Reply from the Authors: We were pleased to read the letter by Reches et al demonstrating that L-dopa-fed mice (similar to those we used) have elevated brain levels of 3-0-methyldopa, DOPAC, and HVA. However, our hypothesis does not necessarily require the decarboxylation of exogenous L-dopa within dopaminergic neurons. Any other brain structure might transform L-dopa to toxic compounds which then might act upon the dopaminergic neurons. This concept requires only that dietary L-dopa should reach the brain. That L-dopa indeed has penetrated into the brains of mice fed L-dopa-containing diet is indicated by the elevations of 3-0-methyldopa, DOPAC, and HVA reported by Reches et al. Additionally, elevation of striatal concentrations of DOPAC and HVA after administration of exogenous L-dopa without an inhibitor of peripheral L-aromatic amino acid decarboxylase does not indicate that L-dopa has been decarboxylated within dopaminergic neurons. We have shown that striatal formation of DOPAC and HVA after administration of L-dopa alone is not reduced by total lesions of the dopaminergic nigrostriatal neurons.[1] These findings suggest that DOPAC and HVA are formed mainly outside of dopaminergic neurons under these circumstances.

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CT after metrizamide myelography

To the Editor: Complications of intrathecal injection of metrizamide for myelographic examinations include seizures and encephalopathy.[1,2] We now report CT findings in the brain after metrizamide myelography.

A 12-year-old boy with acute flaccid paraplegia had a lumbar-thoracic myelogram to exclude a compressive lesion. Ten milliliters of metrizamide (200 mgI/ml) was injected into the lumbar subarachnoid space; the myelogram was normal, and the patient was positioned with his head elevated at least thirty degrees for the next 12 hours. There were no adverse effects except for mild headache. Twenty hours after completion of the myelogram a non-enhanced CT (figure) revealed diffuse hypodensity of the white matter, a pattern resembling that of diffuse leukoencephalopathy. However, the density of the white matter was 31 Hounsfield units and the gray matter density was 62 units. By comparison, a normal CT gave a white matter density of 31 units, and a gray matter density of 48 units. Thus, the apparent white matter hypodensity was, in fact, an optical illusion because of diffuse gray matter enhancement. CT 72 hours later was normal. After ultrathecal lumbar injection, metrizamide may pass over the convexities[4] and enter the cerebral cortex; it may have an epileptogenic effect.[3] After 6 to 12 hours, the superficial brain substance adjacent to the subarachnoid space shows enhancement within 24 hours. In patients with normal CSF circulation this blush is decreased, and at 48 hours it is gone.[4] Penetration of metrizamide has been observed on the pial surface in normal patients, and in those with communicating hydrocephalus it has also been observed on the ependymal surfaces.[4,5] Therefore transcortical and transpial movement of metrizamide may occur, and would correlate with the CT change. Diffuse gray matter CT enhancement of the cerebral cortex is to be expected within 24 hours after intrathecal instillation of metrizamide. If CT scan is indicated, it should ideally be delayed until 48 hours after metrizamide myelography, when the cerebral gray matter blush is no longer evident.

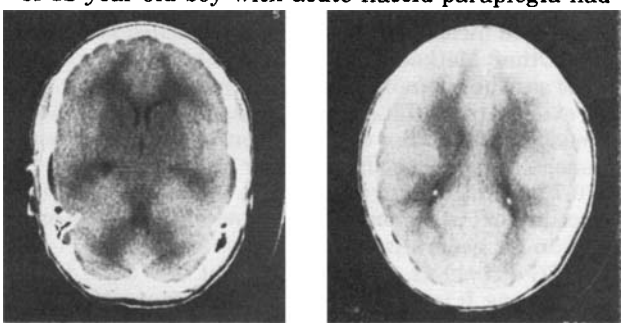

Figure. Nonenhanced CT 20 hours after metrizamide myelography. Diffuse gray matter enhancement is present (Hounsfield unit 62 within the small square on the right), which leads to the illusion of diffuse white matter hypodensity (white matter is of normal density—Hounsfield unit 31).

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5. Winkler SS, Sackett JF: Explanation of metrizamide brain penetration: a review. J Comput Assist Tomog 1980;4:191-3.

Correction

"Localizing and prognostic value of auditory evoked responses in coma after closed head injury" by Dean S. Karnaze, Lawrence F. Marshall, Carol S. McCarthy, Melville R. Klauber, and Reginald G. Bickford, March 1982, p. 301. In paragraph 2, line 4 should read "in 100%, and absent AER and abnormal BAER implied poor quality of survival or death in 80%."

Correction

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