

the hepatic vein it will almost immediately be seen to pass to the right and downward into the right upper quadrant of the abdomen. The position at this time is quite distinctive, as shown in Fig. 1. Instead of passing into the hepatic vein the catheter may pass into the right ventricle or almost straight downward in the inferior vena cava. In either event it may be returned to a higher level, rotated, and further attempts made to manipulate the tip into the hepatic vein. At all times the manipulations should be gentle, force never being used.

As pointed out by Cournand and Ranges,³ the catheter may not always pass in the desired direction, but on repeated manipulation we have been able to reach the right auricle in over 90% of the cases attempted. The most frequent cause of failure has been spasm of the peripheral vein, which makes rotation and movement of the catheter difficult and painful. A rest period and additional local anesthesia at the site of insertion may relieve this. If the auricle has been reached, and there is little or no spasm, we have been able to enter the hepatic vein in most instances, but failure may occur if the catheter repeatedly passes into the right ventricle or the inferior vena cava and cannot be guided into the hepatic vein.

On removal of the catheter the skin is closed with several silk sutures, but the vein is not tied off. If bleeding occurs, it is controlled

with a pressure bandage. A small linear venous thrombosis may develop locally, but in most instances the vein recanalizes within 2 or 3 weeks.

Although this was not the primary purpose of our work, we have made a few observations on hepatic vein blood. The hematocrit reading and hemoglobin content of blood from the liver did not differ from simultaneously obtained arterial blood specimens. The oxygen content of the hepatic vein blood in 6 patients has been somewhat lower than that of mixed venous blood obtained from the right auricle. In the fasting subject the non-protein nitrogen content was not significantly different from arterial blood, while the glucose level was only slightly lower in hepatic vein blood.

In addition to the above data we have made observations upon the bacterial colony counts in hepatic vein blood in patients with subacute bacterial endocarditis. This work, done in collaboration with Dr. Paul B. Beeson, is to be reported in detail elsewhere.

Summary. A method is described for obtaining samples of blood from the hepatic vein in man by means of a catheter in the venous system. Only a moderate amount of skill is required, and if carried out with care, samples can be obtained with minimal discomfort and danger to the subject.

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Delayed Contraction of Denervated Muscle with Intravenous Barbiturates.*

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It is a known fact that denervated striated muscle contracts when in contact with cholinergic substances that have nicotinic properties. Intravenous injections of minute doses of acetylcholine and certain carbaminocholine derivatives produce a contraction of denervated facial muscles within the circulation

time.¹ Large doses of epinephrine also cause the denervated facial muscle to contract, but

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¹ Bender, M. B., Spirtes, M. A., and Sprinson, D. B., *J. Pharm.*, 1943, **77**, 107.

this does not occur until 60 to 90 seconds after the intravenous injection. Such delayed muscular contraction is probably the result of a secondary, rather than direct effect of epinephrine.²

In the course of these experiments, it was noted that intravenous sodium pentobarbital anesthesia produced a delayed contraction of the denervated facial muscle in 11 of 12 tested monkeys. The contraction appeared after a latency varying from 16 to 60 seconds following the administration of 25 mg of sodium pentobarbital per kilo of body weight. The contraction could be elicited only by the intravenous route with a speed of not less than 10 mg per second. So long as the injection was rapid, the effect could be obtained even with subanesthetic doses.

The contraction induced with sodium pentobarbital lasted from 40 to 75 seconds. It could be so elicited so long as the muscle remained denervated. It was slightly enhanced by eserine and never blocked by atropine.

Similar results, and under the same condi-

tions, were obtained with other barbiturates, such as sodium thiopentobarbital, sodium amytal, and sodium evipal. Intravenous injection of other anesthetics, such as paraldehyde, urethane, or ether inhalation did not produce contraction in the denervated face.

The mechanism of this contraction is difficult to explain. Barbiturates have no significant direct stimulation effect on striated muscle *in vitro*.³ This, and the fact the contraction is delayed, suggest that the effect is indirect and secondary. It is possible that the rapid intravenous administration of barbiturate causes a physiologic disturbance in the body leading to the formation or release of substance causing the denervated muscle to contract. The nature and site of formation of this hypothetical substance are obscure. Because of its contractile effect on denervated muscle, the substance may be related to the cholinergic group.

³ Goodman, L., and Gilman, A., *The Pharmacological Basis of Therapeutics*, Macmillan Co., N.Y., 1941.

² Bender, M. B., *Am. J. Physiol.*, 1939, **126**, 430.

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Genetic and Certain Non-genetic Factors with Reference to Leukemia in the F Strain of Mice.*

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MacDowell and Richter¹ found that the incidence of leukemia in hybrids could be roughly correlated with the total heredity from the high leukemia C58 strain when C58 mice were crossed with low leukemia StoLi animals. A "maternal influence" was suggested, for if the female parent was of the C58 strain the

incidence of leukemia was greater than in the reciprocal cross. Cole and Furth,² working with the Ak and Rf stocks, concluded that susceptibility to leukemia is probably inherited as a multiple factor character, and is influenced by undetermined environmental factors. In various crosses the common logarithm of the percent leukemia was a simple function of the percent heredity from the high leukemia stock. Female mice had a higher incidence of leukemia than males. It was

* This investigation was aided by grants from The Jane Coffin Childs Memorial Fund for Medical Research and the Cancer Fund of the Graduate School of the University of Minnesota.

¹ MacDowell, E. C., and Richter, M. N., *Arch. Path.*, 1935, **20**, 709.

² Cole, R. K., and Furth, J., *Cancer Research*, 1941, **1**, 957.