EEG and Clinical Neurophysiology

Proceedings of the 2nd European Congress of EEG and Clinical Neurophysiology, Salzburg, Austria, September 16-19, 1979

Editors:
H. Lechner and A. Aranibar

1980

EXCERPTA MEDICA, Amsterdam-Oxford-Princeton

International Congress Series No. 526
ISBN Excerpta Medica 90 219 0457 8
ISBN Elsevier North-Holland 0 444 90172 8

Publisher:
Excerpta Medica
305 Keizersgracht
1000 BC Amsterdam
P.O. Box 1126

Sole Distributors for the USA and Canada:
Elsevier North-Holland Inc.
52 Vanderbilt Avenue
New York, N.Y. 10017

Printed in The Netherlands by Casparie-Amsterdam

2nd European Congress of EEG and Clinical Neurophysiology

Organized by:

The Austrian Society of EEG and Clinical Neurophysiology

Presidents:	Prof. Dr. H. Lechner
	Prof. Dr. K. Pateisky
	Prof. Dr. E. Deisenhammer
Secretaries:	Prof. Dr. E. Scherzer
	OA. Dr. S. Enge
Treasurer:	Dr. H. Feldner-Bustin

Organizing Committee of the 1st European Congress of EEG and Clinical Neurophysiology

President:	Prof. Dr. E. Lugaresi
Secretary:	Prof. Dr. R. Muntani

ORGANIZING COMMITTEE

Board:
Prof. Dr. H. Lechner, Graz
Prof. Dr. K. Pateisky, Vienna
Prof. Dr. E. Scherzer, Vienna

Secretarial Committee:
Dr. A. Aranibar, Graz
Mrs. Ch. Sulzer, Graz

Contents

I. Position of EEG in diagnosis and treatment of epilepsy with consideration of findings of computerized axial tomography

II. Value of EEG in the classification, treatment and follow-up of epilepsies

III. EEG in the study of cerebrovascular disorders

IV. EEG in children and developmental disorders

VI. Evoked responses

VII. Special neurophysiological studies

COMPONENTS OF EVOKED HYPERPOLARIZATION IN THE SPINAL MOTONEURON OF THE CAT

B. Sutor and J. Vieth

Department of Experimental Neuropsychiatry, University Neurology Clinic, Erlangen, F.R.G.

By means of intracellular recordings from pyramidal cells of the cat's sensorimotor cortex, we have described two kinds of evoked hyperpolarization (1,2). Depending on the stimulus intensity, a short lasting hyperpolarization, up to 100 msec, and a long lasting one, longer than 100 msec, can be evoked. By artificial alteration of the membrane potential and combination of different stimulus conditions, these two kinds of evoked hyperpolarization have been shown to depend on different mechanisms. The question arose, if the spinal motoneurons of the cat behave similarly to cortical pyramidal cells.

MATERIAL AND METHODS

The experiments were carried out under light pentobarbital anaesthesia, immobilization with gallamin and artificial respiration. After a laminectomy from L2 to S1, the spinal cord was covered with paraffin oil held constantly at 37°C. Then the dura mater was opened. Stimulation to evoke a response in motoneurons was done at the transsected ventral and dorsal roots of the lumbar segments L6 and L7. All recordings were made in the ventral horn of the lumbar segments L6 or L7.

RESULTS

Figure 1 shows an intracellular recording of a motoneuron in lumbar segment 7 during the stimulation of the dorsal root L6. With a stimulus intensity of 0.4 mA no response could be seen. A stimulus intensity of 0.5 mA, however, evoked an EPSP followed by a 75 msec lasting hyperpolarization. Further increase of the stimulus intensity also increased the duration of the hyperpolarization. With 0.9 mA a threshold-like prolongation of the hyperpolarization, up to 148 msec, appeared. Further increase of the stimulus intensity did not have any effect on the duration. Thus, also in spinal motoneurons we were able to distinguish two kinds of evoked hyperpolarization: Firstly, a short lasting, stimulus dependent hyperpolarization of a duration up to 100 msec and a threshold between 0.4 and 0.5 mA. Secondly, a long lasting evoked hyperpolarization with a duration of more than 100 msec which was not stimulus-controlled and with a threshold between 0.8 and 0.9 mA.

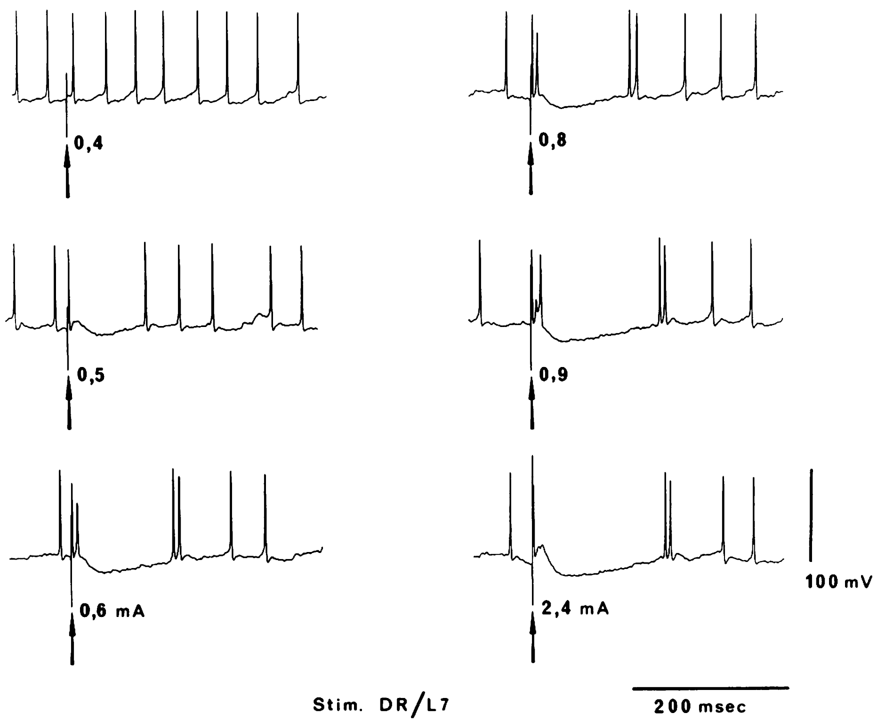

Figure 1. Intracellular recordings of a motoneuron of spinal cord segment L7 of cat. Short and long lasting hyperpolarization as response to single electric stimulus of dorsal root L7. Stimulus intensities are indicated in mA. The stimulation threshold of the short lasting hyperpolarization is between 0.4 and 0.5 mA. Further increasing intensity causes also an increase of duration of hyperpolarization. Between 0.8 and 0.9 mA a limit is reached, when further increase of intensity has no effect on the duration. The duration remains constant.

To analyze the components of the different kinds of evoked hyperpolarization, the membrane potential was altered artificially by means of current injection.

Figure 2 shows recordings of short lasting evoked hyperpolarization in a lumbar motoneuron under normal conditions and under artificial current injection. The

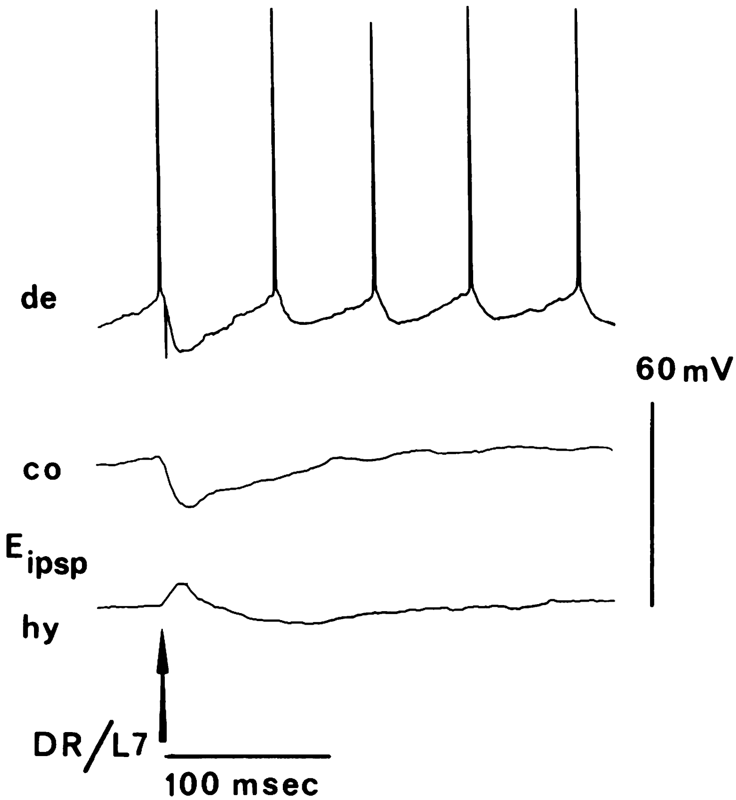

Figure 2. Intracellular recordings of a motoneuron of spinal cord segment L7 of cat. Short lasting hyperpolarization, elicited by an electric single stimulus of dorsal root L7. Change of membrane potential by artificial alteration. co = control recording; de = recording with artificial depolarization; hy = recording with artificial hyperpolarization; Eipsp = IPSP equilibrium potential. In trace hy the Eipsp has been surpassed and the hyperpolarizing IPSP potential changed to a depolarizing one, and the slightly depolarized potential of the disinhibition changed to a hyperpolarizing one. The recordings have been arranged without reference to absolute potential values.

duration of the hyperpolarization was 95 msec. The trace with depolarizing current injection, (de) shows increase of IPSP amplitude, increase of steepness of repolarization, and increase of discharge activity. The trace with hyperpolarizing current injection is marked (hy) in Figure 2. Here the IPSP equilibrium potential had been surpassed. After the thus depolarized IPSP, a hyperpolarization occurred, which corresponded to the depolarized part in normal recording and that under artificial depolarization. Wilson (3) already in 1962 made this observation and he called it disinhibition. This short lasting evoked hyperpolarization in the motoneuron thus consisted of an initial summated IPSP followed by a disinhibition.

To explain these phenomena a description of the discharge characteristics of spinal interneurons is necessary. Curtis and Ryall (4) showed that spinal interneurons have typical discharge patterns. This pattern was namely an EPSP with a high frequency discharge, or burst, consisting of a discharge pause and an afterdischarge (2). Many authors (1,3,5-9) have assumed the motoneurons to be under constant excitatory and inhibitory influence, the so called tonic background excitation and the tonic background inhibition. Commonly it is accepted that the burst discharge of inhibitory interneurons cause the initial summated IPSP by temporal and spatial summation (5). The discharge pause of inhibitory interneurons has been supposed to be the reason for disinhibition (3). Which means that the background inhibition was diminished. Since the background excitation now prevailed in the motoneuron a depolarization would happen after the IPSP, if the membrane potential was not higher or equal to the IPSP equilibrium potential.

Figure 3 shows a long lasting evoked hyperpolarization of 195 msec as response to a ventral root stimulus of segment L7. After the current injection, which changed the membrane potential to values beyond the IPSP equilibrium potential, the membrane potential after the thus depolarized initial IPSP became hyperpolarized in addition to the former value. In the lower part of the Figure 3 this effect is accentuated by superposition.

Long lasting evoked hyperpolarization in spinal motoneurons has been described repeatedly in the literature (5,9-13). Concerning the mechanism of its development, however, there are different opinions. On the one hand a prolonged transmitter effect was supposed to be the reason for the long duration of the hyperpolarization (5). On the other hand the so called remote inhibition was claimed to be the cause of delayed hyperpolarization (10,12,13) i.e. generation of IPSPs in remote regions of the dendritic tree, when especially the longitudinal constant of the dendrites must be taken into account. Our results, however, lead to the assumption of another mechanism: a disfacilitation.

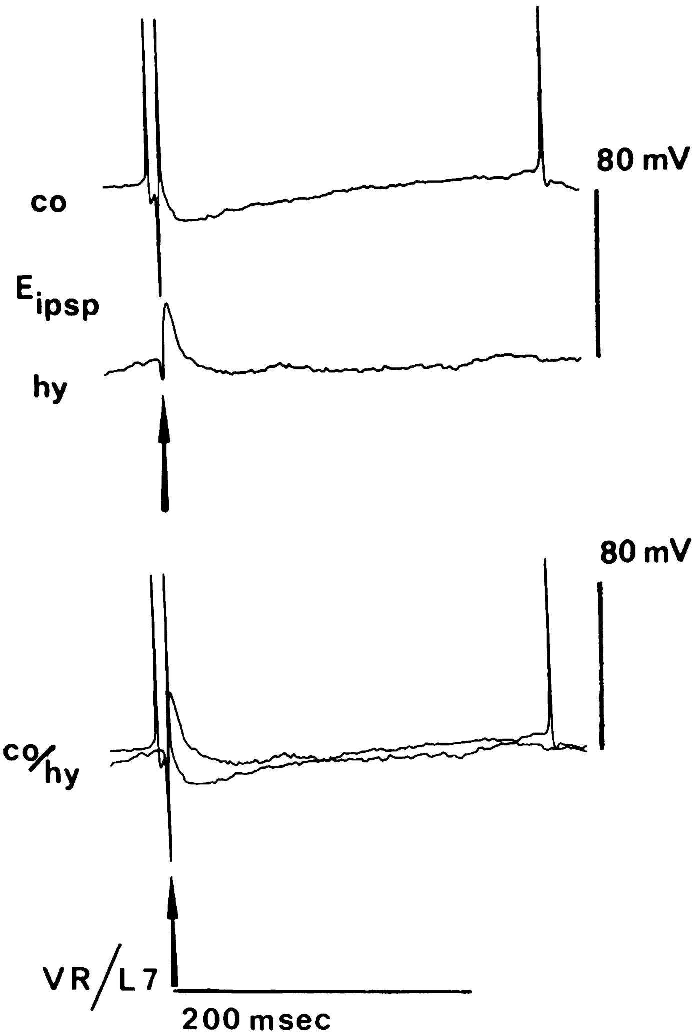

Figure 3. Intracellular recordings of a motoneuron of the spinal cord segment L7 of cat. The long lasting hyperpolarization, elicited by an electric single stimulus, of the ventral root L7. Change of the membrane potential by artificial alteration. Abbreviations as in Figure 2. The lower trace (co/hy) is a projection of the upper traces one upon another to clarify the effect. In trace hy the Eipsp has been surpassed and the hyperpolarizing IPSP potential changed to a depolarizing one and the hypolarizing part after the IPSP is hyperpolarized in addition. The recordings have been arranged without reference to absolute potential values.

This assumption is possible, since injected hyperpolarizing current caused an additional hyperpolarization of the evoked hyperpolarization following the IPSP, which itself was changed to a depolarizing potential. Conductance measurements of the membrane only showed meaningful increases during the initial IPSP, but not during the late part of the evoked hyperpolarization. If one assumes a prolonged transmitter effect, also during the late part then the conductance should be increased.

Against the assumption of a prolonged transmitter effect, also the additional hyperpolarization must be mentioned, which does not fit for an inhibition. If one assumes the late part of the long lasting evoked hyperpolarization to be originated by remote inhibitory synapses, this hyperpolarization should decrease. It should even be reversed just as the initial IPSP. Therefore we conclude that this hyperpolarization was a disfacilitation.

Tonic background excitation as well as tonic background inhibition are probably due to the activity of spinal interneurons (6-8,14). Since the disfacilitation is an interruption of excitatory synaptic influence on the motoneuron, we may conclude that this interruption would predominantly be due to a discharge pause in excitatory interneurons. The threshold of interruption of tonic background excitation is higher than that of the tonic background inhibition, because higher stimulus intensities must be used to obtain a disfacilitation. At the time we are not able to distinguish between excitatory and inhibitory interneurons in the spinal cord, but in our recordings of 35 spinal interneurons we always saw, with weak and with strong stimuli, after the burst discharge a discharge pause. And after strong stimuli which caused a disfacilitation in motoneurons, we always observed in interneurons a long discharge pause corresponding to disfacilitation. Thus we have to assume that during disfacilitation also disinhibition still exists.

DISCUSSION

The results described are consistent with those we obtained in cortical pyramidal cell (1,2). In cortical pyramidal cell it is possible to interrupt the long lasting evoked hyperpolarization by a second stimulus. This effect is called potentiation. The second stimulus has to have a certain intensity which must be less than that of the first one and must have a certain delay to the first stimulus. Furthermore, by means of current injection it could be shown, that the second stimulus interrupted the disfacilitation and not the disinhibition. Figure 4 shows that also in the motoneuron it is possible to interrupt the long lasting evoked hyperpolarization by

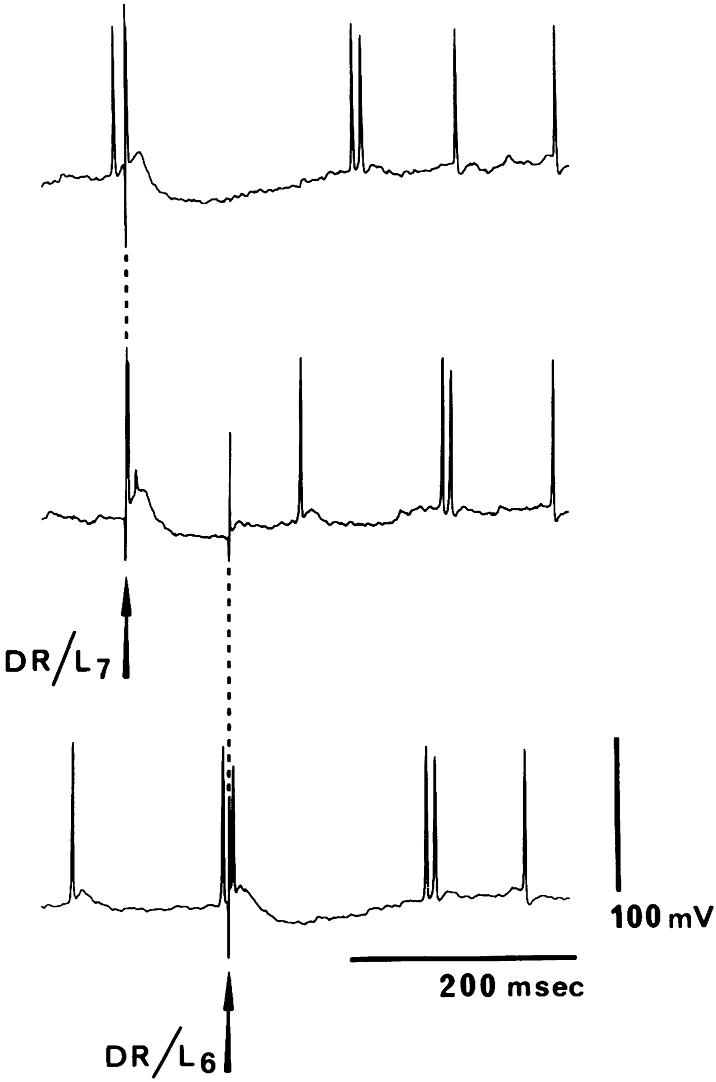

Figure 4. Intracellular recordings of a motoneuron of spinal cord segment L7 of cat. Potentiation of the evoked long lasting hyperpolarization (upper trace; stimulus of 3.0 mA) during the late part by a second shock of 1.0 mA at the dorsal root L6 (middle trace). The delay between either stimuli is 80 msec. The lower trace shows the response to the potentiating stimulus, when it is applied alone.

a second stimulus. The intensity of this stimulus must be between 0.8 and 1.1 mA, in other words, it must be weaker than the first one. The delay to the first stimulus must be at least between 70 and 80 msec. This means that potentiation only can be obtained, when no IPSP is present anymore. These values are independent of the intensity of the first stimulus. It is only necessary that the first stimulus has at least intensity to evoke a long lasting hyperpolarization. That means, that a disfacilitation exists. In most cases the second stimulus applied alone only evoked a short lasting hyperpolarization. Up to now we do not have the evidence, but it can be assumed - corresponding to our results in the cortex -, that also in the motoneuron the second stimulus will only interrupt the disfacilitation and not the disinhibition.

SUMMARY

In spinal motoneurons there are two kinds of evoked hyperpolarization: a short and a long lasting one. The short lasting hyperpolarization is stimulus dependent and consists of an initial summated IPSP followed by a disinhibition. This disinhibition apparently is caused by the interruption of the tonic background inhibition, due to a discharge pause in inhibitory interneurons. The components of long lasting evoked hyperpolarization are also initial summated IPSPs followed by still present disinhibition and additional disfacilitation which probably is caused by the interruption of the tonic background excitation, due to a discharge pause in excitatory interneurons. It is possible to interrupt the long lasting evoked hyperpolarization by a second but weaker stimulus. The reported results are consistent with those we obtained in cortical pyramidal cells and interneurons.

ACKNOWLEDGEMENT

Supported by the Deutsche Forschungsgemeinschaft (Vi 36/1-8).

REFERENCES

1. Vieth, J., Kneise, U. and Kaeferlein, J. (1974): Evozierte Enthemmung und Bahnungsminderung in corticalen Nervenzellen. Arch.Psychiat.Nervenkr. 218: 271-290.
2. Vieth, J. and Faust, J.: Excitatory and inhibitory interneurons in the cortex of the cat. In this volume.
3. Wilson, V.J. and Burgess, P.R. (1962): Disinhibition in the cat spinal cord. J.Neurophysiol. 25: 392-404.
4. Curtis, D.R. and Ryall, R.W. (1966): The synaptic excitation of Renshaw cells. Exp.Brain Res. 2: 81-96.

5. Eccles, J.C. (1964): The Physiology of Synapses. Springer, Heidelberg.
6. Hunt, C.C. and Kuno, M. (1959): Background discharge and evoked responses of spinal interneurons. J. Physiol. 147: 364-384.
7. Koizumi, K., Ushiyama, J. and Brooks, C.M.C. (1959): A study of reticular formation action on spinal interneurons and motoneurons. Jap.J.Physiol. 9: 282-303.
8. Terzuolo, C.A. (1959): Cerebellar inhibitory and excitatory actions upon spinal extensor-motoneurons. Arch.ital.Biol. 97: 316-339.
9. Sutor, B. and Vieth, J. (1979): Kurz- und langdauernde evozierte Hyperpolarisation in Motoneuronen der Katze. EEG-EMG 10, 50.
10. Cook jr., W.A. and Cangiano, A. (1972): Presynaptic and postsynaptic inhibition of spinal motoneurons. J. Neurophysiol. 35: 389-403.
11. Davenport, J., Schwindt, P.C. and Crill, W.E. (1978): Presynaptic and long-lasting postsynaptic inhibition during penicillin-induced spinal seizures. Neurology, 28: 592-597.
12. Kellerth, J.O.(1968): Aspects of the relative significance of pre and postsynaptic inhibition in the spinal cord. In: Structure and Function of Inhibitory Neuronal Mechanisms. Ed.: C. von Euler, S. Skoglund and U. Soederberg, Pergamon, Oxford.
13. Schomburg, E.D., Meinck, H.M., Haustein, J. and Roesler, J. (1978): Functional organization of the spinal reflex pathways from forelimb afferents to hindlimb motoneurons in the cat. Brain Res. 139: 21-33.
14. Frank, K. and Fuortes, M.G.F. (1956): Unitary activity of spinal interneurons of cats. J. Physiol. 131: 424-435.