Modulation of the soleus H-reflex during pedalling in normal humans and in patients with spinal spasticity

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Abstract
Soleus H-reflexes were recorded in 10 normal subjects and seven patients with spasticity caused by incomplete spinal cord injury while they pedalled on a stationary bicycle which had been modified to trigger electrical stimuli to the tibial nerve at eight precise points in the pedal cycle. Stimulus strength was adjusted to yield M-waves of constant amplitude at each pedal position. During active pedalling, all normal subjects showed modulation of the H-reflex with the amplitude being increased during the downstroke portion of the pedal cycle and the reflex suppressed or absent during the upstroke. This modulation was not present during passive pedalling, with the experimenter cranking the pedals by hand, or when the pedals were locked at each of the eight positions. In five of the seven patients with spasticity, there was reduced or absent modulation of the H-reflex during active pedalling and the reflex remained large during pedal upstroke. It is concluded that descending motor commands that produce patterned voluntary activity during pedalling normally cause cyclical gating of spinal reflexes by either presynaptic or postsynaptic inhibitory mechanisms. Loss of supraspinal control over these spinal inhibitory systems could result in failure to produce appropriate suppression of reflexes during patterned voluntary movements such as pedalling or walking, and may be an important factor contributing to the functional disability in spasticity.

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During voluntary movement descending motor commands and afferent inputs from the periphery to the spinal cord interact to produce continuous modulation of the excitability of motoneurons and spinal reflex circuits. The role of stretch reflexes during voluntary movement is not fully defined. In some circumstances, reflex activity may be strengthened to assist the movement; alternatively, it may be advantageous to suppress reflexes in situations where stretch reflex activity may interfere with the movement. There is evidence that changes of this type occur during locomotion in human subjects walking on a treadmill where it has been shown that the soleus H-reflex undergoes modulation at different phases of the step cycle. The reflex is relatively large during the stance phase, particularly during late stance, and is then suppressed during the swing phase.

In patients with spasticity caused by spinal cord injury, stretch reflexes are characteristically increased. With incomplete lesions and preservation of voluntary power, hyperreflexia alone may not have major functional significance. If the damage to descending pathways interferes with the mechanisms that normally modulate stretch reflexes during locomotion, the functional consequences could be much greater.

The purpose of this study was twofold: first, to determine whether reflex modulation similar to that reported during walking also occurs during pedalling movements, and secondly, to investigate how the ability to modulate reflexes is altered in subjects with spasticity caused by spinal cord injury.

Pedalling on a stationary bicycle has been used previously to assess quantitatively some aspects of spasticity. Pedalling permits investigation of some aspects of reflex modulation which are difficult to study during treadmill walking. The patterns of EMG activity are well defined and remain relatively constant during pedalling. By changing the load on the bicycle it is possible to increase or decrease levels of muscle activation and study the effects of varying levels of background EMG on the H-reflex. Furthermore, with the bicycle it is possible to investigate passive pedalling where the effects of afferent inputs associated with pedalling movements can be dissociated from the descending motor commands controlling active pedalling. Some of these results have been published previously in abstract form.

Methods
Subjects
Experiments were performed on seven male subjects with spasticity following incomplete spinal cord injury in the cervical or upper thoracic regions. Clinical details for the subjects are presented in the table. All subjects had at least moderate strength in plantar-flexion at the ankle joint and were able to dorsiflex the foot to some degree. All had recovered sufficient function to be able to walk, in most cases with assistance of a cane or crutches. Control experiments were performed on 10 healthy male volunteers with ages ranging from 21 to 48. The experimental protocol was approved by our local ethics committee and informed consent was obtained in all cases.
Apparatus

A modified bicycle ergometer (Corvil Model 300) was used for these experiments. A circuit board with eight photoelectric sensors was fitted to the flywheel. These sensors were located at 45° intervals around the circle representing one pedal cycle. Each time a tab on the flywheel passed a sensor, a pulse of current was generated so that stimulation and data collection could be initiated at eight precise points in the pedal cycle.

The height of the seat was adjusted so that the knee angle was approximately 170° when the foot was at the lowest point in the pedal cycle. The subject’s feet were strapped firmly to the pedals with the metatarsal-phalangeal joints positioned directly over the centre of the pedals. Subjects were required to pedal at a constant rate of 20 RPMs. For most recordings, the resistance was maintained at a constant moderate load. In some cases, additional recordings were obtained with the subjects pedalling against varying levels of resistance.

To study effects of passive pedalling, the experimenter cranked the pedals by hand at the same speed as during voluntary pedalling. Subjects were instructed to relax their legs as completely as possible. Some subjects had difficulty relaxing fully, and EMG feedback was provided on an oscilloscope to assist relaxation.

EMG recordings

Surface electrodes (Ag-C1 non polarising) were placed over the soleus muscle with the proximal electrode just below the point at which the two heads of the gastrocnemius diverge. Interelectrode distance was 3 cm. Surface electrodes were placed over the belly of the tibialis anterior muscle. The signals were amplified by miniature pre-amplifiers attached to the leg in close proximity to the recording electrodes. This minimised problems related to artefact occurring from movement of the wires during pedalling. The unrectified EMG was filtered at 10 Hz and 1 kHz and led to the A-D converters of a PDP 11–40 computer. The sampling rate was 4 kHz with 12-bit resolution.

Stimulation

The stimulating cathode consisted of a 5mm diameter metal sphere attached to a small plastic block. This was placed over the tibial nerve in the popliteal fossa and held in place by a wide elastic band around the knee. The anode was a 2 cm × 3 cm metal plate which was attached to the anterior thigh just above the patella. Stimulus duration was 0–5 ms. Before each experiment, the subject pedalled at the standard speed and load while stimuli were delivered repeatedly at position 4 (see fig 1) and stimulus intensity was set at a level that produced a maximum H-reflex at this position. The amplitude of the corresponding M-wave was noted and throughout the experiment stimulus intensity at the other positions was adjusted to produce an M-wave of the same amplitude.

Initially we attempted to deliver the stimuli at the eight positions in a random order. This proved difficult in most subjects because the stimulus intensity required to produce M-waves of comparable amplitude was different at different positions, presumably because of slight movement of the stimulating electrode in relation to the tibial nerve during pedalling. Each run therefore consisted of 40 responses to stimuli delivered at one position in a pseudo random manner every second, third or fourth rotation of the pedals. This resulted in H-reflexes being elicited at intervals of not less than 5 s. The M-wave was monitored on an oscilloscope and slight adjustments in stimulus intensity were made to keep its amplitude constant. For the first 20 responses the subject pedalled actively. The subject then relaxed and the experimenter cranked the pedals by hand so that the final 20 responses were obtained during passive pedalling.

Effect of joint angles

To investigate the effects of leg position and hip and knee angles on the H-reflex, additional recordings were made in some subjects with the pedals locked at each of the eight different positions and the subject at rest. In another experiment infrared emitting diodes were placed on the lower leg and foot, and movement at the ankle joint during voluntary pedalling was monitored using a WATSMART motion analysis system. In four subjects, a plastic splint was used to prevent movement at the ankle joint while the subject performed the pedalling experiment.

Data analysis

Individual recordings consisting of EMG from the soleus and tibialis anterior muscles for 25 ms before and 50 ms after the electrical stimulus were analysed. The M-wave was examined initially, and recordings in which the
peak-to-peak amplitude fell within a narrow amplitude window were selected. From each set of 20 responses (active or passive pedalling) we obtained between five and 15 responses that met the defined M-wave criteria. The peak-to-peak amplitude of the H-reflex at each position was then averaged for both active and passive pedalling and the resulting averages were plotted as a function of pedal position. Integrated background EMG during the 25 ms pre stimulus period was also measured for each trial.

Results
Figure 1 shows representative responses to single stimuli to the tibial nerve during active pedalling for a normal subject and for a patient with spinal spasticity. Stimuli were delivered at eight different points in the pedal cycle indicated by the numbers on the illustration. In the normal subject there is considerable modulation of the size of the H-reflex. A small response is present at position 1 at the top of the pedal cycle. It becomes progressively larger at positions 2, 3 and 4 during the downstroke, then diminishes and is completely absent during the upstroke at positions 6, 7 and 8. The M-wave amplitude is the same at all eight positions, indicating that stimulus strength has remained constant. By contrast the spastic patient whose responses are shown in fig 1 showed very little modulation of the H-reflex. In this example the reflex response is slightly smaller at position 7 but just as large at the other upstroke positions as during the downstroke.

Our recordings were carried out using stimulus strengths which produced near maximal H-reflexes. This was necessary to ensure that at least a small M-wave was present to allow matching of responses in which the effective stimulus was approximately the same. As other investigators have emphasised, the importance of using a smaller H-reflex to demonstrate excitatory and inhibitory effects, we carried out more detailed studies in two normal subjects using a wide range of stimulus intensities. The results are shown in fig 2, which displays a full recruitment curve with H-reflex size plotted against M-wave amplitude for each of the eight different pedal positions. It can be seen that the suppression of the H-reflex during the pedal upstroke in the normal subject was a constant finding regardless of the stimulus intensity or the H-reflex size.

Not all of the spastic patients showed loss of H-reflex modulation to the extent indicated by the example shown in fig 1. Figure 3 shows H-reflex amplitude at the eight different pedal positions for all seven spastic subjects. Two subjects (1 and 2) showed essentially normal modulation of the reflex. These were the two subjects with the least clinical deficit among the group—but both were able to walk without support. In two other subjects (3 and 4) there was some suppression of the reflex during the pedal upstroke but a sizeable H-reflex reappeared at position 8, something that was never seen in normal subjects. In the other three subjects (5, 6 and 7) there was little or no modulation of the H-reflex. Spasticity in these subjects ranged from mild to severe, but they were the three subjects who showed the greatest degree of weakness of ankle dorsiflexion. They also showed the most disability in terms of walking with the exception of case 7 in whom the tested leg was severely affected but walking ability was relatively preserved because the contralateral leg was less affected by the spinal cord injury. (see the table).

Figure 4 shows the pooled results for all 10 normal subjects and for five spastic subjects. As subjects 1 and 2 showed modulation of the H-reflex very similar to that seen in control subjects, they were not included in this analysis. In addition to mean H-reflex amplitude, the figure documents the size of the M-wave and mean levels of background EMG activity in the soleus and tibialis anterior at each of the eight different pedal positions. In most of the normal subjects, the H-reflex reached its maximal amplitude at position 3, but in some this occurred at positions 2 or 4. As there was considerable variation in the absolute amplitude of the H-reflexes from different subjects, the data were normalised by assigning a value of 100 to the averaged H-reflex or EMG amplitude at position 4 for each subject and expressing the values at the other positions as percentages of this.

In the normal subjects the changes in the size of the H-reflex roughly parallel changes in the amplitude of background EMG activity in the soleus, but the position with the largest H-reflex did not always coincide with the position with the highest soleus background EMG activity. Moreover, a similar pattern of background EMG change was seen in the soleus of the spastic subjects, but in most of them there was reduced or absent modulation of the H-reflex. The suppression of the H-reflex during pedal upstroke in the normal subjects was accompanied by an increase in tibialis anterior EMG, particularly at positions 7 and 8. There was some increase in tibialis anterior EMG during pedal upstroke in the spastic subjects, but this was less prominent than in the normals.
During voluntary movements, spinal motor neurons receive inputs not only from descending pathways from higher motor centres but also from peripheral receptors which have been activated by the movements. This raises a question as to whether the modulation of the H-reflex during active pedalling is simply due to changing levels of background EMG in the soleus muscle, rather than representing a gating of reflex activity associated with the descending motor commands for pedalling. The relationship between soleus background EMG and H-reflex amplitude is illustrated for two normal subjects in fig 6. In each case, the load on the bicycle was adjusted so that responses were obtained with the subject pedalling against a number of different loads. H-reflexes are plotted for positions 2 and 4, and each data point represents the mean of 20 responses. At both positions, the H-reflex increases as background EMG levels increase. It is obvious, however, that for any given level of background EMG, the H-reflexes are considerably larger at position 4 than at position 2.

Figure 7 shows EMG activity in the soleus and tibialis anterior and associated changes in the ankle joint angle during active pedalling by a normal subject and one of the spastic subjects. In both subjects there is a moderate amount of movement at the ankle joint with plantar flexion during the downstroke and dorsiflexion during the upstroke component of the pedal cycle. Although proximal leg muscles provide the major driving force during pedalling, there are well defined bursts of EMG activity in muscles acting at the ankle joint with alternating bursts occurring in the soleus and tibialis anterior. This reciprocal pattern of activation was maintained in this spastic subject (subject 4) who also retained some degree of modulations of the H-reflex during the pedal cycle (see fig 3).

Because changes in the ankle joint angle may affect the size of the H-reflex, we carried out additional studies in four normal subjects using a plastic splint to prevent movement at the ankle joint. With the ankle immobilised, the amount of H-reflex modulation during active pedalling was similar to that in the unsplinted position.

Changes in hip and knee angle and leg position could also conceivably cause some modulation of the H-reflex. To investigate this possibility we recorded H-reflexes in the normal subjects with the pedals locked to keep the leg stationary at each of the eight positions. Under these conditions we did not see any modulation of the H-reflex and, in particular, there was no apparent suppression of the reflex at pedal positions 6, 7 and 8 where it was markedly suppressed during active pedalling.

Discussion
The results presented here clearly show that, in normal subjects, the soleus H-reflex undergoes

Figure 2 H-reflex amplitudes plotted as a function of M-wave size at each of the eight different pedal positions during active pedalling in a normal subject. The H-reflex is markedly reduced in amplitude during pedal upstroke relative to pedal downstroke over a wide range of stimulation strengths.

Figure 3 H-reflex amplitude as a function of pedal position during active pedalling in seven subjects with spasticity caused by spinal cord injury. The pedal positions are as indicated in fig 1.
considerable modulation during active pedalling on a stationary bicycle. This modulation was diminished or absent in five of seven subjects with spasticity caused by incomplete spinal cord injury.

Although cycling and walking are quite different forms of locomotion, our results bear some similarity to the modulation of the H-reflex during treadmill walking which has been described by Capaday and Stein and Crenna and Frigo. In those studies it was shown that the H-reflex was relatively large during the stance phase of walking and was suppressed during the swing phase. The downstroke portion of the pedal cycle, where the H-reflex was maximal in our subjects, could be considered to be similar to the stance phase of gait and the upstroke portion equivalent to the swing phase. Quadriceps H-reflex modulation has also been reported during walking.

Possible mechanisms for reflex modulation in normal subjects

Several factors could contribute to the reflex modulation that was observed during pedalling. Firstly, it is possible that repeated flexion and extension of the knee during pedalling causes movement of the stimulating electrode in relation to the tibial nerve so that the effective stimulus strength is not constant. By matching the M-wave amplitude at each recording position, however, we could assume that the number of afferent fibres being stimulated at each position was approximately the same.

It has been reported by some investigators that the amplitude of the soleus H-reflex increases in proportion to the level of motor-neuron excitability or the amount of background EMG activity in the soleus at the time the stimulus is delivered. A recent study, however, reported very little change in H-reflex amplitude when the reflexes were superimposed on steady plantar flexion contractions at different force levels, although the H-reflexes were inhibited during steady dorsiflexion. In our experiments the largest H-reflexes were obtained during the downstroke part of the cycle when the soleus background EMG was increased. The results shown in fig 6, however, indicate that for comparable levels of background EMG the H-reflex can be quite different depending on the position of the leg within the pedal cycle.

During cycling, as with other voluntary movements, there is continuous flow of afferent information back to the spinal cord from various receptors in the moving limb. Some of this afferent feedback might modify reflex transmission to change the size of H-reflexes independently of any effects from descending motor commands. To investigate this possibility, we recorded reflexes during passive pedalling movements and, when subjects were able to fully relax, there was very little H-reflex modulation. Because of changes in fusimotor drive and spindle sensitivity, however, feedback in muscle afferents may be different during active and passive cycling, even when the movement of the leg is the same. Nevertheless, the observations during passive pedalling suggest that the H-reflex modulation seen during active pedalling was related to descending motor commands and not simply an effect of sensory feedback.

Changes in the position of the limb or joint angles at the hip and knee may also cause variation in the size of the soleus H-reflex. In our experiments this did not seem to be a contributing factor. H-reflexes elicited with the
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Figure 6  Relationship between H-reflex size and background EMG activity in the soleus muscle. Results obtained at positions 2 and 4 of the pedal cycle (see fig 1) are shown for two normal subjects. The resistance load on the bicycle was set at six different levels to generate six levels of background EMG. Each point represents a mean of 20 H-reflex measurements. The H-reflex size increases as soleus background EMG increases but, in both subjects, H-reflexes are larger at position 4 than at position 2 for comparable levels of background EMG. H-reflex size and background EMG levels are expressed in arbitrary units. Background EMG was integrated over a 25 ms pre-stimulus period.

Figure 7  EMG activity in soleus and tibialis anterior muscles and associated changes in the ankle joint angle during active pedalling at 20 r.p.m. in normal subject and in a spastic subject. The pedal positions are as indicated in fig 1.

limb relaxed and stationary were essentially the same at each of the eight positions.

Changes in ankle joint angle may also modify the size of the H-reflex. In particular, passive dorsiflexion of the ankle can cause depression of the H-reflex. The results shown in fig 7 reveal that some movement does occur at the ankle joint during unrestrained voluntary pedalling. When a splint was applied to prevent movement at the ankle, however, the H-reflex modulation seen during active pedalling was unchanged.

It can be concluded that the H-reflex modulation observed during active pedalling represents a patterned change in the excitability of spinal reflexes which is associated with the descending motor commands producing the movement. Changes in presynaptic inhibition during movement may be responsible for H-reflex modulation seen during walking and pedalling. Changes in the H-reflex/EMG relationship (see fig 6) may also be due to changes in presynaptic inhibition on Ia afferent terminals. Postsynaptic inhibition through Ia inhibitory interneurons may also be involved in the soleus H-reflex suppression observed during pedal upstroke when the soleus motoneuron pool is quiescent, as occurs during voluntary ankle dorsiflexion.

Additional factors may be responsible for the H-reflex suppression in our experiment. In most of our normal subjects, the H-reflex was also markedly reduced at position five in the early part of the pedal upstroke before there was any evidence of EMG activity in the tibialis anterior. Muscle history might be a contributing factor in soleus H-reflex modulation, as H-reflexes have been reported to be inhibited at rest following a shortening contraction.

Mechanisms for changes in spastic subjects

In five of the seven spastic subjects the H-reflex failed to suppress normally during the pedal upstroke and in four subjects did not show changes in amplitude paralleling the changes in background EMG in the soleus during the downstroke. This lack of reflex modulation could be due to abnormal mechanisms acting at the presynaptic level, the postsynaptic level, or possibly a combination of factors. In spastic subjects, failure to suppress the H-reflex with vibration has been interpreted as evidence for a defect in presynaptic inhibition. In our spastic subjects, failure to suppress the H-reflex during the pedal upstroke could also be due to a lack of normal reciprocal inhibition acting through Ia inhibitory interneurons. In a recent study on patients with spinal spasticity using conditioning stimuli to the peroneal nerve to inhibit the soleus H-reflex it was shown that reciprocal Ia inhibition was increased rather than decreased, and other investigators using a different technique have also presented evidence for increased reciprocal Ia inhibition in patients with spinal spasticity. Nevertheless, one would expect that in normal subjects the descending motor commands that activate the tibialis anterior during the pedal upstroke would also excite Ia inhibitory interneurons to inhibit soleus motoneurons. Tibialis anterior
EMG activity was diminished during pedal upstroke in most of our spastic subjects and it could be argued that activation of reciprocal inhibition of soleus motoneurons by descending motor pathways was similarly diminished. H-reflexes have also been studied during treadmill walking in patients with spinal spasticity. The results were variable with some subjects showing normal reflex modulation and others showing almost complete loss of modulation.

**Functional implications**

During the swing phase of gait or the upstroke part of the pedal cycle, when the tibialis anterior muscle is normally active, there may be some stretching of the soleus. A large stretch reflex in the soleus could interfere with ankle dorsiflexion and there would appear to be clear advantages to having reflex activity suppressed at this time. It is a common observation in patients with spasticity that ability to walk may be severely impaired even when voluntary strength is relatively well preserved. Failure to produce appropriate suppression of hyperactive reflexes during locomotion may be an important factor contributing to the functional disability in spasticity.

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