BLOOD VOLUME AND DISTENSIBILITY OF THE PULMONARY VESSELS AFTER DIVISION AND STIMULATION OF THE VAGUS NERVES

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KEY WORDS: lungs; electroplethysmography; blood volume; distensibility.

Nervous regulation of pulmonary vascular tone remains a su[illegible]for discussion [7, 8]. Research into the elastic properties of these vessels has been una[illegible]n. These properties are closely connected with the concept of vascular tone and they are [illegible]ged by stimulation of autonomic nerves, although in the investigations cited mainly the effects of the sympathetic innervation were examined [9, 10], and as a rule no attention was paid to regional differences in the pulmonary circulation. The present writers showed previously [2, 4] that distinct regional differences in the character of the response to stimulation of parasympathetic and sympathetic nerves are observed within the same lobe of the lungs.

The object of this investigation was to study the blood volume and elastic properties of the pulmonary vessels in response to electrical stimulation of the peripheral ends of the vagus nerves divided in the neck.

EXPERIMENTAL METHOD

Experiments were carried out on 15 adult mongrel dogs weighing 10-18 kg with an intact chest, anesthetized with pentobarbital (0.02 g/kg). The animals were fixed lying on their back, and the trachea and jugular vein were exposed in the neck. A catheter was introduced under roentgenologic control through the jugular vein into the artery of the posterior lobe of the lung and the blood pressure was recorded with an electromanometer (Elema). Natural respiration was stopped by injection of listhenon. Under fluoroscopic control the detector probe of an electroplethysmograph was introduced into the trachea until it became wedged in the small branches of the bronchi in the same lobe of the lung (in its central or peripheral part). Both cervical vagosympathetic trunks were divided at the level C4 and the peripheral ipsilateral ends of the divided nerves were stimulated by an electric current (50 Hz, 2.5 V, 10 sec). Artificial respiration was stopped at the time of stimulation. According to the results of an electroplethysmographic investigation [1, 3, 5] the blood volume per unit volume of the lobe of the lung ($\underline{V_k}$), and its pulse increase ($\underline{\Delta V}$), determined by averaging the measurements in 3-5 cardiac cycles, were estimated. Changes in blood volume during stimulation of the nerves also were estimated and the distensibility ($\underline{D}$) of the pulmonary vessels was calculated as the change, in per cent, of blood volume per unit (cm H_2O) of blood pressure:

$$\underline{D} = \frac{\underline{\Delta V}}{\underline{\Delta P} \cdot \underline{V_k}} 100\ \%.$$

Since it follows from the investigations cited above [1, 3, 5] that

$$\underline{\Delta V} \approx 1.5 \frac{\Delta\gamma}{\gamma_{\underline{k}}} \text{ and } \underline{V_k} \approx 1.4 \frac{\gamma}{\gamma_{\underline{k}}},$$

$$\text{we have: } \underline{D} \approx \frac{\Delta\gamma}{\underline{\Delta P} \cdot \gamma} 100\ \%,$$

where $\Delta\gamma$ is the increase in specific electrical conductivity of the lungs during systole, γ the specific basic electrical conductivity of the organ. However, the values obtained for

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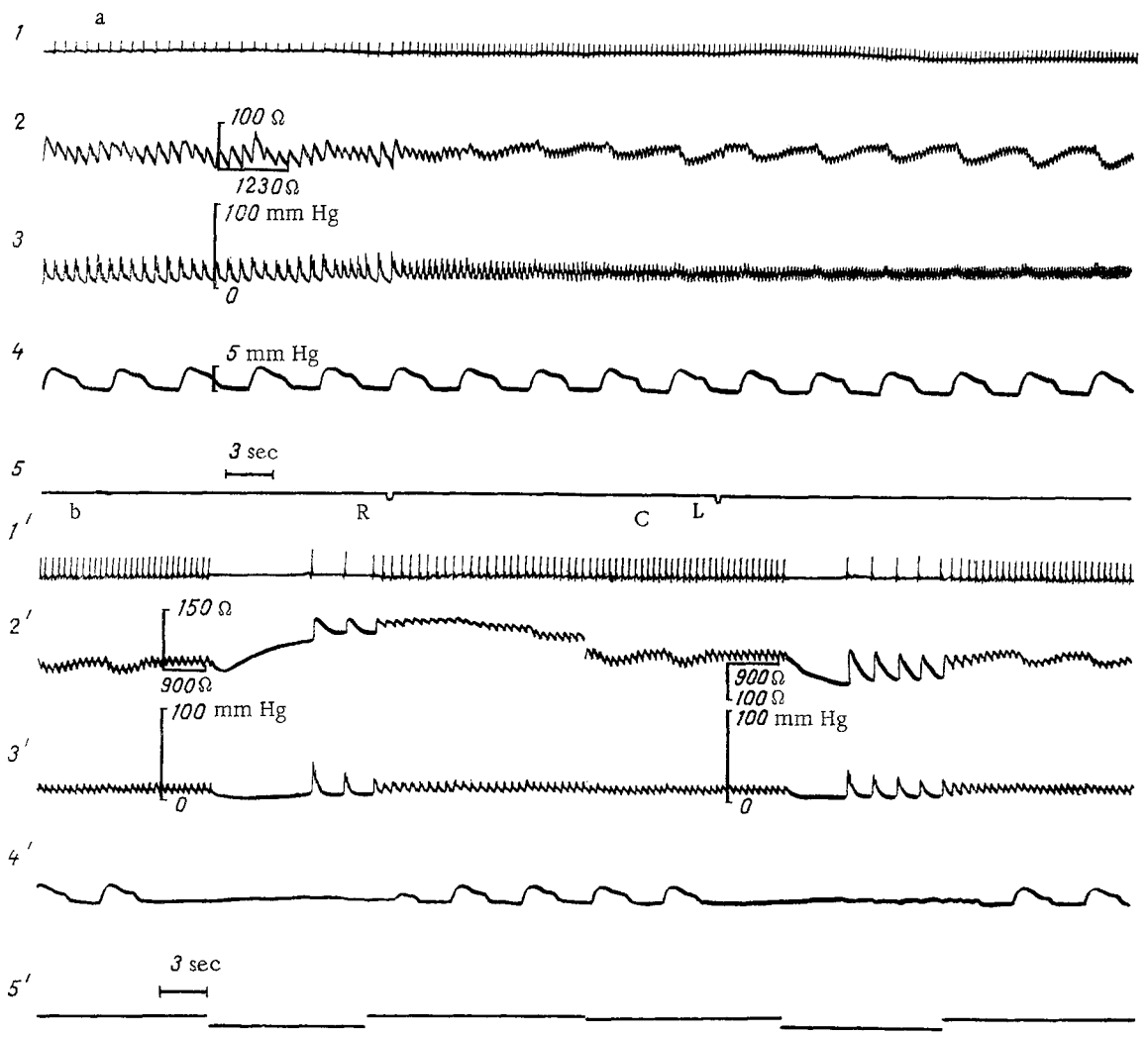

Fig. 1. Effect of vagotomy (a) and division (b, c) of distal ends of divided vagus nerves by electric current. On left — central part, right — peripheral part of lobe. 1) ECG, 2) electroplethysmogram, 3) pressure in lobar branch of pulmonary artery, 4) pneumogram (artificial respiration), 5) marker of intervention: a) division of vagus nerves (R — right, L — left), b) stimulation of vagus nerve.

distensibility of the vessels cannot be compared directly with data in the literature obtained by other, more complex methods [12], for in the case under consideration the pulse increase in blood volume is an integral parameter, reflecting the state of the blood volume in small vessels from arteries to veins, and not only in the arterial part of the vascular bed. The pulse increase in pressure, on the other hand, applies only to the arterial part of the vascular bed, and for that reason the values mentioned will be lower than those given in the literature.

The experimental results were subjected to statistical analysis and the significance of differences was determined by Student's t test. The difference method was used in some experiments.

EXPERIMENTAL RESULTS

Division of the vagus nerves (Fig. 1a) caused, simultaneously with tachycardia, a marked fall in the pulse oscillations of the electroplethysmogram, due to a corresponding reduction in distensibility of the pulmonary vessels. However, comparison of the mean values of this parameter before and after vagotomy showed that the difference was not statistically significant, for in some experiments vagotomy had no effect, or only a minimal effect, on this parameter. Its mean values before and after vagotomy, both in the central part of the lobe and at its periphery, are shown in Fig. 2 (1, 2).

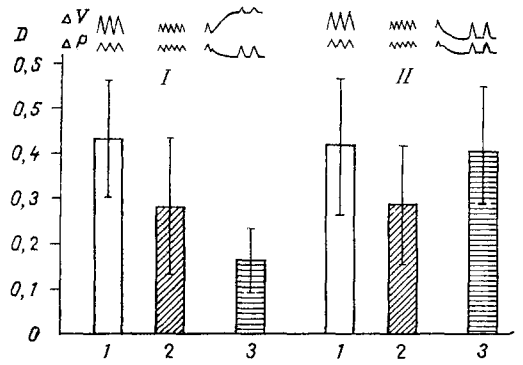

Fig. 2

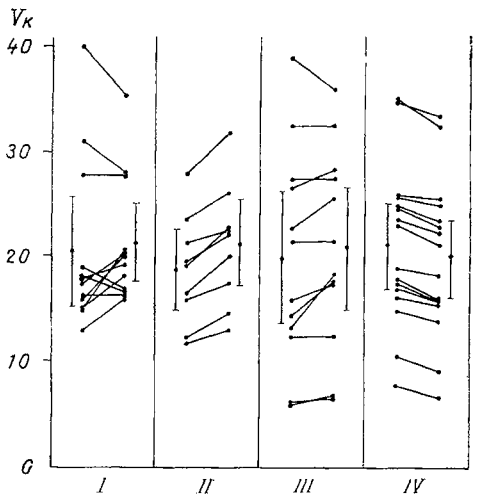

Fig. 3

Fig. 2. Mean values and confidence intervals for distensibility of pulmonary vessels in intact dogs after division and during electrical stimulation of vagus nerves. I) Central part, II) peripheral part of lobe of lung. 1) Before, 2) after vagotomy, 3) during stimulation of peripheral ends of ipsilateral vagus nerves at height of response of increase (I) and decrease (II) in blood volume.

Fig. 3. Mean values and confidence intervals for blood volume (in ml/100 cm^3 volume of the organ) of posterior lobes of dog lung after division and during electrical stimulation of vagus nerves. I-II) Central part of lobe of lung; III, IV) peripheral part of lobe of lung. In I and III, points on left — before vagotomy, on right — after vagotomy. In II and IV points on left — before stimulation, on right — during stimulation of ipsilateral nerves.

The blood volume in the pulmonary vessels during vagus nerve stimulation changed on average by 13% in the central part of the lobe and by 5% at the periphery (Fig. 3, II, IV). Calculation by the difference method showed that the results in both cases (Fig. 1b, c) were statistically significant ($P < 0.001$). However, the mechanism of the change in blood volume was quite different in these two cases. Reduction of the blood volume at the periphery of the lobe was caused by a cardiac mechanism, i.e., it was due to sudden bradycardia (Fig. 1c) or cardiac arrest during stimulation, accompanied by a fall of blood pressure in the pulmonary artery. In the central part of the lobe, despite the same fall of pressure, the blood volume was increased, and this can be explained only by active, neurogenic lowering of the tone of the vessel wall. This corresponds to modern views of the vasodilator function of the vagus nerve fibers relative to the pulmonary vessels [7, 8]. Artificial electrical stimulation of the vagus nerve probably leads to very substantial relaxation of the vessels in the center of the lobe. Whatever the case, at the height of this response a further increase in volume of the vessel during pulse oscillations is limited, as is shown by the substantial fall in distensibility of the pulmonary vessels to values only half as high as initially (Fig. 2, 3), at a time when the blood volume is already considerably increased. Meanwhile, against the background both of passively reduced (as a result of the abrupt bradycardia or temporary cardiac arrest) blood volume (periphery of the lobe) the distensibility of the vessels, also estimated from the ratio of pulse increases in blood volume to pulse increases of pressure, was 1.5 times higher than the initial distensibility, or three times higher than the distensibility of the pulmonary vessels at the center of the lobe at the height of the response of increased blood volume.

On the basis of the facts described above it can be postulated that at rest neurogenic vasodilator influences on the pulmonary vessels may be virtually absent or very weak, in agreement with views expressed previously [6]. However, in other situations, during muscular work [6] or changes in the gravitational load, or for other reasons, for example, the role of this mechanism may become much more important and it may alter the state of elasticity of the pulmonary vessels.

The increase in the relative blood volume of the lungs during electrical stimulation of the nerves may reach on average 13% of the initial value, and this may account for the significant increase in the blood flow through the lungs without any appreciable rise of pressure in the pulmonary arterial bed [11], for the blood flow, other conditions being the same, depends on the fourth power of the width of the lumen of the blood vessel. An important role in this situation is probably played also by the central part of the lobe.

Further increase in blood volume is evidently limited by the lowering of distensibility of the pulmonary vessels, and it could hardly be possible without an appreciable rise of blood pressure in the pulmonary circulation.

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IONIC MECHANISM OF THE EXCITATORY ACTION OF NORADRENALIN ON SMOOTH-MUSCLE CELLS OF THE RABBIT PULMONARY ARTERY

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KEY WORDS: pulmonary arteries; noradrenalin; transmembrane ionic currents.

The excitatory effect of noradrenalin (NA) on smooth-muscle cells of the pulmonary artery is manifested as their contraction, to an amount which depends on the NA concentration. If the NA concentration is 10^{-8}-10^{-7} M, this contraction is not accompanied by any significant changes in membrane potential (MP). Under the influence of higher doses of NA the contractile response is increased and depolarization of the smooth-muscle cell membrane is observed [4, 5]. Some workers also have reported an increase in the electrical excitability of muscle cells of the pulmonary artery under the influence of NA, by a mechanism not yet explained [3, 4, 7]. Investigations of the turnover of radioactive Na^+, K^+, and Cl^- ions have shown that membrane permeability for them is increased under these conditions [5]. However, if membrane conductance is judged from the value of the electrotonic potentials, under different experimental conditions both an increase [5] and a decrease [3, 4, 8] in conductance have been found under the influence of large doses of NA.

The aim of this investigation was to continue the study of the mechanism of the excitatory action of NA on smooth-muscle cells of the pulmonary artery. Attention was directed mainly to the study of the mechanism of noradrenalin depolarization and of the increase in electrical excitability of the membrane.

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