VASOMOTOR EFFERENT EFFECTS ON THE LUNGS (STIMULATION OF THE VAGUS NERVE IN RABBITS)

B. I. Mazhbich and L. P. Osadchuk

UDC 612.227.2:612.819.912

Changes in the blood volume of the posterior lobes of the rabbit's lung were studied in response to regular electrical (50 Hz, 2.5 V, 10 sec) stimulation of the peripheral ends of the vagus nerves divided in the neck. In the central part of the lobe the predominant response to stimulation was an increase in the blood volume (the vascular component), whereas at the periphery the predominant response was a decrease in the blood volume (the cardiac component).

The role of the efferent vasomotor nerves, especially the vagus nerve, in the control of the pulmonary circulation is not a new problem [2, 7, 11, 13, 15], but a final solution is still awaited [3, 15]. Among the reasons for discrepancy among the experimental results are the possibility of variation of the pulmonary circulation through changes in cardiac activity and bronchial tone [1, 3, 8, 10, 12], the use of animals of different species for the investigation, and the severe operative trauma during the experiment [3, 7].

In the investigation described below, an attempt was made to differentiate the active vascular response after minimal operative trauma and the passive changes in the pulmonary circulation in response to electrical stimulation of the peripheral end of the vagus nerve divided in the neck.

EXPERIMENTAL METHOD

Three series of experiments were carried out on adult rabbits anesthetized with Nembutal (0.04 mg/kg, intravenously), immobilized with paramyon, and maintained on artificial respiration. Recordings were made of the ECG, and air pressure in the trachea, and the electroplethysmograms [4] of the central (near the apices of the segments of the lobe) and peripheral (posterobasal segment of the lobe, closer to its costophrenic border) portions of the posterior lobes of the lung were recorded in 27 rabbits during regular stimulation of the nerve trunks (50 Hz, 2.5 V, 10 sec). In six animals the vagus nerve was stimulated for 3-7 min after total, irreversible cardiac arrest, and in eight animals it was stimulated during compression of one branch of the pulmonary artery (thoracotomy).

Changes in the blood volume of the lungs observed electroplethysmographically were expressed in ml/100 g weight of the organ.

EXPERIMENTAL RESULTS AND DISCUSSION

The character of the changes in the blood volume of the lungs depended on where the electroplethysmogram was recorded and on which of the vagus nerves was stimulated.

Laboratory of Electrophysiology, Institute of Physiology, Siberian Branch of the Academy of Sciences of the USSR, Novosibirsk. (Presented by Academician of the Academy of Medical Sciences of the USSR, V. V. Parin.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 72, No. 12, pp. 3-6, December, 1971. Original article submitted December 21, 1970.

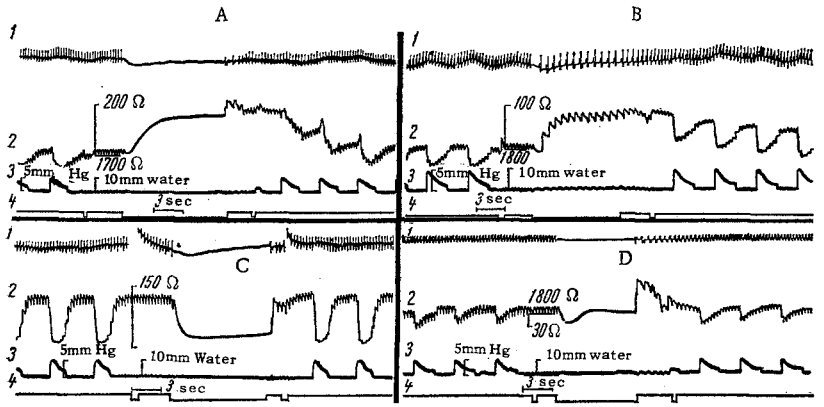

Fig. 1. Changes in blood volume in the "central" and "peripheral" parts of the posterial lobes of the lungs in response to electrical stimulation of the vagus nerve: 1) ECG; 2) electroplethysmogram; 3) air pressure in trachea; 4) signal line. First marker on signal line denotes stopping artificial respiration and airtight closure of air passages, second marker shows stimulation of vagus nerve, and the third shows resumption of artificial respiration; calibration signals and values of original electrical resistance of the lungs are shown on the left; calibration signal (in mm water) applies only to time of stimulation.

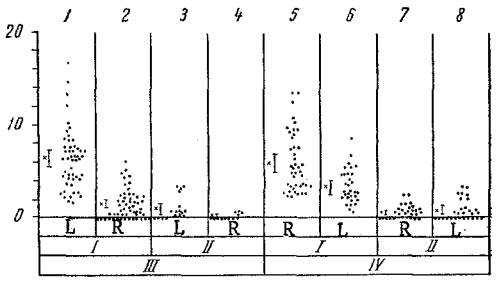

Fig. 2. Phase of increase in blood volume of lungs during stimulation of left (L) and right (R) vagus nerves: I) central area, II) peripheral area, III) left posterior lobe, IV) right posterior lobe. Each point denotes result of one stimulation; crosses give mean values.

In response to stimulation of the ipsilateral nerves, an increase in the blood volume only (in 81% of cases) was observed in the "central" parts of the lobes, or the increase in blood volume was preceded by a slight decrease (19% of cases) (Fig. 1A, B). It was immaterial whether complete cardiac arrest was observed during stimulation, or whether the heart rate was merely slowed.

In the "peripheral" parts of the lobes in 13% of cases the blood volume was only increased during stimulation of the ipsilateral nerves, in 47% of cases it was reduced, and in 40% of cases the response was biphasic in character but with definite predominance of the phase of a reduced blood volume (Fig. 1C, D). In the biphasic responses the phase of a decrease in blood volume always preceded the phase of an increase.

The changes in electrical resistance (the volume) of the lungs in rabbits in response to vagus nerve stimulation are thus the result of two simultaneous reactions in different directions. One of these reactions, the first phase (a decrease in blood volume), is an indirect effect of vagal stimulation through the heart.

If the vagus nerve was stimulated after compression of the ipsilateral branch of the pulmonary artery, the phase of a decrease in blood volume was not observed in any of the 48 cases regardless of whether the electroplethysmogram was recorded in the central or the peripheral portion, whereas the phase of increased blood volume remained. A decrease in blood volume likewise never took place in experiments in which the nerve was stimulated after total cardiac arrest. In other words, if for any reason connected with the experimental conditions the vagus effect on the heart, especially on the right ventricle, cannot be transmitted to the pulmonary vessels or is absent, the phase of reduced blood volume is not found.

The second phase (increased blood volume) during stimulation of the ipsilateral nerves is most marked in the central parts of the lobes (Fig. 2, columns 1 and 5). It was also well-marked in response to stimulation of the contralateral nerves (Fig. 2, columns 2 and 6), but significantly smaller than in the first case ($P < 0.001$).

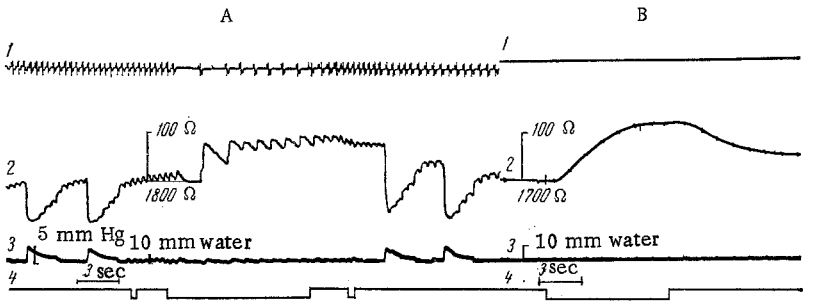

Fig. 3. Changes in blood volume in the right posterior lobe of the lung in response to electrical stimulation of the ipsilateral vagus nerve during clinical death: A) original response; B) 5 min after complete cessation of cardiac activity. Remainder of legend as in Fig. 1.

Electroplethysmograms recorded at the periphery of the lobes of the lungs showed that the phase of increased blood volume was weaker during stimulation of the corresponding nerves (Fig. 2, columns 3, 4, 7, and 8) and was significantly lower than in the central areas ($P < 0.001$).

The increase in blood volume in response to vagal stimulation is regarded as the active vascular component of the response. It cannot be attributed to a simple increase in the inflow of blood from the left atrium, in which the pressure may be slightly increased as a result of the accompanying bradycardia [9, 14]. Experiments on animals with cardiac arrest (clinical death) showed that the electroplethysmographic response of an increase in blood volume at the moment of vagal stimulation still remains in this case (Fig. 3). The results given in Fig. 2 also show that stimulation of the contralateral nerves (the central areas of the lobes) evokes a substantially smaller increase in blood volume than stimulation of the ipsilateral nerves ($P < 0.001$), which would not happen if the observed picture was "cardiac" (left-atrial) in origin.

After airtight closure of the air passages the pressure in them at the time of stimulation remained unchanged (in 70% of cases), or increased very slightly, by not more than 2 mm water. This suggests that the changes observed in the electroplethysmogram cannot be attributed to a response of the bronchial musculature (changes in the air content in the lungs), for during stimulation the electroplethysmographic changes were accompanied by a very slight increase in air pressure in the trachea, whereas the same or even smaller changes in the electroplethysmogram during artificial inspiration were accompanied by a substantial increase (by 50–70 mm water) in the air pressure in the trachea (Fig. 1). Moreover, the change in pressure in the trachea observed during stimulation was always in the same direction, while the electroplethysmographic effects were variable in direction. The change in blood volume, being small compared with the volume of air in the lungs, evidently led to very small and inconstant changes of intratracheal pressure.

Consequently, the effect on the pulmonary hemodynamics mediated through the heart is not the only effect of electrical stimulation of efferent fibers of the vagus nerve, but a vascular response is also induced [2, 3, 6]. However, it is only in the central part of the pulmonary lobe in rabbits, just as in dogs [5], that this vasomotor effect is functionally significant. This effect diminishes progressively from the center toward the periphery of the lobe. Attempts to evaluate the vasomotor response in the lungs from changes at the entrance to and exit from this vascular region have thus not always proved successful, perhaps for this very reason.

LITERATURE CITED

1. D. P. Dvoretskii, Fiziol. Zh. SSSR, No. 3, 337 (1969).
2. V. S. Kupriyanov, Nervous Regulation of the Tone of the Pulmonary Vessels, Doctoral Dissertation, Ufa (1966).
3. Ya. A. Lazaris and I. A. Serebrovskaya, The Pulmonary Circulation [in Russian], Moscow (1963), p. 125.
4. B. I. Mazhbich, Electroplethysmography of the Lungs [in Russian], Novosibirsk (1969), p. 43.

5. B. M. Shershevskii, The Pulmonary Circulation [in Russian], Moscow (1970), p. 142.
6. J. L. Ardisson, C. Grimaud, P. Bernard, et al., J. Physiol. (Paris), 56, 275 (1964).
7. J. L. Ardisson, C. Grimaud, and M. Gastaud, Sem. Hôp. Path. Biol., 15, 417 (1967).
8. G. S. Campbell, F. J. Haddy, W. Z. Adams, et al., Am. J. Physiol., 158, 96 (1949).
9. I. de B. Daly, Physiol. Rev., 149 (1933).
10. I. de B. Daly, Quart. J. Exp. Physiol., 46, 257 (1961).
11. I. de B. Daly, Arch. Exp. Path. Pharmak., 240, 431 (1961).
12. A. P. Fishman, Med. Thorac., 19, 438 (1962).
13. W. Harrison and A. A. Liebow, Circulation, 5, 824 (1952).
14. M. Hochrein and C. J. Keller, Arch. Exp. Path. Pharmak., 166, 229 (1932).