Abolition of Ischemic Response to Atrial Pacing Following Aortocoronary Bypass Surgery

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SUMMARY To determine the effect of successful aortocoronary bypass surgery (ACBS) on left ventricular (LV) function, the ischemic response to right atrial pacing (RAP) was studied in 22 angina patients before and 3.8 ± 1.1 months after surgery. All patients were free of angina after ACBS and had at least one patent graft. Before ACBS, RAP induced angina in 15 patients (Group 1) but not in 7 patients (Group 2). After ACBS, no patient had angina with RAP despite the increased maximum rate of pacing. Post-operation LV end-diastolic pressure (LVEDP) after ACBS decreased in Group 1 from 25 ± 6 to 15 ± 6 mm Hg (P < 0.01), but not in Group 2. Changes in ejection fraction, cardiac output, resting LVEDP, or LVEDP after LV angiography were not significant in either group and were therefore not useful in evaluating the result of ACBS. However the ischemic response to right atrial pacing was abolished by successful aortocoronary bypass surgery, suggesting improved myocardial perfusion during stress.

SUCCESSFUL AORTOCORONARY BYPASS SURGERY relieves angina in the majority of cases indicating improved myocardial perfusion. However, previously reported studies of the left ventricular function in such patients produced variable results and ejection fraction did not improve consistently. This may be due to the fact that these studies were carried out at rest in the absence of anginal symptoms or myocardial ischemia.

In patients with angina, left ventricular function is frequently normal at rest but becomes abnormal during stress, whether induced by exercise or atrial pacing. In such patients, no improvement from the resting left ventricular function can be expected even after successful revascularization. This study was carried out to compare the left ventricular response to stress induced by atrial pacing before and after aortocoronary bypass. Previous studies have indicated that right atrial pacing is a reliable method of assessing anginal threshold and ventricular function in patients with coronary artery disease.

Method

Patients

Our series consisted of 24 consecutive patients with chronic angina who underwent atrial pacing studies and coronary angiography before and after aortocoronary bypass surgery. All patients were suffering from disabling angina pectoris before operation and were found to have 75% or greater stenosis of one or more major coronary arteries. Patients with congestive heart failure, mitral insufficiency, and those having resection of left ventricular aneurysm were excluded from the study. All patients agreed before surgery to undergo the postoperative studies, which included selective coronary and vein graft angiography and were carried out 3 to 5 months after operation (mean 3.8 months). There were 21 men and 3 women. The average age was 51 ± 7 years. Two male patients had persistent angina after operation and their grafts were found to be occluded. These two patients were excluded from the subsequent analysis as revascularization was unsuccessful. All the patients analyzed were therefore angina free and had at least one patent graft.

Aortocoronary bypass surgery was carried out using the standard technique of cardiopulmonary bypass with normothermic perfusion. The aorta was intermittently cross-clamped during distal anastomosis of each graft. A total of 58 grafts were inserted (2.6 grafts per patient), and 45 grafts were found to be patent and perfusing distal coronary arteries at the postoperative angiography.

A total of eight patients were on propranolol therapy before surgery but the drug was discontinued at least 24 hours before the preoperative investigation in all except four patients (3, 7, 9 and 15); only one (#15) had received the drug within 12 hours of the study. No patient was receiving propranolol at the time of the postoperative investigation. One patient (#22) was receiving digoxin until 24 hours before the preoperative study while five patients were receiving digoxin until 24 hours before the postoperative study.

Hemodynamic Studies

Patients were studied in the fasting state an hour after oral premedication with diazepam (10 mg) and secobarbital sodium (100 mg). Using lidocaine local anesthesia, a no. 6 Courand catheter and a no. 8 pigtail catheter were introduced into the femoral vein and artery respectively using a percutaneous technique. Pressures were recorded using a Statham strain gauge (23Pd) and a photographic recorder (Electronics for Medicine, DR-8). Cardiac output was measured using the Fick principle.

Sequential right atrial pacing was carried out using a bipolar pacing catheter at increasing rates from 90 to 150 beats/min, at increments of 20 beats/min. Patients were paced for two minutes at each rate allowing a brief pause between pacing periods to record left ventricular pressures. The electrocardiogram (lead II) and left ventricular pressures were recorded during pacing and immediately after pacing was abruptly stopped. Left ventricular end-diastolic pressure (LVEDP) was determined at the point of rapid pressure increase after the "a" wave. In cases where "a" waves were not identified, LVEDP was measured at a point corresponding to the peak of the R wave on the electrocardiogram. The pacing rate was increased until definite anginal symptoms or atrioventricular block appeared, or a

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maximum rate of 150 beats/min was reached. We did not use atropine to increase the pacing rate so as to avoid any possible effect it might have on ejection fraction. Ten minutes after the pacing study, left ventricular cineangiography was performed in 30° right anterior oblique position with power injection of 40–48 ml Hypaque in four seconds. A 6-inch image intensifier and 35 mm cinecamera were used and left ventricular pressures were recorded two minutes after the angiography. Selective coronary angiography was then performed by Judkins technique using cineangiography as well as cut-films in multiple projections. At the postoperative study, aortic root angiography was obtained using large cut-films and power injection of 60 ml of 75% Hypaque over 2.5 to 3 seconds. In addition, selective angiography of each graft and coronary artery was performed. A graft was assumed occluded if it failed to opacify on aortic root angiography and could not be selectively cannulated.

Left ventricular volumes were calculated by the area-length method of Sandler and Dodge. Ejection fraction (EF) was calculated using the equation: EF = end-diastolic volume minus end-systolic volume/end-diastolic volume. The calibration factor was obtained by recording the image of a lead impregnated grid (4 x 4 cm) positioned to the left of the ventricular apex.

**Results**

Before surgery, 12 of 22 patients had electrocardiographic evidence of myocardial infarction (11 inferior and three anterior). Two patients (#2 and 13) sustained peri-
operative myocardial infarction as judged by the electrocardiogram and serum enzymes. Of 58 grafts (56 saphenous vein and two free radial artery grafts) inserted, 45 (44 saphenous and one radial artery graft) were patent at the postoperative angiography giving a graft patency rate of 78%. Seven patients had one patent graft, nine patients had two patent grafts and six patients had three or four patent grafts. All patients were free of angina after surgery.

Resting heart rate increased slightly from 68 per min to 75 per min (not significant) after operation (tables 1 and 2). Similarly, there was no significant change in resting cardiac output (5.9 vs 5.6 L/min), resting left ventricular end-diastolic pressure (LVEDP, 13 vs 12 mm Hg), LVEDP after left ventricular angiography (19 ± 6 vs 17 ± 5 mm Hg). Ejection fraction did not change after surgery (55 ± 15% vs 54 ± 12%). The mean ejection fractions were the same whether the patients had developed an ischemic response to atrial pacing before surgery (Group 1) or not (Group 2).

### Atrial Pacing Study

Before surgery, atrial pacing induced anginal symptoms in 15 patients (Group 1) while the remaining seven patients did not experience angina (Group 2). Before operation in Group 1, LVEDP increased significantly from 14 ± 5 mm Hg at rest to 25 ± 6 mm Hg (P < 0.001) immediately after atrial pacing (tables 2 and 3). However, in Group 2, the change was small and not significant (10 mm Hg at rest and 13 mm Hg after pacing). These findings are consistent with other observations\(^5\) that patients who develop myocardial ischemia evidenced by angina or ST-segment depression are characterized by increased LVEDP after pacing.

In Group 1, LVEDP immediately after maximum rate of pacing decreased significantly from 25 ± 6 to 15 ± 6 mm Hg (P < 0.005) after operation. Before operation, all these patients (table 1) had postpacing LVEDP of 18 mm Hg or more. After operation, postpacing LVEDP (fig. 1) was less than 18 mm Hg in all except four patients. Two of these had electrocardiographic evidence of old infarction before surgery, anterior and inferior wall in one (#3), inferior in the other (#15). The other two patients suffered acute inferior myocardial infarctions perioperatively, mild in one (#2) and severe in the other (#13). In Group 1, sequential pacing before operation demonstrated progressively higher postpacing LVEDP (fig. 2), suggesting the postpacing LVEDP is an indicator of the degree of myocardial ischemia. After surgery, postpacing LVEDP was significantly lower at pacing rates of 110/min, 130/min, and 150/min. In Group 1, the heart-rate-systolic pressure products ranged from 11.3 to 18.7 (mean 14.6 ± 2.5) × 10\(^2\) mm Hg × beat/min before operation, and from 10.3 to 23.7 × 10\(^2\) mm Hg beats/min after surgery (mean 14.8 ± 3.9 × 10\(^2\) mm Hg beats/min), without significant change. Immediately after atrial pacing at maximum rate, the mean rate-pressure product was 7.8 ± 2.0 × 10\(^2\) mm Hg beats/min before operation and 9.0 ± 2.2 × 10\(^2\) mm Hg beats/min after the operation, the increase not being significant. In Group 2, where there was no ischemic response to atrial pacing, postpacing LVEDP did not change after the operation (13 ± 5 mm Hg vs 14 ± 6 mm Hg).

The two patients excluded in this analysis because of unsuccessful ACBS and persistent angina both had an ischemic response to atrial pacing before and after the operation.

### Degree of Revascularization and Ejection Fraction

The 22 patients with successful ACBS were subdivided into three groups (A, B, C) on the basis of vascularization of the three major territories (left anterior descending, circumflex, right coronary artery) using an approach similar to Levine et al.\(^6\) Preoperative and postoperative coronary angiograms were analyzed to determine graft patency and lack of significant stenosis (less than 50%) of the coronary artery distal to the bypass or the coronary artery which was not bypassed.

There were 2.6 grafts patent per patient. In Group A (nine patients), with all three major territories revascularized, ejection fraction changed from 61 ± 13% to 60 ± 10%.

### Table 2. Resting Hemodynamic Data (mean ± sd) before and after Coronary Artery Bypass Surgery

<table>
<thead>
<tr>
<th>Group</th>
<th>Heart rate (beats/min)</th>
<th>LVSP (mm Hg)</th>
<th>LVEDP (mm Hg)</th>
<th>Cardiac output (L/min)</th>
<th>Ejection fraction (%)</th>
<th>LVEDP post LV angiography</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Preop</td>
<td>64 ± 11</td>
<td>124 ± 22</td>
<td>14 ± 5</td>
<td>5.4 ± 1.4</td>
<td>54 ± 16</td>
</tr>
<tr>
<td></td>
<td>Postop</td>
<td>73 ± 14</td>
<td>119 ± 18</td>
<td>11 ± 4</td>
<td>5.4 ± 1.5</td>
<td>53 ± 13</td>
</tr>
<tr>
<td>2</td>
<td>Preop</td>
<td>76 ± 7</td>
<td>124 ± 12</td>
<td>10 ± 2</td>
<td>6.6 ± 2.9</td>
<td>57 ± 15</td>
</tr>
<tr>
<td></td>
<td>Postop</td>
<td>79 ± 11</td>
<td>122 ± 13</td>
<td>13 ± 4</td>
<td>5.8 ± 1.4</td>
<td>55 ± 10</td>
</tr>
<tr>
<td>1 &amp; 2</td>
<td>Preop</td>
<td>68 ± 11</td>
<td>124 ± 19</td>
<td>13 ± 5</td>
<td>5.9 ± 2.2</td>
<td>55 ± 15</td>
</tr>
<tr>
<td></td>
<td>Postop</td>
<td>75 ± 13</td>
<td>120 ± 16</td>
<td>12 ± 4</td>
<td>5.6 ± 1.5</td>
<td>54 ± 12</td>
</tr>
</tbody>
</table>

### Table 3. Hemodynamic Data (mean ± sd) during Pacing before and after Coronary Bypass Surgery

<table>
<thead>
<tr>
<th>Group</th>
<th>Pre-operative angina rate</th>
<th>Maximum rate</th>
<th>HR</th>
<th>SP</th>
<th>LVSP</th>
<th>HR</th>
<th>SP</th>
<th>LVEDP</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HR</td>
<td>RP × 10(^6)</td>
<td>HR</td>
<td>RP × 10(^6)</td>
<td>HR</td>
<td>RP × 10(^6)</td>
<td>HR</td>
<td>RP × 10(^6)</td>
</tr>
<tr>
<td>1</td>
<td>Preop</td>
<td></td>
<td>129 ± 16</td>
<td>115 ± 24</td>
<td>14 ± 9</td>
<td>63 ± 12</td>
<td>124 ± 29</td>
<td>25 ± 6</td>
</tr>
<tr>
<td></td>
<td>Postop</td>
<td></td>
<td>129 ± 16</td>
<td>109 ± 28</td>
<td>10 ± 5</td>
<td>72 ± 14</td>
<td>126 ± 22</td>
<td>15 ± 6</td>
</tr>
<tr>
<td>2</td>
<td>Preop</td>
<td></td>
<td>127 ± 24</td>
<td>118 ± 25</td>
<td>7 ± 7</td>
<td>73 ± 9</td>
<td>123 ± 14</td>
<td>13 ± 5</td>
</tr>
<tr>
<td></td>
<td>Postop</td>
<td></td>
<td>127 ± 24</td>
<td>113 ± 8</td>
<td>9 ± 3</td>
<td>70 ± 8</td>
<td>116 ± 13</td>
<td>14 ± 6</td>
</tr>
<tr>
<td>1 &amp; 2</td>
<td>Preop</td>
<td></td>
<td>128 ± 18</td>
<td>116 ± 21</td>
<td>12 ± 8</td>
<td>66 ± 12</td>
<td>124 ± 25</td>
<td>21 ± 6</td>
</tr>
<tr>
<td></td>
<td>Postop</td>
<td></td>
<td>128 ± 18</td>
<td>110 ± 24</td>
<td>10 ± 4</td>
<td>71 ± 15</td>
<td>123 ± 20</td>
<td>15 ± 6</td>
</tr>
</tbody>
</table>

Abbreviations: LVEDP = left ventricular end-diastolic pressure (mm Hg); HR = heart rate (beats/min); RP = rate-pressure product (mm Hg × beats/min); SP = left ventricular systolic pressure (mm Hg).

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Group B (eight patients), with two of the three territories revascularized, ejection fraction was $51 \pm 19\%$ before operation, and $48 \pm 13\%$ after operation. In Group C (four patients), with only one of the three areas revascularized, ejection fractions were $51 \pm 14\%$ before and $51 \pm 8\%$ after surgery. These changes in ejection fraction were not significant. When all patients with myocardial infarction (14 in all, 12 preoperative, two perioperative) were excluded, ejection fractions before and after operation were $70 \pm 8\%$ vs $63 \pm 8\%$ in Group A (five patients), $62 \pm 15\%$ vs $56 \pm 12\%$ in Group B (two patients), and $66\%$ vs $60\%$ in Group C (one patient).

**Discussion**

Previous studies of resting left ventricular function before and after ACBS have provided conflicting results. Chatterjee et al. reported a significant decrease in left ventricular end-diastolic pressure and an increase in ejection fraction after ACBS, especially in patients with preinfarction syn-
drome. Their study, however, was carried out within two weeks of surgery when resting heart rate and calculated cardiac index were increased by 21% and 58%, respectively, suggesting that there was sympathetic overstimulation secondary to the stress of surgery. Also anemia and fever are common in the immediate postoperative period. Thus, the apparent improvement in left ventricular function may have been partly due to the stress of surgery and increased sympathetic stimulation. Subsequently, Hammermeister et al. found, one to fourteen months after operation (median 4 months), a significant deterioration of ejection fraction (51% vs 40%) in patients with occluded grafts, while in patients with all grafts patent ejection fraction was unchanged (54 ± 12% vs 52 ± 13%). A similar result was reported by Shepherd et al. one to nine months postoperatively. They found that left ventricular function, determined by either left ventricular angiography with analysis of segmental contraction or pressure-flow relationship, did not change after operation in patients with all grafts patent and deteriorated in patients with occluded grafts.

Our study was carried out 3 to 5 months after operation when nonspecific effects of surgery have almost certainly disappeared and the likelihood of significant progression of coronary artery disease is small. In keeping with the last two reports, 2, 3 we did not find any appreciable change in conventional left ventricular function indices at rest such as left ventricular end-diastolic pressure, stroke volume, stroke work or ejection fraction. Levine et al. recently suggested that previous failure to demonstrate improved ejection fraction following ACBS could be explained on the basis that changes in native circulation as well as graft patency were not taken into account and proposed a new index of vascularization. We used a similar approach to classify our patients into three groups, and found that ejection fraction did not reflect the degree of revascularization as judged by both graft patency and the native coronary vascular bed. Although eight patients had taken propranolol 12 hours or more before the preoperative study, our results should not have been significantly altered by this drug as propranolol does not significantly change ejection fraction or left ventricular end-diastolic pressure, 14 and the half-life is short. 19

We have demonstrated that ischemic response to atrial pacing is relieved by successful aorticcoronary bypass surgery suggesting that myocardial perfusion during stress has improved although ejection fraction at rest did not change. Other investigators observed similar improvement of left ventricular response to exercise 10 and atrial pacing 11 following ACBS in a small number of patients. In our opinion, the apparent lack of improvement in resting left ventricular function is based on the fact that abnormal contraction due to old scarring does not change 12 after revascularization and that the potentially ischemic myocardium supplied by a stenotic coronary artery may function normally at rest without anginal symptoms in patients with chronic stable angina. 8 We did not study left ventricular contraction by angiography during induced angina as was done by others. 9, 18, 19

**References**

1. Chatterjee K, Swan HJC, Parmley WW, Sustaita H, Marcus HS, Matloff J: Influence of direct myocardial revascularization on left ven-
SUMMARY Seventeen consecutive patients with chronic recurrent ventricular tachycardia (VT) were studied in an attempt to delineate the reproducibility and mechanism of this arrhythmia. Six patients had nonsustained and 11 had sustained VT. The following electrophysiological techniques were utilized in an attempt to reproduce VT: 1) rapid atrial and ventricular pacing (17 pts); 2) atrial extrastimulus technique (17 pts); 3) ventricular extrastimulus technique (17 pts); 4) V,V,V substitution technique (5 pts); 5) ventricular pacing from two or more sites (5 pts). Ventricular tachycardia was induced in six of 11 (54%) patients with sustained VT. However, in four there was only a single induction and only in the remaining two patients could VT be repetitively induced. In the latter two patients ventricular tachycardia was induced with both atrial and ventricular stimulation. Ventricular tachycardia could not be induced in any patient with nonsustained VT, although three had spontaneous episodes of ventricular tachycardia during study.

In conclusion, in the present series of patients with chronic recurrent VT, this rhythm could not be reproducibly induced in the majority of patients in the cardiac catheterization laboratory utilizing catheter stimulation techniques.

RECENT ELECTROPHYSIOLOGICAL STUDIES in patients with recurrent ventricular tachycardia have demonstrated duplication of the arrhythmia with critically timed electrical stimulation.1-4 All except one of the previous reports have been concerned with selected cases. Recently, Wellens and Lie reported results of electrophysiological studies in a series of 36 patients with ventricular tachycardia, 20 of whom had chronic recurrent ventricular tachycardia.4 They noted that ventricular tachycardia could be induced with ventricular extrastimulus technique in 17 of the 20 patients with chronic recurrent ventricular tachycardia. Electrophysiological observations in these cases suggested that ventricular tachycardia reflected re-entrant mechanisms.

In the present study, we report the results of electrical stimulation utilizing several types of stimulation techniques in 17 consecutive cases of chronic recurrent ventricular tachycardia, in an attempt to further delineate the reproducibility of chronic recurrent ventricular tachycardia. We also attempted to further define mechanisms of this rhythm.

Methods

Patient Selection and Definitions

Seventeen consecutive patients undergoing electrophysiological study because of chronic recurrent ventricular tachy-
Abolition of ischemic response to atrial pacing following aortocoronary bypass surgery.
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