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Spinal Cord Changes in Subacute Combined Degeneration Following Liver Therapy. (A Histopathologic Study).

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Beneficial effects of liver therapy in pernicious anemia complicated by neurologic signs of subacute combined degeneration were reported by Minot and Murphy,[1] Richardson,[2] Ungley and Suzman,[3] and others. A histopathologic study of the spinal cords in

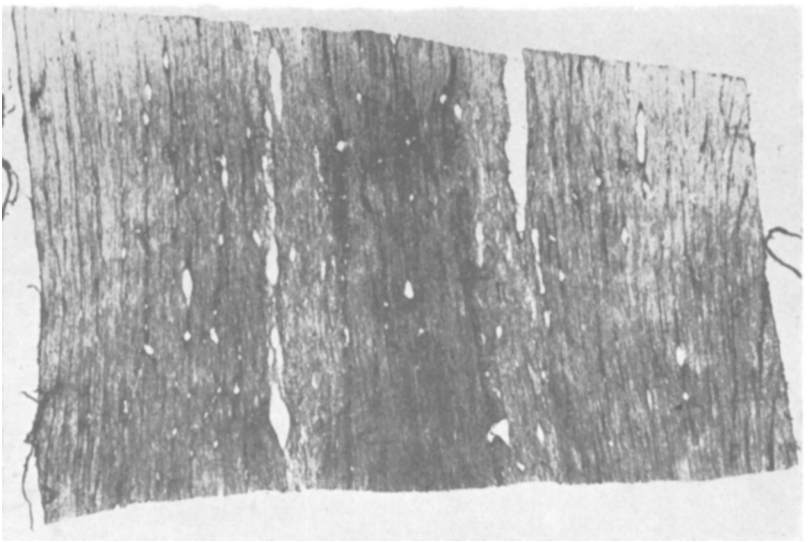

FIG. 1 (a).
Longitudinal section from a normal spinal cord through the posterior and lateral tracts showing the normal glia structure. Victoria blue stain. × 30.

[1] Minot, G. R., and Murphy, W. P., *J. Am. Med. Assn.*, 1926, **87**, 470; 1927, **89**, 759.

[2] Richardson, W., *New Eng. J. Med.*, 1929, **200**, 540.

[3] Ungley, C. C., and Suzman, M. M., *Brain*, 1929, **52**, 271.

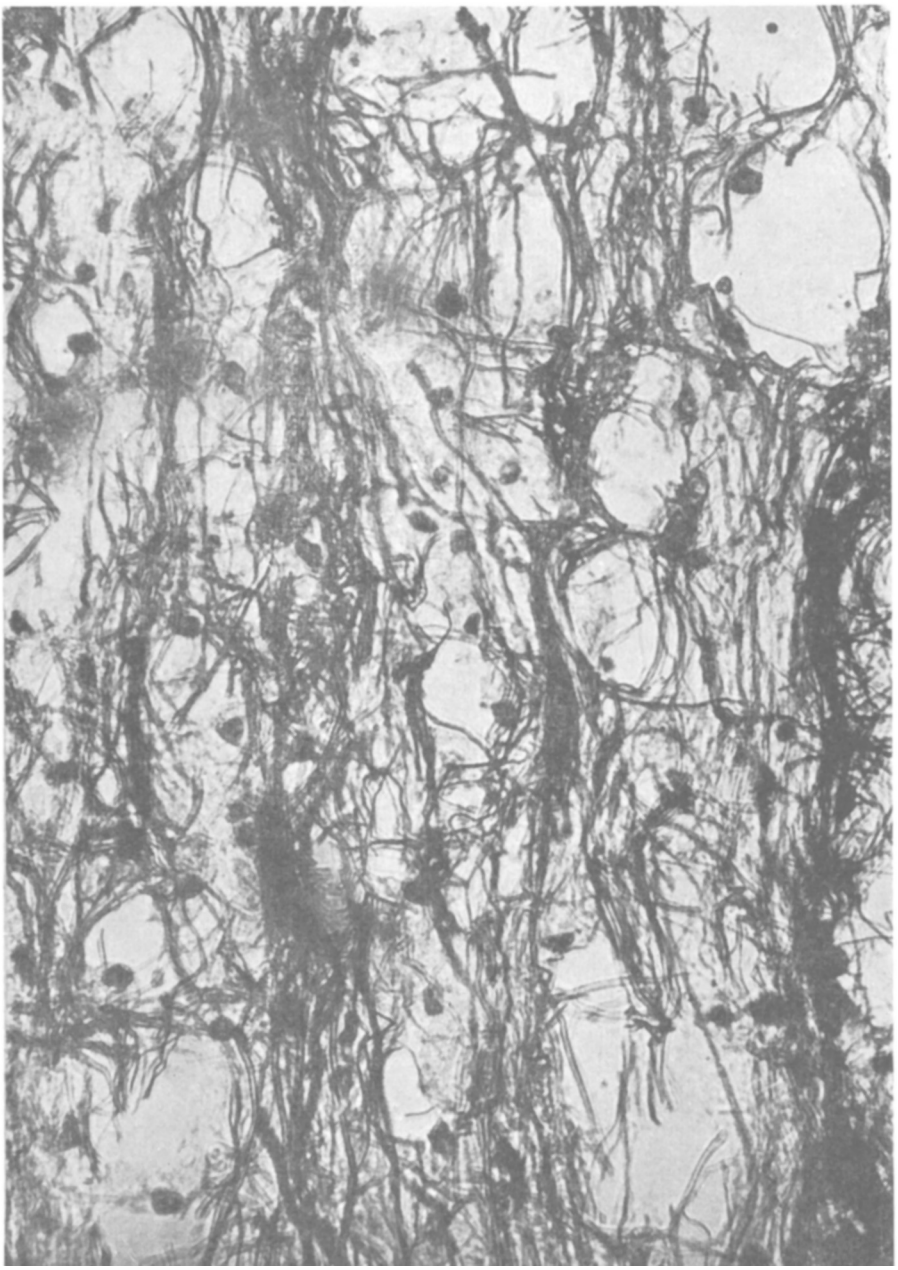

FIG. 1 (b). Same as Fig 1 (a). × 480.

treated and improved cases of subacute combined degeneration has never been reported. The present study was limited to 7 cases of subacute combined degeneration (due to pernicious anemia) which received liver; 2 of the cases showed some improvement in the neurologic symptoms.

Transverse and longitudinal sections of the spinal cord of these cases were stained for myelin sheaths, axis cylinders and glia, and compared with sections from 10 untreated cases of subacute combined degeneration. The myelin sheaths and axis cylinders in the treated and untreated cases showed the same changes. The only histopathologic difference observed was that of the glia. In the untreated cases of subacute combined degeneration the glia destruction ran parallel with that of the myelin sheaths and axis cylinders. The poor glia response in subacute combined degeneration is designated by neurohistopathologists as "a regressive glia change". (Figs. 2a and 2b.) In the treated cases instead of a poor glia response, there was a definite increase in the glia fibers (Figs. 3a and 3b) which is designated as "progressive glia change".

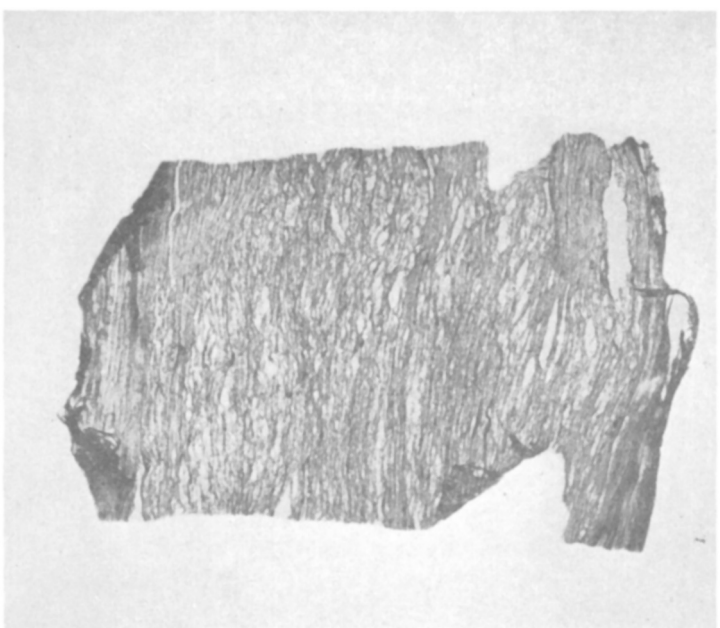

FIG. 2 (a).

Longitudinal section of the spinal cord through the posterior and lateral tracts from a case of pernicious anemia, not treated with liver, complicated by neurologic signs and symptoms of subacute combined degeneration. Notice the poor glia response throughout the section and compare with Fig. 1 (a). Victoria blue stain. × 30.

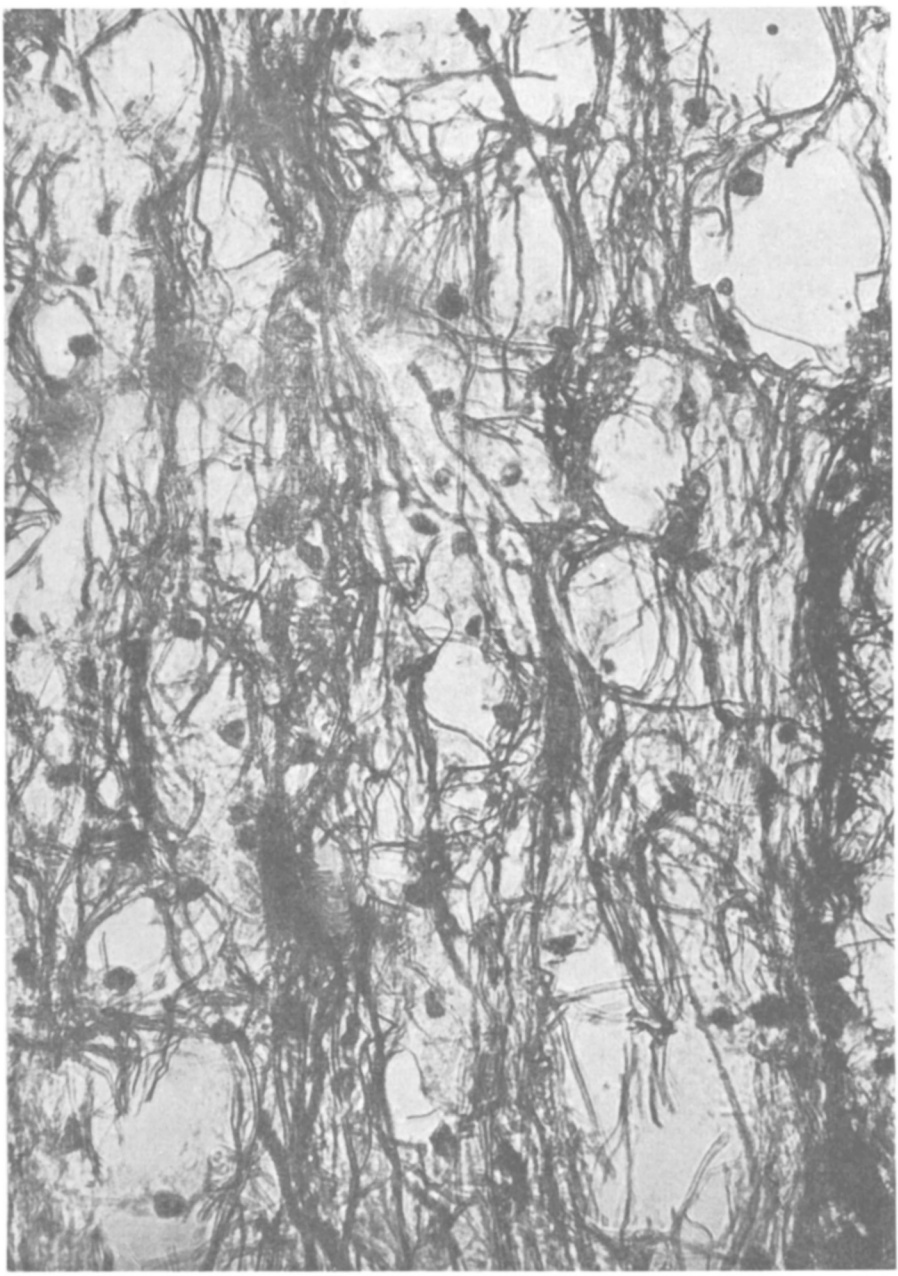

FIG. 2 (b). Same as Fig. 2 (a). × 480.

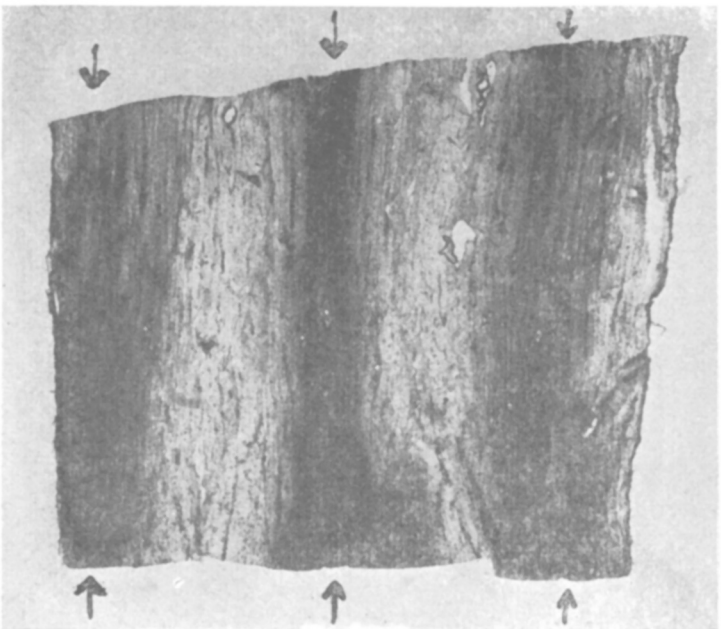

FIG. 3 (a).
Longitudinal section of the spinal cord through the posterior and lateral tracts from a case of pernicious anemia, complicated by subacute combined degeneration, which received liver therapy. Notice the intense glia proliferation (designated by arrows) in the crossed pyramidal tracts and posterior columns and compare with Figs. 1 (a) and 2 (a). Victoria blue stain. × 30.

From the study of these cases the intensity of the glia proliferation did not appear to depend upon the early administration nor upon the duration of treatment with liver. It may be assumed that in these cases the liver therapy either caused a reduction in the hypothetical toxin (pernicious anemia) or in its attenuation, and therefore allowed the glia to proliferate and replace the destroyed tissue. The only possible effect that liver therapy may have on the myelin sheaths and axis cylinders is to arrest the further destruction of these structures. In the light of our present day knowledge of neuro-histopathology, regeneration of destroyed axis cylinders is inconceivable. This finding, however, should not discourage us in the early administration of liver in cases of pernicious anemia with or without neurologic complications. To succeed in delaying the progress of destruction of the axis cylinders and in causing the formation of a glia scar is in itself an advantage.

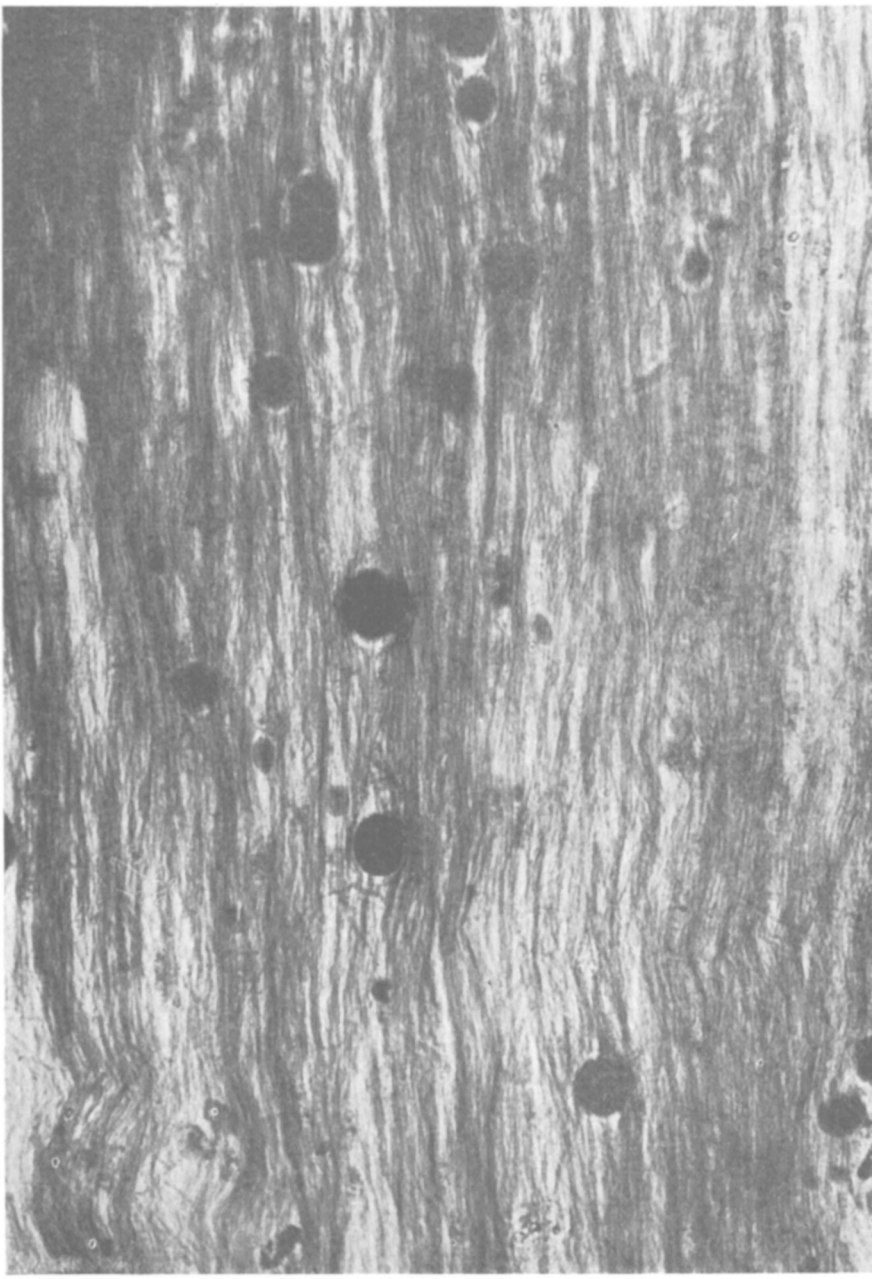

FIG. 3 (b). Same as Fig. 3 (a). × 480.