There has been considerable necrosis and destruction of hepatic parenchyma, leaving many collapsed, empty areas devoid of liver cells; these areas now consist of condensed reticulum, newly formed ductules, inflammatory cells, dilated capillaries and even fresh hemorrhage. The degree of necrosis varies from place to place, resulting in an irregular nodularity of surviving parenchyma. Special stains indicate that the bands which lie between the irregular nodules of parenchyma consist of condensed reticulum rather than true collagen, suggesting the appearance of postnecrotic collapse which has not yet gone on to postnecrotic scarring. The surviving lobules are disrupted and the liver cords broken up. Many liver cells are binucleated or multinucleated. There are many inflammatory cells in the portal areas. Most of these are mononuclear cells and eosinophilic leukocytes, although some neutrophilic leukocytes are also present. Small numbers of the same inflammatory cells are present in the lobules. There are many small intracanalicular bile plugs in the liver lobules, and larger bile plugs in the bile ducts of the portal tracts. This reflects terminal hepatic failure. A small amount of hemosiderin is seen in Kupffer cells, parenchymal cells and in macrophages within portal tracts; this is probably derived from the blood transfusions the patient received. The yellow stellate crystals scattered through the sections may be hematoidin, and probably are artefactual.

On histologic grounds alone, I would strongly suspect that this is a case of viral hepatitis with marked necrosis and collapse of tissue. When I bring together the clinical and pathologic data, I conclude that this case almost certainly is one of fatal homologous serum hepatitis. The onset of jaundice about 60 days after the first of a series of transfusions strongly suggests homologous serum hepatitis, and the low weight of the liver (860 gm.) and its gross and microscopic appearance indicate a necrotizing inflammatory hepatitis, such as viral hepatitis. The patient survived three months after the onset of the liver disease. This is not at all unusual in viral hepatitis. Moreover, when patients with viral hepatitis die 3 months after the onset of the disease, the gross and histologic appearance of the liver is identical with that of the present case.

I believe it is high unlikely that the hepatic lesion could have been caused by irradiation alone or that irradiation was the major factor in the hepatic lesion. It should be noted that there are many inflammatory cells in the liver, - as many as are usually found in the average case of fatal viral hepatitis. Also note that these cells consist of mononuclear cells as well as eosinophilic and neutrophilic leukocytes. I interpret this to meantthat the reticuloendothelial system was in sufficiently good condition to respond by producing adequate number of cells, which ended up in the liver. If the hepatic lesion were caused by irradiation alone, then that amount of irradiation that would produce such a severe liver necrosis would have wiped out the much more sensitive reticuloendothelial system and we would have seen no inflammatory cells to speak of in the damaged liver.