Effects of Dobutamine on Coronary Stenosis
Physiology and Morphology
Comparison With Intracoronary Adenosine

Jozef Bartunek, MD, PhD; William Wijns, MD, PhD; Guy R. Heyndrickx, MD, PhD; Bernard de Bruyne, MD, PhD

Background—The mechanisms leading to dobutamine-induced ischemia are not fully understood. In the present study, we investigated the effects of high-dose intravenous dobutamine on morphological and physiological indexes of coronary stenoses.

Methods and Results—Twenty-two patients with normal left ventricular function and isolated coronary stenoses were studied. At catheterization, mean aortic pressure (Pa), mean distal coronary pressure (Pd), and Pd/Pa as an index of myocardial resistance were recorded at rest, after intracoronary adenosine, and during intravenous infusion of dobutamine (10 to 40 \( \mu \)g·kg\(^{-1} \cdot \)min\(^{-1} \)). Reference vessel diameter and minimal luminal diameter, as assessed by coronary angiography, did not change during dobutamine infusion compared with baseline (2.84±0.49 versus 2.77±0.41 mm and 1.35±0.38 versus 1.27±0.31 mm, respectively; both \( P \) =NS). During peak dobutamine infusion, Pd and Pd/Pa reached similar levels as during adenosine infusion (60±18 versus 59±18 mm Hg and 0.68±0.17 versus 0.68±0.17, respectively; all \( P \) =NS). In 9 patients, an additional bolus of intracoronary adenosine given at the peak dose of dobutamine failed to further decrease Pd/Pa. Furthermore, in patients with dobutamine-induced wall motion abnormalities, the maximal decrease in Pd/Pa was similar during dobutamine and adenosine infusions.

Conclusions—High-dose intravenous infusion of dobutamine does not modify the dimensions of the epicardial coronary stenosis. However, much like the direct coronary vasodilator adenosine, dobutamine fully exhausts myocardial resistance regardless of the presence of mechanical dysfunction. (Circulation. 1999;100:243-249.)

Key Words: dobutamine ■ hemodynamics ■ ischemia ■ coronary disease ■ stress

Dobutamine has a predominant positive inotropic effect associated with an increase in coronary blood flow and a mild positive chronotropic effect.\(^1\) Because of these characteristics, dobutamine leads to an increase in contractility without major changes in perfusion of the acutely ischemic myocardium.\(^2,3\) In current practice, high-dose dobutamine is increasingly used to detect and evaluate coronary artery disease. However, only a few experimental\(^2,4\) and clinical studies\(^5\) have investigated the effects of dobutamine on regional myocardial perfusion. Results of these studies suggested that mechanical dysfunction during dobutamine is usually associated with a blunted increase (or paradoxical decrease) in regional blood flow. However, these studies used models of acute or chronic myocardial ischemia\(^2,4\) or included many patients after myocardial infarction.\(^5\) In addition, it is not clear whether changes in myocardial perfusion during dobutamine infusion are related solely to changes in microvascular resistance or whether they are associated with changes in the morphology of an epicardial coronary stenosis. Therefore, in the present study, we tested the hypothesis that hemodynamic response of physiological indexes of stenosis severity during intravenous dobutamine is similar to the response induced by intracoronary adenosine. We also investigated whether dobutamine-induced changes in stenosis physiology are associated with changes in epicardial stenosis morphology.

Methods

Patients
The study population consisted of 22 patients (3 women; mean age, 58±8 years) with normal left ventricular function and isolated coronary stenoses referred for coronary angioplasty. In all patients, cardiac medications were stopped ≥36 hours before protocol and replaced by aspirin 100 mg/d and molsidomine 4 mg TID. Molsidomine was routinely administered to prevent any vasomotion during wire manipulation. Stenoses were located in the left anterior descending artery in 9 patients, in the right coronary artery in 12 patients, and in the left circumflex coronary artery in 1 patient.

Dobutamine Echocardiography
Echocardiography was performed on the same day as the catheterization as previously described in detail elsewhere.\(^6\)
Catheterization Protocol

A 7.5F or 8F introduction sheath was introduced into the femoral artery, and a 7F guiding catheter without side holes was engaged in the coronary ostium. Distal coronary pressure was measured with a fluid-filled (n=10, Schneider Europe) or high-fidelity (n=12, Radi Medical) pressure-monitoring guidewire as previously described in detail.6,7,8 After baseline recordings, intracoronary adenosine (18 μg into the left coronary artery and 12 μg into the right coronary artery) was given during maximal coronary hyperemia.7,8 After hemodynamic parameters returned to baseline, dobutamine was infused at 10, 20, 30, and 40 μg · kg⁻¹ · min⁻¹ for 3 minutes at each stage. The end points for infusion were maximal dose, target heart rate, unbearable chest pain, ST-segment depression >0.3 mV, or severe side effects. In 9 patients, an additional bolus of intracoronary adenosine was injected during the last 30 seconds of dobutamine infusion. Figure 1 shows an example of pressure recordings performed at rest, during maximal hyperemia, and during dobutamine infusion.

Quantitative Coronary Angiography

Quantitative assessment of the stenosis geometry was performed with the ACA system as previously described.9 The interpolated reference diameter, minimal luminal diameter, percent diameter stenosis, and area stenosis were calculated.

Data Acquisition

Mean aortic pressure (Pₐ), mean distal coronary pressure (Pᵩ), mean translesional pressure gradient (∆P), and the ratio of mean distal coronary pressure to mean aortic pressure (Pᵩ/Pₐ) were monitored under baseline conditions, during intravenous infusion of dobutamine, and before and during adenosine infusion. Angiographic measurements of the segment under study were performed at baseline and at the end of dobutamine infusion.

Statistical Analysis

Data are expressed as mean±SD. Statistical comparison was made by ANOVA, followed by the Newman-Keuls test. Changes in vessel diameters during infusion of dobutamine were analyzed by Student’s paired t test. A value of P<0.05 was considered statistically nonsignificant.

Results

Effects of Intravenous Dobutamine on Stenosis Dimensions

At baseline, reference vessel diameter ranged from 2.10 to 3.82 mm (mean, 2.77±0.41 mm); minimal luminal diameter ranged from 0.64 to 1.89 mm (mean, 1.27±0.31 mm); percent diameter stenosis ranged from 36% to 79% (mean 53±11%); and area stenosis ranged from 60% to 90% (mean 76±9%) (Table 1). Dobutamine had no significant effect on these indexes of stenosis morphology (Figure 2).

Comparative Effects of Intravenous Dobutamine and Intracoronary Adenosine on Systemic and Coronary Hemodynamics.

Major individual and mean data in response to dobutamine and adenosine are given in Tables 2 and 3 and Figure 3. Adenosine had no effect on heart rate, systolic blood pressure, or rate-pressure product. Intravenous dobutamine significantly increased heart rate and rate-pressure product. Heart rate and rate-pressure product continued to increase significantly when dobutamine was increased from 20 to 40 μg · kg⁻¹ · min⁻¹ (Figure 3).

Mean aortic pressure did not change during dobutamine or intracoronary adenosine infusion. Translesional pressure gradient increased, and distal coronary pressure and the Pᵩ/Pₐ ratio decreased to a similar level during both dobutamine and adenosine infusion. Figure 3 shows dose-dependent changes in Pᵩ/Pₐ during intracoronary adenosine and dobutamine. Pᵩ/Pₐ decreased significantly at 20 μg · kg⁻¹ · min⁻¹. There was a mild but statistically nonsignificant decrease in Pᵩ/Pₐ when dobutamine was increased from 20 to 30 μg · kg⁻¹ · min⁻¹. Increasing dobutamine from 30 to 40 μg · kg⁻¹ · min⁻¹ did not produce any additional decrease in Pᵩ/Pₐ. In contrast, heart rate and rate-pressure product did increase further at dobutamine >20 μg · kg⁻¹ · min⁻¹. Figure 4 shows the near identity of the values of Pᵩ/Pₐ after intracoronary adenosine and during peak dobutamine.

In 9 patients, an additional bolus of intracoronary adenosine given during peak dobutamine infusion induced a sig-
significant decrease in blood pressure (89 ± 15 mm Hg before versus 82 ± 15 mm Hg 15 seconds after an additional bolus of adenosine, \( P < 0.05 \)) and a proportional decrease in distal coronary pressure (60 ± 18 versus 53 ± 14 mm Hg, \( P < 0.05 \)) but no change in heart rate (139 ± 17 versus 144 ± 19 bpm, \( P = \text{NS} \)), pressure gradient (29 ± 18 versus 29 ± 18 mm Hg, \( P = \text{NS} \)), and \( P_d/P_a \) (0.68 ± 0.19 versus 0.66 ± 0.18, \( P = \text{NS} \)).

**Coronary Hemodynamics According to the Presence or Absence of Dobutamine-Induced Ischemia**

In 10 patients, dobutamine echocardiography was negative, whereas in 12 patients, dobutamine-induced wall motion abnormalities could be detected by echocardiography (Tables 4 and 5 and Figure 5). There were no differences between the anterior and inferior walls in response to dobutamine. As expected, patients with abnormal dobutamine echocardiograms had significantly higher transstenotic pressure gradients and lower distal coronary pressures and \( P_d/P_a \) ratios during both adenosine and dobutamine infusion. However, the dose-dependent response of systemic hemodynamics and stenosis physiology to dobutamine was similar in both groups. Both distal coronary pressure and \( P_d/P_a \) decreased to a similar level during dobutamine and adenosine infusion regardless of ischemia. Likewise, an increase in translesional pressure gradient during dobutamine was similar in both groups compared with adenosine, suggesting that the changes in myocardial resistance induced by either intracoronary adenosine or intravenous dobutamine were similar in both groups.

**Discussion**

The present study investigates in humans the effects of intravenous dobutamine at dosages used for pharmacological stress testing on coronary atherosclerotic stenoses and resistive vessel function. The results can be summarized as follows. First, in patients taking oral molsidomine, intravenous dobutamine did not modify the severity of the epicardial coronary stenosis. Second, despite a less-than-maximal increase in rate-pressure product, dobutamine induced a de-

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**Table 2. Major Individual Patient Data**

<table>
<thead>
<tr>
<th>Patient</th>
<th>DS, %</th>
<th>MLD, mm</th>
<th>Dobutamine RPP, mm Hg · s</th>
<th>Dobutamine ( P_d/P_a )</th>
<th>Adenosine ( P_d/P_a )</th>
<th>Dobutamine WMA</th>
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<tbody>
<tr>
<td>1</td>
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<tr>
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<tr>
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<td>1.11</td>
<td>19 380</td>
<td>0.63</td>
<td>0.69</td>
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</tr>
</tbody>
</table>

DS indicates diameter stenosis; MLD, minimal luminal diameter; RPP, rate-pressure product; and WMA, wall motion abnormalities.

**Table 3. Comparative Effects of Intravenous Dobutamine and Intracoronary Adenosine on Coronary Hemodynamics**

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Dobutamine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate, bpm</td>
<td>73 ± 12</td>
<td>76 ± 14</td>
</tr>
<tr>
<td>Systolic ( P_a ), mm Hg</td>
<td>128 ± 23</td>
<td>123 ± 20</td>
</tr>
<tr>
<td>Mean ( P_a ), mm Hg</td>
<td>92 ± 13</td>
<td>87 ± 12*</td>
</tr>
<tr>
<td>RPP, bpm · mm Hg</td>
<td>9463 ± 2733</td>
<td>9447 ± 2526*</td>
</tr>
<tr>
<td>Mean ( P_a ), mm Hg</td>
<td>76 ± 21</td>
<td>59 ± 18*</td>
</tr>
<tr>
<td>( \Delta P ), mm Hg</td>
<td>16 ± 17</td>
<td>28 ± 16*</td>
</tr>
<tr>
<td>( P_d/P_a )</td>
<td>0.83 ± 0.19</td>
<td>0.68 ± 0.17*</td>
</tr>
</tbody>
</table>

RPP indicates rate-pressure product.

*\( P < 0.05 \) vs baseline; †\( P < 0.05 \) vs adenosine.
crease in distal coronary perfusion pressure similar to that with adenosine. Because vasodilation was not associated with changes in stenosis morphology, the present data suggest that intravenous infusion of dobutamine decreased myocardial resistance to a minimum much like adenosine. Third, in most patients, myocardial resistances did not further decrease at dobutamine doses greater than 20 μg · kg⁻¹ · min⁻¹.

Dobutamine and Epicardial Vasomotion
Stimulation of β₁-adrenergic receptors by isoproterenol has been shown to induce an endothelium-independent vasodilation of large coronary arteries in dog experiments. In the same species, however, the endothelium is essential for mediation of exercise-induced epicardial coronary vasodilation. Likewise, in humans, the response of epicardial coronary arteries to sympathetic stimulation by the cold pressor test depends on the functional integrity of the endothelium. In contrast, in the present study conducted in patients with angiographically documented atherosclerosis, no changes in minimal luminal diameter or reference diameter were observed during high-dose dobutamine. Yet the effect of dobutamine is difficult to appreciate because all patients received oral molsidomine. The latter, an endothelium-independent vasodilator, might have precluded any dobutamine-induced vasomotion. Nevertheless, preliminary results suggest that in patients with diffuse coronary atherosclerosis not pretreated with molsidomine, dobutamine induces a mild increase in coronary dimensions. Thus, in contrast to sympathetic stimulation by the cold pressor test or exercise, our data suggest that high-dose dobutamine infusion causes either no change or vasodilation rather than paradoxical vasoconstriction even in atherosclerotic conduit vessels.

Distal Coronary Pressure as an Index of Myocardial Resistance
In the presence of an epicardial stenosis, adenosine infusion induces vasodilation, which in turn induces a decrease in coronary pressure distal to the stenosis. This increases pressure gradient and augments transstenotic flow. At constant aortic pressure, changes in distal coronary pressure may be due to changes in the severity of the epicardial stenosis (ie,
TABLE 5. Parameters of Coronary Hemodynamics in Patients Without Dobutamine-Induced Ischemia

<table>
<thead>
<tr>
<th></th>
<th>BL 1</th>
<th>ADO</th>
<th>BL 2</th>
<th>DOB 10</th>
<th>DOB 20</th>
<th>DOB 30</th>
<th>DOB 40</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate, bpm</td>
<td>75±16</td>
<td>80±18</td>
<td>75±17</td>
<td>76±18</td>
<td>98±21†</td>
<td>119±24†</td>
<td>141±15†</td>
</tr>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>133±23</td>
<td>128±20</td>
<td>134±25</td>
<td>138±23</td>
<td>139±21</td>
<td>131±26</td>
<td>124±27</td>
</tr>
<tr>
<td>Mean Pd, mm Hg</td>
<td>97±12</td>
<td>91±12†</td>
<td>96±13</td>
<td>97±11</td>
<td>97±9</td>
<td>90±16</td>
<td>87±16</td>
</tr>
<tr>
<td>RPP, ×10⁴ mm Hg · bpm</td>
<td>10.2±3.5</td>
<td>10.2±3.1</td>
<td>10.2±3.6</td>
<td>10.7±4.2</td>
<td>13.6±3.9†</td>
<td>15.4±4.1†</td>
<td>17.7±5.4†</td>
</tr>
<tr>
<td>Pa, mm Hg</td>
<td>93±12</td>
<td>76±10†</td>
<td>92±12</td>
<td>92±10</td>
<td>86±9†</td>
<td>75±13†</td>
<td>73±13†</td>
</tr>
<tr>
<td>Pd/Pa</td>
<td>0.95±0.04</td>
<td>0.83±0.06†</td>
<td>0.95±0.02</td>
<td>0.94±0.03</td>
<td>0.89±0.05†</td>
<td>0.84±0.04†</td>
<td>0.84±0.04†</td>
</tr>
</tbody>
</table>

Abbreviations as in Table 4. *P<0.05 vs preceding value; †P<0.05 vs BL 1.

Dobutamine and Arteriolar Vasodilation

Several studies have shown that adenosine induces maximal vasodilation.9 In addition, the magnitude of adenosine-induced hyperemia appears to be similar to that induced by exercise-induced ischemia16 and postocclusional hyperemia.17,18 Our data suggest that in humans intravenous dobutamine at doses used for stress testing also induces maximal vasodilation regardless of whether ischemia is present. Furthermore, an additional intracoronary bolus of adenosine given during peak dobutamine did not further decrease myocardial resistances. These findings are in contrast to earlier experimental reports.” However, they corroborate previous studies demonstrating an ∼3-fold increase in myocardial flow during dobutamine infusion19 and an ∼4-fold increase during intravenous infusion of 300 μg · kg⁻¹ · min⁻¹ adenosine.20 In addition, our data are consistent with those from a study of Skopicki et al,21 who reported a similar increase in myocardial blood flow as assessed by [¹⁵N]ammonia in stenotic regions during adenosine and dobutamine infusion.

Furthermore, in the present study, a vasodilation close to that obtained by adenosine was reached already at 20 and 30 μg · kg⁻¹ · min⁻¹ dobutamine. Indeed, the Pd/Pa ratio did not significantly decrease further when dobutamine was increased from 30 to 40 μg · kg⁻¹ · min⁻¹. Because during maximal vasodilation a linear relationship exists between coronary driving pressure and myocardial flow, these data suggest that myocardial flow did not significantly increase when dobutamine was increased to >20 μg · kg⁻¹ · min⁻¹. Of note, corroborating our data, Petroukilas et al22 observed a maximal increase in coronary flow velocity reserve in stenotic coronary arteries already at intermediate doses of dobutamine.

Mechanisms of Dobutamine-Induced Vasodilatation

In the presence of a fixed coronary stenosis, flow maldistribution and myocardial ischemia are ascribed to an increase in cardiac work and oxygen consumption. Yet in this study, as in other studies,22–30 the rate-pressure product reached during dobutamine infusion is far from that usually reported during exercise despite a maximal decrease in myocardial resistance. Despite this modest increase in rate-pressure product, the ability of dobutamine echocardiography and scintigraphy to detect coronary artery disease is similar to that of exercise echocardiography.31–33 Several factors may explain this apparent paradox.

First, dobutamine may increase contractility more than physical exercise. Previous animal experiments have shown a larger dP/dt during dobutamine infusion than during exercise.33 In humans, Dagianti et al34 also showed that at the ischemic threshold, the ratio of systolic blood pressure to end-systolic volume index, a variable related to myocardial contractility, was significantly higher during dobutamine infusion than during exercise.

Second, experimental studies suggested a direct coronary vasodilating effect of dobutamine mediated by β₂-adrenergic receptors contributing to a decrease in myocardial resistance and an increase in myocardial flow in a feed-forward manner.1,35 In chronically instrumented pigs, Duncer et al36 also noticed that β-adrenergic receptor activity contributed in a feed-forward manner to coronary vasodilation during exercise. In patients with congestive heart failure, dobutamine induced an increase in coronary blood flow associated with an increase in coronary oxygen content but without changes in myocardial oxygen consumption,37 which is also consistent with a direct coronary vasodilatory effect of dobutamine. In the present study, a decrease in myocardial resistance reached a plateau...
at 20 μg · kg⁻¹ · min⁻¹ of dobutamine, whereas heart rate and rate-pressure product continued to increase. Using PET, Severi et al 38 also found no significant increase in myocardial blood flow at dobutamine >20 μg · kg⁻¹ · min⁻¹ despite a progressive increase in heart rate at higher doses. This blunted increase in myocardial flow is in contrast with a progressive and continued increase in systolic wall thickening at higher doses of dobutamine.39 Taken together, these data suggest a dissociation between an increase in myocardial flow and an increase in myocardial mechanical work. Furthermore, they are consistent with a direct effect of dobutamine on myocardial resistive vessels that most likely override the metabolic regulation of myocardial perfusion.

Third, myocardial ischemia itself causes vasodilation and might explain a decrease in distal coronary perfusion pressure. However, in the present study, a similar decrease in the Pve/Pa ratio was observed in patients with and without ischemia-induced wall motion abnormalities. This suggests that myocardial ischemia is not a direct cause of the maximal vasodilation observed in our study. In sharp contrast to our finding that myocardial resistance uniformly decreases during dobutamine-induced ischemia, Sambucetti et al.40 using coronary Doppler flow velocity measurements, recently reported an inappropriate, severe microvascular vasoconstriction during pacing-induced ischemia that could be totally abolished by intracoronary adenosine.

Finally, an alternative mechanism that can contribute to dobutamine-induced ischemia is the so-called oxygen-wasting effects of catecholamines, ie, an increase in energy utilization at a comparable workload. This phenomenon has recently been demonstrated to occur in humans during intravenous administration of dobutamine even at doses as low as 10 μg · kg⁻¹ · min⁻¹.41

**Study Limitations**

This study has several limitations. First, patients with coronary atherosclerosis often show an impaired vasodilator response of the microvasculature. Unfortunately, pressure measurements without simultaneous coronary flow velocity measurements cannot assess the presence of microvascular disease. Nevertheless, in the present study, this does not confound the comparison between intracoronary adenosine and intravenous dobutamine because each patient served as his or her own control. Second, our study included only patients with single-vessel disease and normal left ventricular function. Thus, our data must be interpreted with caution in cases of extensive coronary artery disease or left ventricular dysfunction. Furthermore, responses in the stenotic coronary arteries were not compared with contralateral “normal” vessels. Recent studies suggested that in normal segments, the dobutamine-induced increase in coronary myocardial blood flow21 is lower than during intracoronary or intravenous adenosine. Nevertheless, our data suggest that in patients with a mild stenosis, the magnitude of the response of stenosis physiology to dobutamine is similar to that of adenosine. In addition, it seems unlikely that alternative routes of adenosine administration would have different hyperemic effects.9,42,43 Fourth, all patients were off β-blockers. Considering the effect of β-blockade on systemic hemodynamics, it remains unclear whether treatment with β-blockers alters the effects of dobutamine on stenosis physiology.

**Conclusions**

Although high-dose intravenous dobutamine infusion did not affect the geometry of an epicardial coronary stenosis, in patients pretreated with molsidomine, it fully exhausted myocardial resistances at doses that do not cause a maximal increase in cardiac work.

**Acknowledgment**

The secretarial help of Josefa Cano is sincerely acknowledged.

**References**

Bartunek et al Effects of Dobutamine on Coronary Hemodynamics


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Circulation. 1999;100:243-249
doi: 10.1161/01.CIR.100.3.243

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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