# **NIM**/ CONCISE COMMUNICATION

### BRAIN SCANNING WITH 99mTcO<sub>4</sub>- IN MULTIPLE SCLEROSIS

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There have been several reports of abnormal brain scans in patients with acute multiple sclerosis (1-4), as well as in other demyelinating diseases (5-7). A recent study reported focal abnormalities in 36% of 14 patients with acute multiple sclerosis scanned with <sup>203</sup>Hg-chlormerodrin or with <sup>99m</sup>TcO<sub>4</sub><sup>-</sup> (4). We have reviewed our experience using more specific criteria for the diagnosis of multiple sclerosis.

Nineteen patients studied between 1968 and 1972 met the following criteria for the diagnosis of multiple sclerosis (8): (A) objective neurological findings; (B) evidence of involvement of two or more parts of the central nervous system not attributable to a single lesion; (C) involvement predominantly of white matter, i.e., fiber tract damage; (D) two or more exacerbations lasting more than 24 hr separated by at least 1 month, or stepwise progression of disease over at least 6 months; (E) onset of disease between ages 10 and 45; and (F) signs and symptoms not explainable by another disease process.

Most of the brain scans were performed beginning 1 hr after the intravenous injection of 15 mCi of Na<sup>99m</sup>TcO<sub>4</sub> and after pretreatment with 400 mg potassium perchlorate orally to block choroid plexus activity. An Ohio-Nuclear dual 5-in. scanner equipped with  $3\frac{1}{2}$ -in. focusing collimators (resolution—FWHM,  $\frac{3}{8}$  in.) was used in all but two of the patients who had scintillation camera images. Contrast enhancement was employed for the rectilinear scans.

Fourteen of the nineteen patients with definite multiple sclerosis had brain scans during an acute exacerbation of their disease. The remaining five patients had brain scans during a quiescent phase. All degrees of severity of disease were found in this study, but the majority of the patients had moderate to severe disability and had multiple sclerosis for at least several years. Nine patients were felt to have cerebral plaques on the basis of the presence of seizures, behavior changes (inappropriate euphoria), or quadrantanopsia.

Brain scans in all 19 patients were found to be normal.

The failure to detect cerebral plaques by brain scan cannot be attributed to their infrequent occurrence. In most series, up to 34% of patients with multiple sclerosis have signs and symptoms attributable to lesions in the cerebral hemispheres (9). In our series, 47% of the patients had cerebral lesions on clinical grounds. However, autopsy series report that more than 90% of the cases of multiple sclerosis have plaques in the white matter of the cerebral hemispheres and that the majority of the plaques in the average patient are found in the cerebral hemispheres (10,11). Most of the plaques are situated deep in the white matter, particularly in the periventricular regions and are 1-10 mm in diam (10). Their small size may render them undetectable with current techniques. This is in contrast to the large areas of demyelination and marked inflammatory response seen in Schilder's disease (12) in which an abnormal brain scan has been reported (7).

In summary, normal brain scans were found in 19 patients who satisfied strict criteria for the diagnosis of multiple sclerosis. Fourteen of the patients were studied during an acute exacerbation of the disease, and 9 of the 19 had evidence of cerebral cortical involvement. We conclude that an abnormal

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pertechnetate brain scan is unlikely to be obtained in patients with clinically certain multiple sclerosis.

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