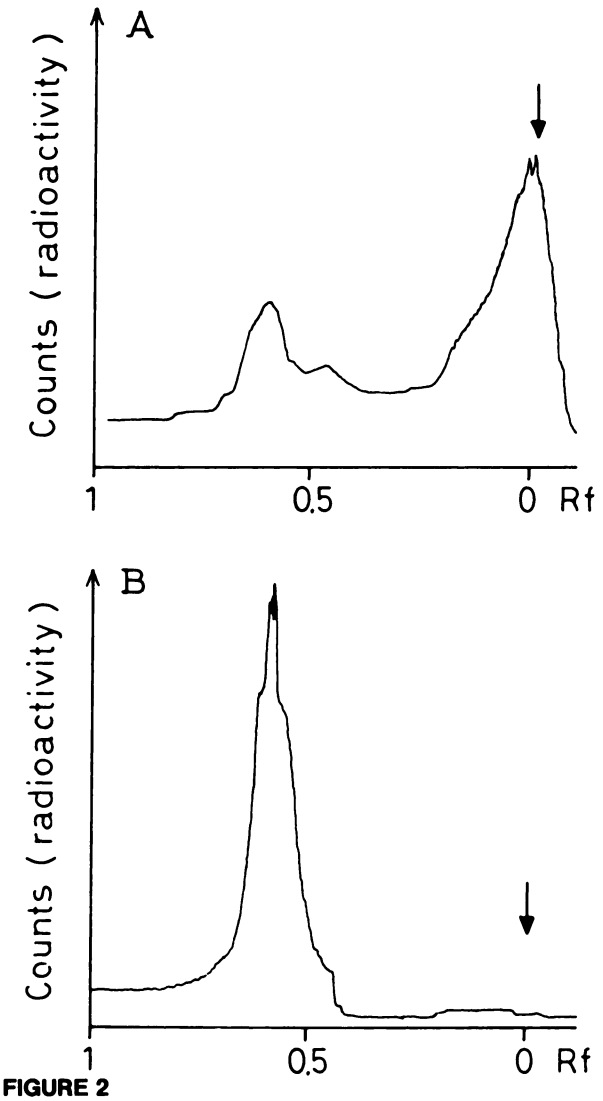

FIGURE 2
Thin layer chromatography of A: [^{99m}Tc]DMSA which remains at origin with, however, one small radioactive peak at an Rf similar to Rf of [^{99m}Tc] 5 DMSA. B: [^{99m}Tc] 5 DMSA which has an Rf between 0.4 and 0.6. Rf of ^{99m}Tc pertechnetate is between 0.7 and 0.9

H_2O 30:20:30) shows that [^{99m}Tc]DMSA (Mallinckrodt Diagnostica) remains at the origin with, however, a few more radioactive peaks, one of them having the same Rf as [^{99m}Tc] 5 DMSA which migrates at an Rf between 0.4 and 0.6 (Fig. 2)

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REPLY: We thank Dr. Jeghers and his colleagues for their suggestion. Although we did not exclude by chromatographic techniques the possibility that there was the pentavalent [^{99m}Tc] (V) DMSA in the trivalent [^{99m}Tc]DMSA we used in our case (*1*), it seems unlikely for the following reasons: (a) The pH needed for [^{99m}Tc] (V) DMSA is much higher (pH 8) than the acid pH used with labeling of the renal imaging agent, (b) the commercial kit preparation of lyophilized DMSA contains a high concentration of tin ($SnCl_2$) unlike the low amounts of $SnCl_2$ needed for the formation of [^{99m}Tc] (V) DMSA, (c) renal uptake in our patient was very high, a finding which is not characteristic of [^{99m}Tc] (V) DMSA as shown by Hata (*2*), (d) our patient showed a rapid soft-tissue clearance of the [^{99m}Tc] DMSA renal imaging agent. Typically the tissue background clearance of [^{99m}Tc] (V) DMSA is slow as evident in the work of Ohta (*3*). Dr. Jegher's image of the [^{99m}Tc] (V) DMSA also shows high background activity.

We agree that a small fraction of [^{99m}Tc] (V) DMSA accounting for tumor localization cannot be excluded; however, other possibilities exist: [^{99m}Tc]DMSA may penetrate into the nuclei and bind to the nucleic acids (*4*). Also, in renal failure or obstruction, nonspecific localization in hypervascular tumors may occur (*5*)—a possibility to be ruled out whenever a high background activity is observed.

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Correction

The following is a reply which was inadvertently omitted to a letter by Caballero et al. appearing in *J Nucl Med* 27:868–869, June 1986.

REPLY: Our initial ten neuroblastoma cases (*1*) demonstrated that the most intense MIBG uptake tended to be associated with tumors which synthesized and secreted catecholamines, but this was by no means an absolute prerequisite for tracer uptake and nonfunctioning tumors can indeed image well as Caballero et al. observed.

It is of interest to note that with pheochromocytomas there is no difference in the biochemical parameters of tumors which do and do not take up iodine-131 metaiodobenzylguanidine ([^{131}I]MIBG), and it is quite reasonable to hypothesize that the metabolic pathways for the synthesis of catecholamines, cytoplasmic granular storage, and catecholamine reuptake may be dissociated from one another in certain tumors. Thus, [^{131}I]MIBG uptake has been demonstrated in nonfunctioning paragangliomas by Smit et al. (*2*) and we have had similar cases. Our observations suggest that the pathways are frequently, but not invariably, linked in neuroblastomas.

The "influence of previous therapy" we believe was misinterpreted by Caballero et al. In patients with tumor unresponsive to preceding radiotherapy or chemotherapy, the possibility exists that the therapy may reduce tracer uptake by the tumor in the absence of tumor shrinkage giving rise to the possibility of false-negative scans. Although the cases with the lowest tracer uptake had all been previously treated, it has been our experience that in general such therapy does not render the MIBG scan useless. We agree that [^{131}I]MIBG scintigraphy, especially when performed serially, is very helpful in following the progress of patients on chemotherapy and radiotherapy. It may on occasion reveal tumor foci in patients who by all other available criteria are in remission. On the other hand it may also be helpful when used along with other investigations in confirming a patient to be disease-free.

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