

RESPONSE OF THE THYROID GLAND TO CHANGES IN THE BLOOD PLASMIN CONCENTRATION

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UDC 612.44-06:612.128.015.13

In experiments on rabbits, plasmin (fibrinolysin), in a dose of 2000 units/kg modified the hormone-excreting function of the thyroid gland: 5 min after injection of the preparation the blood protein-bound iodine level was lowered, but 1 h after injection it was raised. In rabbits with depressed thyroid function fibrinolysin also inhibited the hormone-excretory function of the gland. If plasmin activity was reduced by ϵ -aminocaproic acid, the protein-bound iodine level rose. Heated fibrinolysin did not affect thyroid function.

KEY WORDS: fibrinolysis; plasmin; protein-bound iodine; ϵ -aminocaproic acid.

Plasmin (fibrinolysin), producing lysis of intravascular blood clots, is an essential component of the fibrinolytic system of the body [1, 9, 10]. Thyroid hormones have a marked effect on the plasmin activity of the blood [3, 4, 7, 15]. Changes in the fibrinolysis system, in turn, may lead to changes in the functional state of both nervous and endocrine systems [2].

The object of this investigation was to study the response of the thyroid gland to changes in the blood plasmin concentration.

EXPERIMENTAL

Experiments were carried out on 57 rabbits of both sexes weighing 2000-3200 g. Thyroid function was depressed in one group of animals by administration of 6-methylthiouracil (6-MTU) in a dose of 0.15 g/kg daily for 1 month. An aqueous solution of fibrinolysin (2000 units/kg) was injected intravenously. Fibrinolysin heated on a boiling water bath for 12 h, in the same concentration, was injected into the control animals [1]. To reduce the blood concentration of endogenous plasmin, ϵ -aminocaproic acid (ACA) was given in a dose of 100 mg/kg. The blood protein-bound iodine (PBI) concentration [8] was determined 5 min and 1 h after the injection of fibrinolysin. In parallel tests, changes in the fibrinolytic activity of the blood plasma were investigated [11].

The numerical results were subjected to statistical analysis [6].

RESULTS

In intact rabbits fibrinolysin depressed the hormone-excretory function of the thyroid gland. The blood PBI level 5 min after injection of the preparation was lowered from 14.5 to 6.6 $\mu\text{g}\%$ ($P < 0.001$). The PBI concentration 1 h after injection of the fibrinolysin was slightly increased (to 18.9 $\mu\text{g}\%$; $P = 0.05$). Heated fibrinolysin caused no change in the hormone-excretory function of the thyroid gland.

Parallel with changes in the PBI concentration, changes also occurred in the fibrinolytic activity of the blood plasma. For instance, 5 min after the injection of fibrinolysin, the fibrinolytic activity of the plasma was increased by 42.6% ($P < 0.01$), but after 1 h in most experiments it was reduced by 32.5% ($P < 0.001$). In a minority of animals the PBI remained at a high level.

In the rabbits receiving 6-MTU, fibrinolysin also lowered the plasma PBI concentration (from 3.0 to 2.04 $\mu\text{g}\%$ after 5 min and to 1.5 $\mu\text{g}\%$ after 1 h; $P < 0.05$).

Department of Human Physiology, Saratov Medical Institute. (Presented by Academician of the Academy of Medical Sciences of the USSR N. A. Fedorov.) Translated from *Byulleten' Éksperimental'noi Biologii i Meditsiny*, Vol. 78, No. 12, pp. 15-16, December, 1974. Original article submitted February 18, 1974.

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ACA inhibited the fibrinolytic activity of the blood plasma by 30-40 % ($P < 0.05$). No significant changes in the PBI level could be observed 5 min after injection of ACA into the rabbits, despite the reduced endogenous plasmin activity [12-14]. However, after 1 h the plasma PBI concentration was significantly higher than initially ($20.0 \mu\text{g } \%$; $P < 0.05$).

During changes in the blood plasmin concentration, changes were thus observed in the hormone-excretory function of the thyroid glands both in intact rabbits and in animals whose thyroid function was depressed. With elevation of the blood fibrinolysin level, the PBI concentration fell, but when the plasmin concentration was reduced, the PBI level rose. The latter also occurred 1 h after the injection of fibrinolysin, when because of binding of the enzyme by antifibrinolysin [1, 5], the fibrinolytic activity of the blood decreased.

Conjecturally, fibrinolysin (plasmin), by modifying the activity of the thyroid gland, one of the factors concerned with the hormonal regulation of hemostasis, provides for self-regulation in the fibrinolytic system.

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