phine, a number of centers in Europe and elsewhere are testing the efficacy of subcutaneous apomorphine as "on-demand" treatment of off periods. Clinical studies in these patients could easily address the issue of whether apomorphine is more effective in treating tremor than is levodopa.

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Liver in Creutzfeldt-Jakob disease

To the Editor: We read the article by Tanaka et al¹ on hepatic dysfunction in Creutzfeldt-Jakob disease (CJD) and are impressed with their clinical insight. Although this is a very good paper, we would like to comment on our own experience with hepatic function in CJD. We have had nine cases with CJD in our department in more than 20 years. All were confirmed by autopsy. In all our cases, GOT, GPT, LDH, ALP, r-GTP, and LAP were all within normal range. There were no specific liver findings at autopsy. We would like to know the following about the patients of Tanaka et al: did they have other examinations, such as ultrasound, CT, scintigraphic study, and so on? Were there any specific autopsy findings in the liver? In their paper, elevation of enzymes was not correlated with CNS progression.

Was liver dysfunction transient, or did abnormalities persist through the end stage? Were there any cases in which liver function tests were done prior to disease onset in identical cases? Liver function in CJD deserves more attention, and we agree that further studies are needed to clarify liver dysfunction in CJD. It would be of interest to test liver function in another prion disorder, Gerstmann-Sträussler-Scheinker disease.

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Reply from the Author: Our paper¹ confirmed the previous findings of Dr. Roos et al² of abnormal liver function in patients with CJD. We do not know why the CJD patients reported by Drs. Iwasaki and Kinoshita did not show abnormal liver function. One of our patients, who is still alive, had a mild fatty liver on gross inspection at gastrostomy, and the other six patients showed fatty liver at autopsy. Hepatic dysfunction was not confirmed to be the result of fatty liver because liver damage caused by medication or nutritional change might appear during a later stage in these patients. What is important is that mild but definite hepatic dysfunction is present during the early stage of CJD. Further investigation of this early stage is needed. The accumulation of abnormal prion protein in splenic follicular dendritic cells of CJD-infected mice before involvement of the CNS was suggested to be necessary for the development of the disease in the brain.³ We have suggested elsewhere that hepatic dysfunction in CJD is related to abnormal prion protein in follicular dendritic cells of the spleen.⁴

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Correction

In the December 1992 "Message from the Editor's office" (*Neurology* 1992;42:2237-2240), the name of A. Arturo Leis, M.D., was inadvertently left off the list of ad hoc reviewers. The editors apologize for the error.

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Correction

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