Letters to the Editor

Measurement of Myocardial Fatty Acid Metabolism by Iodine-123 Heptodecanoic Acid Kinetics

TO THE EDITOR: In their article "Measurement of Myocardial Fatty Acid Metabolism: Kinetics of Iodine-123 Heptadecanoic Acid in Normal Dog Hearts," published in *J Nucl Med* 1986; 28:1449–1455) Schön et al. aim at characterizing the kinetics of omega-^{123}I heptadecanoic acid (IHA) in normal dog myocardium and at relating these kinetics directly to the myocardial oxygen consumption as an index of myocardial oxidative metabolism under different levels of cardiac workload. The paper shows that the kinetics of ^{123}I following the bolus injection of IHA into the coronary artery of open chest dog hearts are different from those of carbon-11 (^{11}C) following injection of the physiologic palmitic acid labeled with ^{11}C, as published elsewhere (*1*). This result is expected and agrees with many observations; it may, however, not be used as an argument against measuring myocardial metabolism with IHA.

Indeed, it has been repeatedly emphasized that it is necessary to correct the gross ^{123}I counts that are obtained after IHA injection from the myocardial region of interest by external measurements, for the contribution from the labeled water soluble catabolites, i.e., mainly inorganic ^{123}I (*2,4*). Also in the studies by Schön et al. the catabolically produced ^{123}I enters the relatively large iodine space in the myocardium and is not as readily removed from the myocardium as is CO_2 that is bound to erythrocytes directly after it enters the capillary bed. The larger the myocardial mass, the lower the heart rate, the more extensive the microcirculation and the surface area-permeability product for iodine exchange into tissue. On the other hand, the transit of water soluble ^{123}I from the mitochondria to the blood circulation in the myocardium is relatively rapid; its value was measured ~2 min, and it was less than 1 minute for CO_2 (*5*). Comparable data were obtained in mice (*6,7*); because of principle similarities in histology and function in mammalian myocardium, this transit time should be similar in different mammals (*7*).

Throughout their work Schön et al. fail to consider the difference between the kinetics of IHA and of water soluble ^{123}I; in fact, this lack of consideration was the reason for previous failures of measuring myocardial lipid metabolism from gross counts of ^{123}I after i.v. injection of omega-^{123}I-hexadecanoic acid that in general behaves similarly to IHA (*8*). For solving this problem, the dual tracer analysis was introduced in order to subtract from the gross ^{123}I counts the signals that originate in the decay of water soluble ^{123}I in the myocardium (*4*). After proper separation of the kinetics of water soluble ^{123}I from the gross counts of ^{123}I, the resulting time-activity curve relates to IHA and its lipid conjugates (*5*); also in man the corrected IHA curve is practically the same as that obtained with [^{11}C]palmitic acid (*9*) and both respond comparably to metabolic interventions (*10,11,12*).

Thus, the measurements by Schön et al. do not permit any conclusion relating to the turnover of myocardial lipids, so that the claim of the authors that IHA cannot be used for measuring myocardial metabolism from the half-time or clearance rates of tracer is in no way supported by their data.

References

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REPLY: In response to the letter of Dr. Feinendegen regarding our article, it is well appreciated that the gross iodine-123 (^{123}I) counts of an externally recorded time-activity curve have to be corrected for the contribution of free ^{123}I, however, only for intravenous administration of [^{123}I]heptadecanoic acid