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4675

Utilization of Fat by Resting and Exercising Muscles of Diabetic Dogs.

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Eight normal, 13 phlorizinized, and 9 depancreatized dogs under amytal anesthesia were studied during rest and exercise. Blood entering and leaving muscle and liver was analyzed for fat, using the method of Stewart and White.[1] The experimental error was ±20 mg. %, and differences of 40 mg. % or more were considered significant. The differences of the arterial and venous fat contents of the blood of the muscles of the normal post-absorptive dogs varied, since the muscles liberated fat 5 times and removed it on 3 occasions. Of greater interest are the observations of the diabetic dogs, since in 10 of 13 significant determinations on the phlorizinized dogs, and 12 or 15 on the depancreatized animals, the muscles of the lower extremities removed fat from the blood passing through them.

The results obtained on the blood of the liver were not the same in the phlorizinized and depancreatized dogs. The livers of the phlorizinized dogs usually removed fat from the blood, while those of the depancreatized dogs added fat.

TABLE I.

Typical results of the fat content of the blood of diabetic dogs.

Date	No.	Femoral Artery	Femoral Vein	Portal Vein	Hepatic Vein	Remarks
7/10/27	1	1345	1198	1310	1379	Depancreatized Exercise
7/10/27	2	1408	1221	1337	1488	Depancreatized Exercise
7/19/27	3	1156	1039	1017	1251	Depancreatized Rest
3/21/27	4	825	654	547	529	Phlorizinized Rest
5/17/27	5	574	404	538	466	Phlorizinized Rest
5/23/27	6	879	789	618	592	Phlorizinized Rest

[1] Stewart, C. P., White, A. C., *Biochem. J.*, 1925, xix, 840.

Thus these results obtained by the method of Stewart and White[1] indicate the utilization of the fat of the blood by the muscles of diabetic dogs.

4676

The So-called Hyperglycemic Action of Insulin.

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Collens and Murlin[1] have recently reported that the portal injection of insulin into dogs, in dosage of 0.05 to 0.1 unit per kilo weight, results in an immediate sharp rise of blood sugar of 20 to 80 mg. The rise occurs within 5 minutes and is then followed by a rapid decline. No such rise occurs following the systemic injection of the same dose of insulin. Bürger and Kramer[2] at about the same time reported that the injection of 10 to 20 units of insulin into the cubital vein of human beings produces a rise of blood sugar averaging 11.5%; intrajugular injection of 40 units into dogs of about 20 kilograms causes a rise averaging 28%; intraportal injection results in a rise averaging 46%. The rises occur within 5 minutes and are followed in 10 to 30 minutes by a rapid fall. In both of these reports, the results are interpreted as showing that insulin has a glycogenolytic action on the liver and that the hyperglycemia is a physiological or normal response to this action. Since such an action of insulin would have a bearing on work we were carrying out, we have gone into the matter in order to have a clearer understanding of its significance.

We have carried out experiments on dogs, corresponding to those described in the reports cited. We employed the Lilly insulin, as did Collens and Murlin, whereas the Burroughs Wellcome product was used by Bürger and Kramer. Using small doses, 0.1 unit per kilo, we obtained no rise in blood sugar on intrajugular injection, but a rise followed intraportal injection. Our maximum rise, however, was 15 mg. as compared to the 20 to 80 mg. rise of Collens and Murlin. With the larger dosage, 2 to 3 units per kilo (40 units total), we obtained a rise on intrajugular injection of 5 to 10%, and

[1] Collens, W. S., and Murlin, J. R., PROC. SOC. EXP. BIOL. AND MED., 1929 xxxvi, 485.

[2] Bürger, M., and Kramer, H., *Ztschr. f. d. ges. exp. Med.*, 1929, lxv, 487.