EFFECT OF CHLORALOSE ANESTHESIA ON THE OXYGEN TRANSPORT FUNCTION OF THE BLOOD

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Experiments on waking dogs and dogs anesthetized by intravenous injection of chloralose (60-80 mg/kg) showed that chloralose has practically no effect on most indices of the oxygen transport function of the blood and produces only a very slight decrease in the oxygen saturation of the arterial blood and the minute oxygen transport by the arterial blood. The O_2 consumption is reduced by a greater degree; the balance between the O_2 supplied by the blood and its consumption by the tissues of the body is thereby unaffected. The results indicate that chloralose, given in the dose usually used experimentally, does not give rise to any manifestations of oxygen insufficiency such as are frequently observed when other general anesthetics are given.

KEY WORDS: chloralose; oxygen transport function of the blood; oxygen consumption.

It has been known for a long time that chloralose [11] has the advantage over other general anesthetics because it produces only weak depression of the reflexes [15]. Chloralose still remains widely used in experimental physiology [9]. However, even now it still remains one of the least studied anesthetics [3, 7]. According to some observations chloralose depresses the respiratory center [8] and slows the rate of respiration [12], but according to others it does not disturb respiration and can be used without the risk of development of hypoxia [9]. No information on the effect of chloralose on the respiratory function of the blood could be found in the literature.

This paper describes a study of the effect of narcotic doses of chloralose on the oxygen transport function of the blood and on some hemodynamic indices.

EXPERIMENTAL METHOD

Experiments were carried out on waking and anesthetized (1% chloralose solution, 60-80 mg/kg, intravenously) mongrel dogs. For sampling the arterial (left common carotid artery) and mixed venous blood (from a catheter introduced through the external jugular vein as far as the orifice of the venae cavae) and also for determining the hemodynamic indices, a preliminary operation was carried out on one group of dogs and all the tests were then carried out without anesthesia [1]. The anesthetized animals were investigated in acute experiments. The following blood levels were determined: the O_2 concentration, the oxygen capacity (with the AGK-2 apparatus), and the hemoglobin (Hb) concentration. The minute blood volume (MBV) was determined by the thermodilution method, the arterial blood pressure (BP) with an electromanometer, and the blood temperature by means of a thermistor introduced into the arch of the aorta (the hemodynamic indices were obtained on the anesthetized dogs by M. M. Koganovskaya). The oxygen saturation of the arterial (S_aO_2) and mixed venous (S_vO_2) blood, the arterio-venous O_2 difference, and the coefficient of O_2 utilization from the blood were calculated; the minute O_2 transport by the arterial (q_aO_2) and mixed venous (q_vO_2) blood was calculated from the O_2 concentration in the blood and the value of MBV; the O_2 consumption was determined as the difference between q_aO_2 and q_vO_2.

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TABLE 1. Principal Indices of the Oxygen Transport Function of the Blood and Hemodynamics in Waking and Anesthetized (chloralose) Dogs (M ± m)

Index studied	Waking dogs	Anesthetized dogs	P
Hb concn. (g %)	14,39±0,43	16,05±0,27	<0,001
O_2 capacity of the blood (vols. %)	19,25±0,58	20,99±0,33	<0,01
O_2 capacity of Hb (mg/g)	1,34±0,03	1,32±0,02	>0,5
O_2 concn. in mixed venous blood (vols. %)	18,71±0,58	19,46±0,32	>0,1
S_aO_2 (%)	97,3±0,5	92,6±0,4	<0,001
O_2 concn. in arterial blood (vols. %)	12,56±0,54	13,32±0,37	>0,1
S_vO_2 (%)	65,3±1,8	63,1±1,2	>0,1
Arterio-venous O_2 difference (vols. %)	6,15±0,40	6,20±0,26	>0,5
Coefficient of O_2 utilization (%)	32,8±1,9	32,1±1,3	>0,5
No. of cardiac concentrations per min	129,9±5,1	141,1±5,0	>0,1
BP (mm Hg)	108,7±7,4	143,8±1,4	<0,001
MBV (ml/kg/min)	139,0±7,8	123,1±4,4	>0,1
q_aO_2 (ml/kg/min)	24,9±1,9	21,5±1,4	>0,1
q_vO_2 (ml/kg/min)	17,2±1,7	15,0±1,2	>0,5
O_2 consumption (ml/kg/min)	7,68±0,41	6,42±0,41	<0,05
Temp. of arterial blood (°C)	38,91±0,15	39,15±0,16	>0,5

Legend. Number of dogs and their mean weight shown in parentheses.

EXPERIMENTAL RESULTS

The anesthetized animals differed from the waking dogs by their somewhat higher values of the oxygen capacity of the blood (Table 1). This was evidently explained by random selection of dogs with a higher Hb level in this group, although the Hb values in the animals of both groups were within normal limits of variation of this parameter for dogs [4].

Chloralose anesthesia had virtually no effect on S_vO_2 but was accompanied by a decrease in S_aO_2. The marked decrease in S_aO_2 during drug-induced sleep (chiefly with the use of barbiturates, urethane, and so on) is well known [2, 6, 18]. During natural sleep the degree of lowering of S_aO_2 in healthy persons usually does not exceed 4% [2], and accordingly the decrease of 4.7% in S_aO_2 observed in dogs must be regarded as hardly significant and similar to that observed during normal physiological sleep.

The decrease in S_aO_2 during sleep is considered to be attributable to the depression of respiration and the decrease in pulmonary and alveolar ventilation [6, 17], and also to a disturbance of the normal relations between alveolar ventilation and the blood flow in the lungs [14, 16]. These phenomena evidently also determined to some degree the small decrease in S_aO_2 observed under the influence of chloralose anesthesia. Although data obtained earlier [5] indicate some increase in the minute volume of respiration under the influence of chloralose, this increase took place only on account of the increased respiration rate; the respiratory volume actually showed some tendency to decrease, indicating that the increased ventilation of the lungs was not sufficient to improve the conditions of entry of O_2 into the body.

Administration of chloralose was accompanied by a slight increase in the heart rate, a marked increase in BP, and some decrease in MBV. These observations agree with data in the literature on changes in the heart rate and BP in chloralose anesthesia [13, 15, 19]. No precise data are available for the effect of general anesthetics on MBV [9].

The decrease in MBV produced by the action of chloralose determined the tendency for the minute O_2 transport of the blood to decrease.

Chloralose anesthesia caused a marked decrease (by 16%) in the O_2 consumption of the animal. This agrees with the decrease in the intensity of metabolism observed during anesthesia [18]. In dogs and cats anesthetized with chloralose a small decrease in O_2 consumption was noted [5, 10]. Meanwhile the absence of change in the temperature of the arterial blood during chloralose anesthesia evidently rules out a marked decrease in the basal metabolism.

The most interesting fact, from the writers' point of view, is that the decrease in the O_2 supplied by the arterial blood and in the O_2 consumption of the animal taking place under the influence of chloralose was so proportional that the ratio between them was virtually unchanged, despite the anesthesia. In waking dogs q_aO_2 was 3.25 times higher than the O_2 consumption, but 3.35 times higher in the anesthetized dogs. This indicates that chloralose, in a dose sufficient to cause anesthesia, does not disturb the balance between the O_2 supply by the blood and its consumption by the tissues.

Changes in the principal parameters of the oxygen transport function of the blood under the influence of chloralose are thus either not significant in degree or absent altogether. This suggests that chloralose in the doses usually used experimentally, does not give rise to those manifestations of oxygen insufficiency that are frequently observed when other anesthetics are used.

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