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RESEARCH ARTICLE

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An investigation into mechanisms of reflex reinforcement by the Jendrassik manoeuvre

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Abstract Tendon jerk and H-reflexes are both potentiated by the Jendrassik manoeuvre, but the mechanism of potentiation remains uncertain. We investigated several possibilities in human subjects. Evidence for fusimotor activation during the Jendrassik manoeuvre was sought by recording the tendon jerk reflex as surface EMG in triceps surae after the muscles had been conditioned to leave their spindles in a slack, insensitive state. Interposing a Jendrassik manoeuvre between conditioning and the test reflex should have increased reflex amplitude by restoring spindle sensitivity, but this was not the case. In humans, a close synergist of the triceps surae is the quadriceps. A possible presynaptic disinhibitory mechanism was investigated by testing the effect of a Jendrassik manoeuvre on facilitation of the soleus H-reflex produced by a quadriceps afferent volley. The Jendrassik manoeuvre failed to increase facilitation, contrary to what would be expected if it reduced the level of tonic presynaptic inhibition; the assumption being that the inhibition acts on both homonymous and synergist afferent terminals. The Jendrassik manoeuvre did not increase the level of ongoing EMG in the soleus during a weak voluntary contraction, indicating that it does not operate by direct facilitation of motoneurones. There was found to be less potentiation of soleus tendon jerk and H-reflexes by the Jendrassik manoeuvre under conditions when spindles in the soleus were likely to have a high resting discharge rate. A remaining possibility is discussed: that the Jendrassik manoeuvre operates by modulation of oligosynaptic pathways that may contribute to the largely monosynaptic reflex response. These experiments demonstrate, with new, more sensitive methods than previously used, that neither is the fusimotor system involved in reinforcement nor are direct excitatory or presynaptic disinhibitory effects on motoneurones. While this confirms the previously prevailing view, none of the lingering uncertainties associated with the methods used now remains.

Keywords H-reflex · Tendon jerk · Jendrassik manoeuvre · Human reflex

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Introduction

After many years of service, the tendon jerk reflex continues, today, to be a convenient and useful tool for testing the integrity of peripheral pathways and local, segmental reflexes. In the clinic, it is necessary, at times, to resort to reinforcement manoeuvres, the Jendrassik manoeuvre, to bring out a full-sized reflex. The experiments described here explore possible mechanisms of the Jendrassik manoeuvre.

Following description of the H-reflex (Hoffmann 1922), it was initially thought that the Jendrassik manoeuvre was able to potentiate the tendon jerk, but not the H-reflex (Sommer 1940; Paillard 1955; Buller and Dornhorst 1957). This observation was the basis of the claim that the Jendrassik manoeuvre acted via the fusimotor system, since the H-reflex bypasses the receptors. However, this view was based on a comparison of reflex responses of different size, and subsequent findings demonstrated that reinforcement increases both the tendon jerk and H-reflex, most probably as a result of central influences on motoneurone excitability, skeletomotor and possibly fusimotor (Clare and Landau 1964; Landau and Clare 1964; Bussel et al. 1978). The evidence does not support the view that reinforcement acts selectively through fusimotor neurones (Hagbarth et al. 1975; Burke et al. 1981a, 1981b). For a review of this subject, refer to Murthy (1978). More recent observations using microneurographic recordings have rekindled the debate. Recent studies claim fusimotor activation of spindles during a variety of tasks, including mental activities and reinforcement manoeuvres without concomitant alpha activation or muscle-length changes (Burg et al. 1973; Ribot et al. 1986; Ribot-Ciscar et al. 2000). These findings res-

urrect the idea that reflex reinforcement involves activation of the fusimotor system.

However, can fusimotor activity, in fact, reinforce the stretch reflex? It has long been the established view that activity, particularly that of dynamic fusimotor neurones, is able to increase the size of the tendon jerk by increasing spindle stretch sensitivity. We believe that this is true only under certain circumstances. If the test muscle is conditioned so that its passive spindles show their maximal stretch sensitivity, spindle responses to a tendon tap cannot be increased above the passive level by fusimotor activity, including dynamic fusimotor activity (Wood et al. 1994).

The sensitivity of muscle spindles to stretch, vibration and fusimotor stimulation depends on how they have been conditioned beforehand (for a review, see Proske et al. 1993). When a muscle is passively shortened, the intrafusal fibres of spindles are unable to take up the new, shorter length and fall slack. The spindles are now in a desensitised state since any applied stretch must first take up the slack before they are able to respond. It is fair to assume that, in a normal human subject, in the absence of any deliberate conditioning procedures, most spindles in limb muscles of a reclining, relaxed subject will exhibit various degrees of slack. To standardise spindle stretch sensitivity it is therefore necessary to condition the muscle.

Here, in the face of the new evidence for fusimotor involvement in the Jendrassik manoeuvre, we have taken advantage of the technique of altering spindle stretch sensitivity by introducing intrafusal slack to re-examine the question of the role of the fusimotor system in reinforcement manoeuvres. The method used avoids a number of the potential sources of error inherent in previously applied methods. We report the results of an experiment where the test muscle was conditioned so as to leave its spindles in a slack state, using a technique that has been in use in this laboratory for some time (Gregory et al. 1998). A Jendrassik manoeuvre was then interposed between conditioning and the test stretch reflex. Any fusimotor activation during the reinforcement should have been apparent as an increase in reflex size, because the resulting intrafusal contraction would be expected to remove some of the slack produced by the conditioning. However, no increase in reflex amplitude was seen.

Other possible mechanisms that have been suggested for the Jendrassik manoeuvre include facilitation of alpha motoneurones, a reduction of tonic presynaptic inhibition, a change in motoneurone input impedance and the modulation of oligosynaptic pathways that may contribute to the largely monosynaptic reflex response. Facilitation of alpha motoneurones has been previously reported to not be involved in the Jendrassik manoeuvre (Dowman and Wolpaw 1988), and this result has been confirmed here using a similar technique. Of the remaining possibilities, a reduction in the level of tonic presynaptic inhibition during the Jendrassik manoeuvre is an appealing idea, but there is as yet no evidence for it. Zehr and Stein (1996) looked for interactions between a known presynaptic inhibitory input to the soleus H-reflex and the Jendrassik manoeuvre, but found none, and they concluded that the Jendrassik manoeuvre acted independently of the presynaptic mechanisms available for inhibiting the H-reflex. However, with the method used, that of conditioning the soleus reflex with a common peroneal nerve volley, there was the possibility of occlusion of the Ia presynaptic interneurones by the combination of peripheral and central inputs, thereby masking any disinhibition.

We have re-examined the possibility that the Jendrassik manoeuvre operates by reducing the level of tonic presynaptic inhibition, using a different and, in our view, more sensitive method. The method used sought changes resulting from a Jendrassik manoeuvre in the facilitation of the soleus H-reflex produced by a heteronymous input, that from quadriceps afferents. This method assumes that presynaptic inhibition affects inputs from synergist afferents as well as from the homonymous muscle, as has been previously demonstrated (Hultborn et al. 1987; Wood et al. 1996). If the Jendrassik manoeuvre operates by presynaptic disinhibition, it would be predicted that the facilitation should increase when the quadriceps input, freed from inhibition, becomes more effective during the Jendrassik manoeuvre.

A preliminary account of this work has appeared in abstract form (Gregory and Proske 2000).

Materials and methods

All experiments were carried out on healthy adults, of both sexes. Experimental protocols had been approved by the local Standing Committee on Ethics in Research Involving Humans.

Subjects were seated comfortably in a chair with the hip flexed to about 80° and the knee to about 130°. The right foot was strapped to a footplate, which could be rotated about an axis coincident with the ankle and could be locked in position at 5° increments, enabling the foot to be plantarflexed or dorsiflexed as necessary. Torque about the ankle was monitored by means of strain gauges attached to the axle supporting the footplate. Reflex facilitation was measured as the amplitude of the facilitated reflex divided by the amplitude of the control reflex, expressed as a percentage; 100% therefore indicating that control and facilitated reflexes were of equal size. Where results are quoted with error estimates, these are in all cases SEM values.

Stimulation and recording

H-reflexes were elicited by stimulating the tibial nerve with single constant-current pulses, isolated from ground, delivered between an adhesive electrode (3M Ag/AgCl "Red Dot") attached to the skin in the popliteal fossa and a thin, flexible brass anode taped below the patella. Electrode gel was applied to the anode before attachment to ensure good electrical contact. The recorded H-reflex was invariably preceded by an M-wave, which was monitored to check that stimulating conditions remained constant: a change in the M-wave being taken to indicate that the stimulating electrode had moved.

Tendon jerk reflexes were elicited by tapping the Achilles tendon with a pivoting hammer that delivered blows of constant energy. The hammer was accelerated by a spring and left the spring before striking the tendon; since it was also counterbalanced for

gravity, this ensured that all of the spring's energy was transferred to the tendon. The strength of the blow could be altered by adjusting spring force.

Reflexes were recorded as surface EMG through a pair of adhesive electrodes (3M "Red Dot") attached to the skin over the soleus muscle or with one over the soleus and the other over the gastrocnemius. A third, reference electrode was attached elsewhere on the leg and the three leads taken to an isolated, differential pre-amplifier. Data were recorded on computer with a commercial recording system and software.

Muscle conditioning and the Jendrassik manoeuvre

In some experiments, the triceps surae muscle was conditioned before reflex testing to leave the spindles in a defined, reproducible state. Three conditioning procedures were used, Hold Long, Hold Short and Hold Test. Hold-Long conditioning consisted of dorsiflexing the ankle by 30° for about 10 s before returning it to the initial length, at which the reflex was subsequently elicited several seconds later. At the start of the dorsiflexion, the subject was given a tone instruction to perform a strong, but not necessarily maximal contraction of the triceps surae for about 2 s and then to stay relaxed for the remainder of the dorsiflexed period. Hold-Long conditioning has been shown in animal experiments to leave spindles in a slack, mechanically insensitive condition with a low resting discharge rate. The Hold-Short conditioning procedure was similar, but the ankle was plantarflexed instead of dorsiflexed. Hold-Test conditioning consisted of only the muscle contraction, the foot remaining in the test position. Both Hold-Short and Hold-Test conditioning have been shown in animal experiments to leave spindles in a mechanically sensitive state, with a relatively high rate of resting discharge (Gregory et al. 1990).

The Jendrassik manoeuvre was performed either by squeezing a handgrip or by attempting to pull the clenched hands apart. Initially, it was thought to be important to achieve a reproducible level of muscle force during the performance of the Jendrassik manoeuvre, and this was conveniently arranged by modifying a commercial hand dynamometer so that a tone signal to the subject was generated when a pre-determined level of force was reached. The pulse that initiated the tone was also used to trigger the stimulus for the reflex after a suitable delay. Reflex timing relative to the start of the Jendrassik manoeuvre is important, particularly for the H-reflex, since it has been shown that potentiation is significant over only a narrow range of intervals after the start of the Jendrassik manoeuvre (Delwaide and Toulouse 1983). An LED was illuminated as an instruction to perform the Jendrassik manoeuvre by squeezing the handgrip to a force level set to be about 50% of maximal. The stimulus for the reflex was delivered 300 ms after this level of force had been achieved.

However, no difference was detected between the efficacy of this method and the more conventional procedure of attempting to pull apart the clenched hands. Moreover, subjects reported that repeatedly squeezing the hand grip was uncomfortable and fatiguing, so this method was eventually abandoned in favour of hand clenching. With this method, a tone instruction was given to the subject to commence the Jendrassik manoeuvre, and the reflex was initiated 300 ms later. This interval was occasionally varied in an attempt to improve the situation in subjects with poor potentiation, but this did not produce any noticeable improvement.

Presynaptic disinhibition

In this experiment, the soleus H-reflex was facilitated from two sources, the Jendrassik manoeuvre and the heteronymous input from quadriceps afferents. The early facilitation from the quadriceps input is monosynaptic (Hultborn et al. 1987). To ensure that the effect is monosynaptic, the volley from the soleus afferents must be timed to arrive at the soleus motoneurones within 0.5 ms of the arrival of the quadriceps volley.

For each subject, the correct timing of the two volleys was established in a separate session, since to do this and the main experiment in a single session would have been unnecessarily tiring for the subject. The quadriceps nerve was stimulated with single, constant-current shocks delivered through a hemispherical brass electrode, 2 cm in diameter, placed on the skin over the femoral nerve in the femoral triangle. The anode was a sheet of thin brass placed under the top of the thigh, on which the subject sat. Electrode jelly was used on both electrodes, and the cathode was held down with a weight.

Stimulus strength was adjusted so that there was a significant, but not maximal quadriceps H-reflex, which was recorded with surface electrodes. Maximal quadriceps-reflex amplitude was not determined, because subjects reported unacceptable discomfort before maximal stimulus strengths were reached. The stimulus strength used to elicit the soleus H-reflex was adjusted so that the reflex amplitude was about 20% of the maximal M response in this preliminary session and in the main experiment.

The interval between the soleus and quadriceps stimuli was then varied and a facilitation versus interval curve constructed, from which the optimal interval was determined. Because of the greater length of the soleus afferent pathway, the soleus stimulus had to be delivered appreciably before the quadriceps one for the volleys to arrive at the soleus motoneurones simultaneously. The optimal interval for facilitation presumed to be monosynaptic was between 5.5 and 6.25 ms for the subjects used in this experiment. This interval for a particular subject was found to be quite stable if determined repeatedly in separate sessions. The optimal interval determined in this way was then used in the main experiment at a later date. In all but one subject, the Jendrassik manoeuvre was performed by squeezing a hand grip. The remaining subject commenced with the hand grip, but became fatigued and was re-tested with the hand-clench method.

Facilitation of alpha motoneurones

The experiment was to record the ongoing soleus EMG before and during the performance of a Jendrassik manoeuvre, which in this case was done by hand clenching. Subjects were requested to maintain a weak contraction of the triceps surae muscle, at a strength of 10% of a maximal voluntary contraction, and in most trials were given visual feedback on an oscilloscope of the torque generated by the contraction and of the required torque level. A tone instruction was given to commence the Jendrassik manoeuvre, and 300 ms later an H-reflex was elicited. Some trials were performed without visual feedback of torque. This was done to eliminate the possibility that any increase in EMG and torque during the Jendrassik manoeuvre would not be seen because of voluntary adjustment; although it seems unlikely that appropriate adjustment of torque would be possible in the short time available, less than 300 ms, between the start of the tone and elicitation of the reflex. EMG was recorded from just before the start of the Jendrassik manoeuvre to past the occurrence of the H-reflex. Each of 120 trials with and without a Jendrassik manoeuvre were rectified, averaged and divided into 50 ms bins, then each bin averaged and normalised in each subject to the first control value, just before the Jendrassik manoeuvre, and then averaged across subjects.

Results

Fusimotor activation

This experiment, designed to test for fusimotor activation during the Jendrassik manoeuvre, was performed in five subjects, using the tendon jerk as the test reflex. Before each reflex trial, the test muscle was first systematically conditioned to leave its spindles in either a tight, mechanically sensitive state, by means of Hold-Short conditioning, or a slack, relatively insensitive state by

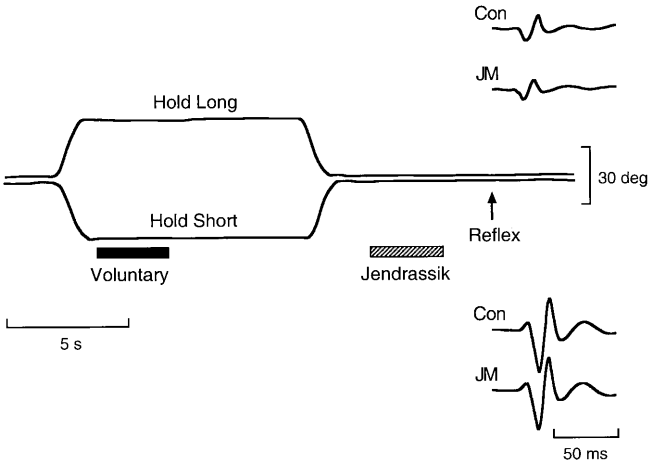

Fig. 1 Illustration of the method used to look for fusimotor activation during the Jendrassik manoeuvre. The *traces* in the centre are records of ankle angle in two trials, one using Hold-Long, the other Hold-Short conditioning. In all trials, a strong voluntary contraction was carried out where shown by the *solid bar*. The *hatched bar* shows the position of the Jendrassik manoeuvre interposed in some trials. The tendon jerk reflex was elicited at the *arrow*. Sample reflexes recorded as surface EMG are shown at the *right*. The *upper pair* show sample control (*Con*) and post-interposed Jendrassik manoeuvre (*JM*) reflexes after Hold-Long conditioning, and the *lower pair* after Hold-Short conditioning. (See text for description of conditions)

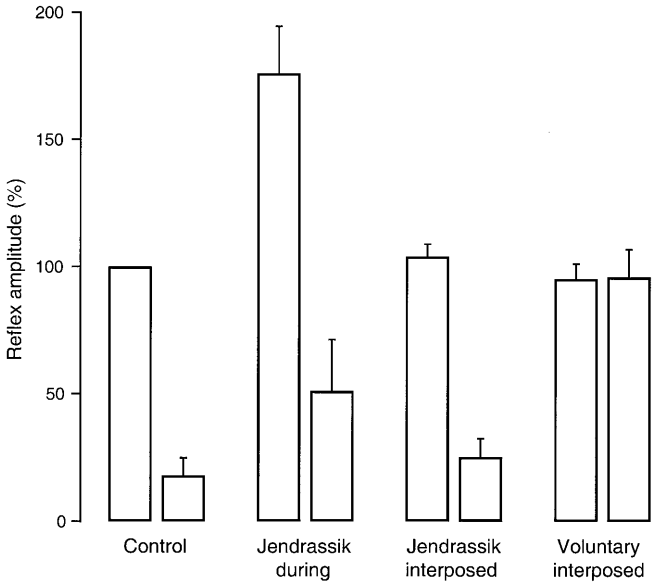

Fig. 2 Summary of the experiment testing fusimotor activation during the Jendrassik manoeuvre. Each *pair of bars* shows means (+SEM) of subjects' mean reflex amplitude after Hold-Short (*left bar*) and Hold-Long (*right bar*) conditioning. From the left are control reflexes (i.e., muscle conditioning alone), reflexes recorded during a Jendrassik manoeuvre, after an interposed Jendrassik manoeuvre and, finally, after an interposed strong voluntary contraction in place of the Jendrassik manoeuvre. Both Hold-Short and Hold-Long reflexes were potentiated during the Jendrassik manoeuvre. An interposed Jendrassik manoeuvre did not significantly increase the Hold-Long reflex, indicating a lack of fusimotor activation, while the interposed voluntary contraction increased the Hold-Long reflex to match the Hold-Short reflex. (See text for description of conditions)

means of Hold-Long conditioning (see Methods and Fig. 1). The reflex was elicited 7 s after the end of conditioning and was recorded with electrodes on soleus and gastrocnemius. That the conditioning procedures were effective is shown by the mean reflex amplitude after Hold-Long conditioning being only 18.0±6.9% of the Hold-Short value.

The results are summarised in Fig. 2. Values quoted are means of the pooled values from the five subjects. For each subject, means of five repetitions were calculated and normalised to that subject's control Hold-Short mean. As expected, both reflexes increased in amplitude when elicited during a Jendrassik manoeuvre, to 175.9±18.6% of the control Hold-Short value after Hold-Short conditioning and from 18.3 to 51.1±20.2% of the control Hold-Short value (an increase of 280%) after Hold-Long conditioning. The Jendrassik manoeuvre was performed by hand clenching.

The test for fusimotor activation required a reinforcement manoeuvre during the period following conditioning and before reflex testing was carried out, the reflex being elicited 2 s after the end of the Jendrassik manoeuvre (see Fig. 1). Fusimotor activation would be expected to restore spindle sensitivity to some degree, because the resulting intrafusal contraction would remove some of the slack left in the spindles after Hold-Long conditioning. The reflex should, therefore, have become larger than the control Hold-Long reflex, possibly approaching the Hold-Short value, while the Hold-Short reflex should have remained unaffected by the interposed Jendrassik manoeuvre. No evidence of fusimotor activation was found. Reflex amplitudes after an interposed Jendrassik manoeuvre were not significantly different from control values, the Hold-Short and Hold-Long means being, respectively, 104.0±4.9% and 25.2±7.2% of the control Hold-Short mean.

Finally, the experiment was repeated, but instead of carrying out the interposed Jendrassik manoeuvre, a strong voluntary muscle contraction was substituted. This time, both the Hold-Short and the Hold-Long reflexes were left about equal in size and, at 95.3±5.9% and 95.8±10.9% of it, were not significantly different to the control Hold-Short reflex. This demonstrated that the small reflex after Hold-Long conditioning could be increased by a preceding intrafusal contraction, produced, in this case, by the voluntary contraction.

Presynaptic disinhibition

The experiment to test the possibility that the Jendrassik manoeuvre might operate by removal of tonic presynaptic inhibition was to look for a change during the Jendrassik manoeuvre in the degree of facilitation of the soleus H-reflex by a heteronymous input, that from quadriceps afferents (Hultborn et al. 1987). The assumption was made that presynaptic inhibition, if present,

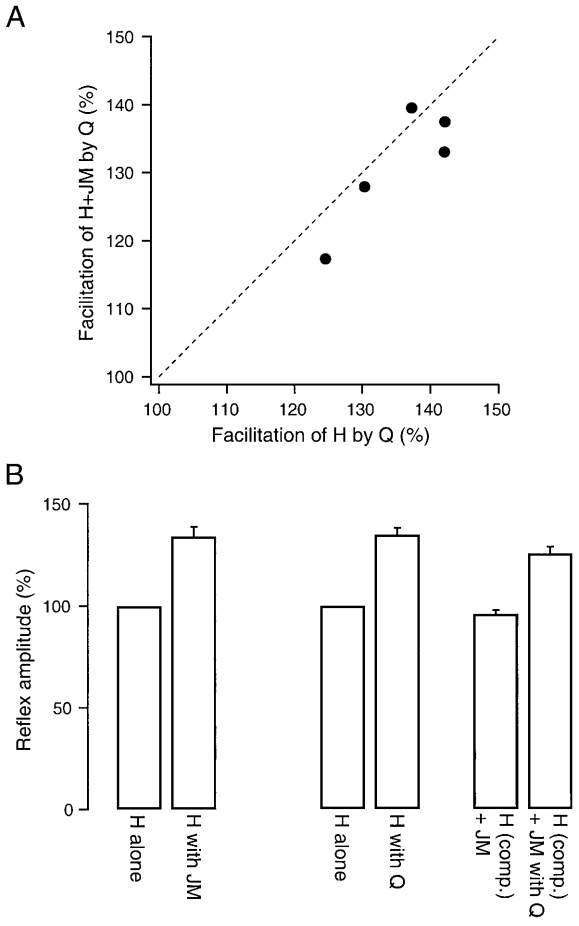

Fig. 3A, B Results from the experiment designed to test for presynaptic disinhibition. **A** Comparison between the facilitation of the soleus H-reflex (*H*) produced by a quadriceps volley (*Q*) (*abscissa*) and the similar facilitation in the presence of a Jendrassik manoeuvre (*JM*) (*ordinate*) for each of the five subjects. Values show the mean amplitude of the facilitated reflex divided by the mean amplitude of the control reflex, expressed as a percentage; 100% therefore indicating reflexes of equal size. The *dashed line* is the line of proportionality. If there was disinhibition during the Jendrassik manoeuvre, the points should lie above the line. **B** Average results from the five subjects. The *left pair of bars* shows that the Jendrassik manoeuvre was effective in potentiating the soleus H-reflex. The *pair of bars in the middle* shows the average facilitation of the soleus H-reflex by the quadriceps volley. On average, facilitation was not increased by the Jendrassik manoeuvre (*right hand pair*), contrary to what would be expected with presynaptic disinhibition

would affect all the afferent terminals on the soleus motoneurones: that is, those of heteronymous as well as homonymous origin. This assumption has been shown to be true in all known cases of presynaptic inhibition. A reduction in presynaptic inhibition should be revealed as an increase in the facilitation of the soleus H-reflex produced by the quadriceps input in the presence of a Jendrassik manoeuvre.

Five subjects were tested in this experiment. Facilitation of the soleus H-reflex was produced by single-shock stimulation of the femoral nerve at a strength weak enough to be comfortable, but sufficient to produce a clear H-reflex in the quadriceps. The quadriceps volley increased the amplitude of the soleus H-reflex to between 125% and 142% of the unfacilitated value in different subjects, with a mean for the five subjects of 135.3±3.4%. The soleus H-reflex elicited during a Jendrassik manoeuvre was 121–147% of the control value, with a mean of 134.1±4.7% (Fig. 3). These and other values quoted in this section were calculated from 15 pairs of control and facilitated reflex trials.

The critical part of the experiment was then to compare the amount of facilitation of the soleus H-reflex produced by the quadriceps volley alone with that observed while the subject performed a Jendrassik manoeuvre. Since the amount of facilitation for a given input has been shown to depend on the absolute size of the reflex (Hultborn et al. 1987), the tibial nerve stimulus was adjusted in strength so that the mean control-reflex amplitudes in the presence and absence of a Jendrassik manoeuvre were within 10% of each other in each subject.

Facilitation during the Jendrassik manoeuvre increased slightly in one subject, but decreased in the other four (Fig. 3A), while the mean facilitation for all subjects fell from 135.3±3.4% to 131.1±4.0% during the Jendrassik manoeuvre (Fig. 3B). This difference was not significant. The lack of a significant increase argues against the idea that the Jendrassik manoeuvre operates by presynaptic disinhibition.

Facilitation of alpha motoneurones

The possibility was investigated that the Jendrassik manoeuvre is mediated by central influences acting to directly facilitate alpha motoneurones, which would raise the excitability of the pool and so should increase the number of motoneurones firing to a given muscle afferent input. It has been recently shown that increases in background EMG are accompanied by increases in H-reflex amplitude (Funase and Miles 1999). An increase in background EMG that facilitates reflexes by a similar amount to a Jendrassik manoeuvre should be detectable by surface electrodes. The experiment consisted of looking for an increase in recorded surface EMG coincident with the performance of a Jendrassik manoeuvre.

When the muscle is at rest, there is no background EMG, and under these conditions there may be facilitation that is effective at potentiating a reflex response, but is insufficient to initiate motoneurone firing and would, therefore, not be detected in the EMG record. The most sensitive condition is to test the effect of a Jendrassik manoeuvre during a weak voluntary contraction. In this experiment, a contraction strength equal to 10% of a maximum voluntary contraction of triceps surae was used. Surface EMG was recorded from the soleus just before and during an H-reflex. In alternate trials, subjects performed a Jendrassik manoeuvre to a tone instruction starting 300 ms before the H-reflex was elicited (Kawamura and Watanabe 1975; Delwaide and Toulouse 1983). The experiment was carried out in six subjects.

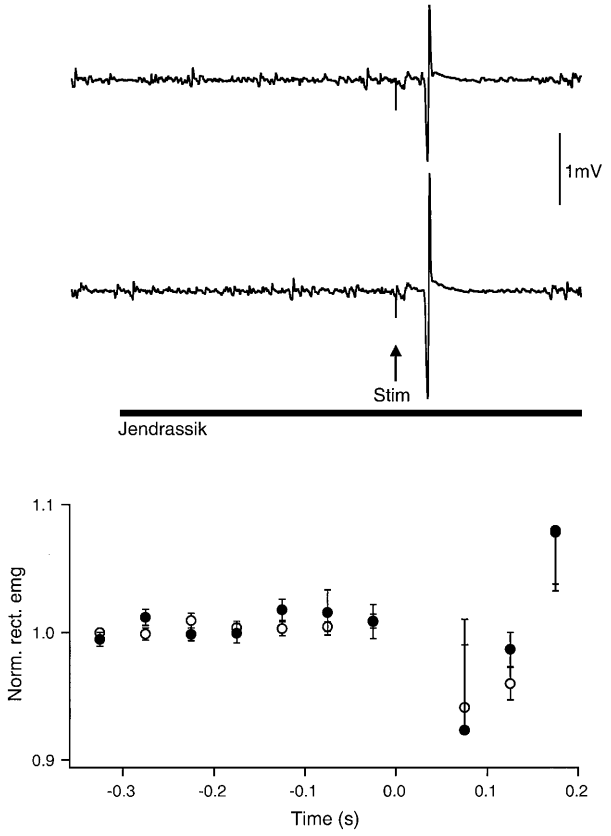

Fig. 4 Testing for facilitation of motoneurones during the Jendrassik manoeuvre. At the *top* is a pair of records of the EMG recorded from soleus while the subject maintained a weak voluntary contraction. An H-reflex was elicited by stimulation at the *arrow*. The *upper trace* is a control record, and in the *lower* the subject performed a Jendrassik manoeuvre starting 300 ms before the reflex. The *graph* below shows the mean (±SEM) results from six subjects with the EMG averaged in 50 ms bins. *Open circles* show control values, *filled circles* values during the Jendrassik manoeuvre. The time axis also applies to the records above

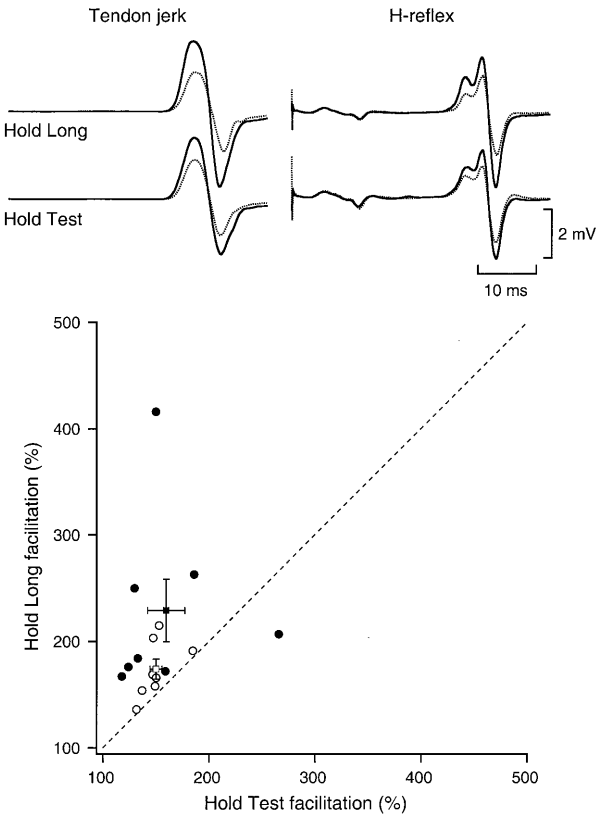

Fig. 5 Effect of muscle conditioning on the Jendrassik manoeuvre. *Above* are shown individual records from separate subjects of tendon jerk and H-reflexes recorded after Hold-Long and Hold-Test conditioning. Records in the absence (*dotted lines*) and presence (*solid lines*) of a Jendrassik manoeuvre are shown superimposed. The graph plots facilitation of the tendon jerk (*filled circles*) and H-reflex (*open circles*) after Hold-Test vs. facilitation after Hold-Long conditioning for eight subjects in each case. Averages across subjects (±SEM) are shown by the *square symbols*. Facilitation was measured as in Fig. 3. It is evident that facilitation was greater after Hold-Long than after Hold-Test conditioning, but there was no significant difference between the H-reflex and tendon jerk. (See text for description of conditions)

The Jendrassik manoeuvre was effective in potentiating the reflex by an average of 119.0±8.2%, but no consistent difference in EMG with a time course similar to that of the Jendrassik manoeuvre was seen between control and potentiated trials (Fig. 4). No difference was noted between those trials where feedback of the torque generated was given to the subject and where it was not.

Effect of muscle conditioning

During the course of these experiments, it was noticed that the Jendrassik manoeuvre seemed to be more effective after Hold-Long than after Hold-Short or Hold-Test muscle conditioning. This suggested that muscle conditioning might provide a tool for revealing the mechanism of reinforcement. It was decided to explore this point systematically, for both the tendon jerk and H-reflex. Eight subjects were tested for each reflex.

The experiment consisted of recording alternating control and Jendrassik manoeuvre-potentiated trials after Hold-Long and Hold-Test conditioning. Since muscle conditioning also affects reflex amplitude, stimulus strength for the H-reflex was adjusted so that the mean control reflexes after Hold-Long and Hold-Test conditioning did not differ by more than 15% in each subject. This adjustment was necessary because, as noted before, the amount of facilitation produced by the Jendrassik manoeuvre varies with reflex amplitude. A similar adjustment was made to the strength of the tendon tap used to elicit the tendon jerk reflex, but in the opposite direction, since the tendon jerk reflex is larger after Hold-Test than after Hold-Long conditioning but vice versa for the H-reflex (Gregory et al. 1990).

For the tendon jerk, one subject's facilitation by the Jendrassik manoeuvre was greater after Hold-Test than Hold-Long conditioning, but the other seven showed more facilitation after Hold Long (Fig. 5). The mean facilitation of the eight subjects was significantly greater

after Hold Long (*t*-test, *P*=0.04). A similar result was obtained for the H-reflex (Fig. 5), but here all eight subjects showed more facilitation after Hold-Long than Hold-Test conditioning, and the mean for all subjects was significantly greater after Hold Long (*t*-test, *P*=0.02). The ratio of the mean facilitation after Hold Long to that after Hold Short was greater for the tendon jerk (1.44) than for the H-reflex (1.16), but this difference was not significant (*t*-test, *P*=0.14).

Discussion

None of the mechanisms investigated here were able to explain the reflex reinforcement seen with the Jendrassik manoeuvre. This is in agreement with previous work (Hagbarth et al. 1975; Burke et al. 1980, 1981b; Dowman and Wolpaw 1988; Zehr and Stein 1996), with the exception of a recent report that, during the Jendrassik manoeuvre, there was an increase in the stretch sensitivity of human spindles without any concomitant activation of alpha motoneurones (Ribot-Ciscar et al. 2000). The increase was quite moderate and was seen during sinusoidal muscle stretch in only four of the nine spindles studied. It is difficult to know how much contribution this would make to the increase in reflex amplitude during the Jendrassik manoeuvre, and, of course, an increase in spindle sensitivity cannot explain the observed potentiation of the H-reflex.

Previous microneurographic studies (Hagbarth et al. 1975; Burke et al. 1980, 1981b) failed to find any evidence of fusimotor activation during the Jendrassik manoeuvre, and this was also the case here, where a quite different method was used to detect the fusimotor activity. Our method used reflex amplitude as the test measure, and this avoids two possible weaknesses of some other methods. The first of these is that fusimotor activation may not lead to any alteration in the size of the test reflex unless the muscle history of contraction and length changes is controlled for. This was the conclusion of Wood et al. (1994), who showed that there is little difference in the size of tendon jerk reflexes elicited in the cat with and without fusimotor stimulation, provided that the spindles in the test muscle are in a tight, mechanically sensitive condition. Therefore, unless muscle history is known, there must be some doubt about any negative conclusions drawn from such an experiment.

A second possible source of error is that there may be some weak, inadvertent contraction of the test muscle while the subject is performing the Jendrassik manoeuvre (Hagbarth et al. 1975), and if weak enough, this may remain undetected in the EMG trace, yet still include fusimotor effects. It is known that there is co-activation of alpha and gamma motoneurones even during very weak voluntary contractions (Wilson et al. 1997; Gregory et al. 1998). The method used here avoids this problem because the Jendrassik manoeuvre is completed before the reflex is elicited. While we cannot rule out some weak activation of the fusimotor system during the Jendrassik manoeuvre, our results indicate that it would be insufficient to significantly potentiate the tendon jerk reflex and, therefore, cannot be the main mechanism by which reinforcement operates.

The possibility that the Jendrassik manoeuvre might involve direct facilitation of alpha motoneurones was first investigated in a study by Dowman and Wolpaw (1988). They were, however, unable to find any evidence in support of such a mechanism. Since changes in background EMG were likely to be small, we chose to increase the number of trials over which EMG was averaged (120, c.f. 10 by Dowman and Wolpaw 1988). However, we have similarly found no evidence of changes in excitability of the motoneurone pool, as expressed in EMG levels, during the Jendrassik manoeuvre.

If both alpha motoneurone facilitation and fusimotor activation can be ruled out as mechanisms for the Jendrassik manoeuvre, the most likely remaining possibility is some mechanism acting presynaptically. Reduction of tonic presynaptic inhibition seems a possibility since it has been found that motoneurones are subjected to some degree of tonic presynaptic inhibitory control, particularly during locomotion (Faist et al. 1996). In a recent study, Zehr and Stein (1996), using reinforcement in combination with a common peroneal nerve volley, failed to find any evidence for presynaptic inhibition and concluded that "the Jendrassik manoeuvre acts independently of the presynaptic mechanisms that can inhibit the H-reflex."

A difficulty with a test for presynaptic inhibition using a common peroneal volley, the D_1 inhibition (Tanaka 1974; El-Tohamy and Sedgwick 1983), is that there is the risk of occlusion of the presynaptic pathway by the combined inputs from the conditioning peripheral volley and activity of central origin generated by the Jendrassik manoeuvre. Thus, Iles (1996) showed complete occlusion between cutaneous and corticospinal inputs in their respective actions to reduce presynaptic inhibition of soleus Ia afferents from common peroneal stimulation. Similarly Faist et al. (1996) suggested that one explanation for the reduced D_1 inhibition during gait in human subjects was occlusion as a result of saturation of the presynaptic network. The advantage of the method of heteronymous testing, which has been used here, is that it assesses ongoing presynaptic inhibition and not the presynaptic inhibition evoked by a conditioning volley (Faist et al. 1996). This avoids the problem of saturation between inputs acting via common pathways. Other saturation effects are equally unlikely. The quadriceps volley was submaximal, and the facilitation produced by it was similar to the facilitation by the Jendrassik manoeuvre. The soleus reflex was only 20% of the amplitude of the maximal M wave and, under these conditions, any increase in the efficacy of the quadriceps input would be expected to be seen. Yet we were unable to detect any increase in heteronymous facilitation, only a small, insignificant decrease. We conclude that presynaptic disinhibition acting at the Ia motoneurone synapse is not responsible for the reflex reinforcement.

A clue about the mechanism of reinforcement might be that the Jendrassik manoeuvre was more effective in facilitating the reflex when it followed Hold-Long conditioning than after Hold-Test conditioning (Fig. 5). How could the effectiveness of the Jendrassik manoeuvre be reduced after Hold-Test muscle conditioning? The two forms of conditioning result in a difference in the ongoing level of resting discharge from spindles. A known effect of the increased resting discharge after Hold-Test conditioning is to depress the amplitude of reflexes originating from volleys in the afferents with the higher discharge, probably by a mechanism involving depression of transmitter release (Hultborn et al. 1996; Wood et al. 1996).

Our studies have revealed that the overriding determinants of the size of the tendon jerk reflex are mechanical factors, while for the H-reflex it is the level of spindle resting activity. This does not mean that the tendon jerk reflex is unaffected by resting discharge, but rather that mechanical effects predominate. Hold-Long conditioning leaves the intrafusal fibres of muscle spindles slack and that accounts for the resultant low level of resting discharge. It leads to a reduced stretch sensitivity of spindles and, therefore, a small tendon jerk. The H-reflex is large because there is little transmitter depletion from the low level of background activity. As mentioned earlier, it is likely that, in subjects whose muscles have not been deliberately conditioned, some slack will be present in spindles. That, in turn, leads to a sizeable potentiation of the tendon jerk from reinforcement, as is routinely observed in the clinic (Fig. 5).

Our finding that there is more reflex potentiation after Hold-Long conditioning suggests that, during post-activation depression, reinforcement is less effective in increasing the reflex. The findings of Nielsen et al. (1993) that reflexes in spastic, spinal-cord-injured patients are less prone to post-activation depression than in normal subjects suggests the experiment of testing potency of reinforcement in these patients to determine whether they show more reinforcement than normal subjects.

Another factor that could be considered is the greater level of subliminal input to the motoneurones from the increased afferent traffic following Hold-Test conditioning. That this is unlikely to be important was shown by Wood (1997), where, in animal experiments using similar methods to those used here, increasing the afferent traffic from a close synergist (lateral gastrocnemius) by selective muscle conditioning had no effect on homonymous (medial gastrocnemius) reflex amplitude. It seems likely then that an increased homonymous input would similarly have no effect either on reflex amplitude or on the relative increase in amplitude produced by the Jendrassik manoeuvre.

Our findings lead us to the conclusion that the Jendrassik pathway does not involve the motoneurones directly, but must act at an interneuronal site somewhere upstream. It has been argued that the tendon jerk and H-reflexes are not strictly monosynaptic and that there are opportunities for oligosynaptic contributions (Burke et al. 1984). The Jendrassik pathway may involve neurones in this oligosynaptic pathway. If the excitability of such postulated interneurones varied inversely with prevailing levels of background activity in the Ia afferents, it would account for the larger effect of reinforcement on the reflex after Hold-Long conditioning. It remains for future experiments to determine if these oligosynaptic pathways are indeed involved in the Jendrassik manoeuvre and whether the effects of their modulation are sufficient to produce the observed degree of reflex potentiation.

To conclude, we have presented a new method for demonstrating that the Jendrassik manoeuvre does not act through the fusimotor system to potentiate the tendon jerk and H-reflex. We have confirmed that there is no detectable direct action on motoneurone excitability nor is there any evidence that reinforcement acts by presynaptic disinhibition. The only clue we are left with is a difference in the amount of reinforcement when muscle spindles are in a mechanical state where their generated level of background activity remains low. That finding will be the subject of future experiments.

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