A STUDY OF SPINAL CORD INHIBITION PROCESSES AND REFLEX EXCITABILITY UNDER CONDITIONS OF ANESTHESIA

A. D. Reva

Department of Animal and Human Physiology and Biochemistry (Head—Prof. P. E. Motsnyi) of the Dnepropetrovsk State University Commemorating the Tricentennial of the Reunion of the Ukraine with Russia

(Presented by V. V. Zakusov, Active Member of the AMN SSSR)

Translated from Byulleten' Eksperimental'noi Biologii i Meditsiny, Vol. 49, No. 6, pp. 75-78, June, 1960

Original article submitted April 21, 1959

The works of I. P. Pavlov, N. E. Vvedenskii and A. A. Ukhtomskii contain valuable ideas and facts which make it possible to approach an explanation of the physiological nature of anesthesia. The physiological aspect of anesthesia has been most profoundly investigated by N. E. Vvedenskii [1], P. V. Makarov [5] and V. V. Zakusov [4]. On the basis of experimental data obtained on nerve-muscle preparations, N. E. Vvedenskii [1] described anesthesia as a condition of parabiosis, the essential feature of which is "that it affects all the contiguous parts simultaneously and is a more or less stable and constant state."

The purpose of this work was to study the changes which occur in the cerebrospinal reciprocal inhibition and reflex excitability processes of warm-blooded animals under conditions of anesthesia.

METHODS

The experiments were performed on spinal cats and on some decerebrate cats. We used ether or chloroform to produce inhalation anesthesia and Pentothal, Nembutal and Hexenal to produce intravenous anesthesia. We investigated the changes which occurred during anesthesia in the reciprocal inhibition and reflex excitability of the flexor and extensor muscles of the posterior extremities. The anterior tibialis muscle was used to determine change in the excitability of the flexor reflex and in the contralateral inhibition. The reflex contractions of this muscle were induced by single inductive stimulations of the popliteal nerve of the same paw, while the inhibition was induced by stimulating the same nerve in the opposite paw. We used the gastrocnemius muscle to study the excitability of the extensor reflex and the ipsilateral inhibition. The reflex contractions of this muscle were induced by single inductive stimulations of the posterior root of the 7th lumbar segment, and the inhibition was induced by similar stimulations of the deep peroneal nerve of the same extremity.

Reflex excitability was evaluated according to height of the recording produced on a kymograph by the contractions of the experimental muscle in response to a single inductive stimulation of its sensory nerve. The degree of inhibition was estimated from the height of the recording produced by the contractions of the same muscle on a background of an inhibitory discharge given 40-60 milliseconds before the reflex discharge in the case of contralateral inhibition and 8-10 milliseconds before in the case of ipsilateral inhibition. The inhibition was most apparent at the specified intervals. The proper intervals between the stimuli were selected with the aid of a Helmholtz's pendulum.

In some experiments, the contraction of the tibialis muscle was induced by stimulation of the descending lateral cerebrospinal tract. For this purpose, the spinal cord was exposed at the level of the 5th lumbar segment, and special bipolar electrodes were inserted from the dorsal surface of the spinal cord in such a way that their non-insulated ends were located in the specified tract. The electrodes were silver wires about 30 μ in diameter, sealed in a glass capillary. In this case, the inhibitory stimulus was produced by stimulating the posterior roots of the opposite side of the 7th lumbar segment.

RESULTS

The investigations showed that the state of anesthesia induced by the inhalation of ether or chloroform, or by the intravenous injection of the barbiturates, intensified the contralateral inhibition and reduced the reflex excitability of the flexor muscles of the posterior extremities. Fig. 1 gives a myogram of an experiment which illustrates the changes which occurred with the intravenous injection of 2 ml of a 2.5 % Pentothal Sodium solution into a spinal cat weighing 1.7 kg. The myogram shows that the inhibition process became stronger in the initial stage of the anesthesia, before any change occurred in the height of the reflex contractions of the muscle (see first and second pairs of recordings after arrow); subsequently, as the anesthesia deepened and reflex excitability decreased, the intensification of the inhibition processes continued. Therefore, the intensification observed of contralateral inhibition cannot be accounted for by the weakening of the reflexes.

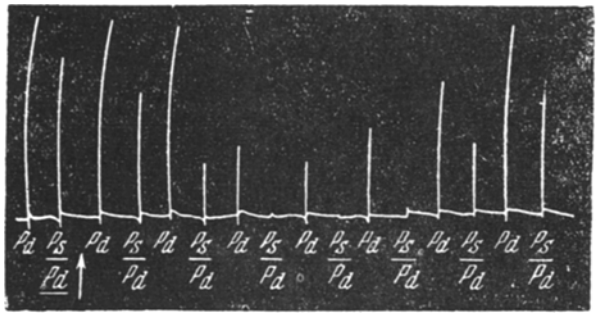

Fig. 1. Change in the contralateral inhibition and excitability of the flexor reflex during anesthesia. Pd) height of contractions of right anterior tibialis muscle in response to a single stimulation of right deep popliteal nerve; $\frac{Ps}{Pd}$) the same on a background of a single discharge from the left popliteal nerve (40 milliseconds previously). The arrow represents the intravenous injection of 2 ml of 2.5% Pentothal Sodium into a spinal cat weighing 1.7 kg. Duration of investigation - 45 minutes.

Intensification of the inhibition processes during anesthesia was also characteristic of the experiments in which the contraction of the tibialis muscle was induced by single inductive discharges of the descending lateral cerebrospinal tract and the inhibition, by single stimulations of the opposite posterior root. The change which occurred in the ipsilateral inhibition and reflex excitability of the extensor muscle is shown in Fig. 2. This type of reflex inhibition, unlike the contralateral inhibition, was observed to become briefly weaker in the initial phase of the anesthesia before it became stronger. In these experiments, as in the preceding ones, the intensification of the inhibition occurred before the decrease in the reflex excitability (see Fig. 2, second pair of recordings after arrow), so that it could not be due to the weakening effect of anesthesia on the reflexes.

The changes which developed in the reciprocal inhibition and reflex excitability during anesthesia were reversible as the animal emerged from the condition of anesthesia.

In contralateral and ipsilateral inhibition, the impulses are known to pass along the intercalary neurons. It is therefore natural to suppose that they become weaker during anesthesia when the transmission of stimulation in the polysynaptic paths is blocked. However, although the inhibitory impulses do become somewhat weaker in the polysynaptic paths under conditions of anesthesia, the conditions created by the anesthesia in the motor cells of the spinal cord are extremely favorable for the manifestation of stronger than normal inhibition of reflex discharges. Our previous investigations [7, 9] showed the decrease in the functional mobility (prolongation of chronaxy) and increase in the accommodation rate (decrease in the constant λ) of the spinal cord motor cells during anesthesia to be such conditions. In [6], P. E. Motsnyi established that functional changes of the same nature also develop in the spinal cord motor cells under conditions of inhibition.

Therefore, the functional changes which develop in the motor centers of the spinal cord under conditions of inhibition and anesthesia are the same in nature. In a previous work [9], we showed that these changes in the motor centers of the spinal cord could be cumulative with inhibition of a background of anesthesia. This evidently is the reason for the intensification of the inhibition processes under conditions of anesthesia.

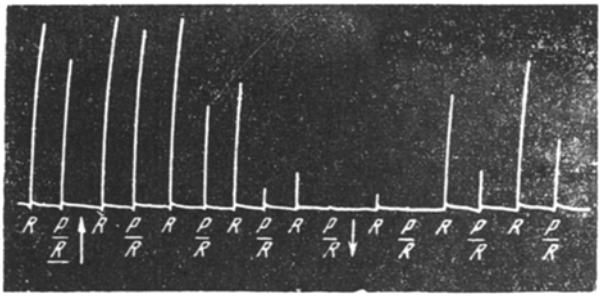

Fig. 2. Change in the ipsilateral inhibition and excitability of the extensor reflex during anesthesia. R) height of contractions of gastrocnemius muscle in response to a single stimulation of the posterior root (L_7); $\frac{P}{R}$) the same on a background of a single impulse from the right deep peroneal nerve (10 milliseconds previously). (Arrows indicate beginning and end of ether inhalation. Inhalation duration - 4 minutes.)

As to the changes in the reflex excitability of the spinal cord during anesthesia, the experiments showed that the changes are different for the flexor and extensor reflexes of the posterior extremities. The decrease in the reflex excitability of the extensor muscles was preceded by a brief phase of facilitation of the reflex. In the case of the flexor muscles, the phase of facilitation of the reflex was absent; the decrease in reflex excitability paralleled the increase of the anesthesia. These differences are evidently due to the higher lability indicated by N. E. Vvedenskii and A. A. Ukhtomskii [2] of the extensor muscle centers as compared with flexor centers.

The "rule of optimal physiological lability for excitability" [3] can be applied to explain how reflex excitability depends on change in lability. According to this rule, excitability is highest with a certain mean value of physiological lability. When the lability becomes either higher or lower than this optimal level, the excitability of nervous tissue decreases and the conduction of stimulation is hampered. Fig. 3 shows N. V. Golikov's rule [3] as applied to explain the dependency of change in the excitability of the nerve centers of flexor and extensor muscles on the change in their lability during anesthesia. Anesthesia lowers the lability of the spinal cord motor centers [7, 9]; in the centers of the flexor reflexes (located in left part of diagram in Fig. 3), which are formations less labile than the extensor centers, the excitability decreases parallel to the lability, and there is no phase of reflex facilitation. In the centers

of the extensor reflexes (located in the right part of the diagram in Fig. 3), the more labile formations, the decrease in lability which occurs under the influence of anesthesia passes through the optimum point at which excitability is highest. Therefore, an initial facilitation of the reflex is observed in the early stages of the anesthesia. As the anesthesia deepens and the lability of the centers falls below the optimal value, the excitability also decreases.

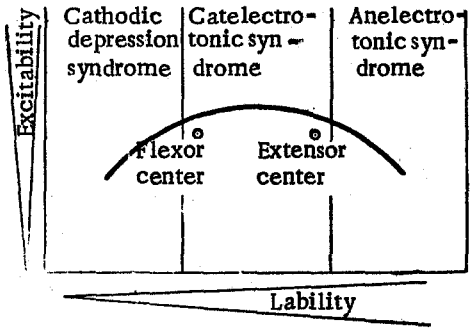

Fig. 3. "Rule of optimal physiological lability for excitability" (N. V. Golikov [3]), used to illustrate how change in the excitability of the motor centers of flexor and extensor muscles depends on change in their lability during anesthesia.

All the experimental anesthetics caused these reversible changes of decreased reflex excitability and increased reciprocal inhibition; the duration and intensity of the changes, however, depended on both the dosage and the physiological activity of the anesthetics. The intensification of reciprocal inhibition during anesthesia, together with the decreased functional mobility and increased accommodation rate of the spinal cord motor cells (respectively, prolonged chronaxy and decreased constant λ) observed earlier by us under conditions of anesthesia [7-9] and by P. E. Motsnyi [6] with reciprocal inhibition, testifies that the functional changes which develop under conditions of inhibition and anesthesia are similar in nature and fits in well with N. E. Vvedenskii's doctrine of parabiosis.

SUMMARY

Myography was used on spinal cats to investigate the changes in the reciprocal inhibition and reflex excitability of extensor and felxor muscles during anesthesia. The reciprocal inhibition was intensified during anesthesia induced by ether or chloroform inhalation as well as during that induced by the intravenous injection of sodium pentothal or nembutal.

LITERATURE CITED

1. N. E. Vvedenskii, Excitation, Inhibition and Anesthesia [in Russian] (Leningrad, 1901).
2. N. E. Vvedenskii and A. A. Ukhtomskii, in: N. E. Vvedenskii, Collected Works [in Russian] (Leningrad, 1938) Vol. 4 (2nd book), p. 90.
3. N. V. Golikov, Physiological Lability and Its Changes in the Basic Nervous Processes [in Russian] (Leningrad, 1950).
4. V. V. Zakusov, The Pharmacology of the Nervous System [in Russian] (Leningrad, 1953).
5. P. V. Makarov, Arkh. Anat., Gistol. i Embriol. 19, 1-2, 5-6 (1938).
6. P. E. Motsnyi, Data on the Characteristics of Central Inhibition [in Russian] Doctorate Dissertation (Dnepropetrovsk, 1955).
7. A. D. Reva, Fiziol. Zhur. AN Ukr. SSR 1, 2, 21 (1955).
8. A. D. Reva, in: Abstracts of the Proceedings at the 8th All-Union Convention of Physiologists, Biochemists and Pharmacologists [in Russian] (Moscow, 1955) p. 502.
9. A. D. Reva, Byull. Éksp. Biol. i Med. 43, 3, 12 (1957).*

*See C. B. translation.