

REFLEX CHANGES IN RESPIRATION DURING A RISE IN PRESSURE IN THE THORACIC DUCT

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Translated from *Byulleten' Éksperimental'noi Biologii i Meditsiny*, Vol. 56, No. 7,
pp. 20-24, July, 1963

Original article submitted June 16, 1962

The highly developed autonomic nerve supply of the lymphatic system, and especially of the thoracic duct, has been demonstrated by anatomical investigations [6, 7, 9, 10, 14, 27]. This has been confirmed physiologically by observations noting the changes in the lumen of the lymphatic trunks during application of various stimuli [12, 13, 17, 22, 24, 25, 26], and also the reflex changes in the arterial pressure and in the lumen of certain lymphatics in response to mechanical and chemical stimulation of the thoracic duct [3, 4, 8]. These facts have led some workers [6] to include the thoracic duct among the "reflexogenic organs of the vascular system" and to claim [17] that, together with other lymphatic vessels, it takes part in the reflex regulation of the circulation.

Because of the close functional connection between the circulation of the blood, respiration, and the lymph flow we suggested that the thoracic duct is also concerned in the interoceptive regulation of respiration, more especially so because reflex changes in respiration have been demonstrated experimentally during stimulation of the receptors of the lymph glands [20] and of the cisterna chyli [5].

EXPERIMENTAL METHOD

Acute experiments were conducted on 18 adult dogs weighing from 9 to 20 kg, anesthetized with morphine and hexobarbital or morphine and urethane. In all the experiments the lateral pressure in the left common carotid artery was recorded with a mercury manometer and the respiration was recorded by means of a Marey's capsule, connected to one section of an endotracheal cannula.

In ten experiments the lateral pressure in the left femoral and left external jugular veins was measured with linear water-air manometers and recorded by means of a Marey's capsule. In the same experiments we used the technique previously described by us [18] to record the lateral pressure in the thoracic duct. To stimulate the receptors of the thoracic duct, the outflow of lymph from the vessel into the venous system was temporarily halted, and instead of the lateral pressure in the duct, the terminal pressure was measured, its tracing showing an increase as lymph accumulated in the thoracic duct. After the terminal pressure in the duct had increased by 30-80 mm water, the clamp preventing the drainage of lymph from the thoracic

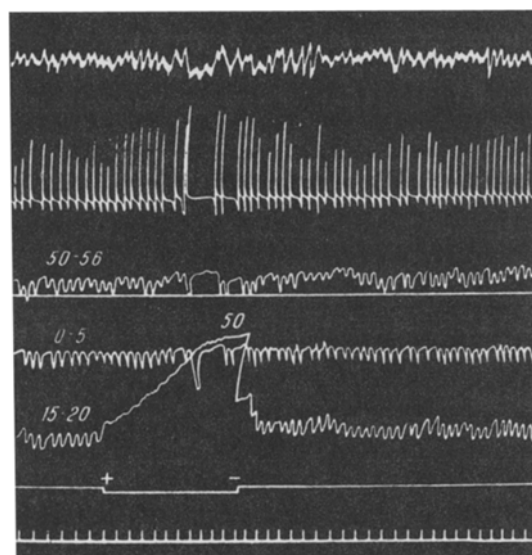


Fig. 1. Changes in respiration during an increase in the pressure in the thoracic duct. Significance of the curves (from above down): arterial pressure; respiration; pressure in the left femoral vein; zero line of the arterial pressure; pressure in the left external jugular vein; lateral pressure in the thoracic duct; marker of occlusion of lymph flow into the venous system (+) and of restoration of the flow (-); time marker (5 sec). The numbers below the curves denote the pressure (in mm water).

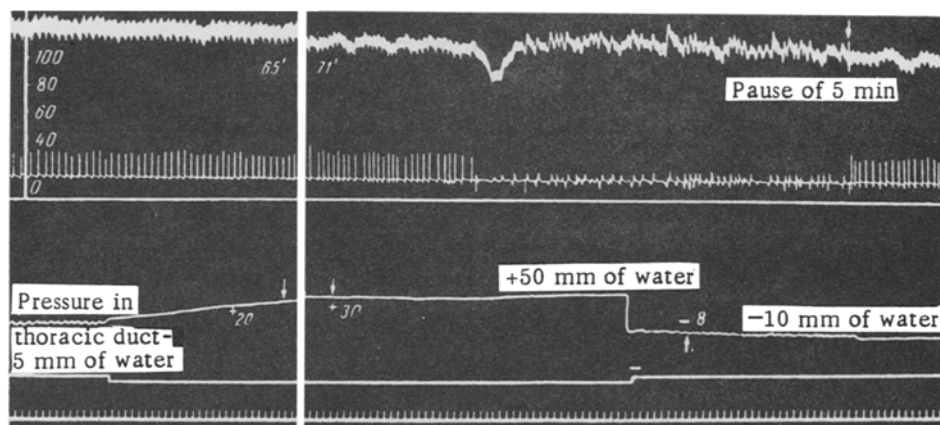


Fig. 2. Appearance of periodic disorders of respiration during prolonged interruption of the outflow of lymph from the thoracic duct to the venous system. Significance of the curves (from above down): arterial pressure; respiration; zero line of arterial pressure; pressure in thoracic duct; marker of occlusion of outflow of lymph into the venous system (+) and of restoration of its flow (-); time marker (5 sec). The numbers on the curve of pressure in the thoracic duct denote its value in mm water.

duct into the venous system was removed and the lateral lymph pressure was again recorded.

In five experiments the thoracic duct was perfused with oxygenated Tyrode solution (temperature 38°) under a pressure of between 4 and 8 mm Hg. The afferent cannula joined by a rubber tube to the pressure vessel was inserted into the cisterna chyli in the abdomen, in a cranial direction, from 3.4 cm below the crura of the diaphragm; the efferent cannula was introduced into the mouth of the thoracic duct. A mercury manometer was introduced between the afferent cannula and the pressure vessel to check the pressure under which the Tyrode solution entered the thoracic duct. Each drop of perfusate flowing from the duct was recorded on the drum of a kymograph. To stimulate the receptors of the thoracic duct in these experiments, the pressure vessel was quickly raised so that the pressure under which the Tyrode solution entered it rose by 20-35 mm Hg. The vessel was then lowered to its original level. The receptors of the thoracic duct were stimulated for 70-90 sec in these experiments.

EXPERIMENTAL RESULTS

A rapid (in the course of 50-70 sec) increase in the pressure within the thoracic duct by 30-80 mm water after interruption of the drainage of lymph into the venous system in eight experiments led to a deepening of the respiratory excursions of the chest wall and a slowing of the respiration rate, these effects taking place 10-12 sec after the onset of stimulation (Fig. 1). In four experiments this phenomenon was combined with a simultaneous decrease in the arterial pressure, a phenomenon observed after stimulation of the receptors of the thoracic duct and cisterna chyli by several workers [3, 4, 5, 8], while in another four experiments changes in respiration were observed in the absence of changes in the arterial pressure. In our opinion, the latter findings may be evidence of the greater lability of the reflex from the receptors of the thoracic duct on respiration compared with the reflex on the blood vessels, especially since no changes were observed in the height of the pressure in the femoral and jugular veins in our experiments during the period of stimulation.

The view that the point at which interoceptive influences on respiration are exerted may be the receptors of the thoracic duct is confirmed by the results of five perfusion experiments. A rapid increase in the pressure (by 10-23 mm Hg) of the perfusate entering the thoracic duct in all these experiments was followed by the slowing and deepening of respiration, developing 7-10 sec after the onset of stimulation.

After the preliminary division of the vago-sympathetic trunks in the neck, the changes in the respiration of the dogs (5 experiments) during the period of increased pressure in the thoracic duct were preserved, mainly in the form of a decrease in the amplitude of the respiratory excursions of the chest wall.

These results demonstrate, firstly, that the vago-sympathetic trunks take part in the transmission of interoceptive influences from the receptors of the thoracic duct on respiration, and secondly, that these nerves are not the only route along which these influences are spread.

In five experiments the lymph flow along the thoracic duct of the dogs was small, as a result of which the tracing of the terminal pressure after occlusion of the outflow of lymph into the venous system rose slowly. In two of these 5 experiments respiration was unchanged for 12-15 minutes of stimulation, while in the other three changes developed 7-10 min after the beginning of stimulation, and took the form of the appearance of periodic respiration. For example, interruption of the outflow of lymph into the venous system (Fig. 2) led after 8 min to a deepening of the respiratory excursions of the chest wall, accompanied by a fall in the arterial pressure. Two minutes later, periodic disturbances of respiration suddenly developed, probably as a result of a change in the functional state of the respiratory center in the medulla [1, 2, 21] following the arrival of a powerful stream of impulses in the central nervous system from the receptors of the lymphatics. Six minutes after the restoration of the lymph circulation, the normal depth and rhythm of the respiratory movements were restored.

These reflex changes in respiration during stimulation of the receptors of the thoracic duct by an increased pressure are extremely important, for slow and deep respiratory movements promote the rapid movement of lymph along the main lymphatic trunks [15, 23], and besides leading to dilatation of their lumen they contribute to the removal of congestive manifestations in the lymphatic system.

The dilatation of the lymphatics during an increase in pressure in the thoracic duct is demonstrated by the fall in the lateral pressure of lymph in the duct by comparison with the initial lateral pressure after temporary occlusion of the outflow of lymph from the duct into the venous system (Fig. 2). It is also confirmed by the experiments in which the thoracic duct was perfused. Stimulation of the receptors by an increase in pressure under which Tyrode solution entered the thoracic duct led to an increase in the flow of perfusate through the duct, even after the pressure reservoir had been lowered to its original level. It can hardly be suggested that this was a residual passive dilation of the thoracic duct, for its wall contains numerous elastic fibers [10, 11, 19] and the outflow of perfusate from the duct during the period of stimulation of the receptors by an increased pressure was not interrupted. Finally, evidence in favor of a probable functional dilatation of the lymphatics in these conditions is given by observations [4] showing that when the pressure in the thoracic duct is increased the lumen of the jugular lymphatic vessel undergoes reflex dilatation, and vice versa.

These results suggest that the receptors of the thoracic duct play an essential role in the interoceptive regulation of respiration. To some extent they explain also the onset of forced respiration in patients with venous stasis, and the consequent impaired drainage of lymph into the vascular system. The observations noting the appearance of periodic respiration when the lymph flow along the thoracic duct is disturbed must impress on surgeons [16] the need for greater care when advising the total and rapid packing of the operation wound or ligation of the thoracic duct if lymphorrhea should develop during operations in the region of the neck, for the collateral channels draining lymph into the venous system probably cannot compensate at once for the disturbances of the lymph circulation after occlusion of the thoracic duct.

SUMMARY

A rapid pressure rise in the thoracic duct following arrest of lymph outflow into the venous system or raising the pressure vessel in conditions of perfusion of thoracic duct leads to the reflex slowing and deepening of respiration. A slow rise in the lymph pressure in the duct following disturbance of its circulation may provoke pathological types of respiration.

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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.
