Reply

To the Editor:

We appreciate the opportunity of answering the points raised in the Letter to the Editor that was forwarded on July 6, 2010.

The first point raised by the author(s) of this letter regarding our article[1] is that ''it is not possible to conclude that the '… specificity of ECG changes during adenosine infusion for the detection of severe obstructive CAD is poor.''' Strictly speaking, this is true, as the number of patients without severe obstructive CAD who did not have ischemic ECG changes—the true negatives that would figure in both the numerator and the denominator of the specificity formula, where we to have included all patients with normal MPI studies in our study and not just those with ischemic ECG changes and normal MPI studies—was not ascertained. By design, these patients were excluded and this number was made to be zero. Thus, the numerator in the specificity formula is zero and anything divided into zero (in this case the number of false positives plus the number of true negatives, which is zero) is zero. Hence, our qualified statement was ''Thus, in the current study, the specificity of ischemic ECG changes occurring during the infusion of adenosine for detecting obstructive CAD in the presence of normal MPI was found to be poor''—i.e., zero.

The second point raised is that ''… patients with multiple coronary risk factors, particularly diabetes mellitus, should undergo further investigation'' and the author(s) asks ''Why?'', noting that ''All patients had a 0% incidence of a hard cardiac event (cardiac death or nonfatal myocardial infarction) during follow-up.'' In fact, what is stated in our article[1] is that ''… our findings do suggest that diabetic patients who have adenosine-induced ischemic ECG changes and patients with adenosine-induced ST-segment depressions lasting for >5 minutes have a higher incidence of obstructive CAD even though they have normal MPI. Such patients should be considered for further work up.'' Our statement was made because diabetic patients made up the majority of those that had soft events, i.e., revascularization procedures, in our group. As noted, the total rate of hard cardiac events was very low, i.e., 0%; this may have been due, in part, to the fact that high-risk patients, in this case, patients with diabetes mellitus and those with prolonged ischemic ECG changes, underwent angiography at an early stage and their CAD was discovered early.

The author(s) further states ''… their data provide no proof that angiography or revascularization was beneficial. A literature review may allow this hypothesis, but their data does not.'' (sic) What we state is that ''The fact that 10 patients in our study underwent revascularization as a result of the index MPI study, however, undoubtedly had a positive impact on the hard event rate (0%) that we report.'' While no definitive proof of this is provided by our data, the 0% hard event rate seen during a follow up period of 24 ± 13 months is very suggestive of a beneficial effect, given the severity of disease in these patients. The ethics of conducting a study to test this hypothesis would be questionable.

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Reference

1. Sharma J, Roncari C, Giedd KN, Fox JT, Kanei Y. Patients with adenosine-induced ST-segment depressions and normal myocardial perfusion imaging: cardiac outcomes at 24 months. J Nucl Cardiol 2010 [Epub ahead of print].

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