EFFECT OF THYROID HORMONES ON ASCORBIC ACID DISTRIBUTION IN THE BODY DURING HYPOTHERMIA

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The development of experimental hyper- and hypothyroidism in rats leads to a decrease in the vitamin C content in several organs. Artificial hypothermia (mild and deep), induced against the background of hyper- and hypothyroidism, is accompanied by a decrease in the vitamin C concentration in the tissues, and the degree of this decrease in the ascorbic acid content in the organs bears a definite relationship to the level of saturation of the body with thyroid hormones.

It is considered [10, 17] that a change in the content of ascorbic acid in the organs and tissues can be used as an objective criterion reflecting the depth and character of metabolic changes under extremal conditions. At the same time, evidence has been obtained that the state of thyroid function affects the distribution of vitamin C in the body [3-5, 6, 15, 16]. The writers have shown [7] that mild and, in particular, deep hypothermia in albino rats leads to a considerable disturbance of the vitamin C topography, and that the development of deep hypothermia is accompanied by inhibition of the hormone-producing activity of the thyroid tissue [8].

The object of the present investigation was to study the distribution of ascorbic acid in hypothermic animals in relation to the level of thyroid hormone saturation.

EXPERIMENTAL METHOD

Experimental hyperthyroidism was produced in albino rats by feeding them with 0.1 g dry thyroid daily, and hypothyroidism was produced by subcutaneous injection of a suspension of 6-methylthiouracil

TABLE 1. Changes in Ascorbic Acid (mg%) in Tissues and Blood Plasma of Albino Rats ($M \pm m$)

Group of animals	Adrenals	Brain	Heart	Liver	Skeletal muscle	Blood plasma
2-(Hyperthyroidism)	$328,2\pm10,4$ $217,6\pm12,36$ $314,9\pm12,76$	$28,6 \pm 1,20$	$7,6\pm 0,37$	21.1 ± 1.17	7.8 ± 0.58	0.79 ± 0.05
4-(Hyperthyroidism+ mild hypothermia)		27,7±1,58	6,8±0,37	15,6±0,91	5,9±0,25	2,4±0,16
5-(Hypothyroidism+ mild hypothermia)	239,7±12,83	29,8±1,78	8,3±0,63	20,2±1,10	6,8±0,42	1,75±0,09
6-(Hyperthyroidism + deep hypothermia)	231,5±10,9	28,2±1,94	8,5±0,53	18,3±0,97	7,0±0,38	3,62±0,28
7-(Hypothyroidism+ deep hypothermia)	214,8±8,56	26,6±1,57	6,8±0,41	14,6±1,0	6,4±0,47	4,03±0,32

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twice a day (total daily dose 20 mg). These substances were administered for 8-10 days [11]. Artificial hypothermia was produced by cooling the animals in hermetically closed cold chambers [19] until their rectal temperature was 32-29°C (mild hypothermia) or 22-19°C (deep hypothermia).

The ascorbic acid concentration in the tissues was determined by the dichlorophenolindophenol method [1]. The vitamin C topography was investigated in albino rats divided into the following experimental groups (16 animals in each group): 1) control, 2) hyperthyroidism, 3) hypothyroidism, 4) hyperthyroidism + mild hypothermia, 5) hypothyroidism + mild hypothermia, 6) hyperthyroidism + deep hypothermia, and 7) hypothyroidism + deep hypothermia.

EXPERIMENTAL RESULTS

In the animals with hyperthyroidism the ascorbic acid concentration in the tissues was significantly reduced (Table 1), evidently as the result of activation of oxidation-reduction processes and increased consumption of ascorbic acid [14]. In addition, saturation with thyroid hormones leads to ascorbic acid mobilization from the organs [3, 12, 15].

In hypothyroidism a significant decrease in the ascorbic acid content was found in the heart, liver, and skeletal muscle. According to some investigators [5, 14], this decrease is due to metabolic changes in the body leading to a disturbance of the ability of the tissue cells to assimilate and utilize ascorbic acid.

Mild hypothermia in hyperthyroid rats was accompanied by marked decrease in the ascorbic acidlevel in the tissues of several organs and by a marked (by 100%) increase in its level in the blood plasma, while in hypothyroid animals the changes in the vitamin C topography were similar but less marked.

Deep hypothermia against the background of hyperthyroidism was accompanied by a decrease in the vitamin C concentration in the tissues and by an increase (by 3 times) in its level in the blood plasma. Cooling the hypothyroid animals to a rectal temperature of $22-19^{\circ}$ C led to similar changes in the vitamin C balance, but in some organs (heart, liver) they were more severe than during deep hypothermia against the background of hyperthyroidism (P < 0.05).

Consequently, deep hypothermia in hyperthyroid animals produces less marked biochemical disturbances in the tissues than in animals receiving the thyrostatic agent. This is in full agreement with results indicating that in fully developed deep hypothermia the functional activity of the thyroid tissue is depressed [2, 6, 9, 13, 18, 20].

These results thus indicate the possibility of hormonal influences of the thyroid gland under development of changes in the vitamin C topography during hypothermia.

LITERATURE CITED

- 1. V. S. Asatiani, Methods of Biochemical Research [in Russian], Moscow (1956).
- 2. R. T. Boiko, in: Collected Scientific Transactions of Dnepropetrovsk Medical Institute [in Russian], Vol. 19, Part 2, Dnepropetrovsk (1961), p. 231.
- 3. S. M. Bremener, Vitamins and Their Clinical Application [in Russian], Moscow (1966).
- 4. A. Sh. Byshevskii, in: Problems in General and Special Hygiene [in Russian], Kiev (1963), p. 296.
- 5. Z. Ya. Dolgova, Abstracts of Proceedings of the 5th Scientific Conference of Semipalatinsk Medical Institute [in Russian], Semipalatinsk (1962), p. 30.
- 6. Z. Ya. Dolgova and E. G. Dolgov, Vopr. Med. Khimii, No. 6, 21 (1965).
- 7. Z. Ya. Dolgova and B. V. Karatysh, Trudy Semipalatinsk Med. Inst., 5, 55 (1969).
- 8. Z. Ya. Dolgova and V. A. Glumova, Byull. Éksperim. Biol. i Med., No. 8, 39 (1970).
- 9. M. G. Kolomiitseva and I. I. Neimark, Goiter and Its Prevention [in Russian], Moscow (1963), p. 16.
- 10. B. A. Lavrov, Abstracts of Proceedings of the 4th All-Union Conference on Vitamins [in Russian], Moscow (1957), p. 26.
- 11. N. V. Lazarev (Editor), Reproduction of Diseases in Animals for Experimental Therapeutic Research [in Russian], Leningrad (1954).
- 12. S. M. Leites and N. N. Lapteva, Outlines of Pathophysiology of Metabolism and the Endocrine System [in Russian], Moscow (1967), p. 328.
- 13. A. I. Makkaveeva, in: Collected Transactions of the Republic Research Laboratory, Ministry of Health of the Belorussian SSR [in Russian], No. 1, Minsk (1957), p. 107.
- 14. M. F. Merezhinskii, in: Fundamentals of Endocrinology [in Russian], Minsk (1963), p. 80.

- 15. S. M. Ryss, Vitamins [in Russian], Leningrad (1963).
- 16. G. S. Sheves, Biokhimiya, 23, 80 (1958).
- 17. B. I. Yanovskaya, Uspekhi Sovr. Biol., 56, No. 1, 3 (1963).
- 18. W. G. Bigelow and S. Sidlofski, Brit. Med. Bull., 17, 56 (1961).
- 19. L. Giaja and R. Andjus, C. R. Acad. Sci. (Paris), 229, 1170 (1949).
- 20. C. Kayser, Ann. Rev. Physiol., 19, 32 (1957).