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Atypical atrioventricular node characteristics in a patient after surgical ablation of atrioventricular nodal reentry tachycardia

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Abstract

This clinical case report describes a patient who previously underwent cardiopulmonary bypass and peri-nodal modification of the atrioventricular node for the management of atrioventricular nodal reentry tachycardia and a mitral valve repair. Twenty years after the initial atrioventricular nodal reentry tachycardia operation, atrial fibrillation and flutter developed. Radiofrequency ablation of the cavo-tricuspid isthmus was performed for atrial flutter. We decided to block all conduction through the atrioventricular node and implant a permanent pacemaker. However, at the time of the planned ablative procedure, a His bundle electrogram could not be identified anywhere except in the paraseptal area. Nevertheless, successful 3rd-degree heart block was accomplished. Interventional specialists should be aware that the electrophysiological characteristics of the AV node late after peri-nodal modification may be unusual and difficult to discern and treat by standard techniques.

Keywords

cardiopulmonary bypass; valve repair; atrial fibrillation

Introduction

Atrioventricular Nodal Reentry Tachycardia (AVNRT) is the most common type of paroxysmal supraventricular tachycardia, comprising about 50% of all cases. The arrhythmia is most common in women under the age of 40 years, with the average age of 28 years [1]. Moe and associates [2] were the first authors who described that AVNRT is based on longitudinal dissociation of conduction through the atrioventricular (AV) node because of the presence of two pathways of conduction. These authors also demonstrated that atrial extra-stimuli could induce and terminate AVNRT in rabbits. Moe's experiments showed that the α-pathway had slower conduction and a shorter refractory period, while the β-pathway had higher conduction velocity and a longer refractory period [2]. Surgical perinodal modification of the AV node using Cardiopulmonary bypass (CPB) was first accomplished clinically by Cox et al in 1982 for the treatment of

AVNRT [3-5].

Subsequently, catheter ablation replaced surgery and has been highly effective for the treatment of AVNRT. These direct approaches to curing AVNRT without creating complete heart block may unexpectedly result in changes in the electrophysiological properties of the AV node, making the subsequent creation of 3rd-degree heart block by catheter ablation more complicated.

Case Presentation

A 64 year-old female was admitted with complaints of weakness, dizziness, increased blood pressure up to 180/100 mmHg and paroxysms of an irregular tachycardia. The patient first became symptomatic in 1988 when a regular tachycardia was recorded electrographically. AVNRT was diagnosed and patient received appropriate drug therapy (trials of beta-blockers and calcium channel blockers in maximally tolerated doses). However, the prescribed therapy was ineffective and paroxysmal tachycardia recurred. Five years later a surgical perinodal modification of the AV node was performed concomitantly with a mitral valve repair for moderate to severe mitral insufficiency.

Perinodal modification of the AVN

Surgical modification of the AV node in patients with AVNRT using multiple discrete cryolesions was subsequently widely adopted. AV node modification was performed on the beating heart during normothermic CPB. Initial AV node antegrade and retrograde conduction was determined, epicardial mapping of the AV sulcus was performed during tachycardia or ventricular pacing. After opening the right atrium, multichannel endocardial AV mapping was performed to determine the area of early retrograde breakthrough. Using the 3 mm cryoprobe, row of serial individual cryolesions was placed from the top of the coronary sinus orifice laterally and continued medially along the tendon of Todaro to the membranous inter-atrial septum. A second row of cryolesions was placed along the tricuspid valve annulus from the level of the coronary sinus to the membranous septum. Additional cryolesions were then placed medial to the coronary sinus orifice across the base of the triangle of Koch. Cryodestruction was performed under continuous monitoring of the PR interval on a storage oscilloscope synchronized to trigger its sweep with each paced atrial beat allowing continuous, real-time monitoring of the AV interval. When no further cryolesions could be placed inside the triangle of Koch without inducing transient heart block, the procedure was terminated. EP testing of the surgical effectiveness included evaluation of antegrade and retrograde conduction through the AV node to document the absence of dual AV nodal pathways and the inability to induce AVNRT [6].

Follow-up

The perioperative period was uneventful and paroxysmal tachycardia did not recur on long-term follow-up. Twenty years later, however, the patient developed symptomatic paroxysmal atrial fibrillation and atrial flutter. Antiarrhythmic therapy (trials of IC and III groups of antiarrhythmic drugs) yielded varying degrees of success and as a result, an Electrophysiological (EP) study was performed 20 years after

perinodal modification and showed there was no evidence of dual AV node pathways. Antegrade Wenckebach point was 420 ms. Antegrade effective refractory period of the AV node was 300 ms with no A-H jump or echo-beats. The effective refractory period of the left atrial was 240 ms, the retrograde Wenckebach point was 600 ms, the retrograde effective refractory period of the AV node was 480 ms, and the effective refractory period of the right ventricle was 220 ms. Maximal corrected sinus node recovery time was 650 ms. During coronary sinus pacing, atrial flutter with a cycle length 240 ms was induced. Radiofrequency ablation of the cavo-tricuspid isthmus was performed. Afterwards, atrial flutter could not be induced using overdrive pacing or programmed electrical stimulation. However, overdrive pacing of the coronary sinus induced atrial fibrillation. Transseptal puncture for the potential treatment of the atrial fibrillation was impossible because of the extremely rigid atrial septum caused by the previous surgery. Postoperatively, antiarrhythmic therapy (trials of IC group, III group of antiarrhythmic drugs) remained ineffective with frequent paroxysms of atrial fibrillation that were poorly tolerated. A dual-chamber BIOTRONIK Evia DR-T (Biotronik Group, Berlin, Germany) permanent pacemaker was implanted to pace and subsequently perform AVN modification.

Several days after pacemaker implantation, the patient was transferred to the X-ray operating room with a pacemaker rhythm in DDD mode with base rate of 70 beats per minute. The pacemaker was switched to VVI mode at 40 beats per minute. An open irrigated ablation catheter BW Celsius Thermocool (Biosense Webster Inc, Diamond Bar, CA, USA) was positioned in the mid-septal area. However, no His bundle spike could be identified despite carefully conducted mapping. Radiofrequency ablations in this area had no effect and neither slow nor accelerated nodal rhythm developed. The left femoral artery was then punctured, and Medtronic Marinr multi-curve catheter (Medtronic, Minneapolis, MN, USA) was introduced into the left ventricular cavity (Figure 1). Again, no His bundle spike could be found (Figure 2). A test radiofrequency ablation (power 30 W, temperature 45-50 ° C, impedance 95-110 ohms) was applied to the mid-septal area for 40 seconds with the development of a rapid nodal rhythm, 3rd-degree of AV block and a ventricular rate of less than 30 beats per minute. Next day, pacemaker interrogation revealed that third-degree AV block was preserved. The patient was discharged in satisfactory condition.

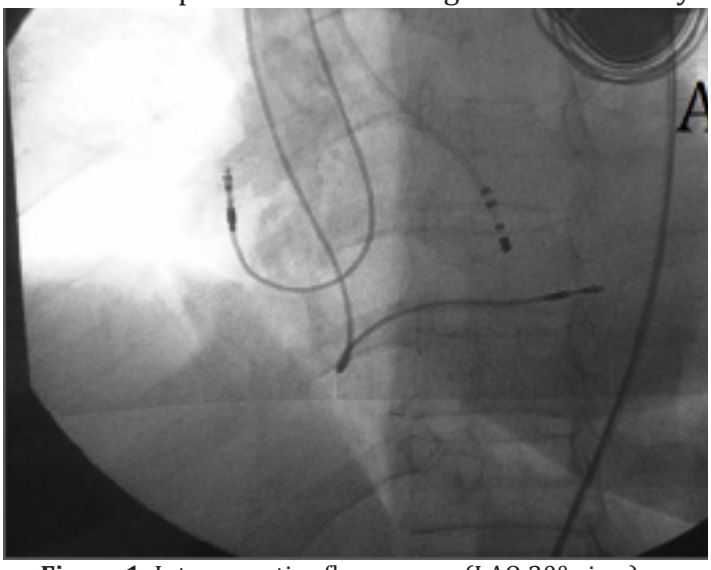

Figure 1: Intraoperative fluoroscopy (LAO 30º view). Abl: ablation mapping catheter in the left mid-septal area introduced through the aorta.

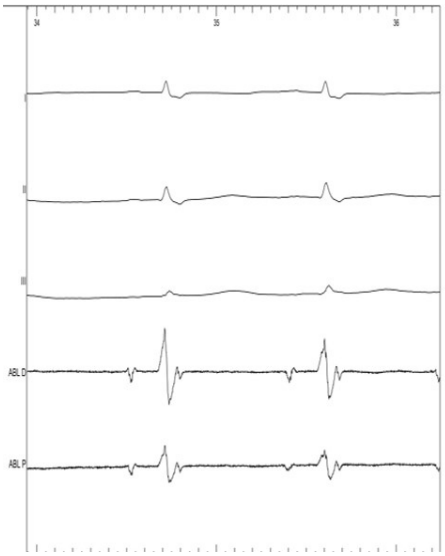

Figure 2: Intraoperative endogram. Mapping electrode introduced through the aorta is located in the left mid-septal area, a His bundle spike is not verified.

Discussion

Typical slow-fast AVNRT constitutes 85% of all AVNRT cases. Also, there are other forms of tachycardia: “fast-slow” and “slow-slow”. Generally, it is assumed that AVNRT emerges in the structures located in the Koch triangle. However, recent evidence suggests that AVNRT may also involve the interatrial septum, left atrium and coronary sinus [7].

The first report of the surgical management of AVNRT by purposely creating a complete AV heart block was published by Gianellis et al. in 1967 [8]. The authors proposed surgical dissection in the AV node area during CPB. In 1977, Harrison et al. described a His bundle cryoablation technique for the same purpose [9]. Previously we described our experience with the laser peri-nodal destruction technique for patients with AVNRT [10].

As mentioned, the first successful clinical attempt to interrupt only one of the dual AV node pathways of conduction, leaving normal AV node conduction intact was accomplished surgically by James L. Cox and colleagues at Duke University using a “discrete cryosurgical technique”, employing a 3 mm cryoprobe. Postoperative EP studies revealed that the slow α-pathway had been blocked and the fast β-pathway had been preserved [5].

Discussion of perinodal cryoablation surgery requires consideration electrophysiological and histological changes in the AV connection tissues. Holman and colleagues provided experimental study of surgery performed on 12 dogs. The authors demonstrated that postoperatively, retrograde ventricular-to-atrial conduction was blocked and that in 4 dogs with documented dual AV node conduction pathways preoperatively, the slow α-pathway had been blocked leaving the fast β-pathway had been preserved. Authors stated that no cryolesions extended into the histologically verified area of AVN but were close to it [11].

Conclusion

It is possible that the inability to find a His spike during final EP procedure was due to fibrous tissue created by the original surgical perinodal cryoablation in this patient. Complete AV node block had to be accomplished by placing lesions in the left paraseptal area because of the absence of a detectable His spike using the usual right-sided approach. Our findings in this patient should be helpful to interventional EP’s who need to create complete heart block late after previous surgical perinodal modification for AVNRT.

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