Electrophysiologic properties of para-Hisian atrial tachycardia

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BACKGROUND Focal atrial tachycardia (AT) originates from preferential sites, including the tricuspid and mitral annuli. AT arising from the atrioventricular annuli is initiated and terminated with programmed stimulation and is, in general, adenosine and verapamil sensitive. Para-Hisian AT arising from the apex of the triangle of Koch has been considered to be a distinct entity, characterized by unique electrophysiological properties.

OBJECTIVE We sought to more fully delineate the electrophysiological and electrocardiographic properties of para-Hisian AT in a large series of patients.

METHODS The study population consisted of 38 patients (63 ± 15 years; 23 female) with AT from the para-Hisian region. The ATs were focal and originated from the anteroseptal tricuspid annulus, in close proximity to the His bundle recording. Proximity to the His bundle was confirmed by electrogram recordings, fluoroscopy, and centrifugal activation during three-dimensional mapping.

RESULTS The mean AT cycle length was 421 ± 69 ms. AT was associated with a distinct P-wave morphology that was significantly narrower than the P wave during sinus rhythm. Adenosine (5.0 ± 1.5 mg) terminated AT in 34/35 patients. Intravenous verapamil terminated AT in three of three patients. Catheter ablation was attempted in 30 patients and was successful in 26 (87%).

CONCLUSION The para-Hisian region is a source of focal AT, with properties consistent with AT arising circumferentially along the tricuspid and mitral annuli, and should be considered a subset of this broader group of "annular" ATs. The electropharmacologic findings in para-Hisian AT are mechanistically consistent with cyclic AMP-mediated triggered activity.

KEYWORDS: Ablation; Adenosine; Arrhythmia; Atrial tachycardia; Mapping

ABBREVIATIONS **AMP** = adenosine monophosphate; **APC** = atrial premature complex; **AT** = atrial tachycardia; **AV** = atrioventricular; **AVNRT** = atrioventricular nodal reentrant tachycardia; **ECG** = electrocardiographic; **LV** = left ventricular; **NCC** = noncoronary cusp; **PPI** = postpacing interval; **RF** = radiofrequency; **TCL** = tachycardia cycle length

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Introduction

Recently, a classification system categorizing atrial tachycardias (ATs) as being either "focal" or "macroreentrant" has been proposed.[1] The rationale of this classification is based on electrophysiological mechanism, as defined by entrainment and three-dimensional mapping, and has important implications regarding the strategy for ablation.

The underlying mechanism of focal AT can be difficult to ascertain clinically but can be inferred from the mode of initiation and termination, response to entrainment, and specific pharmacological sensitivity. Focal ATs arise from preferential sites in the atria and most commonly originate from the crista terminalis, atrioventricular (AV) annuli, pulmonary vein ostia, and coronary sinus ostium/musculature.[2–8] Less frequently, the atrial appendages and superior vena cava are identified as sites of AT origin.[9,10] Three-dimensional mapping systems may help in determining a reentrant mechanism by registration of electrical activity that spans 90%–100% of the tachycardia cycle length (TCL; head meets tail). In contrast, a focal origin of AT shows a centrifugal pattern of activation. The apex of the triangle of Koch, that is, the para-Hisian region, has been reported to be another distinct site of origin of AT, although its underlying arrhythmia mechanism has yet to be identified.[11,12] The boundaries of the triangle of Koch include the tendon of Todaro, the septal leaflet of the tricuspid annulus, and the His bundle. It is unclear whether tachycardias from the apex of this region (i.e., near the His bundle) should be considered a separate entity given its signature origin or as part of the broader category of tachycardias arising from the AV annuli, which share identical electrophysiological properties. To

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clarify this distinction, we sought to fully characterize the pharmacological and electrophysiological properties of para-Hisian ATs.

We have previously proposed that the effects of adenosine on AT can differentiate between focal and macroreentrant ATs.[3,13] That is, in general, adenosine has no effect on macroreentrant circuits but can terminate or suppress focal ATs, depending on their underlying mechanism. In addition to its tissue-specific effects on supraventicular tissue mediated by $I_{KACh,Ado}$, adenosine also has antiadrenergic effects, decreasing intracellular cyclic adenosine monophosphate (AMP).[14] This results in inhibition of the L-type calcium current ($I_{Ca(L)}$) as well as the transient inward current (I_{Ti}).[15] These effects are consistent with adenosine-mediated termination of tachycardia due to cyclic AMP-dependent triggered activity.

Methods

Patient characteristics

Thirty-eight consecutive patients (62 ± 15 years; 23 females) who presented for invasive electrophysiological evaluation and catheter ablation of AT arising from the para-Hisian region comprise this series. This study was approved by the participating institutional review boards.

Noninvasive evaluation

Patients underwent evaluation of cardiac structure, function, and ectopy burden. When possible, this included 24-hour Holter monitoring and/or inpatient telemetry. Presence of coronary artery disease was assessed as clinically indicated by stress testing and/or cardiac catheterization. Left ventricular (LV) systolic function was quantified by echocardiography, radionuclide ventriculography, and/or ventricular cineangiography. Structural heart disease was defined as the presence of coronary artery disease, an LV ejection fraction ≤ 45%, and/or moderate/severe valvular disease.

Baseline electrophysiological study

After giving informed written consent, patients underwent electrophysiological testing after an overnight fast. Patients were locally anesthetized (with 0.25% bupivacaine and/or lidocaine 1%) and sedated with intravenous midazolam and morphine/fentanyl. Quadripolar 6-Fr catheters were advanced to the His bundle position and right ventricular apex. Right atrial electrogram recordings were obtained with either a quadripolar catheter positioned in the high right atrium (RA) or a 7-Fr duodecapolar halo catheter positioned along the tricuspid annulus. A 6-Fr decapolar catheter was positioned in the coronary sinus to record left atrial activity along the mitral annulus. Bipolar intracardiac electrograms were filtered at 30–500 Hz and recorded on optical disk. If mapping and/or ablation of the sinuses of Valsalva were required, access was obtained via transseptal atrial puncture or retrogradely via the aorta.

The stimulation protocol included rapid atrial and ventricular pacing and introduction of atrial and ventricular extrastimuli at several basic drive cycle lengths. Stimuli were delivered as rectangular pulses of 2-ms duration at 4 times diastolic threshold. To facilitate induction of sustained AT, when necessary, programmed stimulation was repeated after isoproterenol or dobutamine was infused to decrease the sinus cycle length by approximately 30%.

Twenty patients underwent electroanatomic mapping using the Biosense CARTO system (Biosense-Webster, Diamond Bar, CA), with a reference locator pad (on the patient's back) for spatial reference and a bipolar intracardiac electrogram (from the coronary sinus) used as temporal reference. A 7-Fr deflectable, 4-mm-tip quadripolar catheter (Biosense-Webster) was used for activation mapping and ablation in these patients. The remainder of the patients underwent activation mapping with either an alternative system (Real Time Position Mapping [RPM] System, EP Technologies, Boston Scientific, Natick, MA; or Ensite NavX, St. Jude Medical, St. Paul, MN) or solely using fluoroscopy.

P-wave analysis

Surface 12-lead P-wave morphology was assessed as described in detail elsewhere.[16] P waves were described as (1) positive (+), (2) negative (−), (3) biphasic (+/− or −/+) deflections from baseline, and (4) isoelectric (arbitrarily defined when there was no P-wave deflection from baseline of >0.05 mV). The duration of the P wave was measured during tachycardia and sinus rhythm.

AT diagnosis

AT was distinguished from other supraventricular tachycardias, including AV nodal reentry and AV reciprocating tachycardia, by standard electrophysiological criteria, as described in detail elsewhere.[3] Criteria included (1) intracardiac atrial activation sequence during tachycardia different from that during sinus rhythm, (2) change in the A-A interval during tachycardia preceding any change in the V-V interval, (3) presence of AV conduction block or delay without affecting TCL, (4) dissociation of ventricular activity from the tachycardia, and/or (5) tachycardia initiation independent of a critical prolongation of the AH interval. In 16 patients, attempts to demonstrate manifest entrainment during tachycardia were made by pacing from the high right atrium and coronary sinus at progressively shorter cycle lengths, beginning 10 ms less than the TCL.

Definitions

Focal AT was defined based on the following characteristics: (1) centrifugal atrial activation pattern, (2) dissociation of nearly the entire atria from the tachycardia with atrial extrastimuli, (3) early local atrial activation relative to the surface P wave, and/or (4) atrial activation map encompassing <50% of TCL. Annular focal AT was identified when the above criteria were met, fluoroscopic and three-dimensional electroanatomic sites were consistent with an annular site, and an atrial and ventricular electrogram were present simultaneously at the site of successful ablation. A location was considered "para-Hisian" when either a His deflection was observed at the site of earliest atrial activation during tachycardia or the successful ablation site along the tricuspid annulus was within 1 cm of a site recording the His bundle potential.