creased sodium content after sodium loading confirms the impression gained from these previous studies; namely, that the sodium content of extracellular fluid and soft tissue cells is closely guarded in the normal animal and can only be altered by extreme situations. The changes in the sodium content of bone mineral, on the other hand, confirm the concept that this phase of the skeleton acts as a reservoir of sodium which is able, under appropriate circumstances not only to release sodium into the extracellular fluid, but also to store this ion.

Summary. 1) The composition of plasma, muscle, liver and bone was examined in adult male rats. Three rats served as controls, and 5 rats were given a daily load of sodium chloride approximating 50% of the total body sodium for 24 days. 2) The sodium content of plasma, muscle and liver was unchanged in the experimental animals at the end of this time. However, bone mineral sodium increased significantly, and bone water declined.

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Effect of Fasting or Underfeeding on Glucose Tolerance of Rats.* (23625)

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Humans(1) and rabbits(2) fasted for several days show glucose tolerance curves of the diabetic type, *i.e.*, increased elevation and delayed decline of blood sugar levels after glucose administration. The degree of diminution in glucose tolerance is proportionate to the duration of the fast. Hill *et al.*(3) found that in rats fasted for 96 hours (sex, weight and previous diet not specified) which received 400 mg glucose/100 g body wt by stomach tube, blood sugar progressively rose and reached 280 mg% 5 hours after the gavage, whereas it did not exceed 130 mg% if the rats were fed a diet rich in glucose during the 96 hours preceding the tolerance test. We have investigated more extensively the glucose tolerance of rats fasted for 1 to 8 days and rats subjected to chronic undernutrition, which causes a variety of changes in carbohydrate metabolism(4,5,6).

Method. Male Sprague-Dawley rats fed Rockland diet were used. Glucose was given by gavage (300 mg/100 g body wt., 30% solution, or 400 mg/100 g, 40% solution) or into the saphenous vein exposed without anesthesia (100 mg/100 g, 40% solution). Oral glucose tolerance tests were performed in rats fasted for 1, 4, 6 or 8 days and intravenous tolerance tests also in rats which were not fasted. Before fasting these animals were fed

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TABLE I. Duration of Fast and Oral Glucose Tolerance of Previously *Ad libitum*-Fed Rats.

Days fasted	No. rats	Body wt, g	Initial blood sugar, mg %	Blood sugar change, mg %* Hours after glucose ½	1	2	3	4	Dose glucose, mg/100 g
1	8	217 ± 6†	73 ± 3	+39 ± 7	+39 ± 7	+35 ± 4	+27 ± 5		300
4	8	185 ± 3	77 ± 2	+41 ± 3	+51 ± 5	+38 ± 4	+30 ± 5		"
6	8	166 ± 4	76 ± 2	+28 ± 4	+32 ± 5	+23 ± 3	+22 ± 2		"
8	4	147 ± 2	62 ± 3	+44 ± 4	+70 ± 4	+69 ± 9	+48 ± 9		"
4	8	253 ± 5	70 ± 1	+33 ± 5	+31 ± 4	+47 ± 4	+52 ± 5		400
4	8	265 ± 5	82 ± 2		+22 ± 5	+23 ± 6	+38 ± 6	+42 ± 7	"

* Compared with initial value. † Mean ± stand. error.

ad libitum. Further rats (230-250 g) were placed for 6-7 weeks on a regimen of 10 g ground Rockland diet daily and water *ad libitum*. Controls fed the same diet without restriction consumed an average of 20 g daily. They were either of the same age as the experimental rats (age controls) or selected to match their final weight (size controls). Experimental and control animals were fasted for 24 or 96 hours before the administration of glucose by stomach tube (300 mg/100 g, 30% solution) or intravenous injection (100 mg/100 g, 40% solution). Blood was taken from the tail vein before and at intervals (*cf.* Tables) after the feeding or injection of glucose. Blood sugar levels were determined as in earlier studies(5). Liver glycogen levels were measured in groups of underfed rats and size controls which were tube-fed glucose (300 mg/100 g) after a 22 hour fast and killed 2 hours later, and in animals which had received no glucose during the terminal 24-hour fast. Under Nembutal anesthesia (5 mg/100 g) liver pieces weighing about 1 g were removed, frozen between blocks of dry ice and weighed in the frozen state. Dissolved alcoholic precipitates of 30% KOH digests were then analyzed with the method of Montgomery(7). In some underfed rats the penis was ligated and bladder urine accumulated in the course of the oral glucose tolerance test examined for sugar with Benedict's reagent.

Results. Table I shows that prolongation of acute fasting up to 6 days failed to result in the progressively diminishing oral glucose tolerance described in rabbits(2). Rats fasted for 8 days were moribund (9 out of 13 had died between the 6th and 8th day of starvation) and their decreased glucose tolerance could therefore have been due to nonspecific changes such as impaired circulation. Even rats given 400 instead of 300 mg/100 g of glucose after a 4 day fast did not show the exorbitant elevation of blood sugar described by Hill *et al.*(3) in similarly treated animals. Reasons for this discrepancy are not obvious, perhaps owing to lack of particulars in Hill *et al.*'s paper. In' intravenous glucose tolerance tests non-fasted animals showed a faster return of blood sugar levels towards normal than fasted rats, but a significant change in slope of the blood sugar decline did not occur as fasting time was prolonged from 1 to 6 days (Table II). The 3 rats fasted for 8 days (survivors of 19 still alive at 6 days) showed a markedly reduced rate of removal of excess glucose from blood, but because of the poor condition of these animals the significance of

TABLE II. Duration of Fast and Intravenous Glucose Tolerance of Previously *Ad libitum*-Fed Rats.

Days fasted	No. rats	Body wt, g	Initial blood sugar, mg %	Blood sugar change, mg %* Min. after glucose 10	30	60
0	6	257 ± 2†	101 ± 3	+ 90 ± 6	+ 36 ± 9	+15 ± 3
1	6	234 ± 3	72 ± 1	+121 ± 4	+ 81 ± 5	+38 ± 4
4	8	190 ± 2	82 ± 1	+ 92 ± 2	+ 73 ± 4	+36 ± 4
6	8	126 ± 2	79 ± 3	+ 90 ± 3	+ 68 ± 6	+31 ± 8
8	3	151 ± 1	52 ± 8	+ 95 ± 8	+108 ± 7	+89 ± 12

* Compared with initial value. † Mean ± stand. error.

TABLE III. Oral Glucose Tolerance of Underfed and Control Rats.

Regimen	Days fasted	No. rats	Body wt, g	Initial blood sugar, mg %	Blood sugar change, mg %* Hours after glucose ½	1	2	3
Underfed	1	15	208 ± 5†	82 ± 1	+37 ± 3	+40 ± 3	+15 ± 3	+ 2 ± 5
Ad lib fed‡	1	8	210 ± 1	69 ± 3	+58 ± 4	+48 ± 4	+47 ± 2	+27 ± 5
Idem §	1	6	350 ± 4	67 ± 3	+24 ± 3	+30 ± 3	+30 ± 5	+23 ± 4
Underfed	4	7	182 ± 2	78 ± 6	+37 ± 8	+29 ± 4	+32 ± 3	+32 ± 3
Ad lib fed‡	4	8	185 ± 3	77 ± 2	+41 ± 3	+51 ± 5	+38 ± 4	+30 ± 5

* Compared with initial value. † Mean ± stand. error. ‡ Size controls. § Age controls.

this change is in doubt.

In the oral glucose tolerance test the blood sugar levels of underfed rats fasted for 1 day declined faster than those of controls. Age controls showed a lower blood sugar zenith than size controls, which agrees with earlier findings on the effect of body weight on glucose tolerance(8). Underfed rats fasted for 4 days showed only a somewhat flatter glucose tolerance curve than controls (Table III). In the intravenous glucose tolerance test (Table IV) underfed rats fasted for 1 day again exhibited the fastest return to preinjection blood sugar levels, but the age controls did not lag far behind, whereas in size controls the blood sugar fall was definitely more sluggish.

Previous studies(5) had shown that intestinal absorption of glucose is not impaired in underfed-fasted rats. Since we had also found(5) that liver glycogen levels of undernourished rats change only slightly when a test meal is fed after a 1-day fast, the possibility was considered that the fleeting blood sugar rise in underfed rats given glucose orally may be due to failure of hepatic glycogen formation. However, 2 hours after tube-feeding of glucose underfed rats had liver glycogen levels commensurate with those of similarly fed size controls (Table V). As in previous studies(5), the fasting liver glycogen concentrations were significantly ($p<.01$) higher in underfed rats than in controls. Glycosuria was not found in any of the underfed-fasted rats tested.

The tendency of underfed rats fasted for 1 day towards increased glucose tolerance (especially in the oral test) may be related to their enhanced insulin sensitivity(5). The faster fall of blood sugar levels in these animals is conceivably due to the exaggerated response to insulin secreted under the stimu-

TABLE IV. Intravenous Glucose Tolerance of Underfed and Control Rats.

Regimen	Days fasted	No. rats	Body wt, g	Initial blood sugar, mg %	Blood sugar change, mg %* Min. after glucose 10	30	60
Underfed	1	8	203 ± 2†	90 ± 4	+ 88 ± 4	+44 ± 5	- 2 ± 2
Ad lib fed‡	1	8	199 ± 1	70 ± 2	+100 ± 3	+79 ± 5	+33 ± 6
Idem §	1	7	332 ± 5	84 ± 2	+107 ± 3	+48 ± 4	+13 ± 2

* Compared with initial value. † Mean ± stand. error. ‡ Size controls. § Age controls.

TABLE V. Effect of Fed Glucose on Liver Glycogen of Underfed–fasted and *Ad libitum* Fed–fasted Rats.

	No glucose			2 hr after glucose		
	No. rats	Body wt, g	Liver glycogen, mg %	No. rats	Body wt, g	Liver glycogen, mg %
Underfed–fasted	11	191 ± 2*	479 ± 70	12	194 ± 3	1276 ± 142
Ad lib fed–fasted	11	199 ± 1	145 ± 7	11	200 ± 1	1190 ± 53

* Mean ± stand. error.

lus of hyperglycemia.

Conclusions. Rats fasted for 1-6 days failed to show a progressive decrease of oral glucose tolerance. Return of blood sugar levels to normal after intravenous glucose was somewhat delayed after 1-6 days of fasting as compared with the fed state, but not proportionately with the duration of fast. Prolonged acute fasting therefore did not lead to typical "starvation diabetes" in rats. Chronically underfed rats fasted for 1 day and given glucose orally or intravenously exhibited a more fleeting elevation of blood sugar than *ad libitum* fed-fasted controls. This was not associated with any marked deficiency in hepatic glycogen formation from fed glucose, nor with glycosuria.

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Blood Chemistry and Parietal Eye of *Anolis carolinensis.** (23626)

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Although the parietal eye of reptiles has received considerable histological study (Tilney and Warren(8); Von Haffner(9)) its possible function remains unclear. In the present study, quantitative determinations of various organic components of the blood of *Anolis carolinensis* were made to ascertain if removal of the parietal eye affected these components. Previous studies concerning possible functions of this structure have indicated that surgical removal of the parietal eye of adult *Anolis carolinensis* caused testicular stimulation (Clausen and Poris(3)) and modification of the rate of oxygen consumption (Clausen and Mofshin(2)).

Materials and methods. The lizards (*Anolis carolinensis*) were collected in New Orleans several days prior to use. Four experiments, each involving 3 groups, were performed between February and April. Two of these experiments were of 26 hours duration and 2 were of 18 days duration. The parietal eye was removed by thermal cauterization of an etherized lizard. A drop of collodion was used to seal the wound in operated and sham-operated lizards. The sham-operation was performed by cauterization of a small midline area on the surface of the head at the level of the anterior border of the lateral eyes. Only lizards in 18-day experiments were supplied with meal worms (larvae of *Tenebrio molitor*) daily except on the last day. Droplets of water were sprinkled on the cage and on dead vegetation within the cage. At the end of each experiment, the blood of each group was analyzed. Blood was obtained by decapitation; potassium oxalate was used as the anticoagulant. Determination of uric acid was according to the method of Folin(7). Other determinations followed the technics put forth in the *Manual of Standardized Procedures for Spectrophotometric Chemistry*, edited by Fister(6). Accordingly, the lipoid-phosphorus method was basically that of Fiske and Subbarow, the urea method that of Gentzkow and Masen. The determinations of glucose, total nitrogen and non-protein nitrogen were based upon the methods and

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