CASE REPORT

Bystander cavo-tricuspid isthmus activation during post-incisional intra-atrial reentrant tachycardia

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We describe a case of post-incisional atrial tachycardia resembling typical atrial flutter on the surface ECG. Typical atrial flutter reentry was ruled out by the results of activation and entrainment mapping. Nevertheless, overdrive pacing from the lateral edge of the cavo-tricuspid isthmus produced tachycardia entrainment with concealed fusion associated with post-pacing and stimulus-to-P wave onset intervals exactly matching the tachycardia cycle length duration and the electrogram-to-P wave onset interval, respectively. Therefore, that site was firstly severed by sequential radiofrequency pulses. However, a transformation of the tachycardia P wave morphology and endocardial activation sequence, not associated with tachycardia termination or cycle length modification occurred. After additional mapping manoeuvres, a relatively small reentrant circuit was identified in the low and mid aspect of the lateral right atrium with the critical isthmus located between the lower border of a cannulation atriotomy and the crista terminalis, close to the inferior vena cava orifice. A single radiofrequency pulse at that site terminated the tachycardia. Both the electrocardiographic pattern and the endocardial mapping data obtained in our case might be explained by a split of the reentrant wavefront into a secondary wavelet which freely propagated through the cavo-tricuspid isthmus without completing the peritricuspid loop.

In conclusion, bystander cavo-tricuspid isthmus activation during atrial tachycardia may simulate a typical atrial flutter pattern on the surface ECG. Further studies should evaluate the prevalence of this propagation pattern in post-incisional atrial reentry and atypical atrial flutters, and identify its implications for ablation strategy.

Key Words: Post-incisional atrial tachycardia, entrainment mapping, radiofrequency catheter ablation.

Introduction

The extensive application of new mapping techniques to radiofrequency catheter ablation procedures provides a better knowledge of the tachycardia circuit in different atrial reentrant arrhythmias[1-6]. In particular, a ‘dual-loop’ intra-atrial reentry has been recently described by Shah et al.[7] using a ‘non-fluoroscopic’, three-dimensional, mapping system in a series of five patients previously having undergone cardiac surgery. In their report, the reentrant wavefront produced a complete figure-of-eight activation path, and the transection of two separate isthmi by radiofrequency current delivery allowed effective tachycardia ablation in all cases.

This paper deals with a case of post-surgical atrial tachycardia resembling typical atrial flutter on the ECG, in whom further information regarding the arrhythmia circuit was provided by the entrainment mapping data.

Case report

A 46-year-old male had previously undergone mitral and aortic valve replacement (single leaflet disc valve; Ultracor, Aortech Europe Ltd., Lanarkshire, U.K.) for rheumatic disease. During surgery, three incisions were made: in the low and high right atrium, close to the caval orifices, for extra-corporeal circulation, and in the left atrial wall for valve replacements. In the post-operative...
period the patient had a sustained atrial fibrillation episode successfully cardioverted by amiodarone i.v. administration. At hospital discharge the ECG showed normal sinus rhythm without conduction disturbances. Eighteen months after surgery, the patient complained of recurrent episodes of palpitations, documented at surface ECG recording as typical atrial flutter. This tachycardia was refractory to several antiarrhythmic drugs (quinidine, propafenone or amiodarone) and he was therefore referred to our Institution for electrophysiological evaluation and catheter ablation. The procedure was performed in the fasting, non-sedated state, after amiodarone discontinuation for five half-lives and written informed consent was obtained. Three multielectrode catheters were inserted through multiple venous accesses: a 6F quadripolar catheter was introduced into the coronary sinus, a 6F decapolar catheter was placed along the antero-lateral right atrium (with the proximal and distal pairs of electrodes at the mid-high level and close to the lateral edge of the cavo-tricuspid isthmus, respectively) and, finally, a 7F, 4 mm-tip, steerable catheter was moved to several locations in the right atrium for detailed mapping and ablation.

At baseline recordings, a sustained atrial tachycardia with a 210 ms cycle length was present. The surface ECG P waves were negative in leads II, III and aVF, and biphasic in leads V1 (first deflection positive) and V6 (first deflection negative) resembling that of typical 'counterclockwise' atrial flutter. At intracardiac recording, the antero-lateral right atrial wall (from high to low) was activated almost simultaneously; the proximal coronary sinus atrial activation coincided with the initial downward deflection of the surface ECG P waves in the inferior leads (Fig. 1).
arrhythmia mechanism was diagnosed as reentrant since tachycardia was entrained by overdrive atrial pacing with a manifest fusion occurring on the surface ECG and intracardiac recordings\cite{8}.

In order to identify the areas involved in the reentry circuit, entrainment mapping was performed delivering 8–10 consecutive beats at 180–190 ms cycle length (first two beats of the sequence) from the distal pair electrodes of the mapping catheter positioned in different locations. Classification of the pacing site as within or outside the reentry circuit was made according to previously published criteria\cite{1,8}. Briefly, the presence of manifest or concealed fusion on both surface ECG or intracardiac recordings during tachycardia entrainment as well as the behaviour of the post-pacing interval (PPI) compared with the tachycardia cycle length (TCL), and of the stimulus time (ST; time interval between the stimulus artefact and the ECG P wave onset) with respect to the activation time (AT; time interval between the local electrogram and the ECG P wave onset) were evaluated after each pacing protocol.

According to the surface ECG P wave morphology and to the presence of diastolic activation at the inferior cavo-tricuspid isthmus (CTI), entrainment mapping was initially performed at different sites in that area (lateral and medial) with controversial results. In fact, pacing from the lateral edge of the isthmus transiently entrained the tachycardia with concealed fusion (Fig. 2A); In addition, the PPI and the ST matched TCL and the AP, respectively. On the contrary, pacing from the medial aspect of the CTI (performed at multiple levels to exclude sub-eustachian ridge tissue capture) produced

Figure 2  Tachycardia response to overdrive atrial pacing from the lateral (A) and medial (B) aspect of the cavo-tricuspid isthmus (CTI). In both panels surface ECG and intracardiac recordings are arranged as shown in Fig. 1(B) except for that obtained from the distal (Abld) and proximal (AbIp) pair of electrodes of the mapping catheter placed in the CTI. (A) Overdrive atrial stimulation at 190 ms cycle length (first two beats of the sequence) is performed during the ongoing tachycardia from the lateral edge of the CTI producing an apparent orthodromic capture of the surface ECG (even if a careful evaluation of P wave morphology could be limited by ST-T segment superimposition) and intracardiac recordings (no change in their morphology and activation sequence). The post-pacing interval (210 ms) matches the tachycardia cycle length duration, while the stimulus time (ST; stimulus-P wave onset interval) is 10 ms shorter than the activation time (AT; local electrogram-P wave onset interval). All these findings indicate tachycardia entrainment with concealed fusion. (B) Overdrive atrial stimulation at the same rate (first three beats of the sequence) from the medial aspect of the CTI produces tachycardia entrainment associated with a change in the intracardiac electrogram morphology and activation sequence along the antero-lateral RA (cranio-to-caudal activation) compared with baseline. The post-pacing interval (270 ms) exceeds the tachycardia cycle length, while a 20 ms difference occurs in the ST and AT interval measurement. See also text for discussion. St, stimulus artefact. Solid lines indicate the P wave references utilized for ST and AT measurements. All values are given in ms.
tachycardia entrainment with manifest fusion (evidenced by the changes in the right antero-lateral wall activation) associated with PPI and ST widely different compared with the TCL and the AT, respectively (Fig. 2B). Even if these findings clearly excluded typical atrial flutter reentry, since the lateral CTI was an ‘anatomically protected’ area participating in the reentrant pathway (as indicated by the entrainment data), catheter ablation was anyway attempted at that site by delivering sequential radiofrequency current pulses (75 W power; 65°C temperature; 45 s duration). As a result, the isthmus ablation produced a modification of the endocardial activation sequence characterized by a marked delay in the coronary sinus activation time compared with baseline, associated with a significant change in the surface ECG P wave morphology (Fig. 3). In addition, also a slight modification in the activation of the antero-lateral right atrium occurred with a change in the electrogram morphology of the second couple of electrodes. No variation in the TCL occurred. For this reason, the mapping catheter was moved and activations encompassing the whole TCL were observed in a relatively small area at the mid- and low-lateral right atrium. In particular, a fragmented, long-lasting (100 ms), mid-diastolic electrical activity was recorded in a narrow area between the lower border of a cannulation atriotomy scar and the crista terminalis close to the inferior vena cava orifice. Overdrive atrial pacing at that level led to transient tachycardia entrainment with concealed fusion associated with a PPI and ST perfectly matching TCL and AT, respectively (Fig. 4). These findings again indicated pacing from a protected area within the reentry circuit and, therefore, another radiofrequency pulse was delivered at that site producing prompt tachycardia termination. To enlarge the lesion slightly, two additional radiofrequency applications were delivered at close locations during sinus rhythm and, subsequently, programmed atrial stimulation (up to two extrastimuli at two basic drives and bursts) failed to induce any sustained atrial arrhythmia. In addition, bidirectional conduction block in the CTI was demonstrated by atrial pacing from both the lateral low right atrium and the

Figure 3  Modification of the tachycardia P wave morphology and intracardiac activation sequence during cavo-tricuspid isthmus (CTI) ablation. In panels A to C, surface ECG and intracardiac recordings are arranged as shown in Fig. 2. (A) Baseline recordings before radiofrequency (RF) current applications at the lateral edge of the CTI. (B) The fourth RF pulse produces a marked delay between the LRA and the CS activations (arrow) associated with a modification in the surface P wave and MHRA electrogram morphologies (asterisk). Tachycardia cycle length is unchanged. (C) The completion of the CTI ablation after two further RF applications resulted in a further prolongation of the LRA to CS conduction time (arrow) again without tachycardia termination or rate modification. (D) On the 12-lead ECG, the tachycardia P waves are now predominantly positive in the inferior leads, negative in lead V1, and positive in leads V2 to V6. All values are given in ms.

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proximal coronary sinus. The procedure was therefore, stopped and the patient remained asymptomatic at 13 months follow-up without arrhythmia recurrences documented at serial 12-lead and ambulatory 24-hour ECG recordings.

**Discussion**

A wide range of reentry circuits, sometimes multiple, has been recently described in patients suffering from atrial tachycardias (mainly post-incisional), by means of several new mapping techniques\[1-4,6,7\]. The primary role of anatomical, surgical, or functional barriers in creating areas of conduction block and favouring reentrant propagation has been established\[5,9\]. This report describes a post-incisional atrial reentrant tachycardia resembling typical ‘counterclockwise’ atrial flutter on the surface ECG recordings. Our ablation strategy was guided by the results of the entrainment mapping, a method which has proved useful in several reentrant arrhythmias of both atrial or ventricular origin\[1,8,10-12\]. Briefly, the presence of an excitable gap between the head and the tail of the propagating wavefront allows transient tachycardia entrainment by fixed rate, overdrive stimulation. If pacing is performed outside the reentry circuit or from a non-protected area within it, entrainment occurs associated with manifest fusion on the surface ECG and intracardiac recordings, resulting from collision of two pacing generated wavefronts (orthodromic and antidromic). Because of unopposed orthodromic conduction, the last paced beat is entrained.

**Figure 4** Baseline recordings (A) and tachycardia response to overdrive atrial pacing (B) at the site of successful ablation. Surface ECG and intracardiac recordings are arranged as shown in Fig. 2. (A) Fragmented, long duration (100 ms), mid-diastolic electrograms are recorded from the mapping catheter (AbI) placed in the low-lateral right atrium between the border of a cannulation atriotomy and the crista terminalis, close to the inferior vena cava orifice. (B) At this site, overdrive atrial pacing (180 ms cycle length) produces tachycardia entrainment with concealed fusion (no changes in P wave morphology and intracardiac activation), with the post-pacing and StP intervals matching the tachycardia cycle length duration and AT, respectively. A single radiofrequency pulse delivered at this location (not shown) promptly interrupted the tachycardia. See also text for discussion. St, stimulus artefact. All values are given in ms.
but not fused. The PPI interval, measured at the stimulation site, is equal to the propagation time through the reentry circuit (and, thus, to the TCL) plus the conduction time from and to the pacing site. On the other hand, if pacing is performed from a protected area (such as that bounded by lines of conduction block) within or adjacent to the reentrant path, the tachycardia circuit is continuously reset by the faster rate pacing without any detectable fusion on the surface ECG or intracardiac recordings since orthodromic and antidromic paced wavefront collision occurs inside a protected part of the reentry circuit (entrainment with concealed fusion). In this case, tachycardia resumes at the end of stimulation with a PPI exactly matching the TCL. In addition, during tachycardia entrainment, the ST is variably prolonged (depending on the distance between the stimulation site and the exit from the protected isthmus, and on the conduction properties of the activated tissues) and it should equal the AT during tachycardia. If all these criteria are met, the participation of the paced area in the reentry circuit (and, thus, to the TCL) plus the conduction time from and to the pacing site. On the other hand, if pacing is performed from a protected area (such as that bounded by lines of conduction block) within or adjacent to the reentrant path, the tachycardia circuit is continuously reset by the faster rate pacing without any detectable fusion on the surface ECG or intracardiac recordings since orthodromic and antidromic paced wavefront collision occurs inside a protected part of the reentry circuit (entrainment with concealed fusion). In this case, tachycardia resumes at the end of stimulation with a PPI exactly matching the TCL. In addition, during tachycardia entrainment, the ST is variably prolonged (depending on the distance between the stimulation site and the exit from the protected isthmus, and on the conduction properties of the activated tissues) and it should equal the AT during tachycardia. If all these criteria are met, the participation of the paced area in the reentry circuit is demonstrated\(^1,8\). Conversely, tachycardia entrainment with concealed fusion associated with wider differences (>20–30 ms) in the measured parameters indicates pacing from protected, bystander areas adjacent to the central isthmus\(^9\). In our case, tachycardia entrainment with concealed fusion occurred only during pacing at the lateral edge of the CTI, excluding, therefore, typical atrial flutter reentry configuration (and the ‘lower loop’ variant\(^6\)). However, since this site participated in the main reentrant pathway (according to the entrainment mapping results), it was initially severed by means of sequential radiofrequency pulses with a transformation of the P wave axis and morphology (due to the modification of the atrial activation sequence) without tachycardia termination or change in its cycle length duration. This behaviour was similar to that reported in the work of Shah \(\text{et al.}\) and raised the question about the possible dual-loop nature of the reentry circuit\(^7\). In fact, tachycardia was actually terminated (and cured) after the ablation of another narrow isthmus bounded by the inferior border of a lateral cannulation atriotomy (identified according to the presence of double potentials in an area just above that of continuous electrical activity recording) and the crista terminalis, close to the inferior vena cava orifice.

**Possible reentry circuit configuration**

According to the activation and entrainment mapping data, it could be hypothesized that the reentrant wavefront circulated in the mid and low aspect of the right atrium around a short cannulation scar. The very delayed conduction occurring at the narrow isthmus close to the lower turning point of the periatriotomy loop (as demonstrated by long-duration electrogram recording at a single site) permitted the tachycardia perpetuation through a relatively small circuit (Fig. 5A). Additionally, since the circulating impulse engaged the CTI before entering the area of slow conduction, the tachycardia wavefront may split into a secondary wavelet and freely propagate through the isthmus (that acts as a protected conduction channel) and the septal portion of the tricuspid annulus. By this means, a large

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**Figure 5** Possible tachycardia reentry circuit configuration and paced wavefront propagation in response to overdrive stimulation from the cavo-tricuspid isthmus (CTI). TV, tricuspid valve; SVC, superior vena cava; IVC, inferior vena cava; CT, crista terminalis; ER, Eustachian ridge; CS, coronary sinus; HB, His-bundle. (A) The reentrant wavefront (solid line) circulates around the atriotomy scar (arrows indicate the direction of the propagation) with a narrow, slow-conducting isthmus located at the low pivotal point of the reentry, close to the CT (grey area). Since the travelling impulse engages the CTI before entering the area of slow conduction of the main reentrant loop, it may generate a secondary wavefront propagating through the isthmus and the septal wall and colliding with that emerging from the upper turning point of the periatriotomy loop. (B) Since the lateral edge of the CTI acts as a protected ‘outer loop’ area of the periatriotomy reentry, overdrive pacing at that site (asterisk) produces tachycardia entrainment with concealed fusion [see also Fig. 2(A)] associated with a post-pacing interval matching the tachycardia cycle length. Dotted lines indicate the paced wavefront propagation. (C) Conversely, stimulation at the medial aspect of the CTI (which is far from the reentry circuit) entrains the tachycardia with surface ECG and intracardiac recording fusion resulting from a greater propagation of the paced wavefront around the tricuspid annulus. The post pacing interval equals the propagation time through the periatriotomy loop plus twice the conduction time from the pacing site to the reentry circuit [see also Fig. 2(B)], and thus exceeds the tachycardia cycle length duration.
amount of right atrial (and probably also left atrial) activation depended on the isthmus propagation and it occurred in the same way as during typical atrial flutter reentry. This behaviour might explain the electrocardiographic pattern of this tachycardia and its changes following the CTI ablation.

Although the different mapping technique does not permit a direct comparison, this case seems to differ from that previously reported as ‘dual-loop’ reentry. In fact, by means of the CARTO mapping system, Shah et al. completely reconstructed the tachycardia activation path and noticed the presence of two circulating loops, combining in a figure-of-eight depolarizing wavefront\(^7\). Invariably, one loop involved the inferior cavo-tricuspid isthmus while the other propagated around an atriotomy scar, and they were believed to be independent since both their propagation times were similar to the TCL duration. Unfortunately, no information was available regarding the tachycardia response to overdrive pacing since this technique was not employed to avoid tachycardia termination or degeneration. Conversely, in our case the secondary peritricuspid loop was probably widely incomplete as demonstrated by the ‘simultaneous’ activation sequence recorded along the antero-lateral aspect of the right atrium, and its change in response to overdrive pacing from the medial CTI. In fact, during stimulation at that site, the paced wavefront propagates both fully around the tricuspid annulus (as demonstrated by the cranio-to-caudal activation sequence recorded from the decapolar catheter in Fig. 2B), and in the medial to lateral direction through the CTI to entrain the tachycardia (Fig. 5C). The PPI at the CTI recording is prolonged since it is equal to the propagation time through the periatrotomy loop plus twice the conduction time from the pacing site to the reentry circuit. Conversely, overdrive stimulation from the lateral CTI resulted in tachycardia entrainment with concealed fusion since this area was immediately adjacent to the primary reentry path, very close to the slow conducting zone, and was believed to be independent since both their propagation times were similar to the TCL duration. Unfortunately, no information was available regarding the tachycardia response to overdrive pacing since this technique was not employed to avoid tachycardia termination or degeneration. Conversely, in our case the secondary peritricuspid loop was probably widely incomplete as demonstrated by the ‘simultaneous’ activation sequence recorded along the antero-lateral aspect of the right atrium, and its change in response to overdrive pacing from the medial CTI. In fact, during stimulation at that site, the paced wavefront propagates both fully around the tricuspid annulus (as demonstrated by the cranio-to-caudal activation sequence recorded from the decapolar catheter in Fig. 2B), and in the medial to lateral direction through the CTI to entrain the tachycardia (Fig. 5C). The PPI at the CTI recording is prolonged since it is equal to the propagation time through the periatrotomy loop plus twice the conduction time from the pacing site to the reentry circuit. Conversely, overdrive stimulation from the lateral CTI resulted in tachycardia entrainment with concealed fusion since this area was immediately adjacent to the primary reentry path, very close to the slow conducting zone, and was bounded by anatomical barriers (Fig. 5B). However, this ‘outer loop’ area was not critical for periatrotomy reentry perpetuation and, therefore, its ablation did not terminate the tachycardia.

A limitation of our report is that the reentry circuit characteristics have been recognized following the CTI ablation. Therefore, pacing from the second isthmus, actually responsible for tachycardia perpetuation, was performed only after the change of the atrial activation sequence. As a consequence, no information is available regarding the peritricuspid wavefront behaviour in response to pacing from the primary reentrant loop. In addition, the need for a dual isthmus ablation in this case remains an unresolved question. Actually, it could be postulated that in our case this strategy has been incorrect since the possible peritricuspid loop was widely incomplete. However, the block of conduction in the periatrotomy loop could not exclude tachycardia perpetuation (with a longer cycle length) due to the unopposed completion of the peritricuspid reentry. Nevertheless, with regard to this point, no information is available from published series since also in those cases the CTI was invariably the first area targeted for catheter ablation\(^7\).

In conclusion, a bystander CTI activation may occur during post-incisional atrial reentry and simulate a typical atrial flutter pattern on the surface ECG. This uncommon propagation route may be identified by careful analysis of the activation and entrainment mapping performed in multiple areas. Further studies should evaluate the prevalence of this activation pattern in patients with post-incisional atrial tachycardias and/or atypical atrial flutters. Finally, in this setting, the possible role of the secondary peritricuspid loop in sustaining the reentrant propagation should be defined in order to identify the best ablation strategy.

References