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# HISTOPHYSIOLOGICAL SIGNS OF HYPERFUNCTION IN THE ANTIDIURETIC CENTRES IN EXPERIMENTAL TRAUMATIC OLIGURIA

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The circulatory theory has found most widespread acceptance to explain the development of oliguria and renal insufficiency associated with secondary and traumatic shock. Since it is known that haemoconcentration, an important stimulus of ADH-mobilisation, is another phenomenon associated with secondary shock, it seemed interesting to study the behaviour of the antidiuretic centres in experimentally induced traumatic oliguria.

A total of 100 white rats of both sexes, kept on a mixed diet, taken from the laboratory stock, was used in the experiments. Renal insufficiency was induced by applying a tourniquet to one or both hind legs of the animals under superficial ether narcosis. Compression was maintained for 4 hours by means of isolated aluminium wire. After killing the animals in different intervals the diencephalon and the hypophysis were fixed in Susa's fluid and embedded in paraffin, then serial sections were made. These were stained with Böhmer's haematoxylin and Gömöri's chrome-alum haematoxylin. (See our previous experiments.)

On comparing the diuresis ensuing after hydration in the controls with that in the experimental animals, the water output of the traumatized rats was found to have stopped after 3½ hours' compression. The quantity of urine of the animals hydrated at 2, 4, 6 and 12 hours after removing the tourniquet, measured in periods of 90 minutes, was very small but after 48 hours it returned to normal.

As early as 3 hours after the tourniquet had been applied, haemoconcentration developed further to increase during the first hours after removal of the tourniquet. The haemoconcentration subsided after 24 hours.

Four hours after removal of the tourniquet characteristic hyperfunctional cell forms could be observed in the antidiuretic centres, viz. in the supraoptic and paraventricular nuclei of the hypothalamus. Beside empty cells almost devoid of secretory granules there were observed ganglion cells with nuclei surrounded by a halo; this was brought about by a shift towards the periphery of the cell of the secretory granules, grown less in number, and the tigroid granules. Intensely secreting cells filled with dark staining material were also

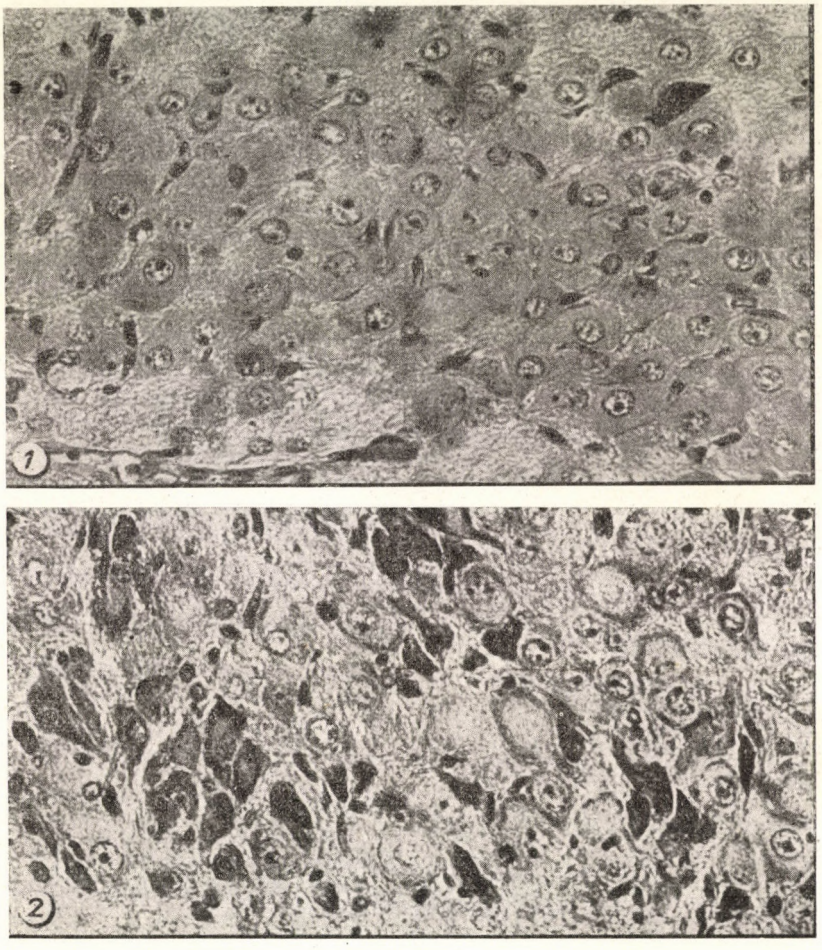

*Fig. 1.* Supraoptic nucleus of control rat. ×350

*Fig. 2.* Signs of hyperfunction in the supraoptic nucleus 4 hours after removal of tourniquet. ×350

observable (Figs. 1 and 2). Similar changes could be seen 8 and 12 hours after removal of the tourniquet. After 24 hours was the depletion of neurosecretory material most pronounced, and at that time there were many swollen, completely empty cells found. It took, as a rule, 70 hours for the histophysiological picture to become normal again. In the neurohypophysis a slight diminution of neurosecretory material or the appearance of swollen pituicytes with empty cytoplasma could be observed in the fourth hour of limb compression. Hyperaemia, oedema, and a marked diminution of the colloid substance ensued 4, 12 and 24 hours, respectively, after the removal of the tourniquet. In most animals the histological picture took 70 hours to become normal.

**Summary**

In experimental traumatic oliguria hyperactivity has been observed to occur at the site of production of the antidiuretic-vasopressor hormone, i. e. in the neurosecretory ganglion cells of the anterior hypothalamus. This makes it probable that antidiuretic-vasopressor hormone is released into the circulation. The results of the present investigations suggest that the described neurohumoral mechanism plays an important part in the development of the oliguria induced by traumatic shock, be it by increasing tubular water resorption, or by promoting renal vasoconstriction.

Details of these investigations will be reported elsewhere.

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## ЯВЛЕНИЯ ГИПЕРФУНКЦИИ В АНТИДИУРЕТИЧЕСКИХ ЦЕНТРАХ ПРИ ЭКСПЕРИМЕНТАЛЬНОЙ ТРАВМАТИЧЕСКОЙ ОЛИГУРИИ

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Согласно исследованиям авторов, место образования вазопрессорных и антидиуретических гормонов, — нейросекреторные ганглиозные клетки переднего гипоталамуса, — проявляют при олигурии, вызванной турникетом, признаки резкой гиперактивности, что говорит за то, что в кровообращение попадает большое количество вазопрессорного и антидиуретического гормонов (см. прежние исследования авторов относительно нейросекреции передних ядер гипоталамуса). Результаты данных исследований указывают на то, что в возникновении олигурии, связанной с травматическим шоком, важную роль может играть вышеизложенный нейрогуморальный механизм, как путем повышения тубулярной резорбции воды, так и путем повышения сужения сосудов почек.

# HYPERFUNKTIONSERSCHEINUNGEN IN DEN ANTIDIURETISCHEN ZENTREN BEI EXPERIMENTELLER TRAUMATISCHER OLIGURIE

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Laut den beschriebenen Untersuchungen zeigt die Bildungsstelle der Hormone mit vasopressorischer und antidiuretischer Wirkung, d. h. das Gebiet der neurosekretorischen Ganglienzellen des vorderen Hypothalamus, während der Tourniquet-Oligurie Zeichen von lebhafter Hyperaktivität, was dafür spricht, dass grosse Mengen der vasopressorischen und antidiuretischen Hormone in den Blutkreislauf gelangen (siehe frühere Untersuchungen der Autoren bezüglich der Neurosekretion der vorderen Hypothalamuskerne). Die erhaltenen Resultate weisen darauf hin, dass an der Entstehung der Oligurie in Verbindung mit traumatischem Schock der besprochene neurohumorale Mechanismus, sowohl durch die Erhöhung der tubulären Wasserresorption, als auch durch die Förderung der renalen Vasokonstriktion in bedeutendem Masse beteiligt sein kann.

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