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SUPRAVENTRICULAR TACHYCARDIA AND ATRIAL ECTOPIC BEATS: ELECTROPHYSIOLOGICAL FINDINGS IN A PATIENT AFTER PREVIOUS SLOW PATHWAY ABLATION

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In this case report we present electrophysiological features of supraventricular tachycardia in a 64-year-old patient after previous slow pathway catheter ablation; ECG features of the tachycardia and results of electrophysiological testing are discussed.

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In the first issue of the "Journal of Arrhythmology" of 2022, the article "Atrial ectopy during paroxysmal supraventricular tachycardia" was published. The readers of the journal were granted access for remote analysis of the Holter electrocardiogram monitoring (HEM) data of a 64-year-old female patient. In the course of the 72-hour HEM were registered paroxysms of supraventricular tachycardia, which in our opinion had several interesting electrophysiological particularities.

Tachycardia initiation with an atrial ectopic beat (AEB) with a marked prolongation of PQ interval allowed us to suggest their conduction through the slow pathway of the atrioventricular node and define it as an atrioventricular node reentrant tachycardia (AVNRT). At the same time, it was impossible to ignore that during sinus rhythm, the patient had atrioventricular (AV) block 1 degree, and a PQ interval length of 300 ms. Interestingly, tachycardia heart rate without physical activity did not increase to more than 115 beats per minute. This certainly required an explanation. It was clear that presence of AV block 1 degree and the long tachycardia cycle length can be associated as a result of antiarrhythmic drug administration, as well as by a previous radiofrequency ablation procedure.

After request information, it was possible to know that the patient underwent AVNRT substrate ablation. It is likely that this procedure is associated with both a relatively low conduction velocity in the slow pathway of the AV node, and, consequently, a low heart rate AVNRT, as well as the presence of 1st degree AV block during sinus rhythm, due to the presence of a zone of immediate conduction located below the re-entry loop.

With the HEM of patients with AVNRT and during sinus rhythm, in some cases, it is possible to identify signs of AV node dissociation into zones of fast and slow conduction. For these purposes, it is possible to use the histogram of the distribution of PQ intervals, the scattergram of the dependence of the PQ interval on RR and the graphic of the PQ interval. In this case, despite the pronounced lengthening of the PQ interval, signs of AV conduction dualism were not revealed, which, of course, does not exclude its presence. These features can be found in the analysis of the PQ intervals of atrial ectopic beats (Fig. 1).

Fig.1a shows a single AEB with a subsequent echo-beat, which does not correspond to the data of automatic analysis. The QRS complex of AEB beat is preceded by an RR interval of 840 and not 654 ms. The P wave is indicated by a red arrow, and the PQ interval is about 470 ms due to excitation through the AV node slow pathway.

QRS complexes begin with pseudo-q-waves (blue arrows), which actually reflect the beginning of atrial excitation as a result of retrograde conduction excitation along the fast pathway of the AV node. Due to a pronounced decrease in conduction velocity below the re-entry loop, excitation spreads to the atrium earlier than to the ventricles (the RP interval is negative). In the Fig.1b, the AEB conduction is carried out through the fast pathway in the AV node. However, the value of the PQ interval exceeds 260 ms, which is associated with a slowdown in AV conduction below the re-entry loop.

The most interesting feature of the AVNRT of this patient, it is the presence of atrial ectopic activity, which does not interrupt the course of tachycardia, but affects the

duration of RR intervals (Fig. 2). It is obvious that these P waves are not conducted to the ventricles, since the value of the minimum PQ of atrial ectopic beat interval does not exceed 130 ms, which is significantly less than the PQ intervals registered during sinus rhythm, and contradicts the idea of the presence of a zone of delayed conduction located below the re-entry loop.

The Fig.2a clearly shows that the RR intervals, including ectopic P waves, are substantially shorter than the following RR intervals. The maximum difference in their value exceeds 70 ms. AEBs conducting to the AV node outside the period of its full refractoriness (AEBs occur quite late in the tachycardia cycle) cause tachycardia resetting with further prolongation of the slow conduction.

The activation delay is due to the fact that the slow pathway has already left the absolute refractory period, but is still in the functional refractory period. On the other hand, due to the depolarization of the atria, the AEB retrograde conduction to the atria is blocked before the QRS

complexes, following after the ectopic P waves, there are no pseudo-q-waves (Fig. 2b). The mechanism of this phenomenon is quite obvious. After an atrial extrasystole, the atria are in the refractoriness, which hinders their coverage as a result of retrograde conduction. It is important that this does not interrupt the re-entry cycle and the tachycardia continues. However, the end of the paroxysm is preceded by an AEB.

To judge whether this played any role in the restoration of sinus rhythm, in our opinion does not seem possible. The listed features of the AVNRT are illustrated by the diagram shown in Fig.2c.

The patient was referred for repeated catheter ablation of the slow AV pathway. During the electrophysiological study, the programmed stimulation of the atria confirmed the presence of slow AV conduction, tachycardia was triggered by single atrial stimuli (Fig.3). Single atrial extrastimuli applied during tachycardia out of the refractoriness of the His bundle system led to a “reset” of tachy-

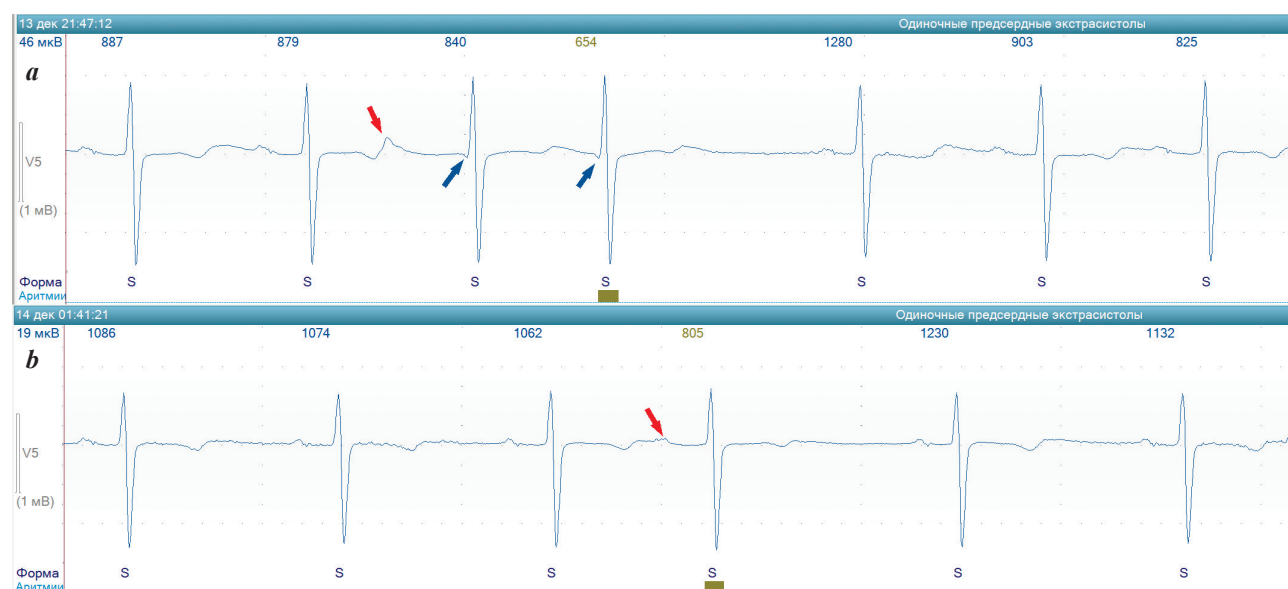


Fig. 1. Atrial ectopic beats with conduction through the slow (a) and fast (b) pathways of the AV node. See explanation in the text.

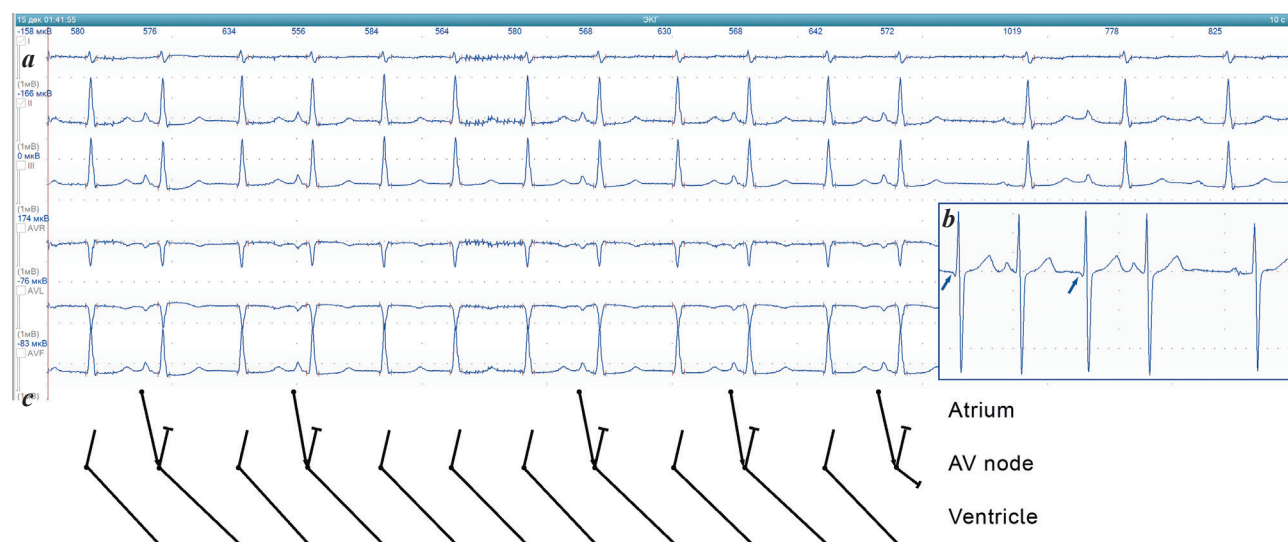


Fig. 2. Atrial ectopic beats during AVNRT: a - a fragment of Holter monitoring (limb leads are shown), b - the lead V5 (pseudo-q-waves are clearly visible - blue arrows), c - the scheme of AVNRT and atrial ectopic beats. See explanations in the text.

cardia with a prolongation of the subsequent RR interval, while the extrastimuli during the refractoriness of the His bundle did not affect the tachycardia cycle (no resetting). A

single radiofrequency application in the posterior septal region of the right atrium resulted in the elimination of slow pathway and rendered the tachycardia non-inducible.

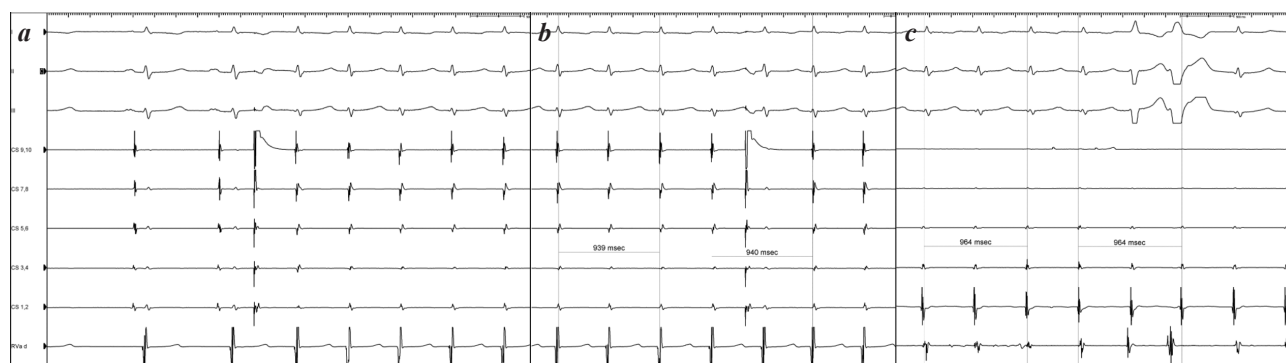


Fig. 3. Intracardiac electrophysiological study. CS - electrograms from a decapolar catheter in the coronary sinus. RVa d - electrograms from a catheter in the right ventricle apex. (a) - induction of a typical AVNRT by a single atrial extrastimulus; (b) a single extrastimulus during tachycardia during the refractory period of His bundle does not reset the tachycardia, indicating the absence of atrial participation in the tachycardia circuit; (c) - a paired premature ventricular beat caused by catheter oscillations in the right ventricle, the first ectopic beat falls within the refractory period of the His bundle, indicating the absence of ventricular myocardium participation in the tachycardia circuit; thus, the tachycardia circuit is entirely within the AV junction, which confirms the AVNRT mechanism. Note the “pseudo-q-waves” in lead III in beats 3, 5, and 6 in panel (a), as well as in panels (b) and (c).