Zusammenfassung

Mittels Papierchromatographie wurde das Bild der freien Aminosäuren der Netzhaut und des Cornea-Epithels des Rindes untersucht und mit den Aminosäuren des Hydrolysats des gesamten Gewebes in Beziehung gebracht. Die Ergebnisse zeigen, dass das Bild der freien Aminosäuren ein charakteristischer Zug eines bestimmten Gewebes ist, obwohl er nicht ganz genau dessen Zusammensetzung an Aminosäuren wiedergibt.

Diurnal Changes in the Brain Glycogen

As changes in the concentration of brain glycogen parallel several altered states of behaviour[1], the question arises what connection this brain metabolite has to autonomic functions which are regulated at the highest level. In the present report, the rhythms of sleep and wakefulness were chosen as representative of these functions.

In 123 rats the concentration of brain glycogen was determined in 6-h intervals in five topographical regions of the brain. The same method was used as described in a preceding paper[2].

Diurnal changes of glycogen in the cerebral cortex and the diencephalon

Astronomic time	Glycogen (mg/100 g of wet weight)*			
	Cerebral cortex	*n*	Diencephalon	*n*
0	91 ± 5·2	31	117 ± 3·8	28
6	86 ± 4·6	34	131 ± 6·2	25
12	89 ± 5·4	28	135 ± 4·2	26
18	90 ± 4·1	30	127 ± 5·1	28

* Mean ± S. E.

In the Table the characteristic results of these experiments are given. From these it follows that, while there are no significant periodic changes in the cerebral cortex, the mesencephalon, cerebellum, and medulla oblongata, there is a significant difference in the diencephalon ($P < 0{\cdot}01$ between the interval 0 and 12).

The maximal concentration of glycogen in the diencephalon coincides with the period during which there is a maximal readiness of the rat to the cerebral inhibitory state[3].

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Zusammenfassung

In 6stündigen Intervallen wurde bei der Ratte die Konzentration des Glykogens in der Hirnrinde sowie im Diencephalon, Mesencephalon, Cerebellum und im verlängerten Mark festgestellt. Innerhalb von 24 h tritt im Diencephalon – und nur in diesem – periodisch eine signifikante Veränderung der Glykogen-Konzentration ein.

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Effect of Aldosterone and Hydrocortisone on Sodium in Red Cells

Studies with aldosterone in the rat have recently led us to the theoretical conclusion that this steroid, and perhaps related ones as well, can act by conditioning sodium flux across cell membranes. We have suggested that the net effect favors an increase in intracellular sodium, possibly by hindering sodium transport (efflux[1]). The red cell seemed particularly appropriate for a direct experimental approach to this postulate. GLYNN also predictively considered this but was unable to demonstrate any effect[2]. He used red cells in a buffered medium, however. It seemed to us that the problem could not be definitively settled unless plasma were used as medium since quite possibly a cofactor or carrier is required. Further, it seemed desirable to subject the cells to forcing procedures favoring separately either influx or efflux. Accordingly, our experiments were designed to use whole blood and to follow the changes in plasma [Na] and [K] during refrigeration or subsequent rewarming. In view of our inexperience in the difficult field of red cell experimentation and of the inherent variability of whole blood studies, experiments were freely repeated. Well defined effects were demonstrated both with aldosterone and with hydrocortisone.

Methods. – Fresh blood from student volunteers was collected in heparin (0·2 ml/10 ml blood), centrifuged, and the buffy coat removed. The blood was then reconstituted. It was divided into 5 ml aliquots and each was sampled separately for the determination of plasma Na and K and of hematocrit. The aliquots were then transferred to prepared tubes containing the steroid in 0·1 ml of vehicle, or the vehicle alone. In cooling experiments the tubes were placed in a refrigerator at 4°C and sampled in the cold 2, 4, 6 or 24 h later. While in the refrigerator, they were inverted gently every half hour throughout the day. In rewarming experiments, untreated 5 ml aliquots were first refrigerated for 24 h and then sampled separately in the cold before transfer to fresh tubes containing the steroid or vehicle. Sampling was again carried out after 0·5, 1, 2, and 3 h of rewarming at 37°C. Aldosterone racemate in ethyl alcohol and hydrocortisone acetate in saline were used.

Calculations were based on the change in each datum from its own base value taken as zero. Regression coefficients were determined where applicable[3]. Further details are given with each experiment.

Results.—Effect of aldosterone on Na, K, and hematocrit during rewarming. Aliquots (5 ml) containing 1, 1·4, 1·8, and 2·0 µg/ml of aldosterone racemate, or the vehicle alone, were rewarmed and sampled as described. Five separate runs were carried out. The figure presents the essential findings. Aldosterone clearly depresses the rate of Na extrusion during rewarming. This is preceded during the first half hour by a fall in plasma [Na] which is most prominent at lower doses. The proportionality of the effect is shown by the decreasing values of the regression coefficient with increasing dose (Table I). The effects on Na are not accompanied by similar changes in K or hematocrit; these show the normal patterns of rewarming.

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[2] I. M. GLYNN, J. Physiol. *136*, 148 (1957).
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