CONNECTIONS BETWEEN THE NEURONS OF THE SYMPATHETIC GANGLION

(UDC 612.891 : 612.822]-019)

V. I. Skok

Laboratory of Electrophysiology (Head, Academician AN Ukr. SSR D. S. Vorontsov),
Institute of Physiology, Ukrainian SSR Academy of Sciences, Kiev
(Presented by Active Member AMN SSR A. V. Lebedinskii)

Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 61, No. 3,
pp. 12-17, March, 1966
Original article submitted July 20, 1964

In the sympathetic ganglia of mammals the process of synaptic transmission from preganglionic to postganglionic neurons has in the main been systematically studied. The connection between the neurons of the ganglion themselves have received little study, although histologists have repeatedly described different contacts between these neurons. Sensory nerve cells and complex networks of dendrites, connecting many neurons, are found in the ganglia [2], together with axon collaterals, the course of which has not been traced [2, 4]. The functions of these complex contacts are unknown, apart from the connections between the sensory neurons, which are the morphological basis for peripheral reflexes through the ganglia, described for the inferior mesenteric ganglion [1] and the solar plexus [3].

Some types of neurons have been found to be interconnected by axon collaterals and intermediate neurons—reciprocal inhibition. This inhibition is found in the motor neurons of the spinal cord [9, 12], the neurons of the cerebral cortex [11], and the neurons of the hippocampus [10]. A single antidromic excitation of the neurons of the sympathetic ganglion is known to be accompanied by strong depression of conduction of the orthodromic impulse through the ganglion, as a result of a lowering of the excitability of the neurons in the ganglion [6-8].

The object of this investigation was to determine if this lowering of excitability is entirely an after-effect or whether it takes place with the participation of synaptic mechanisms, as in the motor neurons of the spinal cord, with which the neurons of the sympathetic ganglia have much in common from an anatomical and physiological point of view.

EXPERIMENTAL METHOD

Experiments were carried out on the stellate ganglion of cats anesthetized with Nembutal (50 mg/kg). All the nerves leaving the left ganglion were divided to isolate it from central or peripheral influences, and the segments of the nerves remaining joined to the ganglion were placed on electrodes for stimulation and for recording the action potentials. The blood supply to the ganglion was left intact, and the body temperature of the animal and the temperature of the ganglion were maintained normal by warming the animal and the wet chamber in which the ganglion was placed. The technique of the operation and of recording the action potentials was described fully earlier [5, 6]. Stimulation was by means of rectangular pulses, 0.5 msec in duration, supplied by an electronic stimulator; after amplification, the action potentials were photographed from the screen of an electron-beam oscillograph.

If two cardiac nerves emerged from the ganglion, the effect of stimulation of each of them was investigated on the amplitude of the action potential of the other in response to a single stimulation of the preganglionic fibers of the lateral sympathetic trunk. If the cardiac nerve was single, but ramified some distance away from the ganglion, the same procedure was repeated with its branches. If the single cardiac nerve did not ramify, it was artificially split into two branches a short distance from the ganglion (10-15 mm). To test further, the fibers of these branches were excited during stimulation; the action potentials were recorded from the cardiac nerve between the ramification and the ganglion.

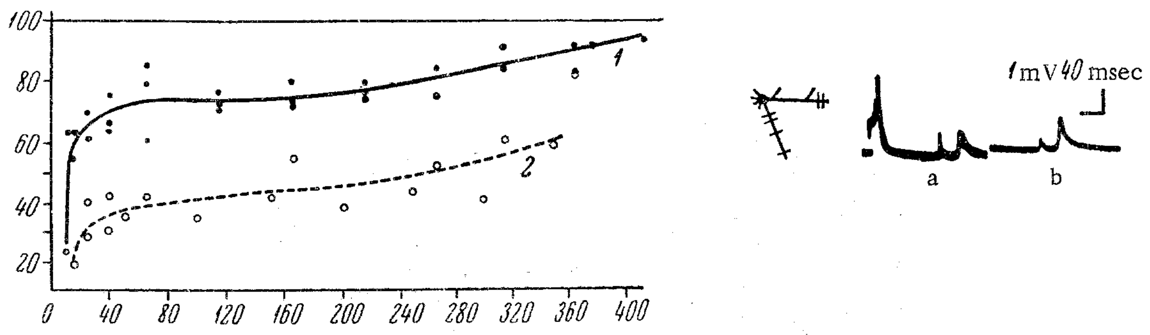

Fig. 1. Effect of an antidromic impulse on synaptic transmission in neurons of the ganglion which have been excited antidromically. Recording electrodes (here and in Figs. 2 and 3 the lines farthest apart) placed on the cardiac nerve, and the stimulating electrodes for antidromic excitation lie on the same nerve between these electrodes and the ganglion. The stimulating electrodes for orthodromic excitation are placed on the lateral trunk below the 3rd thoracic ramus communicans (the stimulating electrodes here and in Figs. 2 and 3 are shown by lines closest together). Along the axis of abscissas—interval between antidromic and orthodromic stimuli (in msec), along the axis of ordinates—amplitude of action potential of orthodromic impulse combined with antidromic (in % of amplitude of orthodromic impulse without preliminary antidromic). 1) Results of three experiments; 2) results of two experiments. From top to bottom—orthodromic impulse after antidromic, separated by interval of 115 msec (a) and orthodromic impulse without preliminary antidromic (b), obtained in one of the experiments.

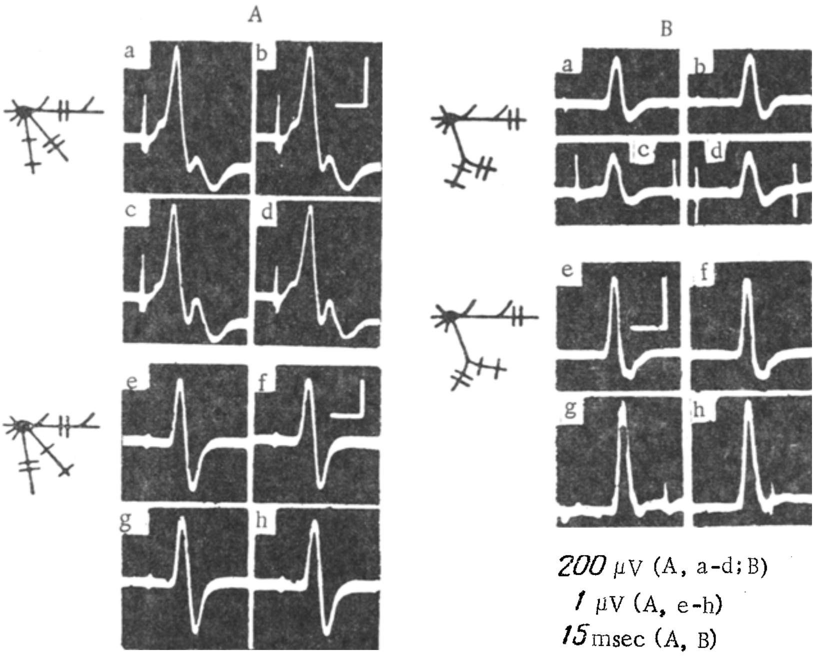

Fig. 2. Effect of antidromic impulses on synaptic transmission in neurons of a ganglion lying next to those excited antidromically. A) Antidromic impulse sent via the cardiac nerve next to that from which the recording is made; B) antidromic impulse sent via one of two branches, into which the cardiac nerve was artificially split, and the recording is made from the other: a) orthodromic impulse 100 msec after antidromic; b) orthodromic impulse without antidromic; c) orthodromic impulse against the background of repeated antidromic stimulation (frequency 15 cps in A and 25 cps in B); d) orthodromic impulse without repeated antidromic stimulation; e-h) the same as a-d (and in the same order), but with recordings made from the nerve of branch through which the antidromic impulses were sent in a-d. The scale of amplitude and time for a-d (A) is shown in b, and for 3-h (A) it is shown in f while for a-d (B) it is shown in e; the dimension of the scale lines is shown beneath the oscillograms. The stimulating electrodes for orthodromic stimulation are placed on the lateral trunk (in A between the 2nd and 3rd, and in B between the 3rd and 4th thoracic rami communicantes).

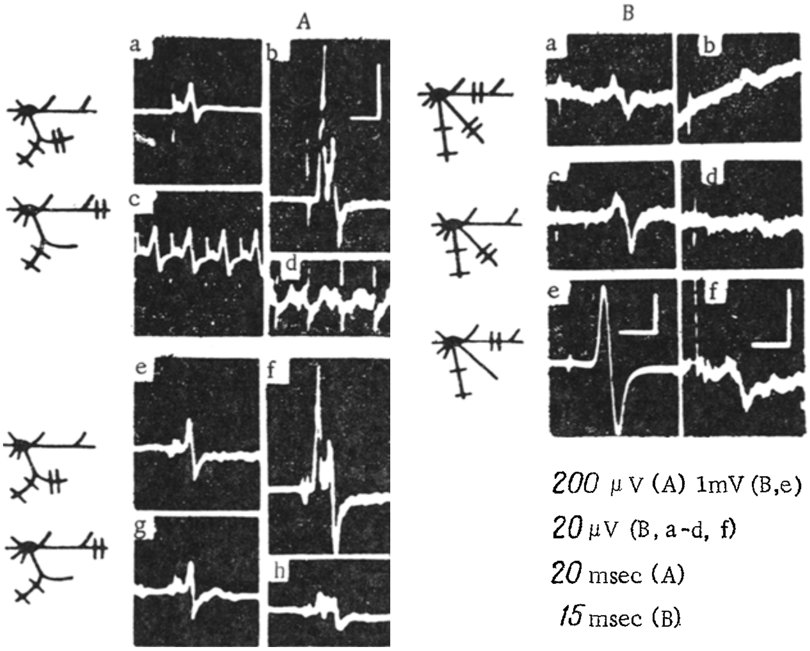

Fig. 3. Responses with a short (A) and a long (B) latent period in one branch of the cardiac nerve (or in one cardiac nerve) to stimulation of the other branch (or of the other nerve of the same ganglion) compared with responses in the same branch (or in the same nerve) to stimulation of the preganglionic fibers of the lateral trunk In A) Natural ramification of the cardiac nerve, in B) two cardiac nerves from one ganglion. A: a) response of one branch to a single; b) to repeated (frequency 50 cps) stimulation of the other; c) response of this same branch to a single; d) to repeated (at the same frequency as in b) stimulation of the preganglionic fibers of the lateral trunk between the 3rd and 4th thoracic rami communicantes; e) response of one branch to stimulation of the other before intravenous injection of diplacin (0.2 ml of a 2% solution per kg body weight); f) 20 sec after injection; g and h) the same as e and f, but during stimulation of the preganglionic fibers of the lateral trunk; B: a) response of one cardiac nerve to a single stimulation of the other cardiac nerve of this same ganglion; b) the same, but a 100 msec after a single stimumulation of the preganglionic fibers of the lateral trunk between the 2nd and 3rd communicantes; c) the same as in a before intravenous injection of diplacin in the same dose as in a; d) 2 min after injection; e and f) the same as c and d, but during stimulation of the preganglionic fibers of the lateral trunk between the 2nd and 3rd thoracic rami communicantes. Scale of amplitude and time for A is given in c and for B (except for B, e) is given in f; dimension of the scale lines is indicated beneath the oscillograms.

EXPERIMENTAL RESULTS

Altogether seven experiments were carried out, in four of which the cardiac nerve was artificially split into two parts, one was performed on two cardiac nerves, and in two the cardiac nerve branched naturally (the two last types of structure are rare). The intervals between the antidromic and the orthodromic stimulus were of 60 and 100 msec; during these intervals the antidromic impulse in the nerve from which the recording was made caused depression of the amplitude of the response to the orthodromic stimulus by 30-60% (Fig. 1). In the case of antidromic stimulation through the neighboring nerve or through the neighboring branch, no consistent changes took placed in the amplitude of the response to the orthodromic stimulus outside the limits of the insignificant spontaneous variations, as shown in Fig. 2.

According to Renshaw [12], during reciprocal inhibition the conduction of the orthodromic impulse in the spinal cord may cease almost completely. In the sympathetic ganglion, on the other hand, in the influence of antidromic stimulation on synaptic transmission in the nerve next to that stimulated was absent; it likewise was not found when the orthodromic stimulus was applied against the background of repeated antidromic stimuli (frequency 15-25 cps; Fig. 2) or it was weakened. Hence, a prolonged (up to 400 msec) lowering of the excitability of the neurons of the ganglion, accompanying their antidromic excitation (Fig. 1) was not reciprocal inhibition like the inhibition of the spinal cord, but was evidently an after-effect of subnormality of the neurons. The reciprocal synaptic connections of each neuron which possesses them involve too few of the neighboring neurons to be revealed by the onset of inhibition in these neurons or by facilitation against the background of the excitation of all the surrounding neurons.

It is interesting that stimulation of one of the two branches (or one of the nerves) caused responses in the other branch (or in the other nerve). The amplitude of these responses was much smaller than the amplitude of the response to stimulation of the preganglionic fibers in the same branch. From their latent period, these responses may be divided into two groups—with a short and a long latent period. The former (3-4 msec, Fig. 3A, a) were observed in nearly all the experiments. Their threshold either corresponded to the threshold of excitation of the postganglionic fibers of the stimulated branch or it was slightly below it. The lability of these responses was above 25 cps, and in some cases it was above 50 cps (Fig. 3A, a, b) ,which is much higher than the lability of the synapses of the ganglion, which does not exceed 25 cps (Fig. 3A, c, d). If such responses were found in one of the branches, they were also observed in the other, i.e., excitation was conducted in both directions. Diplacin—a curare-like substance—had no effect on these responses (Fig. 3A, e, f) whereas the synaptic transmission from the preganglionic fibers to the postganglionic fibers of that particular branch was suppressed (Fig. 3A, g, h).

These facts show that responses with a short latent period are the result of direct stimulation of those fibers from which the recording was made. Evidently few of the postganglionic fibers ramify, and the branches of the same fiber are found in different branches of the nerve (postganglionic axon reflex).

In the case of the responses with a long latent period (15-18 msec) the lability was always below 25 cps, and sometimes below 10 cps, while the threshold of the production of these responses was much lower than the threshold of excitation of the postganglionic fibers of the stimulated branch. The amplitude of these responses was less than the amplitude of the responses of the first group. The number of responses with a long latent period fell sharply if they were observed immediately after synaptic transmission from the preganglionic fibers to the postganglionic fibers of the branch lying on the recording electrodes (Fig. 3B, a, b). Diplacin suppressed these responses just like the responses to stimulation of the preganglionic fibers (Fig. 3B, c-f). In most cases responses with a long latent period were observed only in one of the branches.

Because of these differences, the responses with a long latent period may be regarded as the result of synaptic transmission in the ganglion. This may be either in the form of peripheral reflexes or of axon reflexes (preganglionic). Further experiments are required to explain the mechanism of these reflexes.

LITERATURE CITED

1. I. A. Bulygin, E. I. Balakhina, and M. P Kul'vanovskii, Fiziol. zh. SSSR, No. 9, 1096 (1961).
2. A. S. Dogiel, Arch. mikr. Anat., Bd. 46, S. 305 (1895).
3. O. N. Zonyatima, Fiziol. zh. SSSR, No. 6, 687 (1961).
4. S. Mikhailov, Nevrol. vestn. 16, 2, 320 (1909).
5. V. I. Skok, In book: Physiological Collection [in Russian], No. 10, Kiev (1957), p. 203.
6. V. I. Skok, Electrical Reactions of the Stellate Sympathetic Ganglion of the Cat. Author's abstract of candidate dissertation, Kiev (1960).
7. V. I. Skok, Fiziol. zh. (Ukr). No. 2, 155 (1959).
8. C. Eccles, Physiol. (London), 88 (1937), p. 1.
9. J. C. Eccles, P. Fatt, and K. Koketsu, Ibid., 126 (1954), p. 524.
10. E. R. Kandel, W. A. Spencer, and F. J. Brinley, J. Neurophysiol. 24 (1961), p. 225.
11. C. G. Phillips, Quartz. J. exp.Physiol., 41 (1956), p. 58.
12. B. Renshaw, J. Neurophysiol., 4 (1941), p. 167.