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RECIPROCAL Ia INHIBITION BETWEEN ELBOW FLEXORS AND EXTENSORS IN THE HUMAN

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SUMMARY

1. Reciprocal inhibition between elbow flexor and extensor muscles (biceps and triceps brachii) has been investigated in nine healthy subjects. Two techniques were used to assess changes in motoneurone excitability after stimulation of antagonist muscle afferents: (1) monosynaptic reflexes elicited by a mechanical stimulation of the distal muscle tendon (tendon tap); (2) post-stimulus time histograms (PSTH) of voluntarily activated motor units.

2. Electrical stimulation of the antagonist muscle nerve produced a short-latency and short-lasting inhibition of the flexor and extensor motoneurones. The amount of this inhibition was found to be similar in both motor nuclei.

3. The inhibition could be evoked with conditioning electrical stimuli as low as 0·7 × motor threshold (MT) or by very weak tendon taps applied to the antagonist tendon. In the former case the threshold of this inhibition was found to be consistently increased after raising the threshold of Ia afferent fibres by a long-lasting muscle vibration. Since a contribution from cutaneous afferent fibres was ruled out, it is concluded that this inhibition was Ia in origin.

4. Post-stimulus time histograms of voluntarily activated triceps and biceps motor units were made following electrical stimulation of homonymous and antagonist muscle afferents. This enabled an estimate of the central synaptic delay of the inhibitory process. An average central delay of 0·94 ms in excess of that of monosynaptic facilitation was found, thus suggesting that the inhibitory process could be mediated by only one interneurone.

5. A conditioning reflex discharge elicited in the antagonist muscle by a tendon tap depressed or suppressed this inhibition. This depression was maximal when the reflex discharge was elicited 10–20 ms before the conditioning stimulus for the inhibition and never lasted more than 30 ms. It is argued that the only mechanism compatible with such a depression is the inhibitory activity of Renshaw cells acting on the pathway mediating reciprocal inhibition.

6. We conclude that group Ia afferent fibres from elbow extensor and flexor

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muscles project monosynaptically onto Ia inhibitory interneurones to mediate disynaptic reciprocal inhibition of antagonist motoneurones.

INTRODUCTION

The active inhibition of antagonist muscles during the contraction of an agonist was first stressed by Sherrington (1906), who introduced the concept of reciprocal innervation. Experiments on animals (for references see Baldissera, Hultborn & Illert, 1981) demonstrated that Ia inhibitory interneurones are at least partly responsible for this reciprocal behaviour. These interneurones are powerfully controlled by central descending commands. They are fed by agonist muscle spindle Ia afferents and inhibited only by Renshaw cells belonging to homonymous motoneurones and by Ia interneurones fed by Ia afferents coming from antagonistic muscles (opposite Ia interneurones). In the cat, maximal Ia inhibitory postsynaptic potentials (IPSPs) are generally much larger in flexor motoneurones than in the corresponding extensor motoneurones (Eccles & Lundberg, 1958). In the human, reciprocal inhibition has been studied between ankle flexors and extensors (Mizuno, Tanaka & Yanagisawa, 1971; Tanaka, 1974; Iles, 1986; Crone, Hultborn, Jespersen & Nielsen, 1987; Rossi, Mazzochio & Scarpini, 1988) and between wrist flexors and extensors (Baldissera, Campadelli & Cavallari, 1983; Day, Marsden, Obeso & Rothwell, 1984). In both situations, however, the comparison between the amount of reciprocal inhibition from extensors to flexors and from flexors to extensors is difficult since the experimental conditions are not similar. The tibialis anterior H reflex is rare and small (Tanaka, 1974; Pierrot-Deseilligny, Morin, Bergego & Tankov, 1981; Crone *et al.* 1987) whereas the soleus H reflex is regularly obtained with a sizeable amplitude. At wrist level the extensor carpi radialis H reflex is found only in some subjects (Baldissera *et al.* 1983; Day *et al.* 1984) contrary to what is observed with the flexor carpi radialis H reflex. In addition, the question remains to be answered whether the inhibition seen between antagonistic muscles, at lower or upper limb, is actually a true example of Ia reciprocal inhibition. Indeed it is known that the blocking by Renshaw cells is the only available means to discriminate between non-reciprocal (i.e. via Ib interneurones; Jankowska & McCrea, 1983) and reciprocal Ia inhibition (i.e. via Ia interneurones; Hultborn, Jankowska & Lindström, 1971).

The present experiments in the human are devoted to the study of reciprocal inhibition between elbow flexor and extensor muscles. Evidence is provided that this reciprocal inhibition is actually mediated by Ia interneurones, the activity of which is blocked by orthodromic activation of Renshaw cells.

METHODS

General experimental arrangement

The experiments were performed in nine healthy volunteers from the departmental staff (aged 26–45), all of whom gave their informed consent to the procedure which was approved by the local Ethical Committee. Each of them was tested at least twice and the amount of reciprocal inhibition was systematically compared in four of them.

The subjects were comfortably seated in an armchair. The examined (right) arm was lying on an armrest with a shoulder abduction of about 60 deg; the elbow was semi-flexed and the wrist extended. In all the experiments, the surface electromyogram (EMG) was recorded by two non-polarizable disc electrodes (0·9 cm diameter) placed 1·5 cm apart, about 5 cm above the elbow over the bellies of biceps and triceps brachii.

Test reflexes

Since the H reflex wave of the biceps or triceps brachii muscles is not clearly distinguishable from the direct motor response (Bratzlavski, Isch & Van der Eecken, 1971), the excitability of their motoneuronal pool was tested by eliciting monosynaptic reflexes with a tendon tap. Triceps and biceps brachii tendon reflexes are both easily obtained in humans and their amplitude and frequency of occurrence are similar (Cavallari & Katz, 1989). The tap was applied to the distal tendon of the muscle by an electromagnetic hammer (Brüel and Kjaër model 4809) which produced a very quick transient stretch (8 mm in 5 ms). The intensity of the percussion was graded using a power amplifier and expressed in multiples of the threshold for the tendon-jerk reflex ($\times$TT). Special care was taken to ensure that the hammer struck the tendon at the same position throughout the experiment.

To make valid comparison of reflex size among different motor nuclei the test reflex was measured as the peak-to-peak amplitude of the muscle action potentials and expressed as a percentage of the maximal motor response (M_{max}) obtained by the stimulation of all the motor axons of a given motor nucleus. Biceps and triceps brachii are innervated by several deeply located nerve branches and even using the maximal intensity delivered by the stimulator to evoke the M_{max} response it was nevertheless possible that not all the nerve branches were stimulated. This may have led to an underestimation of the amplitude of the M_{max} response and therefore to an overestimation of the size of the test reflexes, expressed as a percentage of M_{max}. The reflexes were chosen to be large enough so that inhibition as well as facilitation could be clearly seen. After amplification, the reflex responses were computer-analysed on-line and the results stored on disc for further analysis.

Conditioning stimuli

Different kinds of conditioning stimuli were used.

(1). Electrical pulses of 1 ms duration were delivered percutaneously to the musculo-cutaneous nerve and to the radial branches innervating the triceps muscle (triceps nerve). The stimulating electrodes were placed a few centimetres below the shoulder and it was carefully checked that even when using the maximal intensity delivered by the stimulators, there was no encroachment of other nerves. Since the stimulating electrodes were located over the belly of the biceps and the triceps muscles, the electrical stimulations might result not only in the excitation of the nerve branches but also in a local muscle stimulation. Increasing the intensity of a local muscle stimulation results in a contraction of only a limited part of the muscle while increasing the intensity of the nerve stimulation resulted in the contraction of the whole muscle. In each case the site of the stimulation was chosen so that increasing the stimulation strength above motor threshold resulted in a steep increase in the motor response and in a contraction involving the whole muscle, as verified by tendon palpation. Furthermore, in each case, the efficiency of this conditioning stimulus to excite Ia fibres was verified by its ability to induce an increase in firing probability of a homonymous voluntary activated motor unit (see below).

The current delivered by the stimulators was measured by a current probe (Tektronix 6021) and the stimulus intensity was expressed in multiples of the threshold intensity for the direct motor wave ($\times$MT).

(2). Mechanical stimulation of the distal tendon of the triceps or biceps brachii muscles was applied with the same electromagnetic hammer used to elicit the test reflexes.

(3). Since in some cases the electrical conditioning stimuli produced a cutaneous sensation, special experiments were designed to mimic this sensation. This was provided by electrical stimulation of the skin near the site of the conditioning electrodes.

(4). In some experiments the electrical conditioning stimulus was delivered to the nerve branches after a long-lasting mechanical vibration (166 Hz for 25 min), applied to the distal tendon of the biceps or triceps muscle, a manoeuvre that selectively increases the electrical threshold of the Ia afferents, as described in the cat (Coppin, Jack & McLennan, 1970), while the Ia receptor

mechanical threshold to muscle stretch remains unimpaired (Fetz, Jankowska, Johannisson & Lipski, 1979). In humans, it has been shown that after such a prolonged tendon vibration, electrical stimulation of the posterior tibial nerve is markedly less effective in eliciting monosynaptic H reflexes in the soleus muscle (Heckman, Condon, Hutton & Enoka, 1984).

Study of single motor units

The effect of a conditioning stimulus on a voluntarily activated motor unit can be determined by constructing a time histogram of the occurrence of motor unit spikes following repeated presentation of the stimulus. The PSTH extracts from the naturally occurring spike train only those changes in firing probability which are time-locked to the stimulus (Stephens, Usherwood & Garnett, 1976). The validity of the method to detect post synaptic potentials in motoneurones has been established by intracellular recordings (Fetz & Gustafsson, 1983).

A detailed description of the particular method used in this section has been already published (Fournier, Meunier, Pierrot-Deseilligny & Shindo, 1986) and will be only summarized here. The EMG from single motor units of the biceps or triceps brachii was recorded by conventional surface electrodes while the subject performed a very weak and steady contraction. After several training sessions it was possible to isolate one motor unit, either because it was the only one active or because it was larger than the others. This was achieved using auditory and visual feedback of the EMG potentials. In all the subjects thus examined the contraction strength was below 5% of maximal voluntary force. The motor units studied were therefore all in the low-threshold range. The EMG potentials of single motor units were converted into standard pulses by a discriminator with variable trigger levels and were used to trigger first a computer (Apple II) and then the stimulators delivering nerve stimulations. The motor unit potential and the trigger pulse were continuously monitored to detect false triggers due to other active units and to ensure that the motor unit shape and trigger position remained constant within and between sequences.

When the nerve stimulation is delivered at a fixed interval after the previous motor unit discharge, it is possible to choose a delay at which the probability for a new discharge is high (provided that there is a relatively regular firing frequency during voluntary contraction). This implies that the number of triggers necessary to reveal obvious peaks in the PSTH can be much lower than if nerve stimulation is given without reference to the motor unit discharge. The conditioning stimulation of the nerves was therefore triggered at a fixed delay after the preceding voluntary motor unit discharge (Fournier *et al.* 1986). The latency of the motor unit potential following the conditioning stimulus was then computed. The PSTHs of the motor unit discharge were constructed for the period 10–70 ms following the stimulation using bins of 0·4 and 1 ms. This method also implies that the probability of discharge in the PSTH depends not only on the postsynaptic potentials evoked by the stimulation, but also on the motoneurone membrane trajectory during the interspike interval. To take account of the latter, a histogram of firing probability was also constructed in a control situation without stimulation. The control and the different conditioning situations were randomly alternated (same number of triggers) within a sequence. The control histogram represented the background firing probability to which the results following stimulation were compared. To clarify the differences between the results obtained in the two situations, the control value in each bin was subtracted from that obtained after stimulation. Within different time interval windows a χ^2 test was used to determine to what extent the distribution of firing probability after stimulation differed from that obtained in the control situation. Such an analysis was only performed after having checked that in the control situation the firing probability within the window of analysis did not differ from the mean probability of discharge of this unit. The onset of the changes in firing probability following the conditioning stimulation was visually identified. It was then required (1) that the number of counts in the bins following the onset was significantly greater (for excitation: Fig. 3*A*, *B*, *E* and *F*) or less (for inhibition: Fig. 3*C*, *D*, *G* and *H*) than the control values and (2) that the number of counts in the bins preceding this onset did not differ significantly with and without stimulation. To perform the statistical analysis the duration of the bin window was increased by summing individual bins of 0·4 ms so that there were at least 5 counts (validity conditions for χ^2 test). When the conditioning stimulus inhibited the discharge so completely that there were < 5 counts in the entire period of inhibition (Fig. 3*C*), no statistics were considered necessary to prove that the change was significant.

Method of estimating the central inhibitory delay

Day *et al.* (1984) have described a method of estimating the central inhibitory delay of reciprocal inhibition between wrist flexors and extensors in the human. They have used an H reflex technique and their method is based on the assumption that the same afferent fibres are responsible for homonymous facilitation and reciprocal inhibition. Using 0·4 ms bins to get adequate precision in the determination of the onset of increase and decrease in firing probability we have applied a similar method with PSTH experiments.

The latency of the onset of the increase in firing probability following homonymous nerve stimulation is the sum of (1) the afferent conduction time in Ia fibres, (2) the efferent conduction time in the studied motor unit, (3) the monosynaptic central delay. The latency of the onset of the decrease in firing probability following antagonistic nerve stimulation is the sum of (1) the afferent conduction time in Ia fibres, (2) the efferent conduction time in the studied motor unit, and (3) the synaptic central delay.

Homonymous and antagonistic effects were studied in the same biceps (or triceps) motor unit, so that the efferent conduction time was the same in both cases for a given motor unit. The characteristics of the conditioning stimuli (localization, intensity) were identical whatever the motor unit studied, so that the afferent conduction times were the same for the biceps and the triceps motor units.

In such conditions, calculations for the central delay of reciprocal inhibition could be made as follows.

(1) Latencies of the onset of the homonymous increase in firing probability for the biceps motor unit (L_{FB}) and for the triceps motor unit (L_{FT}):

$$L_{FB} = X+M+Y, \tag{1}$$

where X = biceps Ia fibre afferent conduction time, M = monosynaptic delay and Y = biceps motor unit efferent conduction time.

$$L_{FT} = V+M+W, \tag{2}$$

where V = triceps Ia fibre afferent conduction time and W = triceps motor unit efferent conduction time.

(2) Latencies of the onset of the antagonistic decrease in firing probability for the biceps motor unit (L_{IB}) and for the triceps motor unit (L_{IT})

$$L_{IB} = V+M+T_s+Y, \tag{3}$$

where T_s = central delay in excess of the monosynaptic delay.

$$L_{IT} = X+M+T_s+W. \tag{4}$$

(3) Calculation of the central delay of reciprocal inhibition (T_s) was performed by

(i) Subtracting eqn (3) from eqn (1):

$$L_{FB}-L_{IB} = X-V-T_s. \tag{5}$$

(ii) Subtracting eqn (4) from eqn (2):

$$L_{FT}-L_{IT} = V-X-T_s. \tag{6}$$

(iii) Adding eqns (5) and (6)

$$2\,T_s = (L_{FB}-L_{IB})+(L_{FT}-L_{IT}).$$

(iv) Therefore the central delay of the reciprocal inhibition in excess to the monosynaptic one is:

$$T_s = (L_{FB}-L_{IB}+L_{FT}-L_{IT})/2.$$

Experimental protocol and analysis of results

In each experimental run unconditioned and conditioned tendon reflexes were randomly alternated. Two different protocols were used: (1) the intensity of the conditioning stimulus was varied keeping the conditioning–test interval constant, (2) the intensity of the conditioning stimulus was kept constant while the conditioning–test interval was varied.

By convention, the timing of the pulse triggering the tap (test stimulus) is referred to the timing of the conditioning electrical shock. Because of the delay introduced by the electromagnetic coupling of the hammer, and because of the peripheral delays associated with spindle activation and afferent conduction to the stimulation site, simultaneous arrival of the conditioning and test volleys in the spinal cord was obtained when the conditioning stimulus was delivered after the tap trigger. In such a case the conditioning–test interval was said to be negative. A total of twenty control and twenty conditioned reflexes were presented in each sequence. Variance analysis was used for testing the significance of the changes in the test reflex amplitudes.

RESULTS

Time courses of the inhibition between elbow flexors and extensors

Figure 1*A* illustrates the time course of the inhibition of a biceps brachii tendon reflex (control value 17% of M_{max}) following electrical conditioning stimuli at 1·0 × MT applied to the triceps nerve. In this example the inhibition was apparent for a conditioning–test time interval of −5 ms, it reached a peak at −3·5 ms and ended at −1 ms. When the inhibition was maximal, the test reflex amplitude was 57% of its control value. This time course was confirmed in eighteen experiments performed in six subjects. In 30% of these experiments a slight facilitation took place at the end of the inhibition between −1 and +1 ms conditioning–test intervals. The test reflex amplitude at the peak of inhibition ranged between 45 and 80% of its unconditioned value (mean value 69·7 ± 8·8%).

Figure 1*B* shows the time course of the changes in a triceps brachii tendon reflex (control value 20% of M_{max}) when conditioned by stimulation of the musculocutaneous nerve at an intensity of 1·0 × MT. This time course was obtained in the same subject as the time course illustrated in Fig. 1*A*. The inhibition of the triceps reflex started for a conditioning–test interval of −5 ms, peaked at −4 ms (67% of its control value), then declined progressively and was replaced at −2 ms by a slight facilitation. This time course was confirmed in thirteen experiments performed in six subjects and the slight facilitation took place in 60% of the cases. The test reflex amplitude at the peak of inhibition ranged between 65 and 75% of its unconditioned value (mean value 72 ± 3·9%).

In similar conditions, i.e. with a conditioning stimulus at 1·0 × MT in each case and unconditioned test reflexes of the same size (15–25% of M_{max}) there was no significant difference in the amount of inhibition between elbow flexors and extensors.

Determination of the afferent fibres responsible for this early inhibition

Electrical conditioning stimuli at 1·0 × MT certainly involve Ia and Ib fibres. Experiments were therefore performed to activate (or inactivate) Ia fibres selectively. Furthermore since the activation of cutaneous fibres cannot be excluded, experiments involving cutaneous fibres only were also performed.

The effects induced on the test reflex by an electrical conditioning stimulus were compared to those obtained with a slight mechanical tap as conditioning stimulus (0·50 × TT), a stimulus which, at rest, activates primary spindle endings but not Golgi tendon organs (Lundberg & Winsbury, 1960). Eight experiments (including at least 320 test reflexes) were performed in four subjects. In Fig. 2 the changes in the

biceps test reflex amplitude following electrical (*A*) and mechanical (*B*) conditioning stimuli, obtained from the same subject during the same experiment, are compared. When the conditioning stimulus was a tendon tap, the inhibition of the test biceps reflex was apparent for a conditioning–test interval of −3 ms. It peaked at 0 ms and

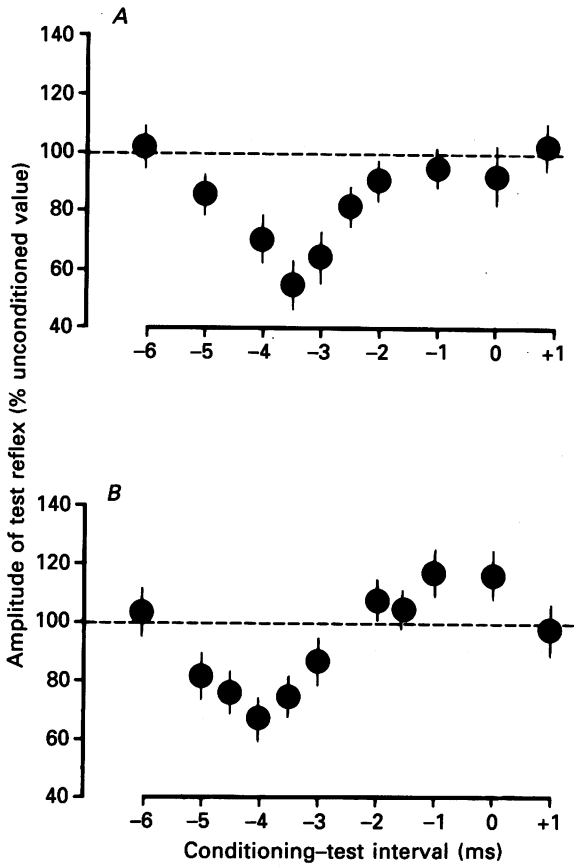

Fig. 1. Time courses of the variations of elbow muscle tendon reflexes following antagonistic electrical nerve stimulation. *A*, the test reflex is evoked in the biceps brachii. The conditioning stimulation is applied to the triceps nerve (intensity 1·0 × MT). Ordinate, amplitude of the test reflex expressed as a percentage of this unconditioned value. Abscissa, time interval between test and conditioning stimuli. By convention the timing of the test stimulation is referred to that of the conditioning one. Negative time intervals correspond to the cases in which the test stimulus was given first. *B*, test reflex evoked in the triceps brachii, conditioning electrical stimulus applied to the musculocutaneous nerve (intensity 1·0 × MT). Same co-ordinates as in *A*. Each symbol represents the mean of sixty measurements. Vertical bars, 1 standard error of the mean.

vanished a few milliseconds thereafter. Therefore in both cases, with an electrical conditioning stimulus and with a conditioning tendon tap, there was an early inhibition. By contrast, contrary to what was observed following an electrical conditioning stimulus (Fig. 2*A*), no late facilitation was observed with a conditioning

tendon tap and such a result was obtained in all the experiments with a conditioning mechanical tap. In the reverse situation (i.e. with a test triceps reflex and a biceps conditioning tap, not illustrated), the results were similar.

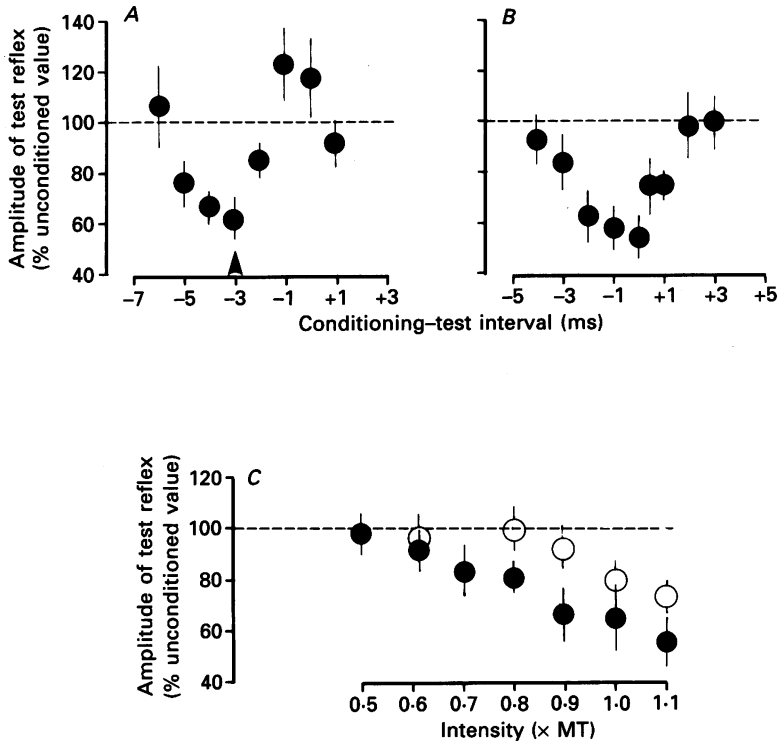

Fig. 2. Changes in the amplitude of the biceps test reflex following various antagonistic conditioning stimuli. *A*, time course of the inhibition of the biceps test reflex following a conditioning electrical stimulation applied to the triceps nerve (intensity 1·0 × MT) obtained in another subject than in Fig. 1. Same co-ordinates as in Fig. 1. *B*, time course of the inhibition of the biceps test reflex induced by a mechanical tap applied to the distal triceps tendon (intensity 0·50 × TT). Same co-ordinates as in *A*. Time courses represented in *A* and *B* were obtained the same day in the same subject. *C*, changes in the amplitude of the biceps test reflex following changes in the intensity of the conditioning stimulus at a fixed conditioning–test interval (−3 ms; arrow in Fig. *A*). ●, variations obtained in control conditions. ○, variations obtained during a selective increase of the electrical threshold of the conditioning Ia fibres. Each symbol represents the mean of twenty measurements.

In all the cases test and conditioning mechanical tendon taps were evoked at the elbow, roughly at the same distance from the spinal cord. The peripheral delays associated with mechanical stimulation should be the same for the conditioning and the test stimulus. In such conditions, the latency of the onset of the inhibition, −3 ms, could seem surprisingly early. This apparent paradox can be explained by the findings of Araki, Eccles & Ito (1960). They have demonstrated that the earliest time at which an inhibitory postsynaptic potential (IPSP) may decrease a reflex discharge corresponds to the moment at which an IPSP is able to suppress the *last* spikes of the desynchronized motoneuronal reflex discharge. Pierrot-Deseilligny *et al.* (1981) have already described this phenomenon with electrically evoked reflexes. The temporal dispersion in the tendon

reflexes is more marked than that in the electrically evoked reflexes, as shown in humans by Burke, Gandevia & McKeon (1984). Therefore the time interval during which the conditioning volley may interfere with the test volley when it arrives later in the spinal cord must be longer with tendon reflexes.

In order to further characterize the afferent fibres responsible for this reciprocal inhibition, the effects of varying the intensity of the electrical stimulation were studied in two experimental conditions: (1) before, and (2) after a long-lasting vibration which is known to increase the threshold for the electrical activation of Ia afferent fibres (see Methods). Eight experiments (including at least 160 test reflexes in each experimental situation) were performed in three subjects. Figure 2*C* represents the results obtained at a conditioning–test interval of −3 ms (arrow on Fig. 2*A*, peak of the inhibition). The strength of the conditioning stimulus was increased by steps of 0·10 × MT. Before vibration (●) the progressive decrease of the biceps test reflex amplitude started at conditioning intensity below 0·60 × MT. After vibration (○), the threshold of the inhibition of the test reflex was enhanced up to 0·90 × MT. It was verified that 20 min after the end of the long-lasting vibration, the threshold of the test reflex inhibition returned to its control value. In the reverse situation, i.e. with a triceps test reflex, the results were the same.

Electrical conditioning stimuli applied to the musculo-cutaneous nerve or to the triceps nerve did not usually result in paraesthesiae and, when they occurred, the induced local sensation was very slight. Control experiments, however, were performed with a purely cutaneous conditioning stimulation mimicking the sensation evoked by the conditioning stimuli. Whatever the test reflex (biceps or triceps), no significant changes in its amplitude were seen following such a cutaneous stimulation applied on the arm 2–3 cm from the nerve trajectory.

Response of individual motor units

The modifications of the firing probability of voluntarily activated motor units of the biceps (or the triceps) brachii muscle were studied following electrical conditioning stimuli (intensity 1·0 × MT) applied to the homonymous and to the antagonistic nerves.

In Fig. 3*A*, *C*, *E* and *G* open bars represent the control histograms obtained without stimulation while shaded bars represent the histograms obtained following stimulation. Figure 3*B*, *D*, *F*, and *H* represents the differences between these two situations.

The early increases in firing probability following homonymous stimulation are shown in *B* for the biceps motor unit and in *F* for the triceps motor unit whereas the early decreases following antagonistic stimulation are shown in *D* (biceps motor unit) and in *H* (triceps motor unit). The possibility that cutaneous fibres may have contributed to these modifications in firing probability was ruled out using the same complementary experiments as those described in the preceding section: no modification of firing probability was found following pure cutaneous stimulation. Previous results (Cavallari & Katz, 1989) indicate that the onset of the increase in firing probability following homonymous facilitation is due to the monosynaptic Ia input. Forty-six biceps motor units were studied in four subjects; a decrease in firing probability, as illustrated in Fig. 3*D*, was observed in 51 % of the cases. No decrease

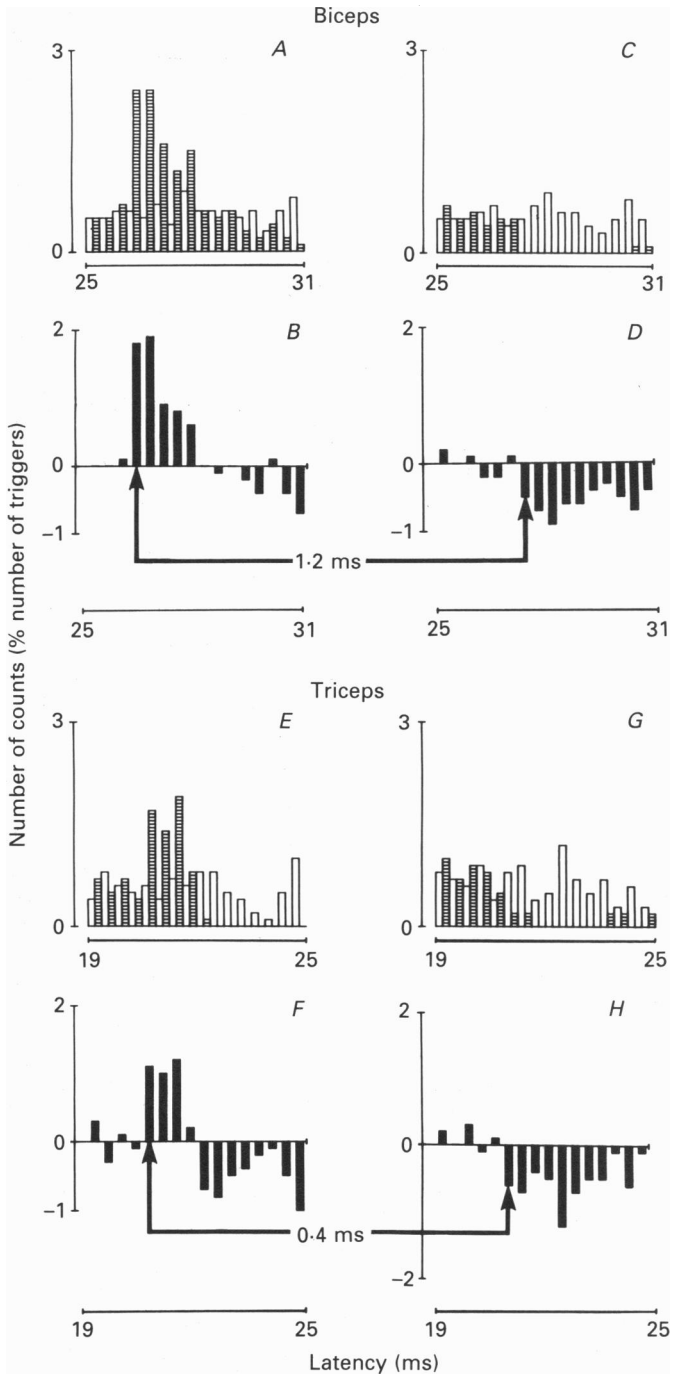

Fig. 3. Comparison between the latencies of the increase in firing probability in response to homonymous nerve stimulation (left) and of the decrease in firing probability in response to antagonistic nerve stimulation (right). *A–D*, time histograms of the discharge of a voluntarily activated biceps motor unit. *E–H*, time histograms of the discharge of a voluntarily activated triceps motor unit. In *A*, *C*, *E* and *G* open bars represent the

in firing probability was therefore observed in 49% of the cases despite the fact that the efficiency of the conditioning stimulus was checked in all cases by studying the homonymous facilitation (Fig. 3*F*). A decrease in firing probability, as illustrated in Fig. 3*H*, was observed in thirty-two out of the sixty-four triceps motor units studied in seven subjects; no modification of firing probability following antagonistic stimulations was therefore seen in half the cases. In both cases the duration of the decrease in firing probability was short, 3·6 ms on average (range 1·6–7·6 ms). The inhibition following the Ia homonymous monosynaptic peak is probably due to the effects of after-hyperpolarization. However, if the homonymous conditioning stimulus had resulted from time to time in a monosynaptic reflex, recurrent inhibition would also have contributed to this late inhibition (Meunier, Penicaud, Pierrot-Deseilligny & Rossi, 1990).

The results illustrated in Fig. 3 were obtained in the same experiment and the same conditioning musculo-cutaneous (or triceps) nerve stimulation was used to study the homonymous and the antagonistic effects. The central delay of the inhibitory pathway was estimated following the protocol described in Methods. The experiments leading to this estimation were technically difficult since they required the subject to keep the same motor unit for the determination of the onset of the homonymous increase and of the antagonist decrease in firing probability and to activate successively a biceps and a triceps motor unit. They also required the onset of the changes in firing probability to be sufficiently abrupt that they could be clearly seen and the duration of the bins to be short enough to distinguish between one or two synapses. Therefore bins of 0·4 ms have been used to estimate the latency of the onset of the changes in firing probability. In the left part of Fig. 3 the onsets of the homonymous peaks are abrupt and their duration short. Statistical analysis of changes in firing probability (see Methods) were nevertheless performed in the time

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histograms obtained in control condition (i.e. without stimulation) while shaded bars represent the histograms obtained in response to nerve stimulations. *B*, *D*, *F*, and *H*, difference between the above two histograms; in each 0·4 ms bin the control value (open bars) was subtracted from that obtained after stimulation (shaded bar). The vertical arrows indicate the bin corresponding to the onset of the increase (*B*, *F*) or the decrease (*D*, *H*) in firing probability. This bin was visually identified and statistical analyses were performed to determine to what extent the distribution of firing probability after stimulation differed from that in control situation. In the time interval 25–31 ms for the biceps motor unit and 19–25 ms for the triceps motor unit, a χ^2 test was performed; for the PSTHs represented in *A* and *B* no significant differences between control situation and after stimulation were seen in the 25, 25·4 and 25·8 bins ($\chi^2 = 0{\cdot}2$) whereas the increases in 26·2 and 26·6 ms bins were highly significant ($\chi^2 = 12{\cdot}8$; $P < 0{\cdot}001$). For the PSTHs represented in *E* and *F* no significant difference between control situation and after stimulation was seen in 19, 19·4, 19·8 and 20·2 ms bin. ($\chi^2 = 0{\cdot}84$) whereas the increases in 20·6, 21 and 21·4 ms bins were each significant ($\chi^2 = 6$; $P < 0{\cdot}05$). Similar calculations were made for PSTHs represented in *C* and *D*: no significant differences between control situation and after stimulation were seen in the 25, 25·4, 25·8, 26·2, 26·6 and 27 ms bins ($\chi^2 = 0{\cdot}81$) whereas the decreases in the 27·4–29 ms windows were so profound that the χ^2 test could not be performed (see Methods); and for PSTHs represented in *G* and *H*: no significant differences were seen in the 19, 19·4, 19·8, 20·2 and 20·6 ms bins ($\chi^2 = 0{\cdot}48$) whereas the decrease in the 21–21·8 window was highly significant ($\chi^2 = 13{\cdot}44$; $P < 0{\cdot}001$). Number of triggers, 1000. Ordinate, number of counts expressed as a percentage of the number of triggers. Abscissa, latency after stimulation (0·400 ms bin). The same conditioning stimulation was used in *A* (and therefore in *B*) and *G* (and therefore in *H*); and in *C* (and therefore in *D*) and *E* (and therefore in *F*).

interval window surrounding the peak; in the three bins preceding its onset (arrow in Fig. 3*B* and *F*) it was verified using a χ^2 test within each 0·4 bin, that the distribution of firing probability after stimulation (shaded bars in Fig. 3*A* and *E*) did not differ from that in the control situation (open bars in Fig. 3*A* and *E*) while the probability was significantly increased during the peak and that its first bin was

TABLE 1. Determination of the central delay of the reciprocal inhibition between elbow flexors and extensors. Using 0·400 ms bins, the onset of the monosynaptic increase in firing probability and that of reciprocal inhibition was determined as the first bin in which the PSTH obtained in control conditions differs significantly from that following stimulation. The central delay in excess of the monosynaptic delay was calculated using the procedure described in the Methods section

	Triceps motor units			Biceps motor units			
Expt	Monosynaptic peak latency (ms)	Reciprocal inhibition latency (ms)	Difference (ms)	Monosynaptic peak latency (ms)	Reciprocal inhibition latency (ms)	Difference (ms)	Central delay (ms)
1	20·8	21·2	0·4	27·8	29·4	1·6	1
2	21·8	22·6	0·8	27·2	29·2	2	1·4
3	22·8	23·2	0·4	27·2	28·8	1·6	1
4	21	21·4	0·4	27·8	28·2	0·4	0·4
5	20·6	23·4	0·8	27	28·4	1·4	1
6	20·6	21·4	0·8	25·4	26·6	1·2	1
7	20·6	21	0·4	26·2	27·4	1·2	0·8

significant ($0{\cdot}05 < P < 0{\cdot}001$). In the right part of Fig. 3 are represented the decreases in firing probability following antagonistic nerve stimulation. The onsets of the decreases are less obvious than the onsets of increases in firing probability despite the fact that the number of triggers was increased to produce a stable firing probability under control conditions (open bars in Fig. 3*C* and *G*). Taking into account that the sites of the homonymous and antagonistic stimulation were roughly at the same distance from the spinal cord, statistical analysis of changes in firing probability were confined to the time interval surrounding the onset of the homonymous increase in firing probability. The onset of the antagonistic decrease in firing probability was therefore visually identified and it was then verified that (a) there was no difference in the distribution of firing probability between control (open bars in *C* and *G*) and following stimulation (shaded bars in *C* and *G*) in each of the five bins preceding the arrows in Fig. 3*D* and *H*; (b) a significant decrease was seen in the subsequent bin windows including the one indicated by the arrows.

The onset of facilitation was 26·2 ms for the biceps motor unit and 20·6 ms for the triceps motor unit. The onset of inhibition was 27·4 ms for the biceps motor unit and 21 ms for the triceps motor unit. The differences between the homonymous and the antagonistic effects are respectively 1·2 and 0·4 ms. The central delay, estimated as described in Methods, is 0·8 ms longer than the monosynaptic effect. Only seven successful experiments were performed (Table 1). The extreme values of the central delay were 0·4 and 1·4 ms, with an average of 0·94 ms (to these values must be added a possible measurement error, equal to one bin window ($\pm 0{\cdot}4$ ms), so that the actual possible range of central delays was 0–1·8 ms). The mean value (0·94 ms) suggests that the inhibition was mediated through one interneurone.

Synaptic linkage responsible for this inhibition

As stated in the Introduction, Ia inhibitory interneurones are only inhibited by Renshaw cells and opposite Ia interneurones (Hultborn *et al.* 1971) (Fig. 4*A*). The following experimental protocol was set up to verify that the reciprocal inhibition

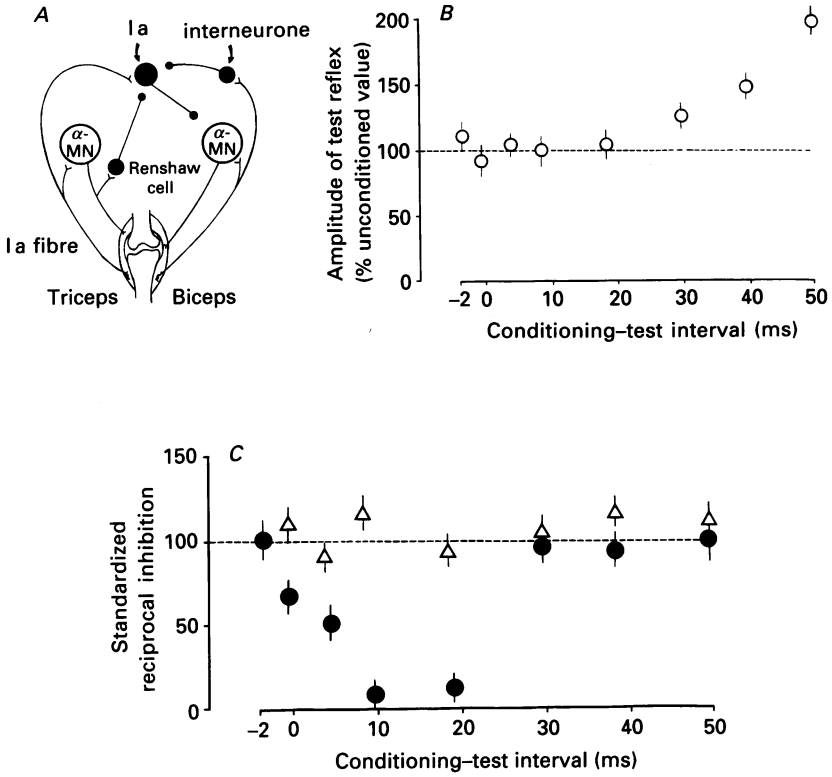

Fig. 4. Changes in reciprocal inhibition induced by a preceding reflex discharge. *A*, schematic representation of the connections between Ia interneurones and Renshaw cells (after Hultborn *et al.* 1981): large filled circle represents the Ia interneurone fed by triceps Ia fibres, inhibited by Renshaw cells belonging to the triceps motoneurone pool and inhibiting the biceps α-motoneurones. *B*, time course of the variations of the biceps test reflex induced by a tendon reflex discharge evoked in the triceps. Ordinate, amplitude of the biceps test reflex expressed as a percentage of its unconditioned value. Abscissa, time interval between test and conditioning stimuli. *C*, time course of the modification of reciprocal inhibition induced by the mechanical conditioning stimulus: the test reflex was evoked in the biceps brachii, a conditioning electrical stimulus applied to the triceps nerve gave rise to the reciprocal inhibition, and the Renshaw cells were activated by a triceps reflex discharge. Ordinate, amount of reciprocal inhibition – conditioned by the antagonistic tendon reflex – expressed as a ratio of its unconditioned value and standardized in order to have $y = 100\%$ (dashed line) when the reciprocal inhibition was not modified by the conditioning tendon reflex and $y = 0$ when the reciprocal inhibition was completely blocked. Abscissa, time interval between the test stimulation and the mechanical conditioning stimulus. ●, results obtained when the mechanical stimulus evoked a triceps test reflex. △, results obtained when the mechanical stimulus was just below tendon reflex threshold.

described above was affected by the activation of Renshaw cells. The test being the biceps tendon reflex, two conditioning stimuli were used: (1) the first was a mechanical tap applied to the distal tendon of triceps to produce a triceps monosynaptic reflex able to activate Renshaw cells (Hultborn, Pierrot-Deseilligny & Wigström, 1979); (2) the second was an electrical stimulus (1·0 × MT) applied to the triceps nerve and eliciting the reciprocal inhibition (cf. Fig. 1*A*). The time interval between the first and the second conditioning stimuli was systematically varied between 0 and 50 ms, while that between the test and the second conditioning stimulus was kept constant. This latter interval was chosen so that the reciprocal inhibition was at its peak. The first conditioning stimulus elicited a triceps tendon reflex and therefore resulted in orthodromic activation of some triceps-coupled Renshaw cells. It is obvious that this activation of triceps-coupled Renshaw cells was accompanied by activation of other pathways: (1) activation of triceps group I afferents; (2) possible activation of cutaneous afferents from the skin where the mechanical tap was applied; (3) activation of some triceps motoneurones and the consequent afferent discharge due to the triceps reflex discharge; (4) possible activation of biceps spindle endings by spread of the vibration wave (Katz, Morin, Pierrot-Deseilligny & Hibino, 1977). Such activation of various pathways could modify the effects induced by the second conditioning and the test stimuli. To be able to distinguish the effects of the activation of the triceps-coupled Renshaw cells on reciprocal inhibition from among all the effects induced by the first conditioning stimulus, the experimental protocol was as follows: the effects of the first conditioning stimulus alone were studied on the test reflex (Fig. 4*B*) and compared to those obtained when the two conditioning stimuli were applied. Since it has been shown (Crone, Hultborn, Mazières, Morin, Nielsen, Pierrot-Deseilligny, 1990) that the amount of inhibition also depends on the test reflex amplitude '*per se*', the intensity of the test stimulus was adjusted so that amplitude of the test reflex conditioned by the mechanical tap was in the same range as that of the test reflex alone. The amount of unconditioned reciprocal inhibition was determined by studying the effects of the second conditioning stimulus on the test reflex alone. Four configurations were therefore randomly alternated and the stimulus protocol was as follows: (a) twenty test reflexes alone; (b) twenty test reflexes + first conditioning stimulus; (c) twenty test reflexes + second conditioning stimulus; (d) twenty test reflexes + the two conditioning stimuli.

An example of the results is illustrated in Fig. 4*C*. The amount of reciprocal inhibition in control conditions (i.e. the difference between the amplitude of the test reflex evoked alone and that obtained following the electrical conditioning stimulus applied to the triceps nerve at a conditioning test interval of −3·5 ms) is represented by the horizontal dashed line. It corresponds to an inhibition of 27 %. Filled circles represent the modifications of the amount of reciprocal inhibition induced by the mechanical tap applied to the triceps tendon. A reduction of reciprocal inhibition was apparent at a time interval between the test and this mechanical tap of 0 ms, then progressively increased and at time intervals of +10 and +20 ms, the reciprocal inhibition had almost completely disappeared. For time intervals longer than +20 ms, the reciprocal inhibition reappeared, regained its control value at +30 ms and kept it for longer time intervals. Such a time course was found in the ten

experiments performed in three subjects: in particular, in all of them, after having been strongly depressed or suppressed, reciprocal inhibition always returned to its control value, the only difference among the experiments was the time interval at which it returned to this control value: between 20 and 30 ms or between 30 and 40 ms. Open triangles represent the results obtained when the strength of the first conditioning stimulus (the mechanical tap applied to the triceps tendon) was slightly reduced to be just below tendon reflex threshold. Since only small changes in the strength of the conditioning mechanical tap were necessary to be just below or just above reflex discharge, the afferent volley induced by the mechanical tap was probably similar. However when there is no reflex discharge, there is no activation of Renshaw cells and, as it can be seen in Fig. 4*C*, no modification of reciprocal inhibition was seen under such conditions.

DISCUSSION

In the present experiments a short-latency, short-duration and low-threshold inhibition between elbow flexors and extensors has been described. Its magnitude was the same in flexors and in extensors. This inhibition was studied using a tendon reflex technique and PSTHs of isolated voluntarily activated motor units. It most probably corresponds to the disynaptic reciprocal Ia inhibition described in the cat. In the Discussion the main evidence which supports this conclusion will be presented.

Ia fibres are responsible for this inhibition

Several arguments support this assumption. (a) The reciprocal inhibition could be obtained both with an electrical conditioning stimulus and a slight mechanical conditioning tap. At rest, such a weak stimulus activates muscle spindle primary endings but not Golgi tendon organs (Lundberg & Wisbury, 1960). (b) The electrical threshold of this inhibition was increased after long-lasting muscle vibration. It has been shown that long-lasting vibration results in a selective increase in the electrical threshold of Ia afferents (Coppin *et al.* 1970; Fetz *et al.* 1979; Jankowska & McCrea, 1983; Heckman *et al.* 1984; Cavallari & Katz, 1989).

When two stimulations (two conditioning stimuli, conditioning and test stimuli) involve the same Ia fibres, this may induce a transmitter depletion at the junction between Ia fibres and motoneurones as first described in the cat by Curtis & Eccles (1960) and studied in the human by Katz *et al.* (1977). However the time course of the effects of the transmitter depletion, beginning early after the second stimulus and lasting between 2 and 3 s, rules out its contribution both in the enhancement of the electrical threshold of reciprocal inhibition following long-lasting vibration and in the reduction of reciprocal inhibition evoked by an antagonistic conditioning tendon reflex.

The slight facilitation which in some cases followed the early inhibition, was not found when the conditioning stimulus was a tendon tap. This and its longer latency suggest that this late facilitation is Ib in origin.

Estimation of the central delay of the inhibition

The central delay of this inhibition was calculated using the PSTHs of voluntary activated motor units to estimate the extra time required for afferent impulses to inhibit antagonist motoneurones with respect to that required to excite homonymous motoneurones. The procedure is based on the assumption that the same afferent fibres are responsible for both the monosynaptic homonymous excitation and the antagonist inhibition.

The central delay of inhibition was 0·94 ms longer on average than that of monosynaptic excitation, therefore suggesting that the inhibition could be mediated through only one interneurone.

The inhibition is blocked by recurrent inhibition

In the cat, the Ia inhibitory interneurones are inhibited by Renshaw cells (Hultborn *et al.* 1971) and this feature provides a unique means of identification of reciprocal Ia inhibition. In this study dramatic changes in the inhibition were observed when a conditioning reflex was elicited in the muscle from which the conditioning Ia fibres originated. It is obvious that such a stimulation could modify the effects induced by the second conditioning and test stimuli. The amplitude of the test reflex conditioned by the antagonistic tendon reflex was therefore used as the reference value to estimate the modifications of the reciprocal inhibition induced by this conditioning tendon reflex. This conditioning antagonistic tendon reflex may affect the pathway of reciprocal inhibition (1) by the stimulation of afferent fibres; (2) by the excitation of Renshaw cells via the conditioning reflex discharge since it is obvious that the appearance of this conditioning tendon reflex is due to the discharge of antagonistic motoneurones. The first possibility was tested using a conditioning mechanical tap just below tendon reflex threshold: roughly the same strength and therefore involving roughly the same afferent fibres, but the Renshaw cells were not activated. In such a situation, the reciprocal inhibition was not modified (Fig. 4*C*). Thus the blocking of reciprocal inhibition obtained with a conditioning tendon reflex is likely to be due to recurrent inhibition. It should be noticed that the time course of this blocking (● in Fig. 4*C*) is similar to the time course of recurrent inhibition described in the cat (Renshaw, 1946) and in the human (Bussel & Pierrot-Deseilligny, 1977). This blocking rules out the possibility that the observed inhibition could result from the activation of non-reciprocal Ia pathway, i.e. Ib interneurones fed by Ia fibres (Jankowska & McCrea, 1983), since Renshaw cells are known to inhibit Ia but not Ib interneurones. Therefore, the reciprocal inhibition we described between elbow flexors and extensors is very likely to be disynaptic Ia reciprocal inhibition.

Characteristics of the reciprocal Ia inhibition between elbow flexors and extensors

It should be noticed that contrary to previous results obtained in the cat (Eccles & Lundberg, 1958) and in the human (see Introduction) the amount of reciprocal inhibition was in the same range in flexor and extensor motor nuclei. As already stressed in the Introduction this discrepancy might be due to differences in experimental conditions since, in the human at wrist, the amplitude and frequency of occurrence of monosynaptic reflexes are different in flexor and extensor motor

nuclei. However, such an explanation does not take into account the differences between the cat and the human and differences in the physiological role of human upper limb and cat forelimb which should certainly also play a role in these differences.

Reciprocal inhibition has been largely investigated in the human between ankle flexors and extensors, and between wrist flexors and extensors (for references see Introduction). Evidence was provided that at its onset reciprocal inhibition in humans is mediated via Ia afferents acting through a single inhibitory interneurone, but the type of this interneurone (Ia or Ib) was not established. We provide evidence that reciprocal inhibition between elbow flexors and extensors is mediated via Ia interneurones and our findings are in accordance with the spinal pathways described at elbow joint in the cat (Hahne, Illert & Wietelmann, 1988).

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