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RESPIRATORY MOVEMENTS OF THE FACIAL MUSCLES AND RESISTANCE TO BREATHING

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KEY WORDS: resistance to breathing; nosebreathing; nasophrenic reflex.

Resistance to breathing intensifies not only activity of the respiratory muscles of the chest, but also the respiratory movements of the facial muscles. The study described below showed that the latter are of great importance in compensating the respiratory disturbances caused by an increase in the resistance to breathing.

EXPERIMENTAL METHOD

Experiments were carried out on 10 tracheotomized rabbits, breathing spontaneously and anesthetized by intravenous injection of pentobarbital (30 mg/kg). Electrical activity was recorded from the muscles of the nostrils and from the diaphragm. The resistance to breathing was raised by occlusion of the tracheotomy tube.

EXPERIMENTAL RESULTS

According to data in the literature occlusion of the trachea causes an immediate increase in the amplitude and duration of the inspiratory volleys of the diaphragm [2, 4]. The corresponding changes in electrical activity recorded from muscles of the alae nasi were absolutely synchronous, but there was one important difference. The increase in the discharges in muscles of the alae nasi soon after occlusion of the trachea was significantly greater than the increase in discharges in the diaphragm (Fig. 1). In some experiments mouth-breathing also began or became more marked. In the tracheotomized animal air can enter the lungs without having to pass through the upper respiratory passages. The fact that respiratory movements of the facial muscles still take place under these circumstances provides an exceptional opportunity of investigating their role in the mechanism of compensation of breathing disturbances caused by an increase in the resistance to breathing.

As already mentioned, an increase in resistance to breathing causes an increase in the amplitude and duration of inspiratory volleys of the diaphragm. If, against this background, the respiratory movements of the facial muscles are obstructed by squeezing by the hand, further intensification of the respiratory volleys of the diaphragm immediately occurs (Fig. 2). Preventing respiratory movements of the facial muscles evidently causes even greater breathing disturbances.

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Fig. 1. Inspiratory discharges of diaphragm (1) and muscles of alae nasi (2) after occlusion of trachea (arrow): Discharges in muscles of alae nasi are increased more than those of diaphragm.

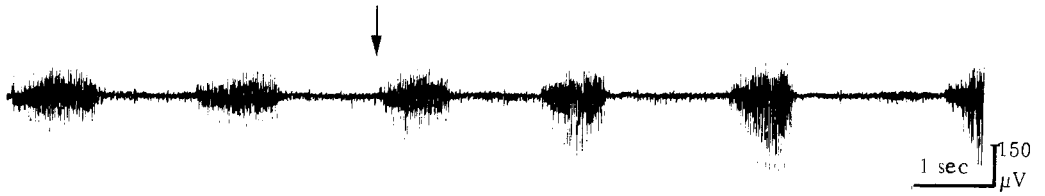

Fig. 2. Obstructing respiratory movements of facial muscles of a tracheotomized rabbit (arrow) immediately intensifies inspiratory volleys in diaphragm.

Similar obstruction of respiratory movements of the facial muscles in the tracheotomized animal before the resistance to the animal's breathing had been increased did not give rise to any clear changes in breathing.

As was observed during occlusion of the trachea, inspiratory volleys of the muscles of the alae nasi were increased much more abruptly than volleys of the diaphragm. The same relationship between activity of the intercostal muscles and diaphragm during mechanical occlusion of the respiratory passages also was found by the writers previously: Volleys of the intercostal muscles were intensified much more than those of the diaphragm [3]. This can evidently be explained on the grounds that Golgi receptors are predominant in the diaphragm, and impulses from them lead to self-inhibition of activity of the diaphragm, whereas in the intercostal muscles stretch receptors predominate, and impulses from them intensify the stretch reflex. This evidently also explains the fact that activity of the facial respiratory muscles is intensified more, when the resistance to breathing is increased, than volleys from the diaphragm.

However, as already stated, compressing the alae nasi before occlusion of the trachea caused no clear changes in respiration. Consequently, the effect of stopping respiratory movements of the facial muscles depends, not on stimulation of the nasal mucosa, but on the interruption of the flow of impulses from stretch receptors, which accompanies respiratory movements of the facial muscles.

It is generally considered that reflex intensification of activity of the respiratory muscles in response to an increase in resistance to breathing is caused by a decrease in the flow of impulses from stretch receptors of the lungs, which normally inhibits the respiratory center, and by an increase in the flow of impulses from stretch receptors of the intercostal muscles, which facilitates spinal motoneurons [2, 4, 5, 7]. The present experiments demonstrate that impulsation not only from the intercostal muscles, but also from the facial muscles, participates in the compensation of the respiratory disturbances caused by occlusion of the respiratory passages. A fact that calls for special attention is that this mechanism continues to act as long as air enters the lungs through the trachea and not through the upper respiratory passages. This is evidence that it is not stimulation of the nasal mucosa by air that determines the compensation mechanism, but impulsation from stretch receptors of the facial muscles.

Almost half of the total resistance to the airflow arises in the nose; nevertheless, we prefer to breathe through the nose [1, 8]. Impulsation arising from muscle stretch receptors during nosebreathing is probably very important for maintaining the normal activity of other respiratory muscles and, in particular, of the diaphragm (the principal respiratory muscle). In fact, after tracheotomy breathing becomes slower and more superficial. In connection with the proprioceptive nasophrenic reflex which has been described, special interest attaches to

the excitatory intercostophrenic reflex, i.e., increased activity of the diaphragm evoked by impulsation from the proprioceptors of the lower intercostal muscles [5, 6]. The two reflexes serve the same purpose: to compensate for the paucity of stretch receptors in the diaphragm, which prevents it from facilitating its own activity.

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THE USE OF HYPEROXIC MIXTURES FOR THE DIAGNOSIS OF LATENT DISTURBANCES OF EXTERNAL RESPIRATION

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KEY WORDS: hyperoxia; diagnosis; external respiration.

The degree of oxygen saturation of the arterial blood is a very important integral parameter of external respiratory function. Under normal conditions venous blood, as it passes through the lungs, is oxygenated up to 95% with oxygen. In the presence of various pathological processes in the effector part of the external respiratory system and the regulatory systems, the level of blood oxygenation may fall. The oxygen saturation of arterial blood (SaO_2) is reduced by alveolar hypoventilation, disturbance of the diffusion properties of the alveolo-capillary membrane, disparity between ventilation and the blood flow in different parts of the lung, and shunting of blood in the lungs [4, 6, 8].

Definite correlation is known to exist between the value of SaO_2 and pO_2 of arterial blood, and this relationship is described by nomograms [5, 7]. High values of p_aO_2, close to 100 mm Hg, always correspond to a high degree of blood oxygenation.

The use of hyperoxic tests widens opportunities for analysis of the role of individual mechanisms impairing gas exchange in the lungs [2, 3, 8, 9]. A leading role in hyperoxic loading must be ascribed to possible changes in the alveolo-arterial oxygen gradient ($\Delta P_{A-a}O_2$), a composite parameter whose value is mainly determined by interaction between reduced alveolo-capillary permeability for oxygen, a disturbed distribution of air in the lungs, and the volume of venous shunting [1, 3, 8].

In the investigation described below values of $\Delta P_{A-a}O_2$ were determined in healthy subjects and patients with chronic nonspecific lung diseases, inhaling hyperoxic gas mixtures, and the characteristics of the disturbances of gas exchange in the lungs were recorded and evaluated by a graphic method, a further development of the technique of comparing p_AO_2 with SaO_2 [4].

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