Repolarization Abnormalities After Catheter Ablation of Accessory Atrioventricular Connections With Radiofrequency Current

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The purpose of this study was to evaluate the serial changes in T wave configuration in patients undergoing successful radiofrequency catheter ablation of accessory atrioventricular (AV) connections. Twenty-nine consecutive patients with overt preexcitation and 16 patients with a concealed accessory atrioventricular (AV) connection were included. An electrocardiogram (ECG) was recorded before ablation and 15 min, 1 or 2 days and 1 and 3 months after ablation. Postablation T wave abnormalities occurred in 22 (76%) of the 29 patients who had overt preexcitation but in none of the 16 patients with a concealed accessory AV connection. The T wave abnormalities were not related to myocardial necrosis or echocardiographic abnormalities. The ECG location and severity of T wave changes were dependent on the accessory AV connection location and degree of baseline preexcitation, respectively. Fourteen of 19 patients with a posteriorly located AV connection (left, right or septal) had T wave inversion or flattening in the inferior leads and 3 patients had precordial T wave peaking. Two patients with an anterosepstral AV accessory connection had both inferior T wave inversion or flattening and precordial T wave peaking. Among seven patients with a manifest left lateral accessory AV connection, two had lateral T wave inversion or flattening and two had precordial T wave peaking.

There was 98% concordance between the directional change of the T wave after ablation and the direction of the delta wave on the baseline ECG. Follow-up ECGs demonstrated complete resolution of the T wave changes in a minority of patients in the 1st 1 to 2 days after ablation; by 3 months, complete or near complete resolution occurred in nearly all patients.

It is concluded that transient repolarization abnormalities that mimic ischemia occur commonly after elimination of overt preexcitation and are dependent on the direction of the delta wave and the degree of baseline preexcitation. These repolarization abnormalities are not due to cardiac injury and may be explained by the presence of cardiac memory.

Methods

Patient characteristics. This was a prospective study of 45 consecutive patients who underwent successful radiofrequency catheter ablation of an accessory AV connection. There were 29 men and 16 women, with a mean age of 35 ± 12 years. Twenty-nine patients had overt preexcitation on the baseline electrocardiogram (ECG) and 16 patients had a concealed accessory AV connection. Among these 29 patients, the accessory AV connection location in patients with overt preexcitation was left posterior in 9, left lateral in 7, posteroseptal in 8, right posterior in 2, anterosepetal in 2 and right anterolateral in 1. In patients with a concealed accessory AV connection, the location was posteroseptal in 2, left lateral in 11 and left posterior in 3.

Catheter ablation protocol. After informed consent was obtained under a protocol approved by the Human Research Committee, an electrophysiologic test was performed with patients in the fasting state. Antiarrhythmic drugs were discontinued at least four half-lives before the procedure. Midazolam was used as needed for sedation. Electrode catheters were positioned in the right atrium, right ventricle. His bundle position, coronary sinus and left ventricle as
needed for pacing, mapping and ablation. An electrosurgical unit was used to deliver radiofrequency energy at a cycle length of 350,000 Hz (Radionics, model 3B). Left-sided accessory AV connections were ablated from the left ventricular aspect of the mitral annulus, whereas septal and right-sided accessory AV connections were ablated with applications of energy in the right atrium. The catheter used for ablation was a 7F catheter with a 4-mm distal electrode, 5-mm interelectrode spacing and a steerable tip (Mansfield/Webster). The creatine kinase-MB fraction was measured every 8 h for 24 h after the procedure.

In patients who had a concealed accessory AV connection, an electrophysiologic test was performed on the 2nd day after the ablation procedure to rule out a recurrence of conduction through the accessory AV connection. Patients were then discharged home and seen in follow-up 1 month later by their referring physician. An electrophysiologic test was performed 3 months after the ablation procedure to confirm successful long-term ablation of the accessory AV connection.

Analysis of the ECG. A 12-lead ECG was recorded at the beginning of the ablation procedure and 15 min after ablation. Additional ECG tracings were obtained 1 or 2 days after ablation and at 1 and 3 months of follow-up. The ECG was recorded at a 25-mm/s paper speed and a 10-mm/mV gain setting.

The baseline ECG was graded for the degree of pre-excitation as follows: none = no discernible delta waves; mild pre-excitation = small delta waves with a QRS width ≤0.1 s; and marked pre-excitation = easily identifiable delta waves with a QRS width >0.1 s.

The 15-min postablation ECG was graded for the severity of T wave abnormalities as follows: none = normal T wave configuration after ablation; mild T wave abnormalities = T wave flattening or inversion ≤3 mm or peaking ≤15 mm; and marked T wave abnormalities = T wave inversion >3 mm or peaking >15 mm. The postablation ECG was compared with the baseline ECG to evaluate the directional change of the T wave after ablation compared with the direction of the delta wave in each lead of the baseline ECG. The follow-up ECG was evaluated for the presence of either complete or partial resolution of the repolarization abnormalities.

Reproducibility was assessed by determining the inter- and intraobserver variability for both the pre-excitation and postablation T wave grading schemes. Ten preablation and 10 15-min postablation ECGs were randomly chosen for analysis in a blinded fashion. Reproducibility within and between two observers was 100%.

Statistical analysis. Data are expressed as mean values ± SD. Statistical analyses were performed with Student's t test or by contingency table analysis.

Results

Immediate postablation effects. Twenty-two (76%) of the 29 patients with overt pre-excitation had repolarization abnormalities 15 min after ablation (Fig. 1) compared with none of the 16 patients with a concealed AV connection. Fifteen (68%) of these 22 patients had T wave inversions, 3 (14%) had T wave peaking and 4 (18%) had both. No patient with postablation T wave abnormalities had symptoms of myocardial ischemia. There was no relation between the presence of repolarization abnormalities and enzymatic evidence of myocardial damage; the mean peak creatine kinase MB fraction was 5.5 ± 3.4 IU/liter in patients who had T wave abnormalities compared with 6.2 ± 4.4 IU/liter in those without T wave abnormalities (normal range 0 to 10, p = NS). There was no difference in the number of radiofrequency applications between patients with (6.5 ± 4.6) and without (7.4 ± 7.6) T wave changes.

No correlation was found between the presence of T wave changes and postablation echocardiographic abnormalities. Fifteen of 16 echocardiograms obtained in patients with T wave abnormalities were normal and one patient with an overt left lateral accessory AV connection had a mild anteroseptal wall motion abnormality. Twenty of 21 echocardiograms obtained in patients without T wave changes were normal and one patient with a concealed left lateral accessory AV connection had a proximal inferior wall motion abnormality.

Of 45 patients, 5 had a functional bundle branch block during orthodromic AV reciprocating tachycardia induced before ablation of the accessory AV connection. The presence of postablation T wave changes was not related to the occurrence of functional bundle branch block during tachycardia before ablation.

Relation to baseline pre-excitation (Table 1). The severity of the T wave abnormalities after ablation was dependent on the degree of baseline pre-excitation. Marked pre-excitation occurred in association with eight of eight posteroseptal, five of nine left posterior, two of two right posterior, two of two anteroseptal, one of one right anterolateral and three of seven left lateral accessory AV connections. Mild pre-excitation was present in the remaining patients. Marked T wave abnormalities occurred in 13 (62%) of the 21 patients with marked baseline pre-excitation but in only 1 (12%) of the 8 patients with mild pre-excitation (p < 0.05).

Relation to location of the accessory AV connection (Table 2). Sixteen of the 19 patients with a posterior accessory AV connection had postablation T wave changes (eight of eight posteroseptal, six of nine left posterior and two of two right posterior). Thirteen had T wave inversion or flattening in the inferior leads after ablation (six posteroseptal, five left posterior and two right posterior). Two had both inferior T wave inversion or flattening and precordial T wave peaking (one posteroseptal and one left posterior) and one patient with a posteroseptal accessory AV connection had isolated precordial T wave peaking. Thirteen of the 19 patients had normalization of the T wave in the precordial or lateral leads, or both.

Among the seven patients with a left lateral AV connection, four had T wave changes after ablation: two had lateral T wave inversion or flattening and two had precordial
T wave peaking (Fig. 2). Both patients with an anterosetal AV connection had inferior T wave inversion or flattening. precordial T wave peaking and normalization of the T wave in the lateral leads on the postablation ECG. One patient with a right anterolateral accessory AV connection showed only normalization of the T wave segments on the postablation ECG. There was a 95% concordance between the directional change of the T wave segments after ablation and the direction of the delta wave on the baseline ECG (that is. if the delta wave was positive, the T wave in that lead tended to change in a positive direction and vice versa).

Serial changes. A follow-up ECG was obtained to evaluate the time course of resolution of the repolarization abnormalities. Twenty of the 22 patients with T wave abnormalities on the immediate postablation ECG had a follow-up ECG 1 or 2 days later: 12 of the 20 showed either complete (n = 3) or partial (n = 8) resolution of the changes. Eight of the 18 patients who had not shown complete resolution at days 1 or 2 after ablation had a follow-up ECG at 1 month: 3 showed complete and 5 showed partial resolution. Thirteen of the 15 patients in whom normalization of the ECG had not occurred by 1 month had an ECG at 3 months: 7 showed complete and 6 showed near complete resolution of the changes.

Delayed effects. Six of the 29 patients with overt pre-excitation had delayed T wave changes noted 1 day after ablation (Fig. 3). Among the seven patients with overt pre-excitation who did not have T wave abnormalities on the immediate postablation ECG, four developed ECG changes 1 day later. Two of the 22 patients with T wave abnormalities on the immediate postablation ECG developed new T wave changes 1 day later. The ECG location of the delayed changes was consistent with the location of the changes noted on the immediate postablation ECG for the particular AV connection locations. In all of these cases, the T wave changes were graded as mild.

Discussion

Main findings. This study demonstrates that T wave abnormalities that mimic ischemia often occur after catheter ablation of manifest accessory atrioventricular (AV) connec-

<table>
<thead>
<tr>
<th>Severity of T Wave Abnormalities</th>
<th>Degree of Baseline Pre-excitation</th>
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<tbody>
<tr>
<td>Marked</td>
<td>(n = 21)</td>
</tr>
<tr>
<td>Mild</td>
<td>(n = 8)</td>
</tr>
<tr>
<td>None</td>
<td>(n = 18)</td>
</tr>
<tr>
<td>Marked</td>
<td>13 (62%)</td>
</tr>
<tr>
<td>Mild</td>
<td>5 (24%)</td>
</tr>
<tr>
<td>None</td>
<td>3 (14%)</td>
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</tbody>
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Table 1. Relation Between the Degree of Pre-excitation and the Severity of Postablation Repolarization Abnormalities in 45 Patients

p < 0.05 between pre-excitation groups.
Table 2. Relation Between Accessory Atrioventricular Connection Location and the Location of T Wave Changes after Ablation

<table>
<thead>
<tr>
<th>Accessory Pathway Location</th>
<th>Major T Wave Abnormalities</th>
<th>Other Observed T Wave Changes</th>
</tr>
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<tbody>
<tr>
<td>Posterior (left, right and septal) (n = 19)</td>
<td>Inferior T wave inversion 14 (74.7%)</td>
<td>Precedial T wave peaking 3 (16%)</td>
</tr>
<tr>
<td>Left lateral (n = 7)</td>
<td>Lateral T wave inversion 2 (29%)</td>
<td>Precedial or lateral T wave normalization 13 (68.4%)</td>
</tr>
<tr>
<td>Anteroseptal (n = 2)</td>
<td>Inferior T wave inversion 2 (100%)</td>
<td>Inferior or precedial T wave normalization 1 (14.3%)</td>
</tr>
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</table>

Possible mechanisms for repolarization changes after elimination of pre-excitation. Ventricular repolarization normally proceeds in a direction opposite from that of depolarization, resulting in a T wave that is in the same direction as the QRS complex (10). Alterations in the sequence of ventricular activation cause changes in the pattern of repolarization that are manifest as abnormalities of the T wave complex (11). Common clinical situations associated with altered ventricular activation are ventricular pacing, intraventricular conduction defects and ventricular pre-excitation. In these settings, there are “secondary” T wave changes that are opposite in direction to the QRS complex.

Rosenbaum et al. (1) demonstrated that in the case of right ventricular pacing and left bundle branch block, the T wave results from a balance of two forces: one that causes the secondary changes that are opposite in direction to the QRS complex and disappear rapidly once normal ventricular activation is restored, and another that develops over time, causes changes that are in the same direction as the QRS complex and that may persist for long periods after restoration of normal ventricular activation. This latter force is believed to be responsible for “cardiac memory” (1). These general principles of ventricular repolarization would be expected to apply to other clinical situations in which there

Figure 2. A. Baseline electrocardiogram (ECG) demonstrating marked pre-excitation in a patient with a left lateral atrioventricular connection that had a long anterograde refractory period. The QRS complexes marked with an “are not pre-excited and show T wave changes from the pre-excited state. B. After ablation, there are mild T wave abnormalities with T wave inversion in lead aVL and flattening in lead I. There is also normalization of the T wave segments in the inferior and preordial leads. The repolarization changes after ablation are the same as those seen on the normally conducted beats in the baseline ECG (A). The directional change of the T wave from before to after ablation is consistent with the direction of the delta wave on the baseline ECG. (Electrocardiograms were retouched for clarity.)
is abnormal ventricular activation, including ventricular pre-excitation.

In ventricular pre-excitation, secondary T wave abnormalities occur and the T wave is generally directed opposite to the major delta wave and QRS vectors (8). The elimination of pre-excitation and restoration of normal ventricular activation would immediately result in loss of the secondary T wave changes and cause a shift in the T wave segment in the direction of the previously present delta wave. The presence of cardiac memory would also be expected to cause a shift in the T wave in the direction of the delta wave vector. The findings of this study are consistent with these principles. There was a 95% concordance between the directional change of the T wave after ablation and the direction of the delta wave on the baseline ECG. Leads in which the delta wave was negative tended to have T wave inversion or flattening after ablation and leads in which the delta wave was positive had T wave peaking or an increase in the T wave height after ablation.

Results of previous studies. A previous study (12) attempted to evaluate the presence of cardiac memory after surgical ablation of accessory AV connections and concluded that the post-ablation T wave changes were not due to memory. However, because T wave abnormalities caused by pericardial irritation commonly occur after thoracotomy and cardiac surgery, repolarization changes due to cardiac memory may have been observed. Another study (8) described the presence of "ischemic" inferior T wave inversion after catheter ablation of posteroseptal AV connections using electric shocks as a potential complication of the
procedure: it was not stated whether the ischemic T wave inversions were limited to patients with overt pre-excitation or whether they also occurred in patients with a concealed AV connection. In addition, these changes were not correlated with measures of cardiac damage. Because of these limitations, it is unclear whether the T wave abnormalities resulted from traumatic effects of the electric shocks or from loss of pre-excitation.

Resolution of repolarization abnormalities. Electrocardiograms obtained 1 or 2 days after ablation showed some resolution of the repolarization abnormalities in approximately 50% of patients but complete resolution in only 20% of patients. The intermediate follow-up ECG at 1 month after ablation was incomplete but did show that there are persistent changes this late after the procedure. The 3-month follow-up ECG demonstrated that nearly all of the T wave abnormalities had resolved by this time and that the remaining abnormalities were minor. The delayed resolution of the repolarization abnormalities after the loss of pre-excitation is consistent with the results of previous studies (4,6) demonstrating that it may take months for normalization of the ECG after cessation of pacing or termination of a tachycardia.

Delayed repolarization changes. A small group of patients showed repolarization abnormalities 1 day after ablation that were not present 15 min after ablation. The location and direction of these delayed changes were consistent with the abnormalities seen immediately after ablation in other patients. Although there is no readily apparent explanation for this phenomenon, the absence of such delayed abnormalities in patients with a concealed accessory AV connection indicates a high likelihood that they were related to the loss of ventricular pre-excitation. It is possible that there is at times a latency period in the expression of cardiac memory.

Clinical implications. This study demonstrates that repolarization abnormalities occur commonly after catheter ablation of overt accessory AV connections using radiofrequency energy and that these abnormalities are a manifestation of cardiac memory rather than a result of myocardial injury or pericarditis. The location and severity of these postablation repolarization abnormalities may be predicted on the basis of the direction of the delta wave vector and the degree of pre-excitation, respectively. Repolarization abnormalities that occur after ablation of a concealed accessory AV connection or T wave abnormalities that are in a direction opposite to that of the previous delta wave in that lead cannot be explained by the phenomenon of cardiac memory and may be an indication of myocardial injury or pericarditis.

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References