It has been shown [6] that in hypoxia or hyperoxia the tissue oxygen tension is controlled chiefly by alterations of the tension in the arterial blood. We may also suppose that in hypoxia the oxygen tension changes in the tissue depends also to some extent on those of the blood supplying the tissue, and possibly on the course of the oxidative processes in them.

Here we have undertaken an investigation into the influence of hypoxia and hyperoxia on the oxygen tension of tissues. Although these factors have little influence on arterial blood oxygen saturation, they nevertheless produce marked changes in the blood supply to the tissues [2, 10, 12, 13, 16, 17, 18, 21, 23], and influence the rate of the oxidative processes in the body. Hypercapnia reduces the metabolic rate, while hypocapnia increases it [1, 3, 4, 5, 7, 8]. We must also take into account any possible influence of hyper- and hypocapnia on the affinity of hemoglobin for oxygen.

**METHOD**

Acute experiments were carried out on cats under urethane anesthesia, and changes of the oxygen tension in cerebral tissue and skeletal muscle were recorded polarigraphically [11, 14]; the method has been described in detail previously [6]. We made simultaneous records of the respiration (pneumogram) and the arterial pressure (mercury manometer), and in some of the experiments we recorded the relative changes in the degree of arterial blood oxygen saturation (by means of a photoelement) and the rate of blood flow in the femoral artery and in the meninges (using a thermoelectric method).

**RESULTS**

The experiments showed that hypercapnia induced by respiration of a gaseous mixture containing 7% of carbon dioxide usually caused different oxygen tension changes in the different tissues: in the brain it was increased, while in the skeletal muscle, in most cases, it was reduced (Fig. 1a).

In analyzing the results of the influence of hypercapnia, in addition to the changes in the blood supply to the tissue we must also take into account the effect on metabolism and on the dissociation curve of oxyhemoglobin, but there is no reason to suppose that there will be any difference in the action of hypercapnia on the last two quantities as between different tissues. Nevertheless, changes of the oxygen tension in the brain and skeletal muscle in hypercapnia are usually in opposite directions, and run parallel to the changes in the blood supply to these tissues (in hypercapnia the blood supply to the brain is increased, while that to the muscles is reduced — Fig. 1b and c). We may therefore suppose that in hypercapnia changes in tissue oxygen tension are principally due to an alteration of the blood supply. It is however possible that during hypocapnia and in the subsequent recovery period oxygen tension changes in the tissues may be influenced by other factors, and the final result will be the result of a number of influences. Our observations indicate indirectly that this is in fact the case.

We must note that in hypercapnia, the direction of the changes of oxygen tension in the brain are more constant than in skeletal muscle. The explanation may be that in cerebral tissues all the factors mentioned above must act in the same direction to increase oxygen tension: in addition to an increased blood supply there is a shift in the
Oxygen tension changes in the tissues in hypocapnia (Fig. 2). Curves, from above downwards: for a) respiration, cerebral oxygen tension, muscular oxygen tension, arterial pressure, time of marker (5 seconds); for b) respiration, cerebral oxygen tension, arterial blood oxygen saturation, arterial pressure, volume of blood per minute flowing through meninges, time marker (5 seconds); for c) respiration, oxygen tension in the skeletal muscle, arterial blood oxygen saturation, arterial pressure, volume per minute of flow in femoral artery, time marker (5 seconds). Here and in the subsequent diagrams, a dotted line indicates the initial levels.

Dissociation of oxyhemoglobin to the right, which facilitates the transfer of oxygen from the blood into the tissue. According to published reports, in hypercapnia, the rate of oxidative processes in the brain either shows no change or is reduced, and if there is a reduced demand for oxygen it would be expected that the oxygen tension in the cerebral tissue would increase.

In hypercapnia, in muscle, besides factors leading towards an increase of oxygen tension (reduction of oxygen demand, change of the dissociation curve), there is another factor acting in the opposite direction, namely a constriction of the blood vessels. In most cases hypercapnia caused a fall of muscle oxygen tension, i.e., the effects of the changed blood supply prevailed over other influences. However, in some experiments with hypercapnia, instead of a reduction there was an increase of muscular oxygen tension, or else there was a reduction at the start of the experiment followed after repeated hypercapnia by an increased oxygen tension; under these conditions, there was no parallelism between the changes in the blood supply to the muscle, which was reduced, and the course of the oxygen tension changes. The probable explanation of the greater variability of the direction of the oxygen tension changes in muscle as compared with the brain is probably that the influences of the different factors in muscle act in different directions.

Hypocapnia, induced by artificial hyperventilation, also causes changes in various directions of the oxygen tension in the different tissues, but the changes are the opposite of those observed in hypercapnia: in the brain, oxygen tension is reduced, while in the skeletal muscle it is usually raised (Fig. 2a). The reduction of the oxygen tension in muscle and in brain at the end of a period of hyperventilation is due to apnea which develops as a result of the hypocapnia. These changes of the tissue oxygen tension are caused mainly by differences in the blood supply to the different tissues: in the brain the hypocapnia causes a reduction of the blood supply, whereas in the muscle it is increased (Fig. 2b and c).