THYROID FUNCTION AFTER PARTIAL PANCREATECTOMY

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The state of the thyroid function was studied in Wistar rats 1, 6, and 24 h after partial pancreatectomy. The criteria of thyroid function were the content of neutron-activated iodine in the gland, the iodine-assimilating power of the gland relative to I¹³¹, the blood levels of protein-bound stable and radioactive iodine, and the morphological state of structures of the gland. Despite initial intensification of synthesis and secretory processes in the thyroid gland, toward the end of the experiment its secretory activity was reduced. This effect is regarded as an adaptive response to partial insulin insufficiency.

KEY WORDS: partial pancreatectomy; thyroid gland; protein-bound I131 of the blood serum.

Soon after partial pancreatectomy an adaptive decrease in activity of the antiinsulin systems takes place [4]. This may reduce the demands on the damaged organ and thus facilitate its regeneration.

In this connection it was decided to study the state of thyroid gland after partial pancreatectomy, for definite antagonism exists between the two glands [1-3].

EXPERIMENTAL METHOD

Male Wistar rats weighing 200-250 g were used. The rats were killed 5 at a time, 1, 6, and 24 h after removal of about one-third of the pancreas. Insulin was injected in a dose of four units into five animals 1 h after pancreatectomy and these animals were killed 5 h later. Intact rats were used as the controls. All the animals received an intraperitoneal injection of $35 \,\mu\text{Ci}\ I^{131}$ 1 h before sacrifice. The protein-bound I^{131} level was determined in the blood serum; radioactivity of a weighed samples of tissue from the thyroid gland and pancreas was determined radiometrically; stable iodine (I^{127}) in the thyroid and protein-bound I^{127} in the blood serum were determined by neutron-activation analysis. The thryoid gland also was studied morphologically.

EXPERIMENTAL RESULTS AND DISCUSSION

The minimal I¹²⁷ concentration in the thyroid gland was observed 6 h after partial pancreatectomy, and after 24 h the I¹²⁷ concentration in the gland was back to normal. Injection of insulin into the pancreatectomized animals led to an increase in the I¹²⁷ concentration in the gland, possibly indicating inhibition of the secretion of thyroid hormones (Fig. 1a). The ability of the thyroid gland of the partially pancreatectomized animals to assimilate I¹³¹, judging from accumulation of the isotope in 1 h, was increased throughout the experiment but especially 6 h after the operation (Fig. 1b). After injection of insulin into the pancreatectomized rats, the ability of the thyroid gland to assimilate I¹³¹ was reduced.

The content of protein-bound I¹²⁷ in the blood serum rose appreciably 1 h after the operation. It then began to fall sharply and by the end of the experiment the blood protein-bound I¹²⁷ level was actually a little lower than in the intact rats (Fig. 2a). The blood protein-bound I¹³¹ concentration fell progressively throughout the experiment (Fig. 2b).

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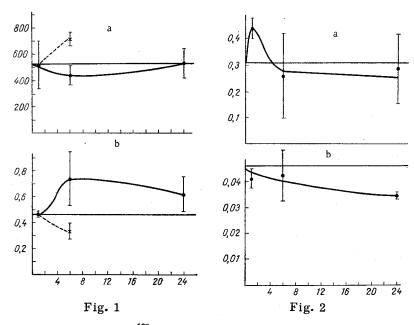


Fig. 1. Changes in I^{127} content in thyroid gland (a) and in ability of thyroid to accumulate I^{131} (b) after partial pancreatectomy. Vertical lines show standard deviations. Horizontal line shows I^{127} and I^{131} levels in control. The broken line – group of animals receiving insulin. Abscissa, time after operation (in h); ordinates: a) I^{127} content (in $\mu g/g$ wet weight of gland); b) I^{131} content (in percent of injected dose per mg gland tissue).

Fig. 2. Change in blood serum protein-bound I^{127} (a) and I^{131} (b) levels after partial pancreatectomy. Ordinates: a) concentration of protein-bound I^{127} (in $\mu g/ml$); b) content of protein-bound I^{131} (in percent of injected dose per ml blood serum). Remainder of legend as in Fig. 1.

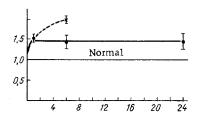


Fig. 3. Change in I^{131} concentration in tissue of pancreas after its partial resection. Ordinate, I^{131} concentration (in percent of injected dose per mg wet weight ($\times 10^{-5}$). Remainder of legend as in Fig. 1.

The results of radiometry of I¹³¹ in weighed samples of the pancreas showed a marked increase in the concentration of the isotope in its tissues in all groups of animals undergoing the operation, but the increase was particularly great in partially pancreatectomized rats receiving insulin (Fig. 3).

Resorption vacuoles appeared in the colloid of the thyroid gland 6 h after the operation and the thyroid epithelium increased in height; these features indicate increased functional activity of the gland. By 24 h after pancreatectomy the colloid of the gland again thickened, and the height of the epithelium returned to the control level.

It can be concluded from these results that during the first 6 h after the operation the response of the thyroid gland took the form of discharge of its hormones. This was shown by the decrease in the $\rm I^{127}$ concentration in the gland and the transient increase in the blood serum protein-bound $\rm I^{127}$ level. Meanwhile, synthesis of the

hormone was stimulated, as shown by the increased ability of the gland to assimilate I¹³¹. The morphological data confirmed discharge of the hormone from the gland (the appearance of resorption vacuoles in the colloid) and intensified hormone synthesis (increase in height of the thyroid epithelium) in the early periods after partial pancreatectomy.

The absence of any increase in the blood serum protein-bound I¹³¹ level indicates that discharge of the hormone from the gland took place at the expense of that held previously in reserve; the rate of secretion

of newly formed (labeled) hormone during the intensification of its synthesis showed an evident tendency to diminish.

Secretion of thyroid hormone was inhibited 24 h after partial pancreatectomy. This was shown, first, by restoration of the normal I¹²⁷ level in the thyroid gland and, second, by a decrease in the blood serum protein-bound I¹³¹ level. The decrease in the blood protein-bound I¹³¹ level could also point to increased utilization of thyroxine at the periphery, which could explain the rapid disappearance of the hormone discharged into the blood stream (Fig. 2a). Increased utilization of the hormone at the periphery was also confirmed by the increase in the I¹³¹ concentration in the pancreatic tissue (Fig. 3).

One of us [5] previously postulated the existence of two iodine depots in the thyroid gland: foci with rapid and slow turnover. A primary effect of thyrotropin is to cause the discharge of iodine from the slow-turnover depot (characterized by the I¹²⁷ dynamics). This is accompanied by activation of metabolism in the first depot (characterized by the I¹³¹ dynamics in the initial stage after its injection).

The primary response of thyroid gland to partial pancreatectomy resembles very closely the response to single injection of thyrotropin. This suggests a role of the thyroid-stimulating function of the pituitary in the response of the organism to partial insulin insufficiency. The decrease in intensity of incretion of thyroid hormone in the late stages after partial pancreatectomy can be interpreted as a decrease in activity of one component of the anti-insulin system.

LITERATURE CITED

- 1. A. S. Breslavskii, in: Endocrinopathies and their Hormonal Treatment [in Russian], No. 3, Kiev (1966), p. 35.
- 2. S. A. Morenkova, Vopr. Med. Khimii, No. 2, 204 (1966).
- 3. I. A. Shevchuk and P. I. Tsapok, Probl. Éndokrinol., No. 5, 98 (1970).
- 4. G. G. Sheyanov and E. A. Rapoport, Byull. Éksperim. Biol. i Med., No. 5, 33 (1972).
- 5. A. W. Tkazcew and W. E. Zajczik (V. E. Zaichik), Endokr. Pol., 24, 11, (1973).