Atrial Pacing in Coronary Artery Disease

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SUMMARY

Atrial pacing was undertaken in 11 patients with coronary artery disease. Angina pectoris occurred in 6 patients. Six of the patients also developed atrioventricular conduction disturbances; the commonest was the Wenckebach block.

Atrial pacing proved to be a relatively simple, safe, and reproducible procedure to assess the angina threshold in patients with coronary artery disease.


It can be difficult to make an objective assessment of the severity of angina pectoris. The angina threshold is determined by important psychological factors. Some patients exaggerate their symptoms, others accept their disability and limit their activities, while placebo therapy is often effective in controlling the pain.

There are several objective methods of evaluating angina pectoris—exercise, atrial pacing, and isoprenaline infusion.

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The heart rate can be increased by atrial pacing.¹ This produces significant myocardial stress as the pressure-time/ min increases.² In the presence of coronary artery disease this leads to regional myocardial hypoxaemia and dysfunction and alteration in left ventricular compliance and in left ventricular end-diastolic pressure;² this may exaggerate the local ischaemic effect. Both mechanisms produce angina pectoris.

We used atrial pacing in patients with coronary artery disease to determine the value of the method to measure the threshold for angina pectoris, to assess its reproducibility and to evaluate the effect of intravenous beta-adrenergic blocking drugs.

PATIENTS AND METHODS

We studied 11 consecutive patients with significant coronary artery disease.

The clinical data are shown in Table I and the clinical diagnosis was confirmed by cardiac catheterization and selective coronary angiography.⁶,¹¹

Oral beta-adrenergic blocking agents were stopped 1 week before the study. Four patients received maintenance digitalis therapy.

TABLE I. THE PATIENTS

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (years)</th>
<th>Race</th>
<th>Sex</th>
<th>Symptoms</th>
<th>ECG</th>
<th>Regional asynergy</th>
<th>Coronary angiography (sites of significant obstruction)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>35</td>
<td>I</td>
<td>M</td>
<td>Previous infarction, no angina</td>
<td>Diaphragmatic infarction</td>
<td>Yes</td>
<td>RCA</td>
</tr>
<tr>
<td>2</td>
<td>45</td>
<td>I</td>
<td>M</td>
<td>Angina</td>
<td>Anterolateral infarction</td>
<td>Yes</td>
<td>RCA</td>
</tr>
<tr>
<td>3</td>
<td>43</td>
<td>E</td>
<td>M</td>
<td>Angina</td>
<td>Trans-septal infarction</td>
<td>No</td>
<td>RCA</td>
</tr>
<tr>
<td>4</td>
<td>53</td>
<td>E</td>
<td>M</td>
<td>Trivial angina</td>
<td>Normal</td>
<td>No</td>
<td>RCA</td>
</tr>
<tr>
<td>5</td>
<td>52</td>
<td>E</td>
<td>M</td>
<td>Angina</td>
<td>Anterior infarction</td>
<td>Yes</td>
<td>RCA</td>
</tr>
<tr>
<td>6</td>
<td>36</td>
<td>E</td>
<td>M</td>
<td>Angina</td>
<td>Anterior infarction</td>
<td>Yes</td>
<td>RCA</td>
</tr>
<tr>
<td>7</td>
<td>62</td>
<td>E</td>
<td>M</td>
<td>Angina</td>
<td>Diaphragmatic infarction</td>
<td>Yes</td>
<td>RCA</td>
</tr>
<tr>
<td>8</td>
<td>62</td>
<td>I</td>
<td>M</td>
<td>Angina</td>
<td>Numerous VPS; LBBB + LAHB</td>
<td>Yes</td>
<td>RCA</td>
</tr>
<tr>
<td>9</td>
<td>56</td>
<td>I</td>
<td>M</td>
<td>Angina</td>
<td>Diaphragmatic infarction + LPHB</td>
<td>Yes</td>
<td>Circumflex</td>
</tr>
<tr>
<td>10</td>
<td>35</td>
<td>E</td>
<td>M</td>
<td>Angina</td>
<td>Anterior infarction</td>
<td>Yes</td>
<td>RCA</td>
</tr>
<tr>
<td>11</td>
<td>31</td>
<td>I</td>
<td>M</td>
<td>Angina</td>
<td>Anteroseptal infarction</td>
<td>Yes</td>
<td>RCA</td>
</tr>
</tbody>
</table>

RCA = right coronary artery; AD = anterior descending coronary artery; VPS = ventricular premature systole; LBBB = left bundle-branch block; LPHB = left posterior hemiblock; LAHB = left anterior hemiblock.
A C52 6F(USCI) bipolar, pervenous, pacing, electrode-wire was passed from a peripheral vein, and the tip placed in a stable position against the lateral wall of the right atrium near the entrance of the superior vena cava. The position of the tip was identified by biplane fluoroscopy. The 3 standard leads of the electrocardiograph (ECG) were recorded on a 6-channel polygraph (Hellige multiscriptor) at speeds of 25 mm/sec and 50 mm/sec. A control ECG was recorded at rest.

The atrium was then driven at faster rates using a Cordis variable-rate external pacemaker. The rate was increased by increments of 10 beats/min until angina developed or until a rate of 160 - 180 beats/min was achieved. Pacing was continued at each rate for 2 minutes and then the rate was increased. The patient was rested for 10 minutes after the first procedure, which was then repeated to assess reproducibility. The patients were again rested for 10 minutes and beta-adrenergic blocking drugs were given intravenously. Five patients were given 2 mg oxprenolol (Trasicor) and 1 patient was given 10 mg practolol. The pacing study was repeated. The patients, again after resting for 10 minutes, were given a further dose of each drug. The patients were then paced for a fourth time.

**RESULTS**

The results are summarized in Table II. Angina pectoris was induced in 6 patients and the pacing rate which corresponded to the anginos threshold was reproducible. Angina disappeared within 2 minutes after pacing had stopped. There was no improvement in the angina threshold after administration of beta-blocking agents during the third or fourth pacing study; in 3 patients angina occurred at lower rates.

Atrioventricular conduction disturbances occurred in 6 patients; the PR interval was prolonged in 2, 1st degree A-V block occurred in 1, and in 3 patients 2nd degree heart block with the Wenckebach phenomenon was induced. The heart rate at which these changes occurred was reproducible and was not altered by administration of the beta-blocking drug. One of these 6 patients was receiving digitalis therapy.

**DISCUSSION**

Atrial pacing is a simple and useful objective technique to assess angina pectoris and to make a definitive diagnosis in the circumstances of nonspecific chest pain.

Atrial pacing increases the heart rate. The cardiac index is not altered and the stroke volume falls. Mean aortic diastolic pressure does not change and in the normal subject the left ventricular end-diastolic pressure decreases. Although left ventricular ejection time is reduced, the overall mean pressure-time/min increases significantly and this increases the myocardial need for oxygen. Moreover, there is a decrease in the diastolic filling period so that any increase in coronary blood flow occurs during a shorter period. Angina pectoris appears when the coronary vascular reserve is unable to accommodate the additional blood flow. Moreover, when coronary vascular disease is present, regional ischaemia impairs left ventricular performance, there is a rise in left ventricular end-diastolic pressure, and this further compromises left ventricular function.19

Our studies have shown that atrial pacing induces angina in some patients. If coronary disease is mild then the additional oxygen which is needed during atrial pacing may not exceed the angina threshold and in these patients exercise, with its greater demand for oxygen, may induce angina. Thus a negative atrial pacing test does not exclude angina pectoris. However, atrial pacing is much simpler to perform; the heart rate at which angina occurs is reproducible, and the pain stops when pacing is stopped. It is much safer than an exercise test.

Many patients have an unusual distribution of pain. The unusual pain could be induced in every patient and was undoubtedly cardiac in origin.

Conduction defects occurred in 6 of the 11 patients. Five had significant obstruction of the right coronary artery and the abnormal conduction time was a consequence of impaired blood supply to the atrial wall or atrioventricular

<table>
<thead>
<tr>
<th>Patient</th>
<th>Angina threshold</th>
<th>Before beta-blocker</th>
<th>After beta-blocker</th>
<th>Conduction defects produced</th>
<th>Rate</th>
<th>Drug and dose</th>
<th>Effect of drug</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>No angina (160)</td>
<td>100</td>
<td>70</td>
<td>Wenckebach</td>
<td>160</td>
<td>Nil</td>
<td>—</td>
</tr>
<tr>
<td>2</td>
<td>No angina (160)</td>
<td>120</td>
<td>Same</td>
<td>Wenckebach</td>
<td>160</td>
<td>Nil</td>
<td>—</td>
</tr>
<tr>
<td>3</td>
<td>No angina (180)</td>
<td>160</td>
<td>140</td>
<td>—</td>
<td>120</td>
<td>Nil</td>
<td>—</td>
</tr>
<tr>
<td>4</td>
<td>Trivial (180)</td>
<td>120</td>
<td>130</td>
<td>PR +</td>
<td>130</td>
<td>Nil</td>
<td>—</td>
</tr>
<tr>
<td>5</td>
<td>100</td>
<td>120</td>
<td>Same</td>
<td>Wenckebach</td>
<td>160</td>
<td>Nil</td>
<td>—</td>
</tr>
<tr>
<td>6</td>
<td>100</td>
<td>160</td>
<td>140</td>
<td>—</td>
<td>120</td>
<td>Nil</td>
<td>—</td>
</tr>
<tr>
<td>7</td>
<td>120</td>
<td>130</td>
<td>Same</td>
<td>—</td>
<td>130</td>
<td>Nil</td>
<td>—</td>
</tr>
<tr>
<td>8</td>
<td>110</td>
<td>130</td>
<td>Same</td>
<td>PR +</td>
<td>130</td>
<td>Nil</td>
<td>—</td>
</tr>
</tbody>
</table>

TABLE II. RESULTS
node. His-bundle electrocardiography, which records the time of arrival of the cardiac impulse in the bundle of His, distinguishes between slow atrioventricular nodal conduction and slow conduction in the distal bundle and ramifications of the Purkinje network. Such studies have shown that the first-degree block and the Wenckebach phenomenon, which may occur in atrial pacing, are consequences of conduction delay in the atrioventricular node.23,24

Intravenous adrenergic beta blockade did not alter the heart rate at which angina was induced. This could be related to the dose of drug used, the method of administration, or the technique of testing the angina threshold. The dose of drug used in this study was inadequate and produced incomplete beta blockade. A dose of 0.2 mg/kg of oxprenolol is needed to abolish the response to an isoprenaline challenge.24 Atrial pacing may also be inadequate to assess the response to beta blockade since these drugs act by reducing heart rate, arterial pressure and velocity of contraction. Beta-blocking agents lower the heart rate on exercise, so that a given workload is achieved at a much lower heart rate. Atrial pacing aboliishes this effect on heart rate and renders ineffective one of the most important actions of the drugs. Moreover, for a therapeutic effect, these drugs are used in large oral doses, and rapid intravenous administration may not be the ideal method of inhibiting an anginous effect.

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REFERENCES

Boeke Ontvang: Books Received


