

THE EFFECT OF INTRAVENOUS INFUSION OF BLOOD SERUM AND PLASMA FROM HYPERTENSIVE AND NEPHRITIC PATIENTS ON ARTERIAL PRESSURE

S. A. Keizer

From the Department of Internal Medicine (Head — Prof. A. A. Nechaev), Academy of Military Medicine, Leningrad

(Received June 18, 1956. Presented by Prof. A. L. Myasnikov, Active Member of the Academy of Medical Sciences, USSR)

To date, the pathogenesis of hypertension has not been completely explored; further study is particularly needed to clarify the role and importance of the humoral factors. According to data given in the literature, the blood of hypertensive patients contains very small amounts of vasopressive substances. At the same time, however, the data concerning the changes in arterial pressure caused by intravenous infusions of blood, blood plasma and serum from hypertensive patients are extremely contradictory.

V. M. Kogan-Yasny [8,9], Voronov and R. Ya. Spivak [3,4] and others observed arterial pressure to increase in people given an intravenous infusion of blood from hypertensive patients. Pikerling, Fridman and Printsmetal, and others did not observe any peculiarities in arterial pressure change under these conditions.

In experiments on cats and dogs, V. M. Kogan-Yasny, B. A. Vartopetov, R. Ya. Spivak and E. M. Shulman [2,7] observed arterial pressure to increase after an intravenous infusion of blood serum and plasma from hypertensive patients. V. M. Kogan-Yasny [8] and A. K. Gorchakov [5] succeeded in bringing experimental animals out of traumatic and peptone shock by an intravenous infusion of blood serum from hypertonics. R. Ya. Spivak [12] obtained a steady elevation of arterial pressure in rabbits by administering a prolonged intravenous infusion of blood serum from hypertensive patients in a dose of 1 cm³ per 1 kg of weight.

According to the data of other authors [6,11], the changes in arterial pressure which occur due to an intravenous infusion of blood serum from hypertensive patients are the same as those which occur due to an intravenous infusion of blood serum from normal people. B. F. Angelidze and G. S. Akhmeteli [1] did not obtain any steady rise in arterial pressure by infusing rabbits intravenously with blood serum from hypertonics for a period of 10 days.

The purpose of this work was to investigate and simplify the problem of determining the effect of an intravenous infusion of serum and plasma from hypertensive and nephritic patients on arterial pressure.

EXPERIMENTAL METHODS

Short experiments were done on cats, weighing from 1.5 to 3 kg, under ether anesthesia (28 experiments) and without anesthesia (6 experiments). Arterial pressure was recorded from the femoral artery with a Ludwig manometer on a smoked kymograph tape. Blood serum and plasma from hypertensive patients (23 experiments) and from nephritic patients (4 experiments) were infused into the femoral vein. In the control, the animals were infused with blood serum and plasma from normal people (7 experiments). We usually infused 8–17 cm³ of plasma and serum, but in a few experiments, 2–5 cm³.

We also studied the background of arterial pressure and pulse in three dogs for a month. The arterial pressure was measured on a hind paw, by Korotkov's method, before and after the intravenous infusions, which were

given every 5 minutes for 1-3 hours. At first, arterial pressure and pulse were observed after vein punctures (3 experiments) and after the infusion of a physiological solution (6 experiments). Later, when the dogs had almost ceased to react to the vein punctures, the changes in arterial pressure and pulse which occurred after the infusion of blood serum from patients with hypertension (17 experiments), chronic nephritis (2 experiments) and from healthy people (2 experiments) were observed. Each dog was infused with serum 7 times over a period of 10 days in a dose of 0.1 - 3 cm³ per 1 kg of weight each time. The total amount of serum infused was from 5-60 cm³.

EXPERIMENTAL RESULTS

The arterial pressure of the cats decreased to 30 mm of mercury 10-31 seconds after the intravenous infusion of blood serum, either with or without anesthesia. The marked decline continued for several seconds and then continued, but not so sharply, for 5-7 minutes. Later, the arterial pressure returned to the original level. Only in 6 experiments with the infusion of blood serum both from healthy people and from hypertensives was a slight, temporary rise of 4-8 mm of mercury in the arterial pressure observed, even at the later period. Analogous changes in arterial pressure were observed with the intravenous infusion of blood plasma.

The character of the changes in the cats' arterial blood pressure was the same with the infusion of blood serum and plasma from healthy people as from hypertensive and nephritic patients, and did not depend on the degree of arterial pressure elevation nor on the stage of the disease. Increasing the amount of serum and plasma infused to 17 cm³ produced a greater decline in arterial pressure, while the infusion of serum in small quantities (2-5 cm³) caused almost no change in arterial pressure.

When the dogs were intravenously infused with the physiological solution and serum, the greatest changes were in maximal arterial pressure. For example, in 15 out of 19 experiments, a temporary increase of arterial pressure, no more than 20 mm of mercury in 10 experiments, occurred after the infusion of blood serum from people with high blood pressure. But a temporary increase in maximal arterial pressure was also observed in every case where blood serum from healthy people or the physiological solution had been infused.

There was comparatively less change in minimal arterial pressure. When blood serum from hypertensive and nephritic patients was infused, minimal arterial pressure increased temporarily in 9 out of 19 experiments; in 7 experiments, this increase did not exceed 20 mm of mercury. Analogous results were produced by the infusion of blood serum from healthy people and of the physiological solution. There was a slight, temporary increase of arterial pressure in 6 out of the 8 control experiments.

In a few cases, a temporary decline of both maximal and minimal arterial pressure was observed immediately after the infusion of the blood serum. The physiological solution had no such effect.

The pulse remained almost unchanged (frequency increase about 4-10 beats per minute) in the short experiments on dogs, except in the first observations made during the vein punctures.

The picture in all of the 3 dogs was completely different on the 10th day of blood serum infusion. In these experiments, a sharp decline in arterial pressure and retardation of the pulse occurred after the infusion of any amount of the serum. Vomiting, panting and strong salivation began immediately after the serum infusion, with, at first, motorial agitation which subsequently changed into total areactivity. At first, the pulse was hardly noticeable, and the arterial pressure could not be determined. In an hour, all pathologic phenomena had passed, and the arterial pressure and pulse returned to the original level. After such a marked pathologic reaction to the blood serum infusion, the experiments were stopped.

Besides the observations of arterial pressure in the short experiment, we also conducted observations during the whole period of the blood serum infusion and afterwards. There was no tendency to regular arterial pressure increase observed in any of the 3 experiments. On the contrary, it was observed to slightly decrease, but returned to the original level 1-2 days after the serum infusions were stopped. During the entire observation period, the pulse fluctuated within normal limits.

SUMMARY

In short experiments it was shown that the effect of intravenous infusion of blood serum and plasma of hypertensive and nephritic patients upon the arterial pressure of cats and dogs is the same as the infusion of blood serum and plasma of healthy individuals.

* In Russian.

In series experiments after 10 days of intravenous infusions of serum and plasma of hypertensive and healthy individuals, arterial pressure of dogs was almost unaltered, and only a slight tendency of lowering was noted.

LITERATURE CITED

- [1] B. F. Angelidze and G. S. Akhmeteli, in the book: Works of the II Enlarged Scientific Session of the Institute of Cardiology on the Problem of Hypertension,* Tbilisi, 1953, pp. 393-397.
- [2] B. A. Vartapetov, R. Ya. Spivak and E. M. Shulman, Byull. Eksptl. Biol. i Med., 1942, Vol. 5-6, No. 11-12, pp. 41-44.
- [3] Ya. K. Voronov and R. Ya. Spivak, Klin. Med., 1941, Vol. 19, No. 1, pp. 95-100.
- [4] Ya. K. Voronov, Vrach. Delo, 1946, No. 11-12, pp. 915-920.
- [5] A. K. Gorchakov, Vrach. Delo, 1945, No. 3-4, pp. 119-124.
- [6] L. N. Karlik and N. I. Burachevsky, Klin. Med., 1945, No. 10-11, pp. 52-56.
- [7] V. M. Kogan-Yasny, B. A. Vartapetov and R. Ya. Spivak, Byull. Eksptl. Biol. i Med., 1937, Vol. 2, No. 4, pp. 91-95.
- [8] V. M. Kogan-Yasny, Byull. Eksptl. Biol. i Med., 1946, Vol. 9, No. 4, pp. 214-217.
- [9] V. M. Kogan-Yasny, Vrach. Delo, 1949, Vol. 26, No. 11-12, pp. 793-800.
- [10] V. M. Kogan-Yasny, Byull. Eksptl. Biol. i Med., 1942, Vol. 14, No. 2, pp. 25-27.
- [11] V. A. Lebedeva and A. Ya. Yaroshevsky, Byull. Eksptl. Biol. i Med., 1950 Vol. 30, part 5, No. 11, pp. 315-320.
- [12] R. Ya. Spivak, Vrach. Delo, 1946, No. 11-12, pp. 841-846.

* In Russian.