period, if stimulation is kept at S_9 at a frequency not superior to 0.1 c/sec. At a frequency of 1 c/sec, however, the response is unstable and disappears in 10 min, at 10 c/sec in 3–4 min and at 100 c/sec in a few seconds. The same applies for the analogous junctions of the other abdominal ganglia.

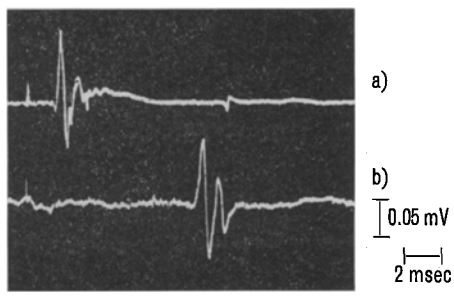

Fig. 2. Response at R following stimulation at S_{13} (a) and S_3 (b). See Figure 1.

The function of this efferent pathway is still to be investigated. A detailed account of this work will be published elsewhere.

Riassunto. Viene descritta una nuova via efferente della catena ganglionare di *Periplaneta americana*, che origina nel 1° ganglio addominale, raggiunge i nervi cercali (NXI) ed è interrotta da sinapsi in ciascuno dei sei gangli addominali. Vengono riferiti la velocità di conduzione nei connettivi ed i tempi di trasmissione a livello dei gangli.

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The Effect of Picrotoxin and Strychnine upon Inhibition of Fusimotor Neuron Discharges Caused by Cutaneous Fiber Stimulation

It was reported by Hunt and Paintal and by Koizumi et al.[1] that some spontaneously firing fusimotor neurons are inhibited for up to several hundred milliseconds following stimulation of various peripheral nerves: most evident with cutaneous nerve stimulation. The present experiments have been designed to observe the effects of picrotoxin and strychnine upon the fusimotor inhibition, since the actions of these two convulsants upon synaptic transmission in the spinal cord appear to be different[2].

Experiments were performed on 16 cats. Under ether anesthesia both carotid arteries were ligated and the trachea was cannulated. The lumbosacral cord was exposed by laminectomy in the usual manner and the L_7 or S_1 ventral root was cut intradurally at its exit from the dural cavity. After the spinal cord was transected at the atlanto-occipital membrane ether was discontinued and respiration was maintained artificially. The central cut end of dissected ipsilateral sural nerve was mounted on silver stimulating electrodes. The L_7 or S_1 ventral root was split into fine filaments containing only one spontaneously firing fusimotor fiber and these filaments were mounted on a pair of silver recording electrodes. Criteria for identification of γ motoneuron is described elsewhere[3].

After the animals were immobilized by i.v. injection of gallamine triethiodide (Gallamine, Teisan, Co.), a saline solution of picrotoxin (0.2 mg/ml) or strychnine nitrate (0.02 mg/ml) was slowly injected in 1 min into the radial vein. The drug was applied at least 3 h after the cessation of ether anesthesia.

Results. In preliminary experiments on 7 units, it was found that the inhibitory effects were strongest when the sural nerve was stimulated at around 7 to 10 times the threshold of the largest fibers. Thereafter, the observation was made at about 10 times threshold where the strength was sufficient to produce clear inhibitory effect, the duration of the rectangular pulses being 0.05 msec and the frequency 0.3 per sec.

One representative example of the effect of picrotoxin on a spontaneously firing fusiomotor neuron is shown in Figure 1A. The firing intervals became more irregular at the dose of 0.4 mg/kg (Figure 1Ab), the number of spikes

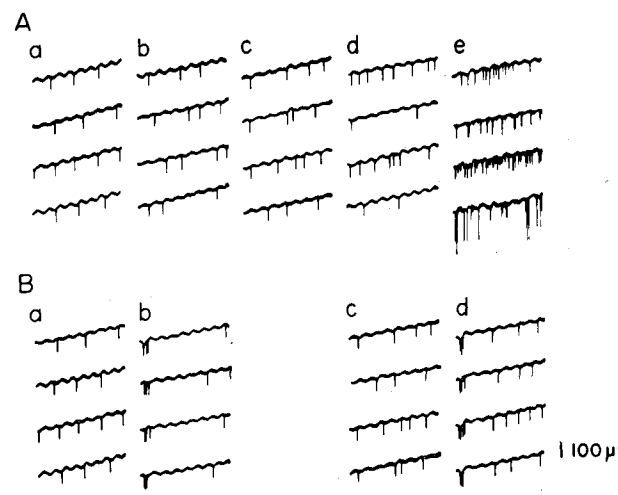

Fig. 1. A) The effect of picrotoxin upon spontaneous firing of a fusimotor neurone. a, control records; b, c, d, e, after injection of picrotoxin at the doses of 0.4, 0.6, 0.8 and 1.0 mg/kg, respectively. At 1.0 mg/kg convulsive discharges of both large and small neurones appeared (e). B) The effect of picrotoxin on inhibition of the fusimotor neurone shown in A. a, b, before injection of picrotoxin; a, spontaneous discharge; b, the spontaneous discharges ceased for about 134 msec on stimulation of sural nerve, stimuli being applied at the beginning of the sweeps; c, d, after picrotoxin injection of 0.6 mg/kg; d, the period of inhibition was shortened to about 74 msec. One sweep represents 180 msec.

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[2] D. R. Curtis, in *Structure and Function of Inhibitory Neuronal Mechanisms* (Ed. C. von Euler, S. Skoglund and U. Soderberg; Pergamon Press, New York 1968), p. 429.

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increased slightly at 0.6 mg/kg (Figure 1Ac) and convulsive discharge appeared at 1.0 mg/kg (Figure 1Ae). The same tendency was recognized for 9 units, but the dose evoking convulsive discharges varied considerably among different animals, ranging from 0.6 mg/kg to 3.0 mg/kg.

The spontaneous firing of the unit, shown in Figure 1A, ceased for 134 ± 14 msec (mean cessation period calculated over 30 trials of stimulation and its standard deviation) on stimulation of the sural nerve at 10 times threshold (Figure 1Bb). After administration of picrotoxin, the cessation period of spontaneous firing was shortened by 45% as shown in Figure 1Bd. Figure 2 shows another example, obtained from a different animal. On this unit the cessation period was shortened by 20 to 30% at the doses of 0.6 to 1.4 mg/kg. Convulsive discharges were observed at 1.5 mg/kg. In all other units the cessation period was shortened by 10.5 to 55%. This suggests that long-lasting fusimotor inhibition by cutaneous volleys is sensitive to picrotoxin and partly blocked.

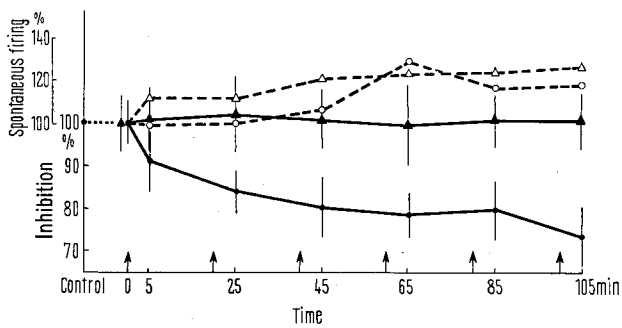

Fig. 2. Graphic representation of the effects of pictotoxin (circles) and strychnine (triangles) upon spontaneously firing frequency (open and broken lines) and fusimotor inhibition (filled and solid lines). Abscissa: time in min. At times indicated by arrows, 0.2 mg/kg of picrotoxin or 0.02 mg/kg of strychnine was injected. Ordinates: Spontaneous firing frequency as percent of control and the lower scale denotes duration of inhibition, also in percent of control, expressed as mean poststimulus pause in spontaneous firing. The data were obtained from 2 different cats. Only 1 kind of drug, either picrotoxin or strychnine, was injected to 1 animal. Records were taken 5 min after each injection. Convulsive discharges were observed at cumulative doses of 1.5 mg/kg of picrotoxin or at 0.14 mg/kg of strychnine. Vertical bars show standard deviation. In the case of picrotoxin, the inhibition is gradually shortened with cumulative doses. In the case of strychnine the duration of inhibition showed virtually no change while the spontaneous firing increased up to about 120%.

Since picrotoxin increases background discharges, there remains a possibility that the above mentioned shortening may be due to the increase in spontaneous activity. However, it may be safe to rule out this possiblity from the following 2 reasons: 1. The inhibition was frequently observed to be shortened at smaller doses which did not increase the spontaneous firing, and 2. these 2 effects did not change in parallel with increasing doses (see Figure 2).

For 7 units strychnine was injected instead of picrotoxin. On a unit shown in Figure 2, virtually no effect was observed on the duration of inhibition, although spontaneous firing was increased with strychnine. On no occasions was the cessation period of firing shortened.

It is widely accepted that picrotoxin blocks presynaptic inhibition in the spinal cord[4]. Although a possible existence of picrotoxin-sensitive, strychnine-resistant postsynaptic inhibition (KELLERTH and SZUMSKI[5]) cannot be fully excluded, it is tempting to postulate that the long-lasting inhibition of fusimotor neurones by cutaneous volleys, studied in the present experiment, is exerted presynaptically on the ground that this inhibition is sensitive to picrotoxin.

Résumé. Les effets de la picrotoxine et de la strychnine sur l'efficacité inhibitrice fusimotrice, évoqués par la stimulation des fibres sensorielles de la moelle du chat, ont été étudiés. Les observations montrent que la picrotoxine réduit l'efficacité inhibitrice, tandis que la strychnine n'a pas d'effet notable.

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28 June 1971.

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Autoradiographic Studies on the Experimental Pulmonary Fibrosis of Rats After H^3-Proline Injection

It is well known that pulmonary fibrosis is caused by various dusts, but the collagen fiber produced varies with the nature of the dust. Silicopneumoconiosis in rats is produced by inhalation or infusion of minute particles of SiO_2 and the collagen fiber developed more when caused by the other dusts. By means of autoradiography[1] we have observed that the label was incorporated into the collagen fiber in experimental silicopneumoconiosis of the rat lung by injection of H^3-Proline.

Experimental silicopneumoconiosis was induced by pertrachel infusion into the lungs with 70 mg/ml of silica saline suspension. The rats with silicopneumoconiosis were observed for 2, 4, 8 and 16 weeks after infusion of silica saline suspension. Those animals were injected i.p. with H^3-Proline (generally labelled, 2 μc/g body weight, specific activity of 360 mc/m*M*, New England Corporation) at definite times and were killed under ether narcosis at 1 h after injection of the label. The excized lungs were fixed in Bouin's solution and embedded in paraffin. The microscopic sections were cut into 10 μm thickness. Following deparaffinization, unstained sections were coated with Kodak's NTB2 emulsion by dipping method. After exposure for 4 and 8 weeks, the sections were developed and

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