

TABLE I. Effect of Stress on K Concentration of Rat Brain.

	Stimulus:			
	225 mm Hg	42°C	4°C	Control
	Time:			
	120 min.	23 min.	120 min.	
K conc.*	29.34	26.47	25.03	37.83
Significance†	.95	.99	.99	

* Microequivalents/g of dry tissue, mean of 8 tests.

† Difference experimental and control value, p value, (t test).

mean of 23 minutes. Subsequent procedures were the same as those under 1-(b).

3. Rats were kept in a refrigerator at 4°C for 120 minutes. The animals showed little effect of the cold (liveliness, eyes, fur). Subsequently, they were treated as those of the other groups 1-(b).

4. Controls were treated as under 1-(b).

Results are given in Table I. The differences between the individual stimuli are not significant. P values indicate degree of significance of the stimulus-control difference.

Conclusion. Systemic stresses of anoxia, heat and cold in rats result in a significant decrease of potassium concentration of the brain.

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Received June 29, 1961. P.S.E.B.M., 1961, v107.

Effect of Stress on Potassium Release from Surviving Rat Brain. (26814)

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In previous work(1) it was shown that various types of systemic stress (anoxia, heat, cold) decrease the potassium (K) concentration of the brain. This paper is an attempt to determine the importance of certain factors which may affect this potassium release.

In a first series of experiments guinea pigs were killed by a blow on the head. The brain was immediately taken out, hemisectioned and immersed in a bath of 10 ml of K-free mammalian Ringer's solution. While one half was exposed to cold (3°C) or heat (42°C) the other half was maintained at control level (23°C). Potassium release for the next 90 minutes was determined from the flame-photometric analysis of the bathing solution. Results are presented in Table I. It is as-

sumed that the potassium loss as indicated by these data is limited to the most superficial layers of the hemisectioned brain.

Extreme heat has no significant effect on K release. Extreme cold produces a decrease of K release. As either stimulus, when applied systemically to the intact animal, results in a marked increase of K release from the brain(1), it appears that the systemic effect in the intact animal is not due to a direct temperature effect on the brain.

As another approach to this problem the following tests were performed on rats: The animal was decapitated, the brain immediately removed, hemisectioned and maintained in separate tubes containing K-free Ringer solution. Oxygen was bubbled through one test tube and nitrogen through the other.

TABLE I. Effect of Temperature on K Release of Surviving Brain Hemisections (Guinea Pigs).

Time, min.	4°C	23°C	42°C
0	3.62	4.23	4.43
30	8.34	9.75	10.05
60	11.63	13.54	13.98
90	13.19	15.58	16.06
Δt	9.57	11.35	11.63

Each value is the mean of 8 tests. Units are microequivalent K released/g dry brain wt. t test analysis shows a significant difference ($p > .95$) 4°:23° and 4°:42°. Δt gives K release during the 90 min. experimental run.

TABLE II. Effect of Anoxia on K Release of Surviving Brain Hemisections (Rats).

Time, min.	O ₂	N ₂
15	12.24	9.50
35	20.59	22.83
55	23.71	32.25
Δt	11.47	22.75

Values are microequivalents K released/g dry wt. t test analysis shows significant difference ($p > .99$).

Each value is the mean of 20 tests. Δt gives the K release during the 40 min. experimental run.

the brains of 10 rats were exposed for 15 minutes to glucose-free Ringer's solution, while the other halves served as controls. Mean control value was 3.84 microequiv. per gram and that of the glucose free solution was 19.90. The difference is highly significant ($p > 0.99$). This is considered an indication that glucose is needed for maintenance of intracellular potassium.

Table III gives the effect of 4 selected drugs on K release. While complete data for 90 minutes analysis are available, listed values are only those obtained after one hour. The results indicate that epinephrine may play a role in the increased K release of stress. Surprising to us was the great increase obtained with gamma-amino-butyric-acid, and the negative results obtained with acetylcholine, while the inhibition observed with nembutal could be expected.

Conclusion. Using *in vitro* preparations of rat brains it was found that temperature had little direct effect on K release. Oxygen and glucose are necessary to maintain K

TABLE III. Effect of Drugs on *In Vitro* K Release of Brain (Rats) within 60 Minutes.

Acetylcholine		Epinephrine		Nembutal		Gamma-amino butyric acid	
Drug conc.*	K conc.†	Drug conc.*	K conc.†	Drug conc.*	K conc.†	Drug conc.*	K conc.†
10	12.49	10	17.93	6.0	9.63	50	24.90
2.0	12.60	2.0	15.79	1.2	11.01	10	19.84
.4	12.46	.4	14.33	.24	12.63	2	16.01
0	12.89	0	12.89	0	12.89	.4	13.70
						0	12.89

* Drug concentrations are mg/ml.

† K concentrations are microequivalents released/g dry tissue.

Each value is the mean of 8 tests. The increase with epinephrine and GABA, as well as the decrease with nembutal are significant ($p > .95$ —t test).

Subsequent procedures were the same as in previous experiments. The mechanical agitation by the gas bubbles increases the release of K. However, the difference between the two conditions (Table II) indicates that anoxia has a direct effect on K release of brain tissue.

In the next experiment, hemisections of

within the brain cells. Acetylcholine has no effect on K release, epinephrine and gamma-amino-butyric-acid increase the release, while nembutal acts as an inhibitor.

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Received June 14, 1961. P.S.E.B.M., 1961, v107.