Spinal reflexes and coactivation of ankle muscles during a submaximal fatiguing contraction

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Lévénez, Morgan, Christos Kotzamanidis, Alain Carpentier, and Jacques Duchateau. Spinal reflexes and coactivation of ankle muscles during a submaximal fatiguing contraction. J Appl Physiol 99: 1182–1188, 2005. First published April 21, 2005; doi:10.1152/japplphysiol.00284.2005.—This study examined the involvement of spinal mechanisms in the control of coactivation during a sustained contraction of the ankle dorsiflexors at 50% of maximal voluntary contraction. Changes in the surface electromyogram (EMG) of the tibialis anterior and of two antagonist muscles, the soleus and lateral gastrocnemius, were investigated during and after the fatigue task. Concurrently, the compound action potential (M-wave) and the Hoffmann reflex of the soleus and lateral gastrocnemius were recorded. The results showed that the torque of the ankle dorsiflexors and the average EMG of the tibialis anterior during maximal voluntary contraction declined by 40.9 ± 17.7% (mean ± SD; P < 0.01) and 37.0 ± 19.9% (P < 0.01), respectively, at task failure. During the submaximal fatiguing contraction, the average EMG of both the agonist and antagonist muscles increased, leading to a nearly constant ratio at the end of the contraction when normalized to postfatigue values. In contrast to the monotonic increase in average EMG of the antagonist muscles, the excitability of their spinal reflex pathways exhibited a biphasic modulation. The amplitude of the Hoffmann reflexes in the soleus and lateral gastrocnemius increased to 147.5 ± 52.9% (P < 0.05) and 166.7 ± 74.9% (P < 0.01), respectively, during the first 20% of the contraction and then subsequently declined to 66.3 ± 44.8 and 74.4 ± 44.2% of their initial values. In conclusion, the results show that antagonist coactivation did not contribute to task failure. The different changes in voluntary EMG activity and spinal reflex excitability in the antagonist muscles during the fatiguing contraction support the concept that the level of coactivation is controlled by supraspinal rather than spinal mechanisms. The findings indicate, however, that antagonist coactivation cannot simply be mediated by a central descending “common drive” to the motor neuron pools of the agonist-antagonist muscle pairs. Rather, they suggest a more subtle regulation of the drive, possibly through presynaptic mechanisms, to the motoneurons that innervate the antagonist muscles.

MATERIALS AND METHODS

Twelve subjects (11 men and 1 woman), aged between 18 and 49 yr (30.4 ± 9.0 yr), volunteered to participate in this investigation. All subjects were well accustomed to electrical stimulation, but to avoid possible influence of practice on coactivation, they were not trained in the force-matching task before data collection. None of the subjects presented with signs of neurological disorders. The Local Ethics Committee approved this study, and the subjects gave their informed consent before participation in the investigation. All experimental procedures were performed in accordance with the Declaration of Helsinki.

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Experimental setup and mechanical recording. The subject was secured on an adjustable seat in a slightly reclined position with the right foot strapped to a footplate of an ankle ergometer. The plate was inclined at an angle of 45° to the floor, and the seat was adjusted so that the ankle and knee angles were at −90° (neutral position) and 120°, respectively. The foot was held in place by a heel block and secured to the plate via two straps. One strap was placed around the ankle and the second strap was placed around the foot, 1–2 cm proximal to the metatarsophalangeal joint of the toes. The isometric torques exerted by the plantarflexor or the dorsiflexor muscles were recorded by a strain-gauge transducer (TC 2000–500, linear range 0–500 N, sensitivity 30 mV/N; Kulite, Basingstoke, UK), and the signal was amplified (AM 502, Tektronix, Beaverton, OR).

EMG recordings. Voluntarily and electronically induced EMG activities in the Sol, LG, and tibialis anterior (TA) were obtained by means of bipolar surface electrodes (silver disk electrode of 8 mm in diameter). One electrode was positioned over the motor point, and a second electrode was placed 3 cm (center-to-center) distal to the first one. The ground electrodes (2 × 3 cm silver plate) were placed over the tibia. The location of the motor point was determined by electrical stimulation and defined as the point of stimulation at which a minimal stimulus evoked a barely perceptible muscle contraction. All EMG signals were amplified (×1,000) and filtered (10 Hz to 1 kHz) by a custom-made differential amplifier. The signals were acquired on a personal computer at a sampling rate of 2 kHz, by using a data-acquisition system and analyzed offline by using the AcqKnowledge analysis software (model MP 100, Biopac Systems, Santa Barbara, CA).

Electrical stimulation. The plantarflexor muscles were stimulated by delivering rectangular electrical pulses of 1-ms duration by means of a two-channel, custom-made stimulator that was triggered by a digital timer (model 4030, Digitimer, Welwyn Garden City, UK). The cathode (silver disk electrode of 8 mm in diameter) was located over the tibial nerve in the popliteal fossa, and the anode (silver plate of a two-channel, custom-made stimulator that was triggered by a

Acquisition system and analyzed offline by using the AcqKnowledge software (model MP 100, Biopac Systems, Santa Barbara, CA).

In some subjects, the maximal stimulation, at the popliteal fossa, activated the common peroneal nerve that supplies the TA. As a consequence, the stimulus also triggered an H-wave in the TA during the sustained fatiguing contraction. This response, called the V-wave when obtained by intense electrical stimulation superimposed to a voluntary contraction (1, 36, 41), represents a reflex response to a volley of Ia impulses. Under resting conditions, maximal stimulation of a peripheral nerve will evoke an M-wave, but not an H-reflex, due to the collision of action potentials that travel antidromically along the α-axons with the action potentials generated by the incoming afferent volley. During a voluntary contraction, however, the antidromic potentials collide with the efferent potentials associated with the voluntary drive, which enables the reflex response generated by the afferent volley to pass along the α-axons and reach the muscle to produce the V-wave. Furthermore, the M-wave recorded in the TA in response to the electrical stimulation of the common peroneal nerve was used to determine the effect of fatigue on the excitability of the muscle membrane and to normalize the V-wave amplitude. The normalized amplitude of the V-wave was used to determine the change in the magnitude of the efferent motor neuronal output during the course of the fatigue task (1). Because the electrical stimulation was optimized for the Sol and LG responses, the TA M-wave was not maximal for all subjects and is therefore referred to as the M-wave, instead of Mmax as for the Sol and LG.

Muscle fatigue and testing procedure. Each experimental session began with the subject performing two MVCs with the plantarflexor muscles and two MVCs with the dorsiflexor muscles. Each MVC had a total duration of 4–5 s, and there were 2–3 min of rest between successive trials. The entire recruitment curve for the H-reflex and M-wave of the Sol and LG was determined at rest by increasing the electrical stimulus progressively at 5-s intervals (39). Thereafter, the intensity of the stimulation needed to evoke an H-reflex in the Sol, accompanied by a small M-wave, was determined during a 50% MVC of the ankle dorsiflexors. Control responses for H-reflexes and Mmax (3 responses each) were then recorded during two submaximal contractions sustained during 10–12 s.

The fatigue task consisted in a sustained isometric contraction of the ankle dorsiflexor muscles at 50% MVC. The contraction ended when the subject was unable to maintain the required force level for a period of 5–10 s (task failure; Fig. 1). The subjects then immediately produced a 5-s MVC with the dorsiflexor muscles. Submaximal (H-reflex) and maximal (M-wave) electrical stimulation of the tibial nerve were induced every 30 s during the fatigue task. To assess the recovery from fatigue, H-reflexes and M-waves were elicited during a 3-s contraction at 50% MVC every 30 s during the first 3 min, every min between minutes 3 and 5, and after 7.5 and 10 min. In addition, the subject performed an MVC with the dorsiflexor muscles after 3 min of recovery and during the subsequent testing periods to assess the recovery of the maximal torque and TA average EMG (aEMG).

![Fig. 1.](http://jap.physiology.org/)
**Measurements.** For both the dorsiflexor and plantarflexor muscles, the MVC torque and associated aEMG activities were determined for a 2-s period during the torque plateau before and after the fatigue task. Before the fatigue task, the trial that yielded the largest MVC torque was taken as the control value and served to normalize all EMG data. The aEMG amplitude of the agonist and antagonist muscles during the fatiguing contraction was measured for a 2-s duration, once every 15 s. These values were normalized to the aEMG recorded during maximal plantarflexion (agonist) or dorsiflexion (agonist) contraction that were performed before the fatigue task. The TA aEMG recorded at the end of the fatigue task was also normalized to the aEMG of the postfatigue MVC. This procedure was used to control whether the decrease in aEMG with fatigue was due to amplitude cancellation that occurs when overlapping positive and negative phases of muscle action potentials are summed (8, 13, 20). The level of coactivation was quantified by computing the ratio between the agonist and antagonist aEMG activities (21). The H-reflexes, V-waves, and M-waves were characterized before, during, and after the fatigue task by their peak-to-peak amplitudes. To exclude fatigue-related changes of the muscle fiber membrane and to assess the neural adjustment during the task, V-wave amplitude was normalized, at each time point, to its corresponding M-wave amplitude. Because the time to failure differed from subject to subject, all data were normalized as percentage of their respective values obtained during MVCs performed before the fatigue task. Significant differences from initial value: *P < 0.05; **P < 0.01.

**RESULTS**

The average time to task failure for all subjects was 315.7 ± 115.7 s (range 184–609 s). Immediately after the fatigue task, the MVC torque produced by the ankle dorsiflexor muscles and the aEMG of the TA dropped by 40.9 ± 17.7% (P < 0.01) and 37.0 ± 19.9% (P < 0.01) of the prefatigue values, respectively. At task failure, the peak-to-peak amplitude of the M-wave for TA also displayed a significant decrease (42.5 ± 19.2%; P < 0.01). In contrast, the antagonist muscles (Sol and LG) did not show any change in Mmax during the fatiguing task.

**Voluntary EMG activities during the fatiguing task.** As illustrated for one subject in Fig. 1, the EMG activity for TA increased progressively throughout the fatiguing contraction. The rise in aEMG amplitude was small during the first half of the task duration but more pronounced thereafter. It increased from 38.3 ± 11.1% (P < 0.05) at the beginning of the contraction to 45.9 ± 14.3% at 80% of the time to failure, before declining slightly thereafter (Fig. 2). At task failure, however, the aEMG normalized to the value recorded during the postfatigue MVC reached 93.3 ± 68.0% (P < 0.01).

Similar to the TA, the aEMG of the antagonist muscles increased continuously throughout the fatiguing contraction (Figs. 1 and 2). At the start of the contraction, the antagonist aEMG for Sol and LG was 17.7 ± 14.2 and 14.5 ± 10.1%, respectively. At the end of the fatigue task, its value reached 30.9 ± 22.9% (P < 0.01) for the Sol and 22.1 ± 15.6% (P < 0.01) for the LG. Although the rate of increase in aEMG for LG was slightly less than that for Sol, the difference was not statistically significant (P > 0.05). At the end of the fatigue task, its value reached 31.7 ± 22.4% (P < 0.01) and 21.9 ± 26.4% (P < 0.05) for the Sol and LG, respectively. When normalized relative to the EMGs during the postfatigue MVCs, however, the coactivation ratio increased slightly but not significantly (P > 0.05) compared with initial values, by 7.2 ± 43.5 and 21.5 ± 48.2% for the Sol and LG, respectively (Fig. 3).

During the sustained contraction of the ankle dorsiflexors, a V-wave induced by the electrical stimulation of the common peroneal nerve was observed in five subjects. Before the fatigue task, the peak-to-peak amplitude of the V-wave and M-wave were 0.39 ± 0.20 and 6.64 ± 2.14 mV, respectively, which corresponded to a V-wave-to-M-wave ratio of 6.2 ± 3.5%. During the fatigue task, the V-wave increased progressively to 168.8 ± 142.9% (P > 0.05) and the M-wave decreased to 57.5 ± 19.2% (P < 0.01) of their initial values. At the end of the contraction, the V-wave-to-M-wave ratio reached 312.5 ± 216.5% (P < 0.01) of the prefatigue value (Fig. 4).

**Antagonist reflex activities during the fatiguing task.** The Mmax evoked in both the Sol and LG during the sustained contraction of the dorsiflexors did not change significantly during the task (Fig. 5). In contrast, the amplitude of the H-reflex changed in parallel for the two antagonist muscles. It increased significantly during the first 20% of the contraction and reached 147.5 ± 52.9% (P < 0.05) and 166.7 ± 74.9% (P < 0.01) for the Sol and LG, respectively, of their control values. Thereafter, the H-reflex declined gradually during the remaining part of the task, reaching 66.3 ± 44.8 and 74.4 ± 44.2% of the prefatigue values in the Sol and LG, respectively. Although not statistically different from prefatigue level, the

![Fig. 2. Changes in average electromyogram (aEMG) of the TA (a), Sol (b), and LG (c) during the sustained contraction of the dorsiflexors at 50% MVC force (left) and during recovery (right). Data (means ± SE) are expressed as a percentage of their respective values obtained during MVCs performed before the fatigue task. Significant differences from initial value: *P < 0.05; **P < 0.01.](http://jap.physiology.org/Downloaded from http://jap.physiology.org by 10.220.32.247 on July 11, 2017)
EMG signal (8, 13, 20). In the present study, the aEMG for TA reached its maximum at ~80% of the time to failure and then declined slightly. When voluntary aEMG was normalized to the aEMG recorded during the postfatigue MVC, however, which provides a more precise estimate of the neural adjustments during fatigue (see Ref. 20), the final value was 93.3% of the maximal capacity. Thus, in addition to possible alterations in neuromuscular transmission and central fatigue, the apparent decline in muscle activation at task failure was probably caused by the loss of signal due to the amplitude cancellation that occurs when overlapping positive and negative phases of muscle action potentials are summed (8, 13, 20). The enhanced muscle activation that occurs during a sustained submaximal contraction is attributable to the recruitment and modulation of discharge rate (3, 7, 15, 25, 26) and contributes to the maintenance of a constant contraction force. Because the modulation of discharge rate during such contractions is modest, the increase in aEMG is largely due to the recruitment of additional motor units (7, 15, 26, 28).

The progressive increase of TA aEMG during the course of the fatigue task was accompanied by a parallel augmentation of the V-wave-to-M-wave ratio. This adjustment results from increases in the number of motoneurons recruited and the number of voluntary motor impulses, which increases the incidence of antidromic collision and allows more motoneuron axons to be cleared for passage of the evoked reflex response (41). Consequently, the V-wave-to-M-wave ratio appears to reflect the magnitude of the efferent motor neuronal output during vol-

![Fig. 3. Changes in normalized coactivation ratios during the sustained contraction of the dorsiflexors at 50% MVC force (left) and during recovery (right). Two normalization procedures are used. The TA aEMG is normalized to the prefatigue MVC value before to compute the ratio for the TA to Sol (●) and TA to LG (○). TA aEMG is normalized to the postfatigue MVC value at the end of the fatigue task and to MVCs performed at the same time period during the recovery period before to compute the ratio for the TA to Sol (●) and TA to LG (○). All ratios (means ± SE) are expressed as percentage of their initial values. Significant differences from initial value: *P < 0.05; **P < 0.01.](image)

final values were significantly different (P < 0.01) from those recorded at 20% of the time to failure.

Recovery. The time course of recovery for the ankle dorsiflexors was quite rapid. The MVC torque and TA aEMG returned to their prefatigue values within 4 min. The M-wave amplitude and V-wave-to-M-wave ratio recovered their initial values after only 30 s of rest (Fig. 4). Similarly, the amplitude of the H reflex for the antagonist muscles (Sol and LG) recovered within 30 s after the end of the fatigue task (Fig. 5). Although H-reflex amplitude remained above control values during recovery, this increase was not statistically significant (P > 0.05). The normalized aEMG of the TA, Sol, and LG during the brief dorsiflexion contractions at 50% MVC force returned to control values within 30 s of rest (Fig. 2).

DISCUSSION

The main finding of the present study was that the progressive increase in EMG activity of the agonist and antagonist muscles during a sustained contraction at 50% MVC force was accompanied by a biphasic modulation of the spinal reflex excitability of the antagonist muscles. The H-reflex amplitude of both the Sol and LG increased during the first 20% of the contraction and then declined gradually during the remaining part of the task. The different changes in voluntary EMG activity and spinal reflex excitability of the antagonist muscles suggest that the level of coactivation during the sustained submaximal contraction was controlled by supraspinal mechanisms, and its increase did not contribute to task failure.

Agonist fatigue. The EMG activity of the agonist muscles increases progressively during a sustained submaximal contraction (11, 17, 37, 38). The increase in EMG activity during such tasks is usually considered to indicate an enhancement of the central drive to the motoneuron pool (3, 14, 15, 17, 24) despite the nonlinear summation of motor unit potentials in the
After the end of the task the aEMG of both muscles had already increased in comparison with the prefatigue value, and the excitatory drive to the two motoneuron pools, despite the different characteristics and function of the mono- and biarticular muscles. This enhancement coactivation occurred without any fatigue of the antagonist muscles. The comparable rate of increase in aEMG amplitude between agonist and antagonist muscles at the end of the fatiguing contraction argues against this possibility. Furthermore, Carpentier et al. (6) previously used similar recording conditions and found that cross talk was quite small and cannot have influenced the results.

Antagonist coactivation. At the beginning of the fatigue task, the antagonist coactivation was 17.7 ± 14.2 and 14.5 ± 10.1% for the Sol and LG, respectively. These values are similar to those observed in previous studies for comparable contraction levels and at different joints (21, 37). As previously reported (12, 17, 37), the EMG activity of the antagonist muscles increased progressively during a sustained submaximal contraction of the dorsiflexors at 50% MVC force and during recovery. Data (means ± SE) are expressed as a percentage of their initial values. Significant differences from prefatigue value: *P < 0.05; ** P < 0.01.

As a result of the slightly greater increase in EMG activity of the antagonist muscles compared with the agonist muscles, the coactivation ratio typically decreases during fatiguing contractions sustained at a submaximal intensity (12, 29, 37). This was also observed at task failure in the present study. As discussed above, however, the use of nonnormalized EMG activity to infer change in voluntary drive may be misleading (8, 19, 20). This is clearly shown in the present study, because when the TA aEMG was normalized to its EMG recorded during the postfatigue MVC, the coactivation ratio was found to increase slightly, but not significantly, at the end of the fatiguing contraction. Therefore, task failure cannot be attributed to the opposite action of the antagonist muscles as a consequence of a greater increase in activation compared with the agonist muscles.

Although short interelectrode distances were used in the present study to minimize cross talk, it may be argued that antagonist coactivation may be caused by volume conduction from the TA muscle. However, the opposite change in the aEMG amplitude between agonist and antagonist muscles at the end of the fatiguing contraction argues against this possibility. Furthermore, Carpentier et al. (6) previously used similar recording conditions and found that cross talk was quite small and cannot have influenced the results.

H-reflex behavior. The potential contribution of spinal adjustments on the activity of the antagonist muscles during fatiguing contractions by the agonist muscles was analyzed by simultaneously recording the H-reflex in the Sol and LG. The data showed that the H-reflex amplitude of both muscles behaved differently compared with the voluntary EMG activity. The amplitude of the H-reflex was augmented significantly during the first part of the sustained contraction and then it declined so that, at task failure, the amplitude was below the control values for both muscles. After the fatigue task, H-reflex amplitude recovered within 30 s and remained slightly, but not significantly, above its control value before returning to the prefatigue level after ~10 min of rest.

In the absence of Mmax impairment, the modulation of the H-reflex response may be attributed to a change in motoneuron excitability and Ia synaptic transmission (4, 30, 33, 40, 45). Because the changes in H-reflex amplitude were observed when the EMG was progressively increasing, they are presumably caused by a decrease in transmission along the neural elements located between the site of nerve stimulation and the motoneurons (10, 11, 23). The modulation of H-reflex amplitude in the antagonist muscles during a fatiguing contraction by the agonist muscles was likely caused by presynaptic inhibition (32, 40). This mechanism is under the influence of diverse afferent inputs and is closely controlled by central mechanisms (16, 27, 33, 34).

The different changes in antagonist aEMG activity and H-reflex amplitude discount a role for spinal mechanisms in controlling the level of antagonist coactivation during sustained contractions. The present data are thus consistent with the hypothesis of a supraspinal control of coactivation (31, 32, 38). The parallel increases in EMG of the agonist and antagonist muscles during fatiguing contractions (37) has led to the suggestion that coactivation is mediated by a central descending “common drive” (9). This hypothesis suggests that the central nervous system may control the motoneuron pool of each muscle of an agonist-antagonist pair by a single input when they are in-
volved in a given task. The present results, however, include a biphasic behavior of the H-reflex in the antagonist muscles during the course of the fatigue task that is not consistent with coactivation being controlled by a common drive. Rather, the results suggest a more subtle regulation of the drive to the antagonist motoneuron pool. An alternative hypothesis is that there is a progressive depression of reciprocal inhibition of the antagonist muscles by the central nervous system, leading to an enhanced coactivation by increasing the excitability level of the antagonist motoneurons (31). The observation that the H-reflex in the antagonist muscles first increased and then decreased during the fatigue task, despite a gradual enhancement of the agonist activation (as tested by V-wave), indicates a role for presynaptic inhibition. Regulation of coactivation during a sustained submaximal contraction, therefore, appears to be centrally modulated at a presynaptic level.

In summary, the results show that the coactivation ratio remained constant during a sustained submaximal contraction of the dorsiflexor muscles and consequently does not contribute to task failure. The different changes in voluntary EMG activity and spinal reflex excitability in the antagonist muscles during the fatiguing contraction support the concept that the level of coactivation is controlled by supraspinal rather than spinal mechanisms. The findings further indicate that the level of coactivation cannot simply be mediated by a central descending common drive to the motoneuron pools of the agonist-antagonist muscle pairs but rather suggests a more subtle regulation of the drive to the motoneurons that innervate the antagonist muscles.

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REFERENCES


