NERVOUS CONTROL OF BLOOD-GAS CHANGES FOLLOWING FOCAL PULMONARY LESIONS

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It has been shown in many experiments, chiefly clinical, that there is very little relationship between the extent of pulmonary damage and the gaseous blood changes resulting from it. As a rule, the oxygen value of the arterial blood returns to its usual level before recovery from the pulmonary damage has been effected [7, 8, 9, 11].

In previous experiments [2, 3], we have shown the importance of the nervous system in compensating respiratory disturbances caused by pulmonary damage. In these experiments, respiratory function was studied chiefly by recording respiratory movements. A blood-gas analysis was performed only in isolated cases. Nevertheless, the blood gases constitute one of the most important indices of respiratory function.

In the present work we have investigated the part played by the nervous system in compensating blood-gas changes following focal pulmonary injury.

EXPERIMENTAL METHODS

The experiments were carried out on unanesthetized rabbits. All 45 animals were used in the experiments. They were placed on their backs and fixed to a support. The damage was effected by introducing 3 ml of water heated to 80-90°C through the chest wall. The advantage of this method is that the damage is immediately effective and there is no incubation period as there is in the case of bacterial pneumonia, or when irritant substances are introduced through the respiratory passages, and there is no possibility of any direct action on the respiratory centers. Samples of arterial blood were obtained from the femoral artery, and venous blood was obtained from the right heart. The blood from the heart was obtained by means of a catheter introduced through the jugular vein. The blood was collected under oil. Blood-gas analysis was carried out using Van Slyke's apparatus, not later than 30 minutes after drawing the blood.

At the beginning of the experiment, the femoral artery and jugular vein were exposed under local anesthesia. After thirty minutes, the first samples of blood were taken. Thirty minutes after the blood had been taken, the lung damage was inflicted and a sample of blood was taken immediately. The next portion of blood was taken 20 to 60 minutes later, and again, as a rule, two to three hours after infliction of the lung damage. A record of the respiration rate was also taken.

EXPERIMENTAL RESULTS

The oxygen saturation of arterial blood has an average value of 95%. Incomplete oxygen saturation of the arterial blood would normally be ascribed to a physiological atelectasis of the pulmonary tissue.
As the experiments lasted 4-5 hours, and as the collection of blood caused a certain anemia, control experiments were carried out to find what effect these factors had on the blood gases. It was found that the effect was quite insignificant, there being only a small reduction in the venous oxygen content, as a result of which the arterio-venous difference increased by 1-2 vol. %.

Blood-gas changes following localized pulmonary damage in rabbits with no damage to nervous system. In these experiments 30 rabbits were used. Blood-gas analysis (carried out immediately after the damage had been inflicted) gave the following results. The oxygen content of arterial blood was reduced by 1.2-3.2 vol. %, The oxygen saturation of arterial blood was reduced by 5.5-15.6%; the CO₂ content was reduced by 1.8-3.8 vol. %.

In the venous blood, the oxygen content was reduced by 2.2-4 vol. %. The CO₂ content was reduced by 2-4 vol. %.

The arterio-venous difference and volume remained within normal limits.

After 20-60 minutes from the time of inflicting the pulmonary damage, the arterial blood was 4-13% below complete saturation. After 2-3 hours, the oxygen saturation, with only a few exceptions, returned to normal. The oxygen content of venous blood remained 1.2-2.9 vol. % below normal, and this caused an increase in the arterio-venous difference of 0.5-2.1 vol. %. The CO₂ content in both arterial and venous blood was reduced by 2-5 vol. %.

Previous experiments have shown that localized pulmonary damage in rabbits, caused by the same method used in the present experiments, led to an increase in the frequency and depth of respiration for the first 15-20 minutes, after which the respiratory movements returned to normal. We found the same thing. We also found that at the same time there is a hypoxia. This hypoxia disappears 2-3 hours after the infliction of the damage, at a time when the injury not only has not healed, but in fact is continuing to develop.

Thus, the present experiments show that the degree of oxygen saturation of arterial blood (see diagram), which is normally taken as one of the principal indices of respiratory function, returns to its normal value before repair of the pulmonary injury has taken place. Postmortem examination carried out after the restoration of respiratory function revealed hemorrhages and edema in the damaged portion, and sometimes regions of atelectasis.

Blood-gas changes following localized pulmonary damage in vagotomized rabbits. In previous experiments in this laboratory [2], we have shown the great importance of the vagi in compensating respiratory damage following localized pulmonary injury. The present investigation of the blood gases in these animals demonstrates this compensatory process particularly clearly.

In these experiments 10 animals were used. A determination of the blood gases was first made in the normal animal, and then repeated, first 30 minutes after resection of both vagi in the neck, then immediately after infliction of the pulmonary damage (which was effected one hour after cutting the vagi), and then 2, 3, and, in some cases, 24 hours later. Section of the vagi in the neck was carried out very rapidly under local anesthesia. After the operation, the breathing became slower and deeper. There was an increase in muscle and facial muscle tone. Thirty minutes after sectioning of the vagi, the breathing became quieter, more regular, and rather more rapid. There was usually no change in the blood gases. These observations agree with other published data [6, 10]. However, it was found that the power to compensate for localized pulmonary damage was considerably reduced in vagotomized animals.

After the pulmonary damage had been effected, the breathing again became deep and labored, but the frequency remained practically unchanged. This agrees with previously published observations [2]. In the arterial blood, the oxygen content was reduced by 1-3 vol. %, and the oxygen saturation was reduced by 5-12%. The oxygen capacity remained within normal limits. In the venous blood, the oxygen content was reduced by 2-3.5 vol. %, and this caused an increase in the arterio-venous difference of 0.5-2 vol. %. The CO₂ content in both arterial and venous blood was reduced by 2-5 vol. %.

Blood-gas analyses carried out after 2, 6, and 24 hours, revealed considerable hypoxia. The oxygen saturation of arterial blood was reduced by 5-16% (see figure). Besides the hypoxia, there was a considerable hypocapnia: the CO₂ content of the arterial blood was reduced by 5-8 vol. %, while the arterio-venous difference