that filling of the gallbladder is not influenced by the state of contraction of the sphincter.

The activity entered the gallbladder before it reached the distal part of the duct. If the theory was correct, it should be the reverse. Shreiner explains this point by surmising that bile already present in the duct prevents the activity from reaching the region of the sphincter. He does not agree with our position that bile already present in the gallbladder is unable to prevent the activity from rapidly reaching the fundus. Nor does he agree with our observation that, in cholecystectomized patients, bile already present in the duct is unable to prevent the activity from rapidly reaching the sphincter (*2*) because, as he puts it, "in cholecystectomized patients resistance to biliary flow would be expected to be similar throughout the ducts." We found this statement puzzling and speculative. If the resistance really were "similar throughout the . . . ducts," would the activity move at all? Cholescintigraphy enables us to study the flow of activity but provides no information on the forces responsible for that flow.

Dr. Shreiner disregarded our data suggesting that the gallbladder is not quiescent during the interdigestive state. The textbook theory is that filling of the gallbladder occurs passively and that it is due to the gradual pileup of bile against the closed sphincter of Oddi. Motor activity during fasting contradicts the theory.

Finally, Dr. Shreiner considers it "unfortunate that (we) did not provide data on the relative rate of filling of the gallbladder." This data was provided in Figs. 1 and 2 of our paper.

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Re: A Comparison of Two Cerebral Perfusion Tracers, N-Isopropyl I-123 p-Iodoamphetamine and I-123 HIPDM in The Human

Holman, Lee, Hill, et al. (*1*) report that the brain uptake of HIPDM is only 50–60% of the uptake of IMP in comparative studies in patients. This value is not consistent with the quantitative measurements for absolute brain uptake of these two compounds previously reported in the literature. This same group (*2*) reported an uptake of 7.45 ± 0.9% for IMP in eight patients. Kuhl et al. (*3*) measured the uptake of IMP in five patients and reported an average of 5% in brain. As part of a Phase 1 clinical study, we have made a careful evaluation (using conjugate counting) of the brain uptake of HIPDM, and have found an average uptake of 6.7 ± 1.4% in seven patients (*4*). This value has been confirmed by an independent measurement at the University of Michigan (*5*), where a range of 6.7–7.2% was found for the brain uptake of HIPDM. Thus, the quantitative uptake measurements of both compounds indicate either an equal uptake or at most a 10 or 15% difference.

No doubt other laboratories have made absolute quantitative measurements of the uptake of IMP and HIPDM in human brain. Since both agents are under Phase 2 clinical trial, we look forward to early publication of these results in the open literature.

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Reply

We thank Drs. King and Blau for their comments concerning our paper (*1*). We also express our appreciation and thanks to them for their most gracious and constructive help during the course of the study. We, too, were surprised at the degree of difference in brain uptake between IMP and HIPDM. It was partially for that reason that we investigated that particular point as extensively as we did, with both planar and tomographic studies, as well as a comparison of the two tracers in the same subject. Perhaps as the ligand is further purified, reducing the quantity of lipophobic tracer, the brain uptake of HIPDM will approach that of IMP more closely.

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