# RELATIONSHIPS BETWEEN CHANGES IN THE OXYGEN TENSION AND THE REGIONAL BLOOD FLOW IN THE TISSUES IN ACUTE HYPOXIA

M. I. Gurevich
and S. A. Bershtein

UDC 616-008.922.1.04-036.11-07:
[616-008.922.1+616-005]072.7

The relationships between the oxygen metabolism, the hemodynamics, and the regional blood flow constitute one of the most important problems in the regulation of the vital functions of the organism. Many aspects of the regulation of the circulation in hypoxia have not yet been explained.

The object of the present investigation was to study the relationships between the changes in the systemic arterial pressure, the regional blood flow, and the oxygen tension in various tissues during hypoxia.

## EXPERIMENTAL METHOD

Acute experiments were carried out on cats under chloralose-Nembutal anesthesia (25 mg chloralose and 25 mg Nembutal/kg body weight).

During the experiments the animals breathed a gas mixture with a lowered oxygen concentration (7.5 and 9.6%) for 5-6 min.

The changes in the oxgyen tension and the tissue blood flow were measured in the thigh muscle of the hind limb, the skin of the abdomen, and the parietal region of the cerebral cortex. The arterial pressure was measured in the femoral artery.

By means of the apparatus constructed jointly by D. A. Golov and the author, synchronized recordings could be made of the dynamics of the changes in the systemic arterial pressure (by an electromanometer), the tissue blood flow (by a thermoelectric method), and the oxygen tension in the tissues (by a polarographic method). The recording apparatus was a type ÉPP-09 multichannel electronic potentiometer.

## EXPERIMENTAL RESULTS

In hypoxia conditions caused by inhalation of a gas mixture containing 9.6% of oxygen, as a rule after 30-60 sec the arterial pressure rose by 10-20 mm (Figs. 1, A and B; 2, A). In only two experiments the arterial pressure fell by approximately the same amount (Fig. 2, B).

Inhalation of a mixture with a still lower oxygen concentration (7.5%) led in most cases to a fall of 20-30 mm in the arterial pressure, and if inhalation of this mixture continued for longer than 5-6 min, the fall in arterial pressure usually progressed until the animal died.

The dynamic pattern of the changes in the oxygen tension and regional blood flow in hypoxic conditions varied from one tissue to another.

In the skeletal muscle and skin, a marked decrease in the oxygen tension began after 20-30 sec, and continued throughout the period of inhalation of the hypoxic mixture. About 1 min later, the oxygen tension began to recover, reaching its initial level at the earliest after 8-10 min (Fig. 1, A and B).

The blood flow in the skeletal muscle during hypoxia usually showed no significant changes, and only occasionally was there a tendency for it to rise (Fig. 1, A). In the skin, on the other hand, there was a marked tendency for the blood supply to diminish (Fig. 1, B).

---

Laboratory of Physiology of the Circulation, A. A. Bogomolets Institute of Physiology, Academy of Sciences of the UkrSSR, Kiev (Presented by Active Member of the Academy of Medical Sciences of the USSR N. N. Sirotinin). Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 63, No. 3, pp. 31-35, March, 1967. Original article submitted August 7, 1965.

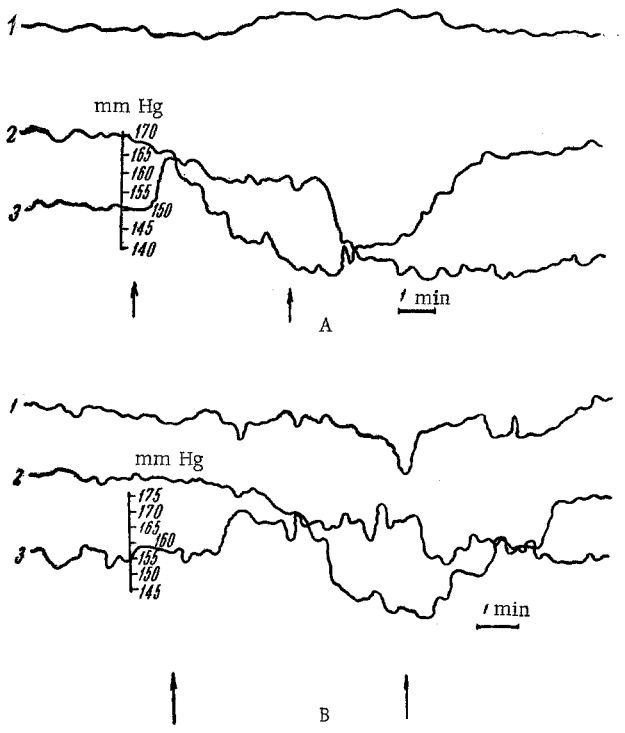


Fig. 1. Dynamics of changes in the velocity of the blood flow (1) and the oxygen tension (2) in a skeletal muscle (A) and in the skin (B), and changes in arterial pressure (3) in hypoxia. Here and in Fig. 2, the arrows indicate the period of inhalation of gas mixture with a lowered oxygen concentration.

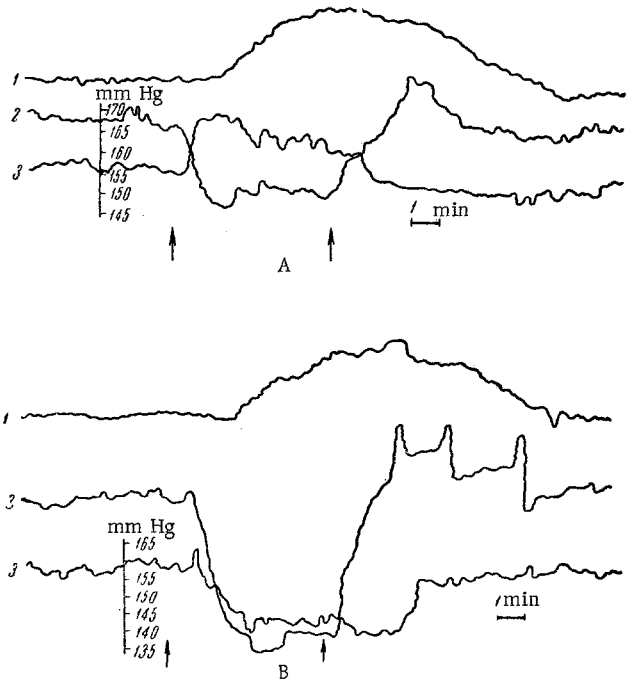


Fig. 2. Dynamics of changes in the velocity of the blood flow (1) and the oxygen tension in the cerebral cortex (2), and changes in the arterial pressure (3) in hypoxia. A—Arterial pressure raised; B—arterial pressure lowered.

The dynamics of the changes in the investigated indices was of a different pattern in the cerebral cortex. The oxygen tension began to fall almost as soon as exposure to hypoxia began, but it did not fall below a certain level despite continued inhalation of the hypoxia mixture. Approximately 30 sec after resumption of inhalation of atmospheric air, the oxygen tension in the brain tissure began to recover quickly. After 3-4 min the oxygen tension was significantly above its initial level, to which it returned after a further 1-3 min (Fig. 2, A and B).

The blood supply to the brain increased sharply during hypoxia. The blood flow was maintained at a high level for some time after the inhalation of the hypoxic mixture ended, and fell to its initial level only after 7-8 min (Fig. 2, A). An increased blood flow in the cerebral cortex during hypoxia was observed even when the arterial pressure showed a substantial fall (Fig. 2, B).

The experiments thus showed that in hypoxic hypoxia not all the tissues are equally supplied with oxygen. Whereas in the skeletal muscle and skin the oxygen tension falls steadily during hypoxia, in the cerebral cortex it falls a little and then becomes stabilized at definite level. Consequently, the cerebral cortex receives the best oxygen supply, at the expense of the skeletal muscles and skin, and possibly of the other organs less sensitive to oxygen deficiency also.

The redistribution of the blood flow observed in the body in these experiments may be explained by divergent changes in the tone of the blood vessels. In fact, the tendency for the velocity of the blood flow in the skin to diminish while the blood flow in the skeletal muscles remained substantially unchanged, despite the increasing systemic arterial pressure, in all probability was due to an increase in the tone of the blood vessels of these tissues.

From the available information it is difficult to judge the tone of the blood vessels of the cerebral cortex because the velocity of the blood flow and the systemic arterial pressure as a rule changed in the same direction. However, the results of individual experiments in which, despite the fall in the arterial pressure, the blood flow in the cerebral cortex was sharply increased, demonstrated that the tone of the cerebral vessels was lowered.

Consequently, the maintenance of a definite oxygen balance corresponding to the oxygen needs of the various tissues, in the conditions of hypoxic hypoxia is due largely to regulation of the regional vascular tone.

The problem of the mechanism of regulation of the regional vascular tone in hypoxia remains unsolved and is one of the more urgent problems facing the physiology of the circulation.

## LITERATURE CITED


1. A. I. Vyshatina, Byull. éksp. Biol., No. 11, 56 (1963).
2. M. I. Gurevich, A. I. Vyshatina, and M. A. Kondratovich, In the book: Abstracts of Proceedings of Symposia of the 10th Congress of the I. P. Pavlov All-Union Physiological Society [in Russian], 1, 132, Moscow–Leningrad (1964).
3. M. I. Gurevich and M. A. Kondratovich, In the book: Abstracts of Proceedings of the 7th Congress of the Ukrainian Physiological Society [in Ukrainian], 115, Kiev (1964).
4. N. V. Lauer, A. Z. Kolchinskaya, and V. V. Turanov, In the book: Proceedings of a Conference on the Problem of Adaptation, Training, and Other Methods of Increasing Body Resistance [in Russian], 71, Donets! (1960).
5. M. E. Marshak, L. I. Ardashnikova, G. N. Aronova, et al., In the book: Regulation of Respiration, the Circulation, and Gas Exchange [in Russian], 65, Moscow (1948).
6. M. E. Marshak, Byull. éksp. Biol., 1, 121 (1957).
7. M. E. Marshak, In the book: The Oxygen Balance and Its Regulation [in Russian], 191, Kiev–Kanev (1965).
8. N. V. Sanotskaya, Byull. éksp. Biol., No. 9, 46 (1962).
9. M. M. Sirotinin, Life at High Altitudes and Altitude Sickness [in Ukrainian], Kiev (1939).
10. L. L. Shik, In the book: Abstracts of Proceedings of Symposia of the 10th Congress of the I. P. Pavlov All-Union Physiological Society [in Russian], 1, 140 Moscow–Leningrad (1964).
11. R. M. Berne, Am. J. Physiol., 204, 317 (1963).
12. R. M. Berne, Circulat. Res., 15, Suppl. 1, 261 (1964).
13. D. G. Crawford, H. M. Fairchild, and A. C. Guyton, Am. J. Physiol, 197, 613 (1959).
14. T. E. Driscol and R. M. Berne, Proc. Soc. Exp. Biol. (N. Y.), 96, 505 (1957).
15. B. Folkow, Physiol. Rev, 40, Suppl. 4, 286 (1960).
16. A. C. Guyton, J. M. Ross, O. Jr. Carrier, et al., Circulat. Res., 15, Suppl. 1, 60 (1964).
17. R. Hilton and F. Eichholtz, J. Physiol. (London), 59, 413 (1925).
18. M. I. Jacob and R. M. Berne, Am. J. Physiol., 198, 322 (1960).
19. G. P. Lewis, J. Physiol. (London), 147, 458 (1959).
20. J. Markwalder and E. H. Starling, J. Physiol. (London) 47, 275 (1913).
21. P. Nicoll and R. Webb, Angiology, 6, 291 (1955).
22. B. Oberg, T. Q. Richardson, and A. C. Guyton, Physiologist, 4, 84 (1961).
23. J. M. Ross, H. M. Fairchild, J. F. Weldy, et al., Am. J. Physiol., 202, 21 (1962).
24. C. F. Schmidt, The Cerebral Circulation in Health and Disease, Springfield (1950).
25. F. Solti, M. Iskum, G. Mark, et al., Acta Physiol. Acad. Sci. Hung., 23, 269 (1963).
26. L. R. Yonce and W. F. Hamilton, Am. J. Physiol., 197, 190 (1959).