NERVOUS CONTROL OF BLOOD-GAS CHANGES FOLLOWING FOCAL PULMONARY LESIONS

Z. N. Sergeeva

From the Laboratory of Comparative Pathology of the Nervous System
(Head – Prof. S. I. Frankshtein) Institute of Normal and Pathological Physiology
(Director – Active Member AMN SSSR Prof. V. N. Chernigovsky) AMN SSSR, Moscow

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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. In 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 collections 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 CO_2 content was reduced by 1.8-3.8 vol. %.

In the venous blood, the oxygen content was reduced by 2.2-4 vol. %. The CO_2 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 CO_2 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 methods 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.

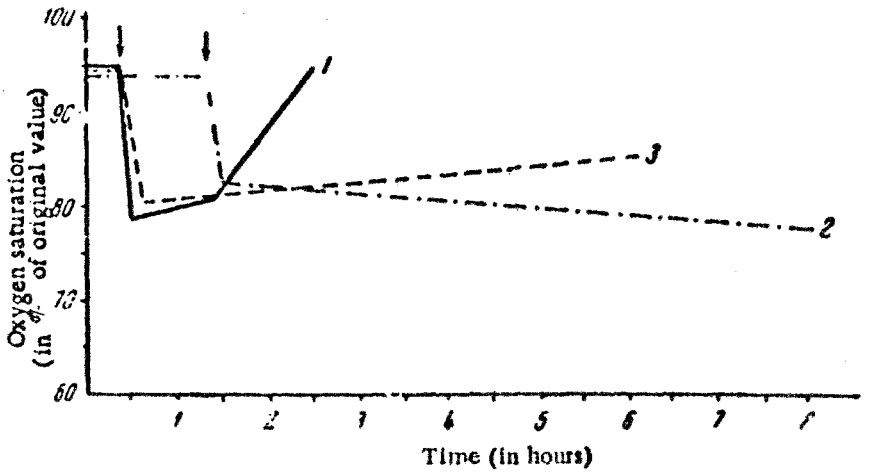
Thus, the present experiments show that the degree of oxygen saturation of arterial blood (see diagram), which is normally taken as one of the principal indices of respiratory function, returns to its normal value before repair of the pulmonary injury has taken place. Postmortem examination carried out 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 CO_2 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 CO_2 content of the arterial blood was reduced by 5-8 vol. %, while the arterio-venous difference



Changes in the degree of oxygen saturation of arterial blood (in % of original value) after damage to lungs by scalding: (1) in animals without interference to nervous system; (2) in vagotomized animals; (3) in animals with impaired nervous trophic functions. The arrows show the times at which the lung damage was effected.

was increased by 1-2 vol. %. The animals died after 24 hours. Postmortem examination revealed a pulmonary edema which was particularly marked on the damaged side.

Investigation of blood gases following localized pulmonary damage in rabbits with impaired nervous trophic function. In the control animals of the first experiments, the blood gases returned, as a rule, to their normal value 2 to 3 hours after the pulmonary damage had been effected. Certain animals which were an exception to this rule were very carefully investigated. We noticed that two rabbits in whom the blood gases did not return to their normal value within 5 to 6 hours showed effects of impaired trophic function in the limbs.

A. D. Speransky [5] and his co-workers [1, 4] showed that a dystrophy of this type is not a localized phenomenon, but reflects a general disturbance of nervous trophic function.

It seemed reasonable to suppose that in animals with signs of dystrophy the failure to effect compensation was due to a generalized impairment of trophic function. We therefore started another experiment to investigate compensation of respiratory function in animals with experimentally induced impairment of nervous trophic function. The simplest form of impairment consists of section of the sciatic nerve.

Five animals were used for this experiment. The sciatic nerve was cut, under local anesthesia, in the upper third of the thigh. After two to three weeks, ulcers developed on the foot.

The blood gases of these animals, determined before and immediately after effecting the pulmonary damage, were within normal limits. However, the oxygen saturation of the arterial blood of these animals did not return to normal within 6 hours. The blood remained 7.4-10.1% below saturation, while in the intact animals the oxygen saturation returned to normal within 2-3 hours after infliction of the pulmonary damage (see figure).

These results show that the hypoxia cannot be explained in the terms of the failure of the oxygen to diffuse into the damaged zone. This follows from the fact that the oxygen saturation of the blood in animals with an intact nervous system is restored to its normal value before the pulmonary damage has been repaired.

It is clear that the oxygen saturation of the blood does not depend directly on the change in respiratory movements or on the pulmonary ventilation. After the pulmonary damage has been inflicted, the breathlessness continues for 15-20 minutes, while the hypoxia lasts for 2-3 hours.

We considered above the possibility that the hypoxia is due to a small part of the blood continuing to circulate through the damaged portion of the lung without becoming oxygenated. We must also suppose that in these present experiments the hypoxia arose in this way.

Lung damage in normal animals caused a hypoxia of a relatively short duration of 2-3 hours, while in vagotomized animals the condition became progressively worse.

In addition to the nervous mechanism controlling pulmonary ventilation and blood circulation through the lungs, the general trophic action of the nervous system is also involved to a considerable extent. As our experiments have shown, elimination of this trophic function hinders the development of compensatory changes.

SUMMARY

In focal injury of lung tissue in experiments on rabbits, the restoration of the gaseous content of the blood takes place when the pathological foci are not only present, but are even in a condition of development. The restoration of the normal content of blood gases is due to the change in the relationship between the ventilation and circulation of the lungs. Resection of the vagus nerves hinders this mechanism. Due to that, injury of the lungs in animals with resected vagus nerves induces progressive hypoxemia. Disturbance of the nerve trophics, likewise reduces the ability of the organism to compensate the disturbance of respiration, caused by focal injury of the lungs.

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