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UNUSUAL FINDINGS DURING TRANSESOPHAGEAL ELECTROPHYSIOLOGY STUDY

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The article presents the results of a transesophageal electrophysiological study of a 39-year-old patient with a combination of an accessory pathway and dissociation of the atrioventricular node into fast and slow conduction zones. The criteria for identifying slow anterograde conduction along the accessory pathway and a rare mechanism for inducing paroxysmal reciprocal atrioventricular nodal tachycardia are discussed.

Key words: transesophageal electrophysiology study; accessory pathways; Wolff-Parkinson-White syndrome; dissociation of the atrioventricular node into fast and slow conduction zones; double atrioventricular conduction; paroxysmal reciprocal atrioventricular nodal tachycardia

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The examination and treatment of patients with the Wolff-Parkinson-White (WPW) phenomenon and syndrome have been extensively documented in numerous publications. In recent years, particular attention has been focused on the non-invasive and invasive risk assessment of patients with manifest, intermittent, and latent WPW phenomenon [1-3]. We have published a series of observations highlighting the potential of transesophageal (TE) electrophysiological study (EPS) in the risk stratification of patients with WPW phenomenon and syndrome [4, 5]. One such observation discusses the validity of diagnosing WPW phenomenon in a patient whose ventricular dyssynchrony, associated with the presence of an accessory pathway (AP), resulted in a marked reduction in the left ventricular ejection fraction (LVEF) [5].

In patients with WPW syndrome (or those with concealed APs exhibiting only retrograde conduction), the occurrence of paroxysmal tachycardia in early adulthood is more typical compared to patients with zones of fast and slow conduction in the atrioventricular (AV) node. Thus, knowing the onset age of palpitations in a patient allows us to preliminarily hypothesize the anatomical substrate responsible. However, exceptions to this rule undoubtedly exist. Below, we present the results of a TE EPS.

A 39-year-old patient, G., sought medical care at the North-West Center for Diagnostics and Treatment of Arrhythmias. The primary reason for the visit was to adjust antihypertensive therapy. During anamnesis collection, it was revealed that the patient had been experiencing brief episodes of rhythmic palpitations with sudden onset and termination since childhood. These episodes were self-terminated or resolved using vagal maneuvers but had never been captured on an electrocardiogram (ECG) or Holter ECG monitoring. As a result, the patient was advised to undergo a TE EPS.

Before initiating TE EPS, the patient presented with sinus tachycardia at a heart rate of 90-105 bpm. The P-wave width was 100 ms, the PQ interval measured 150 ms, the QRS complex width was 90 ms, and the QT interval duration was 350 ms (Figure 1). A contour analysis revealed smoothing of the ascending segment of the R wave in leads V4-V5 (where the onset of the QRS complex was most distinct), the presence of a broad Q wave in lead III, and an almost isoelectric onset of the QRS complex (lasting up to 10 ms) in the right precordial leads. This isoelectric initiation of the QRS complex in certain precordial leads may suggest that ventricular excitation begins from a single focal point rather than multiple areas within the Purkinje fiber distribution. Such an initial depolarisation pattern is typically observed in ventricular ectopy or anterograde conduction along an AP.

Clearly, the patient did not exhibit the "classic" Wolff-Parkinson-White syndrome, as evidenced by a normal PQ interval, absence of a delta wave, normal QRS complex width, and unaltered repolarisation. However, this pattern may occur in the presence of a slow-conducting accessory pathway, where excitation via the AP engages only a very small portion of the ventricular myocardium. The criteria for identifying such APs were detailed in a prior study [6]. In that work, the authors used coherent summation of QRS complexes—traditionally applied to detect late ventricular potentials—to identify low-amplitude potentials preceding the QRS onset and developed quantitative criteria for diagnosing "concealed anterograde conduction via an AP."

We also employed the late ventricular potential analysis option to investigate signs of anterograde conduction via an AP in this patient. The results are presented in Figure 2. In lead X (Frank lead system), the very gradual onset of the QRS complex is clearly visible, and in leads Y and Z, low-amplitude potentials, corresponding temporally to the start of the QRS complex, can be observed. These can be interpreted as equivalents of a minimally expressed delta wave. The delta wave is most distinct in the filtered QRS complex. Thus, ECG analysis using the coherent summation method suggested the presence of an AP with slow anterograde conduction in this patient. This hypothesis, of course, required further confirmation.

Before initiating transesophageal cardiac stimulation (TECS), a transesophageal ECG was recorded and displayed instead of lead V3. The P waves on the transesophageal ECG were of considerable amplitude and biphasic in nature (Figure 3). The sinus node recovery time, determined using orthorhythmic stimulation at a frequency of 150 impulses/min, voltage of 14 V, and pulse duration of 20 ms, was measured at 900 ms, with a corrected value of 250 ms (Figure 4). It is essential to note that the Holter ECG system used to record

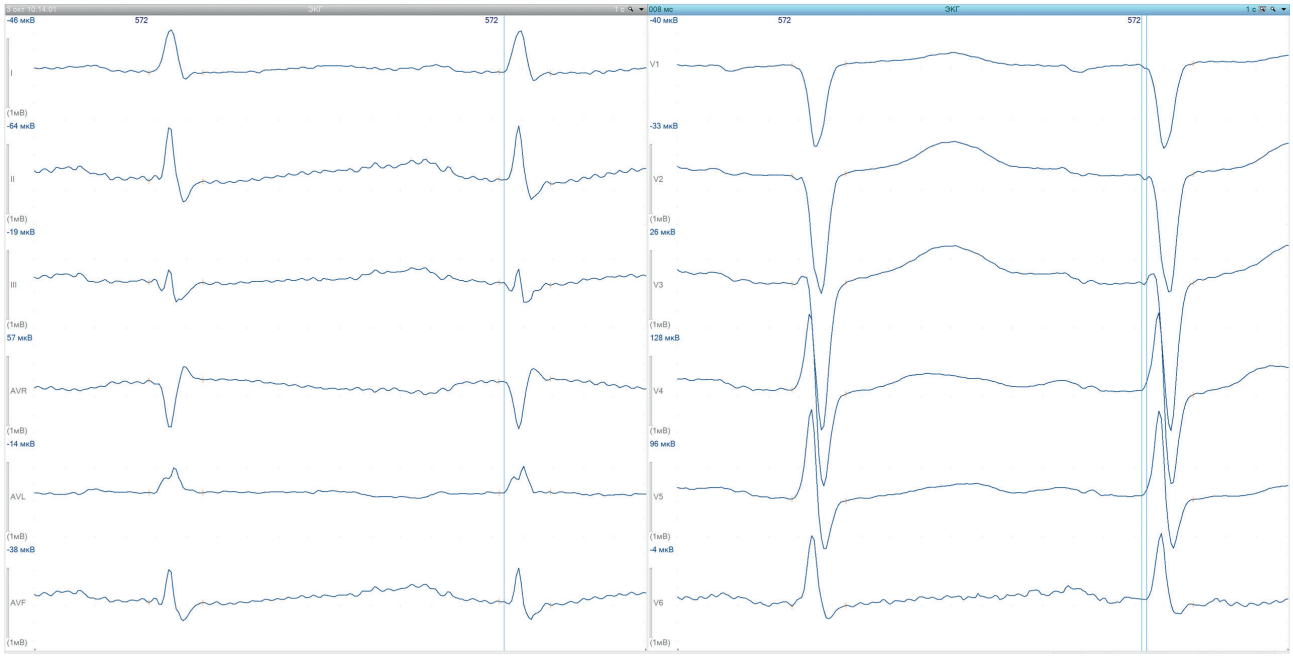

Figure 1. Fragment of Holter ECG monitoring of patient G. in the standard twelve leads, recorded prior to the study. Explanations in the text.

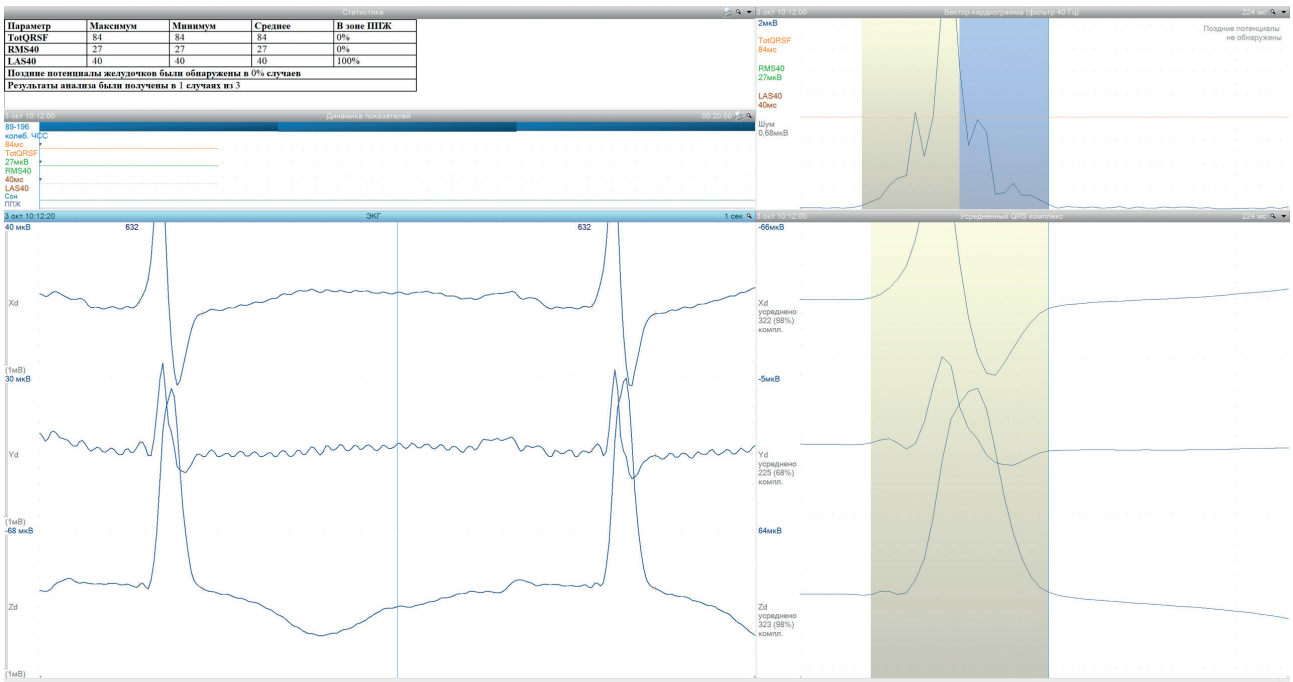

Figure 2. Application of the ventricular late potential detection option to identify signs of anterograde conduction via the accessory pathway (AP) in patient G. The Frank leads are shown (bottom left), along with the result of coherent averaging of QRS complexes (225 to 323 complexes) until the required noise level was achieved (bottom right), and the filtered QRS complex (top right). Explanations in the text.

the cardiac signal is not designed for transesophageal electrophysiology study (TE EPS) data analysis. Consequently, it may incorrectly classify TECS impulses as QRS complexes. Therefore, the numerical results should be interpreted with caution, and particular attention should be paid to the placement of small red markers indicating where the system identified the start and end of QRS complexes or stimulation impulses.

During programmed TECS, the basic rhythm was set at 100 impulses/min, with a testing impulse delay of St1-St2 = 340 ms, St1-R1 interval = 200 ms, and St2-R2 interval = 260 ms (Figure 5). The QRS complex induced by the testing impulse was significantly different from both the basic stimulation QRS complexes and the spontaneous sinus rhythm QRS complexes. Notably, the QRS complex no longer exhibited even minimal signs of pre-excitation. This was most evident in lead V3, where the R-wave amplitude tripled. This absence of anterograde conduction via the AP can serve as a diagnostic criterion in further analysis. It is crucial to highlight that the effective refractory period (ERP) of the AP was at least 340 ms. Further prolongation of the testing impulse delay was not conducted, as it was unlikely to influence therapeutic strategies.

When the testing impulse delay was reduced in 10 ms increments to 300 ms, the St2-R2 interval increased to 270 ms, and an echo beat was observed with an RR interval of 330 ms (Figure 6). A retrograde P' wave of sufficient width and negative polarity was clearly visible in the inferior

Figure 3. Registration of the transesophageal electrogram of patient G. (displayed in place of lead V3). Explanations in the text.

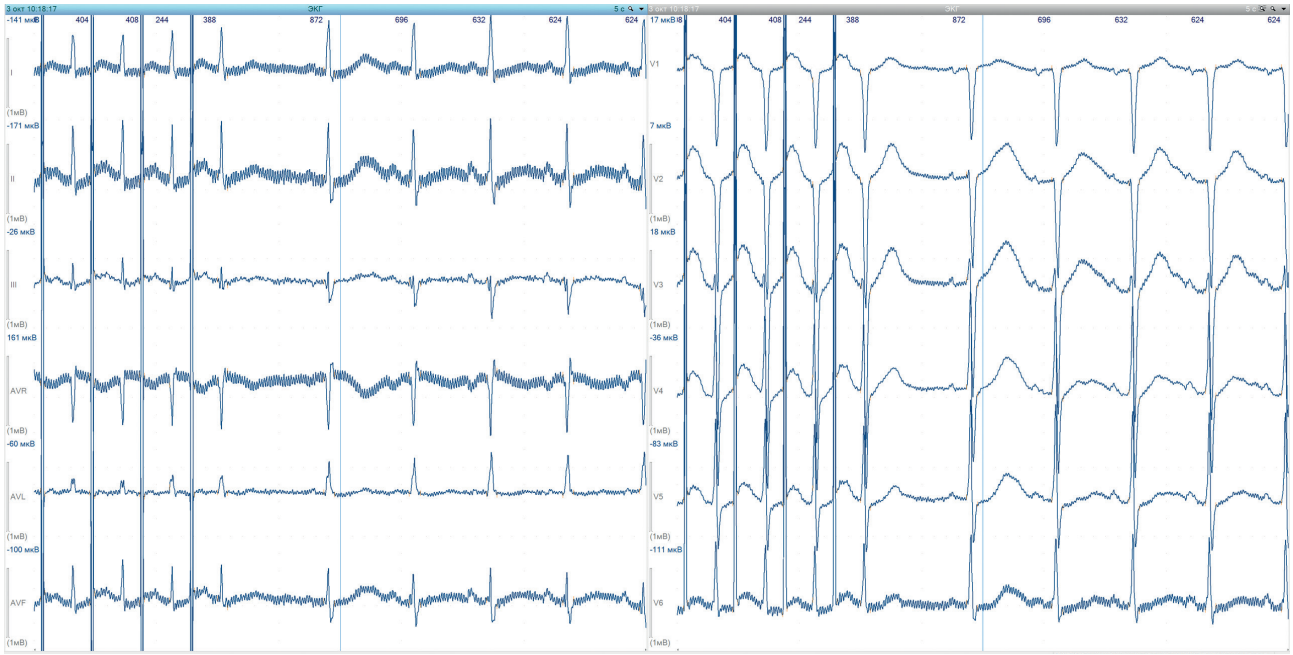

Figure 4. Conducting orthorhythmic cardiac pacing (CP) for determining the sinus node recovery time in patient G. Explanations in the text.

leads, with an RP' interval of approximately 120 ms. This indicates that the testing impulse was conducted through the AV node without AP involvement (as the AP was in a refractory state) and subsequently spread retrogradely to the atria via the AP, which had exited its refractory period. The RR interval of 330 ms suggests the theoretical possibility of inducing paroxysmal orthodromic reciprocating AV tachycardia with a rate of approximately 180 bpm.

When the St1-St2 interval was reduced to 290 ms, the St2-R2 interval increased to 280 ms, and no echo beats were observed. A subsequent reduction in the testing impulse delay to 280 ms resulted in the induction of tachycardia comprising four narrow QRS complexes (Figure 7). The tachycardia rate exceeded 200 bpm, and no distinct retrograde P waves were visible. This raised the possibility of paroxysmal AV nodal reciprocating tachycardia (AVNRT). Typically, AVNRT is induced when a testing impulse conducts to the ventricles via the slow AV node pathway and returns to the atria via the fast pathway, completing a re-entry loop. However, in this case, such a mechanism was excluded because the St2-R2 interval was 300 ms, and its prolongation with decreasing testing impulse delays occurred gradually without abrupt jumps or discontinuities in the AV conduction curve.

An alternative mechanism for AVNRT induction involves double AV conduction, where a single P wave or stimulation impulse produces two QRS complexes due to conduction via both the fast and slow AV node pathways. This induction mechanism has been previously documented during endocardial electrophysiology studies [7]. It appears that this was the mechanism observed in this patient. Supporting this interpretation are pseudo-Q waves in the

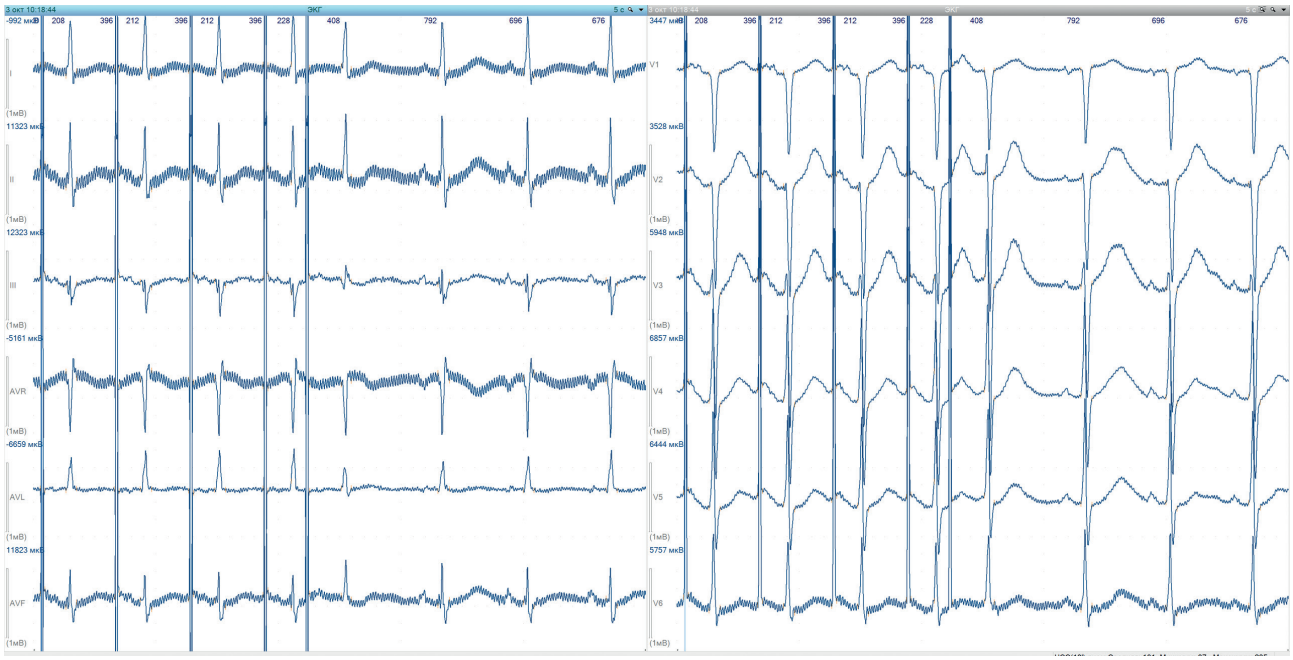

Figure 5. Result of programmed CP in patient G. with a test stimulus delay of 340 ms. Explanations in the text.

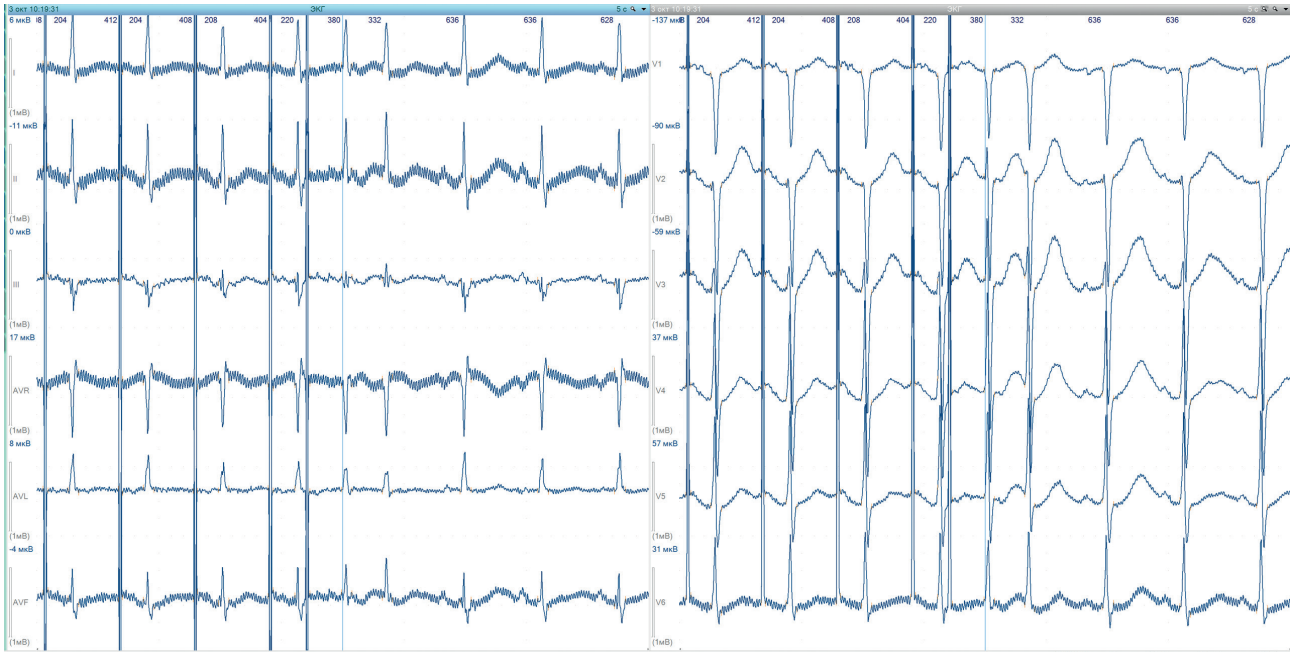

Figure 6. Result of programmed CP in patient G. with a test stimulus delay of 300 ms: recording of an echo beat. Explanations in the text.

second to fourth QRS complexes of the tachycardia, most prominently visible in lead V3. These pseudo-Q waves are, in fact, narrow retrograde P' waves originating from the AV node and propagating concentrically through the atria. Their position preceding the QRS complex (negative RP' interval) indicates that re-entry excitation conducts retrogradely faster than anterogradely.

This tachycardia pattern, characterized by four narrow QRS complexes, was also observed during programmed TECS with testing impulse delays ranging from 280 to 260 ms. This establishes the presence of a tachycardia zone between 290 and 260 ms, although the nature of the tachycardia requires further clarification via transesophageal ECG recording and RP' interval assessment. At an St1-St2 interval of 250 ms, the ERP of the AV node was reached.

During Wenckebach point determination (Figure 8), the value was measured at 220 impulses/min, inducing a brief tachycardia episode exceeding 200 bpm with wide QRS complexes characteristic of complete right bundle branch block. In the figure, the left blue arrow marks an impulse that failed to conduct to the ventricles, while the right arrow indicates an impulse that induced tachycardia, likely through double conduction. A transesophageal ECG recording during tachycardia was not obtained (the red arrow indicates electrode V3 disconnection).

By manually delivering three impulses, we induced a sustained paroxysm of tachycardia (Figure 9). The mechanism of tachycardia induction appears to follow the "classic" pattern: the second impulse propagated via the fast conduction zone of the AV node, while the third impulse

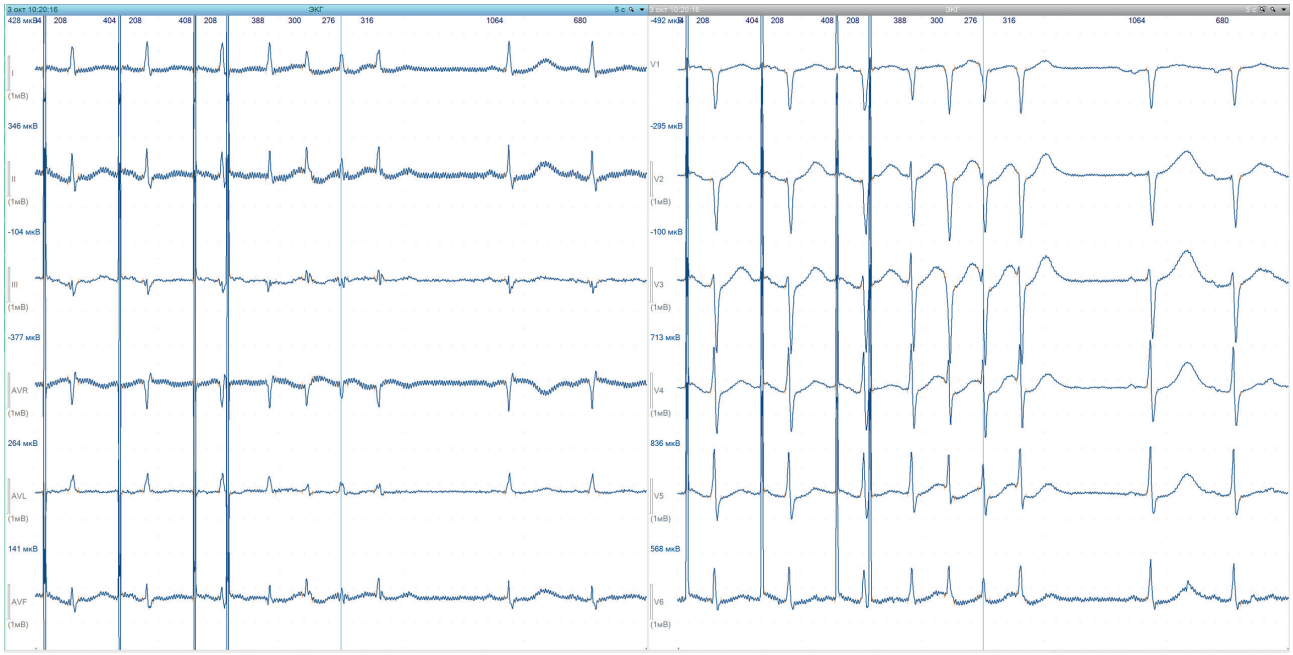

Figure 7. Result of programmed CP in patient G. with a test stimulus delay of 290 ms: induction of tachycardia comprising four QRS complexes. Explanations in the text.

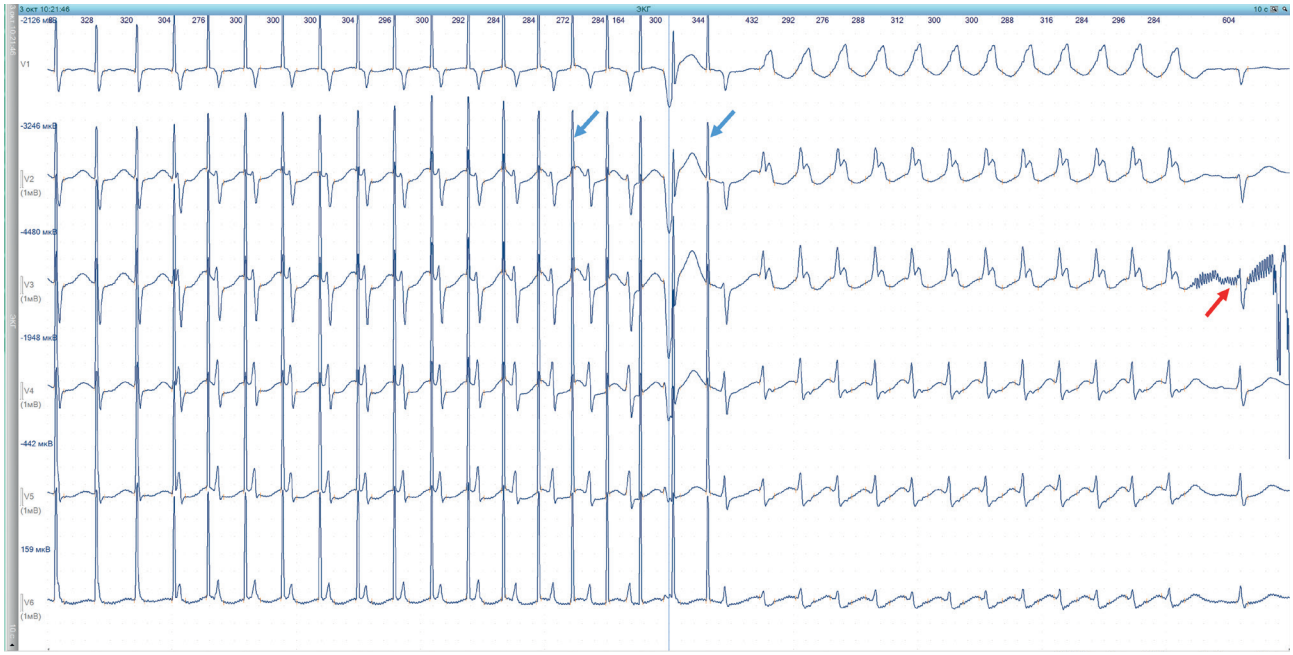

Figure 8. Result of Wenckebach point determination in patient G.: induction of tachycardia with a pattern of complete right bundle branch block (RBBB) at a frequency exceeding 200 bpm. Explanations in the text.

conducted via the slow conduction zone, completing the re-entry loop. An unusual signal morphology was noted in lead V3, which resulted from its disconnection. This was intentionally left disconnected to allow for a quicker recording of the TE ECG in the event of short paroxysms.

The induced paroxysm, with a right bundle branch block (RBBB) pattern, was sufficiently prolonged to permit the recording of a TE ECG (Figure 10). The RP' interval (noted in lead V3) measured 60 ms, confirming the diagnosis of paroxysmal reciprocating AVNRT. Interestingly, attempts to terminate the paroxysm using TECS were unsuccessful, necessitating the administration of adenosine triphosphate (ATP) to restore sinus rhythm.

Given the tachycardia rate exceeding 200 bpm, a rapid intravenous administration of 20 mg ATP was performed (Figure 11). The final event in the tachycardia sequence was marked by a P' wave, followed by a ventricular extrasystole with retrograde conduction to the atria (RP' interval = 160 ms), after which sinus rhythm resumed with AV conduction block. The maximum RR interval reached 4600 ms, which was asymptomatic. The rhythm strip displaying TE ECG illustrates that during ATP action, conduction initially propagated through the AV node's slow pathway (PQ interval = 260 ms), transitioning sharply to the fast pathway (PQ interval = 160 ms). This abrupt shift may have been influenced by retrograde conduction to the atria. Sinus node discharge and the subsequent increase in the RR interval from 600 to 770 ms facilitated the resumption of anterograde conduction through the fast AV nodal pathway, corroborat-

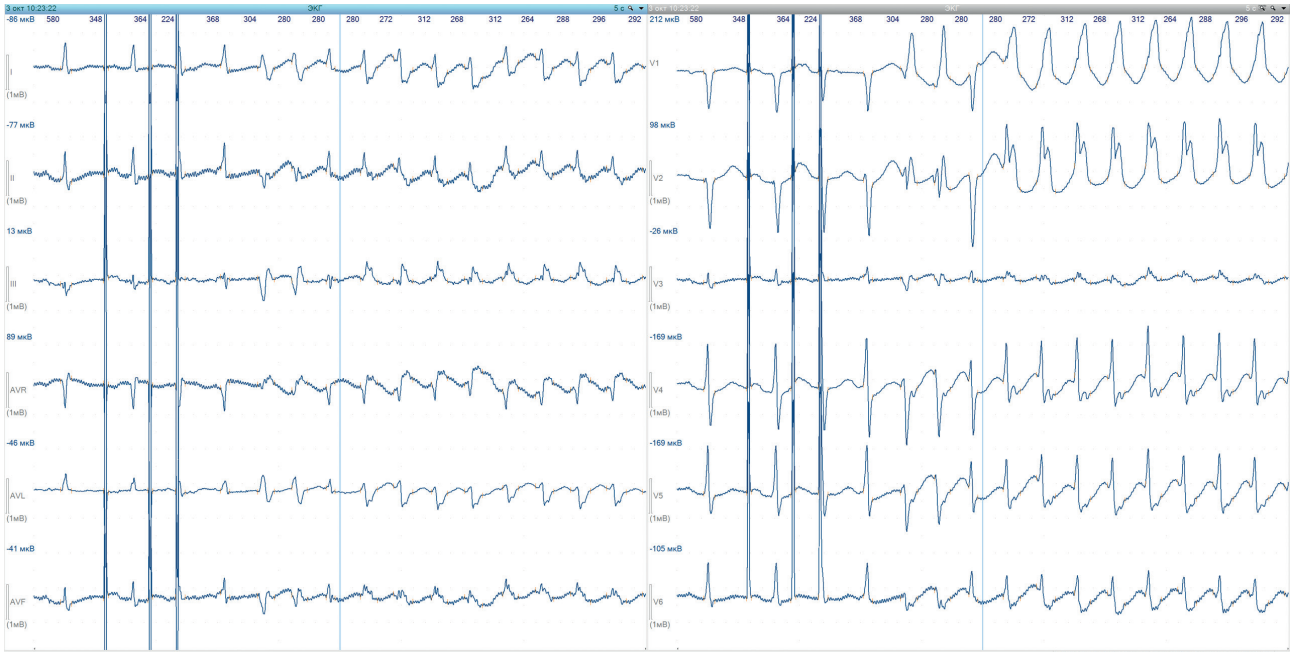

Figure 9. Induction of tachycardia with an RBBB pattern by delivering three impulses in manual mode. Explanations in the text.

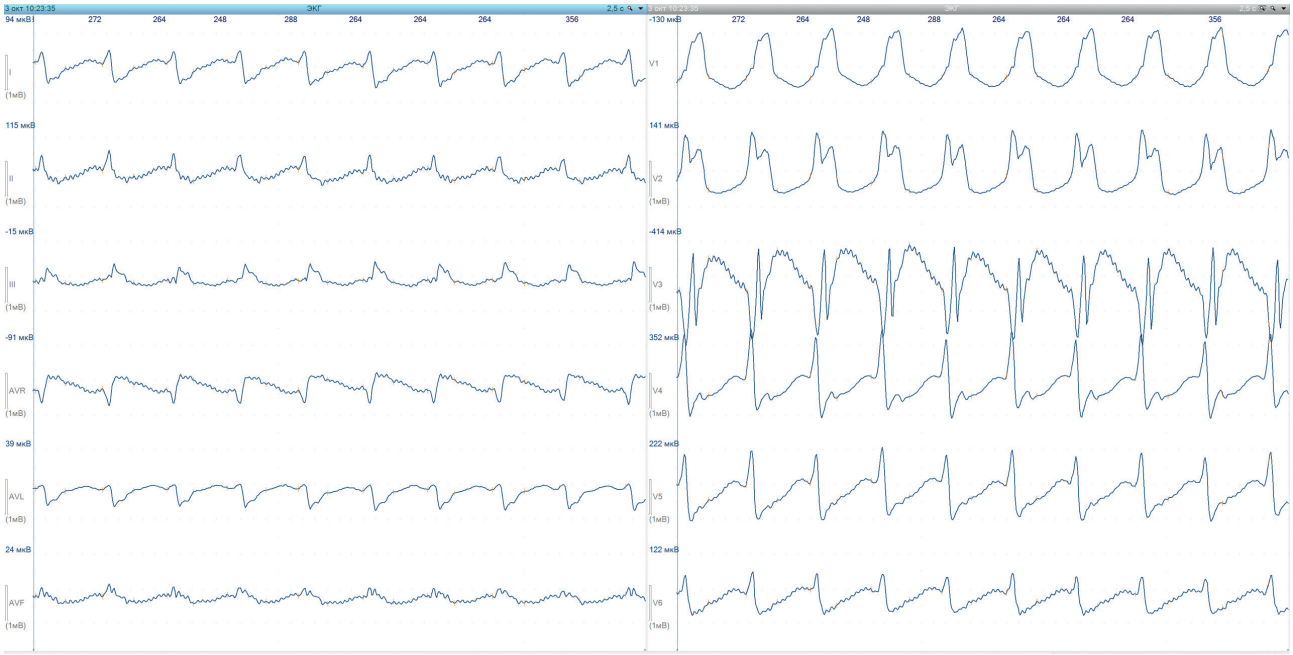

Figure 10. Registration of the transesophageal electrogram of patient G. during tachycardia (displayed in place of lead V3). Explanations in the text.

ing the presence of both fast and slow conduction zones within the AV node.

After the restoration of sinus rhythm via ATP administration, ST segment depression exceeding 200 μV was observed, most prominently in leads V4 and V5 (Figure 12). It is well-documented that ATP can cause a coronary steal phenomenon in patients with fixed coronary obstruction. This finding underscores the importance of thorough clinical evaluation, including stress echocardiography, to assess the patient's coronary status comprehensively.

Thus, the results of the TE EPS conducted on patient G., whose anamnesis suggested the presence of a concealed accessory pathway (AP) and paroxysmal reciprocating orthodromic AV tachycardia, revealed not only the concealed AP but also the existence of fast and slow conduction zones in the AV node. Anterograde conduction via the AP with minimal ventricular involvement was demonstrated, confirmed by the recording of ventricular late potentials and the dynamic assessment of QRS complex morphology during programmed cardiac stimulation.

AVNRT was induced at a rate exceeding 200 bpm and was terminated by administering 20 mg of ATP. A rare mechanism of tachycardia induction was identified, along with indirect signs that allowed its characterization prior to TE ECG recording. The patient was referred for radiofrequency catheter ablation of the slow pathway zone in the AV node.

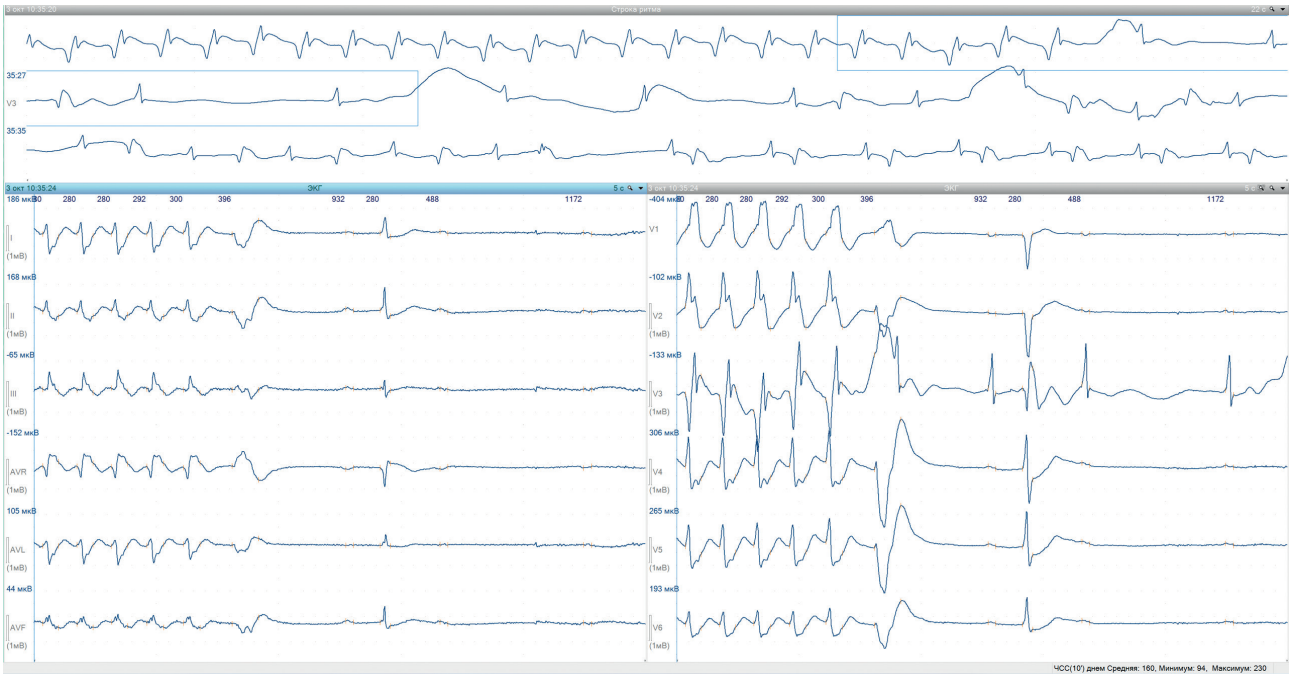

Figure 11. Registration of sinus rhythm restoration after intravenous bolus administration of 20 mg adenosine triphosphate (ATP). The rhythm strip (top) shows the transesophageal electrogram. Explanations in the text.

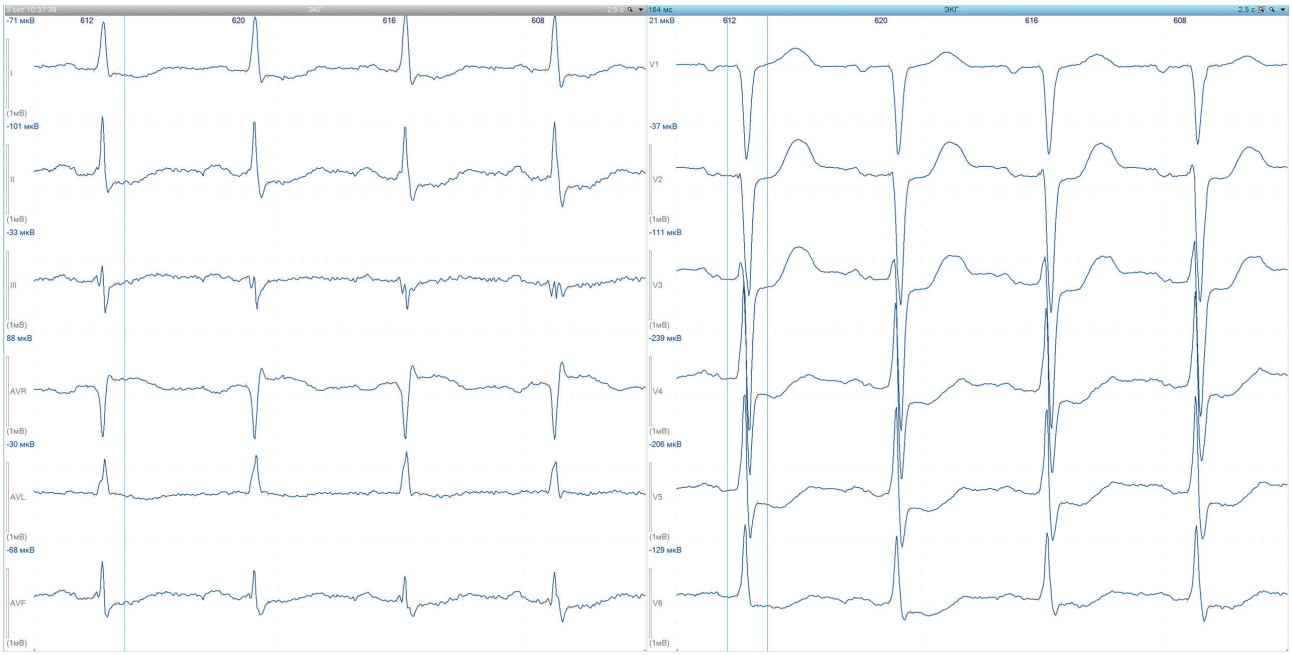

Figure 12. Assessment of ST-segment depression in patient G. following sinus rhythm restoration. Explanations in the text.

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