CHANGES IN TISSUE OXYGEN TENSION IN HYPO- AND HYPEROXIA

N. V. Sanotskaya

Laboratory of the Physiology and Pathology of Respiration
and Circulation (Head, Corresponding Member AMN SSSR
Prof. M. E. Marshak),
Institute of Normal and Pathological Physiology (Director,
Active Member AMN SSSR V. V. Parin), AMN SSR, Moscow
(Presented by Active Member AMN SSSR V. V. Parin)
Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 51, No. 6,
pp. 33-37, June, 1961
Original article submitted July 1, 1960

Results on changes in the tissue oxygen tension may be of great value in determining the oxygen supply to the tissue. Tissue oxygen tension measurements have been made possible by the method of polarography [5]. It has been used frequently in recent years to determine the effect of various factors on the oxygen tension in the brain [1,2,3,5,8] the heart [11,12] and in the skin and muscles [9,10,etc.].

It must be remembered that the changes in tissue oxygen tension may be brought about by at least three factors. There may be alteration in the blood gases, in the blood supply to the tissues, and in the rate of the oxidative processes in them. To determine the cause, the effect of each of the three factors must be determined separately.

We have studied alterations in the tissue oxygen tension caused by altering the oxygen content of arterial blood.

METHOD

Thirty-five acute experiments were carried out on cats under urethane anesthesia.

Records were made of respiration (on a pneumograph), of arterial pressure (by mercury manometer), of relative changes in the oxygen saturation of the arterial blood (using a photocell), and of the oxygen tension in skeletal muscle and in brain tissue by a polarographic method [4,6,7].

To record changes in tissue oxygen concentration we used the following electrodes: a cathode consisting of a platinum wire 0.3 mm in diameter and insulated except for the tip by a polyurethane varnish, and a nonpolarizable anode, made of a silver wire 0.5 mm in diameter, covered with a layer of silver chloride deposited electrolytically. The platinum electrode was inserted into the tissue under test, and the silver electrode into a muscle, usually at a distance of 3–4 cm from the first electrode. A potential of 0.6 v was applied to the leads (and it has been shown that when oxygen is being reduced, the current at the platinum cathode is proportional to the oxygen tension in the solution, or in the tissue [4,10]). The current and its variations were recorded by a GZS-47 galvanometer using the electrical circuit described by Montgomery and Horwitz [10].

The arterial oxygen concentration was altered by arranging for the animal to breathe mixtures containing an increased or decreased amount of oxygen from a bag.

RESULTS

It was found that in hypoxia, changes in the oxygen tension in the tissues follow the variations in the oxygen content of the arterial blood quite closely, lagging behind it somewhat in time: a reduced arterial oxygen concentration usually began 1–5 seconds after breathing a mixture poor in oxygen, and a reduction in the tension in the tissues followed a few seconds later (Fig. 1 a,b; Fig. 2 a,b).

The lag in the change of the tissue oxygen tension behind arterial tension occurred not only at the beginning but during the time that the mixture poor in oxygen is breathed: when after 1–1½ minutes the arterial tension settled at a reduced but almost constant level, the tissue tension continued to fall and settled at a fairly constant level 5–7 minutes after the reduced oxygen mixture was given.

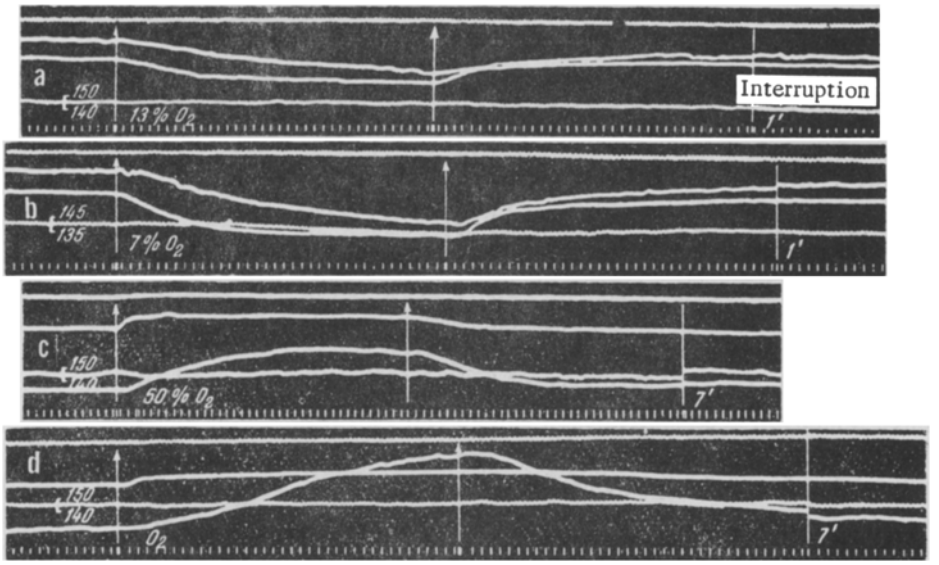

Fig. 1. Changes in oxygen concentration in the brain of a cat weighing 2.5 kg; experiment on 25/12/1959. Inspiration of mixture (↑↑), containing a) 13% of oxygen; b) 7% oxygen; c) 50% oxygen; d) pure oxygen. Curves from above downwards: a,b) respiration, oxygen tension in brain tissue; arterial oxygen saturation; arterial pressure; time marker (5 seconds); c,d) respiration, arterial blood oxygen saturation; arterial pressure; tissue oxygen tension; time marker (5 seconds).

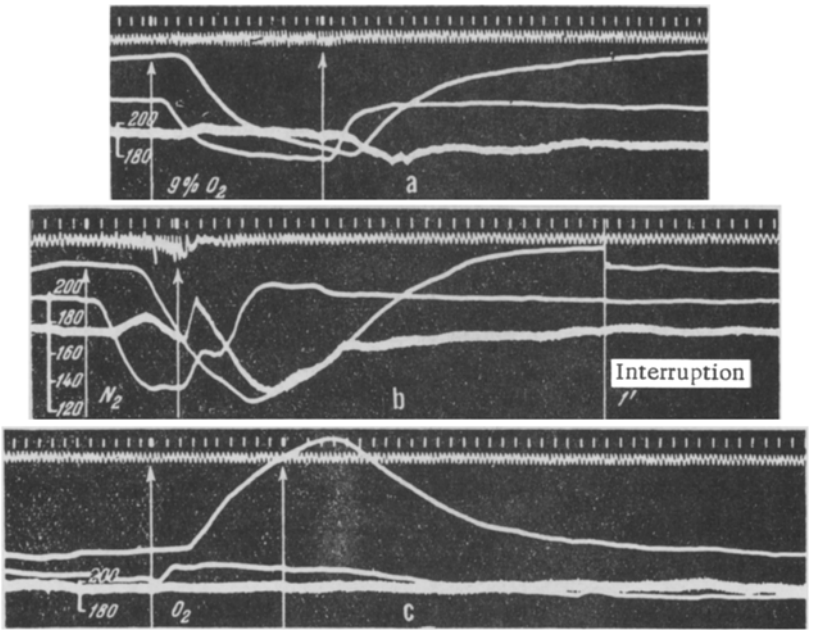

Fig. 2. Changes in oxygen tension in the gastrocnemius muscle. Experiment on cat weighing 1.9 kg, 9/10/1959. A) Breathing a mixture ( ), containing 9% oxygen; b) breathing pure nitrogen; c) breathing oxygen. Curves, from above downwards: time marker (5 seconds); respiration, oxygen tension in muscle; arterial blood oxygen concentration; arterial pressure.

The reduced arterial tension may be to some extent compensated by a reflex increase in respiration. At the same time, to prevent the reduction of the oxygen tension in the tissues a whole number of mechanisms are brought

into play, which include increase in blood supply to the tissue, and a change in the oxidative processes; however, these processes take longer to be established.

After the oxygen-deficient mixture has been replaced by normal air, the recovery of the arterial blood oxygen saturation usually occurs quite rapidly, after 30-40 seconds; only when more severe and repeated oxygen lack has been inflicted does the arterial blood oxygen saturation take longer to recover.

Even after breathing air of a reduced oxygen content for as little as 1–3 minutes, usually a longer time was required for recovery, which however was different for the different tissues. Oxygen concentration in the brain was usually restored in 3–4 minutes, and in many cases, particularly after a considerable hypoxia had developed after breathing a mixture containing 6–9% oxygen, there was an increase in the blood oxygen to a level higher than the original. The oxygen tension in muscle was frequently not fully restored in less than 6–8 minutes; only after very acute hypoxia was there sometimes a more rapid but short-lasting increase in tissue oxygen tension, after which it usually fell below the original value.

Increasing the partial pressure of oxygen in the inspired mixture (Fig. 1 c,d; Fig. 2 c) caused only small increase in the arterial blood oxygen concentration; at the same time there was a very considerable increase in the tissue oxygen tension which was better shown, the higher the concentration in the air breathed. Although when pure oxygen was given (Fig. 1 d) there was same increase in arterial blood oxygen concentration as with a 50% mixture (see Fig. 1 c), the increase in the tissue oxygen tension was better shown in the former case.

It is known that when atmospheric air is breathed, the arterial blood oxygen concentration is 95–96%; even when the partial pressure of oxygen in the inspired air is 40–50%, the hemoglobin is almost 100% saturated. When the oxygen partial pressure is increased further, its concentration in the blood plasma increases, and although no further saturation of the hemoglobin is possible, there is a considerable increase in the partial pressure of oxygen in the blood, which is particularly important in connection with its diffusion from the blood into the tissues. This circumstance explains the changes in the degree of arterial blood oxygen concentration and tissue oxygen tension as related to an increased partial pressure of oxygen in the inspired air.

When a changeover is made to breathing pure oxygen, changes take place in the same sequence as when the oxygen content of the air mixture is reduced: the changes in the tissue oxygen tension lag somewhat behind the arterial blood oxygen concentration. The same thing usually occurs at the changeover from breathing oxygen to breathing atmospheric air. However, quite frequently when the oxygen supply is removed, the fall in the tissue oxygen tension begins at the same time or even somewhat before the recovery in the saturation of the arterial blood.

The explanation is that the initial reduction in the oxygen partial pressure in the blood plasma may produce an immediate reduction in tissue oxygen tension; at the same time, the plasma concentration may be sufficiently high to produce complete saturation of the hemoglobin.

The restoration of the tissue oxygen concentration to its original level usually occurs 3–5 minutes after the cessation of breathing oxygen.

It was pointed out above that changes in tissue oxygen tension accompanying a change in the partial pressure of the air breathed lags somewhat behind changes in arterial blood oxygen concentration. Here it should be noted that this time lag is shown to a different extent in the various tissues. Thus, in the brain the tissue change occurs from 1 to 5 seconds, and usually between 2 and 3 seconds after the beginning of the change in arterial oxygen saturation (see Fig. 1), in muscle the time delay is from 6 to 20 seconds, and usually lies between 8–12 seconds (see Fig. 2).

A condition such as hypoxia produces a whole series of adaptive changes in the body, and in particular an increase in pulmonary ventilation and in the minute volume of the heart; there is also a redistribution of blood throughout the body and a change in the course of the oxidative processes.

The reason for the differences in the latent period of the oxygen tension change for the different tissues is probably that the blood supply to these tissues changes unequally, and their oxidative processes are affected differently.

The blood supply to the brain is more abudant than to the muscles, and under normal conditions is greatly increased during hypoxia and in the subsequent recovery period; the effect is due to the greater surface area of the capillaries through which diffusion of gases takes place, so that there is a greater rate of diffusion toward the surface

of the electrode. In addition, brain tissue has a higher metabolic rate, which is comparatively little reduced during hypoxia; because of the greater demand for oxygen, the drop in oxygen tension in the brain may occur earlier than in muscle.

After the oxygen deficient mixture has been withdrawn, the recovery of the oxygen tension in the brain also begins earlier (after 1–4 seconds) than it does in muscle, where it is delayed until 5–25 seconds after the onset of the recovery of the arterial blood oxygen saturation. As has already been pointed out, complete recovery of the oxygen tension in brain occurs more rapidly than in muscle. It is probable that this is mainly due to an increased blood supply to the brain, which occurs in hypoxia and in the subsequent recovery period.

SUMMARY

Experiments were performed on cats under urethane anesthesia. The effect of increased or decreased oxygen supply on oxygen tension were investigated. It was found that in hypoxia, changes in the tissue oxygen tension follow the degree of oxygen saturation of arterial blood, and are delayed slightly in time compared to the latter. The higher the oxygen concentration in the inspired air, the greater is the increase of the oxygen tension in the tissues.

The latent period for the change in oxygen tension is shorter in the brain than in skeletal muscle.

LITERATURE CITED

1. A. D. Snezhko, Biofizika 1, 6, 585 (1956).
2. I. D. Éntina and V. A. Yakovelev, Biokhimiya 16, 6, 567 (1951).
3. L. C. Clark, G. Misrahy and R. Fox, J. Appl. Physiol. (1958) 13, p. 85.
4. P. W. Davies and F. Brink, Rev. Sci. Instr. (1942) 13, p.524.
5. P. W. Davies and D. W. Bronk, Fed. Proc. (1957) 16,p. 689.
6. J. Heyrovský, The Technique of Polarographic Investigation [in Russian] (Moscow, 1951).
7. I. M. Kolthoff and J. J. Lingane, Polarography (New York, 1941).
8. J. S. Meyer, H. C. Fang and D. Denny-Brown, Arch. Neurol. Psychiat. (Chicago, 1954) 72, p. 296.
9. H. Montgomery, Fed. Proc. (1957) 16, p. 697.
10. H. Montgomery and O. Horwitz, J. Clin. Ivest. (1950) 29, p. 1120.
11. J. J. Sayen, et al.,J. Clin. Invest. (1951) 30, p. 932.
12. J. J. Sayen, et al.,Circulat. Res. (1958) 6, p. 779.

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.
