Retrograde Ventriculoatrial Conduction in Patients with Acute Myocardial Infarction*

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Temporary transvenous pacemakers were inserted in ten patients with acute myocardial infarction complicated by atrioventricular (A-V) block. With return of sinus rhythm, the ventricular rate was intentionally increased above sinus rate by graded increases of the pacemaker rate and stimulus amplitude to a maximum of 120 beats/minute and to 12 milliamperes for each paced rate. Four patients had ventriculoatrial (V-A) conduction at paced rates greater than the sinus rate. Two of four had V-A conduction at slower rates (80-100 beats/minute), but A-V dissociation developed with more rapid rates (100-140). Three of six patients who had sinus rhythm with normal PR interval demonstrated V-A conduction while only one of four with first degree block showed retroconduction. The pattern of conduction was independent of stimulus amplitude. Retrograde V-A conduction occurs commonly in patients with acute myocardial infarction with slow ventricular rates (80-100 beats/minute) and less commonly with rapid rates.

Correct diagnosis of ventricular arrhythmias is often of critical importance in the management of patients with acute myocardial infarction. The electrocardiographic differentiation of ventricular tachycardia from aberrantly conducted supraventricular rhythms may be extremely difficult. For example, atrioventricular (A-V) dissociation, traditionally considered one of the hallmarks in the diagnosis of ventricular tachycardia, can occur in patients with A-V junctional rhythms. Furthermore, recent studies have documented the frequent occurrence of retrograde ventriculoatrial (V-A) conduction in ventricular tachycardia.

The latter studies were performed during cardiac catheterization of patients mostly with pulmonary or valvular heart disease. The most common etiology of ventricular tachycardia is acute myocardial infarction and the frequency of V-A conduction in this group is unknown. The purpose of our study was to determine the incidence of retrograde V-A conduction in patients with acute myocardial infarction.

METHODS

Temporary transvenous pacing was accomplished by means of a bipolar electrode catheter (no. 6 Goetz) inserted into the apex of the right ventricle in all patients with acute myocardial infarction complicated by A-V block. With return of sinus rhythm, the pacemaker was set on demand mode at a rate less than the sinus rate. The catheter was removed three days after a stable sinus rhythm and a normal heart rate were achieved. Just prior to removal of the pacing catheter the ventricular rate was increased above the sinus rate by graded increases in the pacemaker rate to a maximum of 120 beats/minute. In addition, the electrical stimulus amplitude was gradually increased to a maximum of 12 milliamperes at each pacing rate.

The "P" waves were monitored by either esophageal or right atrial electrograms. The latter were obtained by filling a catheter (previously inserted into the right atrium for monitoring central venous pressure and/or oxygen saturation) with hypertonic saline and connecting a metal three-way stopcock at the end of the catheter to the central terminal of the electrocardiograph with alligator clamps. Esophageal or right atrial electrograms were recorded during sinus rhythm and at varying paced rates with the electrocardiograph dial in the "V" position. The QRS complex was defined by the preceding pacing artifact.

Both the electrocardiograph and the pacing unit were connected to a common ground outlet and the electrocardiograph was carefully checked for significant electrical ground "leaks" prior to each study.

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†Abbott 3% NaCl solution.

‡Electronics for Medicine pacemaker PAI-8.

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RESULTS

Over an 18-month period, 194 patients with acute myocardial infarction were treated in the coronary care unit at the San Francisco General Hospital. Twenty of the patients had bradyarrhythmias due to either advanced A-V block* or A-V dissociation due to partial or complete A-V block and underwent temporary transvenous pacing by a bipolar electrode catheter inserted into the apex of the right ventricle. One patient remained in complete A-V block, nine patients died, and ten patients had return of sinus rhythm and survived. The last group of patients were studied after informed consent was obtained. The patients were urged to tell immediately of any symptoms of chest discomfort or dyspnea during the procedure.

Table 1 summarizes the data collected. All patients showed electrocardiographic evidence of acute inferior or inferolateral myocardial infarction. Six patients were studied one week after insertion of the catheter pacemaker and the remainder, within two weeks. The catheter was inserted within 24 hours of acute myocardial infarction in seven patients and within 72 hours in three.

*Advanced A-V block—Mobitz type II block in which the conduction ratio is 2:1 or less.

<p>| Table 1 — Pattern of Conduction and Related Data in Patients During Ventricular Pacing. |
|---------------------------------|---------------------------------|---------------------------------|---------------------------------|---------------------------------|---------------------------------|</p>
<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>Location of myocardial infarction</th>
<th>Day of study after admission</th>
<th>Initial electrocardiographic data</th>
<th>Slowest ventricular rate</th>
<th>After return to 1:1 conduction</th>
<th>Pacing rates and pattern of conduction*</th>
</tr>
</thead>
<tbody>
<tr>
<td>78</td>
<td>Inferior</td>
<td>14</td>
<td>A-VD due to block</td>
<td>Nodal</td>
<td>48</td>
<td>Normal PR interval</td>
</tr>
<tr>
<td>62</td>
<td>Inferior</td>
<td>7</td>
<td>Advanced A-V block</td>
<td>Nodal</td>
<td>46</td>
<td>Normal PR interval</td>
</tr>
<tr>
<td>42</td>
<td>Inferior</td>
<td>11</td>
<td>A-VD due to block</td>
<td>Nodal</td>
<td>54</td>
<td>Normal PR interval</td>
</tr>
<tr>
<td>63</td>
<td>Inferior</td>
<td>3</td>
<td>Advanced A-V block</td>
<td>Nodal</td>
<td>50</td>
<td>Normal PR interval</td>
</tr>
<tr>
<td>52</td>
<td>Inferolateral</td>
<td>5</td>
<td>CHB</td>
<td>Ventricular</td>
<td>40</td>
<td>Normal PR interval</td>
</tr>
<tr>
<td>84</td>
<td>Inferior</td>
<td>7</td>
<td>CHB</td>
<td>Nodal</td>
<td>38</td>
<td>1st block</td>
</tr>
<tr>
<td>82</td>
<td>Inferolateral</td>
<td>14</td>
<td>CHB</td>
<td>Ventricular</td>
<td>34</td>
<td>1st block</td>
</tr>
<tr>
<td>53</td>
<td>Inferior</td>
<td>4</td>
<td>A-VD due to block</td>
<td>Nodal</td>
<td>52</td>
<td>1st block</td>
</tr>
<tr>
<td>41</td>
<td>Posterior inferior</td>
<td>9</td>
<td>A-VD due to block</td>
<td>Nodal</td>
<td>54</td>
<td>1st block</td>
</tr>
</tbody>
</table>

* A-V = atioventricular, D = dissociation, CHB = complete heart block, V-A = ventriculoatrial, and C = conduction
†After return of sinus rhythm
‡A-VD developed when paced at a rate of 140 beats/minute.

Four of the ten patients showed ventriculoatrial conduction at paced rates greater than the sinus rate. Two of the four had V-A conduction at slower rates (80-100 beats/minute) but A-V dissociation developed when the rate was increased. Two patients had both A-V dissociation and retrograde V-A conduction at rates of 85-95 beats/minute, but exhibited retrograde conduction alone with more rapid rates (Fig 1).

Three of six patients with sinus rhythm and a normal PR interval showed retrograde conduction (Table 1) while only one of four patients with first degree block showed retrograde V-A conduction. However, A-V dissociation occurred in this patient when the paced rate was inadvertently increased to 150 beats/minute (Fig 2).

The pattern of conduction in all patients was independent of the stimulus amplitude. Patients with A-V dissociation did not show V-A conduction when the stimulus amplitude was gradually increased to 12 milliamperes, nor did V-A conduction change at a given heart rate with graded increases in stimulus strength. In patients showing retrograde V-A conduction the RP interval was usually greater than the PR interval and increased with increases in heart rates (Fig 2).
Two patients noted palpitations at the highest paced rate, but no patient complained of angina or dyspnea and no new arrhythmias developed during or after the study. All of the subjects survived and eventually left the hospital.

**DISCUSSION**

Electrocardiographic analysis seldom allows for a definitive diagnosis of ventricular tachycardia; hence in our present study ventricular rhythms were induced by a bipolar electrode catheter inserted into the right ventricle in patients with acute myocardial infarction complicated by A-V block. After reversion to sinus rhythm, patients were paced at a rate greater than the sinus rate in order to determine the presence of retrograde V-A conduction. Retrograde conduction was demonstrated in four of the ten patients studied when the right ventricle was paced at 85-95 beats/minute. Only three patients showed retrograde conduction at paced rates of 105-120 beats/minute and one of the three failed to exhibit V-A conduction when the pacer was accidentally set at 150 beats/minute. The pattern of conduction at any given rate was independent of the amount of current used to induce the ventricular rhythm.

This study demonstrated the frequent occurrence of retrograde conduction in patients with acute inferior myocardial infarction initially complicated by A-V block. It is reasonable to assume that retrograde conduction is at least as common in patients with acute myocardial infarction uncomplicated by electrocardiographic evidence of damage to nodal- or infranodal-conducting tissue. Previous studies of V-A conduction were performed largely in patients with valvular or pulmonary disease and demonstrated the frequent occurrence of retrograde conduction in catheter-induced ventricular tachycardia. The present study extends these observations to patients with acute myocardial infarction and clearly shows that arrhythmias of ventricular
origin occurring in these patients may or may not be associated with A-V dissociation.

The incidence of retrograde conduction was less in the patients who reverted to first degree block compared to those who had a normal PR interval. The small number of patients in our study makes these observations of only marginal statistical significance, but other investigators have reported similar

findings in larger numbers of subjects with varying cardiac diagnoses.

Although the pacing rates used in this study were well tolerated by the patients, it was thought unwise to increase the paced ventricular rate to ranges usually observed in spontaneous ventricular tachycardia (ie 150-250 beats/minute). However, other investigators have documented the frequent occurrence (36 percent) of slower ventricular rhythms (60-100 beats/minute) in patients with acute myocardial infarction. Our data would appear to indicate that the incidence of retrograde V-A conduction is quite common (at least 40 percent) in patients with acute inferior myocardial infarction complicated by relatively slow ventricular rates and less common with more rapid ventricular rates.

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