Evidence of facilitation of soleus-coupled Renshaw cells during voluntary co-contraction of antagonistic ankle muscles in man

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1. The amount of recurrent inhibition onto soleus motoneurones was compared during plantar flexion and co-contraction of antagonistic ankle plantar and dorsiflexors at matched levels of background activity in the soleus muscle.

2. During weak plantar flexion and co-contraction (less than 10% of maximal voluntary plantar flexion effort) a test reflex discharge (H′ reflex), which was conditioned by a previous reflex discharge, was found to be significantly more depressed in relation to rest than an unconditioned reference H reflex. During strong plantar flexion (more than 50% of maximal voluntary plantar flexion effort) the H′ reflex either increased more or to the same extent as the reference H reflex in relation to rest. In contrast to this, the H′ reflex was strongly depressed during co-contraction, whereas the reference H reflex was not significantly different from its resting value.

3. At the end of the ramp phase of a phasic contraction, large variations of the H′ reflex were observed during plantar flexion (large increase in relation to rest) and during co-contraction (marked decrease), whereas the reference H reflex was facilitated in the two situations.

4. These observations provide evidence that soleus-coupled Renshaw cells are differently regulated during co-contraction and plantar flexion. It is suggested that the Renshaw cells are inhibited during strong plantar flexion but not during strong co-contraction. The functional significance of the findings is discussed.

Several lines of evidence have suggested that spinal segmental pathways are differently controlled during co-contraction of antagonistic ankle muscles and during isolated plantar- or dorsiflexion (Nielsen & Kagamihara, 1992, 1993, 1994; Nielsen, Petersen, Deuschl & Ballegaard, 1993). During co-contraction the transmission in the reciprocal Ia inhibitory pathway is thus significantly depressed as compared with extension–flexion movements (Nielsen & Kagamihara, 1992). Several pathways can contribute to this differential control. Firstly, there is evidence that during extension–flexion movements the motor command is conveyed by descending pathways projecting in parallel to the motoneurones and their corresponding Ia interneurones, whereas during co-contraction it is thought to be conveyed by pathways without projections onto Ia interneurones (Fetz & Cheney, 1987; Nielsen *et al.* 1993). Secondly, presynaptic inhibition of Ia afferents onto motoneurones has been demonstrated to be increased during co-contraction and it is likely that presynaptic inhibition of Ia terminals on the Ia inhibitory interneurones is similarly regulated (Llewellyn, Yang & Prochazka, 1990; Nielsen & Kagamihara, 1993). Finally, cat experiments have demonstrated that Ia inhibitory interneurones receive recurrent inhibition from Renshaw cells activated by motor axon collaterals (Hultborn, Jankowska & Lindström, 1971*a*,*b*,*c*). Increased activity of Renshaw cells during co-contraction as opposed to extension–flexion movements could therefore explain the depression of the activity of the Ia inhibitory interneurones. The present study was undertaken to investigate this possibility using the following paired H reflex method. When an H reflex (H′) is preceded by a conditioning reflex discharge, it is subject to orthodromically evoked recurrent inhibition (Bussel & Pierrot-Deseilligny, 1977). Changes in the size of the H′ reflex without similar changes in an unconditioned reference H reflex during different tasks may thus reflect changes in recurrent inhibition (Hultborn & Pierrot-Deseilligny, 1979).

METHODS

General experimental arrangement

A total of thirty-five experiments were performed on fifteen healthy subjects (aged 20–60 years), who gave informed consent to the experimental procedures, which had been approved by the appropriate institutional ethics committees. Experiments were performed in Paris and in Copenhagen with the same standardized protocol and were conducted by the same experimenters.

The subjects were seated in an armchair with the leg to be examined semiflexed at the hip (120 deg), the knee flexed to 160 deg and the ankle at 110 deg plantar flexion. The foot was mounted to a torquemeter and the torque was displayed on an oscilloscope placed in front of the subject. The electromyograms (EMG) from the soleus and the tibialis anterior muscles were recorded from surface electrodes, filtered (100 Hz to 1 kHz), full-wave rectified, integrated and displayed on the same oscilloscope. Recurrent inhibition was assessed at rest, during voluntary plantar flexion, during voluntary dorsiflexion and during voluntary co-contraction of ankle flexors and extensors.

At the beginning of each experiment the subject was asked to produce the strongest tonic plantar flexion that was possible to maintain for more than 10 s. The amount of soleus rectified EMG activity recorded during all subsequent contractions was expressed as a percentage of the EMG activity recorded during this maximum.

During experiments on tonic contraction the torque was displayed as a continuous line on the oscilloscope. The subject was asked to perform a tonic plantar flexion so as to superimpose the torque line on a pre-set horizontal target line corresponding to a known degree of contraction. In the case of co-contraction the subject was first asked to make a contraction of the plantar flexors to a given torque level and then, while maintaining the same EMG level in the contracting soleus muscle, bring the torque back to zero by contracting the dorsiflexors. Subjects were also asked to perform a voluntary dorsiflexion, in which the tibialis anterior EMG was matched to that observed during weak co-contraction.

During experiments with phasic contractions the subjects performed first a ramp contraction during which the rectified soleus EMG activity progressively increased during 700 ms and then maintained a holding phase for about 500 ms where the EMG activity was 60% of the EMG recorded during the maximum tonic contraction defined above. The desired time course of the contraction was drawn on the oscilloscope and the subject was asked to perform plantar flexions which caused the rectified and integrated soleus EMG to follow the drawn line. In the case of co-contraction the subjects were trained to perform contractions which caused the rectified and integrated EMG of both plantar and dorsiflexors to follow the same time course while the output from the torquemeter was maintained near zero. Only three subjects with a sizeable H′ test reflex (see below) were able to perform such dynamic co-contractions. The subjects were finally requested to perform a ramp-and-hold dorsiflexion in which the tibialis anterior EMG trajectory was matched to that observed during co-contraction. Plantar flexions, dorsiflexions or co-contractions in which the rectified and integrated EMG deviated significantly from the drawn line on the oscilloscope were discarded from further analysis. The subject initiated the movement after an audible signal and the first EMG spike of the contraction was used to trigger stimulations at the very onset of the contractions or after various time delays.

Method of estimating recurrent inhibition

Changes in excitability of the soleus-coupled Renshaw cells during the different tasks were evaluated by the paired H reflex technique developed by Bussel & Pierrot-Deseilligny (1977).

Principle of the method. A detailed description of the paired H reflex technique used here to estimate changes in the homonymous recurrent inhibition of soleus motoneurones is given elsewhere (Bussel & Pierrot-Deseilligny, 1977) and some of the assumptions on which it relies have been examined in animal experiments (Hultborn, Pierrot-Deseilligny & Wigström, 1979). In this method, only motorneurones which have already fired in the first H1 reflex discharge (evoked by the S1 conditioning stimulus) have their excitability assessed by the subsequent test volley. This arises because the test stimulus is supramaximal for α-axons and induces an antidromic α-volley at the site of stimulation. Providing that the conditioning–test interval is adequate, the H1 conditioning reflex discharge collides with this antidromic motor volley and eliminates it in the corresponding motor axons so that the H′ test reflex evoked by the test stimulus can pass along these axons. Thus all motorneurones responsible for the H′ test reflex evoked by test stimuli and recorded in the EMG, because they have already fired in the H1 conditioning reflex, undergo the post-spike after-hyperpolarization. At low conditioning reflex amplitudes the H′ and H1 reflexes remain equal, but further increases in H1 amplitude result in a progressive decrease in the H′ reflex. 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 discharge. This strongly suggests that, in addition to the after-hyperpolarization, the reflex depression is caused by an increased recurrent inhibition, itself elicited by growing conditioning reflexes. The finding that this inhibition of the H′ test reflex was significantly increased after injection of a cholinergic agonist (L-acetylcarnitine) provides further evidence for its Renshaw origin (Mazzocchio & Rossi, 1989).

Comparison with a reference H reflex. If the amplitude of the H′ test reflex depends on the recurrent inhibition by the H1 conditioning reflex, it also depends on the changes in motorneurone excitability accompanying voluntary contraction of ankle muscles. Thus the excitability of the motorneurone pool was also evaluated by an ordinary H reflex (reference H reflex) of the same size as the H′ reflex at rest. Except from the depression from H1, H′ and reference H reflexes are subject to the same peripheral and supraspinal influences during contraction. An estimate of changes in recurrent inhibition during contraction may therefore be obtained by comparing changes in the size of these two reflexes. To be valid, this comparison must be made while reference H reflexes are obtained with a constant effective stimulus intensity, despite possible changes in the position of the electrodes with regard to the nerve during various contractions. An M wave (M1), the amplitude of which was 10–40% of the maximum motor response (M_{max}) (at a point of the M recruitment curve where a small variation of the stimulus intensity produces a significant change in M amplitude) was used to check the stability of the stimulating conditions and the site of the electrode was carefully chosen so that the M1 response remained constant during various contractions.

Contamination by V1. During weak plantar flexion and co-contraction it was confirmed that the supramaximal stimulus, when applied alone, did not produce any sizeable (more than 1% of M_{max}) V1 response (see Upton, McComas & Sica, 1971). With

stronger contractions and co-contractions the V1 response was systematically measured and subtracted from the H′ reflex.

Stimulation protocol. Conditioning and test stimuli (1 ms duration, 10 ms conditioning–test interval) were delivered to the tibial nerve in the popliteal fossa through the same unipolar electrode in order to evoke various motor and monosynaptic reflex responses. Four or five kinds of stimuli were alternated every 4 s: (1) the conditioning stimulus alone so that the H1 conditioning reflex discharge could be recorded; the H1 reflex was chosen so that it was maximum at rest and thus, in most cases, its size was not significantly modified during the various contractions explored here; (2) the combined conditioning and (supramaximal) test stimulations which resulted in M_{max} followed by the H′ test reflex; (3) the stimulus eliciting the reference H reflex; (4) a stimulus strong enough to activate some α-motor axons, thus causing an M1 wave, the stability of which was used to ensure that, during contractions, stimulation conditions remained constant. A fifth alternative (test stimulus alone) was added during strong tonic contractions to assess V1 (see Hultborn & Pierrot-Deseilligny, 1979). In each sequence the same number (10–20) of each kind of stimulus was presented. When possible, sequences were repeated several times using the same parameters. Sequences where the size of M1 or the H1 reflex deviated significantly from their values at rest were discarded.

Analysis. Reflex and motor responses were measured as the unrectified peak-to-peak amplitude and expressed as a percentage of M_{max} in the corresponding sequence. Having checked that there were significant changes using analysis of variance (Snedecor's F test), differences between the H′ and reference H reflex and between values in the different situations were analysed using Student's t test. The population mean and standard error of the mean were calculated for data from all subjects and differences in the reflex responses between the different situations were tested using Student's paired t test.

RESULTS

Modifications of H′ and reference H reflexes during tonic contraction

Figure 1 illustrates the basic finding that, at an equivalent amount of tonic soleus EMG activity, the H′ test reflex is smaller during co-contraction than during voluntary plantar flexion. Sample records in *A–O* show voluntary EMG and torque (*A–E*), M_{max} and H′ test reflex evoked by combined stimulation S1 plus test stimulus (*F–J*) and the reference H reflex (*K–O*) at rest (*A*, *F* and *K*), during weak (10% of maximum defined above) plantar flexion (*B*, *G* and *L*) and co-contraction (*C*, *H* and *M*) and during strong (50% of maximum) plantar flexion (*D*, *I* and *N*) and co-contraction (*E*, *J* and *O*). The soleus EMG is shown in the top row in *A–E*, the tibialis anterior EMG on the second row in *A–C* and *E* (bottom row in *D*) and the torque on the bottom row in *A–C* and *E* (second row in *D*). Changes in the size of M_{max} in the different situations (*F–J*) reflect the fact that the size of the EMG response to a constant electrical stimulus to the muscle nerve may vary significantly at different muscle lengths (Inman, Ralston, Saunders, Bertram Feinstein & Wright, 1952). The H1 conditioning reflex does not appear on the records shown in *F–J* (because of the collision; see Methods) and was measured separately. Its very large amplitude (between 80 and 90% of M_{max}) in the different situations explains why the H′ test reflex was small at rest (*F*).

A quantitative analysis of all results obtained in the subject during this session is shown in *P–S*, where open columns represent the control (at rest) size of H′ and reference H reflexes. During weak voluntary plantar flexion the H′ reflex was smaller than at rest (filled column in *P*) whereas it was increased during strong contraction (filled column in *Q*). Note that the reference H reflex, in contrast to the H′ reflex, increased during weak plantar flexion (filled column in *R*), but did not increase to the same extent as the H′ reflex during the strong contraction (compare *S* and *Q* or *N* and *I*). This suggests, as discussed in detail by Hultborn & Pierrot-Deseilligny (1979), that soleus-coupled Renshaw cells are facilitated during weak plantar flexion and inhibited during strong plantar flexion. Since it was ensured that during co-contraction the subject activated the soleus muscle voluntarily to the same extent as during the two levels of plantar flexion (compare the top row in Fig. 1*B* and *C* and in *D* and *E*), one might expect that the H′ reflex would be regulated in the same way as during plantar flexion. Actually, Fig. 1*H* and *J* and hatched columns in *P–Q* show that this was not the case, since at the two levels of co-contraction, the H′ reflex decreased with respect to its control size. Here again this decrease was not accompanied by a similar decrease in the size of the reference H reflex (hatched columns in *R–S*), since the reference H reflex was somewhat facilitated during strong co-contraction (*S*) and less inhibited than the H′ reflex during weak co-contraction (*R*).

Changes in the H′ and reference H reflexes during tonic plantar flexion, dorsiflexion and co-contraction were compared in fourteen subjects. Eight subjects with a sizeable H′ test reflex at rest were able to perform a co-contraction of about 50% of the maximal voluntary plantar flexion effort. In eleven subjects, including five of this latter group, data were also obtained with weaker contractions (10% of maximum or less). It was not possible to obtain a sufficiently large H1 discharge in two of the subjects during weak dorsiflexion nor in any of the eight subjects during strong dorsiflexion. Individual and mean (thick lines) values for weak and strong contractions are shown in Fig. 2*A* and *B*, and *C* and *D*, respectively, and the difference between the values at rest and during contraction were calculated for the H′ and reference H reflexes (ΔH′ and ΔH_{ref}). During weak dorsiflexion and co-contraction, the H′ reflex was depressed with regard to rest in all subjects, whereas it was depressed in only seven out of eleven subjects during weak plantar flexion. When pooling data from all the experiments the H′ reflex was found to be significantly depressed in relation to rest during co-contraction and dorsiflexion ($P < 0{\cdot}01$), but not

significantly during plantar flexion. However, since the reference H reflex increased during plantar flexion and was only marginally smaller than at rest during co-contraction, the H′ reflex was found to be significantly smaller than the reference H reflex during both tasks ($\Delta H' - \Delta H_{ref} = -5{\cdot}7$ and $-3{\cdot}9$, respectively; $P < 0{\cdot}001$ and $P < 0{\cdot}05$, respectively). Since the reference H reflex decreased with dorsiflexion, there was no significant difference in the two reflexes during this task ($P > 0{\cdot}1$), although the H′ reflex was marginally more depressed than the reference H reflex ($\Delta H' - \Delta H_{ref} = -0{\cdot}6$). As already discussed in detail by Hultborn & Pierrot-Deseilligny (1979) and Katz & Pierrot-Deseilligny (1984), neither the different sensitivity to pre- and postsynaptic inhibition of reference and H′ reflexes, nor the stimulation of afferent fibres by the conditioning stimulation can account for the significantly smaller size of the H′ than of the reference H reflex during weak plantar flexion and co-contraction. This must therefore reflect an increased recurrent inhibition from the H1 conditioning reflex during the two tasks. During strong plantar flexion, the H′ reflex increased in relation to rest in all eight subjects (and in 5 subjects this increase was larger than that

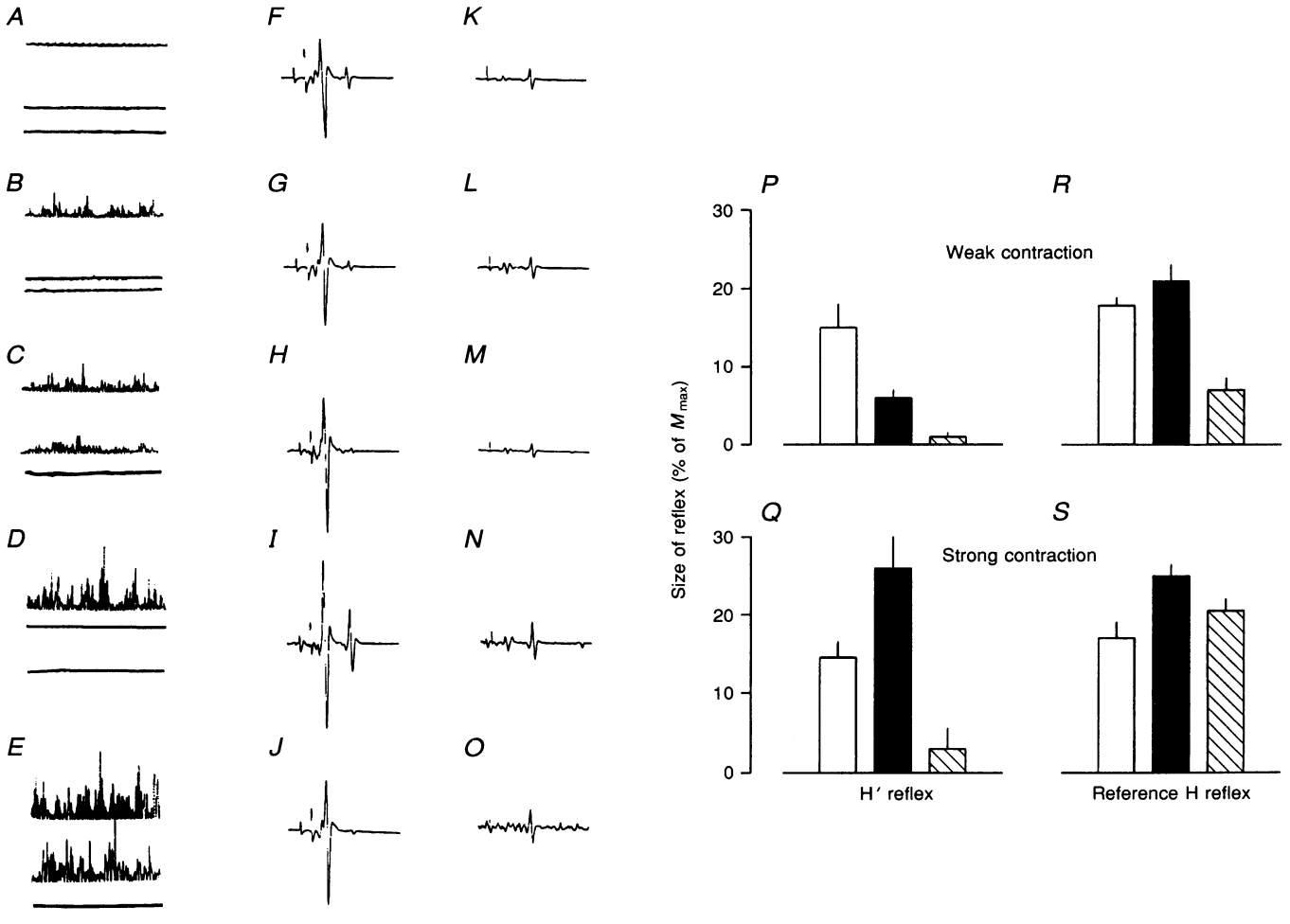

Figure 1. Changes in H′ and the reference H reflexes during weak (10% of maximal voluntary effort) and strong (50% of maximal voluntary effort) co-contraction and plantar flexion at matched levels of voluntary soleus EMG in a single subject

A–E, the voluntary soleus (upper traces), tibialis anterior EMG (middle traces, except in *D* where it is lower trace) and the torque exerted on the foot plate (lower traces, except in *D* where it is middle trace). Sample records of M_{max} (*F–O*), and H′ (*F–J*) and the reference H reflexes (*K–O*). From top to bottom measurements were made at rest, during weak plantar flexion, during weak co-contraction, during strong plantar flexion and during strong co-contraction. The time base was 20 ms in *A–E* and 10 ms in *F–O*. The voltage calibration was 100 μV in *A–E* and 1 mV in *F–O*. *P–S*, average size of the H′ (*P* and *Q*) and reference H reflexes (*R* and *S*) as a percentage of M_{max} in the different situations. Ten reflexes were averaged in each situation. The subject performed weak contractions in *P* and *R* and strong contractions in *Q* and *S*. Open columns represent measurements at rest, filled columns represent measurements during plantar flexion and hatched columns represent measurements during co-contraction. The vertical bars are the standard errors of the mean.

of the reference H reflex). By contrast, the H′ reflex decreased during co-contraction, whereas the reference H reflex increased or was hardly modified. When combining data from all eight subjects the H′ reflex was found to be significantly depressed in relation to the reference H reflex during co-contraction ($\Delta H' - \Delta H_{ref} = -14{\cdot}6$; $P < 0{\cdot}05$), whereas there was no difference in the size of the two reflexes during plantar flexion. As discussed by Hultborn & Pierrot-Deseilligny (1979), the finding that the H′ reflex was not smaller than the reference H reflex despite the strong motor discharge can be attributed to an inhibition of the soleus-coupled Renshaw cells during strong plantar flexion. The strong depression of the H′ reflex during co-contraction on the other hand suggests

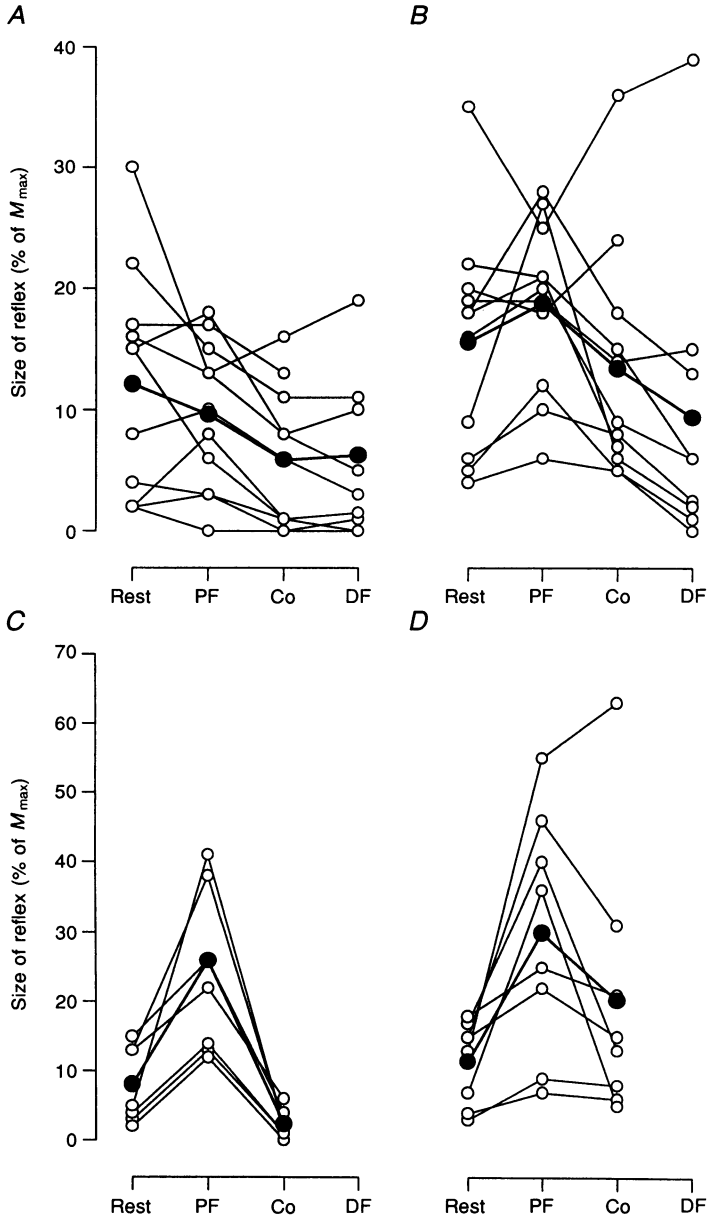

Figure 2. Diagrams illustrating the size of H′ (*A* and *C*) and reference H reflexes (*B* and *D*) during weak (*A* and *B*) and strong (*C* and *D*) contractions in all investigated subjects

Each thin line and each circle represents one subject. At least 10 reflexes were averaged in each situation in each subject. The filled circles and the thick lines represent the population mean of the reflexes during the different tasks. Eleven subjects were studied during weak contraction and eight subjects were studied during strong contraction. Five subjects were studied at both levels of contraction. Two subjects were not studied during weak dorsiflexion and none of the subjects were studied during strong dorsiflexion, since it was not possible to obtain a sufficiently large H1 reflex. Measurements were made at rest, during plantar flexion (PF), during co-contraction (Co) and during dorsiflexion (DF). The average level of contraction in *A* and *B* was around 10% of the maximal voluntary effort, whereas it was around 50% in *C* and *D*.

a very pronounced transmission in the recurrent inhibitory pathway during this task.

Modifications of H′ and reference H reflexes during phasic contraction

Sample records in Fig. 3*A* and *B* show the integrated traces (time constant 120 ms) of the EMG from soleus (top row) and tibialis anterior (bottom row) during a phasic contraction, the ramp phase of which lasted for 700 ms. Soleus EMG during plantar flexion (*A*) and soleus and tibialis anterior EMG during co-contraction (*B*) progressively increased during this ramp phase at the end of which the EMG activity was 60% of the maximum (as defined in Methods). The H′ (10 ms conditioning–test interval) and reference H reflexes (*C* and *D*, respectively) were measured at rest, at the very onset of contraction and at various intervals (200, 500 and 700 ms) after the onset of the ramp. The H1 reflex (maximum H reflex at rest) varied between 65 and 75% of M_{max} in the different situations and M1 was constant around 40% of M_{max}.

Figure 3*C* and *D* illustrates the time course of the variations in the H′ and reference H reflexes, respectively, both reflexes having the same size at rest. During plantar

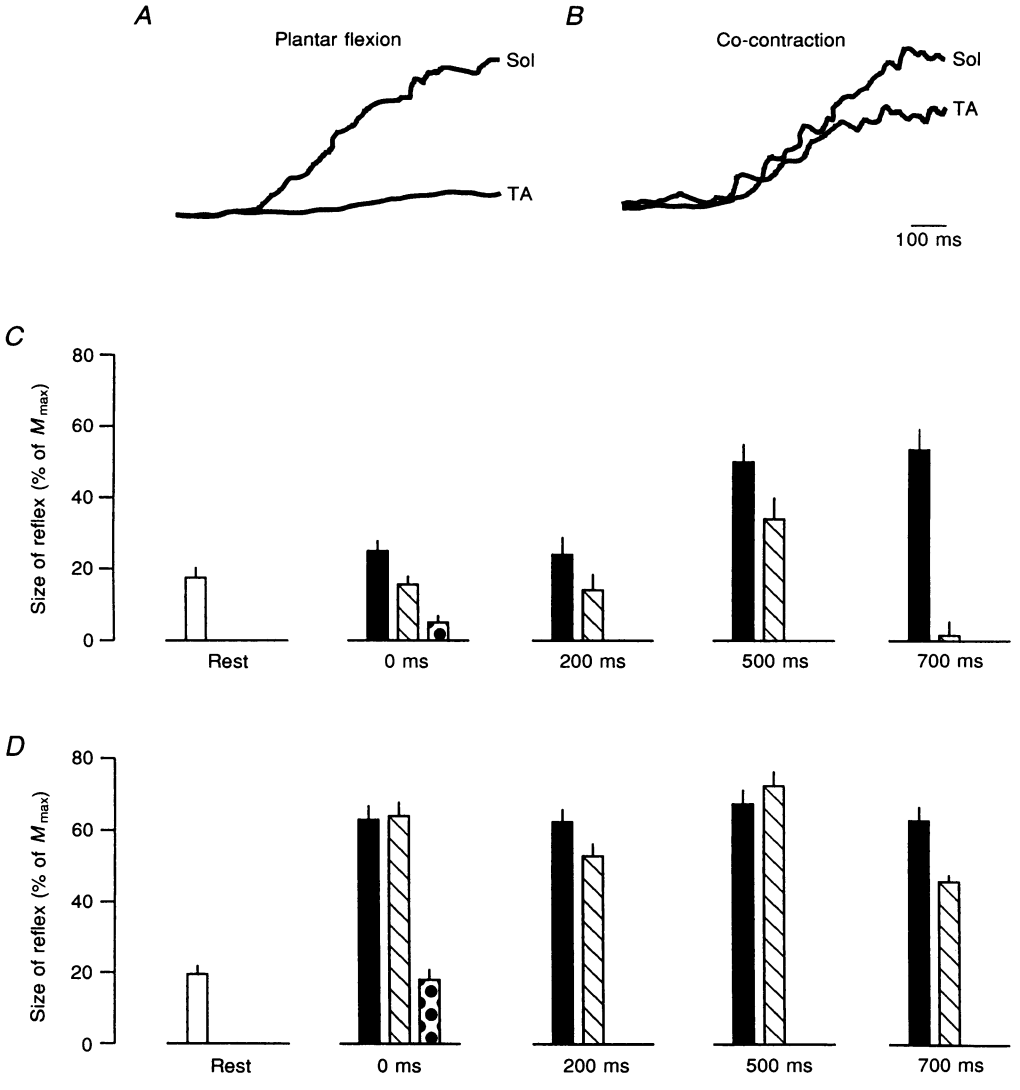

Figure 3. Changes in the size of H′ (*C*) and the reference H reflexes (*D*) during the ramp phase of a ramp-and-hold plantar flexion, and co-contraction

In all cases the ramp phase of the contraction lasted 700 ms. The data are from a single subject. The voluntary soleus (Sol) and tibialis anterior (TA) EMG during the ramp are shown during plantar flexion (*A*) and co-contraction (*B*). Each column is the mean of 10 reflexes and the vertical bars are the standard errors of the mean. Filled columns represent measurements during plantar flexion and hatched columns represent measurements during co-contraction. Measurements were made at the onset of contraction and at delays of 200, 500 and 700 ms. The open columns in *C* and *D* represent the control measurements at rest. Columns with dots represent measurements at the onset of dorsiflexion. It was not possible to make measurements at later intervals during the dorsiflexion ramp, since H1 reflex was strongly depressed.

flexion, the amplitude of the H′ reflex (filled columns in *C*) remained near to its rest value at the earliest intervals following EMG onset, then markedly increased at 500 ms and reached a peak at the end of the ramp. Note that these changes cannot be explained by changes in motoneuronal excitability since the amplitude of the reference H reflex was stable throughout the ramp plantar flexion (filled columns in *D*). As discussed in detail by Hultborn & Pierrot-Deseilligny (1979) this strongly suggests that recurrent inhibition to soleus motoneurones is decreasing throughout the ramp phase of a phasic soleus contraction. In contrast, the H′ reflex (hatched columns in *C*) was completely depressed at the end of the ramp, the difference between the size of the H′ reflex in the two situations being highly significant ($P < 0{\cdot}001$). Here again changes in motoneuronal excitability cannot account for the dramatic changes in the size of the H′ reflex since the corresponding reference H reflex was relatively constant throughout the co-contraction (hatched columns in *D*). At the end of the ramp phase, a similar and highly significant ($P < 0{\cdot}001$) difference was found between the facilitation of the H′ reflex during plantar flexion and its inhibition during co-contraction, in the two other subjects so explored. In two of the three subjects (including the one used for the illustration in Fig. 3) the reference H reflex also decreased during the co-contraction ramp in relation to plantar flexion. However, in all three subjects the H′ reflex was significantly more depressed than the reference H reflex towards the end of the co-contraction ramp as compared with the same time during the plantar flexion ramp ($P < 0{\cdot}01$), thus suggesting a specific increase of recurrent inhibition towards the end of the co-contraction ramp.

In two of the three subjects it was possible to evoke a sufficiently large H1 reflex at the onset of a ramp-and-hold dorsiflexion with the same tibialis anterior EMG trajectory as that observed during co-contraction. The H′ reflex was significantly more depressed than the reference H reflex already at this early time of the contraction (compare columns with dots in Fig. 3*C* and *D*; $P < 0{\cdot}01$). In the subject used for the illustration in Fig. 3, it was not possible to obtain a sufficiently large H1 reflex later during the ramp, but in the other subject the H′ and reference H reflexes were both found to be strongly depressed at the 200 and 500 ms intervals. The pattern of changes in the size of the H′ reflex is thus different during the course of each of the three types of contraction.

DISCUSSION

In the present study recurrent inhibition of soleus motoneurones was assessed during co-contraction of antagonistic ankle plantar and dorsiflexors as compared with isolated plantar flexion using the paired H reflex technique (Bussel & Pierrot-Deseilligny, 1977). In confirmation of the study by Hultborn & Pierrot-Deseilligny (1979) an H reflex (H′), which was conditioned by a previous reflex discharge, was found to be depressed during weak plantar flexion, but facilitated during strong plantar flexion. In striking contrast to this, the H′ reflex was always strongly depressed during co-contraction regardless of the level of contraction. Furthermore, whereas the H′ reflex was facilitated towards the end of a voluntary ramp-and-hold plantar flexion, it was strongly depressed at a similar time during a ramp-and-hold co-contraction.

The arguments favouring the idea that changes in the H′ reflex, when not explained by similar changes in the reference H reflex, may be attributed to changes in *orthodromically* evoked recurrent inhibition were discussed in detail by Hultborn & Pierrot-Deseilligny (1979). The observations in the present paper thus provide evidence that recurrent inhibition onto soleus motoneurones is very differently regulated during plantar flexion and co-contraction. Since both tasks involved the same amount of soleus EMG activity, the motoneuronal discharge driving the Renshaw cells should be roughly similar in the two tasks. The question then arises as to why the amount of recurrent inhibition was, nevertheless, much more pronounced during co-contraction than during plantar flexion. The increase of the H′ reflex during strong plantar flexion and towards the end of the ramp contraction suggests that the activity of the Renshaw cells was depressed despite the pronounced motoneuronal discharge during such strong contractions. Stimulation of supraspinal motor centres in the cat (for references see Baldissera, Hultborn & Illert, 1981) as well as in man (Mazzochio, Rossi & Rothwell, 1994) has been shown to cause an inhibition of Renshaw cells and it is therefore likely that the decreased recurrent inhibition during strong plantar flexion and towards the end of a ramp contraction is caused by descending inhibitory control of the Renshaw cells as already suggested by Hultborn & Pierrot-Deseilligny (1979). The fact that recurrent inhibition was nevertheless very pronounced during strong co-contraction and towards the end of a ramp co-contraction could thus be explained if this descending inhibitory control was not active during those tasks. The large amount of recurrent inhibition during co-contraction would then be explained simply by the motor discharge-induced excitation of Renshaw cells unopposed by any descending inhibition. There is thus no need to suggest an hypothesis for a direct descending facilitation of the Renshaw cells during the co-contraction tasks although this certainly is a possibility.

Irrespective of whether the Renshaw cells are facilitated directly by the brain during co-contraction or simply not inhibited as during strong plantar flexion, the different regulation of the pathway in the two tasks is in line with previous studies which have suggested that the descending control of the spinal segmental motor system is conveyed by different descending pathways during co-contraction and extension–flexion movements (Fetz & Cheney, 1987;

Nielsen & Kagamihara, 1992, 1993, 1994; Nielsen *et al.* 1993). It would not be surprising if this differential control was also reflected in the supraspinal control of Renshaw cells.

In fact, two arguments point against the alternative possibility that the control of soleus-coupled Renshaw cells during co-contraction is only the net result of the effects observed during isolated voluntary plantar and dorsiflexion. Firstly, the net result of the large facilitation of the H′ reflex observed during strong plantar flexion (Hultborn & Pierrot-Deseilligny, 1979; see also Figs 1 and 2) and of the moderate inhibition observed during strong dorsiflexion (Katz & Pierrot-Deseilligny, 1984, their Figs 1 and 2) could very unlikely be the strong inhibition constantly observed here during strong co-contraction (Fig. 2*C*). Secondly, the strong inhibition of the H′ reflex observed during the first 100 ms of voluntary dorsiflexion (Katz & Pierrot-Deseilligny, 1984, and shown here in Fig. 3*C*, column with dots) does not fit the finding that during co-contraction the H′ test reflex was mainly inhibited at the end of the ramp. This strongly suggests a specific control of recurrent inhibition during co-contraction.

We would like to emphasize that this does not exclude the possiblility that the Renshaw cells were also differentially controlled during weak contractions as well as in the first part of the ramp contractions, but if a differential control exists in these cases, it could not be disclosed with the method used in the study.

In addition to the differential supraspinal control, other mechanisms may also contribute to the depression of the H′ reflex during co-contraction. Nielsen, Kagamihara, Sinkjær & Toft (1994) demonstrated that the medial gastrocnemius and peroneal muscles are more strongly activated during co-contraction than during a simple plantar flexion at matched background soleus EMG activity. Since stimulation of group Ib, II and III afferents has been shown to facilitate Renshaw cells (Piercey & Goldfarb, 1974; Anastasijevic & Vuco, 1978) a contribution from discharges of these afferents evoked by contraction of the different muscles acting on the ankle joint during co-contraction is a likely possibility.

Functional considerations

In observing that the distribution of recurrent inhibition onto Ia inhibitory interneurones in the cat always seemed to parallel that of the Ia excitatory input to their corresponding motoneurones, Hultborn *et al.* (1971*c*) suggested that this would 'allow complex movements or postures involving co-contraction of flexors and extensors at the same joint'. It has indeed been demonstrated that the transmission in the Ia inhibitory pathway is depressed during co-contraction tasks in human subjects, thereby allowing an unopposed parallel activation of the two antagonistic muscles (Nielsen & Kagamihara, 1992). From the findings in the present study, and assuming that the measurements reflect not only the excitability of the Renshaw cells but also their activity, it may be suggested that facilitation of Renshaw cells contributes importantly to this depression. In the study by Hultborn & Pierrot-Deseilligny (1979) it was argued that the inhibition of the Renshaw cells observed during strong plantar flexion would assist the voluntary movement both by reducing the inhibition of the agonist motoneurones as well as by increasing the reciprocal Ia inhibition from the contracting muscle onto its antagonists. During co-contraction a compromise between the effects on these two targets (motoneurones and Ia interneurones) is required. By facilitating (or at least not inhibiting) the Renshaw cells during co-contraction it is ensured that the Ia inhibitory interneurones do not compromise the parallel activation of the antagonistic muscles, but at the same time the voluntary motor discharge will be inhibited directly by the recurrent inhibitory action onto the motoneurones. The fact that Renshaw cells are facilitated during co-contraction thus probably explains why subjects are able to produce less voluntary EMG during co-contraction tasks than during isolated contractions of single muscles (Tyler & Hutton, 1986). The findings in the study thus support the hypothesis that the modulation of the motor output through the recurrent inhibitory pathway may help to adjust the muscle tension to provide appropriate levels of muscle stiffness according to any specific task.

The facilitation (or lack of inhibition) of the Renshaw cells during co-contraction may also be advantageous for another reason. Hultborn, Lindström & Wigström (1979) suggested that the recurrent inhibition could serve as a variable gain regulator for the motor output. The inhibition of the Renshaw cells according to their hypothesis would ensure a high input–output gain for the motoneuronal pool, whereas a facilitation would decrease the gain. The facilitation (or lack of inhibition) of Renshaw cells during co-contraction would thus ensure that the gain of the stretch reflex is diminished at the output stage. Previous evidence has suggested that increased presynaptic inhibition of the Ia afferents, which mediate the stretch reflex, diminishes the gain of the stretch reflexes at the input stage during co-contraction (Nielsen & Kagamihara, 1993). If the excitability of both antagonistic motoneuronal pools were allowed to increase excessively during co-contraction, the agonist–antagonist system would have a high risk of breaking into oscillations and clonus (Matthews, 1972). The restriction on the motoneuronal excitability exerted by the Renshaw cells during co-contraction may be of importance in preventing this. This is especially important as stretch reflexes have been shown to increase significantly with strong co-contraction despite a significant presynaptic inhibition of the transmission across the Ia afferent synapses (Nielsen *et al.* 1994).

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