Chapter 11

Anatomical perspective on radiofrequency ablation of AV nodal reentry tachycardia after Mustard correction for transposition of the great arteries

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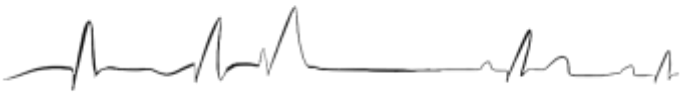

Abstract

A case of radiofrequency catheter ablation of atrioventricular (AV) nodal re-entry tachycardia, in a patient with transposition of the great arteries after venous rerouting according to Mustard, is described. An electroanatomical map of the His and AV nodal region was created from inside the systemic venous atrium. Retrograde mapping of the pulmonary venous atrium was performed and the arterial catheter retracted to a position in close proximity to the venous catheter inside the intra-atrial baffle. This position was chosen to deliver radiofrequency current.

Introduction

Transposition of the great arteries (TGA) is a congenital cardiac malformation characterized by concordant atrioventricular (AV) and discordant ventriculo-arterial connections that accounts for approximately 2.4% of all congenital heart diseases.[1] Currently, anatomical correction of TGA by performing an arterial switch procedure is the treatment of first choice. This operation is regularly performed since the 1980s, before which surgical treatment consisted of physiological correction with venous rerouting of blood according to Mustard or Senning (Figure 1).[2,3] Thus, the majority of adults with TGA have undergone either a Mustard or Senning procedure. Common complications following Mustard and Senning procedures include conduction disorders and arrhythmias, often of atrial origin. The mechanism most commonly described is intra-atrial re-entry based on scar tissue,[4] whereas AV nodal re-entry is far less common. In patients with normal cardiac anatomy, radiofrequency catheter ablation (RFCA) is highly effective.[5] Literature on RFCA in patients after venous switch procedures is, however, limited.[4,6-8]

In this paper, we describe a case of RFCA of AV nodal re-entry tachycardia (AVNRT) performed in a patient with TGA treated by venous rerouting according to Mustard, with focus on the anatomical situation following this operation and implications for the ablation procedure.

Case presentation

A 24-year-old man was referred for recurrent episodes of narrow complex tachycardia, despite the use of different antiarrhythmic drugs. The patient was born with TGA, for which a Mustard operation was performed 2 weeks after birth. Tachycardia events had a sudden onset and its clinical signs were palpitations and laryngeal discomfort. Chemical cardioversion using adenosine

was necessary to terminate events. On physical examination, blood pressure was 140/85 mmHg and heart rate 65 beats per minute (bpm). The electrocardiogram showed right ventricular (RV) hypertrophy, consistent with a systemic RV after the Mustard procedure. Laboratory tests were normal. Transthoracic echocardiography showed a dilated RV with reasonable function (tricuspid annular plane systolic excursion 14 mm) and minor AV leakage. Given the frequent symptomatic recurrences, it was decided to perform an electrophysiological examination and subsequent ablation.

Electrophysiological examination and ablation

The procedure was performed under local anaesthesia and guided by fluoroscopy and a three-dimensional (3D) non-fluoroscopic mapping system (CARTO XP™, Biosense Webster, Diamond Bar, CA, USA). A 7-F ablation catheter (Navistar, Biosense Webster) and two 6-F quadripolar electrophysiological catheters were introduced through the right femoral vein into the systemic venous atrium (His position, systemic venous atrium, and left ventricle, respectively). A second 7-F ablation catheter was later inserted through the right femoral artery and guided via the aorta, RV, and tricuspid valve to the pulmonary venous atrium, containing the AV node. A bolus of intravenous Heparin (7,500 IU) was administered.

At the beginning of the electrophysiological study, the patient was in sinus rhythm (cycle length [CL] 1,100 ms, AV 82 ms, His ventricular 40 ms). The antegrade Wenckebach point was at 300 ms and a significant A-His jump (53 ms) was observed. The retrograde Wenckeback point was at 340 ms and there was concentric decremental ventriculoatrial (VA) conduction, suggesting that retrograde conduction occurred through the AV node. During catheter manipulation, there were spontaneous episodes of tachycardia with a cycle length of 370–390 ms and a short VA interval of 58 ms. Atrial and ventricular

extrastimulations could not reset the tachycardia and it was consistently noted that the tachycardia started with a prolongation of the atrial-to-His interval, all of this being compatible with the diagnosis common-type AVNRT.7 Importantly, the tachycardia could reproducibly be induced by atrial electrical stimulation with a cycle length of 600/400 ms and two extrastimulations (Figure 1).

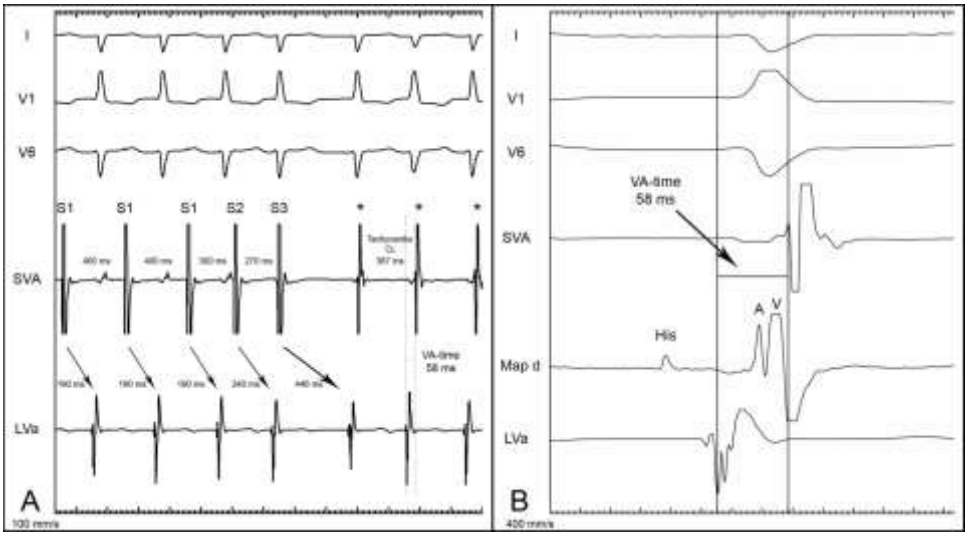

Figure 1. (panel A) Induction of the tachycardia by atrial programmed stimulation (100 mm/s). The tracing includes surface electrocardiogram leads I, V1, V6, and intracardiac recordings from electrophysiological catheters inside the systemic venous atrium (SVA) and left ventricle apex (LVa). In this tracing electrical stimulation with 400 ms (S1) and extrastimulation with 300 ms (S2) and 270 ms (S3) resulted in a critical prolongation of the atrioventricular (AV) time (190–240-446 ms) after which the tachycardia started (*). The tachycardia cycle length (CL) was 387 ms and the ventriculoatrial (VA) time was 58 ms. (panel B) Tracing of the tachycardia including a His registration (400 mm/s). The tracing includes surface electrocardiogram leads I, V1, V6, and intracardiac recordings from electrophysiological catheters inside the SVA, LVa, as well as from the mapping catheter, which was placed in His position. On the distal electrodes of the mapping catheter (Map d) a clear His potential can be seen followed by a superimposed atrial (A) and ventricular (V) signal.

Next, to obtain a 3D reconstruction of the complex atrial anatomy, an electroanatomical map of the His and AV nodal region was created from inside the systemic venous atrium using the CARTO system. Anatomical references (including His position and AV node) were marked on the map (Figure 2). Well-defined His signals as well as fragmented signals could be obtained from inside the intra-atrial baffle. However, since these signals were most likely derived from the AV node positioned in the posterior wall of the pulmonary venous atrium on the other side of the baffle, ablation at this site would mean that radiofrequency current had to be applied through the baffle. Although this was considered as an option, a direct approach via the pulmonary venous atrium was preferred to minimize the risk of injury to the baffle. Subsequently, retrograde mapping of the pulmonary venous atrium was performed by inserting the catheter through the femoral artery, passing the aorta, RV, and tricuspid valve into the pulmonary venous atrium. The arterial catheter was retracted to a position in close proximity to the venous catheter inside the intra-atrial baffle (Figure 2). This position was chosen to deliver radiofrequency current. Radiofrequency current was applied at 60 W with a maximum of 30 seconds on each ablation site. Location markers were placed at each ablation site on the electroanatomical map to obtain optimal insight in catheter positions in relation to the AV node and His bundle. During ablation of the slow pathway, an accelerated junctional rhythm consistently occurred. After ablation, AVNRT could no longer be induced with induction pacing even after intravenous administration of isoprenaline. The patient remained free of tachycardia during follow-up (3 months), without antiarrhythmic medication.

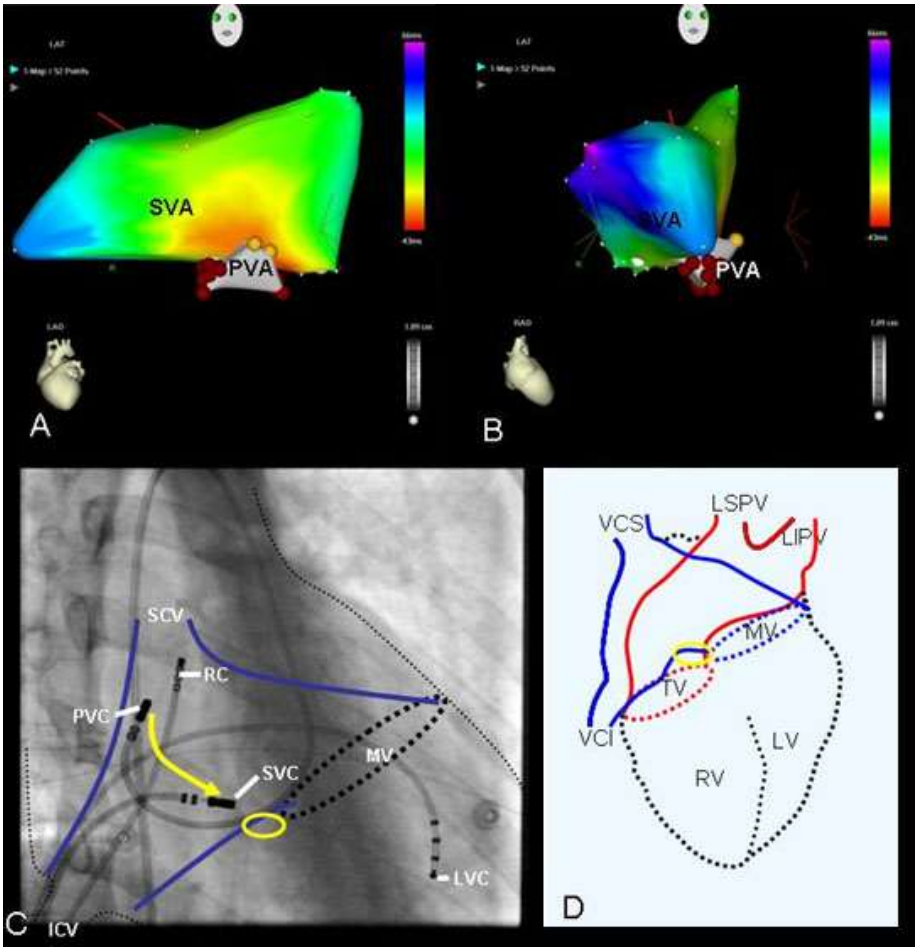

Figure 2. Three-dimensional electroanatomical map of the atrioventricular junction. Left anterior (A) and right anterior (B) oblique views. Anatomical maps were obtained from the systemic venous atrium (SVA) and pulmonary venous atrium (PVA). Red dots implicate ablation points in the area of the AV node, posterior in the PVA, inferior to the intraatrial baffle. Yellow dots indicate the His position. (C) Fluoroscopy. A reference catheter (RC) is located high in the SVA and a mapping catheter (SVC = systemic venous catheter) inferiorly in the intraatrial baffle pointing in the direction where the AV node is located just inferior to the baffle. The arterial catheter has been inserted in a retrograde fashion via the aorta, right ventricle, and tricuspid valve into the pulmonary venous atrium (PVC = pulmonary venous catheter) that contains the AV node. This catheter was retracted to a position in close proximity to the catheter in the SVA, close to the AV node, as indicated by the yellow arrow. At this location, radiofrequency current was applied. (D) Scheme of the anatomy. The AV node is indicated as a yellow oval. LIPV = left inferior pulmonary vein; LSPV = left superior pulmonary vein; LVC = left ventricular catheter; MV = mitral valve; TV = tricuspid valve; VCI = inferior caval vein; VCS = superior caval vein.

Discussion

This report describes RFCA of AVNRT in a patient after venous rerouting according to Mustard. In patients with normal cardiac anatomy, ablation of AVNRT is usually performed by a transvenous approach. In patients with TGA, the location of the AV node inside the right atrium is similar to patients with normal cardiac anatomy. However, after venous rerouting the caval veins drain into the systemic venous atrium/intra-atrial baffle, connected to the LV. As a consequence, the AV node is localized inside the pulmonary venous part of the atria and cannot be reached through a transvenous approach. Although a transbaffle approach to ablate AVNRT has been described,[8] this approach constitutes the risk of damaging the intra-atrial baffle, thereby creating a shunt between the systemic venous and pulmonary venous atria with the risk of hemodynamic overload or paradox embolism. Cryoablation is an alternative; however, there are reports indicating that this approach may be more time consuming with higher recurrence rates.[9] Still, its application might prove safer in terms of catheter stability in patients with complex anatomy. In the current situation, we preferred the arterial, retrograde approach. A disadvantage of this approach may be the difficult positioning of the catheters. Therefore, optimal insight and knowledge of the anatomy is mandatory. In the present report, we describe this procedure using intraoperative fluoroscopy and CARTO mapping.

A disadvantage of the retrograde arterial approach in ablation of AVNRT in Mustard patients is the relatively sharp angle that the ablation catheter must make in order to reach the location of the AV node. The venous catheter in the baffle toward the mitral valve (below which the AV node is located) can be used to guide the arterial catheter in the pulmonary venous atrium, as was described in our case. Also, the use of multidirectional steerable catheters may facilitate ablation using the arterial approach.

References

1. Warnes CA Transposition of the great arteries. Circulation 2006;**114**:2699-2709.
2. Mustard WT, Keith JD, Trusler GA, Fowler R, Kidd L The Surgical Management of Transposition of the Great Vessels. J Thorac Cardiovasc Surg 1964;**48**:953-958.
3. Senning A Surgical correction of transposition of the great vessels. Surgery 1959;**45**:966-980.
4. Khairy P, Van Hare GF Catheter ablation in transposition of the great arteries with Mustard or Senning baffles. Heart Rhythm 2009;**6**:283-289.
5. Akhtar M, Jazayeri MR, Sra J, Blanck Z, Deshpande S, Dhala A Atrioventricular nodal reentry. Clinical, electrophysiological, and therapeutic considerations. Circulation 1993;**88**:282-295.
6. Greene AE, Skinner JR, Dubin AM, Collins KK, Van Hare GF The electrophysiology of atrioventricular nodal reentry tachycardia following the Mustard or Senning procedure and its radiofrequency ablation. Cardiol Young 2005;**15**:611-616.
7. Kanter RJ, Papagiannis J, Carboni MP, Ungerleider RM, Sanders WE, Wharton JM Radiofrequency catheter ablation of supraventricular tachycardia substrates after mustard and senning operations for d-transposition of the great arteries. J Am Coll Cardiol 2000;**35**:428-441.
8. Khairy P, Fournier A, Mercier LA, Dubuc M Wolff-Parkinson-white syndrome in d-transposition of the great arteries and mustard baffle. Heart Rhythm 2006;**3**:853-856.
9. Zrenner B, Dong J, Schreieck J, Deisenhofer I, Estner H, Luani B, Karch M, Schmitt C Transvenous cryoablation versus radiofrequency ablation of the slow pathway for the treatment of atrioventricular nodal re-entrant tachycardia: a prospective randomized pilot study. Eur Heart J 2004;**25**:2226-2231.