13. Doherty PW, Lipton MJ, Berninger WH, Skiolebrand CG, Carls-

The author replies:

To the Editor:

We appreciate the attention given to our paper by Bianco and Taylor. They emphasize the concept that $^{99m}$Tc-PYP concentration to the level of blood flow in myocardial infarction is not the diffuse cardiac uptake of $^{99m}$Tc-PYP, which enhances myocardial infarctions on computed tomography. As indicated by Zaret et al., we accepted these earlier results of Zaret et al. and Marcus et al. To demonstrate this relationship, the number of samples obtained through the infarct in these previous studies was greater and the size of samples smaller than in our study. As indicated by Zaret et al., the PYP ratios were different (lower) when large myocardial samples were used. The infarction model used in their study is similar to the dense infarct model in our study. When one compares these models, which were similar, our results for $^{99m}$Tc-PYP distribution do show a rough relationship to residual blood flow and are actually consistent with the results of Zaret et al.

As indicated in the discussion of our paper, we accepted these earlier results of Zaret et al. and Marcus et al. and had no desire to reproduce their experimental model or experimental procedure.

The purpose of our study was to demonstrate that contrast medium, which enhances myocardial infarctions on computed tomographic (CT) scans, has a distribution similar to that of the infarct avid radionuclide agent, $^{99m}$Tc-PYP. We have learned empirically, as have others who study myocardial infarctions with CT scans, that the patterns of enhancement in some instances involve the entire infarct, whereas in others it involves the periphery of the infarct. Since such patterns are also observed with $^{99m}$Tc-PYP imaging, our hypothesis was that the images produced by contrast media on CT and $^{99m}$Tc-PYP were dependent upon the same factors. The accumulation of $^{99m}$Tc-PYP throughout the infarct has been found to be a pattern observed with subendocardial infarctions in experimental animals and in man. The diffuse cardiac uptake of $^{99m}$Tc-PYP is indeed nonspecific, as discussed in the letter by Bianco and Taylor. However, this pattern of diffuse cardiac uptake was not even considered in our experiments. Careful consideration of the Methods and Results portions of our paper should indicate that diffuse distribution of the radionuclide throughout the infarct is not the same as the diffuse cardiac uptake of $^{99m}$Tc-PYP addressed in the paper by Massie et al. and shown by others to be a nonspecific pattern.

The scope of our paper was not to define the precise relationship of $^{99m}$Tc-PYP concentration to the level of blood flow in myocardial infarctions, but to demonstrate that contrast media, like $^{99m}$Tc-PYP, can be used as a marker of myocardial infarctions on CT scans; that contrast media have a distribution in myocardial infarctions similar to that of $^{99m}$Tc-PYP; and that increased concentration of both contrast medium and $^{99m}$Tc-PYP within myocardial infarctions is primarily dependent upon the presence of myocardial necrosis and a threshold level of residual myocardial blood flow.

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References

Problems of QT Measurement

To the Editor:

Like Ahnve and Vallin, we examined at the influence of autonomic tone on the QT interval, but in the Romano-Ward syndrome. We were interested in features that might distinguish those most at risk of developing further arrhythmias. Measurement of QT interval was frequently impossible because the TU wave complex varied so much in shape in each patient during autonomic maneuvers and encroached on the P wave at high heart rates. Abnormal changes in the TU configuration itself were therefore examined in addition. Five symptomatic members from two families with the syndrome were studied, and all but one had clear prolongation of the QT interval at rest, and four others had apparently normal relatives. All those affected had syncpe while swimming in cold water as well as with a combination of severe phyiscal and emotional stress, but attacks were infrequent.

The Frank orthogonal lead system coupled to a Siemens Elema recorder was used and continuous recordings were made during handgrip, Valsalva, staged maximal exercise using the Bruce protocol, and cold immersion of the face coupled with prolonged breath holding (dive reflex). None of these maneuvers produced arrhythmias, and significant repolarization changes, i.e., giant U or bifid T waves, only occurred in the symptomatic group. Exercise repolarization was abnormal and remained so for more than 10 minutes after stopping, whereas the T wave was virtually the same as control by this time in the unaffected members. Exercise tolerance was normal in everyone. Duration of the dive reflex varied, but was associated with marked bradycardia in the majority and abnormal repolarization in the affected. Valsalva and handgrip rarely produced such changes. There was no T-wave alternans with any of these maneuvers.

After these control studies, the three most symptomatic patients (ages 10, 11 and 11 years) were treated with $\beta$ blockers. This led to fatigue and loss of concentration and, despite switching to cardioselective forms, symptoms persisted and the medication was stopped. Because of the family histories of similar stress-related syncope or sudden death in

Problems of QT measurement.
A Rozkovec, E Rowlands, D Singh and C M Oakley

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