F-18 Fluorodeoxyglucose (FDG), J Nucl Med 23:P6, 1982

 BARON JC, LEBRUN-GRANDIÉ FH, COLLARD PH, et al: Noninvasive measurement of blood flow, oxygen consumption, and glucose utilization in the same brain regions in man by positron emission tomography: Concise communication. J Nucl Med 23:391-399, 1982

Reply

Dr. Selikson's comments are appropriate in stressing the wellknown fact that the FDG rate constants may change unpredictably, not only from patient to patient, but also from one brain region to another within the same patient, particularly in pathologic conditions. The effects of such changes on the calculated G1MR have been the subject of theoretical (1-2) as well as patient (3)studies. The results of these studies demonstrated that large changes in rate constants could result in relatively marked errors on calculated G1MR. In our paper (4), we pointed out that the GIMR values found in stroke patients using the in vivo autoradiographic approach of Phelps et al. (5) (which includes k_4 , the FDG-6P dephosphorylation rate constant) should be interpreted with caution, a fact stressed earlier by Kuhl et al. (6). We also indicated that more accurate G1MR measurement could be obtained if kinetic analysis of the F-18 cerebral accumulation curve is performed, a procedure by which the FDG rate constants can be estimated regionally. We are currently analyzing the data in this way and comparing in the same patients the G1MR values measured by both methods in order to establish further the validity of the autoradiographic approach using adapted (e.g., "ischemic") rate constants. We therefore agree with Dr. Selikson's suggestion that more work is needed if reliable measurement of regional G1MR in pathologic conditions is desired.

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J. C. BARON Service Hospital D'Orsay 91400 Orsay, France

Reply

No one can argue with the fact that deviations in the dynamic rate constants for the transport of deoxyglucose will affect values of glucose metabolic rate (GMR) as determined by the Sokoloff method. However, the situation is not as black as Dr. Selikson indicates. For one thing, he failed to point out that the simplified formula presented in his Ref. 4 reduces the error caused by these deviations, and this reduction ranges from 10% to 50% for the two examples cited. Another fact not mentioned is that, by doing patient scanning during the dynamic uptake period, one can completely eliminate the effect of the deviations (1).

It is not correct that the cerebral glioma project at our institute did not include a rigorous monitoring of the rate constants. As mentioned in the article (2), and as reported in more detail at the American Neurological Association meeting (1), we have measured these constants in gliomas by dynamic scanning and found them to be relatively unchanged.

Finally, one should be very cautious about recommending the abandonment of noninvasive clinical measurements because their accuracy is in question. It is often difficult in medicine to measure basic quantities with absolute accuracy. The point is that the quantity measured, even if it includes "contaminating" effects, may be of great clinical value. This has proven to be the case in our glioma study, where any shifts in rate constants and lumped constant (which may be of even more concern) have not prevented this method from becoming the most successful technique for diagnostic grading of these lesions.

> RODNEY A. BROOKS GLOVANNI DI CHIRO NICHOLAS J. PATRONAS National Institutes of Health Bethesda, Maryland

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Re: Indium-111 Tropolone Versus Oxine

We should very much like to support the comments of Dr. Goedeman (1) on the following subjects.

1. The solubility of oxine in aqueous acidic solutions is good, and crystalline oxine may be dissolved directly in concentrated acid at concentrations of 100 mg/ml, thus avoiding the need for ethyl alcohol as a solvent. This is apparently the case for both commercially available European oxinates (2,3).

2. The comments regarding the labeling efficiency with tropolonate (4), subsequently supported by the work of Danpure et al. (5), show that in plasma, cells can be labeled only if concentrated five- to tenfold, and the labeling efficiency is then only 50-70%. In contrast, the indium-binding ligands, oxine, acetylacetone, and tropolone, will all, in the absence of plasma and in physiological cell concentrations, give almost quantitative labeling efficiency in very short labeling times of less than $2 \min(\delta)$. This is the case for both platelets and leucocytes of common laboratory animals and man.

3. It is dangerous to extrapolate platelet labeling data obtained from canine experiments, since canine plasma is less effective than human plasma at inhibiting indium-oxine uptake. This is probably due to a decreased concentration of lipoproteins in the dog (7).

We stress further that in the search for the ideal cell-labeling method, cell function in vivo must be retained at all costs. This can