patients." Our experience,[2] as that of Ogawa et al,[1] highlights the fact that there is really no "safe" or "small" or "insignificant" infarction, particularly if the ECG characterization is too stringently adhered to. Thus, we have stated[2] that "acute nontransmural MI is not benign and an unstable period exists for 3 months thereafter. Because of this, more aggressive diagnostic measures should be instituted during this period in order possibly to improve prognosis in this group."

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REPLY

To the Editor:

I am interested to read the comments and observations of Dr. Przybojewski. I describe the cause of difference in the results of the two investigations[1,2] as far as I understand. About the incidence of previous myocardial infarction, we did not look at all impacts of prior myocardial infarctions in our patients. In the cases in which we did not look at the impacts, we examined the ECG changes (especially abnormal Q waves, ST-T changes); we conducted precise interviews with patients, families, or the physicians in charge; we examined two-dimensional echocardiograms, especially asynergy and wall thickness; we did thallium myocardial images; and we performed autopsies. We judged prior myocardial infarctions from these data overall. Therefore we think our estimation about prior myocardial infarctions was almost exact. However, we cannot deny the possibility that we did not detect very small prior myocardial infarctions (especially small non-Q wave infarctions).

When the incidences of postinfarction angina and reinfarction were analyzed, I think the differences of therapy methods to acute myocardial infarctions became the subject of discussion to no small extent. In principle, we treated acute myocardial infarction patients by preventive use of nitrates if they did not suffer from post infarction angina. For the patients who suffered from post infarction angina with nitrate therapy, we added calcium antagonists or beta blockers. Furthermore, all patients continuously received these similar treatments in hospital and thereafter as outpatients.

Generally in the acute phase, the mortality is not higher in patients with non-Q wave myocardial infarction than in those with Q wave myocardial infarction. In some patients, however, non-Q wave myocardial infarction is complicated by severe left ventricular failure or cardiogenic shock. They have ECG changes with severe and extensive ST segment depression at the onset.[3] In our study group, there were not a few non-Q wave myocardial infarction patients with severe left ventricular failure. Therefore, it is thought that there was no difference in in-hospital mortality between our groups of non-Q wave and Q wave myocardial infarction patients. In conclusion, we agree with the statement made by Przybojewski et al. Non-Q wave myocardial infarction is more unstable than Q wave myocardial infarction in the clinical course. Thus, more aggressive diagnostic and therapeutic methods are needed for non-Q wave myocardial infarction.

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SERUM MAGNESIUM AND MONONUCLEAR BLOOD CELL MAGNESIUM

To the Editor:

Several studies have not found a significant correlation between the concentration of serum magnesium and the content of magnesium in mononuclear blood cells.[1-5] A recent article by Ryzen et al.[6] reported a significant correlation (correlation coefficient = 0.74, $p < 0.001$) by combining hypomagnesemic and normal control subjects. We believe an inappropriate merging of samples from two different populations led to this conclusion.

The authors[6] had two control groups for their study. One consisted of 27 normal individuals in good health, and the other had 33 individuals with hypomagnesemia as a result of chronic alcoholism and/or malabsorption. They combined both of these control groups to determine their correlation coefficient and p value. In Table I the authors show a significant difference ($p < 0.001$) for the mean value for serum magnesium and mononuclear blood cell magnesium between the normal and hypomagnesemic control groups. Thus, these two groups represent samples from two statistically different populations. We took the data presented in Fig. 3 and determined the classic correlation coefficient and p value for the relationship between the serum magnesium and the mononuclear blood cell magnesium for each group. We found the normal control group had a correlation coefficient of 0.13 with a p value of 0.52. The hypomagnesemic control group had a correlation coefficient of 0.25 with a p value of 0.17. Thus, each of the control groups does not have a significant correlation coefficient. There is a statistical requirement for a homogeneous population to compute a meaningful correlation coefficient.[7] Thus, we believe the data by Ryzen et al.[6] and the previous studies noted above document a lack of correla-