

THE THYROID FUNCTION IN COMPENSATORY HYPERTROPHY OF THE KIDNEYS AND OVARIES

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Translated from *Byulleten' Éksperimental'noi Biologii i Meditsiny*,

Vol. 53, No. 6, pp. 19-23, June 1962

Original article submitted June 13, 1961

Several workers have shown [1-6] that extirpation of the thyroid gland causes inhibition of compensatory hypertrophy of the kidneys and that the administration of thyroidin stimulates such hypertrophy. Sh. M. Chikvashvili has observed a similar relationship between the thyroid gland and compensatory hypertrophy of the ovaries: removal of the thyroid gland depresses and thyroidin stimulates hypertrophy.

In continuing the study of the regulatory relationships between the thyroid gland, the kidneys, and the ovaries, we thought it would be interesting to investigate the thyroid function during the development of compensatory hypertrophy of the kidneys and ovaries after unilateral nephrectomy and ovariectomy.

EXPERIMENTAL METHOD

To study the thyroid function we determined: 1) the maximal accumulation of radioactive iodine (I^{131}) in the thyroid gland, 2) the time taken to achieve the maximal accumulation of I^{131} , 3) the time taken for excretion of half and the whole of the I^{131} from the thyroid gland, and 4) the oxygen consumption of the animals.

Because of the relatively slow excretion of I^{131} from the thyroid gland into the animal organism during the time of development of marked compensatory hypertrophy (1 month), we administered I^{131} twice (after 4 and 20 days) after the operation in doses of $0.1\mu C$ in 1 ml of physiological saline subcutaneously into the right hind limb.

The I^{131} content of the thyroid gland of the rats was determined from the γ -radiation after 2, 4, 6, 8, 12 and 24 hours, and then once daily until no further counts were obtained on the "Las" apparatus, using a scintillation counter and discriminator. Counting was done through a collimator, 10 mm in diameter, at a distance of 60-65 mm. The counting time was selected so that the error of an individual measurement would not exceed 10%.

The oxygen consumption was measured for each rat individually by Kalabukhov's method, using an automatic apparatus suggested by Skvortsov. The measurements were made for a period of 1 hour 14, 15, 21, 22, 28, and 29 days after operation.

Altogether 4 series of experiments were conducted: 1) control experiments on healthy rats, 2) control experiments on laparotomized rats, 3) experiments on unilaterally castrated rats, and 4) experiments on unilaterally nephrectomized animals. Each series comprised 8 rats. The experimental results were analyzed statistically.

EXPERIMENTAL RESULTS

At the end of the experiment the rats were sacrificed, and the ovaries, kidneys, and thyroid gland weighed. The weight of the ovary rose by 114%, and the weight of the kidney by 38% compared with the paired organ removed at the beginning of the experiment. The weight of the thyroid glands was the same as in the controls.

The maximal absorption of I^{131} when administered 4 days after operation is shown by the following figures (mean values): healthy rats $32 \pm 3.4\%$, laparotomized rats $20 \pm 1.5\%$, castrated $19 \pm 1.3\%$, and nephrectomized $15.8 \pm 1.5\%$.

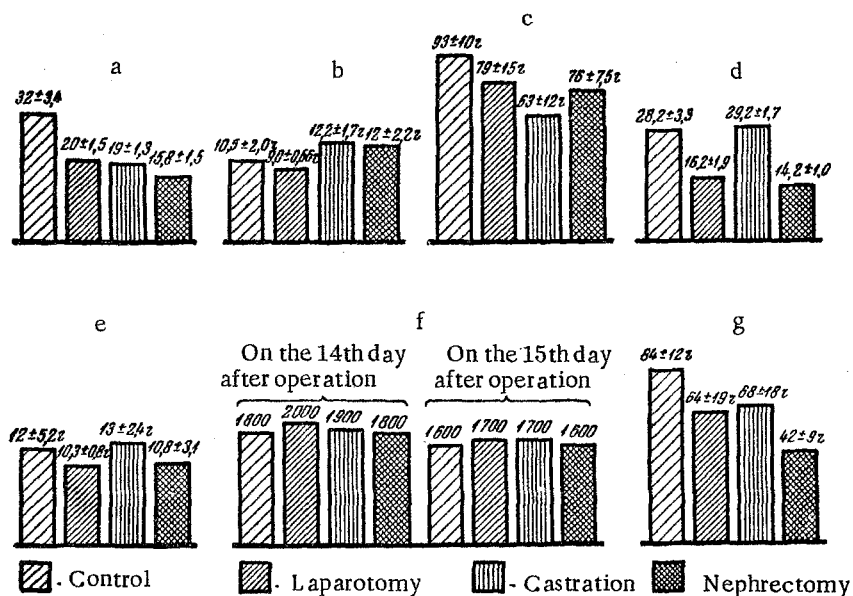
Thus after the operations of laparotomy, unilateral castration, and unilateral nephrectomy, the ability of the thyroid gland to accumulate iodine was lowered, and the extent of the fall was statistically significant (see Fig. 1).

The time taken to reach maximal accumulation of I^{131} was slightly longer in the castrated (12.2 ± 1.7 hours) and nephrectomized (12 ± 2.2 hours) animals than in the healthy (10.5 ± 2) and laparotomized animals (9 ± 0.6 hours), although the difference between the results of the experimental and control groups was not statistically significant.

After the first injection the time taken for excretion of half the I^{131} from the thyroid glands of the rats undergoing operation was slightly less (castrated 63 ± 12 hours, nephrectomized 76 ± 7.5 hours, laparotomized 79 ± 15 hours) than in the intact animals (93 ± 10 hours), although the difference between the figures for the individual groups was not statistically significant.

The investigations of the thyroid function of rats after unilateral castration, nephrectomy, the laparotomy, performed 2-9 days after administration of I^{131} by three different methods of radioindication, thus gave inconsistent results. From the values of absorption of I^{131} the thyroid function not only was not stimulated, as might have been expected from the above-cited literature, but was actually slightly depressed. This does not confirm the results obtained by means of the other two tests, which revealed no significant difference in thyroid function between the intact and the experimental animals.

These inconsistent results forced us to turn our attention to the gas exchange in the animals of all the groups.



Changes in the function of the thyroid gland after castration and nephrectomy in rats. a) Absorption of I^{131} in the thyroid gland (in % of administered dose) on the 4th day after operation; b) time taken to reach maximal absorption of I^{131} in the thyroid gland after first injection of I^{131} ; c) time taken for excretion of half the I^{131} from the thyroid gland after the first injection; d) accumulation of I^{131} in the thyroid gland of the rats (in % of administered dose) on the 20th day after operation; e) time taken to reach maximal absorption of I^{131} in the thyroid gland of the rats after the second injection of iodine; f) oxygen consumption of rats in ml/hour/kg body weight; g) time taken for excretion of half the I^{131} from the thyroid gland of the rats after the second injection.

The oxygen consumption on the 14th day after operation was as follows: in the healthy rats 1800 ml/hour/kg, in the laparotomized rats 2000 ml/hour/kg, in the castrated rats 1900 ml/hour/kg, and in the nephrectomized rats 1800 ml/hour/kg; on the 15th day the corresponding figures were 1600, 1700, 1700 and 1600 ml/hour/kg.

Thus according to the oxygen consumption there was no essential difference between the experimental and control groups.

Consequently by 3 of the 4 tests we were unable to detect any significant difference in the thyroid function between the operated and unoperated rats during the first 10-15 days after the operation. The animals differed only in the degree of absorption of I^{131} by the thyroid gland. However, the results of this test alone cannot be used to form a reliable conclusion regarding the changes in the hormone-excretory and hormone-forming functions of the thyroid gland.

After the second injection of I^{131} the following results were obtained. On the 20th day after the operation the percentage of maximal absorption of I^{131} in the castrated animals was the same (29.2 ± 1.7) as in the intact animals (28.2 ± 3.3); in the laparotomized and nephrectomized animals it was much lower (16.2 ± 1.9 and 14.2 ± 1 respectively). As after the first injection, no significant difference was observed in the rate of absorption of I^{131} between the rats of the different groups (healthy 12 ± 5.2 hours, laparotomized 10.3 ± 0.8 hours, castrated 13 ± 2.4 hours, nephrectomized 10.8 ± 3.1 hours).

On the 20th-25th day after operation the excretion of I^{131} from the thyroid gland was accelerated, although only in the nephrectomized animals was the difference from the control values statistically significant (healthy 84 ± 12 hours, laparotomized 64 ± 19 hours, castrated 68 ± 18 hours, nephrectomized 42 ± 9 hours).

Thus, 20 hours after the operation the difference in absorption of I^{131} between the intact and laparotomized and nephrectomized animals persisted, but the difference between the control and the castrated rats observed 4 days after the operation had disappeared. A new feature was the more rapid excretion of I^{131} from the thyroid gland of the nephrectomized animals. Compared with the healthy rats, in these animals, firstly, the absorption of I^{131} was decreased, and secondly, its excretion from the thyroid gland was accelerated.

Investigation of the oxygen consumption by the rats of the various groups 21-29 days after the operation showed no significant difference between them on each day of the investigation (see Table 1).

TABLE 1. Average Volume of Oxygen Adsorbed by Rats (in ml/hour/kg) at Various Times after Operation

Operation	Time (in days)					
	14	15	21	22	28	29
Unilateral ovariectomy	1890	1704	2132	1660	1313	1332
Unilateral nephrectomy	1831	1563	2164	2058	1163	1146
False operation (Laparotomy)	1955	1687	2011	1816	1254	1211
Intact animals (controls)	1784	1614	2016	1683	1293	1117

Analysis of the data relating to the thyroid function during the period from the 20th to the 30th day after operation also revealed no significant evidence of its stimulation. It may thus be suggested that although the presence of the thyroid gland is essential for the normal course of compensatory hypertrophy of the ovary and kidney after extirpation of its paired organ, no increased thyroid function can be observed under these circumstances. On the contrary, the ability of the gland, for example, to accumulate iodine may actually be diminished.

Only in the case of nephrectomy was the excretion of I^{131} from the thyroid gland accelerated between 20 and 30 days after the operation. This, however, does not always indicate an increased hormone-excreting function of the thyroid gland. The iodine content of the thyroid is a function of several factors, such as the total iodine content of the body and the ability of the thyroid gland to absorb it and to incorporate it into hormones. It is possible that in nephrectomized rats the rapid decrease in the iodine content of the thyroid is associated with a slowing of its absorption and with its rapid excretion from the body. In any case we cannot yet conclude from our results that the hormone-excreting function of the thyroid gland is stimulated after nephrectomy.

In view of our results and those obtained by other workers relating to the effect of stress on the thyroid function, it may be suggested that the thyroid function is not directly connected with the compensatory reactions to removal of one kidney or ovary, but is modified as a result of the involvement of the hypothalamic region and the hypophysis in the compensatory reactions and of the disturbance of the normal relationships in the production of the trophic hormones. Stimulation of one of the hypophyseal functions may lead to some degree of depression of the others. The removal of the thyroid gland thus may probably affect the compensatory hypertrophy of the kidneys and ovaries not only because of the absence of thyroïdin but also because of the depression of the gonadotrophic and other trophic functions of the hypophysis, which is required to produce additional thyrotrophic hormone.

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All abbreviations of periodicals in the above bibliography are letter-by-letter transliterations of the abbreviations as given in the original Russian journal. *Some or all of this periodical literature may well be available in English translation.* A complete list of the cover-to-cover English translations appears at the back of this issue.
