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ATRIAL ECTOPIA ASSOCIATED WITH PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA

M.M.Medvedev

Research, Clinical and Educational Center "Cardiology" FSBEI HE "St. Petersburg State University", Russia, St. Petersburg, 7-9 Universitetskaya emb.

Fragments of Holter monitoring of a 64-year-old patient with paroxysms of supraventricular tachycardia are presented, atrial ectopic activity during tachycardia is recorded, which does not interrupt the tachycardia, but changes the sequence of RR intervals. The possibility of remote analysis of the data of 3-day monitoring of the patient's electrocardiogram in 12-channel is provided.

Keywords: Holter monitoring; supraventricular tachycardia; atrial ectopic beats; atrioventricular conduction; electrophysiology study; radiogrequency catheter ablation

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Corresponding author: Medvedev Mikhail, E-mail: mikhmed@mail.ru

M.M.Medvedev - ORCID ID 0000-0003-4903-5127

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The differential diagnosis of supraventricular tachycardia (SVT) on Holter monitoring (HM) presents a significant challenge in the absence of clearly distinguishable atrial waves, evidence of accessory pathways or atrioventricular (AV) node dissociation into fast and slow conduction zones. However, the presence of P waves does not always permit an unequivocal determination of the type of SVT. While positive P waves in the inferior leads indicate atrial tachycardia, the presence of negative P waves with a downward-upward vector can lead to different mechanisms of SVT, depending on the ratio of PR to RP intervals and the absolute values of these intervals.

If negative P waves are detected in the leads II, III and aVF closer to the preceding QRS complex than to the following one, we are inclined to consider SVT as paroxysmal orthodromic AV reentry tachycardia due to the presence of Wolf-Parkinson-White (WPW) syndrome. However, it should not be overlooked that atrial tachycardia with delayed AV conduction can look exactly the same. In our opinion, it is impossible to differentiate these tachycardias based on an ECG recorded at the "peak" of the paroxysm without having records of the onset and termination of the paroxysm.

A similar situation occurs when negative P waves are closer to the following QRS complex than to the preceding one. In most cases, this pattern masks atrial tachycardia. However, paroxysmal orthodromic AV reentry tachycardia may appear this way if the RP interval is prolonged due to retrograde conduction via "slow" additional conduction.

The shape of the P waves must also be considered. For example, the presence of narrow (already sinus P waves) negative P waves in the inferior leads may indicate that the atria are covered by the excitation of the AV node and facilitate a correct diagnosis. Such P waves are characteristic of paroxysmal AV nodal reentry tachycardia and paroxysmal antidromic AV reentry tachycardia, in which the excitation propagates anterogradely along an additional conduction pathway and retrogradely along AV node. The identical P waves can be recorded in AV nodal tachycardia and atrial tachycardia, with the source of excitation located in close proximity to AV node.

A correct diagnosis is greatly facilitated if ECG or an ECG with sinus rhythm indicates WPW syndrome or signs of AV nodal dissociation into fast and slow conduction zones. The criteria for the diagnosis of WPW syndrome are well established, whereas signs of dissociation of the atrioventricular node from sinus rhythm are less common and are usually interpreted as first-degree AV block. AV node dissociation is indicated by signs similar to those found on a programmed stimulation in the form of a discontinuity in the AV conduction curve. In a sinus rhythm ECG, if we observe a rapid increase in the PQ interval of 80 ms or more from one P-QRS-T complex to the next, followed by an equally rapid decrease, we suspect that the fast channel of AV node is blocked and conduction has occurred via the slow channel, which has then returned to its original position. This can be validated using a PQ interval diagram or a histogram of the distribution of this interval. Unfortunately, these options are not available in all HM systems. However, it should be emphasized that the presence of evidence of WPW or AV nodal dissociation is not a guarantee that a patient's SVT paroxysms are related to this arrhythmic substrate.

Nevertheless, it can be stated that ECG and HM allow determination of the type of SVT without electrophysiological study in most cases. In some cases, it helps to form an idea of the "details and peculiarities"

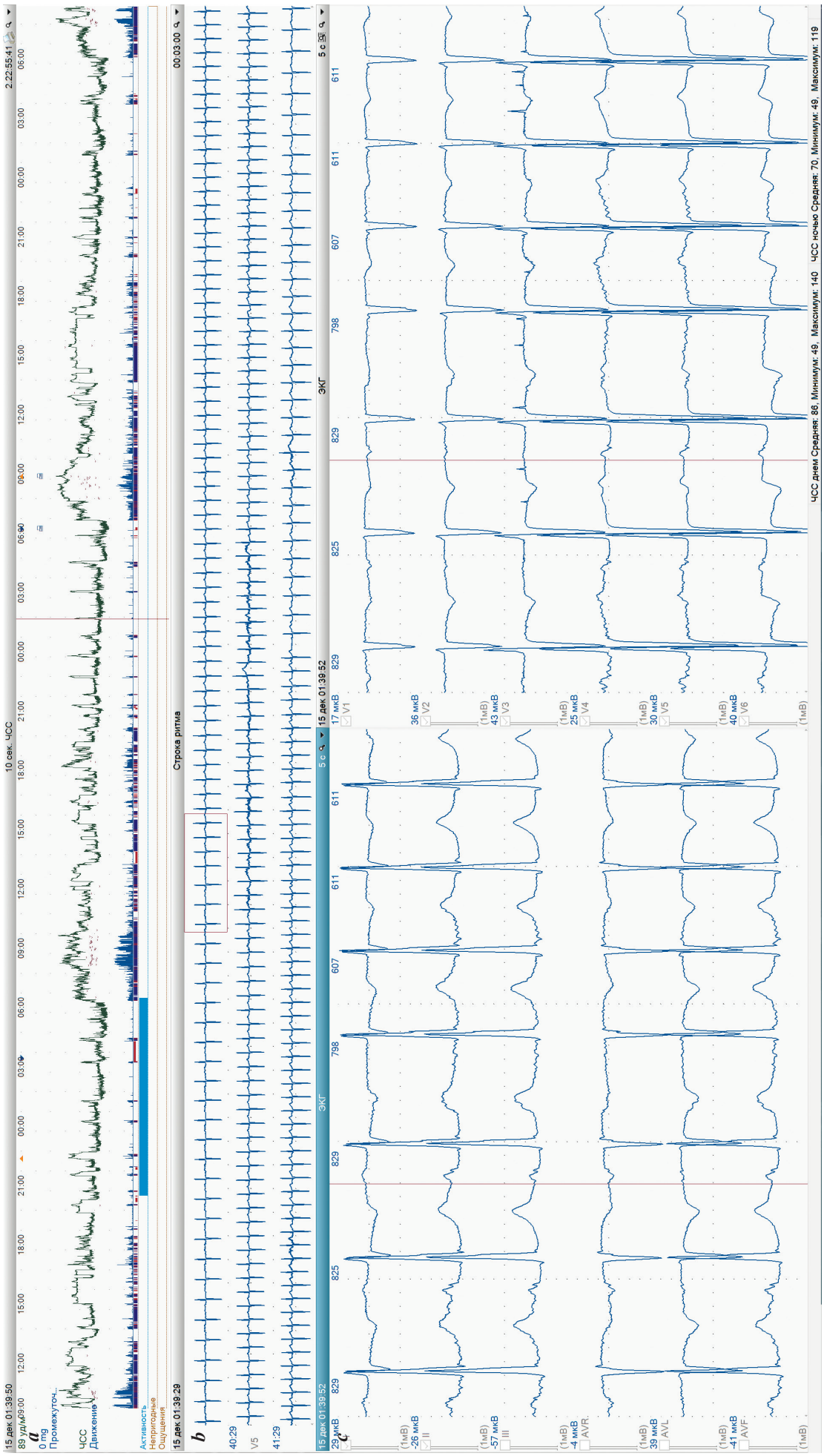

Fig. 1. Fragment of three-day electrocardiogram monitoring in twelve standard leads from patient B. 64 years old: a - HR graph (the cursor position indicates one of the tachycardia paroxysms), b - rhythm line of three minutes duration (the rectangular cursor marks the onset of the paroxysm), c - ECG fragment corresponding to the rectangular cursor on the rhythm line (after three sinus complexes P-QRS-T, a paroxysmal tachycardia with a rate not exceeding 100-110 bpm begins).

(as E.A. Berezniy said) of the electrophysiological mechanisms of arrhythmia. We therefore invite you to analyze what we consider to be very interesting HM data or fragments thereof, presented in this publication. We will present our interpretation of the electrophysiological features of SVT recorded during this monitoring in the next issue of the journal.

We obtained the recording of three-day ECG monitoring in twelve standard leads from patient B. 64 years old. At the time we analyzed the recording, we had no information about the patient. The first "encounter" with the monitor was nothing unusual. However, it should be noted immediately that the combination of persistent first-degree block AV with SVT paroxysms occurring at a comparatively low frequency was thought-provoking. One of the SVT paroxysms was characterized by ectopic atrial activity that did not interrupt the tachycardia but caused some changes in the interval dynamics (Figure 1-3). This elicited quite natural questions from the doctor recording the monitor and this was the reason for the consultation.

Determining the nature of SVT would not present any major difficulties. Nevertheless, it is the "details and

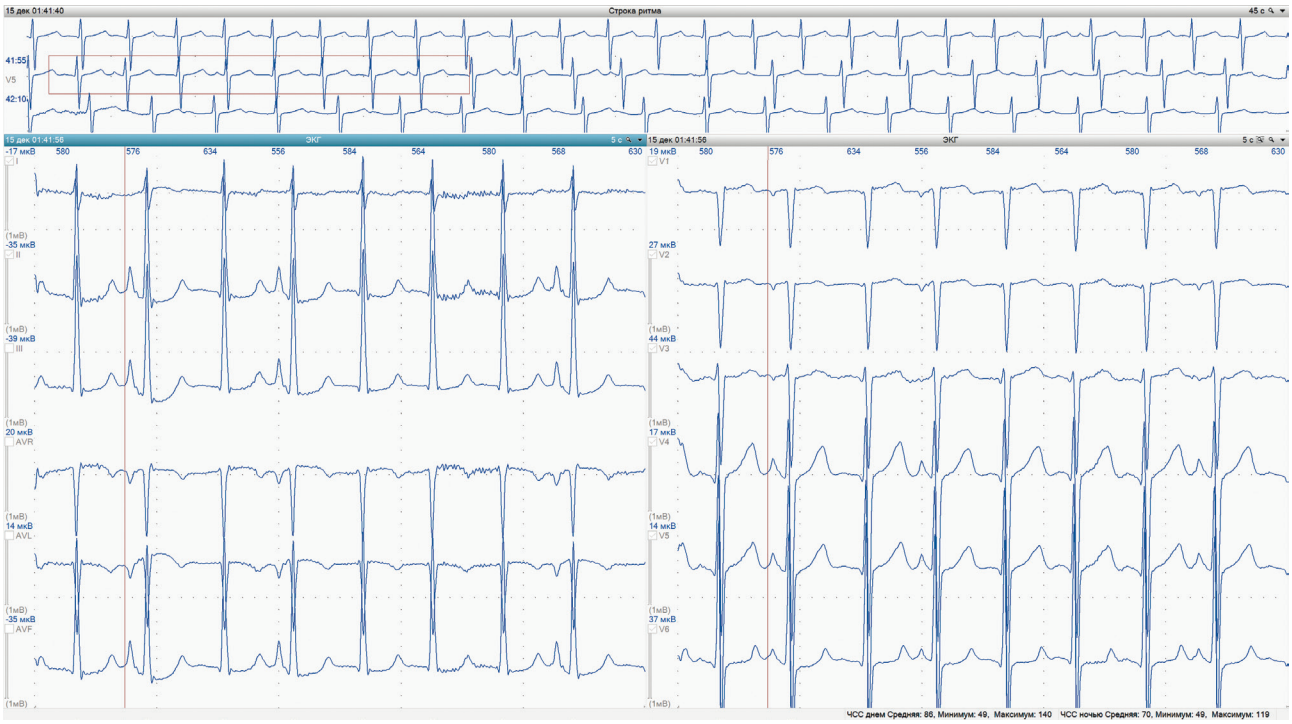

Fig. 2. Fragment of electrocardiogram for monitoring patient B. 64 years old: the ectopic activity of the atria does not interrupt the paroxysmal tachycardia but does influence RR intervals.

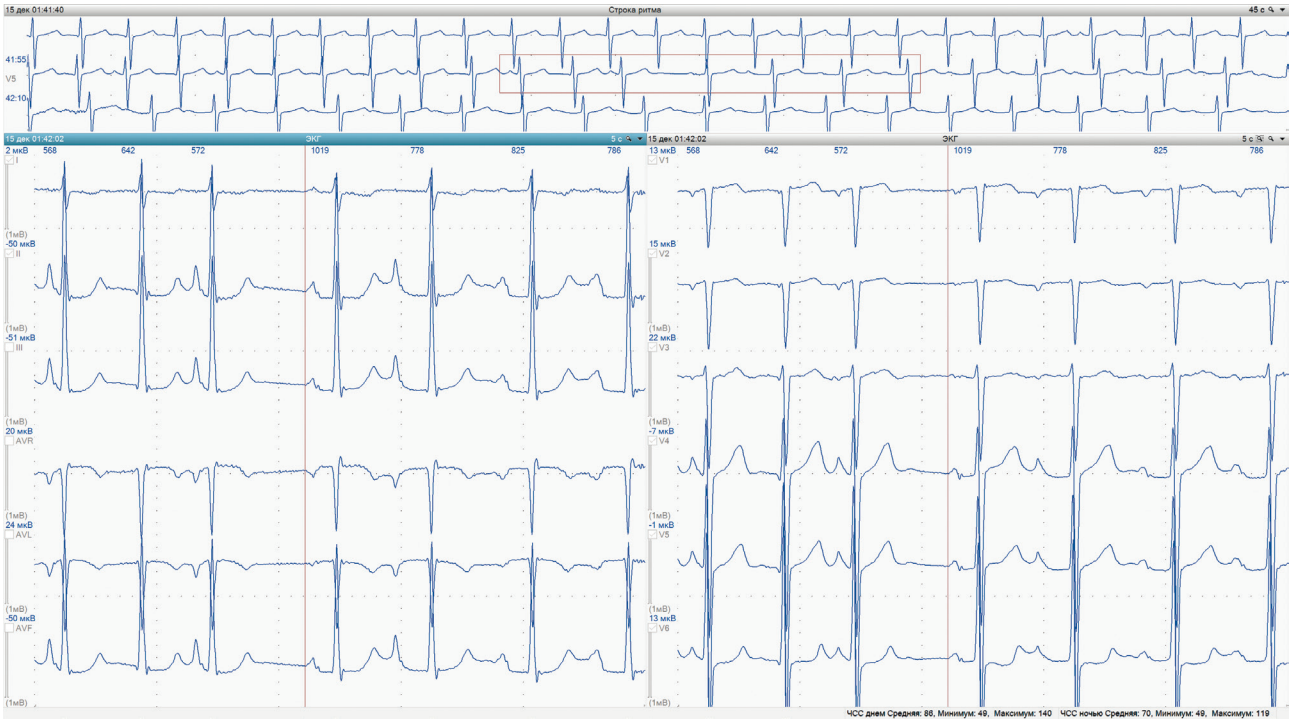

Fig. 3. Fragment of electrocardiogram monitoring of patient B. 64 years old: end of paroxysmal tachycardia with restoration of sinus rhythm.

peculiarities“ of this recording that we believe are of undeniable interest. We have noted ventricular extrasystoles on several occasions without interruption of the course of SVT. We observe for the first-time recordings of atrial ectopy that do not interrupt SVT but alter the sequence of RR intervals. No such descriptions could be found in the literature either. However, it must be emphasized again that the salient features of this SVT are unlikely to have a significant impact on further treatment strategy.