solid, 150 ml water) ranged from 10–30 min in normal subjects (*3*). The sample rate used by Moore et al. ("10–30 min intervals, depending on meal size") is clearly too prolonged for the calculation of this parameter. Their suggestion that the "apparent early delay" in solid emptying may be caused by failure to correct for tissue attenuation is misleading. In our study, anterior imaging without attenuation correction overestimated the lag period (average 48%) while the use of posterior imaging underestimated this parameter by 55% (*2*).

The lag period for liquids can also be measured during our current technique. Using a frame rate of 1 frame every 30 sec for the first 30 min, we regularly observe a lag period for liquid emptying. This period is of the order of 1–3 min for most subjects, though prolonged lag periods (in excess of 10 min) have been observed in some clinical situations.

The accurate measurement of the lag period is of importance in both clinical (*4*) and physiologic (*3*) studies of gastric emptying. This parameter can be measured easily using a technique which corrects for tissue attenuation and has frequent data sampling (at least 1 frame every 4 min).

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Peter J. Collins
Michael Horowitz
Barry E. Chatterton
Royal Adelaide Hospital
Adelaide, South Australia

REPLY: Apparently there is some misunderstanding as to whether or not we believe a "lag period" exists. We never said "negligible" or "probably artifactual." We did say "We do not conclude that such an early emptying period does or does not exist but rather that an early emptying delay can be artifactually created by not employing appropriate techniques." We, in fact, do encounter in *individual* studies early emptying delay periods (or "lags") but the *grouped* data most closely conforms to a linear emptying pattern. Collins et al. are correct in their observation that a sampling interval of 10 min may be too prolonged to detect a lag occurring within <10 min.

J.G. Moore
P.E. Christian
A.T. Taylor
N.P. Alazraki
Veterans Administration Medical Center
Salt Lake City, Utah

Iodine-131 Metaiodobenzylguanidine

TO THE EDITOR: We would like to comment on the article by Geatti O, et al. on the usefulness of scintigraphy with metaiodobenzylguanidine (MIBG) in the diagnosis and treatment of neuroblastoma (*1*).

The above authors observe that tumors with a higher catecholamine secretion show a better uptake of MIBG. They consider that previous treatment may reduce tracer uptake but does not preclude it.

We have studied 11 neuroblastoma patients in activity and 25 in complete remission. In two cases with normal catecholamines excretion the tracer uptake by the tumor was similar to the nine others with high catecholamine levels. A further five cases have been published of patients having normal catecholamines and positive MIBG (one suprarenal hyperplasia, one paraganglioma and three cases of neuroblastoma) (*2–6*). On the other hand, two cases of pheocromocytoma with high excretion of noradrenaline and its derivatives with negative MIBG have been reported (*7*). These apparently contradictory results could probably be due to a disturbance in the process of synthesis, storage and release or in the recaptation of catecholamines that in normal adrenergic cells are closely linked and synchronized.

The cases of positive MIBG with normal catecholamine excretion could be explained by the disappearance or reduction of the synthesis, storage or release phases while recaptation persists. In the other hand the cases of negative tracer uptake and high levels of catecholamine would preserve the synthesis storage and release mechanism with some disturbance in the recaptation.

As to the influence of the previous treatment on the iodine-131 (^{131}I) MIBG uptake by the tumor, in our experience it is directly correlated with the response to this treatment. When the treatment effectively reduces or erradicates the tumoral volume, the posivity diminishes or even disappears. On the other hand, when the treatment does not produce a lessening of the tumor mass, the radiopharmaceutical uptake does not change. In our opinion this clearly demonstrates the usefulness of [^{131}I]MIBG scintigraphy in the evolutive control of neuroblastoma patients.

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