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## REPLY

We thank Dr. Hardebeck for his comments about our study; his comments provide an opportunity to clarify our concepts regarding the finding of "transient ischemic dilation" or "TID" of the left ventricle.

First, research from our laboratory and others clearly established that the finding of TID is a worrisome finding on myocardial perfusion single-photon emission computed tomography (MPS), and one that is often associated with severe and extensive coronary artery disease (CAD) (1–4). After the initial case report by Stolzenberg (5), our first contribution on this subject with planar thallium studies (1), we reasoned that the finding of TID was likely to represent extensive ischemia, because: 1) it was reversible; 2) the amount of ischemia had to be large enough to cause apparent transient ventricular enlargement even in a planar study; and 3) such ischemia was likely to be severe because it lasted for at least one-half hour after stress, a time at which usual exercise-induced ischemia would be expected to have been resolved. This hypothesis was supported by our observations that TID is predictive of proximal left anterior descending (LAD) coronary artery or multiple vessels with >90% stenosis. That the finding represented ventricular dilation and was not due to extensive subendocardial ischemia was suggested by the fact that the regions of interest were drawn on the epicardial and not the endocardial boundaries (6). It has subsequently been suggested that TID can also be caused by extensive subendocardial ischemia, in the absence of true left ventricular (LV) dilation (2,3). The relative frequencies of these underlying processes are likely to vary with the population being studied. In either case, the finding is suggestive of extensive pathology. The relationship of TID to severe and extensive disease was subsequently investigated by our laboratory with planar dipyridamole thallium studies (7) and then with dual isotope MPS (3), with findings that supported our initial observations.

Second, our current study (8) describes the largest population of patients with perfectly normal rest/stress MPS that has ever been reported. Our primary goal was to evaluate the prognostic and not the diagnostic implications of TID (as measured by the TID ratio) in this population, which is at low risk, but not necessarily at very low risk, of subsequent cardiac events. Our results clearly demonstrated that TID has independent and incremental prognostic value over clinical variables in these patients with perfectly normal MPS after all clinical variables—hemodynamic data, historical variables (including history of hypertension), and resting electrocardiogram (ECG) abnormalities—were considered in the multivariable analysis for predicting future cardiac events.

Third, the principal concern of Dr. Hardebeck is that severe CAD was seen in only a minority (5 of 20) of the patients with TID who were subsequently catheterized. Of importance, only 65 of 1,560 patients were catheterized, indicating that this was a very selected group. Our findings did indicate that severe and extensive CAD was found solely in the patients with TID; however, owing to the very small proportion of our population represented by these patients, it is difficult to draw conclusions from these findings. The diagnostic accuracy of TID is yet to be evaluated in this clinical setting.

Fourth, again, our intent was not to further assess the diagnostic value but rather the prognostic value of the finding of TID with perfectly normal MPS. Dr. Hardebeck's comments that subendocardial hypoperfusion, left ventricular hypertrophy (LVH), and high left ventricular end-diastolic pressure (LVEDP) might represent a reason for the TID to be valid, but at the same time his comments do not detract from the prognostic value of the finding. All of these conditions are pathological and are by themselves likely to indicate a worse prognostic outcome compared to normal patients. The higher prevalence of TID in these patients (especially in those without apparent perfusion defects) only makes our point stronger.

Finally, we would like to note that the material in Robinson et al. (9) is a pictorial essay and not a clinical report, and that the material cited dealing with LVH represents only preliminary reports from 1997. The authors of these reports should be encouraged to submit full manuscripts for peer review so that their interesting preliminary observations can be more completely evaluated.

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