Changes in Reciprocal Inhibition Across the Ankle Joint With Changes in External Load and Pedaling Rate During Bicycling

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INTRODUCTION

Bicycling is a recently developed movement form, which is almost exclusively seen in human subjects. Bicycling resembles other forms of rhythmic locomotion such as walking, running, and swimming, and it is reasonable to assume that the brain has used similar strategies to control bicycling as these more “natural” forms of locomotion. Although we have detailed knowledge of how the brain and spinal cord control locomotion in animals, it is only quite recently that it has become possible to address similar issues in human experiments (see reviews by Capaday 2002; Dietz 2001; Nielsen 2002), and only a few studies regarding the central control of bicycling have been published (Boorman et al. 1992; Brooke et al. 1992; Pyndt and Nielsen 2003). Consequently, we still have insufficient information about the central control of human locomotion in general and of bicycling in particular. In this study, we addressed how reciprocal inhibition between antagonistic muscles is regulated during bicycle movements.

Reciprocal inhibition is essential for normal extension-flexion movements to occur because it ensures that antagonistic muscles are relaxed when the prime mover is activated. Experiments in the cat have documented that so-called Ia inhibitory interneurons are organized to ensure this inhibitory control (see Lundberg 1970 and Hultborn 1972 for reviews). The interneurons in the pathway are activated in parallel with the antagonist motoneurons, both by descending motor tracts and peripheral sensory afferents, such as the group Ia afferents, which have given the interneurons their name (reviewed by Edgley 2001). Subsequent human experiments have documented that the activity of the interneurons is regulated accordingly: prior to and during activation of ankle dorsiflexors transmission in the Ia inhibitory pathway to ankle plantarflexors is increased, thereby ensuring that these muscles remain relaxed during the movement and that unwanted stretch reflex activity is not evoked (Crone et al. 1987; Nielsen et al. 1992; Tanaka 1974).

It was an early idea that reciprocal inhibition via the Ia inhibitory pathway could be an integrated part of the spinal network responsible for eliciting the alternating flexor-extensor activity, which characterizes locomotion. However, Pratt and Jordan (1987) demonstrated that, although Ia inhibitory interneurons were modulated rhythmically during fictive locomotion in the cat (i.e., alternating rhythmic activity in motor nerves evoked in the decerebrated cat by stimulation in the mesencephalic locomotor region) and probably contributed to the inactivation of the antagonists in the appropriate phases of the movement, the rhythmic alteration continued after the...
The inhibitory effect of the interneurons was blocked by strychnine. The interneurons are therefore not an integrated part of the network generating the rhythmic alternation but seem to be controlled by this network as well as by sensory and descending pathways. In human subjects, transmission in the Ia inhibitory pathway seems to be regulated in a similar way as in the cat (Petersen et al. 1999). In the stance phase of walking reciprocal inhibition from ankle dorsiflexors to the active ankle plantar-flexors is thus small, whereas it is large in the swing phase where the ankle dorsiflexors become active.

The aim of this study was to investigate whether reciprocal inhibition is also regulated in this way during bicycling. This would further strengthen the idea that the brain uses similar strategies to control bicycling as other forms of locomotion. Bicycling furthermore provides an opportunity to study how the brain controls reciprocal inhibition in relation to changes in external load and the rate of movement. We report that reciprocal inhibition to ankle plantar-flexors measured in early downstroke during bicycling gradually decreases as the load is increased. These findings demonstrate that reciprocal inhibition is specifically controlled in relation to bicycling.

METHODS

Twenty subjects, ages 22–39 yr (10 females; 10 males), participated in the study. Not all subjects participated in all parts of the study. The subjects received written and oral information about the procedures of the experiments before giving their written consent. The experiments were conducted in accordance with the Helsinki declaration and approved by the local ethics committee.

The study consisted of two parts: part 1, reciprocal inhibition during tonic plantar flexion and bicycling; and part 2, reciprocal inhibition during bicycling at different loads and cadences.

General setup

The subjects pedaled with their feet strapped to the pedals on a bicycle ergometer (Monarch 834E), modified to monitor the position of the crank continuously, at an external load from 0.5 to 2.5 kg (frictional torque ranging from 5 to 25 N) at 60 RPM (power output 30–150 W) and/or at an external load of 1.0 kg (frictional torque of 10 N) at 30, 60, and 90 RPM.

Recordings

EMG recordings were made from the right medial tibialis anterior (TA) and soleus (SOL) with bipolar (1-cm² recording area; 2 cm between poles) Ag-AgCl electrodes placed over the belly of the muscles. The EMG signals were amplified (2,000–5,000 times), band-pass-filtered at 25–1,000 Hz, and sampled at 2,000 Hz before being stored on a PC for later analysis. The signals were recorded in a window from 50 ms before until 200 ms after stimuli (see Fig. 1B).

Stimulations

To evoke reciprocal inhibition of SOL, stimulation of TA afferents was applied by bipolar stimulation of the peroneal nerve (PN). The anode was placed distal and lateral to the insertion of the patellar ligament and the cathode just below the neck of the fibula above PN. The stimulation electrodes were secured with adhesive tape to prevent them from moving during the experiment. Care was taken that the electrodes were placed so that the TA motor threshold was below the motor threshold of the peroneal muscle group. This was done by the investigator holding his fingers on the insertion tendons of TA and peroneus muscles, simultaneously perceiving the threshold of the muscle activation. The stimulation intensity of PN was adjusted to 1.2 times TA motor threshold throughout the experiments. At this stimulation, intensity activation of the part of the PN innervating the peroneus muscle group could not be avoided in most subjects, but

FIG. 1. Effect of peroneal nerve (PN) stimulation on soleus (SOL) EMG during tonic contraction. A: connection between the tibialis anterior muscle (TA) afferents and the SOL motoneurons, believed to be responsible for the observed effects. B: rectified averaged SOL EMG of 100 recordings with (black line) and 100 recordings without (gray line) stimulation of PN during a moderate level of tonic plantar flexion (approximately 10% of maximal voluntary contraction). C: inhibition of the SOL EMG during 6 different levels of tonic plantar flexion. To illustrate the inhibition, the abscissa of the plot is limited to the part where the inhibition is observed, from 35 to 65 ms after PN stimulation. Note that the ordinates are different in the right and left columns. Black line shows the rectified averaged SOL EMG after stimulation; gray line shows the SOL background EMG. Each line is an average of 100 recordings. D: linear regression between the level of SOL background EMG and recorded inhibition at 4 levels of plantar flexion. $R^2 = 0.90; P < 0.05$. 

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activation of the peroneus muscles were only minor compared with TA.

The SOL H-reflex was evoked by monopolar stimulation of the right tibial nerve (TN). The anode was placed above the patella, and the ball-shaped cathode was pressed into the popliteal fossa. The stimulation electrodes were secured with adhesive tape to prevent them from moving during the experiment. Seven to 10 crank-angles during the crank-cycle were investigated. At each crank-angle, the maximal muscle response \( M_{\text{max}} \) after TN stimulation was recorded, and the H-reflex was afterward adjusted to approximately 5% of \( M_{\text{max}} \). The H-reflex was kept low, since it is not possible to elicit an H-reflex larger than 5–10% of \( M_{\text{max}} \) during late downstroke and upstroke in most subjects.

**Part I: reciprocal inhibition during tonic plantar flexion and bicycling**

This part of the study consisted of two different types of experiments. In the first experiment, the inhibition of the SOL EMG evoked by PN stimulation was measured at different levels of tonic plantar flexion and compared with the inhibition during bicycling. In the second experiment, the SOL H-reflex was conditioned by stimulation of PN at rest and during bicycling at 60 RPM and an external load of 1.0 kg.

**EXPERIMENT 1: INHIBITION OF THE SOL EMG DURING TONIC PLANTAR FLEXION AND BICYCLING.** The subjects \((n = 12)\) sat in a reclining armchair performing four to six different levels of tonic plantar flexion and compared with the inhibition during bicycling. In the second set-up, the SOL H-reflex was conditioned by stimulation of PN at rest and during bicycling at 60 RPM and an external load of 1.0 kg.

**EXPERIMENT 2: CONDITIONING OF THE SOLEUS H-REFLEX BY PN STIMULATION.** At rest, a time-course of the effect of PN stimulation on the SOL H-reflex \((5\% \text{ of } M_{\text{max}} \text{, see below})\) was constructed. Conditioning test intervals of 0, 1, 2, 3, 4, and 5 ms were used. The conditioning test interval, at which the largest inhibition of the SOL H-reflex was observed, was used in the rest of the experiment (see Fig. 3A). Crone et al. \(1990\) showed that the amount of inhibition of the SOL H-reflex depends on the size of the control H-reflex and that the largest inhibition is usually recorded for reflexes around 20–50% of \( M_{\text{max}} \). When comparing the amount of inhibition in different situations such as different crank-angles during bicycling, it is therefore important to ensure that the control H-reflex has a comparable size in the investigated situations. In most subjects, it is not possible during upstroke of bicycling to obtain a test-reflex larger than 5% of \( M_{\text{max}} \). Caution was therefore taken to adjust the test-reflex to approximately 5% of \( M_{\text{max}} \) throughout the experiment. During the crank-cycle, at 0, 23, 45, 67, 90, 135, 180, 225, 270, and 315° after TDC, \( M_{\text{max}} \) was found, and the unconditioned SOL H-reflex was adjusted to approximately 5% of \( M_{\text{max}} \) before evoking a minimum of 10 conditioned and 10 unconditioned H-reflexes. Conditioned and unconditioned H-reflexes were altered randomly and evoked every 2–3 s. The very first H-reflex in each trial was discarded to avoid any influence of postactivation depression (Crone and Nielsen \(1989\)).

**Part II: reciprocal inhibition during bicycling at different loads and cadences**

This part of the study consisted of three different set-ups. In the first set-up, the inhibition of the SOL EMG after PN stimulation was measured at different levels of tonic plantar flexion. In the second and third set-up, the subjects pedaled on the ergometer-bicycle with three to five different loads at 60 RPM and with different cadences at an external load of 1.0 kg. Whether the second or third set-up was performed first was randomly chosen. This part of the experiment was performed as described above in **part I, experiment 1**. In brief, 100 sweeps with and without stimulation of PN were recorded at different levels of tonic plantar flexion and during different bicycling set-ups. Inhibition was, similar to above, calculated as the difference between the rectified and averaged SOL EMG with and without stimulation during bicycling (see Fig. 1B). Trials with and without stimulation were alternated randomly with \(\geq 2\) s between stimulations. During tonic contraction, the amount of inhibition and the level of SOL background EMG data were fitted to a first order regression line, and the equation of the regression line was used to calculate the expected inhibition at the different levels of SOL background EMG during bicycling (see **Fig. 1, C and D**, as previously described by Petersen et al. \(1999\), comparing tonic contraction and walking). The calculated inhibition was later compared with that actually recorded (see below). Since the regression between background EMG and inhibition was essential for the comparison between inhibition during bicycling and tonic plantar flexion, only data from subjects where a significant regression between background EMG and inhibition was found during tonic contraction were used for further analysis (15 subjects volunteered for the experiments; in 12 subjects, a significant linear regression was found between background EMG and inhibition during tonic plantar flexion). The reason why we found no regression in the three subjects excluded was mainly because the post hoc analysis showed that the stimulation intensity used was not strong enough to evoke a clear detectable inhibition in the background EMG.

During bicycling, the subjects pedaled at 60 RPM with an external load of 1.0 kg. The effect of PN stimulation on SOL EMG was investigated at 0, 23, 45, 67, 90, 135, and 180° after top dead center (TDC; where the pedal-arm of the investigated leg is at the top position, see **Fig. 2A**). In some subjects, SOL EMG activity was present from 23 to 135° after TDC, only. The inhibition was therefore not calculated for 0 and 180° after TDC. The inhibition of the SOL EMG during bicycling was calculated as described during tonic contraction, because the area between the integrated and averaged SOL EMGs with and without PN stimulation (see Figs. 1C and 2A).

**Data analysis**

Inhibition was calculated as the area between the rectified averaged **unconditioned SOL EMG** and the rectified averaged **conditioned SOL**
EMG. The start- and end-time for the inhibition was determined by visual inspection. During the different tasks (tonic contraction and bicycling at different loads and cadences), the time where the inhibition started and especially the time where the inhibition ended varied. To be able to compare the levels of background EMG during the different tasks, the window, where the level of background EMG was measured, was set to be the same during all tasks for each individual subject. The size of the window was determined by the subject’s first start-time and latest end-time of inhibition of all the measurements recorded.

Data analysis was performed with SigmaStat statistical software (SigmaStat for Windows Version 2.03, SPSS). The difference between calculated and recorded inhibition was tested using a Wilcoxon’s signed rank nonparametric test. The difference between conditioned and unconditioned SOL H-reflex was tested using the Student’s t-test. To test for differences in the amount of inhibition during bicycling at different loads and at different cadences, a one-way repeated ANOVA was used. Throughout the study, the level of significance was set at 0.05. Where nothing else is stated, the values reported are mean ± SE.

RESULTS

Part I: reciprocal inhibition during tonic plantar flexion and bicycling

Figure 1. C and D, shows the amount of inhibition of the SOL EMG during tonic plantar flexion at different levels of
background EMG for one representative subject. The correlation coefficient ($R^2$) for the relation between recorded inhibition and background EMG was 0.98 for this subject. For the population of subjects, the average $R^2$ was calculated to 0.88 ± 0.03 ($n = 12$). Figure 2A shows the background EMG, and the insets show the effect of the PN stimulation on the SOL EMG at seven different crank-angles investigated during bicycling at 60 RPM and an external load of 1.0 kg. From the regression line between the inhibition and background EMG during tonic plantar flexion, it was possible to calculate the size of inhibition corresponding to each of the EMG levels recorded during bicycling. The result of this calculation is shown as black columns in Fig. 2B for each of the seven crank-angles. In the same graph, the amount of inhibition that was actually recorded at the seven crank angles is shown as gray columns. It is noticed that the amount of inhibition recorded during bicycling was smaller than the amount of inhibition calculated from the regression line between the inhibition and the background EMG activity during plantar flexion. This was also the case in the other subjects as evidenced from the population average shown in Fig. 2C. To pool data from different subjects, the size of the regression line for the data recorded during bicycling was on average 63% ± 21% of the slope during tonic contraction.

**FIG. 3.** Conditioning of the SOL H-reflex with PN stimulation. A: time course of the SOL H-reflex conditioned with stimulation of PN for 1 representative subject at rest. Arrow indicates conditioning test interval (CTI) where the largest inhibition of the reflex was observed (conditioned reflex was 28% of the unconditioned reflex. Unconditioned reflex was on average 6% of $M_{max}$). This CTI was hereafter used throughout the experiment. B: size of the PN conditioned SOL H-reflex at 8 different crank-angles during pedaling at 60 RPM at an external load of 1.0 kg for the same subject as in A. During bicycling, the average test reflex was 7 ± 1% of $M_{max}$. Bottom: EMG of SOL and TA during the crank-cycle. C: population average of PN conditioned SOL H-reflex during the crank-cycle. On average, the conditioned H-reflex was significantly smaller than the unconditioned H-reflex at 225 and 270° after TDC ($P < 0.05$, *Student’s $t$-test; **at 315° after TDC, the $P$ value was 0.055). Bottom: average SOL and TA EMGs are superimposed.
Modulation of disynaptic reciprocal inhibition

It is not possible to evaluate the amount of reciprocal inhibition from averages of EMG activity when no EMG activity is present, and a full evaluation of the modulation of reciprocal inhibition during the crank cycle is not possible with that method. We therefore also investigated the modulation of reciprocal inhibition evaluated by the H-reflex technique. Data from these experiments are presented in Fig. 3. Figure 3A shows a time course of the effect of PN stimulation on the SOL H-reflex for one representative subject at rest. The largest inhibition was found at a conditioning test interval of 2 ms (the unconditioned H-reflex for one representative subject at rest. The largest inhibition was found at a conditioning test interval of 2 ms (the unconditioned H-reflex was on average 4.27 ± 0.96% of $M_{\text{max}}$) for the same subject. In Fig. 3C, the same is presented for the population average ($n = 9$). As can be seen, the conditioned H-reflex was significantly smaller than the unconditioned H-reflex at 225 and 270° after TDC (Student’s t-test, $P < 0.05$). At 315° after TDC, the $P$ value was 0.055. Figure 3C reflects the trend observed in seven of nine subjects that reciprocal inhibition was most pronounced prior to or when TA was active.

Figure 3, B and C (bottom), shows the activity of SOL and TA. On average, SOL was active from 10 ± 5 to 149 ± 1° (28 ± 15 to 413 ± 6 ms) after TDC, whereas TA was active from 16 ± 7° before TDC to 19 ± 9° after (44 ± 20 ms before TDC to 53 ± 26 ms after).

Part II: reciprocal inhibition during bicycling at different loads and cadences

For the subjects participating in this part of the study, the regression between the average background EMG and the average recorded inhibition at different levels of tonic contraction showed an average $R^2 = 0.82 ± 0.03$. During bicycling, with different loads, the level of SOL background EMG increased with increasing external load in 10 of the 11 subjects (Fig. 4C; thin lines). The population average (Fig. 4C; thick line) showed a statistically significant increase of the background EMG with the external load ($R^2 = 0.95, P < 0.01$). Contrary to the background EMG, the recorded inhibition decreased with increasing external load as shown in Fig. 4D.
When the size of the inhibition was expressed as a percentage of the background EMG for each subject, a significant decrease between the lowest and the highest load was found (Fig. 4E, Student’s t-test, \(P = 0.05\)). Thus, there was a positive relation between inhibition and background EMG during tonic plantar flexion (Fig. 4F; black line; \(R^2 = 0.92, P < 0.01\)), but a negative relation during bicycling (Fig. 4F; gray line; \(R^2 = 0.72, P = 0.07\)).

Figure 5A shows the SOL and TA EMG during bicycling at 30, 60, and 90 RPM, and Fig. 5B shows examples of the effect of PN stimulation on the averaged SOL EMG activity at these three cadences of bicycling. The stimulation was applied at the ascending part of the SOL EMG activity at the same crank-angles. Figure 5C shows the soleus background EMG during bicycling at different cadences. As during bicycling with increased load, there was a linear increase in the SOL background EMG with increased pedaling rate (\(R^2 = 0.96, P = 0.03\)). The regression between recorded inhibition and speed (Fig. 5D), however, showed no correlation (\(R^2 = 0.598, P = 0.437\), and no differences between the amounts of inhibition recorded at the different cadences could be detected using the one-way repeated ANOVA. In Fig. 5E, the amount of inhibition is presented as percentage of the background EMG. No differences in the values were found. This is also illustrated in Fig. 5F, where the relation between background EMG and recorded inhibition is plotted for tonic contraction (grey line; \(R^2 = 0.95, P < 0.01\)) and during bicycling at different speeds. The regression during tonic contraction showed a linear relation between increased background EMG and recorded inhibition (black line; \(R^2 = 0.39, P = 0.57\), whereas during bicycling with increased speed, we found no relation (gray line; \(R^2 = 0.4, P = 0.6\)).

**DISCUSSION**

In this study, we have demonstrated that the inhibition in the SOL background EMG induced by stimulation of PN is smaller during downstroke of bicycling compared with a matched level of background EMG during tonic plantar flexion and that the inhibition of the SOL H-reflex after PN stimulation is modu-
lated during the crank-cycle of bicycling, being largest during upstroke when the antagonist muscle (TA) is active. The inhibition in the SOL background EMG, when expressed as a percentage of the background EMG, decreased as the background EMG increased with increasing load during bicycling. The inhibition in contrast remained constant when the background EMG was increased with increasing tonic plantar flexion.

Disynaptic Ia inhibition

Previous studies have demonstrated that the short latency depression of the SOL H-reflex evoked by PN stimulation is mediated by the disynaptic reciprocal Ia inhibitory pathway (reviewed by Crone and Nielsen 1994). In all likelihood, the depression of the SOL background EMG after PN stimulation is also mediated, at least partly, by this pathway. Petersen et al. (1998) demonstrated that the two inhibitions have the same threshold, and Petersen et al. (1999) showed that the inhibition of the SOL background EMG disappeared when transmission in large-diameter afferents were blocked by ischemia, suggesting that group Ia afferents are also responsible for this inhibition. Finally, Crone et al. (2001) found that the inhibition in both the SOL background EMG and the SOL H-reflex is absent in patients with a known defect in the glycine receptor, which is known to be involved in mediating reciprocal inhibition (Bradley et al. 1953). Although other mechanisms may contribute to the inhibition in the EMG, it is thus our opinion that disynaptic reciprocal inhibition via Ia inhibitory interneurons is the main responsible mechanism.

Decreased reciprocal inhibition in downstroke during bicycling

The inhibition in the SOL background EMG during bicycling was reduced compared with a similar level of tonic SOL background EMG. This resembles what has previously been reported during walking by Petersen et al. (1999), but it is contrary to what was reported by Capaday et al. (1990). As discussed by Petersen et al. (1998, 1999), there may be several reasons for this discrepancy. In this study and in the studies by Petersen et al. (1998, 1999) special care was taken to ensure that the PN stimulation was as selective as possible for the branch supplying the TA muscle and rather weak stimuli compared with the study by Capaday et al. (1990) were used. Furthermore, in the study by Capaday et al. (1990), the regression line calculated for all data recorded in different parts of the stance phase was used for comparison to the regression line calculated for data recorded during tonic plantar flexion. In this study and in the studies by Petersen et al. (1998, 1999), on the other hand, a direct comparison was made for each individual recording during walking to that obtained during tonic plantar flexion at a corresponding level of EMG activity. This was done since changes in the amount of inhibition in specific parts of the gait cycle may be overlooked when all the data are pooled together.

It was ensured that the PN stimulation evoked an M-response of a constant size in the TA muscle in all situations and the smaller amount of inhibition in downstroke during bicycling as compared with tonic plantar flexion therefore is not likely to be caused by changes in the stimulation conditions. As we ensured that the background SOL EMG activity was comparable in the two situations, simple differences in the excitability of the SOL motoneurons are also not likely to be involved. We cannot entirely exclude the possibility that different motoneurons were active in the two situations, but as the soleus muscle is rather homogeneous and consists mainly of type S motor units (Gollnick et al. 1974), which receive an almost equal amount of inhibition (Binder and Powers 1999), we find that this is unlikely to explain our observations. We also cannot fully exclude that it may not be valid to compare the very stable background EMG during tonic contraction to the rapidly changing EMG during bicycling. The motoneurons may be very differently sensitive to inhibition when the background EMG is rapidly changing than when it is stable, and it may in any case be difficult to compare the levels of EMG exactly. However, Petersen et al. (1999) found that reciprocal inhibition was still depressed during walking compared with the inhibition measured in sitting subjects, who were asked to perform a voluntary dynamic plantar flexion where both the level and rate of change of the background EMG was matched to that recorded in the stance phase of walking. We therefore think that the decreased inhibition during bicycling that we have observed in this study is most likely explained by a reduced transmission in the inhibitory pathway at a premotorneuronal stage.

Another concern could be that SOL contract eccentrically during early downstroke but concentrically during late downstroke. This could change the background afferent activity and interfere with measurements of reciprocal inhibition. However, similar amount of inhibition was observed during early and late downstroke (cf. Fig. 3C).

Mechanisms involved in controlling the transmission in the disynaptic reciprocal inhibitory pathway

Several different mechanisms may be involved in setting the transmission in the disynaptic reciprocal inhibitory pathway (Enriquez-Denton et al. 2000; Hultborn 1972), and we can only speculate on which of these mechanisms is responsible for the observed decrease of inhibition. Ia inhibitory interneurons receive inhibitory influences from the Ia inhibitory interneurons projecting in the opposite direction (i.e., interneurons that inhibit extensor motorneurons are inhibited by interneurons that inhibit flexor motorneurons), and it would not be unlikely if increased activity of such interneurons were responsible for the decreased inhibition. The interneurons are also inhibited by Renshaw neurons, which have been shown to be modulated during various voluntary movements in human subjects (Katz and Pierrot-Deseilligny 1999) and during fictive locomotion in the cat (Pratt and Jordan 1987). Increased recurrent inhibition during bicycling may therefore also contribute to decreased reciprocal inhibition. Iles et al. (2000) showed that recurrent inhibition of quadriceps motorneurons from SOL was stronger during downstroke of bicycling compared with matched contraction in sitting subjects. The authors argued that their findings may also apply to recurrent inhibition of SOL motoneurons, but whether they also apply to recurrent inhibition of the inhibitory interneurons projecting to these motoneurons is unknown. The transmission in the disynaptic reciprocal pathway is also controlled at the level of the synapses of the Ia afferents on the interneurons (Enriquez-Denton et al. 2000). Crone and
Nielsen (1989) demonstrated that the long-lasting (more than 10 s) change in transmitter release from Ia afferents following their previous activation (postactivation depression) may interfere with the determination of the amount of reciprocal inhibition. If postactivation depression is more pronounced during downstroke of bicycling, possibly secondary to larger dorsi- flexion Ia afferent activity, than during tonic plantar flexion, this would explain the smaller amount of reciprocal inhibition.

Pyndt and Nielsen (2003) demonstrated that presynaptic inhibition of Ia afferents on soleus motoneurons is increased in the late part of downstroke compared with early downstroke. If these observations are also relevant for presynaptic inhibition of Ia afferents on the antagonistic TA motoneurons and their corresponding interneurons, it would be expected that a similar difference was seen in the modulation of reciprocal inhibition in early and late downstroke. However, this was not the case (cf. Figs. 2 and 3).

It could also be argued that stretch of the TA muscle would increase Ia afferent activity and lead to refractoriness. This could explain the smaller amount of inhibition. However, TA is only stretched during late downstroke (e.g., Chen et al. 2001), and this is therefore not likely to explain our observations during downstroke.

It is very likely that the regulatory mechanisms in the spinal cord may also be influenced by a vast variety of afferent inputs arising from muscles from the ipsilateral as well as the contralateral leg (Brooke et al. 1997).

Modulation of reciprocal inhibition during the crank-cycle

Using the H-reflex technique, we demonstrated that reciprocal inhibition was much larger during upstroke than during downstroke (Fig. 3, B and C). Petersen et al. (1999) similarly reported that reciprocal inhibition was increased in the swing phase of walking compared with the stance phase. In this study, the increase of reciprocal inhibition was observed just prior to and during the time when the TA muscle was active. The inhibition disappeared again when the soleus muscle became active in the early part of downstroke. This underscores the parallel control of Ia inhibitory interneurons and their corresponding motoneurons during extension-flexion movements (Crone et al. 1987; Hultborn 1972; Lundberg 1970). It should also be noted that the pattern resembles that observed for the activity of Ia inhibitory interneurons during fictive locomotion in the cat (Pratt and Jordan 1987).

Pyndt and Nielsen (2003) observed that the soleus H-reflex was depressed in the late part of downstroke and especially during upstroke (see also Brooke et al. 1992). The observation in this study that reciprocal inhibition to soleus motoneurons is not increased until well into upstroke shows that increased disynaptic reciprocal inhibition cannot be responsible for the H-reflex depression. A more likely mechanism for that depression is, as also suggested by Pyndt and Nielsen (2003), increased presynaptic inhibition of the soleus Ia afferents.

Changes in reciprocal inhibition as a function of load and cadence

During tonic contraction the amount of depression of the SOL background EMG was linearly related to level of background SOL EMG. When expressed as a percentage of the background EMG the inhibition in other words remained nearly constant. This phenomenon has been described as “automatic gain compensation” (Marsden et al. 1972; Matthews 1986) and has previously been demonstrated for the PN induced inhibition of the SOL EMG (Capaday et al. 1990; Petersen et al. 1999). Automatic gain compensation is thought to ensure that reflexes remain appropriate to the level of contraction. In our study “automatic gain compensation” was observed during bicycling in 8 of 12 subjects (Fig. 2D). It is, however, meaningless to compare the measurements during bicycling, which include measurements from both early downstroke, where the background EMG is increasing and late downstroke where the EMG is decreasing. As we have already pointed out, reciprocal inhibition is likely to be controlled very differently in these two phases of the movement, and it is therefore not reasonable to calculate regression lines for such data or make any conclusions regarding the existence of automatic gain compensation. However, we also investigated the amount of inhibition as a function of the external load and the rate of pedaling. In these two cases, the amount of inhibition was measured at the same crank-angle during downstroke, but with increasing background EMG as the external load or the rate of pedaling was increased. In striking contrast to what was observed during tonic plantar flexion, the amount of inhibition when expressed as a percentage of the background EMG decreased with increasing background EMG produced by increased external load. The principle of “automatic gain compensation” thus does not apply to bicycling against different external loads. The data again rather suggest that reciprocal inhibition is differently controlled during bicycling than during tonic voluntary plantar flexion. It seems that reduction of the amount of inhibition of the soleus motoneurons is of importance during bicycling to increase the activation of the soleus muscle and overcome the increased external load. A similarly strong reduction of reciprocal inhibition is apparently not necessary during voluntary plantar flexion.

The SOL and TA EMG patterns during pedaling at increased cadences were very similar to the EMG patterns during pedaling with increased load. Although it was not significant, we also observed a small decrease in the amount of reciprocal inhibition with increased pedaling rate. The lack of significance may be caused by the large variability of the measurements (cf. Fig. 5D), but it may also reflect a difference in modulation of afferent inflow (Staines et al. 1997) or central command (Christensen et al. 2000) with increased pedaling rate compared with increased load.

Comparison with walking

The modulation of reciprocal inhibition observed in this study during bicycling is very similar to the modulation reported during walking by Petersen et al. (1999). This strengthens the idea that the brain uses similar strategies to control bicycling and walking. In both cases, less inhibition is observed than during voluntary tonic plantar flexion, and in this study, we found a gradual decrease of inhibition with increasing activation of the soleus muscle. This may be related to differences in sensory inflow in the two situations as already pointed out by Brooke et al. (1997) or it may be related to a different central control of the segmental circuitry. During walking and bicycling it is reasonable to assume that a spinal
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DISCLOSURES

This difference may underlie the differential control of reciprocal inhibition that we have documented here.

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