Dynamic Intraventricular Obstruction During Dobutamine Stress Echocardiography
A New Observation

Patricia A. Pellikka, MD; Jae K. Oh, MD; Kent R. Bailey, PhD; Barbara A. Nichols, BS, RN, RDCS; Kristi H. Monahan, RN, RDCS; and A. Jamil Tajik, MD

Background. The implications of hypotension occurring during dobutamine stress echocardiography have not been elucidated. We observed in some patients that hyperdynamic left ventricular function developed during dobutamine stress echocardiography and hypothesized that intracavitary obstruction was occurring and might account for hypotension in some patients.

Methods and Results. Fifty-seven consecutive patients undergoing dobutamine stress echocardiography underwent pulsed-wave and continuous-wave Doppler examination of the left ventricular cavity at rest and at peak dobutamine infusion. The development of an intracavitary gradient with dobutamine stress was defined as a late-peaking left ventricular Doppler velocity profile that exceeded basal velocity by at least 1 m/sec. During dobutamine stress testing, left ventricular outflow velocity or intracavitary velocity increased in all patients. Obstruction occurred in 12 patients (21%, group 1). Group 2 was the remaining 45 patients. Peak velocities in group 1 ranged from 2.0 to 5.0 m/sec (mean, 3.5 m/sec), and the mean increase from velocity at rest was 2.3 m/sec. The mean change in systolic blood pressure was significantly lower in patients in group 1 (−15 versus 4 mm Hg, p=0.02). When the 18 patients with an ischemic response to stress testing (evidenced by new or worsening wall motion abnormalities) were excluded from analysis, systolic blood pressure response was still significantly different for the two groups (−19 versus 2 mm Hg, p=0.03).

Conclusions. Dynamic left ventricular obstruction is a new observation; it may develop frequently in patients undergoing dobutamine stress echocardiography. Obstruction rather than ischemia may explain a decrease in blood pressure during dobutamine stress echocardiography. (Circulation 1992;86:1429–1432)

KEY WORDS • echocardiography • dobutamine • Doppler • stress • ischemia, myocardial • intracavitary obstruction

Dobutamine stress echocardiography is valuable as an alternative stress imaging test for evaluation of coronary artery disease in patients who are unable to perform standard treadmill or bicycle exercise.1 Whereas exercise-induced hypotension has been recognized as an indicator of severe coronary artery disease,2 the implications of hypotension occurring during dobutamine stress testing have not been elucidated. In preliminary studies, some investigators have noted that a decrease in blood pressure of ≥10 mm Hg during infusion is unrelated to myocardial ischemia3 and others have proposed that a decrease in blood pressure may identify a group of patients with ischemia.4

During dobutamine stress echocardiography, we observed in some patients the development of hyperdynamic left ventricular function with systolic cavity obliteration. We hypothesized that intracavitary obstruction was occurring and might account for hypotension in some patients. Therefore, we began a prospective study in patients undergoing dobutamine stress echocardiography to determine the incidence and significance of the development of intraventricular obstruction.

Methods

Study Population

Fifty-seven consecutive patients undergoing dobutamine stress echocardiography underwent pulsed-wave and continuous-wave Doppler examination of the left ventricular cavity at rest and at peak dobutamine infusion. The patients included 25 men and 32 women (mean age, 67 years; range, 39–85 years). All patients had a physical limitation that precluded standard exercise testing. Dobutamine stress echocardiography was performed for preoperative evaluation for noncardiac surgery in 24 patients (42%) and for evaluation of chest pain or coronary artery disease in 33 patients (58%). Thirty-nine patients (68%) had a history of hypertension.

Dobutamine Infusion Protocol

After a 3-hour fast, dobutamine was administered intravenously by an infusion pump at a starting dose of 5 μg·kg⁻¹·min⁻¹. At 3-minute intervals, the dose was increased to 10 μg·kg⁻¹·min⁻¹, then by increments of 10 μg·kg⁻¹·min⁻¹ until one of the end points or a
maximum dose of 40 μg·kg⁻¹·min⁻¹ was reached. Continuous ECG monitoring was performed. Heart rate and 12-lead ECGs were recorded every minute. Blood pressure was recorded every third minute and also at the time of Doppler examination. Criteria for termination of the dobutamine infusion were angina pectoris, ventricular tachycardia or sustained supraventricular tachycardia, severe hypertension (systolic blood pressure ≥200 mm Hg or diastolic blood pressure ≥110 mm Hg), decrease in systolic blood pressure of 20 mm Hg from the previous level of infusion, target heart rate (85% of the age-predicted maximum heart rate), new regional wall motion abnormalities, ECG evidence of ischemia, or adverse effects from the infusion, including nausea, vomiting, or headache.

**Echocardiography/Doppler Protocol**

Two-dimensional echocardiography was performed with the patient in the left lateral decubitus position. Baseline images were obtained, and two-dimensional echocardiography was continuously performed throughout dobutamine infusion. Quad-screen images (parasternal long-axis and short-axis and apical four-chamber and two-chamber views) were obtained at rest, at low dose of dobutamine infusion (10 μg·kg⁻¹·min⁻¹), at peak dose of dobutamine infusion, and during recovery. Pulsed-wave and continuous-wave Doppler examination of the left ventricular cavity was performed from the apical window at rest and at peak dose of dobutamine using a Duplex and a stand-alone Pedoff transducer, respectively.

**Echocardiographic Analysis**

Wall motion was assessed with a 16-segment model. The development of hyperkinesis during dobutamine infusion was considered normal. Failure to develop hyperdynamic function or development of hypokinesis, akinesis, or dyskinesis was interpreted to represent ischemic response. Segments that were hypokinetic or akinetic at rest and did not change with dobutamine were considered to represent infarcted tissue.

The development of an intraventricular gradient with dobutamine stress was defined as a late-peaking left ventricular Doppler velocity profile that exceeded the basal maximum velocity by at least 1 m/sec (Figure 1).

Presence of left ventricular hypertrophy was determined by two-dimensional echo-derived M-mode measurement of wall thicknesses, with normal values based on age and body surface area, or subjectively assessed from resting two-dimensional echocardiographic images. Resting ejection fraction was determined using a modified method of Quinones et al. or by visual estimate.

**Statistical Analysis**

Data summarization used means and standard deviations for continuous variables and percentages for dichotomous or qualitative variables. Group comparisons used t tests for continuous variables and χ² tests for dichotomous variables. Before and after comparisons were based on the paired sample t test. A significance level of 0.05 was used.

**Results**

During dobutamine stress testing, there was an increase in left ventricular outflow tract velocity or intracavitary velocity in all 57 patients (mean, 0.88 m/sec; range, 0.04–3.7 m/sec; p<0.001).

Among the 57 patients, 12 (21%) had an intracavitary obstruction during dobutamine infusion (group 1) defined as a late-peaking left ventricular velocity that exceeded the velocity at rest by at least 1 m/sec. Group 2 consisted of the remaining 45 patients. Peak velocities of patients in group 1 ranged from 2.0 to 5.0 m/sec (mean, 3.5 m/sec), corresponding to a 16–100-mm Hg gradient. The mean increase from velocity at rest was
TABLE 1. Comparison of Patients With Obstruction (Group 1) and Without Obstruction (Group 2)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group 1, velocity ≥1 m/sec (n=12)</th>
<th>Group 2, velocity &lt;1 m/sec (n=45)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resting ejection fraction (%)</td>
<td>65±6</td>
<td>55±13</td>
<td>0.003</td>
</tr>
<tr>
<td>Change in systolic blood pressure (mm Hg)</td>
<td>-15±18</td>
<td>4±26</td>
<td>0.02</td>
</tr>
<tr>
<td>Resting LVOT velocity (m/sec)</td>
<td>1.2±0.4</td>
<td>1.0±0.2</td>
<td>0.07</td>
</tr>
<tr>
<td>Change in heart rate (beats per minute)</td>
<td>57±18</td>
<td>48±19</td>
<td>0.11</td>
</tr>
<tr>
<td>Age (years)</td>
<td>67±8</td>
<td>67±11</td>
<td>NS</td>
</tr>
<tr>
<td>Sex (% men)</td>
<td>33</td>
<td>47</td>
<td>NS</td>
</tr>
<tr>
<td>Peak-dose dobutamine (μg·kg⁻¹·min⁻¹)</td>
<td>31±8</td>
<td>34±8</td>
<td>NS</td>
</tr>
<tr>
<td>History of hypertension (%)</td>
<td>67</td>
<td>69</td>
<td>NS</td>
</tr>
<tr>
<td>Left ventricular hypertrophy by echocardiography (%)</td>
<td>33</td>
<td>29</td>
<td>NS</td>
</tr>
<tr>
<td>Change in diastolic blood pressure (mm Hg)</td>
<td>-7±13</td>
<td>-7±14</td>
<td>NS</td>
</tr>
<tr>
<td>Receiving β-blocker therapy (%)</td>
<td>17</td>
<td>27</td>
<td>NS</td>
</tr>
<tr>
<td>Receiving calcium channel blocker therapy (%)</td>
<td>58</td>
<td>50</td>
<td>NS</td>
</tr>
<tr>
<td>Receiving digoxin therapy (%)</td>
<td>8</td>
<td>11</td>
<td>NS</td>
</tr>
<tr>
<td>New regional wall motion abnormalities (%)</td>
<td>17</td>
<td>36</td>
<td>NS</td>
</tr>
<tr>
<td>Symptoms (%)</td>
<td>42</td>
<td>31</td>
<td>NS</td>
</tr>
</tbody>
</table>

LVOT, left ventricular outflow tract. Continuous variables are expressed as mean±SD.

2.3 m/sec (range, 1.0–3.7 m/sec). Mean peak velocity of patients in group 2 was 1.5±0.3 m/sec (range, 0.9–2.2 m/sec). Concentric left ventricular hypertrophy was present in four patients in group 1 and was mild in two and moderate in two; all had a history of hypertension. Mild basal septal hypertrophy was present in two patients. One patient with moderate concentric left ventricular hypertrophy had mild obstruction at rest and developed a marked increase in left ventricular outflow tract velocity from 2.2 to 5.0 m/sec. Resting left ventricular velocities were within normal limits for the remainder of the patients in group 1 (mean, 1.1±0.2 m/sec; range, 0.8–1.5 m/sec). In no patient was systolic anterior motion of the mitral valve (SAM) present at rest. However, in three of the 12 patients in group 1, SAM developed with dobutamine infusion; in the nine other patients, SAM did not develop, and intracavitary obstruction occurred as a result of hyperdynamic left ventricular function and cavity obliteration at the midventricular (papillary muscles) and apical regions. Mean resting ejection fraction was significantly higher in group 1: 65% compared with 55% in group 2 (p=0.003) (Table 1).

The mean change in systolic blood pressure was significantly lower in patients in group 1: -15 versus 4 mm Hg (p=0.02). Although a broad range of blood pressure responses was noted in patients without obstruction (from an increase of 50 mm Hg to a decrease of 53 mm Hg), patients with obstruction exhibited either little change or a decrease in systolic blood pressure (Figure 2). When the 18 patients with an ischemic response to stress testing (as evidenced by new or worsening wall motion abnormalities) were excluded from analysis, the systolic blood pressure response was still significantly different for the two groups, with a mean decrease of 19 mm Hg in the 10 patients with obstruction compared with a mean increase of 2 mm Hg in the 29 patients without obstruction (p=0.03). There was a trend toward a greater increase in heart rate in group 1.

The two groups did not differ significantly with regard to age, sex, peak-dose dobutamine, history of hypertension, left ventricular hypertrophy by echocardiogram, change in diastolic blood pressure, medications, new regional wall motion abnormalities, or development of symptoms of chest pain, dyspnea, or light-headedness during the test. Of the five patients with obstruction in whom symptoms developed during the dobutamine stress test, only one had evidence of ischemia. Of the 14 patients without obstruction in whom symptoms developed during the dobutamine stress test, seven had evidence of ischemia.

Of the 12 patients in group 1, 10 had a history of chest pain and exertional dyspnea. Eight of 10 patients (80%) with symptoms had no evidence of stress-induced ischemia.

Six patients in group 1 subsequently underwent noncardiac surgery, including peripheral vascular surgery in five and major abdominal surgery in one. None of these patients had ischemia by dobutamine stress testing. Only one patient experienced a short episode of chest

![Figure 2](http://circ.ahajournals.org/)

**Figure 2.** Values for the change in systolic blood pressure (SBP) during dobutamine infusion are plotted for individual patients with obstruction (group 1) or without obstruction (group 2).
pain postoperatively without ECG changes; the patient was treated with a β-blocker.

Four patients in group 1 underwent changes in their antihypertensive regimens as a result of the stress tests, including discontinuation of diuretic therapy in two patients and a change from nifedipine to verapamil in two patients.

**Discussion**

Dobutamine is a synthetic catecholamine with predominately β1-receptor agonist activity. At the doses used in stress echocardiography, cardiac output is increased by augmentation of myocardial contractility and by an increase in heart rate. Stress-induced myocardial ischemia results in abnormalities in regional wall motion that can be detected echocardiographically. The utility of dobutamine stress echocardiography in the evaluation of coronary artery disease has been demonstrated.1

Doppler ultrasonography has been shown to accurately measure the peak pressure gradient across the left ventricular outflow tract in hypertrophic cardiomyopathy. In addition, a characteristic late-peaking pattern consistent with the dynamic nature of the pressure gradient is well recognized.8,9

Dynamic intraventricular obstruction during dobutamine stress echocardiography is a new observation; it developed in 21% of patients in our series. However, the development of a "functional obstruction," or gradient, between left ventricle and aorta has previously been shown to be caused in normal subjects without hypertrophic obstructive cardiomyopathy by interventions that increase the force of left ventricular contraction or decrease venous return to the left ventricle.10

The development of obstruction with the inotrope dobutamine may be more likely to occur in patients with higher resting ejection fractions and in those who exhibit a greater increase in heart rate with dobutamine stress. The development of dynamic obstruction is associated significantly with a decrease in systolic blood pressure. This remains significant even when patients with ischemia are excluded.

Among the patients in whom intraventricular obstruction develops with dobutamine, the double product of heart rate and blood pressure may significantly underestimate the actual increase in myocardial oxygen demand imposed on the mid-to-apical cavity by the obstruction. It is possible that dynamic obstruction may account for clinical symptoms of chest pain and dyspnea in some patients without flow-limiting coronary artery disease.

We do not think that the potential for development of intracavitary obstruction should limit application of this test. Obstruction resolves after termination of dobutamine infusion, and we have noted no clinically impor-

tant complications related to this obstruction. However, because of concern about hypotension induced by intracavitary obstruction, we have incorporated in our protocol administration of a bolus of 250–500 ml of normal saline in selected patients in whom hyperdynamic function develops during dobutamine stress echocardiography. The impact of this change in our protocol requires further assessment.

We recommend, therefore, that Doppler interrogation be performed in patients in whom hyperdynamic function develops with dobutamine infusion. Intracavitary obstruction rather than ischemia may explain a decrease in blood pressure during dobutamine stress echocardiography and may account for cardiac symptoms in some patients.

**Acknowledgment**

The authors appreciate the assistance of Allen R. Kunsman, Section of Biostatistics.

**References**


P A Pellikka, J K Oh, K R Bailey, B A Nichols, K H Monahan and A J Tajik

Circulation. 1992;86:1429-1432
doi: 10.1161/01.CIR.86.5.1429

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/86/5/1429