

DISTURBANCE OF FUNCTIONS OF SPINAL CENTERS IN COMATOSE PATIENTS

M. Kh. Starobinets, T. S. Sbitneva,
A. Ya. Vernik, and Z. A. Demina

An investigation of the H-reflex of the soleus muscle and of the F-wave of the abductor hallucis muscle in patients in a varied depth of coma revealed successive disappearance of the responses of the α -motoneurons, initially to stretching of the corresponding muscle, later to electrical stimulation of the Ia afferents, and finally, to antidromic stimulation.

Inhibition of the functions of the nonspecific structures of the brain stem, the basis of development of deep coma, leads to the gradual disappearance of tendon reflexes. The mechanism of this areflexia has not been adequately explained.

The object of the present investigation was to study reflex and antidromic excitability of the α -motoneurons of the sacral segments of the spinal cord in patients in stage II-IV of coma, with deprivation of supraspinal impulses [1].

EXPERIMENTAL METHOD

The investigation was carried out on 20 healthy subjects and 12 patients in coma of varied depth and etiology: vascular coma (7 patients), traumatic (3 patients), and toxic (2 patients). Reflex excitability of the α -motoneurons was determined by recording the H-reflex [14, 16]. For this purpose the tibial nerve was stimulated in the popliteal fossa with square pulses of increasing strength with a frequency of 0.1 Hz and duration of 0.5 msec. Potentials of the central and motor responses were recorded by percutaneous electrodes from the soleus muscle. The ratio between the maximal amplitudes of these potentials (H_{\max}/M_{\max}), expressed as a percentage, was used as a measure for quantitative assessment of the monosynaptic excitability of the spinal center [4, 8]. Antidromic excitability of the α -motoneurons was judged from the character of the F-wave [12, 17, 18] — the central response of the abductor hallucis muscle, recorded by coaxial needle electrodes. The action potentials of the muscles were recorded on a Meditor electromyograph.

EXPERIMENTAL RESULTS

Depending on the severity of the coma, the patients were divided into three groups. Group 1 included three patients in stage II of coma. Clinical picture was characterized by unconsciousness, depression of the cutaneous reflexes, inhibition of tendon reflexes in the upper limbs and their absence in the lower limbs, although pyramidal signs were present in the feet. Despite the absence of ankle jerks, all three patients had H-potentials of high amplitude in the soleus muscle and their H_{\max}/M_{\max} ratio varied from 47 to 67%. The mean value for this ratio in healthy persons was 68%, with variations ranging from 38 to 94%. Just as in the control group, the threshold of the H-reflex in every case was below the threshold of the M-potential. The central response of the abductor hallucis muscle in these patients, by contrast with the healthy sub-

Department of Physiology of Man and Animals and Department of Nervous Diseases, O. V. Kuusinen Petrozavodsk University. Department of Anesthesia and Resuscitation, City Hospital, Petrozavodsk. (Presented by Academician V. N. Chernigovskii.) Translated from *Byulleten' Éksperimental'noi Biologii i Meditsiny*, Vol. 74, No. 12, pp. 29-32, December, 1972. Original article submitted February 29, 1972.

© 1973 Consultants Bureau, a division of Plenum Publishing Corporation, 227 West 17th Street, New York, N. Y. 10011. All rights reserved. This article cannot be reproduced for any purpose whatsoever without permission of the publisher. A copy of this article is available from the publisher for \$15.00.

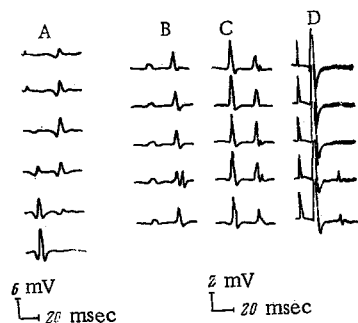


Fig. 1

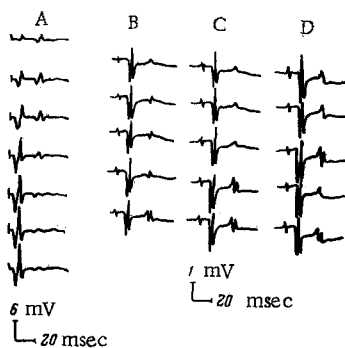


Fig. 2

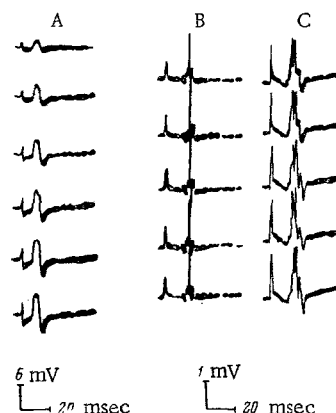


Fig. 3

Fig. 1. Central and motor responses of leg and foot muscles of a patient in stage II of coma. Patient K-n, aged 27 years. Diagnosis: concussion, subarachnoid hemorrhage. A) Dynamics of H-reflex and M-response of soleus muscle with increasing strength of stimulation; B) motor and central responses of abductor hallucis muscle to threshold; C) submaximal; and D) supra-maximal stimulation of motor axons.

Fig. 2. H-reflex and F-wave of leg and foot muscles in a patient with stage III of coma. Patient A-va, aged 32 years. Diagnosis: epilepsy with dementia. State after clinical death as result of obstruction of the respiratory passages by a foreign body. Legend as in Fig. 1.

Fig. 3. Motor responses of soleus and abductor hallucis muscles in stage IV of coma. Patient I-v, aged 43 years. Diagnosis: symptomatic hypertension (anomaly of the kidneys). Multiple subarachnoid and intracerebral hemorrhages. A) Dynamics of M-response of soleus muscle during increase in stimulus intensity (H-reflex absent); B) M-response of abductor hallucis muscle to suprathreshold; C) to supramaximal stimulation (F-wave absent).

jects, was evoked regularly and was constant in amplitude and shape, being inhibited by supramaximal stimulation (Fig. 1), showing that its reflex component was predominant.

Group 2 included five patients in stage III of coma. In these cases complete areflexia was observed, accompanied by disturbance of cardiac activity and severe disorders of respiration, as a result of which the lungs were artificially ventilated with the RO-2 apparatus. The electrophysiological characteristics in this case were a low amplitude of the H-reflex and a decrease in the H_{max}/M_{max} ratio (10-37%). In four cases the thresholds of the H- and M-potentials were equalized. Although the F-wave did not contain a reflex component, it was regular. Sometimes in response to a single stimulus, a double antidromic discharge of the α -motoneurons was recorded (Fig. 2).

Group 3 included four patients whose life had been artificially supported for a day or for several hours by artificial respiration and by drugs acting on the cardiovascular system (the terminal stage of coma). Characteristic results of the electromyographic investigation of the patients of this group were absence of the H-reflex and F-wave of the leg and foot muscles and preservation of the motor responses (Fig. 3).

With deepening of coma, the tendon reflexes thus disappeared consecutively: first the knee and ankle jerks, then the H-reflex of the soleus muscle and, finally, the F-wave of the abductor hallucis. The same pattern was observed not only in patients with coma of different severity, but also in the same patient during worsening of his state.

Consequently, despite disappearance of the ankle jerks, in patients in stage II of coma the reflex excitability of the sacral α -motoneurons remains intact. This is shown by the presence of an H-potential and also by predominance of the reflex component in the central response of the foot muscle. The same effect has also been observed in healthy subjects under 20-22 years of age [5].

According to the literature, comparison of the ankle jerk and H-reflex of the soleus muscle can provide indirect evidence of the activity of the γ -system [9, 11, 15, 20]. Although activation of Ia-afferents during electrical stimulation of a nerve and mechanical stimulation of a tendon differs in its degree of synchronization, and there are technical difficulties in the way of comparing changes in the tendon reflex and H-potential [2], it can nevertheless be supposed that the loss of the ankle jerk at a near-normal value of the H_{\max}/M_{\max} ratio was due to selective inhibition of γ -motoneuron function.

During the development of coma, in the initial period of a decrease in supraspinal influences the excitability of the γ -neurons and, in consequence, of the intrafusal spindles of the skeletal muscle probably falls. Only when the coma reaches stage III-IV, does the reflex excitability, followed by the antidromic excitability, of the respiratory cells of the anterior horn diminish and disappear, i.e., atony of the entire spinal center develops [3].

Both a decrease in the influences arriving via the γ -loop and a gradual development of spinal ischemia are evidently significant factors in the mechanism of the relatively late inhibition of γ -motoneuron function. The resulting asphyxia, through disturbances in the presynaptic system, leads to the suppression of the reflex response whereas the antidromic response still continues [10]. The mismatching of the responses of the spinal center to stretching of the muscle spindles and to electrical stimulation of Ia-afferents is also observed in other lesions of the CNS [6, 7, 11, 19].

The possibility cannot be ruled out that these results may be of practical importance in assessing the severity of coma, evaluating the efficacy of resuscitation measures, and in assessing the patient's prognosis..

LITERATURE CITED

1. N. K. Bogolepov, Comatose States [in Russian], Moscow (1962).
2. V. S. Gurfinkel', Ya. M. Kots, and M. L. Shik, The Regulation of Human Posture [in Russian], Moscow (1965).
3. G. N. Sorokhtin, Responses of Excitable Systems to Deprivation of Excitation [in Russian], Moscow (1968).
4. M. Kh. Starobinets, Byull. Éksperim. Biol. i Med., No. 11, 5 (1971).
5. M. Kh. Starobinets, in: Problems in Deprivation of Excitation. Proceedings of an All-Union Symposium [in Russian], Petrozavodsk (1971), p. 198.
6. M. Kh. Starobinets, A. Ya. Vernik, and A. D. Pshedetskaya, Abstracts of Proceedings of an All-Union Symposium on Demyelinating Diseases of the Nervous System in Experimental and Clinical Practice [in Russian], Minsk (1970), p. 128.
7. M. Kh. Starobinets, A. Ya. Vernik, and A. D. Pshedetskaya, Zh. Nevropat. i Psikhiat., No. 8, 1184 (1971).
8. R. W. Angel and W. W. Hoffmann, Arch. Neurol (Chicago), 8, 591 (1963).
9. C. H. M. Brunia and W. Knijft, Electroenceph. Clin. Neurophysiol., 28, 427 (1970).
10. H. Collewyn and A. Van Harreveld, J. Physiol. (London), 185, 1 (1966).
11. E. Diamantopoulos and P. Z. Olsen, J. Neurosurg. Psychiat., 30, 427 (1967).
12. L. Fra and F. Brignolio, J. Neurol. Sci., 7, 251 (1968).
13. B. Fujimori, M. Kato, T. Yamauchi, et al., Electroenceph. Clin. Neurophysiol., 27, 717 (1969).
14. P. Hoffmann, Untersuchungen über die Eigenreflexe (Sehenreflexe) Menschlichen Muskeln, Berlin, (1922).
15. W. M. Landau and M. H. Clare, Arch. Neurol. (Chicago), 10, 128 (1964).
16. J. W. Magladery, Pflug. Arch. Ges. Physiol., 261, 302 (1955).
17. R. F. Mayer and R. G. Feldman, Neurology (Minneapolis), 17, 147 (1967).
18. I. G. McLeod and S. H. Wray, J. Neurol. Neurosurg. Psychiat., 29, 196 (1966).
19. H. E. Reichenmiller and E. A. Zysno, Electroenceph. Clin. Neurophysiol., 27, 623 (1969).
20. P. Zapata, Acta Physiol. Latino-Amer., 16, 266 (1966).