PATTERNS OF INTERRUPTION OF ATRIAL FLUTTER INDUCED BY RAPID ATRIAL PACING

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We evaluated the patterns of interruption of atrial flutter (AFL) induced by rapid atrial pacing in 10 patients using standard electrophysiologic techniques. We observed 3 patterns of interruption of AFL: 1) interruption resulting from block of an orthodromic wavefront within the reentry loop in 5 patients; 2) interruption when pacing impulses no longer captured all of the recording sites in the atrium during rapid atrial pacing in 2 patients, and 3) interruption with 1 echo wave after the cessation of pacing in 3 patients. These findings suggest that there are patterns of interruption of AFL other than that resulting from a simple block of an orthodromic wavefront within the reentry loop.

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The most common underlying mechanism of atrial flutter (AFL) has been found to be macrorrentrny. AFL can be interrupted when an orthodromic wavefront within the reentry loop is blocked. In the present study, we used standard electrophysiologic techniques to evaluate the patterns of AFL interruption induced by rapid atrial pacing.

SUBJECTS AND METHODS

After informed consent was obtained, we used standard electrophysiologic techniques to evaluate the patterns of AFL interruption induced by rapid atrial pacing in 10 subjects (4 with common type AFL and 6 with uncommon type AFL) (Table I). The study population consisted of 2 men and 8 women aged 2—75 years (mean 56 years). Associated heart diseases included sick sinus syndrome in 6 patients, complete atrioventricular (AV) block in 2, and pericarditis in 1 patient. At the time of the study, none of the patients was receiving antiarrhythmic drugs.

Quadripolar electrode catheters with an interelectrode distance of 1 cm were inserted percutaneously and positioned with fluoroscopic guidance in the high right atrium (HRA), low right atrium (LRA), right ventricular apex (RV), and coronary sinus (CS). An esophageal lead (Eso) was substituted for the left atrial (LA) recording. All of the electrograms were recorded using an ink-jet recorder (Nihon Kohoden, Tokyo, Japan) at a paper speed of 100 mm/sec. ECG leads I, II, V1 and V6 were recorded simultaneously.

Atrial pacing was performed with a programmable stimulator (BC-02, Fukuda Denshi, Tokyo, Japan) using rectangular stimuli at twice diastolic threshold and 2 msec duration.

Rapid atrial pacing was also performed us-

Key words:
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TABLE I PROFILES OF THE 10 PATIENTS WITH AFI

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Associated heart disease</th>
<th>Common type or Uncommon type</th>
<th>Cycle length of AFI (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>74</td>
<td>F</td>
<td>AV block</td>
<td>C</td>
<td>290</td>
</tr>
<tr>
<td>2</td>
<td>75</td>
<td>M</td>
<td>AV block</td>
<td>C</td>
<td>200</td>
</tr>
<tr>
<td>3</td>
<td>60</td>
<td>F</td>
<td>SSS</td>
<td>U</td>
<td>300</td>
</tr>
<tr>
<td>4</td>
<td>68</td>
<td>F</td>
<td>SSS</td>
<td>U</td>
<td>240</td>
</tr>
<tr>
<td>5</td>
<td>2</td>
<td>F</td>
<td>SSS</td>
<td>U</td>
<td>280</td>
</tr>
<tr>
<td>6</td>
<td>60</td>
<td>F</td>
<td>Lone AFI</td>
<td>U</td>
<td>210</td>
</tr>
<tr>
<td>7</td>
<td>10</td>
<td>F</td>
<td>SSS</td>
<td>U</td>
<td>230</td>
</tr>
<tr>
<td>8</td>
<td>75</td>
<td>F</td>
<td>SSS</td>
<td>C</td>
<td>290</td>
</tr>
<tr>
<td>9</td>
<td>64</td>
<td>M</td>
<td>CP</td>
<td>C</td>
<td>230</td>
</tr>
<tr>
<td>10</td>
<td>68</td>
<td>F</td>
<td>SSS</td>
<td>U</td>
<td>240</td>
</tr>
</tbody>
</table>

AV block: atrioventricular block, SSS: sick sinus syndrome, CP: constrictive pericarditis

TABLE II PATTERNs OF INTERRUPTION OF AFI INDUCED BY RAPID ATRIAL PACING

<table>
<thead>
<tr>
<th>Patient</th>
<th>Block CL</th>
<th>Pattern of interruption</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>180</td>
<td>block of the orthodromic wave front</td>
</tr>
<tr>
<td>2</td>
<td>150</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>180</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>200</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>210</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>140</td>
<td>interruption without capture</td>
</tr>
<tr>
<td>7</td>
<td>160</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>260</td>
<td>interruption with an echo wave</td>
</tr>
<tr>
<td>9</td>
<td>160</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>200</td>
<td></td>
</tr>
</tbody>
</table>

Block CL: paced cycle length that achieved AFI interruption

ing 10—13 impulses at 10 msec cycle length decrements, beginning at a cycle length slightly shorter than that of AFI and continuing to conversion into sinus rhythm. Patients whose AFIs were converted into sustained atrial fibrillation during rapid pacing were excluded from this analysis.

RESULTS

The flutter cycle length ranged from 200 to 300 msec (251 ± 39 msec, Table I) and the pacing cycle length that interrupted AFI ranged from 140 to 260 msec (182 ± 37 msec) (Table II). Three patterns of AFI interruption were observed: 1) AFI interruption resulting from the block of an orthodromic wavefront within the reentry loop (Figs. 1, 2) in 5 patients; 2) interruption when stimuli suddenly ceased to capture all of the recording sites in the atrium during rapid atrial pacing (Figs. 3, 4) in 2 patients; and 3) interruption with 1 echo wave after cessation of pacing (Figs. 5, 6, 7) in 3 patients. In 1 patient whose AFI was interrupted by the third pattern, rapid atrial pacing converted 1 type of AFI into another type of AFI. In this patient, the second type of AFI was interrupted by rapid pacing with 1 echo wave. The electrogram morphology of the echo wave in all of the atrial recording sites was similar to that of the initial AFI; the atrial activation sequence produced by the echo wave was identical to that of the initial AFI. In the other 2 patients who exhibited interruption with 1 echo wave, the echo wave was a new flutter wave.

Examples are shown in Fig. 1 to 7.

Fig. 1 shows constant fusion beats on the ECG during transient entrainment of AFI in a patient whose AFI was interrupted by the first pattern (patient No. 1). The last entrained atrial beat (*) at the HRA and LRA immediately followed the last stimulus artifact. The last entrained beat at the LA (Eso) was 1 cycle length beyond the last stimulus artifact. On cessation of pacing, there was a long pause at a cycle length that clearly exceeded the pacing cycle length at all of the recording sites except for the LA (Eso) site, where the cycle length 1 beat beyond the last pacing cycle was identical to the pacing cycle length. The morphology of the atrial complexes recorded at the LA (Eso) during pacing was similar to that of

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Patterns of Interruption of Atrial Flutter

Fig. 1. Recording of rapid atrial pacing at a cycle length of 200 msec in a patient whose AFI was interrupted by the first pattern (patient No. 1) during common type AFI with a cycle length of 290 msec. Constant fusion was observed in the ECG leads. The last entrained atrial beat (*) at the HRA and LRA immediately follows the last stimulus artifact. However, the last entrained beat at the LA (Eso) was 1 cycle length beyond the last stimulus artifact. This interpretation is supported by the fact that with cessation of pacing, there is a long pause at a cycle length that clearly exceeds the pacing cycle length at all of the recording sites, except the LA (Eso), where the cycle length 1 beat beyond the last pacing cycle was identical to the pacing cycle length. The morphology of the atrial complexes recorded at the LA (Eso) during pacing was similar to that of the AFI. (St: stimulus, HRA: high right atrium, LRA: low right atrium, LA: left atrium, Eso: esophageal lead, A: atrial wave, V: ventricular wave)

Fig. 2. AFI interruption by rapid atrial pacing at a cycle length of 180 msec in the patient represented in Fig. 1. Progressive fusions were initially observed in the ECG leads. Thereafter, the conduction time from the stimuli to the LA (Eso) site gradually increased, and then suddenly decreased from 320 msec to 190 msec. The morphology of the atrial complexes also changed.

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Fig. 3. Recording of rapid atrial pacing at a cycle length of 180 msec in a patient whose AFI was interrupted by the second pattern (patient No. 7) during an uncommon type AFI with a cycle length of 230 msec. The last captured arial beat (*) at the HRA, LRA, RA septal and LA (Eso) immediately followed the last stimulus artifact. Every other pacing was blocked, and the conduction time from the stimuli to all of the recording sites in the atrium was decreased. On cessation of pacing, the AFI was interrupted.

Fig. 4. AFI interruption by rapid atrial pacing at a cycle length of 160 msec in the patient represented in Fig. 3.

AFI, indicating that the LA (Eso) site was activated orthodromically within the reentry loop with a long conduction time during pacing and that the LA site was activated by a wavefront of activation distal to the area of a slow conduction within the reentry loop.

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Fig. 5. Recording of a rapid atrial pacing at a cycle length of 200 msec in a patient whose AFI was interrupted by the third pattern (patient No. 9) during an common type of AFI with a cycle length of 230 msec. The asterisks indicate the last entrained atrial beat that immediately followed the last stimulus artifact. However, the last entrained beat at the SLRA was 1 cycle length beyond the last stimulus artifact. On cessation of pacing, there is long pause at a cycle length that clearly exceeds the pacing cycle length at all of the recording sites except the SLRA, where the cycle length 1 beat beyond the last pacing cycle was identical to the pacing cycle length. The morphology of the atrial complexes recorded at the SLRA during pacing was similar to that of the AFI. The atrium was activated during AFI in the following sequence: SLRA, HRA, LA (Eso), LRA.

Fig. 2 shows AFI interruption in the patient represented in Fig. 1. Progressive fusion was initially observed in the ECG leads. Thereafter, the conduction time from the stimuli to the LA (Eso) site gradually increased, and then suddenly decreased from 320 msec to 190 msec. The morphology of the atrial complexes also changed, indicating that AFI was interrupted by a block of an orthodromic wavefront within the reentry loop between the pacing site and the LA site, where slow conduction area is considered to be located.

Fig. 3 shows rapid atrial pacing during AFI in a patient whose AFI was interrupted by the second pattern (patient No. 7). The last captured atrial beats at the HRA, LRA, RA septal and LA (Eso) immediately followed the last stimulus artifact, indicating that all of the recording sites were activated by a wavefront proximal to the area of slow conduction.

Fig. 4 shows AFI interruption in the patient represented in Fig. 3. Every other pacing was blocked and the conduction time from the stimuli to all of the recording sites in the atrium was decreased. On cessation of pacing, AFI was interrupted.

Fig. 5 shows constant fusion beats in the ECG during transient entrainment of AFI in a patient whose AFI was interrupted by the third pattern (patient No. 9). The last entrained atrial beats at the HRA, LRA and LA (Eso) immediately followed the last stimulus artifact. However, the last entrained beat at the SLRA was 1 cycle length beyond the last stimulus artifact. On cessation of pacing, there was a long pause at a cycle length that clearly exceeded the pacing cycle length at all of the recording sites except for the SLRA site, where the cycle length 1 beat beyond the last pacing cycle was identical to the pacing cycle length. The morphology of the atrial complexes recorded at SLRA during pacing was similar to that of the AFI, indicating that the SLRA site was activated orthodromically with a long conduction time through an area of slow conduction.
duction within the reentry loop during pacing. During AFL, the atrium was activated in the following sequence: SLRA, HRA, LA (Eso), LRA. Subsequently, rapid atrial pacing at an increased rate converted AFL into transient atrial fibrillation. AFL then spontaneously converted into another common type of AFL.

Fig. 6 shows rapid atrial pacing during another AFL in the patient represented in Fig. 5. The last captured atrial beats at the HRA, SLRA, LRA, and LA (Eso) immediately followed the last stimulus artifact. The morphology of the atrial complexes recorded at SLRA during pacing was similar to that of the AFL, indicating that the SLRA site was activated orthodromically with a short conduction time during pacing and that the SLRA site was activated by a wavefront proximal to an area of slow conduction within the reentry loop. The atrial activation sequence in this AFL was: HRA, LRA, SLRA, LA (Eso), which differed from that of the initial AFL.

Fig. 7 shows AFL interruption in the patient represented in Fig. 5. The captured atrial beats at the HRA, LRA, SLRA, and LA (Eso) immediately followed each stimulus artifact and all of the recording sites appeared to be activated by a wavefront proximal to an area of slow conduction. However, 1 cycle length beyond the last stimulus artifact at the SLRA site was identical to the pacing cycle length, which differed from the entrainment previously observed in Fig. 5. Thus, the last captured beat at SLRA appeared to be 1 cycle length beyond the last stimulus artifact. It was not clear if the SLRA was activated orthodromically with a short or long conduction time. The morphology and the activation sequence of the last captured atrial beat at the SLRA and the subsequent atrial beat recorded at other sites (echo wave) were similar to those of the initial AFL represented in Fig. 5. This echo wave was blocked between the LRA and SLRA sites. The AFL was interrupted at the point where a slow conduction area was be-

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Fig. 7. AFI interruption with 1 echo wave by rapid atrial pacing at a cycle length of 160 msec in the patient represented in Fig. 5. The entrained atrial beat at the HRA, LRA, SLRA, and LA (Eso) immediately follows each stimulus artifact and orthodromic activation of a site through an area of slow conduction was not recorded. However, the last entrained beat (*) at the SLRA is suggested to be 1 cycle length beyond the last stimulus artifact. On cessation of pacing, there is long pause at a cycle length that clearly exceeds the pacing cycle length at all of the recording sites except the SLRA, where the cycle length 1 beat beyond the last pacing cycle is identical to the pacing cycle length. The morphology and the activation sequence of the last entrained atrial beat at SLRA and the subsequent atrial beat at other sites (echo wave) is similar, if not identical, to that of the initial AFI represented in Fig. 5. This echo wave was blocked, and AFI was interrupted, between the LRA and SLRA sites, where the slow conduction area in the reentry loop of the initial AFI is believed to be located.

Believed to be located within the reentry loop of the initial AFI.

**DISCUSSION**

The most common explanation for AFI is based on a macroreentry mechanism. AFI is generally considered to be interrupted by a block of an orthodromic wavefront within the reentry loop.

We observed 3 patterns of AFI interruption.

In 1 pattern, interruption of AFI by rapid atrial pacing was associated with a localized conduction block to a site, followed by activation of that site by the next pacing impulse from a different direction and with a shorter conduction time. This pattern of AFI interruption is consistent with the third entrainment criterion proposed by Waldo et al. and is generally supported by most investigators.

In the second pattern, AFI was interrupted after the stimuli ceased to capture all of the recording sites in the atrium during rapid atrial pacing. In this pattern, the activation conduction time from the pacing site to all of the recording sites was short, and we could not observe the orthodromic activation of any site through an area of slow conduction within the reentry loop. After every other pacing was blocked, the conduction time from the stimuli to all of the recording sites in the atrium decreased. However, it is not
clear if this decrease was associated with a block of an orthodromic wavefront within the reentry loop.

In the third pattern, AFI was interrupted with 1 echo wave immediately after cessation of rapid atrial pacing. In this pattern, the entrained atrial beats at the HRA, LRA, SLRA, and LA (Eso) immediately followed each stimulus artifact and we did not observe the orthodromic activation of a site through an area of slow conduction. It is not clear if an orthodromic wave front within the reentry loop was blocked during rapid atrial pacing. However, the last entrained beat at the SLRA site appeared to be 1 cycle length beyond the last stimulus artifact. The morphology and the activation sequence of the last captured atrial beat at the SLRA and the subsequent atrial beat recorded at other sites (echo wave) were similar to those of the initial AFI represented in Fig. 5. The echo wave was blocked and AFI was interrupted. These features suggest that AFI was interrupted while being converted from one type of AFI to another AFI. However, it is unclear when this transfer occurred. The mechanism of this pattern of interruption is also unclear. There are other possible explanations for this pattern. For example, premature atrial contraction may have occurred by chance just after pacing was discontinued. However, this explanation is unlikely.

The ability to map the right atrium precisely was limited by the techniques used in this study. Although we observed 3 patterns of AFI interruption, we were unable to clarify the mechanisms of the second and third patterns because of the inherent limitations of the study techniques. Our findings suggest that there are mechanisms of AFI interruption other than the simple block of an orthodromic wavefront within the reentry loop.

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