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The effects of postsynaptic inhibition on the monosynaptic reflex of the cat at different levels of motoneuron pool activity

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Summary. The motoneurons to the Soleus muscle in the decerebrate cat were activated by the crossed extensor reflex, elicited by stimulation of the contralateral common peroneal (CP) nerve. Monosynaptic reflexes were obtained from the Soleus motoneuron pool by stimulation of the cut L7-S1 dorsal roots. The amplitude of the reflex increased approximately linearly with the recruitment level of the motoneuron pool. Tonic postsynaptic inhibition was induced in the Soleus motoneuron pool by repetitive antidromic stimulation of the Lateral Gastrocnemius (LG) and Medial Gastrocnemius (MG) nerves at a rate of 17–47 stimuli/s. This reduced the size of the monosynaptic reflex at rest by at least 40%. However, when the motoneurons were active, the amplitude of the monosynaptic reflex obtained during repetitive stimulation of the LG-MG nerve increased with the recruitment level along the same curve as the control reflexes. Thus, tonic postsynaptic inhibition of the motoneurons per se cannot control the amplitude of the monosynaptic reflex independently of the recruitment level of the motoneuron pool. These experimental results verify predictions from computer simulations and suggest by exclusion that presynaptic inhibition is needed to control the amplitude of the monosynaptic reflex independently of the recruitment level of the motor pool.

Key words: Motoneuron pool – Inhibition – Monosynaptic reflex – Recruitment level – Cat

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Introduction

We showed in a series of experiments in normal human subjects that the amplitude of the H-reflex (an electrically induced analog of the monosynaptic stretch reflex) was strongly task dependent and that it could be controlled by the central nervous system (CNS) independently of the level of EMG activity (Capaday and Stein 1986, 1987a). For example, the reflexes were much larger during maintained postures ranging from quiet standing to standing on the toes of one leg compared to those obtained during walking when measured at the same level of EMG activity. Similarly, the H-reflex was smaller during running, at the same level of motor activity, than during walking. It has also been observed that the H-reflex amplitude is smaller during lengthening compared to shortening contractions of the ankle extensors at the same level of EMG activity (Romano and Schieppati 1987). These characteristic changes in the H-reflex, which occur in going from one natural motor activity to another, are specifically adapted to the functional requirements of each task as discussed in previous publications (Stein and Capaday 1988; Romano and Schieppati 1987).

A priori, one might assume that the changes in reflexes at a given level of motor activity could be produced either by presynaptic inhibition or by the shunting effects of postsynaptic inhibition (Araki et al. 1960; Capaday and Stein 1986). Thus, during standing the reflexes would be large because only the excitatory conductance of motoneurons is activated. During locomotion motoneurons are recruited by an increased excitatory conductance and a reduction of the inhibitory conductance (Perret 1986; Shefchyk et al. 1984), and reflexes might be smaller because of the shunting effect of the inhibitory conductance on the monosynaptic EPSP.

However, more careful analysis and the results of

computer simulations led to a different conclusion. In the simulations presynaptic inhibition decreased the size of the monosynaptic reflex at all recruitment levels, whereas postsynaptic inhibition did not decrease the size of the reflex output independently of the recruitment level. The detailed reasons for this striking result are discussed elsewhere (Capaday and Stein 1987b) but can be explained in a relatively simple way. In order to maintain the recruitment level at a constant value any increase in tonic postsynaptic inhibition must be counteracted by an equal and opposite excitatory current. The increase in the excitatory conductance necessary to counteract the added inhibitory conductance is a constant multiple of the latter and is independent of the resting conductance of the motoneuron or the distribution of resting conductances within the motoneuron pool (Capaday and Stein 1987b). Therefore, postsynaptic inhibition decreases the recruitment level of the motoneuron pool and only coincidentally the reflex output. Of course, both presynaptic and postsynaptic inhibition will decrease the size of the reflex output when the motoneuron pool is quiescent.

In this paper we verified using decerebrate cats the more controversial prediction of the simulation studies. We found experimentally that postsynaptic inhibition does not change the amplitude of the monosynaptic reflex independently of the recruitment level of the motoneuron pool. An implication of this result is that the H-reflex method tests only the efficacy of synaptic transmission between the Ia-afferents and the motoneurons when comparisons are made at the same level of motor output (see also Hultborn et al. 1987). A brief account of the present results has been published (Capaday and Stein 1988).

Methods

Experiments were done on eleven cats decerebrated at the precollicular postmammillary level. These animals often show spontaneous episodes of motor activity and can exhibit well coordinated locomotion (see for example, Akazawa et al. 1982; Nichols 1985). The animals were operated on under Halothane anesthesia. A cannula was inserted in the right carotid artery for recording the arterial blood pressure; the left carotid artery was doubly ligated. A cannula was also inserted in one of the jugular veins to inject Dextran, a plasma volume expander, and solutions of L-Dopa and Nardil (Monoamine oxidase inhibitor) as described below. The triceps surae muscle group in the left hindleg was exposed and the Soleus muscle prepared for attachment to a strain gauge transducer. The nerves to the LG and MG muscles were dissected from the surrounding tissues, separated from the nerve to the Soleus, cut at their site of entry into the muscle belly, and mounted together on silver hook electrodes for stimulation. The right common peroneal nerve was also exposed, a cuff electrode was placed around it, and the wound was then closed. A laminectomy was made extending from the S1 to the L5 spinal vertebra. The S1 and L7 dorsal roots were cut while the animal was under anaesthesia and mounted on a pair of silver hook electrodes for stimulation. A pool was made with skin flaps at both the left leg and the vertebral column and filled with parafin oil. The leg and spinal cord were kept at approximately 37 C by radiant heat.

Experimental protocol

Variations in the level of Soleus motor output were produced by stimulation of the contralateral common peroneal nerve at a strength 2–5 times motor threshold and at a stimulation rate of between 50–100 stimuli/s. This produces the classic crossed-extensor reflex, with flexion of the ipsilateral leg and extension of the contralateral one. The electrical stimulation was sometimes supplemented by pinching the skin on the stimulated leg. Monosynaptic reflexes were obtained by stimulation of the L7-S1 dorsal roots at twice the threshold for obtaining a monosynaptic response. The reflex responses were recorded electromyographically from the Soleus muscle using a pair of intramuscular multistranded stainless steel wires. Sometimes reflexes were obtained during periods of spontaneous activity. In some experiments solutions of L-Dopa (20 mg/kg) and Nardil (20–30 mg/kg) were injected intravenously in order to potentiate the crossed extensor reflexes (see for example, Grillner, 1986). The results obtained from animals in which L-Dopa was used were qualitatively similar to those obtained from animals where L-Dopa was not used.

In order to introduce a tonic level of postsynaptic inhibition without introducing any presynaptic inhibition, the nerves to the Lateral and Medial Gastrocnemius muscles (LG, MG) were stimulated repetitively at physiological rates between 17–47 stimuli/s. Stimulation of the LG-MG nerves excites the Renshaw cells of the LG and MG motoneurons, which also project to the Soleus motoneurons (Granit 1972; Kuno 1959; Renshaw 1941). The strength of the stimulus to the LG-MG nerves was adjusted to produce at least a 50–60% reduction of the monosynaptic reflex by using the conditioning/test stimulus paradigm. The interval between the conditioning stimulus (LG-MG nerve stimulus) and the test stimulus (dorsal root stimulus) was between 5–10 ms (Renshaw 1941). When the stimulation is applied repetitively, the Soleus motoneurons receive a tonic level of postsynaptic inhibition (Granit 1971; Haase 1963) and they alone are tested in response to the dorsal root stimulus. Furthermore, since the dorsal roots are cut, activation of the afferent fibres in the LG-MG nerve cannot influence the Soleus motoneurons.

To summarize our experimental protocol, we activated the Soleus muscle via the crossed extensor reflex and obtained monosynaptic reflex responses over a wide range of muscle activity. We then repeated this procedure during repetitive stimulation of the LG-MG nerve. This gives monosynaptic reflex responses in the presence of a tonic level of postsynaptic inhibition in the Soleus motoneurons. A schematic representation of the experimental preparation is shown in Fig.1.

Method of analysis

The data obtained during an experiment and the markers for the various stimulus pulses were stored on FM magnetic tape for later analysis on a digital computer. On playback, the Soleus EMG was amplified to a suitable level, high-pass filtered at 10 Hz and then processed directly (eg. Figs. 2 and 4), or rectified and low pass filtered at 300 Hz (eg. Fig. 3). The tension record was

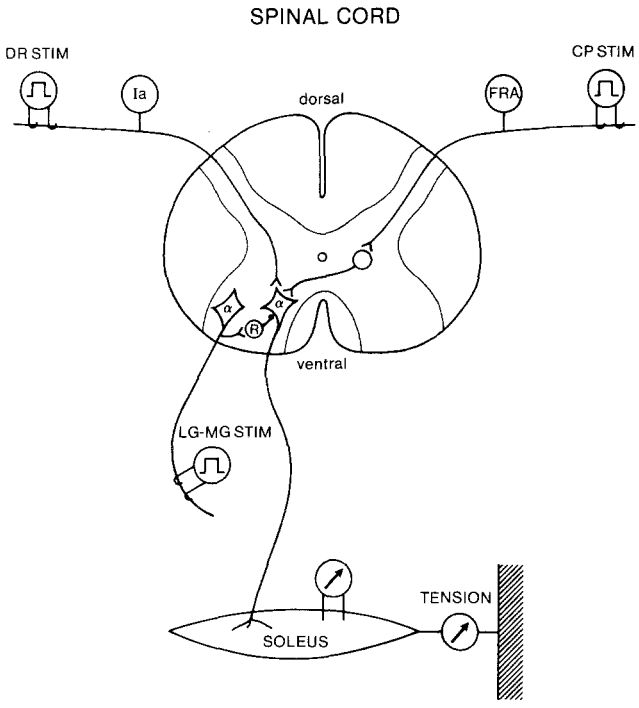

Fig. 1. Schematic representation of the experimental preparation used to study the effects of postsynaptic inhibition on the monosynaptic reflex. Activation of the Soleus motoneurons was produced by stimulation of the flexor reflex afferents (FRA) in the contralateral common peroneal nerve (CP). The Soleus monosynaptic reflex was elicited by stimulation of the L7–S1 dorsal roots (DR) and recorded electromyographically and myographically. Postsynaptic inhibition of the Soleus motoneurons was induced by antidromic stimulation of the Lateral and Medial Gastrocnemius nerves (LG-MG), which activates the Renshaw (R) cells

low pass filtered at 50 Hz. The computer sampled the Soleus reflex EMG response for a period of approximately 30 ms following the stimulus to the dorsal root(s). It then averaged this signal with others that occurred at the same level of motor output as measured from the Soleus tension record. Finally, the peak to peak value of each averaged reflex response was calculated. Thus, a plot of average reflex amplitude versus the level of motor activity (tension) was obtained under control conditions. The same procedure was applied to the data obtained in the presence of tonic postsynaptic inhibition. The reflexes obtained in the control condition could then be compared to those obtained during repetitive stimulation of the Renshaw cells.

Results

Stimulation of the Renshaw cells was chosen as a method to induce postsynaptic inhibition of the Soleus motoneurons because this pathway was the most likely to produce postsynaptic inhibition only (Baldissera et al. 1981). There are no known pathways by which stimulation of Renshaw cells can produce presynaptic inhibition of the Ia-terminals projecting to the motoneurons. Antidromic stimulation of ventral roots does not evoke any dorsal root potentials (DRP) (Barron and Matthews 1938; Hultborn et al. 1971). Furthermore, the excitability of the Ia-afferent terminals is not changed (Wall 1958), nor is the size of the Ia EPSP recorded in motoneurons (Hultborn et al. 1971). Indeed, in our experiments we never detected any DRP in response to stimulation of the ventral roots. It is therefore unlikely that stimulation of the Renshaw cells produces any change in the level of presynaptic inhibition of the Ia-terminals projecting to the motoneurons.

In Fig. 2 reflex responses obtained at rest during repetitive stimulation of the LG-MG nerve and following a single conditioning stimulus are shown. The reflex response conditioned by a single stimulus to the LG-MG nerves is decreased by 80% compared to control. When repetitive stimulation at 47 stimuli/s was used, the reflex response is decreased by 40% compared to control. In other instances repetitive stimulation could decrease the size of the reflex response by as much as 60–70%. These observations demonstrate that repetitive stimulation of Renshaw cells is an effective method for producing a tonic level of postsynaptic inhibition in the motoneurons. However, repetitive stimulation at physiological rates does not inhibit the monosynaptic reflex as potently as an appropriately timed single stimulus. This is probably because the Renshaw cells do not fire with a high frequency burst during repetitive antidromic stimulation, as they do in response to a single stimulus, but instead discharge

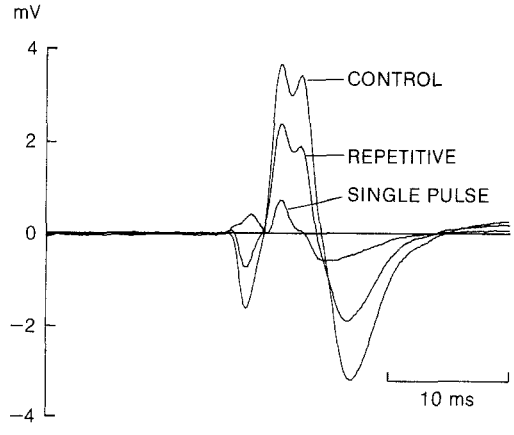

Fig. 2. Examples of monosynaptic reflexes obtained at rest in the three experimental conditions investigated. The largest reflex is the control (uninhibited) response. The reflex of intermediate size is one obtained during repetitive stimulation of the LG-MG nerves at 47 stimuli/s. The smallest reflex was obtained when a single conditioning stimulus to the LG-MG nerve preceded the test stimulus by 10 ms. Each record is the average of 16 responses

repetitively at a lower rate (Haase 1963). Therefore, the average change in inhibitory conductance may be less when repetitive stimulation is used.

Furthermore, the effects of repetitive antidromic stimulation on the amplitude of the monosynaptic reflex at rest were tested over prolonged periods of stimulation of up to 1 min. For example, the average reflex response during repetitive stimulation shown in Fig. 2 was obtained during a period of about 20 s, with the reflex responses elicited at a rate of 1 stimuli/s. There was no tendency for the reflex responses to increase during the course of the trial. This indicates that the Renshaw cells are capable of maintaining their inhibitory capacity during the course of prolonged stimulation at physiological rates.

Stimulation of the contralateral common peroneal nerve produced a crossed extensor reflex in the Soleus which usually consisted of a relatively rapid increase of tension to a maximum value followed by a slow decay to zero after one or more peaks (Fig. 3). Monosynaptic reflexes were elicited at a rate of one per second during such a period of activity. Stimulation of the CP nerve was repeated several times to obtain a wide range of recruitment levels, as well as a large number of monosynaptic reflex responses for averaging. It can be seen in Fig. 3 that the monosynaptic reflex amplitude was related to the recruitment level. It was largest at high recruitment levels, small at low recruitment levels, and varied approximately linearly with the recruitment level (see for example, Fig. 5). Such a correlation has been repeatedly found in normal humans and has been termed automatic gain compensation (Matthews 1986; Marsden et al. 1972; Gottlieb et al. 1970).

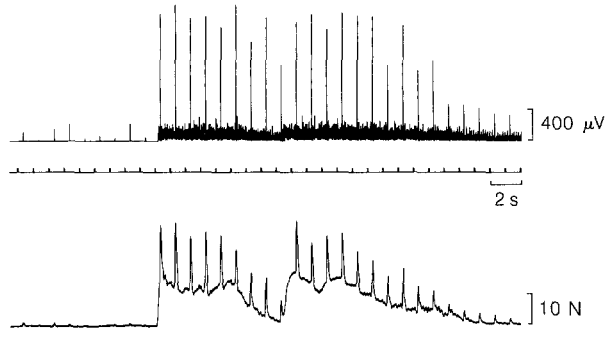

Fig. 3. Example of a crossed extensor reflex recorded from the Soleus muscle during stimulation of the contralateral CP nerve (60 stimuli/s at 4 times motor threshold). The electromyographic activity is shown in the upper record and the muscle tension in the lower record. Monosynaptic reflex responses were elicited at a rate of 1/s and appear as sharp, brief, deflections superimposed on the background activity level. Note how the amplitude of the reflex response is related to the background level

In Fig. 4A the average monosynaptic reflex responses obtained over a range of Soleus muscle tension are shown. Monosynaptic reflex responses obtained during repetitive stimulation of the LG-MG nerve at 47 stimuli/s are shown alongside the control reflexes in Fig. 4B. Two observations are pertinent to this figure and are indeed the basic result presented in this paper. First the amplitude of the control reflex is essentially the same, at the same level of motor output, as that of the reflex obtained during tonic inhibition of the Soleus motoneurons.

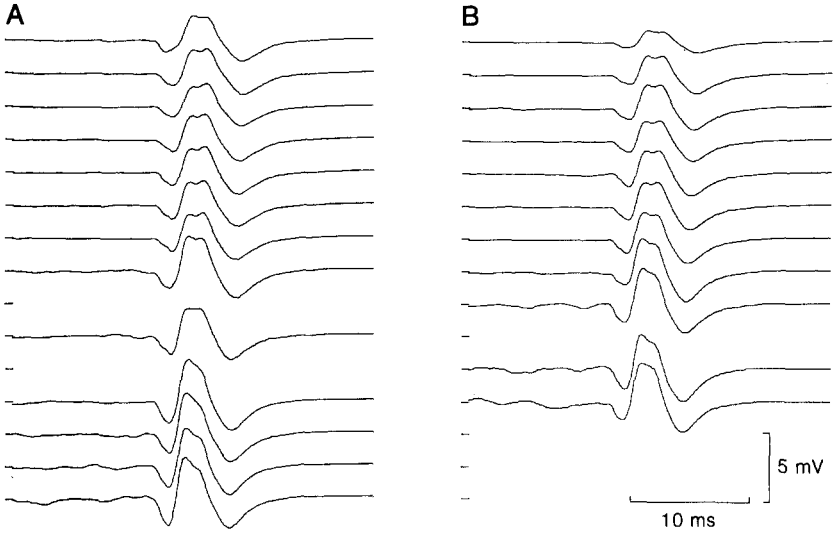

Fig. 4A, B. Soleus monosynaptic reflex responses obtained during stimulation of the contralateral CP nerve. Reflex responses obtained at progressively greater force levels are plotted from the top downward. The topmost response was obtained when the tension was between 0–0.92 N, the second between 0.92–1.84 N and so on until the lowermost response obtained between 12.9–13.8 N. Each record is the average of between 1 and 23 responses. The gaps where no responses are seen are for tension ranges where no stimulus occured. In **A** responses obtained under control conditions are shown. Those obtained during repetitive stimulation of the LG-MG nerve at 47 stimuli/s are shown in **B**. Note the generally similar amplitude of the reflex responses at the same background level of muscle tension in the two conditions. Note also that during repetitive stimulation of the LG-MG nerve the highest level of muscle tension attained was less than during the control condition

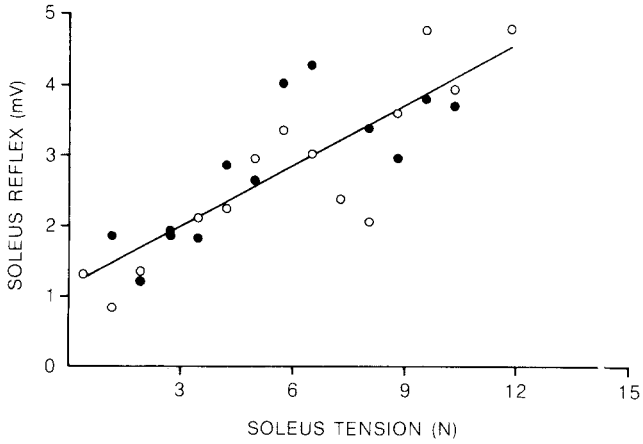

Fig. 5. Plot of the peak to peak value of the Soleus monosynaptic reflex response versus the background level of Soleus muscle tension. Open circles (○) indicate values obtained in the control condition. Those marked by the filled circles (●) were obtained during repetitive stimulation of the LG-MG nerve at 37 Hz. A single line is fitted through all the data because there were no significant differences between the two data sets

A one tailed t-test for correlated samples confirms that there is no significant difference between the reflexes obtained in the two conditions. The other observation is that the maximum tension level attained during repetitive stimulation of the LG-MG nerve is smaller than that attained in the control situation. It appears therefore that the Soleus motoneurons were being inhibited by the antidromic stimulation and that this resulted in a smaller motor output as measured by the tension recording. It should be noted that the same antidromic stimulation at rest inhibited the monosynaptic reflex by 80%. Furthermore, before averaging together responses obtained in different episodes of crossed extension activity, 5–7 reflexes obtained at rest just before stimulation of the CP nerve were compared in order to insure that the state of synaptic transmission between the Ia-afferents and the motoneurons was comparable in each case.

The peak to peak amplitude of the Soleus monosynaptic reflex is plotted against the isometric muscle tension in Fig. 5. The amplitude of the control reflexes increases with the level of motor activity (points labelled with (○) symbol). The data are well fitted by a straight line having a slope of 0.31 mv/N (SE = 0.043 mv/N) and a correlation coefficient (r) of 0.89. The reflexes obtained during stimulation of the LG-MG nerve are plotted on the same graph (points labelled with (●) symbol). The slope for these data is 0.26 mv/N (SE = 0.06 mv/N). The y-intercept for the line fitted to the control data was 0.94 mV (SE = 0.29 mV) and that for the one fitted to the reflexes obtained during repetitive antidromic stimulation was 1.43 mV (SE = 0.41 mV). The values are therefore not statistically different and a common line fitted to all the data is shown in Fig. 5. It can also be seen in this figure that the highest value of muscle tension attained during stimulation of the LG-MG nerve was less than that attained in the control situation (see also, Fig. 4). As already mentioned, this indicates that indeed there was a tonic postsynaptic inhibition acting on the Soleus motoneurons.

The experiment described above, done with repetitive stimulation of the LG-MG nerves, was repeated using only a single stimulus to the nerves which preceded the test dorsal root stimulus by 5–10 ms. In this situation the IPSP is timed to have a maximal effect on the EPSP. The IPSP would temporarily hyperpolarize the membrane potential of the motoneurons and shunt the EPSP, thereby decreasing the amplitude of the reflex output. This can be seen in Fig. 6 where reflexes obtained under control conditions (○), repetitive antidromic stimulation (●), and conditioned by a single antidromic stimulus (△), are plotted against the muscle tension on the same graph. The responses obtained during repetitive stimulation of the LG-MG nerve superimpose almost exactly on those obtained in the control condition. The amplitude of the reflexes conditioned by a single antidromic stimulus were on average about half the size of those in the other two conditions. The best fitting straight lines, according to a least mean Sp squares criterion, are drawn for all three sets of data.

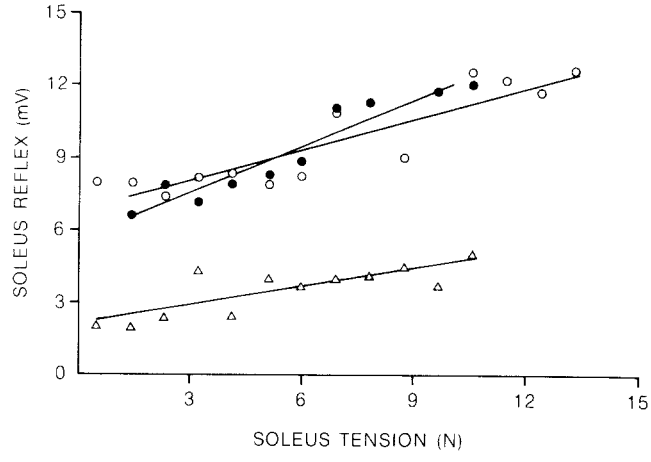

Fig. 6. Plot of the peak to peak value of the monosynaptic reflex versus the background muscle tension for the three experimental conditions investigated. Value obtained under control conditions (no inhibition) are marked by the (○) symbol; values obtained during repetitive stimulation of the LG-MG nerves at 47 stimuli/s are marked with the (●) symbol and those obtained when a single stimulus preceded the test stimulus by 10 ms are marked by the (△) symbol. All three data sets have been fitted by straight lines according to a least mean squares criterion

Discussion

When the motoneuron pool is quiescent an increase of either presynaptic inhibition or postsynaptic inhibition can decrease the amplitude of the monosynaptic reflex. However, in this study we have shown that, when the motoneurons are active, postsynaptic inhibition per se cannot decrease the amplitude of the monosynaptic reflex independently of the level of motor activity. The amplitude fo the monosynaptic reflex output is tied to the recruitment level of the motor pool and is independent of the particular combination of excitatory and inhibitory synaptic currents acting on the motoneurons. The main prediction from our earlier modelling studies thus appears to be supported by experiment (Capaday and Stein 1987b).

Methodological issues

There are three methodological issues that warrant further discussion. Firstly, the efficacy of the Renshaw cell inhibition of the motoneurons may be tied to the recruitment level such that, as the recruitment level increases, the Renshaw cells are progressively more inhibited (Hultborn and Pierrot-Deseilligny 1979). This can account for the observed lack of effect of tonic levels of postsynaptic inhibition on the monosynaptic reflex response when the motoneurons are active. Secondly, the stimulus intensity, used to obtain a reflex response (2 × reflex threshold), may have been too strong to reveal an inhibitory effect produced by the Renshaw cells. Both of these potential factors are not consistent with the observation that a single stimulus to the Renshaw cells inhibits the reflex response at all levels of motor activity that we investigated (Fig. 6). Therefore, the Renshaw cells in our experimental condition retained their capacity to inhibit the motoneurons.

Thirdly, it is possible that during the prolonged period of repetitive stimulation of the Renshaw cells, which sometimes lasted for up to a minute, their neurotransmitter content became depleted, or transmitter release depressed, so that no inhibition of the motoneurons occurred. However, we used stimulation rates of between 17–47 stimuli/s which is within the natural firing range of gastrocnemius motoneurons. If such rates of stimulation of Renshaw cells can rapidly deplete their neurotransmitter content, then this must also happen during natural motor activity. More to the point, we verified throughout the course of each experiment that the Renshaw cells retained their inhibitory capacity by recording monosynaptic reflex responses obtained at rest following a single conditioning stimulus to the LG-MG nerves. We also observed that during repetitive stimulation of the Renshaw Sp, cells reflex responses obtained at rest were decreased throughout the period of stimulation, with no tendency for the reflexes to increase with time during the course of the stimulation. In summary, the physiological range of stimulation used and the periodic checks on the efficacy of the Renshaw inhibition make it unlikely that the synaptic terminals of the Renshaw cells were depleted, or transmitter release depressed, at any time during our experiments.

Neural mechanisms

The lack of effect of postsynaptic inhibition observed in this study is reminiscent of the observation that this mechanism does not change the critical firing level of a motoneuron in a monosynaptic reflex (Clamann et al. 1974). The critical firing level, as defined by these authors, is the minimum level of the monosynaptic reflex response at which the motoneuron first discharges. Thus, in the presence of a maintained postsynaptic inhibitory input, a motoneuron begins to discharge at the same level of recruitment of the motor pool as in the control condition.

What is new in the present study is that the amplitude of the monosynaptic reflex output is also tied to the recruitment level, regardless of what balance of synaptic excitation and inhibition is used to achieve that recruitment level. Therefore, to change the size of the monosynaptic reflex independently of the level of motor output, the efficacy of synaptic transmission between the Ia-afferents and the motoneurons must be altered. This can be done by controlling the level of presynaptic inhibition onto the Ia-terminals (Eccles 1964; Burke and Rudomin 1977). Such a presynaptic control mechanism can be very specifically directed onto one class of terminals within the spinal cord to the exlusion of other nearby terminals (Rudomin 1980). The present observations are also consistent with those made by Hultborn et al. (1987). They found that the amount of facilitation produced by a conditioning Ia stimulus on a control Ia reflex was not affected when the motoneuron pool was subjected to pure postsynaptic inhibition, but was affected by presynaptic inhibition. Although their studies involved a conditioning/test reflex paradigm and transient inputs

their major conclusion is consistent with the present one based on maintained synaptic inputs.

A key point regarding both the modelling studies and the present experimental tests of the model is the assumed distribution of postsynaptic inhibition in the motor pool. Our assumption is that all the motoneurons in the pool receive an inhibitory input. The distribution of inhibition in the pool, however, need not be uniform for our prediction to apply. We have shown, for example, that if the strength of the inhibition, measured as the magnitude of the inhibitory conductance, is made directly proportional to the motoneuron's resting conductance (i.e. large motoneurons receive proportionately more inhibition than small motoneurons), the qualitative effects of postsynaptic inhibition on the amplitude of the monosynaptic reflex remain the same (Capaday and Stein 1987b).

A clear difference between the effects of repetitive stimulation and those of a single stimulus to the Renshaw cells was observed. A tonic level of postsynaptic inhibition does not affect the amplitude of the monosynaptic reflex response independently of the level of motor output. On the other hand, a single IPSP produced by an antidromic stimulus to the LG-MG nerves decreases the monosynaptic reflex response at all levels of motor output (Fig. 6). This reduction can result from two mechanisms: 1) shunting of the EPSP by the recurrent IPSP and/or 2) transient hyperpolarization of the membrane. However, the IPSP being a brief event, occuring nearly simultaneously with the EPSP, will probably exert the greater part of its effect by shunting rather than by hyperpolarization. Therefore, the reduction of the reflex responses shown in Fig. 6 is probably due to shunting of the EPSP by the recurrent IPSP.

An increase in the level of presynaptic inhibition would result in a smaller Ia-EPSP and therefore in a smaller reflex response at all levels of motor output. Presynaptic inhibition is therefore a possible mechanism that allows for the control of the monosynaptic reflex amplitude independently of the level of motor output. Thus, the characteristic changes in the relation between H-reflex amplitude and EMG, which occur in going from the postural state to the locomotor state (Capaday and Stein 1986, 1987a), may be due to changes in the level of presynaptic inhibition of the Ia-terminals.

Alternatively, if the CNS could control the amount of postsynaptic inhibition produced in the motoneurons of the subliminal fringe, independently of that produced in the active population of motoneurons, then this would have the same control action as presynaptic inhibition. However, the range of effectiveness of this mechanism is more restricted, because it can only modulate the response of the neurons in the subliminal fringe and not those in the active population. Furthermore, this hypothetical mechanism would require that the CNS distinguish between motoneurons in the active population and those in the subliminal fringe which seems rather unlikely.

Finally, it may be possible to change the relation between monosynaptic reflex output and recruitment level if the CNS could change the recruitment profile of the motoneuron pool. Descending pathways exist for the selective activation of slow or fast motor units (Kanda et al. 1977; Burke et al. 1970) and such pathways may be involved in producing different recruitment profiles of the motor pool (see for example Nardone and Schieppati 1988). However, it remains to be shown to what extent such potential mechanisms may be involved in the modulation of reflex responses during natural motor activities.

The present experiments and subsequent discussion have dealt with the influence of postsynaptic inhibition on the electrically induced monosynaptic reflex, a highly synchronized response of the motor pool to an excitatory input. The conclusion derived from this study should also be applicable to the repetitive firing of motoneurons. For example, during tonic stretch of a muscle the contribution of the Ia-afferent input to the net firing rate of the motoneurons could only be controlled, independently of the level of motor output, by changes in the level of presynaptic inhibition of the Ia-terminals. Control of the effects of different converging excitatory pathways on the firing rates and recruitment level of postsynaptic neurons may be done in the same way in other parts of the nervous system.

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