Incidence, Significance, and Pharmacological Responses of Catheter-Induced Mechanical Trauma in Patients Receiving Radiofrequency Ablation for Supraventricular Tachycardia

Chern-En Chiang, MD; Shih-Ann Chen, MD; Tsu-Juey Wu, MD; Chin-Juey Yang, MD; Chen-Chuan Cheng, MD; Shih-Pu Wang, MD; Benjamin N. Chiang, MD; Mau-Song Chang, MD

Background Catheter-induced mechanical trauma is unfavorable during electrophysiological study. However, its incidence, significance, and pharmacological responses in patients receiving radiofrequency ablation for supraventricular tachycardia have not been investigated.

Methods and Results A prospective study was performed in 666 consecutive patients with documented, symptomatic supraventricular tachycardia. All had been referred for electrophysiological study and radiofrequency ablation. Catheter-induced mechanical trauma was defined by either disappearance of or change in preexcitation pattern induced by the electrode catheters or noninducibility of tachycardia after the electrode catheter-induced termination of tachycardia, confirmed by electrophysiological study. Adenosine, isoproterenol, and atropine were serially administered 1 hour after the mechanical trauma to study pharmacological response. "Rescue" radiofrequency ablation was defined as delivery of radiofrequency energy just at the presumed ablation site immediately after the mechanical trauma. Of the 666 patients, 254 had atrioventricular (AV) nodal reentrant tachycardia, 367 patients had accessory pathways, 30 patients had atrial tachycardia, and 15 had atrial flutter. Catheter-induced mechanical trauma occurred in 17 patients (2.6%): 4 patients had AV nodal reentrant tachycardia, 9 had accessory pathways, and 4 had atrial tachycardia. Five patients had such episodes during the placement of electrode catheters and 12, during mapping and ablation procedures. Of the 4 patients with AV nodal reentrant tachycardia, 3 had mechanical trauma on the retrograde fast pathway and 1, on the antegrade slow pathway. In the 9 patients with accessory pathways, those pathways were located in the left free wall in 4 patients, right free wall in 1, right postero septum in 1, and right anteroseptum in 3. Atrial tachycardia was more easily traumatized than AV nodal reentrant tachycardia (P<.01) and than accessory pathways (P<.01). The clinical courses of mechanical trauma were variable: 1 patient had spontaneous recovery within 1 week, 5 patients had recurrence of tachycardia within 3 months, and the rest have been free of tachycardia from 3 to 35 months. The recurrence rate was higher in patients with mechanical trauma than in those without (33.3% versus 3.5%, P<.0001) despite rescue radiofrequency ablation given in 7 patients. Pharmacological agents were generally unable to revive the traumatized tissues, and recurrence was unpredictable.

Conclusions Catheter-induced mechanical trauma was not common in patients receiving radiofrequency ablation for supraventricular tachycardia. Their clinical courses were variable, and pharmacological manipulation offered little assistance. More than half of the patients had long-term cures. However, the recurrence rate was, on the whole, significantly high despite rescue radiofrequency ablation. There is a need for great caution in the placement of electrode catheters in every patient during electrophysiological study and radiofrequency ablation. (Circulation. 1994;90:1847-1854.)

Key Words • catheters • tachycardia • radiofrequency • catheter ablation

Catheter-induced mechanical trauma in conducting tissues has been reported during right heart catheterization1-5 and electrophysiological study.6-8 It has been suggested that the catheter tip may not be the only culprit; the rigid shaft of the catheter might also be responsible.9,10 The traumatized tissues may take several hours or days before recovery8 or may become permanently damaged.11 A repeated ablation session becomes necessary when the traumatized tissues revive. The tip electrode of the electrode catheter used for radiofrequency ablation is larger (4 mm), and manipulation in the heart increases during mapping and ablation procedures. However, reports describing the electrophysiological characteristics of catheter-induced mechanical trauma in patients with supraventricular tachycardia were not available. Thus, this prospective study was designed to determine the incidence and significance of catheter-induced mechanical trauma and to investigate pharmacological responses in patients receiving radiofrequency ablation for supraventricular tachycardia.

Methods

Patient Characteristics

From May 1990 to June 1993, 666 consecutive patients were referred to this hospital for electrophysiological study and radiofrequency catheter ablation of documented, symptomatic supraventricular tachycardias. There were 393 male and 273 female patients, with a mean age of 49±10 years (range, 13 to 93 years). Before referral, a mean of 3.4±0.2 antiarrhythmic drugs either had been ineffective or had not been tolerated.
Electrophysiological Study

As described previously, informed consents were obtained from all patients under an investigational protocol approved by the Human Research Committee of this institution. Electrophysiological study was performed while the patient was fasting and not sedated, and all the antiarrhythmic medications were discontinued for at least 5 half-lives before study. A complete 12-lead ECG was performed in the electrophysiological laboratory immediately before the catheterization procedure. The preexcitation pattern (delta wave) and spontaneously occurring tachycardia were recorded.

Three 6F multipolar electrode catheters (Mansfield, Boston Scientific) were inserted percutaneously into the right or left femoral vein and positioned in the right atrium, the His bundle area, and the right ventricle. One or two orthogonal electrode catheters (or other multipolar electrode catheters for recording coronary sinus electrogram) were inserted into the right internal jugular vein and placed in the coronary sinus. Any damage to the putative arrhythmogenic tissues resulting in disappearance of the delta wave or termination of tachycardia during the placement of electrode catheters was recorded, and 12-lead ECGs were repeated immediately.

The diagnostic catheter portion of the electrophysiological study included (1) measurement of the conducting properties of the atrium, AV node, ventricle, and accessory pathways (if present); (2) initiation of supraventricular tachycardia; and (3) determination of the mechanism of tachycardia. If tachycardia could not be induced in the baseline state, isoproterenol (1 to 4 μg/min) or atropine (1 to 2 mg) was used to facilitate the induction of tachycardia. Induced tachycardias were classified as AV nodal reentrant tachycardia (typical or atypical), atrioventricular reciprocating tachycardia involving an accessory pathway, atrial tachycardia, and atrial flutter, according to the classic criteria.

Radiofrequency Ablation

A 7F quadrupolar electrode catheter with a 4-mm distal electrode, an interelectrode space of 2 mm, and a deflectable curve (Mansfield, Boston Scientific) was used for ablation. Radiofrequency current (generated from Radionic-3C) was delivered between the tip electrode and an indifferent patch electrode positioned on the left side of the chest. The unit was coupled to a device that provided real-time monitoring of root-mean-square voltage, current, and impedance. The ablation techniques used in various types of tachycardia have been described previously. In brief, the patients with AV nodal reentrant tachycardia received radiofrequency ablation of the slow pathway, and the ablation site was located in the right posteroseptal area or near the coronary sinus ostium. In patients with accessory pathways, the accessory pathway potential (described by Jackman et al), local electrogram showing fusion of A and V waves with V wave earlier than the delta wave (in patients with manifest accessory pathways), and local electrogram showing fusion of V and A waves during ventricular pacing or reciprocating tachycardia (in patients with concealed accessory pathways) were used to guide the ablation catheters. In patients with atrial tachycardia and atrial flutter, earliest atrial endocardial activation or concealed entrainment pace mapping with short stimulus-P interval (<40 milliseconds) could identify the optimal ablation site. After the ablation procedure, bolus injection of adenosine (6 to 12 mg) for patients with Wolff-Parkinson-White (WPW) syndrome, atropine (1 to 2 mg), and isoproterenol (1 to 4 μg/min) for patients with various types of tachycardia were administered 20 to 30 minutes later to ensure successful results. If the delta wave or any tachycardia recurred, mapping and ablation procedures were repeated.

Study of Catheter-Induced Mechanical Trauma

The 12-lead ECG was continuously monitored during the whole electrophysiological study and ablation procedure. Catheter-induced mechanical trauma was defined by either (1) disappearance of or change in preexcitation pattern caused by the electrode catheters or (2) noninducibility of tachycardia after the electrode catheter-induced termination of tachycardia as confirmed by electrophysiological study.

Patients who suffered mechanical trauma from the small-tip diagnostic electrode catheter during catheter placement were designated group 1. Recovery of the traumatized tissues was defined as "immediate" (<1 hour) or "late" (>1 hour and ≤1 week). Recurrence was defined as recurrence of tachycardia or delta wave after 1 week. If there was no immediate recovery, serial pharmacological agents were administered to revive the traumatized tissues and to predict recurrence. Adenosine was injected within 1 second into the right femoral vein in patients with WPW syndrome with an initial dose of 6 mg to a maximum of 18 mg, with an increment of 6 mg every 5 minutes. After 5 minutes from the last dose of adenosine, isoproterenol was infused in patients with various tachycardias from an initial dose of 1 μg/min to the maximal dose of 4 μg/min, with an increment of 1 μg/min every 5 minutes. Twenty minutes after the last dose of isoproterenol, atropine was administered in patients with various tachycardias, from an initial dose of 0.5 mg with an increment of 0.5 mg every 5 minutes to a total dose of 2 mg. Responses to these three agents were recorded. If spontaneous recovery or response occurred, radiofrequency catheter ablation proceeded; otherwise, the procedure was stopped. Follow-up electrophysiological study and pharmacological tests were repeated 1 week later. If there was no late recovery and still no response to pharmacological agents, the patient was discharged.

Patients who suffered from mechanical trauma caused by the large-tip mapping/ablation catheter during mapping and ablation procedures were designated group 2. They were further divided into two subgroups depending on whether they were administered "rescue" radiofrequency ablation (group 2B) or not (group 2A). "Rescue" radiofrequency ablation was defined as delivery of radiofrequency energy immediately after mechanical trauma, just at the presumed ablation site. Rescue radiofrequency ablation was not performed when the tip electrode of the mapping/ablation catheter was away from the presumed ablation site. Rescue radiofrequency energy was applied in the following manners. In patients with AV nodal reentrant tachycardia, if tachycardia stopped and was rendered noninducible during manipulation of the mapping/ablation catheter near the presumed ablation site, the mapping/ablation catheter was frozen at that site, and programmed stimulation was repeated. If tachycardia was not inducible and the local electrogram of the mapping/ablation catheter was optimal, rescue radiofrequency energy was applied at this site for 60 seconds. In patients with WPW syndrome, once the delta wave disappeared during manipulation of the mapping/ablation catheter around the AV ring and the local electrogram showed small A wave and large V wave, the ablation catheter was frozen and rescue radiofrequency energy was delivered immediately for a duration of 60 seconds. In patients with concealed accessory pathway, when the tachycardia was terminated during mapping around the AV ring, the ablation catheter was frozen and ventricular pacing was performed immediately to ensure that retrograde accessory pathway conduction was lost. Rescue radiofrequency energy was delivered at once at this site for 60 seconds. In patients with atrial tachycardia and atrial flutter, the rescue radiofrequency energy was delivered at the site at which the tachycardia was terminated by the mapping/ablation catheter during mapping around the presumed ablation site. After the ablation procedure, pharmacological agents were administered as in group 1 to all patients to test their responses. Follow-up electrophysiological study and pharmacological tests were repeated 1 week later.

The patients of group 2A who did not receive rescue radiofrequency energy were observed for 1 hour. If there was no immediate recovery, serial pharmacological agents were administered, just as in group 1 patients. If there was no response, follow-up electrophysiological study and pharmacological tests were repeated 1 week later.
After the ablation procedure, all the patients underwent continuous ECG monitoring for 24 hours in the Coronary Care Unit. Any recurrence of tachycardia or delta wave was recorded. After discharge, they were regularly followed at the outpatient clinic at intervals of 1 week, 1 month, and then every 3 months. A history of symptoms was taken, and 12-lead ECG and 24-hour Holter monitoring were performed.

**Statistical Analysis**

All values are expressed as mean±SD. Comparison of the incidence of mechanical trauma between groups was performed by χ² test with Yates’ correction. Comparison of electrophysiological parameters between groups was performed with the Mann-Whitney rank-sum test. A value of P<.05 was considered statistically significant.

**Results**

**Electrophysiological Study and Radiofrequency Ablation**

Baseline electrophysiological studies found that 254 patients had AV nodal reentrant tachycardia, 236 patients had AV reciprocating tachycardia involving a concealed accessory pathway, 131 patients had manifest preexcitation, 30 patients had atrial tachycardia, and 15 patients had common-type atrial flutter. Of the 254 patients with AV nodal reentrant tachycardia, 228 patients had the typical form, 12 had atypical, and 14 had both typical and atypical forms. Four patients (1.6%) suffered from mechanical trauma. Radiofrequency ablation was successful in all 250 patients (100%) who did not have mechanical trauma; 6 patients (2.4%) had recurrence. Of the 367 patients with accessory pathways, 337 had a single accessory pathway, and 30 had multiple accessory pathways. Among the total 401 accessory pathways, 196 (48.9%) were in the left free wall, 86 (21.4%) in the right free wall, 81 (20.2%) in the posteroseptal area, and 38 (9.5%) in the anteromidseptal area. Nine accessory pathways (2.2%) suffered from mechanical trauma. Among the 392 accessory pathways that did not have mechanical trauma, 377 (96.2%) had successful radiofrequency ablation, and 15 (4.0%) had recurrence. Of the 30 patients with atrial tachycardia, 4 (13.3%) suffered from mechanical trauma. Radiofrequency ablation was successful in 25 (96.2%) of the 26 patients who did not have mechanical trauma; 1 patient (4%) had recurrence. Among the 15 patients with atrial flutter, none suffered from mechanical trauma. Radiofrequency ablation was successful in 14 patients (93.3%), and no recurrence was found. Overall, 17 patients suffered from catheter-induced mechanical trauma. Of the 649 patients without mechanical trauma, 632 (97.4%) had successful radiofrequency ablation, and 22 (3.5%) had recurrence.

**Catheter-Induced Mechanical Trauma**

These episodes occurred in 17 patients (2.6%) (Table). Five patients (29%) suffered from mechanical trauma caused by the small-tip diagnostic electrode catheter and 12 (71%) by the large-tip mapping/ablation catheter. Rescue radiofrequency ablation was performed in 7 patients.

**AV Nodal Reentrant Tachycardia**

Four patients (1.6%) with the slow-fast form of AV nodal reentrant tachycardia suffered from mechanical trauma, including 3 patients with injury on the retrograde fast pathway and 1 with injury on the antegrade slow pathway. The tachycardias were readily inducible in the 4 before the episodes. Of the 3 patients with injury on the retrograde fast pathway, 2 were injured by small-tip electrode catheters near the AV junction during electrophysiological study (Fig 1); 1 was injured by the large-tip mapping/ablation electrode catheter, which accidentally bounced during the mapping procedure. In the single patient with injury on the antegrade slow pathway, the episode happened when the large-tip mapping/ablation catheter was manipulated around the right posteroseptal area near the coronary sinus ostium (Fig 2); this patient underwent rescue radiofrequency ablation of the slow pathway.

**Accessory Pathway**

Nine patients (2.5%) suffered from catheter-induced mechanical trauma. All had a single accessory pathway. The pathways were located in the left free wall in 4 patients, the right free wall in 1, the right posteroseptal area in 1, and the right anteroseptal area in 3. Two patients suffered from mechanical trauma from the coronary sinus electrode catheter (Fig 3), making rescue radiofrequency ablation impossible, and accessory pathway potential was not found in them. The other 7 patients suffered from mechanical trauma from large-tip mapping/ablation catheters, and 4 of them received rescue radiofrequency ablation (Figs 4 and 5). There was no significant difference in the incidence of mechanical trauma between right free wall and septal pathways versus left free wall pathways (2.4% versus 2.0%, P>.05), between septal pathways versus free wall pathways (3.3% versus 1.8%, P>.05), or between manifest versus concealed pathways (3.8% versus 1.7%, P>.05). The electrophysiological characteristics, including effective refractory period, the shortest cycle length capable of 1:1 conduction, did not differ significantly in the accessory pathways with or without mechanical trauma.

**Atrial Tachycardia and Atrial Flutter**

Four (13.3%) of the 30 patients with atrial tachycardia suffered from catheter-induced mechanical trauma. The episodes occurred during catheter placement in 1 patient and during mapping and ablation procedures in 3 others. The mechanism of tachycardia was not clear in the former, but in the latter, it was reentry. The 3 had exhibited readily inducible tachycardia by electrophysiological study. Two patients received rescue radiofrequency ablation. Atrial tachycardia was more easily traumatized than AV nodal reentrant tachycardia (13.3% versus 1.6%, P<.01) and than accessory pathways (13.3% versus 2.5%, P<.01). Catheter-induced mechanical trauma was not found in patients with atrial flutter.

**Clinical Courses**

Immediate recovery was demonstrated in only 1 (5.9%) of the 17 patients by electrophysiological study, and she underwent subsequent radiofrequency ablation (Table). None had late recovery. However, 5 patients had recurrent tachycardia at <1 month (3 patients) or at about 3 months (2 patients). The patient with immediate recovery and another patient who responded to isoproterenol were not included in the calculation of recurrence rate. Thus, overall recurrence rate in pa-
Characteristics of 17 Patients With Catheter-Induced Mechanical Trauma

<table>
<thead>
<tr>
<th>Group and Patient</th>
<th>Age/ Sex</th>
<th>Diagnosis</th>
<th>Traumatized Site</th>
<th>Recovery of Traumatized Tissue</th>
<th>Pharmacological Response</th>
<th>Isoproterenol</th>
<th>Atropine</th>
<th>Adenosine</th>
<th>Rescue</th>
<th>RF</th>
<th>Recurrence</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>64/M</td>
<td>AVNRT</td>
<td>Retrograde FAVN</td>
<td>-</td>
<td></td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>26/F</td>
<td>AVNRT</td>
<td>Retrograde FAVN</td>
<td>-</td>
<td></td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>22/M</td>
<td>MWPW</td>
<td>Left free wall AP</td>
<td>-</td>
<td></td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>46/F</td>
<td>MWPW*</td>
<td>Left free wall AP</td>
<td>-</td>
<td>+†</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>40/F</td>
<td>AT†</td>
<td>Right AT</td>
<td>-</td>
<td></td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2A</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>45/F</td>
<td>AVNRT</td>
<td>Retrograde FAVN</td>
<td>+</td>
<td>U</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>66/M</td>
<td>CWPW</td>
<td>Left free wall AP</td>
<td>-</td>
<td></td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>18/M</td>
<td>MWPW</td>
<td>Right anteroseptal AP</td>
<td>-</td>
<td>+†</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>46/M</td>
<td>MWPW</td>
<td>Right anteroseptal AP</td>
<td>-</td>
<td></td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>79/M</td>
<td>IART</td>
<td>Right AT</td>
<td>-</td>
<td></td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2B</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>44/F</td>
<td>AVNRT</td>
<td>Antegrade SAVN</td>
<td>-</td>
<td></td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>60/F</td>
<td>CWPW</td>
<td>Left free wall AP</td>
<td>-</td>
<td></td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>59/F</td>
<td>MWPW</td>
<td>Right free wall AP</td>
<td>-</td>
<td></td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>45/M</td>
<td>MWPW</td>
<td>Right posteroseptal AP</td>
<td>-</td>
<td>+</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>33/F</td>
<td>CWPW</td>
<td>Right anteroseptal AP</td>
<td>-</td>
<td></td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>62/F</td>
<td>IART</td>
<td>Right AT</td>
<td>-</td>
<td></td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>38/M</td>
<td>IART</td>
<td>Right AT</td>
<td>-</td>
<td></td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

RF indicates radiofrequency ablation; AVNRT, atrioventricular nodal reentrant tachycardia; FAVN, fast AV nodal pathway; ND, not done; MWPW, manifest Wolff-Parkinson-White syndrome; AP, accessory pathway; AT, atrial tachycardia; U, unknown; CWPW, concealed WPW syndrome; IART, intra-atrial reentrant tachycardia; and SAVN, slow AV nodal pathway.

*Intermittent WPW syndrome.
†Definite mechanism was not clear.
‡Responsive.

The recurrence rate in patients receiving rescue radiofrequency ablation was also higher than the overall recurrence rate (28.7% [2/7] versus 3.5% [22/632], P < .0001). The rest of the patients have been asymptomatic from 6 to 38 months.

Pharmacological Responses

Only 2 patients (11.7%) responded to pharmacological agents: 1 patient with a left free wall accessory pathway responded to isoproterenol 1 hour after mechanical trauma, and she received radiofrequency ablation under continuous infusion of isoproterenol (2 μg/ min); for another with a right anteroseptal accessory pathway who responded to adenosine (Fig 6) 1 hour after mechanical trauma, only the retrograde conducting property responded, and the possibility of secondary sympathetic activation could not be excluded (Table). Radiofrequency ablation could not be performed because of the short duration of action of adenosine. However, that patient had recurrent tachycardia <1 month later. The accuracy of pharmacological manipulation in prediction of recurrence was poor. Four of the 15 patients (26.7%) who had no response had recurrent tachycardia.

Discussion

This is the first study to determine the incidence, significance, and pharmacological responses of catheter-induced mechanical trauma in a large group of patients who had received radiofrequency ablation for supraventricular tachycardia. The major new findings were that (1) the incidence (2.6%) was low, but the recurrence rate was significantly high; (2) the clinical courses were variable, but more than half of the patients achieved long-term cures; (3) the damages were often caused by the large-tip ablation catheter, although rescue radiofrequency energy delivered through the same catheter did not ensure permanent cure; and (4) pharmacological manipulation was generally unable to revive the traumatized tissues, nor could recurrence be predicted.

Catheter-Induced Mechanical Trauma on Supraventricular Tachycardia

All 4 patients with AV nodal reentrant tachycardia had readily inducible tachycardia before mechanical manipulation. However, catheter-induced mechanical trauma to the right anteroseptal accessory pathway led to a 1-hour recurrence of tachycardia with no response to pharmacological agents.
trauma. Furthermore, the electrophysiological studies were done in the nonsedated state. The possibility of noninducibility of tachycardia, independent of trauma, in these patients was low. Three patients had mechanical trauma presumed to be on the retrograde fast pathway. The episodes happened as the electrode catheters were manipulated near the anterosuperior aspect of the AV node. Right ventricular pacing performed immediately after the trauma showed complete ventriculoatrial conduction block. Although the conducting property of the slow pathway was preserved, AV nodal reentrant tachycardia or echo beat could not be induced by atrial extrastimulation. One patient had mechanical trauma on the slow pathway. This happened during manipulation of the ablation catheter in the right posteroseptal area near the coronary sinus ostium. Although the slow AV nodal pathway was presumed to be approximately here,15 mechanical trauma has not previously been reported. Alteration in autonomic tone might have been a cause, but sinus cycle length, AH interval, and systolic blood pressure were unchanged before and immediately after the trauma, making the possibility unlikely. Because rescue radiofrequency energy was applied at this site, the long-term effect of mechanical trauma in this particular patient cannot be evaluated.

Of the 4 patients with left free wall accessory pathways, 2 suffered from mechanical trauma induced by coronary sinus electrode catheters. The possibility of phase 3 and phase 4 block was excluded because the sinus cycle lengths were the same before and immediately after the episodes.18 Furthermore, large accessory pathway potential was not found in these patients. Several authors19-21 have reported experience in ablation of left-sided accessory pathways via the coronary sinus, and these pathways could be identified by large accessory pathway potential within the coronary sinus. In this laboratory, the left-sided accessory pathways could be ablated successfully by retrograde LV approach or transeptal approach.19 The other two patients had mechanical trauma by the large-tip ablation catheter in the left ventricle. The above findings that left-sided pathways may be injured either by the ablation catheters subendocardially or by the coronary sinus catheters subepicardially would indicate that the left-sided accessory pathways traverse the AV groove at a variety of depths.

Septal and right-sided accessory pathways were generally located in the subendocardial position22 and are said to be especially susceptible to mechanical trauma by electrode catheters.11 In the report by Novick et al,6 catheter-induced mechanical trauma occurred exclusively in patients with septal accessory pathways. The present study found no preponderance of mechanical trauma in patients with septal and right-sided accessory pathways. This suggests that the depth of the accessory pathways traversing the AV grooves was not the only determinant of mechanical trauma. Other factors might have been operative, including the vigor with which the catheter was being manipulated.

Fig 2. Catheter-induced mechanical trauma on the slow AV nodal pathway in a patient with a slow-fast form of AV nodal reentrant tachycardia (patient II) A. Incremental pacing from high right atrium (HRA) with cycle length of 400 milliseconds during baseline electrophysiological study reveals slow pathway conduction and initiated slow-fast form AV nodal reentrant tachycardia. B. After termination of the tachycardia by use of a large-tip ablation catheter in the right posteroseptal area, incremental atrial pacing with the same cycle length (400 milliseconds) shows no slow pathway conduction and AV nodal reentrant tachycardia cannot be induced thereafter. Rescue radiofrequency energy was delivered at this site. ABL indicates ablation catheter; HBE, His bundle electrogram; MCS, middle coronary sinus; OCS, coronary sinus ostium; and PCS, proximal coronary sinus.
A high incidence of mechanical trauma was found in patients with atrial tachycardia, a finding not reported previously and difficult to explain. In the patient with mechanical trauma caused by a small-tip electrode catheter, the precise mechanism of atrial tachycardia was not clear. The warm-up phenomenon was not found in her ambulatory ECG before electrophysiological study, and spontaneous tachycardia was found just before catheter manipulation. Furthermore, the tachycardia has been quiescent for 10 months after the episode. Although the possibility of spontaneous cessation of an automatic focus could not be ruled out, mechanical trauma was believed to be the cause. It is obvious that the catheters should be manipulated with extreme caution in patients with atrial tachycardia.

In the report of Novick et al., all the damaged pathways recovered within 14 hours. Robinson et al.11 reported a case with permanent unintentional ablation of an accessory pathway during the placement of a coronary sinus catheter. The present study showed

**Fig 3.** Catheter-induced mechanical trauma on a manifest left free wall accessory pathway (patient 3). A. The accessory pathway was traumatized when the coronary sinus electrode catheter was being inserted. The episode happened to be recorded by the 12-lead ECG, which showed disappearance of delta wave from the sixth beat (arrow). The sinus cycle lengths before and after the episode were the same. This excluded the possibility of phase 3 or phase 4 block. B. Right ventricular pacing with cycle length of 450 milliseconds after the trauma showed complete VA block and suggested that the retrograde conduction property of the accessory pathway was damaged simultaneously. CS2 to CS5 indicate bipolar electrogram of the most proximal pair (CS1) to the most distal pair (CS5) from the decapolar coronary sinus electrode catheter (interelectrode space, 2 mm; interelectrode pair space, 5 mm); HRA, high right atrium; and HBE, His bundle electrogram.

**Fig 4.** Catheter-induced mechanical trauma in a patient with a concealed left lateral accessory pathway (patient 12). Right ventricular pacing demonstrated complete VA block after the second paced beat after insertion of the large-tip ablation catheter beneath the lateral mitral annulus by retrograde LV approach before application of radiofrequency energy. The ablation catheter was frozen in place, and rescue radiofrequency energy was delivered immediately (arrow). Complete VA block was noted in the electrophysiological study at the end of the procedure and 1 week later (not shown). ABL indicates ablation catheter; other abbreviations as in Fig 3.

**Fig 5.** Catheter-induced mechanical trauma in a patient with a manifest right posteroseptal accessory pathway (patient 14). When the large-tip ablation catheter was manipulated in the right posteroseptal area through the femoral vein approach, it happened to be placed at the optimal ablation site, with local electrogram showing fusion of A and V waves with possible interpolated accessory pathway potential (arrowheads). The mechanical trauma resulted in progressive loss of preexcitation, as evidenced by the progressive diminution of the delta waves in the surface ECG and by the gradual prolongation of the A-AP interval from the first to the fourth beat. Preexcitation was completely lost with the disappearance of accessory pathway potential from the fifth beat. Rescue radiofrequency energy was applied immediately (not shown). ABL indicates ablation catheter; AP, accessory pathway potential; MCS, middle coronary sinus; PCS, proximal coronary sinus; HRA, high right atrium; and HBE, His bundle electrogram.
highly variable courses of recovery from mechanical trauma. These seldom occurred within 1 week. One third experienced recurrence within 3 months, but the others had apparent long-term cures, lasting up to 38 months in 1 patient. Some patients received rescue radiofrequency ablation, on the presumption that the mechanical trauma site might be a good marker for ablation. Still, the recurrence rate was high. It appeared that the mechanical trauma was caused by the shaft, not by the tip, of the electrode catheter in certain patients.

Role of Pharmacological Agents

Adenosine was frequently administered to unmask preexcitation during electrophysiological study. Isoproterenol was known to improve conduction of cardiac tissues and increase the inducibility of cardiac arrhythmias. Because resting vagal tone exerts a direct depressant effect on conducting tissues, atropine was also administered to improve the conduction of accessory pathways and to facilitate the induction and maintenance of AV nodal reentrant tachycardia and atrial tachycardia. However, these pharmacological manipulations were generally unwarranted, since only 2 patients responded to them: 1 to isoproterenol and 1 to adenosine (indirect sympathetic activation could not be excluded). Besides, more than one fourth of the patients had recurrent tachycardia within 3 months despite nonresponse to pharmacological agents in electrophysiological study. These findings indicated that such responses in patients with catheter-induced mechanical trauma were variable.

Study Limitations

We have included all catheter-induced disappearance of conduction in the present study. However, occurrence of immediate recovery of conductive property could not be accurately determined because more than 40% of patients received rescue radiofrequency ablation on the spot. If more patients with mechanical trauma, such as those with right-sided or septal accessory pathways, were included and no rescue radiofrequency ablation had ever been given, the occurrence of immediate recovery might be higher, as in another study. The true recurrence rate for catheter-induced mechanical trauma may well have been underestimated because some patients have had only a 6-month follow-up and follow-up electrophysiological studies have not been done in all patients after their discharges. Longer observation will be essential to the report of long-term outcomes.

Conclusions

Catheter-induced mechanical trauma was uncommon in patients receiving radiofrequency ablation for supraventricular tachycardia. Its clinical courses were variable, but a long-term cure could be expected in more than half of the patients. This study demonstrated that trauma episodes were more often caused by use of large-tip ablation catheters but that the presumed site of mechanical trauma was not a good marker for ablation. Pharmacological agents were generally unable to revive the traumatized tissues, nor could their use ensure against recurrence. A need for great caution is suggested in the placement of electrode catheters in any patient during electrophysiological study and radiofrequency ablation.

Acknowledgments

This study was supported in part by grants from the National Science Council (NSC 81-0412-B-075-525, 82-0115-B-075-110, and 83-0412-B-075-028) and Academia Sinica, Taipei, Taiwan, ROC.

References


Circulation. 1994;90:1847-1854
doi: 10.1161/01.CIR.90.4.1847

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1994 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/90/4/1847

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/