

# VASOMOTOR REACTIONS OF THE ABDOMINAL VISCERA AT DIFFERENT ARTERIAL PRESSURES

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Experimental hypertension, like hypertensive disease, is accompanied by a disturbance of the nervous regulation of the cardiovascular system, expressed primarily by changes in the reactivity of the vascular apparatus.

Burton and Girling [7, 8] observed changes in the effectiveness of sympathetic vasomotor impulses at different levels of the arterial pressure. During hypertension caused by administration of promethin, stimulation of the posterior hypothalamus, or compression of the carotid arteries, several workers [1] have observed an increase in the resistance in different parts of the vascular system.

In renal hypertension, some workers [4] recorded changes in the vasomotor reflexes to the hindlimbs, while others found changes in the reactivity of the renal vessels to adrenalin, histamine, and hypertension [12] and a lowering of the threshold of stimulation of the sciatic nerve to pressor agents [2].

Comparison of the reactivity of vascular sections from normal and hypertensive rats to noradrenalin showed [9] a considerable increase in the reactivity of the smooth muscles of the vessels in the hypertensive animals. It has been suggested [5] that a prolonged increase in vascular tone leads to pathological changes in the vessels characteristic of the various forms of experimental hypertension and of hypertensive disease.

It seems, therefore, that the changes in the reactivity of the blood vessels in hypertension are based on structural changes in the peripheral vessels. At the same time, however, it has been pointed out [10, 11] that if the blood pressure is lowered as a result of bleeding, the vascular reactivity is lowered.

We can now make a direct study of the changes in the reactivity of blood vessels in hypertensive and hypotensive states and express them more accurately in quantitative terms. In our previous paper [3] we mentioned that different degrees of nervous influence on vascular fields (number of nerve elements involved in the activity, frequency of vasomotor impulsation, duration of the volley of impulses) may have functionally different vasomotor effects: dilatation or constriction of the vessels. Meanwhile, it has been found that the effects of identical nervous influences may differ, depending on the functional state of the peripheral vessels themselves.

The object of the present research was to study the reactions of the blood vessels in relation to the changes brought about in their condition by changes in the arterial pressure.

## EXPERIMENTAL METHOD

The vascular reactions of the abdominal viscera (intestine, kidney, spleen) were studied in 35 experiments on cats under ether-urethane anesthesia, using the method of resistography [6], the general arterial pressure in the femoral artery being recorded at the same time. Blood from the central end of the superior mesenteric artery was injected by a perfusion pump into the main artery of the organ to be investigated, the supply of blood being so regulated that the pressure in the artery to the organ was equal to the animal's mean arterial pressure. When the blood supply was constant, the perfusion pressure in the vessels of the test organ was determined by their resistance (if the vessels were constricted the pressure rose, and vice versa). Vasomotor reactions were elicited by stimulating the peripheral end of the greater splanchnic nerve on the left side after dividing the nerve on the right side and extirpating the splanchnic chains bilaterally in the lumbar region. Blood clotting was prevented by injecting the animal with heparin (10 mg/kg).

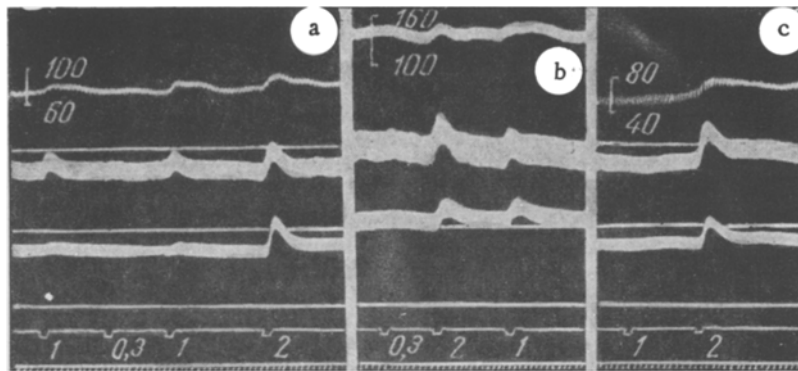


Fig. 1. Relationship between level of general arterial pressure and threshold values of vasomotor responses during stimulation of the peripheral end of the splanchnic nerve by an electric current with a strength of 1, 0.3, and 2 V. a) Vasomotor responses at an arterial pressure of 80 mm; b) 140 mm; c) 60 mm. Significance of the curves (from above down): arterial pressure; resistogram of the superior mesenteric artery; resistogram of the left renal artery (marker of zero pressure in these areas); marker of stimulation with indication of strength (in V); time marker (5 sec).

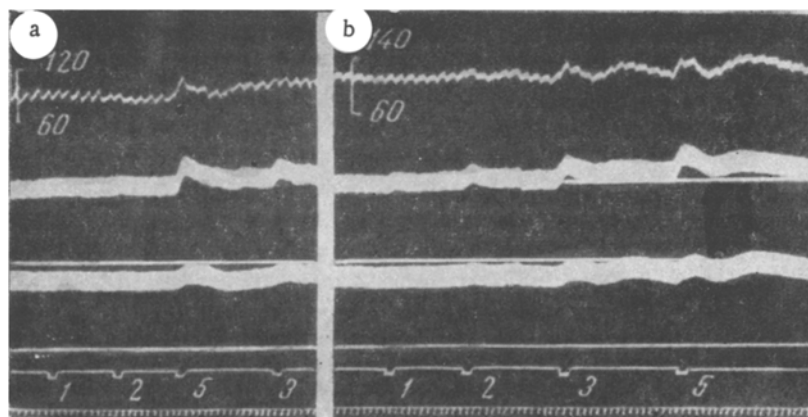


Fig. 2. Relationship between level of the general arterial pressure and frequency thresholds of vasomotor responses during stimulation of the peripheral end of the splanchnic nerve. a) Vasomotor reactions at an arterial pressure of 90 mm; b) the same at an arterial pressure of 120 mm. Significance of the curves (from above down): arterial pressure; resistogram of the superior mesenteric artery; resistogram of the splenic artery (marker of zero pressure in these areas); marker of stimulation with indication of frequencies (in cps); time marker (5 sec).

#### EXPERIMENTAL RESULTS

The threshold levels of stimulation causing vasomotor reactions were determined during stimulation of the splanchnic nerve. When the general arterial pressure was elevated the vasomotor reactions developed in response to relatively weaker stimulation of the splanchnic nerve. When the pressure was lowered no vasomotor reactions were observed in response to stimulation of the same character.

The results of one such experiment are shown in Fig. 1. The splanchnic nerve was stimulated with a constant frequency of 20 cps, a constant duration of each stimulus of 3 millise, and duration of volley of impulses of 5 sec. When the general arterial pressure was 80 mm, vasomotor reactions were not present in the kidney if the strength of stimulation was 0.3 V, but they began to appear at a strength of 1 V, and increased as the strength of stimulation rose to 2 V (Fig. 1, a).

When the general arterial pressure was increased by injection of Ringer's solution and polyglucin (to 140 mm), a weak vasomotor response appeared in the intestinal region when the strength of stimulation was 0.3 V (Fig. 1, b). Conversely, a fall in the general arterial pressure to 60 mm by deliberate bleeding increased the threshold of the vasomotor responses. In these conditions, stimulation with a strength of even 1 V had no effect. Only when the strength reached 2 V were vasomotor effects observed in the intestine and kidney (Fig. 1, c).

A similar relationship between the vasomotor reactions of the vessels of the abdominal viscera and the level of the general arterial pressure was also observed when the frequency of stimulation of the splanchnic nerve was varied. In this case, lower frequencies of stimulation, not causing vasomotor reactions at a relatively low level of the general arterial pressure, produced responses when the arterial pressure was raised.

In Fig. 2, for instance, we show the vascular reactions of the intestine and spleen during stimulation of the splanchnic nerve at a constant strength of 1 V, a constant duration of each individual stimulus of 3 millisecc, and a duration of the volley of the impulse of 5 sec. When the general arterial pressure was 90 mm, a frequency of 1 cps caused no vascular reactions, while at a frequency of 2 cps a weak reaction of the intestinal vessels developed, although the vessels of the spleen did not react to this frequency of stimulation. At frequencies of 3 and 5 cps obvious reactions appeared in both vascular fields (Fig. 2, a).

An increase in the general arterial pressure to 120 mm as a result of compression of both carotid arteries was followed by the appearance of vasomotor responses in the intestine at a frequency of stimulation of the splanchnic nerve of 1 cps, and at a frequency of 2 cps the splenic vessels also began to react. A further increase in the vasomotor reactions was observed at frequencies of stimulation of 3 and 5 cps (Fig. 2, b).

These results indicate that the reactions of the blood vessels of the abdominal viscera are dependent on the level of the general arterial pressure. It was found that when the pressure was elevated vasoconstriction and vasodilatation arose in response to weaker stimulation than when the pressure was normal. Conversely, when the initial pressure was lowered the thresholds of these vascular reactions were raised.

The vasomotor reactions of the intestine and kidney during stimulation of the splanchnic nerve at a constant frequency of 20 cps, a constant duration of each individual stimulus of 3 millisecc, and a duration of the volley of 5 sec, are shown in Fig. 3. When the general arterial pressure was 80 mm, stimulation at strengths of 0.3 and 1 V caused dilatation of the intestinal vessels, whereas at a strength of 2 V they were constricted. Vasoconstriction was increased by stimulation at a strength of 5 V (Fig. 3, a). If the general arterial pressure was raised to 120 mm, as before a strength of 0.3 V caused dilatation of the intestinal vessels, whereas at a strength of 1 V constriction took place, the degree of which increased as the strength of stimulation increased to 2 and 5 V (Fig. 3, b).

Hence, in response to the same strength of stimulation (1 V), different vasomotor effects developed, depending on the level of the general arterial pressure: dilatation at a relatively low pressure (90 mm) and constriction at a higher pressure (120 mm). In the renal vessels no clearly defined vascular reactions were found in this experiment.

It follows from these facts that identical stimulation of the splanchnic nerve gives rise to vasomotor responses of different functional significance, depending on the level of the general arterial pressure. Vasodilatation develops

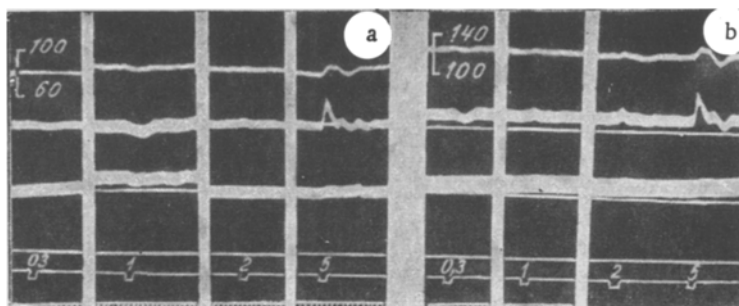


Fig. 3. Relationship between the level of the general arterial pressure and the character of the vasomotor response during stimulation of the peripheral end of the splanchnic nerve. a) Vasomotor reactions at an arterial pressure of 80 mm; b) the same at an arterial pressure of 120 mm. Significance of the curves as in Fig. 1.

more rapidly at a relatively low arterial pressure, whereas the same stimulation during hypertension gives rise to vasoconstrictor effects.

In order to produce an identical change in vascular tone at different levels of the initial arterial pressure, it therefore follows that the central nervous system must exert quantitatively different influences. Another factor concerned here is the initial state of the vessels, which is characterized by changes in their reactive properties in response to nervous stimulation.

#### SUMMARY

A study was made of the vasomotor reactions of abdominal organs (the kidneys, spleen, intestine) by resistography in electric stimulation of the peripheral end of the splanchnic nerve with various intensity and frequency. It is shown that against the background of increased pressure, both vasodilatation and vasoconstriction occurred in lower intensity and frequency in comparison with the vascular reactions in the normal blood pressure level. Conversely, the thresholds of vasomotor reactions increase when the initial pressure level is diminished. This indicated that the vasomotor effects could be replaced by functionally opposite ones in the same nerve influences, depending on the initial state of vessels.

#### LITERATURE CITED

1. A. M. Blinova, G. N. Aronova, and K. E. Serebryanik. In the book: Problems in Experimental Hypertension and Hypertensive Disease [in Russian], No. 3, p. 46. Moscow, 1953.
2. N. N. Gorev, M. A. Kondratovich, and M. F. Shuba. In the book: Problems in the Physiology and Pathology of Vascular Tone [in Russian], p. 17, Kiev, 1961.
3. I. M. Rodionov and V. P. Kulagina. Byull. éksper. biol., 2, 13 (1962).
4. Z. T. Samoilova. In the book: Hypertensive Disease [in Russian], No. 5, p. 112. Moscow, 1958.
5. M. L. Tarakhovskii and V. L. Fastovskii. Vrach. delo, 5, 489 (1960).
6. V. M. Khayutin. Fiziol. zh. SSSR, 7, 645 (1958).
7. A. C. Burton and F. Girling, J. physiol. (Lond.), 1951, Vol. 115, p. 57.
8. F. Girling, Amer. jour. Physiol., 1952, 170, 131.
9. D. B. Gordon and A. Nogueira, Circulat. Res., 1962, Vol. 10, t. 1, p. 269.
10. I. H. Page and R. D. Taylor, Circulation, 1950, Vol. 1, p. 1233.
11. Z. Supek and B. Uroic, Biol. glasnik. Hrvatsko pridodose drustvo, 1960, Vol. 13, p. 387.
12. J. Tripod and H. J. Bein, Helv. physiol. pharmacol. Acta, 1960, Vol. 18, p. 394.

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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.

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