was apparent from the observation that a fall of the blood pressure through bleeding and a rise of the blood pressure through the injection of dextran were associated with characteristic alterations in autonomic reactivity. On the basis of these experiments it may be said that a moderate hemorrhage leads to a reversible state of sympathetic tuning whereas the injection of dextran induces a state of parasympathetic tuning[2].

Since variations of the blood pressure regardless of the mechanism involved seemed to lead to characteristic changes in the reactivity of the autonomic nervous system it appeared likely that the barorecepetor reflexes of the sino-aortic area would be involved. This hypothesis was verified by the observation that the characteristic effects of autonomic tuning were greatly diminished or abolished by denervation of the sino-aortic areas. The tuning effects described in this paper are therefore regarded as the result of the action of baroreceptor reflexes on central autonomic structures.

In view of the fact that earlier investigations[4] had demonstrated the decisive influence of the state of excitability of the hypothalamus on the action of acetylcholine, mecholyl and histamine and on the nor-adrenaline-induced reflex slowing of the heart rate it was thought advisable to investigate the action of these drugs and of the baroreceptor reflexes on the hypothalamus. Advantage was taken of the fact that changes in the excitability of the posterior hypothalamus are accompanied by corresponding changes in the intensity of the hypothalamic-cortical discharges influencing the activity in the whole cerebral cortex[5].

Therefore alterations in the electrocorticogram may suggest changes in the intensity of the hypothalamic-cortical discharge related to the state of excitation of the posterior hypothalamus.

Several groups of experiments were performed. In one group the action of hypotensive drugs on the ECG was investigated before and after discrete lesions had been made in the posterior hypothalamus by high frequency currents[6]. These experiments showed that after unilateral hypothalamic lesions the degree of cortical excitation induced by hypotensive drugs was lessened on the ipsilateral side. In the second group, the action of hypo- and hypertensive drugs on the cerebral cortex was investigated before and after sino-aortic denervation[7]. Under control conditions acetylcholine caused an excitation of the cerebral cortex indicated by an increased asynchrony of cortical potentials and an increase in the integrated amplitude of the fast potentials[8]. Noradrenaline, however, caused the opposite effect: the cortical potentials became more synchronized and the integrated amplitude of the potentials was increased for the low frequencies only. The effects of hypo- and hypertensive drugs on the ECG were practically abolished by sino-aortic denervation.

The conclusion drawn from these experiments that baroreceptor reflexes, modified by the changes in the intrasinusal pressure following the injection of acetylcholine and noradrenaline, act on the posterior hypothalamus and thereby alter the hypothalamic-cortical discharge was borne out by a third group of experiments. The action of acetylcholine and noradrenaline was studied on the potentials of the posterior hypothalamus. It was found that acetylcholine caused an excitation of the hypothalamic potentials (increased asynchrony and recruitment) whereas noradrenaline had the opposite effect[9].

In recent years numerous investigators have shown that protoveratrine excites the baroreceptors of the carotid sinus[10]. If the baroreceptor reflexes act not only on the medulla oblongata but also on the hypothalamus (and thereby on the cerebral cortex) as our experiments suggest, one should expect that the hypotensive action of protoveratrin would depend on the state of excitability of the hypothalamus. Experiments were performed, therefore, on the action of protoveratrine (0·0005 mg/kg intravenously) on the blood pressure of cats[9]. The mean fall of the blood pressure in the control group on administration of this drug was 37 mm Hg whereas following reduction of the excitability of the posterior hypothalamus through the intrahypothalamic injection of nembutal or high frequency coagulation of this area the mean fall of the blood pressure was 5·9 mm Hg.

Zusammenfassung

Fallen des Blutdruckes bewirkt sympathische, seine Steigerung hingegen parasympathische «Umstimmung». Erstere ist durch Sympathicotonie und erhöhte Reizbarkeit des sympathischen Systems, letztere durch entsprechende Änderungen des parasympathischen Systems charakterisiert. Diese Änderungen des autonomen Systems resultieren aus den Blutdruckzügler-Reflexen, deren Wirkung sich auch auf den Hypothalamus posterior und von dort auf die gesamte Hirnrinde erstreckt.

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[5] E. GELLHORN, *Physiological Foundations of Neurology and Psychiatry* (University of Minnesota Press, Minneapolis 1953); Arch. int. Pharmacodyn. *93*, 434 (1953); EEG Clin. Neurophysiol. *5*, 401 (1953). – W. KOELLA and E. GELLHORN, J. comp. Neurol. *100*, 243 (1954). – E. GELLHORN, Brain *77*, 401 (1954). – W. KOELLA and H. BALLIN, EEG Clin. Neurophysiol. *6*, 629 (1954).

[6] Unpublished experiments wirh E. B. SIGG.

[7] H. NAKAO, H. M. BALLIN, and E. GELLHORN, EEG Clin. Neurophysiol. *8*, 413 (1956).

[8] In these experiments the ECG was studied with an Offner frequency analyser.

[9] Unpublished experiments with H. M. BALLIN.

[10] L. CALLIAUW, Arch. int. Pharmacodyn. *107*, 75 (1956). – S. C. WANG, S. H. NGAI, and R. G. GROSSMAN, J. Pharmacol. exp. Therap. *113*, 100 (1955). – G. MATTON, Arch. int. Pharmacodyn. *103*, 13 (1955).

Corrigenda

H. BÄCHTOLD und A. PLETSCHER: *Einfluss von Isonikotinsäurehydraziden auf den Verlauf der Körpertemperatur nach Reserpin, Monoaminen und Chlorpromazin*, Experientia, vol. XIII, Heft Nr. 4, S. 163 (1957).

Auf Seite 163, 11. Zeile von oben, soll es richtig heissen «*Rimifon*» (anstatt Rimiform).

MARIA SZÉKELY: *Die Bedeutung der Mitochondrienstruktur für die Zitronensäuresynthese*, Exper. *13*, Heft Nr. 1, 24 (1957).

Auf Seite 25, linke Kolonne, nach der Tabelle III muss es richtig heissen: «Dabei konnte die Hemmung durch höhere Konzentrationen der an der Reaktion beteiligten Substrate und Coenzyme *nicht* aufgehoben werden» (anstatt aufgehoben werden).
