[9] H. Richter and H. Schrockschnadel, Arch. f. exptl. Pathol. Pharmakol., 1938, Bd. 191, S. 23-29.

[10] H. Schaeffer, Ergebn. d. Physiol., 1950, Bd. 46, s. 71.

SIGNIFICANCE OF THE REFLEX FROM THE SUPERIOR VENA CAVA IN THE REGULATION OF BLOOD AND LYMPH CIRCULATION

V. V. Petrovsky

From the Chair of Normal Physiology (Director: Prof. V. V. Petrovsky, Bashkir Medical Institute, Ufa

(Received March 16, 1956. Presented by V. N. Chernigovsky, Member Acad. Med. Sci. USSR)

The findings of a number of members of our Department (Z. G. Valeeva, D. I. Smirnov, K. V. Kovanov, G. H. Kotova), relating to functional connections between the lymphatic and blood vessels, have been presented in an earlier paper [1].

In the present paper we present some data obtained from further investigation and study of the significance of the reflex from the superior vena cava in the regulation of blood and lymph circulation.

We measured the pressure of lymph in the thoracic duct of a dog, using a mercury manometer, with simultaneous registration of arterial and venous (external jugular vein) pressure, with the object of ascertaining the strength of reflex contraction of the lymphatic vessels. In some cases we perfused the cervical lymphatic duct. Our experiments showed that intravenous injection of large volumes of physiological saline raises arterial pressure by 5-40 mm Hg, and venous pressure from 0-2 to 8-12 cm of water; at the same time the cervical lymphatic duct contracted to a state of complete spasm, and the pressure in the thoracic duct rose to 40-80 mm Hg (Figure 1).

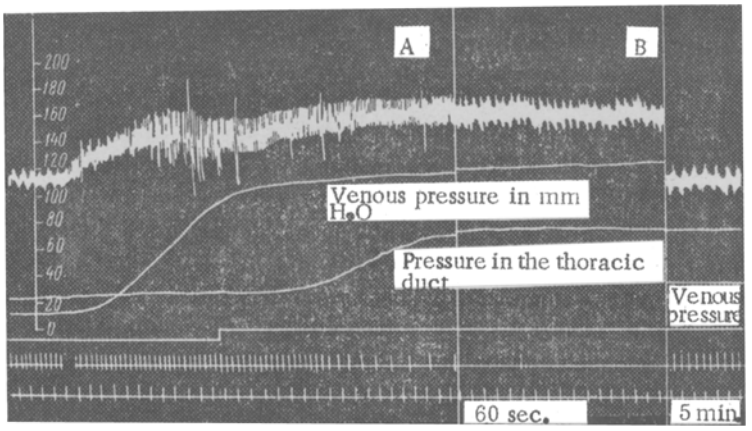

Fig. 1. Effect of intravenous injection of 400 ml of physiological saline on arterial and venous pressure and, pressure in the thoracic lymph duct, and on the tonus of the cervical lymph duct. A) Kymograph stopped for 1 minute; B) kymograph stopped for 6 minutes. Explanation of tracings, from above down: blood pressure in the carotid artery; lymph pressure in the thoracic duct, in mm Hg; venous pressure (right external jugular vein), in mm of water; flow of fluid through the cervical lymphatic duct (number of drops); time marker (5 seconds).

It might be supposed that thoracic duct pressure rose as a consequence of passive distention, due to rise in capillary pressure. In order to check this possibility we measured thoracic duct pressure during introduction of adrenaline. As is evident from Figure 2, the pressure in the thoracic duct, initially 40 mm Hg, rose rapidly after injection of adrenaline to a valueof 75 mm. The rises in lymphatic duct and blood pressure took place simultaneously. The maximum rise in blood pressure also coincided in time with that of lymph. Venous pressure, which was measured separately in this experiment, was 0 cm of water before injection of adrenaline, and rose to 12 cm of water during its action. Since the rise in arterial pressure was synchronous with that in the thoracic duct, it is unlikely that the latter could have been due to intensification of lymph formation, which could have led to over-filling of the lymphatic vessels with lymph, and hence to a rise in lymphatic pressure.

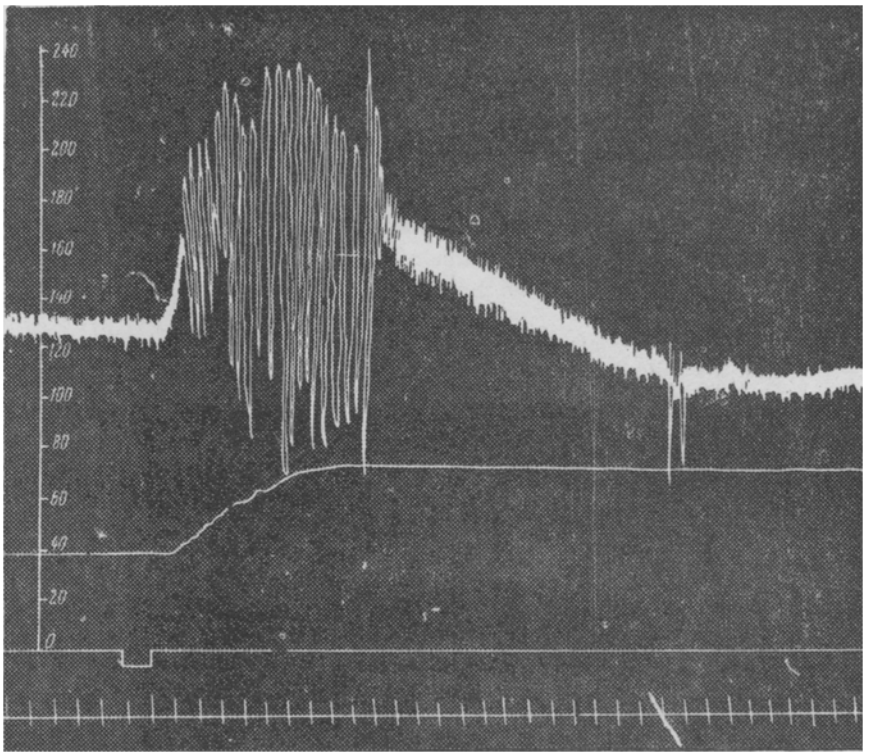

Fig. 2. Effect of intravenous adrenaline (0.000015) on blood pressure and pressure in the thoracic duct.
Explanation of tracings, from above down: blood pressure in the carotid artery: pressure in the thoracic duct, in mm Hg; signal showing time of introduction of adrenaline; time marker (5 seconds).

In a second series of experiments we measured the pressure in the cisterna chyli, into which drain a number of lymph ducts draining the viscera. We raised venous pressure by injection of physiological saline and adrenaline, and in this way raised the pressure in the cisterna chyli to over 60 mm Hg.

The results of our experiments provide evidence of the absence of lymphatic-venous anastomoses; if these existed such rises in thoracic duct pressure as we registered could not have been possible.

It may be supposed that, under conditions of normal blood circulation with raised venous blood pressure, causing increased resistance to the flow of lymph, a reflex contraction of the lymphatic vessels takes place, leading to expulsion of part of their contents into the blood vessels. This causes an increase in the volume of circulating blood. This need not necessarily lead to still further increase in venous pressure, and hence to still further constriction of the lymph ducts. As is known, rise in venous pressure gives rise to a reflex acting on the heart (the Bainbridge reflex). The heart beat becomes more frequent, the minute volume rises, and venous pressure falls. In correspondence with the increased blood volume the capacity of the vessels of the systematic

circulation rises, in response to stimulation from the reflexogenic zones of the carotids and aorta. This explains the significance of the reflex from the superior vena cava under conditions of normal activity of the cardiovascular system.

Venous pressure also rises when the activity of the heart is depressed, but the organism is not able to counteract this tendency by reflex stimulation of cardiac activity. Raised pressure in the superior vena cava evokes a pressor reflex acting on the lymphatic vessels, and impeding the flow of lymph. Lymph is at first retained in the parts of the body most distant from the heart, and then, with increasing venous pressure and striction of lymphatic vessels, edema makes its appearance in other parts of the body. Hulse [3] has reported that in patients displaying progressive edema the thoracic duct and the cisterna chyli are empty, but fill up with lymph as soon as resorption of edema fluid begins. According to Hulse, the incidence of this effect is so regular that it is possible to deduce from it what stage of the edematous process is present, without clinical examination of the patient.

This reasoning applies, obviously, only to edema associated with raised venous pressure. Thus factors tending to lower venous pressure will bring about a weakening of the pressor reflex, and so favor transfer of edema fluid to the blood stream.

Rusnyak [2] has in a recently published paper drawn attention to certain inconsistencies with Starling's theory of causation of edema, such as, for example, the beneficial effect of ligating the inferior vena caca of patients with cardiac decompensation. Not only does this not aggravate the edema of the lower extremities, as would be expected from Starling's theory, but on the contrary, it lessens it.

Rusnyak does not enter into a consideration of the reasons for this effect. We would, on the basis of our experimental findings, suggest the following explanation. Ligature of the inferior vena cava involves a diminution in the flow of blood to the right auricle, at least until the establishment of an adequate collateral circulation. As a result, the pressure in the right auricle and the superior vena cava falls, and the pressor reflex becomes weaker. In addition to this, the pressure in the part of the vein below the ligature rises, causing reflex dilatation of the lymphatic channels, which will tend still further to inhibit the pressor reflex from the superior vena cava. In consequence, the patency of the lymphatic vessels is restored, and the edema of the lower extremities disappears. We would emphasize that Rusnyak also attaches significance to the lymphatic vessels in development of edema.

LITERATURE CITED

[1] V. V. Petrovsky, Fiziol. Zhur. SSSR, No. 3 (1954).

[2] I. Rusnyak, Klin. Med. No. 1, 23-35 (1954).

[3] W. Hulse, Klin. Wschr., 1923, No. 2, S. 63-65.

INFLUENCE OF SPLENIC INTEROCEPTORS ON BLOOD PRESSURE AND RESPIRATION IN ONTOGENESIS

L. A. Palgova

From the Chair of Normal Physiology (Director: Prof. A. P. Polosukhin), Kazakh Medical Institute, Alma-Ata

(Received November 18, 1955. Presented by V. N. Chernigovsky, Member Acad. Med. Sci. USSR)

We know from the literature [6] that the spleen of adult animals (dogs, cats) contains receptors, which when stimulated evoke reflex changes in respiration, arterial and venous pressure, and flow of lymph.

The present paper is devoted to a study of the effects of splenic interoceptors on blood pressure and respiration in ontogenesis.