Use of non-contact mapping in the treatment of right atrial tachycardias in patients with and without congenital heart disease

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Received 29 March 2008; accepted after revision 19 June 2008

Aims Right atrial (RA) tachycardias may involve several mechanisms other than typical isthmus-dependent flutters, particularly in patients with congenital heart disease (CHD) or structural heart disease. We aimed at investigating the clinical utility of non-contact mapping in the diagnosis and ablation of these complex arrhythmias.

Methods and results Non-contact mapping was used to treat RA tachycardias in 22 patients (12 with CHD and 10 without CHD). Ablation strategy consisted of creating linear lesions between scars (in macro-re-entrant circuits) or targeting areas of earliest activation and breakout points (in focal tachycardias). Eleven of the 12 tachycardias in the CHD group were atypical macro-re-entrant flutters. The majority (9 of 12) involved the RA free wall, whereas the remainder involved upper loop re-entry. In contrast, 9 of the 12 tachycardias in the non-CHD group were focal and 3 were macro-re-entrant. Acute procedural success was 88%. During a follow-up of 26 ± 21 months, 90% of the patients reported either no symptoms (60%) or symptoms reduced to ≤ 50% pre-ablation levels (30%).

Conclusions Non-contact mapping can provide important information on the mechanism of complex RA tachycardias in patients both with and without CHD. This can be useful in formulating ablation strategies.

KEYWORDS Atrial tachycardia; Ablation; Non-contact mapping; Congenital heart disease

Introduction

Right atrial (RA) tachycardias are a common late finding in patients with congenital heart disease (CHD) and are a frequent cause of morbidity and mortality.1,2 The most likely mechanisms involve isthmus-dependent atrial flutter (AFL) and atypical macro-re-entrant AFL due to the presence of scar secondary to the structural heart defect or previous atriotomies.3–5 However, more complex mechanisms related to underlying sino-atrial node dysfunction may also play a role.6 Right atrial tachycardias can occur after most forms of surgery for CHD, including repair of atrial septal defect (ASD) and Tetralogy of Fallot, the Mustard operation, and Fontan procedure.7–10 In view of the abnormal electrical substrate and potentially complex re-entrant circuits involved, electro-anatomical mapping systems have been used in the diagnosis and treatment of such tachycardias.5,11 However, in cases in which the tachycardia is unstable, not easily reproducible or poorly tolerated, conventional or electro-anatomical mapping methods may be inadequate. Non-contact mapping with the multi-electrode array (MEA) catheter can be invaluable in these cases.12,13 To date, there have been very few reports on the use of non-contact mapping in patients with CHD. Abrams et al.14 compared non-contact mapping with electro-anatomical mapping in treating patients with RA arrhythmias late after the Fontan procedure and found that non-contact mapping is less accurate in defining areas of scar and low voltage when endocardial sites were > 40 mm away from the MEA.

The aim of the present study was to investigate the clinical utility of non-contact mapping in treating RA tachycardias in patients with CHD (excluding post-Fontan patients). In comparison, we also investigated the use of non-contact mapping in patients with RA tachycardia, but no history of CHD, in which it was felt that non-contact mapping would be useful. Non-contact mapping was used in such cases if patients had electrocardiogram (ECG) characteristics of a RA tachycardia (other than typical AFL) and structural heart disease (primary atrial electrical disease or acquired
structural heart disease) or previously failed conventional ablation for the arrhythmia.

Methods

Patient characteristics

Between January 2002 and July 2007, 22 patients (11 males; total of 24 tachycardias) with RA tachycardias underwent electrophysiological study using the non-contact mapping system (EnSite 3000, St Jude Medical, previously Endocardial Solutions, St Paul, MN, USA). Patients were subdivided into those with and without CHD (Tables 1 and 2). Twelve patients had a history of CHD (nine with previously repaired ASD, one with anomalous pulmonary venous drainage and a patent foramen ovale repair, one with corrected Tetralogy of Fallot, and one with transposition of the great arteries and a ventricular septal defect). Ten patients were in the non-CHD group. Mean (± SD) patient age was 46.5 ± 13.0 (range 29–65) and 45.3 ± 20.5 years (range 20–76) in the CHD and non-CHD groups, respectively.

Electrophysiological study

All anti-arrhythmic drugs (AADs) were stopped at least 5 days prior to the procedure. Procedures were performed with the patient in the fasting state under moderate sedation or general anaesthesia. A 7 Fr deflectable decapolar or quadripolar catheter was inserted via the right femoral vein and placed in the coronary sinus. The mapping/ablation catheter was also inserted via the right femoral vein. A 9 Fr sheath placed in the left femoral vein was used to introduce the MEA catheter, which was advanced over a 0.035 in. guide wire (positioned in the superior vena cava (SVC)) and deployed in the RA (Figure 1). Intravenous boluses of heparin were given during the study to maintain the activated clotting time between 250 and 300 s.

Right atrial anatomy was reconstructed by moving the mapping/ablation catheter along the endocardial surface of the chamber. Particular attention was paid to identify and label important anatomical landmarks such as the superior and inferior vena cava (IVC), RA appendage, tricuspid annulus, and previous atriotomy sites/areas of scar.

If the patient was in sinus rhythm at the start of the procedure, initiation of tachycardia was attempted with single and double extra-stimuli delivered from the coronary sinus or mapping/ablation catheter after a basic drive train (600 or 450 ms) or from burst pacing with cycle lengths between 300 and 200 ms (with 10 ms decrements). If this was successful, or if the patient was already pacing with cycle lengths between 300 and 200 ms (with 10 ms decrements), pacing was performed at cycle lengths 10–30 ms shorter than the tachycardia cycle length. Manifest fusion was considered to be present if there was any change in the atrial activation sequence compared with baseline tachycardia, whereas concealed entrainment was considered to be present if there was no change in the atrial endocardial activation or surface F-wave morphology, but a shortening in cycle length to that of the entraining rate. The post-pacing interval (PPI) was defined as the time interval from the last pacing artefact to the start of the rapid electrogram deflection of the first tachycardia cycle after cessation of pacing. A PPI < 30 ms was taken as evidence that the pacing site was within the tachycardia circuit.

Definitions

We defined RA tachycardias in line with previously published reports.13,17,18

- 'Upper loop re-entry' was defined as an atypical macro-re-entrant AFL involving the upper portion of the RA/peri-SVC.
- 'Lower loop re-entry' was defined as a cavo-tricuspid isthmus (CTI)-dependent atypical AFL involving the lower portion of the RA/peri-IVC.
- Right atrial free wall (RAFW) macro-re-entry was defined as an atypical AFL that involved a circuit around an anatomical area of block (e.g. area of scar) involving the RAFW, which was neither CTI-dependent nor solely confined to the upper or lower RA.
- Focal (or micro-re-entrant) atrial tachycardia was diagnosed if activation appeared to originate from a discrete area within the RA and spread simultaneously in multiple directions. The area of earliest activation (EA) was identified on the activation map as the focal point of origin of the tachycardia in time with the earliest virtual unipolar electrogram, whereas the breakthrough (BO) point was the site at which activation appeared to spread out towards the rest of the RA. The EA and BO regions were found to be at the same point in some cases, but at distinct sites in others.

Ablation strategy

Radiofrequency (RF) ablation was performed using a 4 or 8 mm electrode-tipped ablation catheter connected to an EPT-1000 generator. Cryoablation (6 mm-tipped Freezeor Extra or 8 mm-tipped Freezor Max) was used in three cases in the non-CHD group as these patients were particularly concerned about the possibility of pain during ablation (having previously undergone ablations using...
<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Gender</th>
<th>Structural heart disease</th>
<th>Previous ablations</th>
<th>Tachycardia characteristics</th>
<th>Ablation strategy</th>
<th>Energy source</th>
<th>Catheter tip size (mm) ± cool flow</th>
<th>Acute success</th>
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<td>8 ± Yes</td>
<td>Yes</td>
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<td>Linear lesion between SVC and scar</td>
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PV, pulmonary vein; PFO, patent foramen ovale; ULR, upper loop re-entry; SVC, superior vena cava; IVC, inferior vena cava; CTI, cavo-tricuspid isthmus; RF, radiofrequency; TGA, transposition of the great arteries; VSD, ventricular septal defect; PS, pulmonary stenosis; AVN, atrioventricular node; PPM, permanent pacemaker; RAFW, right atrial free wall; ASD, atrial septal defect; TOF, Tetralogy of Fallot; EA, earliest activation; AT, atrial tachycardia.
<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Gender</th>
<th>Structural heart disease</th>
<th>Previous ablations</th>
<th>Tachycardia characteristics</th>
<th>Ablation strategy</th>
<th>Energy source</th>
<th>Catheter tip size (mm) ± cool flow</th>
<th>Acute success</th>
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<td>Focal AT—upper and lower CT</td>
<td>Cluster lesions around EA and BO regions</td>
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<tr>
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<td>63</td>
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<td>Yes—CTI line</td>
<td>Focal AT—multiple sites in RA</td>
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<td>RF</td>
<td>8</td>
<td>Yes</td>
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<tr>
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<td>Atrial electrical disease</td>
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<td>Focal AT (multiple sites—septum, posterior isthmus, and RAA); CTI- dependent flutter</td>
<td>Cluster lesions around EA and BO regions; CTI line</td>
<td>RF</td>
<td>8/cool flow</td>
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<td>Focal AT (edge of scar)</td>
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<td>Yes</td>
<td>Two atypical flutter circuits; RAFW between scar</td>
<td>Linear lesions between areas of scar</td>
<td>RF</td>
<td>8/cool flow</td>
<td>Yes</td>
<td></td>
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</table>

ULR, upper loop re-entry; SVC, superior vena cava; IVC, inferior vena cava; CTI, cavo-tricuspid isthmus; CT, crista terminalis; RF, radiofrequency; Cryo, cryothermy; RAFW, right atrial free wall; EA, earliest activation; BO, breakout region; DCM, dilated cardiomyopathy; AT, atrial tachycardia; RA, right atrium; RAA, right atrial appendage; MV, mitral valve; TV, tricuspid valve.
RF energy). For macro-re-entrant flutters, a linear ablation line was created, which transected the activating wavefront (as determined from the colour-coded isopotential map) and also joining two areas of low voltage/scar as determined from the DSM (created during tachycardia, sinus rhythm, and coronary sinus pacing). Ablation was performed during tachycardia if possible, although in cases in which the tachycardia was unstable or short-lived ablation was performed during sinus rhythm. Sequential point-by-point energy applications were delivered. Successful ablation was defined as termination and non-inducibility of the targeted tachycardia. Completeness of linear lesions was verified by analysing the isopotential maps of atrial activation while pacing from points on either side of the line. In cases of focal atrial tachycardia, lesions were delivered at sites of EA and BO and along the line between these two points if they were deemed to be at a significant distance from one another.

Follow-up
Patients were routinely seen in the outpatient clinic 3–6 months following the procedure, where their symptoms were re-assessed. If they experienced further palpitations, Holter monitoring was performed to try to document the tachycardia. They were subsequently followed up either via further outpatient clinic visits or telephone interview. Symptoms of palpitations were classified as: none, reduced (~50% pre-ablation level), no change, or worse (greater than pre-ablation level).

Statistical analysis
Continuous data are expressed as mean ± SD.

Results
Tachycardia characteristics and ablation strategies for all cases are summarized in Tables 1 and 2. Nine patients had previously undergone ablation of their CTI for documented typical AFL. In all but one patient, atrial tachycardia was either present at the start of the procedure or could be induced with burst pacing or programmed stimulation. Overall average ablation time in cases in which RF energy was used was 630 ± 307 s, and the average fluoroscopy time was 32 ± 19 min. In cases of macro-re-entrant flutter, these were 527 ± 187 s and 32 ± 23 min, respectively, and in cases of focal atrial tachycardia, the corresponding times were 749 ± 426 s and 34 ± 16 min.

In the CHD group, 11 of the 12 tachycardias involved a macro-re-entrant mechanism (3 upper loop re-entry and 8 involving the RAFW). Figure 2 shows the 12-lead ECG and intra-cardiac electrograms of the tachycardia in Patient 7 in the CHD group, who had previously had an ASD repair. The corresponding MEA images of the macro-re-entrant flutter circuit involving RAFW and ablation lines are shown in Figure 3. One tachycardia in the CHD group (from Patient 9) was found to be focal—this originated from a small area of viable tissue surrounded by scar in the posterolateral free wall. In another patient (12), the tachycardia could not be induced, and therefore no ablation was performed. The tachycardia was subsequently inducible and successfully ablated during a repeat procedure with the MEA. Conduction block across linear lesion sets was determined to confirm the completeness of ablation lines. Figure 4 shows a ‘still’ from the propagation movie during pacing from the coronary sinus following successful ablation of an RAFW tachycardia (Patient 12).

In contrast, nine tachycardias in the non-CHD group were found to have a focal mechanism, whereas three involved a macro-re-entrant circuit. Figure 5 demonstrates two examples of focal atrial tachycardias in this group originating from the triangle of Koch and the low RA anterior wall (Patients 17 and 18 in Table 2, respectively). Ablation lesions were delivered at both the EA and BO points with additional lesions in
All three macro-re-entrant tachycardias in the non-CHD group involved areas of scar and the RAFW.

One patient in the CHD group and one in the non-CHD group (numbers 8 and 22, respectively) had CTI-dependent flutter in addition to other tachycardias, and both these arrhythmias were successfully ablated.

In cases in which ablation was attempted, the acute procedural success rate was 88% (11 of the 12 and 10 of the 12...
The remaining patients were followed up for a period of 26 months. In the CHD group, 7 of the 11 patients (64%) remained free of recurrence—4 of these were off all AADs, whereas 3 were still on at least 1 AAD (one of these patients was subsequently diagnosed with atrial fibrillation). Three patients (27%) reported fewer symptoms (<50% pre-ablation level), although there was no documented recurrence of the clinical tachycardia in any of these patients. All three were still on at least one AAD. One patient had a documented recurrence of the tachycardia 3 months after the procedure and opted for this to be treated medically. In the non-CHD group, five of the nine patients (56%) were free of recurrence (all off AADs), whereas three (33%) reported fewer symptoms (two still on AADs). One patient (11%) felt no difference in symptoms following the procedure. Two patients (one in each group) underwent repeat procedures with the MEA due to the inability to induce tachycardia during the first procedure (Patient 12) and multiple atrial arrhythmias (Patient 22).

Discussion

Right atrial tachycardias in patients with CHD are often challenging to treat in view of the complex anatomy and frequent multiple or atypical circuits involved. The present study demonstrates that non-contact mapping can provide important information on the mechanism of RA tachycardias in this group of patients that can be used to guide ablation strategy. Furthermore, non-contact mapping can also be very useful in patients without CHD but other features, such as structural heart disease or previously failed ablation attempts using conventional methods, are suggestive of a possibly complex tachycardia or atypical mechanism.

An interesting finding of the study is the difference in likely tachycardia mechanism in patients with and without CHD. Activation maps created during tachycardia showed that tachycardias in patients in the CHD group were more likely to be macro-re-entrant (11 of 12), whereas those in the non-CHD group were more likely to be focal in origin (7 of 12). The macro-re-entrant circuits identified in both groups consisted of single loops, with re-entry predominantly around the upper portion of the RA (upper loop re-entry) or RAFW around a central obstacle (usually an area of scar). Our ablation strategy consisted of identifying the tachycardia circuit (in cases of re-entrant flutters) or points of EA and BO regions (in cases of focal tachycardias) and mapping areas of low voltage or scar, as determined from the DSM. We then proceeded to deliver an ablation line between areas of low voltage/scar, ensuring that the line transected critical parts of the tachycardia circuit or focus. These ablation lines were completed even when tachycardia was terminated during ablation. Completeness of the lines was confirmed with differential pacing from either side of the lines and creation of new activation/propagation maps. Where necessary, further lesions were delivered to achieve bidirectional block across the lines.

Several investigators have reported results of using conventional or electro-anatomical mapping systems to diagnose and ablate atypical RA flutters in patients with and without CHD with varying success rates. There have been only a few reports on the use of non-contact mapping in atypical RA flutters and even fewer on focal RA tachycardias. Our study differs in that, the mechanism of the tachycardia was unknown at the outset of the cases. Non-contact mapping was used in the case of RA tachycardia which were felt to be difficult/complex to help elucidate the tachycardia mechanism and to determine an ablation strategy. We treated patients with and without CHD, and our findings highlight the marked differences in tachycardia mechanism (and therefore ablation strategy used) between the two groups.

The use of non-contact mapping has a number of theoretical advantages over conventional or electro-anatomical mapping in the group of patients studied. First, non-contact mapping allows us to identify the tachycardia mechanism even if it is unstable or short lasting. In addition, several different tachycardias and atypical flutter circuits are often
seen in these patients, and non-contact mapping allows these different circuits to be easily and readily identified. If an electro-anatomical mapping system is used instead in such cases, a new activation map would need to be created each time the tachycardia changed. However, despite the use of non-contact mapping, we were unable to terminate all inducible tachycardias in three cases (12%). In our opinion, the use of conventional or electro-anatomical mapping methods in these cases would also have proven to be unsuccessful as the tachycardias were rapidly changing and unstable. It is difficult to know whether these inducible, unsuccessfully ablated tachycardias caused further clinical symptoms as they were difficult to document due to their short-lived nature and infrequent occurrences. Although all three patients reported either improved or no symptoms at follow-up, this may be related to a number of factors (e.g. placebo effect and modification of drug regimen), rather than arrhythmia abolition. In contrast, the fact that some patients experienced further palpitations, despite apparent success during the initial ablation procedure, does not necessarily mean that the procedure had failed or that the clinical tachycardia had recurred (recurrence of the clinical tachycardia was only documented in one patient). Most of the patients had an abnormal atrial substrate for

Figure 5  Multi-electrode array isopotential colour maps showing two cases of focal atrial tachycardia. The tachycardias originate from the triangle of Koch (A) and the low right atrial anterior wall (B). Both patients had structurally normal hearts and had previous failed attempts at ablation using conventional techniques. The red dots represent ablation points. Note that the breakout region and the area of earliest activation are at distinct sites. The ablation strategy involved delivery of lesions at and in between these two sites. BO, breakout region; EA, earliest activation; SR, sinus rhythm; CS, coronary sinus; IVC, inferior vena cava.
arrhythmogenesis and the subsequent occurrence of a different tachycardia or even atrial fibrillation (as was found to be the case in one patient) would not be unexpected. This point is illustrated by Patient 22 who underwent three non-contact mapping procedures over a period of 3.5 years for symptom recurrence and was found on each occasion to have a different tachycardia (focal and atypical macro-re-entrant flutters as well as CTI-dependent flutter).

We diagnosed Patients 21 and 22 in the non-CHD group as having 'atrial electrical disease': a poorly characterized condition with no clear aetiology in which widespread atrial 'scarring' or low-voltage areas are found during electrical mapping. Both patients were young (aged 29 and 30, respectively) with no family history of note and no clear risk factors for cardiac disease. In our experience, there are no obvious structural changes on echocardiography or cardiac magnetic resonance imaging in these patients—the main abnormality appears to be electrophysiological and confined to the atria (at least initially). The appearances are very similar to operated CHD involving the atria and unsurprisingly, the arrhythmias tend to be macro-re-entrant (although focal atrial tachycardias were also found in Patient 22).

In cases of focal RA tachycardias, non-contact mapping allows us to identify the sites of EA and the BO regions, which, as shown in Figure 5, may be at distinct locations. We have found that these sites may vary during ablation (a new site becomes the area of EA as the previous site is ablated, and so on) and suggest that it is important to ablate these sites in addition to the intervening region in between.

Our findings appear to contrast those reported by Tai et al., 13 who also used non-contact mapping to elucidate the tachycardia circuit in atypical RA flutters. They found that over half of the cases (8 of 15) consisted of double-loop re-entry (figure-of-eight re-entry) involving the upper loop and RAFW or lower loop; we did not find double-loop re-entry in any of our patients. The reason for this disparity may in part be due to differing patient populations. The majority of patients in our study had structural heart disease (acquired or congenital), whereas only one-third of patients in the study reported by Tai et al. had cardiovascular disease. In addition, nine patients in our study had previously undergone ablation of the CTI for typical AFL. Consequently, involvement of the lower loop (either alone or as part of a double-loop re-entrant circuit) would not have been possible in these patients.

Limitations of the study

We have reported on a heterogenous group of patients with RA tachycardias, with or without CHD, and used general principles from information obtained using non-contact mapping to guide ablation strategy. These principles may not necessarily be useful in all patients with complex RA tachycardias and ideally, a prospective study would be required to determine applicability of the strategies discussed. None of our patients had previously undergone the Fontan procedure and as such, we cannot extrapolate our data to such patients or those with markedly dilated RA.

Conclusions

Non-contact mapping can be useful in identifying the tachycardia mechanism of atypical re-entrant and focal RA tachycardias in patients with and without CHD. Ablation of these tachycardias between areas of scar/low voltage or at areas of EA and BO regions in re-entrant or focal tachycardias, respectively, allows for successful ablation of this challenging group of tachycardias. Future studies directly comparing non-contact mapping with conventional or electro-anatomical mapping are required to determine whether acute and long-term success rates are superior with any method in these patients.

Acknowledgements

Patient 13 in Table 2 was treated under the care of Dr Edward Rowland and Patients 14 and 22 under the care of Dr Vince Paul. The rest of the patients in this report were under the care of Dr David Ward.

Conflict of interest: none declared.
References


