SYMPATHETIC ORIGIN OF SPONTANEOUS IMPULSES RECORDED IN A MIXED NERVE OF THE FROG

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Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 55, No. 5,
pp. 5-8, May, 1963
Original article submitted July 5, 1962

Spontaneous efferent impulses, i.e., those not caused by obvious stimuli, have previously been described in nerves supplying the skeletal muscles of cold-blooded animals [3]. These impulses approached the threshold level for the muscle. Their rhythm was related to the activity of the respiratory center and of the reticular formation of the brain stem and hindbrain. Section of the brain below the medulla led to disappearance of this pulsed activity. It was subsequently shown that division of the rami communicantes to the 7th, 8th, and 9th pairs of spinal nerves leads to disappearance of the impulses. The sympathetic nature of this pulsed efferent activity has been postulated.

The shape and characteristics of the potentials recorded from a motor nerve in cold-blooded animals during stimulation of the sympathetic chain have been studied [4]. Many workers have examined the spontaneous efferent pulsed activity in nerves to internal organs – the heart, kidneys, thyroid, etc. [5, 6, 7, 8].

The precise localization of the efferent sympathetic pathways in the part of the reticular formation lying in the brain stem has been established [10, 14]. The efferent pathways from the hypothalamus to the various autonomic systems occupy a large zone in the lateral part of the reticular formation. Adrenergic structures have been found [9] in the tegmental portion of the reticular formation. Neurons similar to sympathetic have been demonstrated morphologically in the reticular formation [11]. Marked changes in the functional properties have been found in the tectum of the optic lobes of sympathectomized frogs [2].

Working on the assumption that the spontaneous efferent pulsed activity originates in the sympathetic elements of the reticular formation, we investigated the action of chlorpromazine, which blocks the adrenergic components of the reticular substance [9, 13]. We took into consideration the fact that the effect of chlorpromazine on the intact animal is complicated by the mutual interaction between the different regions of the central nervous system, disturbed by the action of the drug. Nevertheless, the fact that the results followed a uniform pattern justified the drawing of certain conclusions from this series of experiments.

EXPERIMENTAL METHOD

Experiments were conducted on intact frogs. The knee and ankle were securely fixed. Leads were taken from silver electrodes, 3 mm apart, on the cutaneous and muscular branches of the tibial nerve (the membranes having been removed). The nerve was kept moist on the electrodes and the blood supply to the muscle was not disturbed. Chlorpromazine was injected into a cutaneous vein or the femoral vein under local anesthesia. The animal was kept in a screened chamber. A two-channel amplifier with a symmetrical input was used; the sensitivity of the apparatus was 1 μV to 1 mm. The transmission band was 100-1000 cps. Recordings were made by means of a type MPO-2 loop oscillograph. An integrator was used in some of the experiments. In one series of experiments the somatic branches of the 7th, 8th, and 9th pairs of spinal nerves were divided unilaterally above the point of entry of their rami communicantes.

EXPERIMENTAL RESULTS

Altogether 33 experiments were carried out on autumn, winter, and spring frogs of the species Rana temporaria. The pulsed activity from the combined cutaneous and muscular branches of the tibial nerve was recorded. Its central origin was proved by dividing the mixed nerve distally. When leads were taken from the intact nerve, afferent activity was also frequently recorded in the form of fast (2 millisec) biphasic potentials. These afferent impulses became weaker, probably as the limb receptors became adapted. The efferent impulses (Fig. 1) took the form of volleys with

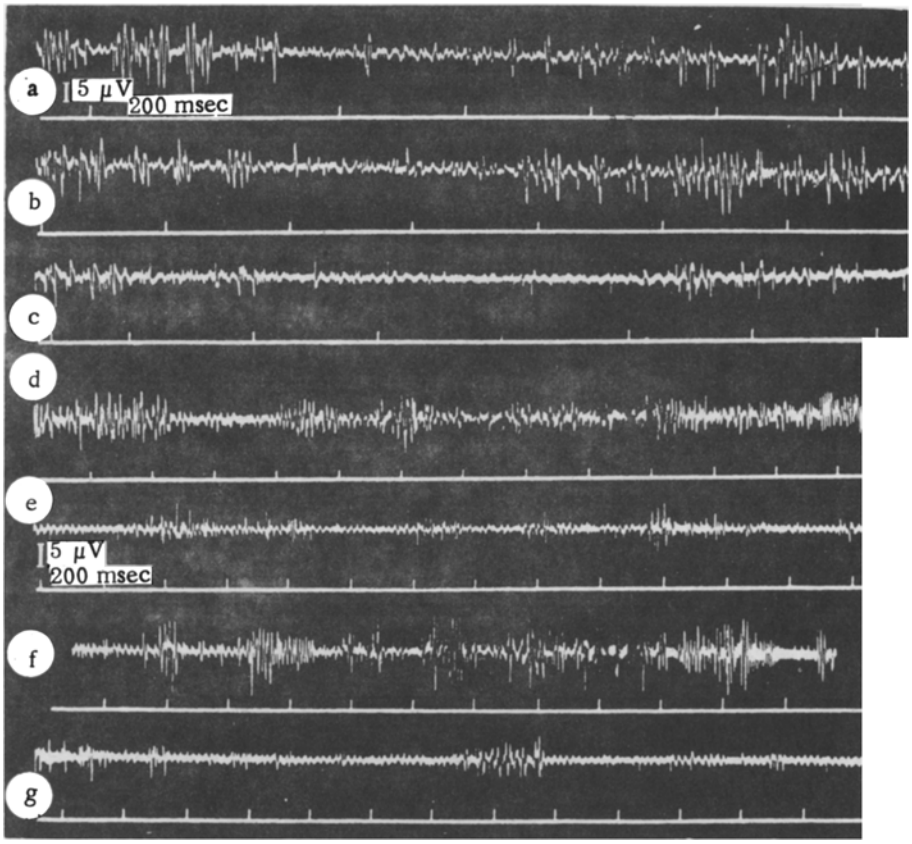

Fig.1. Changes in volleys of impulses from the combined cutaneous and muscular branches of the tibial nerve of Rana temporaria. a and d) Initial levels of spontaneous activity in experiments on February 21 and March 5, 1962; b) after division of the somatic nerves; c) 3 min after injection of 0.08 mg chlorpromazine; e, g) 4 and 50 min after injection of 0.1 mg chlorpromazine; f) increased strength of efferent activity before a generalized movement of the animal, 40 min after injection of chlorpromazine.

a frequency of 4-6/sec: each volley consisted of 6-13 impulses varying in duration (5-10 millisec) and amplitude (4-20 μV). Before the animal made a generalized movement the impulses became continuous and their amplitude increased sharply [3, 4].

The doses of chlorpromazine used were first tested for their action on the respiration and cardiac activity of the frog [2] in a 0.5-1.0% solution. Small doses of chlorpromazine (0.005-0.01 mg/frog or 0.1-0.2 mg/kg body weight) had a slight stimulating effect in only one of seven experiments, and otherwise no effect could be observed or it caused a slight depression of the amplitude of the volleys. It is interesting to note that the motor activity of the animals increased. The changes in the volleys of impulses after injection of a small dose of chlorpromazine are shown in Fig. 2 (A and C). Biphasic changes in activity were frequently seen. The graph shows that the amplitude was depressed during the first 5 min, after which it returned to its original level.

Moderate doses of chlorpromazine (0.08-0.1 mg) had a marked depressing action. A dose of 0.08 mg caused an immediate fall in the amplitude of the potentials (Fig. 1, a, c), although the frequency of the succession of volleys was not changed during the first 5-10 min. If the animal did not move at this period the volleys of efferent spontaneous activity became less frequent, and each volley itself contained fewer impulses and became shorter (Fig. 1, d, e, g). It may therefore be concluded that chlorpromazine caused depression of some of the cells of the adrenergic component of the reticular formation, causing an immediate fall in the amplitude of the potentials in the volley. The intensification of the effect of chlorpromazine, causing (as our previous observations showed) a slowing of respiration after 5-10 min, modified the frequency of the volleys of spontaneous activity. It is an interesting fact that, even when

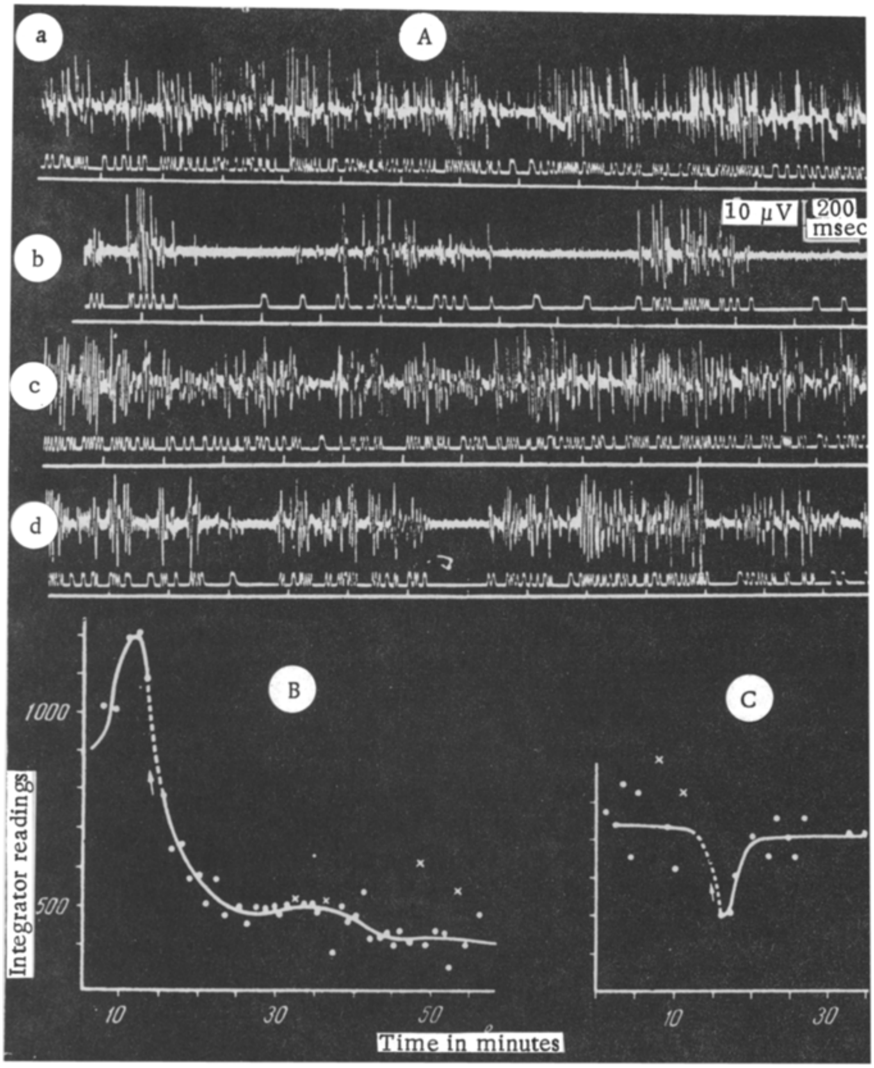

Fig. 2. Oscillogram (A) and graph of the changes in the volleys of impulses after injection of 0.1 mg (B) and 0.006 mg (C) of chlorpromazine. a) Initial level of spontaneous activity of intact nerve; b) 1 min; c) 8 min; and d) 21 min after injection of 0.006 mg chlorpromazine (0.1 mg/kg body weight). Significance of curves for each cut (from above down): spontaneous volleys; readings of integrator; time marker (200 millisec). The arrow on the graphs indicates the time of injection of chlorpromazine. A cross denotes movement of the animal. Broken line – no measurements taken (during injection).

activity was depressed to this considerable degree, a movement of the animal in an attempt to free itself caused a transient (10-15 sec) burst of volleys of almost the same magnitude as initially, i.e., before administration of chlorpromazine (Fig. 1, f). When the animal completed its movement the volleys at once ceased. The amplitude of the potentials fell to 4-5 μV. This activity was not restored.

Doses of 0.1-0.125 mg caused a greater and, in the spring frogs, a more rapid weakening of the activity. In some experiments the volleys of spontaneous activity disappeared immediately after the injection of chlorpromazine, and reappeared only before the animal started to move, which happened more and more rarely (Fig. 2, B).

Hence, the depressing effect of chlorpromazine on the efferent spontaneous volleys of impulses, a manifestation of its inhibiting action on the autonomic components of the reticular formation, confirmed the suggestion [4] that the central apparatuses generating this activity are sympathetic in nature.

SUMMARY

Aminazine depression of "spontaneous" efferent discharge activity as a manifestation of its inhibitory effect on the vegetative components of reticular formation confirmed the assumption of M. V. Kirzon and others on the fact that the central apparatuses provoking such discharge activity were of sympathetic nature.

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