Coronary Artery Injury Due to Catheter Ablation in Adults
Presentations and Outcomes
Kurt C. Roberts-Thomson, MBBS, PhD; Daniel Steven, MD; Jens Seiler, MD, PhD; Keiichi Inada, MD; Bruce A. Koplan, MD; Usha B. Tedrow, MD, MSc; Laurence M. Epstein, MD; William G. Stevenson, MD

Background—Currently, only anecdotal information exists on the presentation and outcome of coronary arterial injury after ablation procedures.

Methods and Results—Four patients who sustained coronary artery injury of a cohort of patients undergoing 4655 consecutive ablation procedures (0.09%) are described. The patients' mean age was 45±11 years, and 1.8±0.5 prior ablation attempts had been unsuccessful. Coronary injury occurred from epicardial ventricular tachycardia ablation in 2 patients (irrigated radiofrequency ablation in one and cryoablation in the other) and ablation within the middle cardiac vein with irrigated radiofrequency in 2 patients. All involved branches of the right coronary artery. Acute occlusion presenting with ST-segment elevation immediately after ablation was recognized during the procedure in 2 cases. Occlusion failed to respond to nitroglycerin or balloon dilation, and stenting was required in both cases. Acute myocardial infarction occurred 2 weeks after epicardial ablation as a result of occlusion of a right ventricular branch of the right coronary artery giving rise to the posterior descending coronary artery in 1 patient. A moderate asymptomatic stenosis was seen on angiography after epicardial cryoablation in 1 patient. All patients recovered and remained asymptomatic from the coronary injury and arrhythmias during 37±53 months of follow-up.

Conclusions—Coronary arterial injury after ablation procedures is rare. It may present acutely or several weeks after an ablation procedure. Acute occlusion appears to require coronary stenting. Unanticipated anatomic variations can predispose to coronary injury. (Circulation. 2009;120:1465-1473.)

Key Words: ablation ■ complications ■ coronary disease

Radiofrequency ablation procedures are safe and effective therapies for the treatment of many tachyarrhythmias,1,2 Although a risk of coronary injury is recognized, it has been reported rarely, despite ablation being performed regularly in close proximity to coronary arteries. This study reviews all recognized coronary injuries at our institution between 1998 and 2008 to describe the variation in the presentations and outcomes.

Clinical Perspective on p 1473

Methods
Between 1998 and 2008, 4 patients (0.09%) of a cohort of patients undergoing 4655 procedures (131 epicardial procedures) had a coronary injury secondary to ablation. Because of changes in data storage over time, details of ablations in relation to cardiac venous structures were not retrospectively obtainable for all patients without coronary injury. To provide some perspective in regard to the range of cases and frequency of coronary venous ablation, data from 1837 consecutive cases of a single operator were reviewed. These comprised ablation for atrial fibrillation (18%), macroreentrant or focal atrial tachycardia (16%), atrioventricular nodal tachycardia (11%), accessory pathways (10%), idiopathic ventricular tachycardia (VT) (9%), VT related to coronary artery disease (16%), VT related to nonischemic cardiomyopathy (18%), and other arrhythmias (2%). During these procedures, ablation was performed at the ostium of the middle cardiac vein (MCV) in 13 procedures (0.3%) and in the body of the coronary sinus in 114 procedures (2.4%).

The patient characteristics are presented in Table 1. The 3 men and 1 woman had a mean age of 45±11 years and had undergone a mean of 1.8±0.5 prior procedures. Two patients were undergoing ablation for scar-related VT: 1 patient for atrioventricular reentrant tachycardia and 1 patient for typical atrial flutter. None of the patients who had coronary injury had undergone stress testing before the ablation procedure.

Electrophysiology Procedure
All patients gave written informed consent. The procedures were performed under conscious sedation with fentanyl and midazolam. Electroanatomic mapping (CARTO, Biosense-Webster, Diamond Bar, Calif) was performed in 3 cases.

Results
In 2 of the 4 cases, coronary angiography was performed before the commencement of ablation. All cases involved
injury to branches of the right coronary artery (RCA), the posterolateral branch in 3 patients, and an unusual anatomic variant of a right ventricular branch in 1 patient. Three cases occurred after the application of radiofrequency and 1 after cryoablation. All of the cases after radiofrequency ablation developed complete occlusion of the vessel, 2 immediately after the application of radiofrequency and 1 at 2 weeks after the procedure. The ablation parameters are presented in Table 2.

### Case 1
This patient was referred for ablation of cavotricuspid isthmus–dependent atrial flutter with the use of a 4-mm internally irrigated catheter (Chilli, Boston Scientific) after 2 prior radiofrequency ablation attempts with solid-tip catheters failed. The maximum power was set at 50 W. During the final application, the catheter slipped into the MCV, an impedance rise was noted, and radiofrequency was discontinued. However, radiofrequency was applied in the MCV for several seconds. The patient experienced some chest discomfort during ablation, but after this last application the discomfort continued. ST-segment elevation was noted in the inferior leads. Immediate coronary angiography (Figure 1) demonstrated occlusion of the posterolateral branch of the RCA at the MCV. Intracoronary nitroglycerin was administered with no effect. A guidewire was inserted, and the vessel was dilated with the use of a 2.0×20-mm balloon but closed repeatedly on deflation. A 3.5×16-mm NIR stent (Scimed, Maple Grove, Minn) was placed, restoring thrombolysis in Myocardial Infarction class 3 flow. There was no rise in creatine kinase or troponin. Bidirectional cavotricuspid isthmus block was demonstrated at the end of the case.

Seven months later, the patient presented with chest pain, and repeat angiography revealed an 80% in-stent restenosis. This was balloon dilated with a good immediate result, but pain recurred 3 days later, and angiography demonstrated a hazy 40% stenosis within the stent. This was ballooned and stented with no residual stenosis. He has had recurrent left atrial flutter and atrial fibrillation but no further episodes of typical flutter over a 10-year follow-up. In addition, follow-up exercise testing has demonstrated no recurrent ischemia and preserved left ventricular function without wall motion abnormality.

### Case 2
This patient had previously experienced an unsuccessful ablation of a posteroseptal accessory pathway causing orthodromic atrioventricular reentrant tachycardia. During this procedure, earliest retrograde atrial activation was at the ostium of the MCV preceded by a sharp potential consistent with an accessory pathway. Angiography of the coronary sinus identified a small diverticulum at this site (Figure 2). Coronary angiography was then performed and demonstrated that the site of earliest atrial activation was between the posterolateral and posterior descending branches of the RCA (Figure 2). Radiofrequency energy was applied with a 3.5-mm externally irrigated catheter (Navistar, Biosense Webster) at a maximum power of 30 W. Pathway conduction disappeared but then recovered. Ablation was then repeated with 35 W with an irradiation rate of 17 mL/min for 90 seconds, abolishing pathway conduction. Immediately after termination of radiofrequency, ST-segment elevation in the inferior ECG leads was noted. Repeat coronary angiography (Figure 3) showed occlusion of the posterolateral branch of the RCA. Intracoronary nitroglycerin was given with no response. A guidewire was inserted through the occlusion, and dilation with a 2.0×12-mm Sprinter balloon (Medtronic, Minneapolis, Minn) was performed, but the vessel immediately reoccluded. A 3.0×23-mm Vision stent (Guidant, Santa Clara, Calif) was inserted, and additional intracoronary nitroglycerin was given, but distal flow remained poor despite the establishment of flow within 29 minutes after termination of radiofrequency. Two additional Minivision stents (Guidant) were placed distally, but flow remained only Thrombolysis in Myocardial Infarction class 1. Aspirin, clopidogrel, and eptifibatide were given after the procedure. There was a rise in creatine kinase to a peak of 910 U/L (normal range, 61 to 224 U/L). A subsequent echocardiogram demonstrated a left ventricular ejection fraction of 60% with a small region of

### Table 1. Patient Characteristics

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age, y</th>
<th>Sex</th>
<th>Structural Heart Disease</th>
<th>Preablation LVEF, %</th>
<th>Arrhythmia</th>
<th>No. of Failed Ablations</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>42</td>
<td>Male</td>
<td>None</td>
<td>55</td>
<td>Typical flutter</td>
<td>2</td>
</tr>
<tr>
<td>2</td>
<td>50</td>
<td>Female</td>
<td>None</td>
<td>55</td>
<td>AVRT</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>55</td>
<td>Male</td>
<td>Unclear</td>
<td>60</td>
<td>VT</td>
<td>2</td>
</tr>
<tr>
<td>4</td>
<td>30</td>
<td>Male</td>
<td>ARVC</td>
<td>60</td>
<td>VT</td>
<td>2</td>
</tr>
</tbody>
</table>

LVEF indicates left ventricular ejection fraction; ARVC, arrhythmogenic right ventricular cardiomyopathy; AVRT, atrioventricular reentrant tachycardia; and VT, ventricular tachycardia.

### Table 2. Ablation Parameters

<table>
<thead>
<tr>
<th>Patient</th>
<th>Catheter</th>
<th>Maximum Power, W</th>
<th>Maximum Temperature, °C</th>
<th>Site of Ablation</th>
<th>Vessel</th>
<th>Preablation Angiography</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>4 mm internally irrigated</td>
<td>50</td>
<td>42</td>
<td>CTI/MCV</td>
<td>Posterolateral branch of RCA</td>
<td>No</td>
</tr>
<tr>
<td>2</td>
<td>3.5 mm externally irrigated</td>
<td>35</td>
<td>47</td>
<td>MCV</td>
<td>Posterolateral branch of RCA</td>
<td>Yes</td>
</tr>
<tr>
<td>3</td>
<td>6 mm cryocatheter</td>
<td>NA</td>
<td>NA</td>
<td>Epicardial LV</td>
<td>Posterolateral branch of RCA</td>
<td>Yes</td>
</tr>
<tr>
<td>4</td>
<td>3.5 mm externally irrigated</td>
<td>40</td>
<td>52</td>
<td>Epicardial RV</td>
<td>Marginal branch of RCA</td>
<td>No</td>
</tr>
</tbody>
</table>

CTI indicates cavotricuspid isthmus; NA, not applicable; MCV, middle cardiac vein; LV, left ventricle; and RV, right ventricle.
inferior wall hypokinesis. After 8 months of follow-up, the patient has had no further arrhythmic or ischemic symptoms. In both cases 1 and 2, the stenotic region had a smooth appearance, without staining to suggest thrombus.

Case 3

Epicardial mapping and ablation were performed in a 55-year-old man with recurrent VT causing implantable cardioverter-defibrillator shocks despite therapy with amiodarone, mexiletine, and metoprolol. Two prior endocardial ablation attempts had been unsuccessful. No structural heart disease was evident on echocardiography, and coronary angiography was unremarkable. Four VTs with a similar morphology ranging in cycle length from 240 to 360 ms were inducible (Figure 4). Percutaneous epicardial access and mapping were performed with the use of previously described techniques.3 A low-voltage (<1.5 mV) region was evident in the posterobasal area of the left ventricle, with middiastolic activity and entrainment features of a reentry circuit site (Figure 4). Coronary angiography demonstrated a large dominant RCA with the posterolateral branch close to the reentry circuit site. An isthmus site was identified at the superior aspect of the low-voltage region, away from this vessel, and irrigated radiofrequency was applied at up to 45 W with termination of VT. The superior and inferior aspects of the low-voltage region were then targeted with a substrate ablation approach. This altered the morphology of inducible VTs, abolishing some of the VTs, but VT with a cycle length of 310 ms remained inducible. Ablation was then performed at the adjacent endocardial sites but also failed to terminate tachycardia. Because the best site had been epicardial, a 6-mm cryocatheter (Cryocath Technologies, Montreal, Canada) was placed in the region of epicardial scarring at the margin of the isthmus site that was estimated to be 5 mm from the posterolateral branch of the RCA. Cryoablation was performed for 4 minutes at −80°C but failed to terminate VT.

Figure 1. Twelve-lead ECGs and coronary angiograms from case 1. A, ECG at the end of the ablation and then 3 minutes after ablation. Note the ST-segment elevation, which is particularly pronounced in lead III. B and C, Occlusion of the posterolateral branch of the RCA (arrow) before (B) and after (C) coronary stenting.
In consideration of another ablation attempt, repeat angiography was performed and showed a 50% to 60% stenosis in the posterolateral branch near the site of ablation (Figure 4). The procedure was terminated, although the morphology of 1 VT remained inducible. The patient remained asymptomatic. On exercise testing 1 week later, he had no symptoms with good exercise capacity. Sestamibi imaging showed a small fixed defect at the posterobasal left ventricle, consistent with the low-voltage region observed on electroanatomic mapping, with some peri-infarct ischemia. Amiodarone and mexiletine were discontinued; therapy with a β-blocker alone was continued. Subsequently, he developed atrioventricular nodal reentry (which had been seen years before amiodarone was initiated). This arrhythmia was abolished with slow pathway ablation, at which time programmed ventricular stimulation demonstrated that the prior VT present at the end of the ablation procedure was still inducible (cycle length of 260 ms). However, he has had no spontaneous VT and no angina over a follow-up of 21 months.

Case 4
A 30-year-old man with arrhythmogenic right ventricular cardiomyopathy was referred for ablation of recurrent VTs after prior failed endocardial ablation attempts and therapy with metoprolol and dofetilide. After ablation was unsuccessful at an endocardial exit site, percutaneous epicardial access was obtained. Extensive regions of low voltage (<1.5 mV) and fractionated potentials were identified over the anterior and inferior free wall of the right ventricle. The first VT (cycle length of 320 ms, left bundle branch–like morphology, and a left inferior axis) was successfully ablated in a reentrant circuit isthmus region in the epicardium of the right ventricular outflow tract. The second VT (left bundle branch–like morphology and left superior axis with a cycle length of 270 ms) was unstable for mapping. Pace mapping identified a likely reentrant circuit exit region in the epicardium at the diaphragmatic surface of the right ventricle in a region that was below the tricuspid annulus and to the right of the defibrillator lead as viewed in the left anterior oblique projection. It was therefore assumed that this area was remote from significant coronary arteries. Externally irrigated radiofrequency at up to 50 W of power was applied with an irrigation rate of 10 mL/min, rendering the region unexcitable to pacing (unipolar at 10 mA, 2-ms pulse width). No further ventricular arrhythmias could be induced with programmed stimulation during isoproterenol infusion.

The postprocedure recovery was uneventful. Two weeks later, the patient developed substernal chest pain and presented to his local cardiologist. His ECG showed inferior ST-segment elevation. Coronary angiography (Figure 5) revealed unusual RCA anatomy. A large right ventricular marginal branch that traversed the floor of the right ventricle gave rise to the distal posterior descending artery. This right ventricular branch was occluded before reaching the interventricular groove, and the posterior descending artery filled retrogradely from the left anterior descending artery. This occlusion was in the region of ablation lesions performed for the second VT (Figure 5). There was minimal detectable effect on left ventricular function, and no intervention was performed. Over 4 months of follow-up, the ejection fraction remained normal, and the patient remained free of VT.
Impedance Changes During Radiofrequency Ablation

In the 2 cases in which radiofrequency ablation was performed in the MCV, the mean impedance fall of the lesions causing the coronary injury was 44 Ω, and a subsequent impedance rise was observed before energy delivery was stopped. In case 4, 4 lesions were placed at the inferior aspect of the epicardial right ventricle. An impedance rise was noted at the end of 1 of these lesions after an impedance fall of 24 Ω.

Discussion

This study demonstrates a spectrum of presentations and outcomes of coronary injuries after the ablation of tachyarrhythmias. The potential for acute coronary occlusion is well recognized. This case series demonstrates that a delayed presentation is also possible. Furthermore, although coronary angioplasty with ballooning and stenting may restore vessel patency, the phenomenon of no-reflow may occur. Finally, unanticipated anatomic variations in coronary artery anatomy may predispose to coronary injury, particularly during epicardial ablation.

The incidence of coronary injuries during ablation procedures is extremely low. In 1998, the North American Society of Pacing and Electrophysiology registry reported only 1 coronary occlusion of 3357 ablation procedures. Similar results have been observed in other large studies. This low risk is somewhat surprising given the close proximity of coronary arteries to common sites of ablation and may be due in part to underrecognition and underreporting. Patients may develop chest discomfort during and after ablation for a variety of reasons, most often pericardial irritation. Hasdemir et al examined the anatomic relation of the coronary arteries to the tricuspid and mitral annulus, sites frequently targeted for ablation. The RCA was <5 mm from the cavitricuspid...
isthmus in 8% of patients, and the left circumflex artery was ≤2 mm from the lateral mitral annulus in 24% of patients. The coronary sinus and its branches were also in close relation to the distal circumflex and the posterolateral branches of the RCAs, with the left circumflex artery within 2 mm of the coronary sinus in 14% of patients and the posterolateral branch within 2 mm of the MCV ostium in 20% of patients. In adults with accessory pathways, Sun and colleagues⁷ found a significant coronary artery within 2 mm of the best ablation site in the coronary sinus or its branches in 65% of patients, and ablation at this site caused coronary stenosis in 66% of patients. In a study of 212 children in whom coronary angiography was performed before and after ablation, the only 2 coronary stenoses observed occurred with the ablation of posteroseptal pathways.⁸ In our study, the only 2 coronary injuries during endocardial ablation occurred at the ostium of the MCV. Angiography was performed before ablation in the second case, but subtle movements of the catheter associated with cardiac and respiratory motion may have contributed to the coronary injury in this case.

This study demonstrates the variability in presentation of coronary injuries after ablation. The majority of prior reports of coronary arterial injury have reported acute coronary occlusion at the time of ablation as the most common presentation.⁹–¹⁶ This presentation occurred in 2 of our 4 cases. Part of this acute response has been attributed to coronary spasm because several authors have reported chest pain with ST elevation after ablation but normal angiography after the resolution of the ECG changes.²,¹⁷ In addition, there are reports of acute occlusions, which untreated have been observed to be patent months after the ablation.¹⁴,¹⁸ Interestingly, spasm of the RCA has also been reported after ablation.
in the region of ganglionated plexi. However, in the present series, spasm seems unlikely because intracoronary nitroglycerin had no effect in the cases in which it was used. In animal models, the application of radiofrequency energy directly on a coronary artery causes acute edema with wall thickening and luminal narrowing, which then resolves to some extent. Within days, there is medial necrosis and loss of intimal and elastic tissue, with the subsequent development of severe intimal hyperplasia. One of our patients developed an acute coronary occlusion 2 weeks after epicardial radiofrequency ablation. To the best of our knowledge, this is the first reported delayed presentation of a myocardial infarction after ablation. Although we cannot exclude spontaneous plaque rupture, the location of the occlusion in relation to the ablation lesions (Figure 5), absence of associated atherosclerotic lesions in other vessels, and young age of the patient suggest that this was unlikely. An embolic event also seems unlikely in the absence of any left-sided ablation. Acute occlusion with later presentation due to increased demand also seems unlikely because symptoms were present at rest. Presumably, in addition to intimal hyperplasia, ablation damaged the arterial wall and endothelium, which provided the nidus for thrombus formation 2 weeks later.

Evidence of persistent injury without acute occlusion has been observed in animal models. After radiofrequency ablation near coronary arteries, minimal angiographic changes are observed in the short term. Histologically, lymphocytic infiltration and medial necrosis are seen. However, between 3 and 9 months after ablation, intimal hyperplasia may develop that is not detectable angiographically. Bertram et al described 2 children who developed coronary artery stenosis, identified over 1 year after the initial ablation procedure. In 1 of the cases, ST-segment elevation was observed shortly after the ablation, but angiography after intracoronary nitroglycerin showed no abnormality. Other angiographic studies in patients with accessory pathways with several months of follow-up have not shown any angiographic abnormalities. Whether the application of radiofrequency in the vicinity of coronary arteries predisposes to late coronary stenoses is not known.

For patients who develop an acute coronary occlusion after ablation, coronary stenting appears to be the treatment of choice. Several authors have reported that balloon angioplasty alone does not open the artery and restore blood flow. In these cases, the insertion of a coronary stent splints the artery open. This was the case in both of the

Figure 5. Electroanatomic epicardial voltage maps and corresponding RCA angiographic views in case 4. Top left, Right anterior oblique (RAO) view of the voltage map. Note the ablation lesions on the free wall of the right ventricular outflow tract, the site of ablation for the first VT. Top right, Corresponding angiographic view of the RCA. Bottom left, Right anterior oblique caudal view of the voltage map. Note the ablation lesions on the inferior (INF) aspect of the right ventricle, which was the site of ablation of the second VT. This corresponds with the site of coronary occlusion shown in the bottom right panel.
patients in this study with acute occlusions. However, in contrast to the other reports, 1 of our patients had a no-reflow phenomenon despite prompt stenting. This phenomenon is thought to relate to endothelial dysfunction, and both functional and morphological endothelial changes have been observed after the application of radiofrequency.27

Epicardial ablation probably poses a greater risk of coronary injury. Not only are the large epicardial coronary arteries in direct contact with the ablation catheter, but there can be movement as the ablation catheter slides in the epicardial space. One of our patients developed a stenosis after cryoablation in the epicardium. In this case, cryoablation was chosen because the ablation site was estimated to be 5 mm away from a small coronary branch. Cryoenergy has been thought to have a lower risk of coronary injury than radiofrequency.20 Using intravascular ultrasound, Aoyama et al20 demonstrated that during cryoablation, the ice ball compressed the artery, but after rewarming, there was no luminal narrowing. In our case, radiofrequency energy at more remote sites had been applied, but the coronary stenosis was not evident until after the freeze that was applied at a site estimated to be closer to the vessel than the radiofrequency lesions. Angiography was performed 10 minutes after the application of cryoenergy; however, the exact mechanism of the narrowing is unclear. The other epicardial coronary injury occurred in a patient with unusual RCA anatomy, with a large marginal branch that went on to supply the posterior descending artery. In this case, coronary angiography was not performed because the epicardial right ventricular free wall was assumed to be devoid of significant left ventricular coronary vessels.

Limitations

This is a retrospective case series. It is possible that some coronary occlusions or stenoses that did not produce symptoms or ECG changes were not detected.

Conclusion

There is a spectrum of presentations of coronary injury after ablation procedures. These may occur with both radiofrequency and cryoenergy. Although the majority of coronary injuries present acutely, delayed presentations may also occur. Coronary stenting appears to be required for acute occlusion that does not respond to nitroglycerin but may be complicated by the phenomenon of coronary no-reflow. Unanticipated anatomic variation may predispose to coronary injury. The awareness of these potential features of ablation should help with the risk/benefit assessment for ablation in locations where coronary arteries are at risk. In addition, coronary injury must be considered in patients who develop persistent chest discomfort, especially if ablation occurs in areas adjacent to the coronary circulation.

Sources of Funding

Dr Roberts-Thomson is the recipient of a Postgraduate Research Scholarship from the National Health and Medical Research Council of Australia (National Health and Medical Research Council grant ID 489417) and the Astra-Zeneca Fellowship in Medical Research from the Royal Australian College of Physicians. Dr Seiler is the recipient of a research grant from St Jude Medical (Switzerland). Dr Steven is the recipient of a research grant from Biosense Webster.

Disclosures

None.

References

19. Yamashita E, Tada H, Tatokoro K, Hashimoto T, Kaseno K, Miyaji K, Naito S, Oshima S, Taniguchi K. Left atrial catheter ablation promotes...


24. Sturm M, Hausmann D, Bokenkamp R, Bertram H, Wibbelt G, Paul T. Incidence and time course of intimal plaque formation in the right coronary artery after radiofrequency current application detected by intra-


27. Demaria RG, Page P, Leung TK, Dubuc M, Malo O, Carrier M, Perrault LP. Surgical radiofrequency ablation induces coronary endothelial dys-

**CLINICAL PERSPECTIVE**

Coronary artery injury is a rare but important complication after catheter ablation. In this study we report 4 patients (0.09%) who sustained a coronary injury of a cohort of patients undergoing 4655 ablation procedures. Coronary injury occurred in 3 patients after radiofrequency ablation, and a moderate asymptomatic stenosis occurred after cryoablation in the other patient. Branches of the right coronary artery were involved in all cases. There is a spectrum of presentations of coronary injury after ablation procedures. Acute occlusion of the artery was seen in 2 patients. Coronary stenting appears to be required for acute occlusion that does not respond to nitroglycerin but may be complicated by the phenomenon of coronary no-reflow. Although the majority of coronary injuries present acutely, delayed presentations of up to 2 weeks may also occur. Coronary arteries appear to be particularly susceptible to injury from ablation in the epicardium and the middle cardiac vein. Unanticipated anatomic variation may also predispose to coronary injury. Awareness of these features should help with the risk/benefit assessment for ablation in locations where coronary arteries are at risk. Coronary injury must be considered in patients who develop persistent chest discomfort, especially if ablation occurs in areas adjacent to the coronary circulation.
Coronary Artery Injury Due to Catheter Ablation in Adults: Presentations and Outcomes
Kurt C. Roberts-Thomson, Daniel Steven, Jens Seiler, Keiichi Inada, Bruce A. Koplan, Usha B. Tedrow, Laurence M. Epstein and William G. Stevenson

Circulation. 2009;120:1465-1473; originally published online September 28, 2009; doi: 10.1161/CIRCULATIONAHA.109.870790
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2009 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/120/15/1465

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/