gency department evaluation of patients with acute myocardial infarction. The addition of a lipid collection tube to an existing clinical/laboratory pathway for acquisition of baseline cardiac enzymes is an ideal collection method. Even though the presence of nonfasting triglyceride levels produced a 5% lower Friedewald LDL estimate, the calculation did correlate well with the gold standard of ultracentrifugation, as does measurement by the immunoseparation method. In other words, the Friedewald measurement provides the same specificity with slightly less sensitivity than the other methods: All patients who were eligible for treatment or who had an LDL level >100 were correctly classified, but 1 patient (2%) with LDL <100 was misclassified as *not* eligible for antilipemic intervention, based on the National Cholesterol Education Program guidelines.[6]

The definition of the LDL cholesterol level at the time of an acute myocardial infarction provides physicians with an opportunity to address risk factor reduction during hospitalization rather than waiting 3 months after the event. This period of time may represent a window of opportunity for patient education about the role of elevated cholesterol and the provision of antilipemic therapy for secondary prevention of coronary artery disease. The initiation of antilipemic therapy for patients experiencing an acute myocardial infarction and who have an elevated LDL cholesterol has not yet been clearly defined, but a recent consensus committee suggested 4 to 6 weeks after the event.[6]

The measurement of LDL cholesterol by any of the 3 methods described above identifies patients eligible for treatment with antilipemic interventions. Accumulating evidence suggests that aggressive lipid-lowering therapy reduces the number of recurrent events, improves cardiac and overall mortality and improves the regulation of coronary arterial tone in patients with atherosclerosis.[7,8] It has also been demonstrated that beneficial effects can be seen as early as 3 months after the start of lipid-lowering therapy.[9] Aggressive lipid lowering may prove to be a key part of therapy immediately after acute myocardial infarction.

In summary, we conclude that lipid screening can be introduced into the emergency department clinical pathway for evaluation and treatment of patients admitted for acute chest pain or myocardial infarction despite the nonfasting state of most such patients by use of any of 3 methods of LDL determination.

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Assessment of Patients After Coronary Artery Bypass Grafting by Dobutamine Stress Echocardiography

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Detection of myocardial ischemia is the hallmark of noninvasive evaluation of graft function[1–5] after coronary artery bypass grafting (CABG). Exercise electrocardiography was shown to have a limited accuracy in this clinical setting.[3] Recent studies have demonstrated a high accuracy of exercise echocardiography for the detection of significant graft or progressive native coronary artery stenosis.[3–5] However, an alternative stress modality is required for patients with limited exercise capacity.

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The value of dobutamine stress echocardiography in the diagnosis and functional assessment of coronary artery disease has been established in various clinical indications.[6–11] However, its role in the assessment of patients after CABG has not been stud-

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ied. Therefore, the aim of this study was to assess the value of dobutamine stress echocardiography in the diagnosis of vascular compromise in CABG patients who were unable to perform adequate exercise stress testing.

The study population comprised 50 patients (mean age 58 ± 9 years; 41 men and 9 women) unable to perform exercise stress testing who were studied by dobutamine stress echocardiography 3 months to 19 years after CABG (mean 5.1 ± 5.4 years, range 3 months to 19 years). Thirty-nine patients (78%) had had a previous myocardial infarction, and 15 (30%) were receiving β blockers. Reasons for stress tests were chest pain in 36 patients and exertional dyspnea in 2; in 12 patients the test was performed for routine functional assessment.

TABLE I Sensitivity, Specificity, and Accuracy of Dobutamine Stress Echocardiography for Diagnosis of Significant Stenosis in Individual Vascular Regions in Patients With Previous Coronary Artery Bypass Grafting

	Sensitivity	Specificity	Accuracy
Left anterior descending artery	(14/22) 64%	(24/28) 86%	(38/50) 76%
95% CI	50–77	76–95	64–88
Left circumflex artery	(10/20) 50%	(24/30) 80%	(34/50) 68%
95% CI	36–64	69–91	55–81
Right coronary artery	(9/20) 45%	(23/30) 77%	(32/50) 64%
95% CI	31–59	64–88	50–77
Left circumflex and/or right coronary artery	(16/25) 64%	(21/25) 84%	(37/50) 74%
95% CI	51–77	74–94	62–86

CI = confidence interval.

Dobutamine-atropine (dobutamine ≤40 μg/kg/min, atropine ≤1 mg) stress echocardiography was performed according to the previously described protocol.[12] Ischemia was defined as new or worsening wall motion abnormalities. Ischemia on the electrocardiogram was defined as ≥0.1 mV horizontal or downsloping ST-segment depression 80 ms from the J point or ≥0.1 mV ST-segment elevation corresponding to noninfarcted regions.[12] Coronary angiography was performed within 3 months. Significant coronary artery disease was defined quantitatively[13] as a stenosis diameter ≥50% in a graft or native artery if the stenosis was distal to the graft or involved nongrafted artery. There was no significant vascular compromise in 18 patients, whereas 32 patients had graft or nongrafted coronary artery stenosis.

Heart rate increased from 70 ± 12 to 135 ± 18 beats/min at peak stress ($p < 0.001$), whereas systolic blood pressure did not change significantly (128 ± 20 vs 127 ± 26 mm Hg). Angina occurred in 15 patients (30%), ST-segment depression in 13 (26%), and ST-segment elevation in 10 (20%); in these patients, the condition was confined to infarction leads. The test was interrupted before the maximal dose or target heart rate was reached because of angina (n = 5), ST-segment depression (n = 1), hypotension (n = 2), and tachyarrhythmias (n = 1). ST-segment depression occurred in 10 patients with vascular compromise (3 had single region and 7 multiregion compromise) and in 3 patients without vascular compromise (sensitivity 31%; 95% confidence interval [CI] 18 to 44, specificity 83%; CI 73 to 94, positive predictive value 77%; CI 65 to 89, negative predictive value 41%; CI 27 to 54; accuracy 50%; CI 36 to 64).

Ischemia was detected during echocardiography in 25 of 32 patients with vascular compromise and in 2 of the 18 patients without it (sensitivity 78%; CI 67 to 90, specificity 89%; CI 80 to 98, positive predictive value 93%; CI 85 to 100, negative predictive value 70%; CI 57 to 82; accuracy 82%; CI 71 to 93). Sensitivity and accuracy were higher than electrocardiography ($p < 0.0001$ and < 0.001, respectively). Sensitivity was 64% (7 of 11) in patients with vascular compromise in 1 region, 83% (10 of 12) in 2 regions, and 89% (8 of 9) in 3 regions. There was a trend to higher sensitivity in patients with multivessel compromise compared with those with 1-vessel compromise (86% [18 of 21] vs 64% [7 of 11], $p < 0.1$). In patients with vascular compromise, peak heart rate was higher in patients with ischemia than without it at echocardiography (135 ± 22 vs 120 ± 18 beats/min, $p < 0.05$). Sensitivity was relatively higher with than without achievement of 85% of the target heart rate (86% [19 of 22] vs 60% [6 of 10]), respectively. An ischemic pattern in 2 different vascular territories, suggestive of multivessel compromise, occurred in 12 of 21 patients (57%) with multivessel and in 2 of 11 (18%) patients with 1-vessel compromise (sensitivity 57%; CI 40 to 74, specificity 82%; CI 68 to 95, positive predictive value 86%; CI 74 to 98, negative predictive value 50%; CI 33 to 67; accuracy 66%; CI 49 to 82). The sensitivity, specificity, and accuracy of dobutamine stress echocardiography for detection of regional vascular compromise are listed in Table I. With the use of 2 myocardial regions (left anterior descending [LAD] and combined left circumflex [LCX] and right coronary artery), ischemia was detected in 30 of 47 regions with vascular compromise (7 of 12 native arteries and 23 of 35 grafts) and in 8 of 53 regions without vascular compromise (2 of 13 native arteries and 6 of 40 grafts) (sensitivity 64%, specificity 85%). Sensitivity and specificity were not different in graft versus native arteries (66% [23 of 35] vs 58% [7 of 12] and 85% [34 of 40] vs 85% [11 of 13], respectively). Sensitivity in each vascular region was not different in the presence or absence of myocardial infarction in the same region (60% vs 65% in LAD and 69% vs 58% in the right coronary artery/LCX region, respectively, p = NS). Of the 8 false-positive regional diagnoses, 3 occurred in relation to lesions between 40% and 50%, and 4 occurred in regions with normal wall motion at rest. A false-positive diagnosis of ischemia in the septum occurred in 2 patients. Septal motion at rest was abnormal in 1.

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Coronary artery bypass grafting is an effective intervention for symptomatic relief, prolonged survival, and improvement of left ventricular function

in a particular subset of patients with coronary artery disease.[14] However, myocardial perfusion after surgery may be compromised by late graft occlusion and progression of coronary artery disease in nongrafted or grafted coronary arteries distal to the bypass.[15] Our study shows that dobutamine stress echocardiography is a moderately sensitive and highly specific technique for detection of vascular compromise in these patients. Sensitivity was relatively high in the presence of vascular compromise in >1 region. In individual vascular territories, sensitivity was not different in the presence or absence of myocardial infarction in the corresponding region. Despite the known effect of CABG on septal motion,[16] only 1 of the 8 false-positive results involved the septum with abnormal wall motion at rest.

The sensitivity (78%) and specificity (89%) of dobutamine echocardiography are aligned with those of the largest exercise echocardiographic study (79% and 82%, respectively) in patients after CABG.[3] Sensitivity and specificity of dobutamine echocardiography in our study are comparable to those reported in patients without CABG,[6-10] denoting the absence of particular limitations in this population.

Localization of inducible wall motion abnormalities identified the area of vascular compromise in a high proportion of segments. Improvement of regional sensitivity and specificity by combining the right coronary artery and LCX territories can be explained by the vascular overlap previously described in these regions.[4] Using the 2-segment model, an equal sensitivity was found in anterior (LAD) and posterior (right coronary artery and LCX) regions. There was a relatively low sensitivity for identifying multivessel disease on the basis of inducible abnormalities in >1 region (57%), which is slightly lower than that of exercise echocardiography (69%) reported by Kafka et al.[3] This can be explained by vascular overlap and different threshold for ischemia in different vascular territories with the potential to reach an end point of the test (angina, electrocardiographic, or hemodynamic changes and maximal dobutamine dose) before the occurrence of ischemia in other territories. In patients with vascular compromise, peak heart rate was higher in patients with ischemia than without at echocardiography consistent with the previous findings on dobutamine[17] and exercise echocardiography.[18]

The limitations of this study are the relatively small number of patients and the inclusion of patients with previous myocardial infarction. Some patients were receiving medication, including 30% who were receiving β blockers, which may decrease the sensitivity of dobutamine stress echocardiography. Nevertheless, we have previously shown that administration of atropine enhances the sensitivity of dobutamine stress echocardiography, especially in patients receiving β blockers.[19] It is not known if the 50% diameter stenosis used to define a significant native artery stenosis would apply to bypass grafts to predict functional abnormalities as defined previously[3-5] and in this study. However, we could not find a difference in sensitivity and specificity in myocardial regions with native or grafted arteries. A particular difficulty would arise in regions of vascular overlap between native and grafted arteries if a different cutoff point were used to define coronary stenosis in each artery.

In conclusion, dobutamine stress echocardiography is an accurate method for diagnosis of vascular compromise in patients after CABG and provides useful data for selection of patients for whom coronary angiography may be indicated.

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