

VASOMOTOR RESPONSES OF THE SYSTEMIC
CIRCULATION IN HEMODYNAMIC ISOLATION
FROM THE HEART

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If a hypertonic solution of sodium chloride is injected intraarterially, vasoconstriction takes place in the perfused regions, and this is expressed by an increase of the arterial pressure [2, 9, 11, 13].

The object of the present investigation was to study systemic vasomotor responses to intraarterial injection of hypertonic sodium chloride solution in animals with complete hemodynamic isolation of the systemic circulation from the heart.

EXPERIMENTAL METHOD

Experiments were carried out on cats anesthetized intravenously with urethane (1 g/kg); all the animals received heparin in a dose of 3 mg/kg. The artificial circulation was maintained in the entire systemic circulation by means of a constant delivery pump [6]. To oxygenate the venous blood the lungs of a donor cat ventilated with an oxygen-air mixture by means of a type DP-1 apparatus were used. The venous blood of the experimental animal (recipient) passed through a catheter inserted into the auricle of the right atrium into the donor's right atrium. The venous blood was returned passively from the recipient as the result of the pressure difference in the recipient and donor, amounting to 50 cm water. After its passage through the donor's lungs, the oxygenated blood was directed through a catheter inserted into the animal's aorta into a reservoir where it was warmed to 37.5°, and then by means of the constant delivery pump it was injected into the recipient's iliac artery. The minute volume was 70-75 ml/kg, corresponding approximately to the minute volume of a cat in resting conditions [8]. The recording system comprised an assembly of tensometric electromanometers [5]. Recordings were made of the perfusion pressure at the outlet of the pump, which in conditions of a constant stroke volume of blood is a function of the total vascular resistance, the pressure in the carotid artery, and the volume of blood returned to the arterialized blood reservoir. The venous return was checked by means of a tensometric pick-up, transforming the weight of the column of liquid in the arterial reservoir into an electric signal. The sensitivity of the pick-up was about 15 mA/ml. A 20% solution of sodium chloride was injected into the recipient's iliac artery in a dose of 2 ml/sec. Altogether 32 experiments with a complete artificial circulation were carried out.

EXPERIMENTAL RESULTS

Intraarterial injection of hypertonic sodium chloride solution (7 experiments) was always followed by a pressor-depressor response. At the moment of maximal increase of pressure it had risen by $11 \pm 2.1\%$ from the initial level, which is below the magnitude of the pressor reaction ($32 \pm 6\%$) observed after injection of the same volume of 20% NaCl solution into the femoral artery in natural circulation conditions (4 experiments). The magnitude of the depressor phase was $9 \pm 5.3\%$ of the original level. A marked increase in the venous return into the arterial reservoir of the artificial systemic circulation system was observed in all experiments. The mean excess of blood flowing into the arterial reservoir throughout the response to injection of 20% NaCl solution into the arterial system was 17.3 ± 6.3 ml.

When the rate of blood flow was 30-40 ml/kg/min, the magnitude of the maximal pressor phase of the reaction was $31 \pm 6.7\%$, and of the depressor phase $-14 \pm 2.1\%$ of the original level. If the blood flow was 110-120 ml/kg/min, the maximal pressor phase of the reaction amounted to $7 \pm 3.6\%$, and of the depressor phase $-4 \pm 0.5\%$. A similar relationship between the response to injection of 20% sodium chloride solution

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and the rate of blood flow was observed by G. P. Konradi [2]. The maximal decrease of perfusion pressure after the first injection of hexamethonium in a dose of 1 mg/kg was 10-4% of the original level. The second injection of the same dose of this drug caused no further decrease of perfusion pressure ($0.2 \pm 5\%$), indicating the presence of a complete block to the conduction of impulses through the autonomic ganglia. The mean value of the maximal pressor phase of the response to sodium chloride solution before injection of hexamethonium was $12.5 \pm 1.8\%$ of the initial level, and of the depressor phase $-7 \pm 2.6\%$. The change in the response to sodium chloride 1-3 min after injection of hexamethonium was not statistically significant (8.8 ± 1.7 and $5.3 \pm 1.1\%$ of the initial value respectively).

The perfusion pressure fell sharply by $31.3 \pm 6\%$ (5 experiments) 20 sec after injection of papaverine 10 mg/kg into the main arterial trunk. In these circumstances the pressor phase of the responses to intra-arterial injection of hypertonic sodium chloride solution was reduced to $1.5 \pm 2\%$, and the depressor phase to $3.3 \pm 2\%$.

Comparison of the responses of the systemic pressure in experiments in which a normal circulation was preserved and in experiments with isolation of the systemic circulation from the heart revealed a more marked pressor response in the first case. This fact could be explained by the increase in venous return detected in response to injection of sodium chloride. These results agree in principle with the results of several previous investigations [1, 3, 4, 7]. Probably the increase in the venous return was associated not only with the expression of blood from resistant vessels, but also with constriction of the capacious venous vessels.

LITERATURE CITED

1. G. P. Konradi and Yu. N. Gal'perin, *Fiziol. Zh. SSSR*, No. 1, 46 (1961).
2. G. P. Konradi, In the book: *Problems in Regulation of the Circulation* [in Russian], Moscow-Leningrad (1963), p. 5.
3. V. A. Levtoy and S. S. Musyashchikova, *Fiziol. Zh. SSSR*, No. 12, 1477 (1961).
4. T. V. Migina, In the book: *Diseases of the Cardiovascular System* [in Russian], Moscow (1961), p. 11.
5. L. I. Osadchii, V. A. Levtoy, and V. V. Orlov, et al., *Byull. Éksp. Biol.*, No. 5, 120 (1964).
6. L. I. Osadchii, In the book: *Pathological Physiology of the Cardiovascular System* [in Russian], Tbilisi, Vol. 2 (1964), p. 81.
7. V. P. Rassolova, *Byull. Éksp. Biol.*, 24, No. 2, 95 (1947).
8. J. I. Baxter, *J. Physiol. (London)* 118, 299 (1952).
9. L. Binet and M. Burstein, *C. R. Soc. Biol.* 145, 1766 (1951).
10. A. Goldflott and D. G. Mason, *Circulat. Res.*, 12, 539 (1963).
11. R. B. Hervey, *Fed. Proc.*, 18, 65 (1959).
12. R. C. Read, J. Vick, and M. Meyer, *Ibid.*, p. 124.
13. R. C. Read, M. D. Read, and J. A. Johnson et al., *Circulat. Res.*, 8, 538 (1960).