THE ROLE OF REFLEXES FROM THE SINOCAROTID ZONE IN REGULATION OF RESPIRATION DURING EXCESSIVE INTRAPULMONARY OXYGEN PRESSURE

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In published work there has been little more than a suggestion that reflex influences from the sinocarotid region play a definite part in the regulation of respiration under conditions of high pressure [2], and information is very incomplete. The object of the present work has been to study this problem.

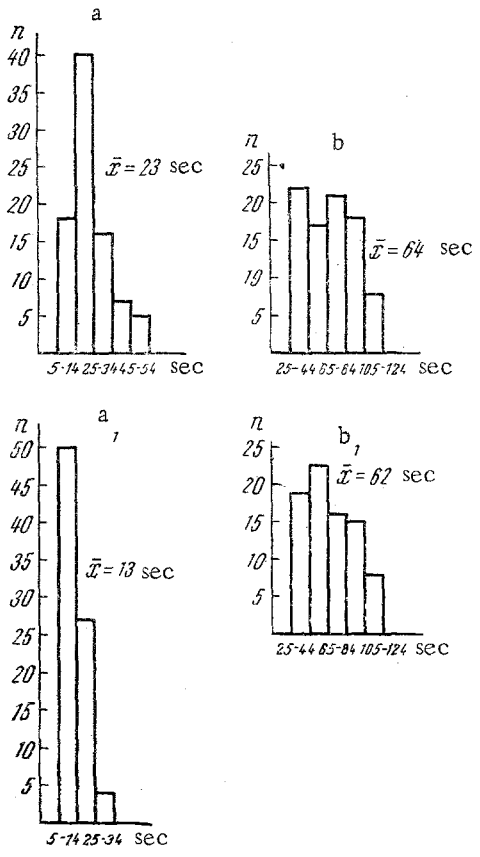

Fig. 1. Histograms showing variation in duration of respiratory delay (a, a_1) and the onset of respiratory delay (b, b_1) before (a, b) and after (a_1, b_1) compression of the common carotid arteries.

EXPERIMENTAL METHOD

Acute experiments were carried out on cats under 30 mg/kg intraperitoneal nembutal. Excess oxygen pressure of 30 mm Hg was produced by means of an oxygen apparatus connected to the lungs via a tracheotomy tube. The increased intrapulmonary pressure caused an arrest of respiration (apnea); then the respiratory movements were renewed—the so-called "escape"—after which respiration was arrested immediately. When the excess intrapulmonary pressure was relieved normal respiration started after a certain interval. The tests were repeated many times. In the intervals between them the animal breathed atmospheric air.

The influence of the sinocarotid reflexes on the work of the respiratory center during excess intrapulmonary pressure was inferred from the electrical activity of the internal intercostal muscles in the region of the cartilaginous portion of the ribs in the third and fourth intercostal spaces; also from the electrical activity of the rectus abdominis and external intercostal muscles, from the duration of the apnea, from the time of onset of the respiratory arrest before and after pressure was applied to the common carotid artery, and before and after denervation of the carotid sinuses.

The electrical activity was led off from the muscles by means of silver plate electrodes sewn in position. In 36 cats we studied the influence of compression applied to both common carotid arteries, and in 4 the effect was found of denervation of the carotid sinus.

EXPERIMENTAL RESULTS

Compression of both carotid arteries normally led to an increased frequency and depth of respiration and to an enhanced

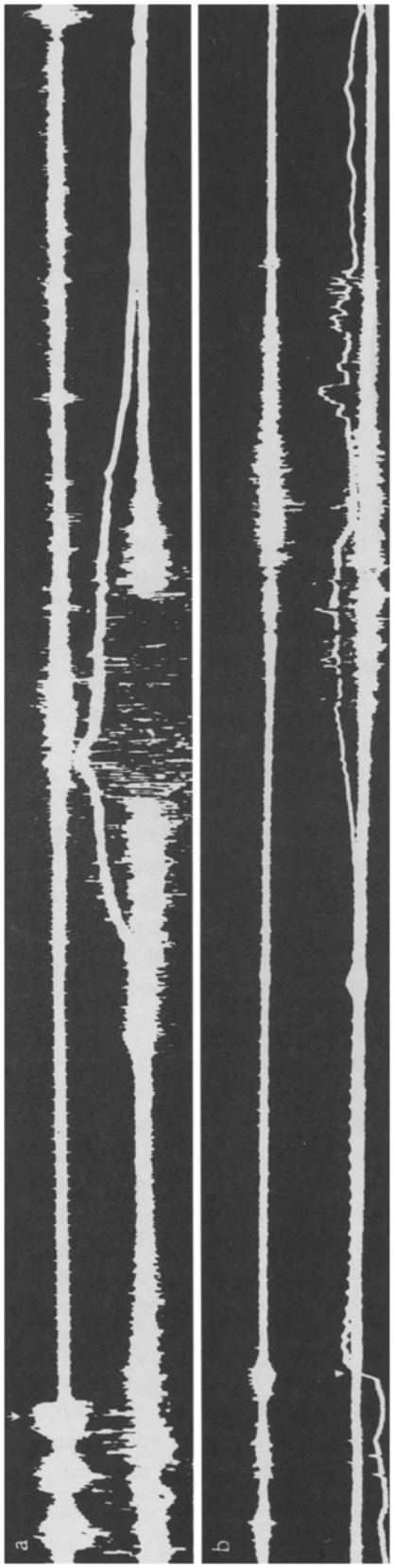

Fig. 2. Effect on the electrical activity of the respiratory muscles of denervation of the sinocarotid region. a) Before denervation; b) after denervation. In each frame: above—electromyogram of internal intercostal muscles; below—external/abdominal muscle. Respiration: inspiration shown by elevation of trace; the arrows show the time at which the excess intrapulmonary pressure was applied. Writing speed 4 mm/second.

electrical activity of the respiratory muscles. However this increase of respiratory activity was observed only for 2-3 minutes, after which respiration returned to normal. If, in this condition the intrapulmonary pressure was increased, in 29 cases of the 36 there was a considerable reduction in the duration of the delay in respiration (Fig. 1, a and a_1). With repeated tests the reduction in the respiratory delay was maintained for a long time (1-2 hours). When blood was once more allowed to flow through the carotid arteries the duration of the respiratory delay returned to the original value. In 7 cases, when the carotid arteries were compressed the duration of the respiratory delay in response to excess intrapulmonary pressure showed no change.

The time of onset of the delay in respiration in response to increased intrapulmonary pressure showed no appreciable change after compression of both common carotids (Fig. 1, b, b_1).

There was no appreciable alteration in the nature of the changes in electrical activity of the respiratory muscles under conditions os excess intrapulmonary pressure after compression of both common carotids.

Denervation of the sinocarotid region reduced the activity of the respiratory center which led to a decrease in the frequency and depth of respiration and to a weakened electrical activity of the muscles investigated (reduction in the total amplitude of the action potentials); when this occurred the electrical activity in the abdominal muscles fell far more than did the activity in the intercosal muscles (Fig. 2), and sometimes it disappeared altogether. In 2 out of the 4 cases there was a brief apnea immediately after denervation of the carotid sinuses.

Denervation of the sinocarotid zone caused an increase in the respiratory delay in response to excess pulmonary pressure. The table shows the mean values from 2-3 tests in each experiment.

There was no essential change in the time of onset of the respiratory arrest under conditions of increased intrapulmonary pressure after denervation of the carotid sinuses.

It is known that in the sinocarotid region baroreceptors are present which respond to an increased blood pressure by a flow of afferent impulses, and that chemoreceptors are present which react to a change in the constitution of the blood and in particular to an oxygen lack [3, 9].

When both the carotid arteries are compressed there is a sharp fall in blood pressure in the carotid sinus [5, 6], and at the same time there is a reduction in the flow of afferent impulses from the baroreceptors [5].

Because of the local hypoxia, after compression of the common carotid arteries chemoreceptors present in the carotid body bring about an increase in the discharge of afferent impulses [7, 10].

The increase in afferent impulses from the chemoreceptors

Effect of Denervation of the Sinocarotid Region on the Duration of Respiratory Arrest Occurring During Increase of Intrapulmonary Pressure

Number of experiment	Duration of respiratory delay during excess intrapulmonary pressure (in seconds)	
	before denervation of the carotid sinuses	after denervation of the carotid sinuses
36	12	16
38	20	35
39	15	22
40	17	24

and the reduction in the number of impulses from the baroreceptors excite the respiratory center, with the result that there is an increase in the frequency and depth of respiration and consequently an increase also in the electrical activity of the respiratory muscles. Other authors [8] have also observed an increase in the activity of the respiratory center after compression of the common carotid arteries.

After a time the blood supply to the carotid body improves through an increase in the anastomoses with other vessels [5], an effect which would be expected to reduce the number of afferent impulses from the chemoreceptors. Nevertheless the formation of anastomoses can scarcely influence the activity of the baroreceptors. In this way we may perhaps explain the prolonged reduction in the duration of the respiratory display.

At each test in which an excess intrapulmonary pressure was established it appears that there was a change in the activity of the chemo- and baroreceptors, a change similar to what is observed immediately after compression of the common carotid arteries.

In our experiments, when the excess intrapulmonary pressure was relieved we observed a reduction in arterial pressure to 60 mm. According to certain results [5], at such an arterial pressure the blood flow in the region of the carotid bodies ceases almost completely. Consequently impulsation from the baroreceptors is reduced, whereas discharges from the chemoreceptors are increased through oxygen lack. As a consequence, under conditions of excess intrapulmonary pressure reflexes from the sinocarotid region probably stimulate activity of the respiratory center, an idea which is confirmed by the increase in the respiratory delay after denervation of the carotid sinuses.

Because when there is excess pressure in the lungs the time of onset of respiratory arrest depends to a large extent upon hemodynamic factors, it would be expected that compression of the common carotid arteries would accelerate the onset of respiratory arrest through reduction in the blood supply to the brain; however no such effect was observed. Probably after compression of both common carotids compensatory mechanisms operate and the blood supply along the vertebral arteries is increased with the result that the brain begins to receive sufficient blood [1].

Reduction in the electrical activity of the respiratory muscles after denervation of the sinocarotid region is in line with results obtained by E. S. Fedorchuk [4], and supports the idea that the afferent flow from this region activates the respiratory center. Thus the results of our experiment indicate that under conditions of excess intrapulmonary pressure afferent impulses from the sinocarotid region exert and activating influence on the respiratory center.

SUMMARY

A study was made in cats of the effect on respiration under an oxygen partial pressure raised to 30 mm Hg of occlusion of the common carotid arteries and of denervation of the carotid sinus.

Occlusion of the common carotid arteries caused complete cessation of respiration when the oxygen partial pressure was raised, whereas denervation of the carotid sinus caused respiration to increase.

When oxygen partial pressure was increased neither occlusion of the common carotid arteries nor denervation of the carotid sinus significantly affected the time of respiratory arrest.

We concluded that the reflexes originating in the carotid sinus activated respiration under conditions of raised intrapulmonary partial pressure.

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