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Muscular timing and inter-muscular coordination in healthy females while walking

Cora Huber[a,*], Corina Nüesch[b], Beat Göpfert[a], Philippe C. Cattin[c], Vinzenz von Tscharner[d]

[a] Laboratory of Biomechanics & Biocalorimetry, University of Basel, c/o Biozentrum/Pharmazentrum, Klingelbergstrasse 50-70, CH-4056 Basel, Switzerland
[b] Orthopaedic Department, University Hospital of Basel, Spitalstrasse 21, CH-4031 Basel, Switzerland
[c] Medical Image Analysis Center, University of Basel, c/o University Hospital Basel, Spitalstrasse 21, CH-4031 Basel, Switzerland
[d] Human Performance Laboratory, The University of Calgary, 2500 University Drive NW, Calgary, Alberta, Canada T2N 1N4

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ABSTRACT

The dynamic interplay between muscles surrounding the knee joint, the central nervous system and external factors require a control strategy to generate and stabilise the preferred gait pattern. The electromyographic (EMG) signal is a common measure reflecting the neuromuscular control strategies during dynamic tasks. Neuromuscular control mechanisms, found in processed EMG signals, showed a precise pacing with a pacing rhythm and a tight control of muscle activity in running and maximally contracted muscles. The purpose of this study was to provide an insight how muscles get activated during walking. The EMG power, extracted by the wavelet transform (92–395 Hz), over a time period encompassing 250 ms before and 250 ms after heel strike was analysed. The study showed that the wavelet-based analysis of EMG signals was sufficiently sensitive to detect a synchronisation of the activation of thigh muscles while walking. The results within each single subject and within the group consisting of 10 healthy females showed that, although there was a lot of jitter in the locations of the intensity peaks, the muscle activation is controlled, on average, by a neuromuscular activity paced at about 40 ms, however with variable amplitudes. Albeit the jitter of the signal, the results resolved the temporal dependency of intensity peaks within muscles surrounding the knee and provided an insight into neural control of locomotion. The methodology to assess the stabilising muscle activation pattern may provide a way to discriminate subjects with normal gait pattern form those with a deteriorated neuromuscular control strategy.

1. Introduction

The knee is a complex joint with many muscles which have to be controlled by the neuromuscular control mechanism. The dynamic interplay between these muscles, the central nervous system (CNS), and external factors do require a control strategy to generate and stabilise the preferred gait pattern. Many studies have shown that small changes in external factors, for instance different shoes, do not change the preferred movement (Nigg, 2001). However, to stabilise a preferred movement the neuromuscular control system must be very flexible. The stabilising neuromuscular control strategy applied while walking can be observed by monitoring the muscle activity using electromyography (EMG) as an indicator.

The EMG signal is a common measure reflecting the neuromuscular control strategies during dynamic tasks (DeMont and Lephart, 2004; Farina et al., 2004; Guidetti et al., 1996; Rutherford et al., 2010; Wakeling, 2009; Wong, 2009; Zebis et al., 2008). Neuromuscular control mechanisms, found in processed EMG signals, showed a precise pacing with a pacing rhythm (Stirling et al., 2011; von Tscharner et al., 2011a, 2011b; Brown, 2000; Brown et al., 1998; Salenius et al., 1996, 1997; Vallbo and Wessberg, 1993). Works done in the late 1990s showed that these rhythms are correlated to the activity of the motor cortex and can therefore be considered as a result of the activity of the CNS (Brown et al., 1998). However, the studies revealing the corticomuscular interaction usually did not explicitly resolve the pacing in the EMG signal. In maximally activated muscles (von Tscharner et al., 2011a, 2011b) and muscle activities measured while running (Stirling et al., 2011) the rhythms were explicitly resolved and showed a tight control of muscle activity. Thus, the presence of rhythmicity within a longer

Abbreviations: BF, biceps femoris; EMG, electromyography; CNS, central nervous system; HAM, hamstring muscle group; QF, quadriceps femoris; RF, rectus femoris; ST, semitendinosus; VL, vastus lateralis; VM, vastus medialis.

* Corresponding author. Tel.: +41 0 61 265 94 64; fax: +41 0 61 265 95 74.
E-mail addresses: cora.huber@unibas.ch (C. Huber), cnueesch@uhbs.ch (C. Nüesch), beat.goepfert@unibas.ch (B. Göpfert), philippe.cattin@unibas.ch (Ph.C. Cattin), vincent@kin.ucalgary.ca (V. von Tscharner).

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lasting muscular event indicates that there is a kind of programmed function controlled by a neuromuscular activity (Arsenault et al., 1986).

The musculoskeletal mechanics of gait depends on multi- and biarticular muscles that are activated in a specific sequence. Especially at heel strike where the human locomotor system is affected by irregular impact forces, controlled muscle activation strategies are essential for counteracting the destabilising forces. A coordinated activity of Mm. quadriceps femoris (QF) is essential to maintain dynamic stability of the patellofemoral joint (Mellor and Hodges, 2005a, 2005b). Such an active control of the patella by precise coordination between the Mm. vastus medialis (VM) and vastus lateralis (VL) is important the more extended the knee joint is (Mellor and Hodges, 2006), as occurring at heel strike. Consequently, as the foot touches the ground, the muscles have to be prepared to absorb the impact shock by a muscle tuning (Nigg and Wakeling, 2001) to control the stabilisation of the knee joint dynamically (Mellor and Hodges, 2006). A balanced VM and VL activity is required to control the translation of the patella to prevent their maltracking (Cowan et al., 2001; Mellor and Hodges, 2005a; Pal et al., 2011). Already a time delay of 5 ms in the onset of the VM relative to VL alters the patellofemoral joint mechanism (Neptune et al., 2000).

Therefore, our hypotheses were: (a) that the EMG signal combined with the wavelet based analysis of this signal is sufficiently sensitive to detect a synchronisation of the activation of thigh muscles while walking and (b) that the neuromuscular control uses a similar timing raster like timing for the synchronisation of muscle activity during gait. The study was designed to investigate how the muscles are controlled by the neuromuscular system before and after heel strike while walking.

The study should provide insight into muscle activation during a movement that does not need absolutely tight control. In decerebrated cats, the gait pattern is still present but not stable (Grillner and Wallen, 1985; Grillner and Zangger, 1984). We therefore have to consider that human muscles have at least two major functions, one is to provide the energy to sustain the movement, and the other is to keep the movement in line by stabilising the joints. It was previously shown that EMG intensity pattern can discriminate between healthy subjects and subjects suffering from osteoarthritis (von Tscharner and Valderrabano, 2010). However, pattern classification did not reveal explicitly the changes in the neuromuscular control strategy. Therefore the important next step is to understand the timing strategy of the neuromuscular control. A methodology to assess the stabilising muscle activation pattern may provide a way to discriminate subjects with a normal gait pattern from those with a deteriorated neuromuscular control strategy.

2. Materials and methods

2.1. Subjects

Ten healthy female volunteers (age: 48 ± 7 years, body mass: 61.1 ± 4.9 kg, height: 1.64 ± 0.05 m) with no history of previous knee or lower extremity surgery, osteoarthritis, neurological, or musculoskeletal disorders participated in this study. They were informed of the experimental risks and signed an informed consent form approved by the local Ethics Committee.

2.2. Experimental design

An instrumented three dimensional gait analysis with synchronous measurement of the lower extremity muscle activity during normal gait was performed. The lower body kinematics were recorded at 240 Hz with a six-camera motion capture system (Vicon MX13+, Oxford, UK) using the Helen Hayes model (Kadaba et al., 1990). The subjects walked barefoot at a comfortable, self-selected walking speed along a 10 m walkway within the laboratory. A minimum of 12 valid trials was collected. The time of heel strike was determined from the position of the heel marker. Two steps per trial were extracted. Due to technical problems, mainly movement artefacts, less than 12 trials were available from seven subjects. Nine trials, thus 18 steps per subject, were used for the calculations. In total 180 steps were analysed.

2.3. Data recording

Surface EMG was recorded from the QF and hamstring (HAM) muscle groups for the right thigh. The muscles of the QF group were: Mm. rectus femoris (RF), VM, and VL, and of the HAM group: Mm. semitendinosus (ST) and biceps femoris (BF). Bipolar Ag/AgCl surface electrodes (diameter: 10 mm, inter-electrode distance: 22 mm, Noraxon U.S.A. Inc., Scottsdale, AZ, USA) were used. After shaving and cleaning the skin with alcohol according to SENIAM-recommendations (Hermens et al., 2000), three electrode pairs were placed side by side on each muscle, in the direction of the muscle fibres, to get additional spatial and temporal information. The first electrode pair, referred to as original, was positioned according to the SENIAM-recommendations whereas the lateral and medial electrode pairs were laterally displaced by 28 mm from the original electrode pair (Fig. 1). The ground electrode was positioned on the tibial tuberosity. The surface EMG was collected with single differential amplifiers (band path of 10–700 Hz, Biovision, Wehrheim, Germany) at a sampling frequency of 2400 Hz without further processing. Cables and electrodes were kept in place by elastic net bandage (Elastofix, Type B-25 m stretched, BSN medical GmbH & Co. KG, Hamburg, Germany) which was pulled over the thigh.

2.4. Data processing

2.4.1. Wavelet transform

The signal analysis was performed using a wavelet transform with 13 non-linearly scaled wavelets characterised by their centre frequency: 7, 19, 38, 62, 92, 128, 170, 218, 272, 331, 395, 457, and 542 Hz (von Tscharner, 2000). Centre frequencies lower than 92 Hz were not considered further because time resolution has to be short enough to detect the rhythm. The theoretical time resolution of the wavelets defined by von Tscharner (2000) represents the time difference of two events that occur at the same frequency and within the same recording that is required for the events to be discriminated. For wavelets with centre frequencies above 92 Hz time resolution is between 25 and 10 ms. In signals of mixed frequency and when comparing peaks recorded in different trials time resolution is much shorter and mainly limited by the sampling frequency. Centre frequencies higher than 395 Hz were omitted because of high frequency noise. EMG power at each time point was calculated by summing the power extracted by the wavelets with centre frequencies of 92–395 Hz. The EMG power over a time period encompassing 250 ms before and 250 ms after heel strike was used for the analysis. The average EMG power of the triplicate electrodes was called a waveform. Thus, heel strike was at time 0 within the waveform. Waveforms were normalized by dividing them by the sum across the waveform. The waveforms indicate the fine structure across time of the step-specific strategy of muscle activation. Each individual waveform represented the average waveform of all steps for a given subject. The group waveform was computed by averaging the individual waveforms.

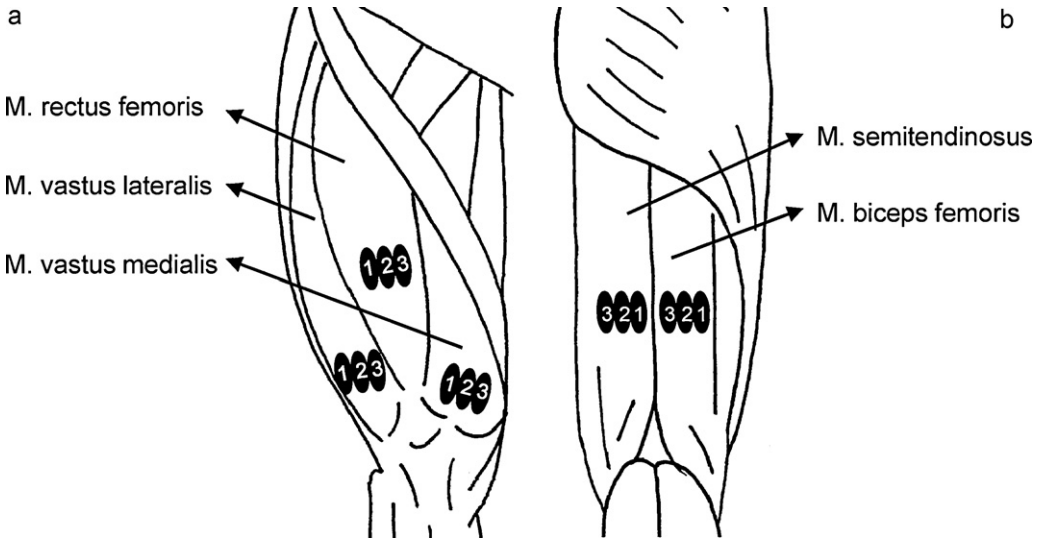

Fig. 1. Placement of the surface EMG electrode pairs for measuring the activity from (a) the quadriceps femoris muscle group: Mm. vastus lateralis, rectus femoris, and vastus lateralis and (b) from the hamstring muscle group: Mm. semitendinosus and biceps femoris is illustrated for the lateral (1), original (2), and medial (3) electrode pairs.

2.4.2. Timing analysis

The time occurrence of all peaks (peak locations) with an intensity level higher than 1% of the maximum peak value was extracted from all waveforms and from the group waveforms in order to avoid the detection of small oscillations around zero (Stirling et al., 2011). Raster plots of the peak locations were generated with a time resolution of 6 ms showing the peak locations (x-axis) for the different steps or muscles (y-axis). A colour mapping was separately applied on the dots in the multi-step raster plots in the pre and the post heel strike period. The order of the colours was equal for all raster plots. It started at heel strike with a backward and a forward mapping in the pre and in the post heel strike period, respectively. As an example, all first detected peaks after heel strike are mapped with the colour pink. The difference in time between two adjacent intensity peaks was calculated for all waveforms. A histogram of these inter-pulse distances was generated representing their probability distribution. A Lilliefors test was used to asses whether the inter-pulse distances were normally distributed. Significance level was set at $p < 0.05$.

All computations were made in custom software implemented in Matlab 7.10.0 (MathWorks, MA, USA).

3. Results

The average walking speed of 10 healthy females was 1.22 ± 0.06 m/s (range 1.14–1.29 m/s). In general, the waveforms indicate the fine structure across time and reveal a step-specific strategy of muscle activation whereas the individual waveforms describe the subject-specific muscle activation strategy while walking.

3.1. Group analysis

The comparison of the group waveforms, the averages of the individual waveforms, is shown in Fig. 2 and illustrates the basic activation patterns at the slightly different positions on the muscle recorded by the triplicate electrode pairs. The similarity between the waveforms of the triplicate electrode pairs was visible checked for each step and subject. This visual inspection showed that only the waveforms of the three BF electrodes had a large variation in shape with the position of the electrodes. This indicated that the EMG signal yielded a non-reliable activation pattern for the BF. Either the patterns of different muscle compartments or of multiple muscles were reflected. The other muscles showed a consistent activation pattern with only slight but detectable spatial differences. Therefore, only the activation pattern of the QF muscles and the ST were used and commented in the detailed analysis. However, the locations of the peaks were not shifted between recordings from the medial or lateral side, as shown in the raster plot of Fig. 3. Thus, a muscle was activated at the same time across the medial–lateral region. The detailed analysis will therefore be limited to the data recorded from the original electrode position.

The group waveforms of the QF muscles revealed three ranges containing distinct peaks or features. The pre heel strike range lasted from −90 ms to heel strike and contained a distinct peak at −50 ms for the RF. For the QF, the first post heel strike range was characterised by a peak at +35 ms and was followed by a second post heel strike range characterised by a peak at +75 ms which rapidly decayed and ended +150 ms after heel strike (Fig. 2a, c and e).

The group waveforms of the ST (BF could not be included) showed its main activation in a peak occurring 125 ms before heel strike (Fig. 2b). The next peak occurred in the pre heel strike range of the QF muscles, thus reflecting a co-contraction.

The time between the peaks are shown in a raster plot (Fig. 3) which reflects the interplay of muscles based on the group waveforms and characterises the basic activation strategies of the group while walking. A time delay of 85 ms (2×42.5 ms) between the pre heel strike peak and the first post heel strike peak of the RF and of 40 ms between the first and second post heel strike peaks of the RF, VM, VL, and ST were found. On average, the peaks occurred in a raster of about 40 ms.

To obtain such distinct peaks as shown in Fig. 3 the individual waveforms have to be well aligned. The details of the alignment will be revealed by the between subject analysis.

3.2. Between subject analysis

The individual waveforms (the average of the waveforms) of the subjects which describe the subject-specific average muscle activation are shown for the QF muscles and the ST in Fig. 4. The individual waveforms were subject-specific; however, similarities between subjects were apparent in the peak locations and not in the shape of the waveforms.

The peaks of the QF muscles that occurred during the pre heel strike range (−90 ms to heel strike) varied in amplitude and

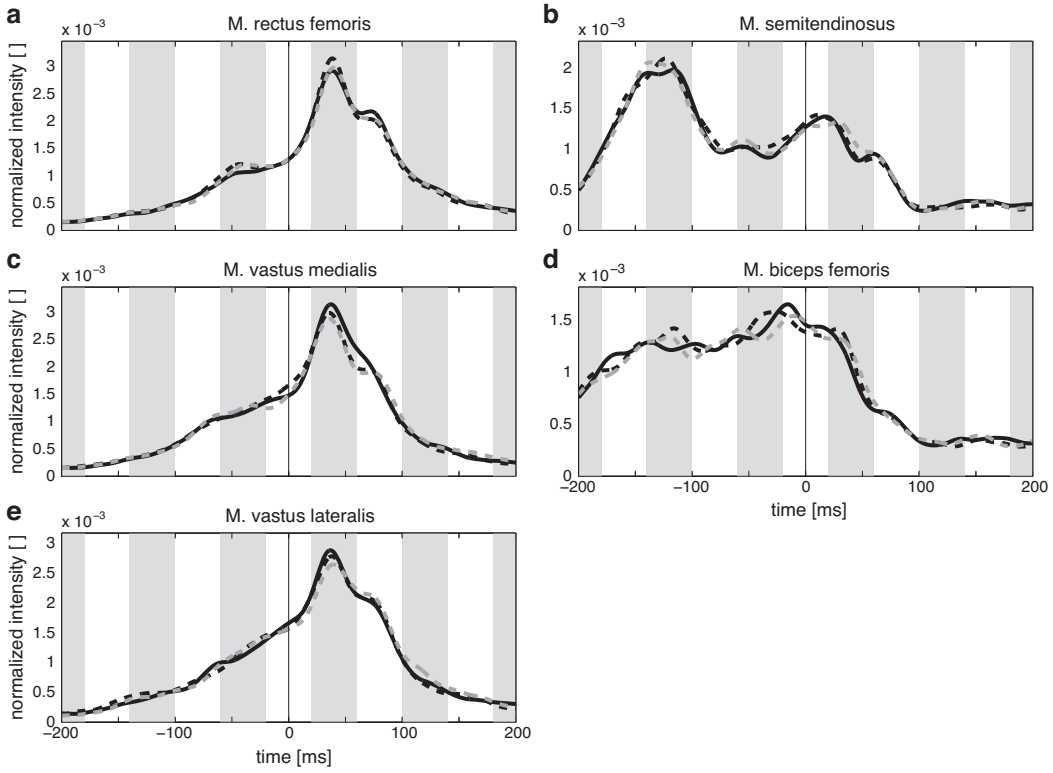

Fig. 2. Line graphs of the group waveforms of the Mm. rectus femoris (a), semitendinosus (b), vastus medialis (c), biceps femoris (d), and vastus lateralis (e) are illustrated for the lateral (black line), original (black dashed line), and medial (grey dashed line) electrode position. Time 0 indicates heel strike (vertical black line).

position between the subjects (Fig. 4a–c). This showed that during the pre heel strike period the timing of the peaks were not tightly controlled. In contrast, the first post heel peak at +35 ms was present in all subjects, however with varying intensity. The peak in the second post heel strike region was also located close to +75 ms, but was not present in all subjects. In summary, the QF muscle activation in the post heel strike period was well synchronised to heel strike and was dominated by peaks occurring in the 40 ms raster that was already observed in the group waveforms.

The ST, in contrast to the QF muscles, did not generally show a systematic alignment of peaks. However, some subjects activated the muscle with a peak at +35 ms and +75 ms, thus in synchrony

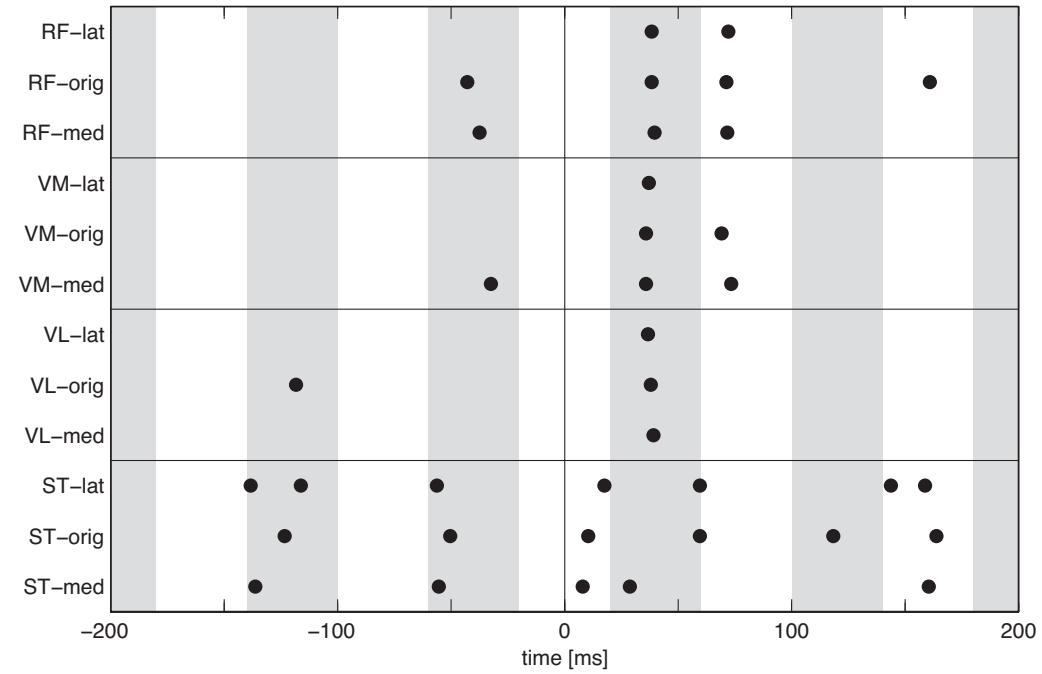

Fig. 3. Multi-muscle raster plot with peak locations (*x*-axis) detected in the group waveforms of the Mm. rectus femoris (RF), vastus medialis (VM), vastus lateralis (VL), and semitendinosus (ST) (*y*-axis, top down) are illustrated for the lateral (lat), original (orig), and medial (med) electrode position. Time 0 indicates heel strike (vertical black line).

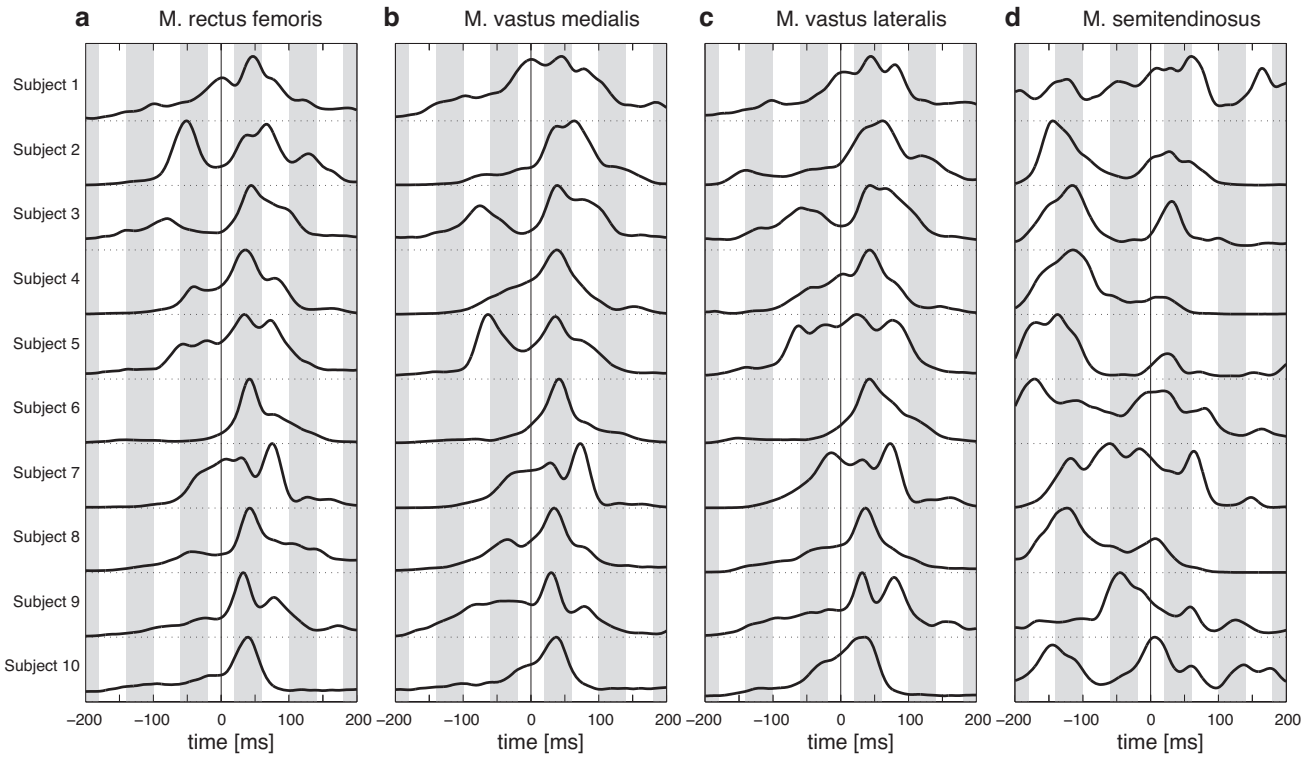

Fig. 4. Line graphs of the individual waveforms of the Mm. rectus femoris (a), vastus medialis (b), vastus lateralis (c), and semitendinosus (d) obtained from subjects 1 to 10 (top down). Each line represents the average across all waveforms. Time 0 indicates heel strike (vertical black line).

with the QF muscles, but this was the exception. In summary, each of the 10 subjects had their individual activation patterns with distinct peaks at mostly the same specific time points.

3.3. Within subject analysis

Only the first two post heel strike peaks of the QF muscles appeared consistently in most individual waveforms. The within subject analysis of any repetitive occurrences of peaks was therefore limited to the QF muscles. The waveforms of the steps of two extreme subjects (subjects 6 and 8), one with high and one with low repeatability between the waveforms, are shown for the RF, as an example, together with multi-step raster plots in Fig. 5. The waveforms reveal the step-specific strategy of muscle activation within a subject. The raster plots show the step-to-step temporal distribution of the peaks across all steps. The same-coloured dots are mostly arranged in a band with loose dots (outliers) of other colours. However, a distinct separation, based on a raster plot that would have allowed a statistical assessment of the separation of the bands failed because the jitter was too large. The within subject variability could therefore only be reported by showing the waveforms of individual subjects and qualitatively relating them to the raster plot.

Subject 6 showed consistent repeats of muscle activation for each step, especially for the main peak at +35 ms (SD = 2.3 ms) which yielded a narrower bandwidth with one outlier in the raster plot (pink dots in Fig. 5e). The second peak occurred with a jitter around +75 ms and occasionally the muscle was activated by the third or higher order peak. The raster plot discriminated the first two post heel strike peaks whereas the other peaks, whether pre or post heel strike, did not visibly fall onto a distinct raster. The similar phenomenon was found in a second subject, subject 9.

Subject 8 showed less consistency of the waveforms. Some well-controlled precise timing was apparent by a nearly vertical alignment of pink dots that consequently led to a relatively narrow band with loose outliers (Fig. 5f). The remaining bands consist of more scattered dots and therefore broader bandwidths. Thus, only the individual waveform (Fig. 5a and b) together with the raster plot (Fig. 5e and f) indicated that the pulses had some systematic occurrences. For instance, the individual waveform of subject 8 showed a preferred activation of the last band before heel strike which corresponded to the yellow dots in the raster plot.

Finally, the analysis of all 20,032 inter-pulse distances revealed a standard right-skewed gamma-distribution with the scale parameter $\theta = 11.22$, the shape parameter $k = 4.57$ and the maximum value at 39.7 ms (Fig. 6). The skewness value was 1.92 and was significantly different from normal distribution ($p < 10^{-3}$) for the inter-pulse distances.

In summary, the within subject analysis showed that, although there was a lot of jitter in the locations of the peaks, the individual waveform revealed a structure showing distinct peaks. The activation during each step distributed the motor unit action potentials in a step specific way but on average on a kind of predefined but not utterly precise raster.

4. Discussion

The basic activation pattern of the RF, VM, and VL in the group analysis indicated a high intra- and inter-muscular coordination (Fig. 2a, c and e) for the entire QF muscle. EMG signal combined with the wavelet-based analysis of this signal was sufficiently sensitive to detect a synchronisation of the activation of thigh muscles while walking. The neuromuscular control uses a raster like timing frame for the synchronisation of muscle activity during gait. The raster like timing frame, on average about 40 ms, seems to control the pacing of the neuromuscular activity. This raster is not limited on one pulse but on the whole sequence of −200 ms to +200 ms and is indicated in all figures by the underling grey shaded areas. The pacing frequency of different subjects were scattered across a small range, thus the

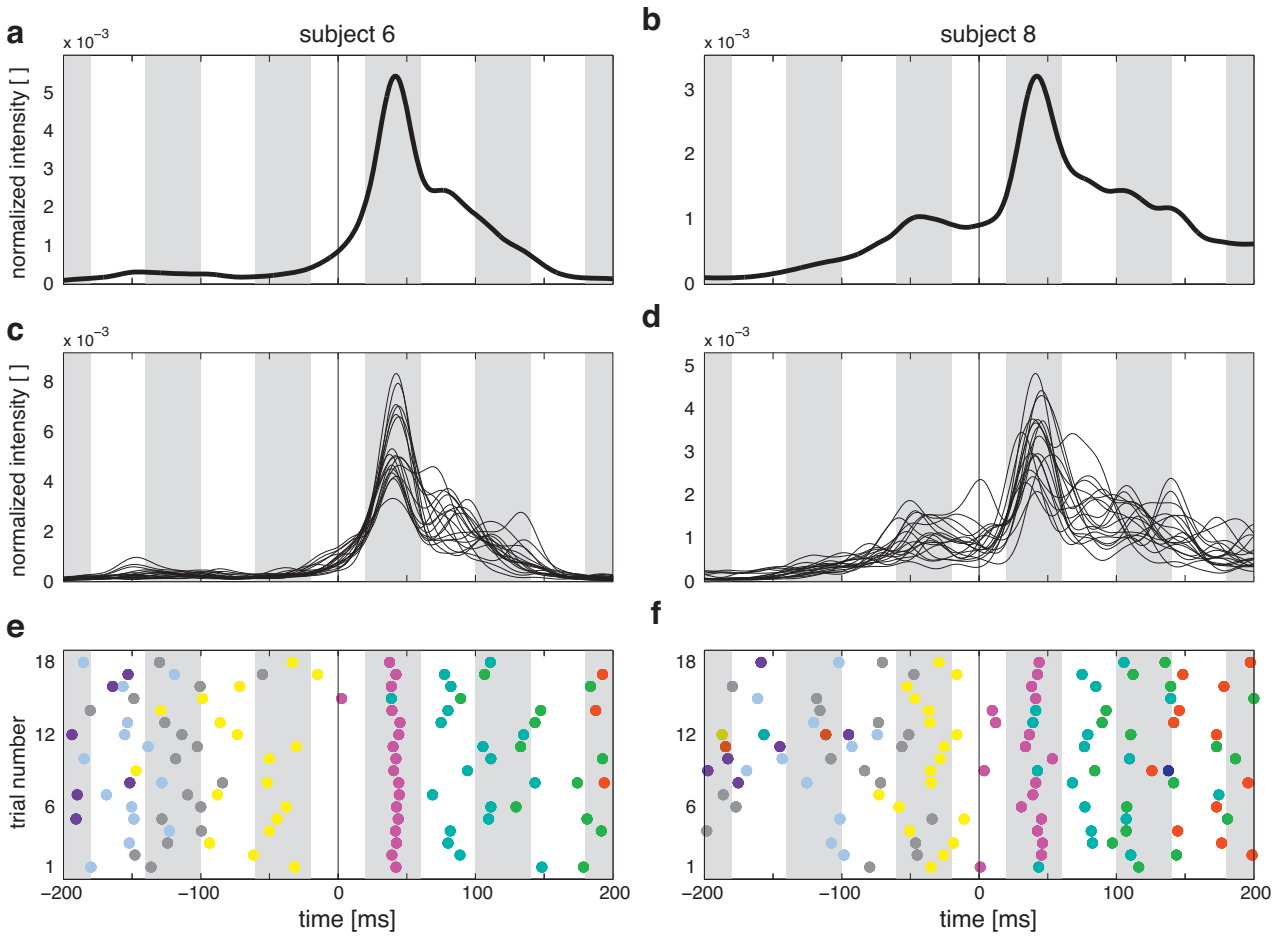

Fig. 5. Line graphs of the individual waveforms (a and b) and the waveforms for all steps (c and d) for M. rectus femoris are shown together with multi-step raster plots with peak locations (*x*-axis) detected in 18 waveforms (*y*-axis) for subjects 6 and 8, respectively. Time 0 indicates heel strike (vertical black line).

averaging of the individual waveforms did resolve the pacing properties of the neuromuscular control. However, in contrast to maximally activated muscles (Brown et al., 1998; von Tscharner et al., 2011a, 2011b) and to running (Stirling et al., 2011) where the neuromuscular rhythms are precisely controlled, walking is a movement that has more degrees of freedom and does therefore not need a tight control of muscle activity. This laxity of the movement is reflected in the jitter of the timing and amplitudes of the muscle

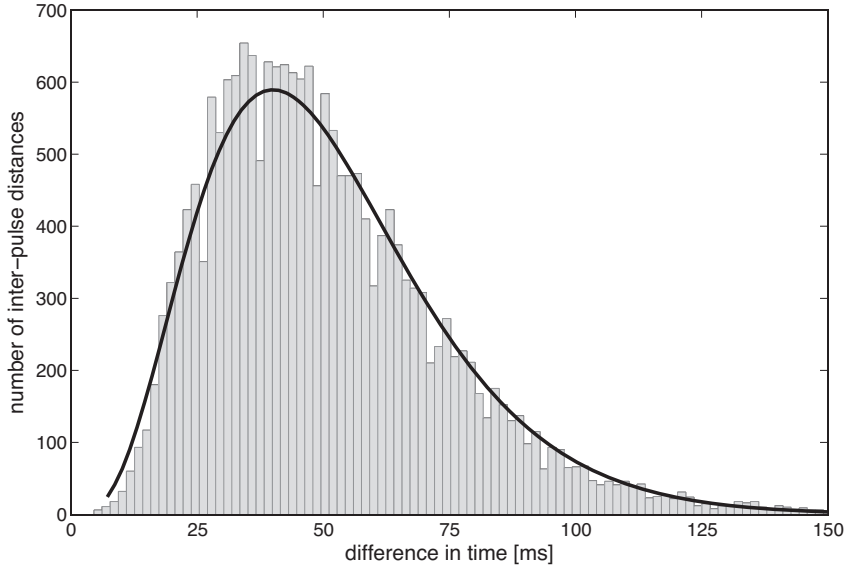

Fig. 6. Histogram of 20,032 inter-pulse distances (grey area) of all 10 subjects across all waveforms (without averaging across the triplicate electrode pairs) is illustrated. The black line is the gamma distribution fit to the data.

activation during individual steps. However, on average, this activity seems to be controlled at 40 ms intervals. The study indicates that there is a balance between a strictly paced neuromuscular control and a more random recruitment of motor units.

The raster of 40 ms is both subject and muscle independent. Our result agrees with earlier findings showing rhythmicity in skeletal muscles (Brown et al., 1998; Stirling et al., 2011; Vallbo and Wessberg, 1993; von Tscharner et al., 2011a, 2011b) and support the idea of previous studies that temporal patterns and rhythms are represented by the central drive to muscles (Vallbo and Wessberg, 1993; Salenius et al., 1996, 1997; Brown et al., 1998; Brown, 2000; Stirling et al., 2011; von Tscharner et al., 2011a, 2011b). It is conspicuous that the raster is synchronised to the time of heel strike. It seems that the neuromuscular system has the ability adapting to an external trigger, in this case the heel strike (Arsenault et al., 1986; Boyer and Nigg, 2004; von Tscharner et al., 2003), whereas the ST and the QF muscles have different controlling strategies. The ST is triggered in the pre heel strike period and the QF muscles, which requires knowledge from previous heel strikes to estimate the time of the next ground contact. The jitter of the pacing seen in the raster plot reflects the muscular coordination at heel strike. It seems that subjects with a more pronounced inhomogeneity of peaks around heel strike required a lower neuromuscular control anticipation of the next heel strike. The control works like a pre-programmed adaptive feedback activation.

The precise peak locations in this study confirms the theoretical predictions of Hof (2003) obtained by a simulation study. Hof stated that equal gait movements could be generated by identically timed muscle activation, only by changing the activation amplitudes. In our view, subjects with a high precision of waveforms seem to have the ability to better adapt the muscular system on exterior influences by changing their pre-programmed activation. Based on the subject-to-subject variation in shape but not in peak locations, the variations could originate from varied muscular recruitments, physiological, and morphological properties of the muscle. Especially, features changing the muscle morphology (e.g. size, fibre type composition (Wakeling et al., 2002)) as well as the interplay between and within muscles caused by various training regimes (Huber et al., 2010; Zebis et al., 2008) can change the neuromuscular control reflected by the waveforms. Furthermore, the between-subject variability most likely reflects that different activation patterns are used to generate the same movement (Hug et al., 2010). To stabilise a preferred movement pattern and react to varying forces, the neuromuscular control has to adapt (Kellis, 1998).

The observed EMG intensities reflect the specific timing around heel strike of the activation of the major thigh muscles while walking. This timing is important to coordinate co-contraction of the QF muscles and its antagonist ST (antagonist–agonist interplay) and to achieve preparatory knee positioning before initial contact. Precisely timed co-contraction is needed to regulate stiffness of the muscular system which is required to tolerate and absorb high impact forces.

The precise timing is also important for regulating the synergistic function of the QF muscles stabilising the knee joint at the time around heel strike. A balanced activation of the VM and VL is known to prevent a patellar maltracking (Cowan et al., 2001; Mellor and Hodges, 2005a, 2005b; Pal et al., 2011), reduce the stress on the anterior cruciate ligament (Hewett et al., 2006), and support a dynamic knee joint stability. A more holistic functional consideration was used by Gizzi et al. (2011), Clark et al. (2010), and Monaco et al. (2010). They refer to muscle synergies also called muscle modules. These articles conclude that there may be a limited number of about four driving sources controlling between 8 and 32 muscles. The activation timing profile in these cases were resolved with a time resolution estimated to be larger than 100 ms and therefore represent a different aspect of muscle activation than the one presented here. The time resolution was so large that the activation timing profiles do not reflect the fine structure of the 40 ms raster observed in the present study. It might well be that the observed activation timing profiles shown by Clark et al. (2010) might show a superimposed rhythm of 40 ms, if resolved with more details. It would be interesting to see whether the activation timing profiles could be seen as structures paced at about 25 Hz, a frequency corresponding to the beta waves of the brain. In that case one could speculate that the modules represent a means of weighing the underlying 25 Hz pacing before sending the commands to the muscles.

In summary, the study shows that the wavelet-based analysis of the EMG signal was sufficiently sensitive to detect a synchronisation of the activation of thigh muscles while walking. The peak muscle activation predominantly occurred at times that fit a raster like frame of about 40 ms, however with variable amplitudes. Albeit the jitter of the signal, the results resolved the temporal dependency of intensity peaks within muscles surrounding the knee joint and provided an insight into neural control of locomotion. The methodology to assess the stabilising muscle activation pattern may provide a way to discriminate subjects with normal gait pattern from those with a deteriorated neuromuscular control strategy. In future, we will apply further analysis methods such as Principal Component Analysis to resolve different activation strategies between the subjects.

Conflict of interest

The authors report no potential conflict of interest or the appearance a conflict of interest with regard to the study presented in this paper.

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