

Neuromuscular alterations during walking in persons with moderate knee osteoarthritis

C.L. Hubley-Kozey^{a,b,*}, K.J. Deluzio^b, S.C. Landry^b, J.S. McNutt^b, W.D. Stanish^c

^a School of Physiotherapy, Dalhousie University, Halifax NS

^b School of Biomedical Engineering, Dalhousie University, Halifax NS

^c Department of Surgery, Dalhousie University, Halifax NS

Received 18 February 2005; received in revised form 11 July 2005; accepted 20 July 2005

Abstract

This paper compared the neuromuscular responses during walking between those with early-stage knee osteoarthritis (OA) to asymptomatic controls. The rationale for studying those with mild to moderate knee OA was to determine the alterations in response to dynamic loading that might be expected before severe pain, joint space narrowing and joint surface changes occur. We used pattern recognition techniques to explore both amplitude and shape changes of the surface electromyograms recorded from seven muscles crossing the knee joint of 40 subjects with knee OA and 38 asymptomatic controls during a walking task. The principal patterns for each muscle grouping explained over 83% of the variance in the waveforms. This result supported the notion that the main neuromuscular patterns were similar between asymptomatic controls and those with OA, reflecting the specific roles of the major muscles during walking. ANOVA revealed significant ($p < 0.05$) differences in the principal pattern scores reflecting both amplitude and shape alterations in the OA group and among muscles. These differences captured subtle changes in the neuromuscular responses of the subjects with OA throughout different phases of the gait cycle and most likely reflected changes in the mechanical environment (joint loading, instability) and pain. The subjects with OA attempted to increase activity of the lateral sites and reduce activity in the medial sites, having minimal but prolonged activity during late stance. Therefore, alterations in neuromuscular responses were found even in this high functioning group with moderate knee OA.

© 2005 Elsevier Ltd. All rights reserved.

Keywords: Neuromuscular control patterns gait knee osteoarthritis pattern recognition

1. Introduction

Osteoarthritis (OA) is a common age-related impairment that causes pain and physical disability with the knee one of the most common joints affected [44]. There is some evidence of neuromuscular impairments of the musculature surrounding the knee joint such as reduced muscle strength [9,14,18,41,42], imbalance between the knee flexors and extensors [41] [31], motor unit inhibition [19,32] [4,7] and proprioceptive

deficits [18,20] for those with knee OA. It is unclear whether the deficits are the result of the disease process or a risk factor contributing to disease progression [6,37,41,42]. How these neuromuscular impairments affect function have not been fully explored. The musculature surrounding the knee joint produces motion, modifies joint loading and provides stability. Therefore, alterations in the neuromuscular strategies associated with OA should modify muscle force distributions changing the mechanical environment of the joint. Knowing how and when these changes occur during OA progression will further our understanding of the pathogenesis of the disease.

* Corresponding author. Tel.: +1 902 494 1941; fax: +1 902 4942635.
E-mail address: clk@dal.ca (C.L. Hubley-Kozey).

There are three proposed explanations of the expected knee neuromuscular responses during walking for those with knee OA i) higher lateral muscle site forces to counteract the high medial joint loading often found in those with OA [1]; ii) higher co-activity of agonist and antagonist pairs to increase joint stiffness to counteract instability associated with joint space narrowing [6] and iii) higher medial muscle site co-activity to increase medial joint stiffness in response to a greater medial joint space narrowing [28]. Co-activity increased joint stiffness and recently it has been demonstrated that medial and lateral knee muscles can be differentially recruited to control abduction-adduction moments and abduction-adduction joint stiffness during static and dynamic loading [57]. All three proposed explanations have merit, but no study of OA gait has simultaneously evaluated the amplitude and temporal characteristics of the muscle activation waveforms during the complete gait cycle.

Much has been learned about the neuromuscular responses of the lower-limb musculature during normal gait using surface electromyography (EMG). The EMG waveforms illustrate clearly established roles for the major muscle groups during walking that have been well documented [39,47,51,55]. More quantitative analysis of these waveforms has been done using pattern recognition techniques based on orthogonal expansion theory [12]. These studies [21,33,40,53,54] have consistently identified similar neuromuscular control patterns for the lower limb muscles of healthy subjects during walking. The pattern recognition techniques allow statistical comparisons of amplitude and temporal (shape) characteristics of the entire EMG waveform. Evidence supports the presence of consistent patterns for specific muscles of healthy asymptomatic subjects, but how these patterns are altered in the presence of OA, in particular early OA, has not been thoroughly investigated. The potential value of using a pattern-recognition approach on the entire waveform is supported by the average EMG waveform data presented by Beneditti et al. [3] for control and severe OA groups. Although no quantitative analysis was performed on the waveforms, the differences were clear between the two groups, indicative of a change in the role of the knee musculature for those with severe OA [3]. Characterizing the neuromuscular responses throughout the entire gait cycle may provide a comprehensive picture of these responses beyond what can be gleaned from discrete measures such as duration of activity [6] or co-activation indices [6,28]. It is important to know how the muscles respond throughout the gait cycle to the changes in joint dynamics.

In summary, the question that has not been addressed and is central to this paper is whether the neuromuscular responses differ during dynamic loading in those with early-stage OA compared to those without

symptoms. The rationale for studying those with mild to moderate knee OA was to determine the alterations that might be expected before severe pain, joint space narrowing and joint surface changes occur. The hypotheses were: i) that the principal neuromuscular patterns are similar between asymptomatic controls and those with OA, reflecting the specific roles of the major muscle groups during walking and ii) that there are subtle differences in the neuromuscular responses of those with OA throughout the gait cycle compared to asymptomatic controls. The expectation was that the subtle differences are manifestations of the neuromuscular deficits, the mechanical environment (joint loading, instability) and pain. To address these hypotheses pattern recognition techniques based on orthogonal expansion theory [17] were applied to the EMG waveforms from seven muscle sites crossing the knee joint and the principal patterns were compared using statistical hypothesis testing between the asymptomatic controls and OA groups.

2. Methodology

Subjects with knee OA were recruited from bi-weekly reviews of waiting lists for investigative arthroscopy, and clinic visits over an 18-month period (February 2003–August 2004). Those in the OA group were included if they had symptomatic unilateral *moderate* osteoarthritis and an assessment including radiographs and a physical exam. All of the patients with moderate OA had standard anterior-posterior and lateral radiographs of the knee taken within 12 months of their visit to the gait lab. These radiographs were graded for severity of osteoarthritis using the Kellgren and Lawrence (KL) global rating [24]. To be included, the subjects with OA had to have a KL grade between 1 and 3, indicative of mild to moderate joint changes. Asymptomatic controls were recruited through postings on the University bulletin board and posters at local hospitals. Subjects in both groups were included if they were over 35 years old, able to walk a city block, jog 5 meters and walk up stairs in a reciprocal manner. This was a further attempt to ensure that the participants with OA had a reasonably high level of function. Exclusion criteria included any neuromuscular disease such as Parkinson's Disease, stroke etc, cardiovascular disorders or surgery to the lower limb (except for exploratory arthroscopy, lavage of knee joint or partial meniscectomy at least 1 year prior to entry into study). All subjects signed a written consent in accordance with the policy of the Research Ethics Board for Health and Medical Sciences at Dalhousie University.

Subjects filled out a WOMAC OA-specific questionnaire [2] and SF36 general health outcome questionnaire [46] prior to testing. A standard gait assessment including motion and force data were collected, however, since

the focus of the present study was on the muscle activation waveforms only the EMG methodology is described in detail. Standard skin preparation over electrode sites was completed before silver/silver chloride pellet surface electrodes (.79 mm² contact area, Bortec Inc, Calgary) were attached in a bipolar configuration (20 mm centre-to-centre), in line with the muscle fibers over the rectus femoris (RF), vastus lateralis (VL), vastus medialis (VM), biceps femoris (LH), semimembranosus (MH), lateral (LG) and medial gastrocnemius (MG) using standardized placements based on palpation of specific anatomical landmarks [16,27]. The test leg was the affected side for the subjects with OA and a randomly selected leg for the asymptomatic controls. A reference electrode was placed over the tibial shaft. Palpation during resisted activations of the selected muscles and assessment of the EMG recordings while subjects performed a series of isolated movements [26] aimed at activating the different muscles were used to validate electrode placement [38,50]. No recordings were made within ten minutes of electrode placement [34] to ensure that the ratio of the skin-electrode impedance to the input impedance of the amplifier was less than the 1% recommended ratio [49]. The raw EMG signals were preamplified (500x) then further amplified (bandpass 10–1000 Hz, CMRR = 115dB (at 60 Hz), input impedance ~10 Gohm) using an eight channel surface EMG system (AMT-8 EMG, Bortec Inc., Calgary, Alberta). The raw EMG signals were digitized at 1000 samples per second, using the analog data capture feature of the Optotrack™ motion analysis system. A subject bias and noise trial in which the subject lay supine and completely relaxed was recorded prior to data collection.

Motion, force and EMG data were collected while subjects walked along a six-meter walkway at a self-selected walking speed. Walking speed was monitored using an infrared timing system and subjects were required to complete five trials within 5% of their self-selected speed. Motion data were captured using an Optotrack™ system and the ground reaction forces were measured using an AMTI™ force plate. The motion and force data were used to define heel contact and toe off for stride and step identification. These events were used to identify one gait cycle and to synchronize the EMG to define the appropriate window of EMG data. The knee joint angular displacements were calculated from the motion data and the maximum knee flexion angle during stance phase was recorded as the peak knee angle.

Following the walking trials subjects performed eight maximal voluntary isometric contractions (seven against a Cybex™ dynamometer, Lumex, NY) to elicit maximal activation amplitudes for normalization purposes and to provide a measure of muscle strength in Nm for the knee extensor, knee flexor and ankle plantar flexor groups. The exercises included 1) plantar flexion in supine position with ankle in neutral and

knee close to full extension, 2) heel rise standing on one foot; the subject was asked to raise his/her heel just off of the floor as forcefully as s/he could against resistance, 3) knee extension in supine with knee at 45° of flexion, 4) knee extension and hip flexion same as 3 but subject simultaneously flexed his/her hip and extended his/her knee, 5) knee extension in supine at 15° of knee flexion, 6) knee flexion in supine with knee flexed to 55°, 7) knee flexion in supine with knee flexed to 15°, and 8) knee flexion in prone position with knee flexed to 55°. All exercises were held for 3 seconds and were performed two times in total. Subjects were asked to give a maximal effort for each exercise and were given practice and then feedback on their performance for torque production. Although concerns existed with respect to eliciting a maximal effort in particular from patient populations, the maximal isometric effort was previously shown to be repeatable [43] and provides a physiological basis on which to make comparisons in EMG amplitudes among muscles [5,22]. In addition Lewek et al. [29] showed that repeating trials, providing feedback and motivation resulted in no difference in the ability to maximally recruit the quadriceps muscles between those with OA and asymptomatic controls, with both groups able to voluntarily elicit over 93% of their maximal stimulated activity. The torque and angle data were recorded at 1000 samples per second simultaneous with the EMG data using the data capture feature of the Optotrack™ system for exercises 1, 3, 5, 6, 7 and 8. A gravity correction trial in which the subject completely relaxed their muscles was also recorded prior to each normalization exercise.

3. Muscle moments of force

A Matlab™ (version 6.0) program was used to calculate the gravity corrected torque (Nm) for the supine knee flexion (15 and 55°), prone knee flexion (55°), extension (15 and 45°) and supine plantar flexion contractions. The program calculated the 1 second maximal steady state window of torque data within the 3-second contraction. The maximal value between the two trials for each exercise was recorded as the maximal torque.

4. EMG processing

All EMG processing was done using Matlab™ (version 6.0). The EMG data were corrected for bias, converted to μ V, full-wave rectified and low pass filtered at 6 Hz using a Butterworth filter [48]. For the normalization exercises a moving window algorithm was used to determine the 0.1 second window in which the maximum EMG amplitude occurred for each muscle

individually [45]. The maximal amplitude regardless of the exercise was considered the maximum voluntary isometric contraction (MVIC) used to normalize the EMG data from the walking trials. The EMG waveforms were amplitude normalized to MVIC and were time-normalized to 100% of the gait cycle. The five walking trials were averaged to create an ensemble average profile for each muscle for each subject [51]. These waveforms were used as input to the pattern recognition algorithms based on orthogonal expansion theory [17]. The pattern recognition was applied to the matrices of waveforms for the three quadriceps ($X = 101 \times 234$); the two hamstrings ($X = 101 \times 156$); and the two gastrocnemius ($X = 101 \times 156$) muscles separately.

Briefly the ensemble-average waveforms form a matrix \mathbf{X} such that:

$$\mathbf{X}(m, n) = [\mathbf{x}_1, \mathbf{x}_2, \mathbf{x}_3, \dots, \mathbf{x}_n].$$

Each x_i is a time normalized profile for a single muscle site and n is the number of waveforms included in the analysis. \mathbf{C} is formed by calculating a cross product, matrix of the columns of \mathbf{X} :

$$\mathbf{C}(m, m) = \mathbf{X}\mathbf{X}^T.$$

The transform matrix \mathbf{T} (m,m) is calculated using an eigenvector decomposition of matrix \mathbf{C} .

$$\mathbf{C} = \mathbf{T}\mathbf{\Lambda}\mathbf{T}^T.$$

Therefore, \mathbf{T} is a matrix of patterns (orthonormal eigenvectors) and $\mathbf{\Lambda}$ is a diagonal matrix of the associated variances (eigenvalues). The transform results from calculating y_i , the vector of *scores* (weighting coefficients) for each of the measured waveforms.

$$y_i = \mathbf{T}^T \mathbf{x}_i.$$

If fewer than m patterns are included then there is data reduction. An estimate of how many patterns (k) are required to explain the variance in the entire measured data set can be determined by calculating the % trace.

$$\% \text{trace}[\mathbf{C}] = \sum \lambda_i \times 100 / \text{trace}[\mathbf{C}] (i = 1, k),$$

where λ are the eigenvalues in descending order of magnitude and k is less than m . The EMG waveforms for each muscle are reconstructed (\mathbf{x}_{ri}) including k patterns,

$$\mathbf{x}_{ri} = \mathbf{T}_r y_{ri},$$

where T_r is the reduced transform matrix including only k patterns with the highest associated variance. Three methods were used to assess how well the principal patterns captured the salient features in the measured data: i) a % trace greater than 90% for the k patterns, ii) an error assessment of the reconstructed versus the measured waveforms using k patterns and iii) patterns with less than 1% variance were excluded. The *scores* for the k principal patterns were then used in the statistical hypothesis testing.

5. Statistical analysis

Means and standard deviations were calculated and t-tests ($\alpha = 0.05$) were used to test for statistically significant differences between the two groups for age, height, mass, BMI (body mass index), Womac, SF36, walking speed, stride characteristics, knee angle during early stance and maximal isometric plantar flexor moments (strength). A two-factor (group, exercise) mixed model ANOVA was used to determine differences in the maximal moment of force (muscle strength) for the two knee extension and the three knee flexion exercises. Two-factor (group, muscle) analysis of variance models were used to test main effects and two-way interactions for the *scores* on the k principal patterns for each muscle group separately. A Bonferonni post hoc procedure was used to test for pair-wise differences correcting α based on six comparisons for the hamstrings and gastrocnemius ($\alpha = 0.008$) and 15 comparisons for the quadriceps ($\alpha = 0.003$) muscles [56]. Minitab™ (version 14) statistical software was used for all statistical analyses.

6. Results

Forty subjects with OA (11 F, 29 M) and 38 (21 F, 17 M) asymptomatic controls completed the entire testing protocol. There were significant differences ($p < 0.05$) between the two groups for mass, BMI and age with the subjects with OA greater on each as indicated in Table 1. There were significant differences ($p < 0.05$) in all of the WOMAC and SF36 values except for mental health and role limitation-emotional as seen in Table 1. Two subjects with OA had a KL grade of I, 23 had a grade of II and 15 had a grade of III. There were no significant differences ($p > 0.05$) in stride lengths or stride times but the OA group had significantly ($p < 0.05$) slower walking speeds (0.08 m/s) and knee flexion angle (3°) during stance as illustrated in Table 2. Two asymptomatic controls had walking speeds greater than 1.8 m/s placing them as outliers. The analysis was rerun without including these two subjects resulting in no significant differences ($p > 0.05$) between the two groups for walking velocity although peak stance phase knee flexion angle was still significantly higher for the controls (3°). Therefore the difference in speed was minimal and the difference in angle was small so the two outliers were included in all subsequent analyses.

There were no significant differences between the two groups for any of the maximal muscle moments during the normalization exercises as shown in Table 2. The differences between the two groups ranged from 0.6 Nm to 6.5 Nm for the different muscle groups and test positions. This was less than a 6.6% difference in maximal muscle moments for all tests. There were significant

Table 1
Descriptive Statistics for asymptomatic controls and moderate OA

Variable	Controls		ModOA		p-value
	Mean	SD	Mean	SD	
Height (m)	1.72	0.10	1.75	0.09	0.16
Mass (Kg)	73.60	16.30	92.10	18.50	0.00
Age (years)	51.08	9.99	58.90	8.07	0.00
BMI (Kg/m ²)	24.74	4.25	29.94	4.86	0.00
SF 36-PF	96.05	5.95	61.20	19.70	0.00
SF 36-RP	96.70	14.40	60.30	42.40	0.00
SF 36-BP	86.60	17.40	52.00	20.30	0.00
SF 36-GH	84.40	14.10	73.20	16.20	0.00
SF 36-VT	73.40	18.00	64.20	18.40	0.03
SF 36-SF	97.04	9.39	87.50	16.00	0.00
SF 36-RE	96.50	17.00	92.30	19.40	0.32
SF 36-MH	83.20	10.40	85.10	13.10	0.47
Womac-Pain	0.29	1.18	6.90	3.47	0.00
Womac-Stiffness	0.16	0.68	3.44	1.55	0.00
Womac-Function	1.45	5.12	22.00	11.80	0.00
Womac-Total	1.89	6.94	32.4	16.00	0.00

BMI – body mass index, Key for SF 36 : PF- Physical function, RP- Role limitations-physical, BP- Bodily pain, GH- General health, VT- Vitality, SF- Social Function, RE- Role limitations-emotional, MH- Mental health.

Table 2

Walking stride characteristics, knee angle and maximal moment of force (strength) for quadriceps, hamstring (s –supine and p-prone) and plantar flexor during normalization contractions for asymptomatic controls and moderate OA

	Controls		ModOA		P-value
	Mean	SD	Mean	SD	
Stride Length (m)	1.46	0.12	1.42	0.13	0.19
Stride Time (s)	1.08	0.10	1.12	0.11	0.09
Speed (m/s)	1.37	0.19	1.29	0.18	0.05
Knee Angle (°)	21.7	4.99	18.4	5.79	0.01
Plantar flexion (Nm)	84.3	30.4	83.7	30.4	0.931
Knee extension 45° (Nm)	114.0	40.4	117.9	37.8	0.659
Knee extension 15° (Nm)	91.7	35.5	98.2	34.0	0.442
Knee flexion 55° (s) (Nm)	58.5	27.4	54.8	22.4	0.532
Knee flexion 55° (p) (Nm)	59.3	15.8	60.2	18.4	0.827
Knee flexion 15° (Nm)	46.5	17.4	44.5	18.0	0.626

Knee angle – peak knee angle during stance.

differences ($p < 0.05$) between the exercises with the knee extension at 45° producing higher moments than the knee extensions at 15°, and the two knee flexions at 55° significantly ($p < 0.05$) higher than the knee flexion at 15° for both groups. Since there were more males in the OA group and more females in the asymptomatic control group, re-analyses of the moment of force data were performed separately for the males and the females. Out of the 12 comparisons the only significant differences were between males with OA (103.6+/-25.1 Nm) and control males (121.3+/-27.1 Nm) for knee extension at 15° ($p = 0.04$) and between females with OA (38.5+/-9.2 Nm) and control females (52.8+/-19.2 Nm) for knee flexion at 15° ($p = 0.02$).

The ensemble average waveforms for the seven muscles for the two groups are found in the left panels of Fig. 1. Three principal patterns captured 96% of the variance in the amplitude and shape characteristics of the

temporal waveforms for the gastrocnemius muscles and 94% of the variance for both the quadriceps and hamstring muscles. Principal pattern one (PP1) captured the general shape and the overall amplitude of the EMG waveforms of the respective muscle groups and is illustrated on the right panels of Fig. 1. The other principal patterns captured the subtle differences in shape of the temporal waveforms. Examples of patterns that resulted in significant group or group by muscle effects are found in Figs. 2–4. Also depicted in Figs. 2–4 are examples of measured waveforms that have high and low scores for the respective patterns. The mean scores for the principal patterns with statistically significant ($p < 0.05$) results for each muscle group are found in Fig. 5.

The ANOVA of the scores for the gastrocnemius muscles in Fig. 5a and b revealed statistically significant ($p < 0.05$) main effects and interactions for PP1 and principal pattern two (PP2). PP1 captures (Fig. 1)

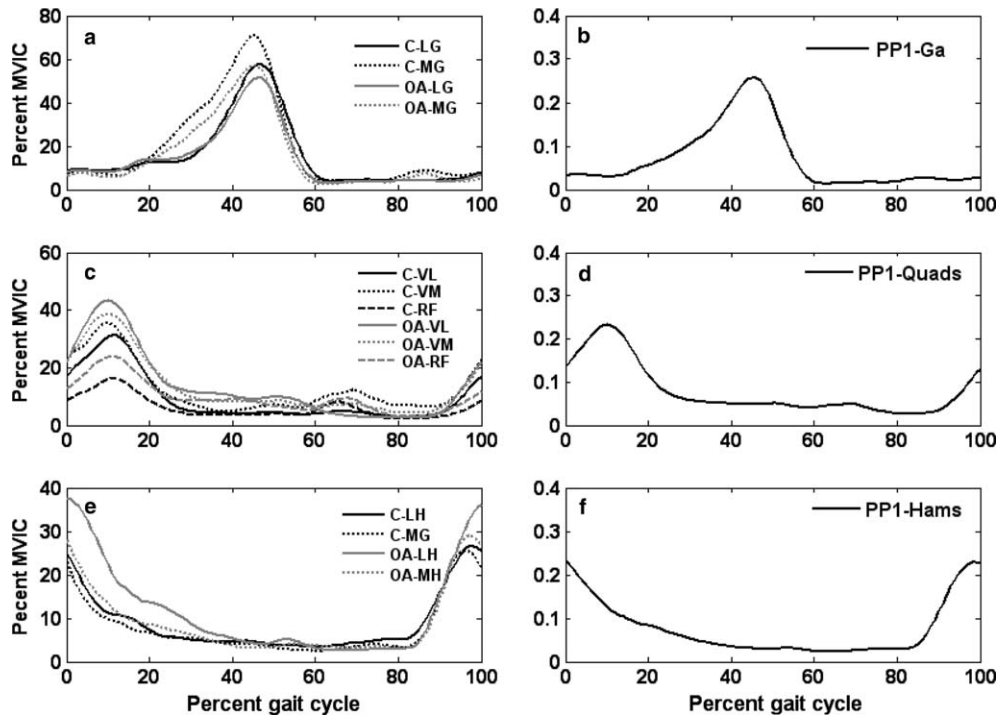


Fig. 1. The sample ensemble average waveforms for the control (black) and the OA (gray) groups are in the left panels. The y-axis is %MVIC. The upper panel a) includes the LG (solid) and MG (dotted); the middle panel c) includes the VL (solid), VM (dotted) and RF (dashed); and the lower panel e) includes the LH (solid) and MH (dotted). The principal patterns explaining the highest percentage of variance in the waveform data (PP1) are in the panels on the right. The y-axis is magnitude. The upper panel b) is for the gastrocnemius muscles (90% trace), the middle panel d) is the quadriceps (87% trace) and the lower panel f) is for the hamstrings (83% trace).

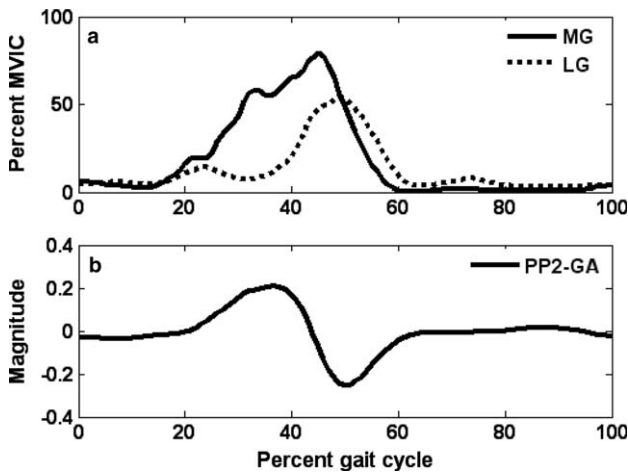


Fig. 2. The upper panel a) illustrates a MG (solid) with a high positive score for PP2 and a LG (dotted) with a negative score for PP2. PP2 in the lower panel b) depicts the phase shift in activity to earlier in the gait cycle. This pattern explains 4.3% of the waveform variance.

minimal activity during early stance followed by a gradual rise in activity, peaking just prior to 50% of the gait cycle then rapidly decreasing until toe off at 60%. The significant pair-wise comparisons illustrated that the control LG score was significantly lower than the control MG ($p < 0.008$), but not different from the OA LG or MG. The control MG score was also higher than

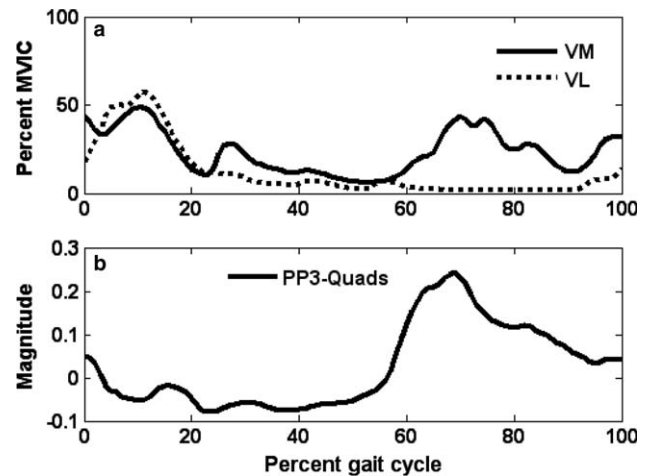


Fig. 3. The upper panel a) illustrates a VM (solid) with a high positive score for PP3 and a VL (dotted) with a negative score for PP3. PP3 in the lower panel b) captures the variation in activity after toe off during early swing. This pattern explains 2.8% of the waveform variance.

both OA gastrocnemius sites ($p < 0.008$). Fig. 5 also shows that PP2 had a statistically significant ($p < 0.05$) muscle effect capturing the phase shift between the lateral and medial sites (bottom panel of Fig. 2). An example of a LG with a negative score and a MG with a positive score for PP2 is found in the upper panel of Fig. 2 clearly demonstrating the phase shift to the left

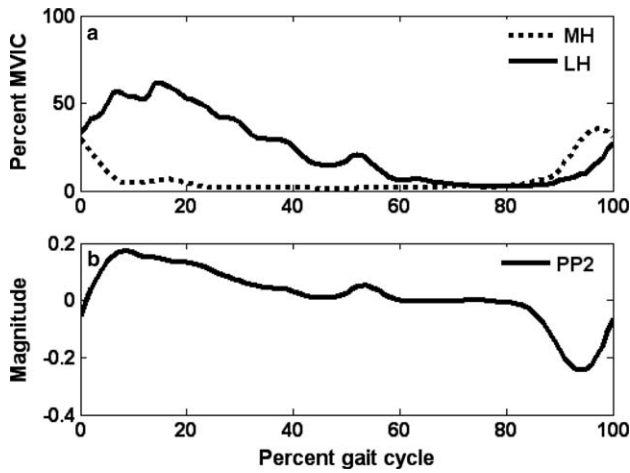


Fig. 4. The upper panel a) illustrates a LH (solid) with a high positive score for PP2 and a MH (dotted) with a negative score for PP2. PP2 in the lower panel b) captures the longer duration of activity during mid stance and the variation in late swing. This pattern explains 6.6% of the waveform variance.

for the MG. This phase shift for the MG is also seen in the ensemble average waveforms in Fig. 1. There were no statistically significant effects for principal pattern three (PP3) score for the gastrocnemius muscles.

The results for the quadriceps illustrated statistically significant ($p < 0.05$) differences between groups and among muscles for PP1, PP2 and PP3 scores (Fig. 5c, d, e). PP1 captured the general shape and overall amplitude of the quadriceps EMG waveforms as illustrated in the middle right panel of Fig. 1. The pattern included a burst of activity during the loading phase peaking at approximately 10% of the gait cycle, gradually decreasing to a plateau after 30% of the gait cycle, then increasing prior to heel contact. The key points from the statistical analysis are that the VL score for the controls was significantly lower than the VL for the OA group ($p < 0.003$), whereas there were no differences for the VM scores between groups ($p > 0.003$). The vasti muscle scores were significantly higher than the RF for both groups ($p < 0.003$), and while RF score for the OA group was higher it was not significantly different from the control ($p > 0.003$). The subtle changes in shape among muscle sites and between groups are illustrated in the ensemble average profiles in the middle left panel of Fig. 1, and occur during mid to late stance and early swing phases. Significant differences for PP2 and PP3 scores between groups and muscles captured these differences. PP2 captured an increase in amplitude during mid-stance reflecting that activity was present for a

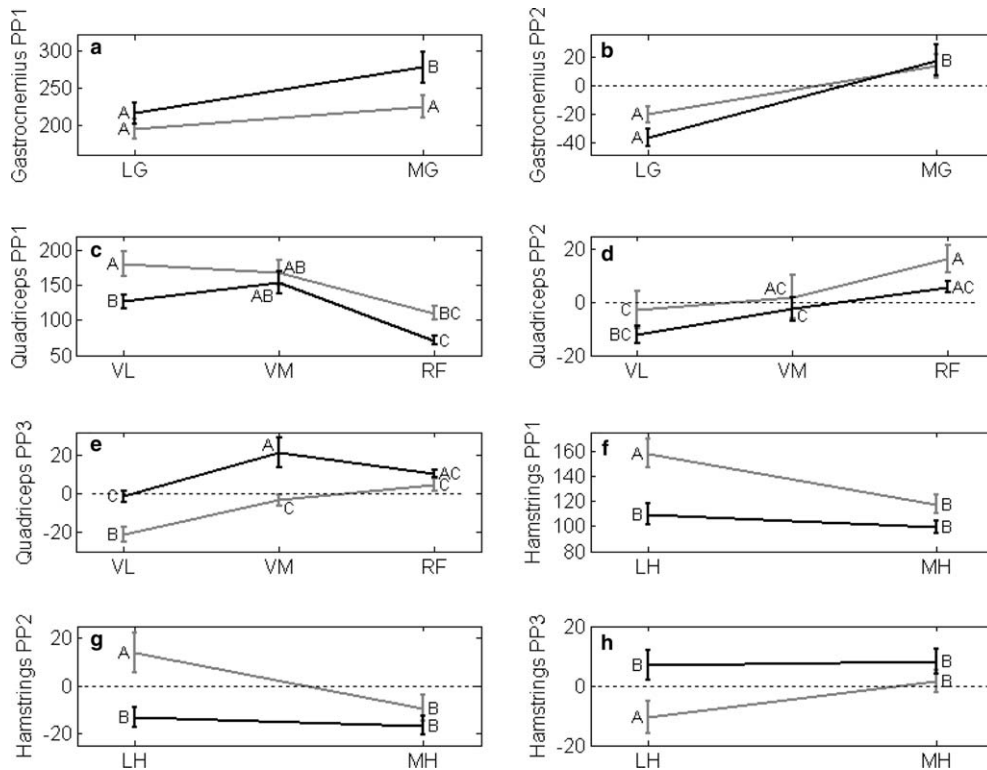


Fig. 5. The interaction plots including mean and SEM for the principal pattern scores. The black lines are for the asymptomatic controls and the gray lines are for the OA group. PP1 and PP2 scores for the gastrocnemius muscles are in panels a and b, PP1, PP2 and PP3 scores for the quadriceps muscles are in panels c, d and e and PP1, PP2 and PP3 scores for the hamstring muscles are in f, g and h. Significant pair-wise comparisons are indicated by the capital letters in the graphs. Means with the same letter are not significantly different from each other whereas those with different letters are significantly different from each other (Bonferroni corrected $\alpha = 0.008$ for gastrocnemius and hamstrings and $\alpha = 0.003$ for quadriceps comparisons).

longer duration. The pair-wise analysis revealed that the OA RF was different from the OA VL ($p < 0.003$) as well as the control VL, control VM; whereas the control RF was only different from control VL ($p < 0.003$). This result indicated that the subjects with OA recruited the RF during mid to late stance phase. PP3 had a significant group by muscle interaction with several pair-wise differences. The lower panel of Fig. 3 illustrates PP3 and a positive *score* captured the burst in activity during late stance-early swing peaking at 70% of the gait cycle. There was a significant difference ($p < 0.003$) between the OA VL (negative *score*) and all other *scores*. The control VM was significantly higher than the OA VM ($p < 0.003$). The upper panel of Fig. 3 illustrates the effect of a positive (VM) and a negative (VL) *score* for this pattern. The key findings for PP3 is that the control subjects activated the VM and RF during early swing; whereas, the OA VL decreased to minimal activity just after toe off.

Fig. 5(f, g, h) illustrates the statistically significant ($p < 0.05$) differences for PP1, PP2, and PP3 *scores* for the hamstring muscles. Fig. 1 shows that the amplitude and shape of the LH waveform for the OA group was different from the LH for the control and the MH for both groups. The statistical comparisons of the principal pattern scores substantiated these visual differences. PP1 captured the high activity at heel contact, decreasing during the first 10–15% of the gait cycle increasing again around 85% of the gait cycle, peaking prior to heel contact (lower right panel of Fig. 1). There was a significantly ($p < 0.003$) higher *score* for the LH for the OA group compared to the MH for the OA and both LH and MH for the controls. PP2 captured the prolonged activation during stance phase gradually decreasing until toe off and the continual increase in activity during late swing (i.e. peak activity does not occur before heel contact). There was a significant ($p < 0.05$) muscle by group interaction for PP2. The LH (positive *score*) of the OA group was significantly ($p < 0.008$) different from the MH for the OA group and both the MH and LH for the control group. The latter three muscle sites had negative *scores*. The effect of a positive versus a negative *score* for PP2 is depicted in the upper panel of Fig. 4. PP3 also had significant pair-wise differences ($p < 0.008$) with the OA LH different from the control LH and control MH but not OA MH ($p > 0.008$). A negative PP3 as found for the OA LH captured the continual rise in activity prior to heel contact with the amplitude peaking just after heel contact. A positive *score* in contrast captured the activity peaking prior to heel contact as illustrated in the bottom left panel of Fig. 1 for the control LH and the MH for both groups. The results for PP2 and PP3 illustrate a lack of synchronous co-activity between the LH and MH for the OA group.

In summary the statistical analysis of the principal pattern *scores* supported the finding of both amplitude and shape differences between the OA and control groups. For the quadriceps and the hamstrings the trend was for the muscles that produced lateral force vectors (VL, RF and LH) to have higher overall amplitudes for the subjects with OA compared to controls. With respect to the gastrocnemius muscle, the controls had higher MG overall amplitude compared to both the MG and LG for OA group. Also the control MG *score* was higher than the control LG, whereas no difference was found between the MG and LG for the OA. The subtle changes in the EMG waveform shapes corresponded to prolonged RF, VL and LH activity during late stance and decreased VL, and VM activity during swing for the subjects with OA compared to the controls and differences in OA LH during late swing.

7. Discussion

The subjects with OA were slightly older (although statistically significant, the mean age was within the same decade) and heavier than those with asymptomatic knees. The difference in BMI between groups is consistent with the literature for subjects of similar ages with and without OA [3,6,8,15,23]. As expected there were significant differences in the disease specific WOMAC scores, and all of the general health items of the SF-36 except role limitation-emotional and mental health. Normative data for WOMAC scores corresponding to osteoarthritic disease severity are not available. However, published WOMAC data from patients with severe OA, evaluated prior to total knee replacement surgery, revealed scores approximately twice as high as our data [30,36]. Therefore the WOMAC scores indicated mild to moderate levels of pain, function and stiffness in the moderate OA sample and confirmed that our control group represented an asymptomatic knee group. The SF-36 data also confirmed the general health of both groups. The mean response is at or above the mean and median norms for asymptomatic subjects in this age group [46]. Furthermore the *radiographic data* indicated that we have a sample of subjects with *moderate* knee OA severity since over 50% of our subjects with OA have grade I or II.

Although walking speed was different between the two groups, the difference was minimal and the other stride characteristics were not. The speeds for both groups were faster than those previously reported for healthy controls [23,35] and much faster than reported for subjects with OA [23,35]. The knee range of motion during stance was on average 3° less for those with OA and is consistent with previous reports of decreased knee flexion during early stance in OA [3,6]. The 18° range of motion during stance for the OA group is within the

range presented in the literature for those with healthy knees [6]. In summary, the demographic and descriptive data for the two groups supported the assumption that the two groups were similar on a variety of measures and that the OA group was moderate and not late in the disease progression.

The lack of significant differences in maximal muscle moments of force substantiated the similarities between groups for functional strength capacity for all three muscle groups tested. The different percentage of males and females in each group was a concern; however, the analysis of males and females separately illustrated minimal differences between controls and OA. Both differences found for the separate analysis occurred at the knee extended position (i.e. 15°). The trends were similar between the groups with higher torques produced by the knee extensors and flexors at mid-range (45 and 55° flexion) rather than the more extended position (15°). Our torque values are higher than some reports [37] and lower than others [6] for subjects of similar age. Therefore both groups were within normal ranges for muscle strength, and the differences were less than 7% between the groups for all test exercises. Many studies of those with OA have reported greater strength deficits when compared to asymptomatic controls [9,14,18,41,42] than our results, perhaps reflecting greater severity of OA in these studies when compared to our moderate OA group.

Distinctly different neuromuscular control patterns for the quadriceps, hamstrings and gastrocnemius muscles were captured by their respective PP1. The patterns reflected the role of each muscle group during the different phases of the gait cycle and are consistent with EMG waveforms previously presented in the literature for similar muscle groups during normal walking [35,53–55]. PP1 explained over 83% of the variance in the waveform data for each muscle group, and the PP1 *scores* in Fig. 5 are higher for both groups, for all muscles compared to the other principal patterns. Since the *score* reflected the weighting of that pattern to the measured waveform, it was concluded that PP1 for each muscle grouping reflected the main neuromuscular control patterns for each muscle, for both control and OA groups. These common patterns among muscles within a grouping corroborated previous reports that the activity patterns have a global control aspect [21,33]. The subtle differences between the individual muscles within a grouping and between the control and the OA group likely reflected responses associated with local feedback from somatosensory and perhaps pain receptors.

The results show that the differences in the moderate OA group were not as profound as the high degree of co-activity among agonists and antagonists shown in EMG waveforms from subjects with more severe OA [3]. The difference between the moderate and more severe group may partially be explained by the slower

walking velocities in the latter group [3] and possible responses to higher pain and joint instability in that group as well.

The key finding for the gastrocnemius muscles was that the subjects with OA had reduced MG activity compared to the control subjects. The differences in the medial and lateral site *scores* illustrated that the control group recruited the MG to higher percentages of MVIC than the LG whereas the OA group recruited both sides to similar amplitudes. For the controls this higher MG amplitude occurred during propulsion. Given that the cross-sectional area for the MG is approximately two times the LG [52] the reduction in MG activity adopted by the subjects with OA may be an attempt to reduce joint loading, in particular to selectively reduce medial joint loading. Determining whether this neuromuscular response is a learned feed forward or feedback response requires further investigation. The phase shift for the MG captured by PP2 reflected an earlier peak in activation during the gait cycle. This was found for both groups and does not support the finding of longer duration of activity for the MG previously reported for subjects with OA compared to controls [6]. In summary, there were no differences in shape between the OA and control gastrocnemius waveforms, with the key difference in amplitude reflecting decreased activation for the OA group.

PP1 for the quadriceps captured the burst of activity just prior to and during the loading phase of the gait cycle. The *scores* illustrated a higher VL recruitment for the OA group compared to controls and similar VM recruitment amplitudes between groups, typical of responses that may serve to decrease medial joint loading. The RF recruitment was lower for both groups compared to the two vasti muscles, although the trend was for a higher OA RF. The peak knee angle during stance was lower for the subjects with OA; therefore, this difference would not explain the trend for higher quadriceps activity for the OA group. The results for the PP2 *scores* illustrated that the RF for both groups was recruited during mid to late stance with a trend for the OA group to be higher. Since there was no difference in walking speed between groups, the increased RF *score* for the OA group during late stance may compensate for the reduced gastrocnemius activity prior to toe off. The higher PP1 *score* for the VL and the positive PP2 *score* for the RF for the OA group supported previous reports of longer durations of VL [6] and RF compared to controls [3]. This increased activity during mid stance phase may be a local response to joint instability or joint loading and it is interesting that it is more prominent for muscles on the lateral side. There were minimal differences between groups for PP1 and PP2 *scores* for the VM and this is verified by only a slightly elevated OA VM ensemble average waveform compared to the control VM in Fig. 1c. The positive PP3 *scores* for the control VM

and RF captured the burst of activity during early swing. This burst has been previously demonstrated for RF [21], and is associated with its' biarticular role of hip flexion. This perhaps has implications with respect to the role the hip joint flexors have in altering joint loading based on modeling work [13]. Gill suggested [13] that a reduced hip flexor moment contributed to higher impact loading at heel contact. In summary the overall differences in the principal pattern *scores* for the quadriceps provided evidence that those with OA had higher lateral site amplitudes during initial loading, prolonged activity during mid to late stance, perhaps to increase joint stability and a slightly altered role for the VM and RF during late stance and early swing.

PP1 for the hamstrings captured that activity peaks prior to heel contact to decelerate knee extension and prepare for initial loading and is consistent with previous reports [21,38,53–55]. The statistical analysis showed that the LH for the OA group was recruited to higher amplitudes than LH for controls and MH for both groups. Again, this recruitment strategy for the OA group appears to be consistent with attempts to decrease medial joint loading during initial contact. PP2 captured the prolonged activity during stance phase and the positive *score* for OA LH confirmed that this muscle was activated during most of the stance phase until just past 40% of the gait cycle. This prolonged activity during the stance phase could be an attempt to increase stability through antagonist co-activity since the VL and RF were also activated during this phase. This result for the LH is consistent with longer durations reported by Benedetti [3] for LH. Benedetti [3] and Childs [6] both reported longer durations for the MH in subjects with OA compared to controls, which is not substantiated by our data. The different finding may reflect the severity of OA and the walking speed differences between studies. The joint instability issue may not be as problematic in our moderate group, whereas decreasing the load on the medial joint may be more important at this stage of OA. The other difference between the groups also occurred with respect to the *score* for PP3. The OA group had a negative LH *score* for this feature indicative of a continued increase in activity until after heel contact, whereas a positive *score* (for the other three sites) indicated that the peak activity occurred prior to heel contact. The differences between the LH and MH in the PP2 and PP3 *scores* were indicative of a lack of coordinated activity between lateral and medial sites for the OA group only. The importance of appropriate patterns of synergistic and antagonist co-activation to maintain dynamic knee joint stability [25] has received considerable attention in the rehabilitation of those with joint stability problems. If the muscle forces are not produced in the correct manner the net result could be high overall forces such as the case with high

levels of agonist and antagonist co-activation [51] or different medial and lateral joint force distributions if muscles are selectively recruited [57].

The results support the view that the neuromuscular alterations in the moderate OA group waveforms were subtle compared to the substantial changes depicted in subjects with severe OA [3]. The overall pattern of neuromuscular control found for the moderate OA group provided support for the idea that the lateral muscle site produce higher forces to counteract the high medial joint loading often found in those with OA [1]. These findings are consistent with the higher lateral site activation for both healthy and those with OA reported by Lewek et al [28], but not the interpretation of their higher medial co-activation index for VM and MG for the OA group. The co-activation index they [28] calculated was for approximately the first 20% of the gait cycle only and unfortunately does not have a unique solution since a combination of activation amplitudes and patterns could achieve similar co-activation indices. They concluded that this higher co-activity for the subjects with OA on the medial side was an attempt to increase muscle stiffness to improve joint stability on the medial side of the knee during initial stance. They measured knee joint stability and it is possible that their subjects had greater medial joint instability than did our subjects. Our results showed that the co-activity among agonists and antagonists is specific to phases of the gait cycle and differs for lateral and medial sites.

Since all the muscle groups produced similar maximal moments of force for both groups, the higher overall amplitude for the agonist and antagonist muscles for the subjects with OA would result in increased muscle forces and subsequently an increased bone on bone force [48,51]. The next step is to begin modeling the muscle and joint forces by combining the EMG data with the joint kinetics. For example, the higher VL, RF and LH activity for the OA group occurred during early stance where others have shown that the adduction moment reaches its' first peak [23] and the preferential reduction in MG activity occurred during late stance (around 40%), when the second peak adduction moment typically occurs [1,23]. Gait kinetic studies showed higher adduction moments [1], reduced external knee flexion moments [23] and reduced plantar flexor moments [10,35] in those with knee OA compared to asymptomatic controls. Further investigation of the muscle forces should help to establish whether the muscle alterations are beneficial or detrimental to the mechanical environment of those with OA. The differences in neuromuscular control patterns found in our moderate OA group provided an indication that some protective mechanisms (higher lateral site activity) and some mechanisms potentially increasing the rate of disease progression (co-activity) were present in early OA.

It is hoped that these results will provide direction for developing non-invasive treatments that can lead to changing the loading distribution on the joint and not simply aspire to increase muscle strength. Sharma [37] argued that if exercises improve local imbalances in malaligned knees, this is a positive response, but if exercise results in altered muscle pull, then unequal force distributions may speed up disease progression. Similarly, if exercise improves dynamic stability then this is positive; if however the result is increased joint loading then this is a problem. Thus exercises that improve load distribution and stability should be the goal [37]. Our study provided a first step toward understanding the muscle responses of those with moderate OA during a dynamic task and with continued work will shed light on the role of the neuromuscular system to alter joint loading.

The three variables that could potentially explain differences in EMG waveforms between the two groups are: i) walking velocity, ii) knee angular displacement and iii) muscular strength. Studies [38,55] demonstrating the effect of walking speed on EMG waveforms used speed variations greater than 30%. The difference found in this study of 0.08 m/s, less than 6%, is unlikely responsible for differences in EMG waveforms between the two groups. The knee angular displacement difference of 3° is small, and it is unlikely that this difference in knee angle alone would result in a change in external moments about the knee joint that would have a substantial effect on the EMG waveforms. Finally, the maximal torque generating capability of the major muscle groups was not different between the two groups. Therefore, the muscle torques produced for each percentage of maximum should be comparable between the two groups. If the OA group produced a lower moment of force compared to the control group during maximum efforts, then amplitude changes in the EMG could be explained by differences in muscle strength between groups. This was not the case; therefore, these three variables alone do not explain the differences found in the EMG waveforms.

The results of this study must be interpreted within the limitations of the data. Using maximal voluntary isometric contractions for normalization purposes has been questioned; however, a recent thorough investigation of normalization techniques for gait [5] illustrates the value of the MVIC. Two recent studies [11,29] show that with appropriate procedures including feedback and motor learning principles that subjects with OA can recruit their muscles to similar percentages of maximum as controls. We utilized a series of muscle contractions in an attempt to elicit maximum activation as well as repeated trials, provided feedback and incorporated an algorithm to detect the maximum regardless of exercise. All these precautions improved the validity of normalizing to maximum and our confidence in stating the percentages. Therefore, the differences between the

groups would not be attributed to a difference in ability to produce maximums. The differences in medial and lateral joint loading were more difficult to interpret with respect to forces on the joint because of the difficulty in modeling individual muscle forces. There was a consistent attempt during both the loading phase (higher LH and VL) and the push off phase (lower MG) to reduce medial active muscle forces in the OA group.

8. Summary

Comprehensive, normalized EMG waveform data illustrated differences in neuromuscular control during walking between those with moderate OA and asymptomatic controls. These results supported the hypothesis that the principal neuromuscular patterns were similar between asymptomatic controls and those with OA, reflecting the specific roles of the major muscle groups during walking. However, the pattern recognition results also showed that there were subtle differences in the neuromuscular responses of those with OA throughout the gait cycle compared to asymptomatic controls. These alterations most likely reflected changes in the mechanical environment (joint loading, instability) and pain but not neuromuscular dysfunction since no strength differences were found between the two groups. The results demonstrated a medial versus lateral site difference, consistent with the OA group attempting to increase activity of the lateral sites and reduce activity in the medial sites. Therefore, even in this high functioning group with moderate knee OA alterations in neuromuscular responses were found.

Acknowledgement

The authors would like to thank Kelly Mckean and Mina Agarabi for their assistance with data collection and analysis. We also acknowledge the financial support of the Canadian Institutes for Health Research.

References

- [1] T. Andriacchi, Dynamics of knee malalignment, *Orthop Clin North Am* 25 (1994) 395–403.
- [2] N. Bellamy, W.W. Buchanan, C.H. Goldsmith, J. Campbell, L. Stritt, Validation study of WOMAC: A Health Status Instrument for Measuring Clinically Important Patient-relevant outcomes Following Total Hip or Knee Arthroplasty in Osteoarthritis, *J of Orthop Rheum* 1 (1988) 95–108.
- [3] M.G. Benedetti, F. Catani, T.W. Bilotta, M. Marcacci, E. Mariani, S. Giannini, Muscle activation pattern and gait biomechanics after total knee replacement, *Clinical Biomechanics* 18 (2003) 871–876.
- [4] M. Brucini, R. Duranti, R. Galletti, T. Pantaleo, P.L. Zucchi, Pain thresholds and electromyographic features of periarticular

- muscles in patients with osteoarthritis of the knee, *Pain* 10 (1981) 57–66.
- [5] A. Burden, M. Trew, V. Baltzopoulos, Normalization of gait EMGs: a re-examination, *Journal of Electromyography and Kinesiology* 13 (2003) 519–532.
 - [6] J.D. Childs, P.J. Sparto, G.K. Fitzgerald, M. Bizinni, J.J. Irrang, Alterations in lower limb extremity movement and muscle activation patterns in individuals with knee osteoarthritis, *Clinical Biomechanics* 19 (2004) 44–49.
 - [7] G.E. Doxey, P. Eisenman, The influence of patellofemoral pain on electromyographic activity during submaximal isometric contractions, *Journal of Orthopaedic and Sport Physical Therapy* 9 (1987) 211–216.
 - [8] S. Eyigor, A comparison of muscle training methods in patients with knee osteoarthritis, *Clinical Rheumatology* 23 (2004) 109–115.
 - [9] N.M. Fisher, D.R. Pendergast, Reduced muscle function in patients with osteoarthritis, *Scand J Rehab Med* 29 (1997) 213–221.
 - [10] N.M. Fisher, S.C. White, H.J. Yack, R.J. Smolinski, D.R. Pendergast, Muscle function and gait in patients with knee osteoarthritis before and after muscle rehabilitation, *Disability and Rehabilitation* 19 (1997) 47–55.
 - [11] G.K. Fitzgerald, S.R. Piva, J.J. Irrgang, F. Bouzubar, T.W. Starz, Quadriceps activation failure as a moderator of the relationship between quadriceps strength and physical function in individuals with knee osteoarthritis, *Arthritis & Rheumatism (Arthritis Care & Research)* 51 (2004) 40–48.
 - [12] J.J. Gerbrands, On the relationships between SVD, KLT and PCA, *Pattern Recognition* 14 (1981) 375–381.
 - [13] H.S. Gill, J.J. O'Connor, Heelstrike and the pathomechanics of osteoarthrosis: a simulation study, *Journal of Biomechanics* (2003).
 - [14] K.D. Hall, K.W. Hayes, J. Falconer, Differential strength decline in patients with osteoarthritis of the knee: revision of a hypothesis, *Arthritis Care & Research* 6 (1993) 89–96.
 - [15] R.S. Hinman, K.L. Bennell, B.R. Metcalf, K.M. Crossley, Temporal activity of vastus medialis obliquus and vastus lateralis in symptomatic knee osteoarthritis, *Am J Phys Med Rehabil* 81 (2002) 684–690.
 - [16] C.L. Hubley-Kozey, E. Smits, Quantifying synergist activation patterns during maximal plantarflexion using an orthogonal expansion approach, *Human Movement Science* 17 (1998) 347–365.
 - [17] C.L. Hubley-Kozey, M.J. Vezina, Differentiating temporal electromyographic waveforms between those with chronic low back pain and healthy controls, *Clinical Biomechanics* 17 (2002) 621–629.
 - [18] M.V. Hurley, Muscle dysfunction and effective rehabilitation of knee osteoarthritis: what we know and what we need to find out, *Arthritis and Rheumatism* 49 (2003) 444–452.
 - [19] M.V. Hurley, D.J. Newham, The influence of arthrogenous muscle ingibition on quadriceps rehabilitation of patients with early, unilateral osteoarthritic knees, *Br J Rheumatol* 32 (1993) 127–131.
 - [20] M.V. Hurley, D.L. Scott, Improvements in quadriceps sensorimotor function and disability of patients with knee osteoarthritis following a clinically practicable exercise regime, *Br J Rheumatol* 37 (1998) 1181–1187.
 - [21] Y.P. Ivanenko, R.E. Poppele, F. Lacquaniti, Five basic muscle activation patterns account for muscle activity during human locomotion, *J Physiol* 556 (2004) 267–282.
 - [22] G.S. Kasman, J.R. Cram, S.L. Wolf, Clinical application in surface electromyography: Chronic musculoskeletal pain, Aspen Publishers Inc., Gaithersburg, Maryland, 1998.
 - [23] K.R. Kaufman, C. Huges, B.F. Morrey, M. Morrey, K. An, Gait characteristics of patients with knee osteoarthritis, *Journal of Biomechanics* 34 (2001) 907–915.
 - [24] J.H. Kellgren, J.S. Lawrence, Radiological assessment of osteoarthrosis, *Ann Rheum Dis* 16 (1957) 494–501.
 - [25] E. Kellis, V. Baltzopoulos, The effects of normalization method on antagonistic activity patterns during eccentric and concentric isokinetic knee extension and flexion, *Journal of Electromyography and Kinesiology* 6 (1996) 235–245.
 - [26] F.P. Kendall, E.K. McCreary, P.G. Provance, *Muscles Testing and Function*, fourth ed., Williams and Wilkins, Baltimore, 1993.
 - [27] B. Leveau, G.B.J. Andersson, Output Forms: Data Analysis and Applications, in: G.L. Soderberg (Ed.), Selected topics in surface electromyography for use in the occupational setting: Expert perspectives, U.S. Department of Health and Human Services, 1992, pp. 69–102.
 - [28] M.D. Lewek, K.S. Rudolph, L. Snyder-Mackler, Control of frontal plane knee laxity during gait in patients with medial compartment knee osteoarthritis, *Osteoarthritis Cartilage* 12 (2004) 745–751.
 - [29] M.D. Lewek, K.S. Rudolph, L. Snyder-Mackler, Quadriceps femoris muscle weakness and activation failure in patients with symptomatic knee osteoarthritis, *Journal of Orthopaedic Research* 22 (2004) 110–115.
 - [30] E.A. Lingard, J.N. Katz, E.A. Wright, C.B. Sledge, Predicting the outcome of total knee arthroplasty, *J Bone Joint Surg Am* 86-A (2004) 2179–2186.
 - [31] R. Marks, J.S. Percy, J. Semple, S. Kumar, Comparison between the surface electromyogram of the quadriceps surrounding the knees of healthy women and the knees of women with osteoarthritis, *Clin Exp Rheumatol* 12 (1994) 11–15.
 - [32] G. Pap, A. Machner, F. Awiszus, Strength and voluntary activation of the quadriceps femoris muscle at different severities of osteoarthritic knee joint damage, *Journal of Orthopaedic Research* 22 (2004) 96–103.
 - [33] A.E. Patla, Some characteristics of EMG patterns during locomotion: Implications for the locomotor control process, *Journal of Motor Behavior* 17 (1985) 443–461.
 - [34] M.S. Redfern, Functional muscle: Effects on electromyographic output, in: G.L. Soderberg (Ed.), Selected Topics in Surface EMG for use in the Occupational Setting: Expert Perspectives, Morgantown National Institute for Occupational Health and Safety, 1992, pp. 104–120.
 - [35] M.J. Robon, K.L. Perell, M. Fang, E. Guerro, The relationship between ankle plantar flexion muscle moments and knee compression forces on subjects with and without pain, *Clinical Biomechanics* 15 (2000) 522–527.
 - [36] E.M. Roos, S. Toksvig-Larsen, Knee injury and Osteoarthritis Outcome Score (KOOS) - validation and comparison to the WOMAC in total knee replacement, *Health Qual Life Outcomes* 1 (2003) 17.
 - [37] L. Sharma, D.D. Dunlop, S. Cahue, J. Song, K.W. Hayes, Quadriceps strength and osteoarthritis progression in malaligned and lax knees, *Annals of Internal Medicine* 138 (2003) 613–619.
 - [38] R. Shiavi, H.J. Bugle, T.J. Limbird, Electromyographic gait assessment part 1: Adult EMG profiles and walking speed, *Journal of Rehabilitation Research and Development* 24 (1987) 13–23.
 - [39] R. Shiavi, N. Green, Ensemble averaging of locomotor electromyographic patterns for level surface walking, *Med Biol Eng and Computing* 21 (1983) 573–578.
 - [40] R. Shiavi, P. Griffin, Representing and clustering electromyographic gait patterns with multivariate techniques, *Med Biol Eng and Computing* 19 (1981) 606–611.

- [41] C. Slemenda, K.D. Brandt, D.K. Heilman, S.A. Mazzuca, E.M. Braunstein, B.P. Katz, F.D. Wolinsky, Quadriceps Weakness and Osteoarthritis of the knee, *Annals of Internal Medicine* 127 (1997) 97–104.
- [42] C. Slemenda, D.K. Heilman, K.D. Brandt, B.P. Katz, S.A. Mazzuca, E.M. Braunstein, D. Byrd, Reduced quadriceps strength relative to body weight: a risk factor for knee osteoarthritis in women? *Arthritis Rheum* 41 (1998) 1951–1959.
- [43] G.L. Soderberg, L.M. Knutson, A guide for use and interpretation of kinesiological electromyographic data, *Physical Therapy* 80 (2000) 485–498.
- [44] M.E. van-Baar, W.J. Assendelft, J. Dekker, R.A. Oostendorp, J.W. Bijlsma, Effectiveness of exercise therapy in patients with osteoarthritis of the hip or knee: a systematic review of randomized clinical trials, *Arthritis Rheum* 42 (1999) 1361–1369.
- [45] M.J. Vezina, C.L. Hubley-Kozey, A comparison of the electromyographic normalization procedures for abdominal and trunk extensors, *Proceedings of NACOB'98* 1998;401–402.
- [46] J.E. Ware, K.K. Snow, M. Kosinski, SF-36 Health Survey: Manual and Interpretation Guide, Quality Metric Inc., Lincoln, RI, 2000.
- [47] D.A. Winter, Biomechanical motor patterns in normal walking, *Journal of Motor Behavior* 15 (1983) 302–330.
- [48] D.A. Winter, *Biomechanics and Motor Control and Human Movement*, second ed., Wiley, New York, 1990.
- [49] D.A. Winter, EMG interpretation, in: S. Kumar, A. Mital (Eds.), *Electromyography in Ergonomics*, Taylor and Francis, London, 1996, pp. 109–125.
- [50] D.A. Winter, A.J. Fuglevand, S.E. Archer, Crosstalk in surface electromyography: theoretical and practical estimates, *Journal of Electromyography and Kinesiology* 4 (1994) 15–26.
- [51] D.A. Winter, H.J. Yack, EMG profiles during normal human walking: Stride to stride and intersubject variability, *Electromyography and Clinical Neurophysiology* 67 (1987) 402–411.
- [52] J.M. Winters, L. Stark, Estimated mechanical properties of synergistic muscles involved in movements of a variety of joints, *J Biomech* 21 (1988) 1027–1041.
- [53] M.E. Wootten, M.P. Kadaba, G.V.B. Cochran, Dynamic Electromyography. I. Numerical Representation using Principal Component Analysis, *J Orthop Res* 8 (1990) 247–258.
- [54] M.E. Wootten, M.P. Kadaba, V.B. Cochran, Dynamic Electromyography II. Normal Patterns During Gait, *J Orthop Res* 8 (1990) 259–265.
- [55] J.F. Yang, D.A. Winter, Surface EMG profiles during different walking cadences in humans, *Electroencephalogr Clin Neurophysiol* 60 (1985) 485–491.
- [56] J.H. Zar, *Biostatistical analysis*, third ed., Prentice Hall, New Jersey, 1996.
- [57] L.Q. Zhang, G. Wang, Dynamic and static control of the human knee joint in abduction-adduction, *Journal of Biomechanics* 34 (2001) 1107–1115.



Cheryl L. Hubley-Kozey received her bachelors' degree from the University of New Brunswick, her MSc from the University of Waterloo and her Ph.D. in Physiology and Biophysics from Dalhousie University. She is a professor in the School of Physiotherapy and is cross-appointed in the School of Biomedical Engineering and the Department of Physiology and Biophysics at Dalhousie University, Halifax, Canada. Since 1997 she has been studying alterations in neuromuscular function associated

with orthopaedic conditions using surface electromyographic waveforms. She is a member of the executive of the Canadian Society for Biomechanics.



Kevin J. Deluzio, PhD received his BSc, MSc, and PhD degrees from Queen's University. He is currently an associate professor in the School of Biomedical Engineering at Dalhousie University and is cross-appointed in the Department of Surgery, Faculty of Medicine. Dr. Deluzio is president of the Canadian Orthopaedic Research Society and is a member of the executive of the Canadian Society for Biomechanics. His research is focussed on the investigation of the biomechanical factors of knee osteoarthritis and its treatment.



Scott Landry received his B.Eng. in Mechanical Engineering from Dalhousie University in 2000 and his B.Sc. (Honours) in Biology from Acadia University in 1997. He is currently a Ph.D. student at Dalhousie University. His research interests are focused on lower limb mechanical and neuromuscular factors associated with anterior cruciate ligament injuries while cutting and moderate knee osteoarthritis during gait. He has presented his work at several national and

international venues.



Jennifer McNutt received her Bachelor of Science in Physiotherapy in 2001 from Dalhousie University. She is currently completing her Masters of Applied Science in Biomedical Engineering at Dalhousie University. In addition to her interest in neuromuscular control and strength in knee osteoarthritis, she is also involved with research in cardiovascular fitness post stroke working as a clinical therapist in a home based exercise study. She has served as a board member on the

Nova Scotia College of Physiotherapists and has been involved with the Nova Scotia Branch of the Canadian Physiotherapy Association. She is currently focusing her research interests in health promotion, injury prevention and health policy.



William D. Stanish, MD, FRCS(C), FACS, Diplomate of the American Academy of Orthopaedic Surgeons; Professor of Orthopaedic Surgery, Dalhousie University, Halifax, Nova Scotia, Canada. He is also Director of the Orthopaedic and Sport Medicine Clinic of Nova Scotia. Dr. Stanish trained in Orthopaedic Surgery in Stoke Mandeville Hospital, in the Oxford University group of hospitals, Aylesbury, England; Dalhousie University, Halifax, Nova Scotia; and Harvard

University, Boston, MA, USA. Dr. Stanish, as a clinical scientist, has fostered research in the area of physiology and biomechanics of ligaments and tendons. His research publications and book chapters by 2004 number 161. He is co-author of the book entitled "Tendinitis: Its Etiology and Treatment", published by Oxford University

Press, which is in its Second Edition. Furthermore, Dr. Stanish is co-editor of the Oxford Textbook of Sports Medicine, which is currently in its Second Edition. He is past Deputy Editor of The

Journal of Bone and Joint Surgery. He has been on the Editorial Board of several publications, including the American Journal of Sports Medicine.