THE REFLEX NATURE OF THE PHENOMENON OF "LAGGING" OF RESPIRATORY MOVEMENTS IN UNILATERAL LUNG LESIONS

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One of the clinical signs of unilateral lung lesions – abscess, atelectasis, pneumonia, etc. – is the so-called "lagging" of the chest cage on the side of the lesion, i. e. the slowing down of respiratory movements and diminution in their amplitude, most marked in the region of the lower ribs.

Such disturbances of function of the respiratory musculature appear to be reflex in nature, as supposed by the majority of clinicians (M. P. Konchalovskii, E. M. Tareev, E. M. Gelshtein, V. F. Zelenin, Stehelin and many others), indicating that when lobar pneumonia, pleurisy and similar diseases are encountered in clinical practice it is difficult to exclude the possibility of involvement of the diaphragm itself by a secondary inflammatory process.

Focal lesions in the lungs of animals can be produced experimentally without simultaneous inflammatory involvement of the diaphragm. However, the phenomenon of "lagging" cannot always be visually demonstrated even in the presence of extensive involvement of the lungs because of the lesser mobility of the chest cage in animals in which the shoulder girdle performs a supporting function [5]. For this reason we considered it expedient to investigate changes in respiratory movements employing electromyographic analysis of diaphragmatic contractions on the side of lung involvement.

EXPERIMENTAL METHODS AND RESULTS

Experiments were carried out on unanesthetized rabbits with laminar electrodes implanted onto the diaphragm. Biological currents were recorded using an encephalographic amplifier made by the Experimental factory of the AMN SSSR and an oscillograph MPO – 2.

Focal injury to the lungs was produced by the introduction into the lung through the chest wall of 2 ml of hot water (90°). In view of the fact that animals withstand poorly the bilateral suturing of electrodes to the diaphragm, experiments were run in two series: in one series the movement of the diaphragm on the side of the injury was recorded, in the other – on the opposite side. In connection with recording biological currents using laminar electrodes which permit taking into account only total changes in the activity of a given muscle segment, this investigation was limited to the study of changes in the amplitude of impulses. When amplification was used during quiet respiration the base line of the discharge on inspiration consisted of impulses of the order of 20 – 30 μv and synchronized impulses of the order of 40 – 50 μv.

In the presence of limited injury to the lungs, changes in the biological currents in the diaphragm follow an established pattern. Immediately upon the introduction of hot water an uninterrupted impulse with amplitudes 4-5 times greater than initially was observed. This reaction is more pronounced on the side of the injury than on the opposite side on which ordinarily there is a greater degree of preservation of the phase of increase and decrease of the impulse amplitudes corresponding to inspiration and expiration.

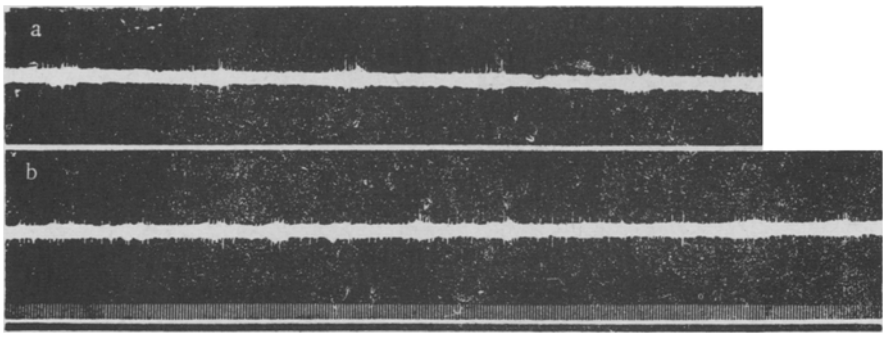

Fig. 1. Reaction of a rabbit diaphragm to injury of the homolateral lung. Electromyograms of the diaphragm: a) initially; b) several seconds after lung injury (diaphragmatic activity does not exceed the initial level despite considerable increase in respiratory rate). Amplification is 50 μv = 5 mm.

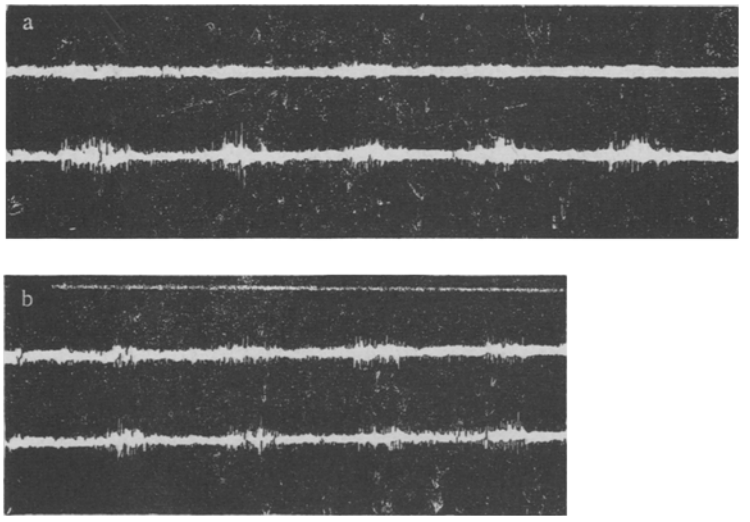

Fig. 2. Difference in reactions of the diaphragm and nasal muscles to lung injury. Tracings (from above down): electromyogram of alae nasi, electromyogram of the diaphragm.
a) Initially; b) 2 minutes after injury of homolateral lung (diaphragmatic activity less than initially, activity of nasal muscles is considerably higher). The recording rate is 3 cm/sec, amplification 50 μv = 5 mm.

On the side of injury the diaphragmatic impulse returns to its initial size in 1-2 minutes, and at times in several seconds, following lung injury, despite the persistent rise in the respiratory rate (Fig. 1). The more common impulses of lesser amplitude return to normal first and those of higher amplitude follow.

In addition, during the next 2-5 minutes the electrical activity of the diaphragm on the side of the lung lesion further weakens in most of the experiments, frequently dropping below the initial level, which is manifested in decrease in the amplitudes of the frequent impulses as well as in the almost total disappearance of high-amplitude impulses (Fig. 2). On repeated injury to the same lung this weakening of activity of the diaphragm lasted much longer (provided that the injected fluid was always introduced into lung parenchyma and not into a bronchus).

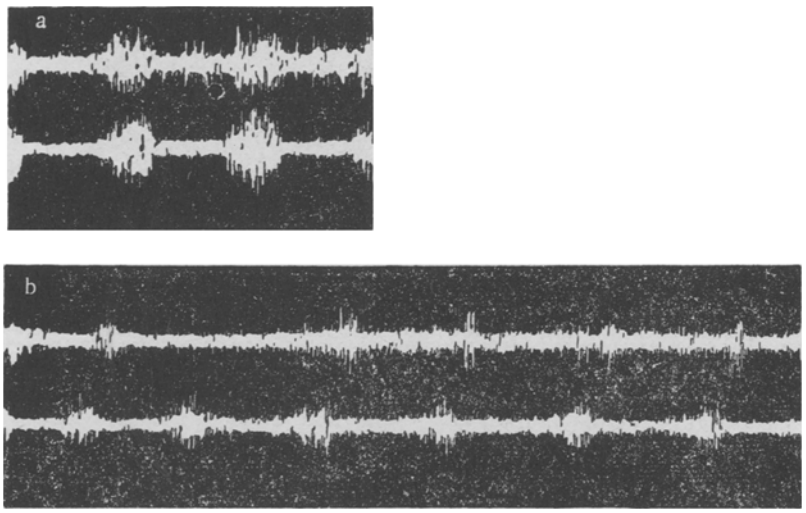

Fig. 3. Reaction of a rabbit diaphragm to injury of the contralateral lung. Tracings (from above down): electromyogram of alae nasi, electromyogram of the diaphragm.
Lower trading — initially; upper — 8 minutes after lung injury (diaphragmatic activity significantly increased). The recording rate is 3 cm/sec, amplification 100 μv = 11 mm.

On the contralateral side a rise in activity of the diaphragm, rather than a fall, was observed (Fig. 3) which lasted not less than 4-5 minutes following a single injury and considerably longer (from 15-20 minutes to several hours) following repeated injuries. This reaction could be abolished by injury to the lung of the same side, and conversely, weakening in activity of the diaphragm could be replaced with strengthening upon additional injury to the lung of the opposite side.

It must be pointed out that the phenomenon of "lagging" is associated not only with a decrease in amplitude of the respiratory movements on the side of the injury, but also with their slowing down, which at times can create the impression of paradoxical movement of the diaphragm (rise on inspiration and fall on expiration). However, some roentgenologists already turned their attention to the phenomenon that paradoxical movements of the diaphragm are possible only when the phrenic nerve is divided or paralyzed or when the lesion is in the diaphragm itself; in the presence of lesions in the lung, one observes only prolongation of the active phases of inspiration and expiration due to slowing down of the contraction of muscle [2, 4, 6, 8].

In experiments on rabbits this phenomenon is difficult to establish because of considerable activity of the diaphragm between inspirations which makes evaluation of the duration of discharge difficult. Preliminary observations on cats (under nembutal anesthesia) indicated that following injury to the lung one could observe alongside the fall in the amplitude of the impulse also a prolongation of discharge on inspiration — up to 2 seconds instead of 1.3 seconds prior to the injury.

Thus, our experiments indicated that at the basis of the phenomenon of "lagging" of respiratory movements on the side of lung lesions are reflex suppression of diaphragmatic activity and simultaneous increase in activity of the contralateral diaphragm.

In connection with this, it must be pointed out that although ontogenetically the diaphragm arises as a paired organ, later on the muscle as well as its innervation unite anatomically as well as functionally to such a degree that some investigators think it possible to regard it as a single unpaired organ [7]. At the same time under pathological conditions when a lesion is present in one of the leaves of the diaphragm, the latter is able like a paired organ to alter its function unilaterally. Thus, compensatory hyperfunction of one of the leaves is well known in the presence of paralysis or paresis of the contralateral phrenic nerve, when the latter is divided or injected with alcohol, and when the muscle tone of the contralateral leaf is diminished following an inflam-

matory process, pneumonectomy, lobectomy, etc. Such compensatory hyperfunction is possible in the presence of severe disturbance of innervation of the diaphragm because of the so-called cross phenomenon of the diaphragm[3].

However, if in the presence of all these pathological processes diaphragmatic hyperfunction could be ascribed to compensation for the injured organ, our experiments indicated that this compensatory exaggeration can be a component of a general reflex reaction which expresses itself in the form of a simultaneous decrease on the side of injury and an exaggeration of contractions on the opposite side. Thus, injury to the lung parenchyma causes reflexly an active dynamic reconstruction of the centers regulating diaphragmatic movements.

At what levels is the force of contraction of the diaphragm determined?

It could be supposed that this depends on the inequality of the strength of impulses from the right and left halves of the respiratory center. Such an assumption is contradicted by the observation that an increase in activity is observed not only in the contralateral diaphragmatic leaf, but also in the respiratory muscles of this same side. Activity of nasal muscles is also increased (see Fig. 2). Apparently the general intensification of the impulse in the respiratory muscles caused by stimulation of the respiratory center when one of the lungs is injured, is actively suppressed only in structures regulating diaphragmatic movement. It can be supposed that for the diaphragm this level is in the 5th and 6th segments of the cervical spinal cord which contain the phrenic nerve center.

Our previous investigations indicated the possibility of such a selective suppression of the diaphragm in the presence of a generalized increase of activity of all other respiratory muscles under conditions of simulated asphyxia [1].

The afferent paths of the reflex "lagging" of the diaphragm on the side of a pulmonary lesion require further study. It is possible to suppose that they must include sensory fibers of the phrenic nerve, inasmuch as there is an abrupt change in the tone of the diaphragm at the moment of lung injury.

SUMMARY

In the presence of a unilateral focal pulmonary lesion the electric activity of the diaphragm is decreased on the homolateral side and increased on the contralateral side. This reflex lies at the basis of the physical sign known as "lagging" of respiratory movements on the side of the pulmonary lesion. The absence of compensatory increase of respiratory movements on the side of the lesion is evidently connected with active suppression of diaphragmatic activity taking place in the phrenic nerve center.

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