Paroxysmal Nodal Reentrant Tachycardia*

Surgical Cure with Preservation of Atrioventricular Conduction


*From the Division of Cardiology and Cardiovascular Surgery, Centro Nacional de Especialidades Medico-Quirurgicas de la Seguridad Social, Clinica Puerta de Hierro (University Hospital), Madrid, Spain.

In a patient with repetitive disabling tachycardias refractory to pharmacologic treatment, the electrophysiologic study suggested the existence of atrioventricular nodal reciprocating tachycardia. During ventricular pacing, endoepicardial mapping of the lower atrium showed the atrial breakthrough point in an area of the lower interatrial septum close to the AV node crista. A selective atriotomy was performed. The postoperative electrophysiologic studies showed absence of antgradreduction at several ventricular pacing rates, while antgradreduction is preserved. The patient remained free of arrhythmias 21 months after surgery, taking no antiarrhythmic drugs.

Electrophysiologic studies using programmed stimulation, 1-3 endoepicardial mapping, 4-5 and surgical results 6,7 have led to a better knowledge of AV junctional reciprocating tachycardias. There is more experience in the surgical treatment of AV junctional reciprocating tachycardias incorporating Kent bundles in the circuit. However, those tachycardias with intranodal mechanism or those incorporating other types of accessory pathways are less well-known, and surgical treatment experience is limited. 8-11

The surgery in the latter type of AV reciprocating tachycardias basically depends on the surgical interruption or curoablation of the His bundle, as demonstrated by Harrison et al 8 and Klein et al. Pritchett et al 9 and Ward et al 10 have recently reported surgical correction of arrhythmia while preserving AV conduction.

We recently operated on a patient with recurrent AV reciprocating tachycardia which was refractory to medical treatment, using endoepicardial mapping of the lower atrium close to the AV junction prior to AV interruption of the tachycardia circuit. The patient remained free of arrhythmias 21 months after surgery, with preserved AV conduction.

Case Report

The patient was a 62-year-old woman with no clinical or hemodynamic evidence of heart disease, who had had bouts of palpitations since 1966. Initially, the tachycardia appeared once or twice a month, lasting for 15 to 30 minutes, and remitted spontaneously. Since 1975 the tachycardia episodes had become more frequent (five to six per month) and lasted longer. From 1979 on, the crises increased to three or four per week, ranging from three to 12 hours, with occasional syncpe. Furthermore, the patient showed evidence of severe impairment of her quality of life and was unable to perform her usual activities.

The arrhythmia remitted temporarily after intravenous (IV) administration of verapamil (10 mg), ajmaline (1 mg/kg), or mexiletine (250 mg as single doses). Multiple oral antiarrhythmic drugs were used without preventing tachycardia, the only ones showing some effect being amiodarone (600 mg/day) and quinidine sulfate (1 g/day), or the association of digoxin (0.25 mg/day) with verapamil (240 mg/day), which lengthened the intercresis interval and shortened the crises.

Preoperative Electrophysiologic Study

This study was carried out to assess the mechanism of arrhythmia and the action antiarrhythmic drugs and diverse modalities of cardiac pacing.

The basal ECG showed sinus rhythm with 0.12-sec P-R interval, left anterior hemiblock and occasionally right bundle branch block (Fig 1A). All antiarrhythmic medication was suspended one week before the study. Three multipolar electrode catheters were inserted...
through the femoral veins and located in the lateral aspect of the right atrium, right ventricular apex, and tricuspid area for His bundle recording. Atrioventricular conduction intervals in sinus rhythm showed a P-R interval of 120 msec, A-H of 60 msec, and H-V of 40 msec.

Right atrial stimulation with a 340-msec coupling interval to the previous P wave produced a lengthening of the A-H interval from 60 msec to 190 msec, with initiation of tachycardia with a 350-msec cycle and antegrade conduction up to a ventricular stimulation frequency of 200 beats/minute and persistence of the ventriculoatrial interval of 200 msec to 230 msec secondary to changes in the antegrade conduction time (A-H interval), with constant retrograde conduction times using the procedure of Narula,10 measuring from the atrial ECG recorded in the right ventricular septum or vice versa, showed second-degree type I supraventricular AV block with atrial pacing at 170 beats/minute and persistence of 1:1 ventriculoatrial conduction up to a ventricular stimulation frequency of 200 beats/minute without modification of the ventriculoatrial interval.

The use of the various types of pacing during tachycardia showed that the arrhythmia could only be controlled with rapid atrial pacing at 220 beats/minute (Fig 2) or with a ventricular pacing program of the "scan type" with two ventricular extrastimuli, which caused several spontaneous ventricular responses and occasionally reinitiated the arrhythmia (Fig 2).13

After medical treatment had failed and the patient did not consent to pacing, she elected surgery for an attempt at proximal atrionodal interruption of the tachycardia circuit or ablation of the His bundle if the former were not possible. The patient had surgery in January 1980 with deep endocardial mapping of the lower atrium and interruption of the tachycardia circuit at atrionodal level.
Endocardial Mapping of the Lower Right Atrium

We used five fixed bipolar reference electrodes, three of them epicardial, located as follows: right atrial appendage; left atrial appendage; and trabecular area at the right ventricular apex. The two remaining electrodes were placed after instituting cardiopulmonary bypass and atriotomy, in endocardial locations—between the coronary sinus and the inferior vena cava close to the anterosuperior angle of the triangle of Koch, and in the lower area of the interatrial septum in a zone of atrial muscle cephalad to the AV node crista between the apex of the triangle of Koch and the fossa ovalis (Fig 3).

The exploring electrode was a standard tripolar one with a 1-mm separation between poles for recordings at eight points in the atrionodal junction from the inferoposterior area of the coronary sinus to the vicinity of the atrial portion of the interventricular membranous septum (Fig 3).

Mapping of the eight preselected points was carried out antegrade with stimulation of the right atrium and retrogradely with pacing of the right ventricular apex to ascertain the point of earliest atrial activation near the AV junction.

This mapping shows that the breakthrough point is located in the lower area of the interatrial septum, anterior to the coronary sinus ostium; the distal and proximal parts of Todaro's tendon are activated 5 and 15 msec later. Activation of the proximal coronary sinus appeared 25 msec after the breakthrough point.

Retrograde atrial mapping during tachycardia could not be performed, due to its self-limitation trends after a few beats during surgery. For these reasons, the retrograde mapping was carried out with right ventricular stimulation.

Interuption of the Tachycardia Circuit and Postoperative Findings

After location of the His bundle ECG with the exploring electrode and with a safety margin of 2 mm within the triangle of Koch, two-pronged atriotomy was performed, with one incision above the AV node and the other following Todaro's tendon but stopping short of the 2-mm margin of the most proximal detected His ECG (Fig 3).

A permanent programmable pacemaker was implanted as prophylaxis for possible postoperative AV block. The antegrade AV conduction after the section shows sinus rhythm with preserved AV conduction and 240 msec P-R interval, with persistence of the preoperative left anterior hemiblock (Fig 4).

AV conduction intervals were measured eight days after surgery and the P-R interval was found to be 190 msec, with lengthened A-H interval and unchanged H-V time.

In this limited postoperative electrophysiologic study it was not possible to achieve atrial captures with ventricular stimulation at different cycle lengths because of the retrograde ventriculoatrial block (Fig 4).

Twenty-one months after surgery, the patient remained free of arrhythmias without antiarrhythmic drugs, and the ECGs resembled the prior ones, with P-R intervals 190 msec (Fig 4).

Discussion

Atrial participation in AV reciprocating tachycardias is a very controversial challenge. One way to demonstrate it would be to achieve exact or paradoxic ventricular captures with atrial stimulation.26 With late atrial extrastimuli applied during tachycardia, one may occasionally achieve captures of the first type, but never have captures of the second type been achieved.26-28 The impossibility of achieving ventricular captures during tachycardias with atrial extrastimuli applied at different locations in an appropriate moment of the cycle in the tachycardia suggests that the atrium does not participate in the tachycardia circuit.23,27,28 In our case the apparition of 2:1 ventriculoatrial block during arrhythmia without interrupting it suggests that the right atrium is not necessary for maintaining it, although part of it (the septal atrium) might be part of the circuit, which would be difficult to demonstrate.
since it is included in the ventricular ECG (Fig 2B).

In our patient, the application of very premature atrial extrastimuli during the study induced ventricular captures, but this was not possible with late stimuli, so that we could not rule out a priori atrial participation in the circuit or state that the atrium was separated by an upper common pathway. The use of ventricular extrastimuli on the upper right septum during AV reciprocating tachycardia suggests that the lower reflection area of the circuit was at His level.

The possibility of dual pathways could be suspected in this patient as ventriculoatrial conduction was maintained up to ventricular pacing cycles of 300 msec, while Wenckebach type grade 2 AV block appears for atrial stimulation cycles of 352 msec. 

Absence of the tachycardia in the patient of Pritchett et al. 10 and in ours after selective actions on the atrionodal junction suggests that this is not a casual phenomenon. The similarity of both cases is evident and emphasizes that in a selective group of patients with AV reciprocating tachycardia refractory to other treatment, surgical intervention may produce partial or total interruption of the circuit at atrionodal level. Even if the atrium is not part of the tachycardia circuit, either the antegrade or the retrograde limb or both, may be obtunded depending on the upper reflection point of the circuit.

In the cases of Ward et al. 11 with an anatomic substrate of left-sided AV accessory pathway and incessant AV tachycardia, cryosurgical ablation of part of the reentrant circuit abolished tachycardia, providing proof of the underlying mechanism. In our patient, the absence of ventriculoatrial conduction after surgery with multiple frequencies of ventricular pacing contrast with the findings of Pritchett et al. 10 of ventriculoatrial conduction in the postsurgical study. This could be explained by assuming that in our patient the retrograde limb of the tachycardia was interrupted in the atrial insertion while producing an incomplete lesion of the antegrade one. In the case of Pritchett et al the lesion of the retrograde limb would be incomplete. This might explain the two types of ventriculoatrial conduction. This diversity of findings suggests that in both cases the atrionodal junction was damaged, although in different zones. Another possibility of two different upper common pathways has been suggested by Coumel et al. 12 As in our patient, when part of the septomedial right atrium participated in the arrhythmia, the presence of atrio-Hisian or atrionodal tracts is probable.

In view of the limitations of atrial mapping during preoperative studies, we believe mandatory the application of more precise mapping of the lower atrial activation during AV reciprocating tachycardia or ventricular pacing.

We used endocardial mapping of the lower atrium described, which permits identification of the points of earliest retrograde atrial activation. The method of construction of this mapping differs from others used for the localization of Kent bundles or for surgery of ventricular tachycardia. 13,14 The location of the reference electrodes close to the physiologic inputs of the crista or dorsal area of the AV node might be operative, according to the work of Janse et al. 15 These reference electrodes in the lower atrium, very close to the AV junction, would inform us of the atrial participation in the AV reciprocating circuit. The fixed reference electrodes in both atrial appendages inform us of the participation or lack of it of other atrial areas in the moment of the tachycardia and of the interatrial conduction derangements that might be observed in retrograde mapping during surgery. The selection of points to be explored by the electrode probe in the atrial endocardium near the atrionodal junction may be variable and have elected a limited number of points in some manner imposed by the diameter of the tripolar probe used.

From the above considerations, we deduce that in selected patients with AV reciprocating tachycardia, the introduction of progressively sophisticated methods of atrial mapping might in the future permit healing some patients while preserving AV conduction.

REFERENCES

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Reversed Rivero-Carvallo's Sign in Right-Sided Hypertrophic Obstructive Cardiomyopathy

Kyozo Ishikawa, M.D., F.C.C.P.; Fukiyo Hirata, M.D.; Shunkichi Hirata, M.D.; and Masamichi Ishikawa, M.D.

An 18-year-old patient had right-sided hypertrophic obstructive cardiomyopathy. His case was quite unique in that the intensity of the systolic murmur was apparently decreased during the inspiratory phase and increased during the expiratory phase (reversed Rivero-Carvallo's sign).

Hypertrophic cardiomyopathy is classically associated with predominant left-sided obstruction, but cases of isolated and dominant right ventricular outflow obstruction (RVO) have been described. It is usually expected that the systolic murmur resulting from subpulmonic stenosis in RVO may be increased in intensity during the inspiratory phase and decreased during the expiratory phase (Rivero-Carvallo's sign). The present patient with RVO in hypertrophic cardiomyopathy was quite unique in that the intensity of the systolic murmur was apparently decreased during the inspiratory phase and increased during the expiratory phase (reversed Rivero-Carvallo's sign).

**CASE REPORT**

The patient, an 18-year-old man, was referred to us for cardiac evaluation because of cardiac murmur. He had been in good health and asymptomatic. He had never experienced chest pain or any kind of unconsciousness. Examination revealed a grade 3/6 systolic murmur with late systolic accentuation. This systolic murmur became louder on expiration and quieter on inspiration (Fig 1).

Intracardiac phonocardiogram recorded in the right ventricular outflow portion apparently demonstrated that the systolic murmur became louder on inspiration than on expiration (Fig 2). Cardiac catheterization revealed no intracardiac shunts, and a systolic gradient of 7 mm Hg was recorded between the right ventricular inflow and outflow tracts (Fig 3). The pulmonary artery pressure was 17/8 mm Hg. No pressure gradient was recorded in the left ventricular cavity. Electrocardiograms showed right axis deviation and right ventricular hypertrophy (Fig 4).

A selective angiogram with right ventricular injection revealed protrusion of the interventricular septum into the right ventricular outflow tract. Left ventricular obstruction was not demonstrated by the left ventricular angiogram. Echocardiography demonstrated a thick interventricular septum (27 mm) but did not reveal systolic fluttering of the pulmonary valve, which was considered to be characteristic of RVO.

**DISCUSSION**

The data from the cardiac catheterization and angiography demonstrated the presence of isolated RVO. The systolic pressure gradient within the right ventricle was slight in degree (7 mm Hg) but apparently nonartifactual. During the cardiac catheterization, the pressure recordings within the right ventricle were carefully repeated several times, reveal-