FACTORS INTERFERING WITH OXYGENATION OF THE BLOOD IN PNEUMOTHORAX

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We have shown previously [6] that processes which cause respiratory changes (as affecting the general level of pulmonary ventilation, its evenness, and coordination of the respiratory movements), may lead to arterial hypoxia; it was also shown that, of these factors, the principal one was that of impaired respiration. The changes in blood oxygenation were brought about, not only by altered pulmonary ventilation, but by a second important factor– namely, the flow of blood in the lesser circulation.

In the present work we have studied the effect on blood oxygenation of changes in the pulmonary circulation under various conditions. As a method of altering pulmonary ventilation as well as circulation, we used unilateral pneumothorax (open and closed).

Pneumothorax causes a marked discoordination between the ventilation and blood supply to the lungs, and therefore, may appear to be a cause of arterial hypoxia. In fact, with open unilateral pneumothorax, one lung collapses, and ventilation in it ceases entirely. Under these circumstances there is a redistribution of blood in the lesser circulation, and blood from the right ventricle flows through the vessels to the second lung, which continues to function; therefore, the ventilation of this lung may be insufficient to saturate the flow of blood, which has increased twofold [4, 7]. Also, it is not known to what extent the blood flow to the collapsed lung has decreased; this factor must also be of significance in the development of hypoxia.

From their very nature, the phenomena which develop in a closed pneumothorax must differ from those in the open operation: According to modern views, in a closed pneumothorax the lungs continue to perform respiratory movements [2, 8, 9, 10]; however, it is possible that even in a closed pneumothorax the normal relationships between ventilation and pulmonary circulation are disturbed.

It seemed worthwhile to learn how arterial hypoxia occurs in pneumothorax; we therefore attempted to determine the importance both of changes in respiration and in the redistribution of blood in the lesser circulation as affecting the development of hypoxia.

METHOD

The experiments were performed on rabbits under urethan or morphine anesthesia. Rabbits were used because with them it is possible to dispense with artificial respiration, since the two pleural cavities are separate: When the cavity on one side is opened, the lung on the opposite side continues to function; the closed pneumothorax was also made unilaterally. Recordings were made of the respiratory movements, pulmonary ventilation, blood pressure, and arterial blood-oxygen saturation (this was done by means of a photoelectric instrument applied directly to an artery).

RESULTS

The experiments showed that when one pleural cavity was opened, the respiratory movements of the thorax increased; the greatest increase was in the inspiration, indicating an expansion of the thorax (Fig. 1a). Despite the increase in respiratory movements, pulmonary ventilation was reduced, sometimes more than twofold (see Fig. 1a). Thus, while the blood supply to the functional lung was improved, its ventilation did not change, and might even have been reduced. By itself, entry of air into a pleural cavity did not cause any great change in blood pressure (see Fig. 1a); only in cases when opening the cavity was associated with a sufficiently intense mechanical stimulation of the pleura was there any fall in blood pressure (followed by recovery).

To determine whether there were changes in the pulmonary circulation, we used an indirect index, which was the degree of arterial blood-oxygen saturation. The reasoning was that if, when the lung collapses, the blood flow through it is stopped completely, the blood-

oxygen saturation, which will have fallen after effecting the pneumothorax, ought after a time to recover partially (partially, because in the second lung some disturbance of blood oxygenation is to be expected). If part of the blood continues to circulate through the vessels of the collapsed lung, it will become mixed with blood which has passed through the functional member, and in this way a reduced arterial blood-oxygen concentration will be maintained. In this case, when the circulation to the collapsed lung is artificially blocked by clamping the main artery supplying it, it is to be expected that the blood-oxygen saturation will be increased.

Immediately after opening a pleural cavity, there was a marked reduction in the saturation of the blood (see Fig. 1a). For the most part, the saturation was either maintained at this reduced level for a considerable time (until the end of the experiment, i.e., for several hours), or else even continued to fall; only in a few experiments did it recover partially.

When a changeover was made from breathing air to breathing oxygen, arterial saturation increased considerably, but it hardly even reached the level corresponding to that obtaining when oxygen was breathed before the pneumothorax. When the artery supplying the collapsed lung was clamped, there was usually a further increase in arterial oxygen saturation (see Fig. 1b); this result indicated that before clamping, some of the blood continued to pass through the collapsed lung. The phenomenon was observed repeatedly during a single experiment for a considerable time after opening a pleural cavity (for several minutes, and sometimes for several hours after the collapse of the lung). E. M. Kreps and V. I. Voitkevich [3] observed a similar phenomenon during pneumonectomy in human subjects: On ligaturing the pulmonary artery (35 minutes after effecting deep pneumothorax), the saturation of the arterial blood was increased by 10-12 %; this fact can be explained only by supposing that blood from the collapsed lung which had not been oxygenated was pre-

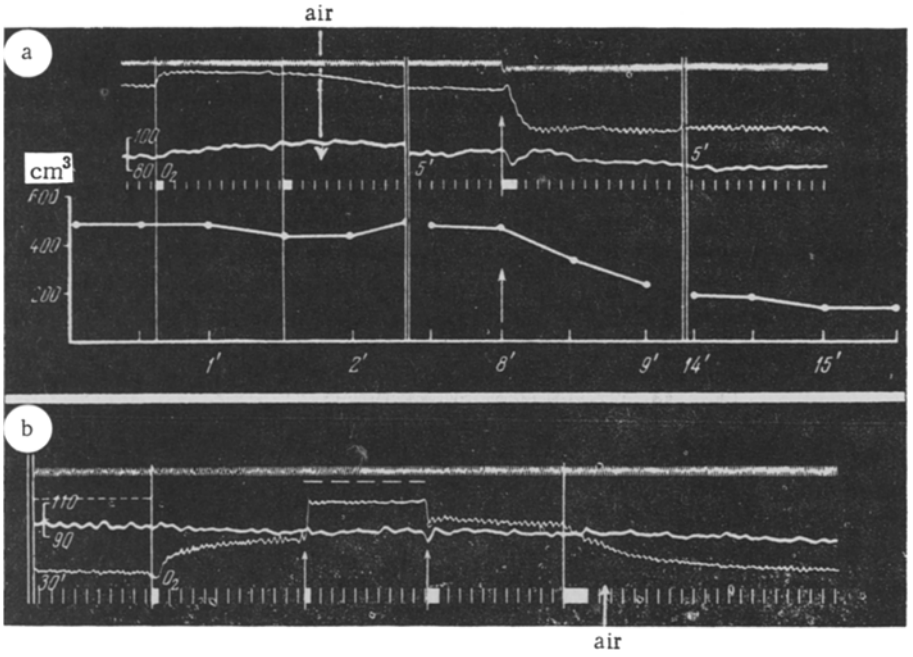

Fig. 1. Changes in the degree of arterial blood-oxygen saturation following open pneumothorax. Experiment of April 14, 1958. (Rabbit weighed 2.75 kg.) a) Response to inspiring oxygen, before pneumothorax (indicated by vertical lines) and to opening of one pleural cavity (shown by arrow). Curves, from above downward: Respiration (pneumogram), degree of arterial blood-oxygen saturation, blood pressure, 5-sec time-marker, pulmonary ventilation (in cm^3 per 30 sec). The two vertical lines indicate a break in the recording, whose duration is shown by the figures alongside; b) response to breathing oxygen after an open pneumothorax (beginning and end of respiration shown by vertical lines) and compression of the main pulmonary artery on the side of the pneumothorax (time of compression of the artery and of removal of the clamp are shown by arrows). Curves, from above downward: Respiration (pneumogram), blood pressure, degree of arterial blood-oxygen saturation. Broken lines indicate level of arterial blood-oxygen saturation before effecting an open pneumothorax: while breathing air - - -; when breathing oxygen — — —. 5-sec time-marker.

vented from mixing with the arterial blood from the functional lung. S. L. Libov and V. I. Burakovskii [4], using angiocardiography, observed that the opaque substance was delayed in the collapsed lung; this result indicated a constriction of the pulmonary vessels on the side of the pneumothorax, which reduced the entry of blood, although, as our experiments have shown, the blood flow is not entirely arrested.

To differentiate phenomena originating in the collapsed lung from those which occur in the functional lung, and which also may be the cause of a maintained hypoxia, we have studied the effect on arterial blood saturation of breathing oxygen. When blood flow to the collapsed lung is stopped, and oxygen is breathed, it is to be expected that the blood will be normally oxygenated, because the increased partial pressure of oxygen in the inspired air should compensate for the insufficient ventilation of the remaining lung. Nevertheless, in many cases, even after tying off the one artery and breathing pure oxygen, the arterial circulation did not reach the level established before the pneumothorax had been effected (see Fig. 1b). This result indicates that in the operating lung, blood must pass along paths in which it does not come into contact with the oxygen of the alveolar air, and most probably it flows along arteriovenous anastomoses, so that the alveolar capillaries are short-circuited. We have previously observed this same phenomenon in humans after pneumonectomy [5], where again there was failure to obtain arterial saturation when oxygen was breathed, which indicated that part of the blood was flowing along arteriovenous anastomoses of the lesser circulation.

It might therefore, be supposed that arterial hypoxia following open pneumothorax is due chiefly to two

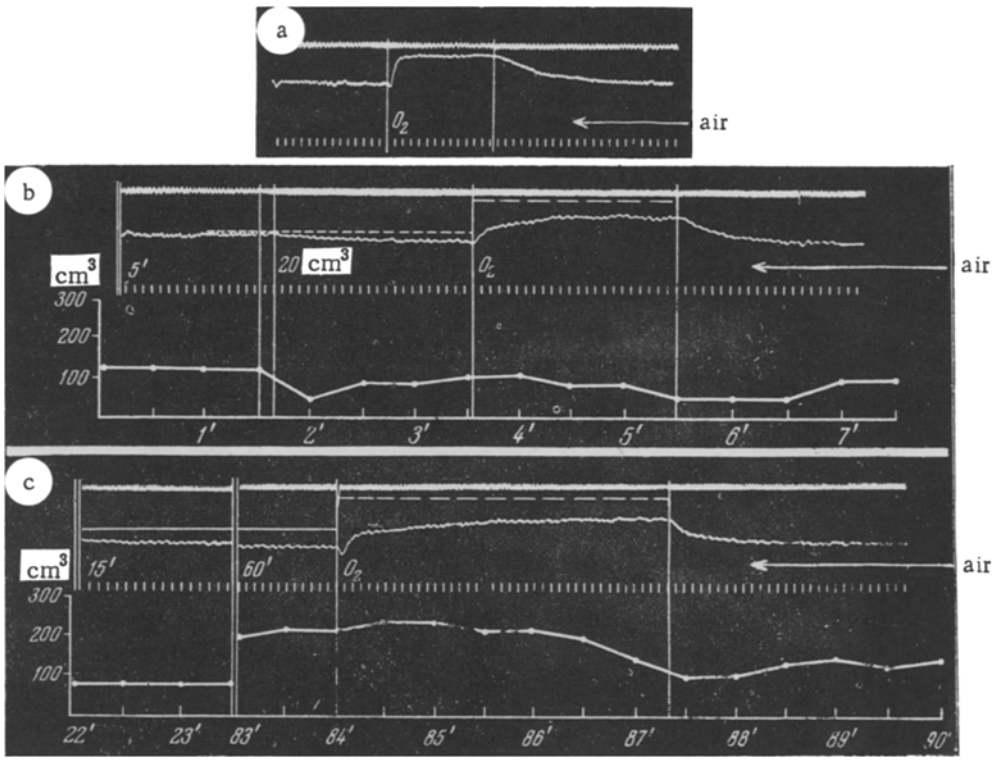

Fig. 2. Changes in the arterial blood-oxygen saturation following closed pneumothorax. Experiment of October 30, 1958. (Rabbit weighing 2.7 kg). a) Response to breathing oxygen before the pneumothorax; b) response to injection of 20 cm^3 of air into the left pleural cavity, and to breathing oxygen 2 minutes after effecting the pneumothorax; c) response to breathing oxygen 1 hour 20 minutes after pneumothorax. Curves, from above downward: For a) respiration (pneumogram), degree of arterial blood-oxygen saturation, 5-sec time-marker; for b) and c) respiration (pneumogram), degree of arterial blood-oxygen saturation, 5-sec time-marker, pulmonary ventilation (in c.c per 30 seconds); for b) the dotted line indicates the degree of arterial blood-oxygen saturation before effecting the closed pneumothorax: while air was breathed (---); when breathing oxygen (— — —). The degree of arterial blood-oxygen saturation before the closed pneumothorax was effected is shown by the continuous horizontal line when air was breathed, and by the line of long dashes when oxygen was breathed.

factors: First, there is a considerable increase in the blood flow to the remaining functional lung, and insufficient compensatory increase in ventilation; secondly, for a considerable time (not less than several tens of minutes or even several hours), blood continues to flow through the collapsed lung without becoming oxygenated. In addition, some of the blood may flow through arteriovenous anastomoses of the functional lung; in this way the resistance of the lesser circulation may be reduced, but the hypoxia then inevitably becomes deeper.

Experiments with closed pneumothorax showed that when 20-40 cm^3 of air are introduced into the pleural cavity, the rate of respiration is increased, and the depth reduced; at the same time there is a marked fall in the arterial blood saturation (Fig. 2b). Usually, after a few minutes the pulmonary ventilation rate recovers or attains a level higher than the original rate. However, the arterial blood-oxygen saturation remains reduced for a considerably longer time, and may remain so until the end of the experiment, i.e., for several hours. When oxygen is breathed, the arterial saturation never attains that maximum level which obtained when oxygen was breathed before the closed pneumothorax was effected (see Fig. 2).

In experiments in which an open pneumothorax was effected, sometimes, in order to let the animal "rest," we closed the opening in the pleura (the open pneumothorax was transformed into a closed one). Subsequently, the following changes could be observed: The respiratory movements immediately became reduced, but the pulmonary ventilation gradually increased, and sometimes attained the level observed before the open pneumothorax had been performed; the arterial blood-oxygen saturation was also increased,

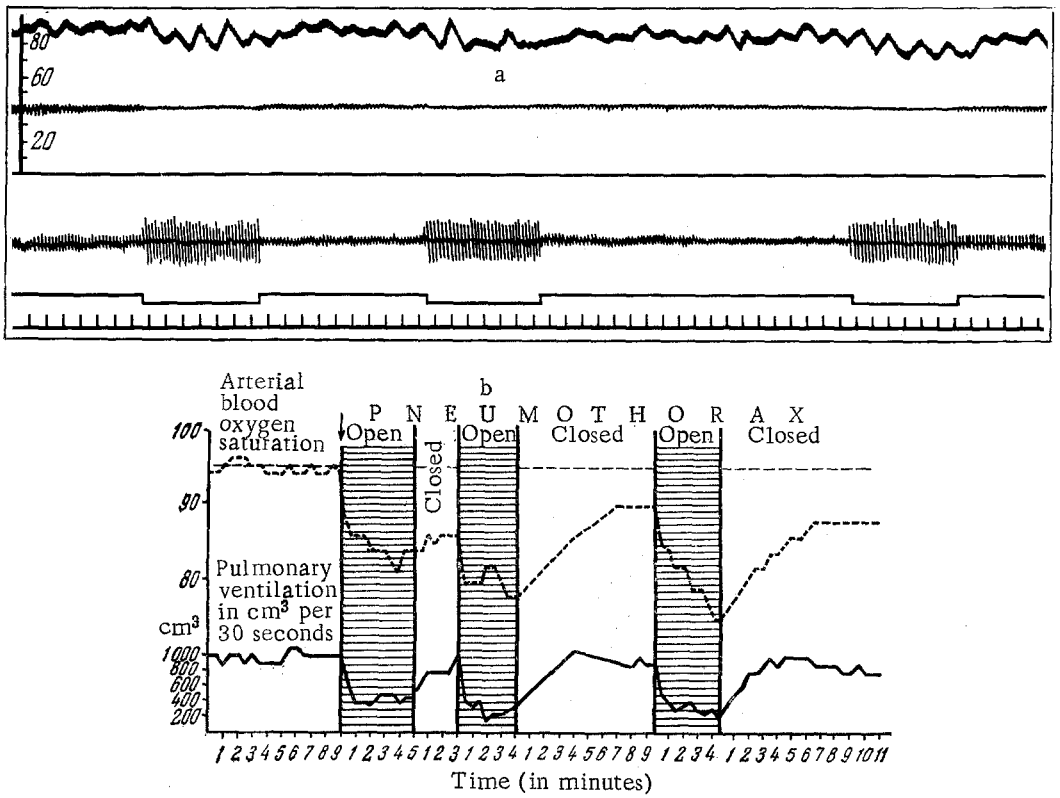

Fig. 3. Effect of hermetically closing the opening in the pleura of an open pneumothorax. Experiment of February 13, 1954. (Rabbit weighed 3.5 kg.) a) Curves, from above downward: Blood pressure, respiration (pneumogram), zero line of manometer, respiration (trace from trachea , stimulus marker (a movement downward corresponds to closing the pneumothorax, and upward to opening it), 5-sec time-marker; b) curves, from above downward; degree of arterial blood-oxygen saturation (in scale divisions of the galvanometer), pulmonary ventilation (in cm^3 per 30 seconds). Shaded columns represent duration of the open pneumothorax. The arrow indicates the moment of opening the pleural cavity. The dotted line represents the level of arterial blood-oxygen saturation before effecting the open pneumothorax.

but remained well below the original level (Fig. 3).

These results indicate that when a closed pneumothorax has been effected, despite the rapid restoration of a normal pulmonary ventilation, the balance between ventilation and pulmonary blood supply is disturbed. It appears that the ventilation on the side of the pneumothorax is greatly reduced. That there is a reduced expansion of the lungs—at any rate, in the first few minutes after producing a closed pneumothorax—has been demonstrated by electrophysiological investigations in which it was found that the number of impulses along the afferent fibers of the vagus was reduced. Nevertheless, even a comparatively small reduction in alveolar ventilation makes the aeration of the normally less ventilated portions insufficient. At the same time, the redistribution of the blood flow in the lungs may not correspond to the altered aeration: Blood flow may continue through the unventilated or poorly ventilated parts of the lungs. Also, as in the case of an open pneumothorax, blood may flow along arteriovenous anastomoses, because, after air has been introduced into the pleural cavity, it is found that the blood is still not saturated even when oxygen is breathed (see Fig. 2).

After a pneumothorax, a considerable time is required for the establishment of normal relationships between pulmonary ventilation and the blood supply which will restore a normal oxygenation of the lungs; it is scarcely possible to observe the development of this condition in an acute experiment.

We may therefore suppose that after an open or closed pneumothorax has been effected, not only disturbed ventilation, but also an impaired lesser circulation is responsible for the abnormal relationships between ventilation and blood supply and for the consequent impaired arterial hypoxia.

SUMMARY

Acute experiments on rabbits under urethan or morphine anesthesia were performed, and the causes of arterial hypoxia following unilateral open and closed pneumothorax studied. It was shown that, in addition to the disturbed pulmonary ventilation, impaired pulmonary circulation also played an important part.

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