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BRIEF COMMUNICATION

THE ULTRASTRUCTURAL CHANGES OF THE HEPATIC MICROCIRCULATION AND THEIR POTENTIAL SIGNIFICANCE IN EXPERIMENTAL *SCHISTOSOMIASIS JAPONICA*

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This paper describes the ultrastructural changes of the hepatic microcirculation in *Schistosomiasis japonica*, which is an important parasitic disease in China. In 8 male rabbits (the experimental group), the abdominal skin was exposed to schistosoma japonica cercariae (100/kg body weight); 4 other male rabbits without exposure served as controls. Four months later, when extensive hepatic fibrosis had developed in the experimental group, liver tissue was obtained from the right lobe by open biopsy and processed for electron microscopy.

The ultrastructural changes in the hepatic microcirculation included notable swelling of the sinusoidal endothelium resulting in narrowing of the lumen and perisinusoidal space. There also was decreased fenestrae in the endothelium *(Fig. 1)* with bundles of collagen fibrils filling the perisinusoidal space with a decrease or disappearance of microvilli on the hepatocyte sinusoidal surface *(Fig. 2)*. Collagen fibrils were also seen between hepatocytes *(Fig. 3)*. The hepatic sinusoids gradually lost their characteristic structure and became transformed into "ordinary" capillaries with loss of fenestrae.

These ultrastructural changes in the hepatic microcirculation with *Schistosomiasis japonica* are likely responsible for increased intrahepatic resistance to portal blood flow. Portal hypertension, therefore, arises not only from presinusoidal obstruction in schistosomiasis, as classically described, but also from increased microvascular resistance at the sinusoidal level. Moreover, these microvascular changes also represent a mechanical barrier between hepatocytes and circulating blood which may seriously interfere with hepatovascular exchanges. Subsequent metabolic injury of poorly perfused and nourished hepatocytes may aggravate hepatic fibrosis and worsen dysfunction of the liver. These findings suggest that restoration of the hepatic microcirculation may be accompanied by improvement in liver function.

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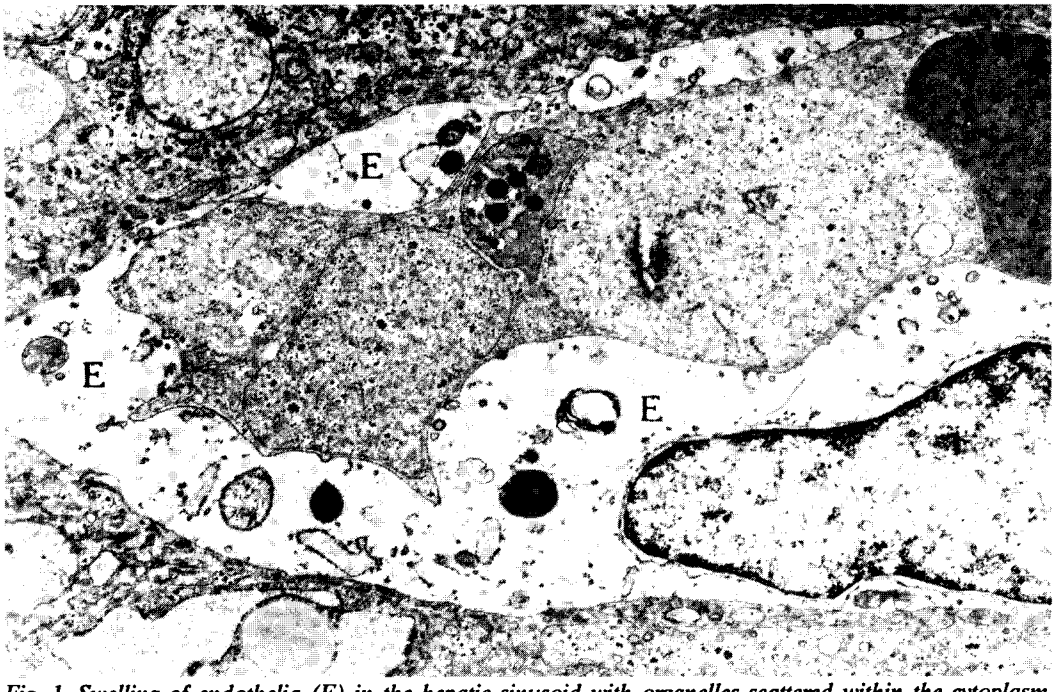

Fig. 1. Swelling of endothelia (E) in the hepatic sinusoid with organelles scattered within the cytoplasm, decreased fenestrae, and endothelial compression of adjacent hepatocytes. x5000.

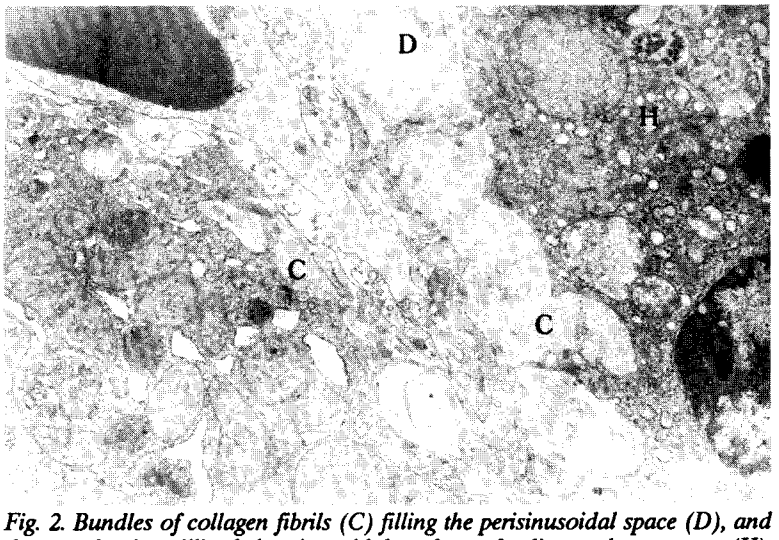

Fig. 2. Bundles of collagen fibrils (C) filling the perisinusoidal space (D), and decreased microvilli of the sinusoidal surface of adjacent hepatocytes (H). x6700.

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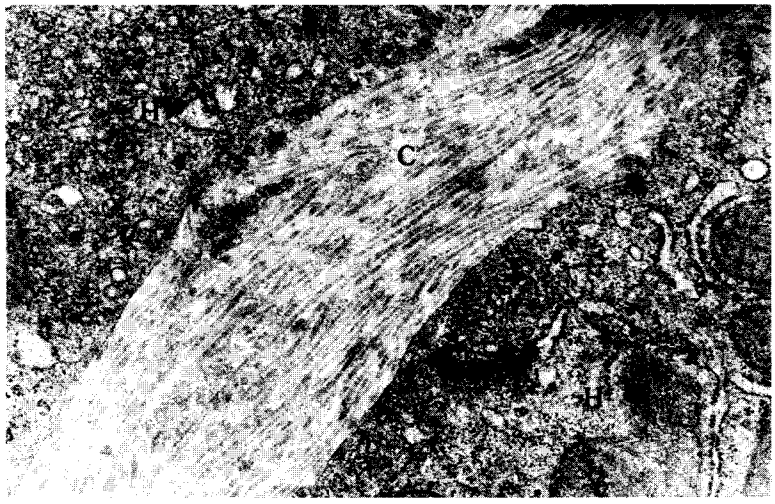

Fig. 3. Dense bundles of collagen fibrils (C) between hepatocytes (H). x14000.

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