

the twelfth day. At the time of discharge the patient still complained of slight tingling of the hands.

Conclusions. The above findings are in accord with the work of Castle and Townsend.¹ Clear dog's gastric juice in a total dosage of 12 liters given during a period of 9 days did not influence the reticulocyte or red cell count or give symptomatic relief in this case of pernicious anemia.

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A Study of the Effect of Insulin on Gastric Motility.

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In continuing the study of the mechanism of pain in gastric and duodenal ulcer a method for increasing the gastric motility was desired. Bulatoa and Carlson¹ showed that the injection of insulin into normal fasting dogs increased the motility of the stomach. Quigley, Johnson and Solomon² concluded from their investigations that the same held true for the stomach of normal fasting man. Clinicians have frequently observed hunger as a symptom of the so-called "insulin reaction". Hence it was assumed that the hunger so seen was due to the increased gastric motility, Carlson³ having correlated "hunger pangs" with activity of the stomach.

This report is based on the results of 18 experiments done on 13 patients, 11 of whom had duodenal ulcer, 1 paroxysmal fibrillation, and 1 mild diabetes mellitus. The last 2 had a complete achylia to the histamine test. Kymographic records of gastric activity were made by means of the usual single balloon method connected to a water manometer, the entire system being inflated with 150 cc. of air. In a number of cases the position was checked by fluoroscopic examination. The tracings were all made with the balloon in the pyloric region of the stomach. Fasting periods ranged from 14 to 21 hours; the average time was 19 hours. The patients were instructed to register the appearance of "hunger pangs" by means of closing a switch set in series with a signal magnet.

In most instances control tracings had been obtained during previous experiments of a different nature. With those in which this

¹ Bulatoa, E., and Carlson, A. J., *Am. J. Physiol.*, 1924, lxxix, 107.

² Quigley, J. P., Johnson, V., and Solomon, E. J., *Am. J. Physiol.*, 1929, xc, 89.

³ Carlson, A. J., "Control of Hunger in Health and Disease," Chicago, 1916.

had not been done, a preliminary period was recorded before the administration of insulin.

Insulin (Mulford) was given subcutaneously in doses of 8 to 20 units. The period of observation after its injection varied from 1½ to 4 hours. Manifestations of insulin reaction were noted in all but 3 instances. The first symptom was usually that of a peculiar nervousness which gradually progressed. Upon this there soon developed a feeling of warmth and a gradually increasing desire for food, followed by profuse perspiration. Throughout the experiment the blood glucose level was followed, the determinations being made by the Folin method.

It seems to us important to differentiate the various factors involved in the phenomenon of hunger. Dr. Carlson has emphasized the difference between hunger and appetite. Appetite, of course, may be present with hunger. However, if one dissociates appetite from hunger there remain at least two factors which properly constitute the hunger complex, namely the epigastric "hunger pang", and a general weakness or desire for food. In some individuals the first dominates the picture of hunger, in others the second. In our work hunger, not appetite, but hunger in the sense of extreme desire for food, not associated with epigastric pain, was found to parallel quite closely the intensity of the insulin reaction. This general desire for food accompanied by weakness and nervousness was frequently present without definite gastric activity and, in 2 such instances especially it became so marked that the patients begged to be released. "Hunger pangs" wherever they occurred were correlated with hunger contractions, although there were occasional exceptions in which the pain was apparently due to activity lower down in the bowel.

The stomach in one patient was in a period of activity at the time of injection of insulin, C, which culminated in vigorous type iii contractions. The control tracings had shown exactly the same type of activity. During the subsequent 90 minutes there was practically no gastric motility, but after 105 minutes another hunger period apparently appeared. The blood sugar fell from 68 to 40 mgm. per 100 cc. and the patient experienced an intense desire for food.

In another patient, in the 2½ hours following the injection of insulin there was no gastric activity different from that seen in the control tracings in spite of the fact that the patient developed a marked insulin reaction. The blood sugar fell from 77 to 42 to 43 mgm. per 100 cc.

In another patient a tracing obtained with insulin on a previous occasion shows essentially the same reaction. There is seen a normal hunger contraction period ending in strong type iii contraction. In none of the control tracings for this individual was there such an extended rest period. Insulin was given at B which represents about one-half of the normal rest period. No definite activity was seen for 2 hours. Although the blood sugar was lowered from 63 to 43 to 47, no increased activity occurred. At the end of this experiment the patient was extremely weak, nervous, and had a marked desire for food.

In one instance we noted a definite prolongation of the contraction period. Insulin was given at B during a period of gastric activity. No rest period occurred during the entire experiment. At the end of 2½ hours the patient suddenly developed a severe insulin reaction which necessitated discontinuing the tracing.

Our studies thus far have failed with 2 exceptions to show any very definite or striking stimulation of gastric activity as the result of insulin. These 2 exceptions were cases in which there was increase in the length of the contraction period but not in the intensity of the contractions. Types ii and iii contractions were rather frequently seen without insulin, but in no case were they seen after the injection of insulin, even at the height of the reaction.

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Experimental Infarction of Bone Marrow.

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In the course of a study of the etiology of solitary bone cysts, a series of experiments was performed, the object of which was to infarct large areas of bone marrow and to determine whether or not a cystic degeneration takes place in these areas with formation of lesions similar to the solitary bone cysts of man.

The animals used were young and adult dogs. Under ether anesthesia and careful aseptic precautions, the periosteum over nearly the entire femoral shaft between epiphyseal lines was elevated, thus severing all blood supply and drainage. Small areas of periosteum were left intact near the extremities of the shaft to afford a ready source of blood supply for revascularization of the infarcted areas.

Twenty-four hours following operation no change was noted.