LOCALIZATION OF DAMAGE IN THE FELINE INFERIOR MESENTERIC GANGLION CAUSED BY THYROID HYPOFUNCTION

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In studying the transmission of excitation through sympathetic ganglia it is important to know what part of the apparatus of the ganglion is first destroyed as a result of various pathological or pharmacological influences. The disturbance of interneuronal connections may be related to changes in the region of the presynaptic terminations, or with disturbance of formation, liberation, or destruction of mediators, or again with changes in excitability of the n-cholinergic structures and with a change in conduction along postsynaptic neurones.

We have studied the influence of thyroid extirpation and hypothyroidism on the conduction of excitation through presynaptic endings and postsynaptic neurones, as well as effects on excitability of n-cholinergic structures of the inferior mesenteric ganglion.

EXPERIMENTAL METHOD

The work was carried out on cats anaesthetized with 40 mg/kg chloralose or 400 mg/kg urethane, and on spinal cats. Stimulation of the preganglionic sympathetic trunk and the collection of action potentials from postganglionic fibers was carried out by the method described by Lloyd [8].

Thyroid function (I series of experiments) by complete extirpation of the gland, and hypothyroidism (II series of experiments) was produced by a daily injection of 250-270 mg/kg of 6-methylthiouracil. In both cases the animals were used for an acute experiment when the basal metabolic rate had fallen by 30-33%.

In the III series of experiments compensation for thyroid hypofunction was brought about by a subcutaneous injection of I-thyroxin sufficient to restore the initial metabolic rate. In addition a control series of experiments was carried out on 32 normal cats serving as controls.

To study the conduction of excitation through the presynaptic terminations we used the phenomenon of postactivational facilitation [2-4].

The functional condition of the n-cholinergic structures was inferred from their sensitivity to a dose of 30 μg acetylcholine, which we used as standard; it was injected subcutaneously into the artery supplying the ganglion. Disturbances observed in the postsynaptic neurones were demonstrated by antidromic excitation [2]. Action currents from the body of the ganglion were picked up by bipolar needle electrodes.

EXPERIMENTAL RESULTS

Under the influence of considerable functional changes in the thyroid (complete lack or hypothyroidism) the indices representative of interneuronal conduction all changed in the same direction; there was a reduction of excitability and lability, an increase in the time of interneuronal conduction, etc.

In normal cats the amplitude of the response to a single stimulus given 1-2 sec after tetanization of the preganglionic trunk was 10-45% higher than the response obtained before tetanization.

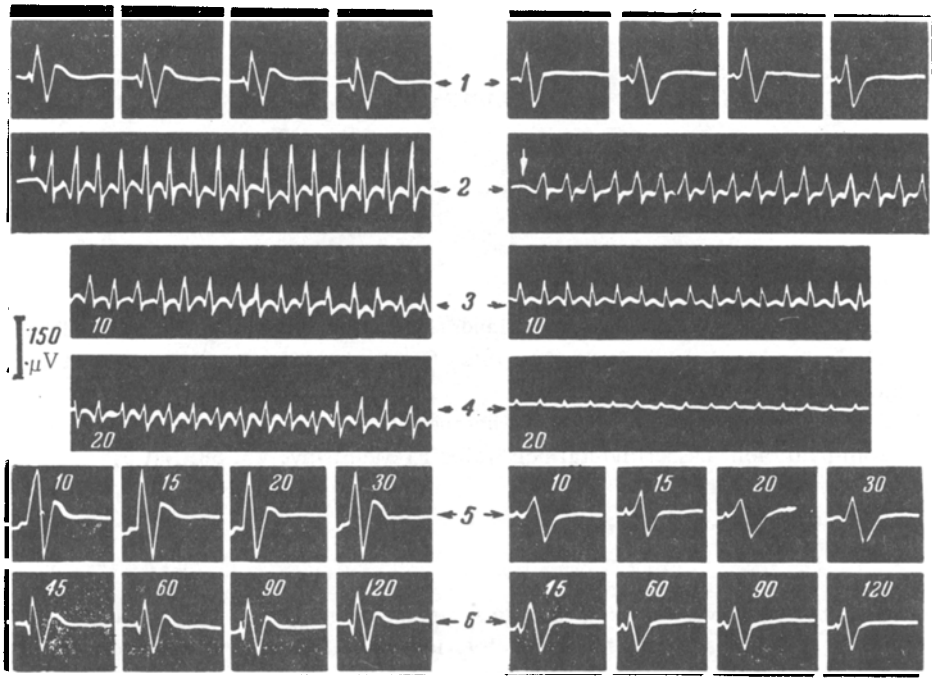

Fig. 1. Influence of thyroidectomy on post-activational facilitation in the feline inferior mesenteric ganglion. Potentials from postganglionic fibers (A) before, and (B) after thyroidectomy: 1) Before tetanus (stimulation of the preganglionic trunk with single stimuli at 5 sec intervals); 2, 3, 4) tetanus (onset of tetanus is indicated by vertical arrow); 5, 6) after tetanization; single stimuli applied to the preganglionic trunk (the figures indicate the time interval after the end of tetanization, in seconds).

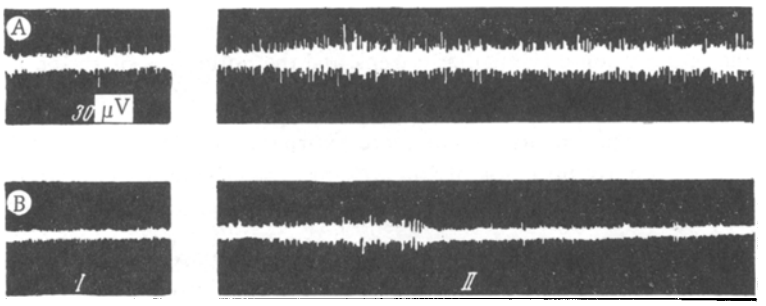

Fig. 2. Influence of thyroidectomy on the excitability of n-cholinergic structures (electrical activity in the postganglionic fibers). I) Before injection of acetylcholine; II) after intra arterial injection of 30 μg acetyl choline; A) normal cat; B) thyroidectomized cat.

The oscillograms shown in Fig. 1 demonstrate that in the thyroidectomized animals the phenomenon of post-activational facilitation, which is present in normal cats, was to a large extent suppressed.

Similar results were obtained also on cats which had been treated with 6-methylthiouracil.

The results of our experiments to study localization of disturbance in the sympathetic ganglia related to complete or partial thyroid deficiency indicated that very important changes developed in the terminations of the presynaptic fibers. The mechanism of the reduction of excitability of the preganglionic nerve fibers is not quite clear. Possibly in these cases not only is there a disturbance of metabolism in the presynaptic terminations but changes in physical properties associated with post activational facilitation in normal animals proceed differently [1, 4-7, 9].

In addition, in this condition we were able to observe disturbance of the functional condition of the n-cholinergic structures (Fig. 2). Whereas in normal cats injection of acetylcholine into the artery supplying the ganglion was associated with a prolonged stimulant action (increase both of frequency and amplitude of impulses), in thyroidectomized animals acetylcholine produced no appreciable influence on the electrical activity of the postganglionic fibers.

We found no important changes in the postganglionic neurones. The potentials in the ganglia of the thyroidectomized cats were of the same amplitude as in normal animals.

In animals in which the thyroid deficiency had been compensated no disturbances developed in the different structures of the inferior mesenteric ganglion, whether the initial condition had been one of complete or partial thyroid deficiency.

The results we have obtained show that partial or complete thyroid lack interferes with the transmission of excitation in the inferior mesenteric ganglion. This disturbance results from changes in the functional condition of the preganglionic terminations and of the n-cholinergic structures, whereas conduction of excitation along the postganglionic nerve fibers is not affected.

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