Exp Brain Res (2014) 232:2931–2943
DOI 10.1007/s00221-014-3975-0

RESEARCH ARTICLE

Unexperienced mechanical effects of muscular fatigue can be predicted by the Central Nervous System as revealed by anticipatory postural adjustments

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Received: 17 February 2014 / Accepted: 22 April 2014 / Published online: 15 May 2014

Abstract Muscular fatigue effects have been shown to be compensated by the implementation of adaptive compensatory neuromuscular strategies, resulting in modifications of the initial motion coordination. However, no studies have focused on the efficiency of the feedforward motor commands when muscular fatigue occurs for the first time during a particular movement. This study included 18 healthy subjects who had to perform arm-raising movements in a standing posture at a maximal velocity before and after a fatiguing procedure involving focal muscles. The arm-raising task implies the generation of predictive processes of control, namely Anticipatory Postural Adjustments (APAs), whose temporal and quantitative features have been shown to be dependent on the kinematics of the upcoming arm-raising movement. By altering significantly the kinematic profile of the focal movement with a fatiguing procedure, we sought to find out whether APAs scaled to the lower mechanical disturbance. APAs were measured using surface electromyography. Following the fatiguing procedure, acceleration peaks of the arm movement decreased by ~27 %. APAs scaled to this lower fatigue-related disturbance during the very first trial post-fatigue, suggesting that the Central Nervous System can predict unexperienced mechanical effects of muscle fatigue. It is suggested that these results are accounted for by prediction processes in which the central integration of the groups III and IV afferents leads to an update of the internal model by remapping the relationship between focal motor command magnitude and the actual mechanical output.

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Keywords Muscular fatigue · Anticipatory postural adjustments · Internal models · First trial

Introduction

Muscle fatigue is an acute neuromuscular state commonly experienced in daily lives as well as in sport activities. It is defined as an increase in the perceived effort necessary to exert a desired force followed by an eventual inability to produce this force (Enoka and Stuart 1992). It is well established that most of the fatigue-related processes occur peripherally, within the exercising muscles (Allen et al. 2008), due to an accumulation of metabolites in the interstitium that disrupt the functioning of the contractile proteins (Sinoway et al. 1993) and impair the neuromuscular propagation (Fuglevand et al. 1993). Fatigue-related changes can also arise from central modifications resulting in a progressive decline in the voluntary activation of muscles (Gandevia 2001; Gandevia et al. 1996; Taylor et al. 1996). It is suggested that these two components of fatigue are closely linked by feedback mechanisms originating from exercising muscles (Bigland-Ritchie et al. 1986; Garland 1991; Amann et al. 2009). Indeed, the increased discharge rates of the small diameter afferents (groups III and IV) associated with the accumulation of muscular by-products during fatiguing contractions exert inhibitory influences on motor brain structures (Taylor et al. 2000; Taylor and Gandevia 2008). As a result, when fatigued, a modulation of the link between the efferent central command and the muscular peripheral result can be observed. In other words, the same motor command generated with and without fatigue will result in different mechanical outputs (Takahashi et al. 2006).

While muscular fatigue has been widely considered in the literature to be detrimental to human motor

performances, numerous authors, however, have emphasized the neuromuscular system capacity to adapt successfully to this internal perturbation in a neurophysiological (e.g., Enoka and Stuart 1992) as well as in a behavioral view (e.g., Forestier and Nougier 1998). Indeed, biomechanical analyses have shown that subjects, when submitted to muscular fatigue, employed compensatory neuromuscular strategies in order to maintain their initial movement performances during goal-directed movements (Côté et al. 2002; Forestier and Nougier 1998; Huffenus et al. 2006; Schmid et al. 2006; Missenard et al. 2009). Moreover, and because these compensatory strategies depended on the spatial localization of muscular fatigue (Huffenus et al. 2006), it was suggested that fatigue should be considered as a contextual signal which is integrated in a feedforward manner by the Central Nervous System (CNS) in order to predict the fatigue-related mechanical effects. However, these assumptions have to be carefully considered since the experimental task used in Huffenus and collaborators' study (2006) (throwing motion) did not allow assessing parameters likely to illustrate any aspects of the predictive control.

Studies actually dealing with feedforward aspects of motor control commonly used movement–posture coordination tasks requiring the generation of Anticipatory Postural Adjustments (APAs). APAs are predictive processes of control mostly revealed by an increase in postural muscle activity prior to the onset of a focal movement. These aim to compensate for the mechanical forces and the associated destabilizing effects generated by the voluntary movement generation (Massion 1992). APAs have been frequently studied using an arm-raising task and have been shown to be specific to the mechanical effects of the upcoming self-generated movement. APA magnitudes and latencies are, for example, specific to arm movement direction (Aruin and Latash 1995a), to biomechanical (Aruin 2006; Li and Aruin 2007; Horak and Nashner 1986) and temporal constraints (Benvenuti et al. 1997; De Wolf et al. 1998; Slijper et al. 2002), to task characteristics (Bonnetblanc et al. 2004) and to peak arm acceleration (Mochizuki et al. 2004; Lee et al. 1987; Bouisset et al. 2000). The efficiency of APAs implies that, in condition of voluntary movement, the CNS is able to predict the kinematic consequences of the focal motor commands (Desmurget and Grafton 2000), thanks to an internal model of the body dynamics (Ahmed and Wolpert 2009; Miall and Wolpert 1996; Wolpert and Kawato 1998; Kawato 1999).

As underlined by Ahmed and Wolpert (2009), APA studies generally do not investigate APA adaptation time course since subjects are classically submitted to familiarization trials that are not included in the analyses. Moreover, APA efficiency in relation to movement characteristics during participants' first exposure to the task is not assessed. Yet, during everyday life activities, subjects do not always have the opportunity to become accustomed to the execution of a particular movement. An efficient motor control needs to be applied regardless of the intended movement and the corresponding environmental constraints, for example, in the case of muscular fatigue.

Some studies have focused on the link between muscle fatigue and APAs by inducing muscular fatigue at the level of postural muscles (Morris and Allison 2006; Vuillerme et al. 2002; Strang and Berg 2007; Strang et al. 2009; Kennedy et al. 2012). Results revealed that fatigue was associated with earlier APA onsets. It was suggested that this APA modulation represents an adaptive strategy offering more time to the fatigued muscles in order to reach the critical force level necessary to ensure postural stability (Strang et al. 2009; Kennedy et al. 2012). These studies, however, give limited information about the prediction capacity of the CNS as regards the mechanical effects of muscular fatigue for several reasons. First, in these studies, the fatigue level of the postural muscles could be integrated and evaluated dynamically before the first movement was executed (e.g., when subjects walked until the experimental recording area and stood upright once the fatigue protocol was achieved). Second, post-fatigue APA features were generally computed on the basis of mean performances and not on individual trial performances (including the first trial). Finally, because muscular fatigue was induced at the level of postural muscles, the mechanical effects of the focal movement were similar over the pre- and post-fatigue conditions. For this reason, the disturbance magnitude was already known and learned by the subjects when they performed the first trial post-fatigue. It has been shown that movement kinematics and its associated mechanical effects decrease after muscular fatigue (Corcos et al. 2002; Enoka and Stuart 1992; Jaric et al. 1997). Therefore, to clearly address the issue of whether the CNS can predict unexperienced fatigue-related mechanical changes in a feedforward fashion, muscular fatigue should not be induced at the level of postural muscles but at the level of the focal musculature. To our knowledge, only one study focused on the effects of focal muscle fatigue (anterior deltoids, AD) on APA features during an arm-raising task (Kanekar et al. 2008). Results revealed that focal muscle fatigue was only associated with minor APA changes. Because those changes were similar to what is observed when fatigue is induced at the level of postural muscles (i.e., earlier APA onsets), the authors concluded that the CNS uses a common feedforward strategy irrespective of the fatigue localization. However, although the participants of that study were asked in both conditions (control and fatigue) to raise their arm as fast as possible, movement kinematics post-fatigue was not significantly affected. Therefore, it is difficult to consider that the prime mover was fatigued since it

is well known and well referenced in the literature (Corcos et al. 2002, Jaric et al. 1997) that muscle fatigue leads to decreases in movement kinematic parameters. Therefore, contrarily to Kanekar and collaborators' study, we assumed that significant and specific APA modulations should be observed when focal muscular fatigue actually occurs, i.e., when movement kinematic parameters decrease.

The aim of the present study was to investigate, through APA analysis, how the CNS predictively deals with an unexperienced focal muscle fatigue state. For this purpose, APA characteristics were recorded during arm-raising movements performed at maximal velocity before and after a focal muscle fatigue procedure. We hypothesized that focal muscle fatigue would lead to slower arm movements and that the system would adapt by exhibiting smaller APAs during the first trial post-fatigue, suggesting that internal models of the body dynamics embody fatigue-related information in prediction processes.

Methods

Participants

This study included 18 voluntary healthy young adults (14 men and 4 women, age 21.3 ± 0.5 years; height 1.74 ± 0.02 m; weight 70.14 ± 2.3 kg) from the physical education department of the Savoie University (UFR-CISM—STAPS). All participants were naive about the goal of the study. The study was approved by the local research ethic committee, and the subjects' informed consent was obtained in conformity with the Declaration of Helsinki (1964) for the experimentation on humans.

Instrumentation

Electromyographic data were collected by a Datalog unit (model MWX8, Biometrics Ltd, UK). Muscle activity was monitored (1,000 Hz) using surface preamplified electrodes (type SX230-1000, Biometrics Ltd, UK). After the skin was shaved and cleaned with alcohol solution, surface electrodes were placed longitudinally over the bellies of the following muscles according to the SENIAM recommendations (Hermens et al. 1999) on the right side of the body: rectus femoris (RF), biceps femoris (BF) and erectus spinae (ES). These muscles have been evaluated because they have been shown, in pilot tests, to exhibit APAs during the arm-raising task. One electrode was also placed over the prime focal muscle of the arm-raising task, i.e., the AD. A common ground electrode was placed over the right wrist. The measured EMG was band-pass filtered (15–450 Hz) close to the recording site. The amplifier had an input impedance of 10^{15} and a common mode of rejection rate of 110 dB. The electrodes were only removed at the completion of each experimental session.

Ground reaction forces were measured with a force platform (Equi+, PF01 model, Aix-les-Bains, France). A wired precision potentiometer was fixed on the rotary hold mechanism of a cable. This cable was fixed to a hand-held spreader to collect the kinematic variables of the focal movement (1,000 Hz). These data have been sampled (1,000 Hz) and synchronized (PCIM-DAS16 card, measurement computing, A/D conversion 16 bits) with the DColl software (GRAME, Laval University, Québec, Canada). Maximal Voluntary isometric Force (MVF) was recorded for the AD using a force transducer (FUTEK, Model LSB300, USA) to assess the level of maximal force loss following the fatiguing procedures.

Experimental procedure

Participants were instructed about the protocol and familiarized with the experimental task by achieving six arm-raising movements at maximal velocity. At the beginning of this familiarization session, subjects were asked to place their feet on the force platform in a position of their choice. This position was then marked for the subjects to replace in at the beginning of each set. Note that participants were barefoot during the entire experiments. They were asked to stand straight on the force platform in a neutral (neither backward nor forward) and comfortable stance. Subjects had to raise their arm as fast as possible while holding a spreader in a self-paced manner within the 5 s following the presentation of an auditory signal. Additional weights specifically adapted to the weight of each participant were fixed to the spreader in order to increase the magnitude of APAs (Aruin and Latash 1995a; Bouisset et al. 2000). It was 0.5 kg for subjects whose weight was 55 kg or less, 1 kg for subjects weighting 56–70 kg and 1.5 kg for subjects weighting 70 kg or more. Subjects were instructed to stop their arm-raising movement at the level of a target placed at eye level. They had to maintain this position until instructed to relax, about 3 s after the completion of the movement.

After the instructions and the familiarization session, participants were equipped with the surface EMG electrodes and then performed three Maximal Voluntary isometric Contractions (MVC) for each instrumented muscle according to the SENIAM recommendations (Hermens et al. 1999). This aimed (1) to express the EMG signals in a relative expression whatever subjects specificity (age, sex, muscle typology, etc.) and (2) to compute the pre-fatigue frequency parameters of the AD EMG signal (Median Frequency, MF).

As illustrated in Fig. 1, the experimental protocol contained two experimental sessions, a *control* and a *fatigue*

session, both formed by two blocks of three sets each. Each set was made up of 6 trials (see explanations in Fig. 1). Subjects were randomly assigned in two groups. One group ($n = 9$) initially performed the control session to complete the fatigue session 2 weeks later and inversely for the other group ($n = 9$). The control session had two main goals. It was carried out (1) to evaluate whether, across trials, APAs decreased due to training or habituation effects and (2) to check that any APA adaptation during the first trial post-fatigue was only due to muscle fatigue and not to 20 min (the time necessary to achieve the fatigue protocol) without performing the arm movement.

Fatigue protocol

Participants placed themselves on their back on a massage table. They held a spreader fixed to the force transducer by an inextensible cable. The length of the cable was set in order to fix the trunk–arm angle at 45°. After a light warm-up involving antepulsion/retropulsion motions, subjects were asked to complete three MVF tests in the supine position to set the workload level for the fatigue procedure. Muscular fatigue was induced by achieving intermittent isometric contractions (20 s of workload–10 s of rest) at 70 % of MVF. Fatigue protocol was completed once participants reached an optimal fatigue level defined as the point when they could no longer maintain a force level of 70 % of MVF during ten consecutive seconds. Afterward, subjects performed a final MVF in order to calculate the loss of force and to record the frequency parameters of the right AD (MF) post-fatigue. They then immediately performed the 6 trials of the post-fatigue set. This procedure was then repeated between each set to keep a constant level of fatigue as the sets proceeded (Fig. 1). At the end of each fatiguing procedure, participants were asked not to move their arms. This was done in order to prevent the level of muscle fatigue from being integrated in a dynamic manner. For this purpose, they were systematically assisted by an experimenter to transfer from the supine to the standing position.

Data reduction and parameters analyzed

The different recorded signals, computation windows and marked points of focal movement parameters, feedforward postural activity and postural variables are summarized in Fig. 2. Position signal from the precision potentiometer was low-pass filtered with a second-order Butterworth filter and a cutoff frequency of 10 Hz. Velocity and acceleration of the arm movement were calculated using finite difference technique. Velocity (V_{peak}) and acceleration peaks (Acc_{peak}) were automatically determined (Analyse, GRAME, Québec). The first visible rise of the acceleration signal (T_0) was marked and used as a reference point to synchronize all the different signals.

Postural and focal muscle EMGs were full-wave-rectified, smoothed with a weighted average moving window algorithm (25 samples) and expressed in relative expression of the MVC values. The feedforward activity of the postural muscles was computed by integrating the EMG signals in an interval ranging from 100 ms before T_0 to 50 ms after T_0 (APA_{iEMG}). The start point of this interval

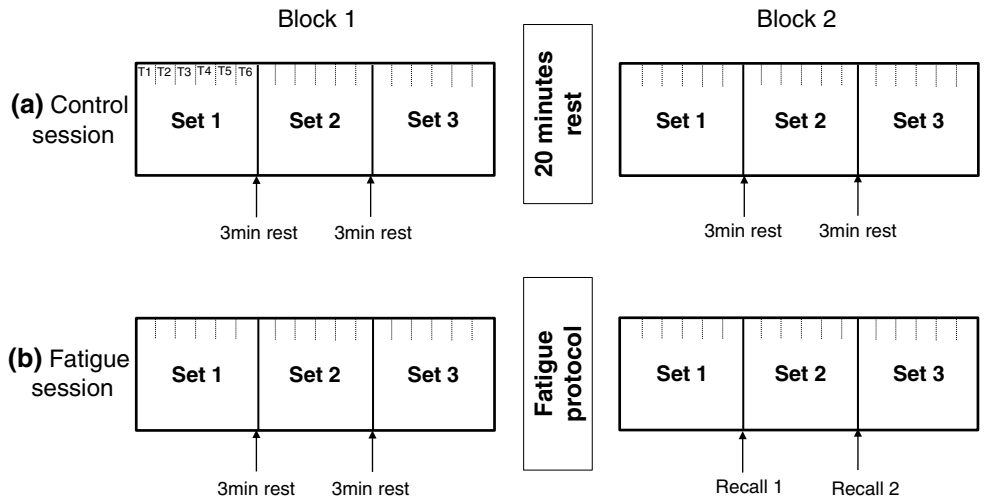

Fig. 1 Organization of the experimental procedure. **a** In the control session, participants performed two blocks of three sets in a normal muscular state. After the first block, participants were engaged in a seated rest period of 20 min (corresponding to the time necessary to perform the fatiguing procedure). **b** In the fatigue session, subjects performed the first block in a normal muscular state. Participants were then submitted to the fatiguing procedure and achieved the second block in a fatigued muscular state. Note that fatigue recalls were performed after the sets 1 and 2 of the block 2 to ensure that the fatigue level kept stable over the entire block

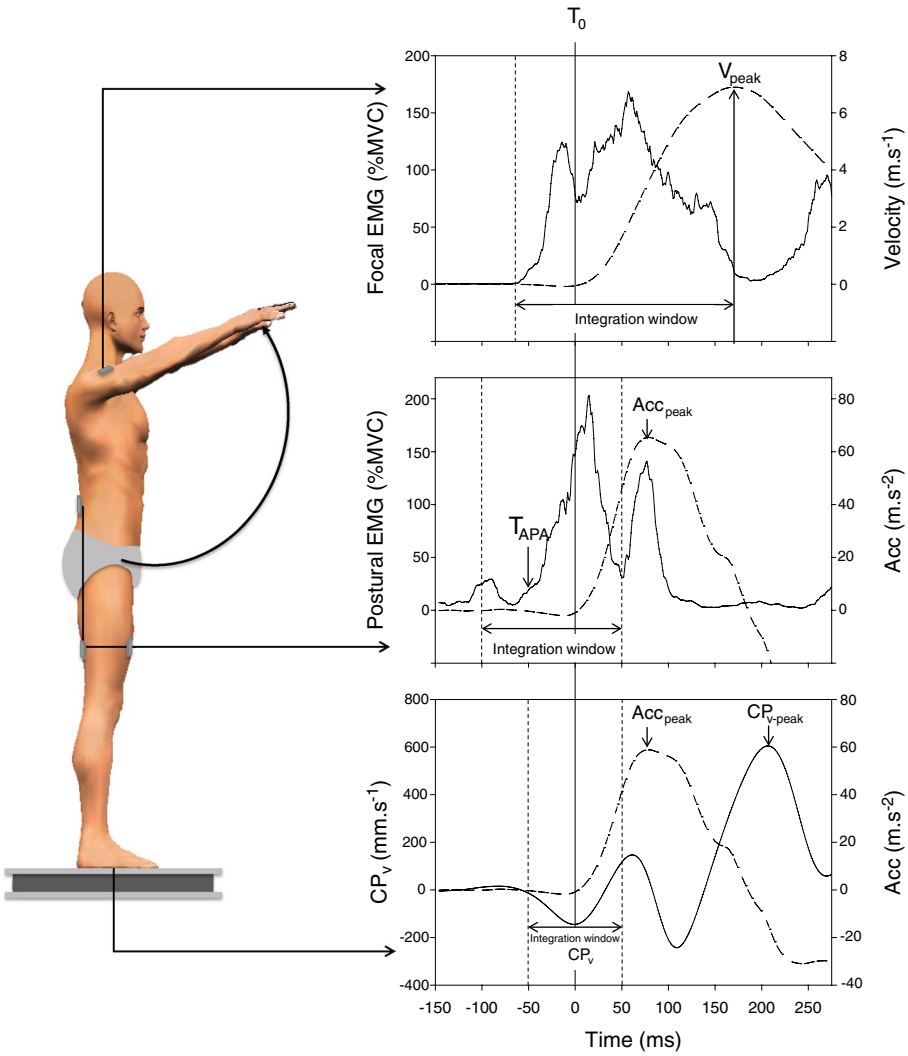

Fig. 2 Summary of the different recorded signals, computation windows and marked points of focal movement parameters (*top*), feedforward postural activity (*middle*) and postural variables (*bottom*). The scaling of the signals illustrated with *dotted lines* is presented on the right

has been selected because postural muscle activity occurring more than 100 ms before T_0 is supposed to be unrelated to the focal movement (Strang and Berg 2007). The end point (i.e., 50 ms after T_0) was chosen based on the fact that no feedback mechanisms can be observed prior to this time (Aruin and Latash 1995a, b). The activation onset of the postural muscles relative to T_0 (T_{APA}) was automatically determined as the point when muscle activity exceeded twice the baseline level of the ongoing muscle activity (Analyse, GRAME, Québec). The focal command magnitude ($Focal_{iEMG}$) was calculated as the integral value of the AD signal in an interval ranging from the activation onset of the EMG signal (determined with the same automatic procedure than for T_{APA}) to the point corresponding to V_{peak}. This integral window was chosen in order to quantify the neural drives responsible for movement acceleration (Corcos et al. 2002). Finally, we computed an index of neuromuscular efficiency (NME) expressed as follows:

$$NME = Acc_{peak}/Focal_{iEMG}$$

Lower NME values indicate a lesser neuromuscular capacity to generate movement acceleration.

Postural effect of the feedforward control was determined by computing the mean anterior–posterior velocity of the center of pressure in an interval ranging from −50 ms before T_0 to 50 ms after T_0 (CP_v). This temporal interval was selected to take into account the electromechanical delay of postural muscle activity. Based on Ahmed and Wolpert's study (2009), the CP velocity peak (CP_{v-peak}) was recorded in order to quantify the reactive control during the trials. According to these authors, higher CP_{v-peak} values indicate increased reactive postural corrections and inefficient feedforward control. Finally, in order to quantify the feedforward postural control efficiency, we computed an index (FCE) expressed as follows:

$$FCE = CP_{v-peak}/Acc_{peak}$$

This enables determination of relationship between the reactive postural control—which indirectly reflects the efficiency of APAs—to the magnitude of the mechanical

disturbance. Indexes referring to the ability to manage the self-generated perturbation by relating focal to postural parameters have already been employed in the literature (see Bouisset and Do 2008).

Statistical analyses

Before the statistical tests, all the data were checked for normality by mean of the Shapiro–Wilk test. Kinematic, postural and electromyographic parameters were submitted to two Sessions (control session vs. fatigue session) × 2 Blocks (Block 1 vs. Block 2) ANOVAs with repeated measures. Post hoc analyses (planned comparisons) were performed whenever necessary. Paired *t* tests were used to compare pre- and post-fatigue values of MF and MVF in order to control the muscular fatigue level. To investigate a potential adaptation to the fatigue-related mechanical changes during the first trial post-fatigue (Post-F1), paired *t* tests were also used to compare Post-F1 parameters to mean parameters of Block 1 and Block 2 in the fatigue session. Similar analyses were conducted over the control session for the first trial of Block 2 (Post-C1) to ensure that any changes during Post-F1 were only due to fatigue and not to 20 min without practicing the movement. Presented values are mean ± SEM. For all analyses, statistical level of significance was fixed at $p < .05$.

Results

Muscle fatigue indicators

MVF values were 213 ± 11 N before the fatiguing procedures and 151 ± 10, 150 ± 10 and 147 ± 11 N following the first procedure, the first recall and the second recall, respectively. All post-fatigue values differed significantly from pre-fatigue values ($p < .001$ for all analyses). MF values were 72 ± 3 Hz prior to the fatiguing procedures and 51 ± 2, 53 ± 2 and 53 ± 2 Hz following the first procedure, the first recall and the second recall, respectively. All post-fatigue values were significantly lower compared to pre-fatigue values ($p < .001$ for all analyses).

Focal movement parameters

Results of the two-way ANOVAs (Sessions x Blocks) for the related-focal movement parameters are presented in Table 1. Analyses of Acc_{peak} revealed a significant Sessions × Blocks interaction effect ($p < .001$). Planned comparisons showed that Acc_{peak} was not different over the two blocks of the control session ($p = .22$), whereas it decreased significantly ($p < .001$) by 27 % during the Block 2 of the fatigue session. Analyses of $Focal_{iEMG}$ demonstrated a significant interaction between Sessions and Blocks ($p < .001$). Post hoc tests yielded a significant decrease in post-fatigue as compared to Block 1 (−11.8 %, $p < .001$), while values were unchanged over the two blocks of the control session ($p = .13$). NME scores exhibited significant Sessions x Blocks interaction effects ($p < .01$) with similar values during the two blocks of the control session ($p = .89$) and significantly lower values post-fatigue during the fatigue session ($p < .001$).

In order to investigate the fatigue-related changes of the focal movement parameters during the first trial post-fatigue, the first trials of the second blocks (Post-C1 and Post-F1) were compared to Block 1 and Block 2 means. Results of the *t* tests are presented in Fig. 3. For the control session, Post-C1 Acc_{peak} was not different from Block 1 mean ($t = .93$, $p = .36$) while it was significantly lower than Block 2 mean ($t = 3.25$, $p < .01$). Post-C1 $Focal_{iEMG}$ was not statistically different from both Block 1 and Block 2 means ($p > .05$ for both analyses). Finally, Post-C1 NME scores was not different from Block 1 mean ($t = 1.5$, $p = .15$), while it was significantly lower than Block 2 mean ($t = 2.81$, $p < .05$).

During the fatigue session, Post-F1 Acc_{peak} was significantly affected by the fatiguing procedure (−27 %, $t = 11.17$, $p < .001$) but remained unchanged as compared to Block 2 mean ($t = 0.02$, $p = .98$). *T* tests revealed that Post-F1 $Focal_{iEMG}$ was not substantially affected by the fatiguing procedure as compared to Block 1 mean (−6.7 %, $t = 1.83$, $p = .09$) while it was significantly greater in comparison with Block 2 mean ($t = 2.46$, $p < .05$). Post-F1 NME scores were significantly lower as compared to both Block 1 ($t = 5.16$, $p < .001$) and Block 2 means ($t = 2.47$, $p < .05$).

Postural muscles feedforward activity

Complete results of the ANOVAs and post hoc tests are summarized in Table 1. APA_{iEMG} analyses revealed significant interactions between Sessions and Blocks for all the assessed muscles. In all cases, APA_{iEMG} remained unchanged over the two blocks of the control session, while values were significantly lower during the post-fatigue block of the fatigue session (−41.6 % for the RF, −11.2 % for the BF and −19.5 % for the ES). Similarly, T_{APA} presented significant Sessions × Blocks interaction effects for all the evaluated muscles. Values did not differ significantly between the two blocks of the control session regardless of muscles. On the other hand, burst onsets were significantly delayed for all muscles during the fatigue session.

In order to quantify a potential adaptation to the mechanical effects of focal muscle fatigue immediately after the fatiguing procedure, paired *t* tests were used to compare feedforward activity of postural muscles during Post-C1 and Post-F1 to Block 1 and Block 2 means within each session. Results are illustrated in Fig. 4a, b. For the control

Table 1 Results of the two-way ANOVAs (Sessions × Blocks) and the post hoc tests for all the parameters assessed

Variables	Session	Block 1	Block 2	*F* values			Post hoc (planned comparisons)
				Sessions	Blocks	Interaction	
Focal movement parameters							
Acc_{peak} ($m\ s^{-2}$)	Control	57.4 ± 0.7	59.1 ± 0.8	9.99**	86.59***	58.41***	ns
	Fatigue	58.6 ± 0.7	43.1 ± 0.6				***
$Focal_{iEMG}$ (% MVC)	Control	69.2 ± 1	70.8 ± 1	2.43	12.02**	27.7***	ns
	Fatigue	82.4 ± 1.2	72.7 ± 1.1				***
NME ($Acc_{peak}/Focal_{iEMG}$)	Control	0.88 ± 0.06	0.88 ± 0.07	9.59**	10.5**	9.67**	ns
	Fatigue	0.75 ± 0.05	0.62 ± 0.04				***
APA parameters							
RF (% MVC)	Control	5.9 ± 0.4	7.2 ± 0.4	0.02	1.7	9.48**	ns
	Fatigue	8.9 ± 0.5	5.2 ± 0.4				**
BF (% MVC)	Control	15.6 ± 0.5	16.3 ± 0.5	6.89*	1.06	10.76**	ns
	Fatigue	12.5 ± 0.3	11.1 ± 0.3				**
ES (% MVC)	Control	53.2 ± 1.4	53.9 ± 1.3	1.25	19.2***	17.6***	ns
	Fatigue	54.9 ± 1.2	44.2 ± 1.1				***
T_{APA} RF (ms)	Control	−37 ± 6	−39 ± 7	5.4*	10.46**	15.51**	ns
	Fatigue	−45 ± 8	−4 ± 8				***
T_{APA} BF (ms)	Control	−84 ± 4	−87 ± 2	8.69**	3.71	7.86*	ns
	Fatigue	−80 ± 1	−69 ± 2				**
T_{APA} ES (ms)	Control	−74 ± 1	−75 ± 1	11.99**	8.88**	6.46*	ns
	Fatigue	−67 ± 1	−56 ± 1				**
Postural and control efficiency parameters							
CP_v ($mm\ s^{-1}$)	Control	−1.1 ± 0.6	−1.3 ± 0.7	0.01	1.08	1.08	
	Fatigue	−1.9 ± 0.5	−0.3 ± 0.6				
$CP_{v\text{-}peak}$($mm.s^{-1}$)	Control	630.3 ± 16.4	653.9 ± 16.1	3.73	6.62*	15.6**	ns
	Fatigue	703.4 ± 16.8	509.5 ± 15				***
FCE	Control	11 ± 0.3	11.2 ± 0.3	0.05	0.02	0.19	
	Fatigue	12.1 ± 0.3	11.9 ± 0.1				

Values are mean ± SEM

* $p < .05$; ** $p < .01$; *** $p < .001$

session, statistical analyses revealed that Post-C1 APA_{iEMG} were not different from Block 1 means for all the assessed muscles. Post-C1 APA_{iEMG} were not different from Block 2 means for the BF and ES muscles while it was statistically lower for the RF ($t = 2.6$, $p < .05$). Post-C1 T_{APA} values of the BF and RF were not statistically different from both Block 1 and Block 2 means. T_{APA} of the ES was significantly earlier during Post-C1 as compared to Block 1 mean ($t = 3.87$, $p < .01$) but was not statistically different from Block 2 mean ($t = 1.9$, $p = .07$).

For the fatigue session, APA_{iEMG} of the RF and ES during Post-F1 were significantly lower compared to Block 1 means (−50 %, $t = 3.14$, $p < .01$ for the RF, −17 %, $t = 2.9$, $p < .05$ for the ES). In contrast, Post-F1 APA_{iEMG} of the BF was not statistically different from Block 1 mean (−3.6 %). On the other hand, all Post-F1 APA_{iEMG} were not statistically different from Block 2 means. Post-F1 T_{APA} values of the BF were not statistically different from both Block 1 and Block 2 means. T_{APA} of the RF and ES were significantly later during Post-F1 as compared to Block 1 means ($t = 3.53$, $p < .01$ for the RF, $t = 2.34$, $p < .05$ for the ES). Block 2 versus Post-F1 analyses for the RF and ES muscles did not reveal any significant differences.

Postural parameters and feedforward control efficiency

Results of the two-way ANOVAs (Sessions × Blocks) for the postural and feedforward control efficiency related-parameters are presented in Table 1. Statistical analyses did not reveal any effect for CP_v and FCE. However, analyses of CP_{v-peak} revealed a significant interaction between Sessions and Blocks ($p < .001$). Planned comparisons showed that values remained unchanged over the 2 blocks of the control session ($p = .54$) while they decreased drastically

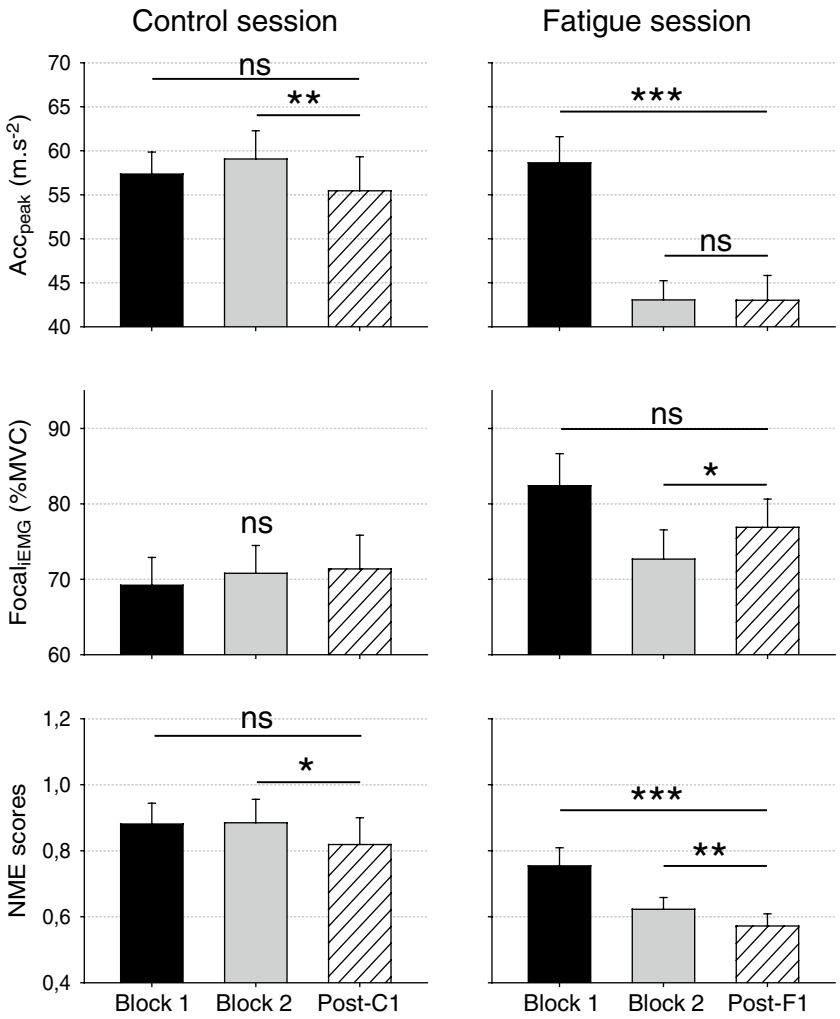

Fig. 3 Means and standard errors of the focal movement parameters (from the top to the bottom: Acc_{peak}, $Focal_{iEMG}$ and NME) for Block 1 (*left*), Block 2 (*middle*) as well as Post-C1 and Post-F1 trials (*right*) during the control and the fatigue session. *T* tests results: *$p < .05$, **$p < .01$, ***$p < .001$

during the second block of the fatigue session as compared to the first block ($p < .001$).

In order to identify a potential adaptation of the postural and feedforward control efficiency related-parameters to the mechanical effects of focal muscle fatigue immediately following the fatiguing procedure, paired *t* tests were used to compare Post-C1 and Post-F1 values to Block 1 and Block 2 means within each session. The results of the paired *t* tests related to Post-C1 and Post-F1 trials are illustrated in Fig. 5. For the control session, *t* test did not reveal any differences whatever the postural parameter (CP, CP_v and FCE). For the fatigue session, Post-F1-related analyses did not exhibit any differences for CP_v and FCE. In contrast, Post-F1 values of $CP_{v\text{-}peak}$ were significantly lower as compared to Block 1 mean ($t = 3.02$, $p < .01$) but were not different from Block 2 mean ($t = .22$, $p = .83$).

Discussion

The aim of the present study was to investigate, through APA analysis, how the CNS predictively deals with an unexperienced focal muscle fatigue state. From a global viewpoint, we hypothesized that focal muscle fatigue would lead to slower arm movements and that the system would adapt by exhibiting smaller APAs, like it is observed for movements with decreased acceleration peaks (Mochizuki et al. 2004; Lee et al. 1987; Bouisset et al. 2000). Until now, only one study focused on the effects of focal muscle fatigue on APA features (Kanekar et al. 2008). In this study, focal muscle fatigue was associated with minor changes in APA characteristics. However, although participants of that study had to perform arm movements at maximal velocity pre- and post-fatigue, the fatigue procedure used did not result in significant changes in focal movement kinematics. Therefore, it can be suggested that focal muscle fatigue did not actually occur and that these results are accounted for by a lack of modifications in the self-generated postural disturbance magnitude. Consequently, in the present study, the level of focal muscle fatigue represented a key element that had to be adequately controlled to generate significant changes in movement kinematics. The muscular fatigue protocol led to drastic reductions in both fatigue parameters

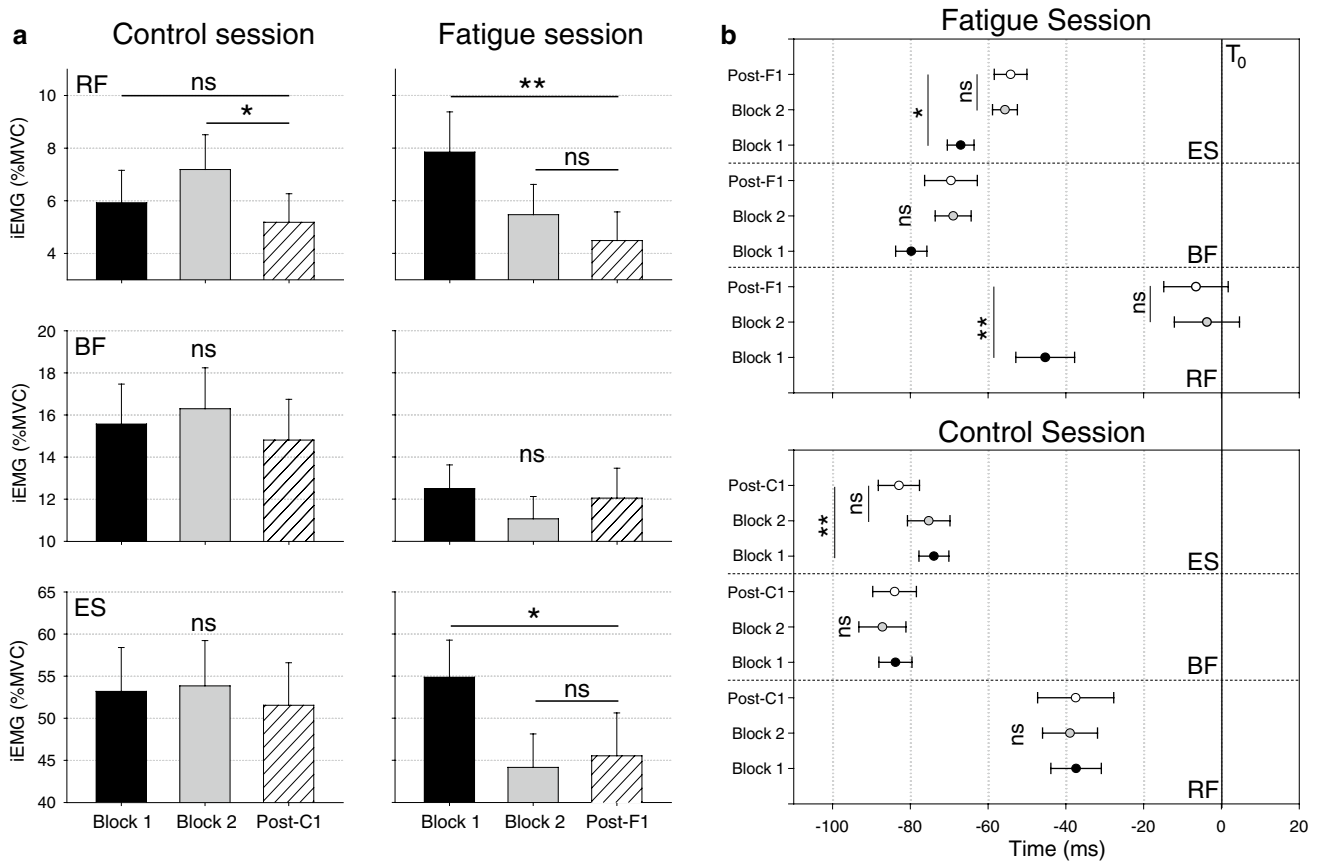

Fig. 4 Means and standard errors of the APA parameters. **a** APA_{iEMG} of the RF, BF and ES muscles for Block 1 (*left*), Block 2 (*middle*) as well as Post-C1 and Post-F1 trials (*right*) during the control and the fatigue session. **b** T_{APA} during the control and the fatigue session for the RF, BF and ES muscles over Block 1, Block 2 as well as Post-C1 and Post-F1 trials. T tests results: $*p < .05$, $**p < .01$, $***p < .001$

MVF and MF (~30 % for both parameters). These modifications were associated with significant decreases in movement kinematics (Acc_{peak} decreased by ~27 %). Although we cannot rule out that small arm movements during or following the first fatiguing procedure enabled participants to partially integrate the level of muscle fatigue, precautions were taken in order to prevent such a phenomenon. Indeed, subjects performed the fatigue protocol in a supine position by the mean of isometric contractions. Therefore, the fatiguing procedure neither allowed the postural system to evaluate the level of focal fatigue, nor to actually experience it in a dynamic manner before the first movement post-fatigue. Furthermore, an experimenter systematically helped participants to transfer from the supine to the standing posture in order to prevent dynamic arm movements before the first post-fatigue movement. Altogether, these conditions appeared to be optimal to investigate a potential adaptation to the fatigue-related mechanical changes during the first trial post-fatigue.

From a general viewpoint, results revealed that the anticipated postural muscle activity scaled to the lower fatigue-related mechanical disturbance. Indeed, the smaller APA magnitudes and the later APA onset latencies clearly demonstrate the implementation of adaptive strategies aiming to grade with the lower kinematics of the arm-raising movement (Mochizuki et al. 2004; Lee et al. 1987; Bouisset et al. 2000). These changes were fully accounted for by focal muscle fatigue as checked out with the control session-related results. The most salient result of this study is that this scaling pattern was obvious during the very first trial post-fatigue (Post-F1). Indeed, despite a potential trial-by-trial adaptation, the feedforward postural muscle commands employed during Post-F1 remained similar over the entire post-fatigue block. Moreover, the postural and control efficiency parameters revealed that these predictive neuromuscular strategies were immediately suited to the new movement context. Finally, these results were entirely attributable to focal muscle fatigue as confirmed by the Post-C1-related analyses.

It was suggested that posture and movement involve two distinct controllers, i.e., one acting on the postural and the other on the focal chain (for reviews, see Massion 1992;

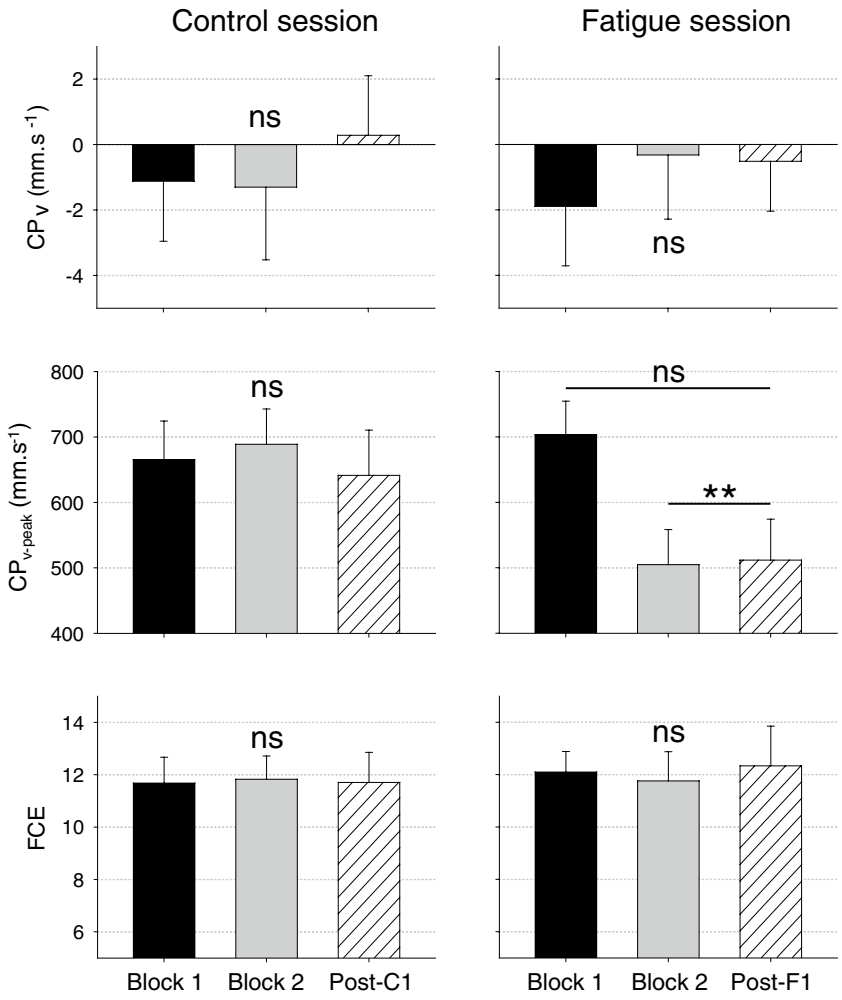

Fig. 5 Means and standard errors of the postural and control efficiency parameters (from the top to the bottom: CP_v, $CP_{v\text{-}peak}$ and FCE) for Block 1 (*left*), Block 2 (*middle*) as well as Post-C1 and Post-F1 trials (*right*) during the control and the fatigue session. *T* tests results: $*p < .05$, $**p < .01$, $***p < .001$

Massion et al. 2004; Ahmed and Wolpert 2009; Kurtzer et al. 2005; Benvenuti et al. 1997; Cordo and Nashner 1982). Within this scheme of control, the scaling of APAs to the kinematic properties of the focal movement implies that the CNS is able to predict the mechanical consequences of the upcoming focal motor commands (Desmurget and Grafton 2000). However, it has been shown that muscular fatigue alters the normal relationship that usually exists between motor command magnitude and mechanical output. For instance, at identical levels of force generated with and without fatigue, the magnitude of the motor commands, as signaled by active muscle EMG size, is greater post-fatigue (de Morree et al. 2012; Carson et al. 2002; Liu et al. 2003). This represents the recruitment of additional motor units to compensate for the loss of muscular force. In the present study, because the movement performed was in both cases (pre- and post-fatigue) maximal, the link between the mechanical output and the magnitude of the focal motor commands (NME) was expressed by relating Acc_{peak} to $Focal_{iEMG}$. Results showed that the efficiency of the neural drives directed toward the focal muscles decreased post-fatigue, i.e., that a given magnitude of command resulted in lower acceleration peaks. Moreover, the size of the focal motor commands was not significantly affected by muscular fatigue during the first trial post-fatigue despite drastic decreases in Acc_{peak}.

The internal model theory (Kawato 1999) proposes that the CNS, given the state of the body (e.g., joint position and velocity) and in response to a corollary of the motor commands (i.e., an efference copy), can predict the consequences of a movement thanks to an internal model of the motor apparatus (Wolpert and Ghahramani 2000). Although some spinal reflexes might notably participate in motor neuron firing rates, it has been assumed that the neural drives recorded peripherally reflect the centrally generated motor commands (Gandevia 2001). As a result, $Focal_{iEMG}$ values represent the motor command magnitude as well as the efference copy integrated and processed in the internal model. If the CNS predicted the postural disturbance caused by the arm-raising movement on the sole basis of the efference copy, one could have expected higher APA magnitudes. Indeed, during the first

trial post-fatigue, focal command magnitude was lower by 6.7 % while anticipated postural commands were lower by 17 % in the ES and 50 % in the RF muscles. The discrepancy in activation magnitude across postural and focal commands suggests that some neural mechanisms operated a reweighting of the motor information contained in the efference copy.

While the efference copy is used to estimate the future state of the body, it can also be transformed into sensory information. Indeed, the processing of the efference copy in somatosensory areas of the brain (Christensen et al. 2007) has been shown to provide a sense of effort, i.e., the conscious awareness of the central motor commands sent to the muscles (McCloskey et al. 1974). This central motor command further gives rise to information on limb position (Gandevia et al. 2006), on limb movement (Walsh et al. 2010) and, in normal circumstances, on the force developed by a muscle or muscular group (Lafargue et al. 2003). However, the force perceived through this central sense of effort has been shown to be altered as a result of fatiguing contractions (Carson et al. 2002). Using a force-matching paradigm, Carson and collaborators asked subjects to equalize the force level of their reference non-fatigued arm with their contralateral eccentrically-exercised arm. Their results revealed that the level of force produced by the reference arm was always underestimated by the fatigued arm despite higher amounts of motor commands (as signaled by higher amplitude of EMG activity and larger cortically evoked potentials). However, when the force level of each arm was expressed as a proportion of its current maximal force capacity, results demonstrated that the levels of force matched very closely. This study demonstrates that the relationship between the perceived and the actual motor command is altered in a way that enables the system to maintain identical proportional motor output with regard to the current force-generating capacity of muscles. The data obtained in the present study support such results but bring new insights about those processes. In Carson and collaborators' study, the force level was regulated through online mechanisms of control. In the present study, this regulation appeared in a predictive fashion and allowed the anticipated postural commands to "match" to the unexperienced acceleration-generating capacity of the focal muscles.

Although it is strongly suggested that posture and movement are organized independently, some authors proposed that posture and movement could be controlled by a unique controller acting on both the focal and the postural chains (see Massion 1992). The main prediction of this theory is the existence of a close relationship between temporal and quantitative features of focal and postural commands (Aruin and Latash 1995b,). The differential changes in activation magnitude across the focal and postural muscles as well as the changes in APA onset latency observed post-fatigue clearly do not support such an hypothesis. The results of the present study are obviously more in favor of the independent mode of control hypothesis and support the implementation of predictive centrally-mediated strategies. Which mechanisms could be responsible for such an adaptation? It could be posited that the changes in central afferent sensory inputs that accompany muscle fatigue, i.e., the increased discharge rate of group III and IV afferents, played a crucial role. Amann (2011) qualified these muscle afferents as «relating "news" to the CNS regarding the status of the muscle». Similar to proprioceptive inputs that are combined with motor outflows to provide an accurate estimate of the body state (Wolpert et al. 1995), muscular fatigue-related information might also play a role in the internal model prediction processes. In some circumstances, like transient exposures to microgravity (Chabeauti et al. 2012; Papaxanthis et al. 2005) or to unexpected force fields (Shadmehr and Mussa-Ivaldi 1994; Takahashi et al. 2006), movements are required to recalibrate internal models and to remap the relationships between feedforward muscle activation and environment-related constraints in a feedback-error-learning fashion. In other cases, some contextual signal, such as the size and the orientation of an object to grasp (Ingram et al. 2010), the size of a target to reach (Bonnetblanc et al. 2004), the Coriolis force generated by voluntary torso rotations during pointing movements (Lackner and Dizio 1994; Cohn et al. 2000) and movement direction related to earth gravity (Papaxanthis et al. 2005), allows the selection of a context-suited controller in a predictive fashion (Wolpert and Kawato 1998). It has also been shown that the brain could exert a dynamic control to facilitate the relevant contextual sensory inputs during movement planification (Saradjian et al. 2013). In this study, it appears that the changes in afferent inputs due to muscle fatigue enabled the internal models to be calibrated without any dynamic sensory experience of the muscular fatigue level. Therefore, it can be suggested that fatigue-related information was integrated in internal models during the preparation of the movement and acted as a relevant contextual signal that enabled to update the relationships between the motor command magnitude and the actual acceleration-generating capacity of muscles. In other words, group III and IV afferents may have recalibrated internal models during movement preparation in order to reweight the size of the efference copy in relation to the impaired status of the focal muscles. This led to accurate predictions with regard to the actual kinematic consequences of focal motor commands and *in fine* to the selection of a context-suited postural controller.

Conclusion and limitations

The main limitation of this study is the absence of a control condition in which subjects would have performed the focal movement in a normal muscular state at similar levels of acceleration peaks than post-fatigue. Such analyses would have enabled to accurately distinguish the effects due to movement slowness from the effects due to muscle fatigue on APA characteristics. Moreover, our analyses do not allow ruling out whether the fatigue level was in part dynamically integrated prior to the first trial post-fatigue and whether internal models were updated during the fatiguing procedure despite the achievement of isometric contractions. Nevertheless, although context-suited APAs require previous knowledge of the upcoming perturbation (Bouisset and Zattara 1981), the results of this study demonstrate that a particular movement practised without fatigue can be predictively controlled as efficiently while fatigued despite no previous actual experience of the muscular fatigue state during dynamic movements. Indeed, despite alterations in the relation between focal motor command magnitude and arm movement kinematics, predictive processes supported by the central integration of the group III and IV afferents enabled subjects to immediately scale APA magnitude and timing to the new mechanical characteristics of the focal movement.

Acknowledgments The authors wish to thank Dr. Grant Handrigan for his help in ironing out last-minute English language problems and Dr. Romain Terrier for his comments and advice. We also thank all the subjects for their cooperation.

Conflict of interest The authors declare that they have no conflict of interest.

References

Ahmed AA, Wolpert DM (2009) Transfer of dynamic learning across postures. J Neurophysiol 102:2816–2824. doi:10.1152/jn.00532.2009

Allen DG, Lamb GD, Westerblad H (2008) Skeletal muscle fatigue: cellular mechanisms. Physiol Rev 88:287–332. doi:10.1152/physrev.00015.2007

Amann M (2011) Central and peripheral fatigue: interaction during cycling exercise in humans. Med Sci Sports Exerc 43:2039–2045. doi:10.1249/MSS.0b013e31821f59ab

Amann M, Proctor LT, Sebranek JJ, Pegelow DF, Dempsey JA (2009) Opioid-mediated muscle afferents inhibit central motor drive and limit peripheral muscle fatigue development in humans. J Physiol 587:271–283. doi:10.1113/jphysiol.2008.163303

Aruin AS (2006) The effect of asymmetry of posture on anticipatory postural adjustments. Neurosci Lett 401:150–153. doi:10.1016/j.neulet.2006.03.007

Aruin AS, Latash ML (1995a) Directional specificity of postural muscles in feed-forward postural reactions during fast voluntary arm movements. Exp Brain Res 103:323–332

Aruin AS, Latash ML (1995b) The role of motor action in anticipatory postural adjustments studied with self-induced and externally triggered perturbations. Exp Brain Res 106:291–300

Benvenuti F, Stanhope SJ, Thomas SL, Panzer VP, Hallett M (1997) Flexibility of anticipatory postural adjustments revealed by self-paced and reaction-time arm movements. Brain Res 761:59–70

Bigland-Ritchie BR, Dawson NJ, Johansson RS, Lippold OCJ (1986) Reflex origin for the slowing of motoneurone firing rates in fatigue of human voluntary contractions. J Physiol 379:451–459

Bonnetblanc F, Martin O, Teasdale N (2004) Pointing to a target from an upright standing position: anticipatory postural adjustments are modulated by the size of the target in humans. Neurosci Lett 358:181–184. doi:10.1016/j.neulet.2004.01.020

Bouisset S, Do MC (2008) Posture, dynamic stability, and voluntary movement. Neurophysiol Clin 38:345–362. doi:10.1016/j.neucli.2008.10.001

Bouisset S, Zattara M (1981) A sequence of postural movements precedes voluntary movement. Neurosci Lett 22:263–270

Bouisset S, Richardson J, Zattara M (2000) Are amplitude and duration of anticipatory postural adjustments identically scaled to focal movement parameters in humans? Neurosci Lett 278:153–156

Carson RG, Riek S, Shahbazpour N (2002) Central and peripheral mediation of human force sensation following eccentric or concentric contractions. J Physiol 539:913–925. doi:10.1013/jphysiol.2001.013385

Chabeauti PY, Assaiante C, Vaugoyeau M (2012) Extreme short-term environmental constraints do not update internal models of action as assessed from motor imagery in adults. Neuroscience 222:69–74. doi:10.1016/j.neuroscience.2012.07.002

Christensen MS, Lundbye-Jensen J, Geertsen SS, Petersen TH, Paulson OB, Nielsen JB (2007) Premotor cortex modulates somatosensory cortex during voluntary movements without proprioceptive feedback. Nat Neurosci 10:417–419. doi:10.1038/nn1873

Cohn JV, DiZio P, Lackner JR (2000) Reaching during virtual rotation: context specific compensations for expected coriolis forces. J Neurophysiol 83:3230–3240

Corcos DM, Jiang H, Wilding J, Gottlieb GL (2002) Fatigue induced changes in phasic muscle activation patterns for fast elbow flexion movements. Exp Brain Res 142:1–12. doi:10.1007/s00221-001-0904-9

Cordo PJ, Nashner LM (1982) Properties of postural adjustments associated with rapid arm movements. J Neurophysiol 47:287–302

Côté JN, Mathieu PA, Levin MF (2002) Movement reorganization to compensate for fatigue during sawing. Exp Brain Res 146:394–398. doi:10.1007/s00221-002-1186-6

de Morree HL, Klein C, Marcora SM (2012) Perception of effort reflects central motor command during movement execution. Psychophysiology 49:1242–1253

de Wolf S, Slijper H, Latash ML (1998) Anticipatory postural adjustments during self-paced and reaction-time movements. Exp Brain Res 121:7–19

Desmurget M, Grafton S (2000) Forward modeling allows feedback control for fast reaching movements. Trends Cogn Sci 4:423–431

Enoka RM, Stuart DG (1992) Neurobiology of muscle fatigue. J Appl Physiol 72:1631–1648

Forestier N, Nougier V (1998) The effects of muscular fatigue on the coordination of a multijoint movement in human. Neurosci Lett 252:187–190

Fuglevand AJ, Zackowski KM, Huey KA, Enoka RM (1993) Impairment of neuromuscular propagation during human fatiguing contractions at submaximal forces. J Physiol 460:549–572

Gandevia SC (2001) Spinal and supraspinal factors in human muscle fatigue. Physiol Rev 81:1725–1789

Gandevia SC, Allen GM, Butler JE, Taylor JL (1996) Supraspinal factors in human muscle fatigue: evidence for suboptimal output from the motor cortex. J Physiol 490:529–536

Gandevia SC, Smith JL, Crawford M, Proske U, Taylor JL (2006) Motor commands contribute to human position sense. J Physiol 571:703–710. doi:10.1113/jphysiol.2005.103093

Garland SJ (1991) Role of small diameter afferents in reflex inhibition during human muscle fatigue. J Physiol 435:547–558

Hermens HJ, Freriks B, Merletti R, Stegeman D, Blok J, Rau G et al (1999) European recommendations for surface electromyography. Roessingh Research and Development

Horak FB, Nashner LM (1986) Central programming of postural movements: adaptation to altered support-surface configurations. J Neurophysiol 55:1369–1381

Huffenus AF, Amarantini D, Forestier N (2006) Effects of distal and proximal arm muscles fatigue on multi-joint movement organization. Exp Brain Res 170:438–447. doi:10.1007/s00221-005-0227-3

Ingram JN, Howard IS, Flanagan JR, Wolpert DM (2010) Multiple grasp-specific representations of tool dynamics mediate skillful manipulation. Curr Biol 20:618–623. doi:10.1016/j.cub.2010.01.054

Jaric S, Radovanovic S, Milanovic S, Ljubisavljevic M, Anastasijevic R (1997) A comparison of the effects of agonist and antagonist muscle fatigue on performance of rapid movements. Eur J Appl Physiol 76:41–47

Kanekar N, Santos MJ, Aruin AS (2008) Anticipatory postural control following fatigue of postural and focal muscles. Clin Neurophysiol 119:2304–2313. doi:10.1016/j.clinph.2008.06.015

Kawato M (1999) Internal models for motor control and trajectory planning. Curr Opin Neurobiol 9:718–727

Kennedy A, Guevel A, Sveistrup H (2012) Impact of ankle muscle fatigue and recovery on the anticipatory postural adjustments to externally initiated perturbations in dynamic postural control. Exp Brain Res 223:553–562. doi:10.1007/s00221-012-3282-6

Kurtzer I, Herter TM, Scott HS (2005) Random change in cortical load representation suggests distinct control of posture and movement. Nat Neurosci 8:498–504. doi:10.1038/nn1420

Lackner JR, Dizio P (1994) Rapid adaptation to coriolis force perturbations of arm trajectory. J Neurophysiol 72:299–313

Lafargue G, Paillard J, Lamarre Y, Sirigu A (2003) Production and perception of grip force without proprioception: is there a sense of effort in deafferented subjects. Eur J Neurosci 17:2741–2749. doi:10.1046/j.1460-9568.2003.02700.x

Lee WA, Buchanan TS, Rogers MW (1987) Effects of arm acceleration and behavioral conditions on the organization of postural adjustments during arm flexion. Exp Brain Res 66:257–270

Li X, Aruin AS (2007) The effect of short-term changes in the body mass on anticipatory postural adjustments. Exp Brain Res 181:333–346. doi:10.1007/s00221-007-0931-2

Liu JZ, Shan ZY, Zhang LD, Sahgal V, Brown RW, Yue GH (2003) Human brain activation during sustained and intermittent submaximal fatigue muscle contractions: an FMRI study. J Neurophysiol 90:300–312. doi:10.1152/jn.00821.2002

Massion J (1992) Movement, posture and equilibrium: interaction and coordination. Prog Neurobiol 38:35–56

Massion J, Alexandrov A, Frolov A (2004) Why and how are posture and movement coordinated? In: Mori S, Stuart DG (eds.) Progress in brain research: brain mechanisms for the integration of posture and movement, Elsevier, Amsterdam, pp 13–27. doi:10.1016/s0079-6123(03)43002-1

McCloskey DI, Ebeling P, Goodwin GM (1974) Estimation of weights and tensions and apparent involvement of a "sense of effort". Exp Neurol 42:220–232

Miall RC, Wolpert DM (1996) Forward models for physiological motor control. Neural Networks 9:1265–1279

Missenard O, Mottet D, Perrey S (2009) Adaptation of motor behavior to preserve task success in the presence of muscle fatigue. Neuroscience 161:773–786. doi:10.1016/j.neuroscience.2009.03.062

Mochizuki G, Ivanova TD, Garland SJ (2004) Postural muscle activity during bilateral and unilateral arm movements at different speeds. Exp Brain Res 155:352–361. doi:10.1007/s00221-003-1732-x

Morris SL, Allison GT (2006) Effects of abdominal muscle fatigue on anticipatory postural adjustments associated with arm raising. Gait Posture 24:342–348. doi:10.1016/j.gaitpost.2005.10.011

Papaxanthis C, Pozzo T, McIntyre J (2005) Kinematic and dynamic processes for the control of pointing movements in humans revealed by short-term exposure to microgravity. Neuroscience 135:371–383. doi:10.1016/j.neuroscience.2005.06.063

Saradjian AH, Tremblay L, Perrier J, Blouin J, Mouchnino L (2013) Cortical facilitation of proprioceptive inputs related to gravitational balance constraints during step preparation. J Neurophysiol 110:397–407. doi:10.1152/jn.00905.2012

Schmid M, Schieppati M, Pozzo T (2006) Effect of fatigue on the precision of a whole-body pointing task. Neuroscience 139:909–920. doi:10.1016/j.neuroscience.2005.12.060

Shadmehr R, Mussa-Ivaldi FA (1994) Adaptive representation of dynamics during learning of a motor task. J Neurosci 14:3208–3224

Sinoway LI, Hill JM, Pickar JG, Kaufman MP (1993) Effects of contraction and lactic acid on the discharge of group III muscle afferents in cats. J Neurophysiol 69:1053–1059

Slijper H, Latash ML, Mordkoff JT (2002) Anticipatory postural adjustments under simple and choice reaction time conditions. Brain Res 924:184–197

Strang AJ, Berg WP (2007) Fatigue-induced adaptive changes of anticipatory postural adjustments. Exp Brain Res 178:49–61. doi:10.1007/s00221-006-0710-5

Strang AJ, Berg WP, Hieronymus M (2009) Fatigue-induced early onset of anticipatory postural adjustments in non-fatigued muscles: support for a centrally mediated adaptation. Exp Brain Res 197:245–254. doi:10.1007/s00221-009-1908-0

Takahashi CD, Nemet D, Rose-Gottron CM, Larson JK, Cooper DM, Reinkensmeyer DJ (2006) Effect of muscle fatigue on internal model formation and retention during reaching with the arm. J Appl Physiol 100:695–706. doi:10.1152/japplphysiol.00140.2005

Taylor JL, Gandevia SC (2008) A comparison of central aspects of fatigue in submaximal and maximal voluntary contractions. J Appl Physiol 104:542–550. doi:10.1152/japplphysiol.01053.2007

Taylor JL, Butler JE, Allen GM, Gandevia SC (1996) Changes in motor cortical excitability during human muscle fatigue. J Physiol 490:519–528

Taylor JL, Petersen N, Butler JE, Gandevia SC (2000) Ischaemia after exercise does not reduce responses of human motoneurones to cortical or corticospinal tract stimulation. J Physiol 525:793–801

Vuillerme N, Nougier V, Teasdale N (2002) Effects of lower limbs muscular fatigue on anticipatory postural adjustments during arm motions in humans. J Sport Med Phys Fit 42:289–294

Walsh LD, Gandevia SC, Taylor JL (2010) Illusory movements of a phantom hand grade with the duration and magnitude of motor commands. J Physiol 588:1269–1280. doi:10.1113/jphysiol.2009.183038

Wolpert DM, Ghahramani Z (2000) Computational principles of movement neuroscience. Nat Neurosci 3:1212–1217

Wolpert DM, Kawato M (1998) Multiple paired forward and inverse models for motor control. Neural Netw 11:1317–1329

Wolpert DM, Ghahramani Z, Jordan MI (1995) An internal model for sensorimotor integration. Science 269:1880–1882