THE MECHANISM OF EXCITATION OF THE RESPIRATORY CENTER DURING PATHOLOGICAL CHANGES IN THE LUNGS

Z. N. Sergeeva, T. I. Goryunova, and S. I. Frankshtein

Laboratory of Comparative Pathology of the Nervous System (Head, Professor S. I. Frankshtein),
Institute of Normal and Pathological Physiology (Director, Active Member AMN SSSR V. V. Parin)
(Presented by Active Member AMN SSSR V. V. Parin)
Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 54, No. 11,
pp. 30-33, November, 1962
Original article submitted March 10, 1962

In our previous investigation we found that pathological changes in the lung tissues do not give rise to diffuse excitation of the receptors of the lungs, but cause dissociation of their activity. The activity of some stretch receptors is increased, while that of others is suppressed, and a third group become active not only during inspiration but also during expiration. Collapse receptors, normally active only when air is sucked from the lungs, after injury to the lungs may become active during natural respiration [3].

It might be suggested that these dissociated changes in the activity of the lung receptors must produce heterogeneous changes in the activity of the neurons of the respiratory center. This formed the subject of the present investigation.

We judged the activity of the neurons of the respiratory center by the activity of single efferent fibers of the recurrent nerve. Several workers have shown [1, 2, 4, 11] that the activity of these fibers corresponds to the activity of the neurons of the respiratory center, as demonstrated by a microelectrode technique [5, 6, 7, 9, 12, 13, 14].

EXPERIMENTAL METHOD

Experiments were carried out on cats breathing naturally and anesthetized by intravenous injection of 50 mg of a 6% chloralose solution and 250 mg of a 6% urethane solution per 1 kg body weight. A localized injury to the lung tissue was caused by injecting 3 ml of water at a temperature of 80-90° into the lung through the chest wall.

The efferent impulses were recorded in single efferent fibers of the recurrent nerve on the injured side. Fine filaments of the recurrent nerve, separated into single functioning fibers, were placed on platinum electrodes which were buried in a hollow filled with vaseline oil. The action potentials were recorded by means of a three-channel amplifier and a cathode-ray oscillograph (manufactured by Diza).

EXPERIMENTAL RESULTS

The activity of the individual fibers of the same nerve differs in respect to the duration of the volleys, the time of the beginning and end of the volley, and the number of spikes present in the volley. In most fibers of the recurrent nerve, when the animal was breathing naturally, the recorded impulsation was synchronous with inspiration. Fibers giving discharges in time with expiration were encountered very rarely. This confirmed that in normal conditions expiration took place mainly passively, and that the expiratory neurons of the respiratory center were associated with the intracentral inhibition of inspiration.

Besides the fibers in which the impulses were grouped in a respiratory rhythm, many other fibers were found with a continuous stream of tonic impulses, in some cases more frequent during inspiration. Besides these groups of fibers described earlier [1, 2, 4, 11], we found inspiratory—expiratory fibers in the recurrent nerve. At inspiration a high-frequency discharge was recorded in these fibers, followed immediately by a discharge at expiration with impulses of lower frequency, and then (in contrast to the tonic fibers) by a pause.

Similar volleys of impulses have also been found in the respiratory center [5, 9]. They are seen especially frequently in the cells of the superior and middle parts of the pons [8]. These neurons, like the tonic neurons, evidently regulate the smooth transition from inspiration to expiration.

Localized injury to the lungs caused changes in the impulses in all the investigated efferent fibers. Most inspiratory fibers gave an increased frequency of volleys after injury to the lungs, corresponding to the increased respiration rate. Each volley contained many more impulses. The duration of the volleys changed in different ways: in some fibers they became longer, in others, shorter (Fig. 1). In individual inspiratory fibers the activity ceased completely immediately after the lung injury (Fig. 2). Conversely, in some fibers in which no activity was recorded before injury to the lungs, inspiratory activity developed (Fig. 3, middle neurogram).

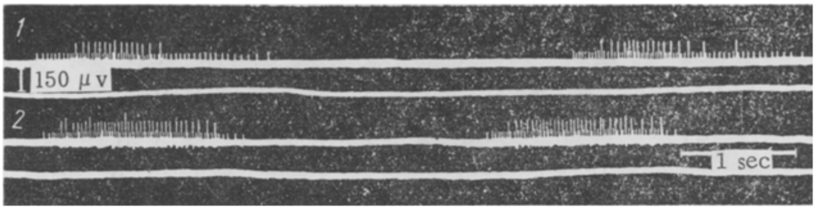

Fig. 1. Neurogram of single efferent fibers of the recurrent nerve. 1) Before injury to the lungs: volleys of impulses at inspiration, two fibers are active; 2) after injury to the lungs: increased frequency of volleys and of impulses in the volley, discharge in one fiber (of greater amplitude) was lengthened, while that in the other (smaller amplitude) was shortened (lower curve—tracing of respiration).

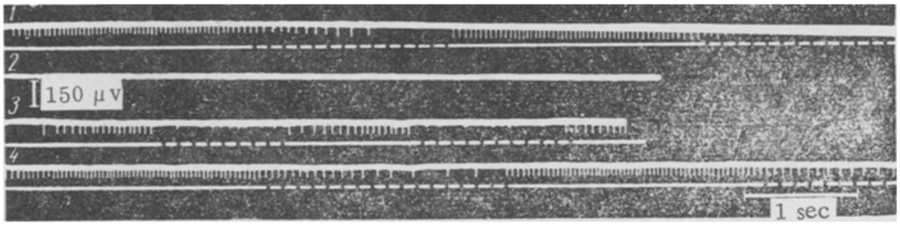

Fig. 2. Neurogram of a single efferent fiber of the recurrent nerve. 1) Before injury to the lungs: impulsation at inspiration, continuing for a short time during expiration; 2) immediately after injury to the lungs: cessation of impulsation; 3) 1 min after injury: appearance of impulses at inspiration, volleys of impulses considerably shortened; 4) after 30 min: restoration of original background.

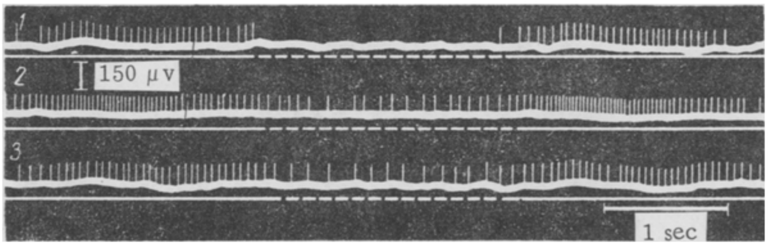

Fig. 3. Neurogram of an efferent fiber of the recurrent nerve. 1) Before injury to the lungs: volley of impulses at inspiration, one fiber functioning; 2) after injury to the lungs: frequency of impulsation increased at inspiration, and impulsation appears during expiration; another fiber has begun to function at inspiration; 3) 35 min after injury: frequency of impulses at inspiration restored, impulsation during expiration continues (———inspiration; ------expiration).

The changes in the type of activity of individual fibers as a result of the injury were particularly interesting. In some fibers, for instance, in which a phased, inspiratory activity was recorded before injury, after the injury a continuous, tonic activity developed (Fig. 3). Conversely, in individual fibers with continuous activity before injury to the lungs, after injury a phased, inspiratory type of activity developed (Fig. 4). Equally definite inspiratory volleys also developed in some fibers in which an inspiratory–expiratory type of activity was recorded before injury to the lungs (Fig. 2).

The changes described above in the efferent activity were transient. As a rule the normal impulsation in the fibers was restored between 3 and 30 min after injury to the lungs, or it approximated to its initial level. The normal respiratory movements were also restored at this time.

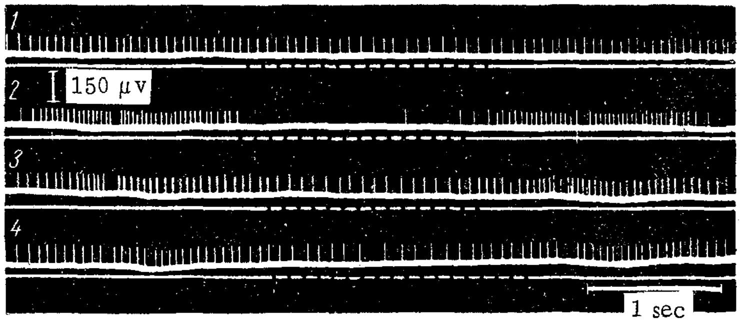

Fig. 4. Neurogram of a single efferent fiber of the recurrent nerve. 1) Before injury to the lungs: impulsation at inspiration and expiration; 2) immediately after injury: frequency of impulses increased at inspiration, disappearance of impulses at expiration; 3) 5 min after injury: increased frequency of impulses as inspiration continues, impulsation at expiration restored to normal; 4) 30 min after injury; restoration of initial level (——— inspiration; ------ expiration).

It is customary to regard the strengthening of the respiratory movements during injury to the lungs as a reflection of the increased activity of the respiratory center. The present investigations show that this increased activity is the result of the complex integrative activity of the respiratory center. The activity in some neurons increases, in others it is inhibited, and in a third group activity arises anew. In some neurons the type of activity is modified—tonic activity changes into phased, and vice versa.

The discovery of neurons capable of modifying their type of activity in this manner demonstrates that the type of activity is not always connected obligatorily with a definite neuron. The same conclusion has been reached comparatively recently in relation to the spinal neurons, which were originally divided particularly sharply into tonic and phased [10].

SUMMARY

Intensification of respiratory movements in affections of the lungs should be regarded as the result of augmented activity of the respiratory center. The present investigations show that this activity intensification is due to a complex integrative activity of the respiratory center. In some neurons the activity is intensified, in others it is inhibited; the third neurons are mobilized anew. In a number of neurons the type of activity changes—the tonic type alters into a phased one and vice versa. Detection of neurons capable of changing the constant type of activity to phased, and conversely, presents special interest. This shows that the type of the neuron activity is not always obligatorily connected with a definite type of neuron, and that under the influence of afferent effects the tonic neurons may function as phased, and conversely.

LITERATURE CITED

1. M. I. Vinogradova, In: Annual Report of the Institute of Experimental Medicine of the AMN SSSR for 1955 [in Russian], p. 113 (Leningrad, 1956).
2. M. I. Vinogradova, In: Annual Report of the Institute of Experimental Medicine of the AMN SSSR for 1956 [in Russian], p. 160 (Vilnius, 1957).
3. Z. N. Sergeeva, T. I. Goryunova, and S. I. Frankshtein, Byull. éksper. biol. 6, 29 (1961).
4. V. I. Filistovich, In the book: Annual Report of the Institute of Experimental Medicine of the AMN SSSR for 1957 [in Russian], p. 100 (Leningrad, 1958).

5. R. Baumgarten, Pflüg. Arch. ges. Physiol. 1956, Bd. 262, S. 573.
6. R. Baumgarten, K. Baltasar, and H. P. Koepchen, Pflüg. Arch. ges. Physiol, 1960, Bd. 270, S. 504.
7. B. D. Burns and G. C. Salmoiraghi, J. Neurophysiol.,1960, v. 23, p. 27.
8. M. I. Cohen and S. C. Wang, J. Neurophysiol.,1959, v. 22, p. 33.
9. M. N. J. Dirken and S. Woldring, J. Neurophysiol.,1951, v. 14, p. 211.
10. R. Granit, The Electrophysiological Investigation of Reception [Russian translation] (Moscow, 1957).
11. J. H. Green and E. Neil, J. Physiol. (London, 1955), v. 129, p. 134.
12. J. Nelson, J. Neurophysiol., 1959, v. 22, p. 590.
13. G. C. Salmoiraghi and B. D. Burns, J. Neurophysiol, 1960, v. 23, p. 2.
14. G. C. Salmoiraghi and B. D. Burns, J. Neurophysiol., 1960, v. 23, p. 14.

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.
