of fasting reported by Dr. Gropler. However, evidence of heterogeneity in normal subjects following longer, yet still physiologic, fasting periods is still lacking. Thus, at the present time there is no evidence for rejecting the use of protocols based upon overnight fasting when metabolic conditions appear to be more stable.

Finally, based upon the above considerations and the results of Dr. Gropler's studies, it is evident that at the present status of knowledge the FDG method can be properly used only in subjects with normal carbohydrate metabolism.

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REPLY: We appreciate the thoughtful comments of Dr. Fragasso and his colleagues with regard to our study recently published in the *Journal* (*1*). We concur with many of the points made by the authors. However, certain comments are in order.

1. We agree that various levels of fasting (with subsequent changes in the pattern of myocardial substrate utilization) can occur and can influence the myocardial uptake of ^{18}F-fluorodeoxyglucose (FDG). The aim of our study was to demonstrate that regional myocardial FDG accumulation differed in the fasted and fed state, but we obviously could not study all possible levels of substrate availability. Our observations have recently been corroborated by others, where similar patterns of heterogenous myocardial accumulation of FDG were observed in normal humans subjected to a more prolonged fast (i.e., >12 hr) (*2*).
2. Occult myocardial disease or regional differences in myocardial contractility certainly would be potentially confounding variables. However, as outlined in our paper, we felt these were unlikely explanations for the differences we observed in regional FDG accumulation. All the subjects studied were under 30 yr of age and healthy, and thus, had a very low likelihood of having occult myocardial disease. Because the regional differences in myocardial accumulation of FDG were sensitive to the pattern of substrates presented to the heart, it would have to be postulated that regional contractile performance is sensitive to levels of serum substrates as well. There is no evidence in the literature to support this hypothesis. Finally, our observations that myocardial oxidative metabolism (based on the measurement of ^{11}C-acetate tissue kinetics) was regionally homogeneous speak against the presence of regional differences in myocardial contractile performance. Although, Fragasso and colleague's observation of mild mitral regurgitation with slight prolapse of the posterior leaflet in three of their subjects demonstrating relatively increased myocardial uptake of FDG localized to the free wall is interesting, without the echocardiographic evaluation of all of their subjects (those with and without localized myocardial accumulation of FDG), the significance of these findings cannot be ascertained.

Finally, as our understanding of myocardial carbohydrate metabolism, under both normal and pathologic conditions, continues to evolve, so will our understanding of myocardial FDG kinetics. We share the concern expressed by Fragasso et al. regarding the accurate interpretation of patterns of myocardial accumulation of FDG in relation to cardiac pathophysiology. However, we believe unique insights into myocardial metabolism can be obtained with this tracer, provided that the relationship of myocardial uptake of FDG to substrate availability is appreciated and that such studies are performed under conditions where the substrate environment is controlled.

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ART: Ambiguous Radioimaging Terminology

TO THE EDITOR: I am awed by your vanity in proposing a new acronym for the already historic term PET, which was coined by a prominent PET pioneer many years ago. I also disagree that the term PET is any more misleading than the term you suggest—ART (*1*). It is true that positrons are not imaged during PET. However your substitution of annihilation radiation is ill conceived because of its ambiguity. You see there are many types of