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Rapid muscle activation and force capacity in conditions of chronic musculoskeletal pain

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Abstract

Background. The association between musculoskeletal pain and decreased maximal muscle strength capacity has been extensively studied, but knowledge about functional rapid force capacity in conditions of chronic musculoskeletal pain is lacking. The objective of this study is to investigate rapid muscle activation and force capacity of chronically painful muscles.

Methods. Cross-sectional study with 42 women with chronic trapezius myalgia, and 20 healthy matched controls. Maximal capacity was determined as peak torque and peak EMG amplitude of the painful trapezius and painfree deltoid muscles during the stable high-force phase of maximal voluntary shoulder abduction, whereas rapid capacity was determined as the steepest slope of the torque–time and EMG–time curves, defined as rate of torque development and rate of EMG rise. Intensity of pain was registered prior to the test on a visual-analogue-scale.

Findings. Peak torque was 18% lower at 115° shoulder joint angle in women with myalgia compared with healthy controls ($P < 0.001$), with a corresponding 29% lower level of peak EMG specifically of the painful trapezius muscle ($P < 0.001$). Rate of torque development was 33–54% lower ($P < 0.001$), with a corresponding 21–35% lower level of rate of EMG rise of both the painful trapezius and painfree deltoid ($P < 0.0001$). Intensity of pain showed higher association with parameters of rapid capacity ($R = -0.33$ to -0.53, $P < 0.001$–0.05) than with maximal capacity ($R = -0.15$ to -0.41, $P < 0.01$-ns).

Interpretation. In conditions of chronic musculoskeletal pain, the ability to rapidly activate painful and painfree synergistic muscles is more severely impaired than maximal muscle activation. These findings have clinical relevance for rehabilitation of chronically painful muscles.

Keywords: Rate of force development; Chronic neck pain; Chronic muscle pain; Myalgia; Musculoskeletal disorder; MVC; Pain; Force; Disability

1. Introduction

Painful conditions of the musculoskeletal system are frequent in the general population (Ferrari and Russell, 2003; Ihlebaek et al., 2007). Musculoskeletal chronic pain is typically negatively associated with physical capacity in terms of maximal muscle strength (Brox et al., 1997; Itoi et al., 1997; Sjogaard et al., 2006), likely due to pain inhibition of motor outflow (Andersen et al., 2008b; Lund et al., 1991; Steingrímsdóttir et al., 2004). Whereas previous studies determined muscle activation and strength capacity as peak values during the stable high-force phase of a maximal voluntary contraction (MVC) (Brox et al., 1997; Itoi et al., 1997; e.g. Sjogaard et al., 2006), many daily tasks involve rapid modulation of force, e.g. during unexpected postural perturbations (Stokes et al., 2006). However, rapid muscle activation and force capacity in conditions of chronic musculoskeletal pain has not previously been investigated.

In healthy individuals, rapid muscle force capacity is influenced by a multitude of physiological factors, e.g. neural drive to the muscle fibers (Aagaard et al., 2002; Grimby

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et al., 1981; Holtermann et al., 2007a; Van Cutsem et al., 1998) and maximal muscle strength (Andersen and Aagaard, 2006). However, in patients with chronic musculoskeletal pain, rapid movements particularly exacerbates fear of pain (Al-Obaidi et al., 2003; Reneman et al., 2007). Therefore, chronic pain is likely to more severely impair rapid force capacity than maximal strength capacity. Furthermore, the fear of increased pain of rapid movements may not only impair rapid force capacity of chronically painful muscle *per se*, but also of synergistic painfree muscles.

In this regard, the shoulder complex provides an ideal model to study effects of chronic musculoskeletal pain on a variety of physiological and biomechanical parameters. The intricate nature of this joint implies synergistic activation of several muscles to provide a functional combination of stability and movement (Veeger and van der Helm, 2007), e.g. between the trapezius and deltoid muscles during shoulder abduction (Inman et al., 1944). Previously, we observed specific inhibition of the chronically painful trapezius muscle during the stable high-force phase of MVC in women with myalgia, whereas synergistic activity of the adjacent painfree deltoid remained unaffected (Andersen et al., 2008b). However, the interplay between synergistic painful and painfree muscles during rapid muscle contraction remains unknown. This interaction is interesting though, not only from a basic physiological point of view, but also for clinicians designing targeted rehabilitation regimes based on different types of training, e.g. powerful versus slowly controlled strengthening exercises.

The objective of this study is to investigate rapid muscle activation and force capacity in conditions of chronic musculoskeletal pain. As a model to investigate this, MVCs of shoulder abduction were performed in subjects with and without trapezius myalgia, while at the same time recording shoulder joint torque and activity of the trapezius and deltoid muscles.

2. Methods

2.1. Study design

The procedure of subject recruitment has been described in detail recently (Andersen et al., in press). Briefly, 42 female office workers with chronic neck muscle pain (MYA; 44 ± 8 yrs, 165 ± 6 cm, 72 ± 15 kg, mean $\pm$ SD) and 20 females comparable with regard to job-type, age, weight and height but without neck muscle complaints (CON; 45 ± 9 yrs, 167 ± 6 cm, 70 ± 11 kg, mean $\pm$ SD) participated. The subjects in each group had to meet a number of self-reported and clinically diagnosed criteria, which have been described in detail recently (Andersen et al., in press). Subjects with serious clinical conditions such as previous trauma, life threatening diseases, whiplash injury, cardiovascular diseases or arthritis in the neck and shoulder were excluded from both groups. All females in MYA were clinically diagnosed with trapezius myalgia, where the main criteria for a positive diagnosis were (1) chronic or frequent pain in the neck area, (2) tightness of the upper trapezius muscle, and (3) palpable tenderness of the upper trapezius muscle (Andersen et al., 2008a; Juul-Kristensen et al., 2006). All subjects were informed about the purpose and content of the project and gave written informed consent to participate in the study which conformed to the Declaration of Helsinki, and was approved by the Local Ethical Committee (KF 01-138/04).

2.2. Pain intensity

Intensity of pain in the trapezius muscle was rated by each subject on a 100 mm visual-analogue-scale (VAS), where 0 mm is "no pain" and 100 mm is "worst imaginable pain" (Huskisson, 1974). Pain at present was registered at rest prior to the dynamometer test.

2.3. Dynamometry and electromyography

A Biodex Medical isokinetic dynamometer (System 3 Pro, Brookhaven R&D Plaza, New York, USA) was used for testing of shoulder abduction. Prior to the test, EMG electrodes were positioned at the upper trapezius muscle and the mid part of the deltoideus muscle with a bipolar surface EMG configuration (Neuroline 720 01-K, Medicotest A/S, Ølstykke, Denmark) according to standardized procedures (Hermens and Freriks, 1997). Shoulder abductions – in a plane 15° from the frontal plane – were performed at two separate static shoulder joint angles (35° and 115°). After careful warm-up and preconditioning, consisting of slow and fast submaximal and near maximal contractions, three MVC's were performed at each joint position. Subjects were instructed to contract the muscles as fast and hard as possible (Bemben et al., 1990). All torque and EMG signals were sampled synchronously at 1000 Hz using a 16-bit A/D-converter (DAQ Card-AI-16XE-50, National Instruments, USA) and stored on a laptop for further analysis.

During subsequent off-line analyses, the torque signal was low-pass filtered at 10 Hz and subsequently corrected for the effect of gravity on the subjects arm by adding the passive torque of the arm to the sampled torque signal. The passive torque of the arm for the two joint positions was calculated as the passive torque measured at 90° – i.e. when the subject held on to the handlebar and relaxed the shoulder – multiplied by sine to the respective shoulder joint angles. All raw EMG signals were filtered using linear EMG envelopes, which consisted of: (1) high-pass filtering at 10 Hz, (2) full-wave rectification, and (3) low-pass filtering at 10 Hz. The filtering algorithms was based on a fourth-order zero phase lag Butterworth filter (Winter, 1990). From the filtered signal the following parameters were extracted:

Maximal capacity of muscle activation and torque: For each trial peak torque (PT; unit Nm) and peak EMG

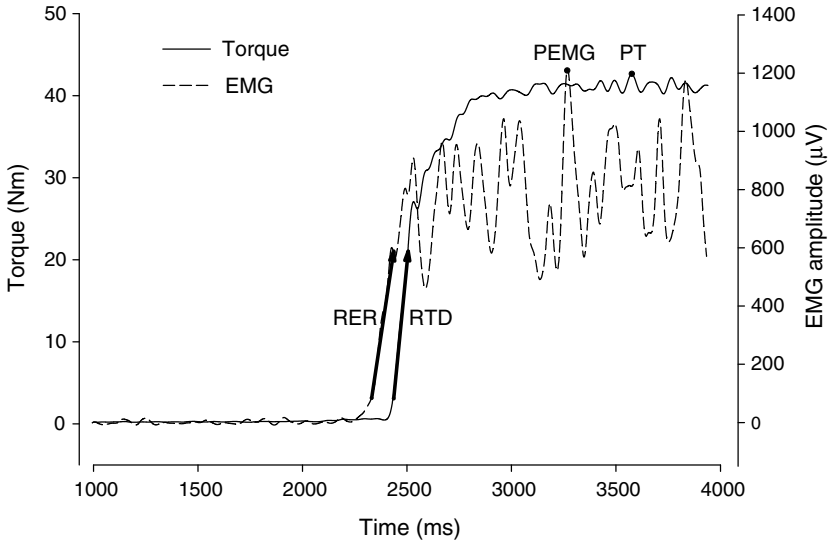

Fig. 1. Representative recording of torque and trapezius EMG amplitude during a maximal voluntary static contraction. Maximal capacity was defined as peak torque (PT) and peak EMG amplitude (PEMG), and rapid capacity was defined as rate of torque development (RTD) and rate of EMG rise (RER).

amplitude (PEMG; unit μV) were determined as the maximal value of the filtered torque–time and EMG–time curve, respectively (Fig. 1). For the statistical analysis the trial with highest PT was used.

Rapid capacity of muscle activation and torque: For each trial the rate of torque development (RTD; unit Nm s^{-1}) was determined as the steepest slope over 100 ms of the rising part of the filtered torque–time curve (Fig. 1). In the same trials the rate of EMG rise was determined as the steepest slope over 100 ms of the rising part of the filtered EMG–time curve normalized to the individual PEMG (RER; unit % PEMG s^{-1}). For the statistical analysis, the trial with highest RTD was used.

2.4. Statistics

Analysis of variance was performed in SAS version 9 using the MIXED procedure. Factors included in the model for PT and RTD were *group* (MYA and CON) and *angle* (35° and 115°). For PEMG and RER the factor *muscle* (trapezius and deltoideus) was added to this model. When a significant main effect was found, Bonferroni corrected post-hoc tests were performed to locate differences. Spearman's correlation coefficient was calculated to determine the association between the main parameters. An alpha level of 5% was considered statistically significant, and results are reported as group mean ± SD unless otherwise stated.

3. Results

3.1. Pain intensity

Pain at rest prior to the dynamometer test was 19 ± 17 and 0.4 ± 0.7 mm on the VAS scale in MYA and CON, respectively ($P < 0.0001$).

3.2. Dynamometry and EMG

There was a significant *group by angle* effect for PT ($P < 0.05$) and RTD ($P < 0.05$). Post-hoc tests showed that PT was 18% lower at 115° shoulder abduction angle in MYA compared with CON ($P < 0.001$), whereas no significant difference was seen at 35° (Fig. 2a). RTD was 33% and 54% lower at 35° ($P < 0.001$) and 115° ($P < 0.0001$) (Fig. 2b).

There was a significant *group by muscle* effect for PEMG ($P < 0.05$). Post-hoc tests showed that PEMG was 29–38% lower in MYA compared with CON in the trapezius muscle ($P < 0.001$), but not significantly different in the deltoideus muscle (Table 1).

There was a significant *group* effect for RER ($P < 0.0001$). Post-hoc tests showed that RER was 21–35% lower in MYA compared to CON for the trapezius and deltoideus muscles ($P < 0.0001$) (Table 1).

3.3. Correlation analyses

Intensity of pain was negatively related to several of the main parameters. The highest degree of association was found between pain and RTD ($R = -0.53$, $P < 0.0001$). In general, pain was more closely related to RTD and RER, than pain was to PT and PEMG (Table 2).

Correlations of EMG activity between the two muscles showed that RER in the deltoid and trapezius were closely related to each other (at 35°: $R = 0.70$, $P < 0.0001$, at 115°: $R = 0.67$, $P < 0.0001$), whereas the relation between PEMG of these two muscles was less pronounced (at 35°: $R = 0.50$, $P < 0.0001$, at 115°: $R = 0.29$, $P < 0.05$).

4. Discussion

The major finding of the present study is the severely impaired ability to rapidly activate painful and synergistic

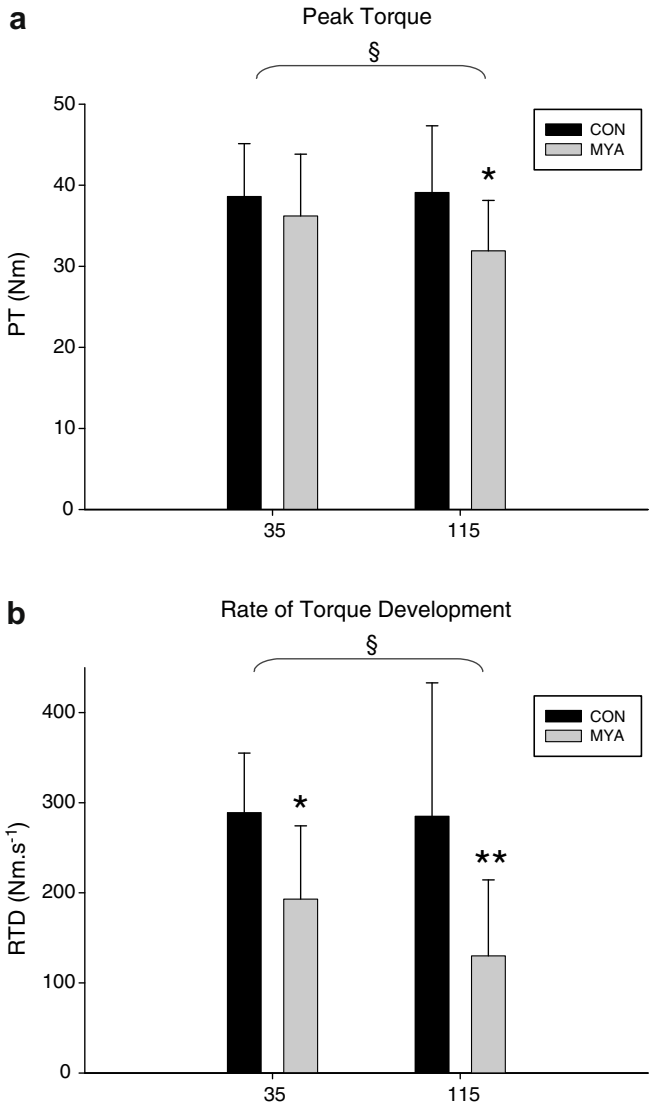

Fig. 2. (a) Peak torque (PT) and (b) Rate of torque development (RTD) at 35° and 115° shoulder abduction in the control group (CON) and the group with myalgia (MYA). Mean (SD). A priori hypothesis testing of main effects: (§) *group by angle* ($P < 0.05$). Post-hoc test: (∗) and (∗∗) MYA < CON ($P < 0.001$ and $P < 0.0001$, respectively).

Table 1
Peak EMG (PEMG) and rate of EMG rise (RER) in the trapezius and deltoideus muscles

		PEMG trap (μV)	PEMG delt (μV)	RER trap (% PEMG s^{-1})	RER delt (% PEMG s^{-1})
35°	CON	1058 (379)	457 (150)	689 (192)	735 (176)
	MYA	660 (382)[a]	394 (234)	544 (241)[a]	477 (192)[a]
115°	CON	1152 (472)	578 (225)	541 (204)	588 (160)
	MYA	822 (407)[a]	498 (259)	374 (187)[a]	384 (193)[a]

Mean (SD).
[a] MYA < CON ($P < 0.001$).

painfree muscles and thereby rapidly generate force in conditions of chronic musculoskeletal pain. In comparison, maximal muscle strength capacity was much less affected.

Table 2
Spearman's correlation coefficient between pain and the other main parameters, separately at 35° and 115° shoulder joint angle

	Pain	
	35°	115°
RTD	−0.52***	−0.53***
RER trap	−0.48***	−0.36**
RER delt	−0.42***	−0.33*
PT	−0.15 ns	−0.41**
PEMG trap	−0.34**	−0.20 ns
PEMG delt	−0.16 ns	–

RTD: rate of torque development; RER: rate of EMG rise; PT: peak torque; PEMG: peak EMG amplitude.
* $P<0.05$.
** $P<0.01$.
*** $P<0.001$.

The present results confirm that activation of painful muscles is specifically inhibited during the stable high-force phase of MVC (Andersen et al., 2008b), i.e. PEMG was significantly lowered in the painful trapezius, but not in the painfree deltoid muscle. As a novel finding, muscle activity during the rising phase of muscle force – i.e. RER – was lowered in both muscles, resulting in markedly lowered RTD. Although maximal muscle strength and RTD are closely correlated (Andersen and Aagaard, 2006), the relation is not necessarily causal (Holtermann et al., 2007b). Therefore, it is of clinical interest to investigate the mechanisms underlying impaired maximal muscle strength and RTD in conditions of chronic musculoskeletal pain. We have recently demonstrated that average trapezius muscle fiber size (Andersen et al., in press) as well as gross muscle thickness (Andersen et al., 2008b) is not significantly different between women with and without chronic neck muscle pain. This suggests that muscle wasting cannot explain impaired maximal muscle strength and RTD, but rather that a neural limitation mechanism exists.

RTD and RER were measured during the very initial phase of MVC – i.e. at the steepest slope of rising muscle force (see Fig. 1). The very short time period from contraction initialisation to the steepest part of the torque–time curve limits the potential influence of pain-related inhibitory feedback due to high-force levels on RER. Thus, neural inhibitory feedback could only have a minor impact on muscle activation during this very initial phase of contraction. Instead, it has been suggested that pain-related beliefs, such as self-efficacy and fear avoidance, are more important determinants of disability in patients with musculoskeletal disorders than intensity of pain *per se* (Denison et al., 2004). Thus, the belief that rapid movement exacerbates pain (Al-Obaidi et al., 2003; Reneman et al., 2007) could potentially limit motor outflow to agonist muscles. RER of the trapezius and deltoid were closely related to each other and lowered to a similar extent, supporting the notion of a generally reduced descending drive to these synergistic muscles during MVC. In comparison, the less pronounced relation between PEMG of these two muscles, and lowered PEMG in trapezius only, indicate

that inhibitory feedback is restricted to chronically painful muscles during the stable high-force phase of MVC. Altogether, these findings indicate that feedforward mechanisms – e.g. fear of increased pain – generally reduces rate of descending drive of both painful and painfree synergistic muscles during the phase of rapid rise in muscle force, whereas neural inhibitory feedback mechanisms limits maximal activation of painful muscles during the stable high-force phase of MVC.

An alternative explanation for the present findings is that people with innate low rapid force capacity are more prone to developing musculoskeletal pain due to a lack of skill to rapidly stabilize the joints, e.g. during postural perturbations. Future prospective studies should examine whether healthy people with a low inherent rapid force capacity are more prone to development of musculoskeletal chronic pain.

A secondary observation was that differences in maximal muscle strength and RTD between women with and without chronic neck muscle pain were larger at the more abducted shoulder joint position, i.e. at 115°. During this position the trapezius muscle contracts in a shortened position, which can be speculated to influence inhibitory feedback. However, this was not the case, since trapezius activity was lowered to a similar extent in MYA compared with CON at both joint positions. Whereas decreased trapezius PEMG at 35° did not results in significantly lower torque, a corresponding decrease of PEMG at 115° did. This indicates that the relative contribution of the trapezius to net shoulder joint torque becomes increasingly important in more abducted positions, likely due to increased need for scapular stabilization and upward rotation (Veeger and van der Helm, 2007).

The findings of this study are of clinical relevance. The severely lowered rapid force capacity, likely due to fear of performing rapid movements in conditions of chronic pain, designates that during the initial phase of rehabilitation, exercises should be performed in a controlled manner to ensure a high level of muscle activation in both painful and painfree muscles. During later phases of rehabilitation, increasingly more powerful execution of exercises may be employed. Further, since higher torque levels are found during less abducted shoulder joint positions, range of shoulder joint motion should be progressively increased as pain decreases. We encourage future studies to evaluate the optimal progression of these parameters in rehabilitation settings.

A limitation of this study is that fear avoidance was not measured. Future studies should measure fear avoidance prior to testing of rapid capacity, e.g. by the Tampa Scale of Kinesiophobia (Feleus et al., 2007). Also, future studies should evaluate pain during, and not only before, maximal muscle exertions. Further, the present MVC's were performed during standardized laboratory conditions. Future studies should investigate whether rapid muscle activation is lowered during real-life unexpected postural perturbations.

5. Conclusions

The ability to rapidly activate painful and painfree synergistic muscles, and thereby rapidly generate force, is more severely impaired than maximal muscle activation and strength capacity in conditions of chronic musculoskeletal pain. These findings elaborate upon the present knowledge about functional biomechanical consequences, physiological mechanisms and clinical rehabilitation of conditions of chronic musculoskeletal pain.

6. Conflicts of interest statement

There are no conflicts of interest.

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