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THREE FACES OF ONE PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA

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Fragments of a transesophageal electrophysiological study of a 35-year-old patient with induction of paroxysmal atrioventricular nodal re-entry tachycardia occurring with three different electrocardiographic patterns are presented.

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Atrioventricular (AV) nodal re-entry tachycardia (AVNRT) is perhaps the most versatile of the supraventricular tachycardias. Depending on the direction of excitation propagation through fast and slow channels in the AV node, it can proceed in a slow-fast and a fast-slow version, and in the presence of a polyvascular structure of the AV node - in a slow-slow version. Against the background of AVNRT, an aberrant conduction of excitation may develop which does not lead to a change in tachycardia frequency because the legs of the branch bundle are not involved in the re-entry circuit. In the presence of obstruction above or below the re-entry loop, AVNRT may be recorded with AV or ventriculoatrial lead in a ratio other than 1:1. In addition, in this tachycardia, atrial or ventricular ectopias may not interrupt its course. Finally, it should be borne in mind

that AVNRT may take on additional features after radiofrequency catheter ablation, related to the procedure performed, if it recurs.

Patient C., 35 years old, was referred for a transesophageal electrophysiological examination because she had been suffering from palpitations for several years, but these had become more frequent in recent months. The patient stopped it herself using vagus techniques, it was not possible to record an ECG during the seizure. The electrophysiological examination showed the signs of AV conduction duality on the background of the initial rhythm, but it was not possible to induce AVNRT. After administration of 1 ml of 0.1% atropine sulphate solution on a background of sinus tachycardia with a heart rate of over 130 bpm, AVNRT was induced (fig. 1). Initially,



Fig. 1. Induction of tachycardia after atropinization. Explanations in the text.

AVNRT flowed with a 2:1 AV conduct. In the middle between the QRS complexes, you can see P' waves, negative in the lower leads. They are narrower than the sinus waves P caused by the concentric covering of the atria by the excitation of the AV node. An interesting feature of this AVNRT is that the P' waves that are in the QRS complexes do not form pseudo-beats s (as is quite common), but pseudo q. They are particularly striking in the right thoracic leads, where the r-waves registered against the background of sinus rhythm have been replaced by q-waves during tachycardia induction.

Against the background of the vagus manoeuvre, the 2:1 AVNRT changed into a 1:1 tachycardia accompanied by a twofold increase in heart rate (fig. 2). The increased

frequency of the ventricular complexes led to the appearance of a tachycardia-dependent complete blockade of the right bundle branch and within a few seconds a tachycardia with «wide» QRS complexes could be observed. Then conduction was restored along the right limb of the bundle branch, which was accompanied by constriction of the QRS complexes. Changes in the width of QRS complexes had no significant effect on the frequency of tachycardia. The electrocardiographic changes shown, in combination with the signs of conduction duality found AV, allowed the tachycardia to be considered AVNRT. The paroxysm was terminated by transesophageal electrocardiostimulation. The patient was referred for modification of the slow pathway in the AV node.



Figure 2. Tachycardia response to the vagus maneuver. Explanations in the text.