

Characteristics of Diffuse Muscular Coactivation (DMC) in

Persons with Fibromyalgia - Part 2

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This study examined the electrical characteristics (Root Mean Square - RMS and median frequency) of Diffuse Muscular Coactivation (DMC) associated with the tender points of fibromyalgia. DMC is defined as an increase from resting levels (tonus) in the electrical activity of any muscle during a movement which does not involve that muscle and is not part of the agonist - antagonist unit. The results show an increase in RMS in fibromyalgia sufferers as compared to controls. Coactivation was stronger proximal to the neck and decreased in intensity as the area recorded moved distally. Median frequency changed over time but not significantly between groups. Possible neurological mechanisms are discussed.

Keywords: fibromyalgia, muscle activity, coactivation, electromyography, root mean square, median frequency

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1. Introduction

Muscles as a possible source of pain in fibromyalgia have been studied extensively. In general, controlled studies of muscle activity have failed to implicate muscular pathophysiology as a key factor in fibromyalgia [1]. Zidar et al [2] indicate there is no important loss of motor units or conspicuous muscle fiber degeneration and go on to suggest that muscle tension cannot be a prominent pathogenic factor. Various authors [3,4] suggest that high muscle tension, anxiety, and paraspinal muscle fatigue are not factors associated with the pain of fibromyalgia. A study by Durette [5] did not demonstrate any evidence of focal motor activity in tender points or of ongoing denervation. Jacobsen [6] supported this opinion and suggested that the pain may be due to inhibition of contraction due to spinal or supraspinal mechanisms. Lindh [7] similarly expressed the view that an impaired control mechanism at the supraspinal level was the source of the muscle pain. Several studies have examined the role of muscle strength in fibromyalgia. Norregaard et al [8] suggested that fibromyalgia pain may be due to a reduction of muscle strength per unit area of about 35% as compared to controls. However, this study was limited due to a lack of control for physical activity and for neuroendocrine factors. Two studies [9,10] using surface electromyographic (sEMG) techniques showed a relationship between sEMG levels and reported pain. Results from the study conducted by Sarnoch [9] indicate a reduction in the sEMG level correlate with a decrease in reported pain,

while Elert et al [10] suggest that the fibromyalgia sufferers had an inability to relax the muscle throughout their bodies between tasks.

Surface electromyography offers the scientist a chance to study muscle activity and nerve fiber activity (Adrian and Bronk, 1929 as cited in [11]) in a functional manner. The ability to measure amplitude, frequency and muscle interactions can shed new light on dysfunctions such as fibromyalgia. Amplitude as measured in microvolts is thought to reflect the measurement of force over time [11]. It is measured in a number of different ways but Root Mean Square (RMS) is the preferred method due to greater features of accuracy. Frequency as measured in cycles per second or Hertz (Hz) is thought to reflect the number of times cells polarize and depolarize producing electrical energy. Two most commonly used measures of this are the median frequency and the mean frequency. Median frequency is thought to more accurately reflect the lower end of the physiological frequencies (i.e. 20 Hz).

Multiple channel sEMG placements allow the scientist a chance to examine the onset of firing, the timing of the interactions amongst muscles and examine for the presence of inappropriate muscle activity (muscles working when they should not). The authors Donaldson [12] and Mueller [13] studied muscle interactions and reported results suggesting the muscle activity of fibromyalgia sufferers differ from that of myofascial pain (MPS) patients. In particular the presence of DMC in

fibromyalgia sufferers, at a ratio of 1.75 compared to 1.0 for controls, is reported in Part 1 of this study.

The purpose of this study was to explore the amplitude and frequency of muscles, showing DMC from individuals suffering from fibromyalgia as compared to controls. It was hypothesized that the muscle activity of fibromyalgia sufferers will differ in RMS values and median frequency values when compared to controls.

2. Method

Subjects

The demographics of the subjects are reported in Part 1 of this study. Approximately four months after the completion of Part 1, 16 of the original 40 subjects were asked to volunteer for a second sEMG study (before they received treatment). Of these 16 subjects, 12 agreed to participate in the second study and 4 refused. During the data collection, one individual became ill and her results were omitted from the study, as it was not known how her condition would affect the results. The same 6 individuals, who had volunteered as controls for Part 1, volunteered as controls for Part 2.

Measures

Prior to the evaluation, the manufacturer calibrated the instrument for accuracy of measurement. Muscle activity from 16 of the 18 ACR tender points (see Table 1

for locations) was studied in the following manner: electrodes were placed on the skin, 2 cm center-to-center, parallel to the direction of the fibers of the muscles involved with the tender point. For example, data was collected at a point 2 cm distal to the epicondyles in order to collect data about the lateral epicondyle tender point. All sites were cleaned with rubbing alcohol and the electrodes assessed for artifact (see manual of the Norodyn™ 8000¹ equipment for details of this) before the testing. Any electrode showing noise above .2 microvolts was replaced and the preparation repeated. Electrode locations are outlined in Table 1 below.

Insert Table 1 here

The subjects lay supine on a massage table, fully supported with a pillow under the knees and the arms at their sides. The sEMG system was utilized to collect data on 8 channels at a sampling rate of 640 Hz (per channel), bandpass filter of 15 – 400 Hz. Channels 1 and 2 were utilized in all trials to monitor for the appropriate movement from the cervical paraspinal muscles at C5 – 7. The other 6 channels monitored muscle activity from 3 or 2 pairs (bilaterally) of muscles. The 14 muscles were grouped as follows: a) upper trapezius (UTP), pectoralis major (PMS), greater trochanter (TFL), b) occiput (C1), lateral epicondyle (EXT), gluteus medius (GMD), and c) vastus medialis (VM), and supraspinatus (SUP). Each group of muscles was monitored during a) resting levels, b) flexion of the

head just up off the table, c) rotation of the head to the left (average 58°), and d) rotation of the head to the right (average 59°). Each movement was repeated 3 times with data captured before movement (resting levels), during the first movement (labeled repetition 1) and the third movement (labeled repetition 3). The degree of rotation was held constant for all rotations for each subject, but varied between subjects. A one-minute rest period was given between the different movements to minimize fatigue. The order of data collection for the different muscle groups was randomized in order to reduce order effect. All data was stored to disk for future data analysis.

During the collection of data in group b (C1, EXT, GMD) the subjects were asked to plantar flex their feet and toes to examine for the possibility that the pattern of DMC could be reproduced by foot movement moving distally to proximally.

Data Analysis - sEMG

Two sets of scores were studied: a) the RMS values in microvolts and b) the median frequency measured in Hz. The sEMG equipment utilized allows for simultaneous analysis of both sets of data by allowing the operator to place a window over the data to be analyzed. For purposes of this study a window with a time sample of .91 seconds was used. First the data were visually inspected for artifact, which, if found, was deleted. The window was placed over the resting levels data at a site at approximately 2 seconds. Because it was impossible to

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delete heartbeat, each set of data was required to contain one heartbeat. The window automatically generated the RMS values, the peak microvolt values, and median and mean frequency for that .91 seconds. The data were collected for resting levels and then for the first and third repetitions of each movement as outlined above.

First the average resting levels RMS values were established for all three movements and all the muscles. This was summed and averaged and then compared between groups for total (sum of left and right sides) resting levels activity using a One Way ANOVA.

The RMS values for each muscle for the 1st and 3rd movements were then compared to the resting level value with the difference calculated and converted to a percentage difference with the resting levels as the divisor. The percent changes and actual microvolt values were calculated for the left and right sides for all three movements; resting levels, movement 1, and movement 3 and for the three conditions (flexion, rotation left and rotation right). The left and right-sided values were then averaged, producing a total score.

A similar procedure was followed to calculate changes in median frequency. First the median frequency from the resting level (.91 seconds) was obtained, followed by similar time samples for the 1st and 3rd movements. The difference between the resting level and each movement was then established. Due to

issues of measurement [11] the results were not averaged for all the muscles and are reported individually.

3. Results

RMS Values

The results for the resting levels activity show an average score of 5.33 microvolts (SD = .89) for the control group and a score of 4.81 microvolts (SD = .83) for the fibromyalgia group (see Table 2). The One Way ANOVA for between group differences was not significant ($F = 1.460$, $p = .246$).

Insert Table 2 here

The average total change in microvolts from resting levels activity during movements 1 and 3 for all conditions are reported in Table 2. As may be seen, the fibromyalgia average activity increased from 4.77 microvolts to 8.92 for movement 1 and to 9.55 for movement 3 as compared to the controls score of 5.32 microvolts (resting levels) to 6.95 and 7.19 for movements 2 and 3 respectively. A Repeated Measures ANOVA showed no significant differences between groups ($F = 2.145$, $p = .154$) although change over time approached significance ($F = 3.014$, $p = .064$). It is expected that with a larger sample size significant results would be obtained.

The fibromyalgia sufferer's average level of muscle activity increased by 80% as compared to the control's increase of 21.0%, almost four times higher. The increase in average activity was higher on the right side (87.0% - fibromyalgia, 26.6% - controls) than on the left (73.6% vs. 14.9% - respectively).

Rotation showed increased activity to the ipsilateral side, although this was more pronounced for rotation left then to the right. In all cases this pattern was consistent for controls as well. In one case this pattern was pronounced, with the controls (during rotation right) showing an increased level of activity (74.9%), which was not evident during the other movements (rotation left – 36.5% and flexion – 26.6%). No explanation for this is known at the present time. Table 3 summarizes the actual and percent changes for the groups and condition.

Insert Table 3 here

Analysis of the individual sites shows a tendency for the muscles of the upper torso to have higher average percent change for the three movements decreasing as the site moves distally from the neck. This is similar for both groups. This pattern is illustrated in Table 4.

Insert Table 4 here

Median Frequency

A Repeated Measures ANOVA was performed on each muscle and showed no significant changes between groups over time ($F = .237, p < .789$) for any of the measures investigated. However, all muscles showed a significant ($p < .05$) change over time between resting levels and movements 1 and 3. Examination of the individual muscles showed similar results for all sites except C1. This change at C1 probably reflects some increased activity due to involvement with the movement. Table 5 shows the results of the median frequency for the various muscles.

Insert Table 5 here

Toe Pointing

No change in RMS values or median frequency for any of the muscles was observed in any of the subjects except one.

Post Hoc Analysis

In view of the possibility of anxiety causing the increase in the RMS levels a post hoc analysis was conducted comparing the results of the SCL 90 R Global Severity Index (from Part 1 of the study) to the RMS changes. These results were not significant.

4. Discussion

The persons suffering from fibromyalgia and the controls both demonstrated an increase in DMC distant from the origin of the movement. This was evident in muscles associated with the ACR defined tender points. While the fibromyalgia sufferers showed a higher incidence of positive findings, statistically significant between- group differences were not found. This is probably due to the small sample size and particularly due to problems of homogeneity of variance in the control group data.

The structural unit of muscle contraction is the muscle cell or muscle fiber, which is innervated by terminal branches of one nerve fiber. This nerve fiber is innervated by the axons whose cell bodies are in the anterior horn of the spinal grey-matter [11]. This is known as a motor unit, which is the functional unit of activation of the striated muscles. Motor units with a small number of muscle fibers are innervated by smaller alpha motoneurons and are excited earlier during a contraction, while larger motoneurons are innervated by larger motoneurons and become activated at progressively higher levels of force [11]. The rate of firing of the muscle is a function of a) force, b) type of muscle, c) type of contraction and d) length of time of contraction. [11]. Little research has been conducted on the firing rates of abnormal muscles, with Andreassen and Rosenfalck (Andreassen and Rosenfalck, 1980 as cited in [11]) reporting the mean firing rate of spastic muscle as reduced when compared to motor units from the contralateral nonaffected muscle. Kranz (Kranz 1981 as cited in [11]) showed a similar pattern for muscles with clinically mild lesions of the central

nervous system, while Holonen (Holonen, 1981 as cited in [11]) reported the average firing rate is increased in myopathic disorders. It is important to note for purposes of this paper the above noted studies of motor recruitment have primarily involved sustained contractions and may not be appropriate here.

Lenz et al [14] suggested that the thalamus contributes to abnormal movements occurring in patients with dystonia. In particular they showed increased simultaneous cocontractions of antagonist muscles while recording thalamic neuronal signals from the ventral intermediate (VIM) and pallidal relay nucleus. The same patterns were not present in controls. The authors go on to indicate that micro stimulation of the VIM produced simultaneous activation of multiple forearm muscles.

Jacobs and Fornal [15] studied the relationship of serotonin to motor activity. They suggest the activation of brain serotonergic neurons facilitates motor output and simultaneously suppresses sensory information processing. It is hypothesized serotonergic neurons play an auxiliary role in coordinating appropriate autonomic and neuroendocrine outputs to the ongoing tonic and repetitive motor activity.

Whatmore and Kohli (Whatmore and Kohli, 1968 as cited in [11]) defined ponesis as the production of nerve impulses in pathways extending from motor and premotor cortical neurons through pyramidal and extrapyramidal tracts to the

peripheral musculature. These impulses participate in all voluntary motor activity. These impulses are associated and/or connected with minute movements usually not observable to the naked eye. For example minute eye efforts participate in the production of visual images (Jacobson, 1930, 1938, Lorens and Darrow, 1962 as cited in [16]). Dysponesis is therefore defined as “a physiopathologic state made up of errors in energy expenditure within the nervous system.” (pg. 380 [16]). The authors go on to state “the detrimental influence of these misdirected efforts results from the facts that action potentials (or nerve impulses) constituting effort not only follow the well-known pathways from motor and pre-motor cortex to anterior horn cells, and thus to muscle fibers, but also feed signals (by way of side branches and feedback mechanisms) into the reticular activating system, the hypothalamus, the limbic system, and the neo-cortex, thus producing wide spread effects. These signals exert excitatory and inhibitory influences that are inappropriate to the immediate objectives of the organism. The result is an interference with many aspects of nervous system function including the organism’s emotional reactivity, its ideation and the regulation of various organs of the body.” (pg. 383 [16]).

The DMC demonstrated the following characteristics: a) not all muscles coactivated all the time, with the muscles nearer the neck more active than those distal, b) the amplitude of the coactivation followed a similar pattern of a greater increase proximal to the neck than those muscles distal, c) the amplitude and percent change from the resting levels was much greater for the fibromyalgia

sufferers than controls, d) the median frequency changed in an upward direction in a similar manner for both groups, e) the direction of activation moves from the head towards the feet and not vice versa, and e) when several muscles coactivate the onset of timing is identical for all involved.

Examination of the RMS data and the median frequency values revealed some interesting trends. The increase in RMS values varied in strength from a 20% increase to an approximate 800% increase as the location moved proximally towards the neck. It can be argued that this increase refers to a stretching of the skin, or some other similar artifact. However, this increase in activity was almost four times higher in the fibromyalgia group than the controls, rendering this explanation meaningless. A similar argument and counterargument can be made concerning the presence of trigger points affecting the results.

No examination of the antagonistic muscles to those monitored was conducted. This needs to be completed for the presence of DMC in the antagonists would be consistent with the definition of stiffness (mentioned above) possibly accounting for this component of this dysfunction.

The increase in the RMS values of the muscles would suggest an increase in the amount of force generated in that muscle. Yet no observable movement occurred. If this phenomenon occurs every time a person moves their head it is possible to see how fatigue and associated muscle pain would occur over time.

An improvement in muscle tonus over time would counteract this, reducing the pain, but severe exercise would fatigue the muscles too rapidly increasing the pain.

The increase in the median frequency is puzzling, as a decrease in level of activity was expected. During sustained constant -force contractions the median frequency shows an immediate decrease in frequency probably reflecting a decrease in the firing rates of the single motor unit [11]. However, it is not known if the same principle applies during coactivation, as no research was found examining this topic. Perhaps if the DMC had been sustained for a longer period of time a downward shift in frequency may have been seen. This needs to be investigated.

As in part 1 the resting levels of activity for both groups showed no significant differences supporting the findings from Zidar et al [2]. Thus it is hard to attribute the presence of the generalized pain of fibromyalgia to this factor. Examination of the data shows the fibromyalgia resting levels in both parts of the study show a slight variation of levels (Part 1 – 3.4 microvolts, Part 2 – 4.8 microvolts), while the control group had much wider findings (Part 1 – 2.99 microvolts, Part 2 – 5.33 microvolts). While some of the increase may be explained by the use of different equipment the greater change in the control group is difficult to explain. Post hoc discussion with the control group indicated they had not done anything out of the ordinary before the evaluations and their activity levels were consistent between

assessments. Examination of the data for outliers showed no presence of any. The reason for this greater difference is not presently known.

5. Conclusion

This study was pilot in design intending to explore the nature of muscle activity in fibromyalgia sufferers. This study notes the presence of unusual muscle activity occurring in all fibromyalgia sufferers in which movement of the head causes increased electrical activity of other muscles throughout the body. An increase in RMS activity is seen, as is an increase in median frequency. Labeled as Diffuse Muscular Coactivation (DMC) by the authors, it appears this phenomena involves the central nervous system although exactly how is not known. In 1965 Janda and Stara (as cited in [11]) reported the presence of muscle activity similar to what is being reported here. Other authors report similar findings of increased neuromuscular activity due to illness, stress, fatigue and aging. Perhaps this appearance of the DMC reflects a breakdown of the motor control systems, which exert an inhibitory control over motor activity leading to the development of the DMC. The presence of DMC establishes a possible link between the musculoskeletal system, other bodily systems and their dysfunctions pointing to directions for further research of this complex phenomenon.

Acknowledgements:

This portion of the study was funded by the British Columbia Fibromyalgia Society.

References:

- [1] R. Simms, Is there muscle pathology in fibromyalgia syndrome?, *Rheumatic Diseases Clinics of North America* 22(2), (1996), 245-266.

- [2] J. Zidar, E. Bachman, A. Bengtsson, K. Henriksson, Quantitative EMG and muscle tension in painful muscles in fibromyalgia, *Pain* 40(3), (1990), 249-254.

- [3] M. Stokes, C. Colter, A. Klestov, R. Cooper, Normal paraspinal muscle electromyographic fatigue characteristics in patients with primary fibromyalgia, *British Journal of Rheumatology* 32(8), (1993), 711-716.

- [4] S. Svebak, R. Anjia, S. Karstad, Task-induced electromyographic activation in fibromyalgia subjects and controls, *Scandinavian Journal of Rheumatology* 22(3), (1993), 124-130.

- [5] M. Durette, A. Rodriguez, J. Agre, J. Silverman, Needle electromyographic evaluation of patients with myofascial or fibromyalgic pain, *American Journal of Physical Medicine and Rehabilitation* 70(3), (1991), 154-156.

- [6] S. Jacobsen, S., Physical biodynamics and performance capacities of muscle in patients with fibromyalgia syndrome, *Zeitschrift fur Rheumatologie* 57Suppl(2), (1998), 43-6.
- [7] M. Lindh, L. Johansson, M. Hedberg, G. Grimby, Studies on maximal voluntary muscle contraction in patients with fibromyalgia, *Archives of Physical Medicine and Rehabilitation* 75(11), (1994), 1217-1222.
- [8] J. Norregaard, P. Bulow, B. Danneskiold-Samsoe, Muscle strength, voluntary activation, twitch properties, and endurance in patients with fibromyalgia, *Journal of Neurology, Neurosurgery and Psychiatry* 57(9), (1994), 1106-1111.
- [9] H. Sarnoch, F. Adler, O. Scholz, Relevance of muscular sensitivity, muscular activity, and cognitive variables for pain reduction associated with EMG biofeedback in fibromyalgia, *Perceptual and Motor Skills* 84(3), 1997, 1043-1050.
- [10] J. Elert, S. Rantapaa-Dahlqvist, K. Henriksson-Larsen, R. Lorentzon, B. Gerdle,. Muscle performance, electromyography and fibre type composition in fibromyalgia and work-related myalgia, *Scandinavian Journal of Rheumatology* 21(1), (1992), 28-34.

- [11] J. Basmajian, C. De Luca, *Muscles Alive (5th Ed) Their Functions Revealed by Electromyography*, Williams & Wilkins: New York, NY, 1985.
- [12] S. Donaldson, G. Sella, H. Mueller, Fibromyalgia: A retrospective study of 252 consecutive referrals, *Canadian Journal of Clinical Medicine* 5(6), (1998), 116-127.
- [13] H. Mueller, S. Donaldson, D. Nelson, M. Layman, Treatment of fibromyalgia incorporating EEG-driven stimulation: a clinical outcomes study, *Journal of Clinical Psychology* 57(7), (2001), 933-954.
- [14] F. Lenz, C. Jaeger, M. Seike, Y. Lin, S. Reich, M. LeLong, J. Vitek, Thalamic single neuron activity in patients with dystonia: dystonia-related activity and somatic sensory reorganization, *Journal of Neurophysiology* 82(5), (1999), 2372-2392.
- [15] B. Jacobs, Fornal, C., Serotonin and motor activity, *Current Opinion in Neurobiology* 7, (1997), 830-825.
- [16] J. Basmajian, Facts versus Myths in EMG Biofeedback, in: E. Peper, S. Ancoli, M. Quinn, M., *Mind/Body Integration*, (eds), Plenum Press: New York, NY, 1979, pp. 377-378.

Table 1
Muscles Examined and Electrode Locations

<i>Tender Point</i>	<i>Electrode Locations</i>
Occiput (C1)	Bilaterally over the upper trapezius at C3-5
Supraspinatus (SUP)	Bilaterally at the origins above the medial border of the scapular spine
Gluteus Medius (GMD)	Bilaterally in upper outer quadrants of buttocks over anterior fold of muscle
Greater Trochanter (TFL)	Bilaterally posterior to the trochanteric prominence
Vastus Medialis (VM)	Bilaterally at the fat pad proximal to the joint line
Pectoralis Major (PMS)	Bilaterally upper surfaces just lateral to the costochondral junctions
Lateral Epicondyle (EXT)	Bilaterally 2 cms distal to the epicondyles
Upper Trapezius (UTP)	Bilaterally at the mid point of the upper border

Table 2
Average Scores of sEMG in Microvolts by Group for Three Conditions

Group		Grand Total Resting levels	Grand Total Movement 1	Grand Total Movement 2
Control	Mean	5.3264	6.9514	7.1944
	N	6	6	6
	Std.D	.89	.8547	1.4299
Fibro	Mean	4.81	8.9242	9.5597
	N	11	11	11
	Std.D	.83	3.6802	3.4549

Table 3
Actual and Percent Change by Condition

	B	Flexion				Rotation Left				Rotation Right			
		M1	%	M3	%	M1	%	M3	%	M1	%	M3	%
TF	4.81	8.66	80.0	9.10	89.2	9.03	87.7	9.79	103.5	9.5	97.5	9.97	107.3
TC	5.33	6.45	21.0	6.73	26.3	6.8	27.6	7.33	37.5	7.99	49.9	7.64	43.3
LF	4.78	8.30	73.6	8.76	83.3	10.74	124.7	10.08	110.9	9.11	90.6	9.54	99.6
LC	5.04	5.79	14.9	6.33	26.0	6.88	36.5	7.58	50.4	6.17	22.4	6.33	25.6
RF	4.83	9.03	87.0	9.43	95.2	7.32	51.5	9.50	96.7	9.88	104.6	10.41	115.5
RC	5.61	7.10	26.6	7.13	27.1	6.71	19.6	7.08	26.2	9.81	74.9	8.95	59.5

Legend:

All values (excluding %) are in microvolts (RMS) 0.91sec.

B=baseline

T=grand total for entire body

L=left side of the body

Right=right side of the body

F=fibros

C=controls

M1=first movement

M3=third movement

%=change from baseline to M1 and change from baseline to M3

Table 4
Average % Change in Microvolts for Individual Muscles

Movement						
	Rotation Right		Rotation Left		Flexion	
	Left	Right	Left	Right	Left	Right
UTP	50	331.6	168.6	243.3	78.3	383.8
PMS	14.8	26.8	11.9	7.7	64.5	111.5
TFL	17.1	9	24.7	17.9	37.3	16.9
C1	499.3	601.7	771.2	636.0	183.2	333.3
EXT	37	6.7	62.8	8.4	10.8	0
GMD	45.2	46.1	14.1	28	15.4	17
VM	22.5	25.3	20.6	35.5	8.3	8.3
SUP	80.2	83.3	155.2	55.2	103.1	35.1

Table 5
Change in Median Frequency in Muscles by Condition

Muscle		Change in Baseline Median Frequency	Change in Movement 1 Median Frequency	Change in Movement 2 Median Frequency
L UTP	Mean N Std.D	18.2400 6 1.2213	22.8417 6 3.5617	23.0983 6 2.3663
R UTP	Mean N Std.D	23.5200 6 2.3537	30.7850 6 5.1056	33.2133 6 4.2635
L EXT	Mean N Std.D	22.1333 6 .7988	23.1617 6 3.4930	23.6833 6 6.2812
R EXT	Mean N Std.D	20.1700 6 .5968	21.0450 6 1.8716	21.1833 6 3.5917
L GMD	Mean N Std.D	18.0617 6 .6143	18.8033 6 1.1575	19.2017 6 1.2601
R GMD	Mean N Std.D	19.5083 6 .8899	18.8000 6 1.0500	19.2800 6 .7321
L VM	Mean N Std.D	20.3333 6 .8650	20.5917 6 1.0925	21.0800 6 1.3956
R VM	Mean N Std.D	19.2317 6 1.8764	21.2267 6 1.6938	20.2167 6 1.1549
L SUP	Mean N Std.D	18.8317 6 .3449	21.6183 6 1.9969	24.4400 6 4.3512
R SUP	Mean N Std.D	19.6950 6 .8273	23.4667 6 2.5442	25.1567 6 3.0380
L PMS	Mean N Std.D	19.2617 6 .7943	22.2767 6 5.2497	24.4467 6 4.9943
R PMS	Mean N Std.D	18.6033 6 .4822	23.5033 6 3.6093	23.1450 6 2.5382
L TFL	Mean N Std.D	19.4800 6 1.5030	21.3167 6 1.6947	20.0933 6 1.8062

R TFL	Mean N Std.D	18.2950 6 .9669	18.2000 6 1.4048	17.9317 6 1.5718
L C1	Mean N Std.D	28.2300 6 6.7492	38.6450 6 16.4443	37.0067 6 10.0593
R C1	Mean N Std.D	26.3167 6 3.1462	44.1600 6 19.8810	39.3267 6 12.5974
Total	Mean N Std.D	20.5420 108 3.3326	23.9305 108 9.1264	24.1231 108 7.4297

Legend:

See Table 1 for muscle locations