EFFECT OF HYPEROXIA ON THE CIRCULATION OF ANESTHETIZED AND UNANESTHETIZED DOGS

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Under chronic experimental conditions inhalation of 90-96% oxygen by unanesthetized animals caused no regular changes in the arterial pressure and velocity of the blood flow in the carotid artery. Inhalation of oxygen by dogs anesthetized with Nembutal in every case caused a fall in arterial pressure and a considerable decrease in the velocity of the blood flow.

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The data concerning changes in the circulation in man and animals following inhalation of hyperoxic mixtures under atmospheric pressure conditions are conflicting. Some investigators [8] in experiments on animals observed a decrease in the systemic arterial pressure under these conditions, while others found no significant changes [2, 4, 11-13]. The regional blood flow of animals under hyperoxic conditions has been studied only in acute experiments [5, 6, 9].

We have investigated the hemodynamics in dogs when inhaling oxygen under chronic experimental conditions.

EXPERIMENTAL METHOD

Experiments were carried out on dogs having previously undergone operations and accustomed to the experimental conditions. Polyethylene catheters were implanted into the central and peripheral ends of the divided common carotid artery of the anesthetized animals. The catheters were brought out from the dorsal surface of the neck at the level of vertebrae C5-6. Between the experiments the catheters were filled with physiological saline containing heparin. The arterial pressure (mercury manometer), respiration (pneumographic method) and the volume velocity of the blood flow (our own method [3]) were recorded graphically on a kymograph. Air and gas mixtures containing 90-96% oxygen in nitrogen were administered through a mask with valves. The experiments were carried out on the same animals when unanesthetized and anesthetized with Nembutal (30-35 mg/kg intraperitoneally).

EXPERIMENTAL RESULTS

Inhalation of hyperoxic mixtures for 3-5 min in the experiments without anesthesia produced changes in the dogs' systemic arterial pressure in different directions. In 33 of the 55 cases the mean arterial pressure was increased by 2-19 mm. In 19 cases it was reduced by 2-12 mm below the initial level and in 3 cases it remained unchanged. The volume velocity of the blood flow in the carotid artery fell by 2-30 ml/min in 30 of 36 cases and in the rest it rose by 7-15 ml. On the average for the group these changes were not statistically significant.

In all experiments the pulmonary ventilation was reduced in the first few minutes of inhalation, while later in most cases it returned to its initial level and sometimes exceeded it.

Comparison of the changes in respiration, in the systemic arterial pressure, and in the blood flow in the carotid artery showed that when ventilation increased to its initial level or higher during prolonged hyperoxia, an increase in arterial pressure and in the blood flow was constantly observed.

In the experiments under anesthetia, inhalation of the same hyperoxic mixtures caused a significant decrease in arterial pressure (on the average from 125.8 ± 2.1 to 113.5 mm; $P < 0.01$). In all the experiments a decrease (by 10-70 ml) in the blood flow in the carotid artery was observed (on the average from 130 ± 3.98 to 110 ± 4.5 ml over a period of 4 min; $P < 0.01$).

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The decrease in pulmonary ventilation lasted throughout the period of oxygen inhalation.

During inhalation of hyperoxic mixtures by both anesthetized and unanesthetized animals no regular changes were observed in the heart rate. This is in agreement with previous findings [7, 10, 14].

The results of this investigation show that during inhalation of hyperoxic mixtures the systemic pressure of unanesthetized animals does not diminish, while in anesthetized animals as a rule it falls. This may perhaps be dependent on influences of the higher levels of the central nervous system of the reflex activity of the vasomotor center, as has been demonstrated in the case of the respiratory center in unanesthetized dogs [1].

The results obtained show that anesthesia significantly modifies the response of the circulation to hyperoxia.

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