High incidence of acute sub-clinical circumflex artery ‘injury’ following mitral isthmus ablation

Kelvin C.K. Wong, Chris Lim, Praveen P. Sadarmin, Michael Jones, Norman Qureshi, Joe De Bono, Kim Rajappan, Yaver Bashir, and Timothy R. Betts*

Oxford Heart Centre, John Radcliffe Hospital NHS Trust, Oxford OX3 9DU, UK

Received 22 August 2010; revised 11 March 2011; accepted 25 March 2011; online publish-ahead-of-print 29 April 2011

Aims Mitral isthmus (MI) ablation is technically challenging, requiring long endocardial ablation times and frequently coronary sinus (CS) ablation. The circumflex artery lies in the epicardium in close proximity to the CS and the mitral annulus and may potentially be injured during radiofrequency ablation.

Methods and results Fifty-four patients underwent catheter ablation procedures that included MI ablation for treatment of atrial fibrillation. Irrigated ablation catheters were used with the following settings: endocardial surface (max power: 40/50 W at the annular end; max temperature: 48°C); CS (max power: 25/30 W; max temperature: 48°C). Coronary angiography was performed pre- and post-ablation and analysed by two cardiologists with quantitative coronary angiography. Mitral isthmus block was achieved in 89% of patients (60% required CS ablation). Fifteen patients (28%) had angiographic changes following ablation: eight had mid-circumflex narrowing only, one had circumflex and obtuse marginal (OM) artery narrowing, one had OM narrowing only, and five had distal circumflex occlusion/narrowing. Five patients had significant narrowing (50–84%), which resolved with intracoronary glycerine trinitrate. Fourteen (93%) of the patients with circumflex ‘injury’ had CS ablation and a longer mean CS ablation time (5.0 ± 3.0 vs. 2.6 ± 3.3 min, P = 0.03). Patients with distal circumflex occlusion had significantly smaller vessel diameter (1.0 ± 0.1 vs. 2.1 ± 0.2 mm, P = 0.03). A shorter distance between the circumflex and the CS was also associated with circumflex ‘injury’ (3.2 ± 1.9 vs. 5.6 ± 3.2 mm, P = 0.04). There were no electrocardiographic or echocardiographic abnormalities and no angina symptoms during follow-up.

Conclusion Acute sub-clinical circumflex ‘injury’ following MI ablation is not uncommon. Ablation within the CS, proximity of the circumflex and the CS, and a small distal circumflex were risk factors for ‘injury’.

Keywords Atrial fibrillation • Radiofrequency ablation • Mitral isthmus • Coronary artery injury • Complication

Introduction Mitral isthmus (MI) ablation in addition to pulmonary vein isolation can improve outcomes in the treatment of both paroxysmal and persistent atrial fibrillation (AF).1–5 Mitral isthmus ablation was ultimately required in 86% of patients who underwent the Bordeaux stepwise approach for catheter ablation of persistent AF.5 Mitral isthmus ablation may modify the left atrial substrate and prevent macro-re-entrant tachycardia,7,8 but an incomplete line may predispose to arrhythmias.6,9–13

It is widely recognized that achieving a MI block is challenging, often requiring extensive ablation with irrigation catheters, the use of high ablation power, and epicardial ablation from within the coronary sinus (CS) in the majority of patients.1,2 The circumflex artery lies on the epicardial surface of the atrioventricular groove and is in close proximity to the CS and the endocardial aspect of the MI area.4 Isolated case reports have described acute and chronic circumflex injury following radiofrequency ablation at the MI area.15–17 Previous retrospective and prospective registries have reported very low incidences of coronary injury following radiofrequency ablation.18–20 Two studies, which actively sought evidence for coronary injury after ablation, have reported incidences in the order of 1%.21,22

In view of the substantial ablation performed at the mitral annulus and the CS during MI ablation, this study sought to investigate the incidence and risk factors for acute circumflex injury.
Methods

Study population
This was a prospective, single-centre study of consecutive patients who underwent first or redo catheter ablation procedures that included MI ablation for treatment of persistent or long-duration paroxysmal (episodes lasting >48 h) AF. Circumferential pulmonary vein isolation, MI, and roof line ablations formed the ablation strategy for persistent AF. Mitral isthmus ablation was also performed if peri-mitral flutter was induced during repeat ablation procedure for paroxysmal AF. Previous MI ablation was an exclusion criterion. Written informed consent was obtained from each participant prior to study inclusion. The study protocol was approved by the Oxford Research Ethics Committee.

Electrophysiological study
The procedures were performed with the patients in the post-absorptive state under general anaesthesia or conscious sedation. All anti-arrhythmic drugs except amiodarone were discontinued for at least 5 half-life periods prior to the procedure. All patients were anticoagulated for at least 1 month and had transoesophageal echocardiography to exclude left atrial appendage (LAA) thrombus. Access was obtained via the left femoral artery and both femoral veins. A steerable decapolar catheter (Dynamic XT, Bard) was positioned within the CS. A quadripolar catheter (Josephson, Bard) was positioned in the aortic root as a positional reference catheter. Double trans-septal punctures were performed for introduction of an F-curve irrigated-tip catheter (Thermocool, Biosense Webster) through a steerable sheath (Agilis, St Jude Medical) and a pulmonary vein mapping catheter (Optima, St Jude Medical) through a fixed curve sheath (SL0, St Jude Medical). A heparin bolus and infusion was used to maintain the activated clotting time between 300 and 350 s throughout the procedure. Electroanatomical geometry of the left atrial chamber was created (EnSite NavX, St. Jude Medical) and circumferential ablation was initially performed to isolate the pulmonary veins in pairs as described before.23,24 Roof line ablation was performed by creation of a contiguous line of ablation lesions from the left to right superior pulmonary veins over the most cranial portion of the left atrial roof. Radiofrequency energy was delivered with powers of 30 W, temperature limited to 48°C, and irrigation rates of 17–30 mL/min. If patients were in AF, they were electrically cardioverted to sinus rhythm. Mitral isthmus ablation was performed during LAA pacing by gradually moving the ablation from a lateral position on the mitral valve annulus to the isolated left-sided pulmonary veins (maximum powers of 40 or 50 W at the annular end and 30 or 40 W at the venous end of the line; maximum temperature: 48°C; irrigation rate: 17–30 mL/min). Ablation was targeted anatomically as a line across the isthmus at sites with atrial activation earlier than electrograms recorded in the distal CS. If isthmus block was not achieved and no further atrial electrograms could be identified along the ablation line, ablation was then performed epicardially in the distal CS (maximum powers of 25–30 W; maximum temperature: 48°C; irrigation rate: 17 mL/min). To position the ablation catheter in the distal CS and great cardiac vein, the Agilis sheath was positioned at the distal CS (maximum powers of 25–30 W; maximum temperature: ≏10 mm to encompass the area overlying the previously ablated endocardial aspect of the MI. A change to a proximal to distal CS activation pattern during LAA pacing was used to signify MI block as previously described (Figure 1).25 The end point of linear ablation was complete block. This was confirmed with activation maps, mapping for widely spaced double potentials along the ablation line and differential pacing. Protamine was given at the end of the procedure to reverse the effects of heparin. A 6 Fr angioseal was deployed in the femoral artery in all patients. All other sheaths were removed, and manual pressure was used to achieve haemostasis.

Study protocol
The ablation catheter (F-curve Thermocool, Biosense Webster) or the steerable decapolar catheter (Dynamic XT, Bard) was positioned in the distal CS prior to coronary angiography. Pre-ablation left coronary angiography was performed at the beginning of the procedure. Post-ablation angiography was performed within 30 min of MI ablation. They were performed in the following three caudal views (20–30°): left anterior oblique (LAO 30–45°), anteroposterior (AP, 0), and right anterior oblique (RAO 30–45°). Levoephase images were also obtained to show the CS venogram. In cases where the levoephase images were not clear, CS venograms were obtained after balloon occlusion (Arrow International, Inc.). This was analysed off-line by two independent observers who were blinded to the procedures.

Quantitative coronary angiography
Coronary angiograms were screened visually for any changes. Quantitative coronary angiography (QCA) was then performed on the circumflex artery (proximal, mid, and distal segments), the proximal left anterior descending (LAD) artery, and the proximal obtuse marginal (OM) branch. This allows for measurement of the mean luminal diameter (LD) over a 1 cm segment. In cases of narrowing, the minimal MLD were measured and the percentage of narrowing was determined. The shortest distance of the circumflex artery to the catheter positioned in the CS (at 3 and 4 o’clock on the LAO view) was measured in all three caudal views in diastole and the maximal distance was taken as the ‘actual distance’ (Figure 2). The distance between the site of circumflex ‘injury’ and CS stenosis was also determined.

Follow-up
Post-procedural echocardiogram was performed on all patients after the procedure to exclude pericardial effusion. Twelve-lead electrocardiograms were obtained immediately post-procedure and the following day. Patients were followed up in the arrhythmia clinic at 3 and 12 months with symptom assessment, 12-lead electrocardiographs, and ambulatory monitoring as per recommendations from the HRA/EHRA/ECAS consensus guidelines.26 Repeat ablation was recommended if there was symptomatic atrial arrhythmia after the 3-month blanking period. Long-term follow-up data were not part of the study protocol.

Statistical analysis
Continuous variables are expressed as mean ± SD unless otherwise stated. Normally distributed parameters were compared using unpaired student t-test. The Mann–Whitney U-test was used for data which were not normally distributed. The statistical software used was GraphPad Prism version 4. All statistical tests were two-sided and statistical significance was established at \( P < 0.05 \).
Results

Baseline characteristics are summarized as follows: mean age was 60 ± 10 years, 72% were males, 78% had persistent AF, 39% had other cardiovascular disease, 15% had impaired left ventricular systolic function, median CHADS2 score was 0 (range 0–3), and mean left atrial diameter was 44 ± 6 mm. Forty-three of 54 patients (80%) were undergoing first time ablation procedures. The mean total procedure time and screening times were 175 ± 35 and 56 ± 35 min, respectively. Circumferential pulmonary vein isolation was achieved in all patients.

Mitral isthmus block was achieved in 89% of patients [30 of 50 patients (60%) required CS ablation to achieve block]. Mean MI ablation and procedure times were 12.7 ± 6 min (endocardium: 9.4 ± 3.4 min; CS: 3.2 ± 3.4 min) and 26.7 ± 18.3 min, respectively.

Following ablation, 15 out of 54 patients (28%) had focal angiographic changes in the circumflex artery or the OM artery (Figure 3): eight patients had mid-circumflex artery narrowing only (Figure 4A), one patient had both mid-circumflex and OM narrowing, one patient had OM narrowing only, and five patients had small distal circumflex narrowing or occlusion (Figure 4B). Five patients had what were deemed as significant narrowing (50–84%). All these narrowing resolved with 300 mcg intracoronary glycerine trinitrate (GTN) boluses (Figure 5). Quantitative coronary angiography analysis is summarized in Table 1.

Ablation within the CS was a strong predictor of acute circumflex 'injury'. Only 1 of 15 cases (7%) occurred with endocardial ablation only. In this case, there was occlusion of a small distal circumflex branch (ablation duration: 547 s; max power: 40 W at the mitral annulus). Patients with circumflex 'injury' had a significantly longer mean CS ablation time (5.0 ± 3.0 vs. 2.6 ± 3.3 min, \( P = 0.03 \)). They also tended to have longer mean total ablation time although that did not reach significance (15.4 ± 3.7 vs. 11.7 ± 6.4 min, \( P = 0.07 \)).

Eighteen out of 54 patients (33%) had a maximum power of 50 W delivered at the annular end of the MI line. Twelve out of 36 patients (33%) who had ablation in the CS had a maximum delivered power of 30 W. The maximum power delivered during
Ml ablation (endocardial annular end: 40 W or 50 W; CS: 25 W or 30 W) did not predict circumflex ‘injury’.

Mitral isthmus ablation was performed nearer the 3 o’clock position (LAO projection) in 31 patients (57%). The ablation position (3 or 4 o’clock) was not associated with circumflex ‘injury’.

Other ablation parameters such as mean impedance did not predict circumflex injury (Table 2).

The pre-ablation incidence of coronary artery disease affecting the circumflex artery was 31% (17 of 54 patients). Most of them were irregularities or mild stenoses involving the proximal circumflex artery (<30%) except for two patients with moderate stenosis (≤50%) seen in the OM branch. The presence of underlying circumflex disease did not predict circumflex ‘injury’, although both patients with moderate disease of the OM artery did have evidence of distal circumflex ‘injury’ following ablation.

The ‘actual distance’ of the circumflex to the catheter positioned in the CS at 4 o’clock was significantly shorter in patients who had circumflex ‘injury’ (3.2 ± 1.9 vs. 5.6 ± 3.2 mm, P = 0.04).

**Figure 2** Top panel, fluoroscopic pictures in right anterior oblique, anteroposterior, and left anterior oblique views showing the relationship between the ablation catheter in the coronary sinus and the circumflex artery. The dotted circle represents the mitral annulus with the arrow pointing at 3 o’clock position. ‘Actual’ distance at 3 o’clock was measured in the right anterior oblique view (longest of the three views). Bottom panel, Fluoroscopic pictures in right anterior oblique, anteroposterior, and left anterior oblique views showing the relationship between the ablation catheter in the coronary sinus and the circumflex artery at the 4 o’clock position. ‘Actual’ distance at 4 o’clock was measured in the left anterior oblique view (longest of the three views). See text for further explanation.

**Figure 3** Pie chart showing the incidence of acute circumflex artery injury. Column chart depicts affected segments. Cx indicates circumflex artery; OM, obtuse marginal artery.
**Figure 4** (A) Coronary angiogram showing mid-circumflex ‘injury’. Ablation catheter had been withdrawn from original ablation site to help visualize circumflex artery; (B) coronary angiogram showing distal circumflex occlusion post-ablation. Black arrows indicate affected artery segments.

**Figure 5** Coronary angiograms showing two examples of circumflex artery narrowing (A and B) resolving with intracoronary glycerine trinitrate (GTN). Black arrows indicate affected artery segments.
In patients with distal circumflex occlusion, the mean LD was significantly smaller (1.0 ± 0.1 vs. 2.1 ± 0.2 mm, \(P = 0.03\)). Other anatomical characteristics of the circumflex artery and patient clinical features did not predict circumflex ‘injury’ (Table 2).

There were no changes detected on electrocardiograms and echocardiograms following the ablation procedures. One patient developed a haematoma which did not require intervention and one patient had a pseudoaneurysm requiring thrombin injection. There was no tamponade or thrombo-embolic event.

**Medium-term follow-up**

After a mean follow-up of 15 ± 3 months, there was no reported angina. Thirteen patients had repeat angiography at a mean of 8 ± 3 months after the index procedure. Five of these patients had acute circumflex ‘injury’ (mid-circumflex narrowing ranging from 40 to 60%) during the index procedure and their repeat angiograms did not demonstrate any abnormality. There were also no new angiographic changes in the remaining eight patients.

**Discussion**

From prospective and retrospective registries, the reported incidences of coronary artery injury after all catheter ablation procedures are 0.03% in children \(^1\) and 0.06–0.1% in adults. \(^18,20\)

This is likely to be an underestimate as acute transient injury of the coronary arteries may not result in symptoms or electrocardiographic changes and may be missed. \(^27,28\) However, there may be long-term sequelae following such radiofrequency-induced injury. \(^17,29,30\)

Two prospective studies which systematically investigated the incidence of coronary artery injury after radiofrequency ablation reported the incidence to be 0.94% in children \(^22\) and 1.3% in adults. \(^19\) In the first study, two moderate coronary stenoses were observed 30 min after ablation, but GTN was not used acutely and no further angiography was performed at follow-up. Hence, the authors were unable to comment on the mechanism of injury. In the other study, acute distal circumflex artery occlusion was secondary to spasm as repeat angiography was normal when performed 6 weeks later. These studies were based mainly on accessory pathway and atrioventricular nodal re-entrant tachycardia ablations, usually requiring limited ablation with non-irrigated catheters via an endocardial approach.

It is well recognized that MI ablation is technically challenging and involves extensive ablation with an irrigated-tip catheter, frequently in the epicardium via the CS. One may expect the incidence of coronary artery injury to be even higher after MI ablation. The finding of a high incidence of sub-clinical circumflex artery injury (28%) after MI ablation is not entirely surprising.

The risk factors which contribute to coronary artery injury after ablation are not well defined. In our study, ablation in the CS, longer CS ablation duration, and close proximity between the circumflex and the CS predispose to circumflex injury after MI ablation. While we cannot conclude definitively that circumflex injury occurred during CS ablation and not during the preceding endocardial ablation, it is likely that the CS ablation led to circumflex injury. This may be explained by the significantly closer relationship between the circumflex artery and the CS when compared with left atrial endocardium. \(^31\) There was a close relationship between the site of circumflex artery ‘injury’ and CS stenosis (Figure 6 and see Supplementary material online, Figures S1 and S2). A small-calibre distal circumflex artery is also associated with a higher risk of distal circumflex occlusion (Figure 4B). This finding is consistent with an in vivo study using a porcine model, which demonstrated a higher percentage of hepatic vein injury in veins smaller than 3 mm due to the absence of a heat-sink effect. \(^32\) This would support the hypothesis that the heat-sink effect protects the coronary artery from the thermal effects of radiofrequency ablation by convective cooling and explain the low incidence of coronary artery damage despite ablation at close proximity.

Maximum ablation powers limited to 40 W or 50 W at the annulus endocardially and 25 W or 30 W in the CS did not appear to affect the risk of circumflex artery (Cx) injury. In our cases, the use of 40 W or 50 W was only limited to the annular end of the line and was reduced to 30 W and only occasionally 40 W at the venous end. While these powers may be considered to be high, many published series have reported maximum power delivered at the endocardium of between 40 W and 45 W and the CS of between 20 W and 30 W. \(^1,2,4,5,12,13,33,34\) Few studies reported a maximum power delivered at the endocardium of <40 W. \(^6,9,35\) Coronary sinus ablation was not performed in two studies, and the reported success rate was only 31% and 73%. \(^35\) Jais et al. \(^1\) reported two cases of cardiac tamponade in 136 procedures felt to be secondary to the use of ablation power >50 W endocardially. The study recommended limiting the power to 42 W at the endocardium. There was no cardiac tamponade observed in our study. In our experience, these ‘high’ powers are often necessary for achieving MI block and are likely to have contributed to the relatively high success this study achieved.

<table>
<thead>
<tr>
<th>Artery segment</th>
<th>Number</th>
<th>Pre-ablation mean LD (mm)</th>
<th>Post-ablation mean minimal LD (mm)</th>
<th>(P)-value</th>
<th>Percentage narrowing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mid-Cx</td>
<td>9</td>
<td>2.6 ± 0.9</td>
<td>1.2 ± 0.6</td>
<td>0.0004</td>
<td>37–84</td>
</tr>
<tr>
<td>OM</td>
<td>2</td>
<td>2.7 ± 0.7</td>
<td>1.8 ± 0.1</td>
<td>0.22</td>
<td>37–48</td>
</tr>
<tr>
<td>Distal Cx</td>
<td>5</td>
<td>1.4 ± 0.8</td>
<td>0.3 ± 0.6</td>
<td>0.04</td>
<td>55–100</td>
</tr>
<tr>
<td>Prox Cx</td>
<td>15</td>
<td>3.4 ± 0.9</td>
<td>3.5 ± 0.8</td>
<td>0.13</td>
<td></td>
</tr>
<tr>
<td>Prox LAD</td>
<td>15</td>
<td>3.1 ± 0.8</td>
<td>3.2 ± 0.9</td>
<td>0.57</td>
<td></td>
</tr>
</tbody>
</table>

Cx, circumflex artery; LAD, left anterior descending; LD, luminal diameter; Prox, proximal.
Incidence of acute sub-clinical Cx 'injury' following MI ablation

Our mean MI ablation time of 12.7 ± 6 min (endocardium: 9.4 ± 3.4 min; CS: 3.2 ± 3.4 min) was shorter when compared with those published in the literature, which were mainly in excess of 15 min.1,2,4,6,9,12,31 The frequency of ablation within the CS (60%) was also lower than in many studies.1,2,9,12,13,33,34 Therefore, it is very unlikely that our ablation technique and parameters have contributed to the high incidence of circumflex 'injury'.

A recent study suggested that radiofrequency ablation causing coronary injury resulted in impedance drop followed by an impedance rise.29 This was not observed in our series.

Our study demonstrated a close relationship between the circumflex artery and the usual site of ablation in the CS during MI ablation (Figure 7): 3.4 ± 1.9 mm at 3 o'clock and 5.1 ± 3.0 mm at 4 o'clock. This finding is consistent with previous studies.34,35 Hasdemir et al.36 found that the circumflex artery was <2 mm from the CS catheter at the lateral and anterolateral mitral annulus in 24% of patients, and Wittkampf et al.14 suggested that the risk of damage to the circumflex artery is increased with more ‘distal’ ablation. In our study, MI ablation at the 3 o’clock position on the mitral annulus (LAO view) was not associated with a higher risk of circumflex injury.

Five out of 15 patients (33%) had significant stenosis (>50%) which resolved with intracoronary GTN, suggesting that the main underlying mechanism may be spasm as described previously.37–39 While this may represent only transient thermal irritability with no longer term consequences, it is clear that vasospasm may not be an entirely benign process.40 In addition, there may be an acute inflammatory component which does not respond to nitrates.29 Animal studies have shown that luminal narrowing may be secondary to acute oedema and wall thickening which may then resolve.41 Subsequent maturation of the inflammatory response through medial necrosis and intimal hyperplasia may be the reason for delayed presentation and long-term coronary stenosis in animal models.42–44 To date, the mechanism of coronary artery injury following radiofrequency ablation in humans is not fully understood. Further studies are needed to elucidate the mechanisms underlying coronary artery injury following ablation and to determine the long-term outcome of these patients.

Our findings are significantly different from the original series of MI ablation reported by Jais et al.1 In that study, no detectable abnormality was reported in coronary angiograms performed after ablation in a subset of 15 patients. This may be explained by our systematic approach to actively look for angiographic changes by comparing pre- and post-ablation coronary angiograms. The techniques and the ablation parameters (power and temperature) used in both studies were comparable except for the use of a deflectable long sheath (Agilis, St Jude Medical) in our study.

While the circumflex ‘injury’ reported in our study was sub-clinical and transient, there is a potential for more severe damage to the circumflex artery as reported in the literature.15,16 In addition, this transient ‘injury’ may have longer term consequences. Consequently, these findings should encourage operators to exercise caution during MI ablation. Coupled with recent concerns that MI ablation may predispose to peri-mitral flutters even when block was achieved acutely (presumed to be secondary
to the resumption of conduction across the line), operators may need to reserve MI ablation for patients who would benefit most from it, i.e. patients with peri-mitral flutter and persistent or long-lasting persistent AF.

Ablation of an ‘alternative MI’, an alternative energy source such as cryo-ablation, and improving techniques to achieve MI block endocardially should be considered.

Study limitations
While the occurrence of circumflex injury was strongly associated with CS ablation, it could have happened during endocardial ablation. As coronary angiography was only performed after the completion of the ablation procedure, we cannot be certain when the ‘injury’ occurred. This study was designed to investigate acute changes in the circumflex artery using coronary angiography. Previous animal models have demonstrated significant intimal hyperplasia despite normal angiographic appearances. Further imaging with optical coherence tomography or intravascular ultrasound and functional vasomotor testing may have added additional information. Biomarker testing was not performed as there is already substantial myocardial damage after left atrial ablation which would make interpretation of cardiac enzymes difficult. It would be advantageous to have longer term follow-up angiography to check if these acute changes lead to further chronic changes.

Conclusion
There is a high incidence of sub-clinical acute circumflex ‘injury’ following MI ablation. Ablation in the CS, long-CS ablation times, proximity of the circumflex artery to the CS, and a small distal circumflex diameter are significant risk factors. Operators should exercise caution during MI ablation and consider performing coronary angiography in patients who have had extensive ablation especially in the CS. Further studies are needed to elucidate the

Figure 6 Fluoroscopic pictures (in right anterior oblique, anteroposterior, and left anterior oblique views) showing a close relationship between site of circumflex ‘injury’ (mid-circumflex artery narrowing just before bifurcation indicated by arrows in the top panel) and the site of CS stenosis (bottom panel) in all three views. Coronary sinus venograms were obtained with balloon occlusion as the levophase pictures were not clear enough.

Figure 7 Fluoroscopic views (right anterior oblique, anteroposterior, and left anterior oblique) showing the close proximity of the ablation catheter (positioned in coronary sinus) and the circumflex artery taken pre-ablation.
mechanism underlying acute coronary artery injury after radiofrequency ablation and its longer term sequelae.

Supplementary material
Supplementary material is available at European Heart Journal online.

Funding
We would like to thank Biomedical Research Council, St Jude Medical, and Medtronic for their financial support.

Conflict of interest: K.C.K.W. is in receipt of an unrestricted educational grant from Medtronic and St Jude Medical.

References


34. Sawhney N, Anousheh R, Chen W, Feld GK. Circumferential pulmonary vein ablation with additional linear ablation results in an increased incidence of left atrial flutter compared with segmental pulmonary vein isolation as an initial approach to ablation of paroxysmal atrial fibrillation. Circ Arrhythm Electrophysiol 2010;3:243–248.


