TABLE II.
Recovery of Glucose Urea from a Jejunal Loop of a Dog. 20 cc. of .09 M glucose urea, equivalent to 324.2 mg. of glucose remained in the loop 35 minutes.

Vol. fluid removed	Reducing Substances as glucose*		Glucose urea removed	Glucose urea hydrolyzed in the loop
	Before Hydrolysis	After Hydrolysis		
cc.	mg.	mg.	%	%
23	0	315	97	0

*Corrected for reducing substances removed from the loop during a control experiment.

the loop. At the end of 35 minutes, the contents of the loop were removed and the loop thoroughly washed, and analyzed for reducing sugar, before and after hydrolysis, by the Hanes method.[6] Correction was made for the small quantity of reducing substances that accumulated in the loop when 20 cc. of physiological saline was introduced into the loop for 35 minutes. The results of this experiment (Table II) show an almost quantitative recovery of glucose urea and are in marked contrast to results observed when a solution of glucose[7] or sucrose[8] was inserted into this loop. These sugars were rapidly and in large part absorbed in the course of a half hour. It may be concluded that glucose urea was not absorbed from the dog's jejunum. Further, no glucose urea was hydrolyzed during its exposure to intestinal enzymes.

The presence in blood or other tissues of any appreciable quantity of urea united with glucose, as glucose urea, seems unlikely since all the urea present in blood is decomposed by urease and the presence in blood filtrates of a non-fermentable substance that yields glucose on hydrolysis has not been demonstrated.

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Glycogen Content of the Rat Heart.

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The cardiac glycogen of albino rats has been determined under a variety of conditions. The figures quoted are for glycogen as glu-

[6] Hanes, C. S., *Biochem. J.*, 1929, **23**, 99.

[7] Unpublished results of Ravdin and Johnston.

[8] Unpublished results of the author.

* With the aid of a grant from the Banting Research Foundation.

cose in milligrams percent, and unless otherwise stated are for 24-hour fasted animals. Amytal anesthesia was used when securing hearts and muscles.

Fifty-two 24-hour fasted animals, used as controls, showed a quite constant cardiac glycogen of 497±56 mg. %; the gastrocnemii of the same animals contained 525±48 mg. %.

Animals fasted for 48 hours had more glycogen in the hearts (578±81) and less in the gastrocnemii (455±37) than controls.

Non-fasted animals showed considerably less cardiac glycogen (341±54) but more glycogen in gastrocnemii (574±74) than controls.

It was found also, that 24-hour fasted animals which had been fed sufficient glucose by mouth to provide for maximum absorption for 4 hours, and which were taken at the end of such time, had somewhat less glycogen in the hearts (449±50), although considerably more in the gastrocnemii (690±48) than controls. When, however, insulin injection accompanied such glucose feeding, both hearts and gastrocnemii contained increased glycogen (hearts 703± 212, gastrocnemii 830±183).

Epinephrine sufficient to reduce the glycogen of gastrocnemii to 55% of its control value did not alter cardiac glycogen appreciably. The cardiac glycogen after subcutaneous injection of 0.02 mg. of epinephrine per 100 gm. of rat, was (a) in ½ hour 475±52, (b) in 3 hours 509±54. One-half hour after large intravenous doses of epinephrine the hearts contained 475±26.

Similarly, exercise, both natural and electrically produced, sufficient to reduce the glycogen of gastrocnemii to 70% of its control value did not decrease cardiac glycogen, the values being (a) after natural exercise 546±40, (b) after electrical stimulation 512±19. When, however, electrical stimulation was severe enough to produce cyanosis through interference with respiratory movements the cardiac glycogen was much reduced, being 226±33. This finding is in agreement with those recorded below under anoxaemia.

Marked changes in the $H_2CO_3/NaHCO_3$ ratio of the blood can occur without altering greatly the cardiac glycogen. Thus, after 3 hours in an atmosphere of 8% CO_2, 92% O_2 animals had a glycogen content in hearts of 458±27, and in gastrocnemii of 532±32. Four hours after sufficient $NaHCO_3$ by mouth to produce a plasma CO_2-combining power of from 76 to 88 vol. % the animals had a cardiac glycogen content of 466±31 and in gastrocnemii of 517±46. When, however, larger doses of $NaHCO_3$ were given, producing a CO_2-

combining power of 108 to 123 vol. % and resulting in tetany, the glycogen content of both hearts and gastrocnemii was much reduced (hearts 261, gastrocnemii 178).

Anoxaemia readily lowers cardiac glycogen. Immediately after asphyxia by coal gas, hearts were found to contain only 25 mg. %. The hearts of animals 2 minutes after clamping the trachea contained 83 mg. %. Less acute experiments were done by placing the animals for 3 hours in atmospheres containing low percentages of oxygen. At the end of such time in 12% to 7.4% O_2 no significant change in cardiac glycogen was found; the glycogen of gastrocnemii was not altered in this nor in the lower oxygen percentages which follow. After 3 hours in 6.2% O_2 the cardiac glycogen was 339 ± 101, and in 5.8% it was 221 ± 52. When the oxygen was reduced further, some of the animals did not survive, although 3-hour survivals occurred in as low as 4% O_2. Between 5.8% and 4% O_2, the cardiac glycogen of survivors was 192 ± 65. Those which did not survive, died of cardiac failure with surviving respiratory movements; the hearts of dying animals caught during the period when respirations still persisted, and including 3 with ventricular fibrillation, had glycogen values averaging 85 ± 15. From these experiments alone it can not be concluded that low cardiac glycogen stands in causal relation to cardiac failure, but it is established that there is a sharp difference in the level of cardiac glycogen in hearts which survive and those which do not survive severe grades of anoxaemia.

The recovery process following anoxaemia is rapid and complete. Thus immediately after 3 hours in 5.8% O_2, cardiac glycogen was found to be 221 ± 52, whereas after ¾ hour recovery from such it was 400 ± 24, after 1 hour recovery 485 ± 17, after 1½ hour recovery 484 ± 38 and after 3 hours 547 ± 20.

Single gastrocnemii were exercised by a uniform electrical stimulation. Immediately after such exercise, the glycogen content was 214 ± 49. Others allowed to recover contained (a) in ½ hour 357 ± 51, (b) in 1 hour, 407 ± 49, (c) in 2 hours 430 ± 47. It is to be observed that the recovery in a single exercised gastrocnemius, which has approximately the same weight as a heart, is not so rapid or complete.

A discussion of the significance of these findings is considered out of place at present. Two observations, however, are thought important: (1) Consistent values for cardiac glycogen in the albino rat have been obtained both for 24-hour fasted controls and for a variety of conditions. (2) In view of (a) the well maintained level of cardiac glycogen under such conditions as fasting, epine-

phrine injection, alkalosis, acidosis, and exercise and (b) the prompt recovery of glycogen after it has been lowered by anoxaemia, it would seem that there exists a quite efficient mechanism for the maintenance of cardiac glycogen.

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Unilateral Overflexion of the Limbs Following Experimental Lesions in the Pons and Upper Medulla.*

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A characteristic and constant unilateral overflexion of the limbs, during progression or isolated movement of the limbs, has been observed in 12 to 15 cats following hemisection of the brain-stem at the level of the pons or upper medulla—the vestibular nuclei being uninvolved. The abnormal action occurs in the limbs on the opposite side from the lesion. Maximal rather than graded flexion occurs at the ankle, knee, shoulder, or hip joints. In some instances in progression the flexion was maintained to the extent that the animal fell to the affected side because of the failure of the fore limb to extend in time to catch the weight of the body. This overflexion has been seen to involve a fore limb or a hind limb individually. In cases where the lesion extended across the mid-line so as to involve a small medial segment of the opposite half of the stem the ability of the animal to right and to maintain the righted position was impaired or eliminated. In these cases the overflexion occurred when rhythmic running movements were executed. If the animal was passively held in the righted position, typical walking movements embodying overflexion occurred.

Overflexion did not occur following: (1) hemisection of the stem through the thalamus or mid-brain, or even after complete unilateral removal of the mid-brain. (2) Unilateral or bilateral section of the lateral quarter segments of the pons or upper medulla. It was therefore not due to the absence of a rubro-spinal tract. (3) Hemisection of the spinal cord at upper cervical levels. (4) Injury or removal of the lateral lobes or vermis of the cerebellum.

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