Haemodynamic alterations during ischaemia induced by dobutamine stress testing

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KEY WORDS: Dobutamine, echocardiography, haemodynamics, ischaemia, myocardial infarction, stress testing.

To identify the haemodynamic response to ischaemia induced by dobutamine stress testing, 15 patients with a first acute myocardial infarction underwent right-sided heart catheterization during dobutamine stress cross-sectional echocardiography. Haemodynamic variables and echocardiography were recorded at rest and during dobutamine infusion at each dose from 5 to a maximum of 40 \( \mu \)g kg\(^{-1}\) min\(^{-1}\). Ischaemia was diagnosed by cross-sectional echocardiography if asynergy appeared in at least two ventricular segments other than the area of acute myocardial infarction. Ischaemia was absent in six patients (group I) and identified in nine (group II). Response curves for each haemodynamic variable in the two groups were compared by applying Zerbe’s method.

The response curves were similar in the two groups for heart rate, arterial, right atrial, pulmonary arterial and pulmonary artery wedge pressures. The response curves were significantly different in groups I and II for thermodilution cardiac output, stroke volume and systemic vascular resistance (P<0.05). An increase in stroke volume was observed at low dosage of dobutamine in both groups. From low to maximum dose, stroke volume remained unchanged in group I and was significantly decreased in group II.

Ischaemia induced by dobutamine stress testing leads to a decrease in stroke volume with no change in pulmonary artery wedge pressure.

Introduction

In patients with coronary artery disease, angina pectoris is associated with an increase in left ventricular end-diastolic pressure and in pulmonary artery wedge pressure\(^1,2\). These haemodynamic changes have been demonstrated during ischaemia induced by exercise and atrial pacing stress tests\(^3,4\): in the latter condition, left ventricular end-diastolic pressure increases immediately after cessation of pacing\(^5,6\). Ambulatory pulmonary artery pressure monitoring also showed an increase in pulmonary artery diastolic pressure during painful and painless myocardial ischaemia in patients with coronary artery disease\(^7\) or variant angina\(^8\).

Pharmacologic stress testing also has been used for evaluating the functional significance of coronary artery disease; dipyridamole, a very potent coronary vasodilator, has been extensively studied\(^9-13\). We have previously shown that dobutamine infusion at increasing doses is a well-tolerated stress test\(^14\). We used dobutamine stress echocardiography, for identifying patients with multivessel coronary artery disease after acute myocardial infarction: the 87% overall accuracy of the technique was higher than that of exercise electrocardiography in our previous study\(^11\). However, the haemodynamic alterations during myocardial ischaemia induced by dobutamine stress testing have not yet been evaluated. To measure the haemodynamic correlates of the ischaemic response induced by dobutamine stress infusion, we studied 15 patients following acute myocardial infarction who underwent simultaneously right-sided heart catheterization and cross-sectional echocardiography.

Methods

PATIENTS

Fifteen patients (mean age 55 years, range 41 to 73 years), hospitalized within a 6-month period for a first acute myocardial infarction were studied. They belong to a greater population of an ongoing study evaluating the value of dobutamine stress...
echocardiography after myocardial infarction. They were consecutive patients who agreed to undergo right heart catheterization during dobutamine stress testing. The study protocol was approved by the institutional ethical committee. All patients gave informed consent. The location of infarction was anterior in four patients, lateral in two and inferior in nine. Six patients were given thrombolytic therapy (streptokinase 1·5 × 10^6 units i.v. over 1 h). Patients did not receive thrombolytic therapy if they were admitted > 4 h after the onset of symptoms, if ST-segment elevation was < 2 mV or if a contraindication to thrombolysis was present. No patient was being treated with β-adrenergic blocking agents. The other cardiac medications (nitrates or calcium-channel blocking agents) were not discontinued before the dobutamine stress testing. The studies were performed in the supine position 5–10 days after acute myocardial infarction.

**DOBUTAMINE STRESS TESTING**

Dobutamine was administered intravenously by an infusion pump at increasing doses from 5 to a maximum of 40 µg kg^{-1} min^{-1} at 5-min intervals. Electrocardiographic, haemodynamic and cross-sectional echocardiographic monitoring was continued during the infusion. Haemodynamic variables, a complete cross-sectional echocardiogram and a 12-lead electrocardiogram were recorded at each dose of dobutamine. Criteria for termination of the dobutamine infusion were angina, significant arrhythmia, severe hypertension (systolic blood pressure > 200 mmHg or diastolic blood pressure > 100 mmHg), decrease in systolic blood pressure > 20 mmHg or heart rate increase to 75% or more of maximum. Twelve-lead electrocardiograms were considered positive for ischaemia when > 1 mm segment depression developed during dobutamine infusion 80 ms after the J point.

Two independent observers reviewed all echocardiograms, without knowledge of the patients' clinical, electrocardiographic, haemodynamic or angiographic data. Segmental wall motion and thickening were assessed as previously described[14]. The recordings obtained in basal conditions were first interpreted to assess the topography of acute myocardial infarction. The recordings obtained during dobutamine infusion were carefully analysed in real-time, slow motion and frame-by-frame. Dobutamine-induced ischaemia was defined by the development of abnormal wall motion and reduced myocardial thickening in two or more ventricular segments outside the infarct zone. Ischaemia was considered adjacent to or at a distance from the infarcted area using our previous echocardiographic study which compared the location of coronary artery obstruction and the topography of akinetic segments[15]. The number of abnormal segments was calculated in basal conditions and at the end of dobutamine infusion.

**HAEMODYNAMIC EVALUATION**

Right-sided cardiac catheterization was performed with a triple lumen flotation catheter for measurement of cardiac pressures and thermodilution output. The variables measured were: heart rate, arterial blood pressure (systolic and diastolic), mean right atrial pressure, pulmonary arterial pressure (systolic and diastolic), mean pulmonary artery wedge pressure and cardiac output. Three thermodilution cardiac output measurements were made and averaged. Stroke volume, right and left ventricular stroke work indices, pulmonary and systemic vascular resistance were determined by calculation.

**ANGIOGRAPHY**

All but one patient underwent ventriculography and selective right and left coronary arteriography, using the Judkins technique, prior to hospital discharge. The infarct-related vessel was determined with consideration of the electrocardiogram and the location of wall motion abnormalities on the ventriculograms. The maximal luminal diameter stenosis for each major coronary artery was measured. Diameter stenoses were considered significant when they were > 50%.

**STATISTICAL ANALYSIS**

In each patient, four sets of measurements were considered for analysis: baseline, low, intermediate and maximum dose of dobutamine. At the low dose of dobutamine (5 or 10 µg kg^{-1} min^{-1}), the increase in heart rate was less than 10 beats min^{-1}. The intermediate step was determined as the dose at which heart rate was approximately midway between baseline and maximum heart rate, usually at approximately 100 beats min^{-1}. The maximum dose was determined using the described criteria for termination. The 15 patients were classified into two groups, according to the absence or presence of at least two new abnormal ventricular segments during dobutamine stress testing, as determined by cross-sectional echocardiography.
The mean value and standard deviation were computed for all quantitative variables. Response curves of haemodynamic variables were determined for each patient by linear interpolation between the serial measurements. Response curves for each variable in the two groups were compared by applying Zerbe's method. This method allows comparison of response curves not only pointwise but also over any fixed period—in this study, the four steps of dobutamine stress testing—thus providing a global assessment of group differences. For each variable, an F-criterion, with 1 degree of freedom was calculated to test the hypothesis of equal mean response curves between the two groups. Results were considered significant at the 5% critical level.

A chi-squared test with Yates' correction was used to compare proportions and a Student's t-test for unpaired data was used to compare means calculated from continuous data.

Results

Ischaemic vs Non-Ischaemic Response to Dobutamine Stress Testing

There were no complications as a result of this study. No patient had significant arrhythmias or severe hypertension.

Of the 15 patients, six were judged to have a non-ischaemic response to dobutamine stress testing, as determined by cross-sectional echocardiography, and these constitute Group I. The baseline echocardiogram revealed a mean of four asynergic segments (range two to five), corresponding to the infarcted area. Hyperkinetic contraction was observed in remote myocardium throughout the test in all six patients. At the low dobutamine dose, systolic myocardial thickening became evident in one patient in the two segments that were akinetic at rest. In this patient (no 5, Table 1) asynergy developed in one segment that was normal before dobutamine. This constitutes a non-ischaemic response according to the criteria used. No Group I patient had angina or electrocardiographic changes during the test.

The remaining nine patients were judged to have an ischaemic response, and they constitute Group II. The baseline echocardiogram revealed a mean of 3-2 asynergic segments (range two to seven). At the low dobutamine dose, hyperkinesis was observed in the unaffected walls and myocardial contraction appeared in two of the affected segments in three patients and in three affected segments in two patients. In all nine patients, asynergy was detected at higher doses in a mean of 4-3 segments unaffected at rest (range three to nine). Ischaemia was considered to occur at a distance from the area of infarction in three patients and within the area of the infarct-related vessel in six. Ischaemia was also already detectable at the intermediate dose level in four patients. Five Group II patients had ischaemic electrocardiographic changes during dobutamine infusion; no patient developed angina.

Patient Characteristics (Table 1)

Thrombolysis was attempted in no Group I patient and in six Group II patients. At angiography, the infarct-related vessel was occluded in five of six Group I patients but only in one of the eight Group II patients who underwent angiography. One-vessel coronary artery disease was observed in five of six Group I patients and in four of eight Group II patients. All six patients who were treated by thrombolysis had dobutamine-induced ischaemia in the area of the infarct-related vessel. Of the five patients with multivessel disease, three developed ischaemia at a distance and four developed ischaemia that was adjacent to the infarct zone.

Haemodynamics at Rest and in Response to Dobutamine Infusion

Table 2 shows the mean haemodynamic data at baseline and at low, intermediate and maximum dose of dobutamine infusion for the two groups. According to Zerbe's method, the response curves of the following variables were not significantly different in Groups I and II: heart rate (Fig. 1), systolic and diastolic arterial pressure; systolic, diastolic and mean pulmonary artery pressure; right atrial pressure, pulmonary artery wedge pressure and left ventricular stroke work. The response curves were significantly different in Groups I and II for thermodilution cardiac output, stroke volume and systemic vascular resistance (P < 0.05) (Fig. 1).

In both groups, mean pulmonary artery wedge pressure remained unchanged or was slightly decreased compared with baseline with low, intermediate and maximum doses of dobutamine. Six of the nine Group II patients exhibited a slight decrease in stroke volume from a low to an intermediate dose of dobutamine; asynergy outside the infarct zone was already detectable at this dose level by echocardiography in three of them. It should be noted that only one of the six Group I patients showed an increase in stroke volume when

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Table I  Clinical, angiographic and echocardiographic features of patients with or without ischaemic response to dobutamine stress testing

<table>
<thead>
<tr>
<th>Patient</th>
<th>Thrombolysis</th>
<th>Infarct location</th>
<th>Infarct vessel</th>
<th>Stenosis (%)</th>
<th>VD</th>
<th>Akinetic at baseline</th>
<th>Improved at low dose</th>
<th>Ischaemic with dobutamine</th>
<th>Same territory</th>
<th>At a distance</th>
</tr>
</thead>
<tbody>
<tr>
<td>GROUP I</td>
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<td>I</td>
<td>LCx</td>
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<td>2</td>
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<td>3</td>
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</tbody>
</table>

A = anterior, I = inferior, L = lateral, LAD = left anterior descending artery, LCx = left circumflex, RCA = right coronary artery, VD = vessel disease, — = not available.
Table 2  Haemodynamic data. Mean changes

<table>
<thead>
<tr>
<th>Variables</th>
<th>Heart rate (beats min⁻¹)</th>
<th>SAP (mmHg)</th>
<th>DAP (mmHg)</th>
<th>Mean RAP (mmHg)</th>
<th>Mean PAP (mmHg)</th>
<th>Mean PAWP (mmHg)</th>
<th>Cardiac output (l min⁻¹)</th>
<th>Stroke volume (ml)</th>
<th>SVR (dynes.s.cm⁻²)</th>
<th>Dose of dobutamine (μg kg⁻¹ min⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I (six patients)</td>
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</tr>
<tr>
<td>Baseline</td>
<td>76 ± 5</td>
<td>108 ± 3</td>
<td>71 ± 4</td>
<td>4 ± 2</td>
<td>15 ± 1</td>
<td>8 ± 1</td>
<td>6-4 ± 0-7</td>
<td>85 ± 7</td>
<td>1354 ± 90</td>
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</tr>
<tr>
<td>Low D</td>
<td>77 ± 7</td>
<td>123 ± 6</td>
<td>64 ± 4</td>
<td>5 ± 2</td>
<td>15 ± 2</td>
<td>8 ± 2</td>
<td>8 ± 8 ± 0-9</td>
<td>117 ± 13</td>
<td>1102 ± 68</td>
<td>7-5 ± 2-5</td>
</tr>
<tr>
<td>Int D</td>
<td>97 ± 5</td>
<td>133 ± 5</td>
<td>68 ± 5</td>
<td>4 ± 2</td>
<td>17 ± 3</td>
<td>8 ± 2</td>
<td>11-2 ± 1-2</td>
<td>120 ± 14</td>
<td>956 ± 68</td>
<td>20 ± 5</td>
</tr>
<tr>
<td>Max D</td>
<td>117 ± 7</td>
<td>138 ± 10</td>
<td>69 ± 5</td>
<td>5 ± 2</td>
<td>16 ± 3</td>
<td>7 ± 2</td>
<td>13-6 ± 1-1</td>
<td>120 ± 14</td>
<td>802 ± 62</td>
<td>37 ± 8-5</td>
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<td>Group II (nine patients)</td>
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<tr>
<td>Baseline</td>
<td>78 ± 3</td>
<td>121 ± 4</td>
<td>71 ± 1</td>
<td>3 ± 1</td>
<td>16 ± 2</td>
<td>8 ± 2</td>
<td>5-9 ± 0-5</td>
<td>76 ± 7</td>
<td>1667 ± 138</td>
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<tr>
<td>Low D</td>
<td>81 ± 3</td>
<td>127 ± 3</td>
<td>64 ± 2</td>
<td>2 ± 1</td>
<td>15 ± 2</td>
<td>6 ± 2</td>
<td>7-6 ± 0-6</td>
<td>95 ± 7</td>
<td>1358 ± 101</td>
<td>7-2 ± 2-5</td>
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<tr>
<td>Int D</td>
<td>100 ± 2</td>
<td>134 ± 5</td>
<td>63 ± 2</td>
<td>2 ± 1</td>
<td>14 ± 2</td>
<td>4 ± 2</td>
<td>9-0 ± 0-6</td>
<td>90 ± 6</td>
<td>1214 ± 86</td>
<td>18 ± 3-6</td>
</tr>
<tr>
<td>Max D</td>
<td>116 ± 3</td>
<td>127 ± 6</td>
<td>64 ± 3</td>
<td>2 ± 1</td>
<td>15 ± 3</td>
<td>6 ± 2</td>
<td>9-0 ± 0-7</td>
<td>78 ± 5</td>
<td>1158 ± 99</td>
<td>33 ± 7-3</td>
</tr>
</tbody>
</table>

*P<0.05. D = dose of dobutamine, DAP = diastolic arterial pressure, F = F value using Zerbe's method, Int = intermediate, Max = maximum, PAP = pulmonary artery pressure, PAWP = pulmonary artery wedge pressure, RAP = right atrial pressure, SVR = systemic vascular resistance.
the dobutamine dosage was increased from a low to an intermediate level.

Discussion

POTENTIAL OF DOBUTAMINE FOR INDUCING ISCHAEMIA

The administration of increasing doses of dobutamine to 15 patients with acute myocardial infarction resulted in asynergy developing in nine outside the infarct zone as determined by cross-sectional echocardiography. Simultaneous haemodynamic measurements indicate that ischaemia induced by dobutamine is accompanied by a decrease in stroke volume with no increase in pulmonary artery wedge pressure.

Previous clinical and experimental studies have shown that dobutamine can improve ventricular performance in acute myocardial infarction without exacerbating myocardial injury\(^\text{17,18}\). Despite coronary dilation in normal areas, dobutamine does not always elicit a coronary steal\(^\text{19}\), but has been found to be associated with a greater increase in myocardial oxygen consumption when compared with other catecholamines such as epinephrine and isoproterenol\(^\text{20}\). When dobutamine is used in patients with severe coronary artery disease at doses that significantly increase heart rate,
coronary perfusion becomes inhomogenous and contractile function deteriorates in ischaemic myocardium \(^{[19,21,22]}\).

Dopamine infusion has been found to be a less sensitive means of producing ischaemia than either exercise or isoproterenol infusion \(^{[23-25]}\), but the maximum dose used in these studies was 15 \(\mu g\) kg \(^{-1}\) min \(^{-1}\). Because of its low arrhythmogenic effect, dobutamine could be infused safely at higher doses in our study patients.

HAEMODYNAMIC RESPONSE TO ISCHAEMIA INDUCED BY DOBUTAMINE

The most striking difference between the two groups of patients in the haemodynamic response to dobutamine stress testing was a decreased stroke volume when ischaemia was identified by echocardiography. Careful frame-by-frame observation of the echographic images revealed that the reduction in systolic myocardial thickening in the ischaemic area was preceded by the appearance of marked regional asynchrony in left ventricular wall motion. The onset of systolic inward motion and of diastolic outward motion became markedly delayed in ischaemic areas, as compared with the normal zone. Since such incoordinate contraction and relaxation causes loss of efficiency in the energy transfer from myocardium to the circulation \(^{[26]}\), this may explain the decrease in stroke volume, which occurs without any change in global loading conditions.

Contrary to the haemodynamic response to ischaemia induced by exercise and atrial pacing stress tests, no increase in pulmonary artery wedge pressure was found in our patients who developed ischaemia during high-dose dobutamine infusion. This may be explained by the difference in cardiac and circulatory adjustments occurring during the different stress tests \(^{[27]}\). Like muscular exercise, dobutamine markedly reduces systemic vascular resistance, but unlike exercise, venous return is not increased. Afterload is not reduced during atrial pacing, but venous return is impaired by the decrease in available diastolic time and probably, at high pacing rates, by atrial contraction occurring against closed atrioventricular valves. Venous return increases after cessation of pacing; this explains why the significant increase in pulmonary artery wedge pressure is observed immediately after pacing in patients who develop ischaemia.

INOTROPIC EFFECT OF DOBUTAMINE

At the low dosage level (5–10 \(\mu g\) kg \(^{-1}\) min \(^{-1}\)), dobutamine increased stroke volume by 38% in Group I patients and by 25% in Group II patients; there was no increase in mean arterial pressure and in heart rate. At that stage, cross-sectional echocardiography revealed an increase in myocardial thickening of the unaffected wall in the majority of patients. Our data suggest that stroke volume may also slightly increase as a consequence of enhanced contractility in segments that were akinetic at baseline. An improvement in myocardial thickening was detected in more than one ventricular segment in six of the 15 patients. Previous experimental studies have shown that the infusion of \(\beta\)-adrenergic agents such as dopamine or isoproterenol may reverse the mechanical failure of reperfused, stunned myocardium \(^{[28-30]}\). Further studies are needed to clarify the significance of these observations. The results of this study indicate that in patients with acute myocardial infarction, the inotropic effect of dobutamine is nearly maximal at doses that do not increase heart rate. From the low to the intermediate (15–20 \(\mu g\) kg \(^{-1}\) min \(^{-1}\)) dosage level, there was no further increase in stroke volume in Group I and a tendency to a slight decrease in stroke volume in Group II (Table 2).

In conclusion, dobutamine infusion at increasing doses was used in the present study as a stress test after myocardial infarction. The haemodynamic correlates of dobutamine-induced ischaemia, as assessed by echocardiography, consisted in a decrease in stroke volume with no change in pulmonary artery wedge pressure.

References


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