with Ca-47 and K-42, however, should help clarify many of the essential ration adjustments required for long term periods.

Summary. Radiochemical studies with ruminant animals indicate that activated verxites, a nearly pure hydrobiotite, effectively bind Sr-89 and Cs-137 *in vitro* and *in vivo,* thus increasing excretion rate and quantity of these radio-nuclides injected or ingested at tracer levels.

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Experimental Hepatic Siderosis Following Portacaval Shunt.* (28910)

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Hepatic siderosis associated with human cirrhosis is well known(1,7,9). Recently attention has been called to the rapid accumulation of hepatic iron in a few cirrhotic patients following construction of a portacaval shunt for relief of portal hypertension(4,11, 12). It has been claimed that cirrhosis is a necessary condition, and that hepatic siderosis does not follow creation of a portacaval shunt for extra-hepatic causes of portal hypertension, such as portal vein obstruction(11). To determine experimentally whether shunting of the portal flow is responsible for hepatic iron deposition, end-to-side and side-to-side portacaval shunts were constructed in rats utilizing microsurgical techniques. The animals were then fed normal and increased amounts of iron.

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Materials and methods. The experimental group consisted of 20 male Sprague-Dawley rats, having an average initial weight of 400 g. They were divided into the following groups:

I: Six animals were subjected to end-to-side portacaval shunts and fed Rockland Farms complete rat diet (0.019% iron) plus 3% ferric ammonium citrate.

II: Four animals were subjected to end-to-side portacaval shunts and fed Rockland Farms complete rat diet.

III: Three animals were subjected to side-to-side portacaval shunts and fed the normal diet plus 3% ferric ammonium citrate.

IV: Sham operations were performed on 4 rats, which were then fed the normal diet plus 3% ferric ammonium citrate.

V: Sham operations were performed on 3 rats, which were then fed the normal diet.

Liver biopsies were obtained at 80 days from all animals by laparotomy. The rats were then sacrificed at 130 days. One animal fed iron supplements after end-to-side shunt appeared weak and was sacrificed at 95 days.

At time of sacrifice, the rats were exsanguinated, and the blood collected for hemoglobin determination. The liver was perfused *in situ* with cold saline, after which blocks of liver, spleen, pancreas and heart were fixed in 10% neutral formalin. Paraffin sections were stained with hematoxylin and eosin and subjected to Perl's reaction for the demonstration of iron.

Results. 1. Liver: *Group I* (End-to-side shunt + iron). Biopsy (80 days): Increased amounts of stainable iron in the liver were found in 5 out of 6 rats. In 4 this was in both Kupffer cells and hepatocytes, while in one, only in Kupffer cells. Iron granules were generally more prominent in Kupffer cells than in hepatocytes. Autopsy (130 days): Increased hepatic iron was found in all animals, including the one sacrificed at 94 days. In 4 the degree of iron deposition was increased over that of the biopsy, while in 2 it was the same.

Group II (End-to-side shunt + normal diet). Very small amounts of iron were in scattered Kupffer cells in biopsy specimens of all rats. At autopsy slightly increased iron in Kupffer cells was noted, while traces of iron were in hepatocytes.

Group III (Side-to-side shunt + iron). No animals displayed stainable iron in Kupffer cells or hepatocytes at the time of biopsy. At autopsy one liver showed slightly increased iron in Kupffer cells only.

Group IV (Sham operation + iron). All biopsy specimens exhibited minimal amounts of iron in Kupffer cells and none in hepatocytes. At autopsy one had slightly increased iron in Kupffer cells, while 3 had none.

Group V (Sham operation + normal diet). No stainable iron was found in biopsy or autopsy specimens.

The hepatic architecture was preserved in all groups, with the exception of minimal proliferation of bile ductules and very slight fibrosis in 2 animals from Group I. The liver cells did not appear damaged, except for large amounts of iron in Group I.

2. Spleen: Large amounts of iron were seen in the red pulp in all groups.

Pancreas and heart muscle: No stainable iron was found in any group.

3. Hemoglobin levels in all animals were normal, averaging 13.2 g per 100 cc.

Discussion. Hepatic siderosis involving Kupffer cells and hepatocytes was noted in rats following construction of an end-to-side portacaval shunt. Iron was found in the spleen in sham operated animals and after portacaval shunt. However, the syndrome of hemochromatosis, in which the pancreas and heart are involved, did not develop during the 18 weeks of the experiment.

Many types of parenchymal liver injury may result in deposition of iron in the liver (2,3,5,6), but no evidence of such injury, or of cirrhosis was found in these experiments. One may conclude that, at least in these experimental animals, shunting of the portal flow causes the increased hepatic deposition of iron. After side-to-side portacaval shunt, when the liver continues to receive portal blood, hepatic iron is not increased. This suggests that one of the following 3 factors may be important after end-to-side shunts: 1) Absence of portal blood from the liver; 2) Decreased total hepatic blood flow; and 3) Increased oxygenation of hepatic blood, since all hepatic blood comes from the hepatic artery.

Anoxia damages mitochondria and increases pinocytic activity of liver cells, with transfer of particulate matter from these pinocytic vacuoles to lysosomes(8). It has further been shown that mitochondrial damage and storage of iron in lysosomes occur during induction of dietary hepatic siderosis(10). Thus Factors 1 and 2 might initiate accumulation of iron in the liver by causing prolonged borderline anoxia, with siderosis secondary to

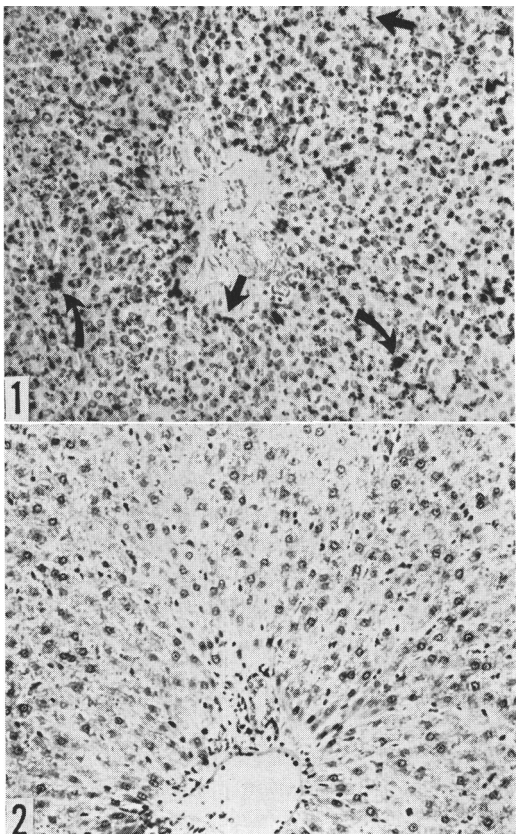

FIG. 1. Section of liver from rat given iron supplement for 130 days after construction of an end-to-side portacaval shunt. Note large amount of iron in Kupffer cells (bent arrow) and hepatocytes (straight arrow). Perl's reaction. Nuclear fast red counterstain (× 90).

FIG. 2. Section of liver from sham operated rat given iron supplement for 130 days, showing absence of stainable iron. Perl's reaction. Nuclear fast red counterstain (× 90).

increased pinocytic activity and mitochondrial damage.

Summary. To determine the role of portacaval shunts in the development of hepatic siderosis, rats were subjected to end-to-side and side-to-side portacaval shunts. Hepatic siderosis developed only in rats with end-to-side shunts, while visible liver cell damage and cirrhosis were absent. It is concluded that shunting of the portal blood flow *per se* results in hepatic siderosis, even in the absence of cirrhosis.

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An Antibody with Alpha-1 Globulin Mobility. (28911)

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Antibodies in man and in most animal species are considered to be generally associated with a group of serum proteins structurally related, known as gamma-globulins (1-3). Their electrophoretic mobility is generally slow and many workers today consider that antibodies with higher mobility reported in the literature(2,3,6,7), are actually gamma-globulins with different mobilities, but belonging to the same antigenetic family (4, 5). There are few data in the literature describing, by indirect methods, antibodies with

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