(UDC 612.815.1:612.014,462.1)

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Department of Normal Physiology (Head—Docent Ya. D. Finkinshtein), Novosibirsk Medical Institute Presented by A. V. Lebedinskii, Active Member, Academy of Medical Sciences, USSR Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 58, No. 8, pp. 6-9, August, 1964 Original article submitted May 27, 1963

According to current concepts the osmomolar concentration of the internal environment of the organism is regulated by neurohumoral systems formed by the aggregate of osmoregulatory reflexes. The afferent portion of these reflexes is effected by specific osmoceptors, which are widely distributed throughout the organism and serve to detect the slightest fluctuations in the osmotic concentration of its internal medium.

Investigations by the students and colleagues of A. G. Ginetsinskii [1-13] established that an osmotic shift may easily be induced by injecting small quantities of a 3-5% hypertonic salt solution, glucose, a NaCl solution isotonic to the aforementioned solutions, or certain other substances into the arterial system of an organ.

It is assumed that the osmoceptors are localized in the tissues and that "hypertonic" blood draws them into reaction as a result of penetration of the osmotically active substance into the interstitial fluid. However, this hypothesis cannot be considered to be sufficiently complete. There is not every reason to believe that physicochemical relationships more complex than previously supposed are created between the blood and the tissue fluid on injection of hypertonic solutions.

In this work we set ourselves the task of further experimentation on the mechanism of osmoceptor stimulation. Using the tagged-atom method and osmometry, we were able to follow the movement of sodium and water in the blood—interstitial fluid system during a hypertonic shift.

EXPERIMENTAL METHOD

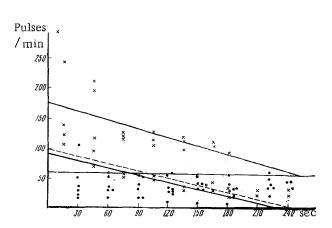


Fig. 1. Fluctuations in radioactive sodium concentration for all experiments of the control and experimental series. The crosses indicate the control series and the dots the experimental series. 1) Pulses/min; 2) sec.

Our investigation was conducted in chronic experiments on 13 dogs weighing 10-20 kg, from which the left kidney was preliminarily removed; a polyvinyl chloride tube was inserted into the renal artery, making it possible to inject the solution to be studied through the aorta into the vessels of the hind legs.

The control experiments were conducted 2-3 days after the operation: 5 ml of physiological solution to which tagged sodium (Na²²) was added in a concentration of 9-10 μ Ci was injected through the tube over a period of 20-30 sec. Experiments involving injection of 5% sodium chloride were later conducted in accordance with the same scheme.

Taking of blood samples was begun simultaneously with the injection, by puncturing the v. saphena. Specimens were taken every 20-30 sec for 2-5 min.

We took 0.2 ml of blood from each sample, applied it to a target in a uniform layer, air-dried it, and

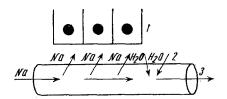


Fig. 2. Diagram of processes which develop in the capillaries in hyperosmia. 1) Cells or organ; 2) interstitial space; 3) capillary.

on the following day determined the Na²² concentration with a Geiger-Muller end-window counter in a B-2 or PS apparatus; this concentration was expressed as the number of pulses per min per 0.2 ml of blood.

We determined the osmotic pressure of these same blood samples by the cryoscopic method [6, 8].

EXPERIMENTAL RESULTS

The tagged sodium content of the blood flowing from the organ depended upon which sodium solution was injected into the blood stream.

Radioactive sodium injected into the blood stream of the hind legs with physiological solution appeared in maximum quantities in the outflow-

ing blood during first 30 sec. The Na²² concentration decreased to 40-50 pulses/min after 90-150 sec and remained at a more or less constant level in all subsequent samples.

When the same quantity of tagged sodium was injected with 5% NaCl its content in the outflowing blood was sharply reduced throughout the entire experiment.

Figure 1 shows the data obtained in all experiments of both series. There was a substantial difference in the Na²² concentrations in the outflowing blood in the control and experimental series, especially during the first 2 min after injection.

In the control experiments the tagged sodium concentration fluctuated between 100 and 175 pulses/min during this time, gradually decreasing. In all cases except one in the experimental series the pulse frequency did not exceed 50 per min, even dropping to 5-10 per min during the interval between the 90th and 180th sec in certain experiments. The sodium contents in the control and experimental series were identical from the 3rd min to the end of the experiment.

The decrease in the quantity of Na²² in the outflowing blood which occurs when a hypertonic solution is injected into the blood stream of an organ apparently results from the fact that radioactive sodium diffuses intensively in the interstitial fluid.

After a certain time the sodium diffusion is joined by an accelerated movement of water from the intertissue space into the blood, the so-called osmotic water flow [14]; as a result, the Na²² content of the venous blood drops still lower 60-90 sec after the injection.

In determining the osmotic pressure of the outflowing blood we obtained data which indicated the existence of a counterflow of water. It was found that the osmomolar concentration of the venous blood was virtually unaltered on injection of physiological solution, while injection of hypertonic solution caused a decrease in osmotic pressure. Thus, in two of the 15 experiments the osmotic pressure remained unaltered, while in the other 13 it decreased by an average of 3% in comparison with the initial pressure.

This drop in pressure cannot be attributed solely to sodium diffusion. Diffusion might have caused the elevated sodium concentration in the blood to return to normal, but it could not have caused it to drop below its initial level.

Injection of hypertonic solution consequently creates conditions for the development of two mutually opposed processes, whose course may be represented in the manner shown in Fig. 2. As may be seen from the diagram, in the capillaries hyperosmia causes development of sodium diffusion into the interstitial space and a simultaneous movement of water in the opposite direction.

The processes which occur in the capillaries when hypertonic solution is injected into the blood stream of an organ are thus apparently more complex than was previously supposed. A counterflow of water into the capillaries probably occurs in addition to diffusion of the injected substance into the tissue fluid.

SUMMARY

By the method of labeled atoms (Na²²) and osmometry it was established that in administration of 5% NaCl solution into the blood there occurs an acceleration of sodium diffusion into the interstitial space and "osmotic flow of water" in the reverse direction.

LITERATURE CITED

- 1. S. A. Borisova and Ya. D. Finkinshtein. In book: Collection of Papers Presented at the 2nd Scientific Conference of Physiologists, Biochemists and Pharmacologists of the Western Siberian Association [in Russian], Tomsk (1961), p. 12.
- 2. L. K. Velikanova, Byull. éksper. biol. (1957), No. 11, p. 62.
- 3. Idem, Abstracts of Papers Presented at the Scientific Conference of Physiologists, Biochemists and Pharmacologists of the Western Siberian Association [in Russian], Tomsk (1957), p. 61.
- 4. Idem, Byull éksper. biol. (1958), No. 4, p. 21.
- 5. L. K. Velikanova and Ya. D. Finkinshtein, Fiziol. zh. SSSR (1959), No. 12, p. 1472.
- 6. L. K. Velikanova, Byull. éksper. biol. (1962), No. 1, p. 15.
- 7. V. L. Inchina and Ya. D. Finkinshtein, In book: Collection of Papers Presented at the 2nd Scientific Conference of Physiologists, Biochemists and Pharmacologists of the Western Siberian Association [in Russian], Tomsk (1961), p. 55.
- 8. L. I. Kurduban, Influence of Extirpation of the Motor Zones of the Dog Cortex on Water Metabolism [in Russian] Candidate's Dissertation, Novosibirsk (1954).
- 9. L. I. Kurduban and Ya. D. Finkinshtein, Byull. éksper. biol. (1960), No. 1, p. 17.
- 10. E. A. Nikolenko and Ya. D. Finkinshtein, In book: Problems of Theoretical and Clinical Medicine [in Russian], Novosibirsk (1959), Book 1, p. 137.
- 11. R. S. Tishenina and Ya. D. Finkinshtein, In book: Collection of Papers Presented at the 2nd Scientific Conference of Physiologists, Biochemists and Pharmacologists of the Western Siberian Association [in Russian], Tomsk (1961), p. 148.
- 12. Ya. D. Finkinshtein, Trudy Novosibirsk. med. inst. (1958), Vol. 30, p. 57.
- 13. Ya. D. Finkinshtein, (1959), Vol. 33, Book 1, p. 130.
- 14. A. Z. Koranyi, Med. (1897), Vol. 33, p. 1, cited by I. Rusn'yak, M. Fel'di, and D. Sabo. Physiology and Pathology of Lymph Circulation [in Russian], Budapest (1957).

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.