MECHANISM OF CHANGES IN THE RESPIRATORY AND CARDIOVASCULAR REFLEXES FROM THE LUNGS ASSOCIATED WITH INTRAPULMONARY STEAM BURNS

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Aviado and Schmidt [4] observed that the characteristic respiratory reflexes in dogs to inhalation of ammonia, to stretching of the lungs, and to intravenous injection of veratrine disappeared following inhalation of steam. They suggested that this effect was due to injury to the corresponding pulmonary receptors under the influence of the high temperature [4, 5]. In that case the intrapulmonary burn may be important as a method of securing selective blocking of the pulmonary receptors.

The object of the present investigation was to study this problem.

EXPERIMENTAL METHOD

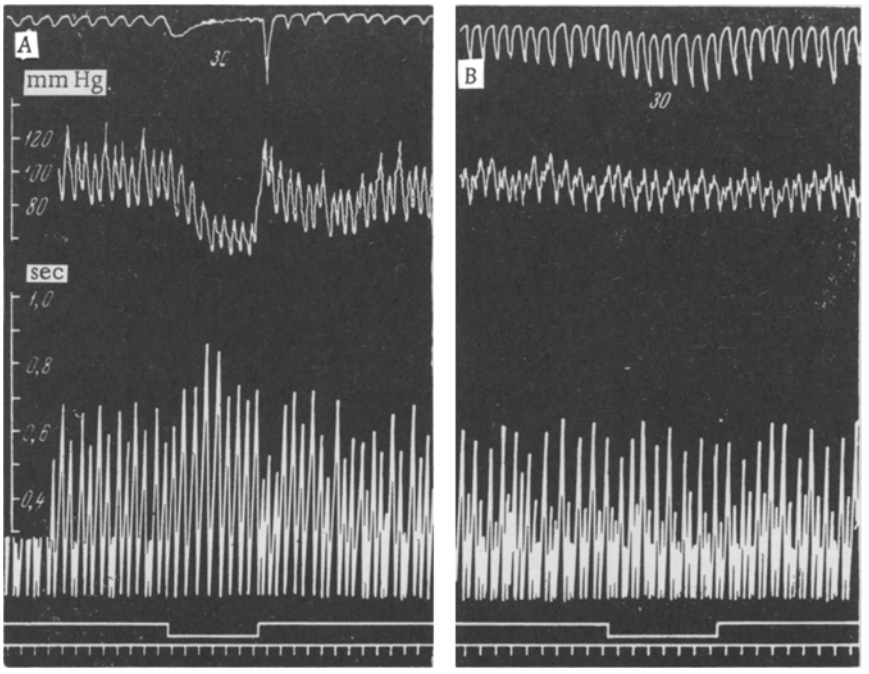
Experiments were conducted on 17 dogs. The animals were anesthetized with morphine (1-6 mg/kg) and urethane (0.8-1.0 g/kg), with the addition of chloralose (10-50 mg/kg) when necessary.

A burn of one or both lungs were caused by steam. In the first case the large flask, in the bottom of which the water was boiled, was connected for 20-120 sec to the intubation tube inserted into one bronchus of the tracheotomized animal; in the second case it was connected to the tracheal cannula. Steam was introduced into the respiratory tract at a pressure of 20-40 mm Hg. The burns were produced repeatedly (15 times) at intervals of 5-15 min.

Before and after exposure to steam, repeated tests were made for the presence of a respiratory [8] and cardiovascular [1] reflex to stretching the lungs, a respiratory reflex to the unilateral [2] and bilateral [8] collapse of the lungs, a cardiovascular reflex to unilateral and bilateral collapse of the lungs [2], and a respiratory [6] and cardiovascular [3] reflex to an increase of pressure in the pulmonary vessels. The lungs were stretched by increasing the pressure in one or both lungs, and collapsed by pumping air from them. To increase the pressure in the pulmonary vessels, a left-sided thoractomy was first performed, and the bronchial and pulmonary arteries and veins on the left side were ligated. Distally to the point of ligation, an artery of the left lung was connected by a polyethylene cannula and tube to a Bobrov's apparatus. The thorax was then closed and the animal breathed spontaneously. In these conditions, when the pressure in the Bobrov's apparatus was raised, a similar increase in the pressure within the blood vessel of the isolated lung was produced.

The respiratory movements (mechanical transmission), the arterial pressure (mercury manometer), and the heart rate (Fleisch's intervalograph [7]) were recorded on smoked paper. To record the respiratory movement in the chest of the experimental animal lying on its back, a strong thread was fixed to the region of the xiphoid process and passed over a pulley to be attached to the pen. Fleisch's apparatus records the interval (with an accuracy of 0.01 sec) between two systoles (which Fleisch calls the "pulse time") in the form of a vertical line, the height of which is directly proportional to the length of the interval, i.e., inversely proportional to the heart rate.

In four dogs the changes in the blood temperature in the thoracic aorta were recorded. For this purpose the detector of an electrothermometer was introduced into the thoracic aorta through the femoral artery under x-ray control.



Effect of stretching the right lung on respiration and the circulation before (A) and after (B) two intrapulmonary burns of the right lung (dog No. 2). From top to bottom: respiration, arterial pressure, "pulse time" (explanation in text), duration of stretching, time (3 sec).

EXPERIMENTAL RESULTS

Repeated intrapulmonary burning with steam led to inhibition, and in most cases to abolition, of all the investigated reflexes (see figure). Inhibition of the reflexes took place as a result of 1-4 burns, and abolition as a result of 1-11 burns. In some cases, despite frequent repetition of the burns, total abolition of the reflexes did not occur. This result was related neither to the depth of anesthesia nor to the duration of the burns. An important factor here was evidently individual variations in the pulmonary circulation, for in experiments on the isolated lung the first burn as a rule caused abolition of the reflexes.

With each burn the blood temperature in the thoracic aorta rose by 0.3-3.0°. This was probably caused by the warming of the blood, mainly in the pulmonary capillaries, by the direct action of the steam. In burns of the lung isolated from the general circulation, the blood temperature was unchanged.

Approximately .5 h after the first burn, a frothy fluid began to appear from the respiratory passages of the injured lung. At thoractomy, edema of the lung was found. It may be postulated that edema of the lung caused a disturbance of the gas exchange and of the chemical properties of the blood. According to some reports [9], an increase in the blood temperature itself leads to a fall in the pH and a rise in the pCO_2 of the blood.

The results suggest that the abolition of the investigated reflexes may have been caused by central mechanisms activated by the direct effect of the warmed and chemically changed blood on the central nervous system. However, some features do not support this suggestion. The intrapulmonary burn did not modify the reflex influences of the carotid sinuses on respiration and the circulation. This shows that the burn did not cause inhibition of the respiratory, vasomotor, and vagus centers. Further, paralysis of the pulmonary reflexes also developed after a burn of the isolated lung, when the mechanism of the increase in temperature and a change in the chemical properties of the blood was for practical purposes excluded.

Consequently, inhibition of the pulmonary reflexes associated with intrapulmonary burns must be attributed to changes in the corresponding receptors. This is also confirmed by the fact that the investigated reflexes, when abolished by a burn of one lung, could be produced afresh in a completely unchanged form by stimulation of the receptors of the opposite, uninjured lung.

In some cases a partial restoration of the excluded reflexes took place for 12-20 min. On the other hand, frequently the excluded reflexes could be evoked again by means of a stronger stimulus. This suggests that the injury to the receptors of the lungs arising as a result of intrapulmonary edema is functional in character.

The results of these investigations thus show that intrapulmonary edema can inhibit or exclude the function of all the known pulmonary receptors.

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