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Research report

Mental simulation of an action modulates the excitability of spinal reflex pathways in man

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Abstract

The question of whether mental simulation of an action has an effect on the spinal reflex circuits was examined in normal humans. Subjects were instructed either to exert or to mentally simulate a strong or a weak pressure on a pedal with the left or the right foot. Changes in the H- and T-reflexes activated by electrical and mechanical stimuli were measured on both legs during motor performance as well as during mental simulation of the same task. Asynchronous EMG activity of the soleus muscles was simultaneously recorded. Reflex excitability increased during performance of the pressure. It was larger when the H-reflex was triggered in the muscle involved in the task as compared to the contralateral side. Because actual performance modified the tension of the tendon and the location of the stimulus, ipsilateral changes of T-reflex amplitude could not be evaluated. Mental simulation of foot presure in this condition resulted in a large increase of spinal reflex excitability, which was only slightly weaker than the reflex facilitation associated with the actual performance. Changes in T-reflex amplitude, but not in H-reflex amplitude, depended upon the lateralization and force of the simulated pressure, being larger in the leg involved in the simulation than in the contralateral leg, and larger for a strong than for a weak simulated movement. EMG activity was found to be weakly increased during mental imagery. This increase was significantly, although slightly, modulated by the lateralization and intensity of the imagined movement. However, no correlation was found across subjects between reflex amplitude and the amplitude of EMG activity.

Keywords: Motor imagery; Spinal reflex; Human; Cognitive neuroscience

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1. Introduction

Actions can be intended, imagined or simulated without being executed. These internally generated cognitive states raise interesting physiological problems as to their neural substrates and to their relationships to actually executed actions. It could be that simulated actions pertain to the same category of neural processes as those which are involved in preparing actually executed actions [11,29].

An argument favoring the hypothesis of common mechanisms for motor imagery and motor preparation is provided by the measurement of autonomic responses. Decety et al. [13] measured cardiac and ventilatory activity during actual and mental locomotion at different speeds. They found a covariation of heart and respiratory rates with the degree of imagined effort. The amount of vegetative activation of a subject mentally running at 12 km/h was similar to that of a subject actually walking at 5 km/h (for a confirmation of this result, see [49]). It could have been that, during motor imagery tasks, the subjects produced a proportional increase in muscular activity by co-contracting antagonistic muscles groups, and that these peripheral changes were sufficient to explain the vegetative changes. This possibility was excluded by another experiment in which intramuscular metabolites (PCr, Pi) and pH were measured directly by nuclear magnetic resonance spectroscopy during both actual and mental leg exercise at two levels of work [14]. During actual exercise, the heart and respiration rates increased in relation to the level of work. End-tidal pCO_2 was unaltered. NMR spectra showed a drop in phosphocreatine (PCr) and an increase in inorganic phosphate (Pi) concentrations. Intracellular pH fell to 6.65 at maximal effort. During motor imagery, both heart and respiratory rates increased immediately after mental exercise started, proportionally to the amount of simulated

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exercise, whereas NMR spectra remained unchanged with respect to the resting values. Thus, because the vegetative activation observed during motor imagery was greater than required by an increase in metabolic demands, it was likely to be due to activation of a central mechanism. This effect can be considered as a vegetative response pertaining to the normal pattern of activation which occurs during motor programming. In this way the central structures responsible for motor programming would anticipate the need for energetic mobilization required by the planned movement, an optimal mechanism for anticipating the forthcoming metabolic changes (by increasing the perfusion of muscles with oxygenated blood) and shortening the intrinsic delays needed for heart and respiration to adapt to effort (for a complete review, see Requin et al. [36]).

These findings raise the problem of determining the degree of activation achieved during motor imagery by neural structures more directly related to motor execution. This can be done by studying the changes in excitability of spinal reflex pathways. Previous work by Bonnet and Requin [3] showed that muscular response to a sudden stretch of the involved muscle varied during the foreperiod of a voluntary movement. The M1 component of the response increased during the early part of the foreperiod and then decreased, whereas the M2 component increased continuously. The M2 increase was interpreted as a presetting of motor pathways for a fast execution of the movement, whereas the selective depression of M1 was interpreted as due to a presynaptic inhibition for blocking the spindle afferents during the preparatory period. Such a sensitivity of spinal reflexes to the central influences associated with motor preparatory processes (for a review see [4]) suggests that a similar modulation should also be observed during simulated movements. In the present experiment, monosynaptic reflexes elicited by direct nerve stimulation or by tendinous stretch were compared during a voluntary isometric muscular contraction and during mental simulation of the same contraction.

2. Methods

2.1. Apparatus

The subject sat in a reclined position in an armchair placed in a dimly illuminated and sound-attenuated room, and especially designed to evoke and record spinal reflexes from the soleus muscles. Subject's feet rested on two pedals. Verbal instructions, pre-recorded on files, were given by a loudspeaker located behind the subject, who was also provided with a switch in order to signal any unexpected problem during the experiment.

2.2. Subjects

Twenty-six naive subjects followed a first training session. Two of them were excluded because of an abnormal reflex asymmetry, and four because of the presence of a large increase in EMG background activity during mental simulation. Twenty subjects (10 males and 10 females), aged 21–33 years (except one of 54), participated in the experiment. All subjects were right-footed, except two (one male and one female) who were ambidextrous. Subjects gave their informed consent to the experimental procedures. The experiments were carried out in accordance with the guidelines of the Declaration of Helsinki.

2.3. Procedure

After a brief explanation of the reflex stimulation techniques and general procedure, subjects were given the Sheehan [43] questionnaire for assessing their ability to evoke mental images, facilitating their understanding of task instructions, and training them for simulation set. About 20 min were necessary to answer this questionnaire, after which the task instructions were given. Instructions required the subject either to exert, or to mentally simulate, an isometric pressure on the pedal, until s(he) received the instruction to relax. Instructions also indicated the side (left or right) of the foot with which the pressure was to be exerted and the strength (strong or weak, approximately 2% and 10% of maximum voluntary contraction) of the pressure (see below). In order to maintain stable the position of the Achilles tendon on which the mechanical stimulation triggering T-reflexes was applied, the subject was instructed that the actual or imagined foot pressure had to be exerted by the toes and foot sole, without lifting the heel. Subjects were informed that in some trials (control trials) they would have nothing to do but rest. They were authorized to close their eyes in order to facilitate the actual or imagined performance of foot pressure.

2.4. EMG and spinal reflex recordings

EMG activity of the soleus muscle on both sides was recorded by bipolar surface electrodes located at the level of the distal third of the leg, 3 cm apart. After being amplified, reflex EMG responses were digitized (1 kHz), full-wave rectified, and stored on hard disk. After the session, data were displayed on a CRT for measuring the latency of the reflex response in order to adjust an electronic time window of 20 ms width (the signal was integrated during this period). In most cases, the latency of the reflex EMG response was around 30 ms for H-reflexes and 35 ms for T-reflexes. Further, the soleus EMG was continuously monitored, after high-gain amplification, on the CRT screen and stored during 0.5 s preceding each reflex stimulation in order to measure background EMG activity. H-reflexes were evoked and recorded simultaneously in the right and left soleus muscles, according to the standard methodology [24]. The intensity of the stimulation was adjusted in order to obtain, at rest, a reflex EMG whose amplitude was between 25% and 50% of the maximum

H-reflex. In some subjects, the presence of a stable, small direct motor response due to the electrical stimulation of alpha motoneuron axons was noticed. This allowed asserting the consistency of the H-reflex stimulation, even at the time of strong voluntary isometric plantar flexion, in contrast with the obvious fluctuations of the mechanical tendinous stimulation which were observed in some subjects. T-reflexes were evoked bilaterally by a tendon tap (8 ms duration) provided by a vibration stimulator (Bruel and Kjaer, type 4809). As usual, the amplitude of the T-reflex EMG response was between two and four times lower than that of the H-reflex EMG response.

2.5. Experimental design

Each of the eight types of trials in which the subject had to either exert or simulate a foot pressure was defined by three parameters: (1) the type of activation, either performing or simulating the movement; (2) the level of activation, either weak or strong; and (3) the leg involved, either left or right. Successive events of a trial were monitored by a 486 PC. Two seconds after the verbal pre-recorded instruction was given, the bilateral reflex stimulation was triggered. The subject had to maintain the required activation until the ''rest'' instruction, which was given 2 s later. After 8 s, a new trial started.

In a series of 72 trials, 24 randomly distributed control trials formed the reference sample for evaluating the EMG reflex response amplitude. In the 48 experimental trials, each combination (type of activation × level of activation × side) was tested six times. Each subject performed the task during two series of trials, T-reflexes being recorded in the first series (session 1) and H-reflexes in the second series (session 2).

2.6. Statistical analysis

For averaging the EMG reflex responses recorded on different subjects in spite of the large between-subject variability, raw data were transformed into standardized Z scores ($Z = \mathrm{M} - \mathrm{m}/\mathrm{S.D.}$). For each series of trials, the mean amplitude (m) and standard deviation (S.D.) of the EMG reflex responses recorded during the 24 control trials were calculated. They were then used to convert the mean amplitude (M) of the EMG reflex responses recorded when the foot pressure was either performed or imagined into Z scores, which express the deviation of M from m in S.D. units. Both the EMG reflex responses and background EMG raw data were analyzed with a repeated measures analysis of variance (ANOVA).

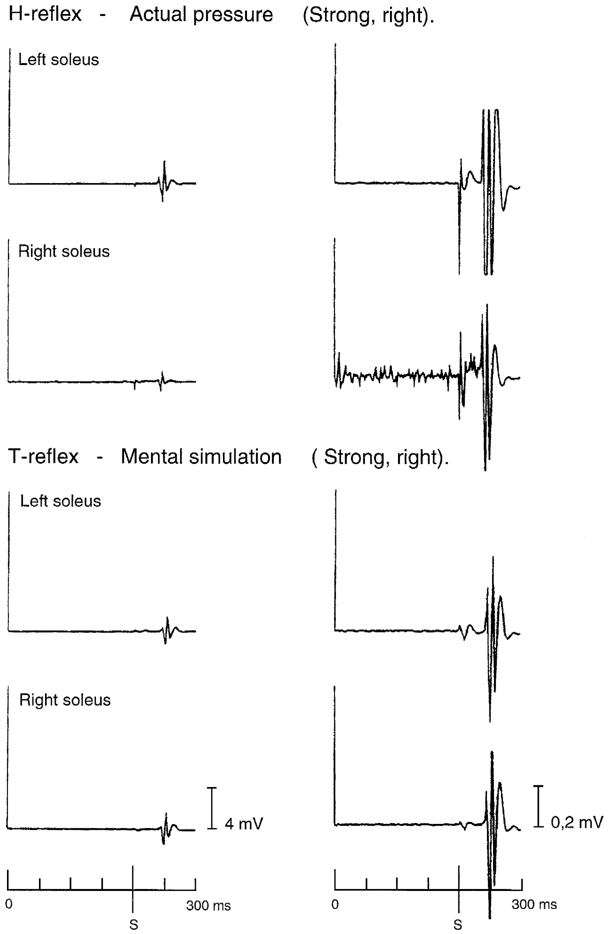


Fig. 1. Raw soleus EMG data from one subject. The upper part depicts an example of H-reflex for actual pressure. The lower part depicts an example of T-reflex during mental simulation. The right column shows the same data with higher amplification.

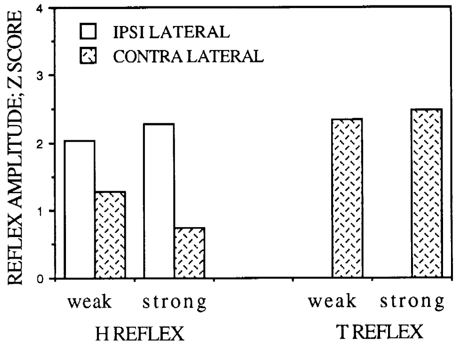
3. Results

Fig. 1 illustrates an individual case for the T-reflex facilitation during mental simulation of a foot pressure. Mean changes in H- and T-reflex amplitudes in the various experimental conditions are shown in Fig. 2. In all these conditions, spinal excitability was found to increase in reference to the distribution of control reflexes. However, because performing the movement modified the tendon tension and/or the location of the vibration stimulator and, therefore, could result in changes in the intensity of the mechanical stimulation, ipsilateral changes in spinal excitability during actual foot pressure could not be correctly evaluated using T-reflex amplitude. For this reason, T-reflex data were analyzed during simulated foot pressure only.

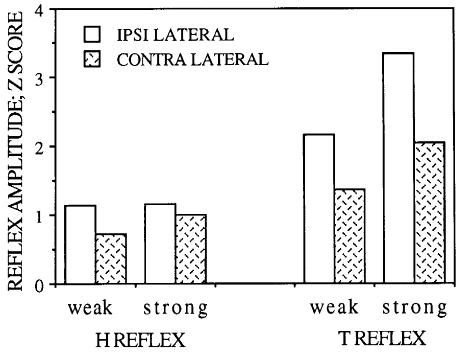
3.1. Changes in H-reflex amplitude during actual foot pressure

A three-factor (right vs. left leg × ipsilateral vs. contralateral leg to movement side × strong vs. weak pressure) ANOVA was performed on H-reflex data only.

A large increase (between 1 and 2 S.D. of the control sample distribution) of spinal excitability was found during



ACTUAL PRESSURE



SIMULATED PRESSURE

Fig. 2. Mean reflex amplitudes (expressed in Z score with reference to the control level) of the Hoffmann reflex (H-reflex, left part) and the tendinous reflex (T-reflex, right part) evoked in both soleus muscles simultaneously as a function of execution (upper part), or mental simulation (lower part) of a 4 s unilateral voluntary muscular contraction (weak or strong) of one of the soleus muscles. Averaged data for 20 subjects. Data for both legs being pooled.

movement performance (Fig. 2, top), which was larger when H-reflexes were triggered in the involved ($Z = 2.17$) than in the contralateral ($Z = 1.01$) muscle ($F_{1-19} = 7.25$, $P < 0.025$), but did not differ as a function of the performing leg ($F_{1-19} < 1$) and of the intensity of the foot pressure ($F_{1-19} < 1$). However, the ipsilateral vs. contralateral difference in H-reflex amplitude was larger when the pressure was strong than when the pressure was weak ($F_{1-19} = 5.49$, $P < 0.05$), this interaction being larger for the left than for the right leg ($F_{1-19} = 4.61$, $P < 0.05$).

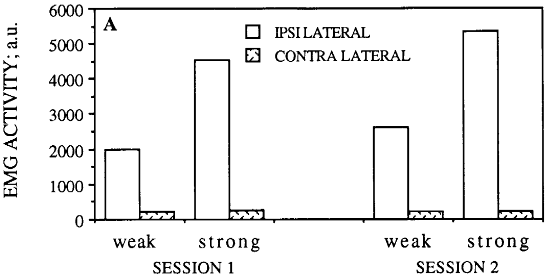
3.2. Changes in H-reflex amplitude during actual vs. simulated foot pressure

A four-factor (actual vs. simulated foot pressure × right vs. left leg × ipsilateral vs. contralateral side × strong vs. weak pressure) ANOVA was performed on H-reflex data only.

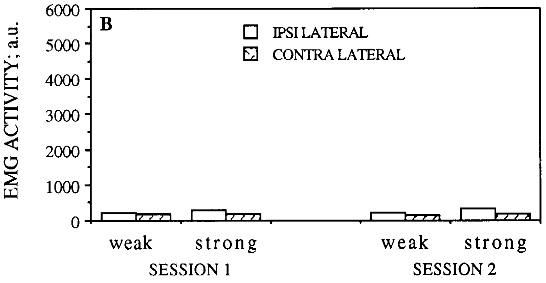
The difference in the increase of H-reflex amplitude between the conditions in which the muscular contraction was either performed or imagined was found to be marginally significant ($F_{1-19} = 3.73$, $P < 0.10$), being only slightly larger in the former than in the latter condition (compare Fig. 2, top left with Fig. 2, bottom left). This increase in H-reflex amplitude was larger when the reflex was triggered ipsilaterally than contralaterally to that contraction ($F_{1-19} = 8.49$, $P < 0.01$), a difference which was larger when the contraction was actually performed than imagined ($F_{1-19} = 4.85$, $P < 0.05$). H-reflex amplitude did not differ between right and left legs ($F < 1$) and did not depend upon the strength of foot pressure ($F < 1$).

3.3. Changes in spinal excitability during simulated foot pressure

A four-factor (H- vs. T-reflexes × right vs. left leg × ipsilateral vs. contralateral side × strong vs. weak pres-



ACTUAL PRESSURE



SIMULATED PRESSURE

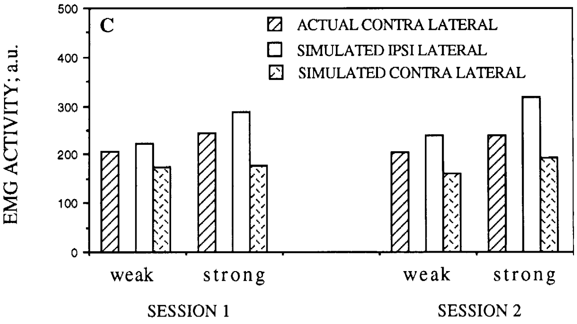


Fig. 3. Mean amplitude (arbitrary units) of the integrated surface EMG activity recorded in both soleus muscles as a function of actual (A) or simulated (B) activation of one unilateral voluntary muscular contraction (weak or strong) of one of the soleus muscles. Two successive sessions. C describes the same data with a different scale in ordinates. Note that the electronic noise of the amplifiers, when disconnected from the subjects, corresponded to 150 units (a.u.) on the ordinates. Data are shown separately for the muscle ipsilateral to the simulation, and for the muscle contralateral to the simulation, data for both legs being pooled. Averaged data for 20 subjects.

Table 1
Values of correlation coefficients calculated between background EMG amplitude and H- or T-reflex amplitude for the four conditions of mental simulation

		Weak left	Weak right	Strong left	Strong right
H-reflex	ipsi	0.16	0.04	−0.02	−0.09
	contra	0.03	−0.03	−0.13	0.63 [a]
T-reflex	ipsi	−0.07	0.08	0.32	0.01
	contra	0.02	−0.01	−0.26	−0.23

Calculation across the 20 subjects.
[a] $P < 0.05$ level of significance (df 18) = 0.44.

sure) ANOVA was performed for both H-reflex and T-reflex data.

The increase in spinal reflex amplitude was found to be larger for T- than for H-reflexes ($F_{1-19} = 16.155$, $P < 0.001$), larger when reflexes were triggered ipsilaterally than contralaterally to the simulated action ($F_{1-19} = 13.373$, $P < 0.01$), and larger when a strong than a weak foot pressure was simulated ($F_{1-19} = 12.924$, $P < 0.01$). Moreover, the effect of foot pressure interacted with the effect of the type of reflex, being larger for T- than for H-reflexes ($F_{1-19} = 11.519$, $P < 0.01$) (Fig. 2).

Since changes in H- and T-reflexes differed greatly and no difference between legs was found, two-factor (ipsilateral vs. contralateral leg to the simulated action side × strong vs. weak foot pressure) ANOVAs were separately performed on T- and H-reflexes, data for both legs being pooled (cf. Fig. 2, bottom). Both H- and T-reflex amplitudes were larger when triggered in the leg ipsilateral than contralateral to the side of the simulated action ($F_{1-19} = 4.901$, $P < 0.05$; and $F_{1-19} = 9.858$, $P < 0.01$, respectively), whereas only T-reflex amplitude differed according to foot pressure, being larger for a strong than for a weak pressure ($F_{1-19} = 14.323$, $P < 0.01$).

3.4. Changes in EMG activity

As shown in Fig. 3A, changes in EMG activity were obviously larger in the muscle actually involved in pressure performance than in the contralateral, non-involved muscle, and larger when foot pressure was strong than weak. The interaction between the effects of these factors was also expected, the difference in EMG changes between the muscles involved and not involved in performing the pressure being larger for a strong than a weak foot pressure.

Changes in EMG activity when the pressure was simulated (Fig. 3B) were considerably weaker than when actually performing the pressure. However, these slight EMG increases were modulated by experimental factors, being larger in the leg ipsilateral than in the leg contralateral to the side of the simulated pressure ($F_{1-19} = 8.38$, $P < 0.01$, in the first T-reflex session, and $F_{1-19} = 10.475$, $P < 0.01$, in the second H-reflex session), and larger when the simulated foot pressure was strong than weak ($F_{1-19} = 5.749$, $P < 0.05$; and $F_{1-19} = 4.573$, $P < 0.05$, in sessions 1 and 2, respectively).

The same EMG data – except for the muscle involved in actually performing the foot pressure – are shown, with expanded ordinates, in Fig. 3C. Note that the electronic noise of the amplifiers when disconnected from the subject corresponded to about 150 units on the ordinates. The increases in EMG activity recorded in the muscle contralateral to the leg involved in the pressure actually performed and in the simulated pressure statistically differed in session 1, but not in session 2.

In order to determine whether these EMG changes actually played a role in increasing spinal excitability and/or spindle sensitivity and were sufficient to explain the large increase of H- or T-reflexes, correlation coefficients were calculated across the 20 subjects, between EMG amplitude and reflex amplitude for the mental simulation condition. As shown in Table 1, only 1 out of 16 coefficients reached the level of significance.

4. Discussion

The main results of this experiment may be summarized as follows: (1) mental simulation of a movement results in a large increase in spinal reflex pathway excitability, which is only slightly weaker than the reflex facilitation associated with the current performance of the same movement; (2) changes in T-reflex amplitude, but not in H-reflex amplitude, depend upon the lateralization and force of the simulated movement, being larger in the leg imagined to be involved in the movement than in the contralateral leg, and for a strong than for a weak simulated movement; and (3) when strongly amplified, the weak EMG activity found during mental imagery is significantly, although slightly, modulated by the lateralization and intensity of the imagined movement.

Although the changes in EMG activity during mental imagery were about 20 times less than during actual contraction and less than twice the amplifier noise, the question arises whether these changes can account for those in reflex amplitude. The question of the relationship between the excitability of spinal reflex pathways and background EMG activity is far from being unequivocal and remains a matter of controversy in the literature. Several experiments have demonstrated a roughly linear positive relationship between the amplitude of the H-reflexes and the EMG activity of voluntary, postural or reflex muscular origin [2,9,10,26,27,30,42]. Similarly, Bedingham and Tatton [1] found that the amplitude of the M1 component of the stretch reflex increased gradually with the force of a voluntary muscular contraction. Such a positive relationship, however, was not always observed. For example, an intense vibration of the tendon triggers simultaneously a tonic muscular activity (the tonic vibration reflex) and a strong inhibition of the H-reflex [19]. Such a depression was also shown for the T-reflex by Scheirs and Brunia [41], when inducing very low levels of voluntary tension in the soleus muscle. Furthermore, Ruegg et al. [39] did not find any effect on H-reflex amplitude of varying a voluntary muscular activation. Similarly, Toft et al. [47] have shown that the amplitude of the M1 and M2 components of the stretch reflex did not depend upon the level of the background soleus muscle contraction. The lack of any systematic correlation at an inter-subject level between the changes we observed in reflex amplitude and in EMG activity thus makes it quite unlikely that the former resulted from the latter.

However, whereas the H-reflex facilitation was only slightly affected by the side of the imagined movement, and not at all by its force, this was not the case for the T-reflexes. Not only were the T-reflexes much more facilitated than H-reflexes but, moreover, they were found to be quite sensitive to the side and intensity of the muscular activation. Both reflexes are conveyed via the same monosynaptic neuronal pathways but the H-reflex, which is triggered by the electrical stimulation of Ia fibers, short-cuts neuromuscular spindles, while the T-reflex, which is triggered by a tendon tap, is a response to stretching these spindles. Insofar as the sensitivity of neuromuscular spindles to muscular stretch is under the control of gamma-motoneurons, an increase in excitability of T-reflex, but not of H-reflex, responses would result from a selective increase in the excitability of gamma-motoneurons.

The comparison between the changes in H- and T-reflex amplitudes for evidencing a spindle activation has often been considered as a disputable method [7,48]. Some differences between H- and T-reflexes may explain why their amplitudes do not necessarily change in parallel: (1) the Ia afferent volley which evokes the T-reflex is less synchronized than that resulting from the electrical stimulation of the nerve [8]; (2) the electrical stimulation also activates the inhibitory Ib afferents; and (3) the threshold for activating the same number of motoneurons being higher for the mechanical than for the electrical stimulation, only those motoneurons which are the most excitable and most sensitive to central influences are involved in producing the T-reflex response. The intervention of all these factors cannot be viewed, however, as sufficient to explain why H- and T-reflexes differed so markedly during mental simulation and, thus, to discard the role played in this difference by spindle activation.

On the other hand, the hypothesis of a spindle activation triggered by mental simulation of a movement fits well the data provided by the present experiment. First, it is well known that spindle proprioceptive afferences play a role not only during movement execution but also for organizing the motor output during self-generated actions [23,34]. For example, in human passively executed movements or vibrations of the tendon strongly facilitate, via spindle activation, the initiation of voluntary movements when such an initiation has became difficult, or even impossible, after a lasting immobilization or a cerebral lesion [5]. Second, the fact that only T-reflexes showed changes in amplitude as a function of the side and force of the imagined movement supports the hypothesis of a spindle activation during mental simulation. Third, the slight changes observed in background EMG activity during mental simulation might also result from such a spindle activation, which could be conveyed, at least partly, by the hypothetical skeleto-fusimotor beta-motoneurons [31,25]. During mental simulation, where peripheral motor effects are blocked, only a weak proportion of the smallest dynamic beta-motoneurons would be activated according to the size principle that would explain both the small increase in background EMG activity and the large increase in T-reflex pathway excitability.

Whatever the precise mechanism of the large facilitation of spinal motor structures during movement simulation, the problem remains to understand how and where motor output is blocked, taking into account that a number of neural structures involved in motor control have been found activated during the process of motor imagery. SMA and premotor cortex were found to be activated by Roland et al. [38], Fox et al. [16], Decety et al. [12] and Stephan et al. [44] using positron emission tomography. Stephan et al. [44] noticed that during imagination the SMA foci were located more anteriorly than during execution. They concluded that functionally distinct premotor areas were involved during preparation and imagery. At the subcortical level, cerebellum [40] and basal ganglia were also found to be activated [15].

At the moment, the available evidence indicates that the primary motor cortex is silent when no execution occurs. However, recent observations using transcranial stimulation showed an increase in excitability of primary motor cortex. Hallett et al. [21] reported that the increase in size of the excitable area devoted to finger movements, which is observed as movements are repeated over training periods (this fact was reported in animal experiments by Grunbaum and Sherrington as early as in 1903 [18]), can be observed for imaginal training as well. A relevant observation was made by Gandevia and Rothwell [17]: they showed that ‘‘concentrating’’ on one hand muscle without activating it increased the effect of subthreshold electric stimulation of the cortical area corresponding to that muscle (and not of other muscles). Thus, there is a selective enhancement of cortical responsiveness during imagery or related processes.

The possibility that motor cortex would be activated suggests that motor output must be blocked somewhere downstream to M1. Hultborn and Pierrot-Deseilligny [26] have claimed that the recurrent inhibition acts like a gain tuning device of the discharge frequency of the motoneurons as a function of corticomotoneuronal excitatory influences. It is well known, indeed, that the activity of Renshaw cells is under the control of central nervous structures [32]. The recurrent inhibition could thus be viewed as the spinal mechanism which would be put into play by descending pathways to reduce the number of motoneurons activated during movement simulation. In man, such a reduction of the motoneuronal discharge frequency is larger as the muscular activation is weaker [33]. In this way, the mental simulation of a movement should be considered as a tiny muscular contraction. This mechanism would offer an example of parallel processes in motor control, whereby motoneuron activation and tuning needed for execution would be paralleled by inhibition during imagery. The possibility of an incomplete inhibition would explain the frequent residual EMG activation observed during motor imagery, as first shown by Jacobson [28] and later also by Shaw [45,46], Hale [20], Wehner et al. [50], Harris and Robinson [22] and the present results.

Note that a spindle activation may also be hypothetized during the preparatory period of an RT task, in which an increase of the background EMG activity was often observed [6]. However, in contrast to mental simulation, motor preparation is characterized by the intervention of a strong inhibitory mechanism which blocks, at the presynaptic level, Ia afferents controlling the motoneurons of the muscles involved in the performance of the forthcoming movement, explaining the corresponding decrease in reflex pathway reactivity [6,37]. In conclusion, it would be more appropriate to compare mental simulation and action, rather than preparation. Mental simulation could be defined as the intention not to execute the movement, i.e., as a negative intention, while preparation is the intention to execute a movement, which is transitorily suspended until the decision to perform is made. The mechanisms by which the motor effect of the primary motor cortex – which may be viewed as very similar to that triggering a real action – is actively inhibited clearly differ for preparation and simulation. In the former case, this motor effect would be blocked by a widespread inhibitory mechanism acting at the spinal level to protect motoneurons against a premature triggering of action [35]. In the latter case, the excitatory motor output would be paralleled by an inhibitory motor output from motor cortical areas, which would be, in some way, the ‘‘negative image’’ of the motor command.

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