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Clinical Neurophysiology 112 (2001) 68–77

www.elsevier.com/locate/clinph

Movement-related cortical potentials associated with progressive muscle fatigue in a grasping task

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Accepted 23 August 2000

Abstract

Objective: The present research was aimed to further address the general empirical question regarding the behavioral and neurophysiological indices and mechanisms that contribute to and/or compensate for muscle fatigue. In particular, we examined isometric force production, EMG, and EEG correlates of progressive muscle fatigue while subjects performed a grasping task.

Methods: Six neurologically healthy subjects were instructed to produce and maintain 70% of maximum voluntary contraction (MVC) for a total of 5 s in a sequence of 120 trials using a specially designed grip dynamometer. Three components of movement-related potentials (*Bereitschaftspotential*, BP, *Motor potential*, MP, and *Movement-monitoring potential*, MMP) were extracted from continuous EEG records and analyzed with reference to behavioral indicators of muscle fatigue.

Results: Experimental manipulations induced muscle fatigue that was demonstrated by decreases in both MVC values and mean force levels produced concomitant to increases in EMG root mean square (RMS) amplitude with respect to baseline levels, and EMG slope. EEG data revealed a significant increase in MP amplitude at precentral (Cz and FCz) and contralateral (C3) electrode sites, and increases in BP amplitude at precentral (Cz and FCz) electrode sites.

Conclusions: The increases in EMG amplitude, EMG slope, and MP amplitudes suggest a possible link between the control signal originating in the motor cortex and activity level of the α-motoneuron pool as a function of progressive muscle fatigue. Overall, the data demonstrate that progressive muscle fatigue induced a systematic increase in the electrocortical activation over the supplementary motor and contralateral sensorimotor areas as reflected in the amplitude of movement-related EEG potentials.

Keywords: Movement-related potentials; Progressive muscle fatigue; Motor control

1. Introduction

The term *fatigue* can refer to both physical and mental exhaustion due to prolonged stimulation or exertion. As such, it is a phenomenon that is of interest to many disciplines and is used in a variety of contexts. Of particular interest is localized muscle fatigue which has been defined as an inability to maintain a required force level after prolonged use of the muscle (Latash et al., 1994). While reduction in force production is obviously detrimental in many circumstances, muscle fatigue has also been shown to impair postural stability (Johnston et al., 1998), muscle coordination (Carpenter et al., 1998), and the control of limb velocity and acceleration (Jaric et al., 1997), and is, therefore, of significance to those interested in the area of human movement science.

One of the major complications that arises in studying this phenomenon is that both *peripheral* (Moritani et al., 1985; Bigland-Ritchie et al., 1986a,b; Enoka and Stuart, 1992) and *central* (McKay et al., 1995; Taylor et al., 1996; Ljubisablejevic et al., 1996) mechanisms contribute to the manifestations of muscle fatigue. These mechanisms are highly interactive and their independent contributions to muscle fatigue are poorly understood.

More specifically, while substantial support exists for metabolic and biochemical changes (Edman, 1992; Hultman and Sjoholm, 1986), changes in motor unit recruitment and firing rates (Moritani et al., 1986; Esposito et al., 1998), and changes in reflex mechanisms occurring with muscle fatigue (Bigland-Ritchie et al., 1986b; Woods et al., 1987; Garland and McComas, 1990; Macefield et al., 1991), evidence has also been provided as to changes in cortical excitability. To assess the excitability of the motor cortex, transcranial magnetic stimulation (TMS) has been applied to the motor cortex and changes

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1388-2457/01/$ - see front matter
CLINPH 2000074
PII: S1388-2457(00)00452-1

in both the motor evoked potential (MEP) in the muscle and the ensuing silent period (SP) seen in the EMG after an MEP, have been analyzed (Taylor et al., 1996; Ljubisavljevic et al., 1996; McKay et al., 1996; Sacco et al., 1997). Despite apparent contradictions, the rather broad conclusions drawn from these studies are that changes occurring in the MEP amplitude and SP duration associated with muscle fatigue are due to complex interactions of both excitatory and inhibitory processes in the motor cortex (Sacco et al., 1997; McKay et al., 1996; Taylor et al., 1996). However, TMS cannot provide evidence of the global changes occurring simultaneously across the cortex, nor can it provide information concerning those changes occurring over time as a result of preparation, initiation, and control of a contraction when the muscle to be used is fatigued. The use of electroencephalography (EEG), however, can potentially overcome the limitations in spatial and temporal resolution found in TMS and thus may provide additional information concerning the changes in the motor cortex during progressive muscle fatigue.

Limited research has been done in the area of muscle fatigue using EEG. Belhaj-Saif et al. (1996) used subdurally implanted electrodes in the cortex of monkeys to record its electro-cortical activity and showed a direct link between motor cortical cells and motor unit activity as revealed by EMG postpike effects in the target muscle. This finding provided evidence regarding the involvement of motor cortical cells in motor unit recruitment during fatigue. The changes in electro-cortical activity in both the frequency and time domains associated with global and local muscle fatigue have also been observed in humans (Ivanova, 1990). Specifically, the reduction of alpha-power in fronto-central areas and an increase in amplitude of movement-related potentials was documented at early stages of progressive muscle fatigue. These findings are consistent with a more recent line of research (Freude et al. 1986; Freude and Ullsperger, 1987) on the effects of muscle fatigue on movement-related potentials observed during repetitive hand contractions. A significant increase in BP amplitude starting 2000 ms prior to movement initiation was shown under a fatigue condition. In contrast, Shibata et al. (1997) used an arterial occlusion technique to induce both metabolic changes in the muscle and force deficits in task performance, similar to that seen in muscle fatigue, and observed no increase of electro-cortical negativity preceding muscle contraction. However, there was a significant increase in negativity during the maintenance phase of an isometric contraction associated with increased EMG activity under the condition of arterial occlusion. It was concluded that increased cortical activation under arterial occlusion may reflect the recruitment of additional motor units to compensate for the reduction of force production.

Following this line of research, our study aimed to further investigate EEG patterns in the time domain associated with dynamic changes in grasping performance as a function of muscle fatigue. Specifically, movement-related EEG potentials preceding (*Bereitschaftspotential*, BP and *Motor potentials*, MP according to Deecke et al., 1969) and accompanying (*Movement-monitoring potentials*, MMP, according to Grünewald and Grünewald-Zuberbier, 1983; Lang et al., 1989) a motor task were examined during progressive muscle fatigue induced by intermittent submaximal isometric contractions of the flexor muscle group involved in a grasping task. The behavioral changes were analyzed by estimating the subjects' maximum voluntary contraction (MVC), nominal force, and force variability as well as examining various indices of EMG changes at different stages of muscle fatigue. As such, this was the first study to rigorously control the task and observe the behavioral and EMG correlates of muscle fatigue while examining the EEG changes preceding and accompanying a grasping task.

2. Materials and methods

2.1. Subjects

Subjects were all right-handed (determined by self-reports), college students ($n = 6$, aged 18–25 years old) with no history of pathologies to either the hand or wrist. Subjects signed an informed consent form approved by the Institutional Review Board of The Pennsylvania State University prior to experimental sessions.

2.2. Experimental procedure

Subjects were seated comfortably in an electrically shielded room with the lights dimmed. Their forearm rested on a table while their hands were kept in a position midway between supination and pronation. Subjects' task involved performing isometric force production using a grip device which was fixed to the table (see Fig. 1). Subjects were asked to use all 5 fingers of their right hand in a power grip configuration when producing the required force. The width of the grip was determined for each subject individually by measuring each subject's hand from the tip of the middle finger to where the wrist meets the hand (see Blackwell et al., 1999). The width of the grip device was determined to be 40% of this measurement.

All subjects were given a practice session to become familiar with the behavioral task. The practice session was done on a separate day from the actual experimental session. Maximum voluntary contraction (MVC) was determined before any task was performed. To assess the MVC, the subject was asked to hold the grip device with a power grip configuration and produce as much force as possible for 5 s. The subject did this twice and the overall MVC was calculated as the average of two trials. For the actual task, the subject was presented with a horizontal target line on the computer screen that represented 70% of their MVC. Online feedback regarding the subjects' current force level (force trace) was also given. Subjects were instructed to

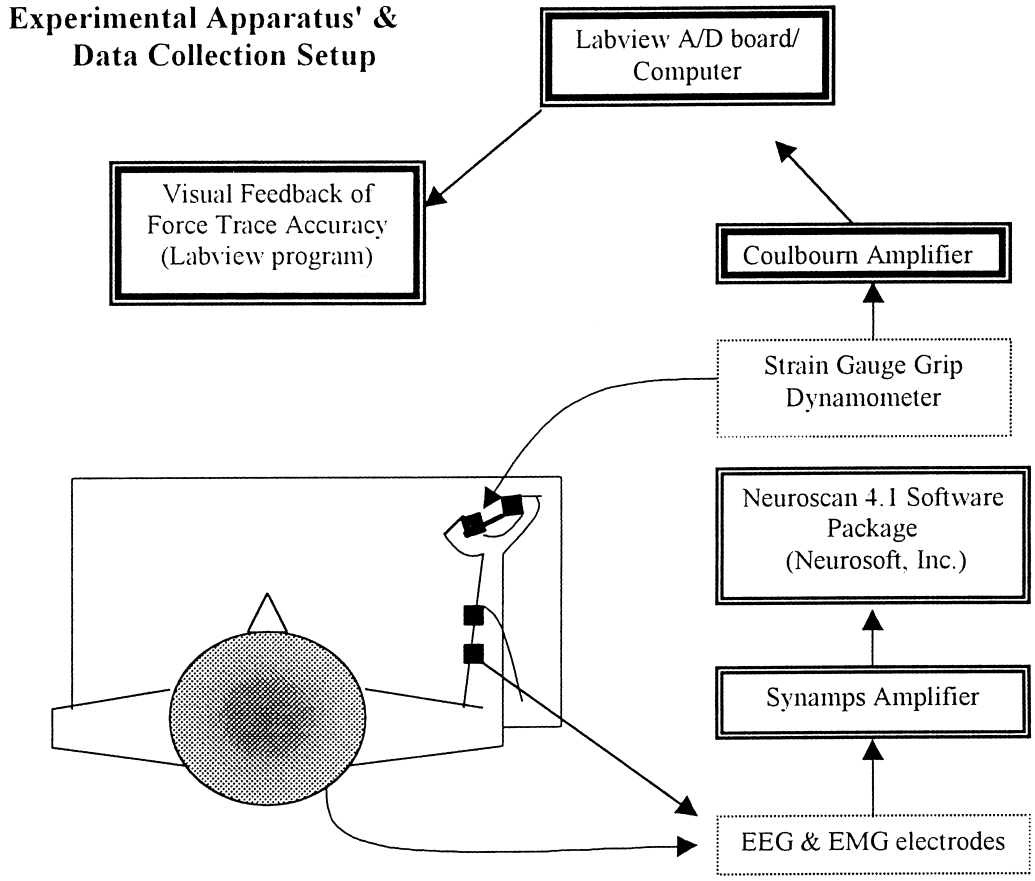

Fig. 1. Experimental set-up.

reach the target line (70% MVC) at its intersection with a vertical line corresponding to 1 s, with a constant slope (rate of force development, Newell and Carlton, 1988) and to match their force trace with the target line for an additional 4 s. Thus, total trial duration was 5 s. The contractions were self-paced and the subjects were instructed to maintain a consistent time interval in between trials. Subjects performed 3 blocks of 40 trials each. MVC was re-calculated between each block and after the last block of trials, but the target force level remained constant at 70% of the original MVC throughout the experiment. Fig. 2 shows the visual display given to the subjects.

2.3. Behavioral data acquisition

The grasping device consisted of a strain gauge dynamometer that was designed to measure the transverse force acting within an aluminum beam causing a sheer stress in the cross-section. The grasping measurement was independent of the point of force application and thus permitted the individual finger forces to sum linearly from both handles. The grip dynamometer output was directed through a Coulbourn Instruments™ Transducer Coupler Type A (strain gauge bridge). The excitation voltage was set at 7.5 V and the gain was set to (500) with DC coupling. The amplified signal was directed to a National Instruments AT-MIO-16E-10 12-bit A/D board, sampled at a rate of 100 Hz and written to the hard drive of a PC 486 computer. Calibration was achieved by using regression analysis to determine the function relating the force applied, by use of weights of various magnitudes, to that of the voltage output from each channel of the dynamometer. The behavioral data were collected and

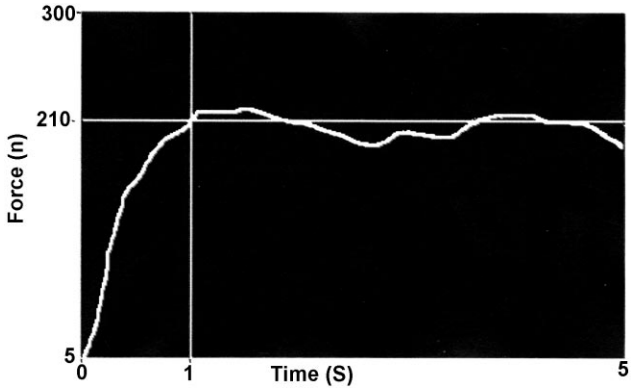

Fig. 2. Visual display given the subjects. The light colored trace is the subjects' force trace and is representative of the online feedback presented the subjects. Notice the force trace crosses the vertical and horizontal lines at the intersection.

visual feedback of subjects' force trace was provided by a specially developed program using the LabView software package (version 5.1, National Instruments, Graphical programming for instrumentation).

2.4. Behavioral data analysis

The mean and standard deviation values of the original MVC and the MVC measured after each block of trials were calculated. The means and root mean square (RMS) error of the last 3000 ms of the force trace for each of the 120 trials per subject were also calculated. Then, the means and RMS error of the 40 trials per block were averaged for each subject and the mean and RMS error were computed between subjects. The means were then normalized as a percentage of the required force level. The slope of the initial 500 ms of the force trace for each block was also determined.

2.5. EEG data acquisition

The continuous EEG was recorded with Ag/AgCl electrodes using a Quik-Cap Electrode Helmet measuring the electrical activity at electrode 30 sites: FP1, FP2, Fz, F3, F4, F7, F8, FT7, FCz, FC3, FC4, FT8, Cz, C3, C4, T3, T4, T7, T8, TP7, TP8, CPz, CP3, CP4, Pz, P3, P4, P7, P8, O1, Oz, O2, according to the international 10–20 system (Jasper, 1958). Linked earlobes served as reference and electrode impedances were kept below 5 kΩ. The signals were measured using a programmable DC coupled broadband SynAmps amplifier (NeuroScan, Inc., El Paso, TX). The EEG signals were amplified (gain 1000, recording range set for ±55 mV) and band-pass filtered in the DC to 100 Hz frequency range. The EEG data were sampled at 500 Hz, using a separate 16-bit analog-to-digital converter for each channel. Data were collected using NeuroScan's Scan 4.1 software package and written to and stored on a Pentium 166 MHz IBM computer. Each trial was visually inspected and those with artifacts were removed. At least 25 trials were kept for averaging. The grip dynamometer trace was used as the trigger with a threshold of 5N. Epochs were measured 1500 ms before and 5500 ms after the trigger threshold.

2.6. EEG data analysis

Electrode DC shift was compensated for off-line by a 4th-order trend correction of each channel over the entire recording epoch in order to remove a drift in the data that extends beyond the sample epoch (linear detrend option of NeuroScan's Scan 4.1 software). The baseline was derived from the average of the segment from 1500 to 1200 ms before the trigger point for each channel. The averaged file was low-pass filtered at 15 Hz, using a 100th order finite impulse response filter. The 3 components of the slow wave were calculated in the following manner. The BP was derived by calculating the mean from 500 ms to 600 ms before onset of the force trace. The MP was derived from calculating the mean from 250 to100 ms before initiation of the force trace. The movement-monitoring potential (MMP) was calculated as the mean from 2000 to 4000 ms after initiation of the force trace. The movement-related potentials were calculated for the FCz, Fz, F3, F4, Cz, C3, C4, Pz, P3, P4 electrode sites representing the frontal, central, and parietal cortical areas that are typically examined in the current EEG literature studying movement.

2.7. EMG data acquisition

Electromyography (EMG) was measured using two Ag/AgCl electrodes placed 3 cm apart over the muscle belly of the flexor digitorum superficialis (FDS). The FDS muscle was located using the procedure similar to Blackwell et al. (1999). The EMG was recorded using the same *SynAmps* amplifier as that of the EEG recordings. The EMG signal was sampled at 500 Hz, amplified at a gain of 200, and band-pass filtered in the 1–100 Hz frequency range. Impedance was kept below 5 kΩ. EMG data were epoched in a similar manner to that of the EEG data.

2.8. EMG data analysis

EMG signals were rectified and all of the 40 trials for each of the 3 blocks were averaged for each subject. A baseline EMG was calculated from the average of the first 3 trials of block 1 for each individual subject. RMS amplitudes (see Merletti et al., 1990, for algorithm) were calculated for the baseline EMG and the averaged EMG per block for every subject. The grand average RMS EMG amplitudes for each block were then normalized as a percentage of the baseline EMG for each subject. These normalized RMS EMG amplitudes were then averaged to obtain the normalized grand average RMS EMG amplitude for each block. In addition, the initial slope of EMG signals was analyzed to provide evidence as to differences in the excitation pulse sent to the motoneuron pool (Corcos et al., 1989) during each block. The slope was determined for the interval between 100 ms prior to and 500 ms after the onset of the force trace.

3. Results

3.1. Behavioral data

The changes in the mean and standard deviation of MVC values as a function of block as well as the mean target force levels are shown in Table 1. Notice that the mean and standard deviation of MVC prior to muscle fatigue induced by experimental tasks (original MVC) was 394.74 ± 65.9 which decreases to 229.97 ± 75 after the last block of trials (MVC 3).

A one-way ANOVA revealed a significant main effect for block, $F(3, 15) = 21$, $P < 0.00003$. Post-hoc analysis

Table 1
Original MVC and the MVCs after each block, and the target force level (70% original MVC) for each subject[a]

	Original MVC (*N*)	MVC 1 (*N*)	MVC 2 (*N*)	MVC 3 (*N*)	Target Force (*N*)
Subject 1	347.28	257.92	182.68	204.43	243.10
Subject 2	407.78	397.78	362.28	323.99	285.45
Subject 3	506.89	448.35	340.45	300.76	354.82
Subject 4	359.46	249.95	209.29	215.72	251.62
Subject 5	325.64	233.72	200.08	220.46	227.95
Subject 6	421.38	256.69	154.14	114.48	294.97
Mean	394.74 ± 65.9	307.40 ± 91.4	241.49 ± 87.4	229.97 ± 75	264.28 ± 46.1

[a] Overall means and standard deviations are also presented. All measurements are in Newtons.

revealed a significant difference between the original MVC and the MVC after block 1 ($P < 0.003$), as well as, between the MVC after block 1 and after block 2 ($P < 0.03$). Overall, the mean MVC significantly decreased to 58% of its original value ($P < 0.0003$).

Fig. 3 shows decrease in mean force production as a percentage of the required force level (70% MVC). After the last block of trials, the overall mean force level decreased to 74.9% of the target force level. A one-way ANOVA revealed a significant main effect for block with regards to the force levels produced, $F(2, 8) = 5.83$, $P < 0.03$. Post-hoc analysis showed a significant decrease in average force levels between block 1 and block 3 ($P < 0.03$).

Fig. 4 shows the initial slope of the force traces. The slopes for these trend lines are 1.4428, 1.2675, and 1.2125 with regression equations: $y = 1.4428x - 5.9659$, $y = 1.2675x - 4.394$, and $y = x - 3.8191$ (block 1, block 2, and block 3, respectively), indicating progressive decrease in slope from block 1 to block 3.

Finally, there was a significant main effect of block for the RMS error during maintenance phase of the force production, $F(2, 10) = 7.06$, $P < 0.03$. A post-hoc revealed a significant differences between block 1 and block 3 ($P < 0.03$) and between block 2 and block 3 ($P < 0.05$), indicating an increased variability in the maintenance phase of the force trace as the subjects progressed from block 1 to block 3.

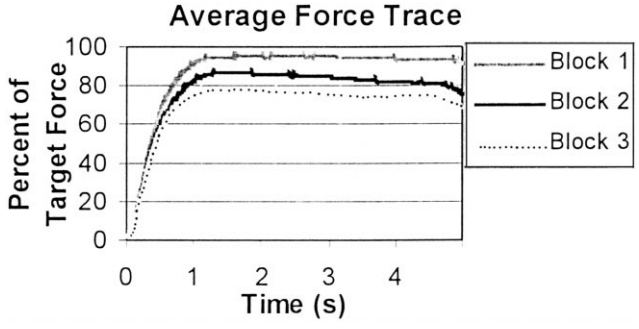

Fig. 3. Grand average force traces for each block shown as a percentage of the required target force level. Notice the increased deviation from the target force in blocks 2 and 3.

3.2. EMG data

Fig. 5 shows the changes in the initial slope of the grand average EMG for each block. Notice the increase in slope in blocks 2 and 3 with respect to that of block 1. The slopes for these trend lines are 0.2661, 0.3449, and 0.3517 with regression equations: $y = 0.2661x + 1.9364$, $y = 0.3449x + 10.658$, and $y = 0.3517x + 13.173$ (block 1, block 2, and block 3, respectively). This apparent trend may suggest an increase in the 'excitation pulse' sent to the muscle (Corcos et al., 1989), as subjects progress from block 1 to block 3.

In addition, the average normalized RMS EMG amplitude increased from block 1 to block 2 and then decreases from block 2 to block 3. While these changes show strong trends in the RMS EMG amplitudes, they are not statistically significant. Two subjects appeared to have qualitatively different EMG responses when compared to the other 4 subjects. The reasons for these differences, whether due to technical difficulties or different motor strategies, has yet to be determined empirically. While running statistical analysis on the 4 subjects showing similar EMG activity, the RMS EMG amplitude became significant at $P < 0.03$, $F(5, 15) = 5.17$. Fig. 6a,b shows the percent increase in RMS EMG amplitude from the baseline for all 6 subjects and the selected 4 subjects, respectively.

3.3. EEG data

An example of the grand average waveform for the C3 electrode site is shown in Fig. 7. As can be seen from this figure, there was gradual increase of negativity preceding the initiation and accompanying the grasping task regardless of block trials.

Fig. 8 displays the average BP, MP, and MMP amplitudes for each block and for all electrode sites. A 3 (block) × 10 (electrode sites) two-way ANOVA was performed to analyze the EEG data separately for each of the 3 EEG components. In addition, statistical analysis was performed for each component by grouping the electrode sites into the precentral (C3, C4, Cz), frontal (F3, FCz, Fz, F4), parietal (P3, Pz, P4), and midline (Pz, Cz, FCz, Fz) areas, similar to Kristeva et al. (1990) as well as on each electrode site individually.

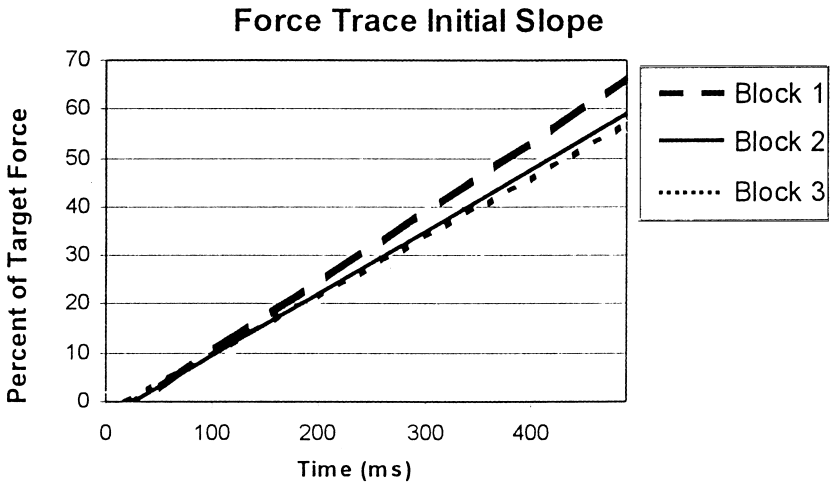

Fig. 4. Slope of the grand average force traces. Slope is calculated from the first 500 ms of the force traces.

Visually, the largest BP amplitudes occur in block 3 at all electrode sites except at F3. Specifically, there is a main effect for block in the contralateral parietal (P3), at the vertex (Cz) and at FCz electrode sites ($P < 0.05$), while a post-hoc analysis revealed a significant difference between blocks 1 and 3 ($P < 0.05$) at these electrode sites. There was also a significant main effect for electrode site in the precentral area, $F(2, 10) = 8.55$, $P < 0.01$. Specifically, negativity at Cz was significantly greater than in both C3 ($P < 0.01$) and C4 ($P < 0.01$). There was also a main effect for block at the precentral area, $F(2, 10) = 5.06$, $P < 0.05$.

Analysis of the MP for individual electrode sites revealed significant main effects for block at FCz, $F(2, 10) = 4.87$, $P < 0.05$, at the vertex, Cz, $F(2, 10) = 5.22$, $P < 0.03$, and in the contralateral sensorimotor area, C3, $F(2, 10) = 5.69$, $P < 0.03$. Post-hoc analysis revealed statistical significances between the amplitudes in block 1 and block 3 at FCz ($P < 0.05$). At Cz, significant differences were found between the amplitudes in block 1 and block 3 ($P < 0.05$) and in block 2 and block 3 ($P < 0.03$). At C3, amplitudes at block 3 were significantly larger than those at both block 1 ($P < 0.03$) and block 2 ($P < 0.03$).

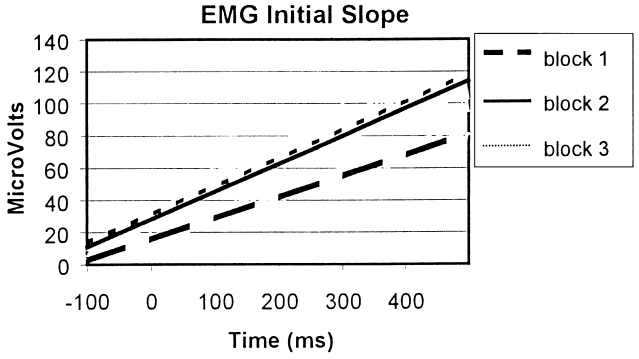

Fig. 5. The slopes of the initial EMG rise. The trend line is measured from approximately EMG onset to its plateau (~100 ms before to 500 ms after force trace onset).

While there was no statistical significance in the MMP amplitudes with regards to block, there seems to be an interesting trend. With the exception of the MMP amplitude at the contralateral parietal area (P3), there is a general decrease in MMP amplitude from block 1 to block 2 and then an increase from block 2 to block 3.

4. Discussion

The present study was designed to investigate electrocortical activity during intermittent submaximal contractions that induced progressive muscle fatigue. The intention was to substantiate claims made as to the association between the changes occurring in the cortex relative to behavioral manifestations of fatigue. The major finding

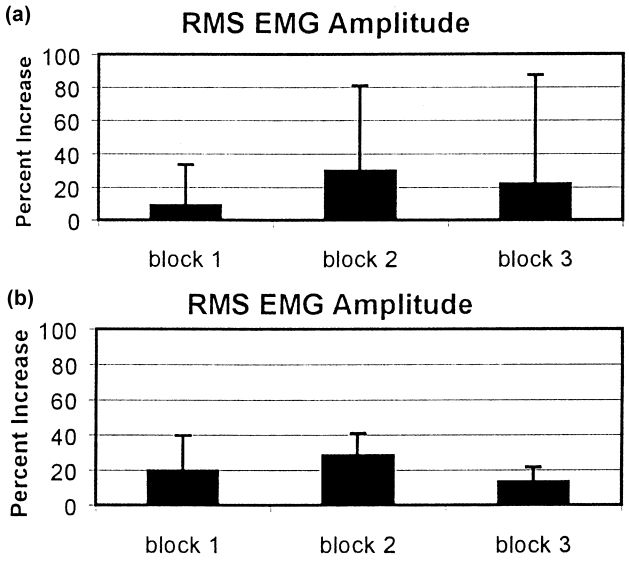

Fig. 6. (a) RMS EMG amplitude for all 6 subjects. (b) RMS EMG of selected 4 subjects. Notice the significant decrease in the standard deviation when including only the 4 subjects.

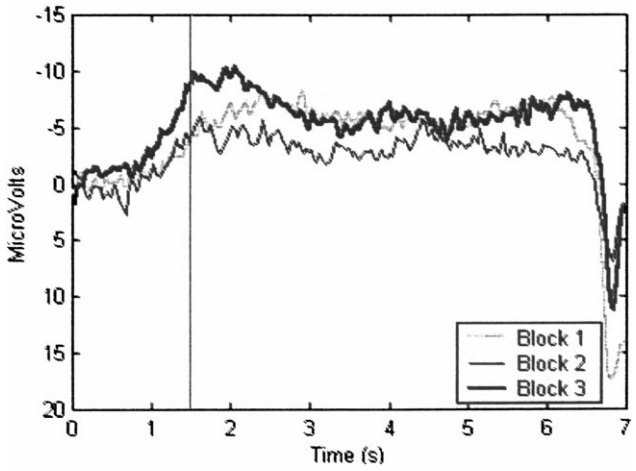

Fig. 7. Grand average EEG waveform at the C3 electrode.

from this study was a significant increase in the activity of the MP component of a MRP over both the supplementary motor (FCz, Cz electrode sites – the SMA has been suggested as the origin of the MRP because it is maximally recorded at Cz which overlies this anatomical structure (Deecke and Kornhuber, 1978; Lim et al., 1994)) and the contralateral sensorimotor (C3 electrode site) areas as a function of progressive muscle fatigue. In addition, there was also a significant increase in the activity of the BP component of the MRP at most of the electrode sites with maximum values at the midline (Fz, FCz, Cz and Pz) as muscle fatigue progressed.

Three major findings from this study suggest that progressive muscle fatigue occurred throughout the 3 blocks of our grasping task. First, fatigue was evident by both the significant decrease in force production relative to the target force level and reduction in the MVC values. These measures were also used by Bigland-Ritchie et al. (1983), Krogh-Lund (1993), and Esposito et al. (1998) as indicators of muscle fatigue. Furthermore, there was an apparent inability to maintain the target force level as the experimental session progressed, as required by the definition of fatigue (Latash et al., 1994). Second, the relationship between EMG and force level, mainly an increase in EMG without an associated increase in force level (Stephens and Taylor, 1972), was also indicative of a fatigued state. Finally, motor performance was impaired as a function of muscle fatigue (Enoka and Stuart, 1992) which was indicated by the

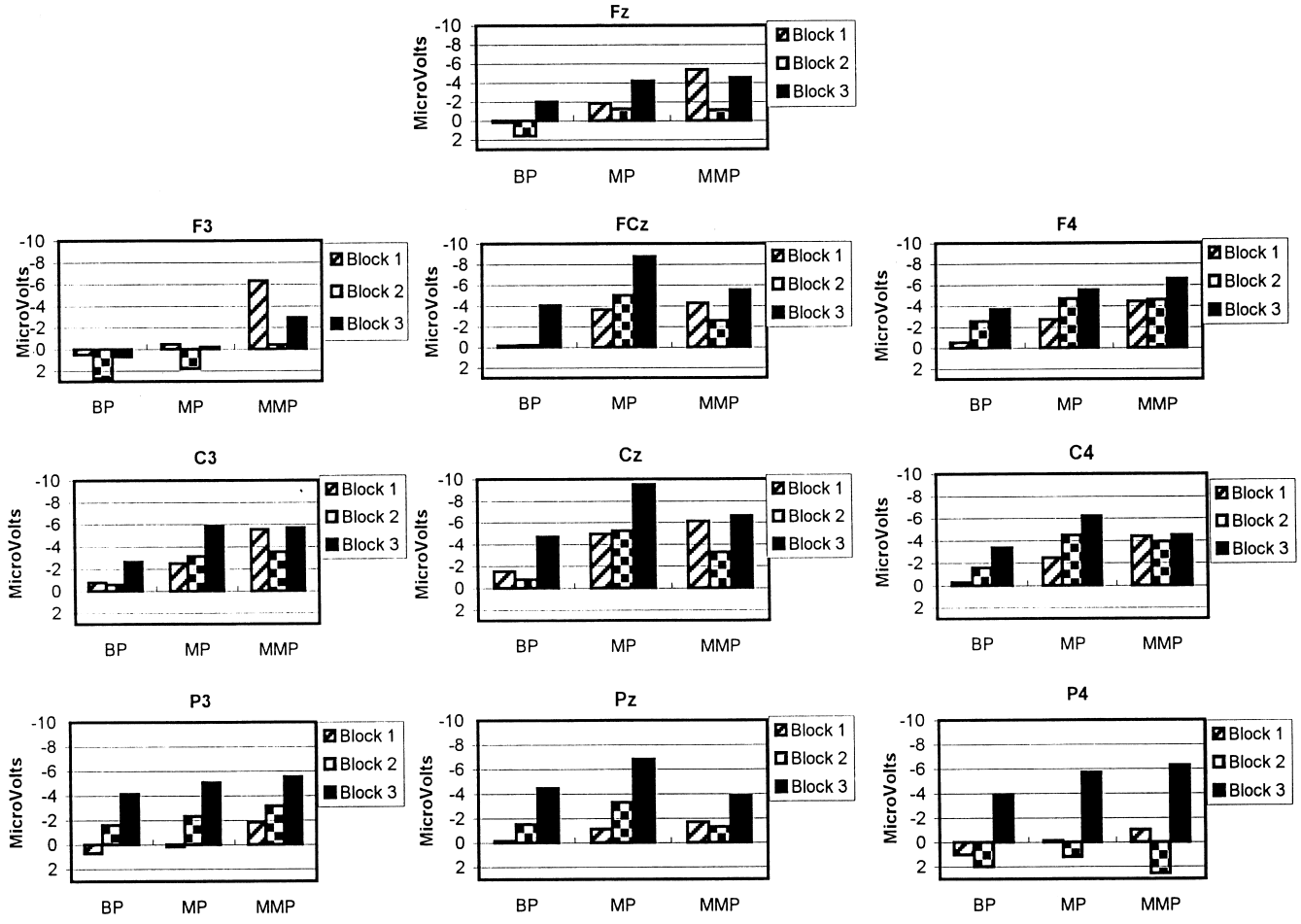

Fig. 8. Grand averaged BP, MP and MMP amplitude for all electrode sites. Note, significant increase in negativity as a function of block over midline, precentral and parietal areas for the MP component.

increased RMS error in the force traces as fatigue developed.

The MP component of the MRP was matched with the time of movement initiation and, according to Kristeva et al. (1990), reflected the scaling of the *control signal* to the muscle group involved in performing the movement task. Kristeva et al. (1990) and more recently Slobounov et al. (1999) equate this *control signal* with what Corcos et al. (1989) and Gottlieb (1993) termed the *excitation pulse*. According to Corcos et al. (1989), the excitation pulse is the signal that activates the motoneuron pool of the target muscle. This signal, which provides for the initial rate of motor unit recruitment and firing frequencies of the α-motoneurons, is scaled depending on the parameters of the movement and is reflected in the initial slope of the EMG. Note, the initial slope of the EMG in the present study also increased as fatigue progressed. It was suggested that this *excitation pulse* might be reflected at the cortical level by changes in the MRP. In the present study, the MP amplitude was significantly increased along the midline with maximum at Cz and over the contralateral sensorimotor areas, indicating an increase in the electrocortical activity as muscle fatigue progressed. This substantiates much of the earlier work (Sacco et al., 1997; McKay et al., 1996; Taylor et al., 1996) using TMS, in which increased excitability of the motor cortex was observed under muscle fatigue.

The present study provided additional evidence to suggest that there are changes occurring at both the motor cortex and the α-motoneuron pool as muscle fatigue develops (Belhaj-Saif et al., 1996). In particular, the trend towards increased RMS EMG amplitude in the early stages of fatigue seen in the present study corroborates the findings of many investigations using submaximal fatiguing protocols (Moritani et al., 1986; Merletti et al., 1990; Krogh-Lund and Jorgensen, 1992; Fuglevand et al., 1993; Krogh-Lund, 1993), and can be indicative of motor unit recruitment, changes in the firing frequencies of active motor units, and/or synchronization of active motor units (Esposito et al., 1998). Thus, the increased amplitude of the MP at the cortical level appears to be associated with changes in the recruitment of motor units and motor unit firing frequencies. In fact, this finding is in agreement with previous research by Kato and Tanji (1972) who provided a direct link between the motor potential in the cortex with the control of single motor units using human subjects. The decrease in RMS EMG amplitude in the present study seen in the later stages of fatigue has also been observed by Bigland-Ritchie et al. (1986c) and may reflect either an inhibition of motor neurons by decreases in muscle spindle activity (Garland and McComas, 1990), or an impairment of the transfer of the impulse from the motor neuron to the muscle (see Fuglevand et al., 1993). Overall, the changes occurring at both the motor cortex and the α-motoneuron pool in the early stages of fatigue might be a reflection of centrally driven mechanisms to compensate for the reduction in force capabilities under fatigue, while the decrease in EMG RMS amplitude with continued increases in cortical activity in the later stages of fatigue may indicate the occurrence of significant peripheral fatigue.

Additionally, our results substantiate the findings of Freude and Ullsperger, 1987 and Freude et al. 1986 who also observed a significant increase in the early components of MRP under fatigued muscle conditions. It has been suggested that an increase in the BP amplitude most likely reflects an enhancement in the subjects' intentional involvement, or effort level (Kristeva et al., 1990; Freude et al., 1986). Moreover, unlike the late components of the MRP, BP amplitude reflects a general preparatory strategy for movement initiation (Deecke and Kornhuber, 1978) and is unrelated to specific parameters of the task (Kristeva et al., 1990; Slobounov and Ray, 1998; Slobounov et al., 1998). Our increase in the BP component may suggest the subjects are developing a different movement strategy in order to accommodate for their inability to reach the target force level due to muscle fatigue. What *new* strategy they may be developing, however, has yet to be evaluated empirically.

Our results contradict the findings of Shibata and colleagues (1997) who found no increase in early components of MRP under muscle fatigue induced by arterial occlusion. They suggested that the decrease in the rate of force development that occurs under muscle fatigue may be the reason no increases in the BP and MP amplitudes were observed. However, these early components of MRP have been found in other work to be insensitive to the rate of force development (Slobounov et al., 1998). Furthermore, the rate of force development in the present study declined as indicated by the changing slope of the initial portion of the force traces, yet both BP and MP still increased. This suggests that the rate of force development can likely be discounted as a factor influencing MRP in this particular task and observed changes in BP and MP amplitudes are indeed reflective of muscle fatigue. The only possible explanation for this contradiction between Shibata and colleagues' work (1997) and our present study is that cortical activity involved in the compensation for metabolic changes occurring in the muscle under arterial occlusion is quite different than compensation for force deficits under movement-induced fatiguing conditions.

In conclusion, given the evidence from the present study, we argue that progressive muscle fatigue induced a systematic increase in the electrocortical activation over the supplementary motor and contralateral sensorimotor areas as reflected in the amplitude of movement-related EEG potentials. The increases in EMG RMS amplitude, EMG slope, and MP amplitudes suggest a possible link between the control signal originating in the motor cortex and activity level of the α-motoneuron pool as a function of progressive muscle fatigue. This is in agreement with a number of studies suggesting the involvement of different neural (McKay et al., 1995; Taylor et al., 1996; Ljubisablejevic et al., 1996; Freude et al., 1986; Freude and Ullsperger, 1987) and peripheral (Moritani et al., 1985; Bigland-Ritchie

et al., 1986a,b,c; Enoka and Stuart, 1992) mechanisms in the development and compensation of progressive muscle fatigue. Additional work, possibly combining TMS, EEG and EMG, needs to be done to further elucidate any definitive conclusions regarding the central and peripheral factors related to voluntary, movement-induced fatigue in various muscle groups during grasping tasks.

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