antiarrhythmic effect according to the drugs of group III. Thus, it is expected that tiapamil may be effective in preventing ventricular arrhythmias in the setting of acute myocardial ischemia. Preliminary results of Thandroyen et al.[3] and a recent study from our laboratory[4] confirmed that tiapamil is highly effective in preventing coronary artery occlusion and reperfusion arrhythmias in a dog model. Kordent and Leasure[1] did not report their observation on ventricular arrhythmia and the causes of death following coronary artery occlusion in their study. However, a careful analysis of this issue might add significantly to the understanding of the mechanism by which tiapamil exerts its beneficial effects during acute coronary occlusion.

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REPLY

To the Editor:

The antidysrhythmic effect of tiapamil and other calcium entry blocking drugs is well known. Because the goal of our study was to evaluate the possible protective effect of tiapamil on limiting infarct size, we wished to suppress this abnormal ECG conduction pattern that might influence this variable. Continuous intravenous lidocaine was used to maintain sinus rhythm during the monitoring and recording periods in both acute groups. It was not possible to continuously monitor the ECG through the chronic period of the study or to continue the intravenous lidocaine administration. In the animals that did not survive the 8-day infarction period, the deaths were assumed to be electrical. The antidysrhythmic effect of tiapamil could therefore not be objectively reported. I agree that rhythmic differences in the ECG would contribute to the evaluation of the overall protective effect of tiapamil in acute myocardial infarction, and if the funds to study this variable would be presented to us, we would be happy to report the results.

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THE Q-WAVE AND TRANSMURAL AMI

To the Editor:

Buda et al.[1] present interesting and potentially valuable material on regional left ventricular function during infarction. However, they may have inadvertently contributed further to the conceptual confusion which can follow the all-too-frequent use of misnomers in science. There has been sufficient literature on the nonspecificity of the ECG for distinguishing anatomically transmural from nontransmural infarcts.[2,3] Indeed, their results with Q wave and non-Q wave infarcts support some of the demonstrated differences and overlap between these two ECG categories that are inadequate to represent anatomy.[3] This would be a mere terminologic quibble if it were not for the importance of getting our descriptors right. Q wave and non-Q wave infarcts do differ in morbidity and the secular distribution of mortality, among other things.[3] Assuming that there is a corresponding anatomic distribution that may be responsible for this when that is incorrect, tends to discourage further investigation as to the real basis for differences. This is not in direct criticism of an otherwise important study.

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REPLY

To the Editor:

Dr. Spodick's point is well taken, and I agree that the ECG is an imperfect marker of infarct localization and transmurality. Several clinical[1-3] and experimental studies[4-6] document the occurrence of Q waves with nontransmural infarction, as well as their absence with transmural infarction. I am well aware of these studies, since it was from this Institution that Wilson et al.[4,5] reported over 50 years ago in this JOURNAL that epicardial qR waves originated from sites in which "the outer layers of muscle were alive and were responding to the excitatory process." And, after half a century, these experimental observations have recently been reevaluated and reconfirmed.[6]

However, in our defense, the terminology "transmural myocardial infarction" is traditional and continues to be employed in recent articles of several other prominent cardiovascular journals.[7-9] This is not to say that this terminology is correct or appropriate. However, tradition has a way of fixing terminology despite advancing knowledge and improved understanding. We have used the traditional term "transmural" rather than the more appropriate term "Q wave" as proposed by Spodick.[10] Although I agree with Dr. Spodick's choice of descriptor, the dichotomy of terms continues to exist, as demonstrated in the 1985 American Heart Association abstracts. Of 55 clinical studies indexed under

myocardial infarction, seven used the term "transmural" and six used "Q wave." It is clear there is no consistency of terminology.

Thus, our intent in using "transmural" was not to contribute further to the conceptual confusion, but merely to be traditional.

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ECHOGRAPHY OF PERICARDIAL MESOTHELIOMA

To The Editor:

Agatston et al.[1] report echocardiographic findings in primary pericardial mesothelioma of considerable interest. I wonder if the authors would reexamine Fig. 2. The structure interpreted to show early diastolic anterior motion followed by abrupt diastasis, a pattern one would anticipate with restrictive physiology, appears to be a Swan-Ganz catheter, rather than the RV free wall.

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REFERENCE

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REPLY

To the Editor:

We reviewed Fig. 2 from our article "Echocardiographic findings in primary pericardial mesothelioma" (AM HEART J 1986;111:986), along with the real-time two-dimensional echocardiogram of our patient. It is apparent that the echo labeled "right ventricular free wall" is in fact the Swan-Ganz catheter. The underlying right ventricular free wall is hypokinetic secondary to pericardial and myocardial tumor. We thank Dr. Spodick for pointing out this misinterpretation.

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CARDIAC ALPHA-RECEPTORS AND CARDIAC HYPERTROPHY IN GENETIC PREDISPOSITION TO HYPERTENSION

To the Editor:

The interesting study of Radice et al.[1] gives evidence for cardiac hypertrophy in normotensive young men with at least one hypertensive parent. Since the nature of myocardial alterations underlying the pathogenesis of this prehypertensive cardiac hypertrophy is still unknown and difficult to investigate in the human heart, it is worth using suitable animal models for experimental studies. Spontaneously hypertensive rats (SHR) develop cardiac hypertrophy before an elevated blood pressure can be detected.[2,3] Furthermore, Syrian hamsters of the inbred strain BIO 8262 with cardiomyopathy (BIO 8262) develop cardiac hypertrophy following myocardial necrosis before heart failure occurs.[4] Since in both SHR and BIO 8262, cardiac hypertrophy occurs before the onset of hypertension or heart failure,[2-4] these animal models provide strong similarities to the subjects studied by Radice et al.[1]

A crucial role of norepinephrine to trigger the cardiac hypertrophy process is well recognized.[5] Recent studies on isolated cardiac myocytes provide strong evidence that this effect is mediated by cardiac α-adrenoceptors.[6,7] We now report an increased sensitivity to stimulation of cardiac α-adrenoceptors in SHR and BIO 8262 before the onset of hypertension and heart failure, respectively. Young SHR and BIO 8262 were investigated in the absence of hypertension and cardiac necrosis, respectively (i.e., twenty-fifth to thirty-second day of life). Age-matched Wistar rats and