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MAIN THEORIES OF EXTRASYSTOLE PATHOGENESIS

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Abstract:
It is shown that supraventricular extrasystole can be single or paired, and also have an allorhythmic character. In this case, the interval between the extrasystolic P wave and the preceding P wave of the sinus rhythm is usually strictly fixed. The signs of the genesis of supraventricular arrhythmia are studied.

Keywords: Supraventricular extrasystole, pulmonary, sinus, rhythm, postextrasystolic pause, block, teeth, compensator.

Introduction

Supraventricular extrasystole (SVE) is called premature (in relation to the normal, sinus rhythm) electrical activation of the heart, caused by impulses whose source is located in the atria, in the pulmonary or cava veins (at the places where they flow into the atria), as well as in the atrioventricular junction. SVE can be single or paired (two consecutive extrasystoles), and also have an allorhythmia character (bi-, tri-, quadrigemy). Cases when SVE occurs after each sinus complex are called supraventricular bigeminy; if it occurs after every second sinus complex - trigeminy, if after every third - quadrigemy, etc.

The occurrence of SVE before the complete end of repolarization of the heart after the previous sinus complex (i.e. the end of the T wave) is called the so-called "early" SVE, a particular variant of which is the SVE of the "P on T" type.

Depending on the localization of the arrhythmogenic source of SVE, the following are distinguished:

• atrial extrasystole,
• extrasystole from the mouths of the vena cava and pulmonary veins,
• extrasystole from the AV junction. The development of ENC may be based on various structural and functional disorders of the myocardial cells of the atria, vena cava/pulmonary veins and AV junction, accompanied by a change in their

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action potentials (AP). Depending on the nature of electrophysiological disorders in the corresponding parts of the heart, ENC may occur by the mechanism of trigger activity (disruption of cell repolarization processes in the 3rd or 4th phase of AP), abnormal automatism (acceleration of slow depolarization of cells in the 4th phase of AP) or re-entry of the excitation wave. The diagnosis of ENC is established based on the analysis of a standard ECG. In atrial extrasystole, the ECG records premature P waves of sinus origin in relation to the expected ones, differing from the latter in their morphology (Fig. 1).

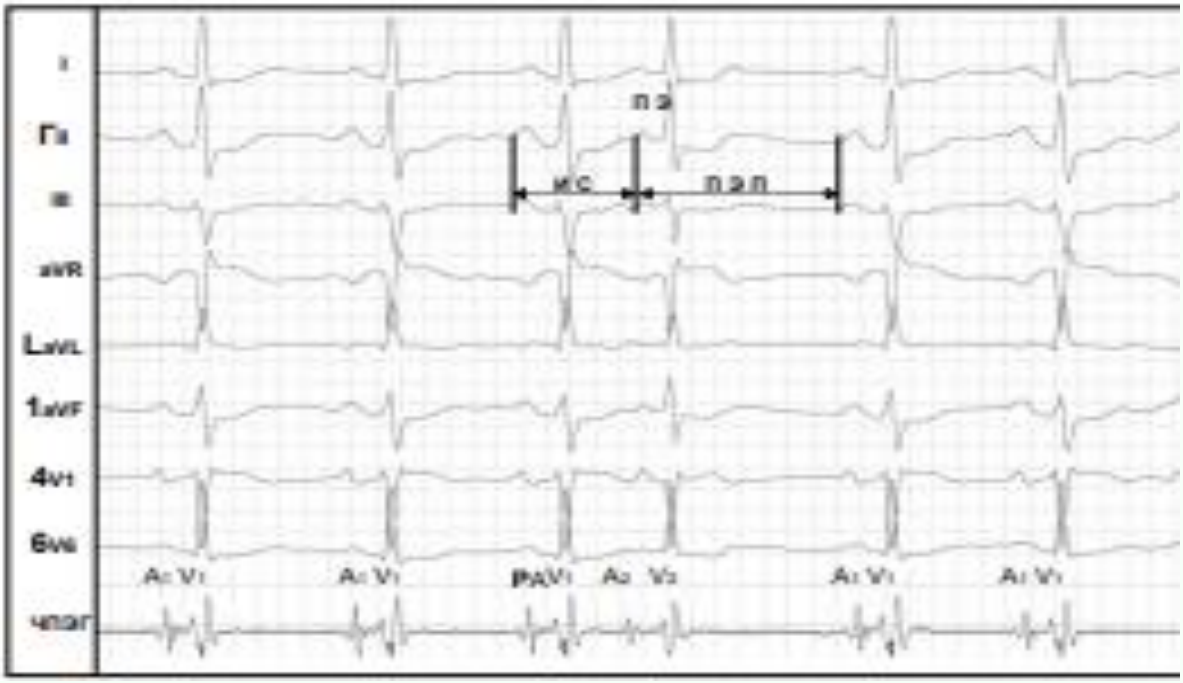

Fig. 1. Atrial extrasystole. Designations: IS – atrial extrasystole (AE) coupling interval, PEP – post-extrasystolic pause, TEE – transesophageal electrogram, A – atrial oscillations, V – ventricular oscillations, index 1 denotes electrical signals of sinus origin, index 2 – electrical signals of AE.

In this case, the interval between the extrasystolic P wave and the preceding P wave of sinus rhythm usually has a strictly fixed value and is called the "coupling interval" of the atrial extrasystole. The presence of several morphological variants of the P waves of atrial extrasystole with different coupling intervals indicates a multiplicity of arrhythmogenic sources in the atrial myocardium and is called polytopic atrial extrasystole. Another important diagnostic feature is the occurrence of the so-called "incomplete" compensatory pause after atrial

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extrasystole. In this case, the total duration of the coupling interval of the atrial extrasystole and the post-extrasystolic pause (the interval between the P wave of the extrasystole and the first subsequent P wave of the sinus contraction) should be less than two spontaneous cardiac cycles of the sinus rhythm (Fig. 1).

Premature P waves can sometimes overlap the T wave (the so-called "P on T" extrasystole), less often - the QRS complex of the preceding contraction, which makes it difficult to detect them on the ECG.

In these cases, recordings of transesophageal or endocardial electrocardiograms make it possible to differentiate the signals of electrical activity of the atria and ventricles.

A distinctive feature of extrasystoles from the AV junction is the registration of premature QRST complexes without preceding P waves. In these variants of extrasystole, the atria are activated retrogradely, due to which the P waves are most often overlapped with the QRS complexes, which, as a rule, have an unchanged configuration. Occasionally, P waves in extrasystoles from the AV junction are recorded in close proximity to the QRS complex; they are characterized by negative polarity in leads II and aVF.

A differential diagnosis between extrasystole from the AV node and the common trunk of the His bundle, as well as between atrial extrasystole and extrasystole from the mouths of the vena cava or pulmonary veins, is possible only based on the results of an intracardiac electrophysiological study. In most cases, electrical impulses from the ventricles are conducted to the ventricles through the AV junction and the His-Purkinje system, which is manifested on the electrocardiogram by a normal (unchanged) configuration of the QRST complex. Depending on the initial functional state of the cardiac conduction system and the degree of prematurity of atrial extrasystoles, the latter may be accompanied by certain manifestations of conduction process disorders. If an impulse from the ventricles, falling into the refractory period of the AV junction, is blocked and is not conducted to the ventricles, they speak of the so-called "blocked" supraventricular extrasystole (Fig. 2-A).

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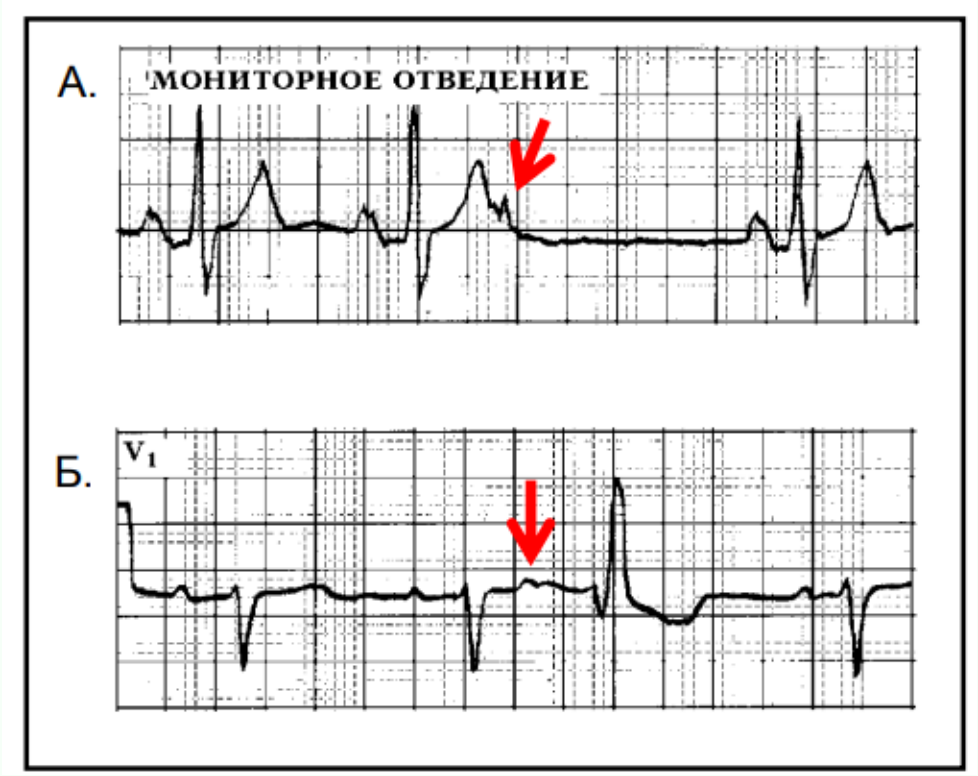

Fig. 2. Atrial extrasystoles. A. blocked atrial extrasystole (AE), B. AE with aberrant conduction to the ventricles (right bundle branch block).

Frequent blocked AE (for example, by the bigeminy type) can manifest itself on the ECG with a picture similar to sinus bradycardia and can be mistakenly regarded as an indication for cardiac pacing. A premature atrial impulse reaching one of the bundle branches in a state of refractoriness leads to the formation of an electrocardiographic picture of aberrant conduction with the corresponding deformation and widening of the QRS complex (Fig. 2-B).

AE, accompanied by an ECG picture of aberrant conduction to the ventricles, must be differentiated from ventricular extrasystole. In this case, the following signs indicate supraventricular genesis of arrhythmia:

1) the presence of P waves before extrasystolic QRS complexes (including a change in the shape and/or amplitude of the T wave of the sinus complex preceding the extrasystole in NVE of the P on T type);

2) the occurrence of an incomplete compensatory pause after the extrasystole,

3) a characteristic "typical" ECG variant of the right or left bundle branch block (example: NVE, accompanied by a right bundle branch block, is characterized by

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an M-shaped QRS complex in lead V1 and a rightward deviation of the EOS of the heart).

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