Case reports
Radiofrequency catheter ablation of atrioventricular nodal reentry tachycardia: selective approach to the slow pathway via the superior vena cava

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Key words:
Superior vena cava; Supraventricular tachycardia; Transcatheter ablation.

Selective radiofrequency catheter ablation of the slow atrioventricular nodal pathway is currently considered the first-line therapy for patients suffering from recurrent symptomatic atrioventricular nodal reentry tachycardia. In most cases slow pathway conduction may be selectively eliminated or modified by the application of radiofrequency current at the posterior portion of Koch's triangle. The ablation site is usually targeted by careful mapping of this area performed using an ablation catheter advanced via the inferior vena cava approach. In this report we describe 2 cases in which the conventional approach to the target site was either impossible owing to the presence of an atresic inferior vena cava (case 1), or contraindicated in view of a history of common femoral vein thrombosis, subsequently extended up to the inferior vena cava (case 2). In both patients a superior vena cava approach was utilized and the slow pathway was successfully ablated. In case of arrhythmias necessitating slow pathway mapping and ablation, such an approach may be considered as a feasible and safe alternative whenever, owing to the presence of anomalies and/or diseases of the inferior vena cava, the conventional approach cannot be employed.

(Ital Heart J 2001; 2 (2): 142-146)

Received October 27, 2000; accepted November 23, 2000.

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Introduction

Radiofrequency catheter modification or elimination of the slow atrioventricular nodal pathway is currently considered the first-line treatment for patients presenting with recurrent symptomatic atrioventricular nodal reentry tachycardia (AVNRT)[1]. The slow pathway is commonly localized at the posterior portion of Koch's triangle[2]. The effective ablation site is generally targeted by careful mapping of this area, accomplished using a deflectable catheter advanced along the inferior vena cava (IVC). In this report, we describe 2 cases in which a superior vena cava (SVC) approach was utilized to successfully ablate the slow pathway in 2 patients in whom the conventional approach to the target site was either impossible or contraindicated owing to congenital (case 1) and acquired (case 2) disease of the IVC.

Description of cases

Case 1. A 44-year-old woman was admitted to our hospital for recurrent episodes of paroxysmal palpitation. A 12-lead ECG, recorded during an arrhythmic episode, showed a narrow QRS complex supraventricular tachycardia at a rate of 180 b/min. P waves were discernible and the RP' interval was much shorter than the P'R interval. Vagal maneuvers as well as intravenous antiarrhythmic drugs administered in the emergency room had successfully terminated the arrhythmic attacks, whereas chronic oral antiarrhythmic therapy was ineffective in preventing recurrences. The patient's past medical history included pleurisy and appendicectomy at 22 and 36 years of age respectively. On admission, physical examination as well as chest X-ray and transthoracic echocardiography were normal. A 12-lead ECG recorded during sinus rhythm excluded manifest ventricular preexcitation or any other abnormality. Having obtained written informed consent from the patient, an off-drug electrophysiologic study was performed in a fasting nonsedated state. Following local anesthesia (0.5% bupivacaine), a 6F tetrapolar catheter (Soloist, Medtronic Puerto Rico, Villalba, PR, USA) was inserted into the right common femoral vein. While maneuvering the catheter under fluoroscopic guidance, the operator failed to advance it directly into the right atrium, the catheter course resulting posterior to the cardiac silhouette, as observed at the left lateral fluoroscopic view. Further advancement of the catheter permitted passage beyond the cardiac silhouette and into the right atrium via the SVC. Subsequently, contrast medium injection into the right and left iliac veins revealed that the IVC was not present. Passage of the catheter via a large azygos vein, into the SVC and then into the right atrium was confirmed. Subsequently, by means of a long 0.032 inch guide wire, similarly advanced through the SVC, the inferior part of the right atrium was probed. No access to the IVC could be found. In view of this evidence, consistent with infrahepatic atresia of the IVC, a second 6F tetrapolar catheter (Soloist, Medtronic) and a 7F bidirectional, deflectable mapping/ablation catheter (Stinger F Curve, Bard Electrophysiology, Lowel, MA, USA) were positioned in the coronary sinus and at the apex of Koch's triangle from the right basilic and the left subclavian veins respectively (Fig. 1A). The ablation catheter deflectability allowed stable recording of the His bundle electrogram (Fig. 1B).

Baseline electrophysiologic study revealed dual atrioventricular nodal physiology but excluded the presence of accessory atrioventricular connections. By programmed atrial stimulation, a typical AVNRT, with a cycle length of 350 ms, resulted reproducibly inducible when the antegrade fast pathway effective refractory period was reached. Then, by deflecting the ablation catheter in the opposite direction, the distal electrode was moved from the His bundle area to the posterior aspect of Koch's triangle, where mapping was carefully performed in sinus rhythm. A possible slow pathway potential was located slightly anterior to the coronary sinus ostium (Fig. 2). By applying a moderate counterclockwise rotation on the ablation catheter, stable contact was achieved. A single radiofrequency energy application delivered at this site for 30 s, in the temperature control mode (target temperature 65°C), resulted in the early occurrence of an accelerated junctional rhythm (Fig. 3). Subsequently, programmed and incremental atrial stimulation showed a continuous atrioventricular nodal antegrade conduction curve and the absence of any reentrant arrhythmia, even during isoproterenol infusion. Ventricular stimulation did not reveal any modification of the retrograde Wenckebach point. No arrhythmias were induced. During the follow-up period of 8 months no arrhythmias recurred, confirming the success of the procedure.

Case 2. A 74-year-old woman was referred to our hospital because of weekly episodes of sustained symptomatic AVNRT, unresponsive to various antiarrhythmic drugs. Patient history included essential hypertension lasting 10 years and a hemorrhagic stroke resulting in

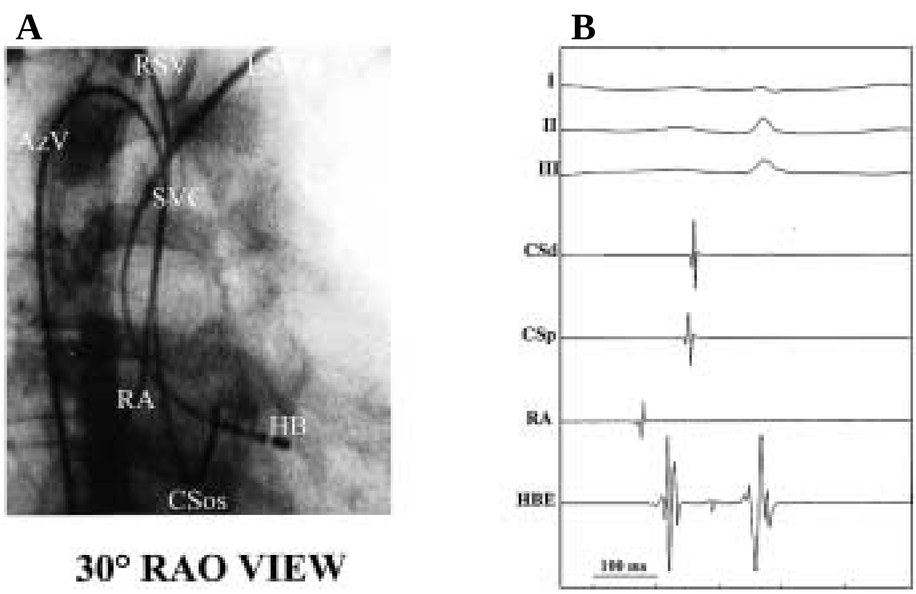

Figure 1. Panel A: *right anterior oblique fluoroscopic view (30° RAO) showing the electrode catheters positioned within the coronary sinus and the mid-right atrium, from the right subclavian and the azygos veins respectively. The mapping/ablation catheter is positioned in the His bundle region from the left subclavian vein. AzV = azygos vein; Csos = coronary sinus ostium; HB = His bundle region; LSV = left subclavian vein; RA = right atrium; RSV = right subclavian vein; SVC = superior vena cava.* Panel B: *surface ECG leads I, II and III simultaneously recorded with bipolar intracardiac electrograms from the distal and proximal coronary sinus, the right atrium and the His bundle region during sinus rhythm. CSd = distal coronary sinus; CSp = proximal coronary sinus; HBE = His bundle electrogram; RA = right atrium.*

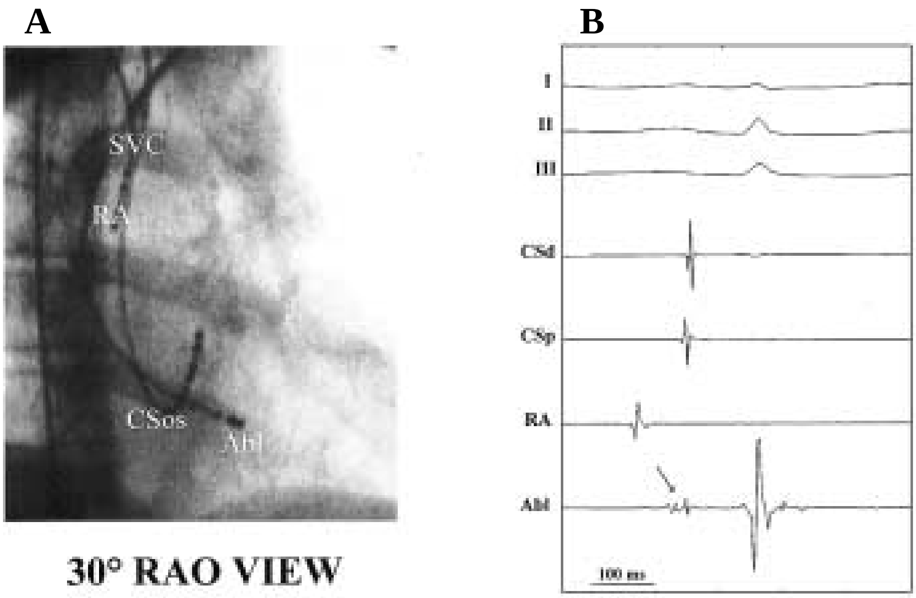

Figure 2. Panel A: *right anterior oblique fluoroscopic view (30° RAO) showing the right atrium catheter in a higher position and the bidirectional mapping/ablation catheter deflected in the posterior portion of Koch's triangle, where a slow pathway potential is recorded. Abl = slow pathway ablation site.* Panel B: *bipolar intracardiac recording, at the target ablation site, showing a slow pathway potential (arrow). Abl = target site electrogram. Other abbreviations as in figure 1.*

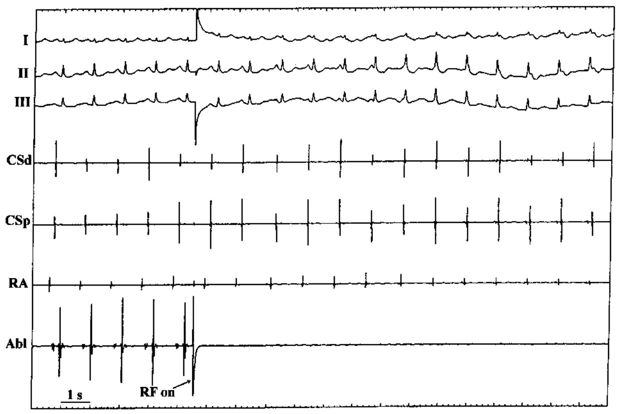

Figure 3. *An accelerated junctional rhythm occurring 5 s after the onset* (arrow) *of radiofrequency application at the target site. RF on = onset of radiofrequency application. Other abbreviations as in figures 1 and 2.*

aphasia and right hemiparesis at 72 years of age. Six months before admission to our hospital, she had undergone an unsuccessful catheter ablation of the slow atrioventricular nodal pathway at another institution, complicated by right common femoral vein thrombosis subsequently extended up to the IVC.

On admission, physical examination did not disclose any signs of congestive heart failure. A 12-lead ECG, recorded in sinus rhythm, revealed only T wave aspecific abnormalities in the right precordial leads. Chest X-ray and transthoracic echocardiography were normal. Owing to the recent thrombotic vasculopathy, it was decided to employ a SVC approach in order to perform electrophysiologic evaluation. Having obtained written informed consent from the patient and administered local anesthesia (0.5% bupivacaine), a 6F decapolar catheter (Response, Daig Corporation, Minnetonka, MN, USA) was inserted into the right basilic vein and then positioned within the coronary sinus. A 7F bidirectional deflectable mapping/ablation catheter (Stinger F Curve, Bard Electrophysiology) was inserted into the left subclavian vein and then positioned across the tricuspid valve. The His bundle electrogram was recorded. Baseline electrophysiologic evaluation, performed in a fasting nonsedated state, confirmed dual atrioventricular nodal physiology and excluded the presence of accessory atrioventricular pathways. Programmed atrial stimuli, applied at the mid-coronary sinus, documented the reproducible inducibility of a typical AVNRT, with a cycle length of 390 ms. Then, by deflecting the ablation catheter from the His position downwards and posteriorly, the posterior aspect of Koch's triangle was mapped in detail during sinus rhythm. A possible slow pathway potential was localized along the tricuspid annulus immediately anterior to the coronary sinus ostium (Fig. 4). Whilst maintaining stable contact by applying a counterclockwise torque on the ablation catheter, radiofrequency current in the temperature control mode (target temperature 65°C) was delivered at this site for 45 s. An active junctional rhythm rapidly ensued. Subsequent electrophysiologic evaluation confirmed successful ablation of the slow pathway and demonstrated the impossibility of inducing any reentrant arrhythmia, even during isoproterenol infusion. No arrhythmia recurrences occurred during the follow-up period lasting 3 months.

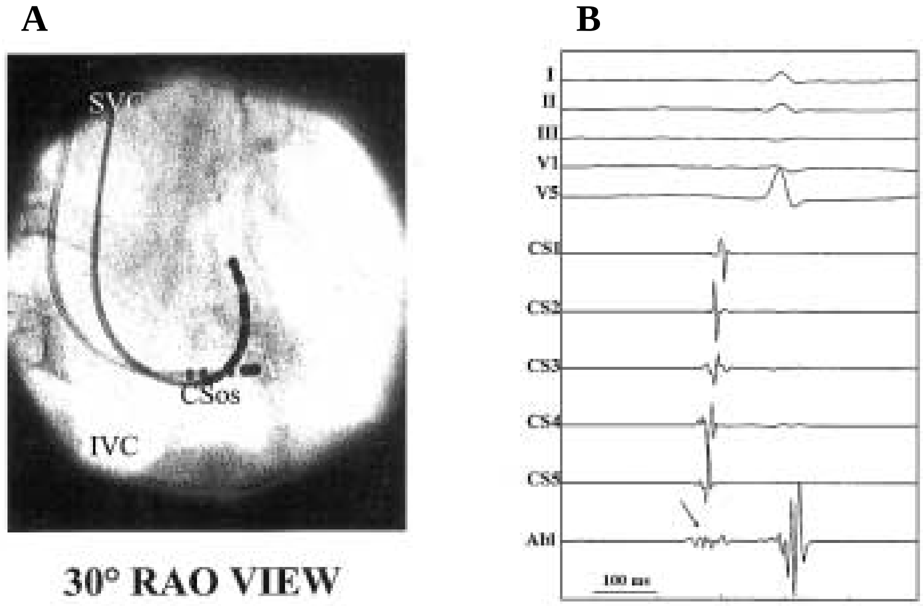

Figure 4. Panel A: *right anterior oblique fluoroscopic view (30° RAO) showing the decapolar catheter positioned within the coronary sinus and the bidirectional mapping/ablation catheter deflected in the posterior portion of Koch's triangle. Abl = slow pathway ablation site; IVC = inferior vena cava.* Panel B: *surface ECG leads I, II, III, V_1 and V_5 simultaneously recorded with bipolar intracardiac electrograms from the decapolar coronary sinus catheter and the mapping/ablation catheter, during sinus rhythm. CS1 to CS5 indicates distal to proximal coronary sinus electrode pairs. Abl = target site electrogram showing slow pathway potential* (arrow). *Other abbreviations as in figure 1.*

Discussion

Selective radiofrequency catheter ablation of the slow atrioventricular nodal pathway is currently considered the first-line therapy for all forms of AVNRT[3]. In the majority of patients, slow pathway conduction may be selectively eliminated by applying radiofrequency current at the posterior portion of Koch's triangle[2]. The different techniques[4-7] used to target the effective ablation site (anatomically guided, electrogram guided and combined techniques) are exclusively performed following right atrium catheterization achieved via the IVC approach. In fact this approach is believed to allow for better stability of the ablation catheter.

In this report we describe 2 cases in which the conventional approach was either impossible or contraindicated, because of the presence of congenital anomalies (case 1) and acquired diseases (case 2) of the IVC. The first patient presented with atresia of the IVC, a rare but generally asymptomatic malformation resulting from failure of the right subcardinal vein branches to join the veins of the primitive liver. In this anomaly the postrenal segment of the IVC joins the azygos-hemiazygos veins and drains into the SVC, whereas the hepatic veins drain directly into the right atrium[8]. The second patient presented with a recent history of thrombosis of the IVC, as a late complication of a previous unsuccessful attempt at slow pathway ablation performed at another institution. In both patients a SVC approach was utilized to ablate the slow pathway. The use of a bidirectional deflectable ablation catheter allowed accurate mapping and stable positioning in both the anterior and posterior areas of Koch's triangle. Thus the atrioventricular node and His bundle were correctly localized and precise ablation of the slow atrioventricular nodal pathway was possible. The SVC approach is commonly employed to ablate the right anteroseptal and anterior accessory pathways when the IVC approach does not permit satisfactory catheter stability[9,10]. Furthermore, this approach has already been employed in a patient with congenital absence of the IVC[11], in whom a right posteroseptal accessory pathway was successfully ablated. Finally, the SVC approach for slow pathway ablation adopted in these two cases can also be used in every patient, in whom an IVC filter has been implanted for recurrent thromboembolism.

In conclusion, although the transvenous femoral approach may allow for easier catheter positioning within the Koch's triangle and for better stability at the target site, the SVC route should be considered as a feasible and safe alternative for patients who present with congenital anomalies and/or acquired diseases of the IVC and who necessitate mapping and ablation of the slow pathway.

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