Effects of Succinylcholine on γ-Fibres

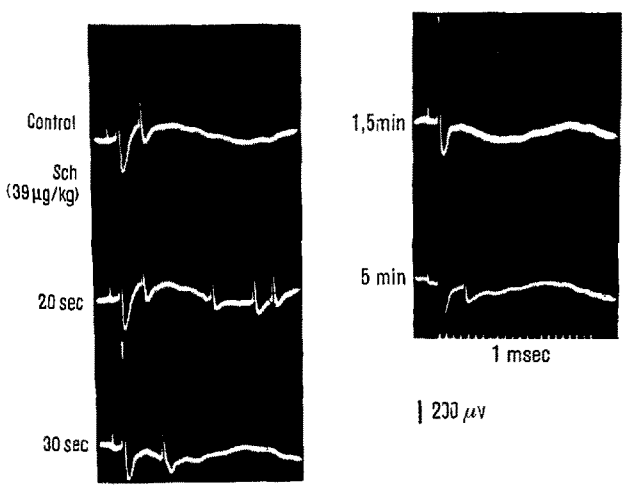
It has been reported that Succinylcholine (Sch) blocks neuromuscular transmission[1], increases afferent discharges from muscle spindles[2], has a variety of secondary effects due to increased afferent discharges from muscle spindles upon spinal reflexes[3–5], and affects the presynaptic terminals of α-motor-fibres[6,7].

The main purpose of the present experiment was to investigate, as an extension of our previous experiments on α-fibres[7], whether Sch affects the presynaptic terminals of γ-fibres.

Experiments were performed on 10 unanaesthetized spinal cats whose spinal cords were transected at the atlanto-occipital membrane. Laminectomy was performed in the usual manner, and hindlimbs were denervated except for the gastrocnemius-soleus (GS) muscles. Great care was taken not to damage the blood supply to the nerves. The ventral roots of L7 and S1 were sectioned close to the spinal cord, and the distal cut end of the roots were divided until a single active axon was obtained. A rectangular pulse of 40 μsec duration was applied at a frequency of $^1/_{2-3}$ sec through bipolar silver electrode to the GS nerve at the popliteal fossa. Sch (20–200 μg/kg) was repeatedly injected into the radial vein and records were taken at least 3 h after discontinuation of ether anaesthesia. Intervals of successive injections were more than 30 min[8].

23 γ-fibres were studied. The conduction velocities of these fibres ranged from 20–41 m/sec. 3 types of Sch effects were observed.

(1) A burst of 2–5 discharges at frequencies of 100 to 300/sec occurred in 11 out of 23 units in response to single shock stimulation of the GS nerve for less than 1 min after the drug application; an example of this effect is shown in the Figure (20 sec). The duration of the effect corresponds closely to the *initial phase* of the evoked EMG of the GS muscle when end-plates seem to be partially blocked[7].



Antidromically conducted action potentials of an α-fibre and a γ-fibre were recorded from thin S_1 ventral root filament containing 1 α-fibre and 1 γ-fibre innervating gastrocnemius-soleus muscles on stimulation of the GS nerve at the popliteal fossa (Control). Conduction velocity of the α-fibre = 115 m/sec, γ = 33 m/sec. Following injection of Sch into the radial vein at a dose of 39 μg/kg, first reaction was repetitive antidromic discharges (20 sec), then latency of the γ-spike delayed (30 sec), and finally the γ-spike disappeared (1.5 min). About 5 min later the spike recovered. No change in the evoked action potential of the α-fibre was observed during these periods. It should be noted here, however, that occurrence of repetitive antidromic discharges from α-fibres has already been reported[7].

(2) Antidromic repetitive discharges were elicited without electrical stimulation to the nerve in 7 out of 23 units. All these 7 units showed the burst discharges mentioned above to electrical stimulation.

These 2 types of Sch-evoked antidromic repetitive discharges were considered to originate from the presynaptic terminals of γ-fibres for the following reasons: (a) it is known that there is an end-plate at some γ-neuromuscular junctions which is morphologically similar to the ordinary extrafusal end-plate (plate-ending of Barker and Ip[9]); (b) it is widely believed that intrafusal neuromuscular transmission is mediated by Acetylcholine as in the extrafusal end-plate[10]. For these reasons it may not be unreasonable to consider that the above-mentioned γ-antidromic repetitive discharges originate from the presynaptic terminals, as they do in the case of α-fibres. This matter is under further investigation.

(3) Impulse conduction of γ-fibres was blocked in 14 out of 23 units after Sch injection. This blocking followed the above-mentioned antidromic repetitive discharges and lengthening of the latency of the evoked spike (Figure, 30 sec). The blocking disappeared after 5–15 min, depending on the dose of Sch; this period of blocking agrees well with that of the stimulating action of the drug on muscle spindles. In a few cases, γ-spikes disappeared without first showing the repetitive antidromic discharges. No change in the shape or latency of the evoked α-spike was observed during the period of γ-blocking. As to the mechanisms of this blocking, it is not clear at present whether it is a direct effect of Sch or a secondary effect due to an increase of potassium ions in blood serum[11,12].

Zusammenfassung. Die Wirkung von Succinylcholin auf die γ-Fasern an unanästhesierten spinalen Katzen wurde analysiert: (1) 2–5 antidrome Entladungen einer Frequenz 100–300/sec bei einer elektrischen Einzelreizung; (2) antidrome, repetierende Entladungen ohne elektrische Reizung; (3) Blockierung der γ-Impulse folgte während 5–15 min den antidromen Entladungen.

M. Kato and B. Fujimori

Department of Physiology, Hokkaido University, School of Medicine, Sapporo (Japan),
16th December 1966.

[1] S. Thesleff, Acta physiol. scand. *27*, suppl. 99 (1952).
[2] R. Granit, S. Skoglund and S. Thesleff, Acta physiol. scand. *28*, 134 (1953).
[3] B. Fujimori, T. Tokizane and E. Eldred, J. Neurophysiol. *22*, 165 (1959).
[4] B. Fujimori and E. Eldred, Am. J. Physiol. *200*, 699 (1961).
[5] W. A. Cook jr., D. R. Neilson jr. and J. M. Brookhart, J. Neurophysiol. *28*, 290 (1965).
[6] F. G. Standaert and J. E. Adams, J. Pharmacol. exp. Ther. *149*, 113 (1965).
[7] M. Kato and B. Fujimori, J. Pharmacol. exp. Ther. *149*, 124 (1965).
[8] J. C. Brinling and C. M. Smith, J. Pharmacol. exp. Ther. *129*, 56 (1960).
[9] D. Barker and M. C. Ip, J. Physiol. *177*, 27p (1965).
[10] C. M. Smith, A. Rev. Pharmac. *3*, 223 (1963).
[11] H. Klupp and O. Kraup, Archs int. Pharmacodyn. Thér. *98*, 340 (1954).
[12] Acknowledgments: The authors express their thanks to Dr. V. J. Wilson of The Rockefeller University for his valuable comments and improvement of the English.