nary angioplasty, or myocardial infarction; and use of digitalis.

Study design and validity This was an investigator-blinded cross-sectional study. All participants underwent multistage Bruce protocol ETT with the standard 12 electrocardiogram leads as well as 3 right precordial leads (V_{3R}, V_{4R}, and V_{5R}) and thallium-201 scintigraphy. During ETT, ischemia was defined as: a horizontal or downsloping ST-segment depression of at least 1 mm 60 µsec after the J point; an upsloping ST-segment with a depression at least 1.5 mm 80 µsec after the J point; in the presence of ST-segment depression at rest, an additional 2 mm of ST-segment depression; or an ST-segment elevation of at least 1 mm. Exercise was terminated if the patient developed severe angina, fatigue, dyspnea or arrhythmias; 3-mm ST-segment depression; 2-mm ST-segment elevation; or a decrease in systolic blood pressure of 20 mm Hg or more. Thallium-201 scintigraphy stress and redistribution images were analyzed by standard qualitative and quantitative techniques. CAD was defined by a narrowing of at least 70% of the left anterior descending, left circumflex, or right coronary arteries, or narrowing of at least 50% of the left main coronary artery, as determined by arteriography.

Results of the 12-lead set and right precordial set were interpreted separately as well as in combination. All medications were discontinued 5 half-lives before testing. Within 2 months of ETT, all patients underwent coronary arteriography and left ventriculography. Investigators in each of the 3 diagnostic arms (ETT, thallium scintigraphy, and coronary arteriography) were blinded to results of the other diagnostic studies. Arteriography was the reference standard for defining CAD.

Outcomes measured The primary outcomes were the sensitivity and specificity of the ETT. Likelihood ratios were calculated by the authors.

Results Arteriography revealed that 14% of patients had normal coronary arteries, 35% had single-vessel disease, 34% had 2-vessel disease, and 17% had 3-vessel disease. In comparison with the standard 12-lead ETT, the additional right precordial leads increased sensitivity for detection of single-vessel disease from 52% to 89%, for 2-vessel disease from 71% to 94%, for 3-vessel disease from 83% to 95%, and for all cases of CAD from 66% to 92% (P <.001). Moreover, sensitivity of detection of single-vessel right CAD increased from 25% to 89% (P <.001) and the sensitivity of left circumflex CAD increased from 45% to 86% (P <.004). Differences in sensitivity between ETT with right precordial leads and thallium-201 scintigraphy were not statistically significant; neither were differences in specificity between the standard 12-lead ETT, ETT with the addition of right precordial leads, and thallium scintigraphy (88%). The positive and negative likelihood ratios for detection of any CAD were 5.5 and 0.4 for standard ETT, and 7.7 and 0.1 with the addition of right precordial leads.

Recommendations for clinical practice **The addition of 3 right precordial leads to the conventional 12-lead exercise electrocardiogram improves detection of CAD, as defined by coronary arteriography, such that sensitivity rivals exercise thallium-201 scintigraphy. This modification also significantly improves the negative likelihood ratio, a measure of how well a negative test rules out disease. These results are intriguing because they propose a low-cost, widely available, and noninvasive improvement to current CAD screening tools. Nonetheless, a prospective randomized controlled trial is needed to determine if this technique has a meaningful impact on mortality and morbidity when compared with standard 12-lead ETT or thallium-201 scintigraphy. A study incorporating more women would also be important, since preliminary findings suggest that greater sensitivity in CAD detection may also be achieved for this group.**

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Comparing Troponin I with Creatinine Kinase

Falahati A, Sharkey SW, Christensen D, et al. Implementation of serum cardiac troponin I as marker for detection of acute myocardial infarction. Am Heart J 1999; 137:332-7.

Clinical question **Should cardiac troponin I (cTn-I) replace creatine kinase and its MB isoenzyme as a biochemical marker for the diagnosis of acute myocardial infarction?**

Background The standard biochemical markers for the diagnosis of acute myocardial infarction (AMI) are elevations in the total creatine kinase (CK) and its MB isoenzyme (CK-MB). Recently, subunits of the troponin complex in myocardial tissue have shown promise as markers of myocardial cellular damage. In particular, cTn-I appears to be unique to cardiac tissue and thus should have fewer false-positive values. Recent studies have shown that cTn-I may be a more sensitive and specific marker, and that it may give a positive result earlier in the course of a myocardial infarction than CK-MB.

Population studied The investigators prospectively enrolled an unselected, consecutive series of 327 patients admitted to an urban midwestern hospital for

evaluation of an acute episode of chest pain. Patient demographics were not reported. Only 62 (19%) met the criteria for AMI.

Study design and validity This was a prospective cross-sectional study. Patients with acute chest pain were admitted to the hospital to receive care appropriate for their clinical situation. Serial total CK, CK-MB, and cTn-I measurements were determined on admission and every 6 to 8 hours for at least 24 hours. The upper limit of normal for CK-MB was 5.0 grams per liter and 0.8 grams per liter for cTn-I. AMI was defined by modified World Health Organization criteria, which required at least 2 of the following: typical chest pain for more than 30 minutes; evidence of ischemic changes on the electrocardiogram; and an elevation in the CK-MB level to > 5.0 grams per liter or a change of ≥ 25% between 2 CK-MB measurements.

The study had several important limitations. Most important is the fact that CK-MB is being tested while also being part of the diagnostic criteria. This generally has the effect of inflating the measured accuracy of CK-MB, although it should not affect the evaluation of cTn-I. Also, only approximately two thirds of the 327 patients enrolled had sufficient data to analyze (unpublished data). Finally, the population was not characterized with respect to demographic or cardiac risk factors.

Outcomes measured The primary outcomes were the sensitivity and specificity of cTn-I and CK-MB during the first 24 hours of hospitalization.

Results The sensitivity, specificity, positive likelihood ratio and negative likelihood ratio are shown in the Table. The specificity is a correction from that quoted in the article, obtained through personal communication with the investigators.

The sensitivity of cTn-I was significantly higher than that of CK-MB, indicating that cTn-I is more likely to detect an AMI that has occurred. The lower negative likelihood ratio for cTn-I suggests that this test is better at ruling-out AMI when negative. The difference in specificity between the 2 tests was not statistically significant. Total CK levels, as expected, were less sensitive and less specific than either CK-MB or cTn-I levels. Although cTn-I appears more sensitive than CK-MB, the test did not reach the peak sensitivity until at least 12 hours after onset of symptoms. Within 6 hours of the onset of chest pain, all tests had a sensitivity of less than 40%.

Recommendations for clinical practice **Of the 3 markers examined, cTn-I was the most accurate test to rule out myocardial infarction in this group of unselected patients. Because of the probable bias from using CK-MB as part of the diagnostic criteria, the apparently better specificity of CK-MB is likely artifactual. The cTn-I marker offers no reduction in time necessary to rule out AMI, but its intermediate half-life may allow it to replace both CK-MB and lactate dehydrogenase in testing for AMI. The cTn-I test may offer other prognostic information as well, since it remained increased longer after a Q-wave than a non-Q–wave AMI. If, as the authors state, the cost and ease of use of CK-MB and cTn-I are comparable, then cTn-I appears to be the best biochemical marker available to determine the presence of AMI. However, a larger study with better attention to detail is needed before widespread acceptance of cTn-I. It would also be of value to closely examine patients with discordant results between the cTn-I and CK-MB tests.**

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TABLE. Sensitivity, Specificity, Positive Likelihood Ratio, and Negative Likelihood Ratio of Cardiac Troponin I (cTn-1)

Marker	Sensitivity, %	Specificity, %	LR+	LR-
CK-MB	88.2	93.2	13.0	0.1
cTn-I	100	90.6	10.6	0.005

LR+ denotes positive likelihood ratio; LR-, negative likelihood ratio.

Bed Rest for Sciatica?

Vroomen PC, de Krom MC, Wilmink JT, Kester AD, Knottnerus JA. Lack of effectiveness of bed rest for sciatica. N Engl J Med 1999; 340:418-23.

Clinical question **Is there any benefit to 2 weeks of bed rest for sciatic back pain?**

Background There is accumulating evidence that bed rest is not helpful for the treatment of uncomplicated low back pain. However, most of these studies have excluded patients with sciatica. This was the first study to test whether bed rest has benefit for patients with lumbosacral radiculopathy.

Population studied Eligible patients were those referred by general practitioners to a neurology department in the Netherlands. Patients were included in the study if they had back pain radiating into one leg (below the gluteal fold) and enough pain to justify 2 weeks of bed rest, which is a standard therapy for sciatica in the Netherlands. Patients were excluded if they had prior back surgery, any pending workers' compensation