3049

Effect of adrenalin upon blood sugar following ligation of the hepatic artery.*

WM. S. COLLENS, DAVID H. SHELLING† and CHARLES S. BYRON.

[*From the Department of Pathology and the Harry Caplin Pediatric Research Laboratory, the Jewish Hospital of Brooklyn, N. Y.*]

In a previous communication,[1] we demonstrated that exclusion of the arterial supply to the liver by ligation of the hepatic artery and its collateral branches causes death in hypoglycemic convulsions within 15 to 60 hours, depending upon the amount of glycogen previously stored.

The present experiments deal with the effect of adrenalin upon the blood sugar level following this procedure. Five-tenths to 1.5 cc. of 1/1000 solution of adrenalin was injected intravenously at varying periods following ligation of the hepatic artery in dogs. The following observations were made:

1. The degree of hyperglycemia following adrenalin injection varies inversely with the period of time following ligation of the hepatic artery; *i. e.*, the longer the time permitted to elapse after ligation the less the increase in blood sugar.

2. When the animal develops all the manifestations of hypoglycemia, adrenalin no longer influences the blood sugar level.

3. When adrenalin fails to cause an increase in blood sugar, hypoglycemic convulsions and death may be predicted in 2 to 5 hours.

4. When adrenalin had no effect on blood sugar level, the tissues of these animals, examined at death, showed complete absence of glycogen. This is in agreement with the results of the work of Ringer,[2] who showed that in a phlorhizinized diabetic dog, totally depleted of glycogen by shivering, adrenalin does not alter the D:N ratio, through an elimination of extra sugar.

* With the aid of a grant from the Therapeutic Research Committee of the American Medical Association.

† Harry Caplin Research Fellow.

1 Collens, William S., Shelling, David H., and Byron, Chas. S., PROC. SOC. EXP. BIOL. AND MED., 1926, xxiii, 361.

2 Ringer, A. I., *J. Exp. Med.*, 1910, xii, 105.

5. The blood sugar level does not necessarily indicate the quantity of glycogen stored in the organism. In Dog 20, 1 cc. of adrenalin, intravenously, had no effect upon the blood sugar, although the blood contained 82 mg. sugar per 100 cc. One hour later this animal was in hypoglycemic shock with a blood sugar of 52 mg. Dog No. 15 showed no effect of adrenalin upon the blood sugar, although the blood sugar was 90 mg., and in dog No. 57, 4½ hours before convulsions and death of the animal, the adrenalin had no effect on the blood sugar level at 86 mg. This is important from a clinical standpoint for it would mean that the sugar content of the blood is not an index of the amount of glycogen stored in the tissues.

These observations seem to confirm our previously reported results, that ligation of the hepatic artery causes an abnormally high degree of carbohydrate oxidation with a total depletion of glycogen stores of the body.

3050

A color reaction associated with vitamin D.

M. J. SHEAR. (Introduced by Benjamin Kramer).

[*From the Harry Caplin Pediatric Research Laboratory of the Jewish Hospital of Brooklyn, N. Y.*]

"The chemical relationship between activated cholesterol and the naturally antirachitic substances, such as cod liver oil, yolk of egg, and bone marrow, is one of prime importance in a consideration of the etiology of rickets."[1]

It is definitely established that substances which contain either cholesterol or phytosterol can be made antirachitic by exposure to ultra-violet light. Cholesterol and phytosterol themselves, ordinarily without any curative effect on rickets, can be made antirachitic by irradiation. The criterion for the presence of the antirachitic factor (here called vitamin D) is the "line test" of

[1] Hess, A. F., Weinstock, M., and Sherman, E., *J. Biol. Chem.*, 1926, lxvii, 420.