

Atrioventricular nodal reentrant tachycardia

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Acronyms

AV: atrioventricular

AVNRT: atrioventricular nodal reentrant tachycardia

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ABSTRACT

Atrioventricular nodal reentrant tachycardia represents a significant part of tachyarrhythmias seen by attending physicians in emergency departments. It is the most common paroxysmal tachycardia, and it occurs primarily in women between their fourth and fifth decades of life. Diagnosis can be made with high reliability by surface electrocardiogram and complex or borderline cases by electrophysiological studies. Its treatment should be aimed towards definitive healing by endocardial ablation technique, but depending on availability or patient preferences drug treatment may be indicated. This article summarizes the diagnostic techniques, types of atrioventricular nodal reentrant tachycardia and major aspects of treatment.

Key words: Tachycardia, Atrioventricular Nodal Reentry, Catheter Ablation, Pulsed Radiofrequency Treatment, Electrocardiography, Cardiac Electrophysiologic Techniques

Taquicardia por reentrada del nodo aurículo-ventricular

RESUMEN

La taquicardia por reentrada del nodo aurículo-ventricular representa una parte significativa de las taquiarritmias que los médicos asisten en los servicios de urgencias. Constituye la taquicardia paroxística más frecuente, y es característico que se presente sobre todo en mujeres entre la cuarta y la quinta década de la vida. El diagnóstico puede hacerse con alta fiabilidad mediante el electrocardiograma de superficie y algunos casos complejos o dudosos, mediante los estudios electrofisiológicos. Su tratamiento debe ser dirigido hacia la curación definitiva mediante la técnica de ablación endocavitaria, pero de acuerdo a su disponibilidad o las preferencias del paciente se puede indicar tratamiento farmacológico. En este artículo se resumen las técnicas diagnósticas, los tipos de taquicardia por reentrada intranodal y los principales aspectos del tratamiento.

Palabras clave: Taquicardia por Reentrada en el Nodo Atrioventricular, Ablación por Catéter, Tratamiento de Radiofrecuencia Pulsada, Electrocardiografía, Técnicas Electrofisiológicas Cardíacas

INTRODUCTION

In the history of diagnosis and treatment of cardiac arrhythmias it is important to remember several important events that have contributed to the current knowledge in this field. In this regard, it can be noted that between 1893 and 1907 the heart's electrical conduction system was described, in the mid-twentieth century its different action potentials were determined and in 1968, it was possible to register the bundle of His depolarization via an intravascular catheter-electrode¹. Since then the evolution of recording and stimulation techniques for the study of arrhythmias has significantly increased the understanding of its mechanisms, and has allowed the development of new therapeutic modalities based on the selective destruction of myocardial areas, on which essential parts of arrhythmogenic substrates are based¹.

In this regard, the development of surgical techniques to treat arrhythmias began in 1968 when Dr. Hill Sealy successfully interrupted an atrioventricular (AV) accessory pathway located by epicardial mapping to the right of the AV groove. This event started the development of surgical techniques for the treatment of Wolff-Parkinson-White syndrome, and subsequently innovative techniques were developed in the treatment of atrioventricular nodal reentrant tachycardia (AVNRT), atrial tachycardia, atrial flutter and atrial fibrillation².

In 1979, there was an accident that changed the history of the treatment of cardiac arrhythmias, when during a study, an AV block through an endocardial catheter occurred. Later, at the beginning of the eighties, the fulguration technique with direct current was described and thus interventional electrophysiology was born. Other forms of energy were sought and that is how radiofrequency came into use¹, a technique introduced in the clinical practice in 1982³, and electrosurgery is the most typical application within it.

According to Ardashev⁴, the first to use this type of energy was Huang in the late eighties. Since then its use has spread worldwide for the definitive treatment of most supraventricular tachyarrhythmias⁴, among these, the AVNRT⁵⁻⁷.

DIAGNOSIS

Epidemiology, interview and physical examination

From the clinical point of view the AVNRT is more

common in women², with a mean age of 40 ± 5 years, and symptoms can begin at any age. Presentation is less common in young children. It is usually seen in patients without structural heart disease. Its presentation is sporadic and familial cases are rare.

Presenting symptoms can range from palpitations, dyspnea, angina (chest pain is often not typical), pre-syncope and even syncope. Some patients are simply interpreted as anxious, so their diagnosis may be much delayed, others may present with symptoms of real heart failure. The most serious forms of presentation are associated with very fast episodes, prior structural heart disease and long pauses at the time the arrhythmia ends, usually explained by a transient depression in sinus node function. The paroxysms may be self-limiting, reversed by the same patient by vagal maneuvers, or may require the assistance of emergency services for their control. In our experience we have found that almost all patients describe the paroxysmal onset of tachycardia, but the ending for some is seen as sudden and for others of progressive slowing-down. In electrophysiological studies and Holter records it can be shown that a good number of patients continue with a sinus tachycardia in the first minutes after self-limiting the AVNRT. This is why some of these patients may not perceive clearly the termination of paroxysmal tachycardia. We point out these elements because the interview of the patient may erroneously suggest a cooling mechanism of focus, as the one that is common in automatic tachycardias, and wrongly lead us to other types of tachyarrhythmias. The annoying feeling of beating in the neck is also noteworthy, which is displayed at inspection as cannon A waves with a 1:1 ratio, with respect to the arterial beats perceived in the radial pulse, although it can also be noted in orthodromic tachycardia by accessory pathways, less evident in this context, because atrioventricular conduction time is longer in these tachycardias, which causes the development of intra-atrial pressures that are lower than those reached during the AVNRT. The mechanism of this symptom/sign is the unison contraction of the atria and ventricles, causing a reverse blood flow into the jugular veins during atrial systole. The maximum heart rate that we have observed in our patients is 290 beats per minute and the lowest of 130, although these values do not reflect the behavior of the majority of cases. These data show that the frequency of tachycardia do not lead diagnosis because it over-

laps with a wide variety of possibilities. A minority of patients reports feeling like urinating during the arrhythmia crises, and there is in fact an increase in diuresis. This seems to be due to the release of atrial natriuretic peptide.

Electrocardiographic diagnosis

It is a regular tachycardia with narrow QRS complex and retrograde P waves which relate 1:1 with QRS (Fig 1). The retrograde character of P waves determines a $RP < PR$ interval in most cases. Pseudo r' waves are produced (false terminal QRS forces) in leads V1 and aVR and pseudo s waves (false terminal QRS forces) in leads with predominantly negative QRS complexes. The P wave should be compulsorily negative in leads DII, DIII y aVF (expressed as a pseudo s or a frankly defined P wave), reflecting the caudo-cranial activation of the right atrium. There are exceptions in almost all areas of medicine, and so there are in this point the AVNRT. In the fast-slow variant a $RP > PR$ interval appears (retrograde arm of the circuit with very slow conduction) and a differentiation with sinus, auricular and permanent reciprocating tachycardia of AV junction (Coummel tachycardia) must be made. It should be noted that $PR < RP$ intervals may appear but of considerable duration, which leads us to misdiagnosis of orthodromic tachycardia, that is why PR intervals between 60-70 ms are in the doubtful zone, and those >80 ms reduce AVNRT possibility almost entirely, but do not exclude it completely. The existence of intermediate types of AVNRT forces us to be prepared to meet, albeit rarely, with various PR intervals. In these cases only the electrophysiological study (EPS) can make the difference. In a now classic article in our environment, Professor Dr. Margarita Dorantes defines that AVNRT electrocardiographic presentations are summarized as: P superimposed on the QRS as terminal force, $PR < 60$ ms, $PR \geq$

60 ms, PR greater than the RP and P wave before QRS⁸.

Occasionally there is not a 1:1 relationship between the P wave and the QRS, thus there is an unusual electrocardiographic presentation of the AVNRT. A slow tachycardia with a $PR = RP$ interval (assuming the doctor informing the electrocardiogram does not notice the false r' or pseudo s waves, and only visualizes the P wave which is found somewhere between two QRS complexes of the tachycardia). This happens by the occurrence of a 2:1 anterograde conduction block at the bundle of His level, with intermittent absence in the QRS generation. Other higher degrees of block, such as high grade or advanced, would be in theory spontaneously possible, which makes the AVNRT an arrhythmia of irregular behavior if such blocking occurs intermittently in short periods of time. In our experience we have never found this assumption in a spontaneous form of presentation, but transient production of this type of block in a patient with common AVNRT during an ablation procedure, allowed proving that this could be possible in patients with Hisian

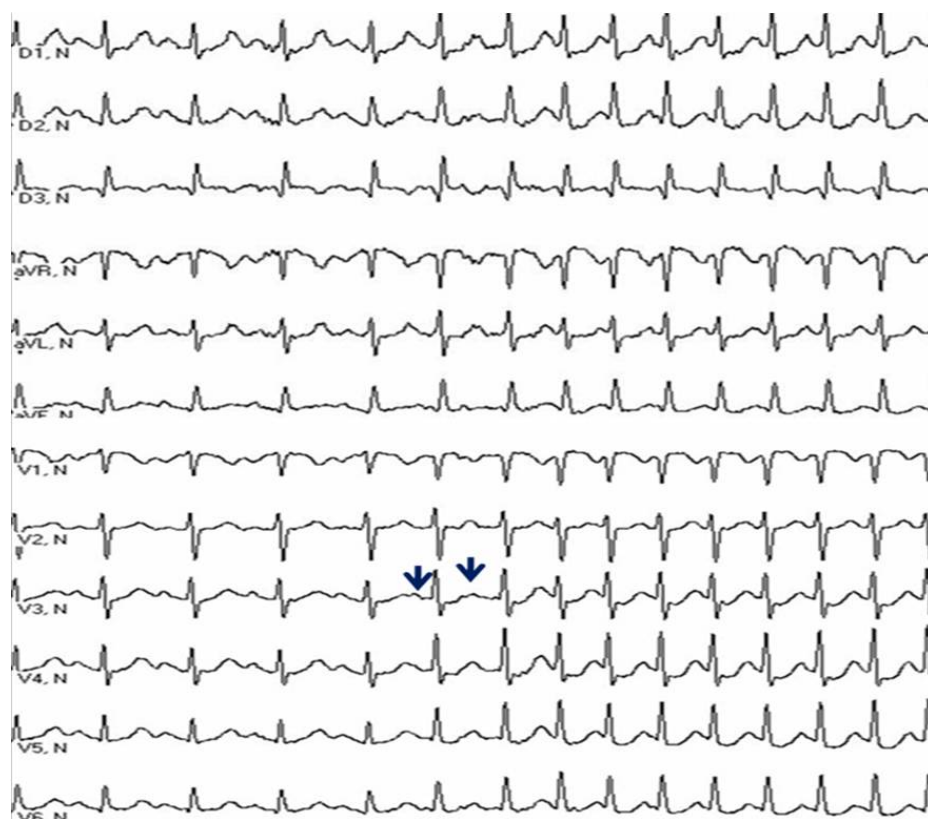


Figure 1. 12-lead electrocardiogram obtained from a 44-year-old woman during the spontaneous episode of a common AVNRT, triggered by a pair of atrial extrasystoles (arrows).

lesions. We must remember that the bundle of His is not a mandatory part of the circuit that determines the AVNRT, so its spontaneous, intentional or iatrogenic injury does not stop the tachycardia.

Occasionally retrograde conduction blocks to the atria could occur, producing certain degree of atrio-ventricular dissociation, but some prestigious authors as Farré⁹ doubt the existence of this possibility, and argue that the few reported cases have probably been wrongly labeled as AVNRT. The existence of this presentation form would support the confining of the circuit to the AV node, when many authors have confirmed that atrial fibers also participate in the AVNRT circuit. The latter determines the impossibility of AV dissociation in this type of arrhythmia.

On multiple occasions, while monitoring these patients during an ablation procedure, there has been a spontaneous transformation of the AVNRT in atrial fibrillation and vice versa, so a story told of tachycardia with beating in the neck and a documentation of atrial fibrillation should lead us to consider such possibility, especially if it is a woman in her fourth decade of life.

Between 25-50% of patients may have an ST segment depression in the presence of normal coronary arteries during an episode of AVNRT, and a transient inversion of the post-tachycardic T wave can be documented in 40% of cases¹⁰.

Finally, it is noteworthy that the AVNRT could manifest with wide QRS upon the occurrence of: phase 3 branch block, preexisting bundle branch block or presence of accessory pathway with anterograde conduction. In the latter case it would be part of the differential diagnosis of pre-excited regular tachycardia.

Electrophysiological diagnosis

The electrophysiological diagnosis of these tachycardias is being performed consistently since the seventies. According to Jackman *et al.*¹¹, in 1973, Denes describes the presence of dual atrioventricular nodal pathway in the EPS of patients with AVNRT. It is now known that dashed curves of refractory period may not be present in all patients with this tachycardia¹¹. From this concept it was classically recognized that AVNRT mechanism was attributed to the presence of nodal double pathway: one fast, with prolonged refractory period and one slow, with short refractory period¹².

In 1981, an important step in the knowledge of reentry in the vicinity of the AV node was taken, when, according to Civera¹³, Sung *et al.* showed that retrograde conduction through the slow pathway produced initial activation in the back of the septum, close to the ostium of the coronary sinus, while retrograde conduction through the fast pathway produces initial activation at the vertex of the triangle of Koch in the distal hisiogram¹³.

The mechanism of this arrhythmia, although not fully understood, is reasonably characterized. Thus, it is now accepted that the AV node is a more widespread structure than previously believed, situated in triangle of Koch¹⁴, which is bounded above and anteriorly by the tendon of Todaro, at the back, by the coronary sinus, and below, by the septal insertion of the tricuspid valve, and into the base of the triangle there is the ostium of the coronary sinus¹⁵.

From the functional point of view, it consists of at least two connections with the surrounding atrial myocardium. The first one, in a more cranial position which is known as fast nodal pathway, and presents a fast conduction velocity and a prolonged refractory period, which causes that at slower atrial rates, the cardiac electrical pulse is conducted through it in a short time, whereas at fast frequencies it cannot pass through this structure. The second connection, of a more caudal location and known as slow nodal pathway, presents a slow conduction velocity and a short refractory period, which causes that, at fast atrial frequencies when there is no conduction through the fast nodal pathway, the electrical impulse is conducted and reaches the ventricles in a longer time.

In some individuals it is possible to prove, in the electrophysiology laboratory, the distinct presence of these two pathways, by atrial electrical extra-stimuli introduced progressively, increasingly early, until conduction through the nodal fast pathway is blocked. At this time, there is a sudden and significant increase (> 50 ms) in the atrio-hisian (AH interval) conduction interval. This phenomenon is known as double nodal pathway physiology and the moment of the significant increase as AH interval jump^{7,16,17}.

The double nodal pathway is a common mode of behavior of AV nodal conduction, as it is present in 10 to 46% of individuals subjected to an EPS, according to different series^{11,18,19}. Its presence is not universal in patients with AVNRT, nor does it explain in any way that the patient will present such AV junctional tachy-

cardia⁹.

These different characteristics of the two nodal pathways also cause that with an atrial extrasystole with short coupling, the electrical impulse can find the fast nodal pathway in its refractory period, and travel very slowly through the slow nodal pathway. The slow conduction through the latter pathway can cause, in turn, that the fast nodal pathway has time to recover its conduction ability at the time the impulse reaches the distal end of the slow nodal pathway in its common insertion with the fast nodal pathway on the bundle of His. In this situation, the impulse will travel in the anterograde direction, via the bundle of His into the ventricles, and also in a retrograde manner through the fast AV nodal pathway toward the atria, from where it can reenter the slow AV nodal pathway, perpetuate this sequence and give rise to the so-called common or slow-fast AVNRT (Figure 2), which can be induced by both atrial and ventricular extrasystoles. This sequence may also occur in reverse, resulting in the so-called uncommon or fast-slow AVNRT, which can be also induced both ways with a high probability from the ventricle, and with an incidence of approximately 10 times lower than the common AVNRT⁷.

The electrophysiological diagnosis of AVNRT is performed taking into account¹³:

- The beginning and the end of tachycardia with programmed stimulation of the heart: with programmed stimulation, the presence of discontinuous AV or VA (ventricular-atrial) conduction can be demonstrated in most cases, however, this is not a necessary nor sufficient criterion for the diagnosis of nodal reentrant.
- An atrial activation sequence during tachycardia consistent with an origin near the AV node (in the atrial septum, between the areas of hisiogram registration and the ostium of the

coronary sinus). The first atrial activation recorded by the catheter of His suggests common AVNRT, and the first activation in proximal coronary sinus, suggests the uncommon type.

- The absence of positive criteria that indicate the use of a septal AV accessory pathway¹³.

TYPES OF AVNRT

Lockwood *et al.*²⁰ in their experience with 734 patients referred to their service for ablation of AVNRT, found that 515 cases (77%) had the slow-fast type, 80 patients (11%) slow-slow variant, while 89 (12%) had the fast-slow²⁰. The slow-fast variant comprises three well-defined electro-anatomic subtypes with practical consequences in the ablation of the slow pathway of the circuit. In the first type (the most common) slow pathway is an inferior right extension, another variant has inferior left extension, and there is a rarer presentation form (2%), where the anterograde slow pathway comes from a region located in the postero-inferior mitral annulus, this location in the posterior region of the mitral annulus is a therapeutic challenge because

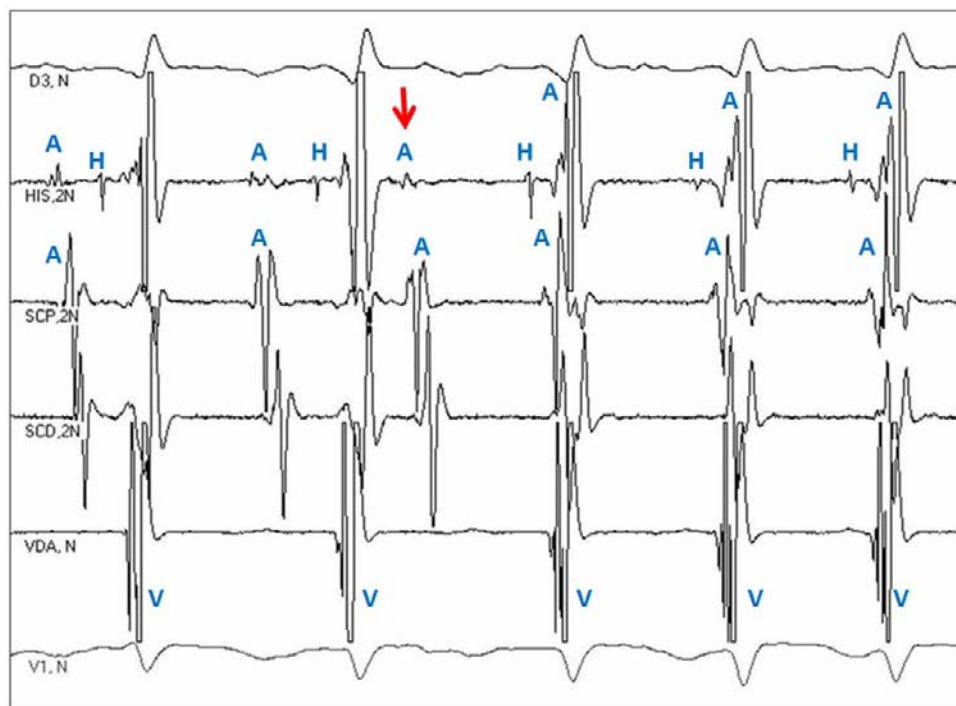


Figure 2. EPS of previous case. The first two are sinus beats. The arrow points to an atrial extrasystole, which induces a considerable prolongation (over 50 ms) of AH interval (compare it with the two previous AH intervals). The occurrence of this phenomenon indicates dual nodal physiology. After this a common AVNRT is established, notice that atrial electrograms coincide with the QRS complex in leads V1 and DIII (first and last recording channel). His: hisiogram recording, PCS and DCS: recordings from the proximal and distal coronary sinus, respectively, RVA: right ventricular apex. A: atrial electrograms, V: ventricular electrograms, H: bundle of His depolarization.

the arrhythmia is presented simulating orthodromic tachycardia, which uses a left posterior accessory pathway. Note figures 3 and 4 for an illustrated view of this explanation.

In the case presented by Montes *et al.*²¹ it is observed that the first retrograde atrial activation occurred at the poles 5 and 6 of a decapolar catheter placed in the coronary sinus. The AV node entry from these odd pathways occurs as follows: 1) through the fossa ovalis and the previous entry, 2) through the coronary sinus musculature and subsequent entry, and 3) directly through the mitral annulus²². In the latter example ablation is achieved from the left heart and can easily simulate left concealed accessory pathway. The occurrence of a 2 to 1 phenomenon allows suspecting this slow-fast AVNRT subvariant. This means that the bundle of His is depolarized twice, first by a wavefront coming from the fast pathway and a few milli-seconds later by another stimulus from the slow pathway, which runs from the left atrium²³.

Slow – fast

In this form of tachycardia, atrial activation occurs before, during or just after the QRS complex while maintaining an AH / HA ratio (Atrium-His/His-Atrium) > 1. In particular an AH / HA > 3 and a AV interval measured from the onset of ventricular activation on the surface electrocardiogram to the earlier deflection of atrial activation in the His bundle electrogram < 60 ms, or a AV interval measured from the right atrium < 95 ms, are diagnosis of slow-fast AVNRT. Ross *et al.*²⁴ classified in 1985 this variety of slow-fast tachycardia in two subtypes: type A or anterior, which is the most common and is characterized by presenting AV and HA short intervals during tachycardia (AV less than or equal to 40 ms)²⁴, atriogram registration within the QRS complex and earliest

atrial activation in the hisiogram derivation. In type B or posterior, which is much rarer, retrograde atrial activation during tachycardia is located on the posterior septum, in the coronary sinus ostium area. AV and HA intervals during tachycardia may vary but they are usually longer than in the previous type (AV greater than 40 ms, the ratio AH / HA < 1)¹³.

Subsequent studies^{13,15,24}, based on analysis of patients with multiple forms of nodal reentrant suggest that anterior and posterior slow-fast types of tachycardia use different circuits and do not merely represent a reversal of the common circuit (pathways α and β), but a different form of reentry. Thus, type B tachycardias use slow pathway (α) as an antegrade way, and as retrograde way a posterior outlet pathway and intermediate conduction times (γ). Some cases of tachycardias, classified as posterior type, show long AH and HA intervals, with similar or superior AH / HA relations¹³.

Fast – Slow

In this form of tachycardia (5-10% of all cases of

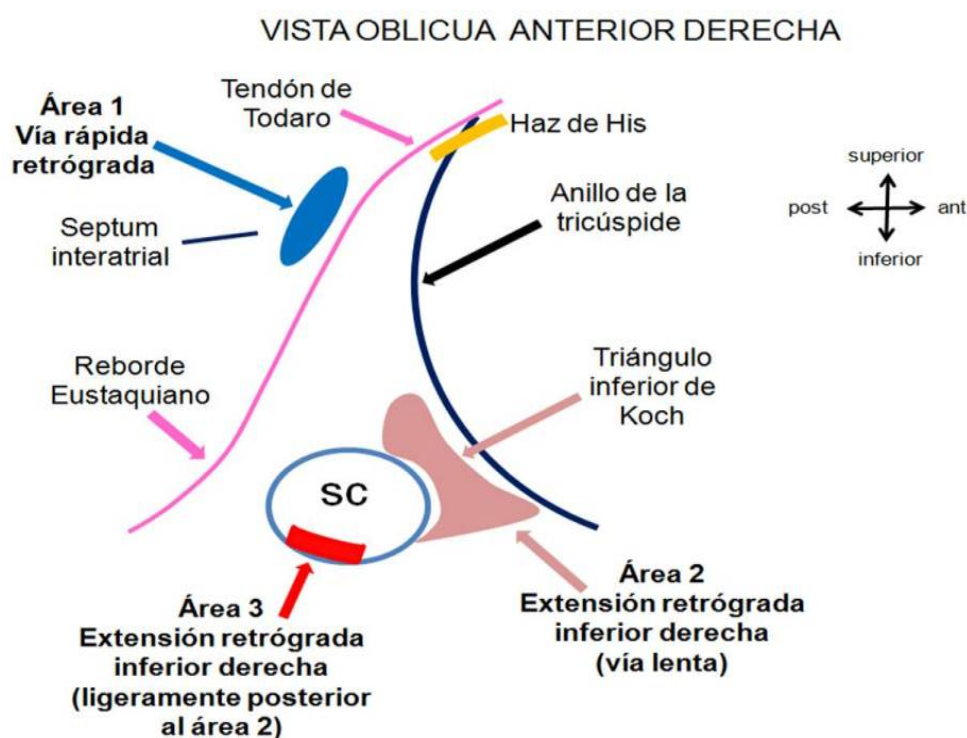


Figure 3. Schematic representation of the earliest atrial activation sites during retro-conducted impulses by fast and slow pathways. Right anterior oblique view. CS: coronary sinus. Taken from: Lockwood D, *et al.* Arrhythmias and Electrophysiology 2008. p. 615-46²⁰. Modified by the authors.

AVNRT), the retrograde atrial electrogram is after a ventricular activation ratio with a $AH / HA \leq 1$ relation, indicating that retrograde conduction is slower than the anterograde conduction. The AV interval, measured from the onset of ventricular activation of surface electrogram, to the earlier deflection of atrial activation in the bundle of His electrogram is > 60 ms and in the high right atrium > 100 ms. From electrocardiographic viewpoint this produces a tachycardia with a PR interval greater than the PR. In most cases of fast-slow tachycardia, the early atrial activation site is posterior to the AV node near the coronary sinus ostium. However, anterior and median forms of fast-slow AVNRT have been described^{23,25}.

Slow – Slow

In this form, the AH / HA ratio is greater than 1, but the VA interval is > 60 ms, which suggests that the two slow pathways are used, for both antegrade and retrograde activation. Usually, but not always, the earliest atrial activation takes place in the posterior septum (ostium of the coronary sinus). In the subsequent tachycardia AV conduction time can be prolonged, in a range from 78 to 168 ms. However, the atrium-His/His-atrium relationship remains greater than 1. This shows that cases reported as subsequent forms of slow-fast AVNRT currently represent the slow-slow type described by Baerman, according to Jackman et al.¹¹.

Fast – Fast

More recently, Wu *et al.*¹⁹ have shown the existence of AVNRT cases that do not conform to the previously described types. This tachycardia, called fast-fast, is characterized by a higher frequency, relatively short AH and HA intervals, a AH / HA ratio of around 1 and

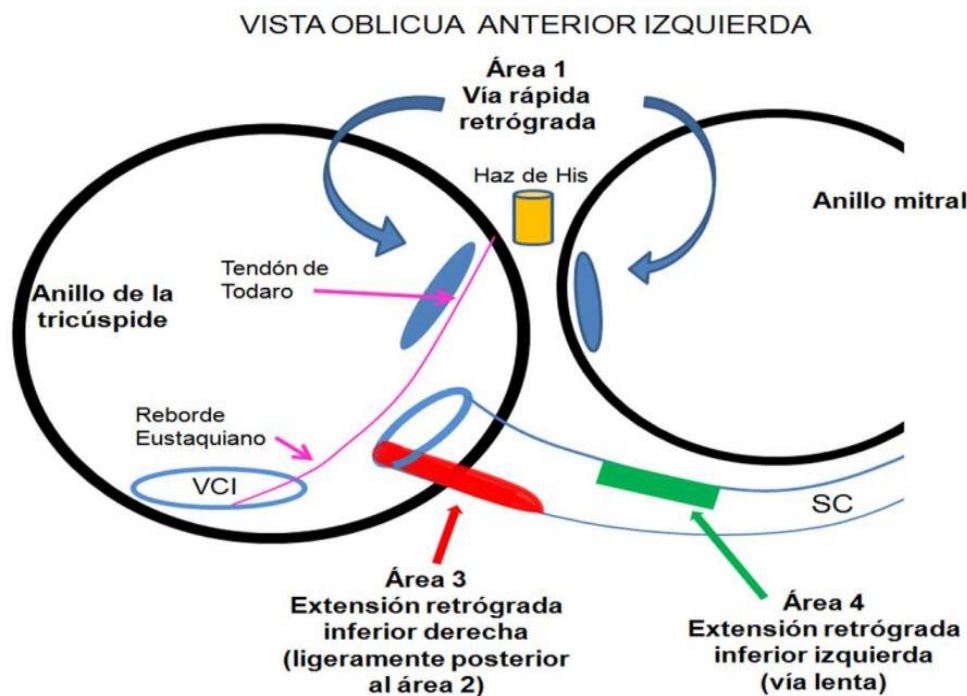


Figure 4. Schematic representation of the earliest atrial activation sites during retroconducted impulses by fast and slow pathways. Left anterior oblique view. IVC: inferior vena cava, CS: coronary sinus. Taken from: Lockwood D, *et al.* Arrhythmias and Electrophysiology 2008. p. 615-46²⁰. Modified by the authors.

an earlier atrial activation, next to ostium of the coronary sinus^{13,23,25}.

On the origin of the circuit

Some authors²⁶ suggest that the AVNRT is the result of functional properties of tissue anisotropy within the triangle of Koch, rather than two or more anatomical pathways, i.e. this arrhythmia is a result of a longitudinal functional dissociation within the AV node. However, more recent studies²⁰⁻²³ suggest that fast and slow pathways involved in the AVNRT reentry circuit represent the conduction on different atrio-nodal connections, instead of being a product of a longitudinal functional dissociation within the compact AV node. These observations include:

1. Differences in the site of earliest atrial activation during conduction through the slow or fast pathway, originally described by Sung *et al.*
2. Electrical treatment of tachycardia with atrial extra-stimuli applied outside the compact AV node at the bottom of the triangle of Koch or in the coronary sinus.
3. Extracellular recordings and optical mapping of AV

conduction in dogs and rabbits.

4. Selective removal of conduction of fast or slow pathway by ablation in the distant atrium of the compact AV node.

PHARMACOLOGICAL AND NON-PHARMACOLOGICAL TREATMENT

TREATMENT OF THE CRISIS

Vagal maneuvers

They are applied in the acute context of a tachycardia crisis because they produce vagal hypertonus in the AV node, which deteriorates the electrophysiological properties that sustain the circular movement of the impulse.

It is recommended as first choice in patients with AVNRT who are hemodynamically stable, and as an ambulatory measure that is explained to the patient. Such maneuvers are: compression of eyeballs that induce some degree of pain (many do not use it because of the danger of causing eye injury), wash the face with cold water, Valsalva maneuver (the most used) and Müller maneuver (similar to Valsalva but the reverse, i.e. inspiration with closed glottis). Also, a massage of the carotid sinus can be performed by a compression of 5-10 seconds, taking into account that the vagal innervation of the heart is lateralized, so the massage on the left carotid sinus exerts greater influence on the AV node, within which the substrate we are dealing with is located. The right sinus has a greater relation with the sinus node. However, tachycardia termination can be achieved from both sides because there are crossed fibers in a greater or lesser degree.

We have seen dramatic cases where, at the touch of eyeballs the arrhythmia terminates, but in the vast majority of cases that come to the emergency department or other scenarios the arrhythmias are not controlled with these measures so other strategies are needed.

Pharmacological treatment

It will be used if there is no hemodynamic deterioration or contraindications to the drug to be used. The most used options are:

- Adenosine: 6-12 mg intravenous bolus. Contraindications: asthma (bronchospasm) and coronary artery disease.
- Verapamil: the intravenous administration of 10 mg in 5 minutes has similar efficacy to adenosine. In

our experience we have successfully interrupted the tachycardia with only 2.5 mg, combined or not with Valsalva maneuver in a considerable number of patients. This reduces the undesirable effects depending on the dose employed. Contraindications: hypotension and poor ventricular function.

- Other drugs during the crisis: diltiazem, esmolol, metoprolol, digoxin and amiodarone.

Electrical cardioversion

Electrical cardioversion is the universal choice for any tachyarrhythmia with hemodynamic deterioration. Sedation with propofol or midazolam is induced. Almost all cases are effective at low loads, hence it is unusual to use doses higher of 100 J in biphasic mode. We believe its use is reasonable if the doctor begins to notice a pharmacologically refractory episode even if there are no signs of hemodynamic deterioration. This recommendation is based on the fact that we have seen the use of different pharmacological options in the same patient, and finally when cardioversion is chosen, there have been enormous pauses and asystole that disrupt the proper treatment of the case. These pauses favored by the indiscriminate use of parenteral antiarrhythmics.

MAINTENANCE TREATMENT

Depressors of AV nodal conduction may be used orally, such as: β -blockers, verapamil, diltiazem, and class IC drugs acting on rapidly conducting tissue (propafenone and flecainide). These class IC drugs seem to exert their effect on the fast pathway of the AV node or may act on the transition fibers. Particularly we have seen they are very effective after classically used drugs have failed. Although Digoxin could be an option, each time it is used less for this indication. Exceptionally, amiodarone may be used in cases refractory to the other proposals, in patients who refuse endocardial ablation or when this is not possible.

Vagal maneuvers without maintenance drugs

They are useful in patients with AVNRT highly-responsive to these maneuvers and behavior non-recurrent of tachycardia.

Pill in the pocket

It is formally indicated as an option in certain patients with atrial fibrillation, but not widely accepted in AVNRT. However, Almendral *et al.*¹⁰ mention it in their

recent review and acknowledge that, due to its delayed effect (1-3 hours), its use may be impractical because the effects of tachycardia could be unbearable if so much time should be waited to control it.

Radiofrequency Ablation

Ablation with this technique was first described in 1989, when Haissaguerre²⁷ published the results of ablation in 21 patients with AVNRT. In the same year, according to Civera *et al.*¹³, Sanjuán *et al.* published the results of radiofrequency ablation in four patients. Initially, the fast nodal pathway was targeted, but this often produced baseline PR interval prolongation and incidence of significant iatrogenic complete AV block. PR interval prolongation could be so exaggerated that some patients developed a pseudo-pacemaker syndrome.

In this regard, there have been multiple studies, which compare the slow pathway ablation or the fast one. The MERFS study (*Multicenter European Radiofrequency Survey*) retrospectively analyzed the incidence of complications related to the procedure in 4 463 patients who had received radiofrequency ablation in 69 European institutions between 1987 and 1992. In this study, it was evident that complete AV block was significantly more frequent in patients that have received ablation through the fast pathway²⁸.

According to Otomo²⁹ and Marine³⁰, in 1990, in a communication to the Congress of the NASPE (North American Society of Pacing and Electrophysiology), Roman *et al.* showed that radiofrequency ablation of the posterior atrial septum, near the ostium of the coronary sinus, was able to eliminate slow pathway conduction and inhibit the onset of tachycardia, without causing any degree of AV block. This aroused great interest and various techniques for this purpose were developed in different laboratories. At present, ablation targeting the slow AV nodal pathway is the method of choice for the definitive treatment of this tachycardia^{29,30}.

Since its inception there have been several studies that have exposed the results of this technique in multiple centers^{31,32}, all with success rates greater than 90%. Tolpinski *et al.*³³ in their study of 14 years of experience in the ablation of this tachycardia, reported a success rate greater than 97%, with a number of complications lower than 5%.

Data from the first Argentine Registry of Catheter

Ablation³⁴ reveal a 98.7% success in the first attempt of ablation in this substrate, after having carried out this therapy in 237 patients. Complications were minimal: a patient with complete AV block and another that showed a hematoma in the puncture site, which represented 0.8% of complications. In Spain, according to the X Official Report of the Electrophysiology and Arrhythmias Section of the Spanish Society of Arrhythmias and Pacemakers³⁵, AVNRT represented 27% (2,321 procedures) of all substrates treated in 2010, which were 8762. The success rate was 98.4%, although it is significant to note that in 34 centers success was achieved in 100% of the ablation procedures. 8 cases with complications were reported (0.34%), with complete AV block as the most frequent (3 cases, 0.13%) requiring permanent pacemaker implantation, and also three patients who had complications related to vascular access. Also, one case with stroke and another with pulmonary thromboembolism were reported.

This procedure is also performed in the pediatric³⁶⁻³⁸ and geriatric³⁹ populations, with very good results.

Recommendations of radiofrequency ablation in AVNRT⁴⁰⁻⁴⁴

Recently the Colombian Society of Cardiology published its Cardiovascular Electrophysiology Guides⁴¹⁻⁴² after evaluating all available information about it from the most recognized practice guidelines.

Class I

- The EPS with mapping and ablation is indicated in patients with symptomatic and sustained AVNRT, which is resistant to drug therapy or in patients who are intolerant of antiarrhythmic medication, do not wish long-term drug treatment or have a profession that is considered high risk (commercial pilots) (level of evidence B).

Class II

- The EPS with mapping and ablation is reasonable in patients with AVNRT identified in an EPS or in the ablation of another cardiac arrhythmia.
- The EPS with mapping and ablation is reasonable in patients with clinical suspicion of AVNRT in who, during an EPS, dual nodal pathway and atrial echoes are found, but without AVNRT.

The Colombian guidelines do not recognize situations leading to a class III indication in the context of this arrhythmia⁴². However, the ACC/AHA⁴³ does fore-

see such a situation which we set forth below:

Class III

- Patients with well tolerated AVNRT, which respond to antiarrhythmic drugs and that the patient prefers to ablation.
- The finding of dual nodal pathway physiology (with or without echo complexes) during the EPS of patients in whom no clinical AVNRT is suspected.

Cryoablation has also been used as a catheter technique. This option is considered safer than radiofrequency because although it induces AV block during applications in up to 6% of the cases, they tend to disappear when applications are stopped^{40,43}.

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