CHANGES IN THE OXYGEN TENSION IN THE CEREBRAL CORTEX AND MECHANISMS OF DEVELOPMENT OF OXYGEN LACK IN THE BRAIN IN ASSOCIATION WITH OPEN PNEUMOTHORAX AND LUNG INJURY

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Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 55, No. 3, pp. 29-33, March 1963
Original article submitted September 26, 1961

An important factor in the pathogenesis of open pneumothorax and pulmonary trauma is acute oxygen lack [2, 3, 6, 9], to which the most sensitive part of the body is the brain [8]. The state of the oxygen supply of the brain and the mechanisms of development of oxygen lack in the brain in association with open pneumothorax have not, however, been studied. With the appearance of the polarographic method of investigation, by means of which the concentration of oxygen in the tissues can be determined in vivo, the possibility of studying problems concerning the transportation and utilization of oxygen in various pathological processes has been considerably widened.

In the present paper we examine the changes in the oxygen tension in the cerebral cortex and compare them with changes in the carbohydrate and phosphorus metabolism of the brain, and the indices characterizing the state of oxygen transportation to the brain in cases of open pneumothorax and lung injury.

EXPERIMENTAL METHOD

Experiments were carried out on unanesthetized animals. Rabbits were injured by the production of an open pneumothorax on the left side, and by mechanical irritation of the pleura and the parenchyma and root of the lung, and resection of the lung; in rats the pleural cavity was simply opened.

The criterion of the oxygen supply to the brain was the oxygen tension in the cortex, determined by a polarographic method [4]. The state of the carbohydrate-phosphorus metabolism was judged from changes in the concentration of readily hydrolyzable phosphorus of adenosinetriphosphate, creatine phosphate, inorganic phosphorus, and lactic acid in the brain tissues. The effect of open pneumothorax on the oxygen absorption in the lungs and its supply to the brain tissues was evaluated from the changes in the volume of the pulmonary ventilation, the oxygen demand, the concentration of oxyhemoglobin in the arterial blood, and in the general and the cerebral circulation. The state of the cerebral circulation was evaluated by the techniques of Hürthle [14] and Avrorov [1].

EXPERIMENTAL RESULTS

The symptom-complex of open pneumothorax and lung trauma in our experimental conditions was described in previous papers [5-7]. We consider that as a result of the combined injury a severe pleuropulmonary shock develops, in which, in contrast to other forms of shock, acute respiratory failure appears very early, and is soon complicated by insufficiency of the cerebral, and later of the general circulation. Consequently, the body develops oxygen lack, which is especially marked in the brain. Directly after the pleural cavity was opened, the oxygen tension in the cerebral cortex fell by 32-75% (average 58%) of its original level (Fig. 1, 2, 3). In the subsequent stages of the experiment, in some conditions (see Fig. 2) the oxygen tension in the cerebral cortex continued to fall, while in others (see Fig. 1, 3) it rose slightly, without reaching its original level, and then fell again, its value in the period of the torpid phase of shock being 42% of its original level.

The origin of the cerebral anoxia in pleuro-pulmonary shock is complex. To judge from our findings, its initial link is respiratory failure. In fact, within 5-15 min after opening the pleural cavity of these animals a simultaneous fall was observed in the oxygen demand, the oxyhemoglobin concentration in the arterial blood, and the

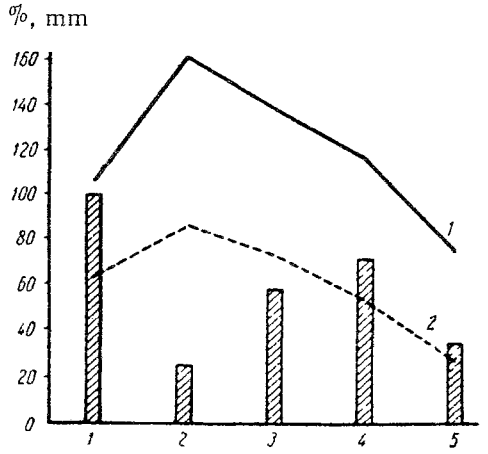

Fig. 1. Change in the oxygen tension in the cerebral cortex and in the arterial pressure during open pneumothorax with lung injury in a rabbit (experiment on April 24, 1961). Columns – oxygen tension in the cerebral cortex (percentages of the original level); continuous line – arterial pressure in the central end of the common carotid artery (in mm); broken line – blood pressure in the circle of Willis (in mm). Stages of the experiment: 1) before trauma (initial); 2) after opeing the pleural cavity; 3) after resection of the lung; 4) 30 min, and 5) 1 h after production of open pneumothorax.

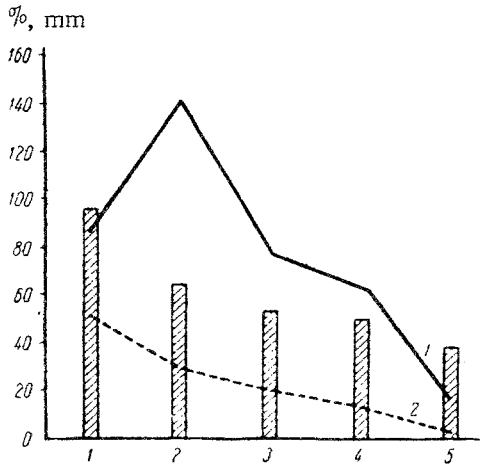

Fig. 2. Changes in the arterial pressure and in the oxygen tension in the cerebral cortex in a rabbit with open pneumothorax and lung injury (experiment on May 29, 1961). Stages of the experiment: 1) before trauma (initial); 2) after opening the pleural cavity; 3) after resection of the lung; 4) 30 min, and 5) 1 h after production of open pneumothorax.

oxygen tension in the cerebral cortex (see Fig. 3, 1, 2). The volume of the pulmonary ventilation at this time, as a rule, was reduced.

After resection of the lung (see Fig. 3, 3) the oxygen tension in the cerebral cortex rose slightly over that in the preceding period of the experiment. It is significant that the oxygen demand and the oxyhemoglobin concentration in the arterial blood were also increased at this time, although they were still below the initial values. This improvement in the indices of the functions of the external respiratory apparatus was due to removal of the collapsed lung from the animal.

The strict parallel between the changes in the volume of the pulmonary ventilation, the oxygen demand, the oxyhemoglobin concentration in the arterial blood, and the oxygen tension in the cerebral cortex was also demonstrable during the torpid phase of pleuro-pulmonary shock (see Fig. 3, 4). Consequently, in open pneumothorax acute respiratory failure plays an important role both in the onset and in the subsequent development of cerebral anoxia.

Meanwhile, the oxygen utilization in the brain increased sharply as a result of the extreme excitation of the nervous system following trauma, as shown by the results of biochemical investigation of the brain. Within 5-15 min after formation of the open pneumothorax, the adenosinetriphosphate concentration in the brain tissues fell by 10% in the rabbits and by 15% in the rats, and the creatine phosphate by 39% in the rabbits and by 46% in the rats. The inorganic phosphorus content of the brain at this stage of the experiment rose by 34% in the rabbits and by 26% in the rats; the lactic acid content of the brain increased in the rabbits by 83% over its value in the control experiments.

Similar disturbances in the carbohydrate-phosphorus metabolism of the brain have been observed by other researchers using different methods of excitation of the central nervous system [10-13]. It has been found [13] that the changes in the metabolism of labile phosphorus compounds in the brain during excitation and anoxia of the organ are identical in their direction. This agrees well with the results of our experiments.

In fact, after resection of the lung a definite connection was observed between the reduction of the creatine phosphate content of the brain and of the oxygen tension in the cerebral cortex, the fall in the volume of the pulmonary ventilation, and the decrease in the oxyhemoglobin concentration in the arterial blood (see Fig. 3). In the torpid phase of pleuro-pulmonary shock the creatine phosphate content of the brain fell by 59%, the oxygen tension in the cerebral cortex by 56%, the oxyhemoglobin concentration dropped to 34% below its initial level, the volume of the pulmonary ventilation fell by 61%, and the oxygen demand by 41%. The content of inorganic phosphorus rose at this time by 54%, and that of lactic acid by 230% by comparison with the control values.

It was mentioned above that as a result of removal of the collapsed lung the signs of respiratory failure showed some improvement, although complete compensation did not take place

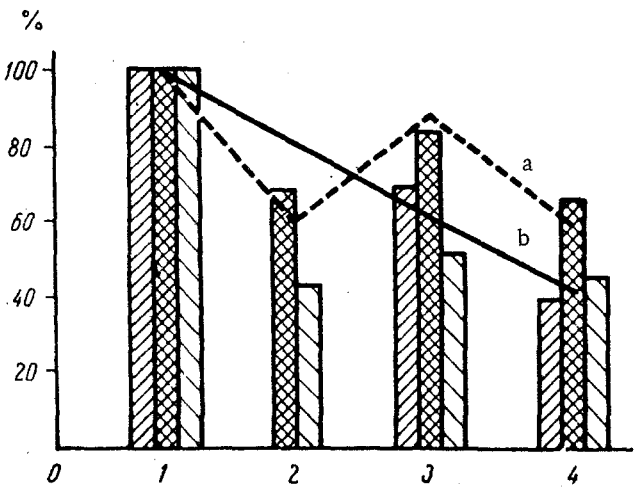

Fig. 3. Change in the volume of the pulmonary ventilation (close oblique shading), the oxyhemoglobin concentration in the arterial blood (cross-hatched), the oxygen tension in the cerebral cortex (open oblique shading), the oxygen demand (a), and the concentration of creatine phosphate in the brain (b) in rabbits with open pneumothorax and lung injury (mean results for groups of experiments). The initial value of each index is taken as 100. Stages of the experiment: 1) before injury (original); 2) after opening the pleural cavity; 3) after resection of the lung; 4) at the end of the experiment (torpid phase of pleuro-pulmonary shock).

because the cerebral, and later the general circulation, became disturbed (see Fig. 1, 2). During the minutes immediately after opening the pleural cavity the fall in the oxygen tension in the cerebral cortex was not, in fact, associated with changes in the general circulation, since the arterial pressure at this period was actually increased. The degree of the fall in the oxygen tension in the cerebral cortex was slightly greater in the rabbit, (see Fig. 1) in which the blood pressure in the circle of Willis rose immediately after formation of an open pneumothorax,than in the animal reacting to trauma by a lowering of the arterial pressure in the cerebral vessels (see Fig. 2). In the first case, spasm of the cerebral vessels probably took place in response to the trauma. After resection of the lung in one experiment (see Fig. 2). In the first case, spasm of the cerebral vessels probably took place in response to the trauma. After resection of the lung in one experiment (see Fig. 2), combined changes affecting both the general and the cerebral circulation, and the oxygen tension in the cerebral cortex appeared, while in another there was no definite relationship between the pattern of the changes in these indices. Only in the stage of severe shock was a clear relationship observed in both rabbits, both between the trend of the changes in the circulation and in the oxygen tension in the cerebral cortex and between the degree of lowering of the arterial pressure and of the oxygen concentration in the brain; moreover, the fall in the blood pressure in the circle of Willis took place relatively more rapidly than in the general system of the circulation. This indicated a diminution of the tone of the cerebral vessels [7].

Hence, injury to the lungs and pleura, associated with open pneumothorax, is accompanied by a fall in the oxygen tension in the cerebral cortex, indicating the development of acute cerebral anoxia. Meanwhile, the oxygen utilization in the brain is increased sharply as a result of the intense excitation of the nervous system by the trauma. Subsequently, after removal of the collapsed lung, the signs of respiratory failure begin to improve, but complete compensation does not take place because disturbances arise, initially in the cerebral, and later in the general circulation. The torpid phase of pleuro-pulmonary shock is associated with both respiratory and vascular failure. In this way, pleuro-pulmonary shock resembles other forms of shock. The specific features of shock associated with open pneumothorax are apparent in its erectile phase, which in pleuro-pulmonary shock, in contrast to other forms, develops against the background of an early and marked respiratory failure.

SUMMARY

As shown in experiments on rabbits, traumatization of the lungs and pleura in conditions of open pneumothorax is accompanied by a drop of oxygen tension in the cerebral cortex, reduction of phosphorus, adenosintriphosphoric acid and creatinphosphate content in the brain with a simultaneous rise of inorganic phosphorus and lactic acid concentration, pointing to the development of acute hypoxia in the brain. At the early states of pneumothorax the leading role in the appearance of cerebral hypoxia is played by respiratory insufficiency and the CNS excitation. Soon disturbances of cerebral circulation, and then the general vascular insufficiency, supervene.

LITERATURE CITED

1. V. P. Avrorov, Byull. éksper. biol., 8, 11 (1957).
2. F. R. Amirov. Proceedings of the Third Usbekistan Conference of Physiologists, Biochemists, and Pharmacologists [in Russian], p. 99. Tashkent, 1951.
3. M. S. Vovsi and T. S. Istamanova. Klin. med., 1, 3 (1947).
4. Kovalenko. Pat. fiziol., 2, 66 (1961).

5. I. D. Kudrin. Transactions of the Military Medical Academy [in Russian], vol. 90, p. 31. Leningrad, 1958.
6. I. D. Kudrin. Transactions of the Military Medical Academy [in Russian], vol. 102, p. 65. Leningrad, 1959.
7. I. D. Kudrin. Pat. fiziol., 5, 26 (1960).
8. V. P. Kurkovskii and I. R. Petrov. Transactions of the Military Medical Academy [in Russian], vol. 21, p. 3. Leningrad, 1940.
9. S. L. Libov. Experience of Soviet Medicine in World War II, 1941-1945 [in Russian], vol. 9, p. 291, Moscow, 1949.
10. A. V. Palladin. In: Biochemistry of the Nervous System [in Russian], p. 7. Kiev, 1954.
11. I. R. Petrov, Z. A. Raiko, and T. E. Kudritskaya. Fiziol. zh. SSSR, 2, 107 (1957).
12. M. I. Prokhorova and Z. N. Tupikova. In: Problems in the Biochemistry of the Nervous System [in Russian], p. 118. Kiev, 1957.
13. V. S. Shapot and K. G. Gromova. In: Biochemistry of the Nervous System [in Russian], p. 139. Kiev, 1954.
14. K. Hürthle, Pflüg. Arch. ges. Physiol., 1889, Bd. 44, S. 561.

All abbreviations of periodicals in the above bibliography are letter-by-letter transliterations of the abbreviations as given in the original Russian journal. *Some or all of this periodical literature may well be available in English translation.* A complete list of the cover-to-cover English translations appears at the back of this issue.
