DISTURBANCE OF γ-MOTONEURON ACTIVITY IN EXPERIMENTAL THYROTOXICOSIS

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Activity of γ-motoneurons was investigated in anesthetized rats after oral administration of thyroid extract for 3 weeks. In experimental thyrotoxicosis the increase in the number of silent filaments isolated from the nerve to the gastrocnemius muscle was accompanied by an increase in firing rate of flexor and extensor γ-motoneurons. High frequencies, not characteristic of γ-motoneurons of intact animals, were recorded in the flexor γ-motoneurons of the animals receiving thyroid.

KEY WORDS: *γ-motoneurons; thyrotoxicosis.*

Experimental thyrotoxicosis leads to changes in the functional state of neuromuscular transmission [3] and in conduction in the central segment of the somatic reflex arc, and to disturbance of all types of segmental inhibition [4] and of the supraspinal regulation of spinal reflex activity [1, 2]. In the modern view, the γ-system plays a role of special importance in the mechanisms of integration of motor activity [7, 16, 17].

It was therefore decided to study the effect of hyperthyroidism on γ-motoneuron activity.

EXPERIMENTAL METHOD

Albino rats weighing 150-300 g were anesthetized with pentobarbital (5 mg/100 g body weight). Experimental thyrotoxicosis was produced by the scheme described previously [3]. Recordings were taken from thin bundles of fibers isolated from the ventral roots or motor nerves (n. gastrocnemius, n. peroneus profundus) at their point of entry into the muscle. Filaments with a minimal (not more than 1-3) number of axons of γ-motoneurons were investigated (Fig. 1). The filaments were dissected with the OM-148 operating microscope in a bath of mineral oil at 37°C.

Platinum recording electrodes were connected to the amplification unit of a Medicor-4M myograph and activity was then recorded on the camera of an oscilloscope with S1-29 memory unit. Spikes were counted in an area of stable frequency and 500 msec in duration (sometimes in shorter intervals). In some experiments the F-552A frequency meter-chronometer was used to count the frequency of spike activity.

EXPERIMENTAL RESULTS

During investigation of the activity of extensor γ-motoneurons in the control animals and flexor γ-motoneurons in both groups, no activity could be found in about 6-9% of filaments isolated for testing. In the animals with thyrotoxicosis, when fibers isolated from n. gastrocnemius were tested, the number of these silent filaments was 23%.

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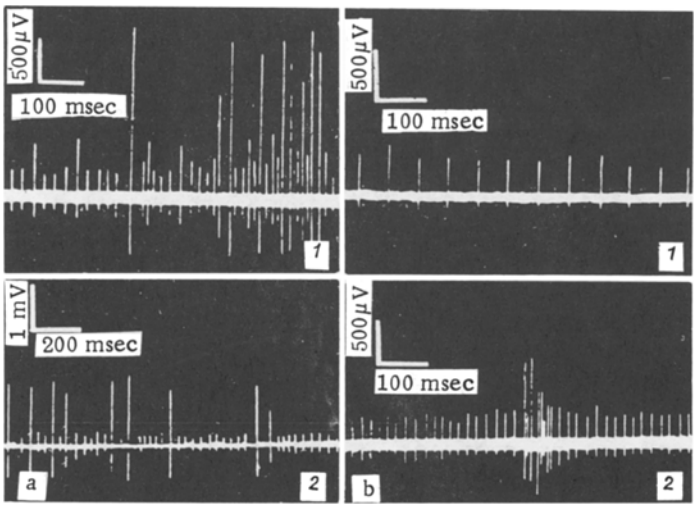

Fig. 1. Efferent activity recorded from flexor and extensor nerves: a) γ-activity of fiber isolated from deep peroneal nerve; 1) control animal (discharge of two γ-fibers and single discharges of α-motoneuron); 2) animal with thyrotoxicosis (discharge in one γ-fiber and one α-fiber); b) γ-activity of fiber isolated from nerve to gastrocnemius; 1) control animal (regular discharge in single γ-fiber); 2) animal with thyrotoxicosis (discharge in one γ-fiber and one α-fiber).

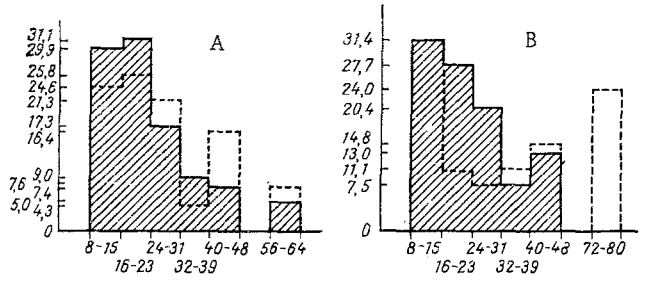

Fig. 2. Histogram of discharge frequency in extensor (A) and flexor (B) γ-motoneurons in animals of control group (continuous line) and animals with experimental thyrotoxicosis (broken line). Abscissa, discharge frequency (spikes/sec); ordinate, percent of total number of fibers tested with particular frequency.

A histogram of the discharge frequency in axons of extensor γ-motoneurons in control animals (121 axons) and animals with thyrotoxicosis (89 axons) is illustrated in Fig. 2A. The distribution of fibers by discharge frequency in the animals of both groups has a maximum in the region of lower frequencies at 8-31 spikes/sec (78.6% of the total number of fibers investigated in animals of the control group and 71.1% in animals with thyrotoxicosis). High frequencies of 40-64 spikes/sec were discovered in 13.4% of fibers in the intact animals and 24% of fibers in the animals receiving thyroid extract.

Despite the fact that the animals of both groups shared a common rule for the distribution of fibers by range of frequencies, in the animals with thyrotoxicosis the discharge frequency was a little higher, although in both cases fibers with both lower and higher frequencies were found, although only in different relative percentages.

The histogram of discharge frequency of the flexor γ-motoneurons for animals of both groups is shown in Fig. 2B. In the control animals 79.5% of fibers showed frequencies of 8-31, and 20.5% frequencies of 32-48 spikes/sec. In the animals with thy-

rotoxicosis the number of fibers with a frequency of 8-15 spikes/sec was the same as in the control (31.4%). The sharp decrease in the relative proportion of fibers with frequencies of 16-31 spikes/sec and the increase in the number of axons of flexor γ-motoneurons in which frequencies of 32-48 spikes/sec were recorded will be noted. A characteristic feature of γ-motoneurons of flexor muscles of the animals receiving thyroid was the appearance of a frequency of 72-80 spikes/sec in 24% of axons tested, although this frequency could not be found in any of the control experiments.

Hyperthyroidism thus leads to a marked redistribution of fibers of flexor γ-motoneurons with respect to their frequency range toward higher frequencies, and to the appearance of high frequencies not characteristic of γ-motoneurons of intact animals.

In the modern view, one result of the consecutive and many-faceted action of thyroid hormones on energy metabolism is depolarization of excitable structures [4, 5, 15]. Depolarization leads to an increase in the spontaneous firing rate of the neuron [6, 9], and this may explain the increased spontaneous firing rate of γ-motoneurons in thyrotoxicosis.

A definite role in the genesis of the disturbances of γ-activity in thyrotoxicosis may also be played by the state of the mechanisms of descending control. This suggestion is supported by the fact that the functional state of the γ-loop is entirely dependent on modulatory influences from the basic motor centers of the brain [7, 8, 10-14, 16-18] and the selective disturbance of descending functions of these centers under the influence of an excess of thyroid hormones [1, 2].

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