Giant T-Wave Inversion in Patients With Acute Coronary Insufficiency

To the Editor:

In the April 1992 issue of Chest, Fisher and colleagues1 reported the cases of an interesting series of patients with giant T-wave inversion and acute coronary insufficiency. In view of our recent report of global T-wave inversion2 and their Figure 1, which is an example of global T-wave inversion fulfilling our criteria,3 I should like to ask the authors to clarify some points in their report.

First, the methodology includes the ECG criterion “T-wave inversion greater than or equal to 10 mm in at least two contiguous precordial leads.” The leads are not specified by case in their table, and it would be important to know how many leads were involved in each case. Was Figure 1, for example, typical? We found that same ubiquitous T-wave inversion pattern to occur in coronary disease, but to be nonspecific; a minority of our patients had coronary disease (and few had giant inversions). If, however, two- or three-lead involvements were more typical than Figure 1 suggests, coronary disease in the authors’ series is better understood.

Finally, the authors’ Methods section excluded patients whose ECGs showed left ventricular hypertrophy (LVH). Table 1, however, lists six of nine patients as having LVH, and the authors conclude that a possible cause of the patterns they report may be ischemia “and some degree of left ventricular hypertrophy.” Clarification would be most helpful; for example, was the LVH in six of nine patients determined by echocardiography or other imaging?

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We thank Dr. Spodick for his interest in our article and offer the following additional data on our patients.

The precordial leads in which the criteria for giant T-wave inversion were met in our nine patients are as follows: patient 5, V3 and V4; patients 2, 6, and 7, V2 and V3; patient 8, V3 through V6; patient 4, V3 through V5; and patients 1, 3, and 9, V2, V3, and V4.

It should be noted that while these were the leads with giant T-wave inversion, patients 2 through 9 had some T-wave inversion in V3 through V5, and patient 1 had T-wave inversion in V1 through V6. It is important to recognize that our patient population was probably different from that screened by Dr. Spodick. We limited our screening to patients admitted to our coronary care unit and, as would be expected, found mainly patients with coronary disease.

Our diagnosis of LVH in six of the nine patients was determined by echocardiogram. We did exclude patients with ECG evidence of LVH but were not surprised to find echocardiographic evidence of LVH since echocardiography is known to be far more sensitive.

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Communications to the Editor