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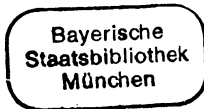
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COMPONENTS OF EVOKED HYPERPOLARIZATION IN THE SPINAL MOTONEURON OF THE CAT

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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 transected 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.

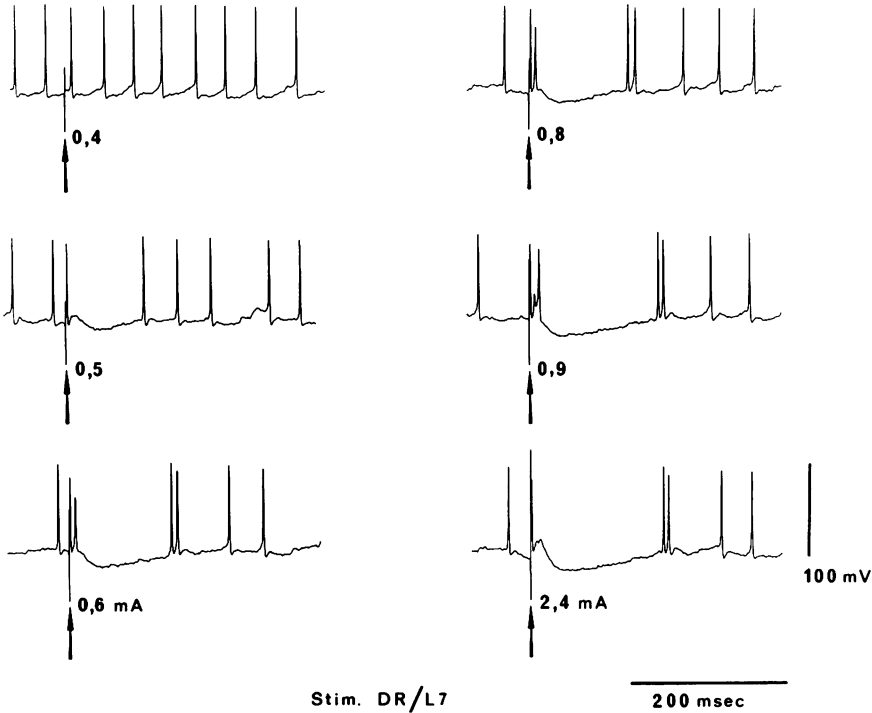


Figure 1. Intracellular recordings of a motoneuron of spinal cord segment L7 of cat. Short and long lasting hyperpolarization as response to single electric stimulus of dorsal root L7. Stimulus intensities are indicated in mA. The stimulation threshold of the short lasting hyperpolarization is between 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

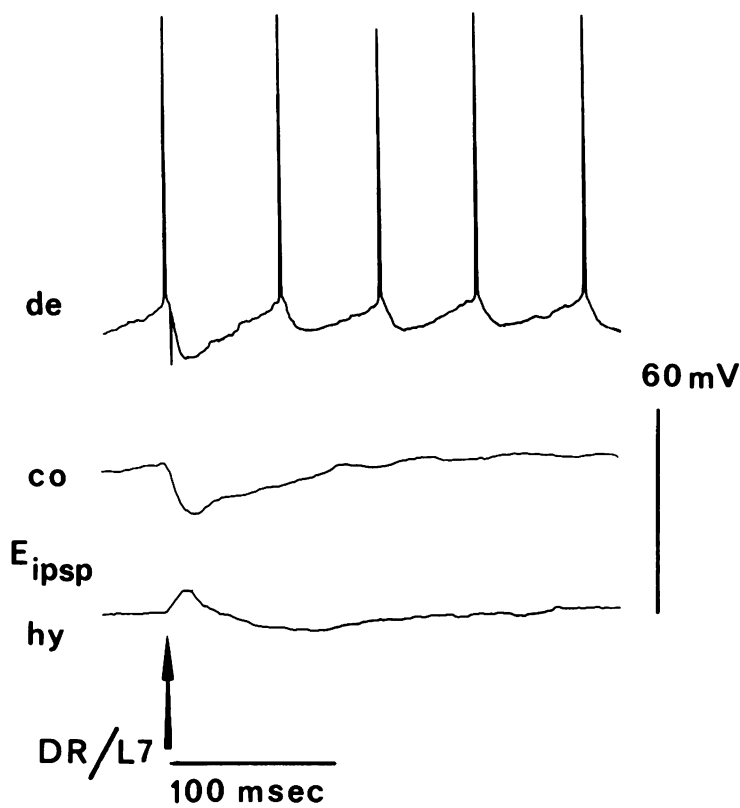


Figure 2. Intracellular recordings of a motoneuron of spinal cord segment L7 of cat. Short lasting hyperpolarization, elicited by an electric single stimulus of dorsal root L7. Change of membrane potential by artificial alteration. co = control recording; de = recording with artificial depolarization; hy = recording with artificial hyperpolarization; E_{ipsp} = IPSP equilibrium potential. In trace hy the E_{ipsp} 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.

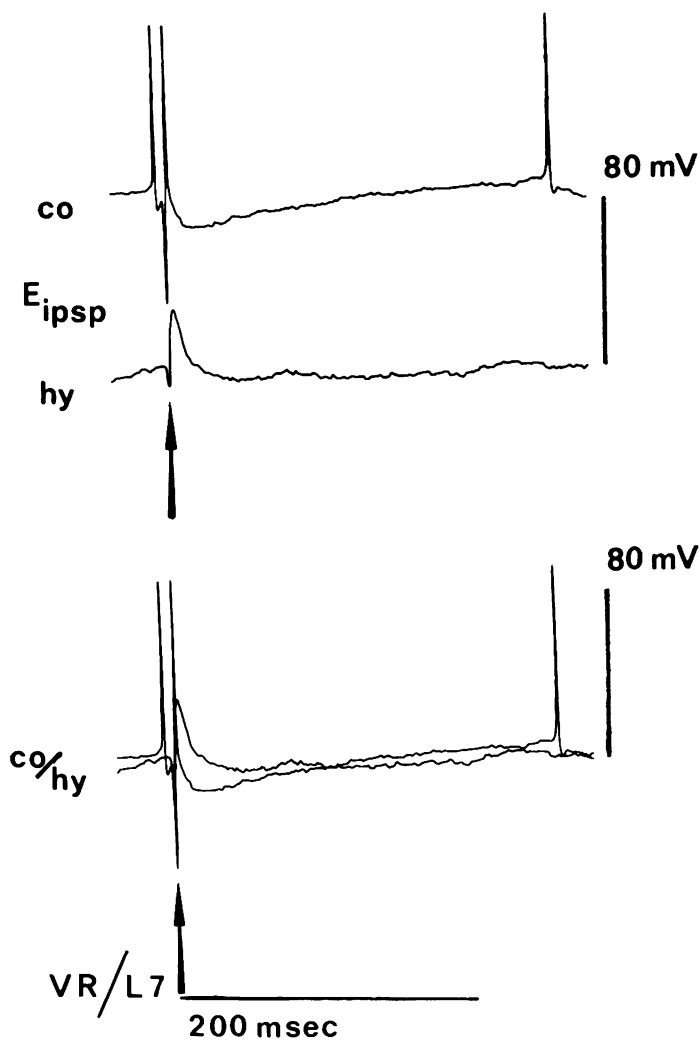


Figure 3. Intracellular recordings of a motoneuron of the spinal cord segment L7 of cat. The long lasting hyperpolarization, elicited by an electric single stimulus, of the ventral root L7. Change of the membrane potential by artificial alteration. Abbreviations as in Figure 2. The lower trace (co/hy) 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 hyperpolarizing 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

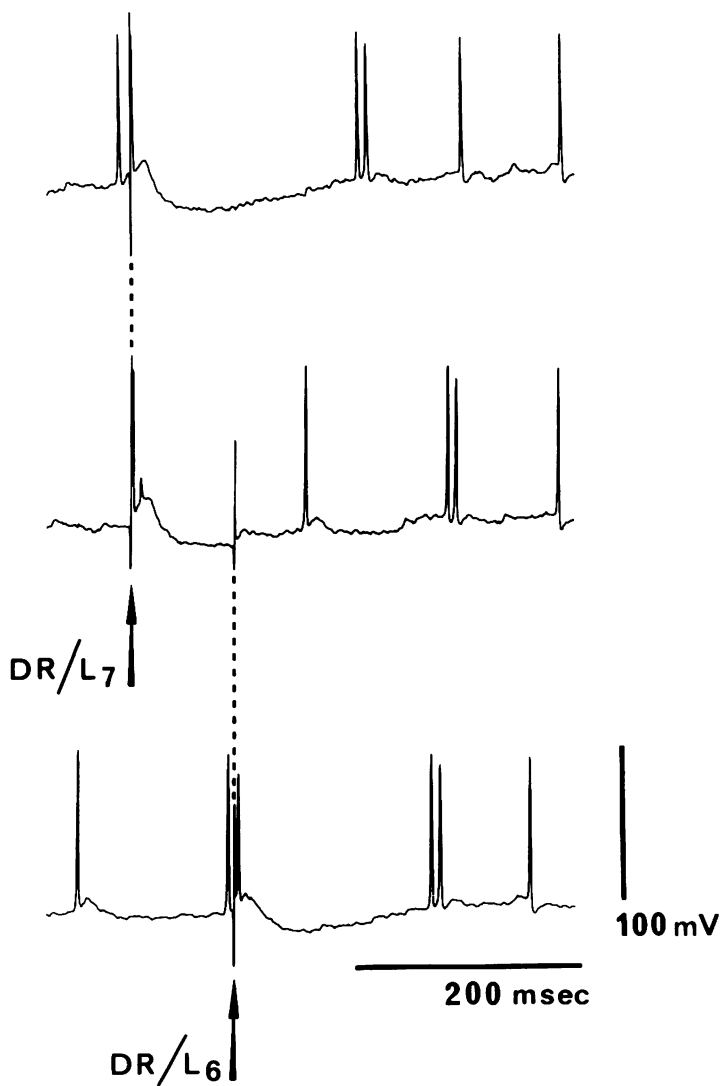


Figure 4. Intracellular recordings of a motoneuron of spinal cord segment L7 of cat. Potentiation of the evoked long lasting hyperpolarization (upper trace; stimulus of 3.0 mA) during the late part by a second shock of 1.0 mA at the dorsal root L6 (middle trace). The delay between either stimuli is 80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

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