DISTAL COMMON PATHWAY IN ATRIOVENTRICULAR NODE REENTRANT TACHYCARDIA

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ABSTRACT - Anatomical boundary of atrioventricular node reentrant tachycardia (AVNRT) is composed of fast and slow pathways, right atrium in upper turnaround and common distal pathways in lower turnaround. We performed electrophysiologic study (EPS) in 152 patients and could show the existence of distal common pathway with decremental conduction properties in approximately 49 patients.


Key Words: AVNRT, distal common pathway, parasympathetic supraventricular tachycardia (PSVT), EPS

INTRODUCTION

Atrioventricular node reentrant tachycardia (AVNRT) is probably the most common form of parasympathetic supraventricular tachycardia (PSVT). Electrophysiologic study (EPS) and programmed electrical stimulation of the heart have had a crucial role in increasing the understanding of the nature of this intriguing cardiac arrhythmia (1,2). During the past three decades, knowledge regarding the electrophysiologic properties of the AVNRT circuit, pharmacologic and nonpharmacologic therapies of the arrhythmia have had a surprising progress in all fields (3,4,5). Also, the anatomical construction of AVNRT and the autonomic nervous system effect on induction and termination of this arrhythmia have been elucidated more (1,6).

In this report, we focus on the electrophysiologic characteristic of this arrhythmia, especially distal common pathway. The reentrant nature of AVNRT is generally accepted and is supported by several electrophysiologic characteristics:

1. The initiation and termination of AVNRT usually by rapid atrial pacing or introduction of premature atrial impulses at critical coupling intervals.

2. The presence of dual pathway physiology demonstrating by jumping phenomenon during atrial pacing.

3. The demonstration of entrainment and resetting of AVNRT during rapid atrial pacing and premature stimulation respectively.

MATERIALS AND METHODS

We performed EPS and radiofrequency catheter ablation in 152 patients at Dr. Shariati Hospital since June 1995 till October 2000. Three quadripolar catheters were located in high right atrium (HRA), His bundle (HB) and right ventricular apex (RVA) in all of the patients, and one decapolar catheter was inserted in coronary sinus (CS) in most of the patients. By atrial decremental pacing, short burst pacing or premature atrial electrical stimulation, we could induce AVNRT in all of them.

RESULTS

During AVNRT, atrial pacing was performed with pacing cycle length 10-20 msec lower than AVNRT cycle length to show entrainment. Also premature ventricular pacing introduced during His bundle refractoriness with no change in AVNRT cycle length or showing premature His deflexion. The above findings prove that AVNRT circuit is confined to the atrium. During atrial pacing, we accepted at least 50 msec increment in all interval as jumping phenomenon, which is due to changing the antegrade conduction pathway from fast to slow pathway. This finding is in accordance with dual physiology of the reentrant tachycardia. Also we could prove that the site of AV block during AVNRT is in the supraventricular region. After atrial pacing with cycle length less than the arrhythmia, AVNRT continued but with 2:1 or Wenckebach periodicity AV conduction. Despite the continuation of AVNRT, Wenckebach periodicity or 2:1 AV block were considered in approximately 40 patients (Fig. 1 and 2).

DISCUSSION

Although the upper boundary of AVNRT circuit is controversial yet (2,3,7), but the lower anatomic boundary of the AVNRT circuit is less controversial (2). It is generally accepted that the structures below the His bundle are not actively involved in the reentrant process. However, participation of proximal His bundle in reentry is still a subject of controversy (2,4).
Fig. 2. 2:1 AV block during AVNRT

Several observations support the concept of a lower intranodal reentrant structure that is separated from the His bundle by a common AV nodal pathway (2,7).

1. The HA interval during ventricular pacing is longer than that during AVNRT, which points toward further conduction delay in a structure below the AVNRT circuit and above the His bundle.

2. Persistence of AVNRT despite a 2:1 or Wenkebach block above the most proximal His bundle recording site.

3. Occurrence of retrograde activation via the fast pathway simultaneously with or prior to His bundle activation (during AVNRT).

4. Preexcitation of His bundle by a premature ventricular beat without change in AVNRT cycle length.

REFERENCES


