# TWO TYPES OF AFFERENT INFLUENCE OF THE MECHANORECEPTORS OF THE SMALL INTESTINE ON THE BLOOD PRESSURE

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Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 50, No. 9, pp. 28-33, September, 1960
Original article submitted August 17, 1959

V. N. Chernigovskii [10, 11] was the first to show that variations in pressure in the vessels of the small intestine were accompanied by corresponding changes in the rest of the blood system. Increasing the pressure in a loop of intestine isolated from the rest of the circulation caused a fall in the blood pressure. A reduction in the pressure of the intestinal vessels produced the reverse reflex change, which suggested that the intestinal vessels constituted a reflexogenous zone mediating blood pressure changes.

Recent experiments [7, 8], in which mechanoreceptor function was studied by increasing the pressure in the lumen of an isolated intestinal loop, showed that there was an arterial pressor response. These results led to the conception of the pressor nature of the response from the intestinal mechanoreceptors. The previous depressor influence from these receptors was now no longer discussed. Thus, the different authors attribute different significance to the signals developed by stimulation of the mechanoreceptors, claiming opposite reflex blood pressure changes. However, when considering the conditions under which the receptors were stimulated by V. N. Chernigovskii and later authors, we have noticed that in the first case the pressure changes were applied to the vascular system of the intestine, and in the second, to the intestinal wall. It is possible that as a result, different afferent endings were stimulated.

It appeared worthwhile to carry out an electrophysiological investigation of potentials developed in the nerves of the intestine and mesentery when the intestinal mechanoreceptors were stimulated in different ways, and to compare the nerve impulses with the reflex blood pressure changes induced. We have attempted to do so in the work reported here.

## METHOD

The experiments were carried out on acute preparations of cats under urethane anesthesia, while perfusing the loop of intestine, which retained only nervous connection with the rest of the body. We caused variation of pressure in the vessels of the intestine in the same way as did V. N. Chernigovskii, by lowering and raising a vessel containing perfusion fluid, or by increasing or decreasing the flow of fluid to the vessels of the intestinal loop by means of a Hoffman clamp. In most experiments, the pressure in the intestinal vessels was increased by injecting Ringer-Locke solution into them through a syringe. The pressure in the isolated intestinal loop was increased by inserting a glass cannula into this section, and connecting it to a closed vessel in which a definite air pressure could be established. For the direct measurement and graphical recording of the changes in intraintestinal pressure, we used a mercury manometer. The electrical potentials from the peripheral ends of the intestinal and mesenteric nerves were recorded with a string oscillograph. At the same time a record was made of the blood pressure in the carotid artery by means of a mercury manometer. The method has previously been described in detail elsewhere [2]. In all, we carried out 22 experiments.

## RESULTS

When recording electrical potentials from the peripheral ends of the cut nerves of the mesenteric plexus, "rapid" impulses are registered which, when the circulatory system is intact, may follow the rhythm of the pulse. When the intestine is perfused, these discharges either do not occur, or occur continuously without any grouping of the impulses. Most authors attribute these impulses to excitation of the Pacinian corpuscles [1, 5, 6, 9, 12, 13, 14].

In our experiments, the increased pressure in the blood vessels to the intestine caused a small fall in arterial pressure and an increased flow of impulses from the Pacinian corpuscles. A reduction in pressure in the intestinal vessels caused a pressor response, and a re-

duced number of impulses. This result led us to propose that impulses from the Pacinian corpuscles had a depressor function [4]. However, the arterial pressure changes are extremely small, particularly when the pressure is reduced. The effect can only be observed under certain conditions. It may occur in a fresh preparation when the receptors have a high excitability, and when the blood pressure is high. When perfusion is continued for a considerable time, reflex blood pressure changes developing in response to variations of blood pressure in the intestinal vascular system become reduced and disappear, and a depressor response may change into a weak pressor reaction. Evidently, this change results from an alteration in the condition of the receptors. An important condition of a development of definite blood pressure changes is that the change in the pressure of the intestinal vessels should be sharp.

The depressor responses illustrated in Fig. 1a were obtained either by rapidly injecting Ringer-Locke solution into the intestinal vessels (traces 1, 2, 3, 5) or by sharply increasing the flow of perfusion fluid by unscrewing the Hoffman clamp (traces 4, 6). However, even when optimal conditions were arranged, it was only occasionally that such extreme depressor responses as are illustrated in traces 5 and 6 (see Fig. 1a) were obtained. A powerful burst of impulses from the Pacinian corpuscles preceded the depressor responses (Fig. 1b).

In this case there was no spontaneous discharge, and impulses were not produced until the Ringer-Locke solution was injected into the intestinal vessels. When the pressure in these vessels was lowered, and the supply of perfusion fluid thereby reduced, there was usually a small but definite increase in the blood pressure (Fig. 2a) and a reduction in the discharge from the Pacinian corpuscles (Fig. 2b).

On increasing the pressure in the lumen of the isolated links of intestine by inflation, a small rise in blood pressure was recorded (Fig. 3a). Besides some increase

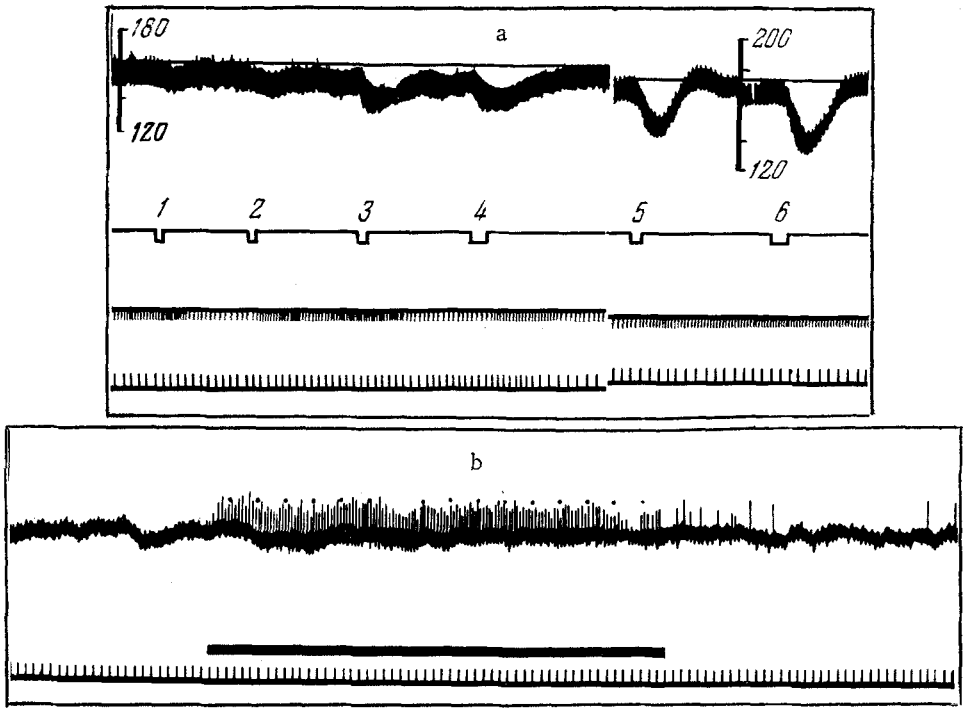


Fig. 1. Reflex changes in the arterial pressure (a) and afferent impulses in the mesenteric nerve (b) caused by increasing the pressure in the intestinal vessels. Curves, from above downward: On the kymogram—blood pressure in the carotid artery; stimulus marker (1 and 2 indicate the injection into the intestinal vessels of 1 ml of Ringer-Locke solution from a syringe; 3 and 5 indicate a similar injection of 5 ml; 4 and 6 show the increased flow of perfusion fluid caused by slackening the Hoffman clamp); perfusion rate indicator; time marker (5 seconds); on the oscillogram—electrical activity of the mesenteric nerve; stimulus marker (injection of 1 ml of Ringer-Locke solution into the vessels from a syringe); time marker (0.02 second).

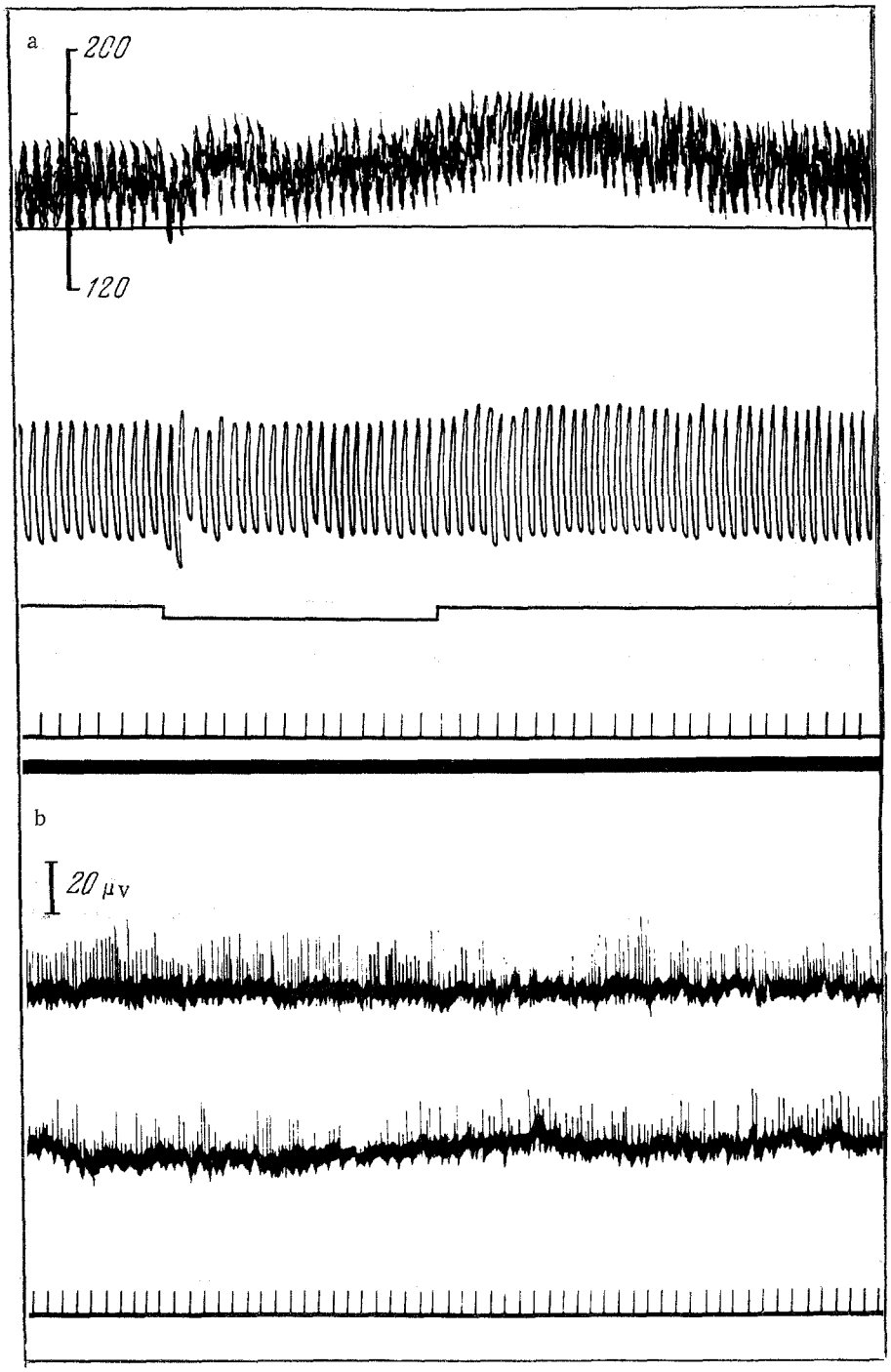


Fig. 2. Reflex changes in arterial pressure (a) and in the afferent impulses from the mesenteric plexus (b) following reduction of the pressure in the intestinal vessels. Curves, from above downward: On the kymogram – blood pressure in the carotid artery; respiration; trace showing time at which perfusion pressure is reduced; time marker (5 seconds); on the oscillogram – initial electrical activity in the mesenteric nerve; activity after reducing the perfusion pressure; time marker (0.02 second).

in the discharge from the Pacinian corpuscles, slow potentials also developed (Fig. 3b); in our experiments they were of quite small amplitude, because they were picked up from comparatively large nerve trunks of the mesenteric plexus, where there is a considerable shunting effect. A more detailed description of these impulses , which, like authors [5, 6], we refer to as "slow," is given by O. N. Zamyatina [6] and by us [2, 3, 4].

Thus, when the intestinal receptors are stimulated mechanically, there is no doubt that different afferent endings are stimulated. Pressure variations in the intestinal vessels cause, in all cases, a change in the discharge from the Pacinian corpuscles. On increasing the pressure in the lumen of the intestine (by inflation), at first the sensory apparatus in the intestinal wall itself is stimulated, and as a result the "slow" impulses are formed. Other workers have previously observed similar potentials in applying strong mechanical stimulation to the intestinal wall [4, 6, 12, 14]. However, under these conditions there is some increase in the discharge from the

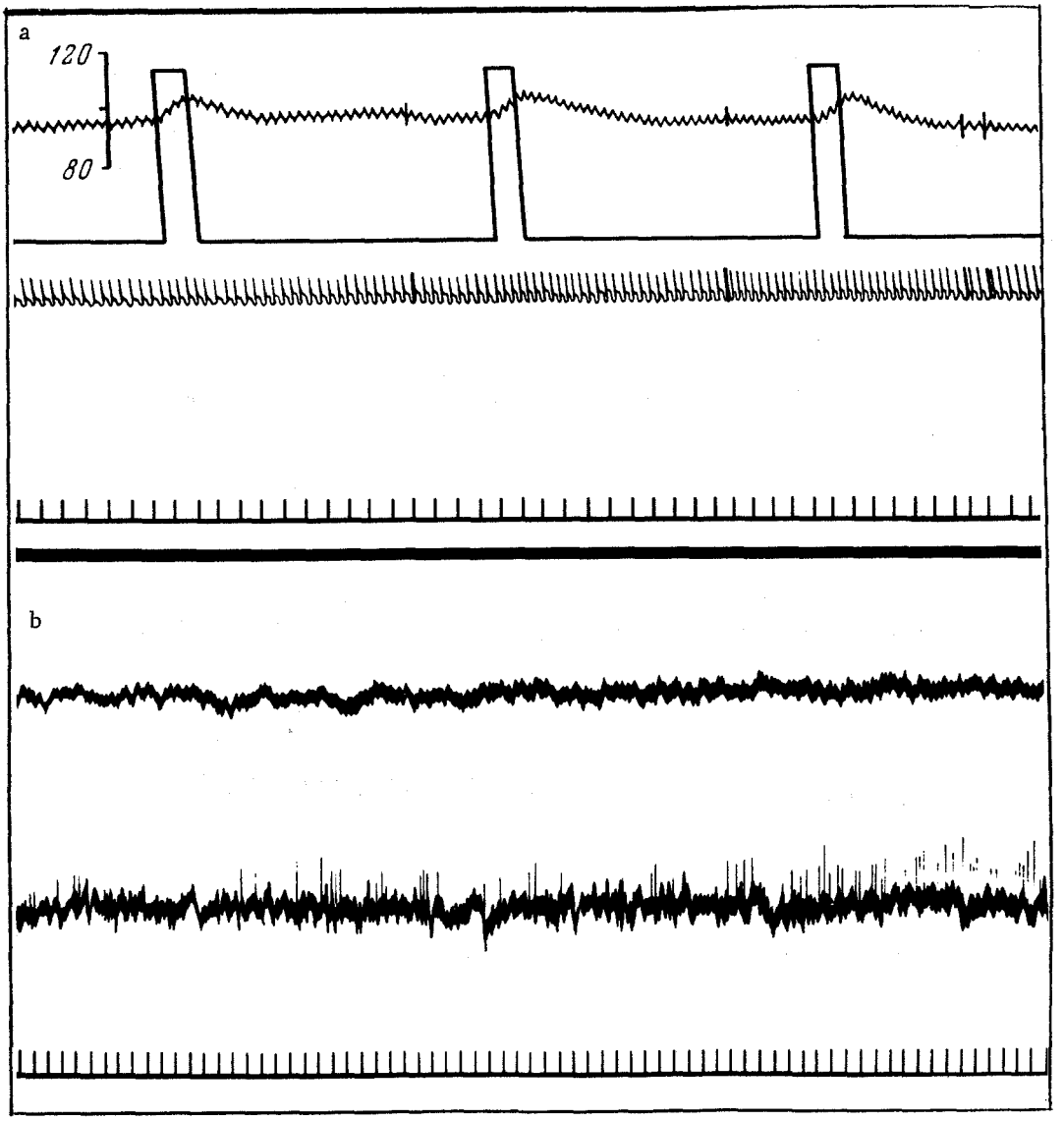


Fig. 3. Reflex changes in arterial pressure (a) and in the afferent impulses from the mesenteric nerve (b) on stretching the intestinal wall by inflation. Curves, from above downward: On the kymogram—blood pressure in the carotid artery; moment of excitation (increasing the pressure in the lumen of the intestine); respiration; time markers: for a) 5 seconds, for b) 0.2 second.

Pacinian corpuscles, because inflation causes some mechanical deformation associated with the displacement of the mesentery.

Despite the well-established relationship between the Pacinian corpuscles and the blood circulation [1, 5, 6, 9, 13], very little work has been done to determine the significance of these impulses in controlling it. Gammon and Bronk [13] were not able to observe any variation in pressure in the greater circulation in response to perfusion pressure variation in the mesenteric artery. Our results enable us to assert definitely that the large sharp increase in the pressure of the messenteric and intestinal vessels causes an increased discharge from the Pacinian corpuscles and a depressor response of the general circulation; on the other hand, decreasing the pressure in the intestinal vessels reduces Pacinian corpuscle activity and leads to an increase in arterial pressure.

Depressor response which develops on inflating the intestine nevertheless brings about some increase in the discharge from the Pacinian corpuscles. This is evidently due to the fact that the "slow" potentials which develop from the receptors of the intestinal wall have a stronger influence on the vasomotor center, and so cause an increase in blood pressure. We have previously demonstrated the relationship between the pressor arterial responses and the "slow" potentials induced by the action of chemical stimuli on the intestinal receptors [2, 4]. O. N. Zamyatina [6] demonstrated an increased rate of discharge from the receptors of the intestinal wall when there was an increased rate of digestion. From our results, it would appear that under these conditions the "slow" impulses cause an increase in arterial pressure and some redistribution in the tone of the peripheral vessels, and so bring about the normal hyperemia of the intestine. However, under the circumstances described [6], there is also an increased discharge from the Pacinian corpuscles, and therefore O. N. Zamyatina attributes the pressor influence from the intestine to an increase in the "rapid" impulses. Our results have led us to the opposite conclusion, and we think that impulses from the Pacinian corpuscles reduce the pressor response induced by the "slow" impulses, and operate together with other buffer zones in bringing about a return to normal of the increased blood pressure. It is probable, therefore, that the intestine is both a pressor and a depressor zone, and that the nature of the response to increasing the pressure in it depends both on the level of activity of the Pacinian corpuscles, and on the relative discharges of the "rapid" and "slow" impulses from the receptors of the intestinal wall.

Possibly some such conception may enable us to understand why dilatation of the duodenal wall, which contains such a large number of Pacinian corpuscles, so seldom causes pressor responses to occur; usually, there are either no changes, or else there is a fall in blood pressure. At the present time we are further investigating the ideas which have been proposed here.


## SUMMARY

An increased pressure in the intestinal vessels causes an increased discharge from the Pacinian corpuscles and reduces systemic arterial blood pressure. A decrease of pressure in the intestinal vessels reduces the discharge rates from the Pacinian corpuscles and causes a rise of blood pressure. Stretching the intestinal wall stimulates the receptors enclosed in it and increases arterial blood pressure. The relationship between intestinal stimulation and blood pressure depends on the interaction between the discharges from the Pacinian corpuscles and from the receptors of the intestinal wall.


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