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Endurance markers are related with local neuromuscular response for the intact but not for the ACL reconstructed leg during high intensity running

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ENDURANCE MARKERS ARE RELATED WITH LOCAL NEUROMUSCULAR RESPONSE FOR THE INTACT BUT NOT FOR THE ACL RECONSTRUCTED LEG DURING HIGH INTENSITY RUNNING

Short title: Endurance correlates of EMG response in ACLR athletes

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ABSTRACT

AIM: It has been demonstrated that the local neuromuscular response during high intensity exercise has a strong relationship with endurance markers. However a diminished neuromuscular response has been reported for the operated leg in athletes having undergone anterior cruciate ligament reconstruction (ACLR). The purpose of the present study was to examine the relationships between endurance markers and the EMG response during high intensity running in ACLR athletes. **METHODS:** Fourteen ACLR soccer players underwent a GXT test to volitional exhaustion and a 10-min bout of high intensity running. During the 10-min bout, EMG data were recorded at the 3^{rd} and 10^{th} minute from the vastus lateralis bilaterally using a telemetric system. The final EMG levels were expressed as a percentage of the initial values. Pearson moment product correlations were used to assess the relationship between the endurance markers of VO_2max, velocity at lactate threshold (vLT), velocity at 4mM (V_4) and the final EMG levels. **RESULTS:** Final EMG levels for the intact leg had a very strong relationship with vLT ($r=0.77$, $p=0.001$) and a strong relationship with V_4 ($r=0.68$, $p=0.008$). Final EMG levels for the reconstructed leg had moderate relationship with vLT ($r=0.47$, $p=0.09$) and V_4 ($r=0.52$, $p=0.06$). **CONCLUSION:** The neuromuscular response of the intact leg during high intensity running shows strong to very strong relationships with endurance markers. Failure of the ACLR leg to present relationships of similar strength may indicate that chronic perturbations modify the ability of the local muscular environment to tolerate sustained high intensity efforts.

Key words: EMG, lactate threshold, ACLR reconstruction, fatigue, running

INTRODUCTION

The goal of the reconstruction of the anterior cruciate ligament in competitive athletes is to achieve return to pre-injury exercise levels. In this regard, optimal muscle activation is of great importance, especially during sustained athletic efforts. Previous research indicated that muscle activation levels (as assessed by surface EMG) do not differ between the operated and the intact leg during activities of moderate intensity [1, 2, 3]. However, it has recently been demonstrated that during high intensity exercise the operated leg exhibits a diminished EMG response over time as compared to both the intact and a healthy control leg [4**,** 5]. These studies demonstrated that during a 10-min high intensity running bout, both the intact and the healthy control leg significantly increased EMG activity of the vastus lateralis by an average of ~8% compared to the initial values. However, the operated leg did not increase EMG activity of the vastus lateralis as compared to baseline.

From a physiological perspective, the most commonly used endurance markers that are considered prerequisite for optimal performance during high intensity sustained efforts are the maximal oxygen uptake (VO_2max), and various aspects of the lactate threshold (LT). Such aspects are mathematically derived thresholds and fixed blood lactate thresholds (e.g the 4mM threshold) [6, 7, 8, 9, 10]. A number of previous studies have confirmed that these endurance markers correlate positively with the local neuromuscular response measured as change in EMG activity [11, 12, 13, 14, 15, 16, 17]. Thus, a higher velocity or power output at lactate threshold is associated with increased EMG activity, indicating that during high intensity exercise the local neuromuscular response is largely determined by physiological factors [18, 19, 20].

However, since our previous research [4, 5] has identified that ACLR athletes display different local neuromuscular responses, it is possible that in such individuals the relationships between endurance markers and the local neuromuscular response are also

altered. Such potential alterations may have important implications for the rehabilitation as well as the conditioning of ACL reconstructed athletes. Therefore the purpose of the present study was to assess the relationships between selected endurance markers and the relative increase in local EMG activity during high intensity running in ACLR athletes. Specifically we hypothesized that in these athletes higher EMG levels will be related with higher values in endurance markers and that the intact leg will display different relationships than the operated leg.

MATERIALS AND METHODS

Fourteen amateur male soccer players [mean (SD) age, body weight and height, 24.8 (5.3) years, 77.3 (7.5) kg and 177 (5.3) cm] with ACL-reconstructed knees were recruited for the present study. The operated athletes had undergone ACL reconstruction with bone-patella tendon-bone (BPTB) graft, 18.5 (4.3) months before testing. ACL reconstruction was performed sub-acutely within 6 months after the injury from the same surgeon. All operated athletes had a unilateral ACL tear confirmed by MRI and arthroscopy. They all underwent the same rehabilitation protocol, starting from the first post operative day with the use of passive exercises. Return to sports was permitted 6 months after reconstruction according to the following widely accepted criteria [21]: (1) Full range of motion, (2) KT-1000 side-to side difference <3mm, (3) quadriceps strength >85% compared to the contralateral side, (4) hamstrings strength 100% compared to the contralateral side, (5) hamstrings-to-quadriceps strength ratio >70% and (6) functional testing >85% compared to the contralateral side. At the time of data collection, no clinical evidence of knee pain and effusion was found in the ACL reconstructed subjects. All subjects agreed with the testing protocol and signed a consent form according to the Declaration of Helsinki. Ethical approval was granted from the Institutional Review Board policies of our Medical School. Prior to any data collection, a clinical

evaluation was performed in all athletes by the same clinician. During this evaluation, the Tegner and Lysholm scores were obtained, while anterior tibial translation was evaluated using the KT-1000 knee arthrometer (MEDmetric Corp., San Diego, California) [22]. The athletes reported to the laboratory having abstained from caffeine or food consumption for 4 hours and without vigorous training for 24h on two different occasions separated by 48 hours. Experimental testing took place at an ambient temperature of 20 ºC to 22 ºC.

For their first visit to the laboratory, the athletes performed a GXT test to volitional exhaustion to determine VO_2max, LT and the 4mM threshold [23]. During warm-up the athletes performed 3 minutes walking at self-selected pace and 5 minutes jogging on a treadmill (Technogym Runrace 1200, Italy) at a speed of 8 $km \cdot h^{-1}$ where heart rate and blood lactate were measured. The incremental test begun at a speed of 10 $km \cdot h^{-1}$ with stage increments of 2 $km \cdot h^{-1}$ every three minutes. During the test, expired gas samples were measured using a breath-by-breath metabolic measurement system (CPX Ultima, Medical Graphics, St Maul, MN, USA). This system uses a zirconium O_2 analyzer and an infrared CO_2 analyzer. The subjects wore a facemask and inspired-expired gas samples were collected through a pneumotachograph (pre-Vent, Medical Graphics, St Maul, MN, USA) and analyzed for O_2 and CO_2. At the end of each stage, capillary blood samples were collected and analyzed for lactate (Accutrend, Roche Diagnostics, Germany). Prior to each test, all analyzers were calibrated according to the manufacturer's instructions. According to the American College of Sports Medicine, criteria for VO_2max were (a) plateau in VO_2 (an increase <2.1 $ml \circ kg^{-1}min^{-1}$ despite an increase in running speed, (b) a respiratory exchange ratio (RER) > 1.10, (c) , and (d) maximal blood lactate values (LAmax) >8 $mmol \circ l^{-1}$[23]. In all cases at least 2 out of 3 criteria were met. The velocity associated with LT (vLT) was determined according to the Dmax method proposed by Cheng et al [24]. Velocity at the 4 mM fixed blood lactate threshold (V_4) was determined from the individual velocity-blood

lactate curves using linear interpolation [8, 9, 10]. The velocity that was used for the subsequent high intensity running was set at 40% of the difference between VO_2max and lactate threshold (vD) and represents the upper limit of high intensity exercise [25**]**. All three velocities were also expressed as % of VO2max [7, 8, 9, 25].

In the second visit to the laboratory, athletes were required to perform a 10-minute high intensity run at the vD velocity. During the 10 minute running bout, EMG data were collected for 15 seconds at the 3rd and 10th minute. Gas exchange data were recorded simultaneously breath-by-breath and heart rate was measured throughout the test. Blood lactate was measured prior to running and immediately after termination of exercise. EMG traces were obtained from the vastus lateralis muscle bilaterally using bipolar, circular, pre-amplified, pre-gelled Ag/AgCl electrodes with 10 mm diameter and fixed inter-electrode spacing of 20 mm (Noraxon Inc, Scottsdale, AZ, USA). It has been demonstrated that vastus lateralis and rectus femoris show similar high relationships between EMG activity and lactate threshold during cycling [12, 13, 14]. However, vastus lateralis was selected on the basis that shows higher levels of activity compared to rectus femoris during the first part of stance phase of running and acts as a shock absorber, thus protecting the cartilage and the graft from high impact forces [26]. EMG data were recorded with a wireless 8-channel EMG system (Telemyo 2400T, Noraxon Inc, Scottsdale, AZ, USA) and displayed real-time on a personal computer using dedicated software (MyoResearchXP, Noraxon Inc, Scottsdale, AZ, USA). The surface of the skin was prepared by shaving hair, rubbing it with abrasive paper and cleaning it with alcohol. The electrodes were fixed longitudinally over the muscle belly. Electrodes were placed at the antero-lateral muscle bulge at 2/3 of the proximo-distal thigh length [27, 28]. The visually largest area of muscle belly was selected using a contraction against manual resistance. The ground electrode was placed on the lateral femoral condyle of the right leg

[27]. Electrodes and cables were secured with surgical tape, in order to avoid any interference with the running pattern of the subjects.

Footswitches (Noraxon Inc, Scottsdale, AZ, USA) placed under the heel and big toes of both legs were used to denote heel-strike and toe-off events. EMG was acquired at a sampling rate of 1500 Hz. The raw EMG was measured in a band of 10 to 500 Hz, was full-wave rectified, was high pass filtered (cut-off frequency at 20 Hz) with an 8^{th} order Butterworth filter to remove movement artifacts and was smoothed with a 100 ms RMS algorithm. Values from 20 strides were averaged to calculate the mean peak amplitude during stance. A preliminary review of the relevant literature revealed that EMG amplitude has been used in many previous studies of similar design [11-13, 16, 17] and is considered to be a reliable parameter to evidence EMG activity. Mean peak amplitude values from the 10^{th} minute were expressed as percentage of the initial values during the 3^{rd} minute and were defined as the final EMG values.

Statistical analysis: Data were expressed as means and standard deviation (mean ± SD). Pearson product moment correlations were used to determine the relationships between final EMG values and the VO_2max (both as $lt \cdot min^{-1}$ and $ml \cdot min^{-1} \cdot kg^{-1}$), vLT, vLT expressed as percentage of VO_2max (vLT %), V_4, V_4 expressed as percentage of VO_2max (V_4 %), vD and vD expressed as percentage of VO_2max (vD %). These relationships were identified for both the ACLR and the intact leg.

RESULTS

Negative Lachman and pivot shift tests indicated that the static knee joint stability was regained. The subjects had all returned to their previous level activity engaging running and soccer specific training for 3-5 sessions/week. Lysholm score was 95 (94-96) and Tegner score was 8 (range, 7-9). KT-1000 results revealed that the mean difference between the

anterior tibial translation of the reconstructed and intact sides was 1.6 mm (range, 1 to 2mm) for the 134-N test and 1.8 mm (range, 1 to 2 mm) for the maximal manual test.

Mean values for the endurance markers are presented in Table 1. The vD high intensity run was performed on average at 87.6% (4.4) of VO_2max. Blood lactate values during the vD run rose from an initial baseline value of 2.1 (0.3) mM to 7.6 (1.7) mM. All athletes completed the 10 min vD run without being completely exhausted and indicated that they could continue for "a few more minutes".

INSERT TABLE 1 ABOUT HERE

Pearson moment product correlations and coefficients of determination between final EMG activity of the intact ($EMG10_{int}$) and reconstructed leg ($EMG10_{rec}$) and each one of the endurance markers are presented in Table 2. $EMG10_{int}$ demonstrated a very strong relationship with vLT (r=0.77, p=0.001) and V_4% (r=0.75, p=0.002), a strong relationship with V4 (r=0.68, p=0.008) and vD% (r=0.57, p=0.03) and moderate relationships with vLT% (r=0.46, p=0.09) and vD (r=0.44, p=0.11). Finaly, $EMG10_{int}$ displayed only weak relationship with VO_2max expressed either as absolute (r=-0.19, p=0.51) or relative values (r=-0.18, p=0.54). $EMG10_{rec}$ demonstrated moderate relationships with vLT (r=0.47, p=0.09), V4 (r=0.52, p=0.06) and V4% (r=0.5, p=0.07), weak relationships with vD (r=0.2, p=0.49) and vD% (r=0.24, p=0.42) and very weak relationships with vLT% (r=0.08, p=0.87) and VO_2max expressed as either absolute (r=0.05, p=0.87) or relative (r=-0.03, p=0.92)values.

TABLE 2 ABOUT HERE

DISCUSSION

The purpose of the present study was to assess the relationships between endurance markers and the relative increase in local EMG activity during high intensity running in ACLR athletes. Specifically we hypothesized that in these athletes higher EMG levels will be

related with higher values in the endurance markers and that the intact leg will display different relationships than the operated leg.

The major finding of the present study was that EMG activity of the intact leg during high intensity running demonstrated very strong relationships with vLT, V_4 % and strong relationships with V_4 and vD %, while EMG activity of the operated leg showed only moderate relationships with vLT, V_4 and V_4 %. The strong to very strong relationships (r=0.57-0.77) established for the intact leg are in line with previous studies that have suggested a strong physiological link between myoelectric changes at fatigue and the lactate threshold [15, 18, 19, 20].

During sustained fatiguing running performed at the vD velocity which averaged 87.6% (4.4) of VO_2max, EMG of the working muscles tends to increase over time as a result of (i) increased firing rate (rate-coding) of the already activated muscle fibers, (ii) recruitment of previously inactive fibers, especially the type II and (iii) both of above mechanisms [18, 29, 30]. On the other hand at these high exercise intensities there is substantial accumulation of metabolic by-products such as lactic acid, H^+, P_i, which may lead to drop out of mechanically failing muscle fibers and decreased EMG activity over time [31]. Thus, final EMG levels during sustained high intensity running depend upon the balance between these two opposing trends. Furthermore, given the fact that lactate threshold represent the balance between accumulation and clearance of metabolic by-products in the exercising muscles [32, 33], it is not surprising that EMG activity of the intact VL showed strong to very strong relationships with the two markers indicative of the lactate threshold, namely vLT and V_4. We hypothesize that the athletes that had higher vLT and V_4 values were able to increase their EMG activity of the intact leg to higher levels probably due to the fact that were better able to sustain the accumulation of metabolic by-products which tends to suppress EMG amplitude.

We were able to demonstrate only a weak relationship between EMG activity of the intact leg and VO_2max. This is line with Bassett and Howley [6] who concluded that VO_2max is mainly linked to central adaptations, presumably maximal cardiac output, while lactate threshold incorporates both central and peripheral adaptations [7, 34]. Thus, lactate threshold may more closely reflect the ability of the local muscle environment to tolerate fatiguing exercise without compromising myolectrical activity.

Our results further indicated that the EMG activity of the reconstructed leg showed only moderate relationships with vLT, V_4 and V_4 %. Several explanations can be given for the lack of a strong relationship between EMG activity in the reconstructed leg and the endurance markers. Recent studies have presented evidence supporting the notion that selective fiber II atrophy may occur in the involved quadriceps after ACL reconstruction [35, 36, 37]. Furthermore, alterations in activity patterns following surgery and subsequent retraining may alter motor unit activation [38]. In addition, loss of joint afferent information may lead to quadriceps weakness and subsequent selective hypotrophy of type II muscle fibers [39]. Thus, the reconstructed leg may display a decreased potential for progressive recruitment and this would only be apparent when performing at high intensities [30]. This seems plausible since it has been suggested that in the case of large muscles, additional recruitment and not increases of firing rate is the major strategy used to generate the required forces over a wide range of submaximal exercise intensities [40]. The above hypothesis is also supported by the fact that the reconstructed leg does not increase EMG activity over time during sustained high intensity exercise [4, 5]. On the other hand, greater suppression of the EMG signal of the reconstructed leg due to higher accumulation of metabolic by-products as compared to the intact leg, may also explain the lack of relationship between EMG activity and endurance markers and the lack of increase in EMG activity over time [31]. This hypothesis is supported by the fact that long-term muscle detraining in endurance athletes, such as soccer players

undergoing knee surgery, induces a transformation of IIa fibers (fast-oxidative, fatigue resistant) to IIb fibers (fast-glycolytic, fatiguable) [41, 42]. Thus loss of the aerobic metabolic characteristics of IIa fibers of the operated leg may suggest that when performing at high intensities (above the lactate threshold) these fibers are subjected to greater metabolic disturbances compared to the "normal" IIa fibers of the contralalteral leg. In any case, the above two hypotheses imply that high intensity exercise is not tolerated in the same manner by the VL of the intact and the reconstructed leg and that fatiguing exercise may provoke greater physiological strain on the VL muscle of the reconstructed leg compared to the VL muscle of the intact leg.

Given the interdependence between neuromuscular and metabolic systems, an important implication of the present study is the potential for improved neuromuscular response of the reconstructed leg following endurance training. Usually, improvement of aerobic fitness of athletes following ACL reconstruction is performed after the athlete is discharged from formal rehabilitation and only in the case of elite athletes during accelerated rehabilitation [43]. It should be anticipated that an aerobic endurance training program aiming at peripheral (muscle) adaptations would improve neuromuscular response by improving lactate tolerance and accumulation of metabolites implicated in the fatigue process during high intensity exercise [7, 8, 29, 34, 44]. To the best of our knowledge this is the first study that investigated directly the relationship between the local neuromuscular response and endurance markers in ACL reconstructed athletes. Previous studies on healthy subjects have assessed extensively the relationship between the local neuromuscular response and endurance markers during high intensity cycling [11, 12, 13, 14, 16, 17]. Our approach enabled us to extend our findings to intense running which represents a highly functional activity for the ACL reconstructed athlete. Furthermore, using preliminary VO_2max and

lactate threshold testing we assigned high intensity exercise according to individual fitness levels. Thus, all subjects exercised under identical controlled conditions.

In the present study only ACL reconstructed athletes with bone-patellar tendon-bone autograft were recruited. Thus, it is unknown if a similar response pattern will be observed in athletes with a different graft such as hamstrings. Furthermore it is accepted that proprioceptive deficits occur in most patients after ACL rupture and that persist to some degree after ACL reconstruction [45]. However we believe that this should have a minimal effect in our study because, a) our players were all treated with the same rehabilitative protocol, which emphasized on proprioceptive re-training and b) our study design involved treadmill running which is a cyclic highly repeatable two-dimensional motion. It should also be acknowledged that EMG recordings should be performed with great care and the results should be interpreted with caution when it comes to dynamic muscle contractions and especially whole body exercises such as running. With that in mind, signal capturing, recording and processing were performed according to established guidelines [27, 28]. We selected a fixed epoch for the period of contraction in our study. Thus, we examined electrical activity developed solely during the stance period, thereby reducing to some extent the role of the signal non-stationarities with respect to the other effects being studied [46]. Furthermore, the peak activity of many (successive) steps was averaged providing a reasonable estimation of peak electrical activity during every recording period and minimizing within subject variability. In addition, our study design involved repeated measures, that is, the final value of the EMG activity was normalized to its original value while the electrodes remained attached during the whole task, thereby overcoming the between-subject variability of EMG amplitude [47].

CONCLUSIONS

In conclusion, the neuromuscular response of the intact leg during high intensity exercise shows strong to very strong relationships with endurance markers that are indicative of local muscle adaptations to endurance training. Failure of the ACLR leg to present a similar response may indicate that chronic perturbations may impair the normal physiological response and/or that variables other than endurance markers may also be implicated in the ability of the local muscle environment to tolerate sustained high intensity efforts.

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REFERENCES

1. Bulgheroni P, Bulgheroni MV, Andrini L, Guiffanti P, Giughello A. Gait patterns after anterior cruciate ligament reconstruction. Knee Surg Sports Traumatol Arthrosc 1997; 5:14–21.
2. Knoll Z, Kiss RM, Kocsis L. Gait adaptation in ACL deficient subjects before and after anterior cruciate ligament reconstruction surgery, J Electromyogr Kinesiol 2004; 14:287–294.
3. Lewek M, Rudolph K, Axe M, Snyder-Mackler L. The effect of insufficient quadriceps strength on gait after anterior cruciate ligament reconstruction, Clin Biomech 2002; 17:56–63.

4. Patras K, Ziogas G, Ristanis S, Tsepis E, Stergiou N, Georgoulis AD. High intensity running results in an impaired neuromuscular response in ACL reconstructed individuals. Knee Surg Sports Traumatol Arthrosc. 2009; 17:977-84.
5. Patras K, Ziogas G, Ristanis S, Tsepis E, Stergiou N, Georgoulis AD. ACL reconstructed patients with a BPTB graft present an impaired vastus lateralis neuromuscular response during high intensity running. J Sci Med Sport 2010; doi:10.1016/j.jsams.2009.12.001
6. Bassett DR Jr, Howley ET. Limiting factors for maximum oxygen uptake and determinants of endurance performance. Med Sci Sports Exerc. 2000; 32:70-84.
7. Coyle EF. Physiological determinants of endurance exercise performance. J Sci Med Sport 1999; 2:181-9.
8. Sjödin B, Jacobs I. Onset of blood lactate accumulation and marathon running performance. Int J Sports Med. 1981; 2:23-6.
9. Bishop D, Jenkins DG, MacKinnon LT. The relationship between plasma lactate parameters, Wpeak and 1-h cycling performance in women. Med Sci Sports Exerc 1998; 30:1270 –5.
10. Bishop D, Jenkins DG, McEniery M, Carey MF. Relationship between plasma lactate parameters and muscle characteristics in female cyclists. Med Sci Sports Exerc 2000; 32:1088–1093.
11. Graef JL, Smith AE, Kendall KL, Walter AA, Moon JR, Lockwood CM, et. al. The relationships among endurance performance measures as estimated from VO_{2PEAK}, ventilatory threshold, and electromyographic fatigue threshold: a relationship design. Dyn Med. 2008; 10:7-15.

12. Camata TV, Lacerda TR, Altimari LR, Bortolloti H, Fontes EB, Dantas JL et. al. Association between the electromyographic fatigue threshold and ventilatory threshold. Electromyogr Clin Neurophysiol. 2009; 49:305-10.
13. Candotti CT, Loss JF, Melo Mde O, La Torre M, Pasini M, Dutra LA et.al. Comparing the lactate and EMG thresholds of recreational cyclists during incremental pedaling exercise. Can J Physiol Pharmacol. 2008; 86:272-8.
14. Lucía A, Sánchez O, Carvajal A, Chicharro JL. Analysis of the aerobic-anaerobic transition in elite cyclists during incremental exercise with the use of electromyography. Br J Sports Med. 1999 Jun;33(3):178-85.
15. Hanon C, Thépaut-Mathieu C, Hausswirth C, Le Chevalier JM. Electromyogram as an indicator of neuromuscular fatigue during incremental exercise. Eur J Appl Physiol Occup Physiol. 1998; 784:315-23.
16. Hug F, Laplaud D, Savin B, Grélot L. Occurrence of electromyographic and ventilatory thresholds in professional road cyclists. Eur J Appl Physiol. 2003; 90:643-6.
17. Hug F, Faucher M, Kipson N, Jammes Y. EMG signs of neuromuscular fatigue related to the ventilatory threshold during cycling exercise. Clin Physiol Funct Imaging. 2003; 23:208-14.
18. Borrani F, Candau R, Millet GY, Perrey S, Fuchslocher J, Rouillon JD. Is the VO_2 slow component dependent on progressive recruitment of fast-twitch fibers in trained runners? J Appl Physiol 2001; 90:2212–20.
19. Bouissou P, Estrade PY, Goubel F, Guezennec CY, Serrurier B. Surface EMG power spectrum and intramuscular pH in human vastus lateralis muscle during dynamic exercise. J Appl Physiol 1989; 67:1245–9.

20. Taylor AD, Bronks R. Electromyographic correlates of the transition from aerobic to anaerobic metabolism in treadmill running. Eur J Appl Physiol 1994; 69:508–15.
21. DeLee J, Drez D, Miller M. DeLee & Drez's orthopaedic sports medicine: principles and clinical practise. Philadelphia: Saunders; 2003.
22. Daniel DM, Malcom LL, Losse G, Stone ML, Sachs R, Burks R. Instrumented measurement of anterior laxity of the knee. J Bone Joint Surg (Am) 1985; 67:720–6.
23. Whaley MH, Brubaker PH, Otto RM. ACSM's guidelines for exercise testing and prescription. Philadelphia: Lippincott Williams & Wilkins; 2005.
24. B. Cheng, A. Kuipers, Snyder AC, Keizer HA, Jeukendrup A, Hesselink M. A new approach for the determination of ventilatory and lactate thresholds. Int J Sports Med 1992; 13:518–22.
25. Carter H, Pringle JSM, Jones AM, Doust JH. Oxygen uptake kinetics during treadmill running across exercise intensity domains. Eur J Appl Physiol 2002; 86:347–54.
26. Montgomery WH 3rd, Pink M, Perry J. Electromyographic analysis of hip and knee musculature during running. Am J Sports Med. 1994; 22:272-8.
27. Hermens HJ, Freriks B, Disselhorst-Klug C, Rau G. Development of recommendations for SEMG sensors and sensor placement procedures. J Electromyogr Kinesiol. 2000; 10:361-74.
28. Merletti R, Hermens H.J. Detection and conditioning of the surface EMG signal. In: Merletti R, Parker P, editors. Electromyography: physiology, engineering and non-invasive applications. New Jersey: Wiley-IEEE; 2004. p. 107-32.
29. Gaesser GA, Poole DC. The slow component of oxygen uptake kinetics in humans. Exerc Sport Sci Rev 1996; 24:35–71.

30. Jones AM, Pringle JSM, Carter H. Influence of muscle fiber type and motor unit recruitment on VO_2 kinetics. In: Jones AM, Poole DC, editors. Oxygen uptake kinetics in sport, exercise and medicine. London: Routledge; 2005. p. 261-93.
31. Pringle JS, Jones AM. Maximal lactate steady state, critical power and EMG during cycling. Eur J Appl Physiol. 2002; 88:214-26.
32. Brooks GA. Anaerobic threshold: review of the concept and directions for future research. Med Sci Sports Exerc. 1985; 17:22-34.
33. Svedahl K, MacIntosh BR. Anaerobic threshold: the concept and methods of measurement. Can J Appl Physiol. 2003; 28:299-323.
34. Holloszy, JO, and Coyle, EF. Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. J Appl Physiol 1984; 56:831-8.
35. Bryant AL, Kelly J, Hohmann E. Neuromuscular adaptations and correlates of knee functionality following ACL reconstruction. J Orthop Res 2008; 26:126–35.
36. McHugh MP, Tyler TF, Nicholas SJ, Browne MJ, Gleim G.W. Electromyographic analysis of quadriceps fatigue after anterior cruciate ligament reconstruction. J Orthop Sports Phys Ther 2001; 31:25–32.
37. Snyder-Mackler L, Binder-Macleod SA, Williams PR. Fatigability of human quadriceps femoris muscle following anterior cruciate ligament reconstruction. Med Sci Sports Exerc 1993; 25:783–9.
38. Drechsler WI, Cramp MC, Scott OM. Changes in muscle strength and EMG median frequency after anterior cruciate ligament reconstruction. Eur J Appl Physiol 2006; 98: 613–23.
39. Konishi Y, Fukubayashi T, Takeshita D. Mechanism of quadriceps femoris muscle weakness in patients with anterior cruciate ligament reconstruction. Scand J Med Sci Sports 2002; 12:371–5.

40. Green HJ and Patla AE. Maximal aerobic power: neuromuscular and metabolic considerations. Med Sci Sports Exerc 1992; 24:38-46.
41. Mujika I and Padilla S. Muscular characteristics of detraining in humans. Med Sci Sports Exerc 2001; 33:1297-303.
42. Mujika I and Padilla S. Detraining: loss of training-induced physiological and performance adaptations. Part II: Long term insufficient training stimulus. Sports Med 2000; 30:145-54.
43. Roi GS, Creta D, Nanni G, Maracci M, Zaffagnini S, Snyder-Mackler L. Return to official Italian First Division soccer games within 90 days after anterior cruciate ligament reconstruction: A case report. J Orthop Sports Phys Ther 2005; 35:52-66.
44. Fitts RH. Cellular mechanisms of muscle fatigue. Physiol Rev 1994; 74:49-94.
45. Lephart SM, Kocher MA, Fu FH, Borsa PA, Harner CD. Proprioception characteristics following ACL reconstruction. Journal of Sports Rehabilitation. 1992; 1: 188-96
46. De Luca CJ. The use of surface electromyography in biomechanics. J Appl Biomech 1997; 13:135–63.
47. Rahnama N, Lees A, Reilly T. Electromyography of selected lower-limb muscles fatigued by exercise at the intensity of soccer match-play. J Electromyogr Kinesiol (3) 2006; 16:257–63.

TABLE LEGENDS

TABLE 1. Mean group values for the endurance markers. Values are mean±1 standard deviation.

TABLE 2. Correlation coefficients (r) and coefficients of determination (r^2) between EMG activity and endurance markers.