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medycyna praktyczna

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DIAGNOSTIC PROBLEMS

CLINICAL INTERPRETATIONS

PREFACE

Electrocardiography has already existed for over 100 years. Its centenary was celebrated even twice, because according to the English the first human electrocardiogram (ECG) was recorded by Augustus Waller in 1887, while according to the Dutch—by Willem Einthoven in 1903. Since then, some great scientists and experts in this field (for example, Thomas Lewis, 1925, and Luis Katz, 1956) repeatedly declared that an electrocardiography had already been fully explored and there was no need for further investigations. Meanwhile, the alterations and modifications of the ECG criteria and their interpretations, as well as the values of different quantitative parameters, occur almost incessantly. A good example of such modifications is the upper limit of the normal QRS complex duration: from 0.09 s (in the first half of the 20th century), through 0.1 s (1965), to 0.11 s (since 2009). Therefore, in the descriptions and interpretations of the ECGs in this set, I use the most current 2009 ECG guidelines (authorized by the American Heart Association, American College of Cardiology, and Heart Rhythm Society) as a reference, following them strictly with regards to numerical criteria and less so with regards to terminology and recommendations.

To users of this ECG set, I pass on the following most important (but frequently forgotten) advice: the interpretation of an ECG tracing must take into account the sex, age, and clinical data of the patient. The best example for this recommendation is the interpretation of horizontally depressed (4 mm) ST segments in several precordial leads (V_4–V_6) without the intraventricular conduction defects. Such ST-segment depression in a 60-year-old man should be interpreted as acute myocardial ischemia, while the same ECG recorded in a young woman just before an oral examination should be treated as an increased sympathetic tone. I hope that the examples of ECG interpretations presented in this set will encourage all users to aim at an accurate analysis of ECGs (always including the clinical data!) in their own medical practice.

Prof. Barbara Dąbrowska, MD, PhD

DIAGNOSTIC PROBLEM

A seemingly wandering atrial pacemaker

This electrocardiogram (FIGURE 1) shows a rather rarely seen wandering of a pacemaker from the sinus node to the lower parts of an atrium, with no change in heart rate (86 bpm). Between these events, a couple of beats with P waves of intermediate morphology are seen. This feature is analogous to the concertina effect, which is usually observed in QRS complexes in patients with preexcitation syndrome or isorhythmic atrioventricular dissociation. The recording seemingly allows a diagnosis of a wandering atrial pacemaker because at least 3 different morphologies of the P waves are seen. However, the wandering is only apparent because the variability of these morphologies is not due to the origin of subsequent electrical events from different active pacemakers, but rather due to the superimposition of 2 rhythms: from the sinus node and from a single focus located in the lower part of the atrium, as described above. Therefore, the heart rhythm presented in FIGURE 1 would be better described as isorhythmic dissociation of 2 supraventricular rhythms: a sinus rhythm and a low atrial rhythm. Such a parasystolic atrial rhythm is usually associated with increased parasympathetic tone, but in this case, a spontaneous increase in the activity of an ectopic pacemaker or an atypical increase in sympathetic tone may be suspected, considering a quite rapid heart rate. Of note, in the 2 sets of recommendations by the American College of Cardiology, American Heart Association, and Heart Rhythm Society (2006 and 2007), there was a minor change in terminology, and so we currently diagnose wandering of an atrial pacemaker instead of wandering of a supraventricular pacemaker.

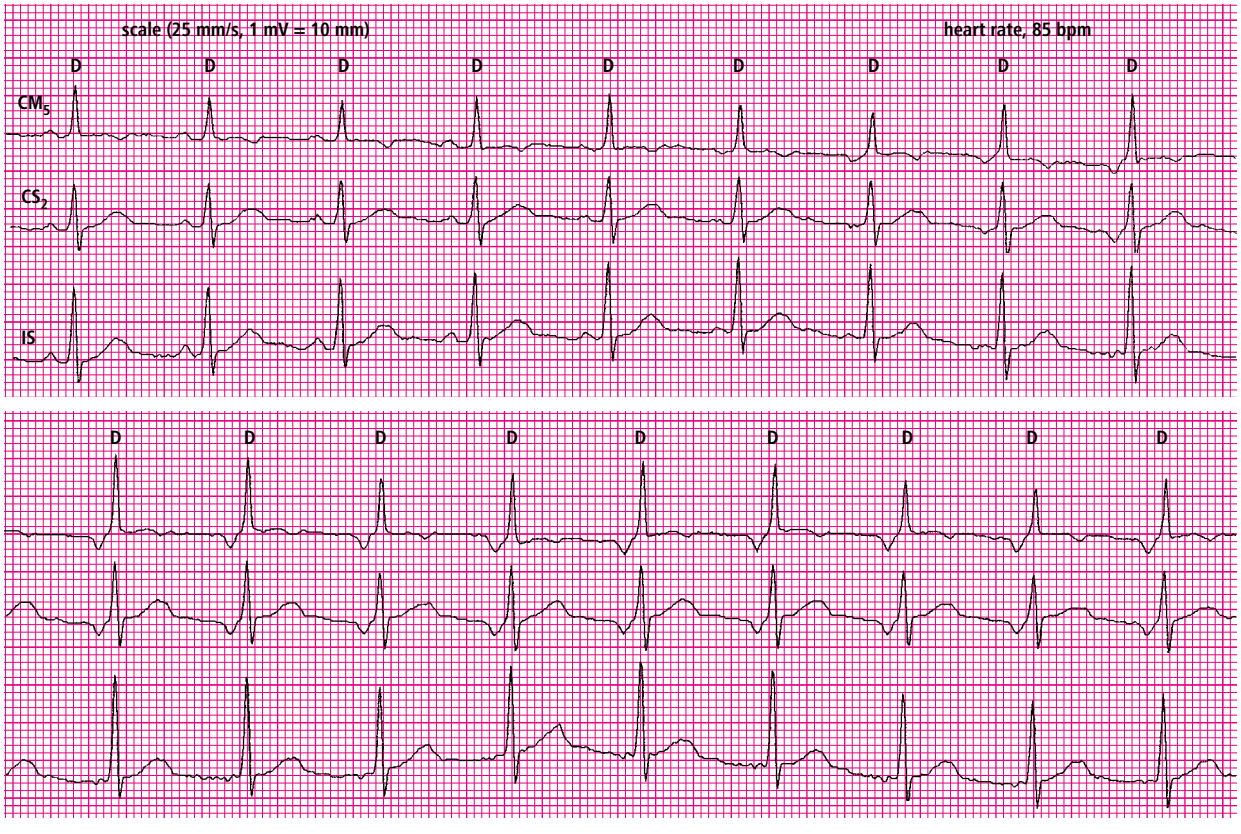

FIGURE 1

DIAGNOSTIC PROBLEM

Capture of sinus rhythm by an artificial cardiac pacemaker

This routine electrocardiogram (ECG; FIGURE 1) was recorded in a 60-year-old woman with an artificial cardiac pacemaker, who was scheduled for a gynecologic surgery. Sinus rhythm at a rate of 60 bpm is smoothly captured by the artificial atrial pacemaker. Particularly interesting are low-voltage QRS complexes in the limb and left precordial leads, with poor R-wave progression in leads V_2 to V_4. These changes of QRS complexes are accompanied by symmetric but not very deep negative T waves in leads V_2 to V_5. How to read such an ECG?

The only conclusion that can be drawn with some certainty is a high probability of sick sinus syndrome, which in the present case resulted in a decision to implant the artificial pacemaker. A low amplitude of QRS complexes may be caused by heart diseases, for example, constrictive pericarditis or restrictive cardiomyopathy (although this ECG does not show the commonly observed features of more prominent electrical activity of either the left or the right atrium). Other possible causes include endocrine or metabolic disorders (obesity, hypothyroidism). However, sometimes the low amplitude of the QRS complex cannot be explained, and the results of echocardiography are completely normal. In the present case, the coexistence of negative T waves raises the suspicion of heart disease and warrants further diagnostic workup (including targeted medical history) before deciding on a surgery. It should be noted that symmetrical negative T waves are a nonspecific feature, which is found in numerous diseases but also in patients without clinical symptoms of heart disease.

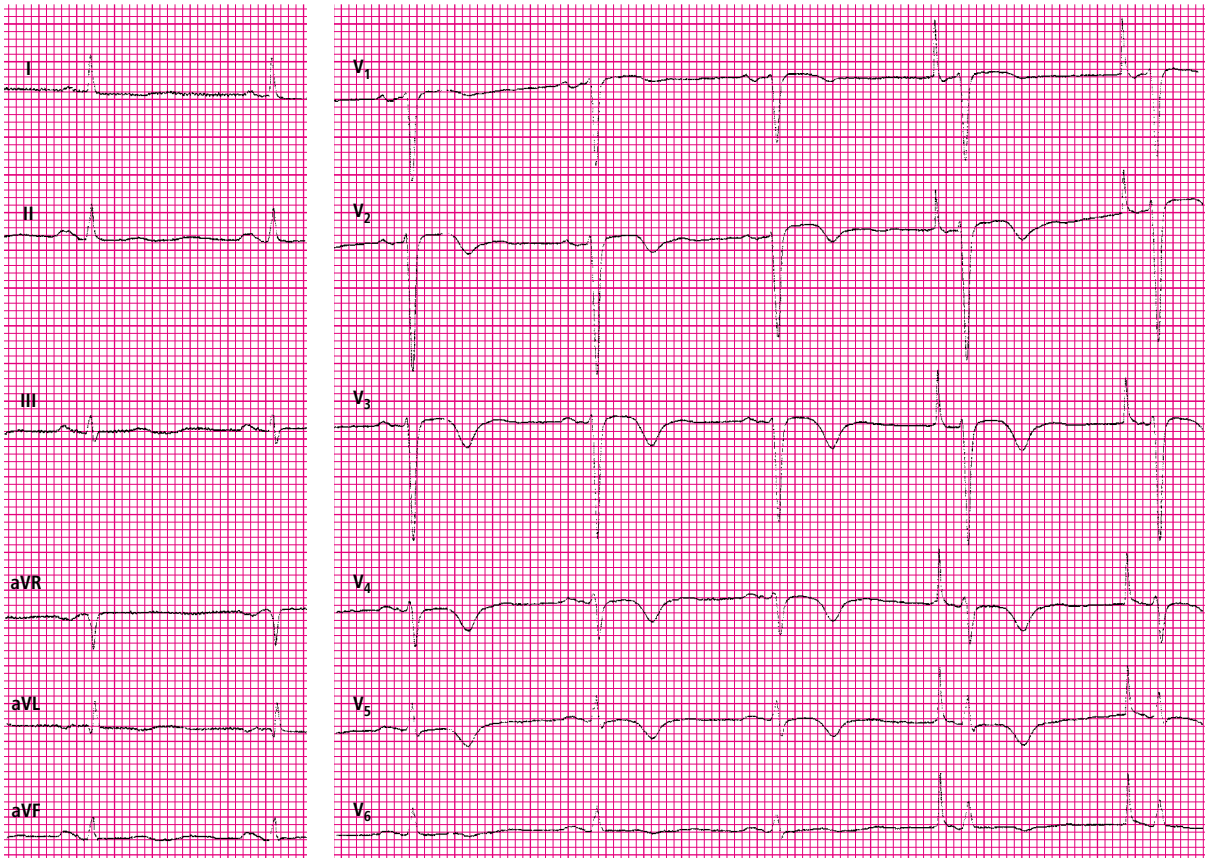

FIGURE 1

Also another symptom suggesting the presence of heart disease—the lack of R-wave progression—is a nonspecific feature. It can be seen in patients with left ventricular overload or cor pulmonale (in such a case, it is accompanied by other characteristic ECG features), in those with anterior wall myocardial infarction, as well as in individuals without cardiovascular disease who are obese or have a deformity of the anterior chest wall. Therefore, this ECG should be subject to further clinical interpretation.

DIAGNOSTIC PROBLEM

Atrial flutter or focal atrial tachycardia?

This is a routine electrocardiogram (ECG; FIGURE 1) showing a regular cardiac rhythm of 66 bpm. The ECG raises a common question: is this atrial flutter or focal atrial tachycardia with a heart block? Of note, experts of the American College of Cardiology, American Heart Association, and European Society of Cardiology (2003) and American College of Cardiology, American Heart Association, and Heart Rhythm Society (2006) currently classify atrial flutter as a type of atrial tachycardia resulting from a large reentry circuit.

The rate of atrial deflections is 240 per minute, so it does not help in the differential diagnosis because the typical range for atrial flutter is from 200 to 400 per minute, and for focal atrial tachycardia—from 100 to 250 (or even up to 300) per minute. In such a case, it may be helpful to assess whether the ECG shows the baseline between atrial waves (P or F) in leads II, III, and aVF. Its presence, as in this case, provides the basis for diagnosing P waves and atrial tachycardia with second-degree atrioventricular block. This type of arrhythmia is typically associated with toxic effects of digitalis and, less frequently, with heart diseases, usually of the right heart and especially with cor pulmonale. However, in this ECG, the only morphological characteristic—left ventricular hypertrophy—does not allow a determination of the cause of arrhythmia.

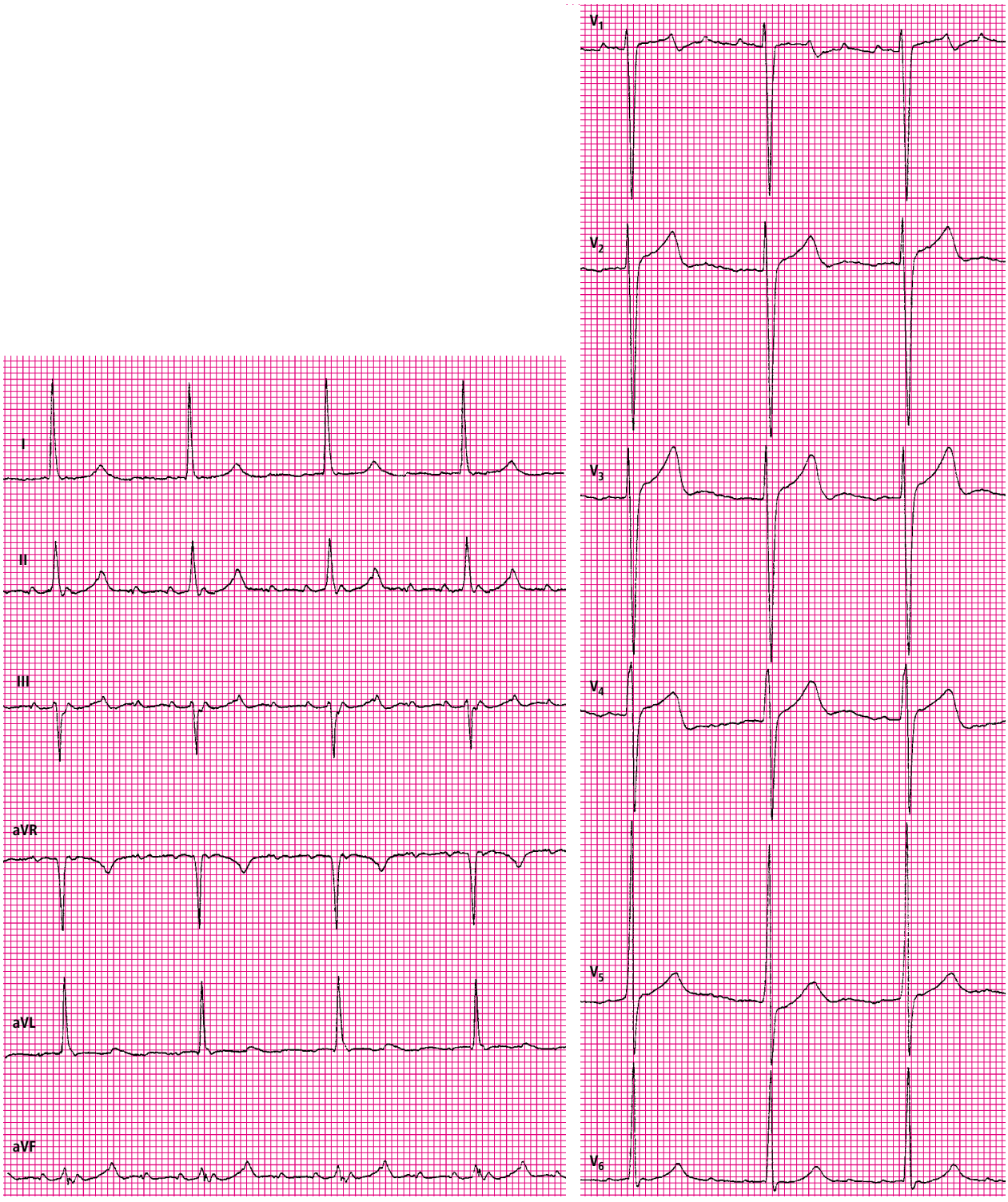

FIGURE 1

DIAGNOSTIC PROBLEM

Bidirectional tachycardia

At first glance, this electrocardiogram tracing, recorded during Holter monitoring, shows bidirectional tachycardia (FIGURE 1A). Tachycardia is diagnosed because the heart rate is 117 bpm. This rhythm does not originate from the sinus node, and it is classified as bidirectional because the QRS complexes are wide and distorted with alternate directions.

Classic bidirectional tachycardia is an ectopic rhythm (reentry or automatic) originating from a group of pacemaker cells and spreading over the ventricles using, alternately, one or the other conduction pathway. Such tachycardia is one of the features characteristic for the toxic effects of digitalis. It originates from a focus located either in the ventricles near the bifurcation of the His bundle or in the atrioventricular junction. However, such a recording may be observed in a few other types of arrhythmia, for example, sinus rhythm with bundle branch block and ventricular bigeminy. In such a case, usually every second QRS complex is preceded by a P wave, but the ventricular rhythm is not regular since it is characterized by alternating RR intervals.

However, in this case, the intervals between the beginnings of the QRS complexes are the same (520 ms). Only further in the recording, at the end of tachycardia (FIGURE 1B), permanent atrial

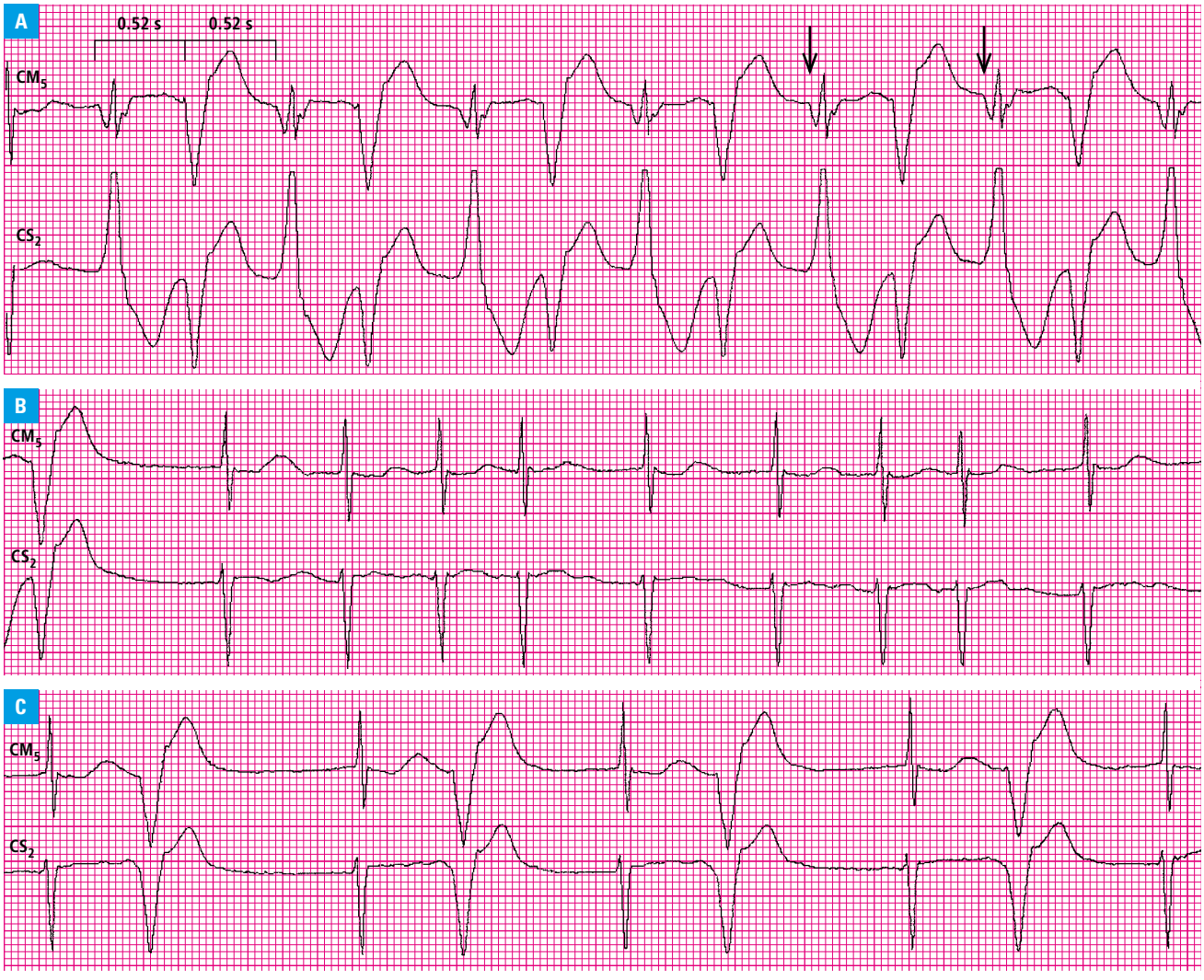

FIGURE 1

fibrillation in this patient can be identified. So what is the mechanism for bidirectional tachycardia shown in FIGURE 1A? The explanation is provided by the tracing in FIGURE 1C, which was recorded immediately before that shown in FIGURE 1A. This patient had ventricular bigeminy lasting a few seconds, which after another few seconds was accompanied by another ectopic ventricular rhythm. The second rhythm was still interfered by the ventricular bigeminy shown in FIGURE 1C.

The history of this patient was unknown, and there was no information on a previous treatment with digitalis glycosides. The recordings from 2 leads of the Holter monitor only indicate that the patient could have suffered an anterior or lateral wall myocardial infarction (an abnormal Q-wave duration of 0.06 s in lead CM_5 in the course of tachycardia [arrows]). It is interesting that this infarction does not manifest within the supraventricular QRS complexes, which are conducted normally. Although the clinical causes of this arrhythmia are unknown, it is still worth to study this case as an unusual example of bidirectional tachycardia induced by an acute episode of idioventricular rhythm and ventricular bigeminy.

DIAGNOSTIC PROBLEM

Indeterminate QRS axis (right superior axis deviation)

This electrocardiogram (ECG; FIGURE 1) was recorded in the 6-channel system at a standard paper speed of 25 mm/s and the 10-mm calibrating deflection representing a 1-mV signal.

A single premature ventricular contraction in the limb leads can be clearly seen (arrow). This contraction is premature, but occurs after a P wave of sinus origin. It is probably a fusion beat and therefore without any impact on the sinus rhythm.

Even without this ectopic evolution, the ECG cannot be considered normal because there are $S_I S_{II} S_{III}$ complexes with the R <S wave in the limb leads (although the complexes may be equal in some parts of the recording). Also in lead aVF, the S waves have a greater amplitude than the R waves, which together with the R <S wave in lead I indicates the presence of an indeterminate axis (right superior axis deviation in the frontal plane). Such a direction of the axis is usually associated with specific diseases: severe right ventricular hypertrophy, hypertrophic cardiomyopathy, or significant intraventricular conduction delay. However, the ECG shows no other abnormalities,

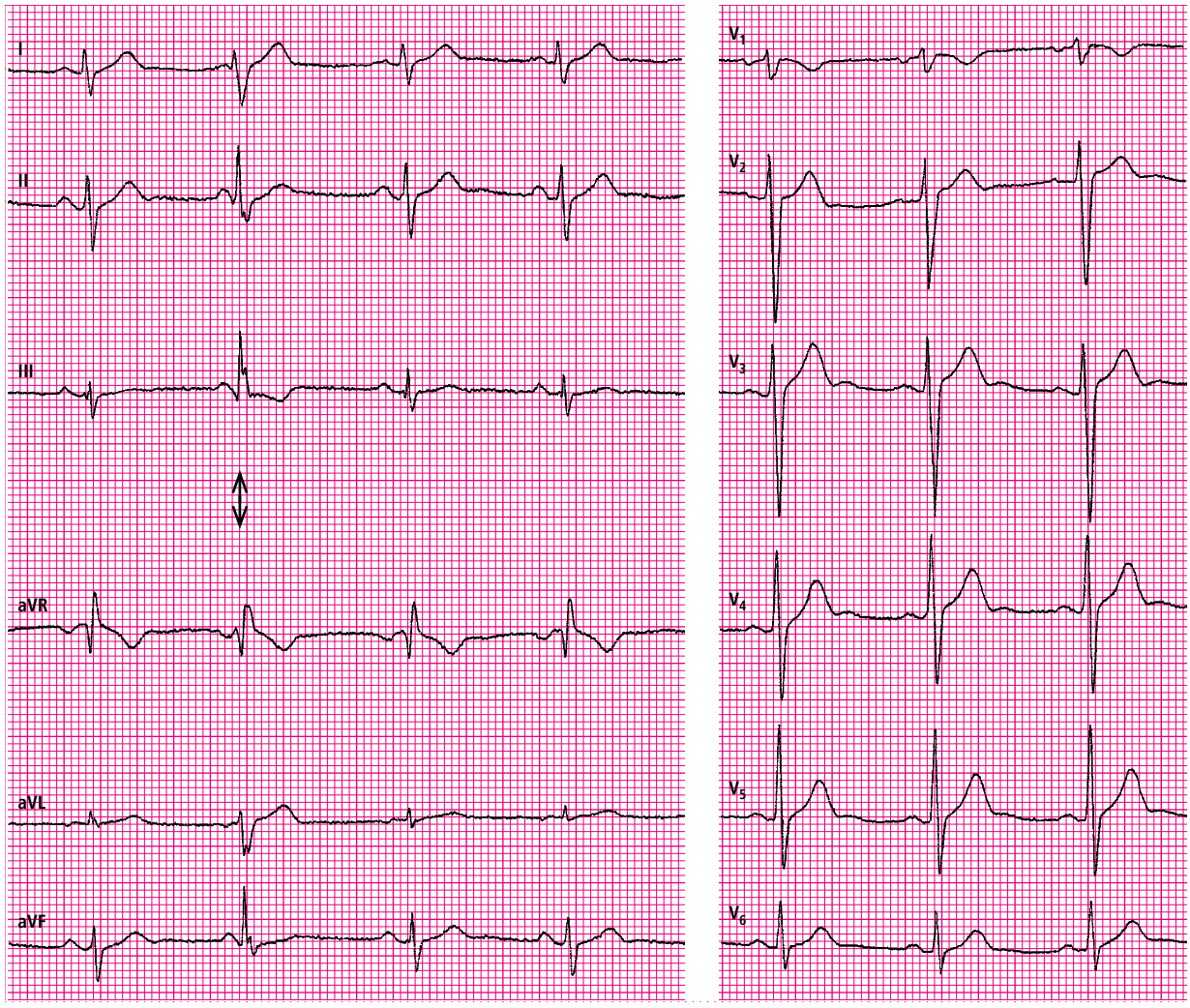

FIGURE 1

particularly in the precordial leads. In such cases, when the history of a patient is unknown, the ECG might be described rather vaguely as "for further clinical interpretation". However, a rare possibility should be considered that we are dealing with some normal variant and that the attending physician will refer the patient for echocardiography, in addition to the analysis of the available clinical data.

DIAGNOSTIC PROBLEM

Left bundle branch block with left axis deviation

This short electrocardiogram (ECG) tracing (FIGURE 1) shows quite a few abnormalities.

It was recorded at a standard paper speed of 25 mm/s, although this may be surprising when looking at its various components, especially the wide P waves and QRS complexes. It is a sinus rhythm (which is the only normal feature of this ECG), and it is quite slow (58 bpm). The P waves meet the criteria for both P mitrale (widened to 140 ms, split in lead I, and characteristically biphasic with a larger negative final phase in lead V_1) and P pulmonale (P-wave amplitude in lead II, 3 mm). The PR interval is prolonged (0.24 s). Left axis deviation (–50°) with a q wave and an extended time to the peak of the R wave (≥45 ms) in lead aVL would allow a diagnosis of left anterior fascicular block, according to the criteria of the American College of Cardiology, American Heart Association, and Heart Rhythm Society (2007 and 2009). However, the wide QRS complexes (0.2 s) would allow this type of diagnosis only in the presence of right, not left, bundle branch block, while in this ECG the prolongation of conduction to the peak of the R wave to 0.12 s in lead V_6 clearly indicates that this is the left bundle branch block. According to most experts, the assessment of an intraventricular conduction abnormality in such a case should be limited to a diagnosis of left bundle branch block with left axis deviation. A change in this overcautious approach will be possible when contemporary researchers will be curious enough to continue the research began already after 1950s that indicated the anatomical possibility of the left bundle block on 2 levels, with a characteristic ECG tracing. In the meantime, we may support the European Society of Cardiology experts who in their 2009 guidelines on syncope went as far as to include "bifascicular block, that is, left or right bundle branch block with accompanying

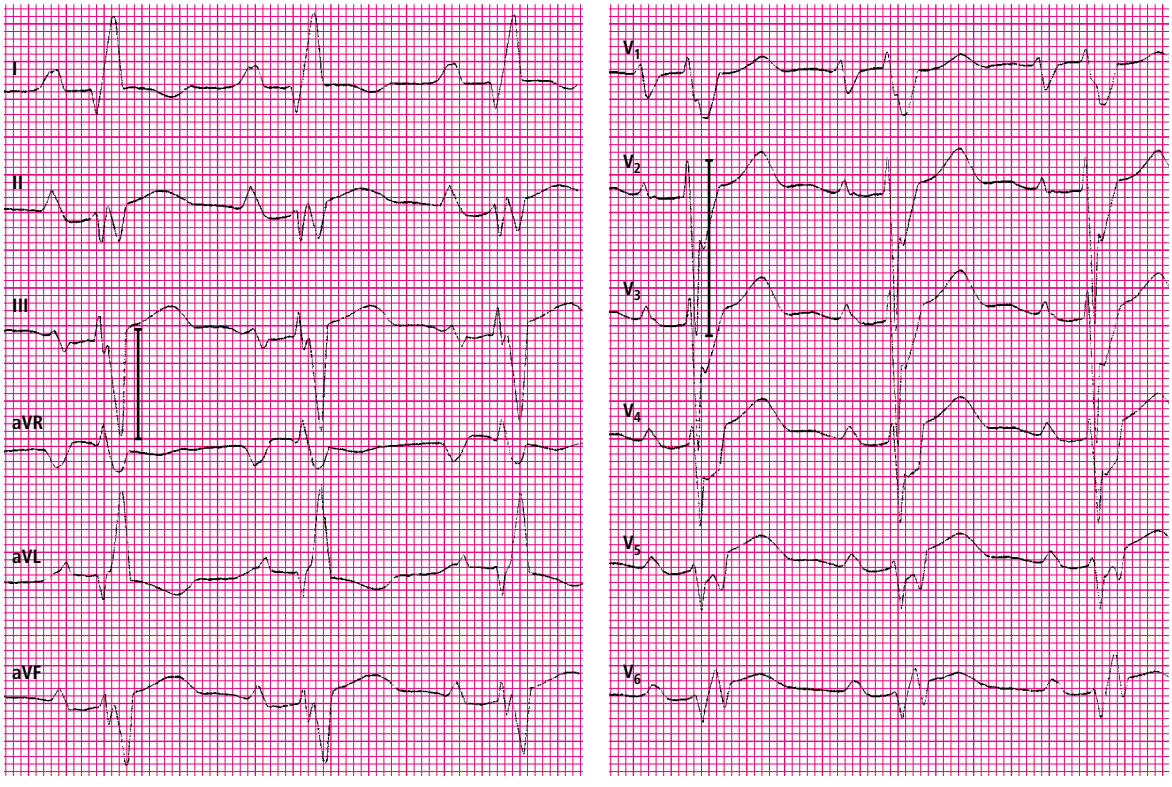

FIGURE 1

block of the left anterior or posterior fascicle" as one of the ECG features suggesting arrhythmia as the cause of syncope.

The abnormal Q waves in leads I, II, aVL, and V_6 that coexist with this bundle branch block (although in leads II and V_6, residual r waves in the QRS complexes known as rSR'S' complexes can be seen) are features of lateral wall myocardial infarction. The elevation of ST segments in leads V_5 and V_6 (in patients with left bundle branch block, these segments would rather show downsloping depression) allows an identification of persistent Pardee wave, probably as the feature of a postinfarction left ventricular aneurysm.

Although the R-wave amplitude in lead aVL in the presence of left anterior fascicular block (provided it actually is the fascicular block) is not a reliable criterion for left ventricular hypertrophy, the sum of the S_{III}-wave amplitude and the largest QRS complex in the precordial leads exceeds 30 mm (S_{III} + QRS_{V2} = 14 + 23 = 37 mm), which supports this diagnosis. Finally, the QT interval (0.5 s with a heart rhythm of approximately 60 bpm) is prolonged, which is not exclusively due to the left bundle branch block because its presence increases the QT interval by no more than 0.03 s.

The description of these few cardiac cycles should thus include the numerous abnormalities presented above, in addition to a possible reference to a clinical diagnosis or a comparison with the previous ECG, if available.

DIAGNOSTIC PROBLEM

Is this really sinus tachycardia?

FIGURE 1 shows a routine electrocardiogram (ECG) with 2 premature ventricular beats.

The basic rhythm is accelerated to 110 bpm, while the shape of P waves in leads I and II allows an identification of sinus tachycardia accompanied by first-degree atrioventricular (AV) block (PR interval, 0.24 s) and the 2 premature ventricular contractions described above. A "rigid" (very regular) rhythm of P waves, which remains unaffected by premature ventricular beats, raises the possibility of ectopic atrial tachycardia. However, to confirm or exclude this arrhythmia, a longer ECG tracing (preferably including the start of tachycardia) or a previous ECG with a slower heart rate would be necessary. The latter would allow a comparison of the shape of P waves between the 2 tracings. Of note, in the 2007 statement of the American Heart Association, American College of Cardiology, and Heart Rhythm Society on standardization and interpretation of ECG, experts recommended to no longer use the previously popular diagnosis of "paroxysmal

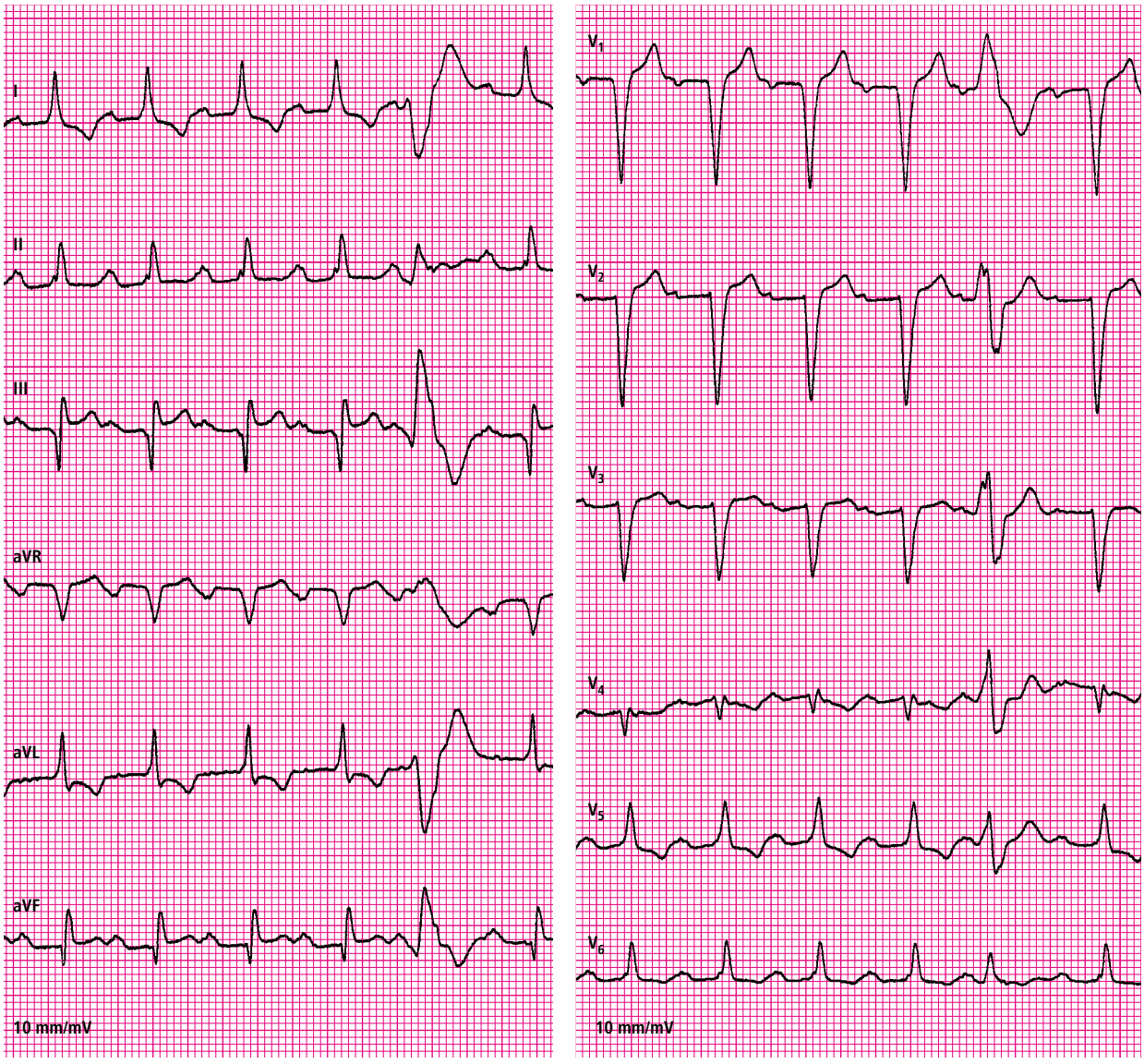

FIGURE 1

atrial tachycardia" (PAT; including also PAT with AV block) but rather to drop the word "paroxysmal" in such contexts.

What other abnormalities can be found in this ECG? The incomplete left bundle branch block may be responsible for ST-T changes and for the lack of R-wave progression in leads V_1 to V_3; however, a small rSr' (or even Qr)-type QRS complex in lead V_4 raises the suspicion of a scar after anterior wall myocardial infarction. Undoubtedly, features of inferior wall myocardial infarction can be identified. The ST-segment elevation in leads III and aVF (although concave) requires differentiation between the evolution of the ECG during a myocardial infarct and persistent elevation indicating the possibility of a postinfarction ventricular aneurysm.

DIAGNOSTIC PROBLEM

Tombstone-like QRS distortion

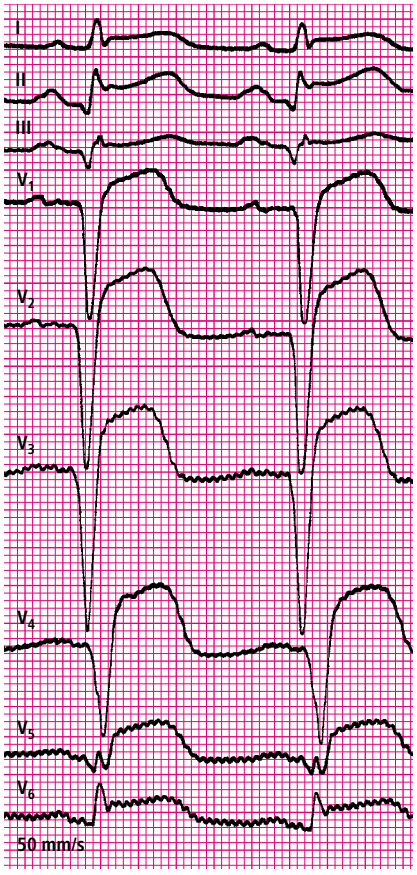

FIGURE 1

This electrocardiogram (ECG; FIGURE 1), recorded at a speed of 50 mm/s, shows a specific configuration of QRS-T complexes, sometimes seen in acute myocardial infarction (MI) and constituting a very unfavorable prognostic factor. The ECG pattern has a characteristic tombstone appearance. It consists of 2 ECG features: 1) QRS complexes without R waves or with residual (very small and narrow) R waves, and 2) dome-shaped elevation of all ST-segments, merging with the descending limb of the T waves.

Such configuration can be seen in leads V_1 to V_4. It is accompanied by features of acute MI, including the lateral and inferior walls, which allows a use of a rather imprecise term of apical MI. Such a location of changes indicates the proximal occlusion of the left descending anterior coronary artery above the first diagonal branch.

In patients with tombstone-like ST-segment elevation in the course of acute MI, death during hospitalization occurs 10 times more frequently than in those without such elevation. In 2006, it was reported that not only the amplitude of ST--segment elevation but also the characteristic shape of the QRS-T complex is an ECG feature associated with unfavorable prognosis.

DIAGNOSTIC PROBLEM

Left bundle branch block with left ventricular hypertrophy

In this interesting electrocardiogram (FIGURE 1A and 1B), only 2 abnormalities do not raise doubts: sinus bradycardia (approximately 50 bpm) and left bundle branch block. The exceptionally deep S waves in leads V_2 and V_3, reaching 45 mm, deserve particular attention. This feature, despite the presence of very small R waves in the left precordial leads, allows an identification of left ventricular hypertrophy on the basis of the criteria that are rarely used but are recommended in the 2009 guidelines of the American Heart Association, American College of Cardiology, and Heart Rhythm Society (AHA/ACC/HRS). These criteria include the sum of the amplitudes of the R and S waves in one of the precordial leads exceeding 35 mm or the sum of the amplitudes of S_{V2} and $R_{V5,6}$ waves exceeding 45 mm. On the other hand, such a marked clockwise rotation of QRS in the horizontal plane in a patient with left ventricular hypertrophy is most likely a sign of a significant hypertrophy causing the displacement of the apex downwards and to the left.

What is the origin of the 2 QRS complexes marked as numbers 4 and 5 in FIGURE 1B? The shape of these complexes, which resembles that of the QRS complex seen in sinus rhythm, indicates that these are supraventricular beats. Interestingly, before the QRS complex no. 5, a P wave can be seen that overlaps the peak of a preceding T wave (arrows) and is best visible in leads V_3 to V_5, while the PP interval between beats 3 an 5 is similar to the intervals between beats 1 and 2, 2 and 3, as well as 5 and 6. Therefore, the QRS complex no. 5 is simply a sinus beat, while the prolonged PR interval in this cardiac cycle is a result of a physiological slowing of atrioventricular nodal conduction, which remains in a relative refraction period after the premature beat. Thus, beat 4 can be described as an interpolated beat because it did not discharge the sinus node. How was that possible? Most probably, it originated from the atrioventricular junction. Beats from this area sometimes do not conduct back to the atria.

And the next question: what is the cause of a prolongation of the RR interval to 1.48 s between beats 6 and 7? The diagnosis of postextrasystolic abnormalities of the sinus rhythm is tempting; however, this premature beat is not of ventricular origin, it is not accompanied by compensatory pause, and finally, no initial short postextrasystolic acceleration of the sinus rhythm can be seen. Therefore, the only diagnosis left is a physiological sinus arrhythmia.

The variability of the T waves in the precordial leads, especially V_4 and V_5, is another interesting feature. The mere presence of symmetrical negative T waves in lead V_4 during slow sinus rhythm is also interesting because their shape is not typical for repolarization abnormalities secondary to bundle branch block or left ventricular overload (seen in leads I, aVL, and V_6). What is more, they are accompanied by negative QRS complexes. When clinical data are not available, it is hard to tell what may be the specific cause of this symptom (eg, previous nontransmural myocardial infarction). On the other hand, the development of positive T waves in beats 4 and 5 (with short RR intervals) is a manifestation of transient "pseudonormalization" during ventricular repolarization caused by the shortening of the cardiac cycle, a symptom that is usually seen in patients with primary abnormalities of the T waves (ie, not related to repolarization abnormalities). This finding is not clinically significant.

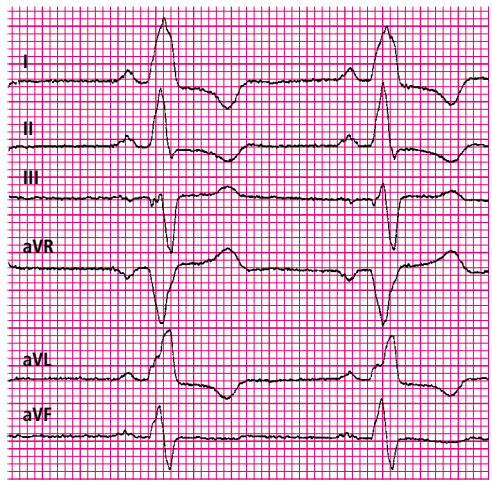

FIGURE 1A

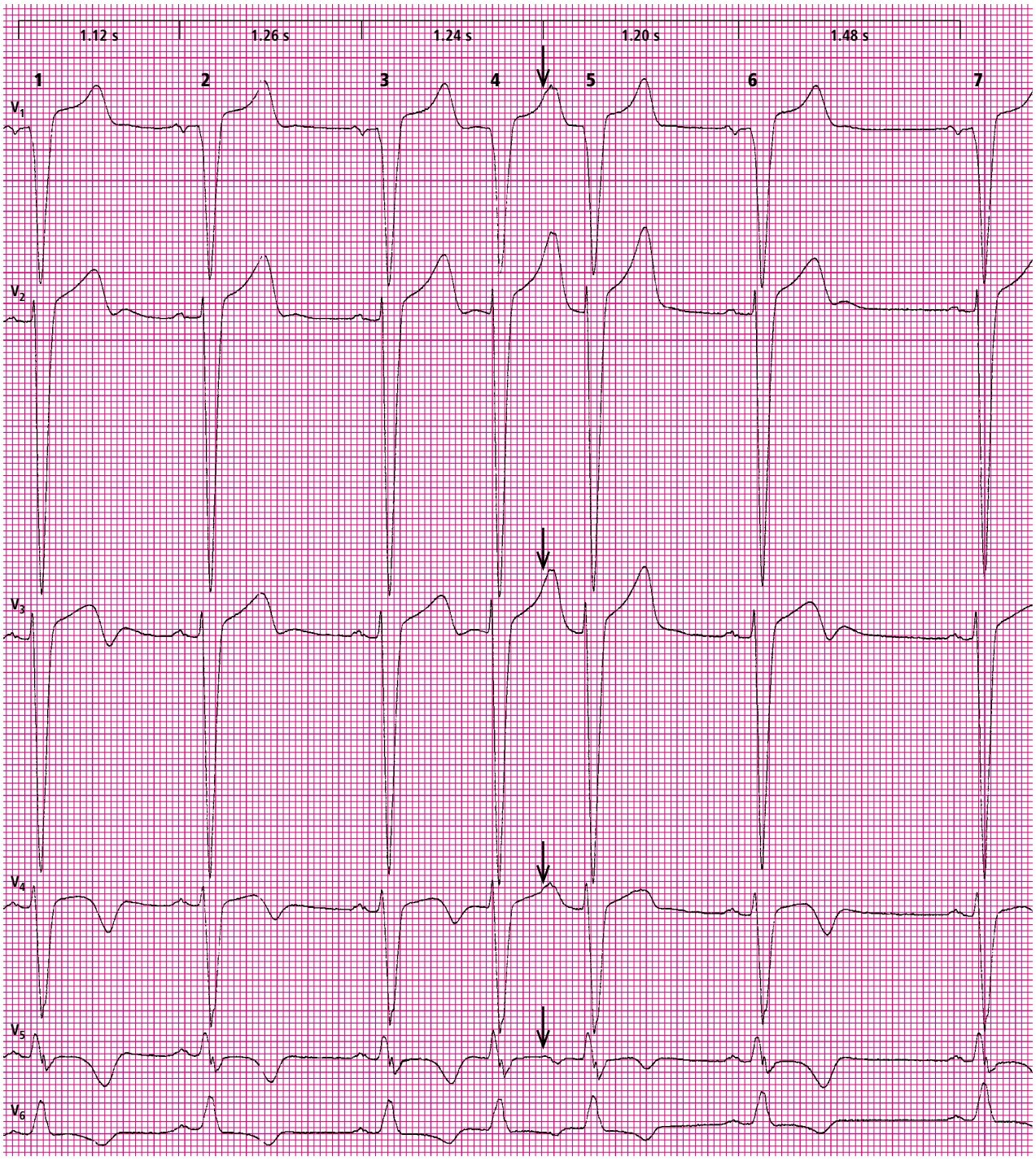

FIGURE 1B

DIAGNOSTIC PROBLEM

Right bundle branch block with left anterior fascicular block

This is a simple electrocardiogram (FIGURE 1) showing the late evolution of an extensive anterior wall myocardial infarction in a 56-year-old man with persistent right bundle branch block and left anterior fascicular block. Although according to the experts of the American Heart Association, American College of Cardiology, and Heart Rhythm Society (2009), QRS complexes in the left anterior or posterior fascicular block should be shorter than 0.12 s, the experts of the Polish Cardiac Society (2010) allow an exception, namely, the simultaneous presence of the right bundle branch block with left anterior or posterior fascicular block. However, all these societies consider the popular term "bifascicular block" to be inadequate.

Nonetheless, it is worth noting that simultaneously with the release of these guidelines, their authors published textbooks of electrocardiography providing the basis for diagnosing bifascicular and trifascicular blocks as well as containing information on their clinical significance.

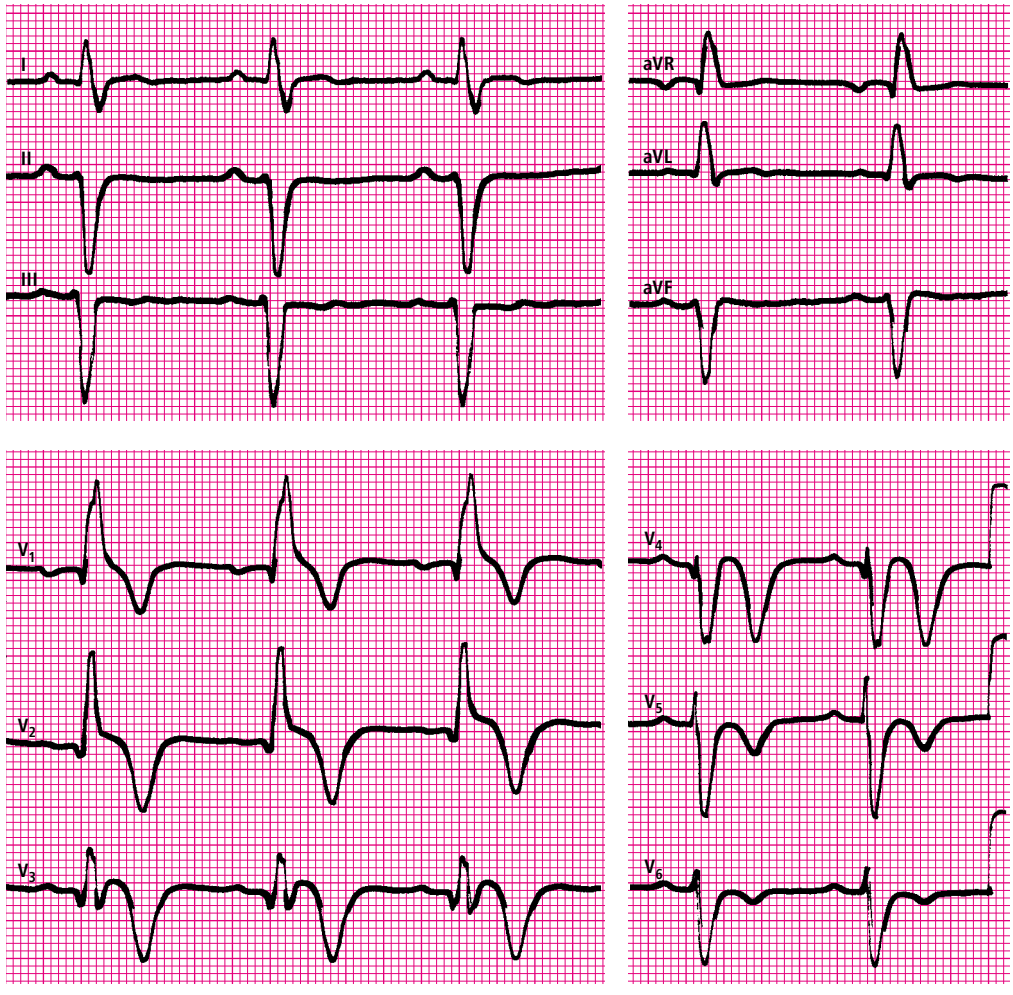

FIGURE 1

DIAGNOSTIC PROBLEM

Premature beats in a patient with left bundle branch block

FIGURE 1 presents a rather simple electrocardiogram (ECG) with normal sinus rhythm, isolated premature beats, and left bundle branch block. What is the origin of these 2 premature beats?

If these were supraventricular beats, they would have the shape of a left bundle branch block, while the shape of a right bundle branch block indicates that they originated from the left ventricle. The beats cannot be of supraventricular origin also because they are not preceded by premature P waves and, finally, there is no retrograde discharge of the sinus node (the premature beats are accompanied by a compensatory pause). Moreover, both nonconducted sinus

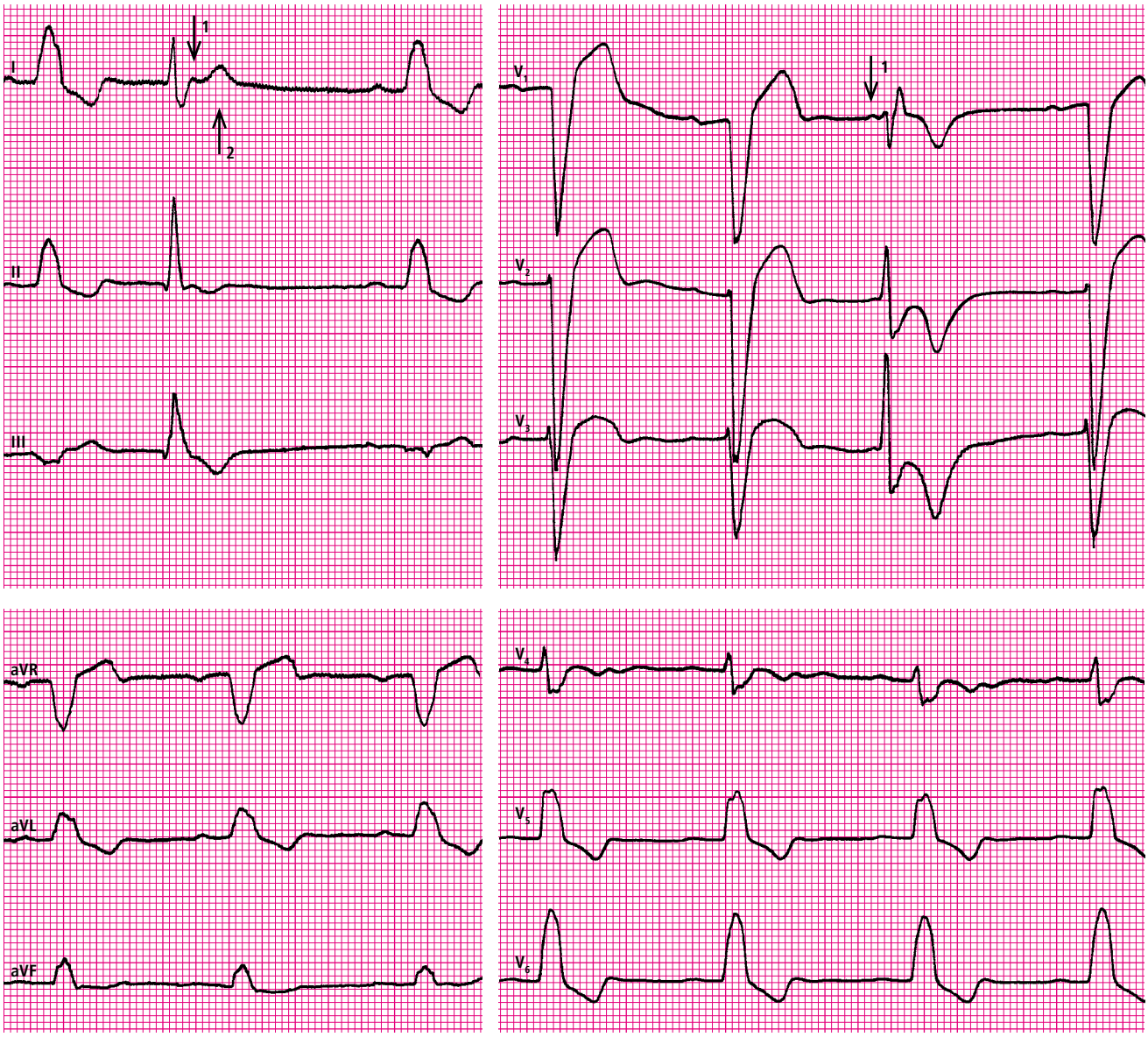

FIGURE 1

P waves are present: in the case of the first premature beat, the P wave is seen after the QRS complex in leads I and II, and in the case of the second ectopic beat—immediately before the QRS complex in leads V_1 and V_3 (arrows 1). The different locations of the sinus P waves in relation to the ectopic QRS complexes obviously result from a different coupling of premature beats in relation to the preceding beats.

A surprising feature are unusually deep and wide ectopic T waves in leads V_1 to V_3. They could be assumed to reveal acute myocardial ischemia, although it is more likely that they reflect the so called cardiac memory of abnormal deep and wide QRS complexes observed during the basic heart rhythm. This is rather an unusual possibility, typically considered in the context of sinus rhythm. It is supported by the presence of a positive ectopic T wave in lead I (arrow 2), in which sinus QRS complexes are positive, as well as the presence of a negative T wave in lead III, which is "less negative" than the ectopic T waves in the precordial leads. Both criteria support the presence of cardiac memory rather than ischemia.

Another issue is the shape of the QRS complexes of sinus origin in lead III, resembling a shallow and notched bowl. Because the neighboring lead (aVF; obviously this is adjacent not in a standard ECG but in the panoramic [hexaxial] reference system for the limb leads [ie, the Cabrera sequence]) does not show features of the loss of potential, this QS complex cannot be interpreted as a manifestation of prior myocardial infarction (MI). However, it should also be noted that the duration of the QRS complexes with the left bundle branch block in this tracing exceeds 150 ms, which suggests the concomitant slowing of conduction in the damaged ventricular myocardium.

Finally, the most alarming symptom (suggesting acute MI) is the dome-shaped ST-segment elevation in leads V_1 to V_3, which corresponds to the Pardee wave but is atypical for the left bundle branch block in a patient without acute MI. In addition, this elevation also meets the Sgarbossa criterion for acute MI in a patient with bundle branch block, because it slightly exceeds 5 mm. This presentation requires a clinical diagnosis to confirm MI. The diagnosis in this case is unknown, but taking into account the normal heart rate (65 bpm) and unnotched QRS complexes in the bundle branch block, it may be assumed that this ECG is one of the 10% of false positive diagnoses of acute MI established on the basis of this criterion.

DIAGNOSTIC PROBLEM

An abnormal heart rate turbulence after a premature ventricular beat

FIGURE 1 shows a standard example of 2 premature ventricular beats followed by a compensatory pause. However, an interesting finding is an escape beat from the atrioventricular junction after a ventricular beat in leads V_1 to V_6, which appears directly after the P wave of sinus origin (arrow 1). Why such an escape beat occurred at all? It is known that after premature ventricular beats that do not discharge the sinus node, after the so called compensatory pause, complete

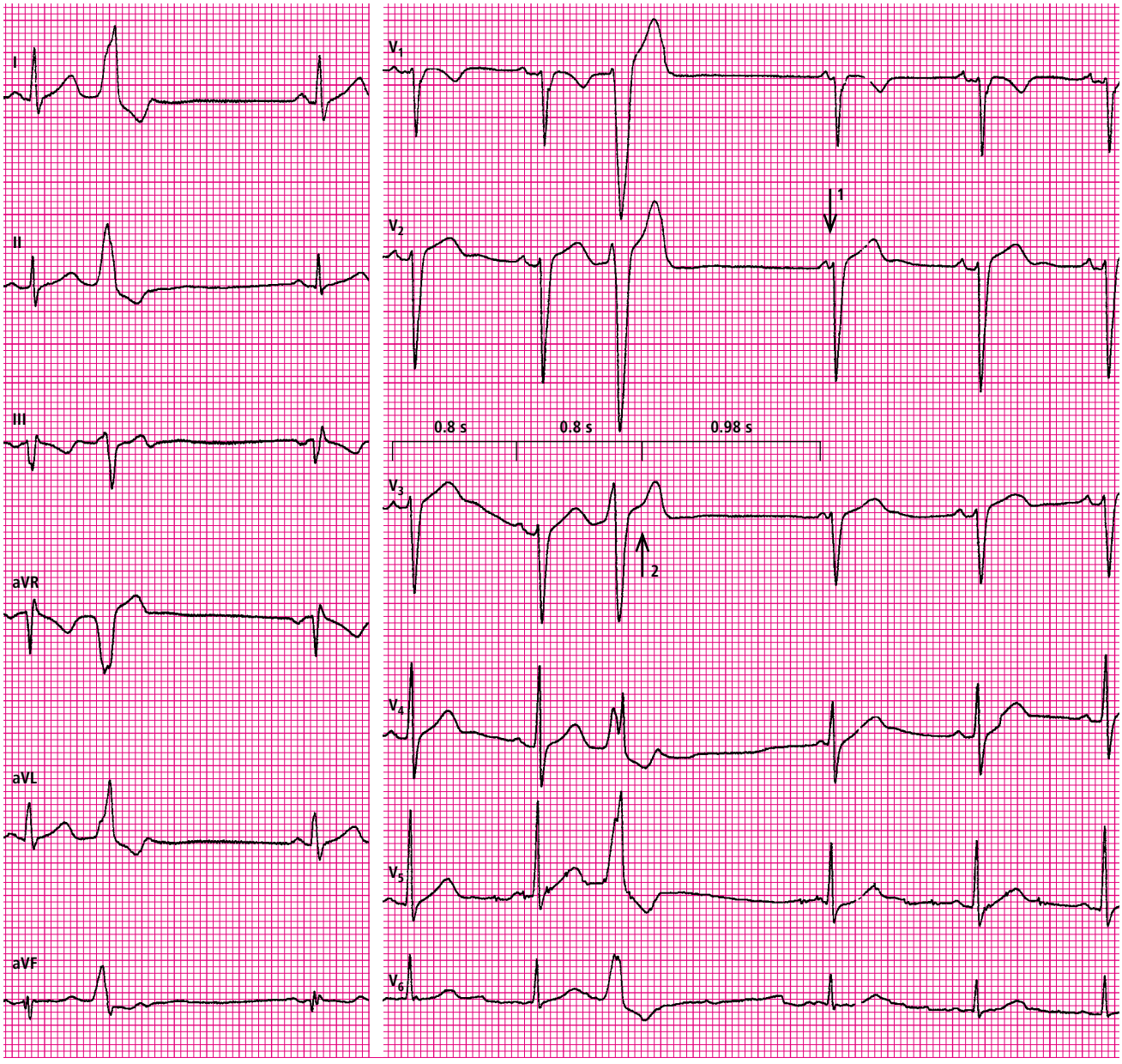

FIGURE 1

sinus beats rather than premature beats can usually be observed.

In comparison with the duration of the RR intervals of sinus origin, separated by a premature ventricular beat, the time after which the premature beat occurs seems short, but in fact these 2 premature beats do not occur too early. They appear after the T waves and are additionally separated from them by the baseline. Therefore, it is rather the R'R interval (between the premature beat and the next beat) that is unusually long. This interval, summed up with the preceding RR' interval, should equal a double RR interval of sinus origin and could be even shorter than the duration of the 2 RR intervals preceding a premature ventricular beat, owing to a physiological phenomenon of short-term acceleration of sinus rhythm after a premature beat. This acceleration causes additional pulse wave in the sinus node artery. After this short acceleration in heart rate, including 2 to 3 cardiac cycles, usually an opposite reflexive reaction can be seen, namely, a gradual extension of several consecutive RR intervals. The sequence following a premature beat is called the heart rate turbulence, and its intensity reflects the sensitivity of arterial baroreceptors in the carotid arteries. In this case, the PP intervals rather than the RR intervals should be measured (because of the escape beat originating from the atrioventricular junction, which caused the shortening of the RR interval following the premature beat). The P wave of sinus origin blocked by extrasystole is seen (poorly but undoubtedly in leads V_3–V_6) on the ascending arm of the ectopic T wave (arrow 2). In this tracing, no shortening of the PP intervals occurred; quite the contrary, while the duration of the PP intervals in the precordial leads before and during the premature ventricular beat was 800 ms, the PP interval after this beat increased to 980 ms. In the subsequent beats, it decreased to 940 ms and finally down to 880 ms.

This observation not only explains the origin of the escape beat (which was obviously the excessive pause after the premature beat), but also allows an assumption that this patient has a 5 times higher risk of sudden cardiac death in comparison with patients with preserved heart rate turbulence following a premature beat. In this particular case, such a conclusion could be reached without the use of special software for calculating turbulence parameters, because the stage of the initial acceleration of sinus rhythm after the premature beat did not occur at all. The accuracy of prediction of mortality based on the assessment of the severity of heart rate turbulence is not high because it reaches 33% in the case of abnormal results. Hence, experts of cardiac societies do not recommend this method for prognosis assessment (class IIb recommendation). However, when the heart rate turbulence is readily at hand, it is worth having a closer look at an ECG tracing and search for other possible threats to the patient. In this ECG, not many abnormalities can be seen. Only the notching of the QRS complex in leads III and aVF may suggest "loss of potential", that is, the possibility of a scar after prior inferior wall myocardial infarction. This slightly increases the significance of the above prognostic factor, namely, the lack of physiological heart rate turbulence after a premature beat.

DIAGNOSTIC PROBLEM

An isolated QRS complex of less abnormal appearance compared with the other complexes

This electrocardiogram tracing (FIGURE 1) was recorded at 11:14 during Holter monitoring in a 65-year-old man. What might be the origin of a single QRS complex in the middle, which is slightly narrower and less abnormal than the remaining QRS complexes and has narrow S waves in both leads?

First of all, the basic heart rate should be assessed, which obviously reveals sinus bradycardia (41 bpm) with left bundle branch block. Because this diagnosis includes the QS complexes with ST-segment elevations not exceeding 5 mm in leads V_1 to V_3, which corresponds to the features seen in lead CS_2, there is no point in considering whether this patient suffered anterior wall myocardial infarction. Sinus bradycardia is probably a result of sleep.

And what is the origin of the QRS complex in the middle, which differs from the others? Obviously, this is not a premature atrial beat (since the shape of the P wave is the same as that of the other P waves, and it occurred precisely within the expected time designated by a sinus rhythm). Also, this is not an intermittent ventricular preexcitation beat using an accessory conduction pathway (which could be suspected because of a short PR interval) because the QRS complex does not reveal features characteristic for this preexcitation. Therefore, the only possible diagnosis is an isolated ectopic ventricular beat (probably coming from the interventricular septum), which bypasses the block area in the left bundle branch and is perhaps partially summed up with the sinus beat. However, is it really conducted without any obstacles? The shape of the QRS complex with a deep S wave in lead CM_5 raises the possibility of an additional left anterior fascicular block that was not modified by this premature ventricular beat.

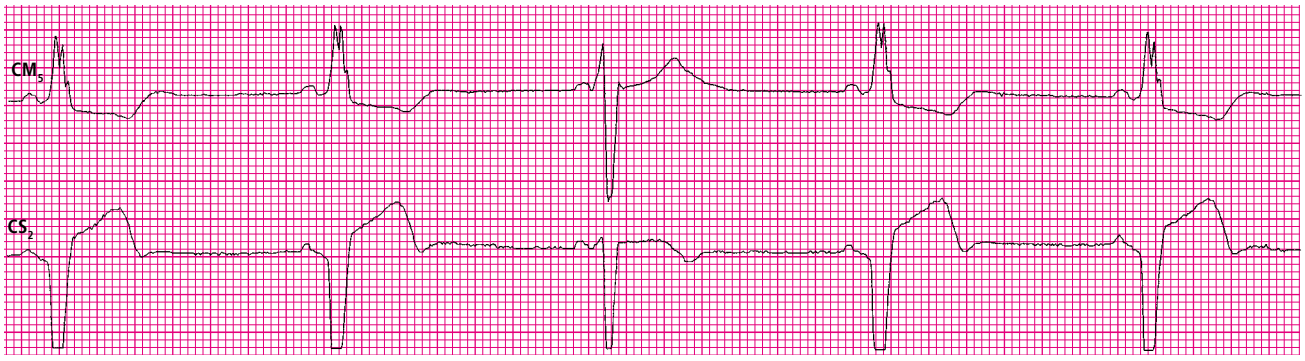

FIGURE 1

DIAGNOSTIC PROBLEM

A double-fusion QRS complex

The lower electrocardiogram tracing, recorded during Holter monitoring (FIGURE 1), shows a sinus rhythm with ventricular preexcitation. In lead CS_2, which corresponds to lead V_2 in a standard electrocardiogram, a negative delta wave is clearly marked on the descending arm of the S wave.

The upper tracing is quite extraordinary. The beginning shows the end of a typical atrioventricular reentrant tachycardia: negative P waves corresponding to a retrograde atrial excitation in lead CM_5 (arrows) with RP intervals exceeding 70 ms. Once tachycardia resolves, the sinus rhythm with features of preexcitation returns; however, the first cardiac cycle is additionally distorted by an ectopic ventricular beat (which in this case should be called an escape beat). Because the beat reaching the ventricles from the sinus node in the presence of an additional conduction pathway is a fusion beat, the overlapping ventricular beat results in a double-fusion QRS complex caused by excitation from 3 different sources. Such a phenomenon is rare.

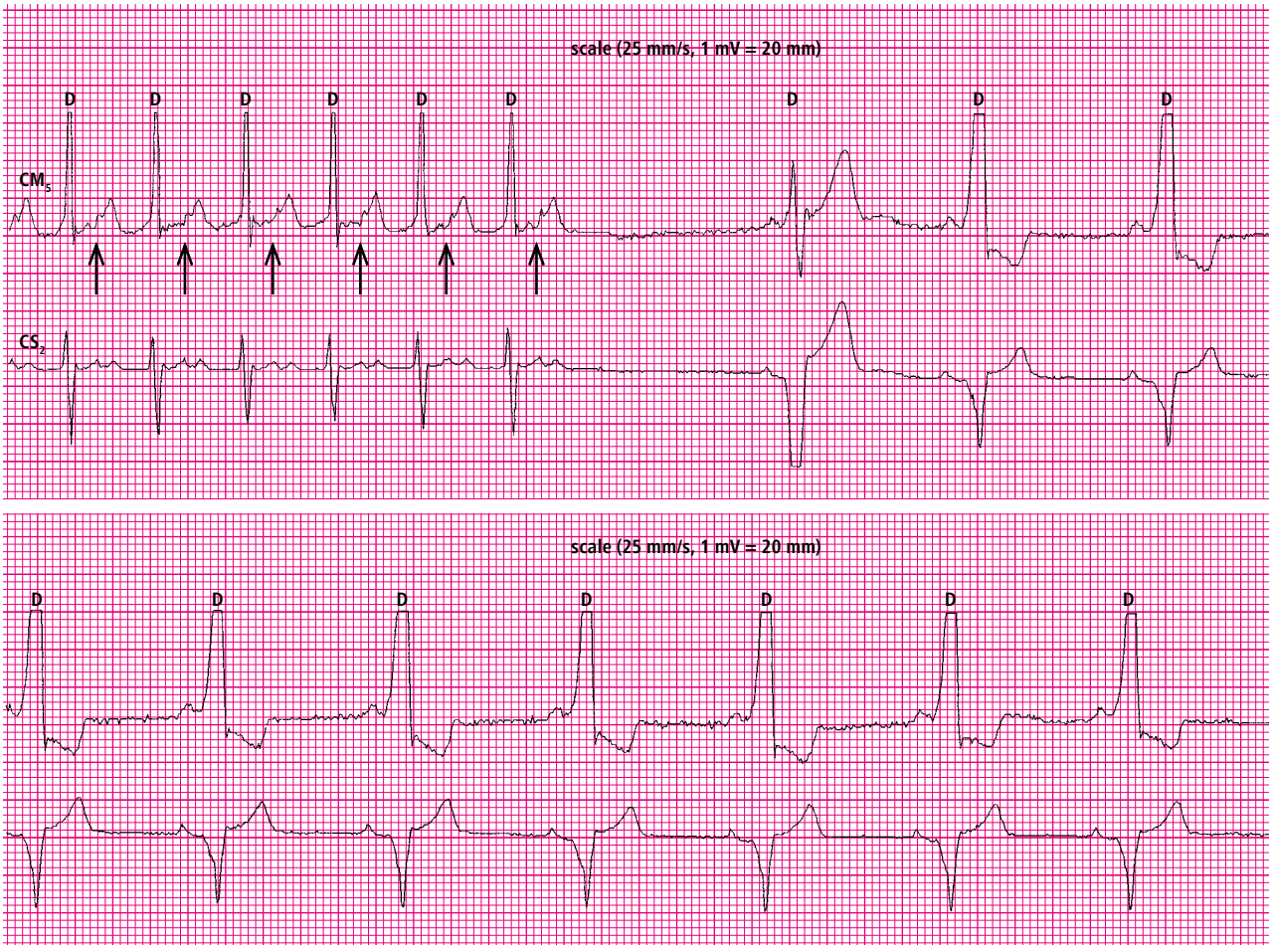

FIGURE 1

DIAGNOSTIC PROBLEM

A premature beat with a narrower QRS complex than that in beats of sinus origin

This electrocardiogram (FIGURE 1) shows the evolution of inferior wall myocardial infarction (MI) spreading to the apex (negative T waves in leads V_4–V_6,). The lack of R-wave progression in leads V_1 to V_3 with accompanying notching of the QRS complexes in leads V_3 and V_4 suggests the possible coexistence of anterior wall MI, which has probably developed before this coronary event. The widening of the P waves in leads II and V_3 to V_5 allows an identification of atrial conduction abnormalities.

The sinus rhythm (90 bpm) is interfered by 2 premature beats. The second premature beat, with a QRS complex duration of 160 ms and the shape resembling the left bundle branch block rather than the right bundle branch block, probably comes from the edge of a septal MI, from where it can faster reach the right bundle branch rather than the left bundle branch. In patients after MI, such a location of the ectopic focus is more likely than the location in the right ventricle. The origin of the first premature beat (occurring only 0.3 s from the beginning of the preceding QRS complex of sinus origin) is more interesting. It is located on the descending arm of the T wave with a narrower QRS complex than the complexes of sinus origin.

As regards tachycardias with the QRS complexes that are narrower than those of sinus origin, such feature is considered a reliable evidence of their ventricular origin from the focus located near the base of the heart, usually near the left bundle branches or in an area with no conduction disturbances. However, in the case of a single

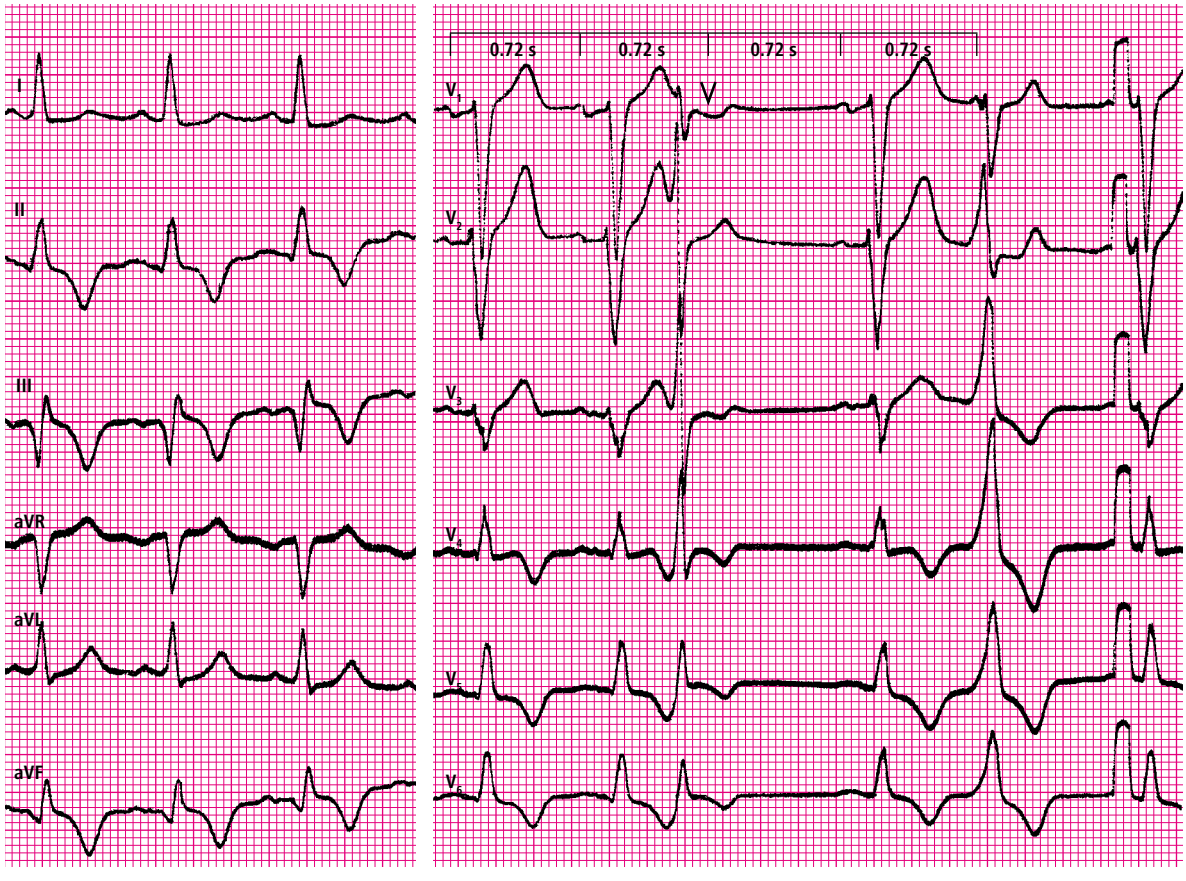

FIGURE 1

premature beat, the presence of a supraventricular focus might be considered, with the stimulus reaching the ventricles in the "supernormal" phase, that is, the phase of hyperactivity. This phase is characteristic for stimuli superimposed in the end part of the third phase of an action potential, but then we would expect to see an ectopic P wave before the QRS complex or to find an indirect evidence for a discharge of the sinus node. Yet, in this case, there is no track of a P' wave and a typical compensatory pause is present, notably, without heart rate turbulence (a dashed arrow indicates the location of the invisible sinus P wave superimposed on the ST segment of the premature beat). Moreover, a supraventricular beat that occurs so close would have to reach the atrioventricular node in the period of its functional refraction, and at least for that reason, it would cause a delay in the supraventricular stimulus reaching the ventricles. Therefore, for many reasons, it is a highly unlikely option, and the only possibility left is the premature ventricular beat. The different shape of these 2 beats is not the evidence for their initiation in 2 different foci because septal beats often propagate through different pathways, depending on the temporary state of their conduction within the various fascicles. A shorter duration of this QRS complex in comparison with QRS complexes of sinus origin should be associated with the "supernormal" conduction mentioned above, usually lasting no longer than 80 to 100 ms. This phenomenon was confirmed experimentally by stimulating the heart in this narrow scope of coupling with the preceding stimulus.

Ventricular extrasystole with short coupling always raises concerns that a stimulus might reach a "vulnerable" phase located nearby the peak of a T wave (a phenomenon known as "R-on-T"), while in patients after MI nonhomogeneous repolarization, which poses a risk of life-threatening ventricular tachyarrhythmias, is not uncommon. This situation is characterized by extended QT intervals and their spatial dispersion, among other features. In the present case, the QT interval is normal (400 ms) at first glance, and after the correction for heart rate using the Hodges method, it is 453 ms. Therefore, it only slightly exceeds the upper limit of normal established for a corrected QT interval in men (ie, 450 ms). The QT--interval dispersion in this tracing is quite small, around 20 ms. Thus, except for a single premature beat (short-coupled and without postextrasystolic turbulence) and features of MI evolution, there are no other sings in this electrocardiogram indicating a risk of dangerous ventricular tachyarrhythmias.

DIAGNOSTIC PROBLEM

Supraventricular tachycardia

The first 2 electrocardiogram (ECG) tracings, recorded during Holter monitoring (FIGURE 1), show the onset and end of a paroxysmal supraventricular tachycardia. Between these points, a tachycardia of 24 s in duration and 163 bpm in frequency is seen.

Let us start from establishing the type of tachycardia. It begins with a wide QRS complex preceded by a premature ectopic P wave, imposed on a T wave from the previous cycle (arrow 1). This ectopic atrial stimulus reached the ventricles with delay (PR interval, 0.3 s), probably via a slow nodal conduction pathway, and returned to the atria using the fast pathway shown on this ECG as a retrograde P wave immediately after the QRS complex (arrow 2). This sequence initiates a recurrent tachycardia originating from the atrioventricular node. Ventricular depolarization is initially associated with aberrant conduction, but after 10 cycles the normal pathway of intraventricular conduction is restored. Throughout the recording, in leads CM_5 and CS_2, negative P waves can

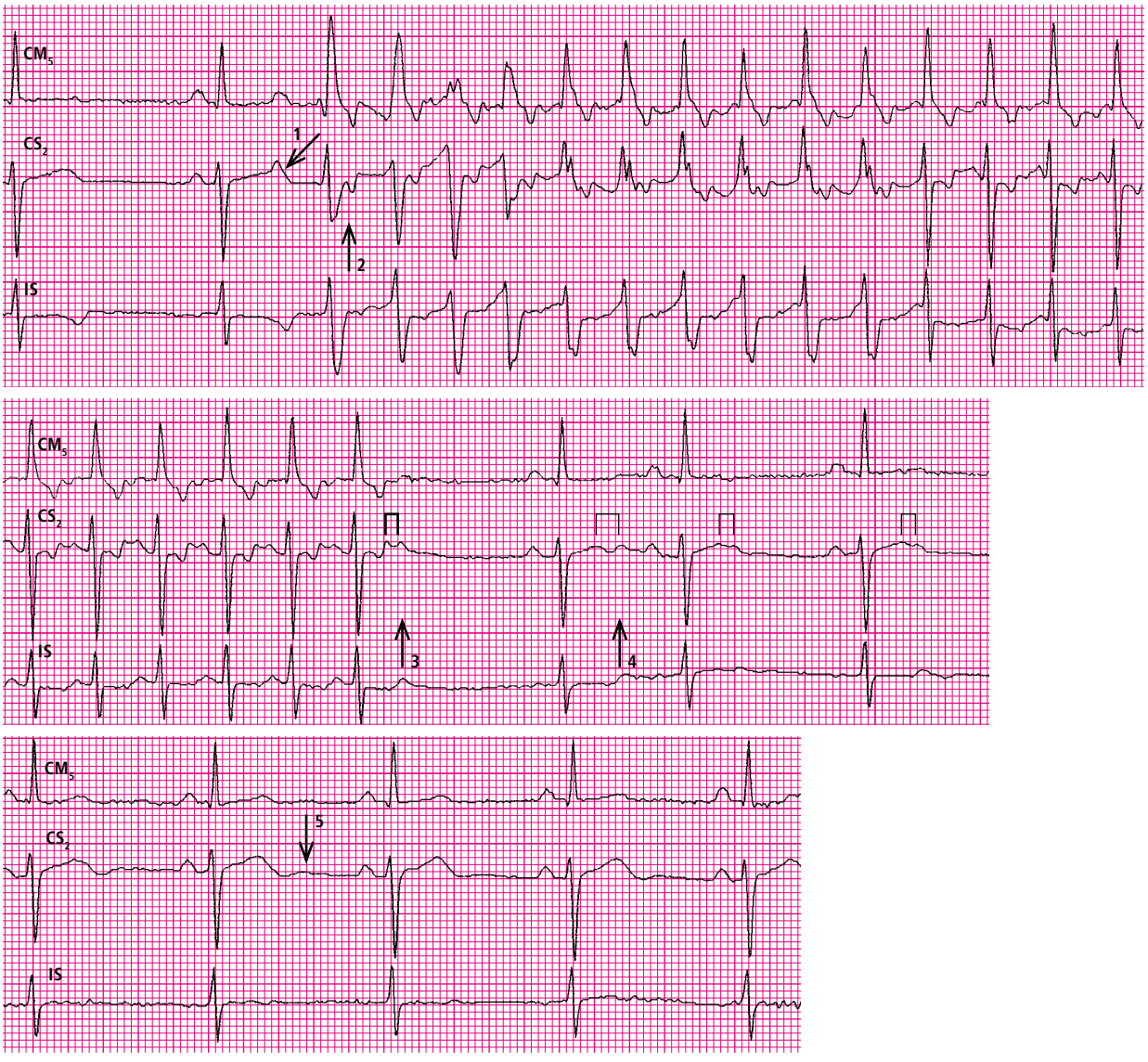

FIGURE 1

be seen after the QRS complexes. After less than 30 s, tachycardia resolved.

Another interesting ECG feature during this supraventricular tachycardia is variability of the T waves. After the resolution of an aberration within the QRS complexes, the T waves seem to be split (bold brackets). In the last beat of supraventricular tachycardia shown in the middle tracing, it is clear that the peak of the T wave in lead IS (corresponding to limb lead III) is synchronized with the second peak of the split T wave in lead CS_2 (arrow 3). Perhaps the supposed first peak of the split T wave is not the peak of the T wave but the second phase of the negative–positive P wave? However, in the first beat of sinus origin, the pattern of these 2 components is stretched (arrow 4, brackets), probably due to the prolongation of the cardiac cycle, and one can get the impression that it is the T wave followed by the U wave (although still synchronous with the monophasic T wave in lead IS). In subsequent beats, both components of the T wave (or perhaps T+U) overlap, and a few seconds later, the third tracing already shows normal T waves with a trace of a physiologic U wave following the T wave (arrow 5). Therefore, it is now clear that during tachycardia and immediately after, the split T waves are seen. However, the reason for this manifestation is unclear.

DIAGNOSTIC PROBLEM

Sudden sinus pauses

These 5 electrocardiogram tracings (FIGURE 1) were recorded during Holter monitoring at different times of the day.

A sinus rhythm is diagnosed on the basis of the presence of positive P waves in leads I and II of a standard electrocardiogram. In the present case, the sinus rhythm can be identified, although it should be remembered that leads I and II have no equivalent in Holter monitoring leads. Recurrent sinus pauses can be seen, with the longest reaching 4.4 s. However, it is difficult to determine their origin.

Tracings 1 and 2 allow us to examine whether the PP intervals seen during the pauses are a multiple of the basic PP interval during a sinus rhythm. In the first tracing, the pause is only 0.16-second longer than the 5-fold duration of the subsequent PP interval, while in the second tracing, the first pause is not the multiple of the preceding PP interval but the second pause is 2-fold longer than the PP interval. What is more, the regular rhythm of a normal frequency preceding the sudden pauses on tracings 3–5 speaks against the diagnosis of episodes of a sinus arrest, usually preceded by a gradual slowing of the rhythm. Therefore, on the basis of the available records, the diagnosis of a sinoatrial block type II seems more likely. Regardless of its pathomechanism, the presence of the pauses longer than 3 s (in the first tracing almost 4.5 s) and of the simultaneous first-degree atrioventricular (AV) block (PR intervals reaching 0.4 s) supports the diagnosis of sick sinus syndrome.

Another interesting feature is the diversity of supraventricular escape pacemakers during the pauses. In tracings 3 and 4, the escape rhythm

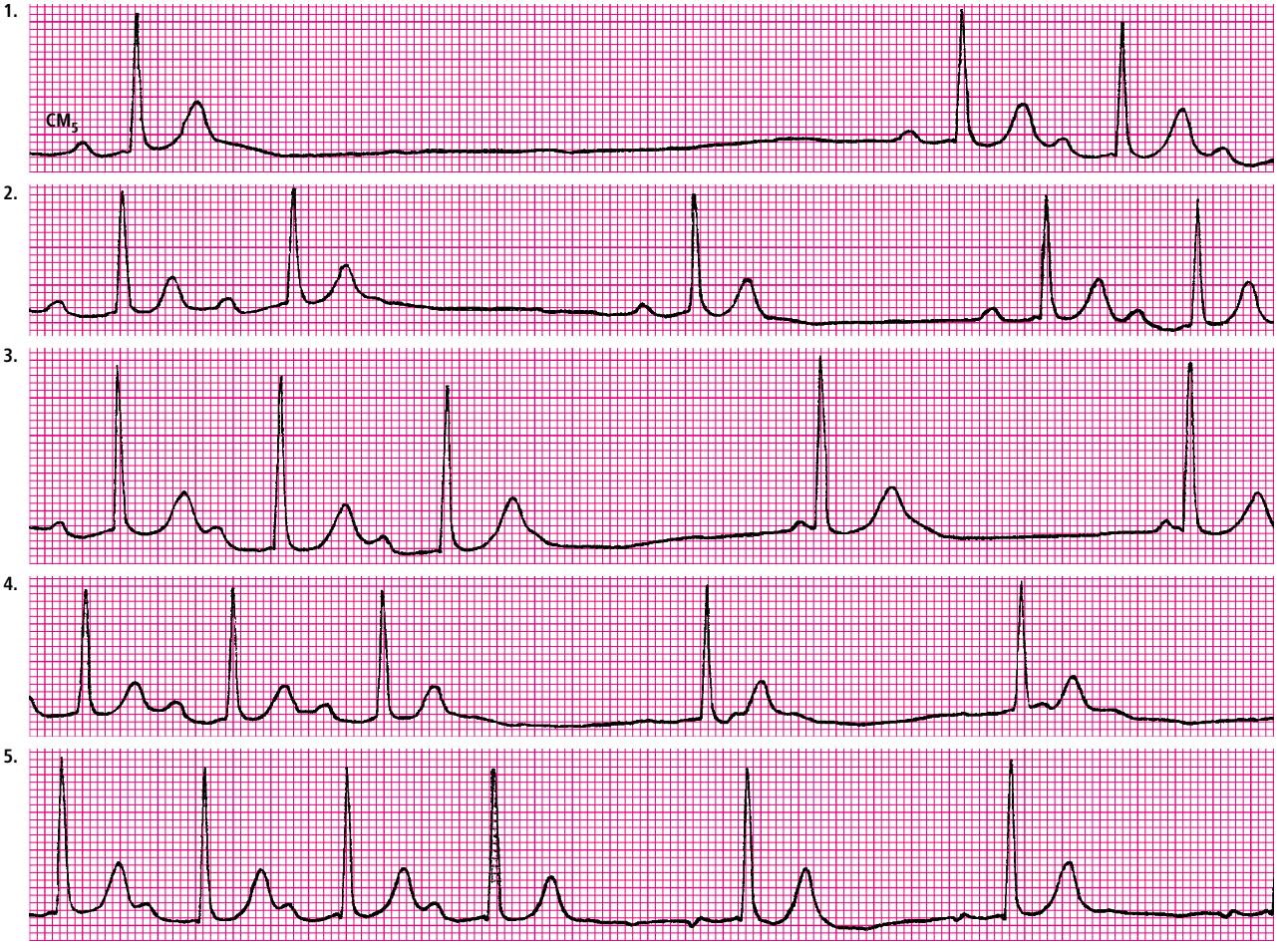

FIGURE 1

from the top of the right or the left atrium interferes with the rhythm of a similar frequency originating from the AV junction, while in tracing 5, a rhythm originating in the lower atrial area and conducted with the first-degree AV block is seen.

DIAGNOSTIC PROBLEM

Relationship between wide QRS complexes and QT interval

This electrocardiogram tracing (FIGURE 1), recorded during Holter monitoring, shows sinus tachycardia (100 bpm), seemingly disturbed by ventricular premature beats or periodic preexcitation. However, in fact, nothing disturbs this basic rhythm but the patient has periodic left bundle branch block, which is evidenced by the absence of atrioventricular dissociation and fixed PR intervals, regardless of the conduction pathway. The shape of the QRS complexes without a bundle branch block in lead CS_2 suggests that the patient had suffered anterior wall myocardial infarction (notched QS complexes). Interestingly, the Holter analyzer does not distinguish the bundle branch block from ventricular premature beats and marks all wide QRS complexes with the letter "K", thus

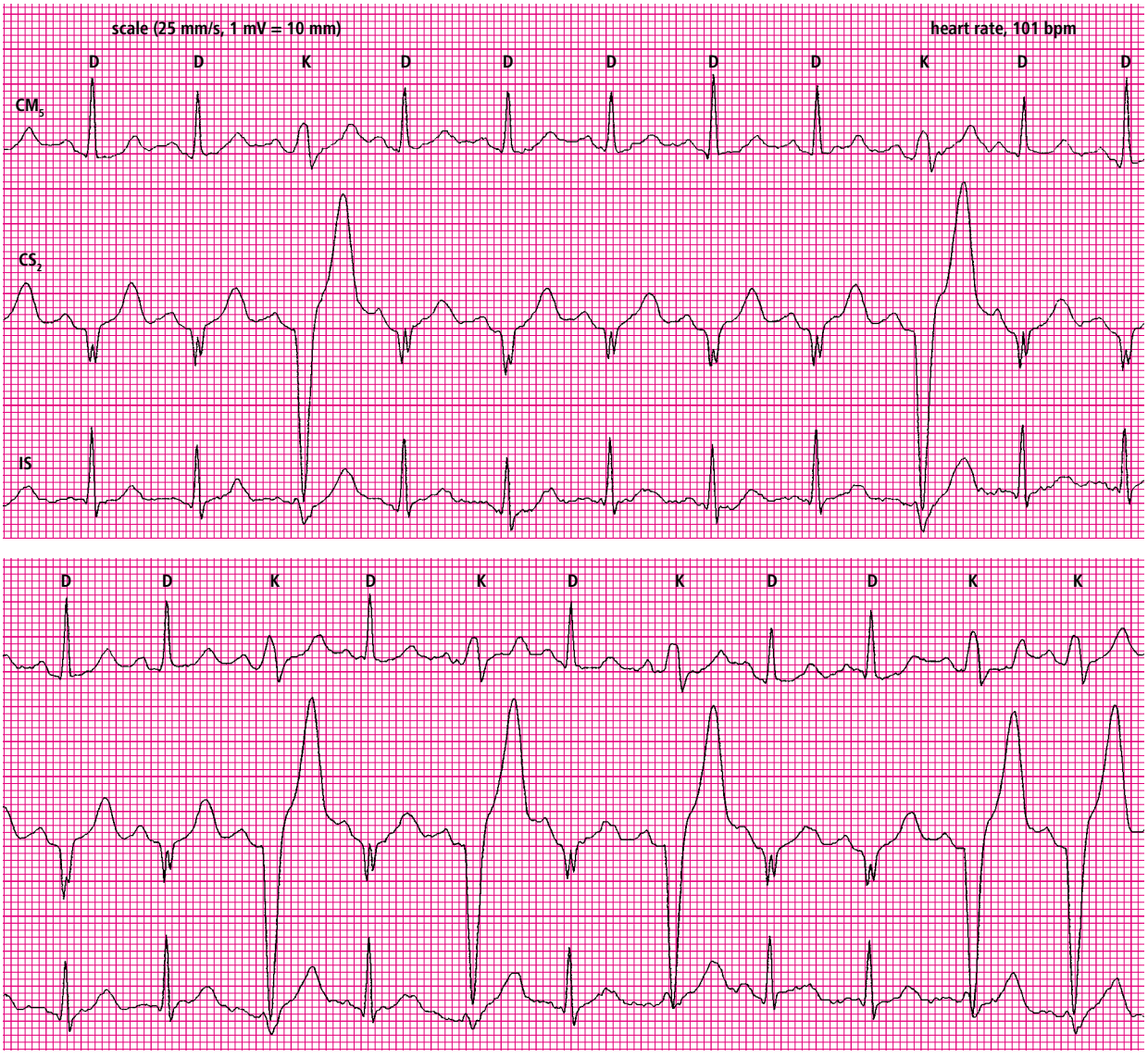

FIGURE 1

increasing the number of episodes of ventricular arrhythmia recorded during the day.

This electrocardiogram also clearly illustrates that the left bundle branch block does not necessarily prolong the QT interval. In the lower tracing, the QT interval in both conduction variants lasts 0.36 s, while in the upper tracing in 2 beats with a wide QRS complex, it reaches 0.4 s. It has been suggested that the bundle branch block may cause a minor prolongation of the QT interval (to 0.03 s in the case of left bundle branch block and shorter in the case of right bundle branch block). The question arises as to why the QT interval sometimes remains unchanged, although in the case of left bundle branch block there is undoubtedly a significant delay in left ventricular depolarization. Experimental studies demonstrated that delayed depolarization, whether caused by a block or an artificial stimulation of the ventricles, reduces the duration of an action potential in the fibers showing abnormal depolarization, as reflected by the shortening of the JT interval. The shortening is most pronounced in the left ventricle and can reach up to 0.03 s. Therefore, the final change of the QT interval is either minor or undetectable.

DIAGNOSTIC PROBLEM

Effect of quinidine treatment on electrocardiogram

This electrocardiogram (FIGURE 1) was recorded in the 1970s, on the 3rd day of quinidine treatment in a 45-year-old man with hypertension, severe left ventricular hypertrophy, long QT interval (520 ms), and atrial premature beats (misdiagnosed as ventricular premature beats). The patient was administered quinidine, which at that time was considered the treatment of choice for premature beats and its effects on the QT interval and the risk of ventricular tachyarrhythmia were yet unknown.

First, the QT interval should be measured. It cannot be measured in leads I, II, and V_5 because it is difficult to determine the end of the T waves. In leads III, V_1, and V_6, the QT interval lasts 520 ms, and in lead aVL, 600 ms. In leads V_2 and V_3, the T wave is split and a large U wave superimposes on its descending arm. Therefore, the measurement has to be made by plotting the tangent to the descending arm of the T wave (after the point where it is split) and measuring the QT interval to the intersection of the tangent with the line marked out by the TP segments. In both these leads, the QT interval is 620 ms.

Now, the QT interval should be corrected for heart rate (50 bpm). TABLE 1 presents the results of the correction obtained using exponential regression according to the Bazett formula compared with those obtained using linear regression according to the Hodges formula. The Bazett formula is too common to be presented here, while the Hodges formula (one of the numerous formulas based on linear regression) is as follows: corrected QT = QT interval [ms] + 1.75 × (HR-60), where HR is the heart rate per minute. If the heart rate is below 60 bpm (in this case 54 bpm), the product of the difference of the 2 heart rates is multiplied by 1.75 and subtracted from the measured QT interval.

TABLE 1 shows that the Bazett formula significantly reduces the real value of the QT interval if the heart rate is below 60 bpm (the opposite happens in the case of higher heart rates, and particularly in the case of tachycardia exceeding 90 bpm). If we were to use this formula, we would conclude that quinidine increased the degree of QT-interval prolongation by less than 60 ms compared with its duration before treatment. According to the 2005 US Food and Drug Administration guidelines for the pharmaceutical industry, such a value indicates a necessity to discontinue a drug. The longest QT interval corrected by the Hodges formula exceeds the reference value recorded 3 days earlier by as much as 83 ms. Moreover, the duration of the QT interval in individual leads indicates their wide dispersion, namely, 100 ms. It is a feature independent of significant QTc prolongation, indicating the risk of malignant ventricular tachyarrhythmia.

The present case corroborates the 2009 guidelines of American cardiac societies, which recommended the use of linear regression methods to correct the QT interval for heart rates and suggested the following upper reference ranges for the corrected QT interval: 450 ms for men and 460 ms for women.

However, the Bazett formula has a single advantage over the linear regression methods. QT prolongation measured by this method better reflects the risk of cardiac death than that measured using linear regression. The reason for this advantage is simple: QT-interval prolongation in the Bazett method is related not only to the actual duration of the QT interval but also to the accelerated heart rate, and it is well known that the higher the resting heart rate, the higher the risk of cardiac death (regardless of the presence of other risk factors). Yet it is obviously better to evaluate

TABLE 1

Leads	Measured QT interval, ms	Bazett correction	Hodges correction
III, V_1, and V_6	520	475	503
aVL	600	548	583
V_2 and V_3	620	566	603

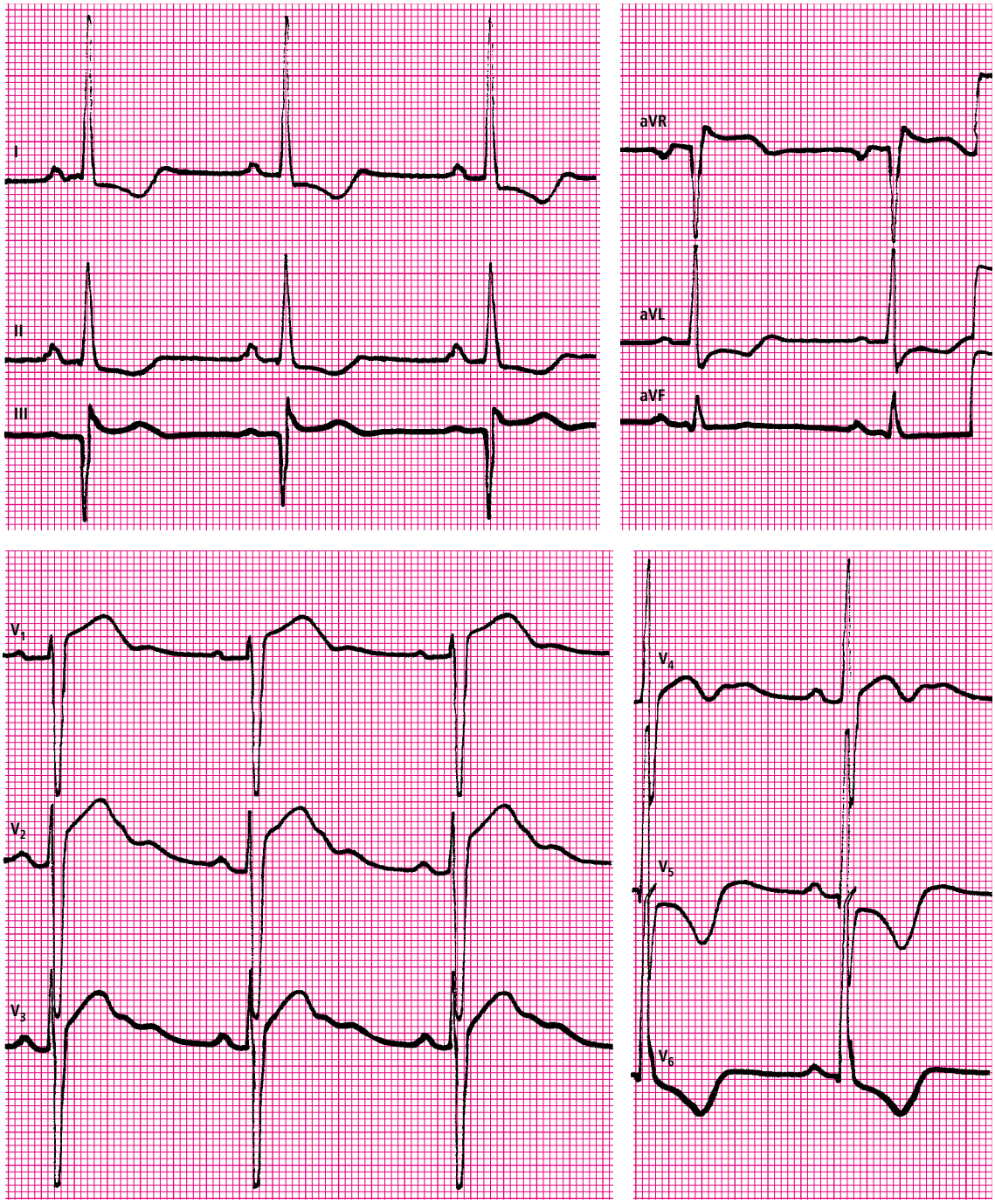

FIGURE 1

this risk not using one common formula, which does not reveal the significance of each individual parameter, but rather independently—by precisely correcting the QT interval for the heart rate and separately evaluating the clinical significance of heart rates.

DIAGNOSTIC PROBLEM

Problems with identification of electrocardiographic features of left ventricular hypertrophy

FIGURE 1 shows an electrocardiogram (ECG) recorded in a 61-year-old man with atypical angina. In this description, the ECG will be interpreted according to the 2010 Polish Cardiac Society guidelines, and the results will be compared with the 2007 and 2009 guidelines of the American Heart Association, American College of Cardiology, and Heart Rhythm Society (AHA/ACC/HRS).

A slow regular sinus rhythm (58 bpm), normal electrical axis of the heart in the frontal plane, and rotation of the heart to the left (counterclockwise in the apical view; R = S between leads V_1 and V_2) can be identified.

The P waves are wide (0.11 s), although still within the reference range, and they are also slightly "notched". The dimensions of a negative phase of the P wave in lead V_1 are also within the reference range. Therefore, it would not be wrong to consider these waves as normal.

The QRS complexes are of normal duration, but the interpretation of their amplitude is problematic. They do not meet any of the criteria of left ventricular hypertrophy, recommended by the Polish Cardiac Society (R-wave amplitude in $V_{5/6}$ <26 mm; the sum of $SV_1 + RV_{5/6}$ <35 mm; and the sum of SV_3 + RaVL <28 mm), while they fulfill some of the 36 criteria listed in the AHA/ACC/HRS guidelines: the sum of R + S exceeding 35 mm in any precordial lead (in this case, 40 mm in lead V_3) and the R wave exceeding 26 mm in any precordial lead (in this case, 32 mm in lead V_3). Thus, the diagnosis of left ventricular hypertrophy is not unequivocal and will depend on what guidelines are used as reference, if any. If hypertrophy was diagnosed, then severe abnormalities in ST-T would be identified as related with the hypertrophy and they would be described either in a standard way (although not in accordance with the guidelines) as the features of left ventricular overload, or in accordance with all of the above guidelines as the features of left ventricular hypertrophy with secondary ST-T abnormalities. However, it is important to note that ST-T abnormalities associated with ventricular hypertrophy can be either secondary (resulting from abnormal ventricular depolarization) or primary (associated with myocardial ischemia). This is also emphasized by the AHA/ACC/HRS guidelines. In the present case, the extent of ST-T abnormalities may raise some doubts as to whether they are associated with left ventricular hypertrophy only. Yet the counterclockwise heart rotation makes the QRS complexes in leads V_2 and V_3 seem to correspond with the left ventricular complexes. However, if we do not diagnose left ventricular hypertrophy, the only possible diagnosis left is the features of myocardial ischemia, owing to the presence of negative T waves with significant ST-segment depression.

As for the counterclockwise heart rotation, it has recently become popular to describe such a feature as "prominent anterior QRS forces". Both these terms describe the presence of R>S--type QRS complexes in leads V_1 to V_3. Moreover, those who identify prominent anterior QRS forces interpret them as a feature of left septal fascicular block, but they have no proof for the existence of such a relationship beyond the imagined course of depolarization. Meanwhile, such a picture is quite common and is seen not only in various diseases but also in healthy individuals. The R>S-type QRS complexes in lead V_1 may be present in patients with right bundle branch block, ventricular preexcitation syndrome type A, right ventricular hypertrophy, and inferoposterior myocardial infarction. When R>S-type QRS complexes are present only in leads V_2 and V_3, the following conditions might be considered: heart displacement due to chest distortion or lung disease, congenital heart disease with hypertrophy of both ventricles, and sometimes also transient disturbances of intraventricular conduction of unknown origin (these conduction disturbances also tend to cause the opposite configuration of the QS complexes in these leads, which is similarly transient and unrelated to myocardial infarction). Finally, considering the variable location of the septal fascicle, it can be assumed that patients with a dominant posterior fascicle that sends its branches to the septum may show

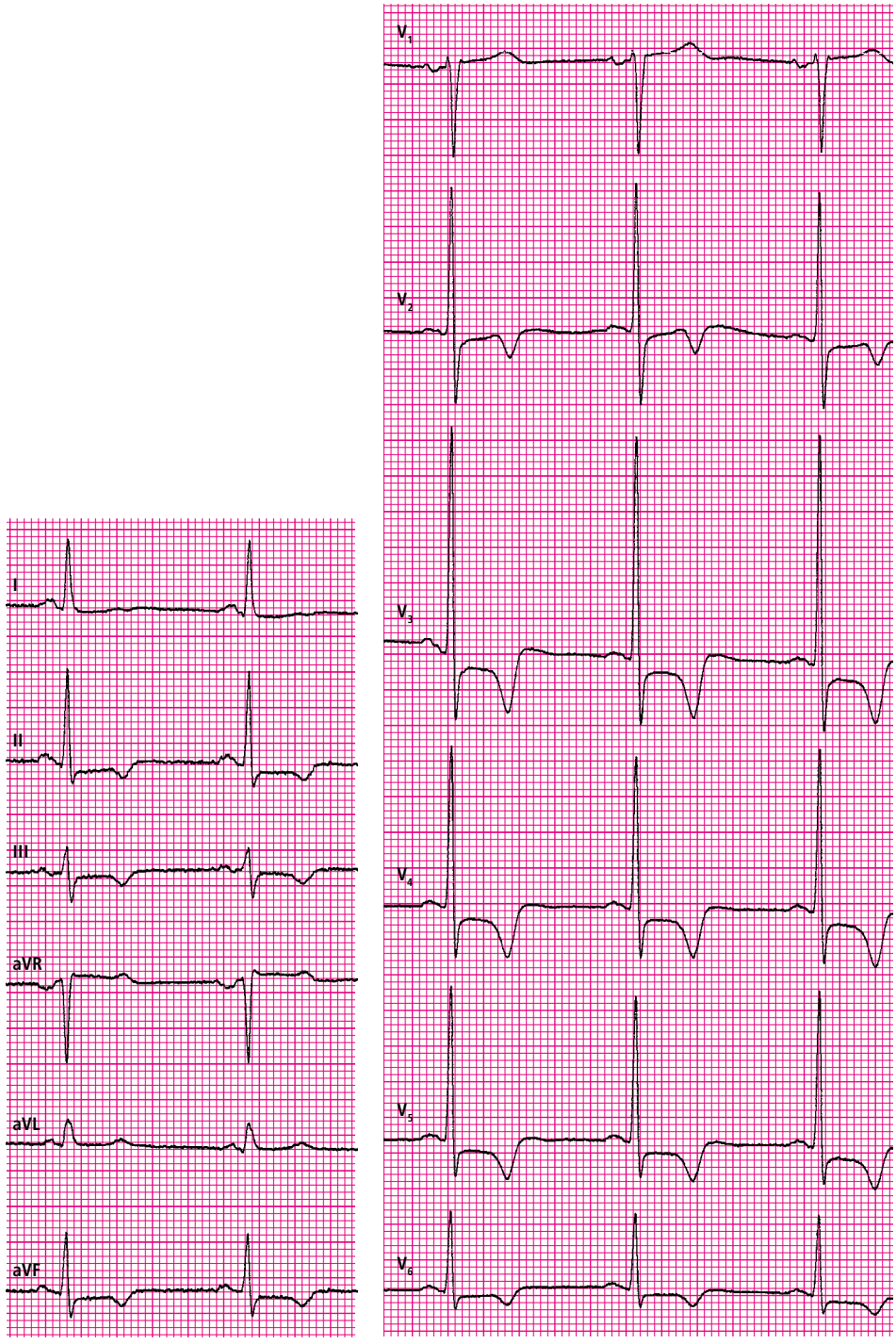

FIGURE 1

a physiological depolarization of the interventricular septum that manifests with the presence of R>S-type QRS complexes, instead of a more common depolarization from the left to the right side of the septum, which is manifested on an ECG by small Q waves in leads I and $V_{5/6}$, called "septal Q waves". Thus, counterclockwise heart rotation with R>S-type QRS complexes in lead V_3 or in leads V_2 and V_3 is a nonspecific feature and usually requires no further diagnostic workup.

DIAGNOSTIC PROBLEM

Atrial flutter with a questionable left bundle branch block

In the limb leads of this electrocardiogram (FIGURE 1), only in leads I and aVL small QRS complexes can be seen, while in the remaining limb leads these complexes are embedded in the flutter waves (the rate of approximately 280 per minute). A small amplitude of the QRS complexes is not specific for any particular disease. It can be seen in patients with pericarditis (both exudative and constrictive) and in various other conditions associated with large pericardial effusion (not always inflammatory); in cardiomyopathy, mainly restrictive (eg, amyloidosis) but also dilated (including inflammatory and ischemic); sometimes in chronic or acute cor pulmonale (in the case of chronic disease, it is related to obstructive pulmonary disease); and occasionally also in healthy individuals (in whom it results from a specific vector of ventricular depolarization, perpendicular to each axis in the frontal plane).

So what are other possible clues to an underlying disease in this electrocardiogram? Of note, the precordial leads show moderate nonspecific features of clockwise heart rotation and focal disturbances of intraventricular conduction (notched QRS complexes in leads V_4 and V_5). The notches in lead V_5, only slightly visible also in lead V_6, as well as an increase in the time to peak R in these leads to more than 0.06 s raise the suspicion of incomplete left bundle branch block. The T waves in these leads, which are opposed to the QRS complexes, may in part result from the negative phase of the F waves that superimpose on the T waves (they can also be seen outside the T waves within the longer RR intervals). However, since

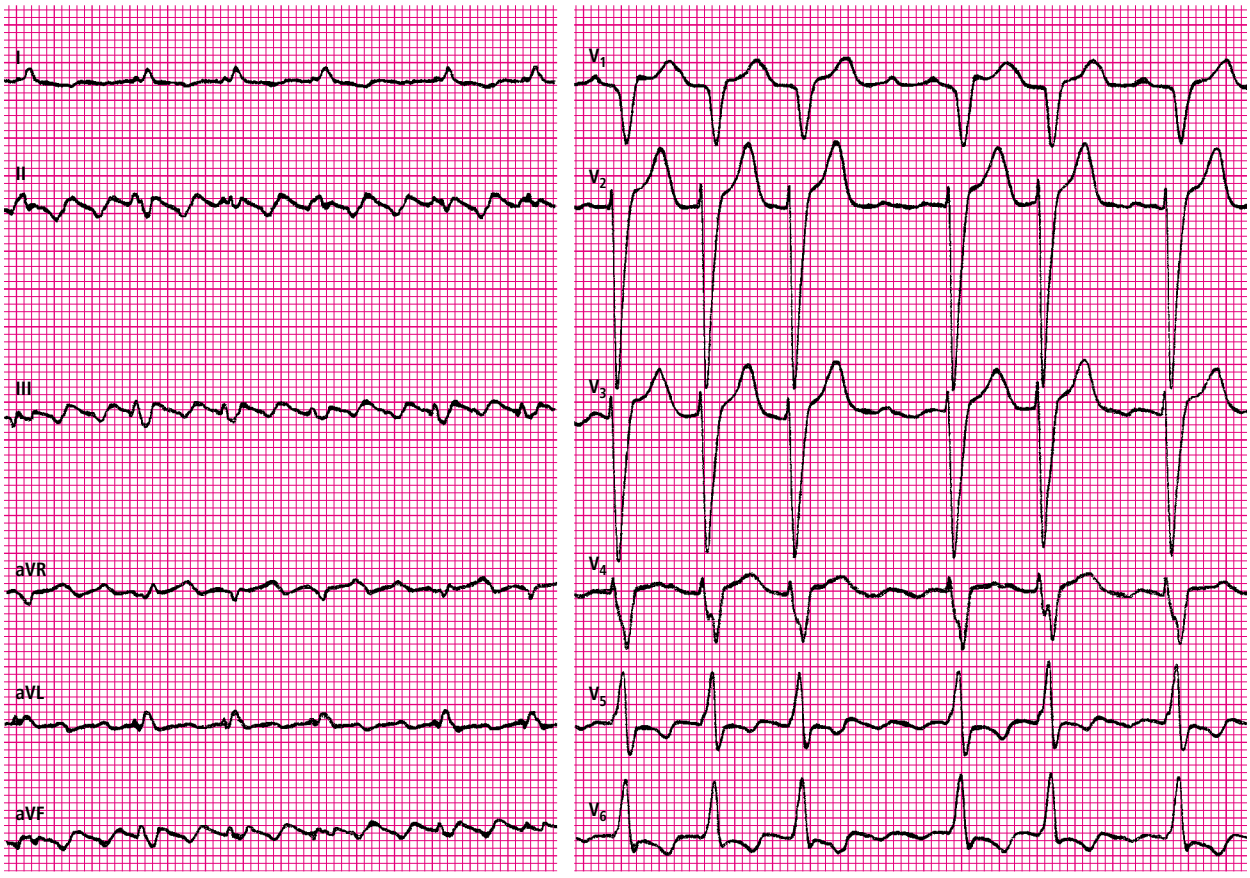

FIGURE 1

the duration of the QRS complexes is 0.12 s, complete left bundle branch block is the only possible diagnosis according to arbitrary expert criteria. Such diagnosis will be in line with the current criteria (American Heart Association / American College of Cardiology / Heart Rhythm Society, 2009), although in fact, it may not reflect the actual status. This is because despite the current criteria, numerous reports have shown that the duration of QRS complexes in complete bundle branch block exceeds 0.12 s. Nevertheless, this issue is of little practical importance, and moreover, it has not been clearly resolved to date.

CLINICAL INTERPRETATION

An apparent myocardial infarction in a woman with left bundle branch block

This simple electrocardiogram (FIGURE 1) was recorded at a speed of 50 mm/s in a 75-year-old woman with hypertension, who was admitted to the hospital with symptoms of transient ischemic attack. The patient was informed that she had suffered myocardial infarction in the past, which she was unaware of. Apart from the diagnosis of left bundle branch block, a physician included the following information in the description of the electrocardiogram: "the lack of R-wave progression in leads V_1 to V_4 indicates prior anterior wall myocardial infarction." However, the lack of R-wave progression in leads V_1 to V_4 is one of the characteristic features of the left bundle branch block. Moreover, it is quite common to see QS complexes in leads V_1 to V_3 in patients with this type of a block, and in this setting they also do not indicate a myocardial infarction. In patients with left bundle branch block, anterior wall myocardial infarction can be identified only when QS complexes are seen up to lead V_4, or when wide Q waves are present in leads V_5 or V_6 (or both).

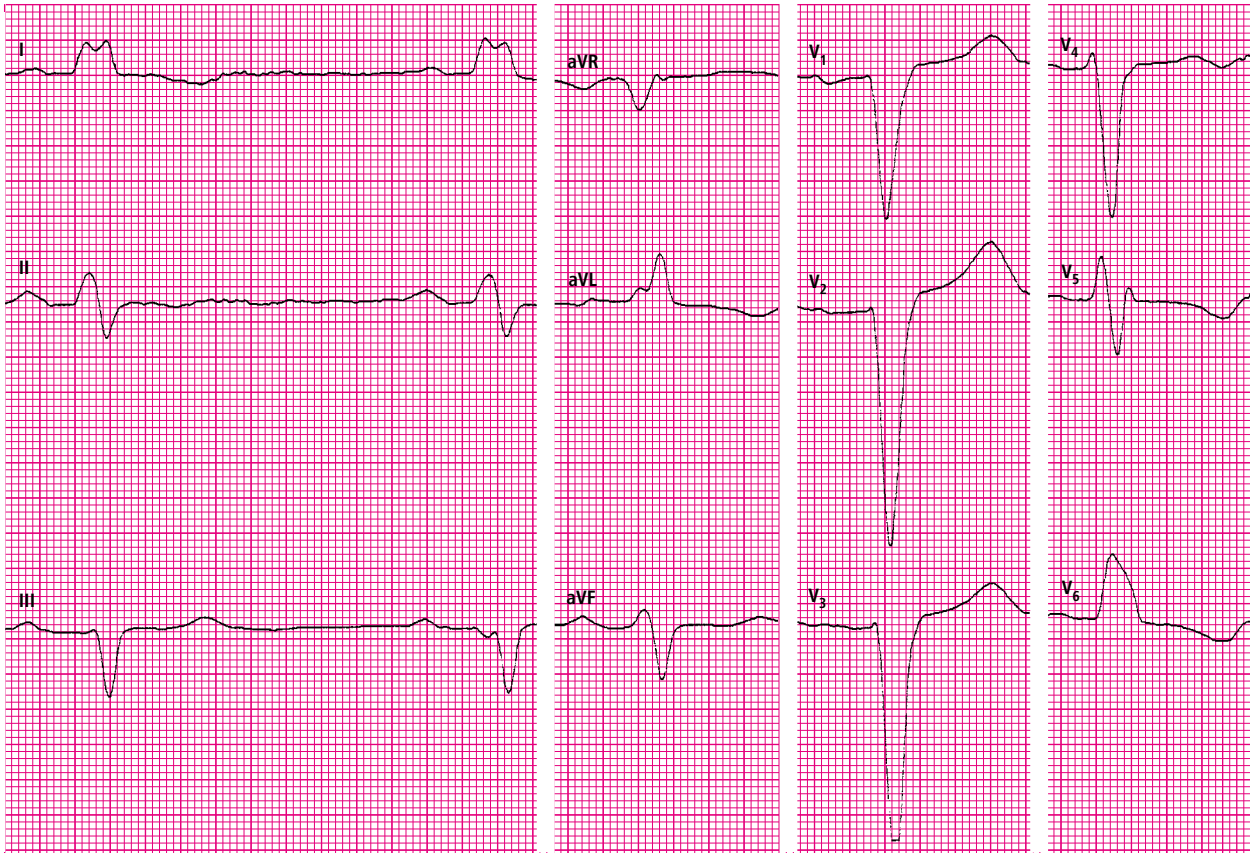

FIGURE 1

An 18-year-old man with muscular dystrophy

These 2 electrocardiograms (ECGs) were recorded within 1 month in a young man with Emery–Dreifuss muscular dystrophy.

The first ECG (FIGURE 1) shows atrial tachycardia with a rate of the P waves of 180 bpm, complete atrioventricular (AV) block, and an escape rhythm from the AV junction with a heart rate of 45 bpm. Although the ratio of the atrial and ventricular rates and the shape of the P waves and QRS complex in leads I to III may suggest otherwise, this is not a second-degree AV block (4:1). The variable PQ intervals in the subsequent leads, sometimes very short, indicate that the atrial stimuli are not the source of the QRS complexes that occur immediately after them.

The second ECG (FIGURE 2) shows only the rhythm from the AV junction with a heart rate of 37 to 45 bpm, with no visible P waves. In both

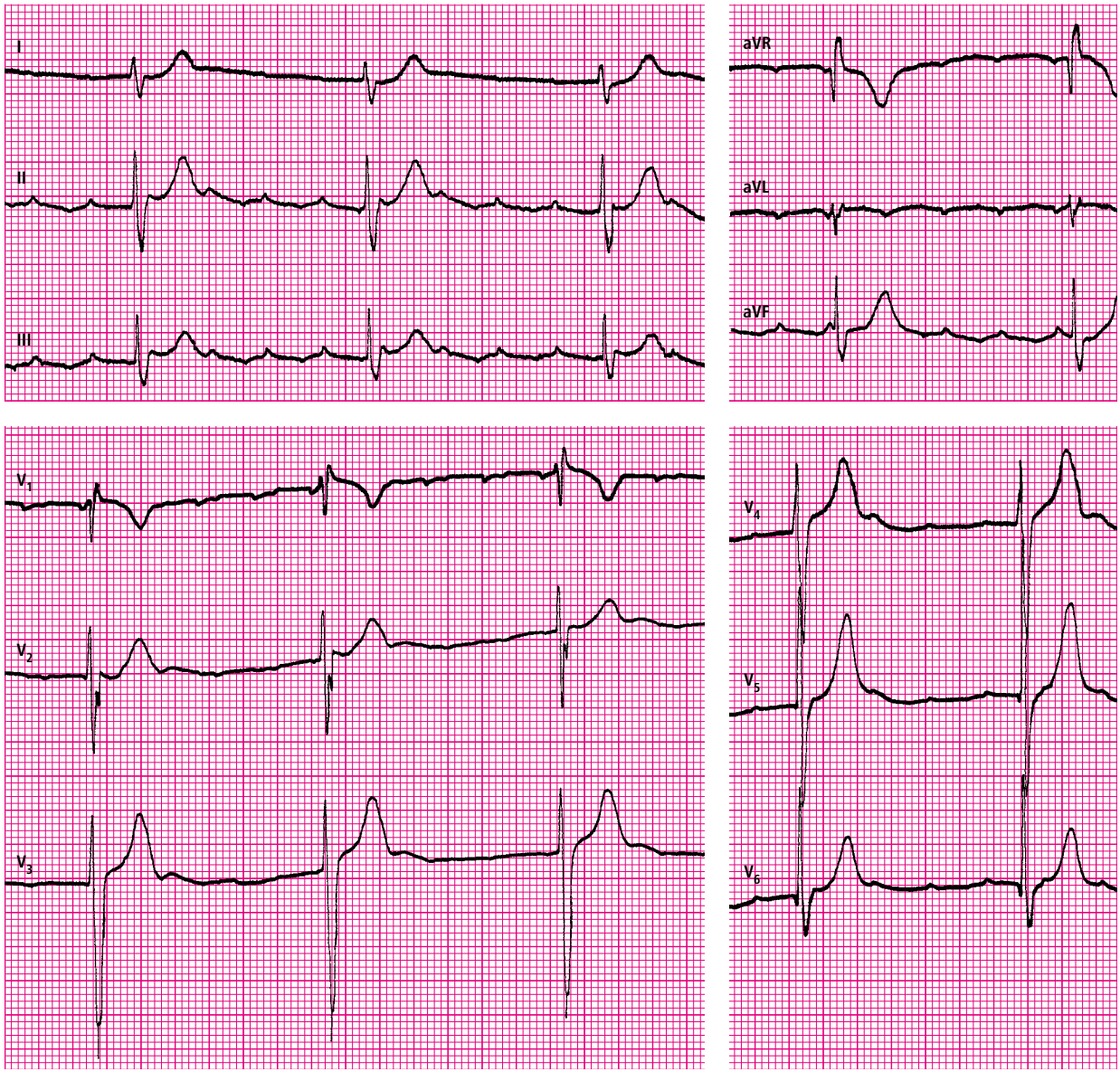

FIGURE 1

ECGs, the QRS complexes have a similar shape: in the form of $S_IS_{II}S_{III}$ with incomplete right bundle branch block.

This rare hereditary muscular dystrophy damages skeletal muscles to a lesser extent (and only in men), while in all patients (both men and women) with the genotype of the disease, it leads to progressive cardiomyopathy, first affecting the atrial muscle (with atrial conduction system) and then the ventricular muscle. Characteristic types of arrhythmia include sinus arrest, AV block (usually first-degree), episodes of atrial fibrillation or atrial flutter, and atrial arrest, a condition in which atrial muscle fibers lose their excitability and do not produce action potential, and therefore do not contract under stimulation. Such a possibility may be considered when interpreting the origin of the rhythm from the AV junction in the second ECG.

The shape of the QRS complexes deserves attention. The $S_IS_{II}S_{III}$ complex with S>R deflections is usually found in patients with cor pulmonale or extensive myocardial infarction, sometimes also in patients with hypertrophy of both the left and right ventricles and in a rare type of intraventricular conduction disorder with right superior axis deviation of unknown pathophysiology. In this case, the results of the echocardiogram were (yet) completely normal, and in the present ECG, the amplitudes of the R and S waves were more or less the same. Therefore, the cardiac axis should be considered indeterminate, and together with the presence of the incomplete right bundle branch block, it should be regarded

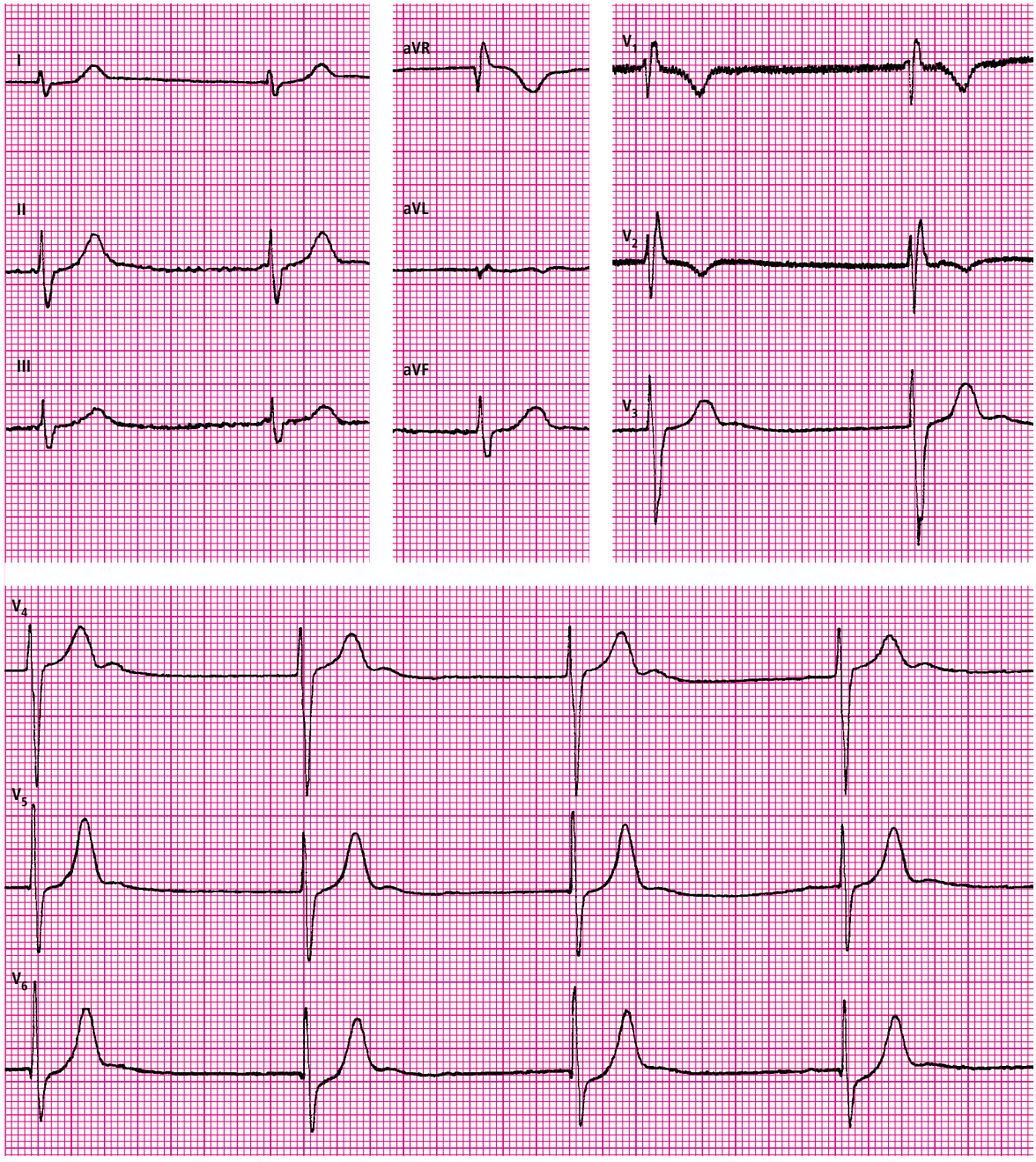

FIGURE 2

as an early sign of intraventricular conduction system damage.

Because of the risk of a complete AV block (not only during rapid atrial tachycardia) and its spread to the intraventricular conduction system, as well as because of a deteriorating pacemaker function, patients with Emery–Dreifuss muscular dystrophy usually require implantation of VVI pacemakers.

A 58-year-old woman with a history of 2 myocardial infarctions

This electrocardiogram (ECG, FIGURE 1), recorded at a speed of 50 mm/s, shows a regular sinus rhythm (84 bpm) and a minor upsloping ST-segment depression in leads V_4 to V_6, which has no clinical significance.

The ECG was recorded in a woman who, a few years earlier, suffered anterolateral wall myocardial infarction without an abnormal Q wave, complicated by sudden cardiac arrest (an ECG showed ventricular tachycardia, followed by asystole, and then by transient third-degree atrioventricular block). After these events, a cardiac team decided to perform percutaneous coronary intervention to dilate the right coronary artery, which was the only occluded vessel at that time. The intervention caused an arterial spasm, which led to another myocardial infarction, this time affecting the inferior wall. The patient survived and

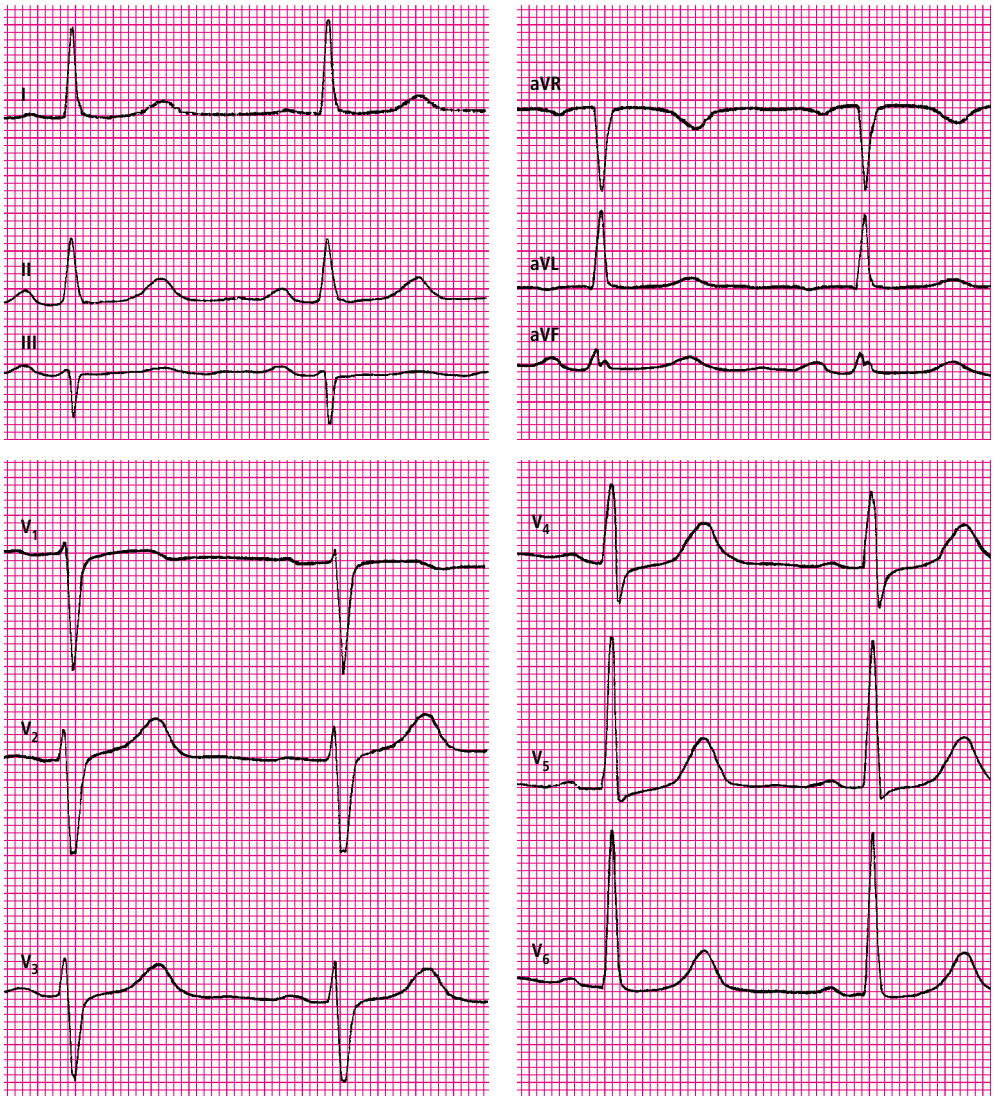

FIGURE 1

remained in a good general condition, except for mild hypertension and hypercholesterolemia.

The present case shows that normal ECG findings do not exclude heart disease, such as life-threatening Prinzmetal angina seen in this patient.

A 92-year-old man with critical aortic stenosis

The greatest challenge in this routine electrocardiogram is to determine the origin of a heart rhythm of 115 bpm. It is only in the precordial leads that the P waves imitating U waves (arrows 1) with a PR interval of 0.26 s can be seen; transferring this time interval to leads I to III explains that the P waves are hidden in the T waves and begin to appear only in the last 2 beats (arrows 2),

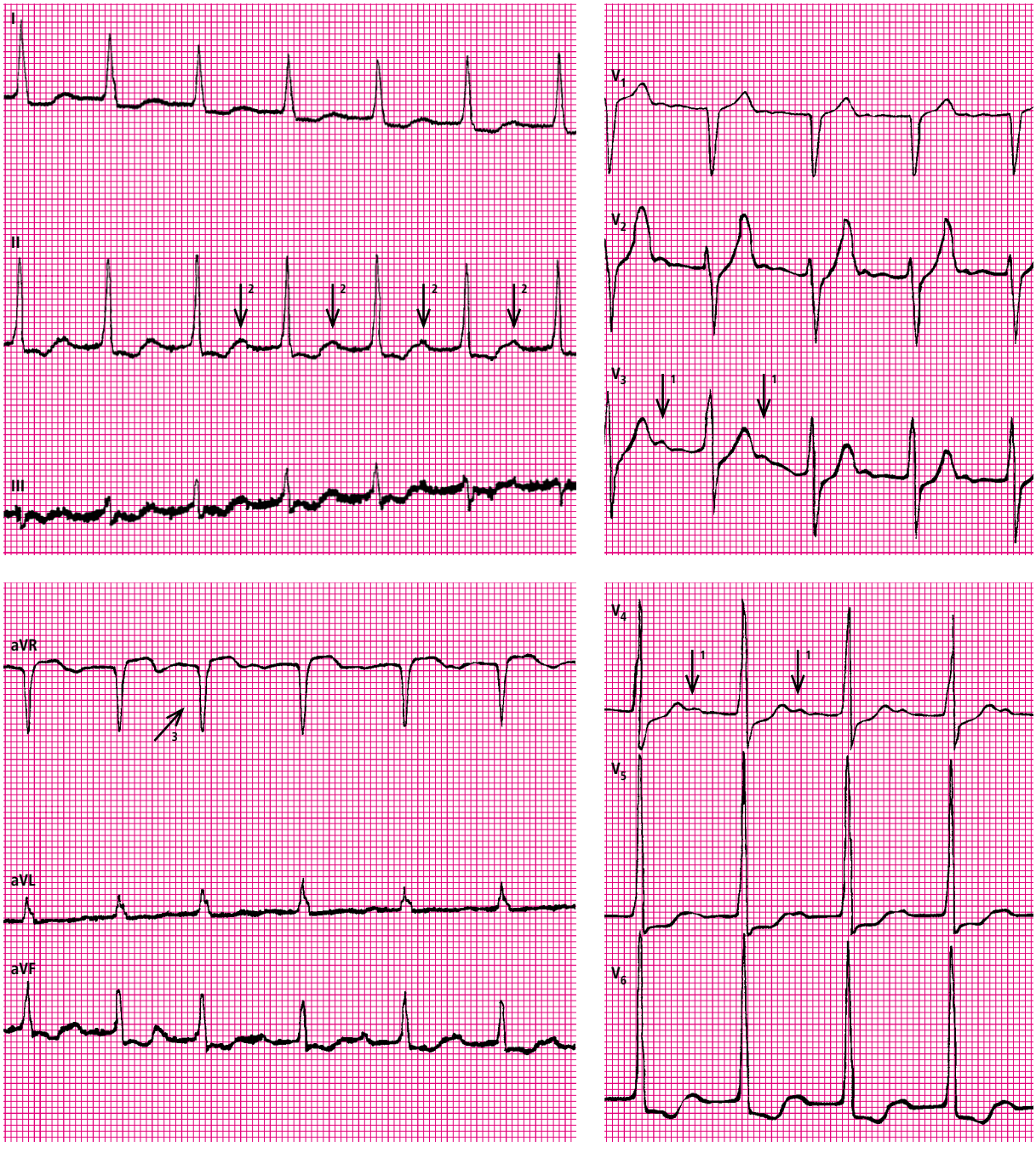

FIGURE 1

during which the prolongation of the RR intervals from the initial 520 ms to 560 ms occurs (and thus the heart rate decreases to 104 bpm). Of note, in leads aVR, aVL, and aVF, the rhythm after a single premature atrial beat (arrow 3) transiently decreases to 96 bpm. Therefore, accelerated sinus rhythm with a single premature atrial beat and first-degree atrioventricular block can be diagnosed.

In addition, downsloping or horizontal ST-segment depression indicates myocardial ischemia, and the R-wave amplitude in leads V_5 and V_6 exceeding 26 mm suggests that this ischemia is probably related to left ventricular overload.

Although sinus tachycardia and features of myocardial ischemia in the elderly indicate a serious heart disease, this presentation does not fully reflect the actual condition of the patient, who had hypertension and critical aortic stenosis (gradient, 108 mmHg; left ventricular wall thickness, up to 18 mm; ejection fraction, 26%), diabetes, and a history of myocardial infarction with pulmonary edema that occurred 2 years earlier. Currently, the patient is in a good general condition; his heart rate is normal and features of ischemia have decreased significantly. This electrocardiogram might be classified as a case of electrocardiographic dissimulation.

Palpitations in a 50-year-old man

FIGURE 1 presents an interesting series of electrocardiograms (ECG) recorded during Holter monitoring in a man with hypertension and periodic palpitations, significant concentric left ventricular hypertrophy (wall thickness, 14 mm), without segmental wall motion abnormalities, and with a negative result of the stress test during which the patient experienced numerous ventricular premature beats, including couplets. The leads are marked as A, B, and C because their origin is unknown. Because the tracings from leads A and C are almost identical, all 3 leads are presented only once. Lead B, unfortunately, was recorded over an area with very small amplitudes of deflections. In tracings 1 to 3, recorded during sleep, bradycardia of up to 30 bpm (tracing 3) was interrupted by episodes of atrial tachycardia of 120 to 130 bpm and, interestingly, with a variable shape of the P waves. With only 2 or 3 bipolar precordial leads used for Holter monitoring, it is difficult to tell what happened in the limb leads at that time. There is insufficient evidence to suspect that it was a chaotic atrial rhythm, that is, multifocal atrial tachycardia (in such a case, we would see occasional beats from the atrioventricular junction with inverted P waves in the leads representing the vertical axis in the frontal plane). Therefore, it is hard to tell whether the variable shape of the P waves indicates intraatrial conduction disturbances or the beats originating from multiple sites. The diagnosis of aberrant intraatrial conduction is supported by features of intraatrial conduction disturbances during the basic heart rhythm, which can be treated as sinus rhythm (with wide and split P waves [arrows 1]). These features, together with the presence of atrial arrhythmia, indicate an underlying condition that may increase the risk of atrial fibrillation.

Apart from short episodes of atrial tachycardia, also occasional single atrial premature beats and episodes of sinus bradycardia were recorded at night. On the other hand, during the day, episodes of parasystolic ventricular rhythm were seen, with heart rates similar to those of the current sinus rhythm, namely, 70 to 80 bpm (tracing 4, recorded at 7:40 PM).

This ECG shows an interesting feature: severe disturbances of ventricular repolarization after resolution of the consecutive episodes of atrial tachycardia, manifesting as a transient significant increase in the amplitude and duration of the U waves (arrows 2) and prolongation of the Q(T + U) interval to about 600 ms. While numerous studies have indicated that postextrasystolic ST-T features (including those seemingly ischemic) are not associated with unfavorable prognosis, it has also been reported (although not yet confirmed) that an increase in the amplitude of a postextrasystolic U wave is a poor prognostic factor. In conclusion, the only abnormality in this ECG series that can be considered as mild and without clinical significance is the parasystolic ventricular rhythm.

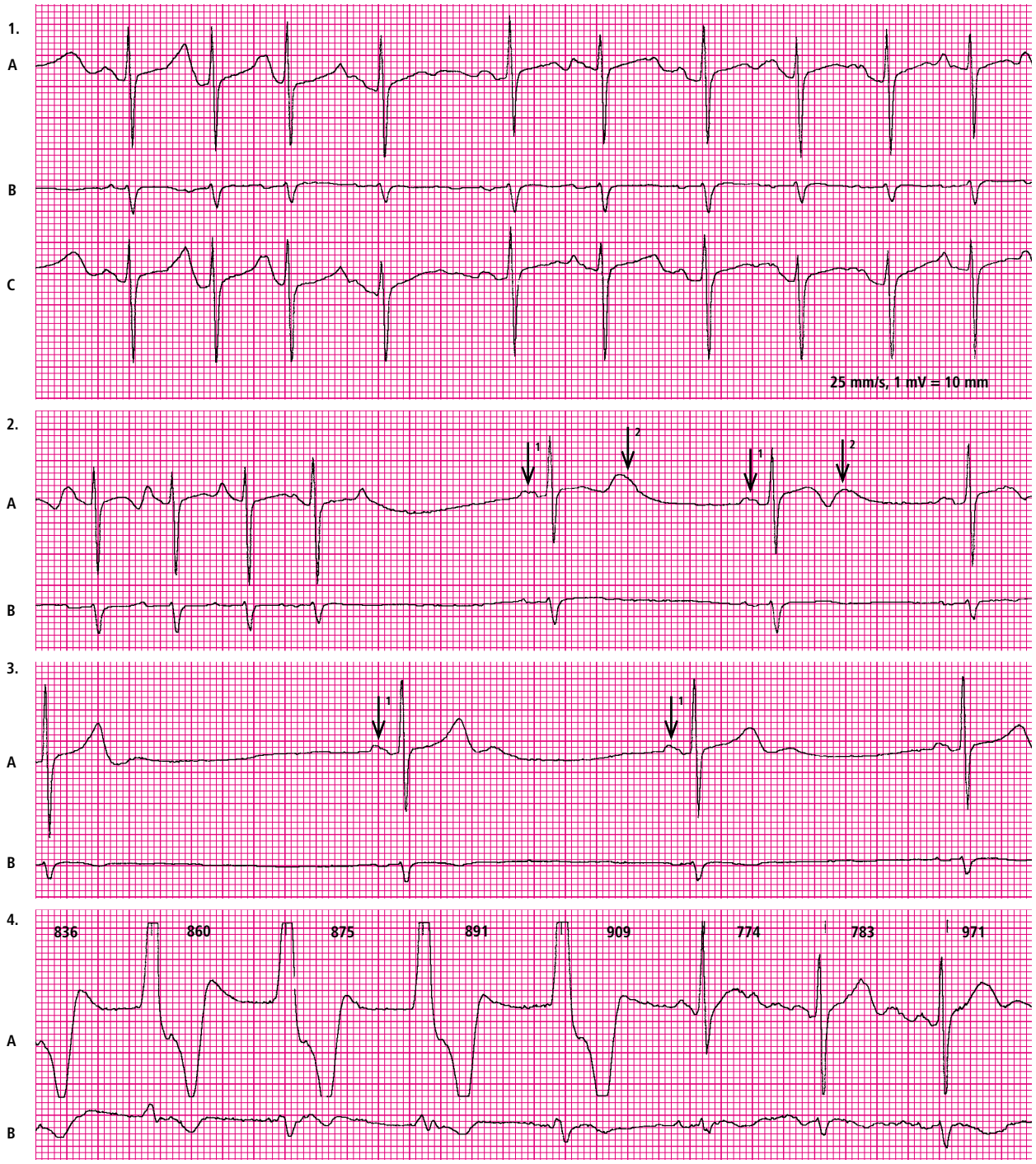
1.
A
B
C
25 mm/s, 1 mV = 10 mm
2.
A
B
1
2
1
2
3.
A
B
1
1
4.
A
B
836
860
875
891
909
774
783
971

FIGURE 1

Exhaust fume poisoning in a 25-year-old soldier

At the end of September 2007, a professional soldier presented to an emergency room (ER) after a short-term loss of consciousness, which he suffered for the first time in his life during a training shooting from a tank. On the spot, it was assumed that he was poisoned with exhaust gases and he was given oxygen; in the hospital, he complained of nausea, headache, and dry cough. A physical examination and laboratory tests revealed no significant abnormalities. Based on data from interviews and an electrocardiogram (ECG), Brugada syndrome was suspected and the patient was transferred to a tertiary referral military hospital. As no abnormalities were detected, the patient was sent back to his military unit.

The ECG tracing in FIGURE 1 was recorded on the first day in the ER (at a speed of 25 mm/s, 1 mV = 10 mm). It shows a regular and fast sinus rhythm (112 bpm). The PR interval is prolonged (0.24 s), the QTc interval corrected by the Hodges method is 0.41 s, and the duration of the QRS complexes in leads II and III is 0.12 s. As for the QRS complex in the precordial leads, first it has to be determined where the R and S waves end. But before that, please take a closer look at Q waves, whose duration (0.05 s) and depth in leads V_1 and V_2 are quite surprising In lead V_3, a residual R wave can already be identified at the beginning of the QRS complex, and thus the subsequent negative wave is the S wave. After Q waves in leads V_1 and V_2, we would typically recognize R waves, and after S waves in lead V_3—R' waves, but from the top they form arches followed by the T waves, with concave ST segments in leads V_3 and V_4 and with a downsloping depression in leads V_1 and V_2. Therefore, as we classify them as J waves rather than R waves, this presentation meets the criteria of type II Brugada syndrome (not type I, because ST segments are not followed by negative T waves; unfortunately, the quality of the tracing from leads V_1 to V_2 is suboptimal, particularly with respect to P waves). All these abnormal ECG features resolved on day 6 of hospitalization (FIGURE 2): the sinus rhythm was no longer accelerated (range, 75–82 bpm), the PR intervals were normal (0.18 s), the QRS complexes had no pathological Q waves and their duration did not exceed 0.1 s, and finally, the changes in the ST-T segment in the precordial leads corresponded to the physiological early repolarization syndrome.

How to interpret these 2 ECGs? In the case of the first ECG, the least likely option (though often seen) is a technical error made during the recording by placing the precordial leads about 1 or 2 intercostal spaces higher than necessary. However, we would then expect narrow and small Q waves or QS complexes, not Qr complexes. Moreover,

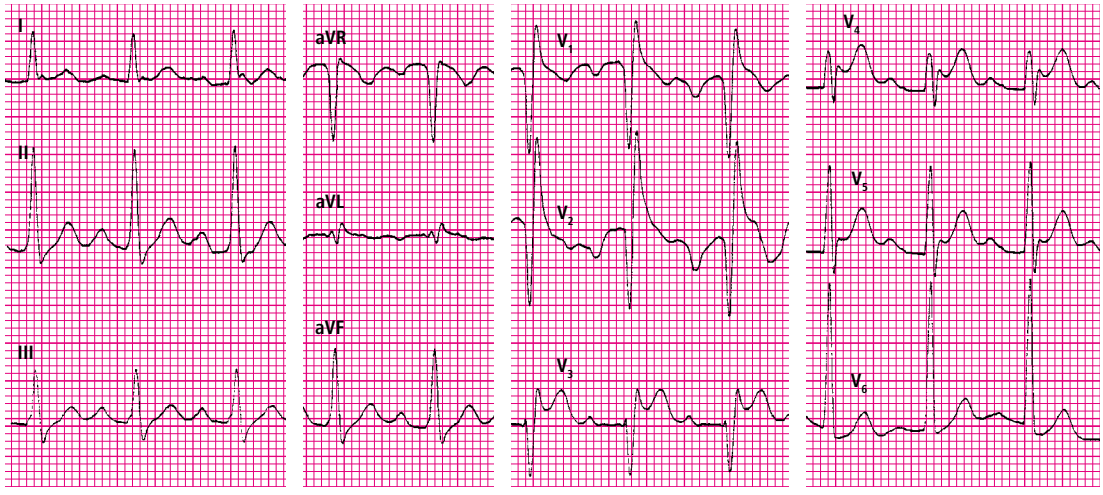

FIGURE 1

this would not explain the wide QRS complexes. Therefore, the suspicion of Brugada syndrome was justified in this case, and before excluding such a diagnosis, we would have to make sure that the patient had no family history of sudden cardiac death and confirm a negative result of sodium channel blocker challenge. In case of doubt, we should consider whether the patient might have been exposed not only to exhaust gases, but also to high ambient temperature during the shooting. There have been reports that high temperatures may trigger or exacerbate ECG abnormalities consistent with Brugada syndrome. However, this is not all. Regardless of the etiology of changes in the ventricular repolarization phase, both the loss of consciousness and complaints after recovery as well as the presence of deep and wide Q waves on the first ECG suggest an important role of the poisoning from exhaust fumes, which contained mainly carbon monoxide but also lead dioxide. As the Q waves quickly subsided and the enzyme markers for myocardial infarction were normal, transient myocardial stunning might be considered in this case (although carbon monoxide poisoning often leads to myocardial necrosis and is associated with poor prognosis).

As regards confusion with the J waves: there is convincing evidence that these waves do not represent the early phase of ventricular repolarization, but they are a manifestation of abnormal depolarization within the right ventricular outflow tract. However, this does not affect the above interpretation of ECG findings.

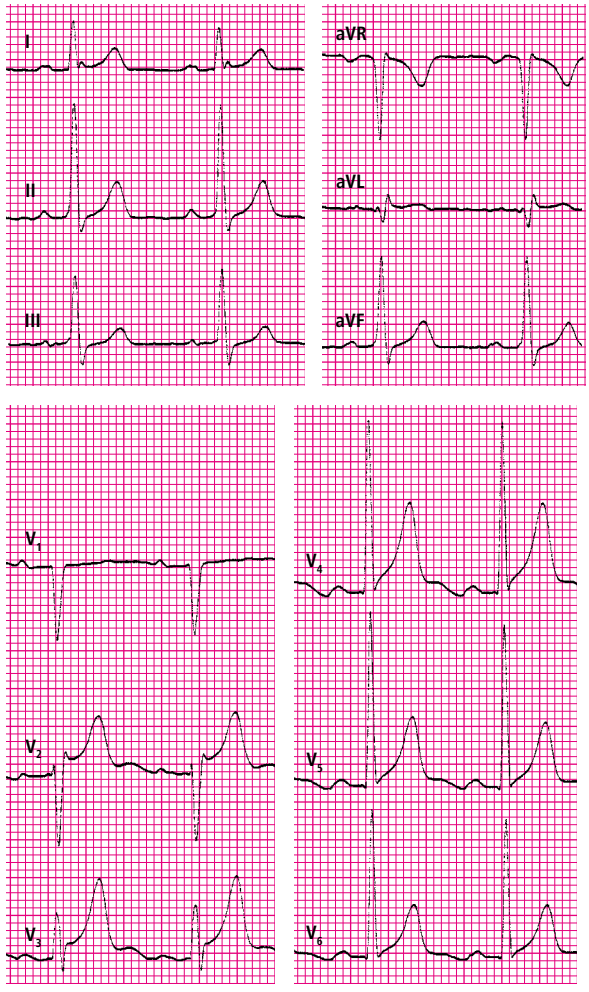

FIGURE 1

A 64-year-old woman with a history of myocardial infarction

FIGURE 1 shows electrocardiogram tracings recorded at 00:30 AM during Holter monitoring.

Sinus rhythm of approximately 85 bpm can be identified, disturbed by numerous premature ventricular beats with compensatory pauses and with one episode of tachycardia with wide QRS complexes.

It can typically be assumed that the leading rhythm in Holter monitoring is a sinus rhythm if P waves seen before the QRS complexes are

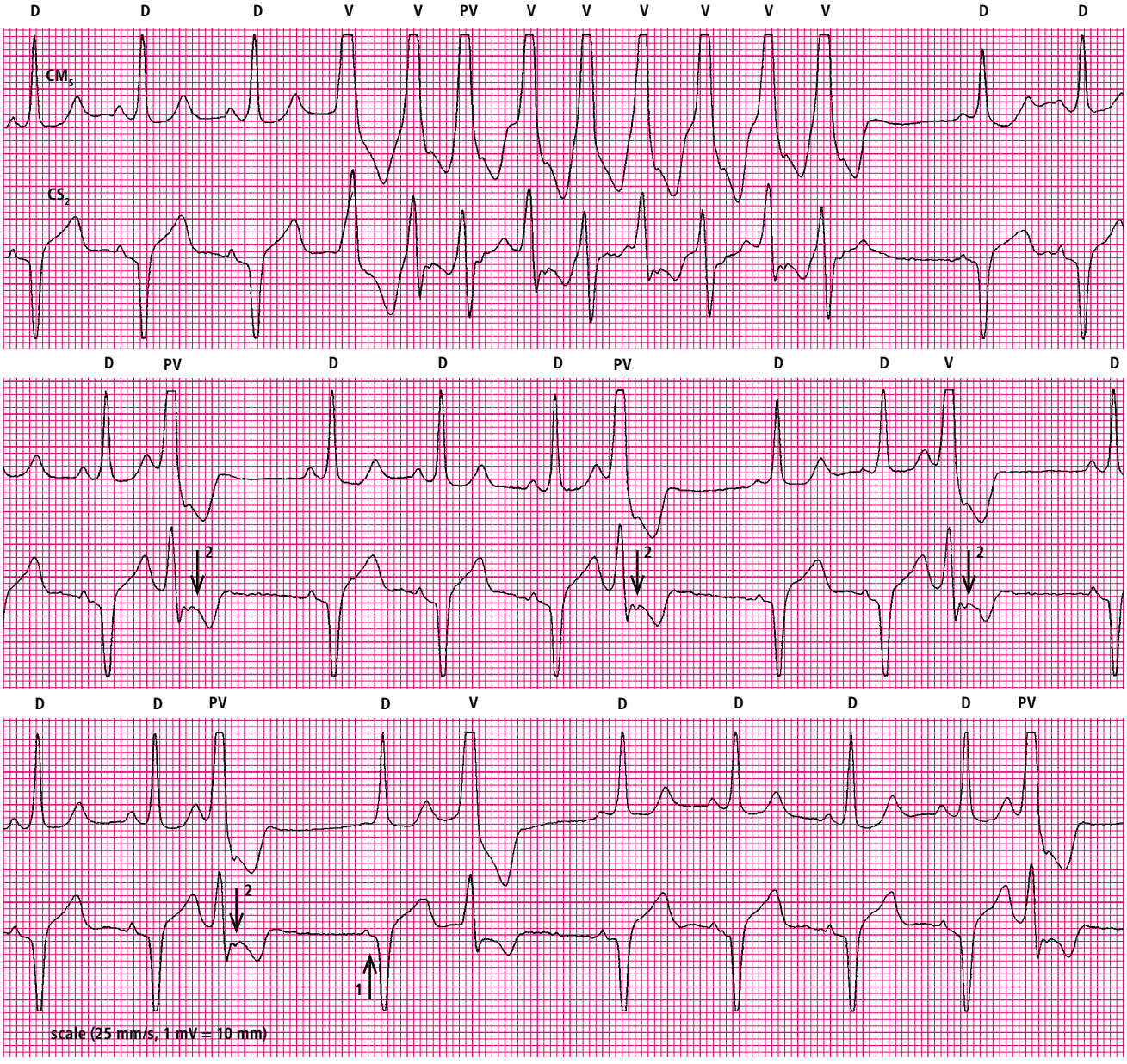

FIGURE 1

identical and are positive in lead CM_5. However, in this tracing, the P waves sometimes lose their similarity to the previous ones, especially in lead CM_5. They are more flattened (eg, in a penultimate beat with sinus rhythm seen in the upper tracing, or in the 2nd and 3rd sinus beats from the end seen in the middle tracing), while the P wave is not seen at all before the 3rd QRS complex of sinus origin in the lower strip in lead CM_5, while it is visible in lead CS_2 (although with an abnormally shortened PR interval [arrow 1]). Because all these smaller P waves do not break out of the rhythm of the PP intervals (0.72 s), it may be safely assumed that they are merely variable, perhaps due to respiratory sinus arrhythmia. And the last of the above beats with a shorter PR interval illustrates an escape beat from the atrioventricular junction (after a premature beat).

This short episode of tachycardia should be recognized as ventricular, although no atrioventricular dissociation can be identified. The ventricular origin is supported by the similarity of the QRS complexes to the complexes seen in premature beats as well as by the shape of the QRS complex in lead CM_5, which follows a pattern of a left bundle branch block with a single positive deflection. However, it is interesting that in lead CS_2 the wide QRS complexes are alternating, which allows a diagnosis of bidirectional tachycardia. Obviously this is not a masked ventricular bigeminy falling between sinus QRS complexes, because QRS complexes in lead CM_5 are of uniform shape. Therefore, this is tachycardia originating from the interventricular septum near the base of the heart (probably from the edge of the scar after myocardial infarction), which preferentially reaches the right bundle branch, but the impulse alternately travels via 2 different paths on its way to the left ventricle.

The notches visible just behind the single ectopic QRS complexes in lead CS_2 in the middle tracing and after the first such complex in the lower tracing (arrows 2) are another interesting and perhaps alarming feature. The notches appear at sites of the expected atrial depolarization originating from the sinus node, which would suggest that these are sinus P waves that have not been conducted to the ventricles depolarized by ectopic premature beats. However, similar notches can also be seen during ventricular tachycardia in the upper tracing, without any connection to a sinus rhythm. Therefore, it has to be assumed that these apparent P waves reflect fragmented ventricular depolarization, that is, the ε waves that are the equivalent of "late potentials", which during a sinus rhythm are best visible in highly amplified averaged tracings.

Death in the postpartum period

This routine electrocardiogram (ECG; FIGURE 1) was recorded in a 28-year-old woman in the 9th month of pregnancy.

The ECG shows a regular sinus rhythm (approximately 80 bpm). An interesting feature is the variable shape of sinus P waves, especially in the limb leads. Such a presentation is called a wandering sinus pacemaker and its clinical significance has not been established yet. Other features that can be identified include extreme right axis deviation (about +180°) with a positive R wave in lead aVR and a trace of R waves in leads I and aVL (arrows); very high R_{V1} waves (22 mm) with ST-T abnormalities corresponding to right ventricular overload and with deep S waves in left ventricular leads; a variable amplitude of

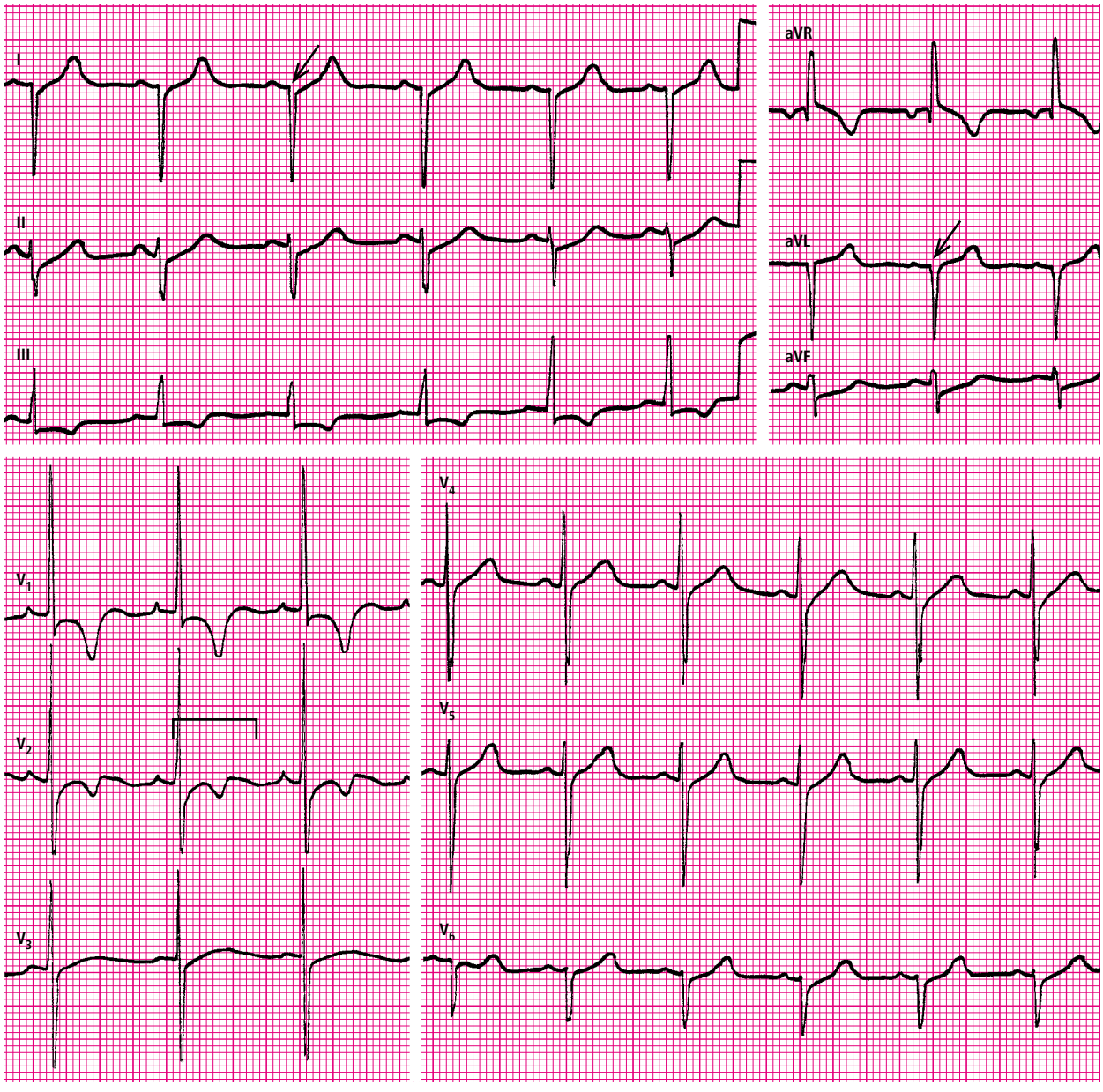

FIGURE 1

the QRS complexes in leads II and III (perhaps due to respiratory sinus arrhythmia); and, finally, a prolonged QTU interval, reaching 500 ms in the right ventricular leads (marked by brackets) and exceeding the QT interval by 120 ms in the remaining leads (a much greater difference than the duration of a normal U wave). The type of QRS and ST-T abnormalities suggests significant right ventricular overload associated with pressure overload-induced hypertrophy rather than volume overload-induced hypertrophy or hypertrophy seen in chronic cor pulmonale with dominant emphysema. Most probably, the prolonged QT intervals in leads V_1 to V_3 are also associated with severe right ventricular hypertrophy. The absence of Q waves in lead V_1 suggests that this is not severe tricuspid valve insufficiency. However, the ECG features do not explain the cause of such a severe right ventricular hypertrophy.

At 15 years of age, the patient was accidentally diagnosed with congenital heart disease. She was hyposthenic, but asymptomatic, and her development was normal. A few months later, cardiac catheterization was performed, which revealed ductus arteriosus with severe pulmonary hypertension and variable shunt: from the left to the right and vice versa. In subsequent years, the patient occasionally had dyspnea on major exertion. She became pregnant at the age of 28 years and the course of pregnancy was normal. For obstetric reasons, she delivered by cesarean section. She delivered a healthy girl with no complications of delivery. On day 2 after delivery, the patient suddenly developed a rapidly progressive dyspnea and cyanosis that spared the right hand (even clubbing, which she had developed earlier, affected only the left hand and both feet). She was transferred to a cardiac intensive care unit with worsening features of respiratory insufficiency and heart failure. Her condition worsened, leading to pulseless electrical activity resulting in death within several minutes on day 4 after delivery. Autopsy confirmed the clinical suspicion of pulmonary embolism, although a microscopic examination revealed thrombosis affecting mainly small pulmonary arteries along minor lipid embolism. Of note, pregnancy in women with Eisenmenger syndrome, regardless of its pathogenesis, is associated with an almost 40% risk of death. In most cases, death occurs immediately after delivery as a result of large fluctuations in blood volume and metabolic disturbances, which increase the risk of thrombosis.

As for this ECG, the dominant feature was the prominent right ventricular hypertrophy (on autopsy, the thickness of the left and right ventricular walls was identical and equal to 13 mm), which masked the coexistent left ventricular hypertrophy typical for patients with patent ductus arteriosus. The appropriate bedside diagnosis of this malformation could have been made on the basis of physical examination: clubbing limited to the left hand and both feet indicated that venous blood from the main pulmonary artery reached mostly the left subclavian artery and the descending aorta, which is in line with the typical location of the ductus arteriosus connecting with the aorta opposite to the ostium of the left subclavian artery.

A 50-year-old man with hypoparathyroidism

This electrocardiogram (ECG, FIGURE 1) was recorded in a patient with primary hypoparathyroidism, admitted to the hospital with a suspicion of sarcoidosis. However, sarcoidosis was excluded, and the analysis of the ECG prompted further diagnostic workup. In fact, this ECG is almost pathognomonic for the diagnosis of hypocalcemia. It shows prolongation of the QT interval solely due to extension of the ST segment, which takes almost 0.3 s of the QT interval of 0.44 s (QTc adjusted for heart rate using the Hodges formula is 0.48 s). Accordingly, the levels of total and ionized calcium (0.94–1.4 mmol/l and 0.61–0.68 mmol/l, respectively) were approximately 2 times lower than the reference range. The levels of parathyroid hormone and magnesium were also significantly reduced, while the levels of inorganic phosphate were increased.

It is known that the change in serum calcium levels is responsible for the change in the shape of the ventricular repolarization phase, although, reportedly, not directly but rather by modifying the activity of potassium channels in cardiomyocytes. A hypocalcemia-induced prolongation of the plateau phase of the action potential is homogeneous, and therefore it is not associated with a risk of ventricular tachyarrhythmia.

However, such a presentation on ECG is only "almost pathognomonic" for hypocalcemia, because there is another rare disorder manifesting with the same ECG features, namely, congenital long QT syndrome (LQTS) type 3. In LQTS type 3, the prolongation of ST segments is associated with genetic sodium channel dysfunction in myocardial ventricular cells. Compared with LQTS types 1 and 2, LQTS type 3 is much rarer but is associated with a much higher risk of sudden cardiac death. Differential diagnosis with hypocalcemia should be based on the measurement of serum calcium levels.

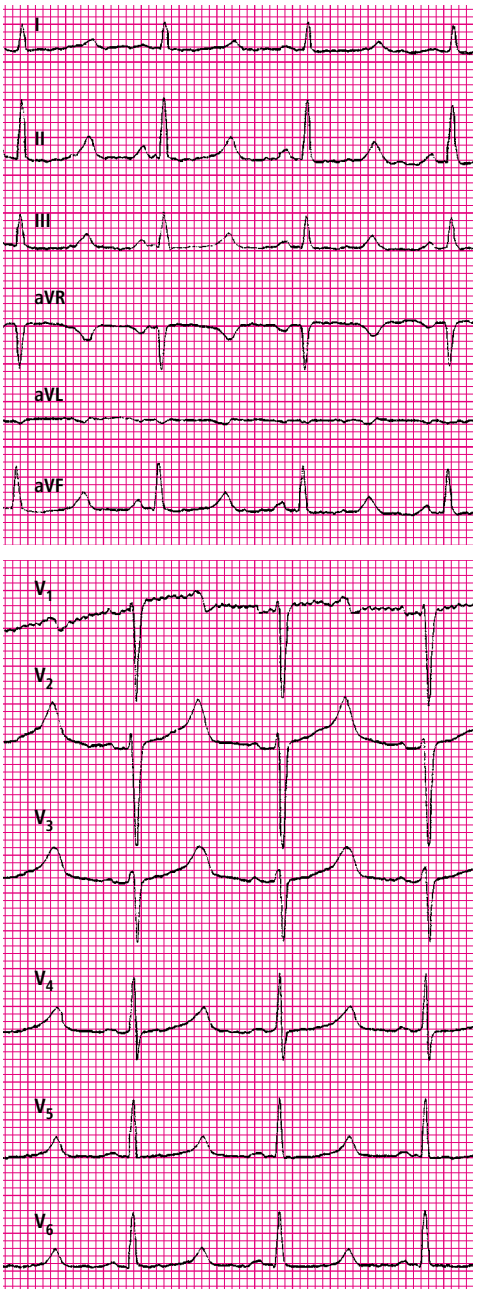

FIGURE 1

A male patient with renal insufficiency

FIGURE 1A shows an electrocardiogram (ECG) tracing from precordial leads V_1 to V_6 (paper speed of 25 mm/s). The QT interval is extremely long (0.7 s), while a heart rate of 58 bpm makes the correction unnecessary. The QT prolongation is caused by a very long ST interval. The first suspicion when reading this ECG is hypocalcemia, and indeed, the ECG was recorded in a patient with renal failure and electrolyte disturbances, including hypocalcemia and hypokalemia. This association was confirmed by the ECG from right ventricular leads (FIGURE 1B) recorded 2 days later, after correction of electrolyte disturbances, showing the shortening of the QTc interval to 0.5 s.

The widening of the QRS complexes up to 0.12 s seen in FIGURE 1A, compared with their normalization in FIGURE 1B, can be attributed to hypokalemia because extracellular calcium deficiency does not affect this parameter. As for the QT interval, the prolongation of the QTc interval in hypokalemia usually does not exceed 140% of the reference value, and if it does, it suggests that it is in fact the measurement of the QU interval, and not of the QT interval. Therefore, this prominent T wave in FIGURE 1A might in fact have been a U wave caused by coexistent hypokalemia. Unfortunately, this ECG does not provide a definitive answer to this question.

If we were to read this ECG without data on the calcium levels, the differential diagnosis would include long QT syndrome type 3 (characterized by ST-segment prolongation), caused by the *SCN5A* gene mutation responsible for sodium transport in phase 0. Mutation of this gene causes a prolonged sodium influx into cells, including the repolarization plateau. However, this serious condition is even less frequent than severe hypocalcemia.

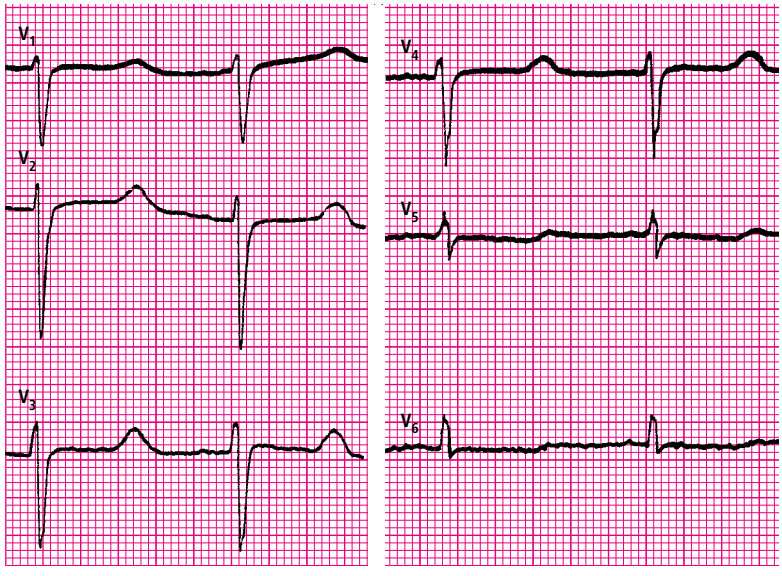

FIGURE 1A

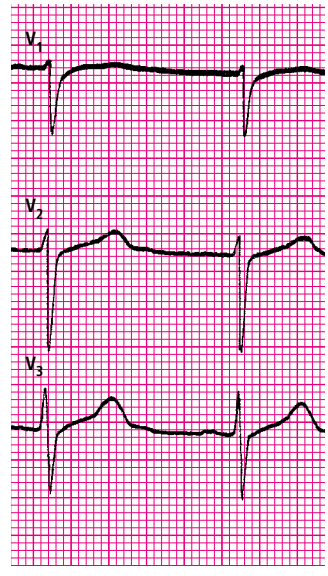

FIGURE 1B

CLINICAL INTERPRETATION

Progressively worsening intraventricular conduction disturbances in a 65-year-old man after myocardial infarction

The electrocardiogram (ECG) tracings in FIGURE 1A and 1B show intraventricular conduction disturbances in a male patient with progressive coronary artery disease.

Already the first ECG (FIGURE 1A) reveals numerous abnormalities, including right superior axis deviation (approx. –95°) and right bundle branch block with an extreme clockwise heart rotation (R/S V_6<1). In addition, features of the late evolution of anterior wall myocardial infarction can be seen: Q waves in leads V_1 to V_3, QS complex in lead V_4, and resolving ST-segment elevation in leads V_2 to V_4 with deep symmetrical T waves in leads V_3 and V_4. The asymmetric negative T waves in lead V_1 should be treated as a change in the course of repolarization secondary to right bundle branch block, and in lead V_2—as a result of the summation of both those mechanisms.

After 7 years (FIGURE 1B), the features of the right bundle branch block are still visible, but now the cardiac axis is directed upwards and to the left (approx. –75°). However, these ECG features do not meet the criteria for anterior fascicular block because there are no q waves in leads I and aVL, while in leads II and III the QRS complexes have a QS rather than an rS pattern. On the other hand, the ECG now shows the features of left bundle branch block, but—although the QRS duration is 0.2 s—it cannot be considered as a complete heart block. This is because the patient had had complete right bundle branch block for years, and in such case the development of complete left bundle branch block would have caused third--degree atrioventricular (AV) block. Therefore, contrary to the current criteria of the American Heart Association, American College of Cardiology, and Heart Rhythm Society (2009), incomplete left bundle branch block should be diagnosed, probably caused by progressive fibrosis and chronic ischemia within the septal fascicle of the left bundle branch. This type of block is also sometimes referred to as a concealed bundle block. The simultaneous presence of first-degree AV block (PR interval, 0.32 s) and right bundle branch block indicates that conduction abnormalities are present also in the other 2 fascicles of the left branch, which may pose a risk of complete AV block.

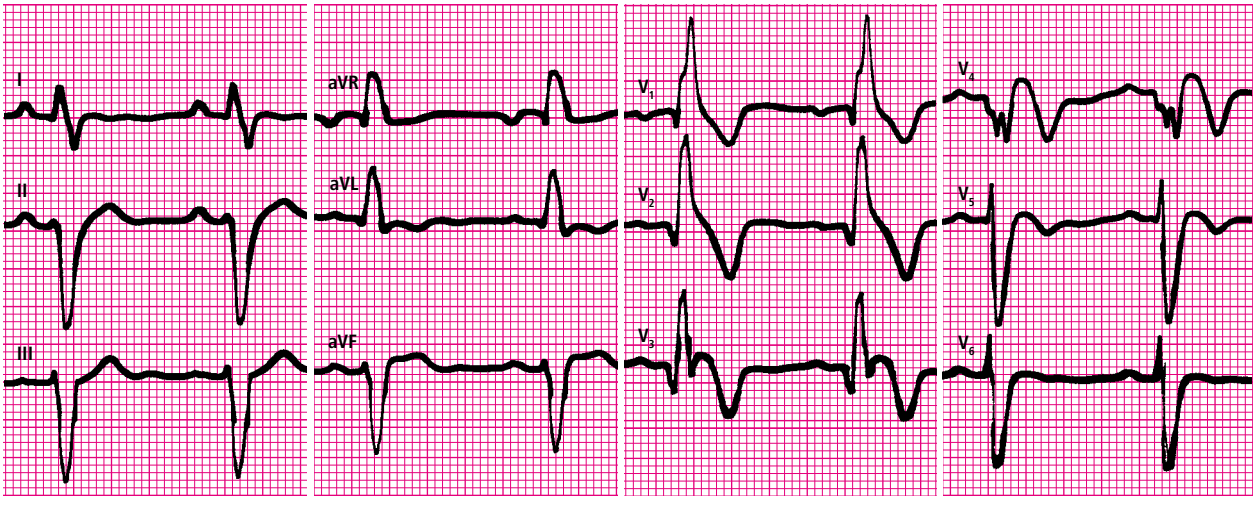

FIGURE 1A

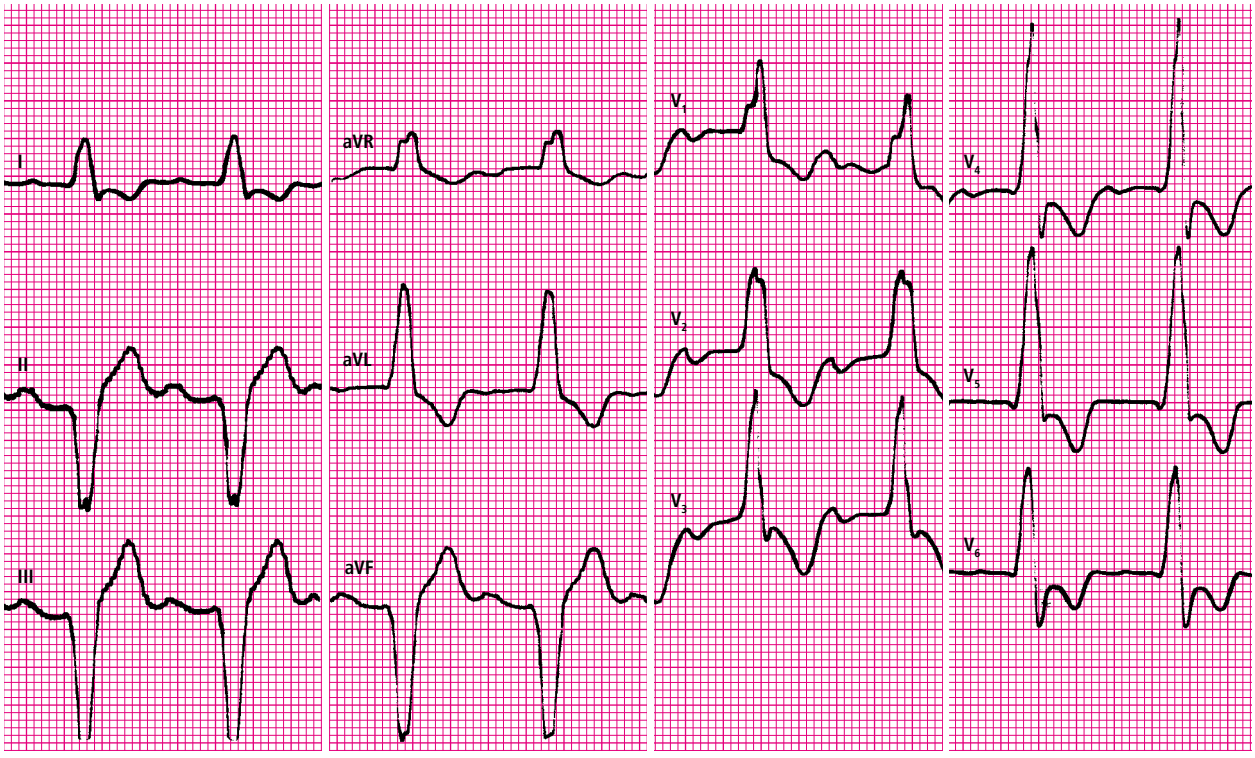

FIGURE 1B

An 82-year-old woman with an undiagnosed acute myocardial infarction

This electrocardiogram (ECG; FIGURE 1A–C) was recorded in an 82-year-old woman, who 7 years earlier suffered anterior wall myocardial infarction (MI) "without a Q wave" and since then has presented with left bundle branch block. She presented to the hospital with retrosternal pain lasting 2 hours, after she fell victim to a street robbery. An ECG was recorded (FIGURE 1A; paper speed of this and subsequent ECGs, 50 mm/s), and sublingual nitroglycerin and intravenous analgesic were administered. With no obvious indications for hospitalization in the opinion of the doctor on duty, she was discharged after resolution of pain, with a diagnosis of stress-induced angina pectoris in a patient with a history of MI, and possible chest pain due to injury.

After 4 days, the patient presented for a previously scheduled consultation with her cardiologist, who raised a suspicion of acute MI on the basis of the current (FIGURE 1B) and previous ECGs (the ECG recorded on the day of the assault showed the previously absent convex ST-segment elevation in leads V_2 and V_3, QS patterns in leads V_1 to V_4, and an abnormal Q wave in lead V_5). The diagnosis was confirmed by a subsequent ECG (evolution of the ST-T complexes in leads V_2–V_5), and the patient was referred for hospitalization at a cardiac unit in the hospital from which she was discharged a few days earlier. She was discharged again after 6 days, and, interestingly, the diagnosis was unstable angina. The basis for excluding the diagnosis of MI was the normal troponin level on the 4th and 5th days after the event. Thus, the clinical features, including the evolution of ECG, were not given priority over a biochemical study performed already after a few days since the coronary event. What is more, echocardiography performed 7 years earlier showed only asynchronous contraction of the interventricular septum and akinesia limited to the anterior part of the septum, while the current echocardiography revealed akinesia of the apical and middle segments of the interventricular septum as well as

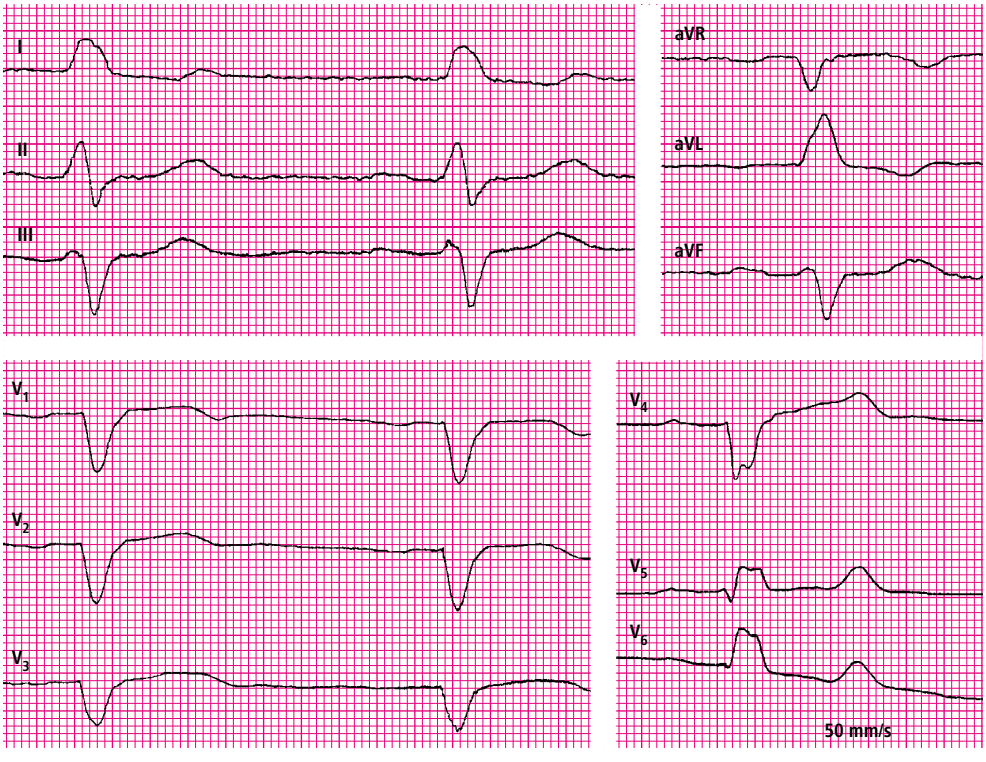

FIGURE 1A

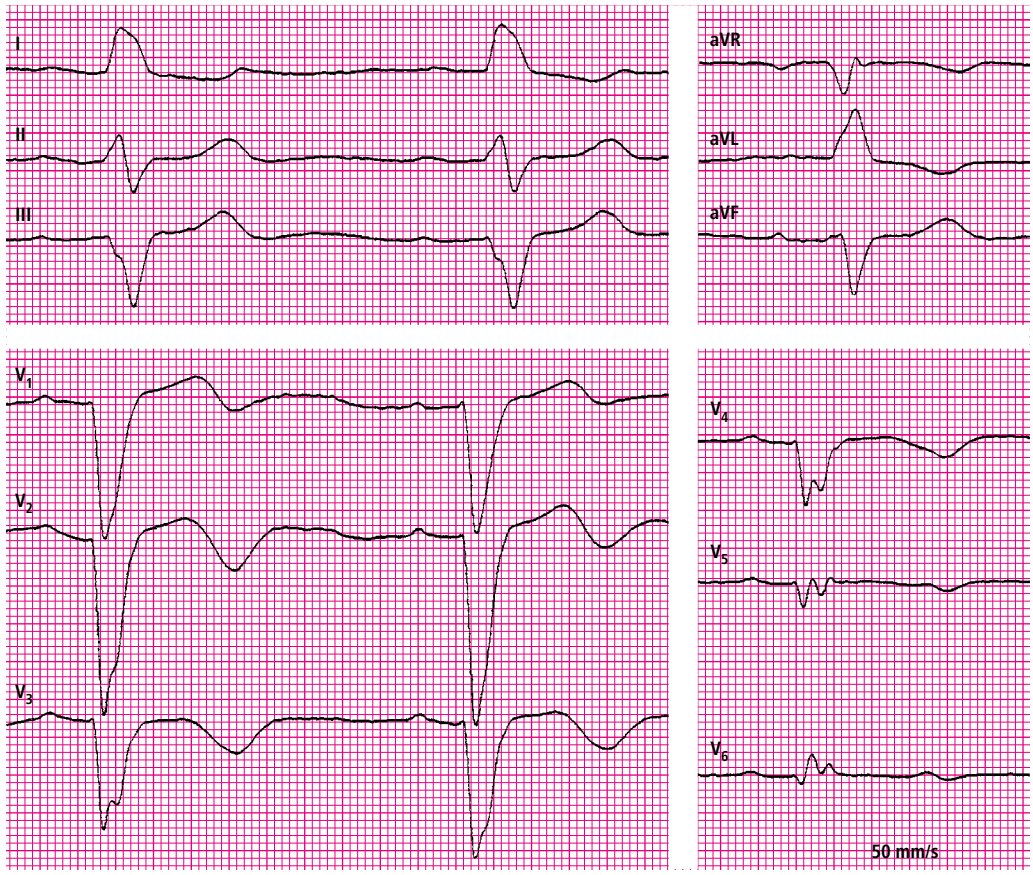

FIGURE 1B

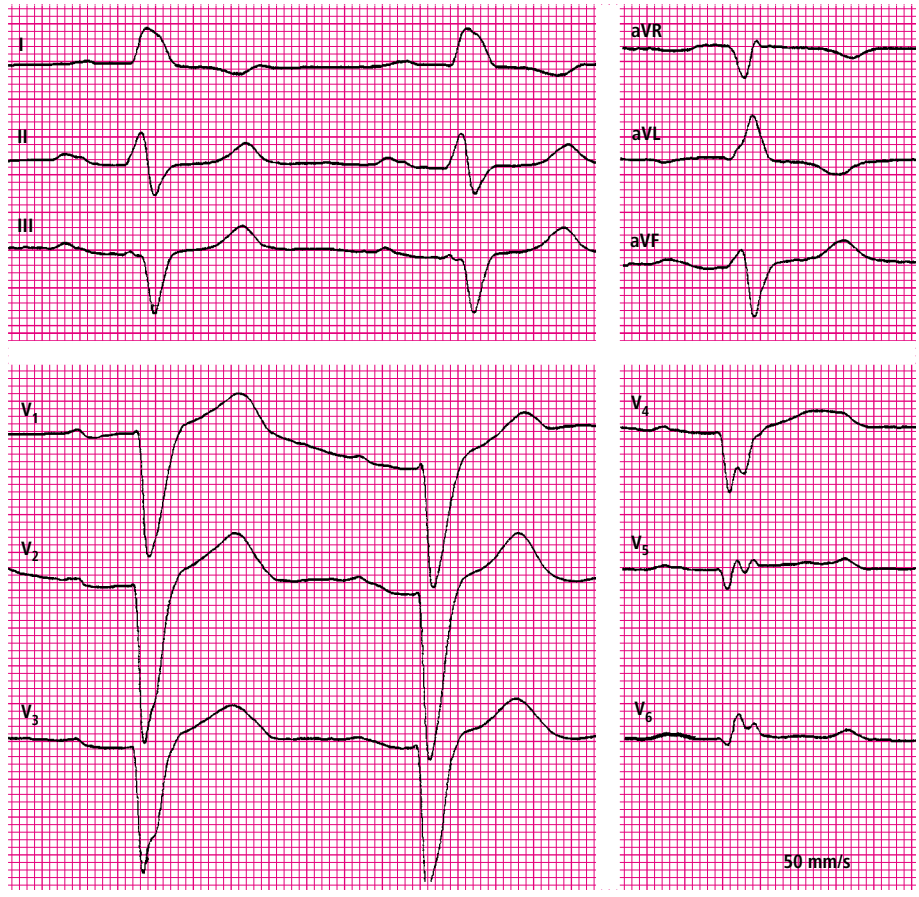

FIGURE 1C

of the apical segment of the lateral wall. However, despite this abnormality the hospital cardiologist did not attempt to revise the diagnosis made on the basis of normal troponin levels. FIGURE 1C shows further evolution of ECG features in this patient.

The present case illustrates that we should avoid:

1 making a diagnosis of angina pectoris in patients with chest pain without comparing the current ECG with previous results (especially in patients after MI) and/or measuring troponin levels in the acute phase of the disease;

2 dismissing the risk of MI in patients with left bundle branch block;

3 making a diagnosis of MI based solely on troponin levels, particularly when assessed only in the first hours of chest pain or a long time afterwards, without considering other clinical features of MI, which include evolution of ECG features and echocardiograms;

4 and street robbers, too.

A 45-year-old man with epilepsy and hyperparathyroidism

This electrocardiogram (ECG; FIGURE 1) was recorded in a man with epilepsy (treated with 2 drugs at high doses), who was admitted to the hospital for diagnostic workup of severe bone loss revealed on densitometry. During the 4-day hospital stay, primary hyperparathyroidism was diagnosed on the basis of laboratory findings (significant hypercalcemia [3.58–3.79 mmol/l], hypophosphatemia, and high parathyroid hormone levels) as well as ultrasound findings (calcium deposits in the kidneys and oval hypoechoic lesion under the right thyroid lobe—probably an enlarged parathyroid gland). Glucose and creatinine levels were normal, while potassium levels were low (2.7–3.1 mmol/l). The patient was administered a bisphosphonate, methylprednisolone, furosemide, spironolactone, and potassium, and was referred for a consultation with an endocrinologist.

If we did not know the diagnosis, we would consider this ECG as normal; in particular, the QT interval is normal (0.40 s corrected for heart rate of 82 bpm using the Hodges formula). The only feature that may draw some attention is a short ST segment (in leads V_3 and V_4, it is slightly longer than 0.04 s, while in leads II, III, aVR, and aVF, the T wave starts almost at the J point). However, the ECG also shows an objective, although not commonly known, quantitative feature characteristic for hypercalcemia. It has been suggested (Surawicz B. Chou's electrocardiography in clinical practice, Saunders Elsevier 2008) that in patients with hypercalcemia, a short interval from the Q wave to the top of the T wave (Q-aT) is a more important feature than the QT interval (which may not always be shortened). In more than 90% of patients with hypercalcemia, the Q-aT interval does not exceed 0.27 s. In this ECG, it is 0.25 s, which is consistent with

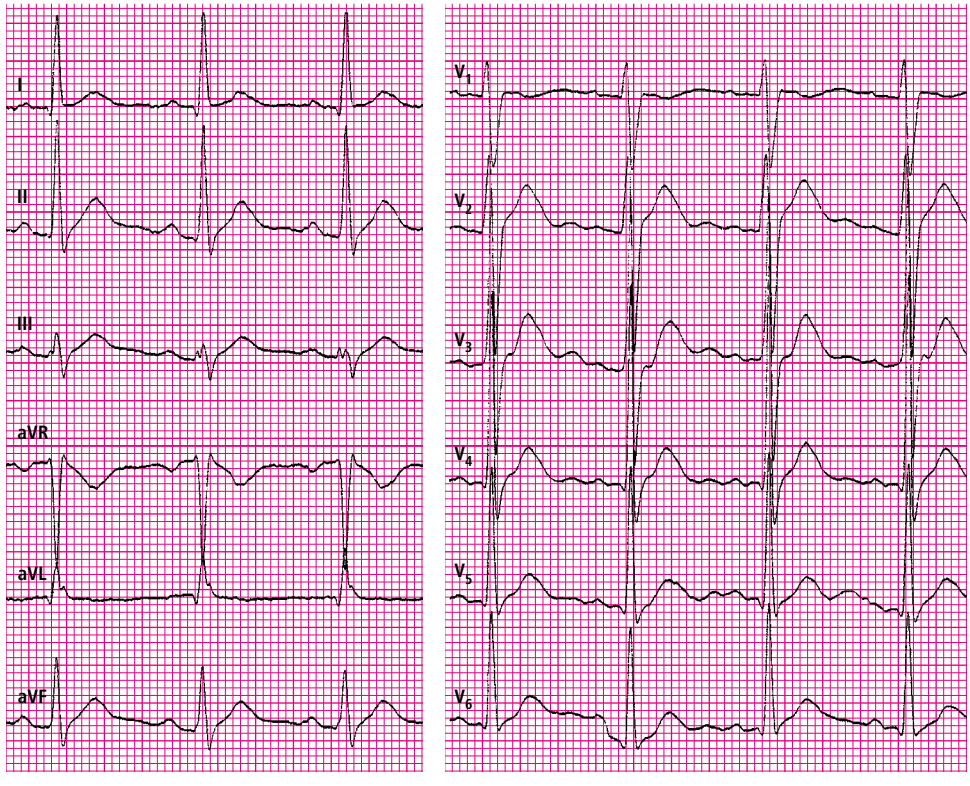

FIGURE 1

the clinical diagnosis. On the other hand, the prolongation of the PR interval and QRS complex, although occasionally associated with significant hypercalcemia, is not characteristic for this electrolyte disturbance and therefore is not mentioned in most textbooks.

The T wave in the present ECG is wide and its early peak makes its descending limb longer than usual (it is even longer than the ascending limb, indicating reversed physiological asymmetry of the T wave). This change, particularly visible in leads V_2 and V_3, was at first (during 4-day hospitalization) linked to concomitant hypokalemia, which in turn was attributed to the use of glucocorticoids. However, is it a correct interpretation? Hypokalemia could have been related to glucocorticoids only if the patient had been taking them before hospitalization, and this is unlikely because the diagnosis was established in the hospital. Therefore, it would be worth considering whether hypokalemia was not linked with an underlying condition: is it possible that it was a parathyroid adenoma coexisting with an adrenal aldosterone-producing adenoma? Such adenomas are often small and difficult or even impossible to identify on ultrasound. If the patient had primary hyperaldosteronism, the diagnosis of multiple endocrine neoplasia type 1 would be the correct one.

Regardless of the cause of hypokalemia, it is rather unlikely that the above disturbances of ventricular repolarization were associated with low potassium levels, because the key feature is missing, namely, T-wave flattening with an increased amplitude of U waves and the reversal of their normal ratio (not T>U, but U>T). Besides, the U waves in this ECG are normal. However, there is one feature undoubtedly associated with hypokalemia (although, unfortunately, not widely recognized), namely, an increase in the amplitude of the QRS complex, which meets the criteria for left ventricular hypertrophy. In this ECG, the sum of the amplitudes of S_{V2} and $R_{V4/5}$ exceeds 45 mm ($S_{V2} + R_{V4} = 18 + 29 = 47$ mm), which is one of the 36 criteria of left ventricular hypertrophy, according to the 2009 guidelines of the American Heart Association, American College of Cardiology, and Heart Rhythm Society.

As for the T waves, although the reason for extension of their descending limb is unclear, it is important to note that this part of the ECG is considered as a measure of a transmural dispersion of ventricular repolarization. However, the extension of the descending limb of the T wave is not used in routine clinical practice because its measurement generates too many errors. Yet there have been numerous reports suggesting that its considerable extension may be associated with a risk of severe ventricular arrhythmia. Then, the final question to answer would be whether the patient in fact had epilepsy. Maybe the loss of consciousness with convulsions was caused by episodes of ventricular arrhythmia? And maybe this would explain the alleged low effectiveness of antiepileptic drugs and the gradual uptitration of their dose?

Two patients in their sixties with near-normal electrocardiograms

These 2 standard electrocardiograms (ECGs) were recorded in men aged 69 and 65 years. In the first case (FIGURE 1A), a doctor referring the patient for ECG established a diagnosis of hypertension and lung tumor, while in the second case (FIGURE 1B) no diagnosis was indicated on the referral.

The first ECG (FIGURE 1A) shows only a slightly prolonged PR interval—0.24 s (however, such a feature may also be seen in healthy persons). The notched and narrow QRS complexes in leads III and aVF ("adjacent" to each other, although not in a routine 12-lead ECG but in a Cabrera display presenting the 6 limb leads in their anatomically ordered sequence at the frontal plane) would not typically be considered as an abnormal feature. However, in the 21st century, there have been reports linking some clinical conditions with QRS notching. First, French cardiologists showed that the notches seen on the ascending limb or on the top of the R wave in leads II, III, and aVF are characteristic for an atrial septal defect (ASD) and are more common in this condition than incomplete right bundle branch block (even if they are present only in one of the above leads, which is a less specific feature than their presence in 2 or 3 leads). This feature is 10 times more frequent in patients with ASD than in healthy persons. However, the mean age of the control group of healthy individuals in this study, corresponding to the age of patients with ASD, was only 23 years. As it is well known that notched QRS complexes become more common with age, further diagnostic workup for a previously undiagnosed congenital heart disease seems unnecessary.

There is also another observation relating to the presence of high-frequency components in the QRS complexes (found also in computerized ECG assessments), which has long been described in patients with myocardial infarction. Recently, American investigators studied the significance of this feature as part of a routine ECG diagnostic workup in a cohort of nearly 500 persons without a branch block referred for stress myocardial perfusion scintigraphy due to diagnosed or suspected coronary artery disease. They showed that the presence of notches in 2 adjacent QRS complexes had a 2-fold higher sensitivity (86% vs 36%) and only slightly lower specificity (89% vs 99%) than the Q waves for prediction of post-myocardial infarction scar, while the negative predictive value (ie, the absence of myocardial infarction if the notches were not present) reached almost 93%. However, as long as these data are not confirmed in other studies and approved by experts, it would not be necessary to include the following statement in the description of the first ECG: "notches of QRS in leads III and aVF for clinical interpretation."

But how do these data affect the interpretation of the second ECG (FIGURE 1B)? The problem here is much more complex. The QRS complexes are quite wide (0.1–0.11 s; according to the 2009 guidelines of the American Heart Association, American College of Cardiology, and Heart Rhythm Society, the value of 0.11 s is the upper normal limit for QRS duration in adults), with notches in 3 adjacent leads (II, III, and aVF) and coexisting incomplete right bundle branch block (unless it is a Qr complex). These features are accompanied by clockwise heart rotation and the RS pattern in lead I (with R>S). Echocardiography would probably be useful in this case to exclude ASD type 2 and to evaluate the inferior and anterior wall motion (because of suspected myocardial scars in these sites). Therefore, in this case, it seems justified to include the presence of QRS notching in the description of the ECG, along with an indication for further diagnostic workup.

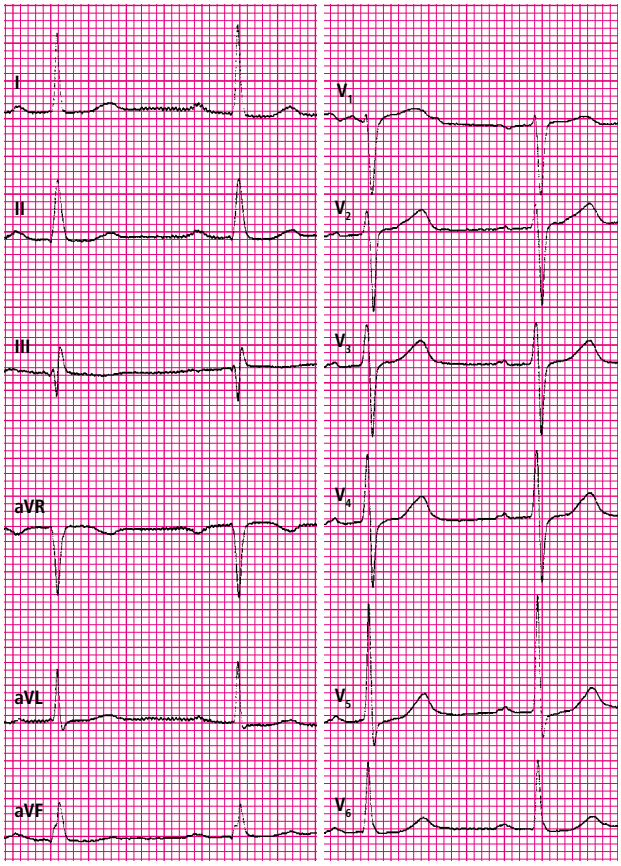

FIGURE 1A

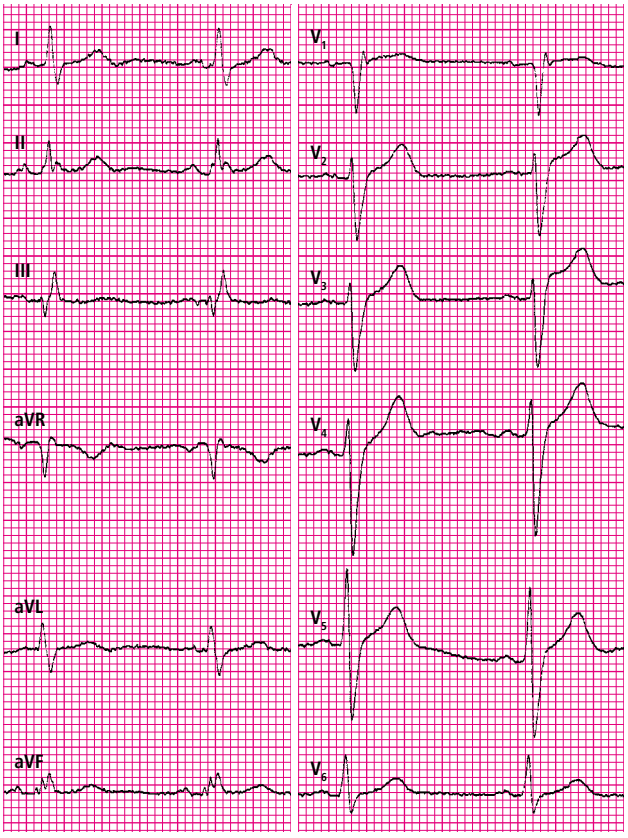

FIGURE 1B

A woman with poorly tolerated palpitations

These 2 standard electrocardiogram tracings (FIGURE 1) were recorded in a woman who presented to the hospital with poorly tolerated palpitations. Before admission, she did not take any antiarrhythmic drugs.

At first glance, the most obvious diagnosis would be ventricular bigeminy. For better assessment, it may be reasonable to start—rather atypically—from the precordial leads. It can be assumed that the basic rhythm is a sinus rhythm of 100 bpm, but every second sinus impulse does not reach the ventricles, since these stimuli are blocked in the atrioventricular [AV] node by ventricular bigeminy. The ectopic rhythm probably comes from the base of the heart, from the area of the bundle of His, because the QRS complexes are only slightly distorted compared with sinus QRS complexes, with a shape typical of an incomplete right bundle branch block. This raises a possibility of an alternative diagnosis, namely, bigeminy from the AV junction, which first reaches the ventricles and is followed by an immediate retrograde conduction to the atria (with alleged R' deflections in lead V_1). However, this diagnosis can be excluded because there are traces of sinus P waves just before some premature QRS complexes, for example, in lead V_3 (arrows 1). The same situation can be seen in leads I to III before the fourth ectopic ventricular beat. In the middle of the tracing, other sinus P waves can be clearly seen, all positive in leads II and aVF, with a rate of 100 to 110 bpm (arrows 2). However, these P waves are followed by conducted QRS complexes only twice (arrows 3). The first complex is completely of sinus origin, while the second one, slightly wider (fourth from the end), is summed up. The other sinus P waves merge with the QRS complexes of nonsinus origin: they sometimes hide in the QRS complexes completely and sometimes not entirely or even slightly position themselves ahead of the nonsinus wide QRS complexes (the third and fifth beats from the onset in the limb leads [arrows 4]). But where is the third active pacemaker located?

The consecutive QRS complexes in the limb leads, just as in the precordial leads, are arranged in bigeminy. These premature QRS complexes, the second of a pair, are slightly different (especially in lead I [arrow 5]) than the preceding QRS complexes, also of nonsinus origin. Therefore, it is obvious that the basic rhythm in the limb leads (coming once from the sinus node and another time from an ectopic supraventricular parasystolic pacemaker, probably located in the AV junction) is disturbed by ventricular bigeminy originating from some other neighboring site.

Such arrhythmia from 3 pacemakers, with sinus tachycardia seen later in the tracing, suggests that the patient was subject to a strong β-adrenergic stimulation (perhaps the use of a β-agonist, eg, in the treatment of asthma), because β-adrenergic stimulation not only accelerates the sinus node but also increases the activity of nonsinus pacemakers.

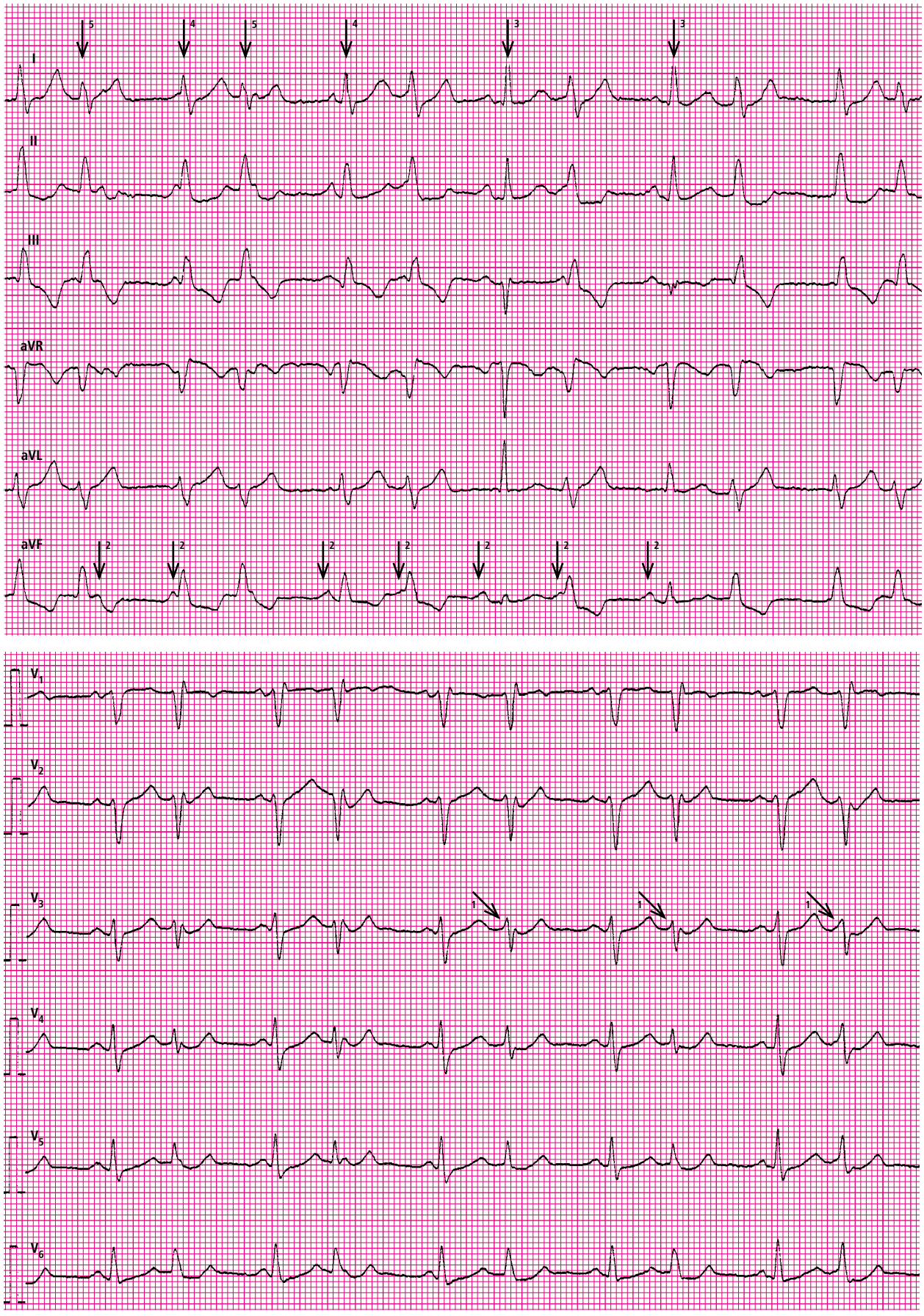

FIGURE 1

A 40-year-old obese man with severe hypertension

This is a standard electrocardiogram (ECG; FIGURE 1) recorded in an obese man (weight, 115 kg; height, 1.78 m), a taxi driver, who presented to the doctor because of high blood pressure persisting for the past 3 months (average range, 180/110–190/110 mmHg, sometimes reaching 200/140 mmHg). He reported smoking of about 30 cigarettes a day for the past 20 years and denied alcohol abuse. His father had hypertension and died at the age of 40 years due to hemorrhagic stroke. On the day of the visit, the blood pressure was 180/120 mmHg.

The ECG shows a sinus rhythm of approximately 70 bpm, with mild arrhythmia, probably respiratory. On the basis of the other ECG features, left ventricular overload might be suspected.

However, despite the typical changes of ST-T, such a diagnosis would not be correct because the ECG does not meet the criteria for left ventricular hypertrophy: the amplitude of the QRS complexes is normal, no features of P mitrale can be seen, and there are no intraventricular conduction disturbances (reflected either by wide QRS complexes or prolonged intrinsicoid deflection in the left ventricular leads).

So how should we interpret such an ECG? If the clinical data on blood pressure and obesity of the patient were unknown, the first diagnosis to consider would be hypertrophic cardiomyopathy. However, considering high blood pressure and obesity, such ECG features are rather a result of "typical" left ventricular hypertrophy in the course of hypertension, and the amplitude of the QRS complexes does not meet the criteria for hypertrophy due to obesity. The differential diagnosis should also account for the fact that the lower part of the S waves in lead V_1 was cut off. However, it seems that the S waves, narrow in the place where they were cut off, would not be deep enough as to meet the criterion of $S_{V1} + R_{V5(6)} > 35$ mm. Therefore, one of the possible descriptions of the ECG would be "features of myocardial ischemia resembling secondary ST-T changes in left ventricular hypertrophy."

There are also 3 other interesting abnormalities. Firstly, although the ECG does not show the features of P mitrale, split P waves in leads V_1 to V_3 (arrows 1) may suggest intraatrial conduction disturbances, and therefore they suggest damage of the atrial myocardium, often promoting a tendency to atrial fibrillation that is frequently associated with hypertension. Secondly, in leads V_2 and V_3, split T waves can be found, and the peak of the T wave in lead V_3 corresponds to the notch on a descending limb of the T wave in lead V_2 (arrow 2). Therefore, these notches are not the U waves, which in this ECG are clearly seen after the split T waves. T-wave splitting in the right ventricular leads occurs even in healthy individuals, but it is sometimes associated with impaired sympathetic tone seen, for example, in patients with withdrawal syndromes.

Finally, in the aVR lead, an almost 2-mm ST-segment elevation can be seen. Previously considered to be of little significance, this feature is now believed to be the most sensitive and specific ECG feature of acute coronary syndrome in the course of left coronary artery stenosis or 3-vessel disease. Of course, in this case such a diagnosis would be incorrect because the elevation is concave (unlike the Pardee wave) and the clinical features are not suggestive of an acute coronary syndrome. Therefore, the ST-segment elevation in lead aVR is only a reflection of extensive subendocardial ischemia associated with left ventricular hypertrophy (not seen in this ECG).

It is worth emphasizing that ischemic features on ECG resulting from left ventricular hypertrophy may be associated with poor prognosis.

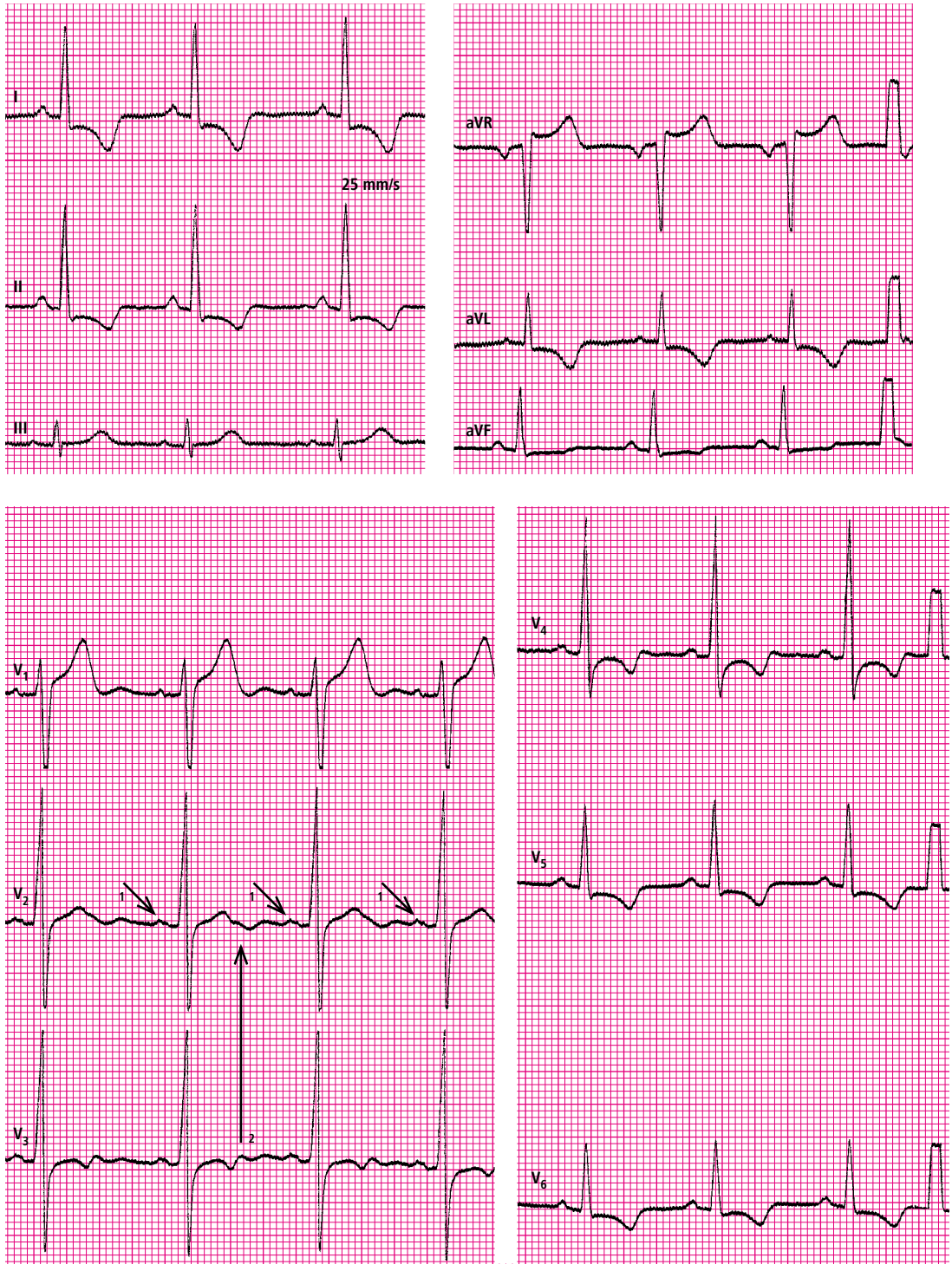

FIGURE 1

A 25-year-old woman after an attempted suicidal quinidine poisoning

These electrocardiograms (FIGURE 1AB) were recorded in a young woman after an unsuccessful suicidal poisoning with quinidine tablets.

An interesting feature is the significant prolongation of the QT interval, perhaps combined with a U wave and ending within a P wave (for this reason, the QT interval is difficult to measure, but if the descending limb of the T wave in lead V_5 is extended to the baseline, then the extrapolated QT interval will be 560 ms, while the QT interval adjusted for heart rate using the Hodges formula [QTc]—620 ms). QT prolongation is due to the blocking effect of quinidine on the outflow of potassium in the 2nd and 3rd phases of the action potential. The simultaneous inhibition of sodium influx into myocardial cells is associated with other characteristic ECG features: the widening of the QRS complexes (here, 0.12 s) and prolongation of the PR interval (here yet normal, 0.2 s) (FIGURE 1A). It may also cause bradycardia. However, in this case, it is sinus tachycardia (96 bpm), which is also typical for quinidine effects. This is because immediately after quinidine ingestion, its additional effects such as a decrease in vagal tone and vasodilation become most apparent, resulting in hypotension and adrenergic response. Moreover, increased adrenergic response promotes ST-segment depression, as seen in this ECG.

Fortunately, in this case, life-threatening ventricular tachyarrhythmia did not occur. At this point, it is interesting to note that, paradoxically, low-dose quinidine triggers early afterdepolarizations more frequently than high-dose quinidine, because the dominant effect of the latter is

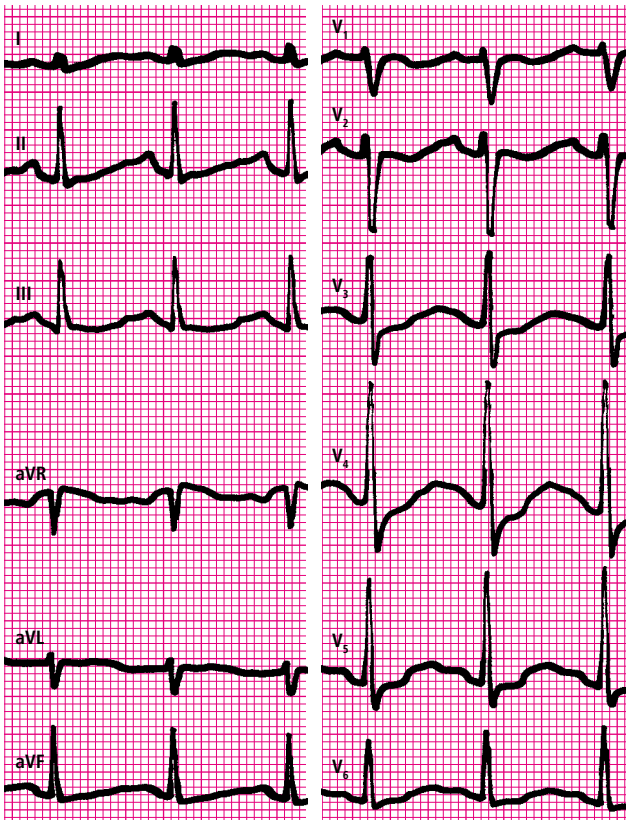

FIGURE 1A

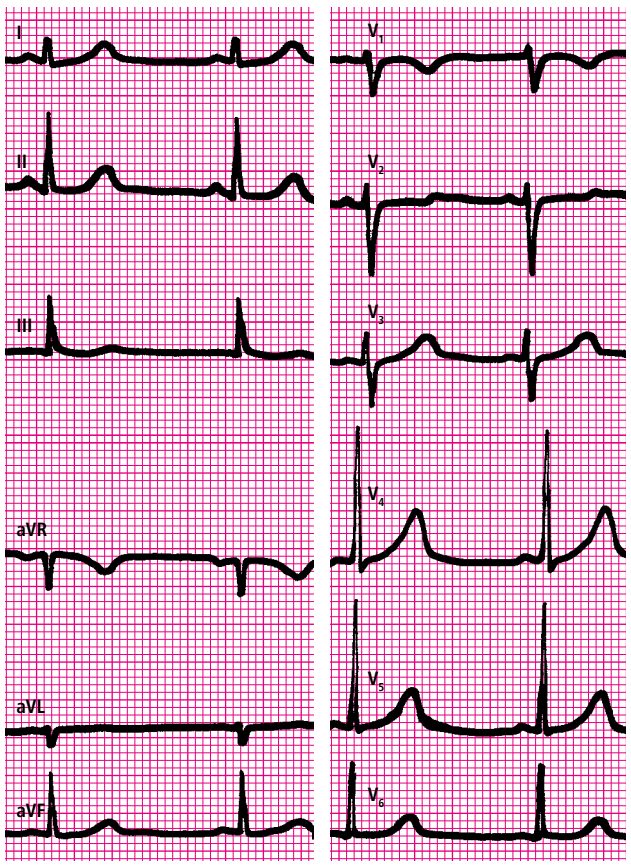

FIGURE 1B

the inhibition of the sodium current rather than potassium channel blockade. In a follow-up ECG (FIGURE 1B) recorded a few weeks later, all the above features returned to normal.

A 42-year-old man with abnormal electrocardiogram

These 3 electrocardiograms (ECGs) were recorded in a man with mitral valve prolapse diagnosed at a young age and with various cardiovascular psychosomatic symptoms effectively controlled by metoprolol. Because of recently diagnosed gastroesophageal reflux disease, a gastroenterologist prescribed treatment with pantoprazole, which coincided with a follow-up ECG performed as part of routine medical checkup. The ECG in FIGURE 1A is almost normal, except for notched QRS complexes over the inferior wall and a quite unusual configuration of ST-T complexes in leads V_2 and V_3. As an artifact was suspected, the ECG was repeated after a few days, but the results were the same (FIGURE 1B). This "rough" area within the ventricular repolarization phase shows a convex ST-segment elevation starting from the J point, which considerably differs from the normal pattern of early ventricular repolarization. Moreover, a U wave superimposed on an unusually long descending limb of the T wave can be seen. The QTU interval in leads V_2 and V_3 is 0.54 s (0.55 s corrected for heart rate [68 bpm] using the Hodges formula). A comparison of these values with QT intervals in other leads without U waves is hardly possible, because such a dispersion of repolarization is difficult to interpret and there are no reference values for QTU intervals in the case when a U wave is superimposed on a T wave. However, the very presence of such superimposition is an abnormal feature. What is possible and worthwhile to do is the measurement of the QT intervals in leads V_2 and V_3, by plotting a tangent to the descending limb of the T wave, with the intersection of the tangent and isoelectric line considered as the end of the T wave. It turns out that due to the less steep than usual inclination of that limb, the end of the T wave falls not much nearer than the end of the U wave. Thus, there is every reason to interpret this untypical ECG as long QT syndrome. After discontinuation of pantoprazole, the ST-T changes resolved (FIGURE 1C). As for the causal relationships between pantoprazole and ECG findings, apart from the apparent resolution of ECG changes after the discontinuation of the drug, there are also some clues related to the pharmacokinetic characteristics of this drug class.

It is believed that more than half of the drugs used in clinical practice are metabolized in the liver by cytochrome P450 enzyme systems, with a large part being metabolized by CYP3A4

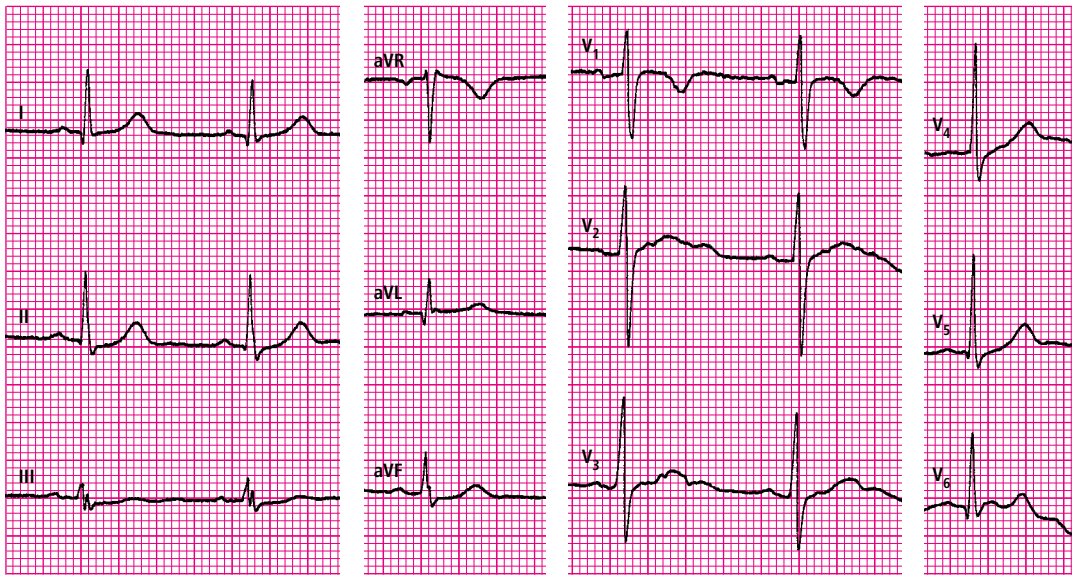

FIGURE 1A

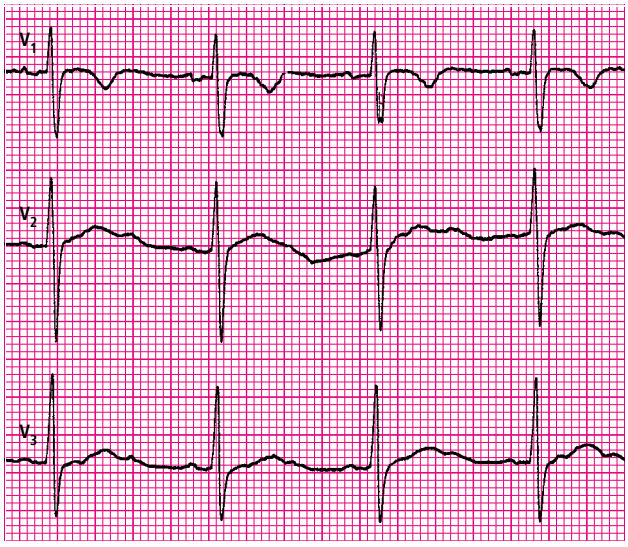

FIGURE 1B

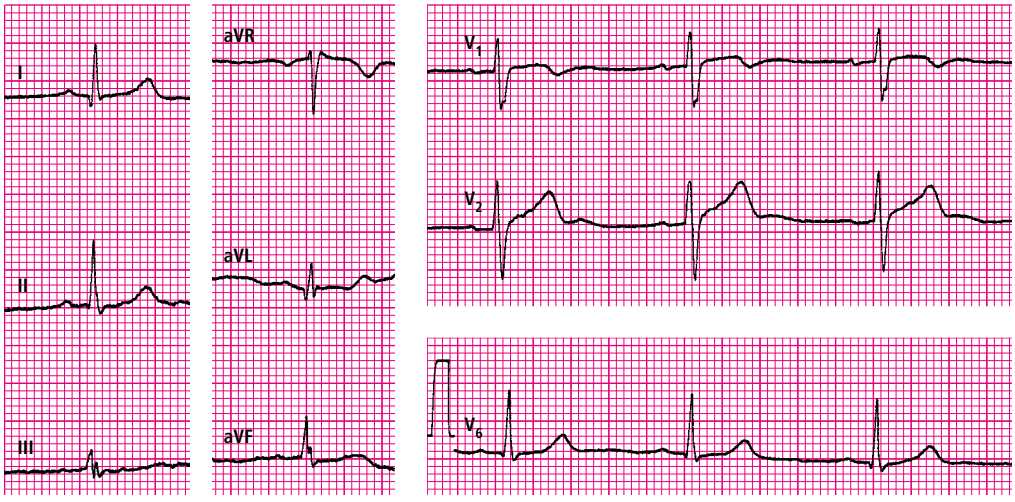

FIGURE 1C

isoenzyme. If a patient takes a drug that is metabolized by this pathway and at the same time prolongs the QT interval (eg, terfenadine or erythromycin), and another physician prescribes the patient another drug metabolized by CYP3A4 (eg, ketoconazole), then both drugs will compete for the same enzyme system. This will result in a significant increase in the concentration of the drug prolonging the QT interval and may cause torsade de pointes. Proton pump inhibitors are also metabolized via this common pathway. Pantoprazole is considered to have low potential for drug–drug interactions, because although it is metabolized by CYP3A4 in the biotransformation phase, conjugation occurs through other metabolic pathways. Numerous studies have reported the absence of interactions between pantoprazole and various other drugs. There are also no data in the literature indicating that proton pump inhibitors directly affect the QT interval; however, it is important to note that the percentage of patients in whom non-antiarrhythmic drugs prolong the QT interval is small, which even allows an assumption that QT prolongation in such cases may indicate latent congenital long QT syndrome. It is also possible that the patient took some other drug (eg, an antiallergic agent) that competed with pantoprazole for access to CYP3A4 (half a year after that incident, he confirmed that he may have sometimes taken cetirizine on his own for allergic rhinitis).

The notched QRS complexes over the inferior wall are not indicative of atrial septal defect, because they are seen in the descending rather than the ascending limbs of the R waves. Such a variant when the QRS complexes are not widened and show no other abnormalities except the notches in the descending limb has been considered normal in the 20th century. However, in recent years, one of the leading research centers in the field of ECG, the Krannert Institute of Cardiology in the United States, has reported that notches in QRS complexes in 2 adjacent leads are a highly specific and more sensitive marker of previous or acute myocardial infarction than the Q waves. Yet this case proves that this ECG feature is not pathognomonic for myocardial infarction because the patient did not show any clinical or echocardiographic features of previous myocardial infarction.

An apparent contraindication to a stress test in a 40-year-old man

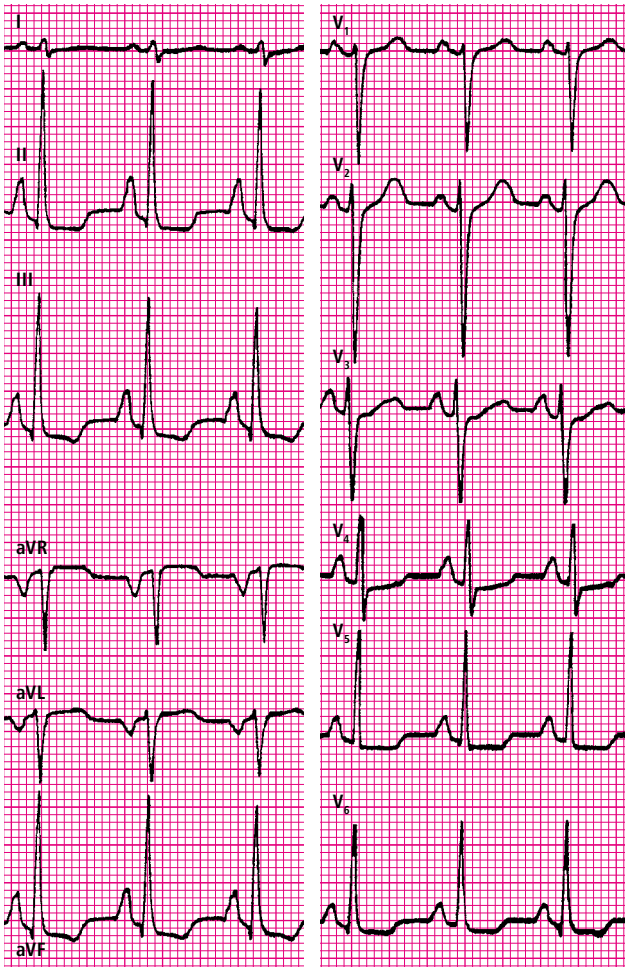

FIGURE 1

This electrocardiogram (ECG; FIGURE 1) was performed in a standing position, using the Mason–Likar system (ie, with the leads placed only on the torso) before a scheduled stress test in a man with atypical chest pain and no abnormalities on routine ECG. The ECG shows sinus tachycardia of 110 bpm, P waves indicative of abnormal conduction within the right atrium (P pulmonale), minor right axis deviation (90°), and an ST-segment depression of up to 1 mm at a distance of 0.06 s from the J point, downsloping in leads II, III, and aVF, parallel to the isoelectric line in leads V_5 and V_6, and upsloping in lead V_4. Of the above features, only the right axis deviation can be associated with a different placement of the limb leads. If such minor right axis deviation with the coexistent q wave in lead III was present in a standard ECG, would it be correct to diagnose left posterior fascicular block? The answer is "no" because right axis deviation meeting the criteria for posterior fascicular block is a nonspecific feature, usually seen in asthenic young men, as well as in patients with chronic cor pulmonale. Therefore, the diagnosis of left posterior fascicular block should be based not only on typical ECG results but also on the coexistent clinical features suggesting left ventricular damage.

As the patient reported no complaints, the physician considered the other abnormal ECG features to reflect increased sympathetic tone and decided that the patient had no contraindications to undergo a stress test. The test was successful and yielded negative results: all the above features normalized already in the first stage of the exercise.

Of note, the ST-segment depression seen in this ECG was caused not only by subendocardial ischemia due to increased sympathetic tone, but also by a negative wave of atrial repolarization (Ta). The latter also affected the ST-segment elevation in leads aVR and aVL.

A 21-year-old man with recurrent palpitations

FIGURE 1 shows a series of electrocardiogram tracings recorded during Holter monitoring in a man with recurrent palpitations. Tracing A was recorded a few months earlier than tracings B and C.

Persistently recurrent episodes of supraventricular tachycardia (tracing B), with the rates ranging from slightly below 140 bpm to 200 bpm, were characterized by negative P waves at a distance of up to 140 ms before the QRS complexes (arrows). A variable shape of the T waves (tracing B), particularly in lead CM_5, is associated both with superimposing ectopic P waves (in different areas, because both episodes of tachycardia are irregular) and with the duration of the preceding RR interval. Moreover, during acceleration of tachycardia, the patient frequently experienced intraventricular conduction disturbances (only one such beat can be seen in FIGURE 1, ie, the first one in tracing B). Between the episodes of tachycardia, particularly at night (tracings A and C), a recurrence of an active ectopic rhythm of 68 to 95 bpm was seen. This electrocardiogram allows an exclusion of: 1) uncommon nodal atrioventricular (AV) tachycardia; 2) AV reentrant tachycardia; and 3) persistent junctional reentrant tachycardia (using an additional pathway), because the rate of ectopic P waves is sometimes too low (below 100 bpm) and they are periodically conducted with second-degree AV block (tracing A). Therefore, the only possible diagnosis is focal atrial tachycardia originating from a very active autonomic pacemaker located in the lower part of the atria.

Antiarrhythmic therapy was ineffective, and within a few months the patient developed clinical features of tachycardia-induced cardiomyopathy. Interesting features of this process are seen in tracings B and C: a change in the shape of the T waves in leads CM_5 and IS (normal or even high in tracing A and asymmetrically negative in tracings B and C) and a significant increase in the amplitude of the R waves in lead IS in tracings B and C (up to 26 mm). The latter feature suggests that persistent tachycardia in this patient led not only to atrial and ventricular dilation but also to myocardial hypertrophy. Tachyarrhythmia and ECG abnormalities resolved after successful ablation of the active pacemaker.

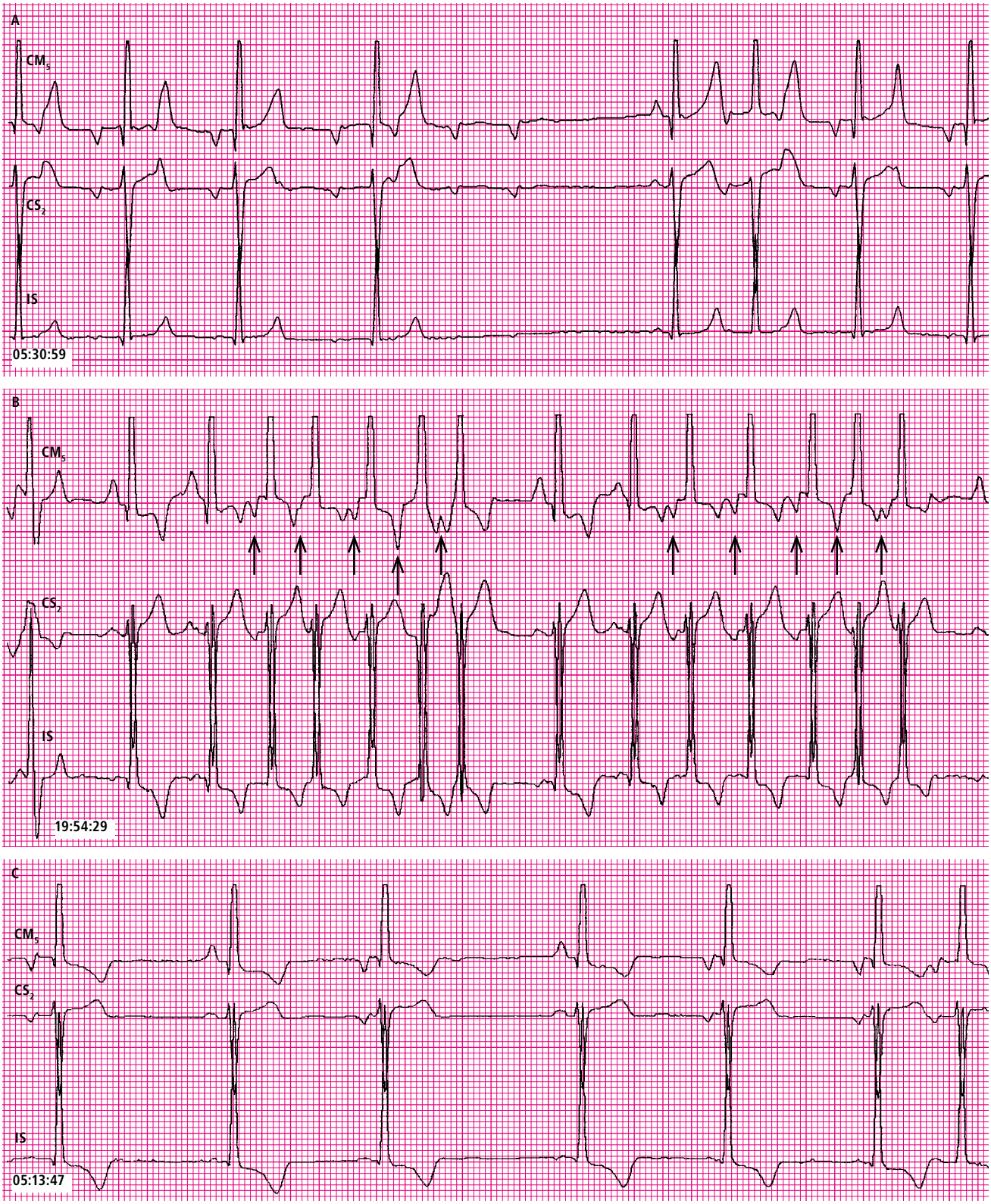

FIGURE 1

A 17-year-old boy with congenital heart disease

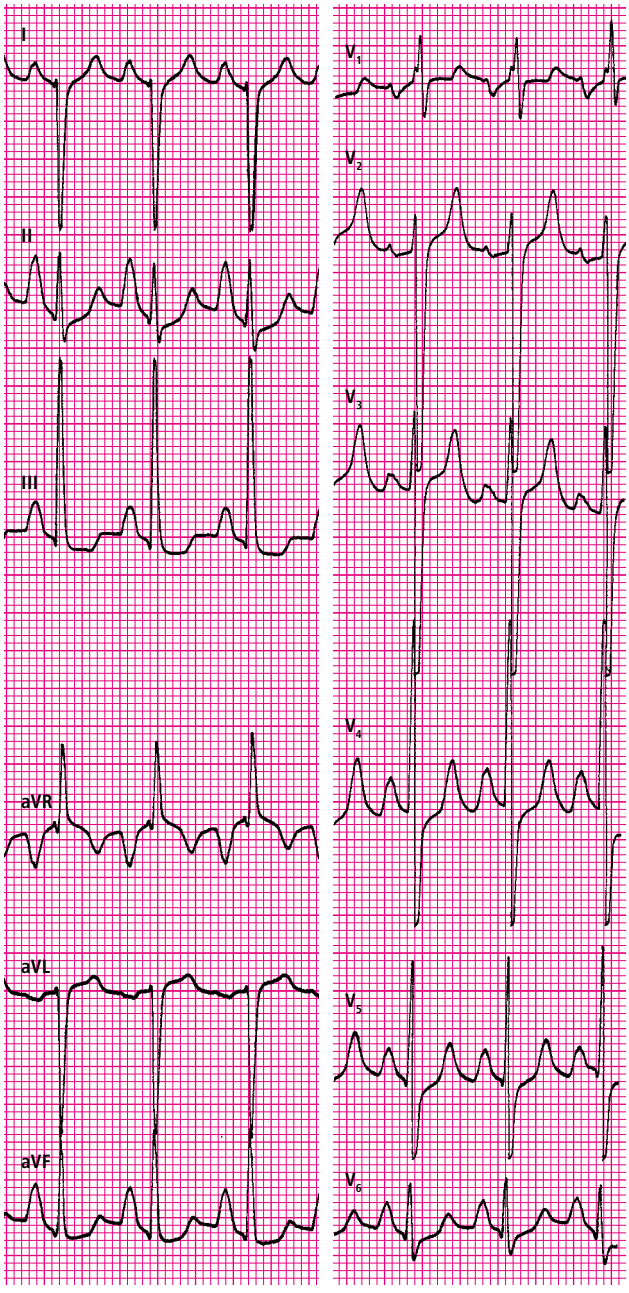

FIGURE 1

This electrocardiogram (ECG; FIGURE 1) was recorded in a 17-year-old boy with congenital heart disease. It shows an interesting feature, namely, exceptionally high P waves.

In 2003, an ECG of an 8-month-old child with tricuspid atresia was published in *Circulation*, with the P waves described as "Himalayan", reaching up to 6 mm in lead II. However, in the present case, they are even higher by 1 mm, and their shape in lead V_1 indicates coexisting left atrial augmentation (hypertrophy?).

Unfortunately, ECG rarely provides sufficient information to diagnose congenital heart disease. However, if no other data are available, a differential diagnosis may be attempted on the basis of the abnormal features seen in this ECG:

1 right axis deviation (approx. 140°, with positive QRS complexes in lead aVR);

2 QRS complexes in lead V_1 fluctuating during respiration, mostly with R>S and the amplitude of the R wave reaching 8 mm, which together with right axis deviation and features of P pulmonale allows an identification of right ventricular hypertrophy;

3 qRS complexes (with R>S) in lead V_6 and a high amplitude of the R waves in the limb leads, with R>S and a high amplitude of the R waves in the limb leads (25 mm in lead III), which may indicate a coexistent left ventricular hypertrophy.

Very high P waves (known as "Himalayan") are seen most often in patients with tricuspid atresia and Ebstein anomaly, sometimes in pulmonary artery stenosis. However, in this case, tricuspid atresia (characterized by left axis deviation) and Ebstein anomaly (characterized by right bundle branch block with a specific multiphase distortion of the end of the QRS complex in lead V_1) can be excluded. Another unlikely possibility is pulmonary stenosis, because it does not explain the coexistence of P mitrale and left ventricular hypertrophy. A possible diagnosis on the basis of all these ECG features is a condition that is extremely rare in adolescents, namely, the transposition of the great arteries, which results in a burden for both the atria and ventricles. In patients with this defect, survival to this age is possible only when specific other defects are simultaneously present (eg, a large atrial septal defect).

An 18-year-old boy with Down syndrome

This electrocardiogram (ECG; FIGURE 1) was recorded in an 18-year-old boy with Down syndrome.

The ECG shows a sinus rhythm of 94 bpm. The P waves are wide (up to 0.16 s) with an amplitude of up to 2 mm in leads I and II, occasionally widely bifid (to 0.06 s in leads V_4 and V_5), and biphasic (+/-) in lead V_1 with a profound negative phase lasting 0.06 s. Such a presentation allows a diagnosis of P mitrale. The PR interval is 0.24 s. Significant left axis deviation can be seen (–70°). The QRS complex in lead V_1 is shaped as qR syndrome with an R wave reaching 8 mm, which together with deep S deflections in leads V_5 and V_6 as well as with typical features of the ST-T complex in leads V_1 to V_3 indicates right ventricular overload. The upper limit of the R-wave amplitude in lead V_5 (26 mm) and the sum of the amplitudes of the S wave in lead III and the largest QRS complex in the precordial leads exceeding 30 mm (a criterion for left ventricular hypertrophy in patients with left axis deviation, indicating left anterior fascicular block, according to the 2009 guidelines of the American Heart Association, American College of Cardiology, and Heart Rhythm Society; in this case, the sum of the amplitudes of S_{III} and QRS in lead V_5 reaches 47 mm), along with typical changes of the ST-T complex in leads V_4 to V_6, indicate the coexisting left ventricular overload. First-degree atrioventricular block, extreme left axis deviation, P mitrale, as well as the overload of both ventricles and the probability of right atrial dilation suggest ostium primum-type atrial septal defect (ASD) with severe mitral regurgitation. Right atrial dilation is probable because of the presence of a narrow q wave in lead V_1 within the QRS complex with features of right ventricular hypertrophy, even though the findings do not generally meet the criteria for P congenitale because the amplitude of the P waves in leads I and II does not exceed 2.5 mm. To establish a full diagnosis, except for P congenitale, features of incomplete right bundle branch block would have to be present (perhaps the initial R in lead V_1 is masked by the Q wave?). However, the above diagnosis is also supported by one additional feature, namely, the notches in the S waves in leads II, III, and aVF, which are equivalent to the notches in the R waves in these leads, seen in patients with ostium secundum-type ASDs.

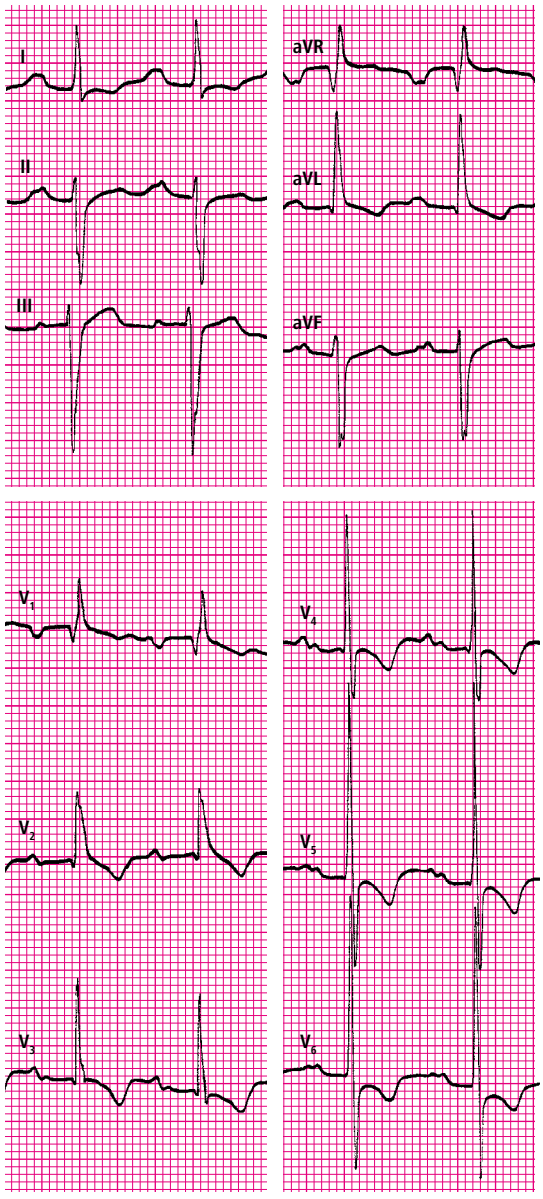

FIGURE 1

Of note, extreme left axis deviation in patients with atrioventricular canal defects, whose "incomplete" type is an ostium primum-type ASD coexisting with a cleft of anterior mitral leaflet (responsible for mitral regurgitation seen in this patient), is not actually a manifestation of left anterior fascicular block, but the result of substantial downward and backward shift as well as underdevelopment of the left anterior fascicle.

Congenital heart disease occurs in approximately 40% of patients with Down syndrome, with ASDs being the most common.

CLINICAL INTERPRETATION

Apparent bradycardia during sleep in a 65-year-old woman with coronary artery disease

The electrocardiogram tracing in FIGURE 1A, recorded during Holter monitoring, shows sinus bradycardia with a rate of 27 bpm. It was recorded before midnight in a patient who had never reported any symptoms suggestive of intermittent abnormalities of cerebral perfusion. The atypical shape of the T waves, which are narrow and peaked, raises the suspicion that in fact they are ectopic P waves that constitute atrial bigeminy blocked within the atrioventricular node.

The tracing in FIGURE 1B, recorded a minute later, confirms the above diagnosis. It shows a minor prolongation of the coupling ectopic P waves with the preceding QRS complexes, which unblocked conduction through the atrioventricular node and revealed premature QRS complexes with aberration of ventricular conduction in the form of right bundle branch block.

What treatment, if any, should be administered for this type of arrhythmia? On the basis of clinical data and the above findings, there are no indications for pacemaker implantation or for further diagnostic workup for sick sinus syndrome.

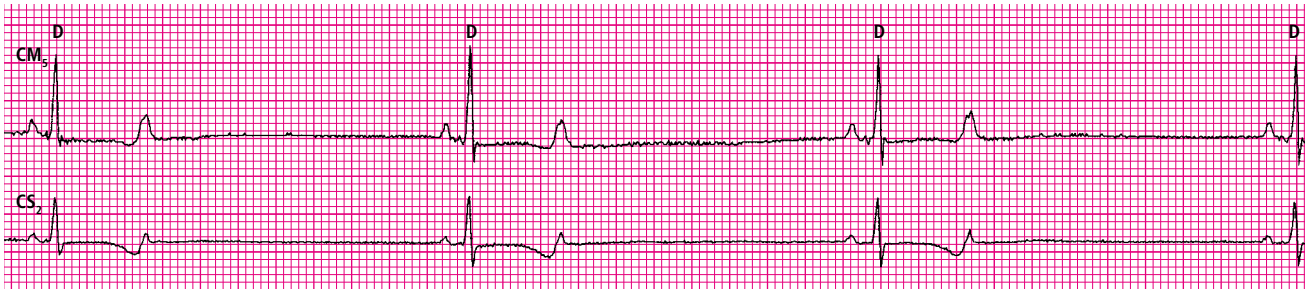

FIGURE 1A

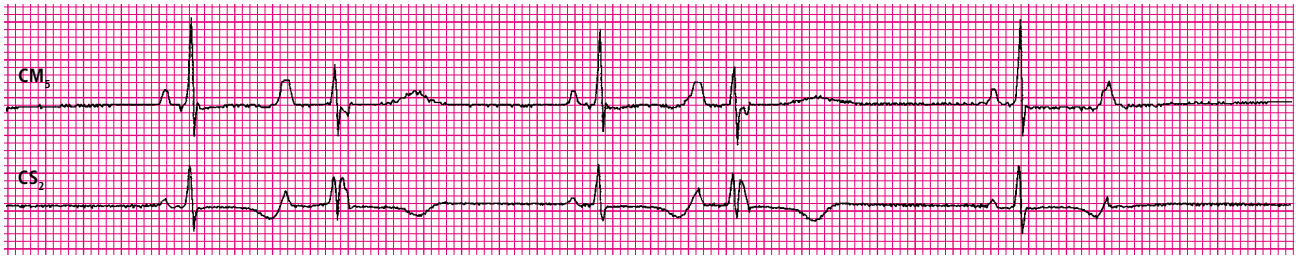

FIGURE 1B