This may perhaps explain the fluctuation in concentration of membranes of individual subcellular structures while the total concentration of membranes of all the organoids studied remained constant.

LITERATURE CITED

1. V. Ya. Brodskii and I. V. Uryvaeva, Cellular Polyploidy, Proliferation, and Differentiation [in Russian], Moscow (1981).
2. P. D. Gorizontov, Homeostasis [in Russian], Moscow (1978).
3. A. A. Pokrovskii and V. A. Tutel'yan, Lysosomes [in Russian], Moscow (1976).
4. Z. A. Ryabinina and V. A. Benyush, Polyploidy and Hypertrophy of Cells in Processes of Growth and Repair [in Russian], Moscow (1978).
5. D. S. Sarkisov, Essays on the Structural Basis of Homeostasis [in Russian], Moscow (1977).
6. I. V. Uryvaeva and V. M. Faktor, Ontogenez, 6, 458 (1975).
7. V. A. Shkurupii, N. B. Khristolyubova, and G. S. Yakobson, Izv. Sibirsk. Otdel. Akad. Nauk SSSR, Ser. Biol., No. 3, 109 (1973).
8. V. A. Shkurupii, Tsitologiya, 17, 985 (1975).
9. V. A. Shkurupii and G. G. Kovrigina, Tsitol. Genet., 17, 6 (1983).
10. I. N. Yashina, Conditions of Regeneration of Mammalian Organs [in Russian], Moscow (1972).
11. A. V. Loud, J. Cell Biol., 37, 27 (1968).
12. A. F. Morselt, Histochemistry, 41, 111 (1974).
13. L. Ranek, Acta Cytol., 20, 151 (1976).
14. L. Ranek, Acta Cytol., 20, 58 (1976).
15. R. Rigler, Exp. Cell Res., 28, 260 (1962).

ACTION OF ANGIOTENSIN ON ULTRASTRUCTURE OF THE RAT THYROID GLAND

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KEY WORDS: angiotensin; thyroid gland; endotheliocyte.

Angiotensin is a peptide hormone which regulates the functional state of cells of various organs [4, 9]. We know, in particular, that angiotensin controls the function of several endocrine organs, namely the hypothalamus, adenohypophysis, and adrenals [5, 14, 15, 16]. After injection of angiotensin into rats the blood flow in the thyroid gland has been shown to be significantly reduced [13]. On incubation of fragments of rat thyroid gland in the presence of angiotensin, accumulation of radioactive iodine by the cells of this gland is considerably inhibited [5]. However, the action of angiotensin on the structure of the thyroid gland has not previously been studied. The aim of the present investigation was accordingly to study the ultrastructure of exchange microvessels and follicles of the thyroid gland in rats receiving angiotensin.

EXPERIMENTAL METHOD

Experiments were carried out on 32 male rats weighing 200-300 g. Sixteen rats were used for the radiometric tests. Angiotensin was injected intraperitoneally into six experimental animals in a dose of 1 mg in 1 ml physiological saline; ten control animals received 1 ml of physiological saline alone. All animals were given an intraperitoneal injection of ^{131}I in a dose of 3 μCi at the same time.

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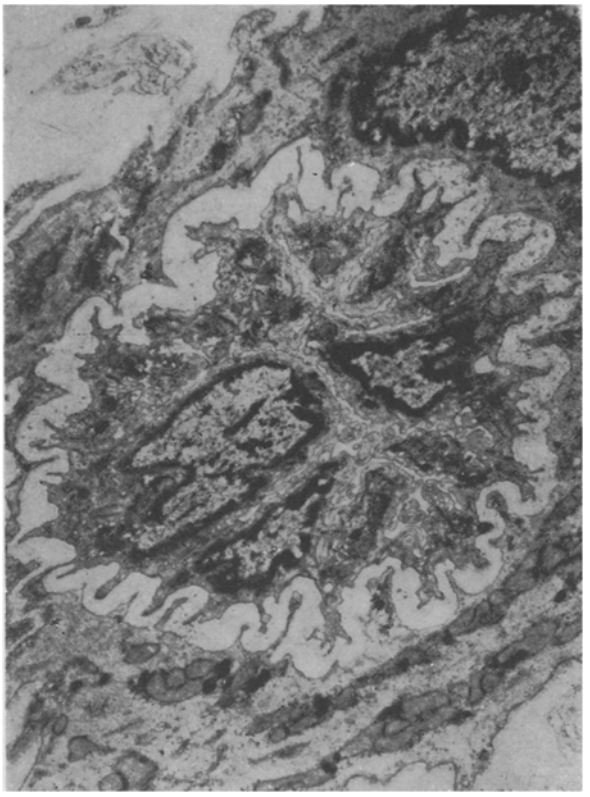

Fig. 1

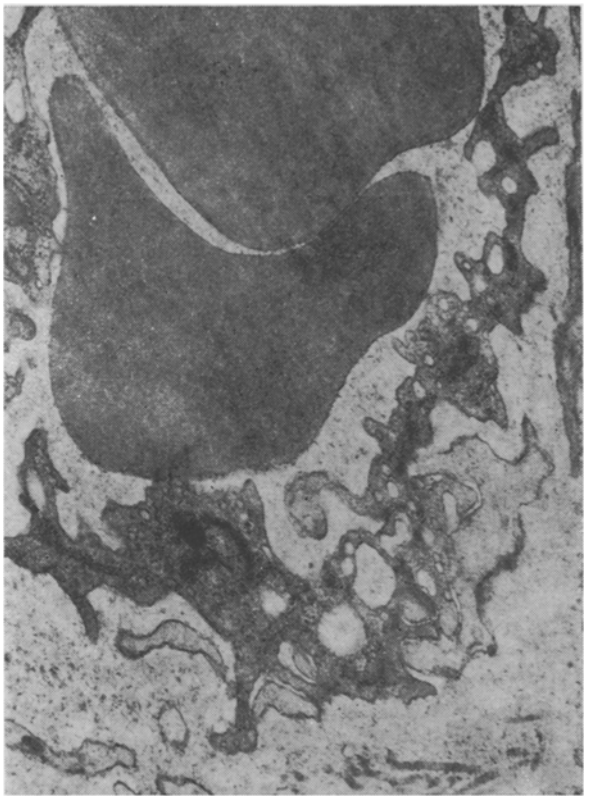

Fig. 2

Fig. 1. Arteriole in thyroid gland of rat receiving angiotensin. Contraction of myocytes, disappearance of lumen of arteriole. 8300 ×.

Fig. 2. Endotheliocyte of exchange microvessel of thyroid gland of rat receiving angiotensin. Condensation of cytoplasm, folding of luminal and basal surfaces of endotheliocyte. 14,400 ×.

The animals were killed under ether anesthesia 4 h later. The thyroid glands were removed and their radioactivity counted on a well-type scintillation counter attached to a URU-64 apparatus. For the ultrastructural investigations six rats were given angiotensin in the same dose and ten rats received physiological saline. The thyroid glands were fixed in glutaraldehyde solution, postfixed in osmium tetroxide, stained in uranyl acetate solution, and embedded in Araldite. Ultrathin sections were cut on the UMTP-2 ultramicrotome, counterstained with lead citrate, and examined in the UEMV-100K electron microscope. The following parameters of the endotheliocytes were measured on negatives by means of a magnifying glass and ocular attachments: the shape factor, characterizing the degree of twisting of the basal and luminal surfaces of the endotheliocytes, the total number of vesicles on the basal and luminal surfaces, and the size and eccentricity of the diaphragmed and pinocytotic vesicles [1, 7].

EXPERIMENTAL RESULTS

After injection of angiotensin into the rats, significant inhibition of accumulation of radioactive iodine by the thyroid glands took place. Whereas the thyroid glands of rats receiving physiological saline accumulated 35 ± 2.5% of the injected dose of radioactive iodine after 4 h, this figure was reduced by angiotensin to 25 ± 3.2% ($P < 0.05$). The ultrastructure of the thyroid gland of rats receiving physiological saline was in full agreement with the results described for intact animals [6, 7]. In a detailed study of the ultrastructure of endotheliocytes of the perifollicular exchange microvessels two types of junctions between these cells were distinguished. Junctions of the first type in the perifollicular exchange microvessels were observed more frequently and were formed by outgrowths of pericytes, penetrating through the basement membrane.

TABLE 1. Effect of Angiotensin on Morphometric Parameters of Luminal and Basal Surfaces of Endotheliocytes of Exchange Microvessels of Rat Thyroid Gland

Parameter	Control $X \pm S_{\bar{x}}$	Angiotensin $X \pm S_{\bar{x}}$	P
Luminal surface			
Shape factor of luminal surface	0,59±0,07	0,34±0,03	<0,01
Number of vesicles connected with luminal surface	0,48±0,14	0,85±0,16	>0,05
Major diameter of diaphragmed vesicles, nm	90±5,6	89±5,2	>0,05
Major diameter of pinocytotic vesicles, nm	92±8,9	82±4,4	>0,05
Eccentricity of diaphragmed vesicles	56±12,9	25±5,0	<0,05
Eccentricity of pinocytotic vesicles	47±9,2	27±6,3	>0,05
Basal surface			
Shape factor of basal surface	0,55±0,05	0,44±0,04	>0,05
Number of vesicles connected to basal surface	0,70±0,18	0,86±0,15	>0,05
Major diameter of diaphragmed vesicles, nm	92±3,5	84±4,5	>0,05
Major diameter of pinocytotic vesicles, nm	96±6,2	88±3,9	>0,05
Eccentricity of diaphragmed vesicles	20±5,5	18±4,7	>0,05
Eccentricity of pinocytotic vesicles	31±4,7	33±7,6	>0,05

Legend. P given by comparison with control.

Sometimes the surface of the endotheliocyte formed an invagination into which the outgrowth of the pericytes entered. Junctions of the second type were formed by outgrowths of the endotheliocyte, in direct contact with pericytes. Complementary invaginations, into which the outgrowths of the endotheliocytes entered, were found on the surface of the pericyte.

Under the influence of angiotensin, spasm of the arterioles (Fig. 1) and substantial changes in the ultrastructure of the exchange microvessels were observed. The cytoplasm of most endotheliocytes of the perifollicular exchange microvessels was considerably condensed, but in some cells translucency of the cytoplasm was observed under the influence of angiotensin. The luminal surfaces of the endotheliocytes became highly convoluted; similar changes affected also the basal surfaces, although to a lesser degree (Fig. 2). Fenestrated areas of endotheliocytes were unchanged. The outlines of the endotheliocyte nuclei became much more convoluted and numerous invaginations of the nuclear membrane appeared. The relative volumes of the condensed and diffuse chromatin were unchanged under these circumstances. No changes in nucleolar structure likewise were observed.

Morphometric measurements showed that angiotensin causes significant changes in certain parameters characterizing the state of the luminal surface of the endotheliocytes. For instance, the shape factor of the luminal surface was considerably reduced. Eccentricity of the diaphragmed vesicles, located on the luminal surface, was reduced, indicating rounding of these structures (Table 1). Meanwhile, the number of vesicles in the cross section of an endotheliocyte was unchanged after administration of angiotensin. There was likewise no change in size of the pinocytotic and diaphragmed vesicles located on the luminal and basal surfaces of the endotheliocytes.

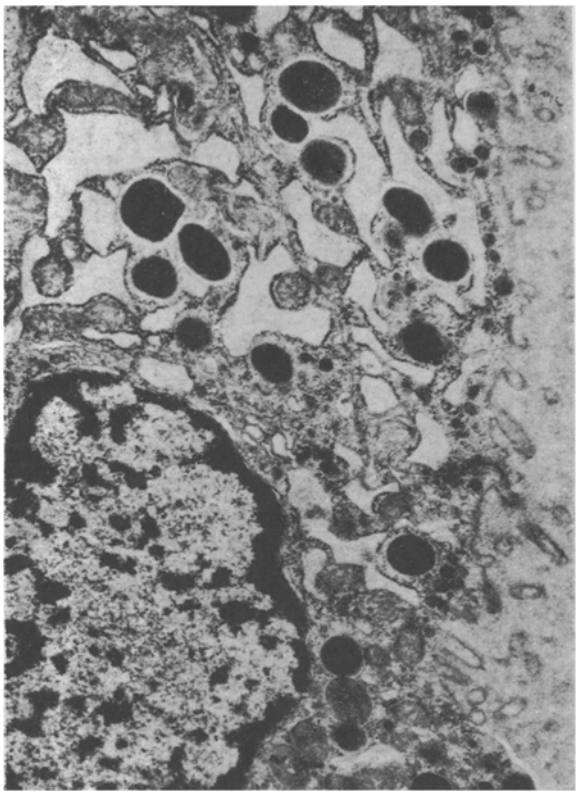

Fig. 3. Thyrocyte of rat receiving angiotensin. Numerous lysosomes in apical zone of thyrocyte. 12,500 ×.

Angiotensin thus caused a considerable change in structure of the endotheliocytes of the perifollicular blood capillaries; these results agree with the view that endotheliocytes are target cells for angiotensin [10]. The reaction of endotheliocytes of thyroid gland blood capillaries to angiotensin is to some degree similar to the reaction of endotheliocytes of blood capillaries of the brain [2, 3]. Parallel with changes in structure of the endotheliocytes, a change also was found in the state of the thyrocytes. On administration of angiotensin the cisterns of the rough endoplasmic reticulum dilated. The number of membrane-bound ribosomes and the number of mitochondria were reduced. The Golgi complex was unchanged. Nuclei of the thyrocytes underwent considerable changes under the influence of angiotensin. Invaginations of the nuclear membrane appeared and the fibrillary component of the nucleoli was condensed, to surround the central part in the form of discrete masses. The relative volume of condensed chromatin was unchanged in this case. The number of lysosomes was significantly increased; these organelles moved into the apical zone of the thyrocyte and often formed groups containing up to 10 lysosomes (Fig. 3). Whereas the mean number of lysosomes in the control in a medium section through one thyrocyte was 3.9 ± 1.0, after administration of angiotensin it reached 10.3 ± 1.8 ($P < 0.01$).

After administration of angiotensin to rats, a parallel change thus takes place in the ultrastructure of the endotheliocytes of the perifollicular exchange microvessels and in that of the thyrocytes. According to data in the literature, angiotensin depresses the blood flow in the thyroid vessels [13]. We found that angiotensin depresses the functional state of thyrocytes (inhibits the accumulation of radioactive iodine).

Consequently, it can be tentatively suggested that angiotensin in the present experiments induced primary modulation of specific target cells, namely endotheliocytes, and that changes in the state of the thyrocytes were secondary. If this is true, our results confirm the view that endotheliocytes are system-forming cells of the thyroid microregion and may have a regulating action on the state of the thyrocytes [8]. The opposite situation arises after experimental narrowing of the lumen of the abdominal aorta in rats, which causes an increase in the blood flow in the thyroid gland and, as a result, stimulates accumulation of radioactive iodine by the thyrocytes [8]. There is also another possible explanation of these results: Angiotensin may have a direct effect on thyrocytes. This possibility is supported by the ability of angiotensin to inhibit the adenylate cyclase of certain epitheliocytes [12].

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LITERATURE CITED

1. G. G. Avtandilov, Introduction to Quantitative Pathologic Morphology [in Russian], Moscow (1980).
2. N. I. Artyukhina, M. G. Airapetyants, O. F. Kuvaeva, et al., Byull. Éksp. Biol. Med., No. 11, 615 (1979).
3. N. I. Artyukhina, K. K. Gekht, O. F. Kuvaeva, et al., Arkh. Anat., No. 3, 16 (1980).
4. S. G. Galaktionov, G. V. Nikiforovich, G. I. Chipens, et al., Angiotensin [in Russian], Riga (1979).
5. V. I. Garets, S. G. Maile-Avgustinovich, E. M. Staroseletskaya, et al., in: Synthesis and Investigation of Biologically Active Compounds [in Russian], Riga (1981), p. 84.
6. S. G. Maile-Avgustinovich, L. V. Gerbil'skii, and V. I. Arkhipenko, Arkh. Anat., No. 6, 106 (1979).
7. G. I. Chipens, L. K. Polevaya, N. I. Veretennikova, et al., Structure and Functions of Low-Molecular-Weight Peptides [in Russian], Riga (1980).
8. V. Buonassisi and P. Colburn, Adv. Microcirc., 9, 76 (1980).
9. K. J. Catt, G. Aguilera, A. Capponi, et al., J. Endocrinol., 81, No. 2, 37P (1979).
10. S. Jard, B. Cantau, and K. H. Jakobs, J. Biol. Chem., 256, 2603 (1981).
11. J. Kapitola, Blood Flow Through the Thyroid Gland in Rats, Prague (1974).
12. C. D. Sladek and R. J. Joynt, Endocrinology, 104, 148 (1979).
13. M. K. Steele, A. Negro-Vilar, and S. M. McCann, Endocrinology, 109, 893 (1981).
14. J. W. Harding, L. P. Stone, and J. W. Wright, Brain Res., 205, 265 (1981).

HISTOCHEMICAL ANALYSIS OF GLYCOSAMINOGLYCAN CONTENT IN THE CHOROID PLEXUS DURING HYDRATION AND DEHYDRATION

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KEY WORDS: vasopressin; water and electrolyte metabolism; choroid plexus; cerebrospinal fluid.

Vasopressin is known to participate in the regulation of cerebrospinal fluid (CSF) production by inducing an iso-osmotic decrease in its volume [4]. It has been suggested that the hormone modifies the transport function of the epithelium of the choroid plexus through its effect on structures of glycosaminoglycan nature sensitive to it, and that this mechanism is similar to that known for its action on the collecting tubules of the kidneys.

This paper describes a histochemical analysis of changes in the choroid plexus during maximal depression of secretion of endogenous vasopressin and in response to injection of posterior pituitary extract. The results obtained in these extreme situations can shed light on the role of glycosaminoglycans (GAG) in the mechanism of the change in permeability of the choroid plexus under the influence of vasopressin and the role of this process in preservation of the volume and composition of the CSF.

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