AFFERENT IMPULSES IN SINGLE VAGAL FIBERS IN PULMONARY DAMAGE

(THE NATURE OF PATHOLOGICAL IMPULSES)

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Until recently, the reflex mechanisms in pulmonary damage could not be demonstrated without eliminating the action of the vagi [2,7,10,18,19]. However, it is not possible by this means to determine which of the pulmonary receptors are stimulated as a result of the damage, or what impulses are generated by them.

To solve this problem, a method for recording impulses in single pulmonary afferent fibers has been developed [8].

Two types of receptor have been distinguished; one responds to expansion of the lungs and the other to their contraction [8,16,17,21]. It is thought that the two receptor groups are not homogeneous, and that they vary in their degree of adaptation [13,16,21].

It is almost universally accepted that during natural breathing at the usual volume only the receptors responding to expansion are stimulated, and those which are sensitive to contraction are excited only when air is sucked out of the trachea, so that no impulses can be recorded from them during a normal expiration.

Electrical recording from single vagal fibers has been used to study respiratory changes caused by an altered composition of the inspired air [8]; by breathing ether or trichlorethylene vapor [8,20], by the action of veratrine, phenyldiguanidine and other substances [11,12,15,16,17]; in pneumothorax [1]; and in congestive pulmonary hyperemia [9,11,14].

In the present investigation, electrical recording from single vagal fibers has been used to study respiratory changes due to localized pulmonary damage.

METHOD

The experiments were carried out on cats under urethane anesthesia (1.5-2 g/kg); the thorax was intact, and respiration was maintained artificially. Local damage was produced by introducing 3 ml of water at 80–90° into the lung through the thoracic wall.

At the site of the damage there was a localized inflammatory infiltration which also contained erythrocytes; sometimes it was joined by a small atelectasis caused by an accumulation of exudate in the bronchials. Under the usual experimental conditions this damage caused an increase both in the frequency and in the depth of respiration which continued for several hours, and sometimes for 1–2 days. In our experiments, the influence of such a change on receptor function was eliminated by applying artificial respiration.

As we have shown previously [4,5] there is no appreciable change in the gaseous composition of the air in the lungs caused by localized damage, a fact which is of great importance in establishing the reflex nature of the induced respiratory changes.

The impulses were recorded from the afferent fibers of one of the vagi, most usually the right (the opposite vagus nerve was sectioned in the neck to reduce reflex influences on bronchial tone).

The splitting of the nerve was carried out in the usual way. Impulses were picked up on platinum electrodes separated by a distance of 3–5 mm. To prevent the nerve from drying up, the electrodes were covered with vaseline saturated with physiological saline. The impulses were recorded on a DIZA electromyograph. During the experiment the animals were in a screened compartment. Respiratory movements were recorded on a pneumograph.

RESULTS

By this means we were able to reveal several different reactions of the pulmonary receptors to the local damage.

Immediately after the pulmonary damage had been caused, the response to expansion of the lungs disappeared (Fig. 1).

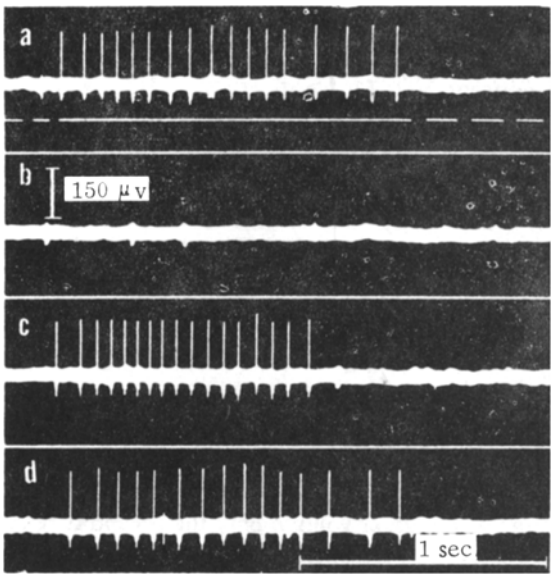

Fig. 1. Impulses from a single afferent vagal fiber recorded from the neck. A) Before pulmonary damage (impulses from stretch receptors responding at inspiration); b) after pulmonary damage (no impulses are elicited by expansion); c,d) recovery of receptor activity.

It was absent for 10–15 minutes, and then returned. Because the damaged area of the lungs was restricted, although most unlikely, there was some possibility that the impulses ceased as a result of an indirect effect on the receptor. Previously we had noticed that regional pulmonary damage, particularly in vagotomized animals, may cause a transient collapse either of the whole lung or of separate portions, and produce atelectasis beyond the damaged area. Quite possibly, therefore, the temporary stoppage of impulses from a tension receptor might result from the temporary collapse of the portion of the lung containing the receptor.

In many cases, after damage, impulses could be recorded during expiration from receptors which before damage had been excited only during inspiration (Fig. 2).

In previous investigations we have shown that beyond the damaged region, and adjacent to the atelectatic areas, there were also portions where there was a localized emphysema. Possibly impulses from a tension receptor occur at expiration by virtue of the fact that the portion of the lung in which it is situated fails to collapse, on account of a local emphysema. The maintained dilatation of these areas would then maintain the flow of impulses during expiration.

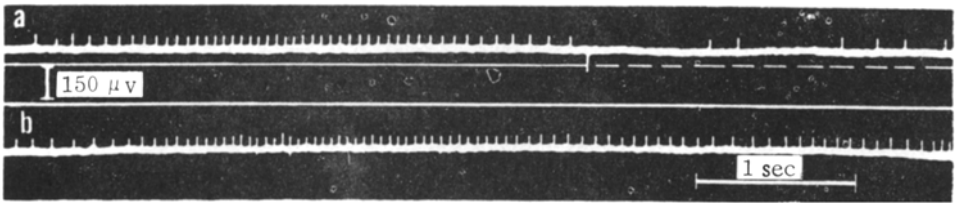

Fig. 2. Impulses from a single afferent vagal nerve fiber in the neck. A) Before pulmonary damage (impulses form a tension receptor at inspiration); b) after damage (impulses from the tension receptor are present also during inspiration).

In many experiments, after pulmonary damage, impulses of small amplitude appeared during expiration, and this effect has not previously been recorded (Fig. 3).

We have already pointed out that receptors responding to collapse are not stimulated by normal expiration, but only when air is vigorously removed from the tracheae. It is, however, known that many chemical substances such as diphenyldiguanidine, 5-hydroxytryptamine, etc., will sensitize such receptors, so that impulses may be recorded from them during a normal expiration [16]. Pulmonary damage on account of breakdown of tissue causes an increase in the amount of 5-hydroxytryptamine in the blood, and could therefore lead to sensitization of the receptors stimulated in our experiments by collapse of the lungs. On this account responses may be obtained at a volume

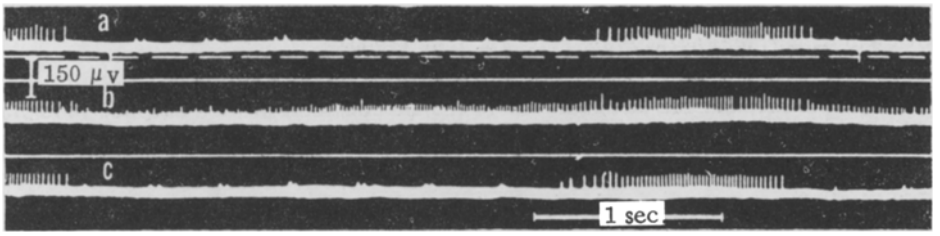

Fig. 3. Impulses from a single afferent vagal fiber in the neck. A) Before damage (impulses from a tension receptor during inspiration); b) after damage (in addition to impulses occurring during inspiration, impulses of smaller amplitude also occur during expiration); c) recovery of activity equal to that recorded before damage).

which was ineffectual before any damage had been inflicted.

There is another possible reason for impulses developing at expiration. Long ago, Adrian [8] reported that when there is infiltration of the lungs in pleuritis, receptors may respond at expiration as a result of a number of mechanical causes. This factor must also be considered in the present experiments in cases where damage caused infiltration.

Finally, in certain experiments, pulmonary damage caused a continuous low amplitude discharge, which was not associated with the respiratory cycle, to appear in fibers which were functionless before the damage. These impulses have not yet been indentified.

Our results appear to be of major importance. Until recently, the study of single-fiber activity has been made by stimulating the whole of the pulmonary receptor apparatus, by changing lung volume, by the use of inhaled drugs or chemical substances, or by congestive hyperemia. Accordingly, changes in the discharges from the receptor were always of the same kind, and consisted of either an increase or a decrease in the frequency of a respiratory volley.

In the case of localized pulmonary damage, more detailed information is available on the origin of the peripheral process transmitted to the central nervous system.

The receptors are inactive during the respiratory phase, when they would normally discharge, and begin to function when normally they would not respond.

This fact is of particular interest in explaining pathological impulses. In localized damage, particularly where the lungs are concerned, these discharges have been interpreted as arising from the damaged zone itself. Without expressing any opinion on the nature of these impulses, we must admit that the pathological condition of the periphery may be of quite a different kind, and may arise from impulses originating in receptors outside the damaged zone, whose normal functioning has been impaired by the damage.

The increase in the rate of breathing which occurs in pathological pulmonary conditions is usually attributed to an increase in the impulses from the pulmonary receptors. The results given here indicate that the pathological process disorganizes receptor activity rather than increases the rate of flow of impulses from pulmonary receptors.

SUMMARY

Recordings from single afferent vagal fibers were made after focal injury to the lungs, and revealed the following alterations in pulmonary receptor activity: 1) temporary disappearance of stretch-receptor activity, evidently due to the transient collapse of the pulmonary area containing the receptor; 2) continuation of stretch receptor activity during expiration, evidently due to the development of focal emphysema in the receptor area; 3) the development of low amplitude activity during expiration, which was not recorded during expiration of the same volume prior to injury. The effect may very likely be explained by receptor sensitization caused by the injury. It is suggested that in addition to impulses from the pulmonary focus itself, the pathological impulses may originate from receptors beyond the focus, the effect being todisorganize rather than to intensify the discharge.

LITERATURE CITED

1. M. I. Vinogradova, in book: Problems of the Regulation of Breathing in Normal and Pathological Conditions [in Russian] (Moscow, 1959) p. 58.
2. T. I. Goryunova, Mechanisms of the Disturbance of Respiration in Damage to the Lungs and Upper Respiratory Tracts. Candidate's dissertation [in Russian] (Moscow, 1949).

3. T. I. Goryunova and S. I. Frankshtein, Arkh. Pat. 12, 1, 40 (1950).
4. Z. N. Sergeeva, Byull. Éksptl. Biol. i Med., No. 8, 38 (1957).
5. Z. N. Sergeeva, Nervous Mechanisms of Regulation of the Gaseous Constitution of Blood in Regional Damage of the Lungs in an Experiment on Animals. Candidate's dissertation [in Russian] (Moscow, 1959).
6. S. I. Frankshtein, Disturbance and Recovery of Function of a Damaged Organ [in Russian] (Moscow, 1948).
7. S. I. Frankshtein, Reflexes of Pathologically Changed Organs [in Russian] (Moscow, 1951).
8. E. D. Adrian, J. Physiol. (1933) 79, p. 332.
9. E. Bülbring and D. Whitteridge, J. Physiol. (1944) 103, p. 477.
10. C. J. Clemedson, Acta Physiol. Scand. (1949) 18, Suppl. 61.
11. L. C. Le Roy, Am. J. Physiol. (1959) 196, p. 49.
12. G. S. Dawes and J. H. Comroe, Physiol. Rev. (1954) 34, p. 167.
13. G. C. Knowlton and M. G. Larrabee, Am. J. Physiol. (1946) 147, p. 100.
14. R. Marshall and J. G. Widdicombe, Quart. J. Exp. Physiol. (1958) 43, p. 320.
15. A. S. Paintal, J. Physiol. (1953) 121, p. 182.
16. A. S. Paintal, Quart. J. Exp. Physiol. (1955) 40, p. 89.
17. A. S. Paintal, Quart. J. Exp. Physiol. (1957) 42, p.56.
18. W. T. Porter, Quart. J. Exp. Physiol. (1957) 42, p. 175.
19. W. T. Porter and L. H. Newburgh, Am. J. Physiol. (1917) 43, p. 455.
20. D. Whitteridge and E. Bülbring, J. Pharmacol. Exp. Ther. (1944) 81, p. 340.
21. J. G. Widdicombe, J. Physiol. (1954) 123, p. 71.

All abbreviations of periodicals in the above bibliography are letter-by-letter transliterations of the abbreviations as given in the original Russian journal. *Some or all of this periodical literature may well be available in English translation.* A complete list of the cover-to-cover English translations appears at the back of this issue.
