RESPIRATORY ELECTROMYOGRAMS DURING RESISTANCE TO BREATHING BEFORE AND AFTER VAGAL SECTION

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A number of studies have been reported of the effect on ventilation, gaseous exchange, and respiratory movements by added resistance to breathing [1, 2, 5, 6, 9, 11, 22, 25, 27, 29, 37]. The respiratory movements were usually recorded pneumographically, and the work performed by the respiratory muscles was inferred only from their total effect. Nevertheless, it is known that respiratory disturbances may result from the impairment of individual muscles. The most convenient method for study has been electromyography (EMG).

The large amount of material on the EMG of the different respiratory muscles has accumulated since Dittler in 1909 first recorded potentials from the diaphragm. Some of the experiments were performed on animals [4, 7, 16, 19, 21, 30, 32, 36] and some on man [8, 13, 18, 24, 26, 31, 34].

There are only occasional references to the electrical activity of the respiratory muscles in man when breathing against a resistance. Campbell made a study of the potentials from the muscles of the abdomen. Normally, when surface electrodes are used, electrical activity from the muscles is not recorded. Campbell showed that it does not appear in these muscles when there is an added resistance. L. L. Shik [8] recorded the electrical activity of the intercostal muscles by means of surface electrodes and showed that it increases with added resistance to breathing.

We have made a simultaneous study of the electrical activity of the diaphragm , intercostal , and oblique abdominal muscles in experiments in which an added resistance to breathing was established chronically.

METHOD

The experiments were performed on 10 unanesthetized dogs weighing 9-17 kg. In the first series, resistance was added to the breathing of normal animals, and in the second series the vagi were divided. All the animals were taught to lie on their side on the table and to breathe through a mask. Potentials were recorded by means of electrodes sewn directly into the respiratory muscles [3]. Silver plate electrodes having an area of 10 × 6 mm were sewn to the left arch of the diaphragm and to the left oblique abdominal muscles through a slit in the peritoneum, the distance between the electrodes being 1.5 - 2 cm; smaller electrodes measuring 5 × 3 mm were attached to the intercartilaginous portion of the left internal intercostal muscles in the fifth intercostal space near the sternum at a separation of 1 cm. The potentials from these muscles were amplified in a three channel amplifer and recorded on a cathode ray oscillograph, the overall sensitivity being 30-100 μv/mm.

The resistance was added by a device which compelled the dog to breathe through a water column of a certain height. The preliminary results showed that there were considerable variations in the amount of resistance which each animal could tolerate. In our experiments therefore we did not use a standard value but one which was maximal for each animal, and which varied from 5 to 20 cm of water. In most experiments the pulmonary ventilation was measured by gas flowmeters. Muscle potentials and pulmonary ventilation were measured before and after introducing the added resistance. The experiment was repeated after dividing the vagi in the neck under local novocaine anesthesia.

RESULTS

Normal EMG of respiratory muscles. Before the added resistance (Fig. 1) the EMG of the diaphragm is asynchronous, gradually increasing at inspiration and decreasing at the end of expiration. With our method of recording, the maximum amplitude of the potential varied between 200 and 500 μv. At expiration the potential was smaller, lying between 50 and 300 μv. Diaphragm activity has previously been recorded both at expiration

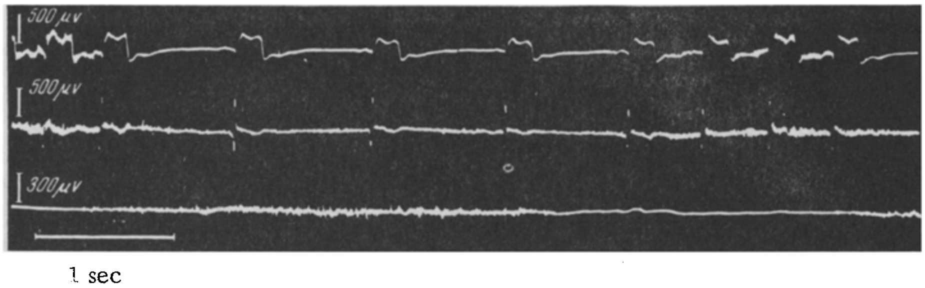

Fig. 1. Normal electromyogram of the respiratory muscles (dog Faithful, experiment of December 18, 1958). Curves (from top down): electromyogram of intercostal muscles, diaphragm, and oblique abdominal muscles.

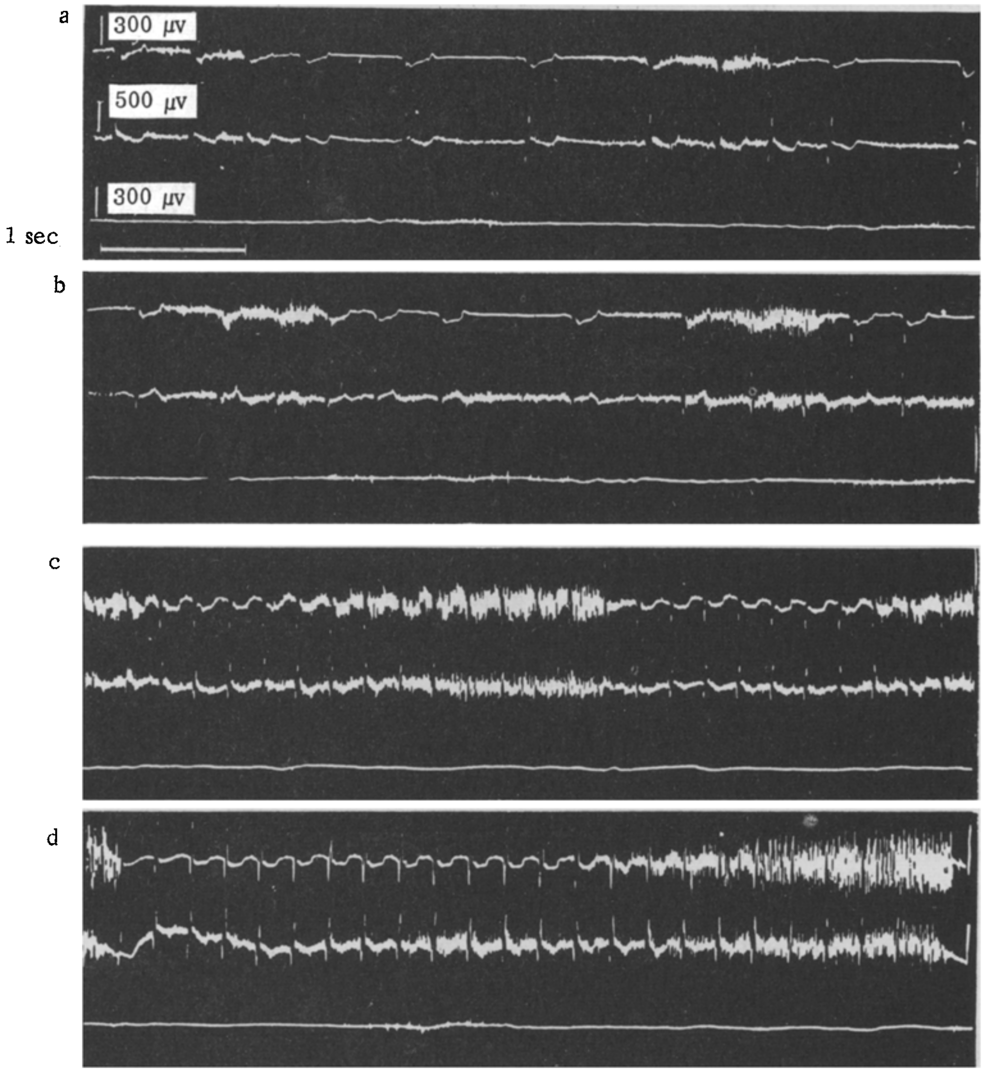

Fig. 2. Reaction of the respiratory muscles to a resistance to breathing before and after vagotomy (dog Faithful, experiment of March 23, 1959). Curves as in Fig. 1. a) Original background; b) after introducing resistance to breathing; c) after vagotomy, before increasing resistance; d) after increasing resistance.

and inspiration by other authors [16, 23, 31, 36]. These results correspond with the x-ray findings which indicate that the diaphragm does not relax completely at expiration.

The results on the electrical activity of the intercostal muscles obtained by previous workers are contradictory. The differences are evidently due to variation in the position of the electrodes. Nevertheless, it has been repeatedly shown that the potentials from the intercartilaginous portion of the internal intercostal muscles arise chiefly at expiration [10, 20, 30]. Our experiments have confirmed that the EMG of this part of the internal intercostal muscles typically shows impulses at inspiration and that they coincide with those from the diaphragm. Their amplitude varies between 120 and 400 μv.

Recordings from the internal oblique muscles showed that in the great majority of experiments there was an increase followed by a gradual decrease of electrical activity only at expiration, though occasionally there was an intermittent, but increasing, discharge during this phase. The maximum amplitude of the potentials was 50-250 μv.

Simultaneous recording from the different respiratory muscles showed that changes in diaphragm activity at expiration are closely associated with the changes in activity of the oblique muscles. The latter increases at expiration together with increased action from the diaphragm. Evidently, the activity of the diaphragm at expiration is brought about reflexly by its extension, prevents the sudden collapse of the lungs and brings about the normal smooth expulsion of the air. It is important to note that the oblique muscles first contract before the action of the diaphragm at inspiration has been completed. It appears that such an overlap enables a smooth changeover to be made between the inspirational and expirational phases [14].

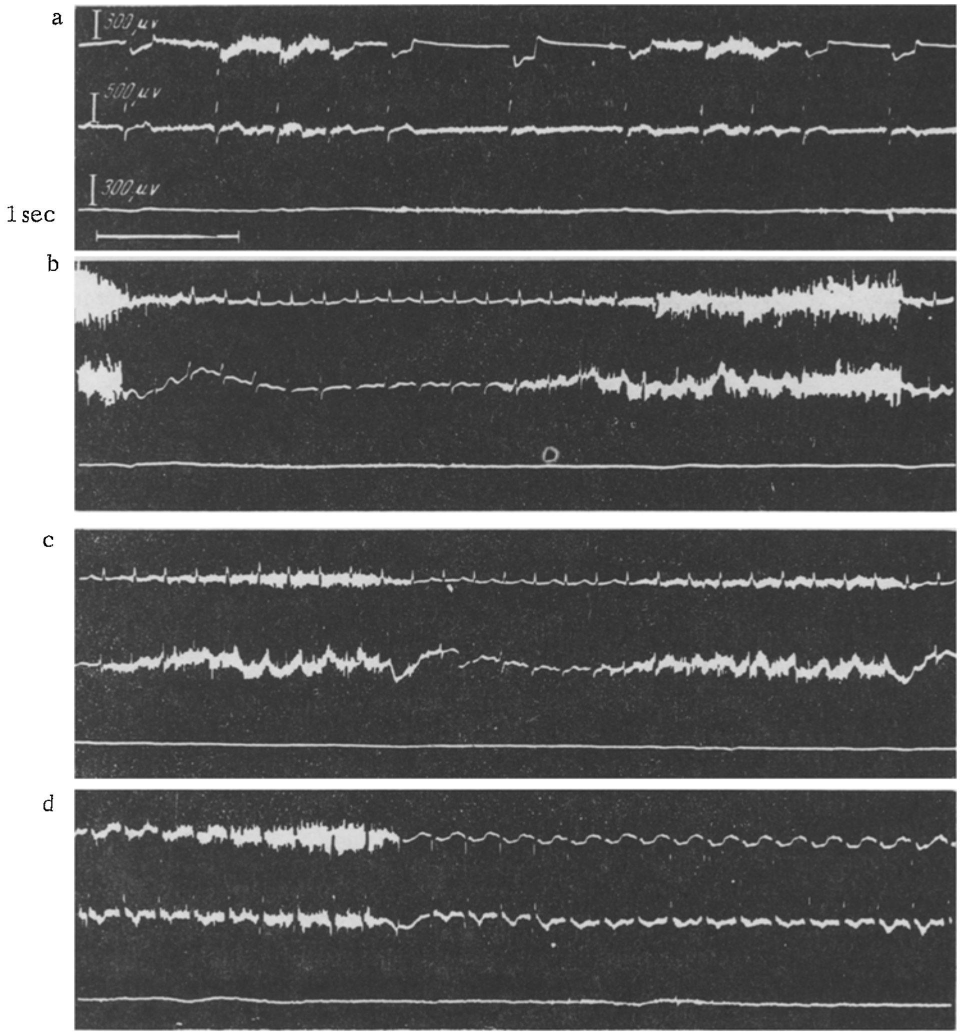

Fig. 3. Changes in the electrical activity of the respiratory muscles after vagotomy (dog Belka, experiment March 23, 1959). Curves as in Fig. 1. a) Before vagotomy; b) immediately after vagotomy; c) one hour after vagotomy; d) one day after vagotomy.

The EMG has shown that when there is a resistance to breathing, there is an increase of both inspiratory and expiratory muscle potentials, the amplitude increasing by 20-100% (Fig. 2a and b). However, despite the increased activity of the respiratory muscles, pulmonary ventilation was reduced by 5-15%.

EMG of the respiratory muscles after vagotomy. Before the increased resistance was introduced (Fig. 3), vagotomy by itself caused a marked change in the activity of the respiratory muscles. The discharge from the diaphragm and intercostal muscles after vagotomy showed a high rapidly increasing amplitude at inspiration, which immediately ceased when the breath was fully drawn, instead of relaxing gradually as in a normal animal. The amplitude of the potentials increased by as much as 2-6 times. Wachholder and McKinley [36] found a similar increase in the response of the diaphragm in vagotomized rabbits, and Meda and Ferroni [28] found the same effect in guinea pigs.

By contrast, the amplitude of the discharge from the internal abdominal muscles after vagotomy was markedly reduced, and often disappeared altogether. There was a corresponding reduction in amplitude or disappearance of the potentials from the diaphragm. The overlap of activities of the diaphragm and oblique muscles, which was commonly observed in normal animals,was not present after vagotomy.

Thus, the increased depth and slowing of the respiration occurring after vagotomy is associated with a marked increase and duration of the activity of the inspiratory muscles, while the activity of the muscles of expiration is reduced or absent. In addition, the respiration is less even.

Some time after vagotomy, the activity of the diaphragm and intercostal muscles at inspiration gradually became reduced, though it remained above normal. At the end of inspiration it broke off less sharply. The oblique muscles were active at inspiration a few hours after vagotomy, though sometimes not until two days afterwards, but the contractions did not have their full strength (see Fig. 3).

In vagotomized animals, added breathing resistance increased the amplitude of the potentials from the diaphragm and intercostal muscles at inspiration to a value 50-200% above the normal level (Fig. 2e and d).

When resistance to breathing was added, the electrical activity of the respiratory muscles was increased while pulmonary respiration was reduced, and this lack of correspondence was greater after vagotomy. Despite the marked increase in the amplitude of the potentials from the respiratory muscles, pulmonary ventilation decreased by 10-40%. By the second day after vagotomy, the increase in potentials from the respiratory muscles caused by increasing the resistance was not so well shown as on the first day (see table).

TABLE. Increase in the Amplitude of Impulses from the Diaphragm in Response to Added Breathing Resistance, on the same day as the Vagotomy, and two days after (in percent).

Name of dog	Amplitude of potentials from diaphragm	
	On the same day	On second day
Mushka	122.0	20.0
Volchok	150.0	80.0
Belka	66.6	13.3

It is known that normally respiratory movements show a characteristic gradual inspiration and expiration and a smooth transition between them. Our experiment showed that this effect is normally brought about by the gradual waxing and waning of the activity of the respiratory muscles, by the action of the diaphragm at expiration, and by the overlap of the periods of action of the diaphragm at inspiration and of the oblique muscles at expiration.

Division of the vagi seriously disturbs the coordination of respiratory movements. After vagotomy there is a marked increase in the activity of the diaphragm and intercostal muscles at inspiration, but it immediately breaks off at the end of this phase; during inspiration the action of the oblique abdominal muscles ends sharply; "during inspiration" lastly, there is no overlap between the inspirational and expirational phases.

It is known that most of the afferent impulses to the respiratory center from the lungs pass along the vagi. It might be thought that the failure of coordination of the respiratory muscles which has been described above might be attributed to disturbance of the respiratory center following its partial deafferentation by vagotomy. At a certain time after vagotomy has been performed, respiration becomes more or less even. Our records showed that this effect is brought about by the respiratory muscles ceasing their activity at input less sharply, and developing an action at expiration. The reason for this change is apparently that at a certain time after vagotomy there is an increase in the afferent inflow along other pathways such as the spinal nerves, sympathetic pulmonary nerves, and the sensory nerves of the pleura and of the respiratory muscles.

There are two views as to what is the mechanism of the change in the respiratory movements which follows increased resistance. Some authors consider that the explanation must be in terms of humoral regulation and particularly the accumulation of CO_2 in the blood [11, 25, 37]; others emphasize the importance of nervous regulation, and particularly the part played by the vagi [1, 8, 15, 17, 27, 35].

Our results favour the latter view. The EMG showed that resistance produced a greater change in vagotomized than in normal animals. The result was less well shown on the second day after vagotomy. It is known that the EMG of the respiratory muscles is an index of the condition of the respiratory center. It may therefore be supposed that the increased response to added resistance immediately after vagotomy is due to an increased excitability of the respiratory center brought about by the elimination of the principal afferent pathways. The reduction in the response on the second day after vagotomy is probably due to a reduction in the excitability of the respiratory center, which is caused by other afferent pathways being established.

Because the oblique muscles are no longer active after vagotomy, the restoration of their function in vagotomized animals when resistance is added indicates that the increased resistance acts as a further afferent stimulus to the respiratory center.

From what has been said, it follows that changes in the excitability of the respiratory center, which depend on an alteration in its afferent supply, play an important part in bringing about the functional changes in the respiratory musculature which are induced added resistance to respiration.

It has long been known that the increase in respiratory movements brought about by added resistance does not correspond to pulmonary ventilation. L. L. Shik and his co-workers found that in various pulmonary conditions ventilation was unchanged despite an increased activity of the respiratory muscles. The marked lack of correspondence between the increased activity of the respiratory muscles and the reduced ventilation which we observed to occur against an added resistance after vagotomy shows that the pulmonary ventilation depends not only on the activity of the respiratory muscles, but also on other factors associated with the vagi, such as control of the tone of the bronchials, innervation of the larynx, and the development of physiological atelectasis.

SUMMARY

Electrical activity of the respiratory muscles, including diaphragm, intercartilaginous part of the internal intercostal and internal oblique abdominal muscles, was recorded from electrodes chronically implanted in dogs. The disturbed coordination of the respiratory muscles which followed vagotomy was indicated by a sharp arrest of the increased electrical activity at the end of inspiration, and its failure to occur during expiration; in addition there was no longer any overlap between the activities in the inspiratory and expiratory phases. These alterations are attributed to functional changes occurring in the respiratory center and caused by the partial deafferentation through vagotomy. The functional changes in the respiratory musculature caused by introducing resistance to breathing depend on the excitation of the respiratory center, which in turn is determined by its afferentation.

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