

Electromyographic analysis and strength of the wrist extensor muscle group during induced ischemia

Análise eletromiográfica e força do grupo muscular extensor do punho durante isquemia induzida

Bandeira CCA, Berni KCS, Rodrigues-Bigaton D

Abstract

Objective: To analyze the effect of induced ischemia on the parameters of electromyographic signals and the strength of the wrist extensor muscle group (WEMG) in healthy women. **Methods:** Thirteen right-handed sedentary subjects aged 23.38 ± 2.32 years old, with body mass index (BMI) of $20.68 \pm 1.87 \text{ kg/m}^2$, took part. To determine WEMG strength, three maximal voluntary isometric contractions (MVIC) were performed using a load cell for 15 seconds, with 2 minutes intervals between contractions. The entire procedure was repeated on three nonconsecutive days. Ischemia was induced for 5 minutes using a sphygmomanometer placed on the dominant arm and inflated until blood flow was absent, as confirmed by Doppler ultrasound. The EMG1000 module (Lynx[®]) was used with differential surface electrodes (Lynx[®]) to record the electromyographic signal of the WEMG. Three MVIC were recorded for 15 seconds, with 30 seconds intervals between them, under the following conditions: pre-ischemia, ischemia, immediate post-ischemia (post-1) and later post-ischemia (post-2: 10 minutes after the onset of ischemia). The MATLAB 6.5.1 software was used to analyze the parameters for the electromyographic signal, the root mean square (RMS) and the median frequency of the signal power spectrum. For statistical analysis, two-way ANOVA and the Friedman test were used. **Results:** Ischemia caused a significant reduction ($p < 0.05$) in WEMG strength. However, there were no significant changes in the RMS electromyographic parameters ($p = 0.05$) or the median frequency of the signal power spectrum ($p = 0.09$). **Conclusion:** Induced ischemia caused WEMG fatigue in relation to muscle strength production. However, it did not cause electromyographic fatigue in the evaluated muscle group.

Key words: electromyography; strength; ischemia.

Resumo

Objetivo: Avaliar o efeito da isquemia induzida sobre os parâmetros do sinal eletromiográfico e a força do grupo muscular extensor do punho (GMEP) em mulheres saudáveis. **Métodos:** Participaram 13 voluntárias, destros, sedentárias, com idade de $23,38 \pm 2,32$ anos e índice de massa corporal (IMC) de $20,68 \pm 1,87 \text{ kg/m}^2$. Para determinar a força do GMEP, foram realizadas 3 contrações isométricas voluntárias máximas (CIVM), utilizando-se uma célula de carga por 15 segundos, com intervalos de 2 minutos entre cada contração, sendo todo procedimento repetido por 3 dias não consecutivos. A isquemia foi realizada por 5 minutos, utilizando um esfigmomanômetro posicionado no braço dominante e inflado até a ausência do fluxo sanguíneo, confirmada pelo ultrassom Doppler. Para coleta do sinal eletromiográfico do GMEP, utilizou-se o equipamento EMG1000 (Lynx[®]) com eletrodo de superfície diferencial (Lynx[®]). Foram coletadas 3 CIVM por 15 segundos, com intervalo de 30 segundos entre elas, nas situações de pré-isquemia; isquemia; pós-isquemia imediata (pós-1) e pós-isquemia tardia (pós-2 - após 10 minutos do início da isquemia). Para análise dos parâmetros do sinal eletromiográfico, root mean square (RMS), e frequência mediana do espectro de potência do sinal foi utilizado o software MATLAB 6.5.1. Para análise estatística, foram utilizados os testes de *Friedman* e ANOVA *two-way*. **Resultados:** A isquemia promoveu redução significativa ($p < 0,05$) da força do GMEP. Entretanto, não provocou alterações significativas nos parâmetros eletromiográficos RMS ($p = 0,05$) e frequência mediana do espectro de potência do sinal ($p = 0,09$). **Conclusão:** A isquemia induzida promoveu fadiga do GMEP quando relacionada à produção da força muscular. Porém, não provocou fadiga eletromiográfica do grupo muscular avaliado.

Palavras-chave: eletromiografia; força; isquemia.

Received: 18/02/2008 – Revised: 14/05/2008 – Accepted: 26/09/2008

Introduction : : : .

It is common knowledge that, under ischemic conditions, the absence of adequate blood flow leads to the diminishment of muscle strength production¹. In this context, the sustained repetitive activities of the upper limbs, which are present in occupational activities, can cause a local reduction in blood flow and tissue oxygenation, thus provoking fatigue, pain, and functional deficit². According to Murphy et al.³, the wrist extensor muscles, particularly the extensor carpi radialis brevis, are the most commonly affected in prolonged and/or repetitive static activities.

Reduced blood flow⁴ and muscle oxygenation⁵ are associated with fatigue. Murphy et al.⁶ evaluated the effect of ischemia on extensor carpi radialis fatigue in 8 healthy individuals and found that a 3 to 5-minute ischemia led to muscle fatigue. In the aforementioned study, the authors evaluated muscle fatigue through the reduction in strength.

Muscle fatigue, whether produced by ischemia or not, can also be evaluated by means of surface electromyography (EMGs), as it is a non-invasive method of muscle function evaluation that detects all electrical potentials from the active motor units by means of electrodes placed on the skin⁷.

When assessed by means of EMGs, muscle fatigue is also known as electromyographic fatigue and, by definition, such a condition occurs when there is an increase in the amplitude of the electromyographic signal (observed through the *root mean square (RMS)* values, suggesting an additional motor recruitment), concomitant to a change in the power spectrum of the electromyographic signal toward the lower frequencies (observed through the median frequency values due to the reduction in the discharge frequency of the motor units)⁸.

Recent studies⁹ have used EMGs to evaluate muscle activity in situations of induced ischemia and reported a reduction in amplitude as well as a change in the power spectrum of the electromyographic signal toward the lower frequencies. This is caused by a pH diminishment and by a change in the electrolytic balance (imbalance of the Na⁺/K⁺ pump) along the muscle fiber membrane, a fact that alters the conduction velocity and the discharge frequency of the motor unit and may cause fatigue or muscle strength reduction⁶.

However, the behavior of electromyographic parameters in an ischemic condition has yet to be well defined in the literature because some studies show changes in the parameters of the electromyographic signals⁹, whereas others do not¹⁰. Another fact to be taken into account is that some studies^{4,5} demonstrate that ischemia is related to muscle fatigue, which, when evaluated through electromyography, promotes an increase in electromyographic signal amplitude. This was not observed by Chung, Callahan and Kent-Braun⁹.

Based on the aforementioned studies, the hypothesis of the present study is that induced ischemia promotes the fatigue of the wrist extensor muscle group in healthy women. This condition was observed through the reduction in strength production levels as well as through the increase in the amplitude and the change in the power spectrum of the electromyographic signal toward the lower frequencies, thus characterizing electromyographic fatigue.

Understanding muscle behavior under ischemic conditions, such as occupational activities, allows the development of better methods of evaluation and treatment of the dysfunctions characterized by ischemia, which is why this study was carried out. Therefore, the aim of this study was to analyze the parameters of the electromyographic signal and the strength of the wrist extensor muscle group (WEMG) during induced ischemia in healthy women.

Methods : : : .

The power of the sample was evaluated based on the RMS electromyographic parameter, obtained in a pilot study with 6 participants. The minimal size of the sample (n=9) was determined by the *t* test for paired samples (mean of the difference between the groups 17.86; standard deviation of the difference, 14.26; power of 0.95, $\alpha=0.05$, and unilateral test), in which the RMS parameter obtained in the pre-ischemic and ischemic conditions was used. The calculations were processed by the BioEstat 4.0.

Fourteen participants were selected for this study (23.38±2.32 years old; 53.75±7.56kg; 1.61±0.06m, BMI 20.68±1.87kg/m²). They were healthy, sedentary, according to the International Physical Activity Questionnaire-IPAQ¹¹, and without orthopedic, neurological, or upper-limb vascular dysfunctions. We opted to evaluate female individuals only, as several studies suggest that women have a higher resistance to fatigue than men in submaximal¹² and maximal¹³ isometric contractions. Women under 20 and over 30 were excluded from the study, as well as those who were left-handed; who had upper-limb dysfunctions; a BMI under 18.5 and over 25; and those not classified as sedentary, according to the IPAQ. All participants signed a consent form, and the research was carried out according to Resolution 196/96 of the National Health Council and approved by the Research Ethics Committee of Universidade Metodista de Piracicaba, under protocol n° 83/2006.

In order to determine the real strength production of the WEMG, a muscle strength baseline was established prior to data collection, for three nonconsecutive days, during which we obtained the mean of the maximal voluntary isometric

contraction value (MVIC), employed as a reference value for strength production at the time of electromyographic signal collection.

The strength measurement (kgf) of the WEMG was taken by means of a load cell (model MM-100, Kratos®, São Paulo, SP, Brazil). The load cell was placed perpendicularly, with one of the ends attached to the participant's hand by means of a metal chain and a leather strap, while the other end was fixed to the floor, making it possible to adjust the length of the chain for each participant (Figure 1D and 1E). The participant was seated with shoulders in a neutral position, elbow at 90°, in the prone position, forearm resting against a support, fingers flexed. This same position was used later to collect the electromyographic signal (Figure 1).

The participant performed 3 MVICs for 15 seconds, with a 2-minute interval between the contractions at each baseline. Throughout this process, the participant received verbal encouragement and visual feedback, in which the participant observed a line in the computer monitor and was requested to raise this line – representing strength – as high as possible. The data was recorded after the stabilization of the muscle strength.

The ischemia was induced for 5 minutes, by using a *Pressure N/C* sphygmomanometer placed on the dominant arm (Figure 1A) and inflated until the blood flow in the brachial artery was absent, confirmed by the Doppler ultrasound (Nicolet Vascular Versalab®), with a 8 MHz transducer. To avoid any irreversible circulatory changes, whether functional, metabolic or muscular, the maximal time-length of ischemia was kept under 2 hours¹⁴.

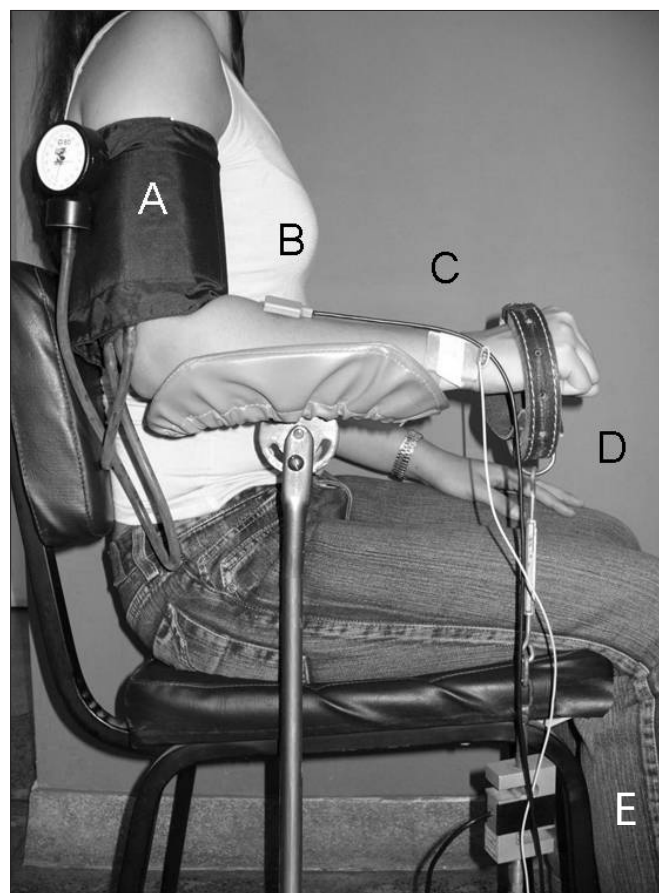
To collect the electromyographic signal, the EMG1000 signal acquisition module was used (Lynx®, São Paulo, SP, Brazil), as well as a differential surface electrode (Lynx®, São Paulo, SP, Brazil), which follow the ISEK and SENIAM guidelines.

The EMG1000 (Lynx®, São Paulo, SP, Brazil) signal acquisition module has a 10⁹ Ohms impedance, a digital/analog converter with a 16-bit resolution and an input band ranging from ±1V to ±10V, with an acquisition frequency of 2000Hz, a Butterworth-type filter with a high-pass of 20Hz, and a low pass of 1000Hz. The EMG1000 (Lynx®, São Paulo, SP, Brazil) was connected to a Pentium III desktop computer. The signal acquisition system was connected to a 12-volt battery with a capacity of 10-ampère-hour (AH), connected to a computer through optical fiber in order to cancel out the interference coming from the wiring on the electromyograph, as described by Guirro, Forti and Rodrigues-Bigaton¹⁵.

The differential surface electrode (consisting of two pure silver poles, 10mm long, 1mm wide and placed 10mm from one another, with a pre-amplifier circuit with a 20-time boost

(±1%), IRMC >100 dB, and signal/ratio <3μV RMS) was positioned perpendicularly to the fibers of the WEMG, in the dorsal region of the arm, approximately 5cm from the elbow, on the muscle mass which emerged when the participant was asked to perform the counter-resistance wrist extension¹⁶ (Figure 1B). Before the electrode was attached, the skin was shaved and cleaned with alcohol at 70%. The reference electrode (30x40mm), made from a metallic plate, was positioned in the ulnar styloid process on the same side that was being evaluated (Figure 1C). To obtain the digital signal, and also to store the data into files, we used the Aqdados software (Lynx®, São Paulo, SP, Brazil), version 7.02 for Windows.

The collection of the electromyographic signal was carried out during three MVICs for 15 seconds, with 30-second intervals, in the following situations: pre-ischemia (under normal blood flow conditions); ischemia (in which the collection began 5 minutes after the sphygmomanometer was inflated and the absence of blood flow was confirmed by the Doppler ultrasound); immediate post-ischemia (post-1; in which the contractions were started upon removing the sphygmomanometer and confirming the blood reperfusion by the Doppler



(A) Sphygmomanometer; (B) Differential surface electrode; (C) Reference electrode; (D) Leather belt; and (E) Load cell.

Figure 1. Position of the participant for electromyographic signal collection and determination of the wrist extensor muscle group strength.

ultrasound); later post-ischemia (post-2; 10 minutes after the onset of the ischemia).

The electromyographic signal was processed in the domains of time and frequency. For the analysis of the time domain, the RMS value was calculated because, according to De Luca¹⁷, this is the processing modality which best represents the amplitude of the electromyographic signal in voluntary muscle contractions. For the analysis of the frequency domain, the Fast Fourier Transform (FFT) was applied to the electromyographic signal to generate the power spectral density. To achieve that, we used 512-point Hanning windows with 256 ms and a 50% overlap. The median frequency was used as suggested by Stulen and De Luca¹⁸, who claim that this statistical parameter has the function of splitting the power spectrum into two isoenergetic regions, and that it is the one which best reflects the physiological changes that occur in the muscle during the sustained contractions¹⁷, such as the conduction velocity of the muscle fibers, and the recruitment of the motor unit¹⁹.

The electromyographic signal was processed by the *off-line* analysis, in the Matlab[®] 6.5.1 software, using specific functions to evaluate the quality of the acquired signal and to obtain the RMS values and the median frequency. The electromyographic signal was not normalized because, according to Soderberg and Knutson²⁰, if in a given experimental procedure the individuals are their own control, and the comparisons are performed on the same day and muscle without removing the electrode, the normalization is not necessary. The experimental procedure of the present study takes the aforementioned guidelines into account.

For the data analysis, programs such as Statistical Package for Social Science for Personal Computer (SPSS/PC version 11.0) and BioEstat 4.0[®] were used, and the Shapiro-Wilk test was applied to determine the normality of the sample, followed by the Friedman test and two-way ANOVA. A level of significance of 5% was set for the analysis of the variables.

Results

The muscle strength values were expressed by mean and standard deviation, and the RMS values and median frequency, by median (MED) and interquartile interval (AIQ).

Figure 2 shows that the ischemia promoted a reduction in the strength of the WEMG (8.63±1.98 kgf in pre-ischemia, and 4.79±1.64 kgf in ischemia, $p<0.01$). In the post-1 and post-2 situations, there was a statistically significant increase in muscle strength when compared with the ischemia situation (7.44±1.65 kgf in the post-1, and 7.13±1.18 kgf in the post-2, $p<0.05$). For the RMS (μv), there was no significant change

($p=0.05$) in the ischemia situations (MED:50.09; AIQ:68.72), post-1 (MED:30.99; AIQ:87.19) and post-2 (MED:30.35; AIQ:95.39), as shown in Figure 3.

With regard to the median frequency (Figure 4), no significant changes were observed ($p=0.09$) in the situations of ischemia (MED:59.89; AIQ:21.32), post-1 (MED:63.47; AIQ:17.25) and post-2 (MED:63.47; AIQ:22.13).

Discussion

Ischemia can cause a series of changes in the metabolic and enzymatic processes in the muscle, and the reversibility of this process is directly related to the duration of the ischemic period²¹. These changes occur due to the fall of the intracellular pH and to the imbalance in the Na^+/K^+ pump, with an increase in the Na^+ inflow, reduction in the intracellular $\text{K}^{+1,21}$ and inability to release the Ca^{2+} ions inside the muscle fiber, bringing about a reduction in the ability to produce strength²².

The present study found that 5 minutes of induced ischemia, in addition to 15 seconds of maximal voluntary isometric contraction of the WEMG, which has a predominance of type-I muscle fibers on the dominant side²², were enough to cause a decline in strength, though it was immediately recovered after the return of the blood flow, reaching levels close to the strength value produced in the pre-ischemic phase. However, even after 5 minutes of blood reperfusion, the strength values did not return to their initial parameters.

Similar results were observed in the gastrocnemius muscle of dogs submitted to an ischemic period of 2 minutes, in which a 30-second reperfusion caused a recovery of 67% of the initial value of muscle strength. Nevertheless, there is no record of total strength recovery through blood reperfusion²³. Muscle strength was analyzed in the knee extensor muscles, submitted to 5 minutes of ischemia and to 5 minutes of ischemia with isometric contraction, and the latter condition resulted in greater strength reduction²⁴.

There was also a change in the muscle strength in the evaluation of maximal torque and total work of the flexor and extensor muscles of the elbow in individuals with pathological ischemia of the upper limbs by means of the isokinetic dynamometry. The most evident strength reduction occurred in the elbow extensor muscles¹.

Concerning the amplitude of the electromyographic signal, there were no significant changes in the mean RMS values during or after the induced ischemia. The results of the present study are in accordance with the findings of Farina, Gazzoni and Camelia¹⁰, who in a situation of ischemia, did not observe any significant change in the amplitude of the electromyographic

signal of the abductor pollicis brevis muscle, which was submitted to 16 minutes of blood flow occlusion. However, they disagree with the findings of Leonard et al.²⁵, who observed a decrease in the RMS values of the soleus muscle after 8 to 12 minutes of ischemia.

In regard to the frequency of the electromyographic signal, there were no significant changes in the mean values of the median frequency of the power spectrum signal during and after the induced ischemia. Contrary to that finding, Merletti, Sabbahi and De Luca²⁷ noted that induced ischemia of the first dorsal interosseous muscle for 10 minutes with isometric contraction brought about a significant reduction in the conduction velocity of the muscle fiber, i.e. the median frequency of the power spectrum of the electromyographic signal originated by the accumulation of metabolites in this condition. The vasodilation and consequent increase in the temperature reached during blood reperfusion led to a quick removal of the metabolic byproducts, resulting in the immediate restoration of the median frequency of the power spectrum of the electromyographic signal²⁶.

The abductor pollicis brevis muscle of 9 male individuals underwent 16 minutes of ischemia in order to analyze the conduction velocity of isolated motor units. It was found that the conduction velocity of that muscle decreased after 13 minutes of blood flow occlusion¹⁰.

By using the same muscle in isometric contractions with normal blood flow and with induced ischemia for 8 minutes, we observed that the muscle strength, muscle fiber conduction velocity, and therefore the median frequency of the power spectrum of the electromyographic signal diminished during the ischemia. Under normal blood flow conditions, the conduction velocity of the inactive muscle fibers was reduced in 5 minutes of muscle contraction; however, in 3 minutes of ischemia, the conduction velocity of the inactive fibers of the muscle under investigation was tripled²².

We believe that the methodology employed, as well as the type of muscle fiber, and the time of ischemia may have been the causes for divergence between the present study and the previously mentioned studies. There is a discrepancy in the literature as for the type of muscle fiber affected by ischemia. Studies suggest that the type-I slow fibers are the most susceptible ones as they depend on an adequate blood and oxygen supply to synthesize the ATP²⁵. Nonetheless, there are reports attesting that ischemia led to more pronounced changes in the conduction velocity of type-II muscle fibers, given the fact that they are quick contraction fibers and consequently produce more metabolic byproducts during muscle contraction²⁷.

The WEMG consists of the extensor carpi radialis brevis, the extensor carpi radialis longus, and the extensor carpi

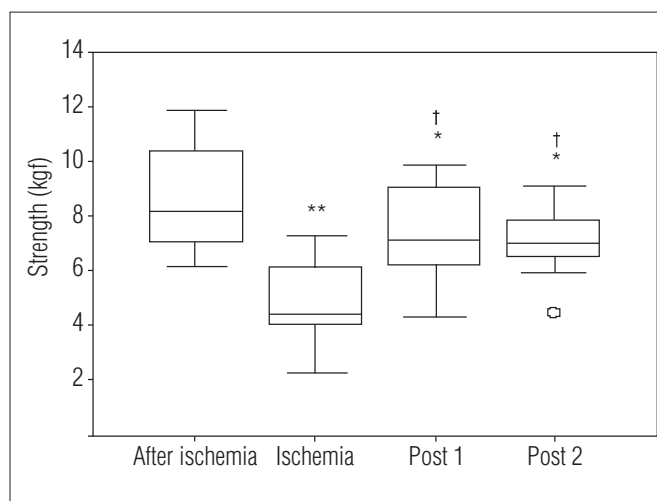


Figure 2. Strength (kgf) of the wrist extensor muscle group during MVIC in the pre-ischemia, ischemia, post-1 and post-2 situations; * and ** differ from pre-ischemia ($p < 0.05$ and $p < 0.01$, respectively) and (†) differs from ischemia ($p < 0.01$).

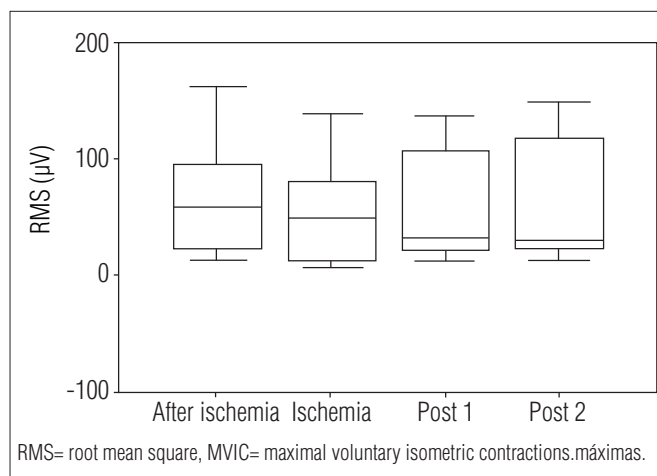


Figure 3. Median RMS values (μV) of the wrist extensor muscle group during MVIC in the pre-ischemia, ischemia, post-1 and post-2 situations ($p = 0.05$).

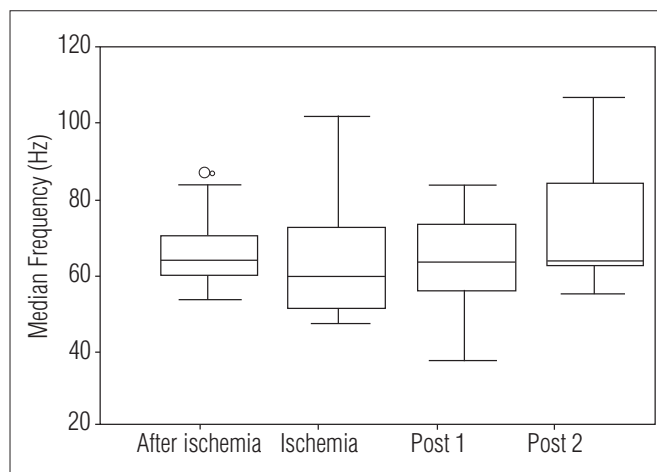


Figure 4. Median frequency values (Hz) of the wrist extensor muscle group during MVIC in the pre-ischemia, ischemia, post-1 and post-2 situations ($p = 0.09$).

ulnar. On the dominant side, they have a predominance of type-I muscle fibers, which may be altered by genetic factors, functional demands, and intra-individual variation²⁸.

It is known that type-I fibers are more resistant to fatigue than type-II fibers, and that the motor units are recruited according to the activation threshold. Thus, because they have a smaller threshold, the type-I fibers are initially activated, and the type-II fibers are then recruited²⁹. Considering that the WEMG has a larger amount of oxidative fibers and that the oxygen supply to these fibers was reduced due to the ischemia, it can be suggested that the type-II muscle fibers were activated at the time, and that the type-I fibers were reactivated when blood flow resumed.

The clinical implications of this study are relevant, given that they emphasize that the production of muscle strength is affected by ischemia and that this must be considered in the

physical therapist's clinical evaluation, as the diminishment of strength production, among other symptoms, may indicate an ischemic condition. Regarding the electromyographic test carried out by means of a differential surface electrode, we suggest that other studies be made in order to lend weight to its use in the clinical evaluation of patients with ischemic dysfunctions.

Conclusion ::::

It can be concluded that a 5-minute induced ischemia caused fatigue of the WEMG when related to the production of muscle strength. However, when related to the electromyographic signal, it can be stated that induced ischemia did not cause electromyographic fatigue in the assessed muscle group.

References ::::

- Nakano L. Avaliação objetiva de isquemia de membros superiores: uso do dinamômetro isocinético [tese]. São Paulo: USP; 2005.
- Rempel DM, Harrison RJ, Barnhart S. Work-related cumulative trauma disorders of the upper extremity. *JAMA*. 1992;268(6):787-8.
- Murthy G, Kahan NJ, Hargens AR, Rempel DM. Forearm muscle oxygenation decreases with low levels of voluntary contraction. *J Ortho Res*. 1997;15(4):507-11.
- Bystrom EG, Kilbom A. Physiological response in the forearm during and after isometric intermittent handgrip. *Eur J Appl Physiol Occup Physiol*. 1990;60(6):457-66.
- Hogan MC, Richardson RS, Kurdak SS. Initial fall in skeletal muscle force development during ischemia is related to oxygen availability. *J Appl Physiol*. 1994;77(5):2380-4.
- Murthy G, Hargens AR, Lehman S, Rempel DM. Ischemia causes muscle fatigue. *J Ortho Res*. 2001;19(3):436-40.
- Farina D, Merletti R, Enoka RM. The extraction of neural strategies from the surface EMG. *J Appl Physiol*. 2004;96(4):1486-95.
- Basmajian JV, De Luca CJ. *Muscle alive: their function revealed by electromyography*. São Paulo: Baltimore, Williams & Williams; 1985.
- Chung LH, Callahan DM, Kent-Braun JA. Age-related resistance to skeletal muscle fatigue is preserved during ischemia. *J Appl Physiol*. 2007;103(5):1628-35.
- Farina D, Gazzoni M, Camelia F. Conduction velocity of low-threshold motor units during ischemic contractions performed with surface EMG feedback. *J Appl Physiol*. 2005;98(4):1487-94.
- Craig CL, Marshall AL, Sjöström M, Bauman AE, Booth ML, Ainsworth BE et al. International Physical Activity Questionnaire:12-country reliability and validity. *Med Sci Sport Exerc*. 2003;35(8):1381-5.
- Fulco CS, Rock PB, Muza SA, Lammi E, Cymerman A, Butterfield G et al. Slower fatigue and faster recovery of the adductor pollicis muscle in women matched for strength with men. *Acta Physiol Scand*. 1999;167(3):233-9.
- Hicks AL, McCartney N. Gender differences in isometric contractile properties and fatigability in elderly human muscle. *Can J Appl Physiol*. 1996;21(6):441-54.
- Bitu-Moreno J, Francischetti I, Hafner L. Lesões de isquemia-reperusão em músculos esqueléticos: fisiopatologia e novas tendências de tratamento, com ênfase em reperusão controlada. *J Vasc Br*. 2002;1(2):113-20.
- Guirro RRJ, Forti F, Rodrigues-Bigaton D. Proposal for electrical insulation of the electromyographic signal acquisition module. *Electromyogr Clin Neurophysiol*. 2006;46(6):355-63.
- Cram JR, Kasman GS, Holtz J. *Introduction to surface electromyography*. Gaithersburg: An Aspen Publication; 1998.
- De Luca CJ. The use of surface electromyography in biomechanics. *J Biomech*. 1997;13:135-63.
- Stulen FB, De Luca CJ. Muscle fatigue monitor: a noninvasive device for observing localized muscular fatigue. *IEEE Trans Biomed Eng*. 1982;29(12):760-8.
- Solomonow M, Baten C, Smit J, Battar R, Hermens H, D'Ambrosia R et al. Electromyogram power spectra frequencies associated with motor unit recruitment strategies. *J Appl Physiol*. 1990;68(3):1177-85.

20. Soderberg GL, Knutson LM. A guide for use and interpretation of kinesiological electromyographic data. *Phys Ther.* 2000;80(5):485-98.
21. Silveira M, Yoshida W. Isquemia e reperfusão em músculo esquelético: mecanismos de lesão e perspectivas de tratamento. *J Vasc Br.* 2004;3(4):367-78.
22. Gazzoni M, Camelia F, Farina D. Conduction velocity of quiescent muscle fibers decreases during sustained contraction. *J Neurophysiol.* 2005;94(1):387-94.
23. Hogan MC, Kohin S, Sary CM, Hepple RT. Rapid force recovery in contracting skeletal muscle after brief ischemia is dependent on O₂ availability. *J Appl Physiol.* 1999;87(6): 2225-9.
24. Pierce JR, Clarck BC, Ploutz-Snyder LL, Kanaley JA. Growth hormone and muscle function responses to skeletal muscle ischemia. *J Appl Physiol.* 2006;101(6):1588-95.
25. Leonard CT, Kane J, Perdaems J, Frank C, Graetzer D, Moritani T. Neural modulation of muscle contractile properties during fatigue: afferent feedback dependence. *Eletroencephalogr Clin Neurophysiol.* 1994;93(3):209-17.
26. Merletti R, Sabbahi MA, De Luca CJ. Median frequency of the myoelectric signal: effects of muscle ischemia and cooling. *Eur J Appl Physiol Occup Physiol.* 1984;52(3):258-65.
27. Gerdle B, Fugl-Meyer AR. Is the mean power frequency shift of the EMG a selective indicator of fatigue of the fast twitch motor units? *Acta Physiol Scand.* 1992;145(2):129-38.
28. Fugl-Meyer A, Eriksson AR, Sjöström M, Söderström G. Is muscle structure influenced by genetical or functional factors? A study of three forearm muscles. *Acta Physiol Scand.* 1982;114(2):277-81.
29. Widmaier EP, Raff H, Strang KT. *Fisiologia humana: os mecanismos das funções corporais.* 9ª ed. Rio de Janeiro: Guanabara Koogan; 2006.