Changes in transmission in the pathway of heteronymous spinal recurrent inhibition from soleus to quadriceps motor neurons during movement in man

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Summary

H reflexes were induced in the human quadriceps muscle by electrical stimulation of the femoral nerve. The reflexes were conditioned by prior stimulation of the inferior soleus nerve. The conditioning stimulus produced an inhibition of long duration (>20 ms). The threshold of this inhibition was at zero soleus motor discharge and the inhibition scaled with soleus motor discharge. It was concluded that the inhibition was a heteronymous recurrent inhibition of quadriceps motor neurons mediated by Renshaw cells which had been activated by soleus motor neuron discharge. This recurrent inhibition declined during voluntary tonic contraction of the quadriceps, falling to zero at around one-third of maximum voluntary contraction. Antagonist contraction and weak co-contraction of the quadriceps and its antagonists did not lead to any significant change in recurrent inhibition. It is concluded that motor commands descending from the brain reduce heteronymous recurrent inhibition during isolated quadriceps muscle contraction, but to a much lesser extent during co-contraction. No evidence was obtained for any descending facilitation of heteronymous recurrent inhibition.

Keywords: recurrent inhibition; spinal cord; motor control; human

Abbreviations: C/T% = conditioned reflex amplitude divided by test reflex amplitude; XMT = electrical stimulation relative to motor axon threshold; %M_max = percentage of the maximum motor discharge; %MVC = percentage of maximum voluntary contraction

Introduction

The function of recurrent inhibition of spinal motor neurons mediated by Renshaw cells has been discussed extensively (Hultborn et al., 1979a; Windhorst, 1996). However, many uncertainties remain. Further insights into recurrent inhibition could be derived from studies of its phylogenetic distribution (Illert et al., 1996) or of the patterns of action in a single species. Monitoring action during movement or monitoring transmission through Renshaw cells during movement may provide further information. The present paper is concerned with monitoring transmission (signal flow) in a heterosynaptic recurrent pathway during simple movements in man.

The techniques currently available for studying recurrent inhibition in man are of restricted use for monitoring transmission during movement. Changes in the firing probability of motor units following motor discharge in other muscles have provided extensive information on the distribution of recurrent inhibition (Meunier et al., 1994), but the method requires constant weak activation of the test muscle, which limits the range of movements that can be studied. Bussel and Pierrot-Deseilligny introduced a method whereby a test H reflex was conditioned by previous reflex discharge in the same pool of motor neurons (Bussel and Pierrot-Deseilligny, 1977). A strong inhibition resulted which was ascribed to a combination of recurrent inhibition and motor neuron after-hyperpolarization. This method can be used to detect changes in homonymous recurrent inhibition, but only in muscles with sufficiently vigorous H reflexes and only by assuming no change in the afterhyperpolarization.
(see review by Mazzocchio and Rossi, 1997a). Mazzocchio and Rossi introduced a pharmacological test (administration of the cholinergic agonist L-acetyl-L-carnitine) (Mazzocchio and Rossi, 1989) that has been valuable for identifying recurrent inhibition, but does not in itself constitute a general method for monitoring transmission other than on a long time scale (Mazzocchio and Rossi, 1997b).

The method described in the present paper stems from the observation made during a study of knee joint receptor actions on human quadriceps motor neurons, that stimulation of soleus motor axons produces a long-latency inhibition of the quadriceps with many of the properties expected of recurrent inhibition (Iles et al., 1990). The present work was designed to test the hypothesis that the inhibition was indeed a heterosynaptic recurrent inhibition of the quadriceps from soleus motor neurons, and to monitor transmission in this pathway during the simple movements of agonist contraction, antagonist contraction and co-contraction. A brief account of some of the observations has been published (Iles, 1996a).

### Methods

#### Subjects

Eight normal subjects aged 20–50 years were studied with informed consent. The procedures were approved by the Oxford Sector ethics committee. Three of the subjects (two male, one female) were studied in greater detail and contributed data to all sections of the results.

#### Procedures

In most experiments the subjects were placed supine. An H reflex was evoked in the right quadriceps muscle by electrical stimulation of the femoral nerve and recorded with surface electrodes over the pure knee extensor vastus lateralis (electrodes ~15 cm proximal to the patella; further details are available in Iles et al., 1990; see also Garland et al., 1994). The stimulus to the femoral nerve was adjusted to produce an H reflex amplitude of ~10% of the maximum motor discharge recorded over the vastus lateralis during supramaximal nerve stimulation.

The quadriceps H reflex was conditioned by prior activation of soleus motor neurons in the same limb. Soleus motor neurons were activated by electrical stimulation of the inferior soleus nerve (single shock; methods are described in more detail in Iles and Pisini, 1992). The strength of electrical stimulation was expressed relative to the motor axon threshold (XMT). Soleus motor discharge (direct with stimuli >1 XMT; H reflex with weaker stimuli) was monitored with recording electrodes placed over the soleus muscle and expressed as a percentage of the maximum motor discharge measured during application of a supramaximal nerve stimulus (%M<sub>max</sub>). If both direct and reflex discharges were present they were summed before being expressed as a percentage of the maximum discharge.

The effects of the conditioning stimulus on the quadriceps H reflex were expressed in the form of conditioned reflex amplitude divided by test reflex amplitude, as a percentage (C/T%). Values of <100% indicate inhibition. Typically 16 or 20 conditioned and test reflexes were measured in a regular or pseudorandom sequence at a repetition rate of ~0.2 Hz (the randomized sequences were used to check that conditioning actions were not a spurious effect of a regular sequence); sequences were repeated four or more times in order to estimate the mean and standard error of C/T%.

Conditioning actions were studied at rest, during quadriceps contraction, during hamstring contraction and during co-contraction of the quadriceps and hamstrings, in all cases with the knee joint maintained in a fully extended position. In order to control the strength of voluntary contraction, additional EMG recording electrodes were placed over the vastus lateralis and biceps femoris. The EMGs were amplified, full-wave rectified and integrated using an averaging filter with an averaging period of 1 s (Garland et al., 1972). The integrated signals were displayed to the subjects so that predetermined levels of contraction could be made [expressed as a percentage of the maximum voluntary contraction (%MVC)].

Two subjects (JFI and SB) were also studied in a sitting position with knee and ankle angles of 135° and 110°, respectively. Other technical details were unchanged.

In one subject (JFI), conditioning actions were studied on a quadriceps motor discharge produced by a corticospinal volley. This was induced by magnetic stimulation of the foot area of the motor cortex (method described in Iles, 1996b).

### Results

#### Time course of conditioning action from soleus to quadriceps

**Assessment with quadriceps H reflexes**

The time course of conditioning of quadriceps H reflexes from direct activation of soleus motor neurons is illustrated in Fig. 1A. In all subjects there was inhibition of the quadriceps H reflex for conditioning intervals of ≥7 ms. The inhibition was maximal at an interval of ~20–25 ms between inferior soleus nerve stimulation and the stimulation of the femoral nerve used to elicit the test reflex. Significant inhibition was found in all seven subjects examined for conditioning intervals between 14 and 35 ms (Wilcoxon test; P < 0.05 for six subjects, P < 0.1 for the seventh).

Significant inhibition was followed by a period of facilitation at conditioning intervals of ~40 ms (except in two experiments in which the time course was investigated while the subjects performed a weak contraction of the quadriceps; see below).

When we used very weak conditioning stimuli (below motor threshold), which elicited an H reflex discharge of soleus motor neurons, there was a smaller inhibition of the quadriceps H reflex extending from 15 to 35 ms (statistically significant for three subjects). There was no facilitation at...
Recurrent inhibition

Fig. 1 Time course of recurrent inhibition. The inhibition of quadriceps motor neuron discharge (expressed as C/T%) is plotted (ordinate) against the time interval between conditioning stimulation of the inferior soleus nerve and the test stimulation (abscissa). The mean and standard error of C/T% were calculated from four or more averages (points plotted ± SE). (A) Data obtained using a quadriceps H reflex as the test reflex and direct stimulation of soleus motor neurons for conditioning (soleus discharge between 50 and 80% M_max in different subjects). Subjects show a significant inhibition for conditioning intervals between 14 and 35 ms. Note that the data for subjects SB and JFI (broken lines) were obtained during a weak voluntary contraction of the quadriceps muscle in a sitting position, and the late excitation seen in the other experiments is absent. The cross-over from inhibition to excitation was consistently at a shorter latency in subject JP (diamonds) than in other subjects. (B) Data obtained using a corticospinal evoked discharge of quadriceps motor neurons as the test discharge. The corticospinal volley was induced by magnetic stimulation of the motor cortex (subject JFI, stimulus set at 71% of maximum output from the Magstim 200 stimulator).

Assessment with a corticospinal evoked motor discharge

The experiments described in the previous section were repeated in one subject (JFI) using a test discharge of quadriceps motor neurons induced by magnetic stimulation of the foot area of the motor cortex in substitution for the H reflex. The conditioning action was very similar: inhibition at an interval of ~30 ms, followed by a tendency towards facilitation at ~50 ms (Fig. 1B).

Scaling of inhibition with conditioning motor discharge

The inhibition of the quadriceps H reflex was measured at the optimal interval for each of three subjects (~25 ms) using conditioning stimuli which recruited varying proportions of the soleus motor neuron pool [expressed as a percentage of the maximal motor discharge recorded from soleus (%M_max)]. Small soleus discharges (<40% M_max) were elicited reflexly as soleus H reflexes with conditioning stimuli below the soleus motor axon threshold, and larger discharges (up to 100%) were elicited by direct stimulation of soleus motor axons with stronger stimuli.

The relationship between quadriceps inhibition and soleus motor discharge is illustrated in Fig. 2A. The curves for the data illustrated in Fig. 2 and from other experiments were slightly convex upwards and were linearized by a power transformation before applying statistical tests (the optimum, i.e. least-squares power transformation, was to raise both C/T% and motor discharge to the power 1.6). The slopes of regressions performed on the transformed data were negative and significantly different from zero (P < 0.001) in all cases.

The regression analyses also enabled the threshold for inhibition to be investigated by projecting the regression lines to the ordinate at zero soleus motor discharge. In no cases were the estimates of C/T% at zero motor discharge significantly different from 100% (98 and 102% for subjects JFI and JP, respectively, in Fig. 2A). In one subject the stimulus to the inferior soleus nerve was increased beyond the value that produced a maximal soleus motor discharge (to 1.22 and 1.59 XMT). There was no further increase in inhibition of the quadriceps.

The relationship between inhibition and soleus motor discharge was also investigated in a second series of experiments. In these experiments the stimulus to the inferior soleus nerve was kept constant at below motor threshold and the soleus H reflex amplitude was modulated by asking the subject to perform small voluntary contractions of the ankle muscles. The intention was to keep the stimulation of sensory axons at a constant level while changing the level of (reflex) activation of motor axons. In these experiments inhibition again scaled with soleus motor discharge (Fig. 2B). The slopes of the regressions of the transformed data were significantly different from zero in four experiments,
including that illustrated in Fig. 2B ($P < 0.01$). The estimates of C/T% at zero soleus motor discharge were not significantly different from 100% (98% in Fig. 2B).

**Effects of muscle contraction**

**Contraction of the test muscle**
The inhibition of the quadriceps H reflex was monitored during voluntary tonic contraction of the quadriceps. Because contraction of the quadriceps leads to an increase in H-reflex amplitude and contraction of the biceps femoris leads to a decrease (for vastus medialis, cf. Sabbahi and Khalil, 1990), the stimulus to the femoral nerve was adjusted to maintain the test reflex amplitude constant at ~10% of the maximum quadriceps motor discharge. Inhibition declined with voluntary contraction of the quadriceps and was completely abolished at around one-third of the maximum voluntary contraction (33% MVC; Fig. 3). The late excitation was abolished at ~20% MVC.

**Agonist, antagonist and co-contraction**
The effects of antagonist muscle contraction and co-contraction were compared with those of quadriceps contraction. Averages were made in the following order: rest; during quadriceps contraction; during hamstring contraction (monitored from biceps femoris); during co-contraction of quadriceps and hamstrings. The cycle was repeated eight or more times in order to estimate the mean and standard error of the measure of recurrent inhibition (C/T%) under the four conditions (Fig. 4).

A non-parametric repeated measures (Friedman) test showed that the medians estimated under the four conditions were significantly different ($P \leq 0.002$). A multiple comparisons (Dunn’s post hoc) test showed that recurrent inhibition was significantly smaller during quadriceps contraction than at rest ($P < 0.05$; see also previous section). Recurrent inhibition during hamstring contraction was not significantly different from rest, but was significantly larger than during quadriceps contraction ($P < 0.05$). Recurrent inhibition measured during co-contraction was not signi-
Fig. 4 Effects of muscle contraction on recurrent inhibition. Estimates of recurrent inhibition (C/T%) were obtained at rest, during quadriceps contraction, hamstring contraction (monitored from biceps femoris) and co-contraction of quadriceps and hamstrings. (A) Subject performing contractions of 10% MVC. Soleus motor discharge (reflex) was 30% M_max. Fifteen averages were used to estimate the mean and standard error. (B) Subject performing contractions of 30% MVC. The soleus motor discharge (reflex) was 20% M_max. Eight averages were used to estimate the mean and standard error. Mean and standard error are plotted for consistency with the other figures even though the data were analysed with a non-parametric test. However, the medians for each column differ by <2% from the means.

Discussion
The nature of the observed inhibitory action on quadriceps motor neurons
The duration of the inhibitory action described in the present experiments is similar to the prolonged reduced firing probability of quadriceps motor units seen after soleus motor neuron activity (Meunier et al., 1994), which has also been interpreted as a recurrent inhibition. The inhibitory time curve (Fig. 1A) is similar to that originally reported for recurrent inhibition in hindlimb extensor motor neurons in the decerebrate cat (Renshaw, 1941). A strong inhibition of quadriceps motor neurons after tibial nerve stimulation was previously noted by Bergmans and colleagues (Bergmans et al., 1978).

At short conditioning intervals (~5 ms) there was a small facilitation, which presumably resulted from group Ia excitation, and this was followed by a phase of inhibition at ~10 ms which has been ascribed to group I non-reciprocal inhibition (discussed in Iles et al., 1990). In four out of five subjects examined there was a period of reduced inhibition at 12–14 ms. It is assumed here that the inhibition at intervals of <13 ms is largely group I-non-reciprocal and that at intervals >13 ms is largely recurrent, but there must be considerable overlap. For further analysis of recurrent inhibition, intervals of ≥20 ms were used, by which time the non-reciprocal inhibition should have been negligible.

The scaling of inhibition with motor discharge seen in Fig. 2 would be expected for recurrent inhibition because Renshaw cell discharge is proportional to monosynaptic reflex motor discharge in the cat (Ross et al., 1972; Hultborn et al., 1979b). The most likely alternative explanation for the long latency and duration of the inhibition is that it is an action of sensory fibres, such as a group I presynaptic inhibition. However, the present inhibition appeared at motor threshold, whereas Ia afferents are recruited at much lower stimulus strengths (around 0.65 XMT), and Iles and Roberts (1987) found no evidence for presynaptic inhibition from soleus to quadriceps Ia afferents (cf. Abbuzzese et al., 1997). Furthermore, the inhibition of cortically evoked quadriceps motor neuron discharge (Fig. 1B) cannot be the result of Ia presynaptic inhibition.

The conditioning stimulus was applied through surface electrodes and would have activated some cutaneous sensory fibres, which could evoke inhibition of quadriceps motor neurons. However, any explanation in terms of sensory actions is rendered less tenable by the experiments in which the stimulus to the soleus nerve (and therefore sensory fibre activation) was kept constant and the H-reflex discharge of soleus motor neurons was modulated in amplitude by voluntary contractions of the soleus or tibialis anterior muscles (Fig. 2B) (cf. Meunier et al., 1990, who use the same argument). Under these conditions the inhibition still scaled with motor discharge, as expected for recurrent inhibition [although in those experiments in which the soleus muscle was contracted some of the effect could have been due to the tonic soleus motor discharge increasing the excitability of soleus-coupled Renshaw cells (cf. Hultborn and Pierrot-Deseilligny, 1979), and the experiment does not exclude the possibility that the strength of recurrent inhibition might have been modulated by the voluntary effort of ankle muscle contraction]. A rather convoluted explanation for our results could be that some sensory action is modulated during
voluntary contraction of ankle muscles to an extent that happens to match the changes in soleus H-reflex amplitude. This explanation can be rejected because the inhibition produced by constant direct activation of soleus motor axons was not modified significantly by voluntary contraction of ankle muscles (examined in two subjects).

The weight of evidence suggests that the long-latency inhibition of quadriceps motor neurons produced by weak stimulation of the soleus nerve is recurrent. However, in the experiments in which a strong soleus nerve stimulus produced a large direct activation of soleus motor neurons, there are at least three possible confounding features: (i) recruitment of high-threshold (group II) muscle afferents from the soleus, which could inhibit quadriceps motor neurons; (ii) stimulus spread to the tibial nerve, leading to activation of group I afferents from plantar muscles, which produce presynaptic inhibition of quadriceps reflexes (Iles, 1996b); (iii) activation of Ib afferents by contraction of the soleus muscle (Binder et al., 1977) and thus group I non-reciprocal inhibition of quadriceps motor neurons. The fact that inhibition did not increase further with supramaximal soleus nerve stimulation tends to diminish the importance of both high-threshold afferents and stimulus spread, but is consistent with a soleus muscle twitch-evoked Ib action. Non-reciprocal group I inhibition from soleus to quadriceps does operate under these experimental conditions (see above) and may well contribute to the long-latency inhibition during strong direct stimulation of soleus motor neurons.

The late excitation seen at ~40 ms in experiments using both H-reflex and corticospinal-evoked quadriceps motor neuron discharge scaled with stimulus strength, not with motor discharge, and is presumably sensory, although there could be an element of post-inhibitory rebound. One way in which cutaneous fibres can produce a long-latency, long-duration excitation is by inhibition of presynaptic inhibition. However, this could not explain the facilitation of quadriceps discharge evoked from the corticospinal tract (Fig. 1B). Furthermore, the excitation disappeared during quadriceps contraction, whereas in the soleus muscle contraction increases the inhibition of presynaptic inhibition by cutaneous afferents (Iles, 1996b). Alternative explanations would be a long-latency spinal or transcortical loop.

**Evaluation of the method for monitoring recurrent inhibition**

The experimental method for assessing recurrent inhibition described in the present paper is simple and direct. It should be possible to extend its use to monitor recurrent inhibition during more complex movements such as those involved in posture and locomotion.

A rather similar method for assessing heteronymous recurrent inhibition, from the medial gastrocnemius to the soleus, was introduced by Rossi and colleagues (Rossi et al., 1994). However, their protocol is limited in two respects. First, because the conditioning and test muscles act around the same joint it would be difficult to activate them independently. Secondly, the conditioning stimulus has to be above the motor threshold, so the inhibition could be contaminated by the effects of twitch-evoked Ib activation. Group I non-reciprocal inhibition is known to exist between the medial gastrocnemius and the soleus (Pierrot-Deseilligny et al., 1981).

Meunier and colleagues (Meunier et al., 1990) demonstrated recurrent inhibition from the quadriceps to the soleus, so our experimental protocol should operate with test and conditioning muscles reversed. However, Iles and Roberts (1987) found some evidence for presynaptic inhibition from the quadriceps to the soleus, which would complicate the interpretation.

**Transmission of heteronymous recurrent inhibition during simple movements**

**Transmission during agonist (test) muscle contraction**

In the present experiments, heteronymous recurrent inhibition declined during quadriceps contraction, with complete abolition at around one-third of maximum voluntary contraction. This result is qualitatively similar to those reported for homonymous recurrent inhibition of the soleus. Hultborn and Pierrot-Deseilligny showed a decline in inhibition during strong tonic and ramp contractions of the soleus (Hultborn and Pierrot-Deseilligny, 1979), though there was no decline during ballistic contractions (Katz et al., 1982). A significant quantitative difference is that in our experiments on the quadriceps the inhibition fell to zero as the contraction exceeded 35% MVC, whereas for the soleus it may still be present at 80% MVC. Two factors could account for this. First, the heteronymous action on the quadriceps is weak anyway (Meunier et al., 1994; their work on motor unit firing probability was done with very weak tonic muscle contraction, which would not by itself lead to much reduction in inhibition). Secondly, homonymous actions will be facilitated because the motor neuron activity during test muscle contraction will activate the coupled Renshaw cells. This could shift the effect of a descending inhibition of Renshaw cells, such as has been demonstrated from the corticospinal system in man (Mazzocchio et al., 1994), to higher levels of contraction.

A second significant difference is that we found no evidence for any facilitation of recurrent inhibition of the quadriceps during weak contraction, whereas this has consistently been found in the soleus. The difference could again be one between heteronymous and homonymous actions, for the reasons described above. Katz et al. (Katz et al., 1982) showed that homonymous recurrent inhibition of the soleus increased just before the start of a ramp contraction, which indicates some descending facilitation. However, there was
no direct evidence for such a descending facilitation during tonic contractions as used in the present experiments.

Loscher and colleagues (Loscher et al., 1996) reported that homonymous recurrent inhibition declines progressively during a 10 min contraction of the soleus at 20% MVC. Our averages each lasted ~1 min (followed by a rest), so there may be an element of such decline in our data. Kukulka and colleagues described experiments from which they concluded that recurrent inhibition increased during a sustained maximal voluntary contraction (Kukulka et al., 1986). We were not able to study contractions of the quadriceps stronger than ~40% MVC because of the requirement to maintain the contraction during the period of data acquisition. Strong contractions may recruit other muscle groups, which could lead to further activation of Renshaw cells (our subjects sometimes tended to co-contract the quadriceps and tibialis anterior).

**Transmission during antagonist muscle contraction**

Katz and Pierrot-Deseilligny reported that homonymous recurrent inhibition of the soleus increases during phasic or tonic (but not ballistic) pretibial flexor muscle contraction (Katz and Pierrot-Deseilligny, 1984). Some of this increase could be due to the activation of peroneal muscles, which send recurrent inhibition to the soleus. However, an increase in soleus recurrent inhibition was also seen just before a ramp pretibial contraction, indicating that there is a descending facilitation of homonymous recurrent inhibition. In our experiments the heteronymous inhibition of the quadriceps did tend to increase during hamstring contraction, but the effect was never significant.

The present experiments have not revealed any significant evidence for descending facilitation of heteronymous recurrent inhibition under any of the conditions investigated, suggesting that the descending controls of homonymous and heteronymous recurrent inhibition may differ.

**Transmission during agonist and antagonist co-contraction**

Nielsen and Pierrot-Deseilligny have reported that the reduction in homonymous recurrent inhibition of the soleus seen during strong plantar flexion is absent during co-contraction of the soleus and pretibial muscles (Nielsen and Pierrot-Deseilligny, 1996). In our experiments the reduction of heteronymous recurrent inhibition of the quadriceps during a weak contraction was much smaller during co-contraction. This fully supports the contention of Nielsen and Pierrot-Deseilligny that during co-contraction the descending inhibition of recurrent inhibition is reduced. The present work does not address the issue of whether heteronymous inhibition of the quadriceps is modulated during co-contraction with ankle muscles. This could be done by performing experiments while the subject is standing wearing a loaded rucksack (which leads to co-activation of the quadriceps and tibialis anterior) (e.g. Iles and Pisini, 1992) and during walking or bicycling movements (which lead to co-activation of the quadriceps and soleus).

**Function of recurrent inhibition in motor control**

The major conclusion of the present work is that heteronymous recurrent inhibition is reduced and eventually abolished during voluntary isolated contraction of a muscle group, but that during co-contraction with the antagonists the reduction is much less. A synthesis of the present results with other published work would suggest that at rest, during weak voluntary and postural contractions, and during antagonist co-contraction, transmission in the recurrent pathway is strong and the gain of the motoneuronal pool and reciprocal inhibitory system is low. During a strong isolated voluntary contraction, transmission in the recurrent pathway is inhibited from the corticospinal system, and the gain of the motoneuronal pool and reciprocal inhibitory system is high. During sustained submaximal contraction there is a progressive shift to the higher gain state.

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