

EFFECT OF MANGANESE ON SYNAPTIC TRANSMISSION

KRISTA KOSTIAL and ZDENKA JURICIC

Toxicology Department, Institute of Industrial Hygiene Zagreb

(Received for publication June 12, 1956)

In the course of our investigations of the influence of metal ions on synaptic transmission and acetylcholine output, we have noticed that manganese ions cause a block of ganglionic transmission when added to the fluid perfusing the superior cervical ganglion.

The experiments were performed on cats. Anaesthesia was induced with ethylchloride and ether and maintained by intravenous injection of chloralose (0.09 g/kg). The superior cervical ganglion was prepared for perfusion by the conventional method modified by Perry (1). The preganglionic trunk was stimulated through platinum electrodes with square voltage pulses of 1.0 msec duration. The shocks were supramaximal. In some experiments the postganglionic fibres were stimulated in the same way as described for preganglionic nerve fibres. Contractions of the nictitating membrane were recorded with an isotonic lever. Manganese chloride was added to the perfusion fluid (Locke's solution) without altering the other components.

Addition of 25 and 50 micrograms of manganese chloride per 1 ml to the perfusion fluid caused only a slight reduction of the nictitating membrane contraction. A nearly complete block of synaptic transmission occurred at a concentration of 100 micrograms of manganese per ml (Fig. 1). The transmission was gradually restored after perfusing

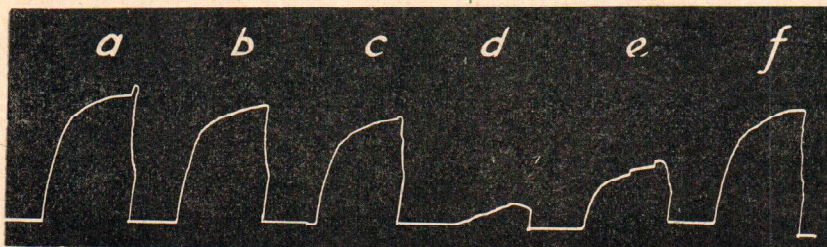


Fig. 1. Cat, chloralose. Contractions of the nictitating membrane in response to stimulation of the cervical sympathetic trunk at 2/sec for 45 sec; a, e, f = ganglion perfused with Locke's solution; b = addition of 25 micrograms of Mn^{++}/ml ; c = 50 micrograms of Mn^{++}/ml ; d = 100 micrograms of Mn^{++}/ml .

the ganglion with Locke's solution. In another experiment (Fig. 2) manganese ions caused a block of ganglionic transmission (100 micrograms/1 ml) with immediate recovery after changing over to Locke's solution.

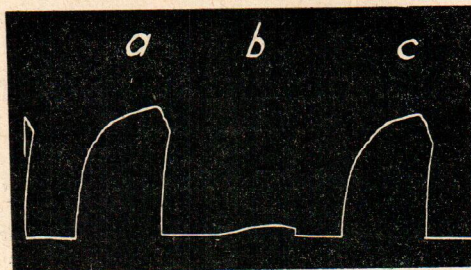


Fig. 2. Cat, chloralose. Contractions of the nictitating membrane. Stimulation of the preganglionic fibres at 2/sec for 45 sec: a, c = perfusion with Locke's solution; b=100 micrograms of Mn^{++} /ml added to the perfusion fluid.

In some experiments nictitating membrane contractions were recorded at pre- and postganglionic nerve stimulation. Addition of manganese caused blocking of synaptic transmission on preganglionic stimulation without affecting the response of the membrane to postganglionic stimulation (Fig. 3). Calcium ions have an antagonising action on the effect produced by manganese. A fivefold increase in the calcium concentration of the perfusion fluid prevents the blocking action of manganese (Fig. 3, 3, f).

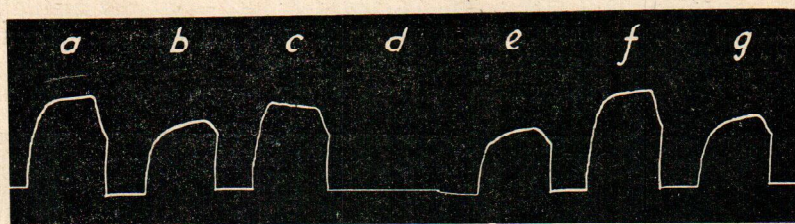


Fig. 3. Cat, chloralose. Contractions of the nictitating membrane in response to stimulation of the preganglionic (b, d, e, g) and postganglionic (a, c, f) nerve fibres at 2/sec for 45 sec: a, b, g = ganglion perfused with Locke's solution; c, d = addition of 100 micrograms of Mn^{++} /ml; e, f = the same as c and d + 10,5 Ca^{++} /l.

Manganese ions affect synaptic transmission in concentrations 100 times higher than lead ions (2).

Further studies on the effect of manganese ions on nerve transmission are still in progress.

References

1. *Perry, W. L. M.*: J. Physiol. 119 (1953) 439.
2. *Kostial, K., Vouk, V. B., Purec, Lj.*: Arh. hig. rada, 5 (1954) 351.

Sadržaj

UTJECAJ MANGANA NA SINAPTIČKU TRANSMISIJU

Dodatak mangana tekućini, kojom se vrši perfuzija izoliranog gornjeg vratnog simpatičnog ganglija mačke, izaziva smanjenje kontrakcija membrane niktitans na stimulaciju predganglijskih vlakana vratnog simpatikusa. Stimulacija postganglijskih vlakana izaziva normalne kontrakcije membrane usprkos prisutnosti manganovih iona. Kalcijevi ioni imaju antagonističko djelovanje na blokadu sinaptičke transmisije izazvanu dodatkom manganovih iona. Mangan izaziva sinaptičku blokadu u koncentraciji od 100 mikrograma na 1 ml.

*Toksikološki odjel
Instituta za medicinska istraživanja,
Zagreb*

Primljeno 12. VI. 1956.