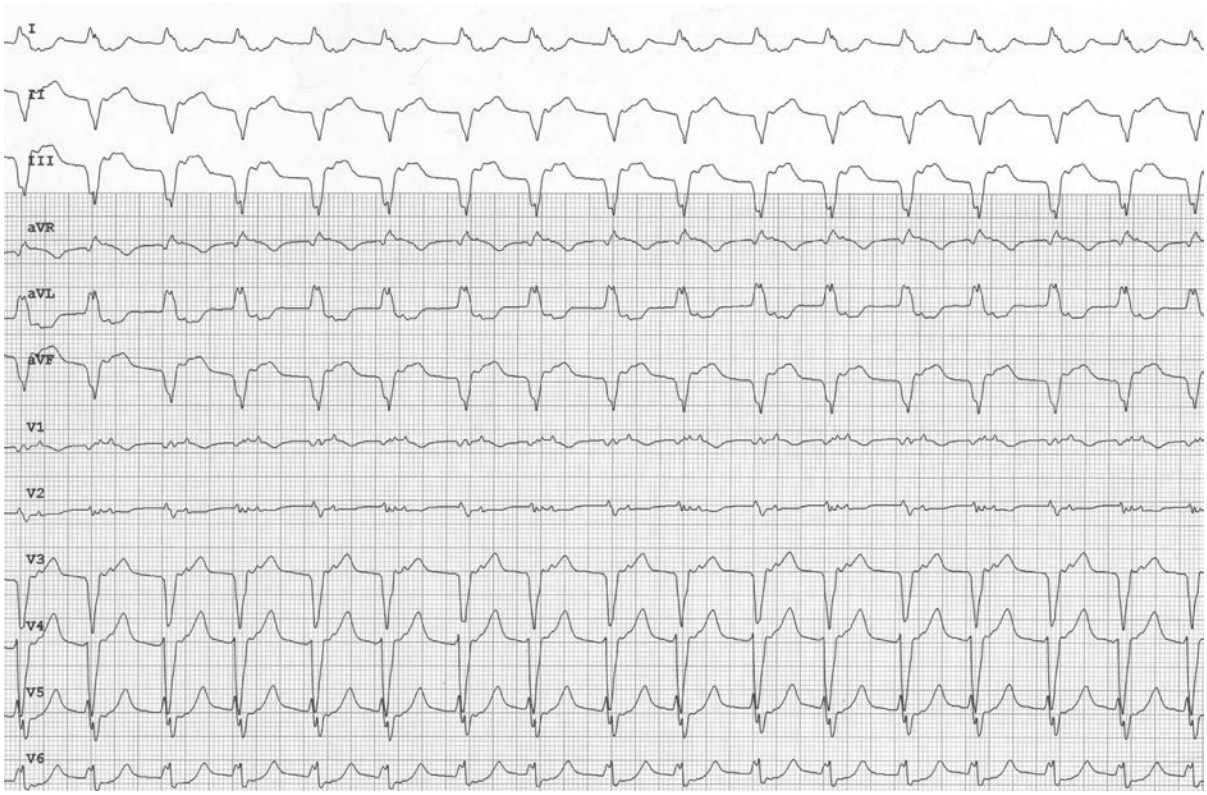


Figure 13A.4.3 12-lead ECG during the second tachycardia (paper speed 25 mm/s).

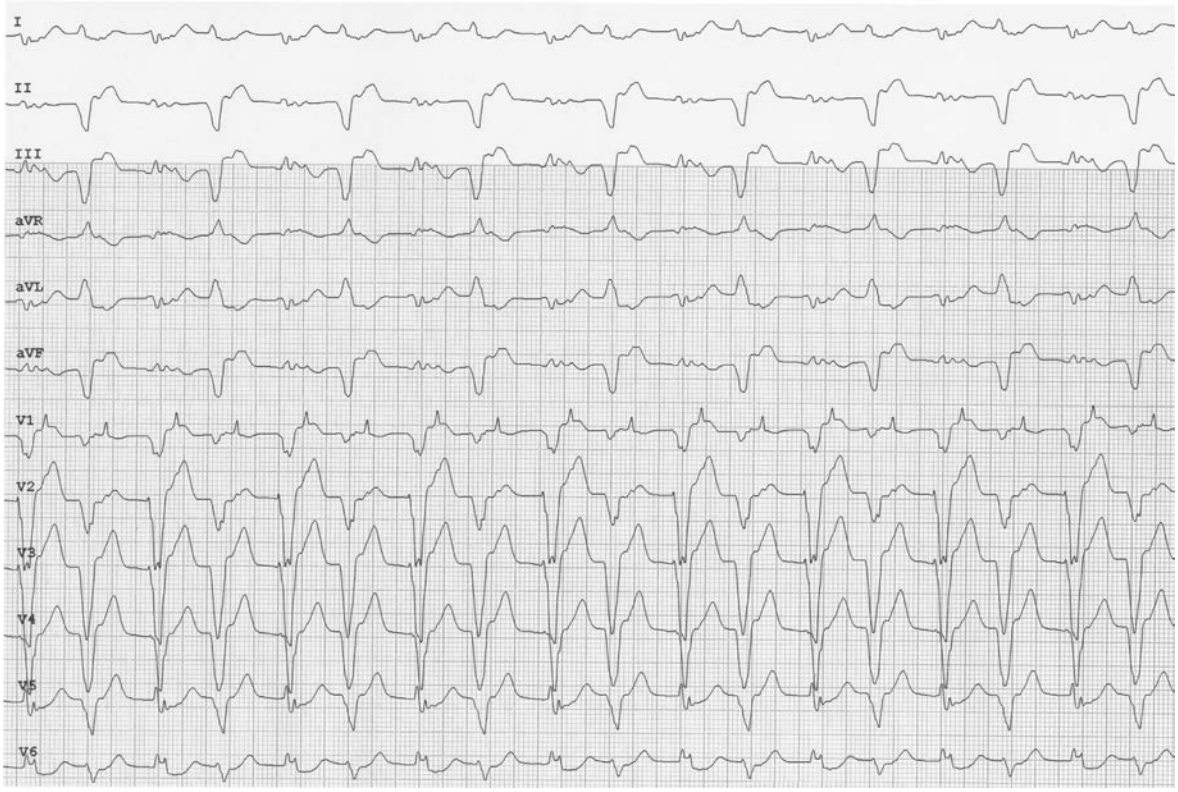


Figure 13A.4.4 12-lead ECG during ventricular tachycardia with ventricular ectopy (paper speed 25 mm/s).

Question

What is the diagnosis?

The first ECG (**Figure 13A.4.1**) at rest showed sinus rhythm at 82 bpm, right atrial enlargement, and right ventricular conduction delay with QRS duration >110 ms in leads V_1–V_3. The QRS morphology showed Q waves in the inferior leads, deeper in DIII, with mild ST elevation and a S1Q3T3 pattern with prolonged S wave–upstroke in leads V_2 and V_3. Epsilon waves and inverted T waves were observed in leads V_1–V_4. Subsequent ECGs showed ventricular tachycardia with left bundle branch block (LBBB) morphology and right axis deviation. Retrograde ventriculoatrial (VA) conduction is almost always present (fixed VA interval, negative P in inferior leads, taller and narrower P wave in V_1). The last ECG (**Figure 13A.4.4**) was acquired a few days after starting amiodarone therapy in addition to a high dose of metoprolol. It captured a very slow, sustained ventricular tachycardia associated with monomorphic ventricular premature ectopic beats at a fixed coupling interval (all ventricular beats had retrograde VA conduction).

Discussion, Interpretation, and Answer

An echocardiogram showed severe right ventricular (RV) enlargement with regional apical dyskinesia, mild left ventricular dysfunction, mild mitral and aortic regurgitation, and slight elevation of systolic pulmonary pressure. A chest x-ray displayed bilateral pleural effusion. Troponin I serum level did not significantly increase (peak 0.214 ng/mL) and had a subsequent gradual normalization. Brain natriuretic peptide serum level at presentation was 702 mg/dL. Coronary

angiography showed no significant coronary disease. Cardiac magnetic resonance imaging was diagnostic for arrhythmogenic right ventricular dysplasia/cardiomyopathy (ARVD/C). Therefore, the diagnosis of different sustained ventricular tachycardia in patients with ARVD/C was established.

ARVD/C is a genetically determined myocardium disorder characterized by fatty or fibro-fatty replacement with thinning of the RV wall in three typical regions (basal RV, apical RV, and RV outflow tract). The disease is often associated with focal myocarditis and the RV usually becomes dilated and dysfunctional. A left dominant variant is also recognized. The disease has an autosomal dominant inheritance, although there are recessive forms associated with a cutaneous phenotype. Clinical presentation varies with the age and stage of disease, and can be characterized by ventricular arrhythmias (usually originating from the RV) and more rarely by RV failure; symptoms like syncope, palpitation, and sudden death are more common during exercise. The main differential diagnosis includes pulmonary embolism, myocarditis, Brugada syndrome, Naxos disease, sarcoid heart disease, and right ventricular outflow tract tachycardia. Clearly, family history, echocardiogram, right ventricular angiography, cardiac magnetic resonance imaging and endomyocardial biopsy are useful tools; however, the ECG is the cornerstone in diagnosis for about 90% of patients and its alterations advance the clinical manifestation.

Major and minor criteria for the diagnosis of ARVD/C have been established and revised in 2010.[1]

References

1. Marcus FI, McKenna WJ, Sherrill D, et al. Diagnosis of arrhythmogenic right ventricular cardiomyopathy/dysplasia: Proposed modification of the Task Force Criteria. *Eur Heart J*. 2010;31(7):806–814.
2. Steriotis AK, Bauce B, Daliento L, et al. Electrocardiographic pattern in arrhythmogenic right ventricular cardiomyopathy. *Am J Cardiol.* 2009;103(9):1302–1308.

SECTION 13G

Pericarditis

Andrés Ricardo Pérez-Riera, MD, PhD
Adrian Baranchuk, MD

CASE 13G.1

Patient History

A 70-year-old female with long-standing hypertension treated with an angiotensin converting enzyme inhibitor. She was admitted to the emergency department (ED) with symptoms of progressive dyspnea on exertion that had worsened over the past 20 days with frank respiratory failure. She had prior history of left nephrectomy for unknown reasons.

Physical Examination

Orthopnea, blood pressure 160/80 mmHg, heart rate 115 bpm.

Laboratory assessment revealed SpO2 of 86% (normal SpO_2 95–100%), and mild hyperkalemia (K^+ 6.2 mEq/L).

Her ECG at admission (**Figure 13G.1.1**) revealed tall, peaked, symmetrical, with narrow-base T waves.

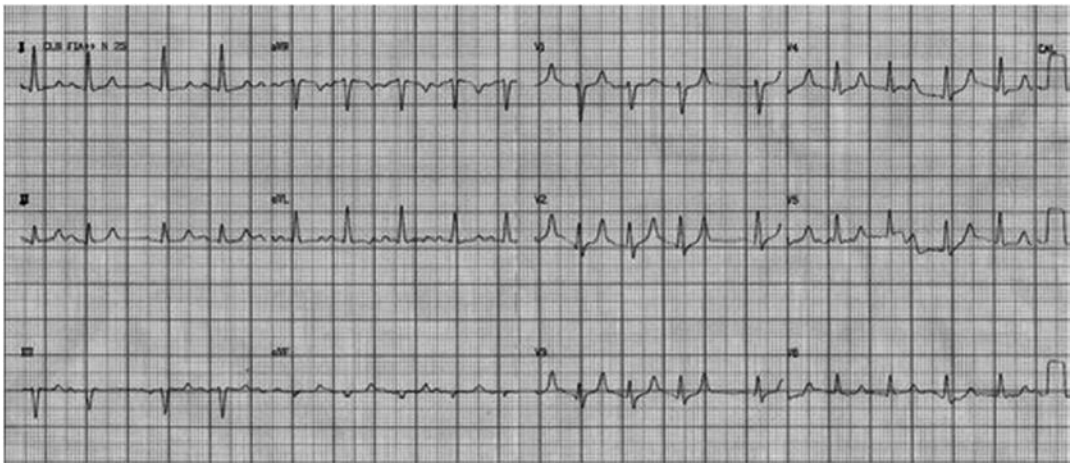


Figure 13G.1.1 ECG diagnosis: Sinus tachycardia with peaked, symmetrical with narrow base T waves ("in desert-tent" or "Eiffel tower-like"). These are the earliest repolarization abnormalities when serum potassium levels exceed >5.5 mEq/L.

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Initial Management

1. Oral tracheal intubation and mechanical ventilation.
2. Intravenous administration of: 10 mL calcium gluconate (to stabilize the myocardial membrane), sodium bicarbonate (1 mEq/kg, IV bolus), and regular insulin with glucose (10 U IV + 50 g of glucose). After these measures, the T-wave abnormalities in the ECG improved.

The patient subsequently developed intense chest pain, and serial ECGs, depicted in **Figures 13G.1.2** and **13G.1.3**, were obtained. The abnormalities observed in these tracings were initially interpreted as an ST-segment elevation myocardial infarction (STEMI). ECG diagnosis of STEMI requires the presence of >1.0 mm ST-segment elevation in at least two contiguous ECG leads in the frontal plane or lateral precordial leads (V_4 and V_5), >2.0 mm in the right precordial leads (V_1–V_3), or presumably new complete left bundle branch block (LBBB). Due to the ECG findings along with chest pain, she immediately underwent coronary angiography that revealed normal coronary arteries.

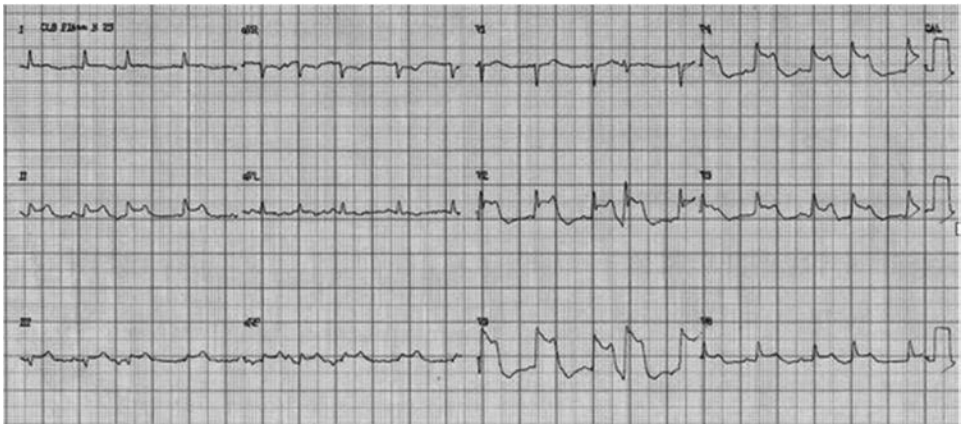

Figure 13G.1.2 A concave upwards ST-segment elevation with great number of involved leads (anterior and inferior walls) (nonsegmentary), premature atrial complexes (during chest pain episode), reciprocal changes or mirror image (ST-segment depression) only in aVR.

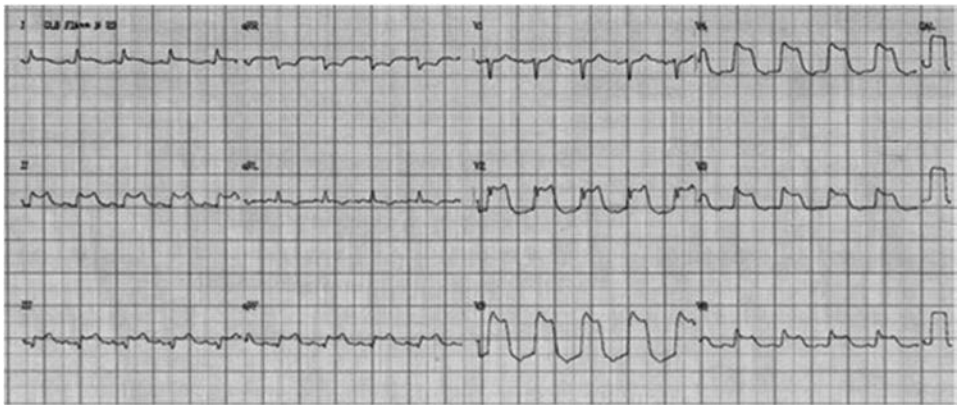

Figure 13G.1.3 Marked diffuse concave upwards ST-segment elevation more prominent from leads V_2 to V_6, II and III with PR segment elevation in aVR (during chest pain). This pattern in the mid precordial leads resembles an intracellular monophasic action potential (a single polarity) and can be misdiagnosed as an acute STEMI. The combined appearance of ST elevation in both anterior and inferior leads along with PR segment shifts opposite to P polarity may also be suspicious of stage I acute pericarditis.[4,16] The typical pattern in acute pericarditis is diffuse ST-segment elevation in leads I, II, aVL, aVF and from V_3 to V_6. ST-segment depression is always seen in lead aVR, frequently in V_1, and occasionally in V_2.[10]

Question

What is the diagnosis?

Discussion

The diagnosis in this case was acute pericarditis. (See below for ECG evidence of pericarditis in this case.) Acute pericarditis is an inflammatory disease of the pericardium due to multiple causes. The real incidence is not known; however, in individuals who are not immunosuppressed, 90% of cases have a viral or idiopathic etiology.[9,17] Acute pericarditis is found in approximately 5% of the patients admitted in the ED for chest pain not due to acute coronary syndrome. It occurs more frequently in men between ages 20–50 years old. Possible etiologies of acute pericarditis include: idiopathic, infection (viral, bacterial, fungal, and parasitic), Dressler syndrome, drugs (e.g., anticoagulants, hydralazine, isoniazid, methysergide, penicillin, phenytoin, procainamide), chest wall trauma, systemic autoimmune disorders, cancer, and uremia. The diagnosis is based on a history of pleuritic chest pain and the presence of a pericardial rub (present in 85% of the cases).[12] Typical ECG changes are characterized by diffuse and widespread concave upwards ST-segment elevation often confused with STEMI.[2,5,9,11] At least two of four criteria are necessary for diagnosis: pleuritic chest pain, pericardial friction rub, diffuse concave upwards ST-segment elevation, and pericardial effusion.[6] A pericardial rub is virtually 100% specific for the diagnosis; however, although the sensitivity is variable (16%–85%) depending on the frequency of auscultation and etiology.[2,9] The rub is best heard at end-expiration with the diaphragm of the stethoscope placed in the low left sternal border while the patient is sitting forward. Erb's point is the ideal location for the hearing the rub. The electrocardiographic alterations in pericarditis involve PR segments, ST segments, and rhythm according to the time course of pericarditis. The ECG can be normal in approximately 6% of cases. In acute pericarditis, the ECG changes evolve over four serial stages.[3,13]

Stage I

Concave upwards ST-segment elevation in multiple leads except aVR and V_1 where ST-segment depression may occur; symmetrical and peaked T waves with narrow base and mild increase in voltage; and PR-segment depression (except in aVR, where there is PR-segment elevation). These alterations are observed in more than 80% of the cases.[1,3]

Stage II

Reversion of ST and PR segments shifts along with T-wave flattening (after some days).

Stage III

Diffuse T-wave inversion resembling myocardial ischemia. Absence of Q waves indicative of myocardial necrosis is a useful clue to differentiate this stage of pericarditis from the diagnosis of myocardial infarction.

Stage IV

T-wave abnormalities resolve to more normal morphology; this may take weeks to months after the onset of acute pericarditis.

Alterations in rhythm may occur during any stage and include sinus tachycardia and various atrial arrhythmias.[14] In the presence of pericardial effusion, the most typical finding is low QRS voltage,[8] which improves after pericardiocentesis. In the setting of pericardial tamponade electrical alternans of QRS voltage may be seen. Low QRS voltage is defined as complexes of less than 5 mm (0.5 mV) in the frontal plane leads and less than 10 mm in the precordial leads. QRS alternans is the beat-by-beat variation in axis, amplitude and/or morphology. Low voltage may also be present in other diseases including emphysema, infiltrative myocardial disease, and pneumothorax. After pericardial drainage the QRS voltage does not normalize immediately suggesting a continued inflammatory process. Electrical alternans is the result of the heart moving around in a large effusion. This pattern is more visible in the precordial leads. Isolated QRS alternans is not specific to pericardial effusion, but the presence of *both* P and QRS alternans is pathognomonic.

The most frequent type of alternans involves only the QRS. When P, QRS, and T waves are included, it is called total alternans and is observed in case of cardiac tamponade. Beck's triad (jugular vein distention, hypotension, and muffled sounds) is a feature of cardiac tamponade.

Table 13G.1.1 Shows the main differences between acute pericarditis and acute coronary syndrome.

	Coronary artery disease (STEMI)	Pericarditis
Number of involved leads	Less (segmentary)	Greater (diffuse) and extensive
Intensity of the phenomena	Greater	Lesser
Reciprocal effect or mirror image	Frequently present	Absent, except aVR
ST-segment elevation convex upward or horizontal	Frequently present. In the initial phases is possibly concave upward.	Absent, always concave to the top
PR segment depression	Possible if atrial infarction	Only in viral pericarditis
ST-segment elevation in leads III > II	Characteristic when present	Absent

Table 13G.1.2 Shows the main differences between acute pericarditis and "benign" early repolarization pattern.

	Acute pericarditis	"Benign" early repolarization pattern
ST-segment elevation degree	2 mm	1 mm
T-wave height	Lesser (4 mm)	Greater (6 mm)
ST / T wave ratio	0.5	0.16
Number of leads involved	Universal elevation (all of them). There may be reciprocal depression in opposite leads.	The ST / T wave ratio < 0.25 followed by prominent T waves
"Fish hook" pattern notched near J-point	Absence of "fish hook" appearance in V_4	This is often best seen in lead V_4.
ST-segment elevation characteristic	Normal T-wave amplitude. It decreases amplitude in hours. Lower voltage. Only increased in early phase.	ST elevation limited to the precordial leads. Reciprocal depression only in aVR.
PR depression	Frequently present	Absent

Table 13G.1.2 (*Continued*)

	Acute pericarditis	"Benign" early repolarization pattern
Time evolution of ECG changes	Slowly over time	Usually stable (i.e., non-progressive)
Response to strain of ST-segment elevation	Not modified	Frequent return of ST to baseline. T wave may normalize.
Hyperventilation	T polarity is not modified	T polarity may be modified
Heart rate	Frequent tachycardia	Frequent bradycardia
Presentation	Transitory	Stable
Clinical picture	Marked alteration	Asymptomatic
Age range incidence	≥ 40 years old	20 to 40 years old

References

1. Baljepally R, Spodick DH. PR-segment deviation as the initial electrocardiographic response in acute pericarditis. *Am J Cardiol.* 1998;81(12):1505–1506. doi: 10.1016/S0002-9149(98)00217-3.
2. Bonnefoy E, Godon P, Kirkorian G, et al. Serum cardiac troponin I and ST-segment elevation in patients with acute pericarditis. *Eur Heart J*. 2000;21(10):832–836. doi: 10.1053/euhj.1999.1907.
3. Bruce MA, Spodick DH. Atypical electrocardiogram in acute pericarditis: Characteristics and prevalence. *J Electrocardiol.* 1980;13(1):61–66. doi: 10.1016/S0022-0736(80)80011-2.
4. Bruch C, Sheinermund A, Dagres N, et al. Changes in QRS voltage in cardiac taponade and pericardial efusion: Reversibility after pericardiocentesis and after anti-inflamatory drug treatment. *J Am Coll Cardiol*. 2001;38(1):219–226. doi:10.1016/S0735-1097(01)01313-4.
5. Imazio M, Demichelis B, Parrini I, et al. Day-hospital treatment of acute pericarditis: A management program for outpatient therapy. *J Am Coll Cardiol*. 2004;43(6):1042–1046. doi:10.1016/j.jacc.2003.09.055.
6. Imazio M, Spodick DH, Brucato A, et al. Diagnostic issues in the clinical management of pericarditis. *Int J Clin Pract*. 2010;64(10):1384–1392. doi: 10.1111/j.1742-1241.2009.02178.x.
7. Imazio M, Spodick DH, Brucato A, et al. Controversial issues in the management of pericardial diseases. *Circulation*. 2010;121(7):916–928. doi: 10.1161/CIRCULATIONAHA.108.844753.
8. Jung HO, Seung KB, Madias JE. Electrocardiographic changes resulting from pericardial effusion drainage. *Am J Cardiol.* 2010;106(3):437–441. doi: 10.1016/j.amjcard.2010.03.044.
9. Lange RA, Hillis LD. Clinical practice. Acute pericarditis. *N Engl J Med.* 2004;351(21):2195–2202. doi: 10.1056/NEJMcp041997.
10. Maisch B, Seferović PM, Ristić AD, et al. Guidelines on the diagnosis and management of pericardial diseases executive summary; The task force on the diagnosis and management of pericardial diseases of the European Society of Cardiology. *Eur Heart J*. 2004;25(7):587–610. doi: 10.1016/j.ehj.2004.02.002.
11. Salisbury AC, Olalla-Gomez C, Rihal CS, et al. Frequency and predictors of urgent coronary angiography in patients with acute pericarditis. *Mayo Clin Proc.* 2009;84(1):11–15. doi: 10.1016/S0025-6196(11)60801-X.
12. Snyder MJ, Bepko J, White M. Acute pericarditis: Diagnosis and management. *Am Fam Physician*. 2014;89(7):553–560. http://www.aafp.org/afp/2014/0401/p553.html. Accessed Oct. 10, 2015.
13. Spodick DH. Diagnostic electrocardiographic sequences in acute pericarditis. Significance of PR segment and PR vector changes. *Circulation*. 1973;48(3):575–580. doi: 10.1161/01.CIR.48.3.575.
14. Spodick DH. Arrhythmias during acute pericarditis: A prospective study of 100 consecutive cases. *JAMA*. 1976;235(1):39–41. doi:10.1001/jama.1976.03260270025020.
15. Spodik DH. *Pericardial diseases*. In: Braunwald E, Zippes DP, Libby P, eds. *Heart Disease*. 6th ed. Philadelphia, London, Toronto, Montreal, Sydney, Tokyo: W.B. Saunders. 2001:1823–1876.
16. Spodick DH. Acute pericarditis: Current concepts and practice. *JAMA*. 2003;289(9):1150–1153. doi:10.1001/jama.289.9.1150.
17. Zayas R, Anguita M, Torres F, et al. Incidence of specific etiology and role of methods for specific etiologic diagnosis of primary acute pericarditis. *Am J Cardiol.* 1995;75(5):378–382. doi: 10.1016/S0002-9149(99)80558-X.

SECTION 15

Special Considerations: Age, Race, Gender, and Athletes

Gemma Parry-Williams, MBChB, MRCP (UK)
Sanjay Sharma, BSc (Hons), MD

CASE 15.1

Patient History

This ECG (**Figure 15.1.1**) was obtained from an asymptomatic cyclist. What further investigation is required?

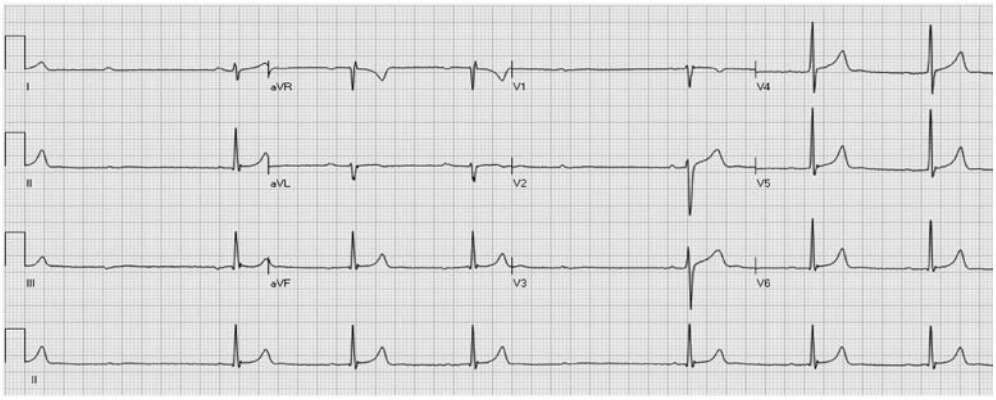

Figure 15.1.1

Question

This athlete's ECG shows a junctional rhythm with a narrow QRS. What would you do next?

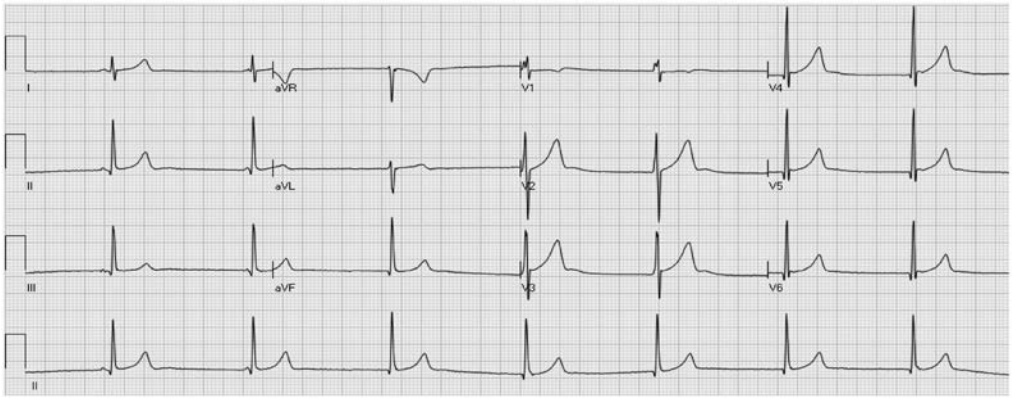

Figure 15.1.2

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Discussion

It has been well established that the ECG in the athletically trained heart can exhibit features of high vagal tone. Sinus bradycardia as low as 30 bpm and a PR interval of up to 400 ms are commonplace and do not require further evaluation. Exercise testing is useful to demonstrate chronotropic competence and PR shortening under these circumstances.

Although less frequent, Mobitz I second-degree atrioventricular (AV) block (Wenckebach) and junctional rhythms are also recognized within the physiological spectrum in the asymptomatic athlete (2.4% and 0.3%, respectively). The ECG in **Figure 15.1.1** shows Mobitz I second-degree AV block (Wenckebach) and as such, no further investigation is required. Likewise, **Figure 15.1.2** shows a junctional rhythm and in an asymptomatic athlete does not require any further management. However, higher degrees of AV block, including Mobitz II and complete heart block, are an exceptionally rare physiological finding and should always be investigated further with echocardiography, exercise tolerance testing, ambulatory ECG monitoring, and possibly cardiac magnetic resonance imaging (MRI).

An interesting finding in athletes, not to be confused with complete heart block, is *AV mismatch*, in which the AV node discharges and captures the ventricle faster than the sinoatrial node as a consequence of high vagal tone, with resulting AV dissociation.

References

1. Sharma S, Whyte G, Elliott P, et al. Electrocardiographic changes in 1000 highly trained junior elite athletes. *Br J Sports Med.* 1999;33(5):319–324.
2. Papadakis M, Basavarajaiah S, Rawlins J, et al. Prevalence and significance of T-wave inversions in predominantly Caucasian adolescent athletes. *Eur Heart J.* 2009;30(14):1728–1735.
3. Sharma S, Drezner JA, Baggish A, et al. International recommendations for electrocardiographic interpretation in athletes. *J Am Coll Cardiol.* 2017;69(8):1057–1075.

Gemma Parry-Williams, MBChB, MRCP (UK)
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CASE 15.2

Patient History

An African-American male soccer player has experienced transient dizziness on two occasions after a strenuous game. He has never lost consciousness. There is no relevant family history. Physical examination and echocardiography were normal.

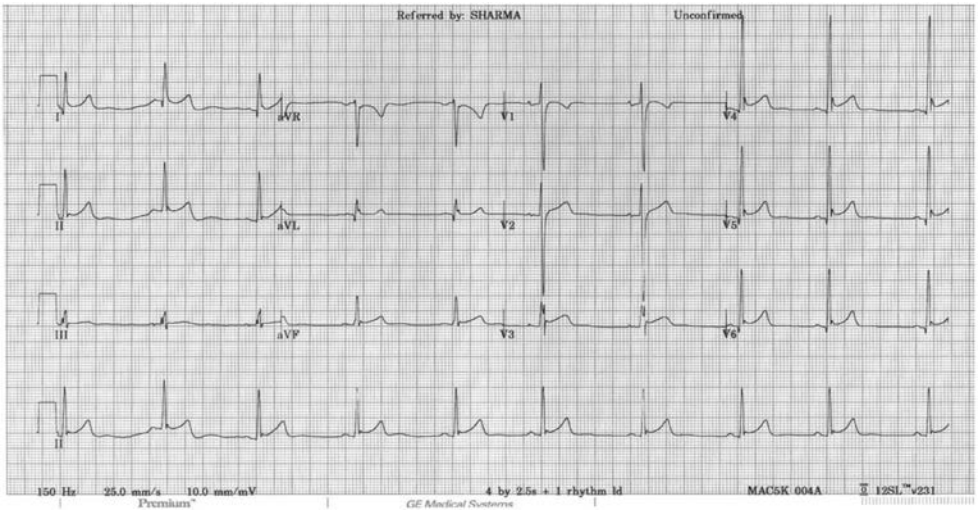

Figure 15.2.1

Question

Is this ECG (**Figure 15.2.1**) normal in the clinical context?

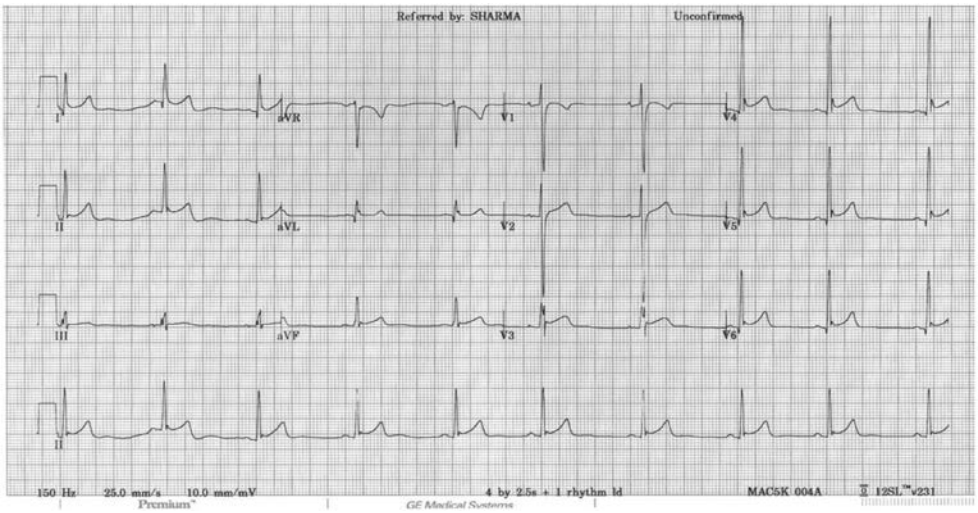

Figure 15.2.2 ECG consistent with early repolarization pattern (ERP). Findings include J-point elevation and notched J waves in leads V_3–V_6, I, and II.

Discussion

The ECG in **Figure 15.2.2** demonstrates the early repolarization pattern (ERP), which is defined as elevation of the QRS-ST junction (J-point) of >0.1 mV, notching of the J-point or slurring of the terminal QRS, with or without ST-segment elevation. The ERP is recognized in 23–44% of athletes, with a predominance in black males (63–91%). **Figure 15.2.3** shows examples of commonly seen ERPs in black athletes. Some small studies including both athletes and nonathletes demonstrated a significantly higher prevalence of ERPs, specifically in the inferior-lateral leads, among patients with idiopathic ventricular fibrillation (VF). However, studies to date have failed to demonstrate any prognostic implication of ERP in athletes, and until studies suggest otherwise, in isolation it should be considered a normal variant in this cohort.

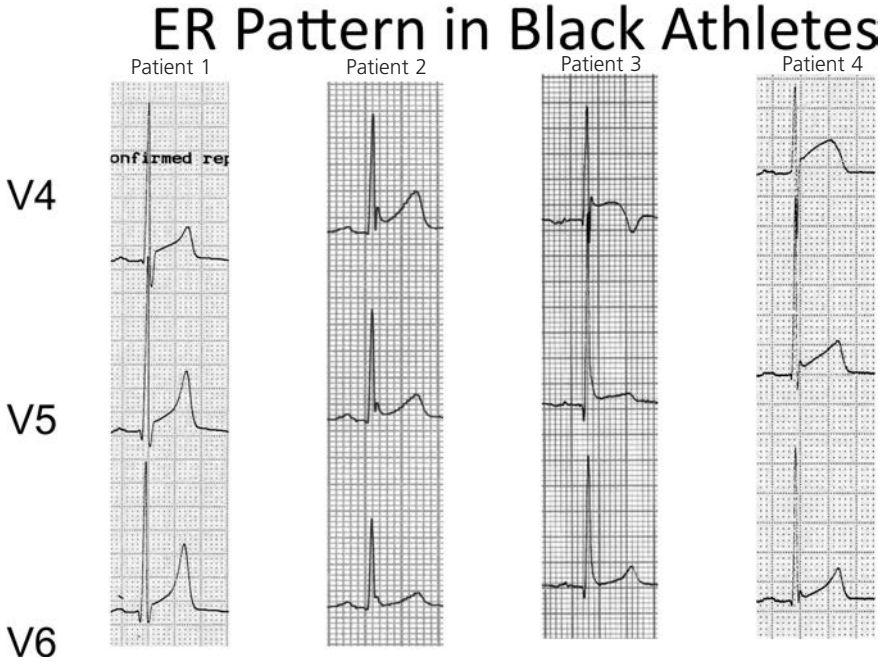

Figure 15.2.3 All four tracings would be considered normal in an otherwise asymptomatic athlete. As mentioned earlier, black athletes are among those most commonly manifesting the ERP, with it being present in more than two-thirds. This can be seen as J-point elevation in isolation, as in all four patients, or in association with notched J waves (patients 2 and 4) or slurring of the terminal QRS (patient 3).

References

1. Sheikh N, Papadakis M, Carre F, et al. Cardiac adaptation to exercise in adolescent athletes of African ethnicity: An emergent elite athletic population. *Br J Sports Med.* 2013;47(9):585–592.
2. Papadakis M, Carre F, Kervio G, et al. The prevalence, distribution, and clinical outcomes of electrocardiographic repolarization patterns in male athletes of African/Afro-Caribbean origin. *Eur Heart J.* 2011;32(18):2304–2313.
3. Noseworthy PA, Weiner R, Kim J, et al. Early repolarization pattern in competitive athletes: Clinical correlates and the effects of exercise training. *Circ Arrhythm Electrophysiol*. 2011;4:432–440.
4. Haissaguerre M, Dervel N, Sacher F, et al. Sudden cardiac arrest associated with early repolarization. *N Engl J Med.* 2008;358:2016–2023.
5. Tikkanen J, Anttonen O, Junttila J, et al. Long-term outcome associated with early repolarization on electrocardiography. *N Engl J Med.* 2009;361:2529–2537.

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CASE 15.3

Patient History

This ECG (**Figure 15.3.1**) was obtained from an asymptomatic 23-year-old rower. Physical examination was normal, with a blood pressure of 110/70 mmHg.

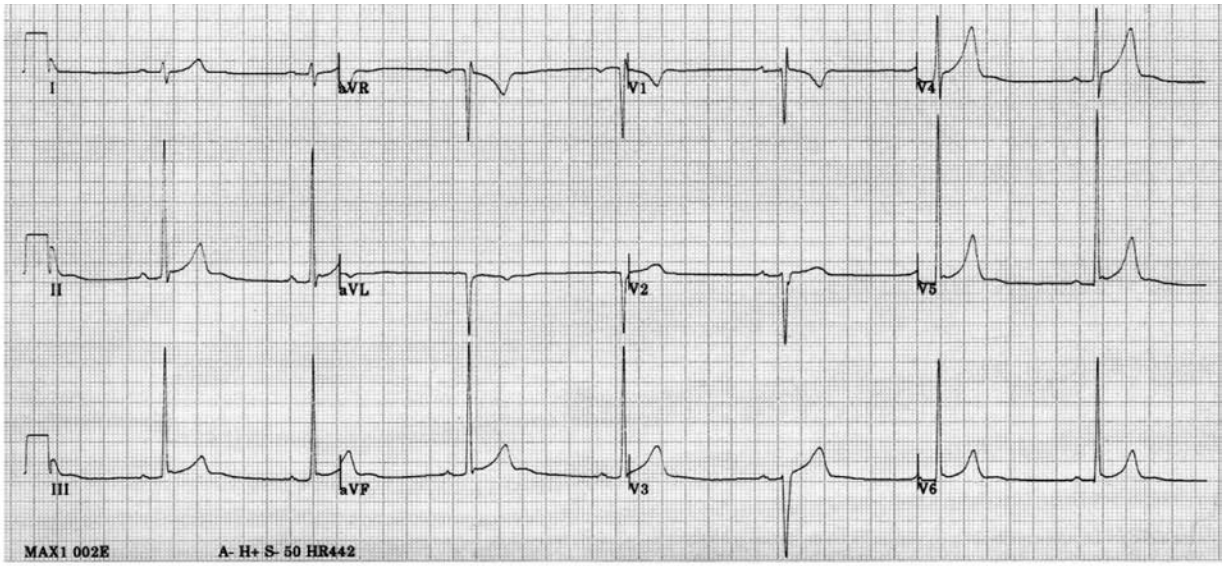

Figure 15.3.1

Question

Is any further investigation required?

Discussion

According to the latest international recommendations, the ECG changes seen in **Figure 15.3.2**, including right axis deviation, incomplete right bundle branch block (RBBB), left ventricular hypertrophy (LVH), sinus bradycardia, and early repolarization pattern (ERP), are all features of athletic adaptation, and therefore this individual does not require further investigation.

Isolated voltage criteria for LVH is seen in up to 50% of ECGs in athletes, most often in young, slender, black males, and does not necessarily correlate with the presence of increased LV wall thickness. It is defined as: $S\text{-}V_1 + R\text{-}V_{5/6} > 35$ mm. LVH is a common ECG manifestation of hypertrophic cardiomyopathy (HCM) but is very rarely found in isolation (2%) and usually associated with T-wave inversion, ST depression, and pathological Q waves. Therefore, isolated LVH is considered a normal variant in the asymptomatic athlete.

Reported in up to 60% of athletes, incomplete RBBB is a feature of physiological athletic adaptation.

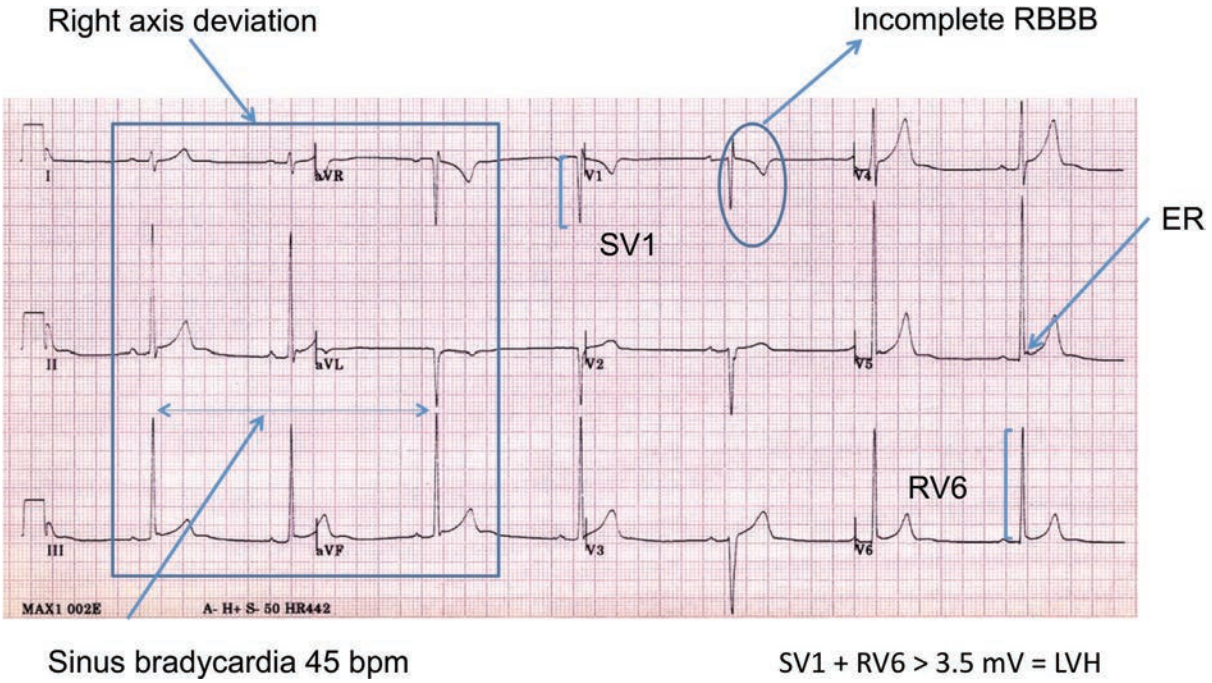

Figure 15.3.2 ECG showing right axis deviation, incomplete RBBB, LVH, early repolarization, and sinus bradycardia.

Isolated right axis deviation, defined as an axis >120 degrees, is considered a normal finding in athletes; however, when seen in association with criteria for atrial enlargement or complete RBBB, further investigation is recommended.

References

1. Pelliccia A, Maron BJ, Culasso F, et al. Clinical significance of abnormal electrocardiographic patterns in trained athletes. *Circulation*. 2000;102(3):278–284.
2. Gati S, Sheikh N, Ghani S, et al. Should axis deviation or atrial enlargement be categorised as abnormal in young athletes? The athlete's electrocardiogram: Time for re-appraisal of markers of pathology. *Eur Heart J*. 2013;34(47):3641–3648.

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CASE 15.4

Patient History

An ECG was obtained from an asymptomatic 15-year-old tennis player (**Figure 15.4.1**). Echocardiography is normal.

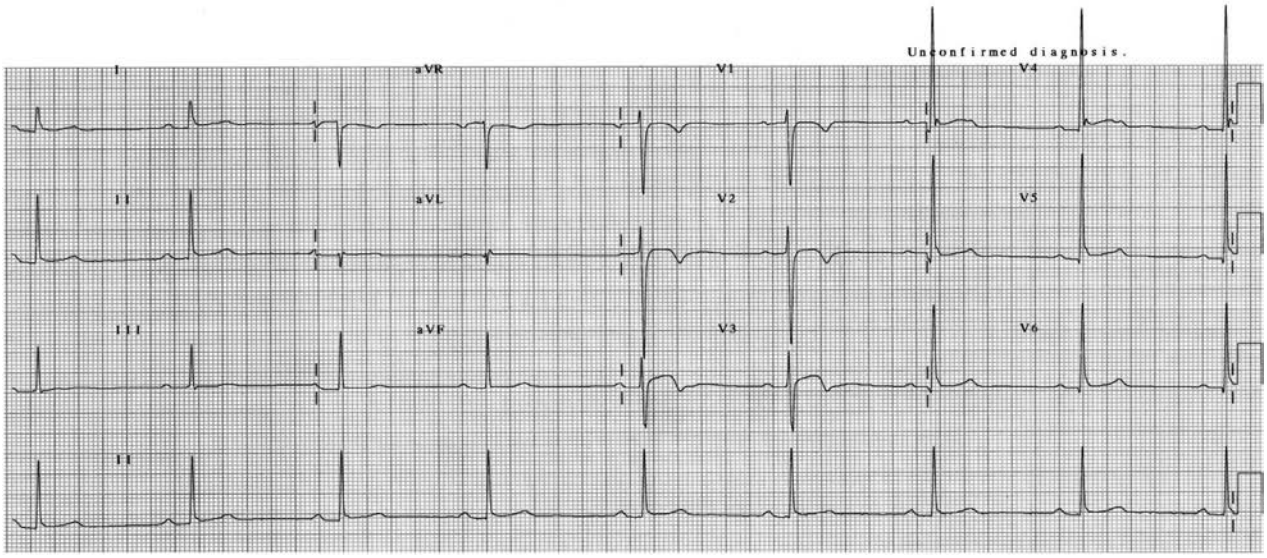

Figure 15.4.1

Question

Would you perform any further investigations at this time?

Discussion

This 15-year-old athlete has an ECG consistent with a benign juvenile ECG pattern with T-wave inversion in V_1 and V_2 and a biphasic T wave in V_3. In the under-16 age group, up to 4% of athletes will have T-wave inversion (TWI) extending beyond V_2. In isolation, in an asymptomatic athlete, this does not require further evaluation; however, a repeat ECG once over the age of 16 is recommended to ensure resolution of the changes. A persistence of TWI beyond V_2 in persons over age 16 is rare (<0.2% beyond V_2) and should be further investigated. If the ECG in **Figure 15.4.2** demonstrated deep T-wave inversion, a much rarer feature in those under age 16 (0.8%), again, further investigation with the minimum of an echocardiogram would be indicated.

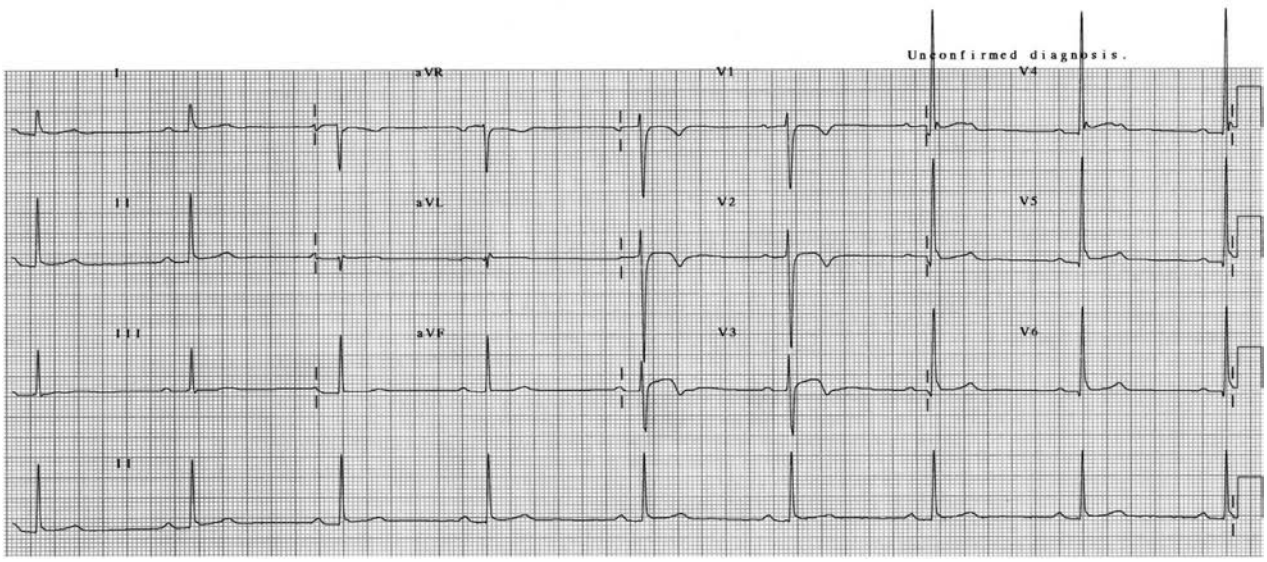

Figure 15.4.2 ECG showing J-point elevation in V_2 and V_3 with convex ST-segment elevation followed by minor T-wave inversion (TWI), consistent with a juvenile ECG pattern.

References

1. Papadakis M, Basavarajaiah S, Rawlins J, et al. Prevalence and significance of T-wave inversions in predominantly caucasian adolescent athletes. *Eur Heart J.* 2009;30(14):1728–1735.
2. Migliore F, Zorzi A, Michieli P, et al. Prevalence of cardiomyopathy in Italian asymptomatic children with electrocardiographic T-wave inversion at preparticipation screening. *Circulation*. 2012;125(3):529–538.
3. Calo L, Sperandii F, Martino A, et al. Echocardiographic findings in 2261 peri-pubertal athletes with or without inverted T waves at electrocardiogram. *Heart.* 2015;101:193–200.

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CASE 15.5

Patient History

An ECG (**Figure 15.5.1**) was obtained from a 26-year-old asymptomatic black professional soccer player.

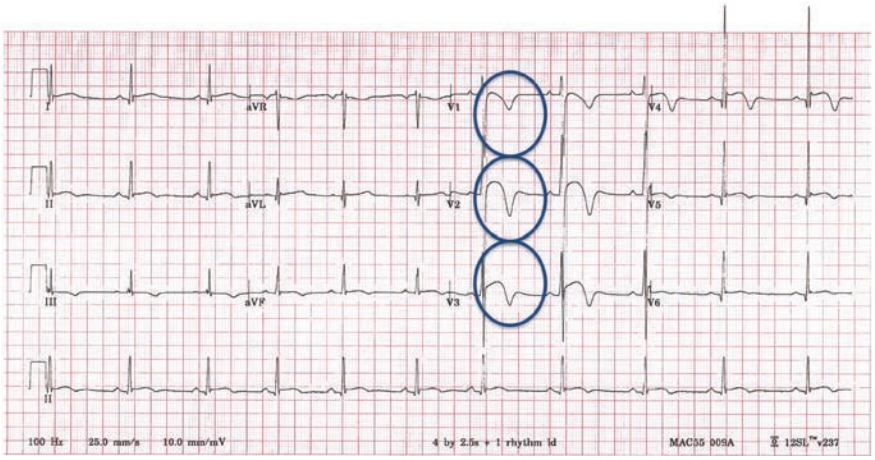


Figure 15.5.1

Question

Is the ECG normal or abnormal?

Discussion

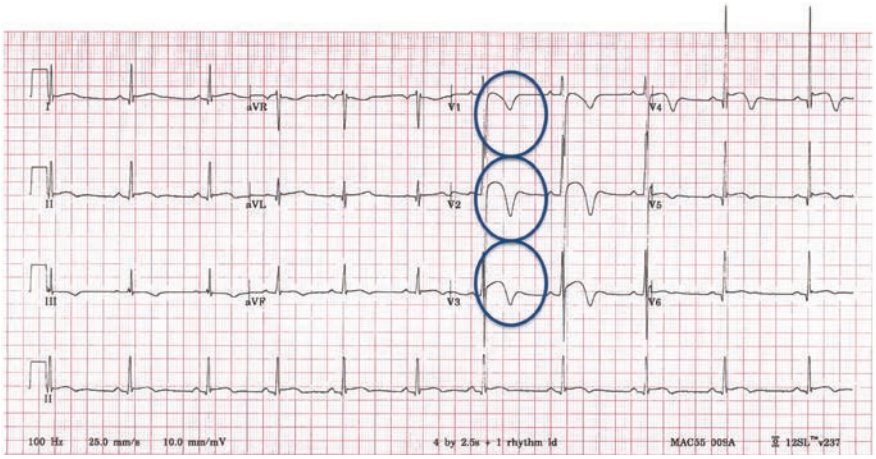


Figure 15.5.2 ECG showing J-point elevation and convex ST-segment elevation with T-wave inversion in V_1–V_4, consistent with physiological changes in a black athlete.

This ECG is consistent with an exercise-related early repolarization pattern (ERP) in an adult black athlete (**Figure 15.5.2**). This is a common manifestation in this cohort and is defined as J-point elevation with convex ST segments in V_1–V_4, followed by inverted T waves. Further examples of the ERP are shown in **Figure 15.5.3**. These findings in isolation do not warrant any further investigation.

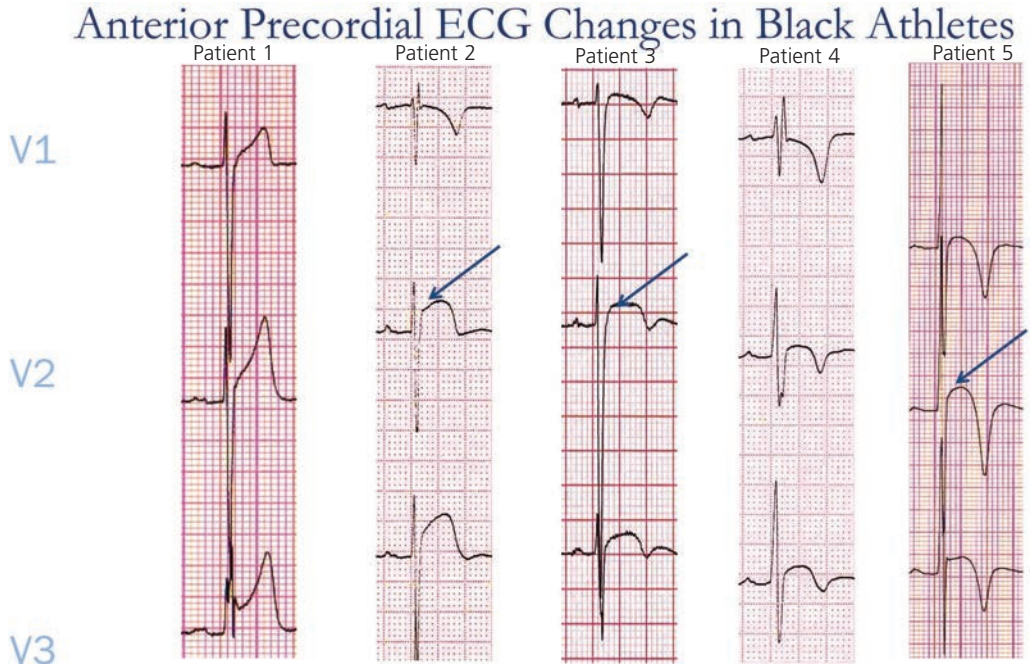


Figure 15.5.3 Patient 1: Anterior J-point elevation with concave ST-segment elevation. Patient 2: rSR′ in V_1 with anterior J-point elevation and convex ST segment elevation. Patient 3: Anterior J-point elevation with convex ST-segment elevation followed by biphasic T waves. Patient 4: rSR′ in V_1 with anterior J-point elevation and T-wave inversion. Patient 5: Anterior J-point elevation with convex ST segments and deep T-wave inversion. All of these anterior lead ECG traces represent a variant of the ERP, which is a normal finding in an otherwise asymptomatic black athlete.

The ERP usually regresses with detraining, which can be useful in cases where the differentiation of adaptive from pathological changes is unclear. An example of such regression is shown in **Figure 15.5.4**.

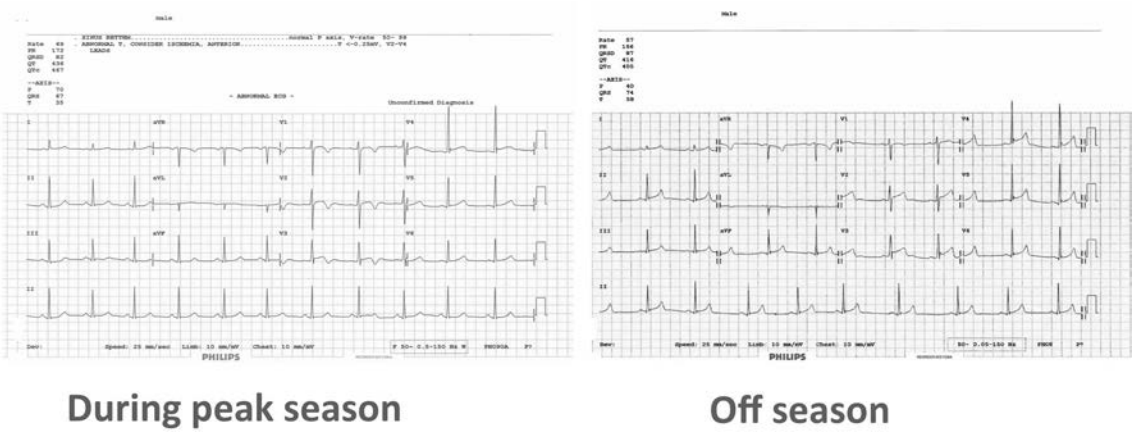


Figure 15.5.4 ECGs taken during peak season and off-season in a black athlete.

The ECG during peak season shows J-point elevation with convex ST segments followed by T-wave inversion in V_1–V_3. In the subsequent ECG off-season, i.e., detrained, although the J-point elevation remains in V_2–V_5, the ST-segment elevation is not concave and the T-wave inversion has resolved.

Other notable features seen in the ECG in **Figure 15.5.4** are a longer PR interval (as a result of high vagal tone) and a longer QTc of 467 ms at peak training. This QTc although acceptable for a male athlete (upper limit of normal ≥470 ms) would be considered prolonged in a sedentary male (cut-off 450 ms) and would require further investigation in a sedentary individual. Similarly, a QTc up to 480 ms is considered normal in female athletes, not requiring further evaluation, in contrast to nonathlete females, where the cut-off is 460 ms.

References

1. Sheikh N, Papadakis M, Carre F, et al. Cardiac adaptation to exercise in adolescent athletes of African ethnicity: An emergent elite athletic population. *Br J Sports Med.* 2013;47(9):585–592.
2. Papadakis M, Carre F, Kervio G, et al. The prevalence, distribution, and clinical outcomes of electrocardiographic repolarization patterns in male athletes of African/Afro-Caribbean origin. *Eur Heart J.* 2011;32(18):2304–2313.
3. Brosnan M, La Gerche A, Kalman J, et al. The Seattle Criteria increase the specificity of preparticipation ECG screening among elite athletes. *Br J Sports Med.* 2014;48(15):1144–1150.

CASE 15.6

Gemma Parry-Williams, MBChB, MRCP (UK)
Sanjay Sharma, BSc (Hons), MD

Patient History

An ECG (**Figure 15.6.1**) was obtained from an asymptomatic black athlete. Echocardiography was normal.

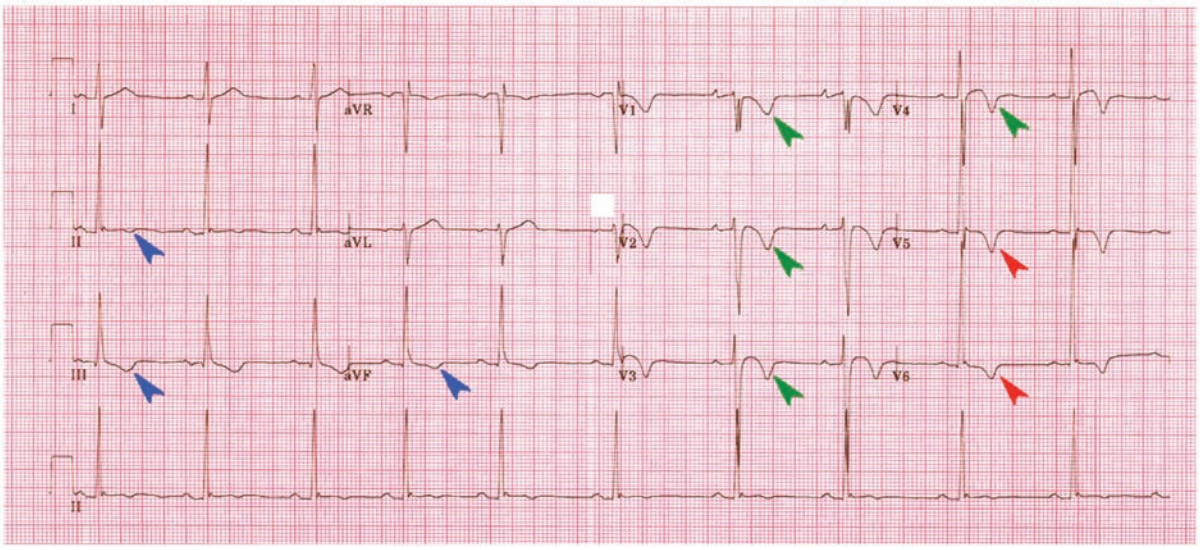


Figure 15.6.1 ECG showing widespread T-wave inversion in the anterior, lateral and inferior leads with J-point elevation and convex ST segments in V_2 and V_3.

Question

Are any further investigations required?

Discussion

T-wave inversion (TWI) is the most common ECG manifestation of cardiomyopathy, found most often in arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVC/D) and hypertrophic cardiomyopathy (HCM). Differentiating benign from pathological T-wave inversion poses a great challenge, especially among those groups of athletes, e.g., juvenile/black, in whom TWI can be very extensive yet not pathological. Recent research has allowed us to further refine our criteria for this differentiation; however, overlap does remain, and under such circumstances further investigation is warranted. A large study demonstrated significant TWI in 23% of black athletes. Eighty-nine percent of these were anterior (V_1–V_4), with inferior and lateral T-wave inversion being much less common (6% and 4.1%, respectively). Inferior TWI is commonly isolated to leads III and aVF and often benign; in contrast, TWI in the lateral leads is a strong predictor of HCM and warrants a higher index of suspicion.

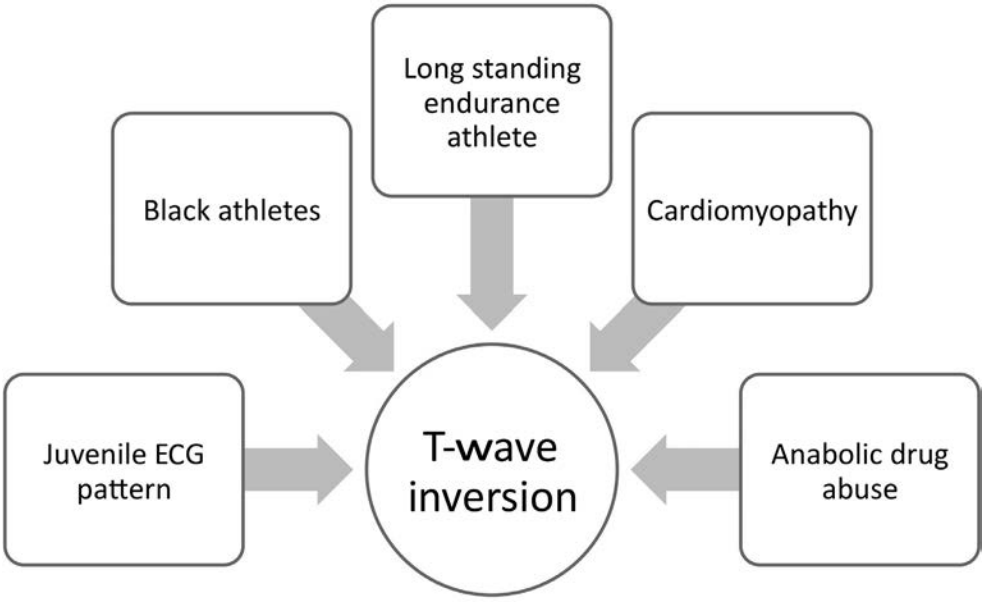


Figure 15.6.2 Image highlighting those common circumstances in which extensive T-wave inversion can manifest.

References

1. Pelliccia A, Di Paolo FM, Quattrini FM, et al. Outcomes in athletes with marked ECG repolarization abnormalities. *N Engl J Med*. 2008;358(2):152–161.
2. Papadakis M, Carre F, Kervio G, et al. The prevalence, distribution, and clinical outcomes of electrocardiographic repolarization patterns in male athletes of African/Afro-Caribbean origin. *Eur Heart J.* 2011;32(18):2304–2313.

CASE 15.7

Gemma Parry-Williams, MBChB, MRCP (UK)
Sanjay Sharma, BSc (Hons), MD

Patient History

An ECG (**Figure 15.7.1**) was obtained from a 32-year-old white female elite rower.

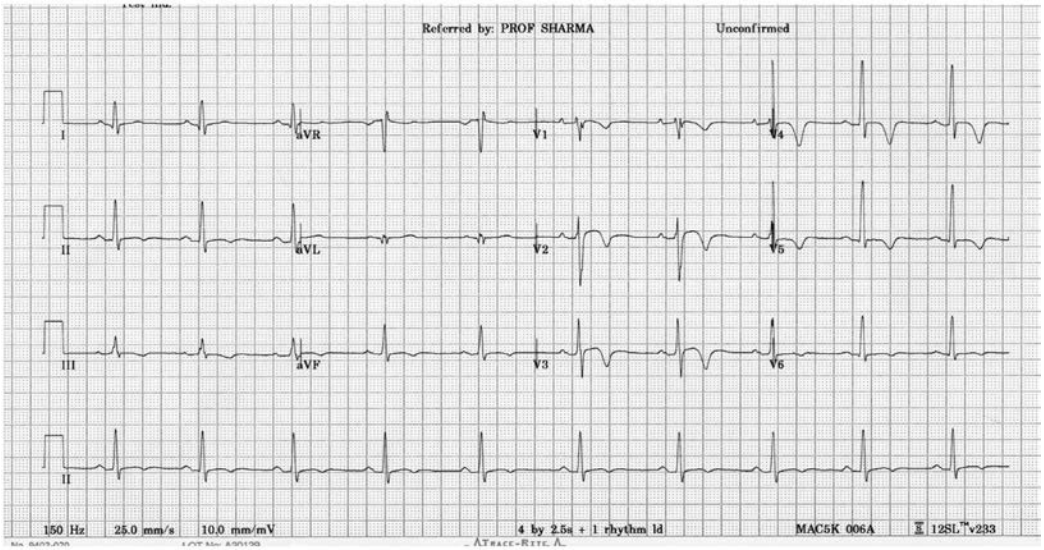


Figure 15.7.1

Question

Is the ECG normal or abnormal?

Discussion

This ECG shows extensive deep T-wave inversion (TWI) from V_1–V_6, II, III, and aVF. There is also J-point elevation with convex ST-segment elevation in V_2 and V_3. This pattern is **not** recognized within the spectrum of ECG changes seen in athletes regardless of age or ethnicity and warrants further investigation. Such investigation should include an echocardiogram, exercise tolerance test, ambulatory cardiac monitor, and cardiac MRI looking for any features of cardiomyopathies such as arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVC/D) and apical hypertrophic cardiomyopathy (HCM).

In a study of predominantly Caucasian athletes, the prevalence of anterior T-wave inversion extending beyond V_2 in individuals over age 16 was very low (0.1%). Inferior-lateral TWI in this cohort was also rare, seen in 1.5%.

Reference

1. Papadakis M, Basavarajaiah S, Rawlins J, et al. Prevalence and significance of T-wave inversions in predominantly Caucasian adolescent athletes. *Eur Heart J.* 2009;30(14):1728–1735.

Gemma Parry-Williams, MBChB, MRCP (UK)
Sanjay Sharma, BSc (Hons), MD

CASE
15.8

Patient History

Below are a selection of ECGs taken in asymptomatic professional athletes between the ages of 14 and 35.

Question

Which of these patients requires further investigation based on their ECG findings?

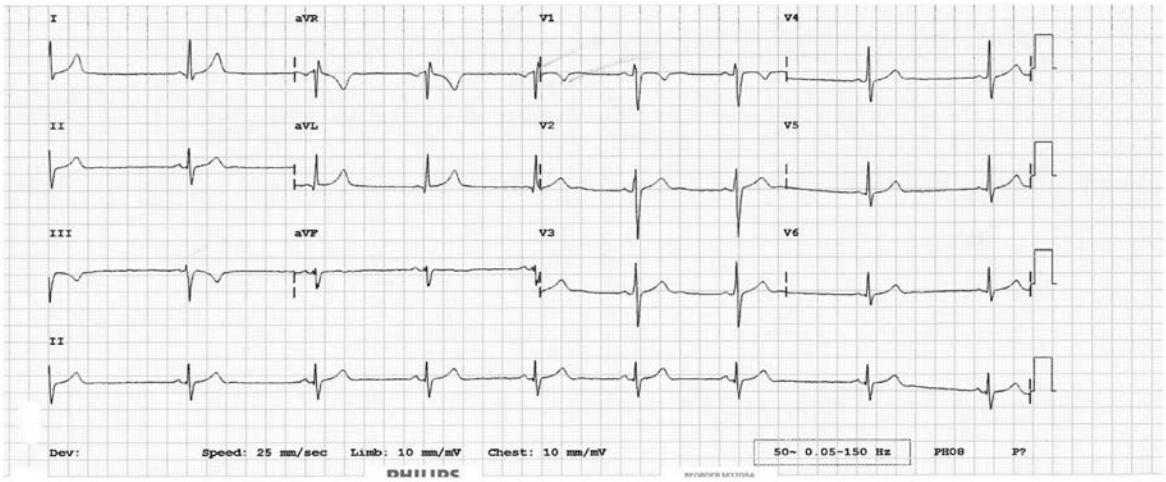


Figure 15.8.1 ECG showing left axis deviation in an athlete.

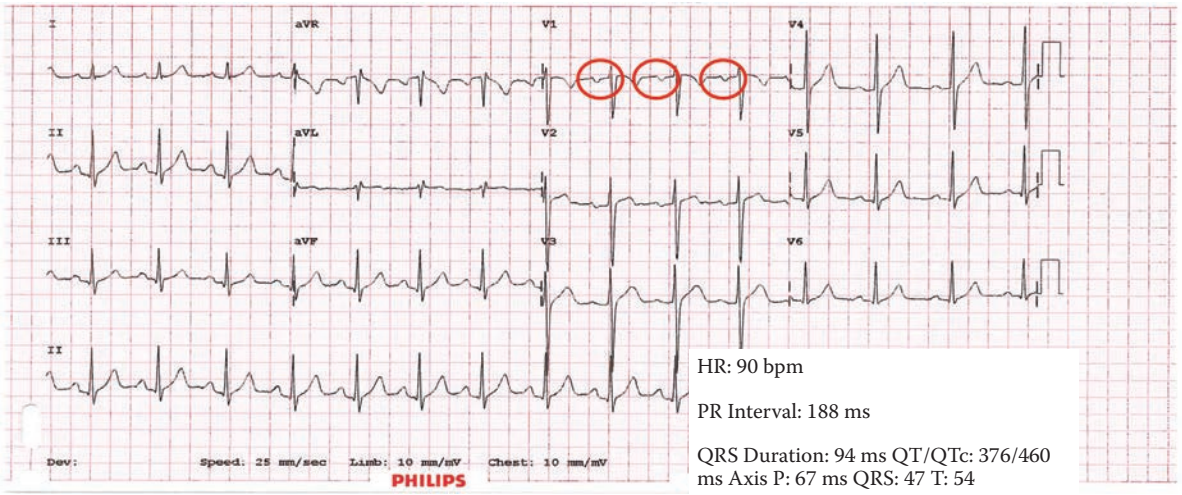


Figure 15.8.2 ECG demonstrating evidence of left atrial enlargement in lead V_1 in an athlete.

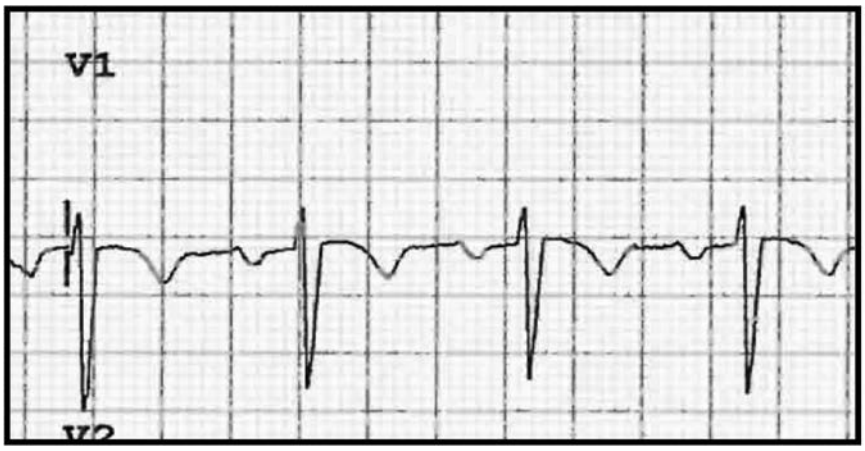


Figure 15.8.3 Focus on lead V_1 showing biphasic P wave with negative portion ≥1 mm in depth and ≥40 ms duration, consistent with criteria for left atrial enlargement.

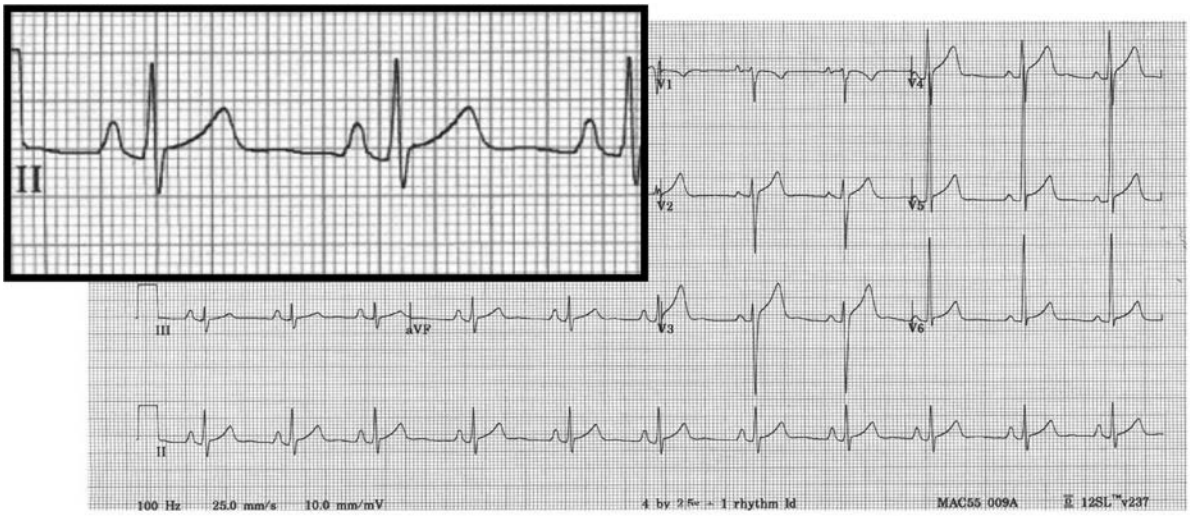
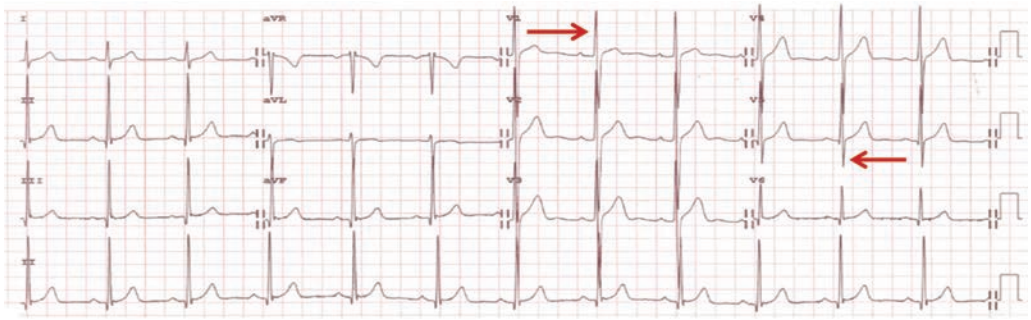


Figure 15.8.4 ECG showing P wave ≥2.5 mm in leads II and aVF consistent with criteria for right atrial enlargement in an athlete.



Sum of R in V1 + S in V5 (or V6)
≥10 mm

Figure 15.8.5 ECG showing tall R wave in V_1 and a deep S wave in V_6, and the sum of their voltages is ≥10 mm, in keeping with voltage criteria for right ventricular hypertrophy in an athlete.

Discussion

These ECGs demonstrate atrial enlargement, left axis deviation, and right ventricular hypertrophy (RVH), respectively, which in isolation are normal findings in an athlete and require no further investigation. Data to support this comes from studies of athletes with isolated atrial enlargement and axis deviation in whom echocardiography failed to demonstrate any significant pathology. Similarly, in athletes with RVH by voltage criteria whose hearts were characterized structurally using both echocardiography and cardiac magnetic resonance imaging, no pathology was identified.

References

1. Gati S, Sheikh N, Ghani S, et al. Should axis deviation or atrial enlargement be categorised as abnormal in young athletes? The athlete's electrocardiogram: time for re-appraisal of markers of pathology. *Eur Heart J.* 2013;34(47):3641–3648.
2. Zaidi A, Sheikh N, Jongman JK, et al. Clinical differentiation between physiological remodeling and arrhythmogenic right ventricular cardiomyopathy in athletes with marked electrocardiographic repolarization anomalies. *J Am Coll Cardiol.* 2015;65(25):2702–2711.

CASE 15.9

Gemma Parry-Williams, MBChB, MRCP (UK)
Sanjay Sharma, BSc (Hons), MD

Patient History

A 30-year-old female cyclist had a routine ECG (**Figure 15.9.1**).

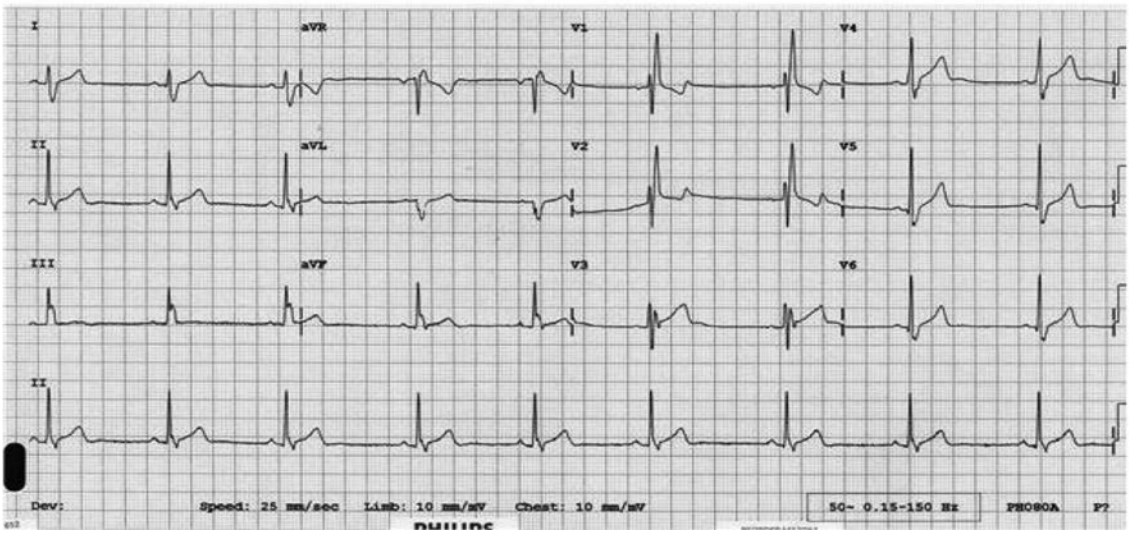


Figure 15.9.1 ECG showing complete right bundle branch block (RBBB).

Question

Are any further investigations required?

Discussion

Complete right bundle branch block (RBBB) is a relatively common finding in athletes (0.5–2.5%); however, its significance is uncertain. It is believed to be secondary to conduction delay as a consequence of increased right ventricle (RV) cavity size. One study demonstrated that athletes with a RBBB were more likely to have a bigger RV and lower RV ejection fraction. In isolation, it does not necessitate further investigation in athletes.

Reference

1. Kim JH, Noseworthy PA, McCarty D, et al. Significance of electrocardiographic right bundle branch block in trained athletes. *Am J Cardiol*. 2011;107(7):1083–1089.

Gemma Parry-Williams, MBChB, MRCP (UK)
Sanjay Sharma, BSc (Hons), MD

CASE 15.10

Patient History

An asymptomatic 21-year-old professional football player underwent pre-participation screening. **Figure 15.10.1** is his ECG.

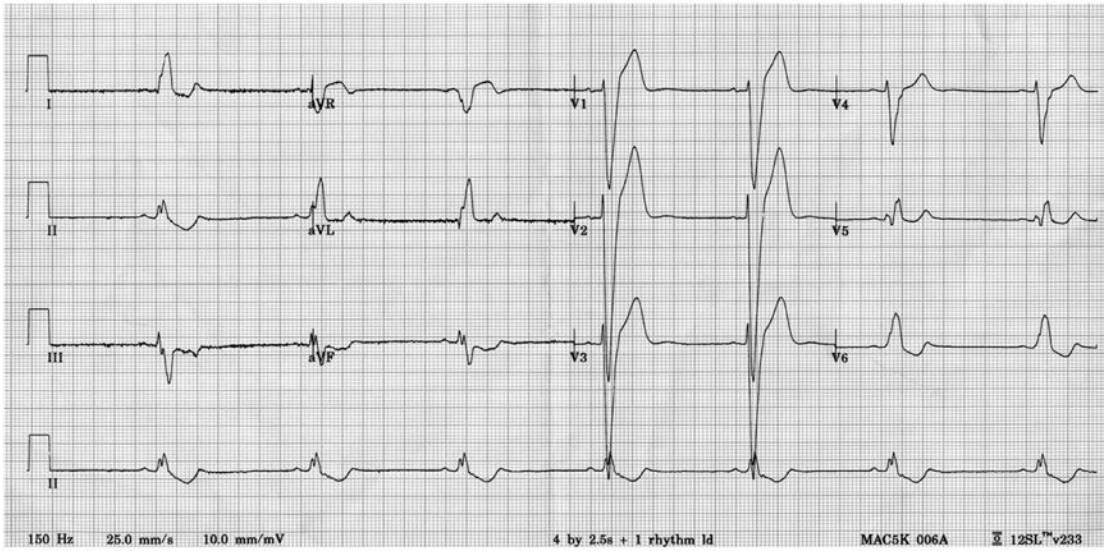


Figure 15.10.1 ECG showing complete left bundle branch block (LBBB).

Question

What investigations would you perform?

Discussion

Left bundle branch block (LBBB) is rare in athletes (<0.1%) but is a well-recognized ECG manifestation of cardiac disease including dilated cardiomyopathy, hypertrophic cardiomyopathy, left ventricular noncompaction (LVNC), sarcoid, and myocarditis and therefore always necessitates further investigation, including echocardiography, cardiac magnetic resonance imaging, and ischemia testing.

Reference

1. Sharma S, Drezner JA, Baggish A, et al. International recommendations for electrocardiographic interpretation in athletes. *J Am Coll Cardiol.* 2017;69(8):1057–1075.

CASE 15.11

Gemma Parry-Williams, MBChB, MRCP (UK)
Sanjay Sharma, BSc (Hons), MD

Patient History

This is the ECG (**Figure 15.11.1**) of an asymptomatic 24-year-old black basketball player. Echocardiography was normal.

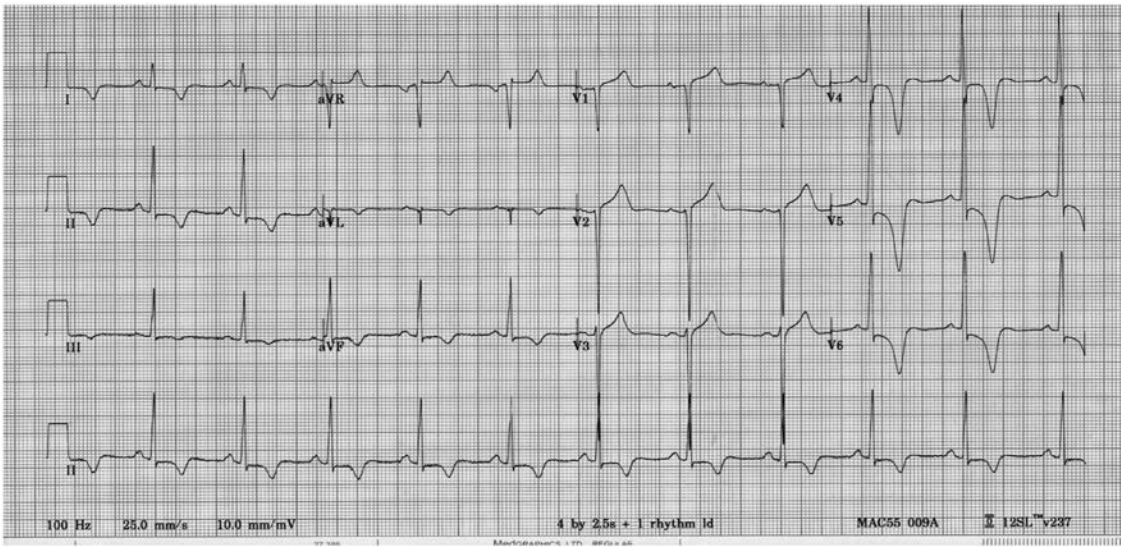


Figure 15.11.1 ECG showing ST depression followed by deep T-wave inversion in the inferior-lateral leads.

Question

Are any further investigations required at this time?

Discussion

This ECG is grossly abnormal and indisputably requires further investigation. It shows ST depression, which is **not** a feature of athletic adaptation, and deep T-wave inversion in the inferior-lateral leads. As mentioned earlier, T-wave inversion in the lateral leads is a strong predictor of hypertrophic cardiomyopathy and the index of suspicion here is even greater, given the presence of left ventricular hypertrophy by voltage criteria. This athlete requires thorough investigation, including echocardiography, cardiac magnetic resonance imaging, exercise testing, and ambulatory cardiac monitoring, and if negative, should be followed up very closely.

Reference

1. Sharma S, Drezner JA, Baggish A, et al. International recommendations for electrocardiographic interpretation in athletes. *J Am Coll Cardiol.* 2017;69(8):1057–1075.

Gemma Parry-Williams, MBChB, MRCP (UK)
Sanjay Sharma, BSc (Hons), MD

CASE 15.12

Patient History

An ECG (**Figure 15.12.1**) was obtained from an asymptomatic 21-year-old white swimmer.

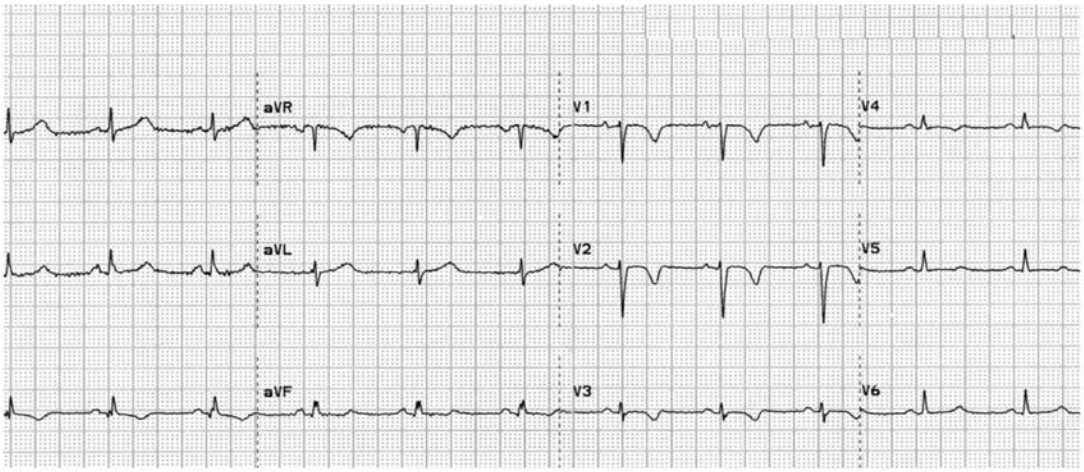


Figure 15.12.1 ECG showing isolated T-wave inversion (TWI) in leads V_1–V_3.

Question

Is this ECG normal?

Discussion

This is an abnormal ECG showing T-wave inversion (TWI) in V_1–V_3 without any J-point or ST-segment elevation. TWI extending beyond V_2 in a white athlete over the age of 16 has a prevalence of only 0.1% and is considered an abnormal finding warranting further investigation. Arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVC/D) is a condition that commonly presents with anterior T-wave inversion. Apical hypertrophic cardiomyopathy (HCM) presents in this way much more rarely. Investigation should therefore be targeted at identifying the recognized features of these cardiomyopathies. Investigations include echocardiography, cardiac magnetic resonance imaging, exercise testing, 24-hour ambulatory cardiac monitoring, and signal-averaged ECG. Other features that would raise the index of suspicion for ARVC/D include epsilon waves and ectopy originating in the right ventricle. The epsilon wave is a small positive deflection ('blip') buried within the end of the QRS complex. Epsilon waves are highly specific for ARVC/D and a major diagnostic criterion.

Reference

1. Papadakis M, Carre F, Kervio G, et al. The prevalence, distribution, and clinical outcomes of electrocardiographic repolarization patterns in male athletes of African/Afro-Caribbean origin. *Eur Heart J.* 2011;32(18):2304–2313.

SECTION 16

Syncope and ECG Troubleshooting

CASE
16.1

Haran Burri, MD

Patient History

The artifacts visible on the ECG (**Figure 16.1.1**) are due to a single limb electrode that has been disconnected.

Questions

1. Which electrode is it?
2. How do you explain the artifacts on the precordial leads?

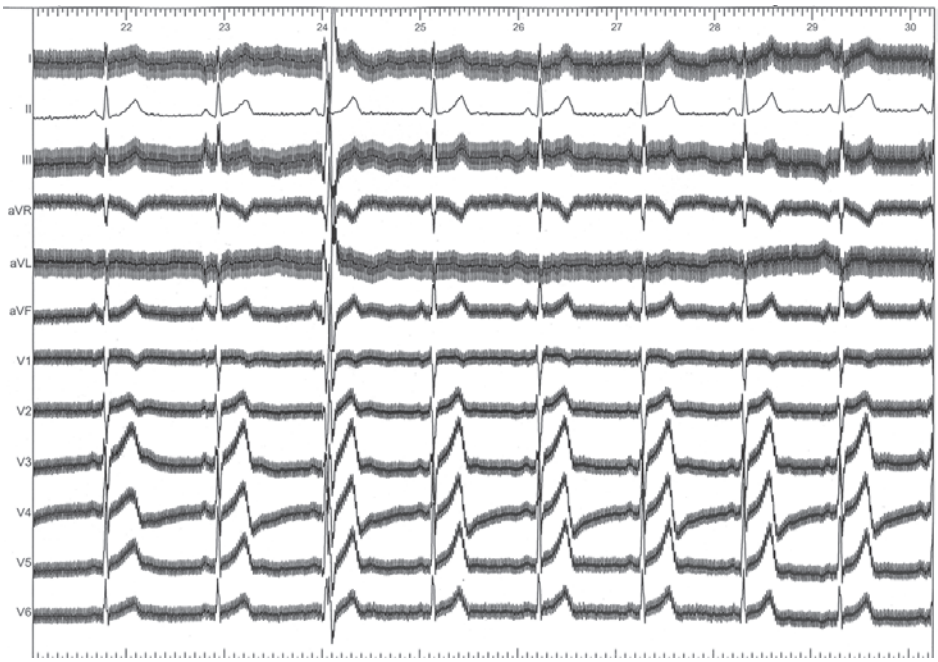


Figure 16.1.1 ECG recording showing artifacts.

Discussion

The only lead without artifacts is lead II, which is independent of the left arm electrode. This electrode was replaced, resulting in disappearance of the artifacts (see **Figure 16.1.2**). The

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reason for the precordial leads displaying artifacts is due to them all being connected to the Wilson central terminal (WCT). The WCT serves as the negative pole for the unipolar leads (aVR, aVL, aVF, and V_1–V_6) and is constructed by connecting the three limb electrodes to a resistance (i.e., it also involves the left arm electrode).

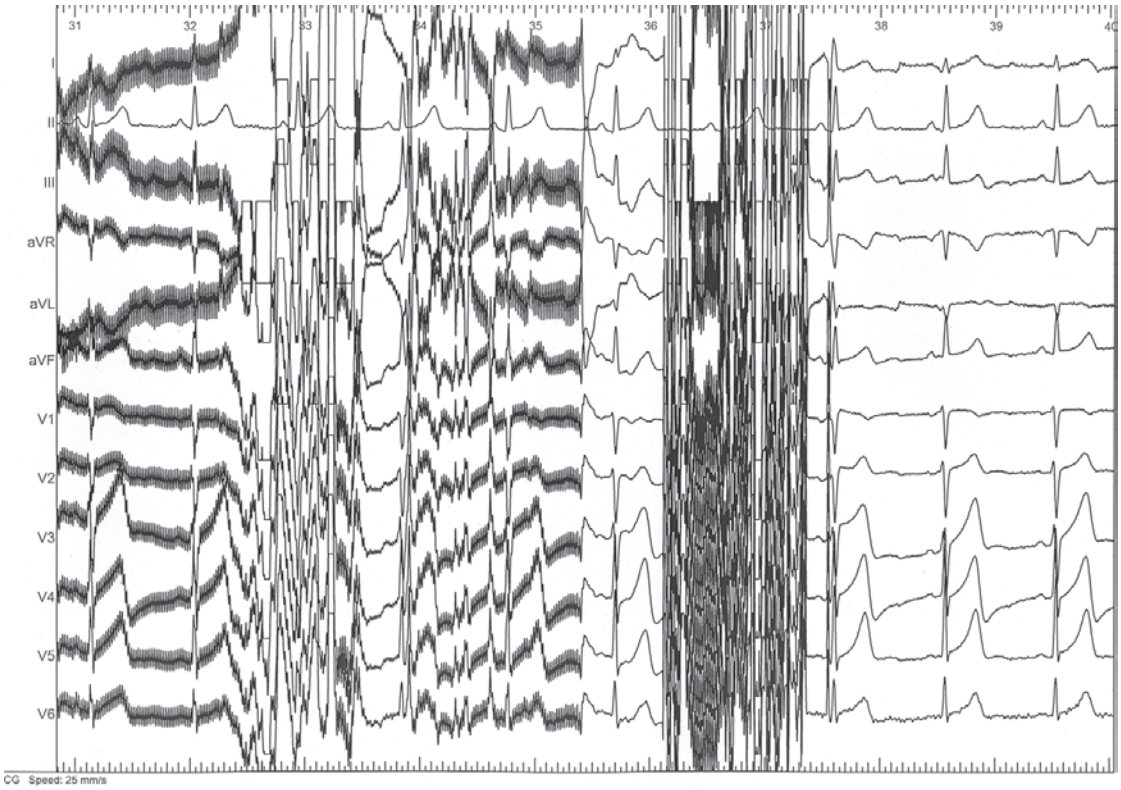

Figure 16.1.2 ECG recorded during and immediately after replacing the electrode on the left arm.

CASE
16.2

Haran Burri, MD

Patient History

A 27-year-old male patient with no previous history was admitted to the surgical unit due to abdominal pain. The ECG recorded is shown (**Figure 16.2.1**). Electrode misplacement was suspected, and the recording was repeated while paying attention to the correct placement of the electrodes; however, it came back with the same findings. The chest x-ray was normal.

Question

How do you explain the findings?

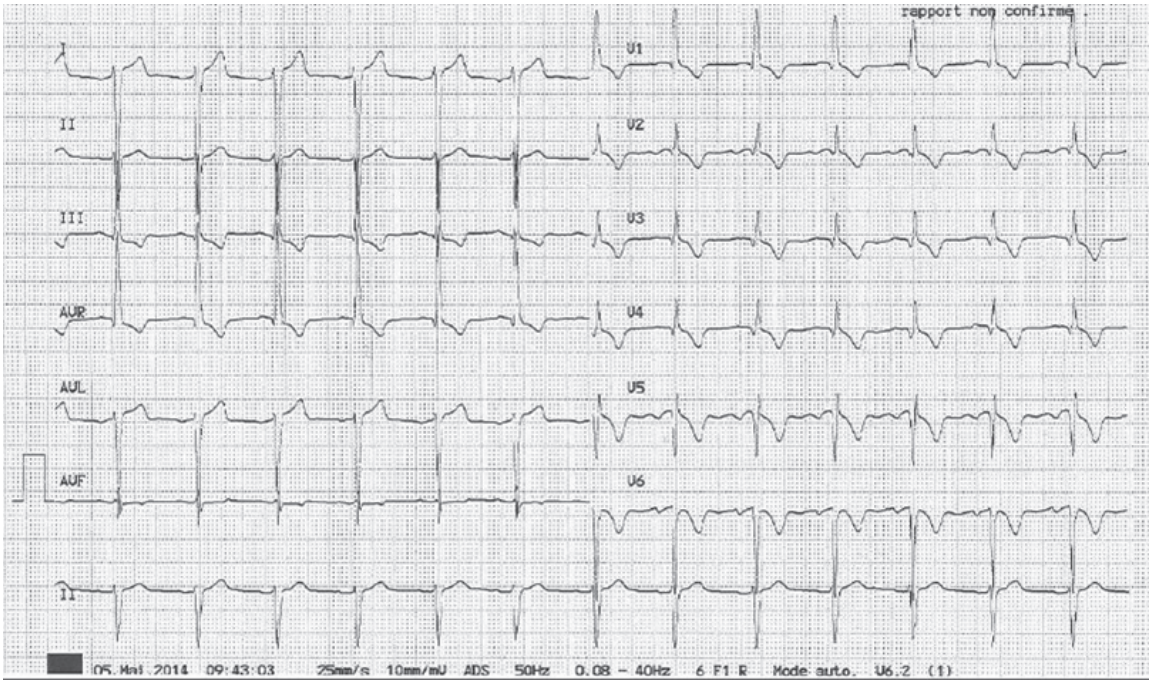

Figure 16.2.1 ECG recorded on the ward, showing unusual QRS morphology.

Discussion

A common error is inversion of the arm electrodes, which would have yielded a tracing similar to what was observed in lead I of **Figure 16.2.1**, but this does not explain the changes in the precordial leads. The abnormal QRS morphology was due to inversion of the connectors of the electrode cables (**Figure 16.2.2**). The connections were corrected, and the new ECG recording was normal (**Figure 16.2.3**).

Figure 16.2.2 Inversion of the connections (note the colors which do not match up).

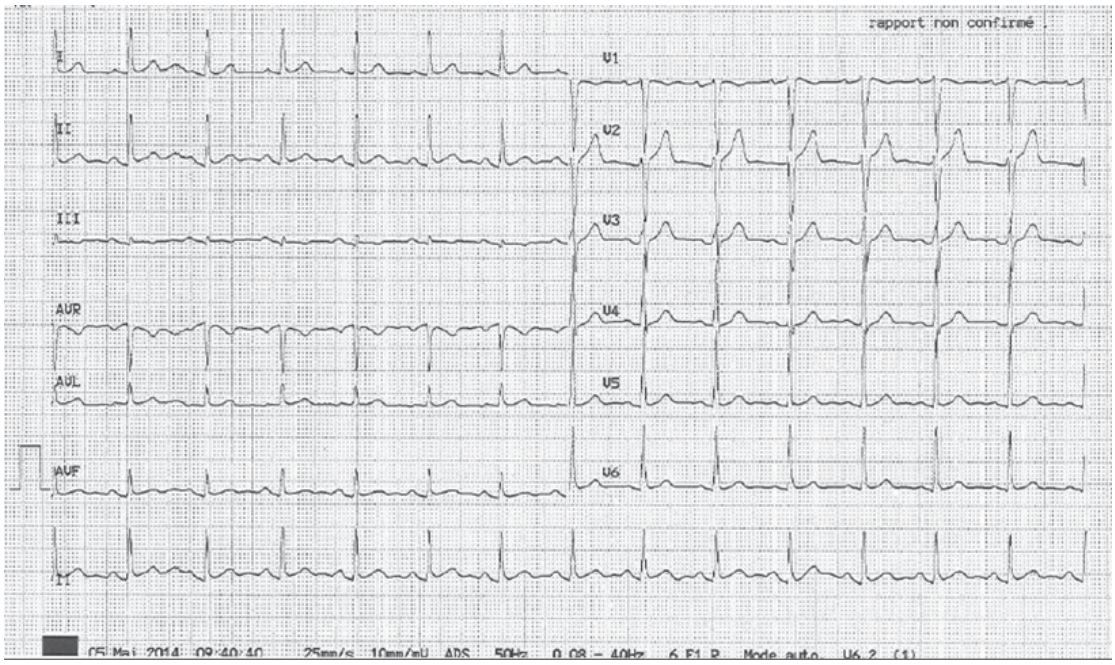

Figure 16.2.3 ECG recorded after correction of the inverted connections.

CASE
16.3

Tamer S. Fahmy, MD, PhD

Patient History

A 65-year-old female patient admitted to the critical care department with acute respiratory failure. She was put on mechanical ventilatory support and given conventional therapy for management of her chest condition. **Figure 16.3.1** shows the rhythm found in the routine ECG that was performed.

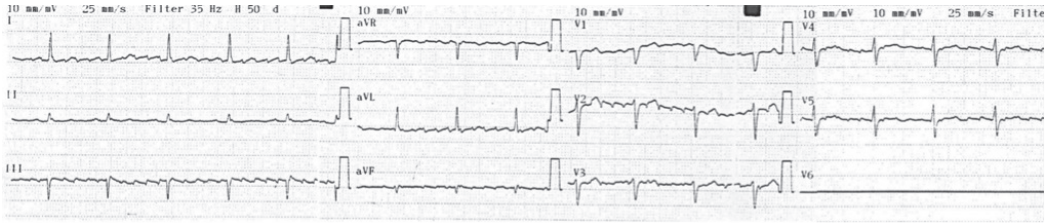

Figure 16.3.1 Routine 12-lead ECG. As seen in most leads, despite the regularity of the rhythm, the sawtooth appearance of the flutter is evident in almost all leads. Evidently, it is more apparent on the chest leads, due to their proximity with the nebulizer vibrations. (Lead V_6 is disconnected).

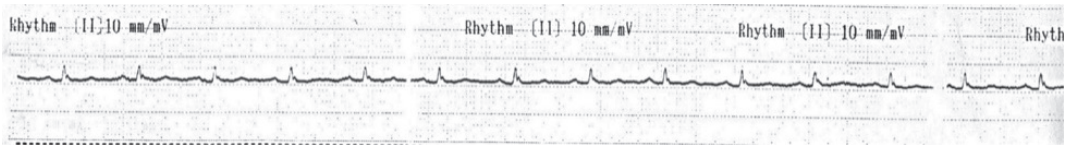

Figure 16.3.2 Rhythm strip of the same patient showing sinus rhythm with well-formed P wave evident.

Questions

1. What is the ECG rhythm?
2. What is the most common rhythms in mechanically ventilated patients with chest problems?

Answers

1. This is sinus rhythm, with pseudo-flutter waves appearing in all leads except lead II.
2. Most common arrhythmias include sinus tachycardia, multifocal atrial tachycardia, atrial fibrillation, and flutter.

Discussion and Interpretation

This is an uncommon deceit that happens when the nebulizer of the ventilator is on and touching the patient. Whether the nebulizer is pneumatic or ultrasonic, it produces vibrations when in contact with the patient; thus, yielding this misinterpretation on the monitor and the ECG. As shown on the surface ECG, these vibrations are seen in almost all leads, except for lead II. Most patients that are on ventilatory support usually suffer from different arrhythmias, especially supraventricular arrhythmias starting with sinus tachycardia, multifocal atrial tachycardia, atrial fibrillation and flutter.

CASE 16.4

Scott Sakaguchi, MD
Baris Akdemir, MD

Patient History

A 35-year-old male was referred by his primary care physician for possible Wolff–Parkinson–White (WPW) syndrome. The patient had no symptoms of palpitations, chest pain, or syncope.

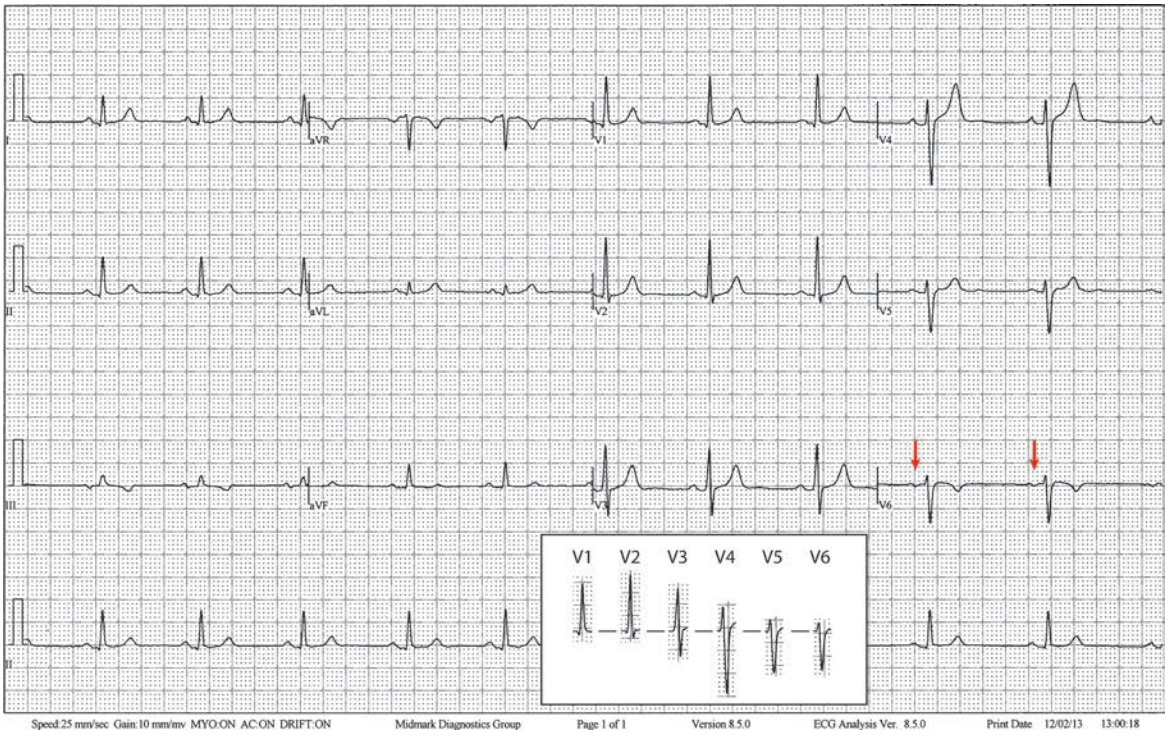

Figure 16.4.1 Presenting ECG. A tall R wave is noted in lead V_1.

Question

What is the differential diagnosis of a tall R wave in lead V_1?

Discussion

The most obvious abnormality of the ECG is the presence of a tall R wave in lead V_1. The differential diagnosis of a tall R wave in lead V_1 includes WPW syndrome, right ventricular hypertrophy, posterior infarction, right bundle branch block (RBBB) and electrode misplacement. Other than cardiac electrophysiologists, most physicians would probably not consider WPW as a first option in their differential diagnosis.

In WPW, a tall R wave in lead V_1 may be seen in the presence of an accessory pathway inserting into the left ventricle. Delta waves may be visible in the ECG, including lead V_1. An inverted or "negative" delta wave in lead I or aVL indicates early activation of the left ventricular free wall.

Similarly, negative delta waves and Q waves in the inferior leads suggest insertion on an accessory pathway into the posterior or posteroseptal aspect of the left ventricle. This pattern may be misread as an inferior infarction. Delta waves are not present in **Figure 16.4.1**, making WPW a questionable possibility.

Right ventricular hypertrophy would be a stronger consideration if secondary findings such as T-wave abnormalities in lead V_1, right axis deviation or right atrial enlargement were present. Likewise, while posterior infarction is occasionally limited to the posterior wall only, it is usually associated with an inferior infarction. A classic RBBB with an rSR′ pattern would rarely be missed by an ECG reader; a QR pattern in leads V_1 and V_2 would be seen in the presence of a concomitant septal infarction.

Abnormalities in precordial QRS progression may be seen in cases of dextrocardia. Case 16.9 in Volume 1 of this series includes the ECG of a patient with situs inversus, in which the location of all viscera (including the heart) is reflected across the midline. Because the left ventricle is in the right chest, the total QRS amplitude becomes smaller towards lead V_6 as the precordial leads are progressively further away from the rightward-directed ventricle. Precordial R waves are small or absent. The term dextrocardia also refers to cases in which the atria are in their normal positions but the heart is displaced towards the right chest, due to a mass effect in the chest (dextroposition) or a developmental abnormality in which the ventricles are rotated toward the right (dextroversion), such as certain forms of transposition of the great arteries. In the latter case, a large R may be seen in lead V_1 reflecting hypertrophy of the ventricle under the electrode. In abnormalities such as transposition, associated anomalies such as ventricular septal defect or pulmonic stenosis may underlie other ECG abnormalities such as right, left or combined ventricular hypertrophy, and left or right axis deviation.

This ECG shows a recording error in which the precordial electrodes were placed in reversed order so that the V_6 electrode is in the V_1 position, V_5 is at V_2, V_4 is at V_3, and so on until the V_1 electrode is in the V_6 position. This error is first suggested by observing that beyond lead V_1 the precordial R/S ratio from V_1 to V_6 is reversed (**Figure 16.4.1**, inset). The R/S ratio is >1 in V_1, decreases across the precordium and is <1 in V_6. The final confirmation of this interpretation is found in the P-wave morphology of lead V_6. Lead V_6 shows a biphasic P wave (red arrows) as would be expected in a correctly positioned lead V_1. Lead V_1 (and the other precordial leads) show a positive P wave.

Appendix

Contributor	Case Number
Abdel Aziz, Ahmed	12.4
Akdemir, Baris	16.4
Amsterdam, Ezra A.	5.1
Andrade, Jason	3.3, 7.1
Baranchuk, Adrian	2.6, 13G.1
Bayés de Luna, Antoni	10.1
Belhassen, Bernard	4.1, 8D.1
Benditt, David G.	2.2, 2.3, 2.4
Blendea, Dan	7.2
Botros, Maichel Sobhy Naguib	5.2
Bui, Jonathan	5.1
Burri, Haran	16.1, 16.2
Buzea, Catalin A.	12.1
Callans, David J.	3.1
Chacko, Sanoj	2.6
Cheng, Alan	2.5, 3.2, 4.2
China, Paolo	9A.4, 12.7, 13A.4
Crosson, Jane E.	2.5, 3.2, 4.2
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Dubuc, Marc	3.3, 7.1
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