ANALYSIS OF THE CHANGES IN TONE OF THE VAGAL INNERVATION CENTER OF THE HEART DURING HYPOXIC AND ANEMIC HYPOXIA IN THE ADULT DOG

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In an earlier communication an analysis was given of the changes in nerve impulses recorded from the vagus nerve during the biphasic reaction of the heart (bradycardia and subsequent tachycardia) under conditions of morphine-ether anesthesia [4].

In laboratory studies the nature of this reaction was established for many forms of stressful stimulation in the dog, beginning at the age of $2^1/_2$ to three months. However, the question remained uninvestigated whether such a reaction is a universal form of maintaining homeostasis and determines subsequent changes in the degree of tonic stimulation of the vagal innervation center of the heart.

The present work evaluates the effect of hypoxic and anemic hypoxia on the tonic stimulation of the center of vagus innervation of the heart according to the characteristics of the nerve impulses recorded in the vagal fibers in the adult dog.

METHODS

The experiments were performed on adult dogs, in ten of which anemic hypoxia was induced and in 15—hypoxic hypoxia. Prior to carrying out the experiment the animals were given morphine (10-20 mg/kg). The central section of the left vagus nerve was freed from its sheath, separated from the sympathetic nerve and secured in a ligature. The basic method of recording the action potential from the vagus nerve was the same as in the preceding paper [14]. The first channel of the myograph (make "DISA") was used to record the action potential of the vagus nerve, the second to record the EKG from standard lead II. The third channel was used to record the action potential from the diaphragm or from the external intercostal muscles. Anemic hypoxia was produced by blood letting via a paraffin-coated cannula inserted into the femoral artery. Intravenous heparin was used to prevent blood clotting. Hypoxic hypoxia was produced by inhalation of a gas mixture poor in oxygen for 50-60 min, from a large Douglas bag.

RESULTS

In our experiments the inhalation of a gas mixture containing 18 and 16% oxygen initiated on the background of bradycardia conditioned by morphine, either provoked a slight speeding up of the cardiac rhythm or was not accompanied by changes in the rate of cardiac contraction. The initial slow cardiac rhythm corresponded to the relatively high level of vagal impulses (Fig. 1, a). Upon transfer to respiration of a gas mixture with a lower oxygen content, the level of vagal nerve impulses rose markedly (Fig. 1, b, c) not only during the modest bradycardial reaction but also during maintenance of the original cardiac rhythm. The increased level of impulses was maintained throughout the entire period of exposure. Upon switching to breathing air, the vagus impulses not only returned immediately to their original level, but in a number of instances became somewhat lower, although the cardiac rhythm during this time did not increase (Fig. 1, d).

Is the described increase in vagal tone during gradual lowering of the oxygen content of the inhaled air an

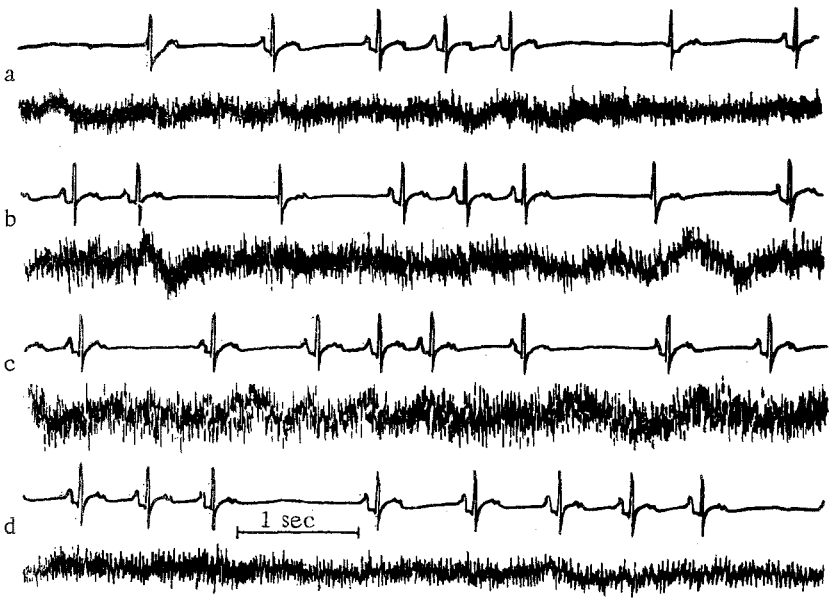

Fig. 1. Effect of moderate hypoxia on the rate of cardiac contraction and vagus impulses. a) Initial morphine background; b) when inhaling a gas mixture containing 18% oxygen; c) when inhaling a gas mixture containing 16% oxygen; d) when switched to inhalation of air. Upper line—EKG, lower line—vagus nerve impulses.

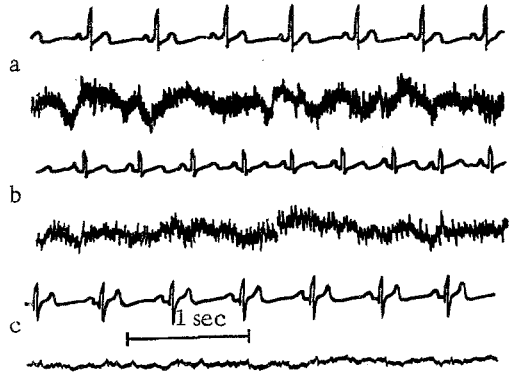

Fig. 2. Effect of profound hypoxia on frequency of cardiac contraction and vagus impulses. a) When breathing a gas mixture containing 7% oxygen; b) when breathing a gas mixture containing 5% oxygen; c) when breathing a gas mixture containing 7% oxygen on a background of urethane anesthesia. Significance of curves the same as in Fig. 1.

expression of increased stimulation of the sino-carotid and cardiac-aortic zone chemoreceptors [2, 3, 5, 6] or a special mechanism of the homeostatic reaction? The answer to that question requires further special study. When the oxygen content of the gas mixture is lowered from seven to five per cent, bradycardia changes to tachycardia (Fig. 2, a, b). With a switch to a respiratory gas mixture containing seven percent oxygen, the tachycardia was still gradually expressed in a number of experiments in comparison with the extremely high rate of cardiac contraction when a gas mixture containing five per cent oxygen was breathed. Vagal impulses in a number of instances remained sufficiently strong (see Fig. 2, a, b) despite a general tendency toward a decreased level. It may be hypothesized that in these cases the stimulating action of hypoxia on the sinocarotid and cardiac-arterial zone chemoreceptors continues to be maintained, while the origin of tachycardia is conditioned by the appearance of catecholamines in the blood. Under urethane anethesia, with hypoxic hypoxia (7-5% O_2), the vagus impulses completely disappeared and tachycardia did not arise (Fig. 2, c).

It must be noted that during the decrease in vagal impulses under urethane anesthesia which goes with tachycardia, the inhalation of a gas mixture containing 16-18% oxygen does not produce an increase in nerve impulses from the vagus or a corresponding slowing of the cardiac rhythm. In distinction from morphine, urethane depresses the capacity of the vagal innervation center of the heart to respond with a reflex reaction to the stimulation of the sinocarotid and cardiac-aortic zone chemoreceptors when the blood oxygen tension is lowered. It may be hypothesized that other narcotics also posses this propertie in doses which provoke transfer to the second, tachycardial phase of the reaction. In distinction from hypoxic hypoxia, in which the capacity to sustain the initial cardiac rhythm or to respond by the first, bradycardial phase of the reaction is retained for a long time, during anemic hypoxia this type of reaction was not detected in its classical form.

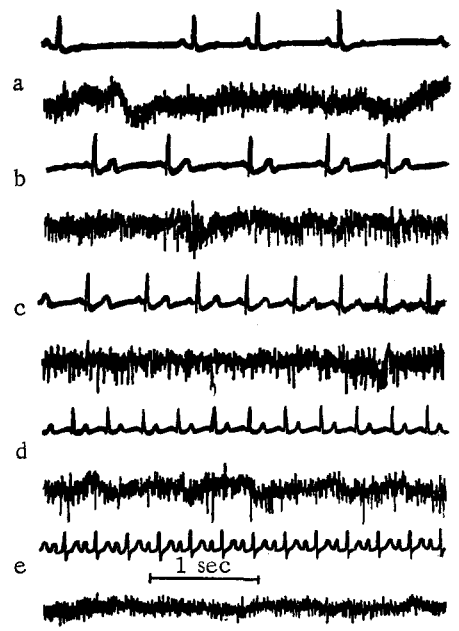

Fig. 3. Effect of blood loss on frequency of cardiac contraction and vagus impulses. a) Initial background; b) when blood loss equals 0.3% of body weight; c) when blood loss equals 1% of body weight; d) when blood loss equals 2% of body weight; e) when blood loss equals 3% of body weight. Significance of curves the same as in Fig. 1.

After blood loss equal to approximately 1% of body weight, the tachycardial reaction immediately appears in dogs (Fig. 3, a, c). When the blood loss comprises 0.5-0.25% of body weight, tachycardia also appears gradually, starting during the bloodletting. In three to five minutes after termination of blood letting the cardiac rhythm is noted to return completely to its initial level. The level of vagal inpulses does not correspond to the tachycardia which arises as the result of blood letting. It either does not change or even increases slightly (Fig. 3, b, c). Even after blood loss reaching 2-2.5% or somewhat more of the body weight, the high level of vagus impulses may be seen to be preserved (Fig. 3, d). Only when the volume of blood loss reaches 3% and the cardiac rate rises to 200-220 per minute, can some decrease in the level of vagus impulses be noted, which does not lead to its complete inhibition (Fig. 3, e). The depression of vagal impulses during marked degrees of both hypoxic and anemic hypoxia may be explained by its indirect effect on the nerve centers. Thus, despite the lack of a bradycardial phase of the reaction in anemic hypoxia, the nature of the reaction in the vagal innervation center of the heart is the same as in hypoxic hypoxia.

It has been discovered by laboratory studies that the tachycardia reaction as one of the adaptions to blood loss appears in puppies only after the 16-18th day of life, when the functions of the sinocarotid zone chemoreceptors first are detected [1]. This suggests that the tachycardia is a reflex, arising in connection with stimulation of the pressoreceptors in the sinocarotid and cardioaortic zones. It was further proved that increase in afferent impulses from these receptors produces inhibition of the vagal innervation center of the heart and thereby depression or even cessation of impulse generation by this center.

The data from the present study induce us to conclude that tachycardia which arises both during hypoxic and during anemic hypoxia does not result from the removal of vagal tonus. It may be suggested, as has been done in the preceding paper [4] that tachycardia which occurs under these conditions is produced by the indirect action of catecholamines on the heart, or, it is possible, by adrenaline-like substances. The outpouring of the latter into the blood may be conditioned by a reflex reaction from the above-mentioned receptor zones. Although the level of vagal impulses is preserved during this, it, evidently, is not able to overcome the more intensive action of humoral factors.

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