The receptors for the axon reflex sweating have a property of being stimulated by nicotine and agents with nicotinic action, in common with the sympathetic ganglion cells and the carotid body chemoreceptors. The similarities and differences between the properties of the latter two structures were comprehensively reviewed by KONZETT and ROTHLIN[6]. The sympathetic ganglion cells have been shown to be selectively paralyzed by the cyanides[7]. Since the work of HEYMANS *et al.*[8], the cyanides, on the other hand, have been known to stimulate the carotid body chemoreceptors. In this respect, the receptors responsible for the axon reflex sweating resemble the carotid body chemoreceptors.

MASAO WADA, TSUYOSHI AOKI, and WATARU KOYAMA

Physiological Laboratory, Tohoku University School of Medicine, Sendai (Japan), July 12, 1957.

Zusammenfassung

Subkutane Einspritzung von Kalium- oder Natriumzyanid in die Zehenballen der Katze führt über einen Axonreflex zu merklichem Schwitzen. Dieser Effekt von Kaliumzyanid wird durch Procain und Atropin gehemmt, nicht aber durch Hexamethonium. Auch beim Menschen ruft intrakutan eingeführtes Natriumzyanid eine entsprechende Schweissabsonderung hervor.

[6] H. KONZETT and E. ROTHLIN, Exper. *9*, 405 (1953).

[7] H. KONZETT and E. ROTHLIN, Wien. klin. Wschr. *64*, 638 (1952). – M. G. LARRABEE and D. W. BRONK, Cold Spring Harbor Symposia on Quantitative Biology (New York) *17*, 245 (1952).

[8] C. HEYMANS, J. J. BOUCKAERT, and L. DAUTREBANDE, Arch. int. Pharmacodyn. *40*, 54 (1931).

The Influence of Frequency of Stimulation on Synaptic Transmission at Different Temperatures

Cooling of the ganglion causes failure of the nictitating membrane contractions on preganglionic nerve stimulation[1]. In some of our preliminary experiments[2], we noticed that at higher frequencies of stimulation nictitating membrane contractions failed earlier on cooling the ganglion. In this communication we report some further experimental results concerning this effect of different frequencies. In some experiments nictitating membrane contractions were recorded, in others the acetylcholine output was determined.

The superior cervical ganglion of chloralosed cats was perfused in the usual way. The cervical sympathetic trunk was stimulated with square voltage pulses of 1 ms duration at frequencies of 2, 10 and 15 shocks per s. The ganglion was heated or cooled in the way described in one of our earlier papers[3]. The temperature was measured by means of a thermocouple connected to a galvanometer. Nictitating membrane contractions were recorded with an isotonic lever. In experiments in which the acetylcholine output was determined, the post-ganglionic trunk was tied and eserine sulphate (1:100000) added to Locke's solution. Acetylcholine was assayed on blood pressure of eviscerated chloralosed cats.

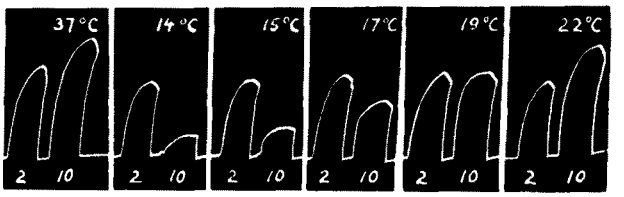

Fig. 1. – Cat, chloralose. Nictitating membrane contractions at different temperatures in the range from 14°C to 37°C. Preganglionic nerve stimulation at 2 and 10 shocks per s.

Figure 1 shows the response of the nictitating membrane to preganglionic stimulation at 2 and 10 shocks per s at 37°C. At 14°C the response of the membrane was considerably more affected for higher rates of stimulation. By increasing the temperature the response to high frequencies gradually returned to its normal value. At temperatures recorded in this experiment (14°, 15°, 17°, 19° and 22°C), the response of the membrane to low frequency stimulation was practically unaffected by temperature changes. These results are in good agreement with those reported by DOUGLAS and MALCOLM[4], who found that an inhibition to high frequency of stimulation preceeded the cold block in cat nerves.

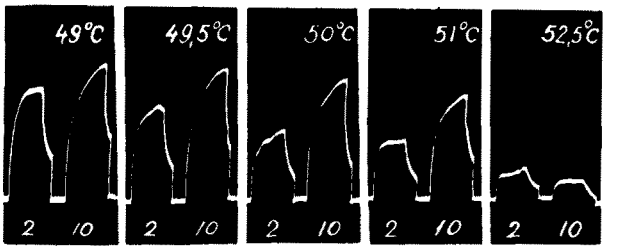

Fig. 2. – Cat, chloralose. Nictitating membrane contractions at different temperatures in the range from 49°C to 52,5°C. Preganglionic nerve stimulation at 2 and 10 shocks per s.

At high temperatures opposite effects were observed. High frequencies of stimulation were more effective. Gradual failure of the membrane contractions to low frequency stimulation started at 49·5°C (Fig. 2). The response to high frequency stimulation was almost unimpaired up to 50·8°C when a steep fall began. At 52·5° C the response to both frequencies was much reduced.

Figure 3 summarises our results. The response of the membrane (on logarithmic scale) has been plotted against the temperature. Two different curves were thus obtained, one for the low frequency (2/s, black dots), the other (circles) for the high frequency of stimulation (10/s). The low frequency curve is shifted to the left in the whole range of temperatures recorded in these experiments.

The influence of frequency of stimulation was also noticable in experiments in which the acetylcholine output was determined at different frequencies. Figure 4 shows the result of four experiments performed at 37°C and at 20°C, respectively. At 37°C the output of acetyl-

[1] K. KOSTIAL, Rad Jug. Akad. Zagreb (in press).

[2] K. KOSTIAL and V. B. VOUK, Abstracts of Communications, 20th Int. physiol. Congress, Bruxelles 1956, p. 520.

[3] K. KOSTIAL and V. B. VOUK, J. Physiol. *132*, 239 (1956).

[4] W. W. DOUGLAS and J. L. MALCOLM, J. Physiol. *130*, 53 (1955).

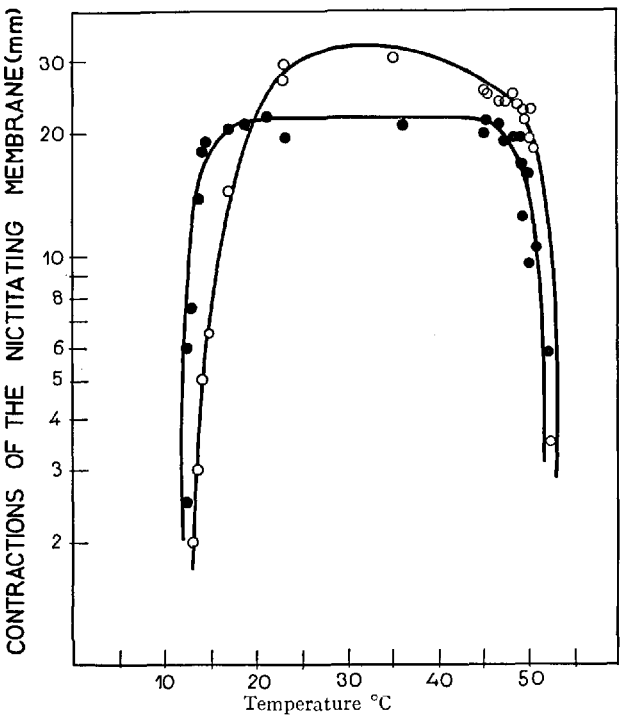

Fig. 3. – Contractions of the nictitating membrane as function of the temperature for two different frequencies of stimulation: 2/s (black dots) and 10/s (white circles).

choline was greatly increased at higher rates of stimulation. When the same experiment was repeated at 20°C an increased number of preganglionic shocks did not increase the total output of acetylcholine.

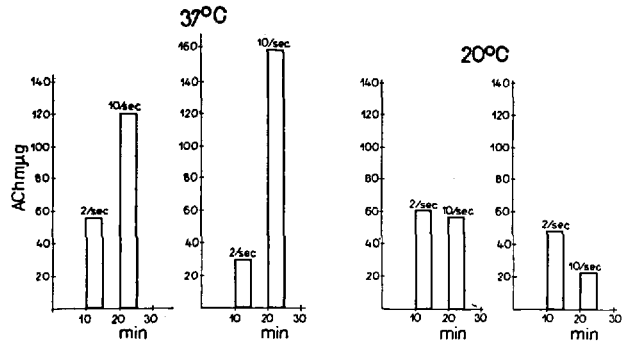

Fig. 4. – Acetylcholine output at 37°C and 20°C for different frequencies of stimulation (2 and 10/s). Each block represents the amount of acetylcholine released during 5 min stimulation of the cervical sympathetic.

These results seem to support our assumption expressed in a previous paper[3] that the disagreement between Browns results[5] and ours was due to the difference in the frequencies of stimulation used in the experiment.

This work has been supported by the Commission for Medical Research (KOMNIS), Beograd.

KRISTA KOSTIAL and V. B. VOUK

Institute for Medical Research, Yugoslav Academy of Sciences and Arts, Zagreb (Yugoslavia), October 22, 1957.

[5] G. L. BROWN, J. Physiol. *124*, 26 P (1954).

Résumé

Durant le refroidissement du ganglion cervical supérieur perfusé du chat, la réaction de la membrane nictitante est notablement plus sensible aux stimulations de haute que de basse fréquence. Aux températures élevées, c'est l'inverse.

L'influence de la fréquence des stimulations a aussi été perceptible lors des expériences, au cours desquelles le rendement de l'acétylcholine a été déterminé. A 37°C, le rendement s'accroît sensiblement lorsqu'on accélère le rythme de stimulation. A 20° C, cependant un nombre croissant de chocs préganglionnaires n'augmente pas le rendement total de l'acétylcholine.

Effects of Intense Noise on Adrenal and Plasma Cholesterol of Mice

The problem of intense noise and its relation to hearing impairment or other systemic injury in animals and man has recently received renewed emphasis. This is partly due to certain legislative measures adopted in some states where hearing loss is now listed as an occupational disease. In part it is also due to the belief that noise is a stressor agent capable of exceeding the limits of physiological hyperactivity of the pituitary-adrenocortical system.

In earlier studies[1] we found that exposure of mice to moderately intense noise (110 db, 10–20 kc) did not result in excessive adrenocortical activation and consequently noise did not appear to constitute a 'harmful' nonspecific stressor stimulus. Criteria used to measure adrenal activation were: adrenal hypertrophy, eosinopenia, thymicolymphatic involution and the appearance of gastric ulcers. Since several workers have reported that noise acts in a fashion similar to other stress agents[2] we decided to extend our studies to include much higher noise levels in an effort to elicit physiological changes characteristic of a stress response.

The sound apparatus used in the present study[3] consisted of a noise generator, a 700 watt University B-24 loudspeaker (having 24 separate 30 watt speakers), a Bogen amplifier and a Navy Beach amplifier. Sound levels attained were about 140 db with most of the energy concentrated in the 150–4800 cy/s frequency range. Such levels approximate those put out by turbo-jet engines and are near the maximal limit one can obtain with conventional diaphragm type loudspeaker systems. Individually isolated mice were exposed to noise for 5 min and autopsied immediately after exposure and at 3 and 6 h intervals for analysis of changes in adrenal weight and in adrenal content of cholesterol and serum Na, K, and Ca levels following noise exposure. Preliminary analyses for total cholesterol were made using the SPERRY-WEBB technique[4]. This was later abandoned in

[1] A. ANTHONY, J. acoust. Soc. Amer. *27*, 1150 (1955). – A. ANTHONY and E. ACKERMAN, J. acoust. Soc. Amer. *27*, 1144 (1955).

[2] E. DAY, D. FLETCHER, G. NAIMARK, and W. MOSHER, J. Aviation Med. *22*, 316 (1951). – R. MILINE and O. KOCHAK, C. R. Ass. Anat., 38e réunion, Nancy *1951*, 1. – C. FORTIER, Endocrinology *49*, 782 (1951).

[3] Authorized for publication as paper No. 2183 in the Journal Series of The Pennsylvania Agricultural Experiment Station. This work was sponsored in part by the Aero Medical Laboratory, Wright Patterson Air Force Base, Dayton, Ohio, under contract No. AF 33(616)-2505.

[4] W. M. SPERRY and M. WEBB, J. biol. Chem. *187*, 97 (1950).