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Kostas Patras
University of Ioannina

Giorgos Ziogas
University of Ioannina

Stavros Ristanis
University of Ioannina

Elias Tsepis
Technological Educational Institution of Patras at Aigion

Nikolaos Stergiou
University of Nebraska at Omaha, nstergiou@unomaha.edu

See next page for additional authors

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Authors

Kostas Patras, Giorgos Ziogas, Stavros Ristanis, Elias Tsepis, Nikolaos Stergiou, and Anastasios D. Georgoulis

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HIGH INTENSITY RUNNING RESULTS IN AN IMPAIRED NEUROMUSCULAR RESPONSE IN ACL RECONSTRUCTED INDIVIDUALS

Kostas Patras[1], Giorgos Ziogas[1], Stavros Ristanis[1], Elias Tsepis[1,2], Nicholas Stergiou[1,3], Anastasios D. Georgoulis[1]

[1]Orthopaedic Sports Medicine Center, Department of Orthopaedic Surgery, University of Ioannina, Greece

[2]Physical Therapy School, Technological Educational Institution of Patra at Aigion, Greece

[3]Nebraska Biomechanics Core Facility, College of Education, University of Nebraska at Omaha and College of Public Health, University of Nebraska Medical Center, USA

<u>Address for Correspondence:</u>

Anastasios D. Georgoulis, M.D
Georgiou Papandreou 2
Ioannina 45444, GREECE
Tel/Fax: +302651064980
P.O BOX 1330
Email:oaki@cc.uoi.gr

ABSTRACT

Introduction: Anterior cruciate ligament reconstruction reestablishes electromyographic activity during moderate activities such as walking but is unclear if this is also the case in sports activities such as heavy intensity running. **Methods:** Telemetric electromyographic recordings from vastus lateralis and biceps femoris muscles were performed bilaterally in nine bone-patella tendon-bone anterior cruciate ligament reconstructed athletes during two 10 minute treadmill runs, one at a heavy intensity and one at a moderate intensity. **Results:** During the high intensity run, electromyographic activity increased significantly [294.2(120.6) μV to 317.1(140.5) μV, p= 0.03] for the vastus lateralis of the intact leg while did not change for the vastus lateralis of the anterior cruciate ligament reconstructed leg [267.8(142.8) μV to 263.8(128.9) μV, p>0.05]. During the moderate intensity run electromyographic activity did not change for either leg. **Conclusions:** High intensity exercise results in an impaired neuromuscular response for the vastus lateralis muscle of the anterior cruciate ligament reconstructed leg.

Keywords: neuromuscular performance, EMG, fatigue, running

INTRODUCTION

Anterior cruciate ligament (ACL) function is associated with coordination of the muscles surrounding the knee joint [1, 2]. As a result, rupture of the ACL leads to alterations in muscle recruitment including diminished electromyographic (EMG) activity of the quadriceps and the gastrocnemius and increased EMG activity of the biceps femoris during walking [3]. In addition, rupture of the ACL generates earlier EMG activity at the biceps femoris during uphill walking [4], reduced EMG activity of the vastus lateralis and the hamstrings during running and reduction in EMG activity of the biceps femoris and hamstrings after 10 minutes of walking [5].

It has been found that reconstruction of the ACL (ACLR) re-establishes EMG activity of the operated leg towards normative values during walking and jogging [6-9]. However, previous studies have demonstrated that the ACLR quadriceps muscles exhibit reduced ability to recruit high-threshold motor units due to reduced neural drive and/or selective type II muscle fibers hypotrophy [10-14]. Thus, although the reconstructed leg may exhibit similar EMG levels with the intact contralateral leg during low demand activities such as walking, it is unclear if ACL reconstruction may affect the behavior of EMG activity during the course of a sustained high intensity running, where the need to recruit high-threshold motor units is more apparent.

Previous studies in healthy individuals demonstrated that during high intensity exercise above the lactate threshold (the point at which lactate levels accumulate in blood), the working muscles increase their EMG activity as a physiological compensatory mechanism [15-19]. However, during moderate exercise performed below the lactate threshold, muscle EMG levels remain constant across time [15, 18]. The underlying mechanism of the increased EMG activity during high intensity exercise is considered to be the enhanced recruitment of high threshold motor units as exercise progresses [16-18]. In this context, ACLR subjects may not increase their EMG activity of the involved muscles due to their above-mentioned diminished ability to recruit high threshold motor units. This may have important clinical implications for the ACLR athlete since a lack of increased EMG activation in the reconstructed muscles during high intensity exercise may indicate premature muscle fatigue and lead to decreased performance. It has been reported that the knee joint provides the major energy-absorption function during the landing phase of single-leg landing tasks,[20] and such diminished neuromuscular response of the surrounding muscles may increase the potential for re-injury, especially during the latter parts of a game when a player is fatigued [21].

Therefore, the purpose of the present study was to investigate the effect of ACLR on the muscle activation levels over time during running at two different intensities, a moderate and a high intensity. The moderate intensity was defined as running at a speed corresponding to 80% of the intensity at the lactate threshold (80%LT). The high intensity was defined as running at a speed corresponding to 40% above the intensity at the lactate threshold [22]. We hypothesized that (a) in both the intact contralateral and ACLR legs the EMG activity will not increase during ten minutes of running at moderate intensity, while (b) ten minutes of running at high intensity will increase the EMG activity at the intact contralateral leg but not at the ACLR.

METHODS

Subjects

Nine amateur male soccer players [mean (SD) age, body weight and height, 27.7 (3.5) years, 79.5 (7.3) kg and 178 (5.9) cm] were recruited for the present study. The athletes had undergone ACL reconstruction with bone-patella tendon-bone (BPTB) graft, 19.2 (5.7) months before testing. ACL reconstruction was performed sub-acutely within 6 months after the injury. All subjects had a unilateral ACL tear confirmed by MRI and arthroscopy. All patients 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 as it is recommended in the literature [23]. At the time of data collection no clinical evidence of knee pain and effusion was found in the ACL reconstructed subjects. All subjects had resumed their sports activities and agreed with the testing protocol by giving their consent to participate in accordance with the Institutional Review Board policies of our Medical School.

Clinical evaluation

Prior to any data collection, a clinical evaluation was performed in all subjects 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) [24]. These measurements were performed using 134N posterior-anterior external force at the tibia, as well as maximum posterior-anterior external force until heel clearance. Repeated anterior tractions were performed until a constant reading on the dial was registered. Negative Lachman and pivot shift tests indicated that the static knee joint stability was regained. The median Lysholm score was 95 (94-96) and the 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.

Torque measurements

For all patients, torque measurements were performed on an isokinetic dynamometer (Biodex, System-3, Biodex Medical Systems Inc., New York, USA). Approximately 45 minutes after termination of the VO_2max and lactate threshold test (at a time where blood lactate levels had returned to baseline values), subjects where tested for maximum torque output of the quadriceps and hamstrings. The subjects sat on the dynamometer chair and were secured with body straps while the hip and knee joints were flexed at 90°. For a warm-up they performed submaximal isokinetic concentric contractions by flexing and extending the knee joint. During testing five maximal concentric reciprocal knee extensions-flexions were performed at angular velocities of 60°/sec and 180°/sec with one minute rest interval between velocities. Angular velocities within this range are typically used for strength testing of ACLR subjects [27-30]. Peak torque was identified as the highest value during the five repetitions. A strength index was calculated for each angular velocity as the % ratio of reconstructed/contralateral peak torque [28]. This muscle torque testing was performed to verify that there were no persistent gross deficits that may have influenced the neuromuscular response of the subjects. We observed no such deficits.

Data collection

Subjects reported to the laboratory having abstained from caffeine or food consumption for 4 hours and without vigorous training for 24h. During warm-up the subjects performed 3 minutes walking at self-selected pace and 5 minutes jogging on a treadmill (Technogym Runrace 1200, Italy) at a speed of 1.94 or 2.22 $m \cdot sec^{-1}$ where heart rate and lactate was measured. Then, subjects performed an incremental exercise test with expired gas and heart rate analysis (CPX Ultima Series, Medical Graphics, USA) to volitional exhaustion to determine maximal oxygen uptake (VO$_2$max) and lactate threshold (LT). Prior to each test, all analyzers were calibrated according to manufacturer instructions. The initial speed was set at 2.5 or 2.78 m◌sec^{-1} depending on the subject fitness and was increased by 0.56 m◌sec^{-1} every 3 minutes until volitional exhaustion. At the end of each stage, capillary blood samples were collected and analyzed for lactate (Accutrend, Roche Diagnostics, Germany). Running was recommenced within 20 sec. Attainment of VO$_2$max was verified according to criteria established by the American College of Sports Medicine [25]. Lactate threshold was determined according to the Dmax method proposed by Cheng et al [26]. Velocities for the moderate and heavy intensity bout were determined using extrapolation from VO$_2$-velocity individual plots. The moderate intensity was set at 80% of the intensity at the lactate threshold and the heavy bout was set 40% above the intensity at the lactate threshold (40%D) [22].

In two subsequent visits to the laboratory, subjects were required to perform a 10-min run, one at the moderate and one at the high intensity [FIGURE 1]. The two runs were presented to the subjects in a random order. Prior to running, the subjects performed 3 minutes of walking at 1.39 m◌sec^{-1} and 3 minutes of jogging at 1.81 m◌sec^{-1} where baseline EMG data where collected at the first 15 seconds of each minute. During running, EMG data was collected for 15 seconds at the 3rd and 10th minute (last minute of the exercise test) [FIGURE 2]. Gas exchange data was recorded simultaneously breath-by-breath. Heart rate was measured throughout the test and blood lactate was measured at baseline (prior to running, after the completion of walking and jogging), and immediately after termination of exercise.

INSERT FIGURE 1 ABOUT HERE

INSERT FIGURE 2 ABOUT HERE

Surface electromyography was obtained from the vastus lateralis (VL) and biceps femoris (BF) muscles bilaterally using bipolar, circular, pre-amplified, pre-geld Ag/AgCl electrodes with 10 mm diameter and fixed inter-electrode spacing of 20 mm (Noraxon, USA). VL was selected on the basis that is the primary force-producing muscle during running. Furthermore the VL acts as a shock absorber during the first part of stance, thus protecting the graft from high impact forces [31]. BF was chosen due to its role in preventing anterior tibial translation and protecting the knee from the pivoting phenomenon [7]. EMG traces were recorded with a wireless 8-channel EMG system (Telemyo 2400T, Noraxon, USA) and displayed on-line on a personal computer using dedicated software (MyoResearchXP, Noraxon, USA).

The surface of the skin was prepared by shaving the hair, rubbing the skin with abrasive paper and cleaning the skin with alcohol. The electrodes were fixed longitudinally over the muscle belly. For the VL the electrodes were placed at the antero-lateral muscle bulge at 2/3 of the proximo-distal thigh length. For the BF the electrodes were placed at the dorso-lateral side of the thigh at 1/2 of the proximo-distal thigh length [32, 33]. The largest area of muscle belly was identified using a contraction against manual resistance. The ground electrode was placed on the lateral femoral condoyle of the right leg. Electrodes and cables were secured with surgical tape to avoid any interference with the running pattern of the subjects.

Footswitches (Inline Foot Contact Sensor, Noraxon, USA) were placed under the heel and big toe of both legs and were used to denote the events of the gait cycle (heel-strike and toe-off). Prior to the bout, subjects performed a “zero offset” function to establish a zero baseline from each of the EMG channels. EMG data was acquired at a sampling rate of 1500 Hz. The Root Mean Squared (RMS) amplitude was calculated for each muscle burst. Specifically, the raw EMG data measured in a band of 10 to 500 Hz, was full-wave rectified, high pass filtered with an 8th order Butterworth filter to remove movement artifacts (with a cut-off frequency of 20 Hz), and smoothed with a 100 ms RMS algorithm. Only the stance period of running was considered in the analysis and the highest value for the RMS amplitude (peak amplitude) was recorded. Values from 15 strides were averaged to calculate the mean peak amplitude during stance for each recording period. The stance period was selected for analysis because the ACL is stressed maximally during this portion of the gait cycle [34].

Statistical analysis

Based on our hypotheses, the dependent variable examined was the mean peak EMG amplitude during stance. First, the Kolmogogov-Smirnov test was calculated to ensure normality of our datasets, Subsequently, our first hypothesis was tested using paired Student's t-tests to compare the mean peak EMG amplitude between the 3rd minute of the moderate intensity bout and the mean peak EMG amplitude during the 10th minute of the moderate intensity bout. This test was performed for both the intact contralateral and ACLR legs. Similarly, our second hypothesis was tested using paired Student's t-tests to compare the mean peak EMG amplitude between the 3rd minute of the high intensity bout and the mean peak EMG amplitude during the 10th minute of the high intensity bout. Again this test was performed for both the intact contralateral and ACLR legs. The level of significance was set at a=0.05. Paired Student's t-tests were also used to compare the VO_2 at minute 3 with the VO2 at minute 10 and the blood lactate values at baseline with the blood lactate values at minute 10.

RESULTS

Electromyographic and physiological responses during the moderate intensity test

EMG amplitude of the VL did not increase for neither the intact nor the ACLR leg (p=0.43; and, p=0.222, respectively] (Table 1). For the BF, the EMG amplitude also did not increase for neither the intact nor the ACLR leg [p=0.637; and p=0.316, respectively]. The paired t-test comparisons did not reveal significant differences for VO_2 and blood lactate values between the 3rd and 10th minute (p=0.444 and p=0.161 respectively) verifying that this exercise was indeed of moderate intensity (Table 2).

Electromyographic and physiological responses during the high intensity test

The EMG amplitude of the VL for the intact leg increased significantly (p= 0.03), while for the ACLR leg remained unchanged (p=0.684) (Table 1). For the BF, the EMG amplitude did not increase for neither the intact nor the reconstructed leg (p=0.325; and p=0.107, respectively). The paired t-test comparisons revealed significant differences for VO_2 and blood lactate values between the 3rd and 10th minute (p=0.026 and p<0.001, respectively) verifying that this exercise was indeed of high intensity (Table 2).

INSERT TABLE 1 and 2 ABOUT HERE

DISCUSSION

The purpose of the present study was to investigate the effect of ACLR on the muscle activation levels over time during running at two different intensities, a moderate and a high intensity. In our study we recruited amateur competitive ACLR soccer players. Their physical characteristics revealed that had good physical conditioning but were not highly trained. To the best of our knowledge only one study has tested ACLR athletes during high intensity running and their physical characteristics were very similar to those of the athletes in the present study [35]. We hypothesized that (a) in both the intact contralateral and ACLR legs the EMG activity will not increase during ten minutes of running at moderate intensity, while (b) ten minutes of running at high intensity will increase the EMG activity at the intact contralateral leg but not at the ACLR. EMG activity was measured bilaterally at the 3rd and the 10th minute of the running tests for the VL and the BF muscles.

Regarding our first hypothesis, the EMG amplitude for the VL and BF muscles did not increase for both the intact contralateral and ACLR leg. Therefore, the results supported our hypothesis. Furthermore and during the moderate running test, VO_2 stabilized around the value obtained at minute 3. Blood lactate also did not increase above the baseline values, averaging 2.4 mM. These results verified the relatively mild physiological strain during the moderate running test [36] [FIGURE 3a]. Previous studies utilizing cycling at intensities similar to our moderate bout, have demonstrated that under moderate physiological strain, EMG activity of the quadriceps muscles did not increase during the course of the exercise [15, 17, 18]. Furthermore, previous research indicates that during moderate intensity exercise, the physiological strain imposed on the athlete is of similar profile to our data with no increase in the VO_2 and the lactate values [17, 36]. Thus, we are confident that the lack of increase in EMG during the moderate intensity running is a result of the low energetic requirements of the task.

Regarding our second hypothesis the increased physiological strain imposed on the subjects [FIGURE 3b] was accompanied with a significant (7.2%) increase in the EMG activity of the VL muscle for the intact contralateral leg. No significant change was found in the EMG activity of the VL muscle for the ACLR leg [Figure 4]. Pilot testing in healthy subjects revealed similar increases in the EMG amplitude that were in the range of 6 to 8%. The 7.2% in the ACLR group is within the expected range. Thus, the results supported our second hypothesis. During the high intensity running test, VO_2 rose well above the values at the 3rd minute. The blood lactate values at end-exercise averaged 7.2mM. These resulted indicated great physiological strain [36]. These values also verified that the high intensity exercise taxed significantly both the aerobic and anaerobic energetic pathways of the subjects in approximating “field” situations. Furthermore, the continuous monitoring of the physiological data allowed each subject to exercise at the same intensity. Previous studies that examined the relationship between EMG activity and exercise intensity indicated that the EMG progressively increases during constant exercise performed at high intensities [16-19]. The progressive increase in the EMG activity during the high intensity exercise reflects the physiological response of the muscles to the accumulating fatigue [19, 37].

INSERT FIGURE 3 ABOUT HERE

INSERT FIGURE 4 ABOUT HERE

Several explanations can be given for the lack of increase in the EMG amplitude for the VL of the reconstructed leg. These may include selective muscle fiber atrophy in the involved quadriceps [10, 13, 14], altered motor unit activation following surgery and subsequent retraining [11], and loss of joint afferent information which may lead to selective muscle fiber hypothophy [12]. These neuromuscular alterations following ACL reconstruction may have a negative impact on performance at high intensities where the need to activate high threshold motor units is more apparent [16-18, 36, 37] and may, at least in part, be responsible for the lack of increase in the EMG amplitude. Therefore, it appears that although reconstruction of the ACL re-establishes EMG activity of the operated leg towards normative values during walking and jogging [6-9], under high intensity running (that generates high levels of fatigue) the involved leg has an altered response showing no increase in EMG activity. In this context our EMG results are in accordance with recent studies suggesting that decrements of functional and neuromuscular performance after ACL reconstruction are more pronounced under fatiguing test conditions [35, 38]. Indeed, Augustsson et al reported that although ACLR subjects had similar hopping performance in the involved and intact legs under non-fatigued conditions, the performance decrements were more pronounced on the involved side as compared to the intact side when the subjects were fatigued [38].

In the present study the BF muscle of the intact leg was not affected by fatigue, showing no increase in the EMG activity during the high intensity running. Large variability in the individual behaviour was evident for this muscle. Similarly, large variability in the EMG activity of the BF has also been reported for an incremental running exercise, where the EMG amplitude of the VL increased linearly with running velocity while the EMG activity of the BF did not display any such relationship with the running velocity [39]. Therefore, it seems that during the stance phase of running the energetic requirements remain relatively stable for the BF.

Importantly and under the fatigued condition, the ACLR subjects demonstrated “asymmetry” in terms of the EMG time-course between the ACLR and contralateral intact leg. This neuromuscular discrepancy under high demand activities may potentially overload the knee joint which has a major energy-absorption contribution during the landing phase of single-leg landing tasks [20]. This may increase the possibility for re-injury, especially during the latter parts of a sporting event when fatigue rapidly accumulates [21]. In this regard, endurance training may delay fatigue occurrence and prevent the development of such neuromuscular “asymmetry” in ACLR athletes.

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. 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 was performed according to established guidelines [32, 33]. 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 other effects being studied [40]. 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 value of the EMG activity of every muscle was compared with its original value while the electrodes remained attached during the whole task, thereby overcoming the between subject variability of EMG amplitude. It is well accepted that EMG amplitude indicates the overall recruitment of motor units but does not provide direct information about the recruitment pattern of type I and II muscle fibers. However, in a recent study, McDonald et al have shown that the RMS amplitude is the most reliable and sensitive EMG variable for a fatiguing cycling exercise at a power output similar to our high intensity [19]. Finally, a limitation of the present study is the small sample size utilized which may affect statistical decisions (Type I and II errors). However, the complexity of the experiment with the long visits in the laboratory by each subject revealed problems with respect to recruitment and affected our sample size. Despite these problems we feel confident with our results since in all statistically significant decisions the effect sizes were quite large.

In conclusion, the present study demonstrated that at the end of a 10-minute run at high intensity, the VL muscle of the intact leg had significantly increased EMG activity by 7.2% (presumably as a response to compensate for the induced metabolic fatigue), while the VL of the reconstructed leg showed no such increase. It appears that under high physiological strain that simulates metabolic fatigue as in “real” conditions, the operated and the intact leg respond in different ways. Future studies should identify whether the lack of increased EMG in the VL muscle of the operated leg is accompanied with other electromyographic alterations (i.e. timing or pre-activity)

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TABLE LEGENDS

Table 1: EMG amplitude (μV) for each muscle during the running tests. Values are given as mean (SD). Asterisks (*) denotes significantly higher than VL EMG amplitude at 3rd min for the intact leg.

Table 2: Metabolic variables during the moderate and high intensity running tests. Values are mean (SD). LA refers to blood lactate values. Asterisks (*) denotes significantly higher than VO_2 3rd minute for the heavy intensity bout, $p<0.001$. Cross (†) denotes significantly higher than LA baseline for the heavy intensity bout, $p<0.0001$.

FIGURE LEGENDS

Figure 1: Subject running on the treadmill during the high intensity test. Respiratory data were measured breath-by-breath with simultaneous EMG recordings. The EMG electrodes were secured with tape to reduce movement artifacts. The configuration was kept exactly the same for the moderate and the high intensity running.

Figure 2: Typical EMG bursts of activity for the vastus lateralis muscle of the involved leg at the beginning (a) and at the end (b) of the high intensity running.

Figure 3: (a) VO_2 response during moderate running at a velocity of 8 km/h (80%LT). Note that after the 3rd minute of exercise, the VO_2 remains stable.

(b) VO_2 response during high intensity running at a velocity of 13.2 km/h (40%D). Note that the VO_2 continues to rise beyond minute 3.

Figure 4: EMG amplitude for the VL muscle. Values are mean (SD) for the ACLR and intact leg during the 3rd and 10th min.

Table 1: Mean values ± SD of the EMG amplitude (μV) for each muscle during the running tests. (N=9). SD refers to one standard deviation. VL refers to vastus lateralis and BF refers to biceps femoris.

	Moderate bout VL		Heavy bout VL		Moderate bout BF		Heavy bout BF	
	3rd min	10th min	3rd min	10th min	3rd min	10th min	3rd min	10th min
ACLR	227.0 (145.3)	210.2 (118.2)	267.8 (142.8)	263.8 (128.9)	158.3 (57.6)	165.4 (53.3)	222.4 (92.4)	233.9 (94.2)
INTACT	235.6 (103.2)	243.7 (114.8)	294.2 (120.6)	317.1* (140.5)	201.4 (155.6)	205.9 (171.4)	208.7 (110.4)	217.1 (106)

Table 2: Mean values ± SD of the metabolic variables during the moderate and high intensity running tests. (N=9). SD refers to one standard deviation. LA refers to blood lactate values.

	Velocity ($m \cdot sec^{-1}$)	VO_2 3rd minute ($ml \cdot min^{1} \cdot kg^{-1}$)	VO_2 10th minute ($ml \cdot min^{-1} \cdot kg^{-1}$)	LA Baseline (mM)	End-exercise LA (mM)
Moderate intensity bout	2.33 (0.19)	31.6 (2.8)	32.5 (2)	2.1 (0.2)	2.4 (0.6)
Heavy intensity bout	3.5 (0.25)	43.9 (2.9)	47.8* (3.7)	2.2 (0.3)	7.2† (1.8)

Figure 1: Subject running on the treadmill.

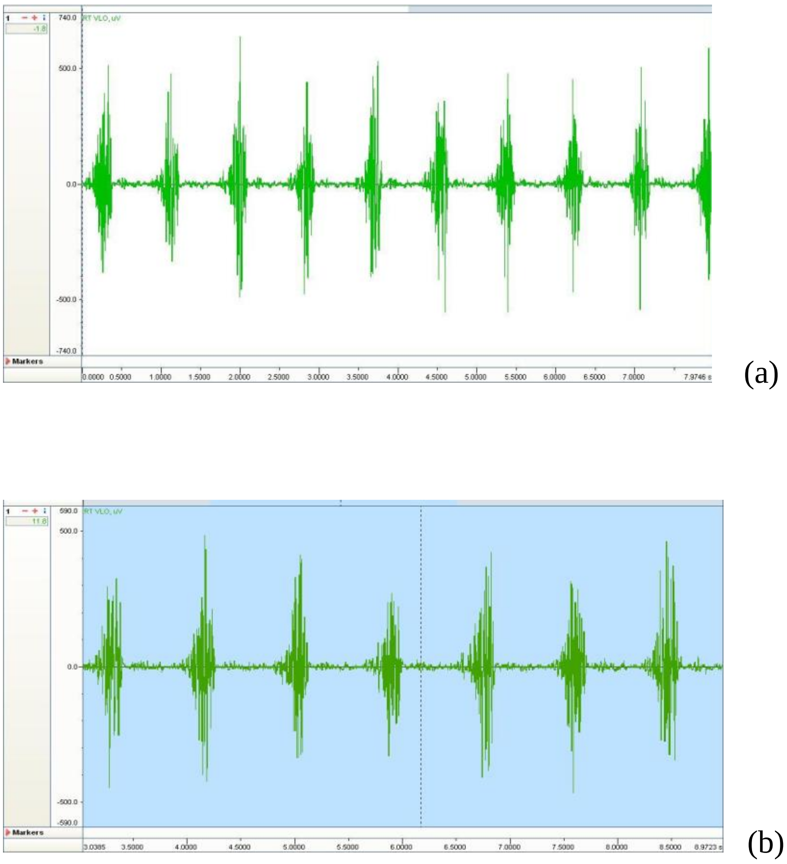

Figure 2: EMG signal of the vastus lateralis at the beginning (a) and at the end (b) of the high intensity running test treadmill running test in a typical subject.

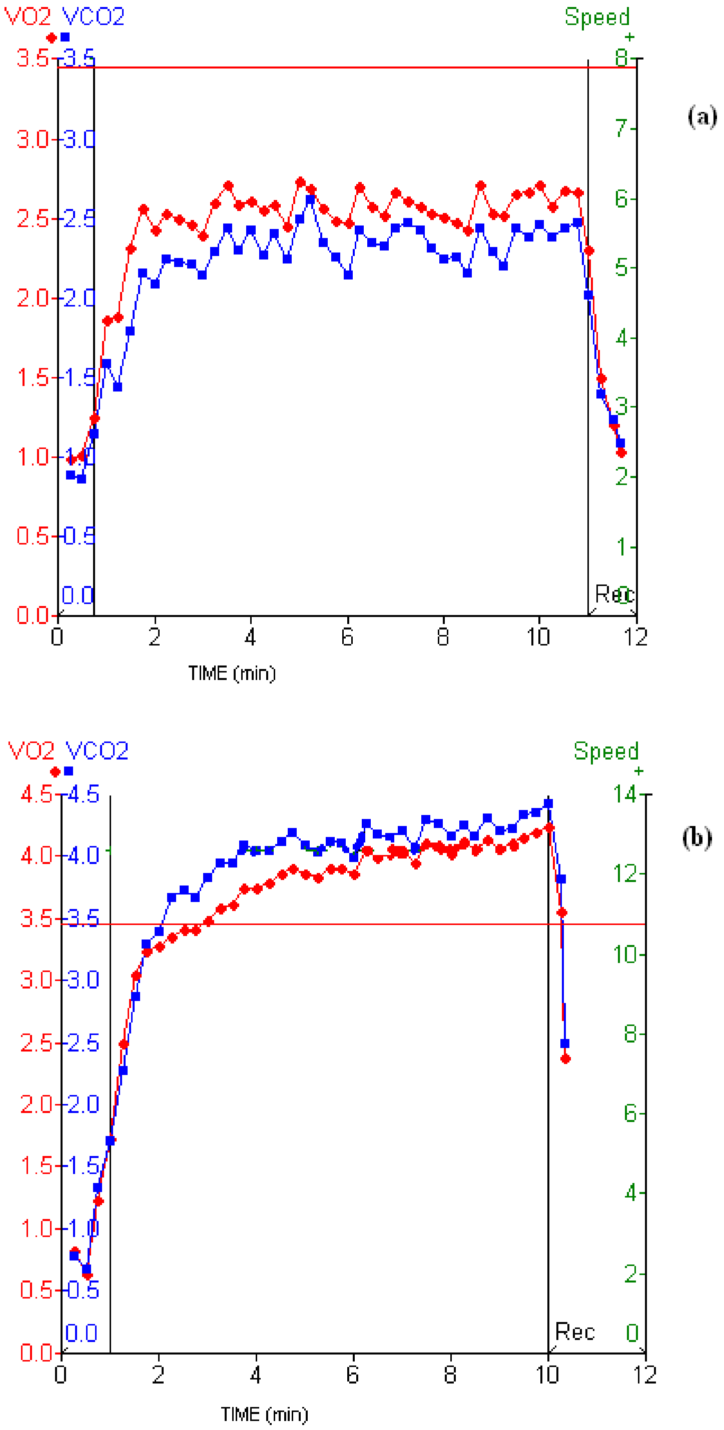

Figure 3: VO_2 response during a moderate running at a velocity of 8 km/h in a typical subject (a) and VO_2 response during a heavy running at a velocity of 13.2h in a typical subject (b).

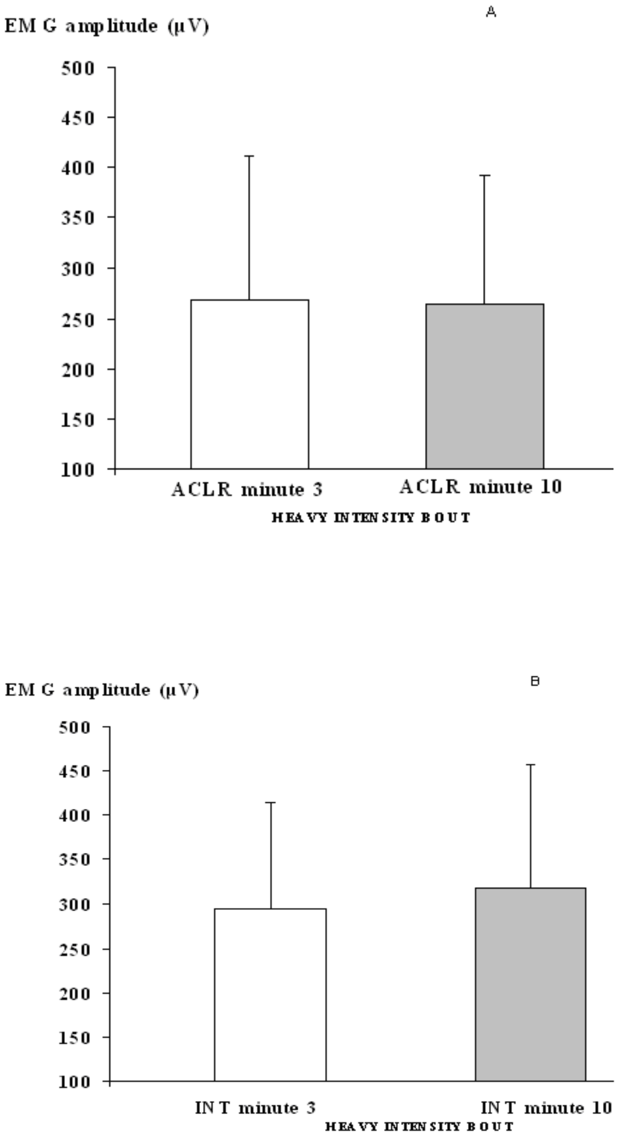

Figure 4: EMG amplitude for vastus lateralis during the heavy intensity running test for (a) the ACL reconstructed leg and (b) the contralateral intact leg. Values are presented as mean ± SD during the 3rd and 10th minute. (N=9).