Arh. hig. rada, *16* (1965) 37

THE INFLUENCE OF LOW TEMPERATURE ON ACETYLCHOLINE AND POTASSIUM SENSITIVITY OF THE SUPERIOR CERVICAL GANGLION

BLANKA ŠLAT and KRISTA KOSTIAL

Institute for Medical Research, Yugoslav Academy of Science and Arts, Zagreb

(Received for publication April 24, 1965)

Results of the effect of low temperature on the sensitivity of ganglion cells to injected acetylcholine or potassium chloride are presented. Contractions of the nictitating membrane of the cat have been recorded.

Kostial and *Vouk* (1, 2) and *Kostial* (3) using the ganglion perfusion technique showed that a decrease of temperature from 20–10° C caused a reduction in the output of acetylcholine (ACh) from the cat's superior cervical ganglion as well as a reversible block of synaptic transmission. Here are presented further experimental results concerning the effect of low temperature. The purpose of our experiments was to establish the sensitivity of cooled ganglion cells to injected ACh or potassium.

The experiments were performed on cats. Anaesthesia was induced with ethylchloride and ether and maintained by intravenous injections of chloralose (0.09 g/kg). The superior cervical sympathetic ganglion was prepared for perfusion by the conventional method modified by *Perry* (4). The preganglionic trunk was stimulated through platinum electrodes with square voltage pulses of 1.0 msec duration. In some experiments the postganglionic fibres were stimulated in the same way as preganglionic nerve fibres. Acetylcholine (55 or 110 mμM in 0.1 ml) or potassium (KCl, 2.7, 4 and 5.4 μM in 0.1 ml) were injected into the arterial cannula. Nictitating membrane contractions were recorded with an isotonic lever. The perfusion fluid was eserinized Locke's solution. In order to attain the desired temperature level the perfusion fluid and Locke's solution surrounding the ganglion were cooled down or heated thermostatically. The temperature was measured by means of a thermocouple connected to a galvanometer. The thermocouple needle was immersed in Locke's solution touching the outer part of the ganglion.

The sensitivity of ganglion cells to acetylcholine was reduced at low temperature. A partial blocking effect could be observed in the temperature range from 15–30° C. Between 11–15° C ganglion cells were no longer sensitive to injected acetylcholine. This effect was reversible (Fig. 1).

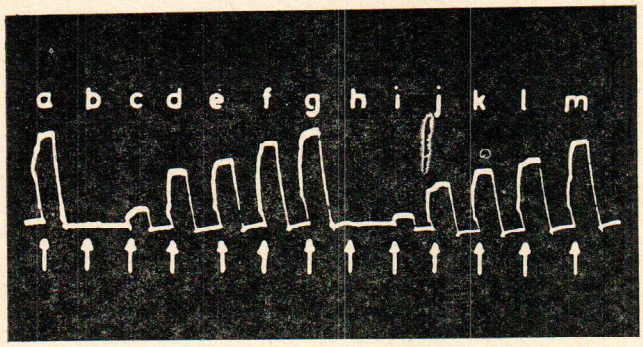

Fig. 1. – Cat, chloralose. Nictitating membrane contractions at different temperatures in response to arterial injection of ACh (↑). a, g = 38°, b, h = 15°, c, i = = 20°, d, j = 25° C, e, k = 30° C, f, l = 35° C. Ganglion perfused with Locke's solution.

In Figure 2. results of six experiments are presented. Contractions of the nictitating membrane at low temperature are expressed in percentage of the height of membrane contractions at 38° C. The results represent the mean and the standard error of the mean.

It was observed earlier by *Punt* (5) that cooling delayed acetylcholine contracture of frog rectus abdominis muscle. Results obtained on the isolated frog heart ventricles (6) also indicate a similar effect of the temperature on acetylcholine action.

The influence of temperature was also observed in our further experiments in which the sensitivity of ganglion cells to injected potassium chloride was registered. During the cooling of the ganglion the sensitivity was also reduced. A reduction of temperature to 11–15° C caused a failure of the nictitating membrane contractions in response to injected potassium. (Fig. 3).

Figure 4 shows the results of four experiments. Contractions of the nictitating membrane at low temperature are presented in the percentage of the height of membrane contractions at 38° C.

In some experiments the reversibility of synaptic transmission after prolonged cooling of the ganglion was investigated. Lowering the temperature of the ganglion to 3–7° C for a period of 70–180 minutes caused

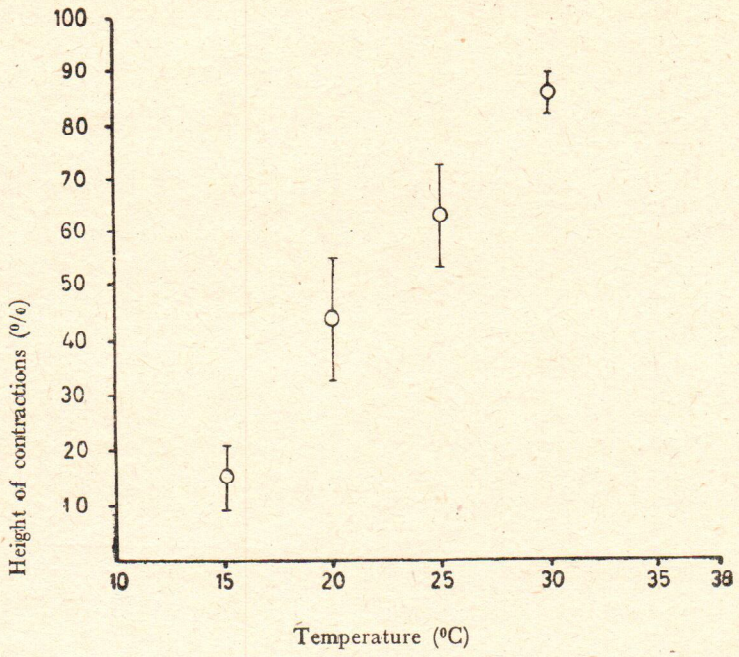

Fig. 2. – Temperature dependence upon contractions of the nictitating membrane of the cat following arterial injections of ACh. Results are expressed in percentage of the height of contractions obtained at 38° C. Each point represents the mean with its standard error for six experiments.

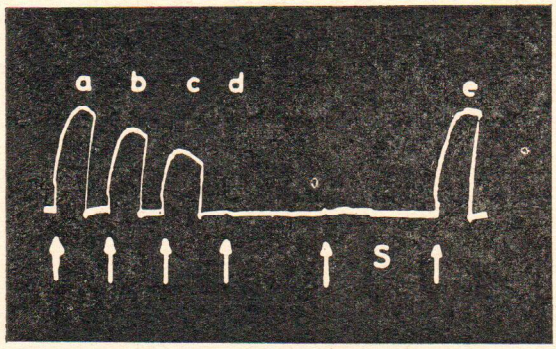

Fig. 3. – Cat, chloralose. Contractions of the nictitating membrane in response to injected potassium (↑) at different temperature: a, e = 38°, b = 25°, c = 20°, d = 15° C. Ganglion perfused with Locke's solution.

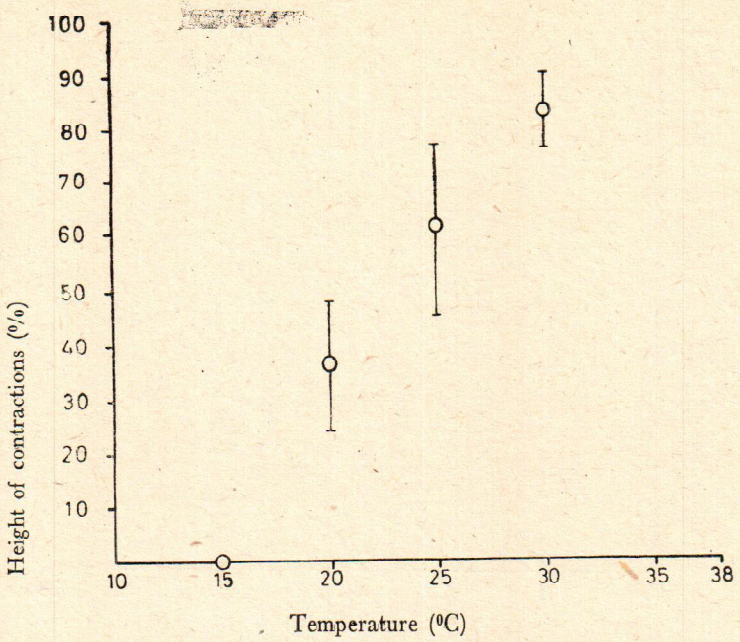

Fig. 4. – Temperature dependence upon contractions of the nictitating membrane of the cat following arterial injections of KCl. Results are expressed in percentage of the height of contractions obtained at 38° C. Each point represents the mean and its standard error for our experiments.

a blocking of synaptic transmission as indicated by the failure of the nictitating membrane contraction to preganglionic nerve stimulation. This effect was also reversible immediately after increasing the temperature of the ganglion.

We wish to express our thanks to Miss M. Pišonić, from our laboratory, for her valuable assistance.

This work was supported by the Federal Council for Scientific Research, Belgrade.

References

1. *Kostial, K., Vouk, V. B.:* J. Physiol., *132* (1956) 239.
2. *Kostial, K., Vouk, V. B.:* Experimentia, XIV/3 (1958) 103.
3. *Kostial, K.:* Rad, Knj. 316 (1958) 175.
4. *Perry, W. L. M.:* J. Physiol., *119* (1953) 439.
5. *Punt, A.:* Arch. Neerland Physiol., *26* (1942) 212.
6. *Turpaev, T. M.:* Doklady Akad. Nauk SSSR, *102* (1955) 323.

Sadržaj

DJELOVANJE NISKE TEMPERATURE NA OSJETLJIVOST GORNJEG VRATNOG SIMPATIČKOG GANGLIJA NA ACETILKOLIN I KALIJ

Registrirane su kontrakcije membrane niktitans izazvane injiciranjem ACh ili kalija kod raznih temperatura. Osjetljivost ganglijskih stanica na injicirani ACh ili kalijev klorid potpuno nestaje pri temperaturi 11–15° C, a djelomično je smanjena u temperaturnom području od 15–30° C. Djelovanje snižene temperature je reverzibilno. Produljena ekspozicija ganglija niskoj temperaturi (3–7° C) uzrokovala je reverzibilnu blokadu sinaptičke transmisije.

Institut za medicinska istraživanja i medicinu rada, Zagreb

Primljeno 24. IV 1965.