J. Physiol. (1984), **355**, *pp.* 587–603
With 5 text-figures
Printed in Great Britain

FACILITATION OF SOLEUS-COUPLED RENSHAW CELLS DURING VOLUNTARY CONTRACTION OF PRETIBIAL FLEXOR MUSCLES IN MAN

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(*Received 17 February 1984*)

SUMMARY

1. Recurrent inhibition to soleus motoneurones, brought about by a conditioning H-reflex discharge, was estimated in human subjects by a subsequent test H reflex. Changes in recurrent inhibition during voluntary ankle dorsiflexion were evaluated by comparing the amplitude of the test H reflex to a reference H reflex: both reflexes were subjected to the same type of influences which modified soleus monosynaptic reflex excitability during pretibial flexor contraction, but only the test H reflex was subject to the recurrent inhibition evoked by the conditioning H-reflex discharge.

2. During tonic or phasic ramp contractions of the pretibial flexors the inhibition of the test H reflex, as compared to rest, was more marked than that of the reference H reflex. Evidence is presented that this may indicate a facilitation of soleus-coupled Renshaw cells. Since this facilitation of soleus-coupled Renshaw cells was also observed before ramp contraction, it is, at least in part, supraspinal in origin.

3. Within the range of forces studied (8–45% of maximum force) there was no evidence that the facilitation of soleus-coupled Renshaw cells increased along with increased force of the pretibial flexor voluntary contraction. During voluntary phasic ankle dorsiflexion, facilitation of soleus-coupled Renshaw cells was maximum at the moment when soleus motoneurones were most facilitated by the stretch-induced soleus Ia discharge. There was no evidence for changes in Renshaw cell excitability during ballistic contractions.

4. It is suggested that this facilitation of soleus-coupled Renshaw cells may be one of the mechanisms preventing the occurrence of a soleus stretch reflex during a voluntary ankle dorsiflexion. Such a mechanism could become important if reciprocal inhibition, via Ia inhibitory interneurones, were not strong enough, e.g. because of a weak γ-drive to the contracting muscles.

INTRODUCTION

Renshaw cells receive excitatory and inhibitory synaptic input from descending tracts (for references, see Baldissera, Hultborn & Illert, 1981), thus allowing higher centres to modulate transmission in the recurrent pathway. Virtually nothing is

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known about the type of motor activity in which this descending control of Renshaw cells is involved. This would require experiments performed during natural movements.

This is possible given a method using a paired H-reflex technique which enables one to study recurrent inhibition directed to soleus motoneurones in man: the test reflex inhibition only depends on the conditioning reflex amplitude, thus suggesting its probable Renshaw origin (Bussel & Pierrot-Deseilligny, 1977). Changes in this inhibition have been observed during gastrocnemius–soleus voluntary contraction and evidence has been presented that these changes may reflect modulations of the excitability of soleus-coupled Renshaw cells: facilitation of Renshaw cells during the weakest contractions, but inhibition of Renshaw cells during the strongest (Hultborn & Pierrot-Deseilligny, 1979*a*).

The present study was undertaken to examine changes in recurrent inhibition directed to soleus motoneurones during voluntary contraction of the antagonistic muscles, i.e. the pretibial flexors. During voluntary tonic and phasic ankle dorsiflexion (but not during ballistic contraction), an increase in the conditioning reflex-induced inhibition of the test reflex was found. Evidence is presented that this may reflect an increased recurrent inhibition to soleus motoneurones. The functional significance of this finding is discussed: it may be one of the mechanisms preventing the occurrence of a stretch reflex in the muscle antagonistic to the contracting one. Some of the preliminary results have been briefly reported (Pierrot-Deseilligny, Morin, Katz & Bussel, 1977).

METHODS

General experimental arrangement. The experiments were carried out on fifteen healthy subjects aged 20–47 years, all of whom gave informed consent to the experimental procedure.

The subjects were comfortably seated in an armchair and the examined leg was mechanically fixed with the knee semi-flexed (120 deg) and the ankle at a right angle. Surface electrodes were used for both stimulation and recording. The soleus H reflex was obtained by stimulating the posterior tibial nerve at the popliteal fossa with rectangular pulses of 1 ms duration, every 4 s. The same unipolar electrode provided the conditioning and test stimuli. The reflex responses were measured in terms of the amplitude of muscle action potentials, recorded by two non-polarizable disk electrodes (0·9 cm diameter) placed 1·5 cm apart on the soleus muscle. A ground electrode was placed between stimulating and recording electrodes. After amplification, the reflex responses were recorded on magnetic tape and analysed by computer.

Voluntary electromyographic (e.m.g.) activity from tibialis anterior was recorded by similar surface electrodes. To ensure that the pretibial flexor voluntary contraction was isolated, the e.m.g. from soleus, gastrocnemius and quadriceps muscles was also recorded and displayed on an oscilloscope. The subjects were trained to keep these muscles silent while performing voluntary dorsiflexion of the foot.

In order to prevent the pretibial flexor contractions from producing an excessive dorsiflexion of the foot, the foot was fastened to a rigid sole by two leather straps, one of which was placed in front of the ankle joint, and the other passed over the toes. This foot-fixation system was not rigid and the contractions were therefore not isometric: pretibial flexor contractions produced a passive stretch of the gastrocnemius–soleus muscle.

Method of estimating recurrent inhibition to soleus motoneurones. The method of exploring recurrent inhibition to soleus motoneurones in man has been previously described in detail (Bussel & Pierrot-Deseilligny, 1977) and its main features will be briefly summarized. This method uses a paired H-reflex technique: a monosynaptic reflex discharge (H1) activates Renshaw cells, and the resulting recurrent inhibition is assessed by a subsequent H reflex, the H′ test reflex. The test volley is supramaximal for α-motor axons, and, because of a special experimental procedure based on a collision in motor axons, all motoneurones responsible for the test reflex recorded in the e.m.g. have

already fired in the H1 conditioning reflex. Since all these motoneurones undergo the post-spike after-hyperpolarization, the tested motoneurone population is homogeneous in this respect. Because of this procedure, the H′ test reflex can be at the very most equal to the H1 conditioning reflex. At low H1 conditioning reflex amplitudes the H′ test reflex remains equal to H1, but further increases in H1 result in a progressive decrease in H′. Provided that the conditioning–test interval is longer than 9 ms, this decrease in the test reflex is only related to the size of the conditioning reflex. This may indicate that the test reflex inhibition is caused by an increased recurrent inhibition, itself elicited by growing conditioning reflexes. The amplitude of the H′ test reflex can therefore be used to estimate the importance of recurrent inhibition elicited by the conditioning reflex.

If the amplitude of the test reflex depends on the recurrent inhibition from H1, it also depends on the background excitability of soleus motoneurones, which is decreased during voluntary ankle dorsiflexion (Hoffmann, 1918). An estimation of this latter factor can be obtained by comparing, during contraction, changes in the H′ test reflex with those in a reference H reflex, i.e. an ordinary H reflex of the same size as H′ at rest (Hultborn & Pierrot-Deseilligny, 1979*a*). Motoneurones responsible for both reflexes are subject to the same descending and segmental influences related to the contraction. If recurrent inhibition from H1 remains constant during contraction, both reflexes should exhibit the same amount of inhibition. A differential net effect on those two reflexes may therefore be ascribed to changes in the recurrent inhibition elicited by the constant H1 conditioning discharge.

Experimental procedure with various pretibial flexor voluntary contractions. The voluntary tibialis anterior e.m.g. activity was recorded on magnetic tape along with the output from a strain gauge fixed on the plate on which the foot was fastened. During phasic *ramp contractions*, the desired time course of the movement was drawn by a line on an oscilloscope screen and the subject was asked to perform contractions which caused the output from the strain gauge to follow this drawn line. Contractions which deviated significantly from the chosen contraction were discarded. The procedure required the subject to start the movement within the second following an acoustic signal. The e.m.g. signal from the tibialis anterior was rectified and connected to a triggering circuit. The e.m.g. onset was then used to trigger stimulations with various time delays. A stimulation could thus be delivered at the very onset of the e.m.g. or at any interval after this onset by introducing variable delay circuits. During *ballistic contractions* the subjects were asked to perform the fastest possible contraction and then to relax immediately (the e.m.g. exhibited a very early peak at 30–40 ms after onset and a rapid decline). Experiments were also performed *before pretibial flexor contractions*: the time separating the test reflex from the tibialis anterior voluntary contraction was measured and only reflexes preceding voluntary contraction by 10–50 ms were retained for further analysis.

During tonic contractions the subjects were asked to match the output from the strain gauge to the chosen strength of the contraction. This strength was expressed as a percentage of the strongest tonic voluntary ankle dorsiflexion of the foot that it was possible to maintain for 15 s. These contractions ranged from 8 to 45 % of this maximum force.

Stimulus protocol. Four kinds of stimuli were alternated: (1) the conditioning stimulus alone, so that the H1 conditioning reflex discharge could be recorded (Fig. 1*A* and *E*); (2) the combined conditioning and (supramaximal) test stimulations (conditioning–test interval 10 ms) which resulted in the H′ test reflex (Fig. 1*B* and *F*); (3) the stimulus eliciting the reference H reflex (Fig. 1*C* and *G*); (4) a stimulus strong enough to activate some α-motor axons, thus causing a small M wave (Fig. 1*D* and *H*), the stability of which was used to ensure that, during contraction, stimulation conditions remained the same as at rest. These different kinds of stimulation were elicited every 4 s and were alternated either regularly or randomly (see below), stimulations being computer triggered. In each session the same number (at least twenty) of each kind of stimulus was presented.

With the method used to study recurrent inhibition it is essential that the H1 conditioning discharge remains constant during contraction. The intensity of the conditioning stimulus was therefore adjusted so that the H1 conditioning reflex size was the same at rest as during contraction, whatever the contraction force or the delay after (or before) the onset of contraction. Only the experiments in which H1 was constant (for a given force or delay) were retained for further analysis. To validly compare the reference H reflex at rest and during contraction it is essential that stimulation conditions are the same. Thus only the experiments in which the small M wave elicited by the fourth stimulus was constant were retained for further analysis.

RESULTS

This method of studying recurrent inhibition in man was first described at rest (Bussel & Pierrot-Deseilligny, 1977), and some of the assumptions on which it is based have been verified in animal experiments (Hultborn, Pierrot-Deseilligny & Wigström, 1979). When it was used to study changes in recurrent inhibition during voluntary plantar flexion, it was verified that: (1) possible differences in sensitivity of the H′ test and reference H reflexes did not invalidate conclusions; (2) the conditioning stimulation was not modifying soleus motoneurone excitability by pathways other than recurrent inhibition (Hultborn & Pierrot-Deseilligny, 1979*a*). Since these prerequisites are crucial for all the conclusions the first section of results is devoted to the experimental support for these assumptions during voluntary ankle dorsiflexion. Results on recurrent inhibition during various contractions of pretibial flexors are then presented in section 2.

(*1*) *Validity of the method of studying recurrent inhibition to soleus motoneurones during pretibial flexor voluntary contractions*

Fig. 1 illustrates the basic finding that during pretibial flexor contraction (tonic contraction of weak force in this case), the H′ test reflex was much more inhibited than the reference H reflex. Sample records in Fig. 1 *A–H* show the responses evoked by the four different kinds of stimulation at rest (*A–D*) and during pretibial flexor contraction (*E–H*). The H1 conditioning reflex was elicited by a 5·5 mA stimulus at rest. Because of the soleus H-reflex inhibition during pretibial flexor contraction, it was necessary to increase the stimulus strength to 6·1 mA to restore its size to the same level (9·5 mV) as at rest (Fig. 1 *A* and *E*). The conditioning reflex discharge reaching Renshaw cells was therefore the same in the two situations. Fig. 1 *D* and *H* shows that the M wave elicited by the fourth stimulus had the same size (6 mV) in the two situations, thus ensuring that stimulation conditions were identical. At rest, the H′ test reflex (Fig. 1 *B*) and the reference H reflex (Fig. 1 *C*) had exactly the same size (5·5 mV), but, during voluntary ankle dorsiflexion, the H′ test reflex was reduced (2·2 mV, Fig. 1 *F*) more than the reference H reflex (3·85 mV, Fig. 1 *G*). Quantitative analysis of all results concerning reference and H′ test reflexes (twenty of each) obtained in this subject and in this session is presented in Fig. 1 *I* and *J*. That the inhibition of H′ was larger than that of reference H was statistically significant ($P < 0·01$). As described in the Methods, such differential changes in H′ test and reference H reflexes should indicate an increase in soleus-coupled recurrent inhibition. This conclusion has been made possible because factors which could have modified the H′ test and reference H reflexes differently were shown not to be responsible for this result: (1) differences in the sensitivity of the two reflexes to inhibitory influences related to voluntary ankle dorsiflexion; (2) 'opening', during contraction, of reflex pathways by which the conditioning stimulus could modify the test reflex (see below).

Intentional modification of the test reflex. Before eliminating these factors which could have modified the H′ test and reference H reflexes differently, a more general cause of bias has to be considered. It is easy to alter an H reflex intentionally, when the different kinds of stimulation are regularly alternated, as in the experiment

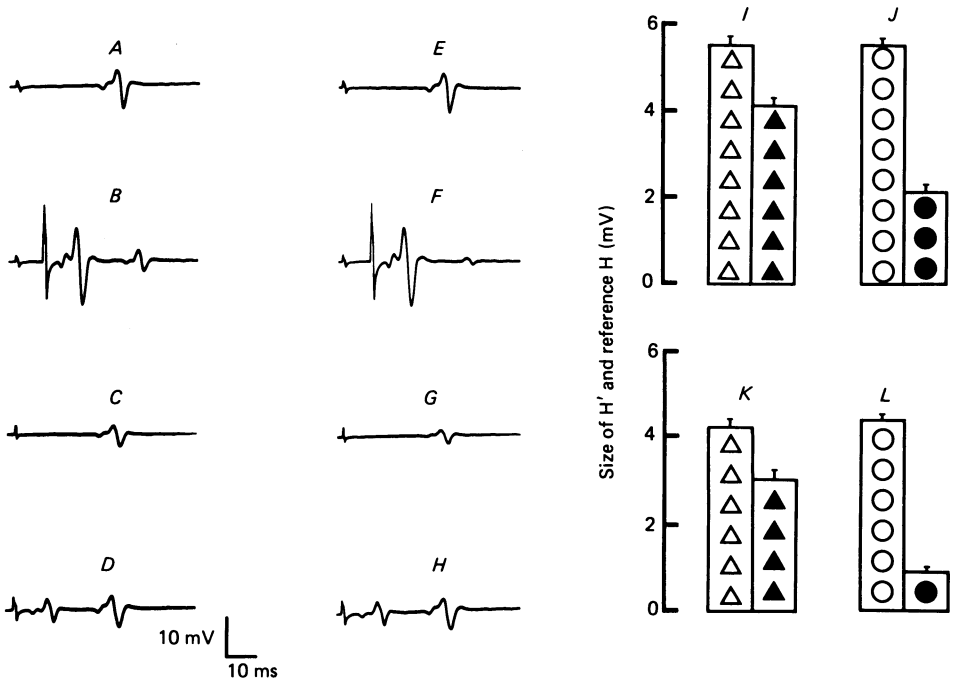

Fig. 1. *A–H*, e.m.g. responses in soleus following the four different kinds of stimulation at rest (*A–D*) and during a weak tonic contraction of the tibialis anterior (*E–H*). *A* and *E*, H1 conditioning reflex; *B* and *F*, combined conditioning and test (supramaximal) stimuli resulting in the H′ test reflex; *C* and *G*, reference H reflex; *D* and *H*, M response elicited to ensure stability of stimulation conditions during contraction. Stimulation of motor axons elicits an antidromic motor volley which in colliding with the reflex volley suppresses part of the latter and reduces the size of the reflex recorded in the e.m.g. (Hoffman, 1918). Because the collision takes place in thick motor fibres (the first to be recruited by electrical stimulation), this amputated part corresponds to the largest motoneurones recruited in the reflex volley. According to the size principle, these motoneurones are the last to be recruited by the Ia afferent volley and the first to be derecruited by inhibitory inputs. It is thus not surprising that during such a weak contraction the H reflex is hardly inhibited (compare *H* to *D*): the very motoneurones which were derecruited during tibialis anterior contraction would be those corresponding to the amputated part of the reflex in the e.m.g. *I* and *J*, quantitative analysis of the results obtained with *regularly* alternated stimulations and illustrated in *A–H*. *K* and *L*, same quantitative analysis but obtained in another subject with *randomly* alternated stimulation. Reference (*I* and *K*) and H′ test (*J* and *L*) reflexes are compared at rest (open symbols) and during a weak tonic flexor contraction (filled symbols). Each column represents the mean of twenty measurements, and the vertical bars 1 S.E. of the mean.

detailed in Fig. 1. In such a case the subject who knows the kind of stimulation to come (e.g. test or reference) might be impelled to influence the results subconsciously. In a recent paper (Fournier, Katz & Pierrot-Deseilligny, 1984), this very general problem has been examined: the effect of a conditioning stimulation on the test reflex was compared when the different kinds of stimulation (test alone, conditioning plus test) were alternated either regularly or randomly. It has been found that regular alternation *can* lead to artifactual results, whereas this never happened when using

random alternation where the subject cannot forecast the kind of stimulation to come. Thus, we have repeated all the experiments dealing with changes in test and reference H reflexes during voluntary ankle dorsiflexion using randomly alternated stimulations. This is exemplified in Fig. 1 *K* and *L* which shows the results obtained during weak tonic contraction of the pretibial flexors in one subject (different from that of Fig.

TABLE 1. Comparison in two subjects of the inhibition of the reference H and H′ test reflexes by a preceding (5 ms) weak (0·8 × the threshold of α-motor axons) stimulation applied to the gastrocnemius medialis nerve. Amplitude of reflexes is expressed as a percentage of their unconditioned value. Each figure represents the mean of twenty measurements

	Size of reflexes (% of unconditioned value)	
Subjects	Reference H	H′ test reflex
Rig.	41	81
Ber.	69	89

1 *A–J*) who also had a large H′ reflex (4·4 mV) at rest. Here too, the reduction of the H′ test reflex during contraction (Fig. 1 *L*) was larger (statistically significant: $P < 0{\cdot}01$) than that of the reference H reflex. All the experiments described hereafter (before and during various contractions of pretibial flexors) were repeated with randomly alternated stimulations and the same result was obtained, thus indicating that intentional alteration of results was not a significant factor here.

Relative sensitivity of H′ test and reference H reflexes to inhibitory influences. Inhibition of the soleus H reflex during (Hoffmann, 1918) or before (Pierrot-Deseilligny, Lacert & Cathala, 1971) voluntary contraction of the pretibial flexors is well-known. This inhibition is probably due in part to the activation of reciprocal Ia inhibition (Tanaka, 1974), although presynaptic inhibition of soleus Ia fibres by the Ia discharge from pretibial flexors has also been postulated to contribute to it (Gottlieb, Agarwal & Stark, 1970; Pierrot-Deseilligny & Lacert, 1973). Regardless of its mechanism, this inhibition of the soleus H reflex contributes to the decrease in the H′ test reflex illustrated in Fig. 1. As explained in the Methods, this contribution has been estimated by comparing the inhibition of the test reflex to that of a reference H reflex. These reflexes are, however, elicited under different conditions (see below), which could cause their sensitivity to those inhibitory mechanisms related to pretibial flexor contraction to be different.

Sensitivity of H′ and reference H reflexes to post-synaptic inhibition. Because of the method used, motoneurones giving rise to the test reflex undergo the post-spike after-hyperpolarization following H1, which could change their sensitivity to inhibitory synaptic inputs. The sensitivity of the H′ test and reference H reflexes to inhibitory inputs has therefore been tested by inhibiting both responses with a preceding (5 ms) weak stimulation (subliminal for α-motor axons) applied to the gastrocnemius medialis nerve. It has been demonstrated that such a stimulation results in a pure post-synaptic Ib inhibition of the soleus H reflex in man (Pierrot-Deseilligny, Katz & Morin, 1979). In Table 1 the comparison is drawn between the

effect of the same gastrocnemius medialis nerve conditioning stimulation on the amplitude of the reference and test reflexes: the size of the two reflexes (which was equal in control conditions) is expressed as a percentage of their unconditioned value. Ib inhibition from gastrocnemius medialis reduced the H′ test reflex much less than the reference H reflex, which indicates that motoneurones undergoing after-

TABLE 2. Comparison in two subjects of the inhibition of the reference and H′ test reflexes by a preceding (50 ms) weak vibration applied to the tibialis anterior tendon. Amplitude of reflexes is expressed as a percentage of their unconditioned value. Each figure represents the mean of twenty measurements.

	Size of reflexes (% of unconditioned value)	
Subjects	Reference H	H′ test reflex
Rig.	32	32
Ber.	58	61
Hed.	58	61

hyperpolarization are less sensitive to Ib inhibitory synaptic inputs, as they are less sensitive to excitatory synaptic inputs because of a membrane conductance increase for potassium ions (Hultborn & Pierrot-Deseilligny, 1979*a*). This very likely also applies to inhibitory synaptic inputs elicited in soleus motoneurones by activating the pathway of reciprocal Ia inhibition from ankle flexors. Thus, during voluntary ankle dorsiflexion this should tend to reduce the H′ test reflex less than the reference H reflex and cannot explain the opposite result illustrated in Fig. 1.

Sensitivity of H′ and reference H reflexes to presynaptic inhibition of Ia fibres. It was not possible to eliminate *a priori* the possibility that presynaptic inhibition of soleus Ia fibres acts differently on the afferent volleys eliciting the two reflexes: the supramaximal test volley activates all Ia fibres, whereas the afferent volley eliciting the reference H reflex activates a part of them. The sensitivity of the two reflexes to presynaptic inhibition has therefore been tested by inhibiting them with a preceding (50 ms) weak vibration (train of three stimuli, interval 5 ms, 0·3 mm amplitude) applied to the tibialis anterior tendon. It has recently been demonstrated that, for conditioning–test intervals longer than 25 ms, such a vibratory stimulus elicits a pure presynaptic inhibition of soleus Ia fibres (Morin, Pierrot-Deseilligny & Hultborn, 1984). In Table 2 the comparison is drawn between the effect of the vibratory stimulation on the amplitude of the reference and test reflexes. Tibialis anterior vibration reduced the two reflexes similarly, thus indicating that they are similarly sensitive to presynaptic inhibition of soleus Ia fibres.

The conditioning stimulus does not modify soleus motoneurone excitability by pathways other than recurrent inhibition. At rest, it was demonstrated that the depression of the H′ test reflex was only related to the size of the H1 conditioning discharge and not to the strength of the conditioning stimulus *per se*, when the conditioning–test interval was longer than 9 ms (Bussel & Pierrot-Deseilligny, 1977). This implied that inhibitory effects elicited by stimulating Ib fibres were no longer efficient at such intervals, which was confirmed when it was demonstrated that Ib inhibition from

the gastrocnemius medialis muscle to soleus motoneurones was always over 8–9 ms after the conditioning stimulus at rest (Pierrot-Deseilligny *et al.* 1979) as well as during voluntary ankle dorsiflexion (E. Pierrot-Deseilligny & M. Shindo, unpublished observations). That the inhibition of the test reflex was not related to the conditioning

TABLE 3. Comparison of the effects of a sural nerve stimulation on the size of the soleus H reflex at rest and during a pretibial flexor voluntary contraction. The size of the soleus H reflex is given in mV (absolute values). Each figure represents the mean of twenty measurements

	Size of reflexes (mV)			
	Rest		Pretibial flexor voluntary contraction	
Subjects	Control	With sural stimulation	Control	With sural stimulation
Rig.	6·8	6·5	6·0	6·3
Ber.	7·0	7·5	7·5	7·4

stimulus strength *per se* also suggested that cutaneous effects elicited by the conditioning stimulus were negligible at such intervals. It would be possible, however, that cutaneous pathways, which are closed at rest, operate during pretibial flexor contractions, since changes in transmission in cutaneous pathways have been described in man during voluntary movement (Pierrot-Deseilligny, Bergego & Katz, 1982). Experiments were therefore performed to determine to what extent pretibial flexor voluntary contractions change transmission in cutaneous pathways to soleus motoneurones. The soleus H reflex was conditioned by a selective cutaneous stimulation applied to the sural nerve at the ankle. Its strength was adjusted so as to evoke the same tactile sensation as the conditioning stimulation eliciting H1, and it preceded the soleus H reflex by 18 ms, which corresponds to a cutaneous stimulation applied 10 ms before the test volley at the popliteal fossa. Hence, the sural nerve stimulation mimicked the stimulation of cutaneous afferents evoked by the conditioning stimulus eliciting H1. Table 3 shows that for such a short delay sural nerve stimulation did not modify the size of the soleus H reflex either at rest (as previously described by Pierrot-Deseilligny, Morin, Bergego & Tankov, 1981), or during pretibial flexor contraction.

In conclusion neither the different sensitivity to pre- and post-synaptic inhibition of the reference and test reflexes nor the opening, during contraction, of inhibitory pathways to soleus motoneurones can account for the test reflex inhibition larger than that of the reference H reflex during voluntary ankle flexion (Fig. 1). By elimination, the larger inhibition of H′ during contraction may therefore be attributed to an increase in the conditioning discharge-induced depression of the test reflex. This is observed despite the lower sensitivity of the soleus motoneurones responsible for the test reflex to inhibitory synaptic inputs related to flexor contraction. It follows that this increase in the depression following H1 is likely to be underestimated. This depression following H1 is caused by the after-hyperpolarization *and* recurrent inhibition following the conditioning discharge (Bussel & Pierrot-Deseilligny, 1977).

Given that the after-hyperpolarization of soleus motoneurones is unchanged during voluntary activation of antagonistic motoneurones (a highly probable assumption), that *the inhibition of the H′ test reflex was larger than that of the reference H reflex may indicate an increase in recurrent inhibition*, as compared to rest.

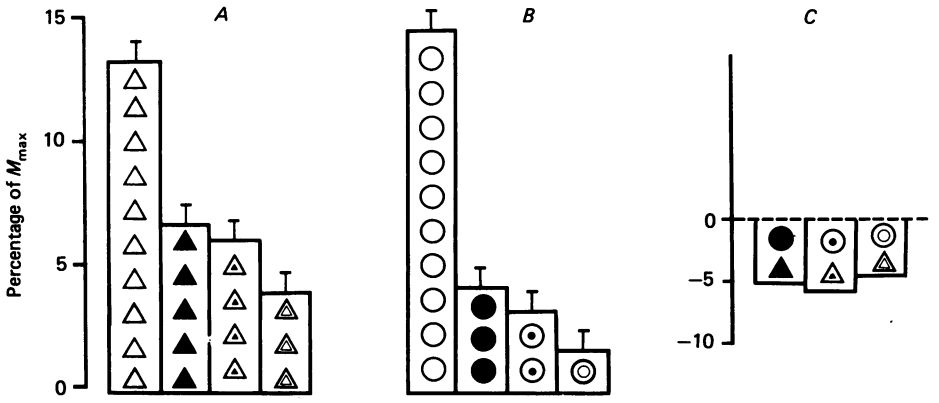

Fig. 2. Variations of reference (*A*) and H′ test (*B*) reflexes during tonic pretibial flexor contractions of various forces. Open triangles and circles, rest. Filled triangles and circles, weak pretibial flexor contraction (8%). Spotted triangles and circles, weak medium pretibial flexor contraction (30%). Double circles and triangles, medium pretibial flexor contraction (45%). *A*, modifications of soleus reference H reflex. *B*, modifications of soleus H′ test reflex. *C*, difference (H′ − reference H) for each level of force. Each column represents the mean of twenty measurements, and the vertical bars 1 S.E. of the mean.

(2) Changes in recurrent inhibition to soleus motoneurones during or before various pretibial flexor voluntary contractions

Tonic contractions of various forces. The variations of the H′ test and reference H reflexes were studied during three levels of pretibial flexor contraction (Fig. 2). These contractions ranged from a weak force of about 8% of the maximum force (filled circles and triangles) to a medium of 45% (double triangles and circles), including also an intermediate force of 30% (spotted triangles and circles).

For technical reasons it was not possible to perform these experiments during stronger pretibial flexor contractions. With our method, the size of the H1 conditioning discharge has to be large enough to elicit a sizeable recurrent inhibition, i.e. it has to be within the range in which the corresponding H′ amplitude is decreasing when increasing H1 (Hultborn & Pierrot-Deseilligny, 1979*a*). Because the inhibition of the soleus H reflex increased with the force of tibialis anterior contraction (see below), this requirement cannot be fulfilled during strong contraction of pretibial flexors.

Fig. 2 illustrates the results obtained in one subject in whom, as rest, the size of the H′ test reflex (2·6 mV) was equal to 14·5% of the maximum of the M wave (M_{max}) when the corresponding H1 amplitude was 50% of M_{max}. Whatever the force of voluntary ankle dorsiflexion, the H′ test reflex was more inhibited than the reference H reflex, as indicated by the negative values of the difference (H′ − reference H) illustrated in Fig. 2*C* (statistically significant: $P < 0{\cdot}01$). As seen above, this suggests

an increase in recurrent inhibition from H1, as compared to rest. Inhibition of both reference and H′ test reflexes increased with each level of contraction force (Fig. 2*A* and *B*) but, within the range of forces studied, as shown in Fig. 2*C*, increasing the force did not result in any significant increase in the difference (H′ − reference H).

The size of the test reflex used in these experiments varied from one subject to another for several reasons. (1) For a given amplitude of the H1 conditioning reflex, the amount of H1 conditioning-induced depression of the test reflex, and thus the size of the test reflex, is not the same in all subjects at rest (Bussel & Pierrot-Deseilligny, 1977). (2) The size of the H1 conditioning discharge had to be the same at rest and during the contractions of different force, in order to provide an identical input to Renshaw cells in the different situations. H1 was therefore not maximum at rest but small enough to be able to restore it during contraction by increasing the conditioning stimulus strength. As the amount of soleus H-reflex inhibition caused by a voluntary ankle dorsiflexion of a given force varied from one subject to another, it was not possible to use the same size of H1 in all the subjects. Since the size of the H′ test reflex is inversely proportional to that of H1 (Bussel & Pierrot-Deseilligny, 1977), these differences in the size of H1 also contributed to the variation between subjects' amplitude of the test reflex. This amplitude varied from 5 to 25 % of M_{max} in the ten subjects studied during tonic contraction of the pretibial flexors. In spite of these variations the inhibition of the test reflex during contraction was always larger than that of the reference H reflex, this difference being statistically significant ($P < 0{\cdot}05$) in all cases.

Phasic ramp contractions. An example of the variations in the H′ test and reference H reflexes during a pretibial flexor ramp contraction is illustrated in Fig. 3. Tension (continuous line) increased for 250 ms and then remained constant for 250 ms during the following medium (45 % of maximum force) tonic contraction. At rest, H′ test (circles) and reference H (triangles) reflexes had the same size: 5 % of M_{max} in this subject. The size of the two reflexes during contraction, expressed as a percentage of their control value at rest, is plotted against the time elapsed between the onset of voluntary e.m.g. activity and the stimulation eliciting the reflexes.

The time course of the reference H-reflex variations corroborates results obtained by Morin & Pierrot-Deseilligny (1977): the inhibition of the reference H reflex seen at the onset of e.m.g. activity (65 % of its control value at rest) started to decrease 40–50 ms after the onset of tibialis anterior e.m.g. activity. This 'relative facilitation', which peaked at 70 ms when the inhibition completely disappeared, was followed by a progressive increase in the inhibition up to 140 ms. Then the deep inhibition, which reduced the reference H reflex by up to 10 % of its value at rest, remained constant.

The time course of the H′ test reflex is extremely different since the H′ size continuously decreased throughout the first 140 ms of the contraction, thus exhibiting no relative facilitation. During this period the H′ test reflex was therefore much more inhibited than the reference H reflex, this differential effect being maximum at the peak of the relative facilitation, when it was highly statistically significant ($P < 0{\cdot}001$). At the end of the ramp, inhibition of both reflexes was so deep that they had almost completely disappeared and were too small to be validly compared.

Ten subjects were explored in this way. Their H′ amplitude, at rest ranged from 5 to 25 % of M_{max} and, as seen in the Methods, the strength of the stimulus eliciting

the reference H was adjusted so that it matched the H′ test reflex at rest. The depth of the inhibition of both H′ test and reference H reflexes during voluntary ankle dorsiflexion varied from one subject to another. In all cases, however, the test reflex was significantly more inhibited ($P < 0{\cdot}001$) than the reference H reflex at the peak of the reference H relative facilitation, which occurred 60–100 ms after the onset of contraction in the different subjects.

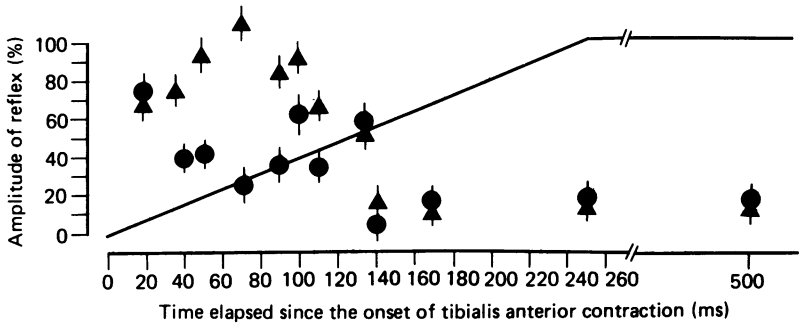

Fig. 3. Time course of the H′ test (circles) and reference H (triangles) reflexes during a tibialis anterior ramp contraction lasting 250 ms. Continuous line, averaged muscle force (arbitrary units). The amplitude of the H′ test and reference H reflexes are expressed as a percentage of their value at rest. Each symbol represents the mean of ten measurements, and the vertical bars represent ± 1 S.E. of the mean. Abscissa, time elapsed between the onset of tibialis anterior e.m.g. activity and the test stimulation.

Ballistic contractions. Experiments were also performed during ballistic contraction of the pretibial flexors. An example obtained in one subject who at rest had an H′ test reflex equal to 2·6 mV (corresponding to 16 % of M_{max}) is illustrated in Fig. 4. In this experiment, the comparison was drawn between the changes in the reference and H′ test reflexes, elicited at rest (open symbols) and 50 ms after the onset of a ballistic contraction (filled symbols). By contrast with what was observed during tonic or ramp contractions, both reflexes were similarly inhibited. Regardless of the time elapsed since the onset of contraction, the same result was obtained in all five subjects studied in this way: there was no evidence for any differential effect in H′ test and reference H reflexes during ballistic contraction.

Effects preceding voluntary ankle dorsiflexion. In order to know whether supraspinal control contributes to the differential changes in reference and test reflexes observed during ramp and tonic contractions, experiments were performed before voluntary ankle dorsiflexion. In Fig. 5 the comparison is drawn between reference and test reflexes at rest (open symbols) and during the period immediately preceding (50–10 ms) a ramp contraction of medium force (identical to the one illustrated in Fig. 3). In this experiment performed on one subject who had at rest a large H′ test reflex (4·2 mV, 24 % of M_{max}), the different stimulations were randomly alternated. As previously described (Pierrot-Deseilligny *et al.* 1971), the soleus H reflex (reference H, filled triangles) was inhibited (passing from 22 to 13 % of M_{max}) before a pretibial flexor ramp contraction, but the H′ test reflex (filled circles) was much more inhibited (passing from 24 to 4 % of M_{max}), this difference being statistically significant

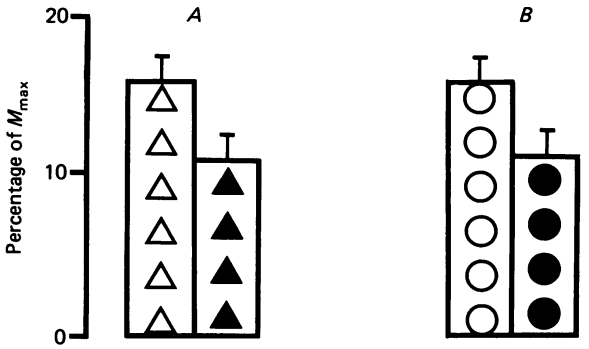

Fig. 4. Variations of the reference (*A*) and H′ test (*B*) reflexes during ballistic contractions. Results obtained during contraction (filled symbols) are compared to those obtained at rest (open symbols). Each column represents the mean of twenty measurements, and the vertical bars 1 S.E. of the mean.

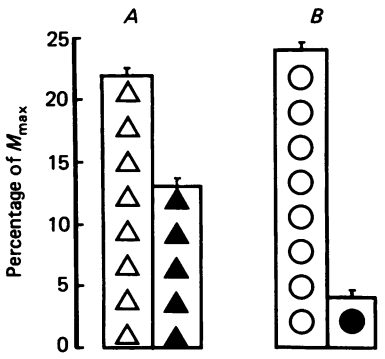

Fig. 5. Variations of reference H (*A*) and H′ (*B*) test reflexes during the period immediately preceding (50–10 ms) a ramp contraction of medium force. Results obtained before contraction (filled symbols) are compared to those obtained at rest (open symbols). Each column represents the mean of twenty measurements, and the vertical bars 1 S.E. of the mean.

($P < 0{\cdot}01$). Here also there was variation between the subjects' amount of reflex inhibition, but, in all eight subjects studied in this way, the H′ test reflex was more inhibited than reference H, this difference being significant ($P < 0{\cdot}05$) in all cases.

DISCUSSION

Modifications of the depression of soleus motoneurones brought about by a homonymous H1 conditioning reflex discharge have been studied during various voluntary contractions of the antagonistic muscles, the pretibial flexors. These modifications were appreciated by comparing changes in the H′ test reflex (which is produced by motoneurones undergoing this depression following H1) to those of a reference soleus H reflex: both reflexes were subject to the same type of influences which modified soleus monosynaptic reflex excitability during pretibial flexor

contractions, but the size of the H′ test reflex depended in addition on the depression elicited by the H1 conditioning discharge. It was observed that, before or during ramp and tonic contractions of the pretibial flexors, the H′ test reflex was significantly more inhibited than the reference H reflex. As seen in section 1 of the Results, neither the different sensitivity to pre- and post-synaptic inhibition of the two reflexes, nor the stimulation of Ib or cutaneous fibres by the conditioning stimulation can account for this differential effect on H′ test and reference reflexes. Under these conditions, the larger inhibition of the H′ test reflex than that of the reference H reflex during flexor contractions may be attributed to an increase in the H1 conditioning discharge-induced depression of the test reflex, thus suggesting an increase in the recurrent inhibition elicited by a given H1 discharge, i.e. a facilitation of soleus-coupled Renshaw cells (as compared to rest).

Origin of the Renshaw cell facilitation

Facilitation of soleus-coupled Renshaw cells may have three possible origins (cf. Hultborn & Pierrot-Deseilligny, 1979*a*,*b*): (1) motoneurone discharge reaching Renshaw cells via motor axon collaterals; (2) segmental afferents; (3) supraspinal influences.

(*1*) *Motoneurone discharge reaching Renshaw cells via recurrent collaterals.* This is the most powerful source of Renshaw cell facilitation (cf. Baldissera *et al.* 1981). Although the pattern of distribution of recurrent inhibition in man is as yet largely unknown, one can assume that it does not differ strongly from that described in the cat (Baldissera *et al.* 1981) and from that, remarkably similar, found in the baboon (H. Hultborn, E. Jankowska & S. Linström, personal communication). If recurrent inhibition has never been found between motor nuclei of strict antagonists at the same joint, like tibialis anterior and soleus muscles, it does exist from peroneus motoneurones to soleus, and, in man, voluntary dorsiflexion of the foot is accompanied by some peroneus muscle contraction. Some facilitation of soleus-coupled Renshaw cells could thus be caused by the voluntary discharge of peroneus α-motoneurones. Another cause of facilitation of soleus-coupled Renshaw cells which must be considered could be the occurrence of a soleus stretch reflex which would cause some motor units to discharge (even though there was no evidence for any soleus e.m.g. activity at the site of the recording electrodes). If facilitation of soleus-coupled Renshaw cells through recurrent collaterals (from peroneus and/or soleus motor axons) cannot be excluded, it cannot explain Renshaw cell facilitation observed before the onset of any e.m.g. activity (Fig. 5).

Mutual inhibition between Renshaw cells is mainly limited to functionally related Renshaw cells, i.e. those which are activated from the same axon collaterals (Ryall, 1970). It is, therefore, likely that Renshaw cells activated from pretibial flexor motoneurones do not inhibit soleus-coupled Renshaw cells. In any event, it must be pointed out that this mechanism (inhibition of soleus-coupled Renshaw cells) would be expected to reduce the recurrent inhibition from H1 and cannot account for the opposite result observed before or during ramp and tonic contractions.

(2) *Facilitation of Renshaw cells by segmental afferents.* This has been described after stimulation of group II and III muscle afferents (Piercey & Goldfarb, 1974) independently of any concomitant motoneurone discharge. It cannot be excluded that group II and III afferent discharges from the contracting pretibial flexors

contribute to facilitation of soleus-coupled Renshaw cells. Neither can this mechanism, however, explain the Renshaw cell facilitation occurring before the contraction.

(*3*) *Supraspinal facilitation of Renshaw cells.* This has been described after stimulation of different higher centres (cf. Baldissera *et al.* 1981). Such a supraspinal influence is the only mechanism which can explain the facilitation of Renshaw cells occurring before pretibial flexor contraction, and thus before any motor discharge and any change in peripheral afferents.

Functional significance of facilitation of soleus-coupled Renshaw cells accompanying voluntary ankle dorsiflexion

A voluntary ankle dorsiflexion produces a passive stretch of the antagonistic soleus muscle. This causes Ia discharge in soleus Ia fibres (Hagbarth, Wallin & Löfstedt, 1975) which could produce a soleus stretch reflex. In the event of a soleus stretch reflex which would oppose the ankle flexion movement, facilitation of soleus-coupled Renshaw cells would help to counteract this 'undesirable effect': the first soleus motoneurones to fire would inhibit other soleus motoneurones and thus curtail the stretch reflex. Because of the dynamic sensitivity of muscle spindles, the risk of a soleus stretch reflex is greater during phasic than during tonic ankle flexor contractions, and the relative facilitation of the soleus H reflex during fast ramp contractions (Fig. 3) has indeed been related to the stretch-induced Ia discharge from soleus (Morin & Pierrot-Deseilligny, 1977). Interestingly enough, facilitation of soleus-coupled Renshaw cells, as estimated by the difference, H′ − reference H, was observed to be maximum just when this relative facilitation of soleus motoneurones was maximum, i.e. when the risk of a soleus stretch reflex was greatest. It is not so surprising that there is no evidence for Renshaw cell facilitation during ballistic contractions: the stretch reflex which is sometimes triggered in antagonists in the course of such rapid movements (Basmajian, 1974) occurs too late to hinder the development of these very brief movements, and, on the contrary, could help to stop them.

Facilitation of Renshaw cells directed to antagonistic motoneurones is obviously not the only factor which prevents stretch reflexes of the passively stretched antagonists. A voluntary contraction of pretibial flexors is known to be accompanied by an active inhibition of ankle extensors, as attested by the soleus H-reflex inhibition (Hoffmann, 1918; Paillard, 1955). This relaxation of antagonists is generally attributed to activation of Ia interneurones, as three mechanisms have been demonstrated to contribute to its increase during voluntary ankle dorsiflexion: (1) due to α–γ-coactivation (Vallbo, Hagbarth, Torejbörk & Wallin, 1979), Ia discharge from the contracting muscle (at least during isometric contractions or slow movements) feeds the Ia interneurones inhibiting soleus motoneurones; (2) facilitation of transmission in the pathway of reciprocal Ia inhibition observed during voluntary movement (Tanaka, 1974) occurs 80 ms before the onset of contraction (Simoyama & Tanaka, 1974), thus indicating that this facilitation is, at least partly, supraspinal in origin; (3) if reciprocal Ia inhibition is an efficient mechanism for inhibiting antagonistic motoneurones during movement, it is necessary for this pathway not to be inhibited by recurrent inhibition from voluntary motoneurone discharge. Thus, it is important that Renshaw cells directed to the motoneurones of the contracting muscle and to

corresponding Ia interneurones (Hultborn, Jankowska & Lindström, 1971) have been shown to be inhibited during voluntary movement by descending control (Hultborn & Pierrot-Deseilligny, 1979*a*,*b*), which thus favours reciprocal Ia inhibition. Furthermore, fusimotor-induced Ia discharge from tibialis anterior very likely elicits presynaptic inhibition of soleus Ia fibres (Devanandan, Eccles & Stenhouse, 1966). Using the method recently developed to study presynaptic inhibition of Ia fibres in man (Morin *et al.* 1984) it has been shown that presynaptic inhibition of soleus Ia fibres also contributes to the soleus H-reflex inhibition during voluntary ankle dorsiflexion (C. Morin & Pierrot-Deseilligny, unpublished observations).

Two mechanisms fed by the Ia discharge from pretibial flexors seem, therefore, to be responsible for soleus monosynaptic reflex inhibition during voluntary ankle dorsiflexion: reciprocal Ia inhibition, and presynaptic inhibition of Ia fibres. Even though the interneurones responsible for these inhibitory mechanisms receive a supraspinal facilitation, the efficiency of the inhibition of the soleus monosynaptic reflex considerably decreases when no longer fed by Ia discharge from the contracting muscles: indeed, during voluntary ankle dorsiflexion, blocking pretibial flexor group Ia fibres by ischaemia markedly reduces soleus H-reflex inhibition (Morin & Pierrot-Deseilligny, 1977). This implies that, to be efficient during natural movement, presynaptic inhibition of soleus Ia fibres and/or reciprocal Ia inhibition of soleus motoneurones should be supplied by a Ia inflow driven by pretibial flexor fusimotor neurones. An α–γ coactivation leading to an increased Ia discharge has indeed been demonstrated in man during tonic or slow phasic contractions (for references see Vallbo *et al.* 1979), but there is evidence for a decrease in Ia discharge from the contracting muscle during rapid unobstructed movements in the cat (for references see Prochazka & Hulliger, 1983). If these results were confirmed in man, this would suggest that during rapid movements, i.e. those which risk triggering a stretch reflex in antagonistic muscles most, neither presynaptic inhibition of Ia fibres nor reciprocal Ia inhibition is working optimally. Under these conditions *facilitation of soleus-coupled Renshaw cells could be of importance to counteract this stretch reflex.* In patients with upper motoneurone diseases, who were able to perform some tonic voluntary ankle dorsiflexion, this facilitation of soleus-coupled Renshaw cells during pretibial flexor contraction was not found (Katz & Pierrot-Deseilligny, 1982). It is tempting to assume that the lack of this control was, at least in part, responsible for the disturbance of voluntary contraction by clonus in antagonistic muscles.

The authors are grateful to Professor Hans Hultborn for his very valuable comments. Our thanks are also due to Annie Rigaudie and Michèle Dodo for excellent technical assistance, and to David MacGregor for scrutinizing the English. This work was supported by grants from the Pierre and Marie Curie University (Paris VI), from INSERM (836025) and from ETP (TW 83/146).

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