ples obtained from eight dogs subjected to a lethal dose of radiation from an atomic detonation demonstrated that a material was present which, when administered intravenously, decreased the rate of vasomotion and decreased the epinephrine sensitivity of the mesoappendix capillary bed of the normal rat. After its appearance in the blood, the concentration of this vasodepressor material decreased as a function of time. This VDM has been shown to fit the criteria established for ferritin.

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Portal Venipuncture. A Percutaneous, Trans-Hepatic Approach. (19442)

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Many clinicians and physiologists have wanted a simple method of access to the portal venous system. If direct access were possible, not only could studies be made on portal venous blood, but also the portal venous system could be demonstrated by injecting a contrast medium and making roentgenograms. Hitherto, samples of blood from the portal vein have been obtained during laparotomy and, in a few instances, polythene tubing has been tied into the portal vein and brought out through the wound for sampling at a later time. In a few patients with hepatic vascular obstruction, small catheters have been passed into dilated abdominal veins and portal blood thus obtained. Portal venograms have been obtained by similar methods. These methods have evident limitations and preclude a more general investigation of the portal blood circulation in man. It occurred to one of us (H.L.S.) that access to the portal vein through the intact abdominal wall should be possible, and the senior author (H.R.B.) conceived the idea of an approach through the liver. He subsequently developed the percutaneous, trans-hepatic technic of portal venipuncture described below with the assistance of the two junior authors (L.P.W. and K.H.K.).

The portal vein is 7 to 8 cm in length, extending from the level of L2 to the porta hepatis at the junction of L1 and T12(1). The portal vein divides to the right and left just before it enters the liver(2), the right branch being shorter and thicker than the left. The division of the portal vein usually takes place at the level of T12, in the transverse plane at approximately 15 degrees caudal to the right in the frontal plane(3). In a transverse cross-section(2) which passes through the twelfth thoracic vertebra the larger radicles of the portal vein within the liver substance lie to the right of the midline so that lines to midpoint of the anterior surface make angles of 10 to 45 degrees with the medial plane. At this level the liver abuts directly upon the anterior abdominal wall so that no other organ is interposed. On the basis of these anatomical considerations, a needle was successfully inserted into the hepatic portal vein of a cadaver. This was followed by studies on living patients in some of which contrast media was injected and venograms

were obtained which demonstrated the intrahepatic portion of the hepatic portal vein.

Method. The patient is placed in the semi-recumbent position inclined upwards approximately 10 degrees. The epigastrium is prepared aseptically and a midline point 1 cm below the xiphoid process is anesthetized with procaine, including the deeper skin, subcutaneous tissues and parietal peritoneum. No additional analgesia is achieved by infiltration of the visceral peritoneum of the liver and of the liver parenchyma. A 16- to 20-gauge needle, 20 cm long, is inserted through the skin and subcutaneous tissue at an angle of 20 degrees to the right. If the liver is of average size, the needle is inserted in the transverse plane without upward or downward deflection. If the liver is enlarged, the needle is best inclined 5 to 15 degrees caudally in addition to the lateral angulation. The needle is advanced steadily and slowly in the proper direction until a sensation of firm resistance is encountered, indicating entrance into the liver. The needle is then advanced slowly until blood can be aspirated or the 15 cm mark is reached. Continual aspiration is attempted as the needle is being advanced. When blood is obtained, a hemostat is clamped firmly about the needle at the skin surface to secure its position. Respiratory movements will move the hub of the needle, but they are minimized in the semi-recumbent position. Nevertheless, the patient should be cautioned not to breathe deeply. Portal blood has shown an increased tendency to clot. Portal blood can be identified by its color, since it is neither as red as arterial blood nor as dark as peripheral venous blood. If the needle is aimed 5 to 10 degrees cranially and 10 to 15 degrees to the right, the larger radicles of the hepatic vein may be entered. Blood from the hepatic vein contains less oxygen than that from the portal vein and closely resembles peripheral venous blood.

Samples for glucose and oxygen determinations have been obtained from the portal vein, a peripheral vein, and an artery simultaneously or within 2 minutes of each other. In most instances the patient had been given sugar by mouth ½ to one hour before the sampling, which resulted in a greater elevation of glucose content of the portal blood than that obtained from peripheral sites. This served to confirm that the blood was obtained from the portal system. Fourteen portal venipunctures have been successfully accomplished on 12 patients without immediate or late reaction. The procedure was first attempted on cadavers, then on patients with a short life expectancy because of cancer. Following their demise, examination of the tract of the needle in the liver revealed that it could be followed into a portal radicle without evidence of bleeding or gross trauma.

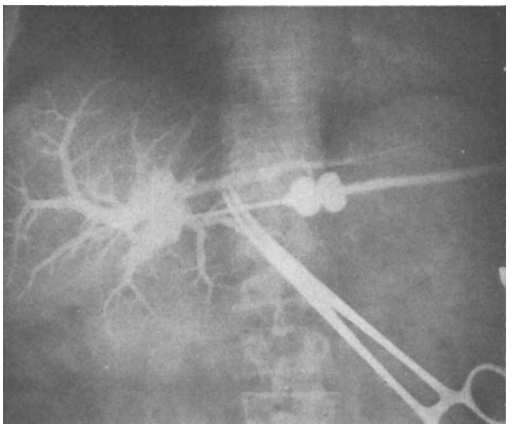

FIG. 1. Portal venogram by direct venipuncture.

In 3 patients who exhibited a hemorrhagic diathesis, a dilute thrombin solution was injected along the tract of the needle after it had been removed from the vein and as it was being withdrawn from the surface of the liver. In 7 others with bleeding tendencies thrombin was not employed and there were no untoward reactions. Following the procedure no special precautions have proved necessary and frequently the patients have walked back to the ward.

In some of the patients who had a portal venipuncture, venograms were performed and were of as good quality as many that had been obtained by injection of a contrast medium during laparotomy (Fig. 1). This will be reported later(4).

Conclusion. This technic permits relatively easy access to the portal venous system in man. It should afford many opportunities

for clinical investigations, including portal venography.

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Tissue Electrolytes in Alloxan-Diabetic Rats with Ketoacidosis.* (19443)

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Metabolic balance studies on diabetic human subjects and experimental animals have afforded abundant evidence that the development of ketonemic acidosis is attended with large urinary losses of water, electrolytes and nitrogen. The nature of the losses suggests that increased cellular catabolism in acidosis leads to a considerable depletion of intracellular materials without breakdown of whole tissues. It may be surmised that functional derangements which occur during severe and prolonged acidosis depend less on changes in constituents of extracellular fluids, much studied and well known, than on changes of intracellular constituents, which have been relatively little studied. The investigations reported here were undertaken to amplify existing knowledge by direct analysis of tissues.

Following is a preliminary report of changes in liver and skeletal muscle of alloxan-diabetic rats in severe ketonemic acidosis. More extensive studies on other animals and other tissues will be reported later.

Methods. Adult white rats from Rockland Farm, weighing around 300 g, fed Purina Chow diet, were kept in glass metabolism cages for collecting urine(1). Three groups, of 36 to 40 rats in each, were studied: a control group, not treated with alloxan, and two alloxan-treated groups. In the latter, diabetes was induced by intravenous injections of alloxan, 50 mg/kg, after a 24- to 30-hour fast. One group was allowed food 24 hours after the injection of alloxan, then starved to induce ketoacidosis. The second alloxan-treated group was fed and treated with NPH insulin, 3 to 5 units per day, until the animals recovered their original weight, usually a period of 7 to 10 days; then ketoacidosis was induced by withholding food and insulin. In both groups severe ketoacidosis developed as a rule within 3 to 4 days, as described in a previous paper from this laboratory(2). All were anesthetized by intraperitoneal injection of nembutal for the taking of blood (drawn from the inferior vena cava) and tissue samples. The whole liver was removed, the gall bladder dissected away, and excess blood removed by blotting with filter paper. The thigh muscles were stripped, dissected free from gross fat and connective tissue. The tissues were transferred as quickly as possible to weighing bottles. Tissue water was determined by weighing and drying the samples at 105°C to constant weight. The dried tissues, liver and thigh muscles, were ground to obtain a homogenous powder. Fat content was determined by extracting the powder with petroleum and ethyl ether in a Soxhlet reflux apparatus. Weighed samples of the fat-free powder were used for determinations of Cl by the method of Van Slyke(3), and of nitrogen by the microkjeldahl method; samples were dry ashed for determinations of sodium and potassium with the Weichselbaum-Varney flame photometer. Samples of blood

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