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PACING MANEUVERS FOR SUPRAVENTRICULAR TACHYCARDIA DIFFERENTIAL DIAGNOSIS: VENTRICULAR OVERDRIVE PACING

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The accurate tachycardia mechanism verification is the most important condition for high effectiveness and safety of the supraventricular tachycardia ablation. Ventricular overdrive pacing - is a simple and useful diagnostic maneuver, frequently used in the supraventricular tachycardia diagnosis. The conditions for its performance and interpretation in the standard and rare situations are described in this review.

Key words: supraventricular tachycardia; electrophysiological study; catheter ablation;, AV nodal reentry tachycardia; orthodromic tachycardia; atrial tachycardia; ventricular overdrive pacing

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Catheter ablation is one of the most common and effective treatments for supraventricular tachycardia (SVT). Since the mapping technique and choice of instruments for catheter ablation depend largely on the type of tachycardia, intraoperative diagnosis is of paramount importance. Both the basic parameters of SVT (cycle duration, ventriculo-atrial (VA) interval, etc.) and the responses to different stimulation techniques need to be considered. The choice of pacing technique and the interpretation of the response to it also depend on the initial parameters of the tachycardia. As each stimulation technique has its own advantages and limitations, the electrophysiologist must be confident in its use and interpretation in order to successfully diagnose SVT. This is what this review is about.

This literature review is first dedicated to the differential diagnosis of the three most common types of SVT: atrioventricular nodal recurrent tachycardia (AVNRT), atrioventricular recurrent tachycardia (AVRT) with involvement of an accessory conduction pathway (ACP) and atrial tachycardia (AT). First we will look at «resetting» and «entrainment» as basic electrophysiological phenomena. In the following we will discuss the role of ventricular overdrive pacing (VOP) in determining the diagnosis in most cases.

ELECTROPHYSIOLOGICAL BASIS OF STIMULATION TECHNIQUES

The absolute majority of supraventricular tachycardias develop by the mechanism of re-entry, therefore a significant proportion of pacing techniques are based on two electrophysiological phenomena: «resetting» and «entrainment» of the tachycardia [1, 2]. With the help of these phenomena, it is not only possible to determine the mechanism of the tachycardia, but also to determine the location of the re-entry circuit, to confirm or exclude the presence of ACP and its involvement in the maintenance of the arrhythmia. Before we turn to the specific issues of differential diagnosis of SVT using stimulation techniques, let us briefly describe the electrophysiological basis of resetting and entrainment.

Resetting the tachycardia

When a single electrical stimulus is applied to the re-entry circuit during a tachycardia, two new excitation waves (two excitation fronts) are generated that move in opposite directions from the point of stimulation (Fig. 1a,b). The wave propagating in the direction opposite to the movement of the tachycardia wave is called retrograde [3]. The stimulated wave propagating along the course of the tachycardia wave is called antegrade. After the tachycardia wave and the retrograde wave have collided (Fig. 1c), they disappear and the antegrade excitation front remains the only wave moving in the re-entry circuit. Thus, the antegrade wave becomes a new tachycardia wave, which will then continue to move in a circuit (Fig. 1d). Since the speed of impulse propagation through the myocardium and the length of the ree-ntry circuit remain unchanged, the frequency of tachycardia will remain the same. Since the retrograde stimulated wave stops the tachycardia and the antegrade wave simultaneously triggers a new tachycardia in the same circuit, this technique is called «resetting» the tachycardia. During resetting, all complexes following the resetting extrastimulus appear «shifted» to the left on the ECG (Fig. 1d).

Entrainment

If a series of pulses, rather than a single pulse, is administered during tachycardia in the re-entry circuit, each

subsequent pulse may terminate the tachycardia that was «reset» by the previous pulse and «resetting» the tachycardia anew. In this case, during a series of stimuli in the re-entry circuit, there are constantly three waves of excitation: an antegrade wave, a retrograde wave and a tachycardia wave (or an antegrade wave from the previous impulse) [2]. At the end of the stimulation, the retrograde wave of the last pulse again stops the tachycardia triggered by the previous (penultimate) pulse, while the antegrade wave resets the tachycardia and continues the cycle of re-entry as the next pulse fails to occur. Since the tachycardia wave is constantly overlaid by retrograde and antegrade waves from both sides during a series of impulses and the tachycardia appears accelerated to the pacing frequency, this phenomenon is called «entrainment». In this case the entrainment is a continuous resetting of the tachycardia during a series of stimuli. Simultaneous excitation of the myocardium from two sources (the main tachycardia wave and the retrograde wave) leads to the formation of ECG complexes (QRS, P waves or F waves) with «merging» morphology, i.e. an intermediate morphology between that during tachycardia and that during pacing (Fig. 2a). It should be noted that in order to assess the ECG shape during entrainment, the configuration of the complexes must be known both during tachycardia and during pacing outside tachycardia.

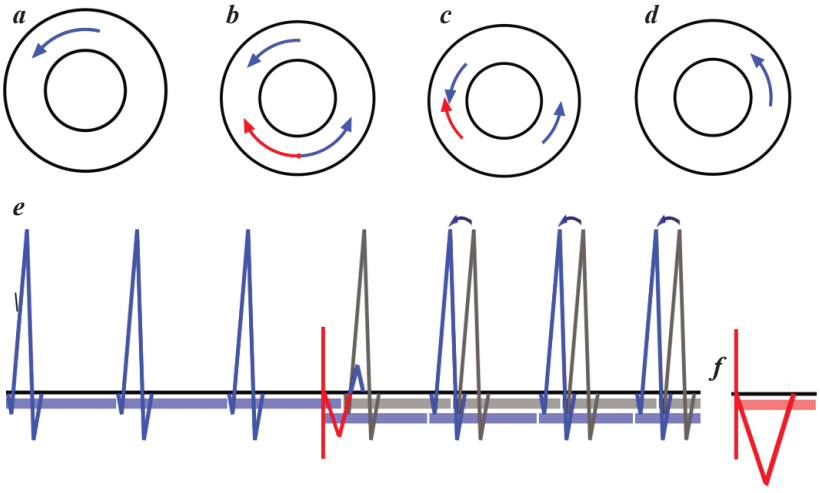

Fig. 1. Schematic representation of the phenomenon of re-entry resetting tachycardia. a) The tachycardia wave propagating in the circuit of re-entry is indicated by the blue arrow. The myocardium outside the arrow is at rest, i.e. it is capable of electrical excitation. b) The extrastimulus applied in the re-entry circuit causes two other excitation waves in addition to the tachycardia wave: antegrade (blue arrow) and retrograde (red arrow). c) The tachycardia wave collides with the retrograde stimulated wave and they cancel each other out. d) The only excitation wave remaining in the re-entry circuit is the antegrade stimulated wave, which becomes a new tachycardia wave that can continue in an endless circular motion. e) Schematic representation of an ECG during a re-entry resetting of tachycardia. A single extrastimulus causes the appearance of a premature complex, which has a confluent morphology (has features of stimulated and spontaneous complexes). The first return tachycardia complex follows the stimulated one at an interval equal to the duration of the tachycardia cycle. Thus, all subsequent complexes (shown in blue) are «shifted to the left» relative to their proper position, which would be observed without «resetting» (shown in gray). f) Schematic representation of the stimulated complex.

Signs of entrainment tachycardia are:

- stable fusion morphology of the complexes, during increasing stimulation with continued tachycardia after termination of stimulation (Stable fusion) (Fig. 2a,c),
- change in the morphology of the complexes during stimulation with gradually increasing frequency with continuation of tachycardia after termination of stimulation (Progressive fusion) (Fig. 2b,d),
- local conduction blockade at the moment of tachycardia termination during increasing stimulation, followed by earlier local activation of this site during the continuation of the series of stimuli,
- change in the morphology of electrograms (EG) and the relative activation time of any part of the circuit of re-entry when the frequency of stimulation changes with continuation of tachycardia after termination of stimulation (Local fusion) [4].

Depending on the presence of signs of confluent excitation on the surface ECG or local ECG, the following main variants of entrainment are distinguished.

- Entrainment with «manifest collision of excitation fronts» (manifest entrainment). During entrainment, the surface ECG has an intermediate morphology between its own and that imposed from stimulation (fusion complexes). The degree of fusion is determined by the volume of myocardium depolarised by the retrograde wave, which depends on the location of the site of stimulation relative to the main circuit of re-entry, the frequency of stimulation and the decremental delay of conduction in the circuit of re-entry. In manifest entrainment, the ECG notes a steady «fusion» with stimulation at a certain frequency and a progressive «fusion» with increasing frequency of stimulation. After termination of stimulation, tachycardia continues, and the last complex accelerated to the stimulation frequency on the ECG already has the morphology of tachycardia without signs of fusion, that is, the same morphology as during tachycardia (Fig. 2a,b) [5].
- Entrainment with a «local collision of fronts». The tachycardia wave (an antegrade wave from the previous pulse) entrains such a small volume of myocardium that it does not cause changes on the surface ECG. In this regard, stimulated complexes are recorded on the ECG, and there is no draining morphology (in contrast to the manifest entrainment). Nevertheless, the collision of fronts in a small area of the myocardium can be recognised with a local intracardiac recording. This requires recording the different morphology and interval from the stimulus to the local excitation at two stimulus series with different frequencies. At a lower frequency, it is possible to find a region in which the St-EG inter-

val is long, and the electrogram will be caused by an antegrade wave from the previous stimulus (Fig. 1a,c). At higher frequency, the St-EG interval will be significantly shorter due to the entrainment of the retrograde wave from the last stimulus (Fig. 1b,d) [4, 5].

• Entrainment with «full retrograde entrainment» If the frequency of stimulation significantly exceeds the frequency of tachycardia or the point of stimulation is far from the circuit of re-entry, the retrograde wave will entrain the entire stimulated chamber (ventricles or atria). In this case, no signs of antegrade activation can be registered neither on surface ECG, nor by intracardiac ECG (Fig. 3a and see Fig. 11b) [5].

• Entrainment with «hidden front collision» (sometimes also called hidden entrainment) is a phenomenon opposite to full retrograde entrainment. The tachycardia is accelerated to the stimulation frequency, but the plum morphology cannot be registered, neither by surface ECG, nor by intracardiac recordings. The morphology of the surface ECG and the sequence of intracardiac activation are the same as during tachycardia. The antegrade front resets the tachycardia, while the retrograde front collides with the main tachycardia front near the point of stimulation and cannot entrain enough myocardial volume to cause changes on the ECG. (Fig. 3b,c) [5].

Post-stimulation interval

If the stimulation point is directly in the re-entry circuit, the time interval between the last stimulus in the series that triggered the tachycardia and the first recurrent local electrogram at that point (post-pacing interval - PPI) corresponds to the duration of the tachycardia cycle. W.Stewenson et al. established that PPI duration equal to tachycardia cycle length (TCL) or exceeding it not more than by 30 ms is a good prognostic factor for detection of points of effective ventricular tachycardia ablation [6]. The use of this criterion has also been described for mapping atrial tachycardia [7]. The longer duration of PPI compared to TCL reflects the remoteness of the stimulation point from the main circuit of re-entry. The further away

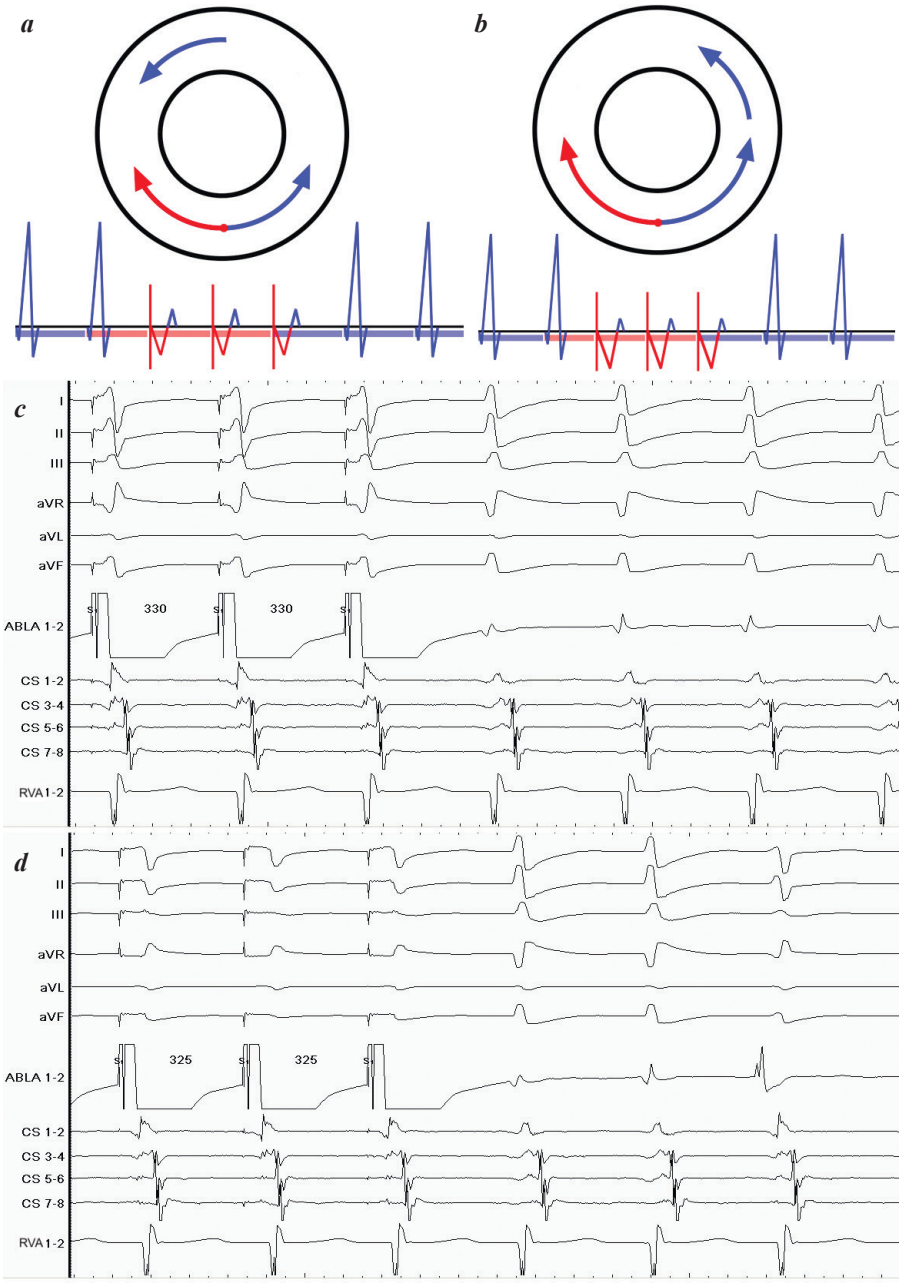

Fig. 2. Schematic representation of the propagation of excitation waves in the tachycardia circuit during entrainment. a) During a series of stimuli with a cycle duration close to the tachycardia cycle, confluent complexes with stable morphology are detected. After termination of pacing, the tachycardia continues. b) With shorter-cycle pacing, the pluminal complexes have a different morphology (due to the greater volume of myocardium captured by the retrograde wave), but the morphology within the pacing is stable. c, d) ECG and EG during ventricular pacing in orthodromic AV re-entry tachycardia. The morphology of the stimulated (merging) QRS complexes changed when the stimulation cycle was shortened from 330 ms to 325 ms. c) At a lower stimulation frequency (stimulation cycle duration of 330 ms), the ventricular myocardium is more strongly entrained by the antegrade excitation wave, the QRS complexes have a shape close to the morphology of QRS during tachycardia (the last 2 complexes). d) At a higher stimulation frequency (cycle duration 325 ms), the retrograde wave started to entrain a slightly larger volume of the ventricular myocardium. Fusion QRS complexes have a morphology less similar to that during tachycardia than in Fig. 2c.

the stimulation point is from the re-entry circuit, the greater the distance the stimulated excitation wave has to travel to reach the re-entry circuit and then return to the stimulation point (after one revolution in the re-entry circuit, which takes the same time as TCL) (Fig. 4).

PRIMARY PARAMETERS FOR EVALUATING SVT

On invasive electrophysiological examination (EPE), SVT is most often tachycardia with narrow QRS complexes and normal H-V interval. Among the parameters presented in Table 1, the first three are the main ones, and their assessment is necessary when analysing any SVT induced during an EPE:

- V:A ratio (quantitative ratio of ventricular to atrial contractions) (Fig. 5);
- V-A interval (from the beginning of the earliest ventricular activation to the beginning of the earliest atrial activation in the bundle branch region);
- atrial activation sequence: concentric (from the interatrial septum to the lateral walls - see Fig. 8a) or eccentric (from the lateral wall of one of the atria to the septum - Fig. 2c,d, Fig. 3c).

If possible, important information about the mechanism of SVT can be obtained by assessing three additional parameters:

- spontaneous termination of SVT with registration of another (not premature) atrial contraction at the end of the paroxysm without changing the atrial activation sequence (Fig. 6);
- with small fluctuations in TCL, changes in the H-H or V-V intervals precede changes in the A-A intervals or, conversely, change after them. In other words, whether fluctuations in the tachycardia cycle are due to changes in the duration of the A-H (A-V) or H-A (V-A) intervals (Fig. 7).
- changes in the V-A interval duration associated with the appearance and termination of functional blockade of one of the legs of the bundle branch.

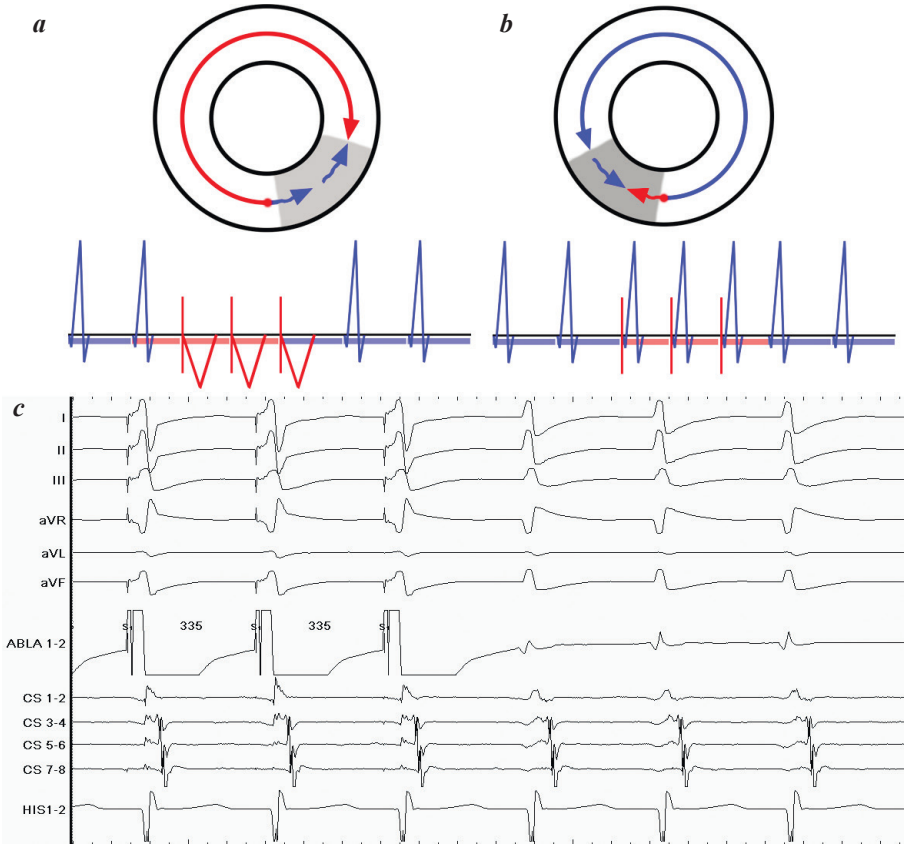

Fig. 3. Schemes depicting rare variants of entrainment. a) Entrainment with full retrograde entrainment. Due to the fact that the stimulation point in the re-entry circuit is in close proximity to the delayed conduction zone, the antegrade excitation wave and the tachycardia wave are significantly delayed in this zone. During this time, the retrograde wave manages to cover in retrograde direction all or almost all of the circuit of re-entry. This leads to the formation of fully stimulated complexes on the ECG without signs of plummeting morphology. Confirmation of the presence of entrainment is the continuation of tachycardia after termination of stimulation. b). The situation is the opposite of that shown in panel a. The delayed-excitation zone is in the path of the retrograde wave. The antegrade wave manages to completely cover the re-entry circuit and form complexes identical to those of tachycardia. Confirmation of tachycardia entrainment is acceleration of tachycardia during stimulation to the stimulation frequency and continuation of tachycardia with the same frequency after termination of stimulation. c) ECG and intracavitary EG in latent entrainment orthodromic AV re-entry tachycardia involving left lateral ACP with ventricular stimulation near the ventricular end of the ACP. The morphology of stimulated (confluent) QRS complexes is almost identical to the morphology of complexes during tachycardia.

In most cases, after evaluation of the listed SVT parameters, the diagnosis is clear and it is possible to perform ablation without additional stimulation techniques. For example, if the V-A interval < 80 ms is registered during SVT in the area of the bundle branch (excludes the possibility of AVRT) and small changes in the H-H interval (or V-V) exactly precede the changes in the A-A interval (excludes the possibility of AT), then the diagnosis of a typical AVNRT (slow-rapid mechanism) can be made and ablation can be performed in the area of the slow pathway of the AV connection.

At the same time, it is not surprising that much attention is paid in the literature to the differential diagnosis of SVT with a ratio of V:A=1:1, a V-A interval duration in the septal area > 80 ms and a concentric (from the centre to the periphery) atrial excitation sequence, since in such a situation any of the SVT types can occur (Fig. 5a). In such

cases, it is absolutely essential to be sure of the diagnosis before proceeding with ablation. In particular, it is critical to differentiate AVNRT from septal AT, or AVRT involving the septal ACP. The latter two mechanisms require mapping and searching for the point of earliest atrial activation. In the case of AVNRT, such tactics will result in exposures in the fast pathway area of the AV junction, where the risk of AV blockade is higher than in the slow pathway area of the AV node (between the coronary sinus mouth and the tricuspid valve ring). It is important to remember that it is easier to choose the right diagnostic technique if the primary parameters are used to narrow down the range of SVT mechanisms to only two possible options (AVNRT and AT or AVRT and AT).

HOW TO QUICKLY RULE OUT ATRIAL TACHYCARDIA?

Since any V-A interval duration and almost any A:V ratio can be recorded in AT, this rhythm disturbance is mentioned in 12 lines of 15 (80%) in Table 1. A quick way to confirm or exclude the diagnosis of AT is a VOP from the right ventricle (RV) with a cycle length 10-30 ms shorter than that of TCL [8]. If, during VOP, the atrial CL accelerates to the CL of the stimulation, and after termination of the stimulation the tachycardia continues and the sequence of spikes represents a «V-A-A-V» type response - this confirms the diagnosis of AT (Fig. 8a). If at the end of stimulation a «V-A-V» type response is recorded, the diagnosis of AT can be excluded, and further differential diagnosis should be made between AVNRT and orthodromic AVRT (see Fig. 10b). The first atrial EG is always the last atrial EG accelerated to the CL of stimulation.

Understanding how VOP confirms or excludes the diagnosis of AT is important to identify specific response variants that can lead to misinterpretation of the result, misdiagnosis, and an adverse outcome of ablation. Let us consider sequentially the options for responding to VOP under each of the SVTs.

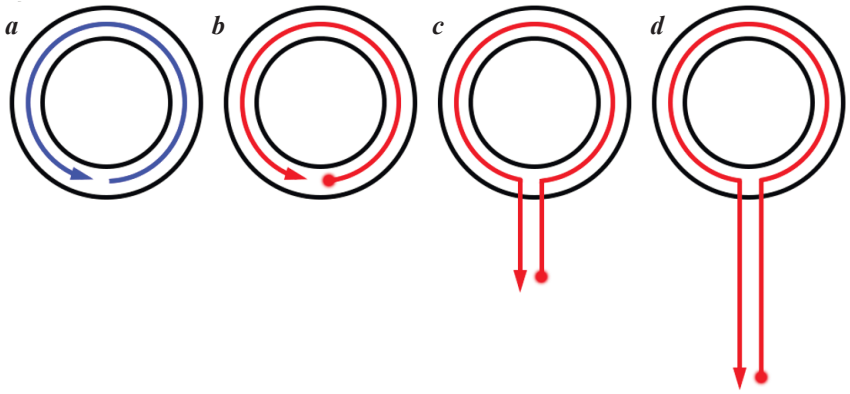

Fig. 4. Schematic depicting the relationship between the duration of the post-pacing interval (PPI) and the distance from the stimulation point during tachycardia entrainment to the circuit of re-entry. a) Tachycardia wave in the circuit of re-entry. b) The stimulation point is on the re-entry circuit: PPI=TCL (0-30 ms). c) The stimulation point is near the re-entry circuit: PPI>TCL (30-60 ms). d) The stimulation point is far from the tachycardia circuit: PPI>>>TCL (>60 ms).

Table 1.

Six basic parameters to be evaluated before selecting a stimulation technique

	Parameter	Note	Mechanism of tachycardia
1.	Ratio of the number of V:A	V=A	AVNRT, AVRT, AT
		V>A ± VA dissociation	oNVRT, oNFRT, AVNRT
		V<A ± AV dissociation	AVNRT, AT
2.	Interval V-A	V-A > 80 ms	aAVNRT, AVRT, AT
		V-A ≤ 80 ms	tAVNRT, AT
		V-A > A-V	aAVNRT, AT, AVRT involving slow-functioning ACP
3.	Atrial activation sequence	Top to bottom	AT
		Concentric	AVNRT, AVRT, AT
		Eccentric	AVRT, AT*
4.	Spontaneous cessation	Last «spike» is A	AVRNT, AVRT
		Last «spike» is V	AVNRT, AVRT, AT
5.	H-H interval changes precede A-A changes	Yes	AVRNT, AVRT
		No	AVNRT, AVRT, AT
6.	V-A interval is increased by more than 30 ms in the development of functional bundle branch block	Yes	AVRT with ACP involvement - on the side of the blocked bundle branch (ipsilateral block)
		No	AVNRT, AVRT, AT

Note: AVNRT - atrioventricular nodal reciprocal tachycardia; AVRT - atrioventricular reciprocal tachycardia; AT - atrial tachycardia; tAVNRT - typical AVNRT; aAVNRT - atypical AVNRT; oNVRT - orthodromic nodo-ventricular reciprocal tachycardia; oNFRT - orthodromic nodo-fascicular reciprocal tachycardia; * - most likely, AVNRT involving left atrial node exit - rare, but AVNRT and AVRT are theoretically possible.

Atrial tachycardia

When VOP is performed during AT, any excitation wave conducted retrogradely to the atria will accelerate the arrhythmia (in a triggered tachycardia mechanism) or inhibit it (in an automatic tachycardia mechanism) or enter the re-entry cycle (entrainment) if AT has a reciprocal mechanism. The last retrogradely conducted excitation wave (responsible for the atrium EG, which we consider the first response to the termination of VOP) cannot return antegradely to the ventricles as an echo response, because the heart's excitation conduction system is still in the refractory phase at this point - it has just conducted the same wave from the ventricles to the atria. Even in the presence of a double AV junction or a bystander accessory pathway, re-entry of the excitation wave that has just come retrogradely from the ventricles into the atria is impossible, as these pathways will simultaneously depolarise parallel to each other in the retrograde direction and will be in the refractory phase for some time (Fig. 9a-c). If AT is not stopped as a result of the series of stimuli, the next atrial EG (i.e., the second atrial EG after the termination of VOP) will be the result of the continuation of AT. Now the cardiac conduction system is out of refractory state, and the next after the atrial EG will be the bundle branch EG (H), followed by the ventricular EG (V2) (Fig. 9d). Thus, the response to the VOP will be «St-V-A-A-H-V», or simply «V-A-A-A-V» (Figs. 8, 9).

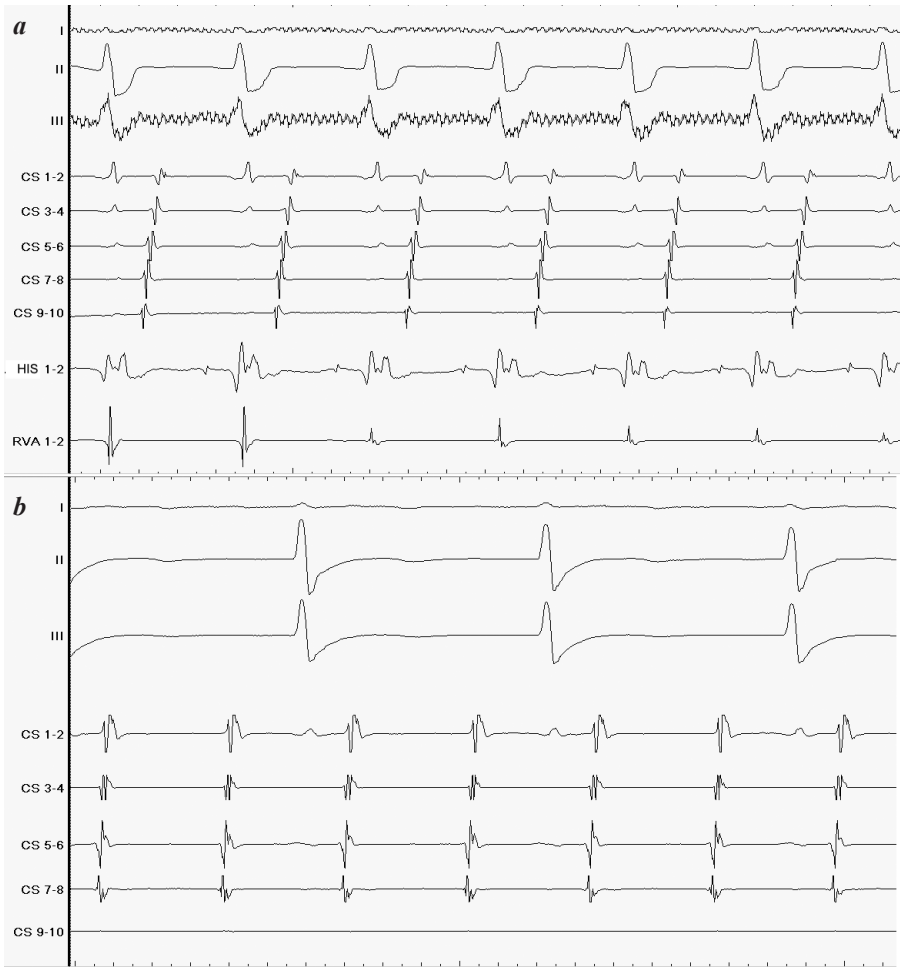

Fig. 5. Examples of ECG and electrograms of patients with tachycardia with narrow complexes and V:A ratio of 1:1 (a), 2:1 (b).

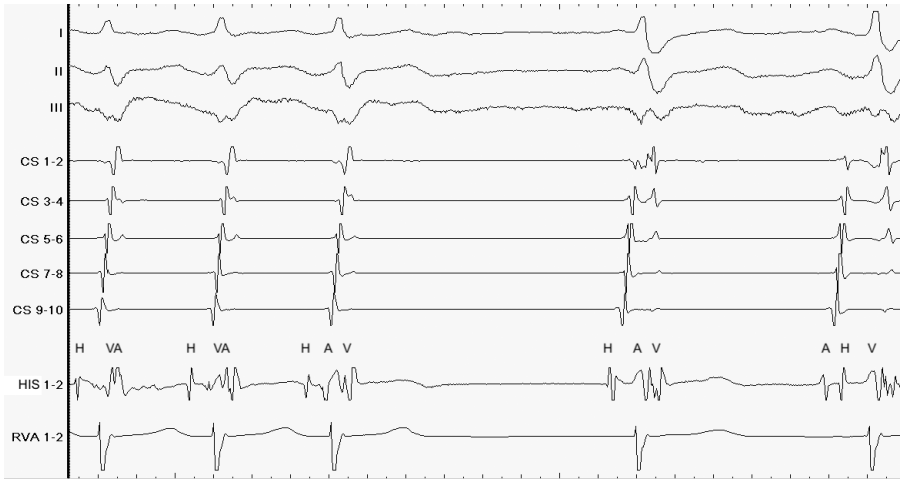

Fig. 6. Capping of AVRT when the next pulse through AV node to ventricles is stopped. At the end of the paroxysm, the H-A sequence is recorded.

Orthodromic AV re-entry tachycardia

In orthodromic AVRT, VOP causes the appearance of two waves of excitation propagating in opposite directions along the circuit of re-entry, and leads to entrainment of tachycardia. The antegrade wave induces atrial excitation through the ACP, so that the sequence of atrial activation coincides with the sequence during AVRT. The retrograde wave propagates through the ventricular myocardium and cardiac conduction system towards the AV node, meeting a tachycardia wave (after the first stimulus) or an antegrade wave of the previous stimulus (after the subsequent stimuli) on its way (Figure 10a). This is how tachycardia entrainment occurs - the tachycardia is resets- «turned on» or «immersed» in a series of stimuli. The best confirmation of entrainment is the registration of confluent QRS complexes (manifest entrainment) during VOP, which combine the morphology of stimulated ventricular complexes and narrow complexes registered during AVRT. If during a series of stimuli an acceleration of atrial activation was observed before the CL

stimulation and the tachycardia continues after the termination of the VOP, then even in the absence of signs of fusion excitation in the ECG, it can be assumed that an entry into the tachycardia circuit has taken place (in this case we speak of entrainment with local collision of the excitation fronts). At the termination of pacing, the last imposed wave will propagate retrograde through the ACP to the atria (thus registering the first atrial EG in response to the VOP), traverse a full circuit of tachycardia in the antegrade direction, without encountering a retrograde wave in the absence of the next stimulus, and leading to the registration of successive bundle branch (H) and ventricular (V) EGs, representing the response to VOP of the «St-V-A-H-V» or simply «V-A-V» type (Fig. 10b).

AV nodal re-entry tachycardia

The situation is similar when the AVNRT entrainment is carried out with the help of VOP. The only difference is that a single stimulated wave of excitation must completely cover the ventricles and pass through the conduction system to the AV node. Then the antegrade wave, having introduced in «fast» way of AV connection, will restart AVNRT and also will accelerate an atrial rhythm to CL stimulation. At the same time, the retrograde wave will enter the «slow» pathway of the AV junction and collide there with the antegrade wave from the previous stimulus (Fig. 11a). The last stimulated wave delivered to the atria in the retrograde direction will result in registration of the first (and the only) atrial EG in response to the VOP, passing through a circuit of tachycardia. Then it will return to the ventricles through the «slow» AV junction pathway and a bundle branch without encountering a retrograde wave (due to the absence of the next impulse). Thus, the response to the VOP «St-V-A-H-V», or simply «V-A-V», is recorded (Fig. 11b). It should be noted that during the AVNRT entrainment, the collision of the retrograde excitation wave and the antegrade wave from the previous stimulus occurs within the «slow» pathway of the AV node. Accordingly, the morphology of the QRS complex is always stimulated and cannot be confluent. Therefore, in AVNRT, entrainment is al-

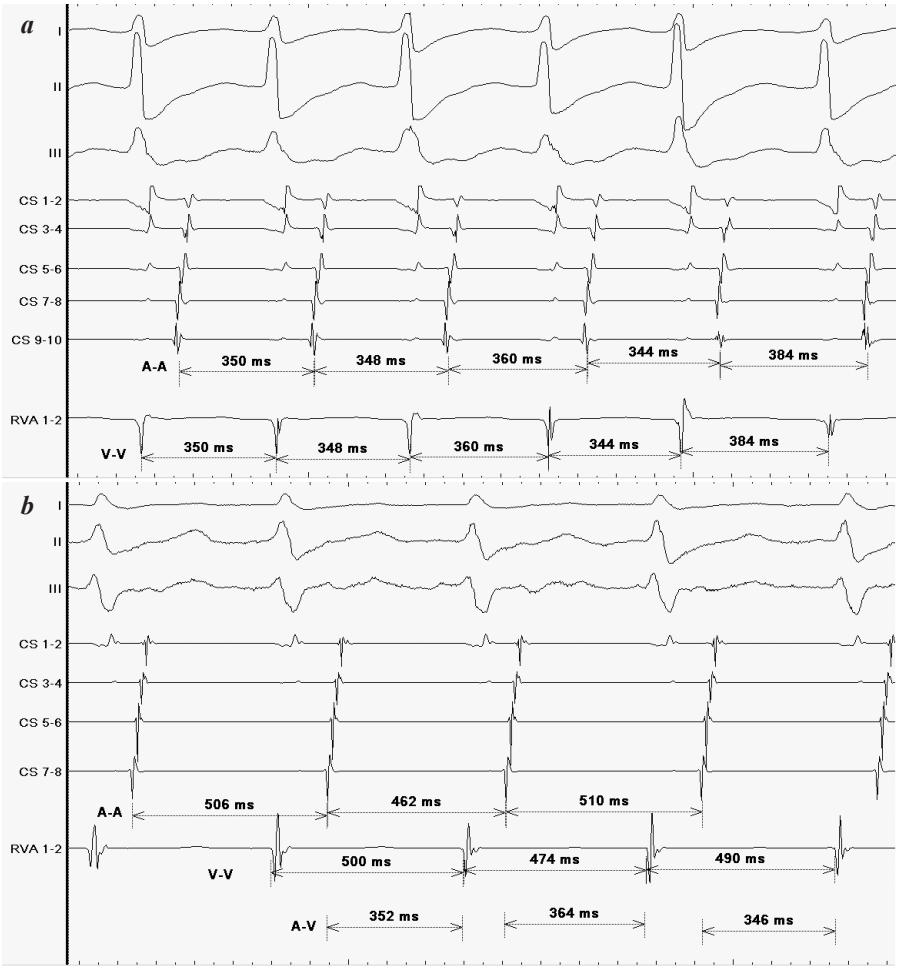

Fig. 7. Changes in the duration of the SVT cycle, allowing for a differential diagnosis. a) Cycle fluctuations in AVRT. Changes in the duration of VV intervals precede changes in AA intervals. b) Cycle fluctuations in AT. Changes in AA intervals precede changes in VV. The duration of the VV intervals does not fully correspond to the duration of AA due to the decremental conduction through the AV node: the shortening of the preceding AA leads to the lengthening of the following AV and vice versa, the lengthening of the preceding AA leads to the shortening of the AV interval.

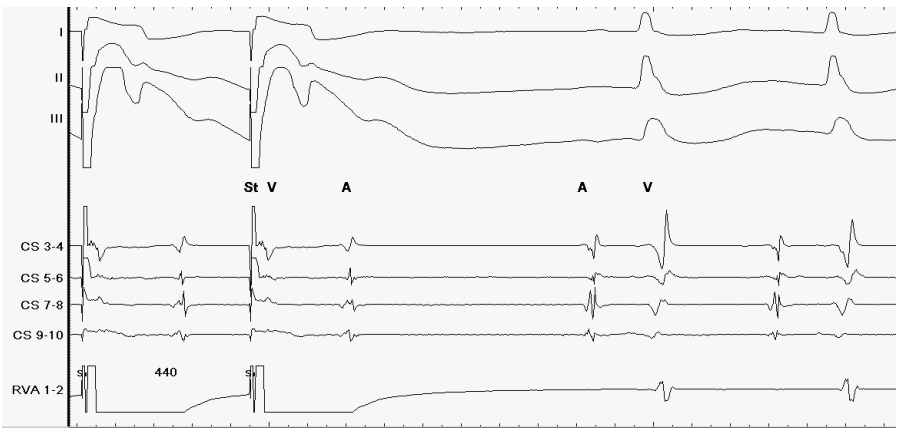

Fig. 8. Entrainment of SVT with the help of VOP. A V-A-A-V response is pathognomonic for atrial tachycardia.

ways characterised by complete retrograde ventricular entrainment and is never manifest neither by surface ECG nor by local EG data.

Thus, a «V-A-V» response to VOP is a sign of entrainment in AVRT or AVNRT and excludes the presence of AT. On the other hand, a «V-A-A-V» type response confirms the presence of AT, excluding the possibility of AVNRT or AVRT. The main disadvantage of this technique is that in 50-80% of cases atrial acceleration to ventricular stimulation rate is impossible. In other words, the atria continue to be excited at the frequency of tachycardia and the ventricles continue to be excited at the frequency of pacing, i.e. slightly more frequently than the atria. As a rule, this phenomenon is caused by low conductivity of the AV node in the retrograde direction. Nevertheless, such response variant shows that atrial rhythm does not depend on ventricular activation (VA dissociation) and allows to exclude AVRT [9-11]. When such a response is detected on VOP, it usually appears to be atrial tachycardia, but additional pacing techniques are needed to be sure that it is AT [9, 11] and not AVNRT.

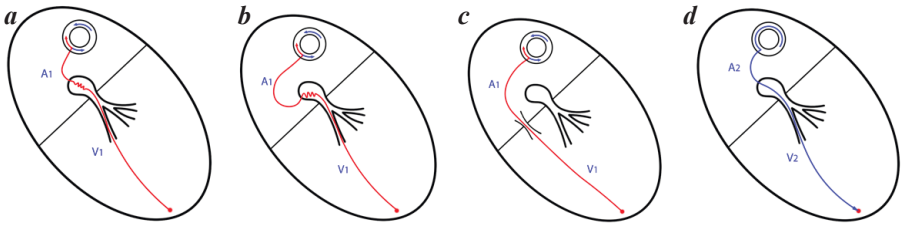

Fig. 9. Schematic of pulse propagation in atrial re-entry tachycardia entrainment during VOP. Retrograde pulse conduction (red line) from the ventricles to the atria can be through a fast AV junction pathway (a), a slow AV junction pathway (b), or an additional conduction pathway (c). Atrial and ventricular re-excitation (d) after one lap (blue line) in the re-entry loop.

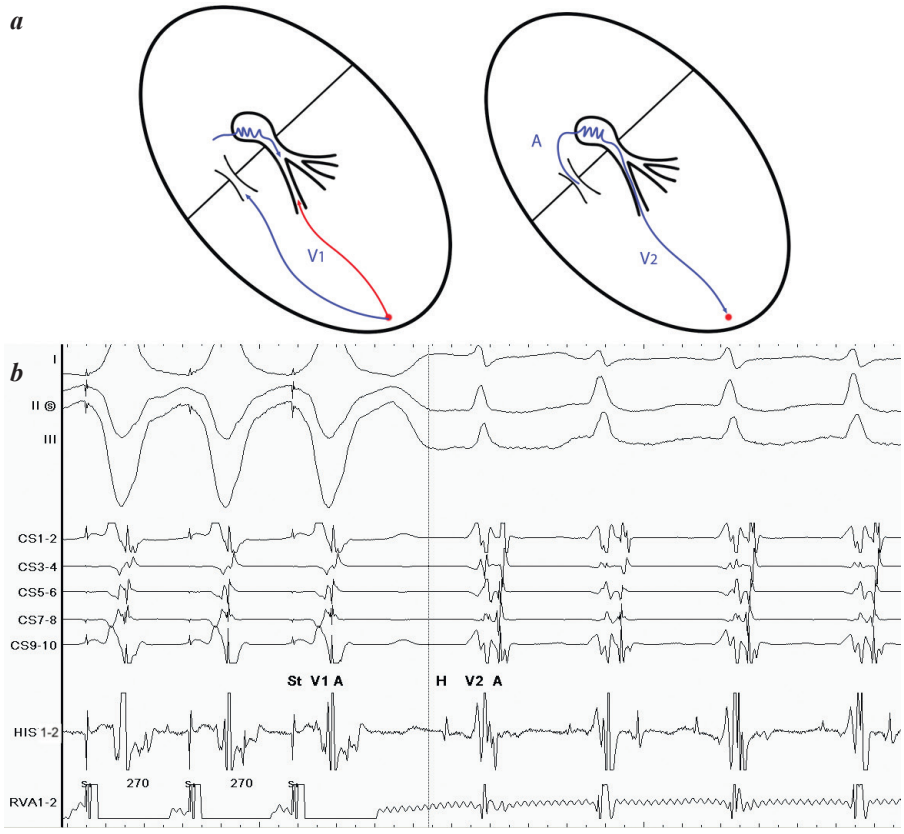

Fig. 10. Entrainment during VOP during orthodromic AVRT. a) Schemes of stimulated excitation wave propagation along the circuit of re-entry. b) ECG and electrograms during entrainment of orthodromic AVRT. During stimulation, the shape of the QRS complexes (the first 3 complexes) has a confluent character, intermediate between stimulated (not shown) and spontaneous (the last 4 complexes). After termination of stimulation, the sequence V-A-H-V is observed.

TECHNICAL DIFFICULTIES IN THE QUALITATIVE ASSESSMENT OF THE RESPONSE TO VOP

Misinterpretation of the response to VOP can be observed in the following situations.

1. During ventricular stimulation there is no 1:1 conduction from ventricles to atria and there is ventriculo-atrial dissociation

The acceleration of atrial activation to the ventricular stimulation rate is critical in VOP. If during VOP atria continue to be excited with tachycardia rather than with stimulation frequency, the test is considered uninformative, and its result cannot be interpreted. To confirm the correctness of this stimulation technique, it is necessary to measure several consecutive A-A intervals at the end of the stimulus series and immediately after its completion. If VOP is performed correctly, A-A intervals equal to the CL of stimulation are recorded during stimulation, then (after termination of stimulation) the A-A intervals increase to the CL of tachycardia. The duration of the broadened first A-A interval may exceed the CL of the tachycardia due to decremental conduction in the re-entry circuit or temporary suppression of the ectopic focus. VA dissociation in VOP may be observed when ventricular stimulation is not frequent enough or not long enough, and when the frequency of tachycardia increases just before or during stimulation. Such a problem can be solved by repeating a longer series of stimuli with the stimulation cycle shortened by 10-20 ms (Fig. 12a).

2. Errors in the assessment of the first atrial electrogram in response to VOP

A. In atypical AVNRT ('slow-slow' and 'fast-slow'), and in AVRT involving a

'slow-functioning' ACP, the V-A interval after the last stimulus may be longer than the CL of stimulation. Then the first atrial EG after the last ventricular stimulus will reflect atrial excitation by the pulse from the penultimate stimulus. The second atrial EG after the last stimulus will be caused by the last stimulus. The interval between the first and second atrial EG (A-A) after the last stimulated ventricular complex will be equal to the CL of the stimulation. If this is overlooked, such a pseudo-«V-A-A-V»-response could be mistakenly interpreted as characteristic of AT (Fig. 12b).

B. The first atrial EG, due to the resumption of AT, may occur at an interval equal to the CL of the stimulation. Since this phenomenon can only occur by chance, it is necessary to repeat VOP several times with different CLs of stimulation to make sure that the «V-A-A-V» response is repeatable.

3. The duration of the H-V interval is longer than the duration of the H-A interval

Sometimes in AVNRT the H-V interval is greater than the H-A interval. Lengthening of the H-V interval may be associated with a decrease in the conduction velocity along the bundle branch or along the «lower common pathway» within the AV node [13]. This ratio of H-V and H-A intervals leads to a shortening or even formation of a negative V-A interval. After the last stimulated atrial EG, a bundle branch EG should follow, and then a ventricular EG. If conduction from AV node to ventricles is delayed, this ventricular EG may be preceded by a second atrial EG due to continuation of AVNRT. This pseudo «V-A-A-V» response can also lead to errors, which can be easily avoided by evaluating the response to VOP not as «V-A-A-V» or «V-A-V» but as «St-V-A-A-A-H-V» or «St-V-A-H-V». [14].

4. Blockage of first-wave tachycardia to the ventricles above the level of the bundle branch

Theoretically, the first excitation return wave can be blocked above the bundle branch (at the level of the «lower common pathway» in the AV node) after stimulation has ended. In this case, the bundle branch potential will not be registered, and the «St-V-A_1-(H_1-V_1)-A_2-H_2-V_2» type response may be formed instead of the «St-V-A_1-A_2-H_2-V_2» response during AVNRT. The registration of such a response to VOP can be expected if a transient spontaneous AV conduction disturbance is observed during SVT. In this situation, the evaluation of the response to VOP may be unreliable, and other diagnostic techniques must be used.

5. The presence of two SVTs in a patient.

An «A-H» type response indicates that the patient is suffering from AVNRT or AVRT. However, this does not rule out the possibility of this patient starting AT. Therefore, after eliminating the substrate of SVT, it is necessary to repeat the diagnostic protocol with programme and pacing to rule out other types of tachycardia.

DIFFERENTIAL DIAGNOSIS OF AVNRT AND ORTHODROMIC AVRT. MORPHOLOGICAL AND QUANTITATIVE ASSESSMENT OF THE VOP RESPONSE

If AT is diagnosed in VOP, you can proceed to mapping and ablation of the tachycardia substrate. If, on the other hand, the possibility of AT is excluded as a result of VOP, the differential diagnosis between AVNRT and orthodromic AVRT can easily be made using the parameters presented in Table 1. Difficulties may arise in situations with concentric atrial activation and V-A interval duration greater than 80 ms. In such cases, in order to distinguish between AVRT involving septal ACP and atypical AVNRT, evaluation of additional quantitative parameters of the response to VOP is necessary.

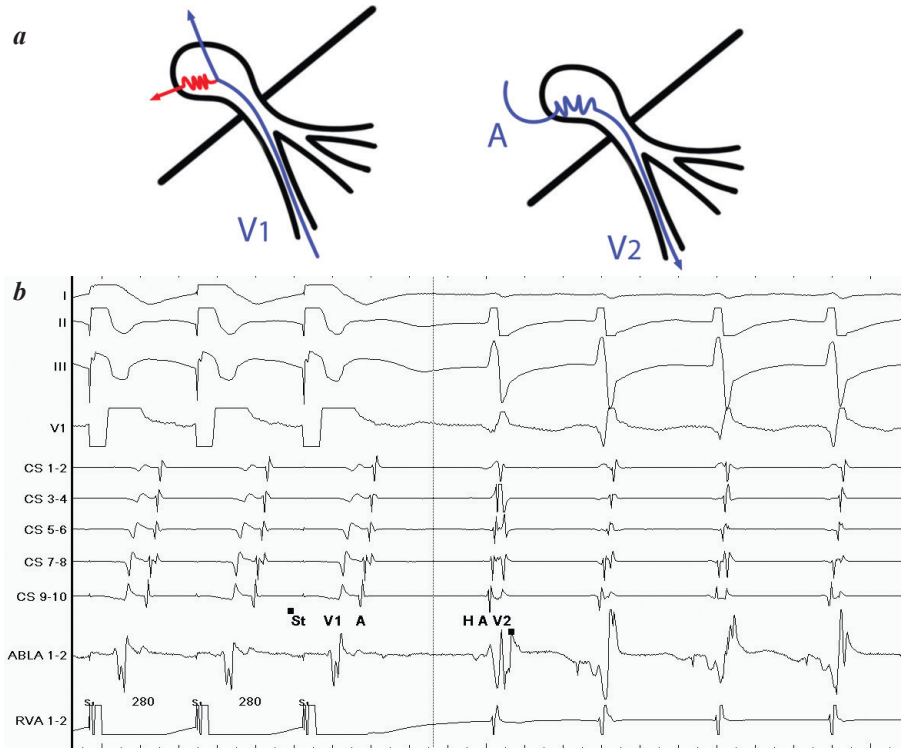

Fig. 11. Entrainment in VOP during AVNRT. a) Schematic depicting the propagation of the stimulated excitation wave along the circuit of re-entry. The retrograde wave (red arrow) travels along the slow pathway of the AV connection, collides with the tachycardia wave (not shown), and disappears. The antegrade wave is conducted into the atria through the fast AV junction pathway and becomes a tachycardia wave after the tachycardia wave and retrograde wave collide. This leads to the formation of a V-A-V type response. b) ECG and electrograms during AVNRT entrainment. During stimulation, the shape of the QRS complexes (the first 2 complexes) has a fully stimulated morphology, and after stimulation (the last 4 complexes) has a spontaneous morphology. After termination of stimulation, the sequence V-A-V or V-A-H-V is observed.

Assessment of ventricular excitation pattern during entrainment

As stated earlier, a V-A-V type response to VOP is indicative of a patient having an AVNRT or AVRT. A sign that helps to diagnose AVRT with high specificity is the formation of confluent QRS complexes during tachycardia entrainment with VOP. Since the formation of confluent QRS complexes is impossible in AVNRT, the registration of this phenomenon during entrainment allows diagnosing orthodromic AVRT (see Fig. 2). Unfortunately, the sensitivity of this sign is relatively low because in AVRT entrainment the collision of excitation fronts occurs more frequently within the conduction system and the QRS complexes on the surface ECG have a stimulated morphology, as in AVNRT (see Fig. 10b), i.e. there is entrainment with local collision of excitation fronts. The following methods can be used to detect the confluent nature of ventricular excitation.

A. Study of local conduction entrainment by antegrade stimulated wave

In order to facilitate the detection of the confluent nature of excitation during an AVRT entrainment (that is, to increase the sensitivity of this diagnostic method), not only the morphology of QRS complexes on the surface ECG, but also the shape and sequence of the intracardiac EGs can be evaluated. Sequential registration of EG the bundle branch and its right limb captured by the antegrade excitation wave shows that the previous impulse crossed the AV node and the bundle branch and collided with the stimulated retrograde wave in the more distal parts of the conduction system or in the working myocardium (see Fig. 10b). It is very important to pay attention to these nuances: the morphology of QRS complexes in these cases will be almost indistinguishable from the stimulated one.

B. Selecting the ventricular stimulation point

It is obvious that the probability of detecting confluent QRS complexes on the surface ECG depends directly on the myocardial volume that the stimulated antegrade wave can entrain before colliding with the retrograde wave. The antegrade excitation wave begins to depolarise the ventricular myocardium only after it leaves the conductive system. At the same time, the retrograde wave begins to excite the myocardium immediately after the next stimulus is applied, and the sooner it enters the conduction system, the less likely the manifest entrainment will be. Therefore, the best stimulation point is the one that is as far away as possible from the cardiac conduction system and as close as possible to the AVRT circuit. Such an area is near the ventricular end of ACP (on the ventricular side of the AV sulcus, opposite the place of the earliest atrial activation during SVT) (Fig. 13). The closer the stimulation point is to the ventricular end of ACP, the more likely it will be possible to register a fusion QRS morphology. Therefore, in AVRT involving septal or right-sided ACP, manifest entrainment (fusion QRS complexes) is easier to detect during stimulation of the apex or basal segments of the RV. Accordingly, at AVRT with participation of the left ventricular ACP, the signs of manifest entrainment are more likely to be received when VOP is conducted from the left ventricle (LV) [11]. By bringing the stimulation point as close as possible to the ACP, one can sometimes achieve a latent collision of the excitation fronts. In this case, the morphology of the imposed QRS complex will be identical to SVT due to complete entrainment of the ventricles by the antegrade wave from the previous impulse (restarted tachycardia wave). (see Fig. 3b,c).

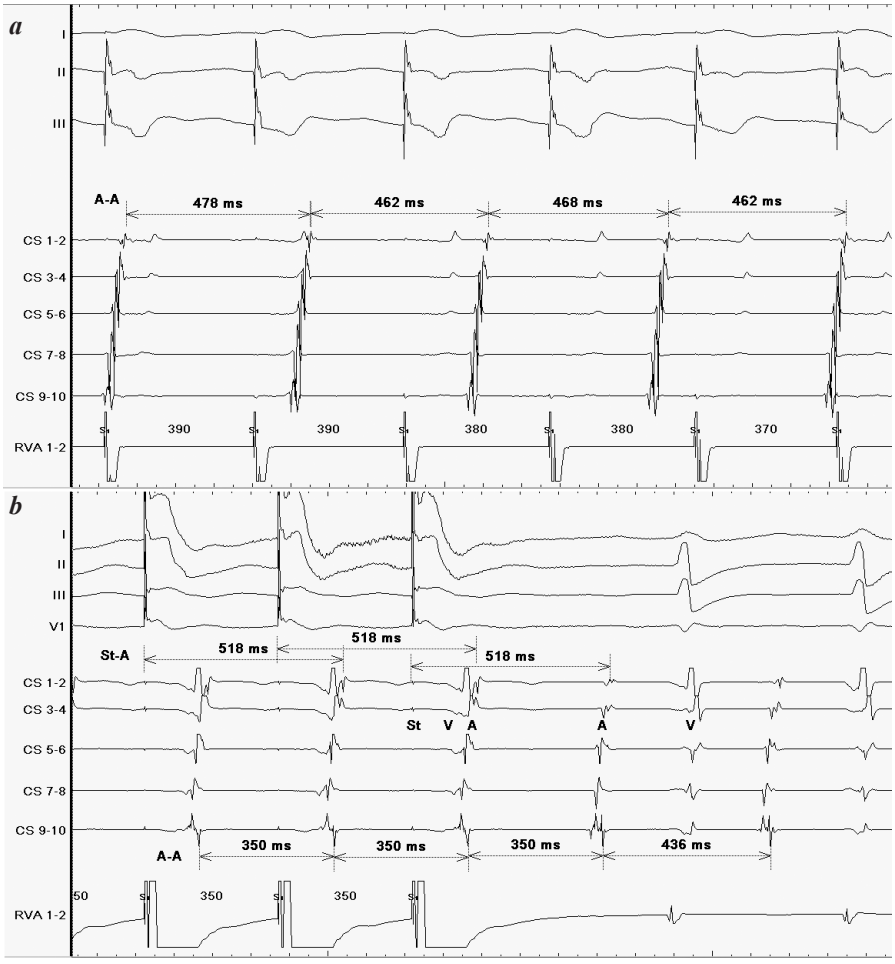

Fig. 12. a) Absence of atrial entrainment in VOP of atrial tachycardia. b) Pseudo-VAAV response in VOP of atypical AVNRT. St-A intervals are longer than St-St intervals, so that the last A-A interval accelerated to the stimulation frequency is entirely after the last stimulus. If this is not taken into account during the electrophysiological study, a false positive diagnosis of atrial tachycardia is possible.

C. Determining the moment of "shift" of atrial activation in VOP

a. The shape of the QRS complexes on the surface ECG.

After the onset of the VOP, there is usually a gradual change in the morphology of the QRS complexes from the form characteristic of SVT, through intermediate forms reflecting different degrees of collision of the excitation fronts, to the stable morphology of the QRS complex (stimulated or drained) (Fig. 14). Identification of the first QRS complex with stable morphology is central to this algorithm.

b. Momentum of acceleration of atrial activity to ventricular stimulation rate

In AVNRT (in the absence of ACP), the excitation wave from the ventricles can reach the atria only through the AV node. For this purpose, successive stimulated excitation waves from the ventricles outpace the corresponding tachycardia waves and entrain the higher parts of the conductive system. In this regard, atrial capture occurs only after complete entrainment of the ventricular myocardium by stimulation (Fig. 14a). In AVRT, the pulses from the VOP will reach the atria immediately after the wave of excitation from the ventricles reaches the ACP (Fig. 14b). In both cases, the «shift» in atrial activation may manifest itself in earlier arousal (shortening of the next A-A interval, the most common variant), slightly delayed atrial activation (prolongation of the next A-A interval, quite rare in decremental ACP), or termination of tachycardia without further atrial activation.

A «shift» in atrial activation that occurs before or simultaneously with the first QRS complex and has stable morphology has a positive predictive value of more than 90% for the diagnosis of AVRT. Similarly, if the «shift» of atrial activation occurs later than the first QRS complex, which has a stable morphology, AVNRT can be diagnosed with a positive predictive value of more than 90% [15-17]. This hallmark is attractive because it does not require the continuation of tachycardia after the termination of the VOP to be assessed. Nevertheless, it is important to keep in mind the possible difficulties in using this technique. These include:

- fluctuations in the duration of the tachycardia cycle;
- rapid atrial entrainment in AVNRT and AT, when using a stimulation CL that is shorter than TCL by more than 40 ms;
- delayed «shift» of atrial activation during AVRT involving decremental ACP;
- rapid atrial entrainment in AVNRT and AT via bystander accessory pathway,
- errors in determining the first QRS complex with stable morphology, the accuracy of which, according to different authors, is about 80% [17].

To increase the diagnostic value of this technique, you can compare stimulation from the apex and basal parts of the ventricles, as close as possible to the place of the earliest atrial activation. In doubtful cases it allows providing earlier acceleration of atrial excitation in AVRT, and later in AVNRT.

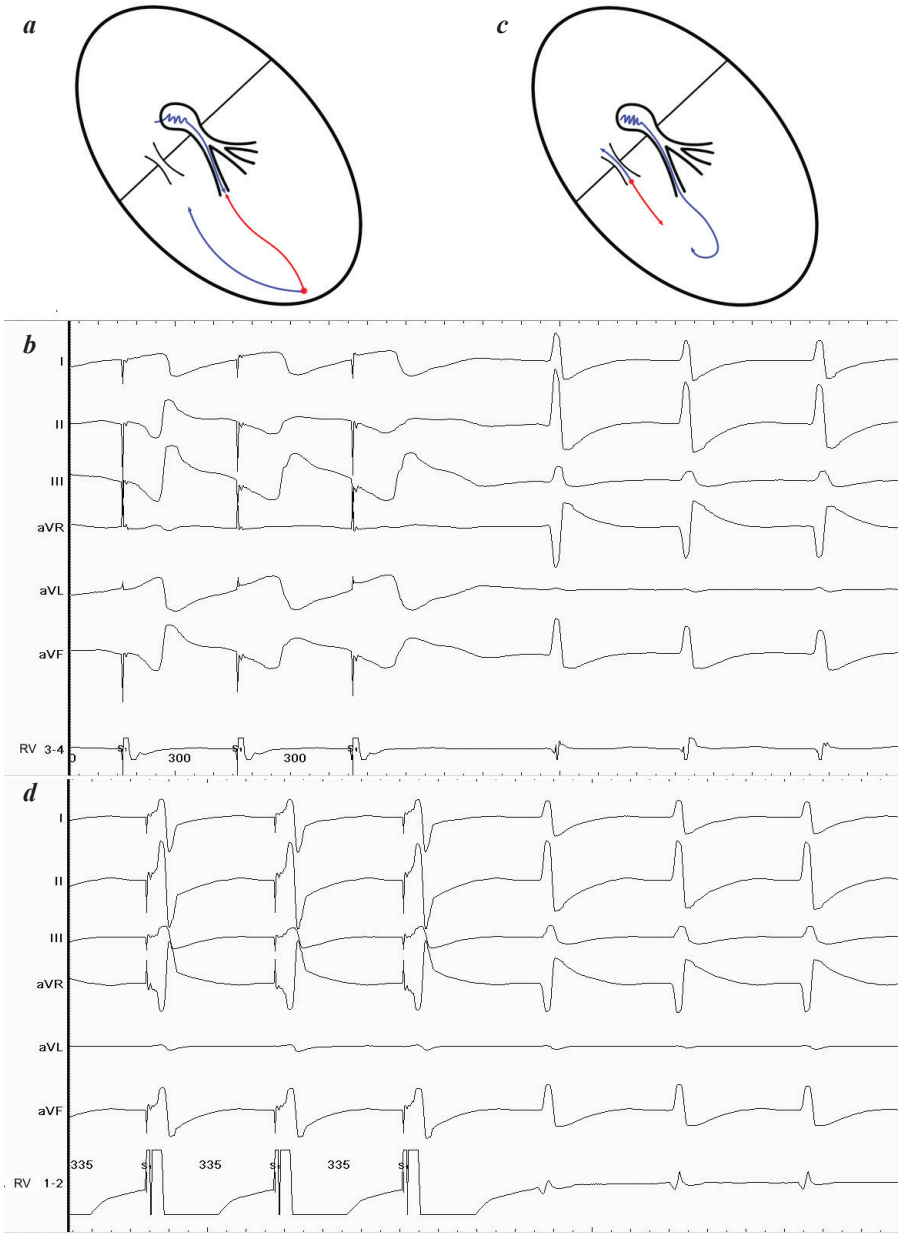

Fig. 13. Differences in the morphology of the QRS complexes during entrainment of orthodromic AVRT from different pacing points in the ventricles. a) Schematic of pacing from the apex region. b) ECG and electrograms during pacing from the apex of the RV. c) Schematic of ventricular pacing versus earliest atrial activation. d) ECG and electrograms during pacing from basal parts of the ventricular myocardium.

Technical difficulties in assessing the character of ventricular excitation in entrainment

A. Concealed bundle branch entrainment during right ventricular basal stimulation

When VOP is performed from the basal septal regions, accidental entrainment of the bundle branch (or proximal left or right leg) can result in relatively narrow QRS complexes. The pseudo-leaky nature of the excitation in this case can lead to overdiagnosis of AVRT. This can be avoided by stimulating above or below the level of the bundle branch and the right leg, checking that the stimulated QRS complexes are fully suspended or «draining» after the tachycardia has subsided.

B. AT entrainment or AVNRT, in the presence of bystander AP

The spillover character of ventricular excitation during SVT entrainment with the help of VOP allows three conclusions to be drawn:

- SVT develops by the mechanism of re-entry,
- impulses conducted along ACP can affect the mechanism of SVT,
- ventricular extrastimuli can lead to «resetting» of re-entry.

The most logical and probable conclusion from these three theses is that the patient has AVRT. Nevertheless, the same signs would theoretically be observed in the presence of a bystander AP whose atrial end is in the re-entry circuit of another tachycardia (AT or AVNRT). Such exceptional circumstances would make it necessary to develop during stimulation two tachycardias, or tachycardia with two circuits, one of which is AVRT. Therefore, such ACP could no longer be considered a «bystander» that is, passively activated, in the full sense of the word. Therefore, ACP ablation would be essential, both clinically and for the detection of a second mechanism of tachycardia in repeated EPI. Thus, the endpoint of SVT with the confluent character of ventricular excitation in VOP indicates the presence of AVRT, irrespective of whether the patient has a second tachycardia with a different mechanism or not [18].

Quantitative parameters of the VOP response

As mentioned above, the detection of the confluent nature of ventricular excitation during SVT ventricular pacing allows the diagnosis of AVRT with a high degree of reliability. The weak side of this technique is the need for additional, sometimes difficult to perform, catheter manipulations (to select the optimal stimulation point or to record local EG) and careful measurement of intracardiac intervals. At the same time, the differential diagnosis between AVRT and AVNRT can be made using simple and objective quantitative parameters: PPI and the interval between the stimulus and the atrial electrogram (St-A).

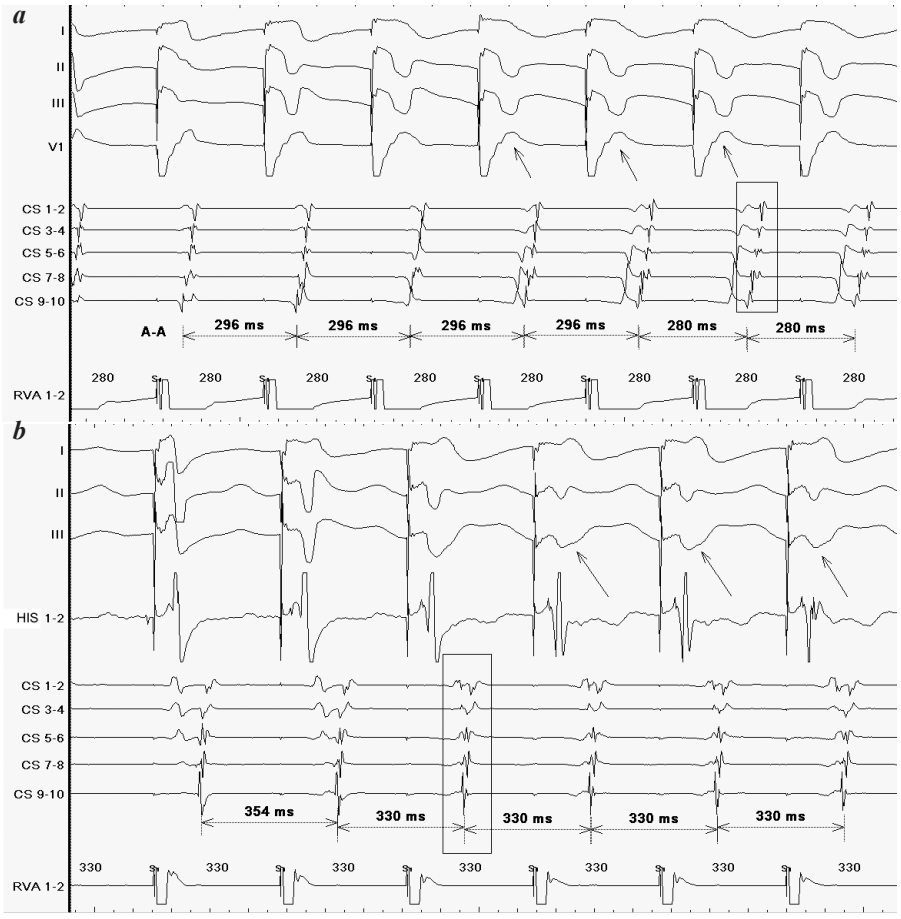

Fig. 14. Determination of the time of the «shift» of the atrial electrogram. a) In AVNRT, the atrial cycle length is accelerated to the pacing cycle length 2 cycles after the first QRS complex with stable morphology (first complex indicated by arrow). b) In AVRT, the atrial cycle length (third A-A interval) is accelerated in one cycle before the stabilisation of the QRS morphology (first complex indicated by arrow). In addition, in the case of AVNRT there is a prolongation of the VA interval, while in AVRT the duration of the VA interval remains stable.

1. PPI-TCL indicator (PPI minus TCL)

The term PPI refers to the time it takes for the last stimulated antegrade excitation wave to complete one revolution in the re-entry circuit and return to the place of stimulation. Accordingly, if the stimulation point is in the re-entry circuit, the PPI value will be almost equal to the tachycardia CL, and the difference between them will not exceed 30 ms [6, 7]. As the distance from the stimulation point to the re-entry circuit increases, the duration of the PPI increases (see Fig. 4). In AVRT, part of the ventricular myocardium is a component of the re-entry circuit (see Fig. 10a). Therefore, in AVRT entrainment, the impulse along the ventricular myocardium reaches the re-entry circuit, passes once through ACP, atrial myocardium, and conduction system, and returns along the ventricular myocardium to the stimulation point. The difference between PPI and TCL is small (usually less than 100 ms) and depends on the mutual location of the stimulation point and ACP. In AVNRT, the ventricular

myocardium is always far away from the tachycardia circuit (see Fig. 11a). Therefore, in AVTRT entrainment, before returning to the point of stimulation, the pulse must pass the conduction system in the direction from the place of stimulation to the re-entry circuit, make one turn in the tachycardia circuit, and then pass through the conduction system a second time in the opposite direction to the ventricular myocardium. Therefore, the PPI-TCL difference is much larger (usually more than 150 ms) in AVNRT entrainment with VOP than in AVNRT entrainment (Fig. 15) [19].

2. Corrected PPI-TCL indicator

The magnitude of PPI can increase not only when the distance from the place of stimulation to the tachycardia circuit increases, but also when there is a decremental slowing of conduction during VOP (the severity of the effect depends on the frequency of contractions). This phenomenon is most typical for the AV node. When performing an AVRT entrainment, the atrial contraction rate increases to the CL of the stimulation. Excitation waves also enter the AV node with higher frequency than during tachycardia, and the time of conduction through the AV node increases decrementally. After termination of stimulation, the first A-H interval (since the H-V interval is fairly constant, it is possible to measure the A-V interval) is often longer than the A-H (or A-V) interval during tachycardia, which has nothing to do with the distance from the stimulation point to the re-entry circuit that we seek to estimate. The changes described can introduce some error into the measurement, so the PPI-TCL difference can be corrected for decremental conduction. To do this, subtract the magnitude of the increase in the duration of the A-H interval (or the A-V interval) in the first return cycle compared to the duration of this interval during the AVRT (Fig. 15) from the result obtained by the PPI-TCL. When corrected for decrementation, this criterion makes it possible to reliably differentiate between AVNRT (kPSI-TCL>110 ms) and AVRT with septal ACP (kPSI-DCT <110 ms) [20].

3. Difference between St-A and V-A intervals

In patients with AVRT, both during tachycardia and during VOP, ventricles and atria are excited sequentially. Both the V-A interval and the St-A interval will be long enough, but comparable with each other. Therefore, the difference between the durations of intervals St-A and V-A will tend to zero (Fig. 16a,b).

In patients with AVNRT, atrial and ventricular activation during tachycardia occur simultaneously, while in VOP it occurs sequentially. Therefore, the duration of the V-A interval during a typical AVNRT tends to zero, and during ventricular stimulation in a patient with AVNRT the St-A interval will be quite long. The difference between the intervals St-A and V-A will be relatively large (Fig. 16c,d).

Accordingly, the difference between the V-A interval values during entrainment and during tachycardia should be greater in AVNRT patients than in AVNRT patients (Fig. 15, Fig. 16). In AVRT the (St-A)-(V-A) difference is usually < 85 ms, and in AVNRT it is (St-A)-(V-A) > 85 ms [19].

4. Differential entrainment

To exclude the possibility of delayed antegrade conduction (due to the decremental AV node or its «double physiology»), we proposed a way to distinguish the parameters cPPI-TCL and (St-A)-(V-A) described above during stimulation of basal and apex portions of the RV. If the values of these parameters during basal stimulation exceed the values of the same parameters during apex stimulation by 20-30 ms, it is more likely that AVNRT is present (if stimulation is closer to the AV node, decremental conduction along the node is more pronounced). Using a single CL for both stimulation points eliminates the decremental prolongation of the A-H interval mentioned above. The term «differential entrainment» is used to describe this phenomenon [21].

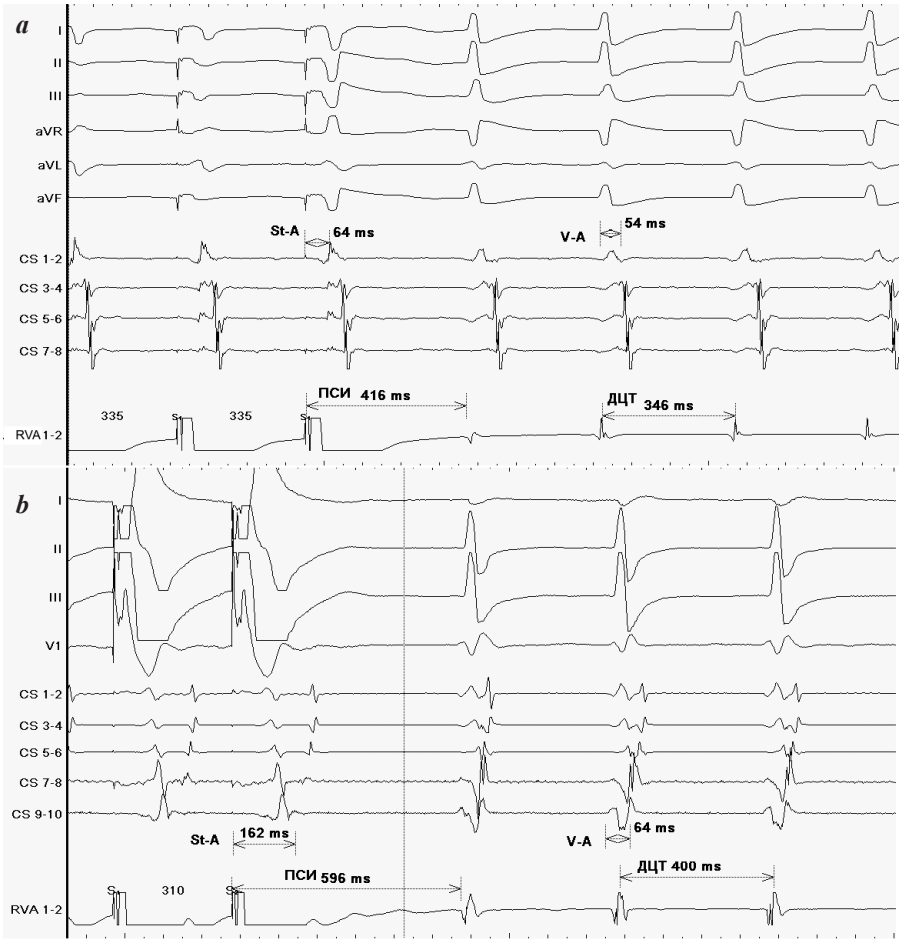

Fig. 15. PPI-TCL and (St-A)-(V-A) difference in AVRT (A) and AVNRT (B). A. PPI-TCL difference is 416-346=70 ms. Difference (St-A)-(V-A)=64-54=10ms. These values are diagnostic for AVRT. B. PPI-TCL difference is 596-400=196 ms. Difference (St-A)-(V-A)=162-64-98 ms. These values are diagnostic for AVNRT.

Accordingly, the cPPI-TCL and (St-A)-(V-A) parameters are also called differential.

Technical difficulties in determining the PPI-TCL, cPPI-TCL, and (St-A)-(V-A) differences

1. Determination of PPI-TCL, cPPI-TCL and (St-A)-(V-A) difference in patients with left lateral ACP

Recall that cPPI-TCL > can be 110 ms and (St-A)-(V-A) > 85 ms and that in AVRT the pacing point is distant from the re-entry circuit (AVRT entrainment involving left ventricular ACP is performed from the apex of the right ventricle. This can easily be avoided by following the algorithm described earlier. A patient with left-sided ACP will have an eccentric sequence of atrial activation during AVRT. It is therefore possible to proceed to mapping and ablation without additional pacing techniques immediately after the tachycardia has been triggered and AT has been excluded from the analysis of the type of response to VOP (VAV or VAAV).

2. Determination of PPI-TCL, cPPI-TCL and (St-A)-(V-A) difference in patients with decremental or slow ACP

In a patient with a slow or decremental ACP, the cPPI-TCL difference score may also be >110 ms, and (St-A)-(V-A) >85 ms. To avoid overdiagnosis of AVNRT in these patients, attention should be paid to the duration of the V-A interval. If a long V-A interval (V-A > ½ R-R) is observed during SVT, it is not appropriate to perform VOP.

3. Determination of PPI-TCL, cPPI-TCL and (St-A)-(V-A) difference in patients with AVRT and dual AV node conduction physiology

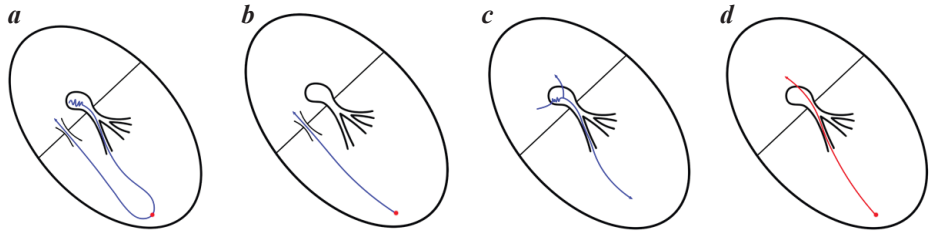

Fig. 16. Schematic representation of the differences in the formation of St-A and V-A intervals in AVRT (a and b) and AVNRT (c and d). a) The excitation wave in AVRT spreads from the apex (exit from Purkyně fibers to the working myocardium) to the base of the heart. A relatively long V-A interval is formed. b) The stimulated excitation wave propagates from the apex of the right ventricle, as in tachycardia. A relatively long St-A interval is formed. c) The excitation wave in AVNRT spreads simultaneously along the fast pathway to the atria and along the conduction system to the ventricles. V-A interval is formed, the value of which tends to zero due to almost simultaneous excitation of atria and ventricles. d) Stimulated excitation wave spreads to atria through conductive system. A relatively long St-A interval is formed.

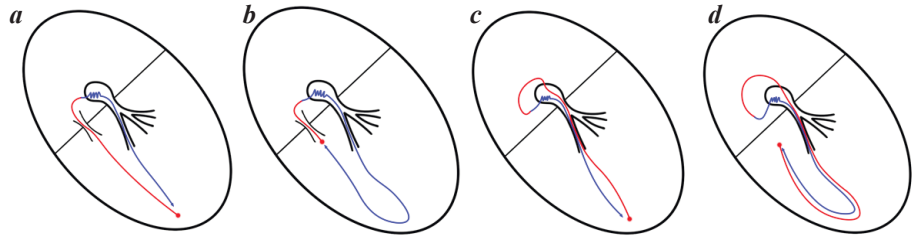

Fig. 17. Differential entrainment in AVRT (a and b) and AVNRT (c and d). a) The stimulated excitation wave in AVRT spreads from the apex (exit from Purkyně fibers into the working myocardium) to the base of the heart, and then returns to the stimulation point through the conduction system. The post-stimulation interval is similar in duration to the tachycardia cycle b) The stimulated excitation wave propagates from the ventricular base through the accessory pathway into the atria, then through the conduction system into the ventricles, and then from the Purkyně fibers in the apical ventricular region to the stimulation point. The post-stimulation interval is similar in duration to the tachycardia cycle. c) The stimulated excitation wave in AVNRT spreads simultaneously along the fast pathway to the atria and along the conduction system to the ventricles. The V-A interval is formed, the value of which tends towards zero due to the almost simultaneous excitation of the atria and ventricles. d) The stimulated excitation wave propagates from the basal region to the apex of the heart, then enters the conduction system and is conducted to the atria, makes one turn in the re-entry circuit within the node AV, returns through the conduction system to the apex of the heart and then reaches the stimulation point in the basal region of the ventricle through the working myocardium. A relatively long St-A interval is formed.

During entrainment AVRT, which uses the «fast» AV connection pathway as the antegrade pathway, the antegrade conduction may switch to the «slow» AV connection pathway during VOP. In this case, the A-H interval in the first recurrent contraction after the termination of stimulation will be significantly prolonged, which may lead to overdiagnosis of AVNRT [22]. The above correction of the PPI-TCL difference (replacing it with cPPI-TCL) helps to avoid complications in patients with a combination of «double conduction» along the AV node and AVRT with septal ACP involvement. The use of (St-A)-(V-A) parameter, in which there is no estimation of conduction along the antegrade knee at all, also allows avoiding errors associated with decremental or «double» conduction along the AV node [23].

4. Selection of the stimulation point for the determination of cPPI-TCL and (St-A)-(V-A)

Ventricular pacing near the sulcus AV means that the pacing point is closer to the ACP involved in the AVRT circuit (when performed in the area of earliest retrograde atrial activation) and further from the AVNRT circuit (due to the extra distance the excitation wave must travel through the ventricular myocardium before entering the conduction system at the apex of the heart) (Fig. 17). There-

fore, in comparison with the apical stimulation, the stimulation of the basal parts of the ventricles:

- increases the values of cPPI-TCL and (St-A)-(V-A) in AVNRT
- decreases cPPI-TCL and (St-A)-(V-A) at AVRT.

Thus, when performing entrainment from the basal segments of the ventricles, the cPPI-TCL and (St-A)-(V-A) values allow for better discrimination between AVNRT and AVRT regardless of ACP localisation. In addition, as mentioned earlier, basal stimulation of the ventricles, as close as possible to the site of earliest retrograde activation, helps to reveal the confluent nature of the excitation (see Fig. 10) and to obtain signs of manifest entrainment in AVRT.

Basal stimulation has some technical difficulties. These include the less stable position of the catheters in this area, the possibility of rhythm influence on the atria or the bundle branch, which can complicate the interpretation of the results and lead to false conclusions. Therefore, VOP from the basal compartments should be used only when the apex stimulation registers a «V-A-V» response, entrainment with full ventricular retrograde entrainment is noted, and the cPPI-TCL and (St-A)-(V-A) values demonstrate borderline values.

What if the answer to VOP is not subject to interpretation?

Thus, VOP is most effective and useful when relatively slow tachycardia continues after the termination of stimulation, and retrograde conduction along the AV node allows acceleration of atrial activation to the rate of ventricular stimulation.

At the same time, there are two types of response to VOP that are often considered inappropriate for interpretation, but can still serve as a source of important diagnostic information.

Atria are not accelerated before CL stimulation. If the atria fail to accelerate in response to stimulation from CL on several VOP attempts, the diagnosis of AVRT can be ruled out and it can be said with a high degree of probability that the mechanism of SVT is AT (see Fig. 12).

Atria are accelerated before CL stimulation, but SVT is terminated after termination of VOP. The cause of termination of SVT during stimulation is usually the blocking of the next antegrade excitation wave by the AV node and its subsequent entrainment by the retrograde wave. After termination of stimulation, the last antegrade wave of excitation cannot return to the ventricles in the form of an echo response and continue tachycardia. In such situations, artificial shortening of the refractory period of AV node by intravenous injection of atropine often helps. If it is still not possible to overcome this problem, the response to VOP may still be of diagnostic value, e.g. in assessing atrial capture time or in performing differential entrainment (although in the situation where SVT is stopped after VOP termination, we cannot speak of entrainment and the term «differential entrainment» is not applicable). If SVT is repeatedly terminated during VOP, frequent ventricular stimulation with a short series of pulses (3 to 6) with a CL of 200-250 ms may be useful. In 60% of cases ventricles will dissociate from SVT (excludes AVRT) or SVT will be stopped without conduction on atria (excludes AT) [9].

CONCLUSION

Thus, VOP is a reliable and relatively simple way of differential diagnosis of sustained SVT. This stimulation technique gives the electrophysiologist a large amount of qualitative and quantitative information without greatly increasing the duration of the EPE.

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