Spinal Mechanisms Contribute to Differences in the Time to Failure of Submaximal Fatiguing Contractions Performed With Different Loads

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Klass M, Lévénez M, Enoka RM, Duchateau J. Spinal mechanisms contribute to differences in the time to failure of submaximal fatiguing contractions performed with different loads. J Neurophysiol 99: 1096–1104, 2008. First published January 9, 2008; doi:10.1152/jn.01252.2007. This study compared the mechanisms that limit the time to failure of a sustained submaximal contraction at 20% of maximum when the elbow flexors either supported an inertial load (position task) or exerted an equivalent constant torque against a rigid restraint (force task). The surface electromyogram (EMG), the motor-evoked potential (MEP) in response to transcranial magnetic stimulation (TMS) of the motor cortex, and the Hoffmann reflex (H-reflex) and maximal M-wave (Mmax) elicited by electrical stimulation of the brachial plexus were recorded in biceps brachii during the two tasks. Although the time to failure for the position task was only 44% of that for the force task, the rate of increase of the average EMG (aEMG; % initial MVC) and MEP area (% Mmax) did not differ significantly during the two tasks. At task failure, however, the increases in normalized aEMG and MEP area were significantly (P < 0.05) greater for the force task (36.4 and 219.9%) than for the position task (22.4 and 141.7%). Furthermore, the superimposed mechanical twitch (% initial MVC), evoked by TMS during a brief MVC of the elbow flexors immediately after task failure, was increased similarly in both tasks. Although the normalized H-reflex area (% Mmax) decreased during the two fatiguing contractions, the reduction was more rapid and greater during the position task (59.8%) compared with the force task (34.7%). Taken together, the results suggest that spinal mechanisms were a major determinant of the briefer time to failure for the position task.

INTRODUCTION

The time to failure for a sustained submaximal isometric contraction of the elbow flexors is briefer when maintaining a constant elbow angle while supporting an inertial load compared with exerting an equivalent torque against a rigid restraint (Hunter et al. 2002; Maluf and Enoka 2005; Rudroff et al. 2007). When the task involved supporting an inertial load, the subject was required to keep the position of the arm constant by matching the elbow angle to a target angle displayed on an oscilloscope. The other task required the subject to sustain a constant force by matching the force exerted by the arm on a transducer to the displayed target force. The rate of increase in the electromyographic (EMG) activity for the elbow flexor muscles was identical for the two tasks, but reached a lower percentage of maximum EMG at task failure for the position task. Nonetheless, mean arterial pressure, heart rate, rating of perceived exertion, and the fluctuations in motor output (force and acceleration) all increased more rapidly during the position task, suggesting greater central neural activity during this task. Despite the different EMG amplitudes at task failure, the level of fatigue experienced by the subjects, as indicated by the decline in the torque during a maximal voluntary contraction (MVC), was similar at task failure for the two tasks.

To assess the influence of load type on the motor output from the spinal cord, Mottram et al. (2005) compared the discharge characteristics of the same motor unit in biceps brachii during the performance of the two tasks. Consistent with the previous observations of enhanced central activity during the position task, the motor unit recordings indicated a greater reduction in discharge rate, a more pronounced increase in discharge rate variability, and an augmented recruitment of motor units during the position task compared with the force task. Because the recordings were obtained from the same motor unit in both tasks, these results indicate that the motor neuron pool receives greater amounts of synaptic input during the position task than that during the force task, despite the two tasks requiring a similar net muscle torque.

The difference in excitation received by the motor neuron pool in the two tasks can originate from either supraspinal or spinal sources. Descending input to the motor neuron pool, for example, can become insufficient during both sustained maximal (Gandevia et al. 1996; Taylor et al. 2006) and submaximal contractions (Søgaard et al. 2006). Furthermore, motor neuron disfacilitation (Bongiovanni and Hagbarth 1990; Maecefeld et al. 1991) and presynaptic inhibition (Duchateau and Hainaut 1993; Kostyukov et al. 2005; Pettorossi et al. 1999) can reduce the excitatory feedback from the periphery to the motor neuron pool. The purpose of the study was to compare selected inputs to the motor neuron pools of the elbow flexor muscles during performance of the force and position tasks. The comparisons involved motor-evoked potentials (MEPs) in response to transcranial magnetic stimulation (TMS) and the Hoffmann reflex (H-reflex) elicited by electrical stimulation of the brachial plexus at Erb’s point. The results suggest that spinal mechanisms, presumably associated with a reduced peripheral excitatory input to the motor neuron pool, have a significant influence on limiting the time to failure of the position task.
with a stimulator (Grass S88K, Astra-Med, West Warwick, RI) that was triggered by a digital timer (Master-8, AMPI, Jerusalem, Israel). The cathode and the anode (silver disks, 8 mm in diameter) were located at the supraclavicular fossa and over the acromion, respectively. The intensity of the electrical stimulus was increased gradually until the M-wave evoked in biceps brachii and triceps brachii reached a plateau while the subject was at rest. The stimulus amplitude was then set to 20–30% above this level.

The H-reflex was evoked in biceps brachii with a submaximal electrical stimulus with the same electrodes used for the M-wave. Because it was difficult to distinguish the H-reflex from the M-wave when the muscle was relaxed, it was elicited during a steady isometric contraction (20% MVC) of the elbow flexors. Stimulus intensity was close to the motor threshold and delivered at a constant rate of 3 Hz. Up to 60 responses were averaged to increase the signal-to-noise ratio. The H-reflex was identified by the criteria proposed by Miller et al. (1995): 1) appearance at a lower stimulus intensity and longer latency than the M wave; 2) disappearance on relaxation of the contracting muscle; when the stimulus intensity was sufficient to induce the reflex response but not the M-wave; and 3) occlusion as stimulus intensity increased and the amplitude of the M-wave increased.

**Experimental setup and mechanical recording**

Subjects were seated in an adjustable chair with the right arm abducted slightly and the elbow resting on a padded support (Hunter et al. 2002). The upper arm was vertical and the forearm horizontal. The forearm was held in a position midway between pronation and supination by a custom-made orthosis. The isometric torque exerted by the elbow flexors in the vertical direction was recorded by a strain-gauge transducer (TC 2000-500, linear range 0–2,200 N, sensitivity 30 mV/N; Kulite, Basingstoke, UK) that was rigidly attached to the orthosis. The signal was amplified (bandwidth DC, 300 Hz; AM 502, Tektronix, Beaverton, OR) and displayed on an oscilloscope in front of the subject. Elbow angle during the position task was measured with an electrogoniometer (SG110 and K100; Penny & Giles Controls, Cwmfelin-fach, Gwent, UK) that was taped to the lateral side of the elbow joint. The output of the electrogoniometer was displayed on an oscilloscope in front of the subject. The subject’s wrist and forearm were placed in the custom-made orthosis and an inertial load equivalent to 20% MVC torque was suspended from it at the same location that contacted the force transducer (distal part of the forearm) during the force task.

**EMG recordings**

Voluntary and electrically evoked EMG activity in the biceps brachii and triceps brachii were recorded with bipolar surface electrodes (silver disk electrodes, 8 mm in diameter). One electrode was positioned over the midbelly of the biceps brachii and the long head of the triceps brachii. The second electrode was placed 3 cm (center-to-center) distal to the first one and the ground electrode was located over a bony prominence on the right elbow. All EMG signals were amplified (×1,000) and filtered (10 Hz to 1 kHz) by a custom-made differential amplifier. The EMG, torque, and angle signals were acquired concurrently on a personal computer at a sampling rate of 2 kHz with a data-acquisition system and analyzed off-line with AcqKnowledge analysis software (Model MP 150, Biopac Systems, Santa Barbara, CA).

**Evoked responses**

**BRAHCHIAL PLEXUS STIMULATION.** Stimuli (rectangular pulses, 0.5-ms duration) were delivered to the brachial plexus at Erb’s point to evoke a maximal M-wave (Mmax) and Hoffmann reflex (H-reflex) with a stimulator (Grass S88K, Astra-Med, West Warwick, RI) that was triggered by a digital timer (Master-8, AMPI, Jerusalem, Israel). The cathode and the anode (silver disks, 8 mm in diameter) were located at the supraclavicular fossa and over the acromion, respectively. The intensity of the electrical stimulus was increased gradually until the M-wave evoked in biceps brachii and triceps brachii reached a plateau while the subject was at rest. The stimulus amplitude was then set to 20–30% above this level.

The H-reflex was evoked in biceps brachii with a submaximal electrical stimulus with the same electrodes used for the M-wave. Because it was difficult to distinguish the H-reflex from the M-wave when the muscle was relaxed, it was elicited during a steady isometric contraction (20% MVC) of the elbow flexors. Stimulus intensity was close to the motor threshold and delivered at a constant rate of 3 Hz. Up to 60 responses were averaged to increase the signal-to-noise ratio. The H-reflex was identified by the criteria proposed by Miller et al. (1995): 1) appearance at a lower stimulus intensity and longer latency than the M wave; 2) disappearance on relaxation of the contracting muscle; when the stimulus intensity was sufficient to induce the reflex response but not the M-wave; and 3) occlusion as stimulus intensity increased and the amplitude of the M-wave increased.

**Muscle fatigue and testing procedure**

The first series of experiments investigated the effects of a submaximal fatiguing contraction on the size of the MEPs in agonist and antagonist muscles (n = 11). To familiarize the subjects with the setup and to determine the force level corresponding to 20% MVC for the elbow flexors, each experimental session began with the subject performing at least two MVCs with the elbow flexor and extensor muscles. Each MVC had a total duration of 4–5 s and the MVC were separated by 2–3 min of rest. Thereafter, the intensity of the electrical and magnetic stimulation needed to evoke the Mmax and MEP responses, respectively, was determined. After having obtained stable recording conditions, at least three control responses for MEPs and Mmax were then recorded during brief submaximal contractions sustained at 20% MVC torque (Fig. 1A). For the experiments involving the position task, control responses for MEPs and Mmax were also recorded while the subject supported an inertial load equivalent to 20% MVC torque. The control measurements concluded with the subject twice performing one MVC with the elbows flexors and one with the elbow extensors, and with a TMS stimulus being superimposed during the MVC with the elbow flexors (Fig. 1A).

The target for the force and position tasks was displayed on the oscilloscope and the subject was verbally encouraged to maintain the target as long as possible. The force task ended when the subject was unable to maintain the required torque level for a period of 5–10 s. The position task was terminated when the elbow angle declined by 10° from the target for >5–10 s despite strong verbal encouragement.
The duration of each contraction was referred to as the time to failure for the task. A TMS stimulus to evoke an MEP and an electrical stimulus at brachial plexus to elicit an Mmax were delivered successively every 30 s during the fatiguing task (Fig. 1A). Immediately after task failure, the subjects performed a 3- to 4-s isometric MVC with the elbow flexors then a 3- to 4-s MVC with the elbow extensors. A TMS stimulus was superimposed during the elbow flexor MVC.

In a second experiment, an H-reflex was recorded in the biceps brachii. The recording of a consistent response was possible in only 6 of the 11 subjects. The fatiguing protocol was similar to that of the first experiment, except that a train of 60 stimuli at a rate of 3 Hz and Mmax responses were recorded every 30 s during the 2 fatiguing contractions (force and position tasks). Immediately after task failure, the subject performed an MVC with the elbow flexors with a superimposed cortical stimulation and then an MVC with the elbow extensors. In the second experiment (B), H-reflex responses, elicited by submaximal electrical stimulation of the brachial plexus, were recorded in the biceps brachii. The fatiguing protocols were similar to those for the first experiment, except that a train of 60 stimuli (3 Hz) followed by a maximal electrical stimulation (Mmax) were delivered every minute during each task.

The amplitude and area of the MEP, H-reflex, and Mmax were measured before, during, and after the fatigue tasks. Area was measured from the rectified signal. Because the amplitude and area of the MEP and H-reflex showed similar changes, only areas are reported. To minimize the influence of activity-dependent changes in the muscle fiber membrane during the fatiguing contraction and to assess the supraspinal, motor neuronal, and spinal adjustments during the task, the MEP and H-reflex areas were normalized, at each time point, to the corresponding Mmax areas. The duration of the silent period following transcranial magnetic stimulation was measured in the contracting muscles as the interval from the stimulus artifact to the return of continuous EMG. The data recorded during the force and position tasks were compared at 20% intervals throughout the time to task failure. When reported in absolute time, either the closest response to each 20% interval is indicated or the average of two equidistant responses is reported. The rate of increase in aEMG or MEP for each muscle during the two fatigue tasks was quantified by the slope of a regression line fit to the data for individual trials.

**Statistical analysis**

Two-factor (task × time) ANOVAs with repeated measures were used to compare the EMG parameters (aEMG amplitude, H-reflex, MEP parameters, and silent period duration) from the biceps brachii and triceps brachii that were observed before, during, and after the fatigue tasks. When a significant main effect was found, a Student–Newman–Keuls post hoc test was used to test for differences among pairs of means when appropriate. The rates of increase in averaged EMG, MEP, and silent period were compared by an ANCOVA. Dependent Student’s t-tests were used to compare the time to task failure, the decrease in MVC torque, and increase in superimposed twitch torque induced by TMS during MVCs for the force and position tasks. A probability of $P < 0.05$
was chosen as significant for all analyses. Data are reported as means ± SD within the text, and means ± SE in the figures.

RESULTS

Despite the similar net muscle torque exerted by the subject for each task and comparable criteria for termination of the tasks, the time to failure for the position task (420 ± 165 s) was equal to 44% of that for the force task (958 ± 371 s; P < 0.001). Average MVC torque before the fatiguing contraction was similar for the force task session (257 ± 81 N) and the position task session (271 ± 99 N; P = 0.75) and was reduced similarly (P = 0.57) immediately after task failure for the force (38.2 ± 22.8%; P < 0.01) and position tasks (39.8 ± 18.2%; P < 0.001). The aEMG for biceps brachii during the brief elbow flexor MVCs decreased immediately after the fatiguing contraction by 35.1 ± 36.9% (P < 0.05) for the force task and by 38.5 ± 37.8% (P < 0.01) for the position task.

Average EMG during the fatiguing tasks

The amplitude of the aEMG (expressed as percentage of aEMG during the initial MVC) for the biceps brachii increased progressively throughout the fatiguing contraction for both tasks (Fig. 2). The aEMG of the biceps brachii was similar at the beginning of the force and position tasks (respectively, 12.9 ± 2.0 and 13.8 ± 3.2%, P = 0.30). However, the aEMG at the end of the force task (36.4 ± 25.6%) was significantly greater (P < 0.001) than that measured at the termination of the position task (22.4 ± 9.4%; Fig. 2). Nonetheless, the average rates of increase of aEMG during the fatiguing contractions were similar for the force and position tasks (1.44 ± 0.3 and 1.27 ± 0.36%·min⁻¹, respectively; ANCOVA, P > 0.05).

The aEMG for the antagonist muscle was also similar at the beginning of the force (2.1 ± 0.1% initial MVC) and position tasks (2.2 ± 1.2% initial MVC; P = 0.96) and increased throughout both tasks (Fig. 2). The rate of increase was slightly greater during the position task (0.23 ± 0.07%·min⁻¹) compared with the force task (0.17 ± 0.04%·min⁻¹), but the difference was not statistically significant (ANCOVA, P > 0.05). The aEMG of the biceps brachii and triceps brachii, normalized to the final MVC value, increased to comparable levels (P > 0.50). At task failure, it increased by 36.1 ± 14.9% (biceps) and 45.6 ± 13.7% (triceps) for the force task and by 32.0 ± 18.5% (biceps) and 44.0 ± 21.0% (triceps) for the position task.

Changes in MEP during the fatiguing tasks

In the control contraction at 20% MVC, the average MEP area in biceps brachii did not differ significantly (P = 0.21) for the force (58.5 ± 29.4% Mmax area) and position tasks (65.7 ± 27.2% Mmax area). Similarly, the MEP area in the triceps brachii corresponded to a same percentage of the Mmax for the force (9.7 ± 10.3%) and position (9.5 ± 6.4%) tasks (P = 0.90). The latency to the onset of the MEP was 12.1 ± 0.8 and 13.9 ± 1.3 ms for biceps and triceps brachii, respectively, before beginning the force task and 11.8 ± 1.1 and 13.2 ± 1.3 ms before the position task. These values did not change significantly (P > 0.40) at task failure (respectively, 11.9 ± 1.1 and 14.0 ± 1.3 ms for the force task and 11.8 ± 1.2 and 13.2 ± 1.6 ms for the position task).

As illustrated for one subject in Fig. 3, the size of the MEP recorded in the biceps brachii increased during the two fatiguing contractions, but to a greater extent for the force task. In contrast, Mmax did not change substantially during either task in this subject. The group data (Fig. 4) show that the normalized MEP area increased during the two tasks, but to a greater extent (P < 0.001) by the end of the force task (219.9 ± 88.2% initial value; from 58.5 ± 29.4 to 111.7 ± 37.7% Mmax area) compared with the position task (141.7 ± 34.7% initial value; from 65.7 ± 27.2 to 89.0 ± 36.5% Mmax area). The rate of increase, however, was comparable for the two tasks (8.04 ± 1.32 and 6.48 ± 1.5%·min⁻¹ for the position and force task, respectively; Fig. 4A). Mmax area declined by 16.7 ± 21.7% (P < 0.05) during the force task and by 9.7 ± 14.9% (P < 0.05) during the position task. The normalized MEP area recovered by 1 min after the fatiguing contraction for both tasks. In contrast, the normalized MEP area for triceps brachii increased to a similar level at the end of the force (from 9.7 ± 10.3 to 19.9 ± 21.5% Mmax area) and position tasks (from 9.5 ± 6.4 to 18.6 ± 16.4% Mmax area; Fig. 4B), but the rate of increase was significantly greater (P < 0.05) for the position task (14.7 ± 2.3%·min⁻¹) compared with the force task (7.9 ± 2.2%·min⁻¹). The MEP area returned to initial values within 1 min for the force task and 2 min for the position task.

The superimposed twitch evoked in the elbow flexors by the motor cortical stimulation during the brief MVC increased similarly (~75%) at the end of the two tasks. The evoked increment increased from 4.6 ± 4.7 to 8.1 ± 4.1% initial MVC torque (P < 0.01) before and after the force task, and from 5.2 ± 4.0 to 9.1 ± 8.1% initial MVC torque (P < 0.05) for the position task.

Silent period following transcranial stimulation

The duration of the silent period that followed the MEP was 131.5 ± 28.6 ms at the beginning of the force task and 137.3 ±
30.4 ms at the start of the position task. The duration of the silent period increased progressively and similarly during the two tasks and reached, at the end of the fatiguing contraction, 118.2 ± 22.9% of the initial value for the force task and 109.2 ± 21.2% for the position task (Fig. 5). However, the increase was statistically significant only for the force task (ANOVA; P < 0.001). The duration of the silent period returned to its initial value within 1 min after the fatiguing contraction.

The duration of the silent period during the brief MVCs of the elbow flexor muscles increased significantly from 93.1 ±

![Fig. 3. EMG traces in response to cortical stimulation (MEPs) and maximal electrical stimulation of the brachial plexus (Mmax) recorded in the biceps brachii of one subject at the beginning, middle, and end of the force (left) and position (right) tasks. MEP size increased more during the force task compared with little change in Mmax amplitude for both tasks.](image)

![Fig. 4. Change in MEP area (% initial) recorded in the biceps brachii (A) and triceps brachii (B) during the force (○) and position (●) tasks. Each data point represents mean ± SE at 20% interval of the time to task failure, with the value normalized to the subsequent Mmax area recorded immediately afterward. Significant difference from initial value: *P < 0.05; **P < 0.01; ***P < 0.001. Significant difference between force and position tasks at task failure: †P < 0.05.](image)
15.4 to 131.4 ms (P < 0.01) from before to after the force task, but not for the position task (96.9 ± 13.0 to 114.8 ± 45.1 ms; P = 0.26).

Change in H-reflex during the fatiguing tasks

A distinct, stable H-reflex was obtained in only 6 of the subjects; their performance was otherwise representative of the whole group of subjects. Nonetheless, the duration of the two tasks was slightly briefer (~15%) for this subset of subjects in the second experiment, presumably because of increased muscle activation induced by the trains of electrical stimuli needed to evoke H-reflex responses. However, the percentage of the time to task failure for the position task compared with that for the force task did not differ statistically (P > 0.05) between the 6 subjects for the second experiment (41%) and the 11 subjects with the first experiment (44%). As in the first experiment, the aEMG during the sustained submaximal contraction for the 6 subjects during the second experiment increased to a greater extent (P < 0.05) for the force task (from 11.5 to 31.6% of initial MVC) than for the position task (from 12.9 to 20.9% of initial MVC). In addition, there was a similar decrease in MVC force (30.9 ± 6.4 and 32.3 ± 12.0%, respectively) and corresponding aEMG (26.7 ± 13.4 and 27.6 ± 13.1%, respectively) at the end of the fatiguing contractions for both the position and force tasks.

The average H-reflex area recorded during the control submaximal contraction was 4.1 ± 1.7 and 4.3 ± 1.9% of the Mmax for the force task and position task, respectively. Although the initial value was similar (P = 0.65) for the two tasks, the change during the fatiguing contraction differed for the two tasks (ANCOVA, P < 0.01; Fig. 6). The normalized H-reflex area remained relatively constant during the first 60% of the force task (Fig. 6B), but declined thereafter to reach 59.8 ± 24.0% initial value (P < 0.05) at task failure. In contrast, the H-reflex area declined rapidly during the position task and was reduced by 36.1 ± 20.8% (P < 0.01) after just 20% of the time to task failure and reached 34.7 ± 10.7% of initial value (P < 0.01) at task failure (Fig. 6B). The recovery of H-reflex area was also rapid and reached control values within 1 min. As for the MEP, the latency of the H-reflex did

FIG. 5. Lengthening of the silent period in biceps brachii after cortical stimulation, expressed as a percentage of the initial value, during the force (○) and position (●) tasks. Each data point represents mean value ± SE at 20% interval of the time to task failure. The silent period was measured from the stimulus artifact to the resumption of voluntary EMG activity. Significant difference from initial value: *P < 0.05; **P < 0.01; ***P < 0.001.

FIG. 6. Change in H-reflex response during the force and position tasks. A: H-reflexes recorded in the biceps brachii in response to electrical stimulation of the brachial plexus of one subject at the beginning, middle, and end of the force (left) and position (right) tasks. Each trace is the average of 60 responses. B: time course of changes for group data (mean ± SE; n = 6). Each data point is expressed as percentage of the initial value at the 20% interval of the time to task failure during the force (○) and position (●) tasks. Significant difference from initial value: *P < 0.05; **P < 0.01. The time course of H-reflex decrease was significantly (ANCOVA, P < 0.01) greater during the position task.
not change significantly from the beginning to the end of the force (10.4 ± 1.0 to 10.8 ± 1.1 ms) and position (10.4 ± 1.1 to 10.8 ± 1.1 ms) tasks (P > 0.10).

**Discussion**

The current study confirmed that although the decline in MVC torque at the end of the two fatiguing contractions was similar, the time to task failure for a sustained isometric contraction of the elbow flexors was briefer when maintaining a constant elbow angle while supporting a submaximal inertial load (20% MVC torque) compared with exerting an equivalent torque against a rigid restraint (Hunter et al. 2002; Maluf and Enoka 2005; Rudroff et al. 2007). The major new finding is that although the rate of increase in aEMG and MEP area for the biceps brachii was similar during the two tasks, the size of the H-reflex declined to a greater extent during the position task compared with the force task. Because there was a comparable decrease in voluntary output from the motor cortex during the two tasks, as estimated by the amount of increase in the amplitude of the twitch elicited by TMS during an MVC, the results suggest that spinal mechanisms, presumably associated with a reduced peripheral excitatory input to the motor neuron pool, were a major limiting factor in the time to failure of the position task.

The average time to failure for the position task in the current study was 44% of that for the force task. This observation is consistent with previous studies on the elbow flexor muscles that reported a time to failure for the position task of 46–60% of that recorded during the force task with the arm in a similar position (Hunter et al. 2002; Rudroff et al. 2007). Despite the substantial difference in the duration of the two tasks, the torque produced during an MVC at the end of both tasks declined by a similar extent (~40%). This observation indicates that the mechanisms responsible for limiting the duration of these tasks differ from those responsible for muscle fatigue (Enoka and Duchateau 2008).

**Supraspinal adjustments**

As classically observed (Duchateau et al. 2002; Fuglevand et al. 1993; Garland et al. 1994; Hunter et al. 2002; Löschner et al. 1996; Søgaard et al. 2006), the EMG activity of the involved muscles increases progressively during a sustained submaximal contraction. Despite the nonlinear summation of motor unit action potentials in the EMG signal (Keenan et al. 2005), the increase in aEMG during such tasks is usually considered to indicate an enhancement of the descending drive to the motor neuron pool (Bigland-Ritchie et al. 1986; Fuglevand et al. 1993; Garland et al. 1994; Griffin et al. 2001; Hunter et al. 2003; Löschner et al. 1996; Søgaard et al. 2006). This adjustment recruits additional motor units and increases the discharge rate of some units to maintain the target force (Carpentier et al. 2001; Garland et al. 1994). Although the rate of increase in aEMG was similar during the force and position tasks, the longer duration of the force task resulted in the aEMG increasing to a greater final value at the end of the force task. The EMG activity of the antagonist muscle (triceps brachii) also increased during the two sustained submaximal contractions of the elbow flexors (Hunter et al. 2002; Lévénez et al. 2007; Søgaard et al. 2006). Because the increase in aEMG (% final MVC) was similar for the antagonist muscles in the two tasks, the briefer time to failure for the position task cannot be attributed to enhanced opposition by the antagonist muscle.

The progressive increase in aEMG for the biceps brachii during the two tasks was accompanied by a gradual increase in the normalized MEP area. The increase in MEP area, which is consistent with an enhanced voluntary drive and augmentation of motor neuronal and cortical excitability for contractions ≤50% MVC (Martin et al. 2006a; Sacco et al. 1997; Søgaard et al. 2006; Todd et al. 2003), achieved a greater final value at the end of the force task compared with the position task. Although the rate of increase in MEP area was slightly greater for the position task than for the force task for triceps brachii, there was no statistical difference between the two tasks for biceps brachii. This result was unexpected because a more rapid recruitment of motor units during the position task (Mottram et al. 2005) should be associated with a greater rate of increase in MEP area during the position task. Perhaps MEP area underestimated the increase in descending drive during the position task due to greater phase cancellation of action potentials producing the MEP (Keenan et al. 2006), a change of the relation between descending drive and EMG activity (Søgaard et al. 2006), or a greater proportion of motor neurons being in a refractory state due to changes in intrinsic properties and the trajectory of the afterhyperpolarization (Martin et al. 2006; Todd et al. 2003).

The silent period that followed the cortical stimulation increased gradually and at a similar rate during the two tasks. Prolongation of the silent period, however, was significant only for the force task. Such lengthening of the silent period has been observed in agonist muscles during sustained and intermittent contractions at submaximal and maximal intensities (Benwell et al. 2007; Sacco et al. 1997; Søgaard et al. 2006). The depression of the ongoing voluntary EMG activity after a MEP is usually attributed to an interruption of the corticomotor output and its duration is often used as an indicator of the level of the inhibition in the motor cortex (Di Lazzaro et al. 2002; Fuhr et al. 1991; Inghilleri et al. 1993). The first 60–70 ms of the silent period likely reflect events at a spinal level, such as afterhyperpolarization and recurrent inhibition of the motor neurons (Fuhr et al. 1991; Garland and Miles 1997; Inghilleri et al. 1993), whereas the remainder results from inhibition of voluntary cortical output (Di Lazzaro et al. 2002). The silent period during the force and position tasks was >100 ms and longer than that occurring after a cervicomedullary MEP (Lévénez et al. 2007), which suggests that the increase in duration after the MEP probably includes extra inhibition at a cortical level. Furthermore, the finding of a similar prolongation of the silent period for the two tasks indicates that the cortical changes represented by the lengthening of the silent period (Benwell et al. 2006, 2007; Gandevia 2001) are not directly related to the difference in time to task failure.

The similar level of aEMG (~36 and 32% final MVC) in biceps brachii at the end of the two tasks suggests a comparable decline in activation of the muscle at the termination of the fatiguing contractions (Löschner et al. 1996; Sacco et al. 1997; Søgaard et al. 2006; Zijdewind et al. 1998). This conclusion is also supported by the similar increase in the amplitude of the superimposed twitch in response to cortical stimulation during the brief MVCs of the elbow flexor muscles after the two tasks.
which suggests a similar decline in voluntary output from the motor cortex during both tasks (Gandevia et al. 1996; Löschner and Nordlund 2002; Søgaard et al. 2006; Taylor et al. 2006). Taken together, these results suggest the adjustment in the supraspinal input to the motor neuron pools was quantitatively similar during the force and position tasks and was not a major determinant of the difference in the time to task failure for the two tasks.

Spinal adjustments

A decrease in the size of the H-reflex has been reported during both submaximal and maximal fatiguing contractions (Duchateau and Hainaut 1993; Duchateau et al. 2002; Walton et al. 2002). The current study demonstrates that the decrease in the normalized H-reflex area during a submaximal fatiguing contraction depends on the type of load supported during the task. The normalized H-reflex area decreased substantially (~40%) within the first 20% of the time to failure for the position task and then more gradually to reach about 35% of the initial value at task failure. Because the decrease in H-reflex area occurred concurrently with an increase in aEMG and MEP size, the decline was presumably due to a decrease in transmission along the pathway from the site of Ia axon stimulation to the motor neurons that responded to the stimulus (Duchateau et al. 2002; Kuwabara et al. 2002). Despite an increase in the descending drive during a sustained submaximal contraction, however, some motor neurons may have been in an inactivated state at the time of arrival of the stimulus and thereby could have contributed to the decrease in the H-reflex. Deactivation of motor units can occur during submaximal fatiguing contractions (Carpentier et al. 2001; Garland et al. 1994; Peters and Fuglevand 1999) and may, in part, explain why the MEP increased less during the position task. It is also possible that the greater decrease in H-reflex area could result from an increased refactoriness of the Ia axons (Burke and Gandevia 1999) due to the heightened activation of this reflex pathway during the position task (Maluf et al. 2005). Nonetheless, the rapid and substantial decrease in H-reflex area and its recovery within 1 min are unlikely attributable to axonal refactoriness alone because the effect is greater in motor axons and it takes about 10 min to recover from the depression (Burke and Gandevia 1999; Vagg et al. 1998).

The remaining two main mechanisms that could explain the greater and more rapid decline in H-reflex area during the position task are motor neuron adaptation and a change in afferent feedback. Recordings obtained in experimental animals have shown that the decline in motor unit discharge during repetitive activation of a motor neuron can be attributed to adaptation of its intrinsic properties (Kernell and Monster 1982; Nordstrom et al. 2007). However, the greater decrease in mean discharge rate and increase in discharge variability of the same motor units during the position task compared with the force task suggest the involvement of other mechanisms (Mottram et al. 2005), such as variation in the synaptic input from peripheral sources to the motor neuron pool.

The earlier decrease in H-reflex area in biceps brachii during the position task, despite the similar rate of increase in the TMS-elicited MEP in biceps brachii during the two tasks, suggests that the difference in the synaptic input received by the motor neuron pool likely involves presynaptic rather than postsynaptic mechanisms (Maluf et al. 2007). The current study cannot differentiate between disfacilitation of the motor neuron pool due to progressive withdrawal of spindle-mediated fusimotor support (Bongiovanni and Hagbarth 1990; Macefield et al. 1991) and presynaptic inhibitory mechanisms (Kostyukov et al. 2005; Pettorossi et al. 1999). Both mechanisms likely contribute to the reduction in excitatory input to the motor neuron pool during fatiguing contractions (Gandevia 2001). This interpretation is consistent with the observation that the application of muscle vibration during a sustained fatiguing contraction, which enhances the excitatory input from Ia afferents to the motor neuron pool, can transiently restore the discharge rate of motor units (Bongiovanni and Hagbarth 1990; Grande and Cafarelli 2003; Griffin et al. 2001). Furthermore, both indirect evidence and direct evidence indicate that input delivered to the spinal cord by group III–IV afferents can increase presynaptic inhibition and reduce the afferent feedback to the motor neuron pool (Duchateau and Hainaut 1993; Kostyukov et al. 2005; Pettorossi et al. 1999). This suggestion is supported by a more rapid increase in mean arterial pressure during the position task (Hunter et al. 2002, 2005; Rudroff et al. 2007). The greater activation of group III and group IV afferents in the position task is accompanied by a faster recruitment of motor units (Mottram et al. 2005) and muscle mass, presumably due to heightened activation of the stretch reflex pathway in this task; the amplitude of the stretch reflex is augmented when a limb acts against a compliant load compared with a rigid restraint (Akazawa et al. 1983; De Serres et al. 2002). Thus the greater group III–IV feedback during the position task probably contributes to the greater presynaptic inhibition during this task. With an increase in presynaptic inhibition, the excitatory input from the muscle spindle to the motor neuron pool is reduced and the descending drive must compensate for reduced peripheral afferent feedback to generate the required net muscle torque. However, it appears that group III–IV feedback can also depress the excitability of the motor neurons that innervate the elbow extensors, whereas there is an opposite action on the elbow flexors (Martin et al. 2006b). Thus in addition to direct inhibition or facilitation of motor neurons that innervate different muscle groups, group III–IV afferents can exert an indirect effect by modulating the level of input received by a motor neuron pool during a fatiguing contraction through presynaptic mechanisms. Regardless of the exact mechanism, the faster increase in descending drive likely explains why the available motor units are recruited more rapidly (Mottram et al. 2005) and the contraction is briefer in the position task.

In conclusion, the current study suggests that the longer time to failure for the force task compared with the position tasks cannot be attributed to a difference in the descending drive to the motor neuron pool or a change in coactivation of the antagonist muscle. Rather, the data suggest that the duration of the position task may be limited by spinal mechanisms, presumably due to a reduction in the peripheral excitatory input to the motor neuron pool. The premature failure in position task may thus be attributable to an inability of central processes to compensate for the decline in peripheral feedback to the motor neuron pool. These results indicate that the spinal adjustments associated with sustaining a submaximal force for as long as possible are functionally less compromising than those associated with maintaining the position of the limb for as long as possible.