Catheter ablation for atrioventricular nodal reentrant tachycardia in patients with congenital heart disease

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BACKGROUND Variability in atrioventricular (AV) node location in congenital heart disease (CHD) can make catheter ablation for atrioventricular nodal reentrant tachycardia (AVNRT) challenging.

OBJECTIVE The purpose of this study was to describe institutional technique and outcomes for slow pathway modification in a cohort with CHD.

METHODS The study consisted of a retrospective review of CHD patients who underwent study from 2001 to 2013 with a diagnosis of AVNRT. Outcomes for slow pathway modification were recorded. In cases in which ablation was deferred, the reasons for this choice were examined.

RESULTS Forty-nine patients (median age 19 years) were included. CHD anatomy involved D-transposition of the great arteries (n = 6), "congenitally corrected" transposition of the great arteries (n = 4), Ebstein anomaly (n = 4), tetralogy of Fallot (n = 5), venous anomalies (n = 8), single ventricle (n = 16), and miscellaneous (n = 6). Ablation was attempted in 39 patients, using radiofrequency energy in 24, cryoablation in 8, and both in 7. Acute success rate was 92% (36/39). One patient had first-degree block in response to cryoablation, but no other complications occurred. At median follow-up 32 months, 1 patient had AVNRT recurrence. Most of the 10 patients in whom ablation was deferred had single-ventricle anatomy with uncertain AV node location.

CONCLUSION Ablation for AVNRT in CHD can be accomplished successfully with attention to underlying anatomy and prior surgery. Patients with single ventricle are a difficult subgroup, and a pharmacologic approach may be indicated in some cases if node localization is ambiguous.

KEYWORDS Supraventricular tachycardia; Congenital heart disease; Catheter ablation; Atrioventricular nodal reentrant tachycardia; Conduction system anatomy

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Introduction

Catheter ablation is effective therapy for atrioventricular nodal reentrant tachycardia (AVNRT) in patients of all ages.[1,2] Because the atrioventricular (AV) node and its extensions are located at predictable sites in a structurally normal heart,[3,4] orientation of the catheter tip relative to the landmarks of the triangle of Koch, combined with certain electrogram characteristics,[5] provides reliable guidance for successful slow pathway modification in nearly all cases.

Patients with congenital heart disease (CHD) have less predictable AV node locations.[6,7] In patients with AV canal defects, for instance, the node develops outside of the triangle of Koch in a region inferior to the mouth of the coronary sinus,[8] whereas in those with L-looped congenitally corrected transposition of the great arteries the node is usually displaced to a superior location medial to the right atrial appendage.[9] The AV node may also be left-sided in some patients with dextrocardia or heterotaxy syndrome and on rare occasions may be duplicated in CHD patients with "twin AV node" physiology.[10] Even if the node is not actually displaced, the landmarks for the triangle of Koch will be distorted in conditions such as tricuspid atresia, Ebstein malformation, or abnormalities of the coronary sinus.[11] Furthermore, surgical patching can complicate catheter positioning near nodal extensions after the Mustard or Senning operation for D-looped transposition of the great arteries or after the Fontan operation for single ventricle. The aim of this study was to review our institutional experience with catheter ablation for AVNRT in patients with various forms of CHD and to offer some insight into the location of slow pathway inputs in this abnormal anatomy.

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Methods

Study population and data collection

A single-center retrospective review was performed with approval of our hospital's Committee for Clinical Investigation. Patients with CHD who underwent electrophysiologic

study with a diagnosis of AVNRT between January 2001 and January 2013 were reviewed. Cases involving minor defects (e.g., small atrial septal defects, mild valve disease) were excluded. Study patients were chosen on an intention-to-treat basis that allowed inclusion even if ablation was deferred for clinical or anatomic reasons after diagnostic testing. Baseline data and technical procedure details were examined, along with acute success rate and associated complications. In cases in which ablation was deferred, the reasons for this choice were investigated. Follow-up status was ascertained by review of hospital records and correspondence from referring physicians.

Electrophysiologic study and ablation

Procedures were performed under general anesthesia (isoflurane) with antiarrhythmic drugs discontinued $\geq$48 hours. Catheter positioning was individualized according to anatomy and vascular access limitations. Procedures varied from a 2-catheter study (reference and map/ablate) to a 4-catheter study (coronary sinus, His-bundle region, ventricular apex, and map/ablate). Three-dimensional electroanatomic mapping was used whenever patient size and anatomy allowed.

The diagnosis of AVNRT was made according to established criteria.[1,5] Slow pathway modification was attempted at the discretion of the operator using radiofrequency energy (RF), cryothermal energy (CRYO), or a combination of both. There was an institutional preference for RF in these cases; CRYO was only used when AV node location was ambiguous or when ablation was attempted closer to the presumptive compact node after failure of RF at more remote sites. Catheters for RF delivery involved a nonirrigated 4-mm tip, and for CRYO a 4- or 6-mm tip. Settings for RF energy involved a maximum of 50 W and 70°C. Energy delivery was interrupted at $\leq$10 seconds (test RF) if a desired effect (e.g., mild junctional acceleration) was not observed or if undesired effects (e.g., rapid junctional acceleration, AH interval prolongation, nonconducted beat) were seen. Energy delivery was continued for 30–60 seconds (full RF) at promising target sites. CRYO involved 240-second freeze/30-second thaw/240-second refreeze at –70° to –80°C (full CRYO). CRYO application was stopped early (test CRYO) if extrastimulus testing after $\geq$20 seconds at $\leq$–30°C showed persistent slow pathway, or if AH prolongation or nonconducted beat was detected.

Target zones were chosen based on multiple criteria: (1) best estimation of AV node location according to underlying anatomy; (2) relative position of the His-bundle signal; (3) relative position of the coronary sinus ostium; (4) proper ratio of atrial:ventricular electrogram amplitudes; (5) identification of high-frequency components on the atrial portion of the electrogram that might indicate a slow pathway potential; (6) in fortuitous cases, mapping of atrial activation during retrograde slow pathway conduction;(7) observation of mild-to-moderately accelerated junctional rhythm without disturbance in fast pathway integrity during RF applications; or (8) selective block of slow pathway during CRYO. Acute ablation success was defined as elimination of all slow pathway conduction or effective attenuation of the slow pathway with a maximum of 1 AV nodal echo beat in response to stimulation off and on isoproterenol over 30 minutes of testing. In cases in which hemodynamic catheterization was combined into the same study, procedure duration is reported only for the electrophysiology portion of the case.

Statistical analysis

This was an observational study on a small and highly selected cohort. Numerical data are presented as median with range.

Results

Patient characteristics

Forty-nine patients met eligibility criteria. Median age was 19 years (range 2–53 years). Thirty-one patients (64%) were female, and median weight was 58 kg (range 13–120 kg). The primary indication for testing was a documented sustained tachycardia (various mechanisms) in 42 cases. The remaining 7 were studied based on concerning symptoms, which included recurrent rapid palpitations (n = 3), palpitations with presyncope (n = 2), or palpitations with syncope (n = 2). Thirty-two patients (65%) had undergone $\geq$1 trials of antiarrhythmic drug therapy before study. Secondary indications for invasive study included the need to assess hemodynamic status (n = 28).

Electrophysiologic study

Anatomic landmarks were registered by biplane fluoroscopy, augmented in selected patients by intracardiac echocardiography (n = 8) and/or angiography (n = 33). Three-dimensional electroanatomic mapping was used during 37 procedures (CARTO in 34, NavX in 3).

In 16 of 49 patients (32%), AVNRT was not the presenting tachycardia but was only uncovered during testing for a different arrhythmia. In these cases, the presenting mechanisms were intraatrial reentry in 8, accessory pathway reentry in 5, focal atrial tachycardia in 1, and ventricular tachycardia in 2. When AVNRT was identified incidentally, the operator made an informed determination whether slow pathway modification was warranted to best manage rhythm status.

AVNRT was typical (slow–fast) in 31 patients (63%), atypical (fast–slow or slow–slow) in 14 (28%), and 4 (8%) had both forms induced. No specific AVNRT pattern could be linked to a particular subtype of CHD. Median AVNRT cycle length was 335 ms (range 250–532 ms). Dual AV node physiology (A_2H_2 interval jump $\geq$50 ms with atrial S_2) was demonstrated in only 38% of cases during baseline testing. More often, S_3 atrial stimulation on isoproterenol, rapid atrial pacing, or ventricular stimulation was required to engage the slow pathway and induce AVNRT.