

LETTERS TO THE EDITOR

Paradoxical Sinus Deceleration During Dobutamine Stress Echocardiography

In the April 1997 issue of the Journal, Attenhofer et al. (1) observed paradoxical sinus deceleration in 14 (8%) of 181 patients during dobutamine stress echocardiography. Significant coronary artery disease was present in eight patients and absent in the remaining six. Activation of the Bezold-Jarish reflex, promoting reflex bradycardia, vasodilation and hypotension, is suggested as a probable mechanism.

In our experience, hypotension (systolic blood pressure decrease >30 mm Hg) without simultaneous deceleration of heart rate occurred in 8% of patients during dobutamine stress echocardiography (2). However, of 114 patients who also underwent coronary angiography for assessment of coronary artery lesions, heart rate increased in 12 of the 15 patients given a bolus of 1 mg of atropine, but only 2 patients achieved 85% of the maximal heart rate, and heart rate remained almost unchanged in 3 (2 without beta-adrenergic drugs). These three patients had significant coronary artery lesions (percent diameter stenosis >70%). Given the difficulty in achieving 85% of the maximal heart rate even in patients given atropine, heart rate changes as an indirect sign of atherosclerotic coronary artery disease were evaluated. We found that a heart rate <112 beats/min at the end of dobutamine stress echocardiography without treatment with beta-blockers showed a sensitivity of 65%, a specificity of 83% and a diagnostic accuracy of 73% for the diagnosis of coronary artery disease. In addition, heart rate <94 beats/min independently of the treatment received and abnormalities of myocardial contractility (multivariate analysis) showed a sensitivity of 69% and a specificity of 70% for the diagnosis of multivessel coronary artery disease (3).

In the study of Attenhofer et al. (1), although the number of patients treated with beta-blockers is not mentioned, seven of eight patients with significant coronary artery disease showed a heart rate <112 beats/min, and three of five patients with two- or three-vessel coronary disease showed a heart rate <94 beats/min. In the six patients without significant coronary artery disease, dobutamine stress echocardiography revealed myocardial contractility abnormalities in two, with atherosclerotic coronary lesions of percent diameter stenosis <50%. However, it is possible that a more accurate measurement of atherosclerotic lesions with the use of calipers could have resulted in a decrease in the number of false positive results.

Heart rate response to dobutamine stress echocardiography may include three possibilities: 1) acceleration up to 85% of maximal heart rate; 2) initial acceleration, followed by deceleration and hypotension through a vagal mechanism; and 3) limited increase in heart rate in patients with or without treatment with beta-blockers. In experimental studies, Vatner et al. (4) showed that in the absence of coronary artery disease, dobutamine increases heart rate in ~20% and cardiac contractility in 125%, whereas in the presence of coronary artery occlusion, there was an increase in heart rate and contractility of 15% and 32%, respectively, as opposed to exercise, in which case, heart rate increased by 102% and contractility decreased by 63%. Dobutamine directly stimulates adenosine triphosphate (ATP)- and cyclic adenosine monophosphate (cAMP)-dependent beta-adrenergic receptors, resulting in an increase in cardiac contractility and chronotropism. Stimulation of cAMP-independent alpha-adrenergic receptors causes an increase in cardiac contractility. In the initial phase of ischemia, there is a decrease in ATP production, which may induce a lower

response to stimulation of beta-adrenergic receptors and, consequently, a lower chronotropic response (5,6) without alteration of cardiac contractility because these events occur when there is an important increase in heart rate (7).

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Reply

Cladellas et al. report their interesting observation that a maximal heart rate <112 beats/min with dobutamine stress echocardiography in the absence of beta-blocker medications indicates significant coronary artery disease with a diagnostic accuracy of 73%. Cladellas et al. believe that the insufficient heart rate increase during high dose dobutamine infusion results from a decrease in adenosine triphosphate production due to myocardial ischemia. However, in 17% of their patients, an insufficient heart increase to <112 beats/min was seen in the absence of coronary artery disease. Thus, in their experience, an attenuated chronotropic response cannot always be attributed to myocardial ischemia.

In the experimental study by Vatner et al. (1), the absolute heart rate increase with dobutamine was modest but similar with and without ischemia (16 and 15 beats/min, respectively), although the baseline heart rate was higher in dogs with ischemia. Thus, despite myocardial ischemia, the chronotropic response to dobutamine was intact. The duration of infusion of 40 µg/kg per min dobutamine was brief, 1 to 3 min, and differentiates that experimental work from the more prolonged infusion typically used with dobutamine stress echocardiography and probably accounts for the much smaller increment in heart rate than is usually observed during dobutamine stress echocardiography.

Our work (2) concerned the paradoxical heart rate decrease occasionally observed during dobutamine stress echocardiography. Thus, in all patients a higher heart rate was obtained during dobut-