INHIBITORY EFFECT OF BULBOCAPNINE ON THE MONOSYNAPTIC REFLEX RESPONSE OF THE RAT

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Bulbocapnine and prochlorperazine are known to produce a cataleptoid behavior in animals. The present report forms a part of the experiments initiated in order to analyse the change of the motor function in the cataleptoid behavior by these drugs and deals exclusively with the inhibitory effect of bulbocapnine on the monosynaptic reflex discharge of the rat.

A decrease in the magnitude of the monosynaptic response is seen by the intravenous administration of bulbocapnine in the dosage of 0.1 to 0.5 mg/kg in the spinal rat (Fig. 1). Larger doses (0.3

FIG. 1. Effect of bulbocapnine on spinal reflex discharges of the spinal rat. The monosynaptic (open circles) and polysynaptic reflex response (filled circles) evoked by a square wave stimulation (0.1 msec, 0.4 c/s, supramax.) to the left tibial nerve were recorded from L4 ventral root. The inserted discharge patterns are the instances corresponding to the points shown by the same alphabetic letters and arrows in the graph.

to 1.0 mg/kg i.v.) are required in the nembutalized rat. Depression of the monosynaptic response occurred very promptly and recovered generally within 30 to 60 minutes. On the other hand, no significant changes were observed on the polysynaptic responses as shown in Fig. 1. Since the effect described above is shown intensely in the spinal rat, it may be concluded that bulbocapnine has a direct action on the spinal cord. Prochlorperazine fails to cause any changes on the spinal reflex discharges of the spinal rat. As depression of the monosynaptic response by bulbocapnine is anta-

gonized by pretreatment with picrotoxin, but not by strychnine, the mechanism of presynaptic inhibition may be concerned with this action to some degree.

More precise investigation for pursuing the mechanism on the spinal reflex and a comparative study with the cataleptoid behavior are needed.

THREE DISTINCT VASOMOTOR MECHANISMS ACTIVATED BY DMPP

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Intraarterial injection of 20 to 100 μ g of dimethylphenylpiperazinium (DMPP) into the perfused hindquarters produced vascular response in the perfused area in autocirculation preparation of the dog. In 2 cases a small rise followed by a fall, and in 6 a moderate rise followed by a slight fall were observed, the hypotensive component tending to be less prominent after transection of the sympathetic chains at L5-L6.

i) Treatment of the animal with β -TM 10, guanethidine or bretylium in 5 to 10 mg/kg, i.v., reversed the pressor effect of i.a. DMPP. ii) Treatment with antimuscarinic doses of atropine or scopolamine strikingly enhanced the pressor effect or reduced the depressor component of DMPP given into the perfused area. iii) Furthermore, a similar enhancement of the pressor effect or reduction of the depressor component of i.a. DMPP was exerted by tripeleennamine or D-chlorpheniramine in doses reducing the hypotensive effect of histamine. These 3 distinct mechanisms activated by DMPP in the perfused area are shown in Fig. 1, which also indicates the response induced by the

FIG. 1. In autoperfusion of dog hindquarters, distal electrostimulation (E) of the divided lumbar sympathetic chains and i.a. DMPP (D) are serially challenged by β -TM 10, tripeleennamine (PBZ) and atropine (Atr).

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