

# Premature Atrial Contractions Managed With Slow Pathway Ablation

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## Abstract

### Key words:

Premature atrial contraction; slow pathway ablation; nonreentrant tachycardia; cardiac arrhythmia; cardiac electrophysiology.

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Received: 12-Oct-2008  
Accepted: 21-Nov-2008  
Online first: 25-Nov-2008

We present data from 18 year old male patient referred to our clinic for evaluation and treatment due to persistent premature atrial contractions (PAC). Up to the time of admission, the patient never experienced paroxysmal tachycardia, and his principal complaint was the irregular heart rhythm refractory to all types of antiarrhythmic drug therapy. Electrophysiological study was performed and it revealed that continuous pacing in the right ventricle produced a retrograde conduction block at rates 500 msec. Decremental programmed electrical stimulation in the right atrium ( $S_1S_2$  540/340 to 540/330 ms induced a sudden prolongation of A – H interval (AH jump of 98 ms) and a nonreentrant tachycardia (cycle length = 492 ms). Intracardiac electrogram showed that supraventricular activity emerged from simultaneous conduction over the slow and fast pathways of the atrioventricular node. This type of conduction is usually seen in patients with atrioventricular nodal reentry tachycardia, but our patient never previously complained of tachycardia, and the electrogram during tachycardia was not suggestive for atrioventricular nodal reentry tachycardia. Subsequent ablation of the slow pathway at the base of the Koch triangle yielded a cessation of the supraventricular premature activity, absence of the nonreentrant tachycardia and disappearance of the A-H jump.

## Introduction

Simultaneous antegrade conduction over the fast and slow atrioventricular nodal pathways was first described by Wu et al in 1975 (1, 2). Following this initial publication, many authors have described this arrhythmia as a nonreentrant tachycardia that has often been mistaken for atrial fibrillation (3-6). Common features of this type of tachycardia are that there is no ventriculoatrial conduction, and patients rarely, if ever, have atrioventricular nodal reentry tachycardia (1). Patients are often treated for atrial fibrillation utilizing almost all different kinds of antiarrhythmic drugs with limited or no effect.

## Case Report

We present an 18 year old male patient who has suffered from short burst of premature atrial contractions (PAC's) and tachycardia for five years. Patient was symptomatic for fatigue and malaise after experiencing narrow QRS tachycardia with heart rate (HR) of 120 bpm. He was unsatisfactorily managed with all 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 atrial contractions. Further evaluation suggested that there might be more

than one atrial activity. These conclusions were presented to the patient, and an electrophysiologic study was suggested and subsequently performed.

## EP Study

After obtaining an informed written consent, the patient was studied in fasting state without medication for one week. The right inguinal region and left cubital region skin was prepped with Betadine solution and were anesthetized with minimal local anesthesia (10 ml 2% Lidocaine). Four quadripolar electrode catheters were introduced in the heart utilizing the Seldinger technique. One decapolar electrode catheter (6F Daig, with 5 mm electrode spacing) was introduced through the left cubital vein into the coronary sinus, and 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), position of His (His), and in height right atrium (HRA).

Basic electrogram intervals showed normal AH and HV intervals of 64 & 52 ms, respectively (Fig. 1).

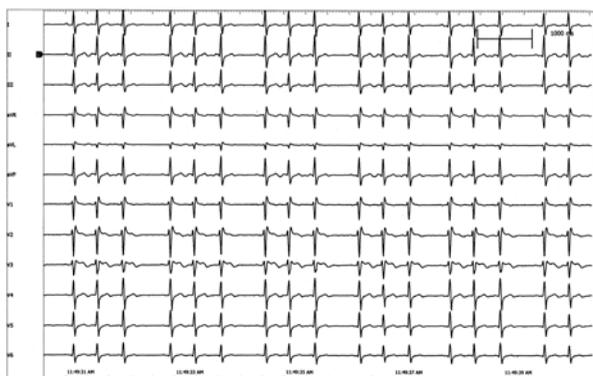


Figure 1: ECG at the start of EPS.

In order to minimize, and reduce the risk of early induction of atrial fibrillation, standard protocol for programmed electrical stimulation (PES) was commenced in the right ventricular apex (RVA). Both programmed and continuous pacing from this site revealed a ventriculoatrial (VA) block at a pacing rate of 500 msec (Fig. 2).

Antegrade programmed electrical stimulation (PES) from the high right atrium (HRA) revealing that a single atrial contraction initiated a dual ventricular response (Fig 3). The presence of stable HV interval on the intracardiac electrogram indicated that dual ventricular depolarization occurred from a single atrial

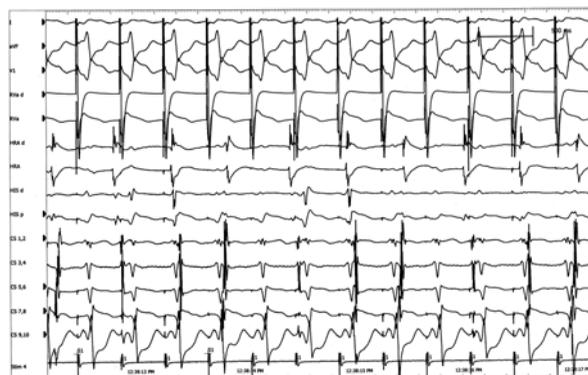


Figure 2: No V-A conduction at pacing rate of 500 msec.

impulse. The atrial impulse splits up into two impulses as it traverses through the fast (Fpw) and slow pathway (Spw). Normally the impulse in the Spw is blocked as



Figure 3: Dual Conduction (see text for description).

it reaches the refractory atrioventricular node from the recently traversed impulse from the Fpw. Instead the impulse is allowed to continue distally and initiate the second ventricular depolarization.

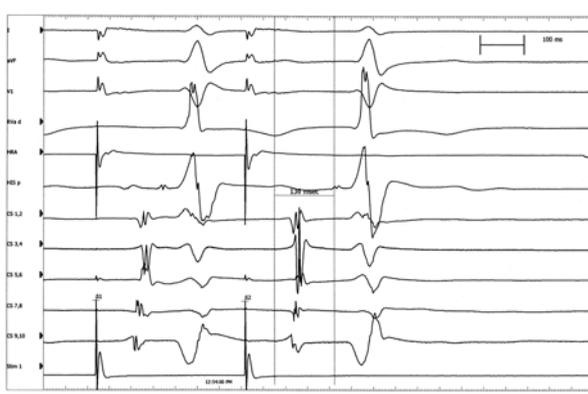


Figure 4: PES at S<sub>1</sub>S<sub>2</sub> 500/340 msec. AH interval is 136 msec.

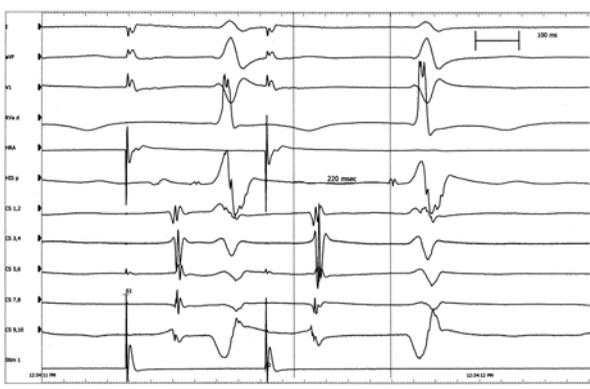


Figure 5: PES at  $S_1S_2$  500/330 msec. With a 10 msec decrement in pacing rate (from 500/340 to 500/330 msec) caused a sudden prolongation of the AH interval from previous 135 to 220 msec. At this point the impulse travels through the slow pathway.

Further evaluation of the conduction system with programmed electrical stimulation (PES) revealed an AH jump of > 100 ms obtained with programmed electrical stimulation at  $S_1S_2$  at 500/330 ms (Figures 4 & 5).

During the study a nonreentrant supraventricular tachycardia could be easily induced with programmed and continuous electrical stimulation from the right atrium (Fig. 6). This sustained nonreentrant tachycardia (TCL: 492 ms) was terminated with antitachycardia pacing.



Figure 6: Non-reentrant tachycardia.

## Radiofrequency Catheter Ablation

After concluding that the dual ventricular response was due to unblocked conduction of a split atrial impulse traveling through the fast and slow pathways, it was postulated that by eliminating the Spw we could eliminate this arrhythmia. Therefore, radiofrequency catheter ablation was applied in the

right atrial posteroseptal region 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 posteroseptal region of the right atrium just above the ostium of the Coronary Sinus (Fig. 7).

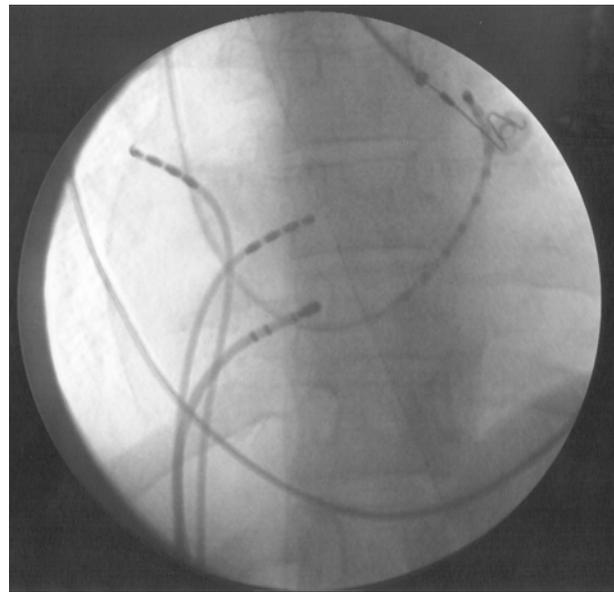


Figure 7: Fluoroscopy view of the ablation site.

Pre-ablation Atrial to Ventricular signal ratio of <1:2 with an initial positive Atrial signal deflection was considered acceptable (Fig. 8).



Figure 8: Pre-ablation signal.

Ablation was carried out in a power controlled mode at an energy level of 30W. We used Osypka HAT 200S as our radiofrequency generator. Each ablation application was timed for a maximum of 30 seconds. Total time of 396 seconds in 14 applications was

delivered. Ablation resulted in complete cessation of all premature atrial activity, as well as loss of A-H jump.

## Discussion

Since the initial description by Wu in 1975, there have been a limited number of papers describing this arrhythmia (1-7, 13). Limited number of publications could arise from the common misinterpretation of this arrhythmia as atrial fibrillation. A number of these papers describe how this arrhythmia was discovered as a coincidental finding while intending to perform other forms of ablation procedure.

This paper describes another case of non-reentrant supraventricular tachycardia with to 1:2 AV conduction which was cured by slow pathway ablation (Fig. 9).

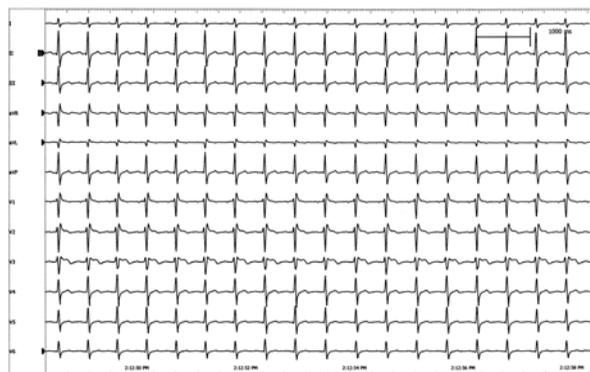


Figure 9: ECG at the end of the study.

Our patient also had a similar fate in that he was misdiagnosed and treated for other arrhythmia before being referred to us. The major electrophysiological feature that determines whether simultaneous conduction over both pathways occurs is the difference in the conduction time between the fast and slow pathways, mainly the slow pathway.

Normally the antegrade conduction occurs over both the fast and slow pathway. In order for dual conduction to occur, slow conduction has to decelerate sufficiently to permit recovery in the distal common pathway of the Atrioventricular Node and the His-Purkinje conduction system, thus allowing conduction of the impulse through to the ventricles.

Patients with this arrhythmia are often misdiagnosed for various atrial arrhythmias, mainly atrial fibrillation. The R-R interval is not always equal as the conduction velocities vary between the fast and slow pathway. Many of the non-invasive diagnostic

procedures are insufficient in presenting the subtle findings that characterize this arrhythmia (presence and absence of the P wave). In the absence of clear cut diagnosis most patients end up being managed only by pharmacological means, and in most cases this produces no clinical effect.

It has been speculated that the lack of retrograde (ventriculoatrial) conduction is the reason why in patients atrioventricular nodal reentry tachycardia is not present. The blocked retrograde conduction simply does not allow for the reentry mechanism to initiate.

It has been published that dual ventricular response can be detected in the presence of other types of arrhythmia such as in the presence of accessory pathways. In our patient we had a consistent HV interval thus indicating that the impulse traveled through the slow and fast pathway (14).

While this mechanism is likely in this case, other possibilities must be considered. From the surface ECG, alternating His bundle ectopic with ventricular activation cannot be ruled out. This uncommon type of tachycardia has to be considered in the differential diagnosis of paroxysmal supraventricular tachycardia and irregular supraventricular rhythms mimicking atrial fibrillation.

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