idol and pyridoxine medication was started, but showed no advantages as compared with haloperidol alone.

In this limited trial, no evidence was obtained for the possible primary or adjuvant secondary role of pyridoxine acting together with isoniazid to corroborate the results quoted in Dr. Perry's report. However, in order to eliminate this possibility entirely, it might perhaps be desirable to test the hypothesis in those few of his patients who were apparently benefited by the combined regimen.

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Reference

1. Perry TL, Wright JM, Hansen S, MacLeod PM. Isoniazid therapy of Huntington disease. Neurology (Ny) 1979;29:370-5.

Reply from the Author: Dr. Braham is correct in assuming that my colleagues and I gave pyridoxine 100 mg daily to our Huntington disease (HD) patients who were treated with isoniazid to prevent toxic peripheral neuropathy. We have only once tried pyridoxine alone in very large doses. Patient 4 in our report,[1] which he cites, was given 1000 mg of pyridoxine daily for 6 weeks without any clinical improvement. But this patient also failed to improve later with combined isoniazid and pyridoxine. In a subsequent double-blind, placebo-controlled, crossover design trial of isoniazid on nine further patients (to be reported), none showed any clinical improvement in a 4-month period on placebo and pyridoxine 100 mg daily.

If the clinical improvement which occurs in a minority of HD patients given high-dosage isoniazid therapy were due to a pyridoxine-induced decrease in striatal dopamine release, there should be similar improvement in HD patients treated with dopamine-receptor blocking drugs such as haloperidol or the phenothiazines. Although these drugs sometimes decrease choreiform movements, my experience is that they usually worsen, rather than improve the mental status of HD patients, and in this way they differ markedly from the occasional beneficial effect seen with isoniazid therapy. I certainly agree with Dr. Braham that there would be little to lose in testing his hypothesis, preferably by givin pyridoxine and placebo in a double-blind crossover trial to a reasonably large number of HD patients.

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Reference

1. Perry TL, Wright JM, Hansen S, MacLeod PM. Isoniazid therapy of Huntington disease. Neurology (Ny) 1979;29:370-5.

Correction

Diagnosis and Treatment of Amyotrophic Lateral Sclerosis, reviewed in the September issue, p. 1207, can now be obtained from John Wiley & Sons, Inc.

"Fatal infantile glycogen storage disease: Deficiency of phosphofructokinase and phosphorylase *b* kinase" by Moris J. Danon, Stirling Carpenter, Jose R. Manaligod, and Louis H. Schliselfeld, October 1981, p. 1306, in the table, line 11, following Acid-α-glucosidase, should read nmoles MU-glucoside. On the same line, the control value should be changed to 1.82 ± 0.24.

Diagnosis and Treatment of Amyotrophic Lateral Sclerosis

Neurology 1981;31;1581-1581-a
DOI 10.1212/WNL.31.12.1581-a