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Myocardial Viability

The major reason why a clinical cardiologist pursues myocardial viability is to reduce cardiovascular mortality. Although there are no data about it, the rationale basis is the preservation of ventricular function by the identification of reversible regional myocardial dysfunction, leading to a lower risk of death, assuming that the better the ejection fraction the lower the mortality. The problem rests on the risk/benefit ratio. The risk is to revascularize through bypass surgery or coronary angioplasty, taking into account clinical condition, inherent procedural complications, mortality and restenosis rates. In the case of coronary angioplasty, low flow conditions may lead to hibernating myocardium. There are widely available data about myocardial viability but none answering the question of how much viable myocardium is worth justifying the risk of the revascularization procedure and a significant increase in ejection fraction. It seems that we have a long way to go because the accepted documentation of viable myocardium is made just *after* revascularization, and there is no "gold standard" method for predicting reversibility of contractile dysfunction. Elhendy et al. (1) in a recent issue of the *Journal* observed that 14 patients with reversible single-photon emission computed tomographic imaging perfusion defects had no evidence of myocardial ischemia as assessed by wall motion abnormalities on stress echocardiography with dobutamine infusion, a finding commented on by Wackers (2). But in this group of patients, four (28.5%) showed improvement of contraction in the infarcted region, a finding that should be interpreted as an evidence of ischemia if we accept the idea that all ischemic myocardium is viable myocardium. In addition, all patients had documented coronary artery disease (diameter stenosis >50%), leading to the hypothesis that improvement of wall motion abnormalities observed in the infarct area is consistent with the presence of hibernating myocardium.

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Reply

We thank Shibata for his interesting remarks on our study of patients with dobutamine-induced hypoperfusion without transient wall motion abnormalities (1). Among the 14 patients without transient wall motion abnormalities in our study, reversible perfusion defects were confined to dyssynergic segments in 4 (29%). However, only two of these four patients showed improved wall thickening at low dose

dobutamine. Improvement was maintained at high dose. These patients were not considered to have ischemia at echocardiography because we used the standard criteria of new or worsening wall motion abnormalities to define an ischemic response. As Shibata points out, myocardial ischemia should also be considered in the presence of evidence of myocardial viability, as in the case of improvement of a dyssynergic segment during low dose dobutamine infusion. Although myocardial ischemia and hibernation are related phenomena, we did not consider the improvement at low dose dobutamine as a definite ischemic response because we have shown (2) that only akinetic segments with a biphasic response to dobutamine have a relatively high prevalence of reversible thallium defects (51% vs. 9% of segments with persistent improvement, $p < 0.005$) as well as a higher prevalence of functional improvement (3) after revascularization (79% vs. 30% of segments with persistent improvement, $p < 0.005$). It is possible that in our study (1), the low rate-pressure product in the group without transient wall motion abnormalities did not allow for the occurrence of a biphasic response, and consequently, dyssynergic segments showed a persistent improvement rather than a biphasic response. This observation supports our recommendation that every effort should be made to attain a high rate-pressure product to elicit myocardial ischemia at dobutamine echocardiography. The conclusion of our study was that the absence of transient wall motion abnormalities in patients with reversible perfusion defects does not imply less severe ischemia but is associated with a lower rate-pressure product. Inclusion of patients with a previous myocardial infarction in our study did not seem to limit our conclusion because we have recently demonstrated (4) that in patients with infarct-related artery stenosis, the prevalence of peri-infarction ischemia at dobutamine stress echocardiography, using the standard criteria of new or worsening wall motion abnormalities, is not significantly different from the prevalence of ischemia according to reversible hypoperfusion on simultaneous 2-methoxy isobutyl isonitrile (MIBI) single-photon emission computed tomography (4).

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Increased Neopterin in Patients With Chronic and Acute Coronary Syndromes

I read with interest the report by Schumacher et al. (1) regarding plasma neopterin levels in acute and chronic coronary syndromes.