Electroanatomical Mapping and Ablation of Upper Loop Reentry Atrial Flutter

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Upper loop reentry is a rare type of non isthmus-dependent atrial flutter with a reentry circuit around the superior vena cava. We present a case of atrial flutter of this type in a 75-year-old man with concomitant sick sinus syndrome. The diagnostic approach used three-dimensional electroanatomical mapping with the upgraded ENSITE 3000 system. This is the first case to be described where, apart from non-contact mapping, the circuit was also tracked in detail with three-dimensional contact mapping. The reentry circuit followed a clockwise course around the superior vena cava and passed through a conduction gap in the crista terminalis. Radiofrequency ablation of this region successfully eliminated the tachycardia.

Case presentation

A 75-year-old man was referred to our Department for ablation of symptomatic, drug-resistant, supraventricular tachycardia. He was already under treatment with amiodarone. The ECG during the episodes showed supraventricular tachycardia with 1:1 or 2:1 ventricular response and an atrial rate of 180 min⁻¹. The morphology of the atrial activation in the classical and monopolar electrograms and isopotential activation maps. The recent upgrade of the system allows the simultaneous creation of further maps, such as the contact voltage map and the local activation time map. The latter map is created based on the timing of the local bipolar contact electrograms in relation to a standard reference point.

We present a case of upper loop reentry atrial flutter that was mapped using the upgraded ENSITE 3000 system. Apart from the dynamic map showing the wave front propagation, a static local activation time map was also constructed for verification purposes.
lar limb leads was isoelectric or isobiphasic and negative on V1 (Figure 1). The ECG free of tachycardia showed stable sinus rhythm at a rate of 45 min⁻¹.

During the electrophysiological study the catheters used were introduced via the femoral veins and placed as follows: a quadripolar catheter to the His bundle, a bipolar catheter to the right ventricular apex, a quadripolar catheter to the coronary sinus, a quadripolar mapping-ablation catheter and the ENSITE catheter, which was placed in the middle portion of the right atrium. The geometric reconstruction of the right atrial cavity and the creation of the isopotential map and local activation time map were carried out through sequential movements of the mapping catheter within the right atrium.

The patient was studied during the tachycardia, which had a cycle length of 320 ms. During the analysis of the isopotential map it became apparent that there was a reentry circuit moving clockwise around the superior vena cava, the posterior and the lateral wall of the atrium. Subsequently, the wave front was blocked at the crista terminalis, apart from a conduction gap in its middle portion, through which the activation passed in order to exit in the region of the right atrial appendage. The conduction gap in the crista terminalis was characterised by slow conduction, demonstrated by recordings of fragmented virtual electrograms and the delaying of the isopotential activation front by a time equivalent to 32% of the tachycardiac cycle length (Figure 2). The analysis of the local activation time map confirmed the existence of the above mentioned reentrant circuit and the conduction block in the crista terminalis, while at the same time precisely reproducing the crista terminalis conduction gap. In addition, the latter map also demonstrated the passive activation of the tricuspid annulus by two activation wave fronts, one clockwise and one anticlockwise, which collided at the level of the coronary sinus ostium (Figures 3, 4). The tachycardia was successfully ablated through the creation of a linear lesion perpendicular to the wave front conduction, from the middle of the crista terminalis as far as the superior vena cava (Figure 4C). The tachycardia could no longer be induced either by extrasystolic atrial stimulation or by rapid atrial pacing (up to 300 min⁻¹). The patient subsequently had a dual-chamber pacemaker implanted because of sick sinus syndrome and remained free of arrhythmias over a 2-month follow up period.

**Discussion**

We describe the case of a patient with upper loop reentry atypical atrial flutter, which was mapped and successfully ablated with the aid of the ENSITE 3000 electroanatomical mapping system. The term “upper loop reentry” was introduced by Yang et al⁴ in a study of atypical forms of atrial flutter that used conventional mapping to describe a circuit located in the upper portion of the right atrium, independent of the isthmus. In their interpretation of the sequence of recordings from multipolar catheters the authors hypothesised that the circuit passed around the fossa ovalis, with a region of blockage posterior to it, but they were unable to describe its complete route in detail. Delivery of energy applied at points demonstrating concealed entrainment was unsuccessful in one out of two patients.
Figure 2. Non-contact mapping. A: Isopotential maps showing the cyclic route of the stimulus around the superior vena cava in antero-posterior view (upper series) and in right posterior oblique view (lower series). The stimulus can be clearly seen to pass from the conduction isthmus to the middle of the crista terminalis (map 6). B: Virtual electrograms placed along the conduction isthmus show low-width, fragmented activity indicative of slow conduction.

Figure 3. Local activation time map. A: Right anterior oblique, cranial view. B: Left anterior oblique view. The activation timing sequence is shown by the colour scale on the left. The reentry circuit (black arrows), which was drawn based on the isochronal curves, rotates around the superior vena cava. The activation of the tricuspid annulus is isochronal in its upper and lower parts and occurs later in the region of the coronary sinus ostium, indicative of 2 activation wavefronts (grey arrows) that collide in that region.
It is clear that conventional mapping suffers from limitations in the detection of complex circuits. Recordings from multielectrode catheters are relatively few for the full tracking of the macro-reentrant circuit and concealed entrainment has relatively low prognostic value for successful ablation guidance, while it may identify sites that lie outside the active circuit. However, even if a protected isthmus region within the active circuit is correctly identified, no information is provided concerning the width of this isthmus or the linear lesion that is required. Tai et al., using non-contact mapping in 8 patients with upper loop reentry atrial flutter, demonstrated a circuit around the superior vena cava and a region of functional blockage in the upper part of the crista terminalis, with the wave front activation being conducted in a clockwise sense in the majority of cases. The specific course of the circuit resembled the case we present here. However, the interpretation of the findings during non-contact mapping requires special care, since a part of the activation of the circuit may be overlapped by ventricular repolarisation, atrial repolarisation, or the more electronegative activation of other atrial segments. Furthermore, the discriminatory capability of non-contact mapping is reduced at distances greater than 4 cm from the centre of the balloon catheter. For these reasons, non-contact mapping focuses on the detection of the tachycardia’s exit site and part of the slow conduction zone, data that are usually sufficient for successful ablation of the tachycardia. Very rarely is it possible to map the complete activation of the cavity. With the addition of the local activation time map the above limitations may be overcome. In the present case of upper loop reentry atrial flutter we describe for the first time a simultaneous contact and non-contact three-dimensional mapping, by the same system, confirming the accuracy of non-contact mapping with regard to the critical points of the circuit.

Conduction through the crista terminalis does not favour the maintenance of a stable circuit, since it is one of the mechanisms through which atrial flutter degenerates into atrial fibrillation via the creation of a short circuit. However, the arrhythmia described above was exceptionally stable throughout the procedure, in spite of the frequent catheter manipulations during the mapping. This stability must
be attributed to the existence of a slow conduction area, which is essential for the maintenance of a reentrant circuit with a relatively short perimeter. The non-contact mapping showed that the region posterior to the crista terminalis was responsible for the delay of the activation wave front, which amounted to 32% of the tachycardia cycle length. This location of the slow conduction area might not be merely due to chance in this particular patient with sick sinus syndrome. Sanders et al., in a recent study using electroanatomical mapping with the CARTO system in patients with sick sinus syndrome, found regions of low potential all over the atrial myocardium and especially in the region of the crista terminalis. In the present case, the long delay in conduction and the location of a region behind the crista terminalis with low potentials are findings consistent with those authors’ observations.

In conclusion, electroanatomical mapping provides new capabilities in the invasive treatment of atypical forms of atrial flutter and should be considered the approach of choice. Simultaneous contact and non-contact mapping using a single system has even more potential, since the advantages of both techniques can complement each other to enable a precise understanding of the mechanism of the tachycardia.

References