

THE EFFECT OF DENERVATION ON THE MORPHOLOGY AND FUNCTIONAL ACTIVITY OF THE THYROID GLAND

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In modern neuroendocrinology, increasing attention is being paid to the study of the transhypophyseal mechanism of thyroid function regulation. Meanwhile, the significance of the parahypophyseal mechanism has not been sufficiently investigated. A frequently used method for studying the role of the autonomic nervous system in regulating the functional activity of the thyroid gland is the extirpation of the cervical sympathetic ganglia, the nerves of which innervate not only the thyroid gland but also many other vital organs. It is possible that this methodological approach has led to the emergence of conflicting data, demonstrating the ambiguity and uncertainty of the autonomic nervous system's influence on thyroid function.

The aim of our study was to determine the extent to which the autonomic innervation of the thyroid gland contributes to the regulation of its secretory function and morphological structure.

The studies were conducted on male and female white laboratory rats. Denervation of the gland was performed using our own methodological approach, which does not compromise the integrity of the superior cervical sympathetic ganglia. The results showed that the secretory activity of the thyroid gland changed only slightly 1–4 days after denervation, indicating the predominant influence of the transhypophyseal regulatory mechanism in animals of both sexes. However, 120 days after denervation of the thyroid gland, there was a significant reduction in its functional activity, characteristic of hypothyroidism. This was evidenced by histological findings revealing a lack of colloid resorption, a reduction in the height of the secretory cells and the diameter of their nuclei, as well as a decrease in the concentration of thyroid hormones in the blood. It was established that prolonged denervation leads to the development of hypothyroidism, which is more pronounced in females, indicating the importance of the parahypophyseal regulatory mechanism in maintaining normal thyroid status.

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