INDUCTION OF ATRIOVENTRICULAR NODE REENTRY
BY SIMULTANEOUS ANTEROGRADE CONDUCTION OVER
THE FAST AND SLOW PATHWAYS

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Abstract: Atrio-ventricular node reentry (AVNRT) is typically induced with an anterograde block over the fast pathway (FP) and conduction over the slow pathway (SP), with subsequent retrograde conduction over the FP. Rarely, a premature atrial complex (PAC) conducts simultaneously over the FP and SP to induce AVNRT. Previous publications have reported that conduction over the fast and slow pathway of the atrioventricular node can occur successively one after the other, thus leading to dual ventricular depolarization from what initially was a single atrial impulse. We report a case of an 18-year-old male patient referred for repeated bursts of ectopic activity. Evaluation of the patient’s electrocardiographic recordings suggested the presence of dual ventricular activations for each atrial beat. The electrophysiological study revealed that the patient had simultaneous conduction over the fast and slow pathways of the atroventricular node giving rise to a non-reentrant tachycardia, along with an absence of retrograde (ventriculoatrial) conduction, and a significant atrio-His bundle jump (A-H jump) through the slow pathway from the fast pathway during programmed electrical stimulation from the right atrium. Ablation of the slow pathway at the base of the Koch triangle yielded a cessation of the dual ventricular response, absence of the nonreentrant tachycardia and no A-H jump.

Key words: dual conduction, slow pathway, ablation of slow pathway, nonreentrant tachycardia.

Introduction

The classic concept of reentry is that an impulse blocks over one conduction pathway, proceeds slowly over an alternative pathway, and then
conducts retrogradely over the initial blocked pathway to complete the reentrant circuit (figure 1A). Simultaneous activation of a single atrial beat over the fast (FP) and subsequently slow (SP) AV nodal (AVN) pathway to initiate AVN reentry (figure 1B), was described in a case report by Wu et al. in 1975.

Following this initial publication, many authors have described this arrhythmia as a non-reentrant tachycardia that has often been mistaken for atrial fibrillation. The most often found clinical features of this tachycardia are that there is no ventriculoatrial (VA) conduction and patients rarely, if ever, have atrioventricular nodal reentry tachycardia. Patients are often treated for atrial fibrillation utilizing almost all the different kinds of antiarrhythmic drugs with limited or no effect.

Figure 1 – A. Classic initiation of slow/fast atrioventricular nodal reentrant tachycardia; B. Dual ventricular activations for each atrial beat because of simultaneous conduction over the fast and slow pathways

Слика 1 – А. Класична активација на споро/брза предкоморно-коморно јазолна кружна тахикардija; Б. Двојна коморна активација за секој предкоморен удар поради истовремено спроведување на импулсот низ брзот и спориот пат

Case Report

We present an 18-year-old male patients who had suffered from short burst of premature atrial contractions (PAC’s) and tachycardia for five years. The patient was symptomatic for fatigue and malaise after experiencing narrow QRS tachycardia with HR of 120 bpm. He was often misdiagnosed and pharmacologically ineffectively managed with various types of antiarrhythmic drugs. Non-invasive echocardiographic evaluation revealed a normal structured and functioning heart. On first glance, evaluation of the electrocardiographic (ECG) recordings revealed numerous bursts of premature multifocal atrial contractions. Subsequent careful evaluation suggested that there might be one atrial activity for two ventricular responses. This conclusion was presented to the patient, and an electrophysiologic study was suggested and subsequently performed.
Electrophysiological study

The patient was examined in a fasting state without having taken any medication the previous week (medication was discontinued for at least 3 half-lives). Written consent was obtained before the electrophysiologic study commenced. The right inguinal region and left cubital region skin was prepped, and anesthetized with minimal local anesthesia. Four quadripolar electrode catheters were positioned in the heart. One decapolar electrode catheter (6F Daig, with 5 mm electrode spacing) was introduced through the left cubital vein into the coronary sinus, and the remaining three quadripolar electrode catheters (6F Daig, with 5 mm electrode spacing) were introduced through separate punctures of the right femoral vein, and positioned in the apex of the right ventricle (RVA), over the His bundle (His), and on the free wall of the high right atrium (HRA) in close proximity to the sinoatrial node. Basic electrogram intervals showed normal AH and HV intervals. In order to minimize the risk of early induction of atrial fibrillation, the standard protocol for programmed electrical stimulation (PES) was commenced from the right ventricular apex (RVA). Both programmed and continuous pacing from this site revealed a ventriculoatrial (VA) block. This finding also ruled out the presence of atrioventricular reentry tachycardia (AVRT).

Antegrade programmed electrical stimulation (PES) from the high right atrium (HRA) revealing that a single atrial contraction frequently initiated a dual ventricular response. [Figure 3] The presence of a stable HV interval on the electrogram indicated that two ventricular depolarizations occurred from a single atrial impulse. The atrial impulse splits up into two impulses as it traver-
sed through the fast (FP) and slow pathways (SP). Normally the impulse in the SP is blocked as it reaches the refractory atrioventricular node from the recently traversed impulse from the FP. Instead the impulse was allowed to continue distally and initiate the second ventricular depolarization.

Further evaluation of the conduction system with programmed electrical stimulation revealed the presence of AH jump of 100 ms obtained with PES at S1S2 at 500/330 ms, [Figure 4].

Figure 3 – Dial conduction in the atrio ventriculare node
Слика 3 – Двојно спроведување низ предкоморно-коморниот јазол

Further evaluation of the conduction system with programmed electrical stimulation revealed the presence of AH jump of 100 ms obtained with PES at S1S2 at 500/330 ms, [Figure 4].

Figure 4 – Atrio-His bundle jump from the fast to the slow pathway in the atrio-ventricular node during the programmed electrical stimulation from the right atrium. The complex 1 and 2 impulses went through the fast pathway (AH under 125msec), the complex 3 impulse went through the slow pathway (AH 219 msec)

Слика 4 – Предкоморен -Хис (ПХ)скок од брз на спор пат во предкоморно-коморниот јазол за време на програмирана електрична стимулација од десната предкомора. Во првот и вториот комплекс импулсот поминал низ брзот пат (ПХ помал од 125 мсек), кај третиот комплекс импулсот поминал низ спориот пат (ПХ 219 мсек)
During the study a non-reentrant supraventricular tachycardia was easily induced at various pacing intervals with both the programmed and continuous electrical stimulation in the right atrium. This sustained nonreentrant tachycardia (tachycardia circle length = 492 ms) was terminated with antitachycardia pacing from the right atrium (ATP). [Figure 5]

**Figure 5 – Non-reentrant atrial tachycardia**

*_Слика 5 – Не-кружна предкоморна тахикардија*_

**Radiofrequency Catheter Ablation**

After concluding that the dual ventricular response was due to unblocked conduction of a “split” atrial impulse travelling through the fast and slow pathways, it was accepted that eliminating the SP would terminate this response. Therefore, radiofrequency catheter ablation was initiated in the postero-septal region of the right atrium on the base of the triangle of Koch. Ablation was directed by both anatomical and signal navigation. The ventricular diagnostic catheter was removed and replaced by the ablation catheter (7F Webster Cordis 5835). The ablation catheter was then positioned in the postero-septal region of the right atrium just above the ostium of the coronary sinus, with a pre-ablation electrogram signal very similar to that obtained during ablation for the slow pathway in patients with atrioventricular nodal reentry tachycardia (AVNRT). A pre-ablation atrial to ventricular signal ratio of less than 1:2 with an initial positive deflection of the atrial signal was considered acceptable. Ablation was carried out in a power-controlled mode at an energy level of 30W. We used the Osypka HAT 200S as our radiofrequency generator, with each ablation application timed for a maximum of 30 seconds. The appearance of a nodal rhythm was considered to be an indication of successful ablation. Ablation resulted in complete loss of dual conduction response (ventricular
depolarization), as well as loss of the A-H jump when later paced in HRA. (Figure 6).

**Discussion**

Since the initial description by Wu in 1975, there have been a limited number of papers describing this arrhythmia. The limited number of publications could arise from the common misinterpretation of this arrhythmia as atrial fibrillation. A number of these papers describe how they discovered this arrhythmia as a coincidental finding while intending to perform a different type of ablation technique.

Our patient also had a similar fate in that he was misdiagnosed and ineffectively treated for his arrhythmia before being referred to us.

The major electrophysiological feature that determines whether simultaneous conduction over the fast and slow pathways will occur is the difference in the conduction time between these two pathways, mainly the slow pathway. Normally the antegrade conduction occurs over both the fast and slow pathway. In order for dual conduction to occur, in some patients slow conduction has to slow down sufficiently to permit recovery of the distal common pathway of the atrioventricular node and the His-purkinje conduction system, thus allowing conduction of the impulse through to the ventricles.

As with ours, patients with this arrhythmia are often misdiagnosed for various atrial arrhythmia, mainly atrial fibrillation. The R-R interval is not always equal as the conduction velocities vary between the fast and slow pathways. Many of the non-invasive diagnostic procedures are insufficient in presenting the subtle findings (such as presence and absence of the P wave) that characterize this arrhythmia. In the absence of clear cut diagnosis most patients end up with antiarrhythmic drugs that we the doctor feel most comfortable with.
In most cases this would produce no effect, because most of these patients do not react favourably to anti-arrhythmia medication.

It has been speculated that the lack of retrograde (ventriculoatrial) conduction is the reason why these patients do not have atrioventricular nodal reentry tachycardia.

REFERENCES

Индукуција на предкоморно-коморна кружна тахикардija преку истовремено антеградно спроведување на импулсот преку спориот пат и брзиот пат

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Предкоморно-коморната кружна тахикардija се добива со антеграден (од предкомора кон комора) блок преку брзиот пат и спроведување на импулсот преку спориот пат, со последователно ретроградно спроведување преку брзиот пат. Ретко, предкоморната екстратисела се спроведува истовремено низ брзиот и спориот пат, со што започнува кружна предкоморно-коморна јазолна тахикардija.

Претходно објавените трудови покажуваат дека спроведувањето преку брзиот и спориот пат на предкоморно-коморниот јазол може да се изврши истовремено низ брзиот и спориот пат, што води кон двојна коморна деполаризация од еден предкоморен импулс. Нашиот случај е 18-годишен момче со повторувачки атаки од ектопични удачи. По евалуација на електрокардиографот беше поставен сомнеж за постојење на двојна коморна активност на еден предкоморен удар. Електрофизиолошката студија покажа дека пациентот има истовремено спроведување на предкоморниот импулс преку брзиот и спориот пат од предкоморно-коморниот јазол, што беше причина за не-кружна предкоморна тахикардija, покрај отсуството на ретроградното (коморно) спроведување и покрај постоињето на значаен предкоморно-Хис гранка скок од брзиот на спориот пат за време на програмираната предкоморна електрична стимулација. Аблацијата на спориот пат на базата на тријаголникот на Кош доведе до прекин на двојниот коморен одговор, отсуство на не-кружна предкоморна тахикардija и губиток на предкоморно-Хис гранка скок.

Клучни зборови: двојно спроведување, спор пат, аблација на спор пат, не-кружна предкоморна тахикардija.

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