

Etienne Delacrétaz, Jens Seiler,
Markus Schwerzmann, Hildegard Tanner

Swiss Cardiovascular Center Bern,
University Hospital, Bern,
Switzerland

Electroanatomic imaging to guide ablation of multiple intra-atrial reentrant circuits in congenital heart disease

Abstract

Patients late after surgical correction of congenital heart disease often develop atrial tachyarrhythmias, that may have complex mechanisms. The availability of three-dimensional electroanatomic map imaging has greatly improved our understanding of the arrhythmia mechanisms and facilitates catheter ablation. We present mapping studies in a 58-year-old patient with repaired tetralogy of Fallot who presented with persistent, symptomatic, atrial flutter. The case illustrates how catheter ablation can be guided by electroanatomic mapping in combination with entrainment mapping.

Introduction

Late after surgical correction of congenital heart disease (CHD), patients frequently develop intra-atrial reentrant tachycardias, which cause significant morbidity [1]. While antiarrhythmic agents have limited efficacy in the treatment of these arrhythmias, radiofrequency (RF) ablation is safe and can be used as primary therapy [1]. Nonetheless, RF ablation may be challenging because of complex arrhythmia mechanisms.

In operated CHD patients, the atria, particularly the right atrium, can support a variety of arrhythmia circuits, and although isthmus-dependent atrial flutter may frequently occur, many other arrhythmia mechanisms have been described. The availability of three-dimensional electroanatomic map imaging has greatly improved our understanding of the arrhythmia mechanisms and facilitates treatment [2].

Case description

A 58-year-old patient with repaired tetralogy of Fallot was referred for treatment of symptomatic, persistent, atrial flutter. He had had se-

veral palliative surgeries including a classic Blalock-Taussing shunt in 1955, a Pott's anastomosis in 1957, pulmonic valvulotomy and infundibular resection in 1962, VSD patch closure and infundibular resection in 1967, and had undergone again infundibular resection with transannular patch in 1979. He developed right heart insufficiency due to severe pulmonic regurgitation and underwent pulmonic valve replacement (stentless Shelhigh™ 23 mm pulmonic conduit) and tricuspid valve reconstruction (32 mm Edwards™ ring) in 2004. In 2007, he developed fatigue and markedly decreased exercise tolerance. On echocardiography, there was no significant valvular dysfunction and biventricular function was mildly depressed. His ECG showed typical atrial flutter with 2:1 AV conduction and a ventricular rate of 115/min.

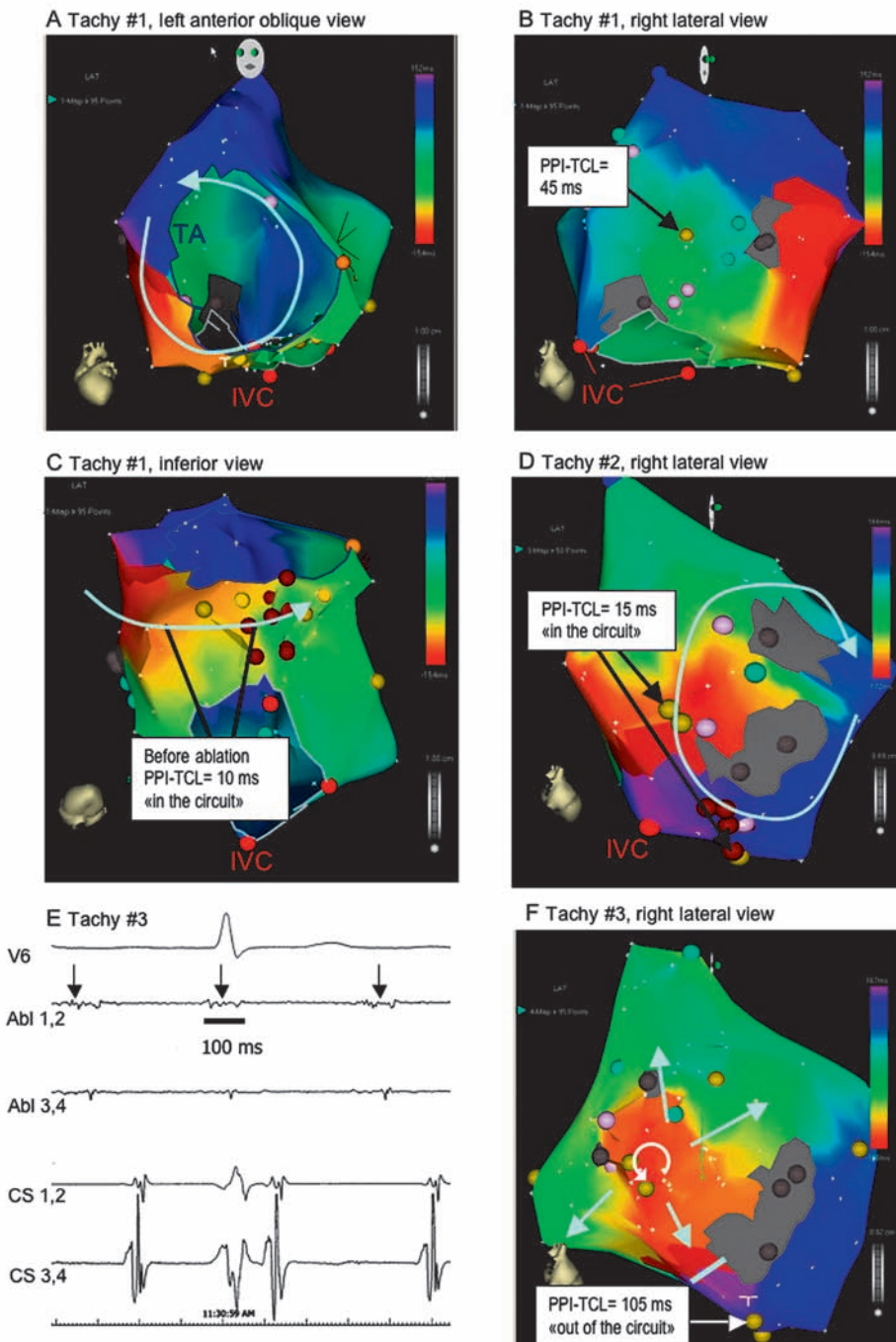
An electrophysiological study was performed using the Carto® System (Biosense Webster, Diamond Bar, CA, USA). The activation map of the clinical tachycardia was consistent with typical atrial flutter, with electric activation of the right atrium spreading in a counterclockwise direction around the tricuspid annulus (fig. 1A–B). Thus, a line of RF lesion was constructed to transect the cavo-tricuspid isthmus. During application of RF energy, the tachycardia changed, with slowing of the cycle length by 25 msec, and change in F wave morphology (not shown).

In such a situation, it is essential to have entrainment mapping data available, that is an analysis of the effect of pacing maneuvers from different sites. Briefly, if a pacing train slightly faster than the tachycardia cycle length is applied at a site remote to the circuit

Correspondence:
Etienne Delacrétaz, MD
Swiss Cardiovascular Center Berne
University Hospital
CH-3010 Bern
Switzerland
E-Mail: etienne.delacretaz@insel.ch

Figure 1

- A–C Electroanatomic mapping of typical atrial flutter. The timing of the local depolarisation is color-coded related to an arbitrary reference. Red denotes early depolarisation, followed by yellow, green, light blue, etc. The electrical wavefront spreads around the tricuspid annulus in a counterclockwise direction (arrows in panel A and C). The dark red dots show the ablation line transecting the cavotricuspid isthmus. TA = tricuspid annulus; IVC = inferior vena cava; PPI-TCL = postpacing intervals minus tachycardia cycle length; Short PPI-TCL (≤ 20 msec) demonstrates that the site studied is "in the circuit". When PPI-TCL is >20 msec, the site studied is "out of the circuit".
- D Right lateral view of the activation of right atrium free wall of the second tachycardia, an incisional arrhythmia circulating clockwise around an area of scar (in grey). There is a relatively narrow corridor at the low pivot point of the circuit, between the inferior aspect of the atriotomy scar, and the inferior vena cava (IVC). Patchy areas of diffuse scars, instead of linear lines expected after a surgical cut, are often seen in patients with enlarged right atrium late after CHD repair. Tachycardia #2 could be interrupted with a short line transecting the narrowest path (dark red dots).
- E Endocardial electrograms registered by distal and proximal electrode pairs of the ablation catheter (Abl. 1, 2 and 3, 4) and by distal and proximal electrode pairs of a reference catheter placed in the coronary sinus (CS 1, 2 and 3, 4). The ablation catheter registers abnormal, fractionated electrograms (arrows) at the site marked with a circular arrow in figure 1F. Application of radiofrequency energy at this site terminated tachycardia within 5 seconds.
- F Activation map of the third tachycardia. At the first glance, the electrical wavefront turns around the lateral scar (in grey) in a counterclockwise direction, as during tachycardia #2 (fig. 1C). However, entrainment mapping rules out such a mechanism, since the region between the scar and the inferior vena cava (IVC) is "out of the circuit" (PPI-TCL=100 msec, see text). Thus, conduction block over the narrow isthmus is confirmed (double white line). Electrical activation spreads in all directions from a relatively large area displaying abnormal, low amplitude, fractionated electrograms (fig. 1E). The mechanism cannot be precisely demonstrated, and is either a focal abnormal activity, or more likely a small reentry circuit encompassing several mm up to 1 cm (small circular arrow).



Electrical activation spreads in all directions from a relatively large area displaying abnormal, low amplitude, fractionated electrograms (fig. 1E). The mechanism cannot be precisely demonstrated, and is either a focal abnormal activity, or more likely a small reentry circuit encompassing several mm up to 1 cm (small circular arrow).

“out of the circuit”), the interval between the stimulus and the next local activation (post-pacing interval [PPI]) will be longer than if the same pacing train is applied in a site within the circuit (“in the circuit”) [2–4]. In the latter case, PPI equals tachycardia cycle length or exceeds it by 20 ms at most.

In this case, short PPI at several cavo-tricuspid isthmus sites had proved that the tachycardia was an isthmus-dependent flutter before ablation (fig. 1C, short PPI in the cavotricuspid isthmus). After the tachycardia change, entrainment mapping from the medial site of the ablation line showed that the cavotricuspid isthmus was not any more “in the circuit”. This means that a block of conduction was created through the isthmus, and that a second loop continued to rotate after disconnection of the first tachycardia limb. Such a circuit is called a double-loop reentry. Activation map of the new tachycardia (tachy #2, fig. 1D, arrow) was consistent with an incisional tachycardia revolving clockwise around areas of scar in the right lateral wall in relation with previous atriotomies. In dual-loop tachycardias, both loops share a common isthmus along the lateral portion of the tricuspid annulus, which is technically very difficult to target with ablation [5, 6]. Tachycardia #2 circuit had a narrow isthmus between the inferior extremity of the scar (in grey) and the inferior cava vein (light red dots). A short line of RF ablation allowed to terminate the tachycardia. Subsequently, control stimulation was performed and reproducibly induced a third tachycardia with a different cycle length (fig. 1E). The activation map suggested again a circuit around the lateral atriotomy scar, but entrainment mapping showed that it was not the case: PPI at the lower pivot point of the tachycardia were very long showing that the ablation line successfully transected this region (fig. 1E). Accordingly, the low anterior right atrium had become a passive bystander with late activation.

Over the area in red of the activation map in figure 1F, fractionated low amplitude potentials were recorded (fig. 1E), and tachycardia termination occurred several times when just touching this region with the catheter. A clear mechanism of the third tachycardia could not be demonstrated but it was either a focal abnormal activity or rather a small reentry circuit that could not be delineated precisely enough with the catheter used. Application of RF energy definitively terminated tachycardia after 5 seconds. Subsequently no further tachycardia could be induced with control stimulation.

In conclusion, this case illustrates that arrhythmia mechanism may be complex in the CHD population. Electroanatomic anatomic mapping is a valuable instrument to delineate anatomical obstacles and circulating activation wavefront, as well as to design ablation lines to interrupt reentrant circuits. For a comprehensive understanding of the arrhythmias, they should be interpreted in the light of entrainment mapping at a few key locations, that rapidly delivers important information.

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