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## ORIGINAL ARTICLE

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## Electromyogram as an indicator of neuromuscular fatigue during incremental exercise

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**Abstract** This study analysed the changes in the electromyographic activity (EMG) of the vastus lateralis muscle (VL) during an incremental maximal oxygen uptake test on a treadmill. A breakpoint in the integrated electromyogram (iEMG)-velocity relationship has already been interpreted in two ways: either as a sign of neuromuscular fatigue or as an expression of the iEMG-velocity relationship characteristics. The aim of this study was to test a method of distinguishing fatigue effects from those due to increases in exercise power. Eight well-trained male runners took part in the study. They completed a running protocol consisting of 4-min stages of increments in power output. Between each stage (about 15 s after the start of a minute at rest), the subjects had to maintain a standard effort: a 10-s isometric leg extension contraction [50% isometric maximal voluntary contraction (IMVC)]. The EMG was recorded during the running and isometric protocols, a change in the EMG signal during the isometric exercise being considered as the sign of fatigue. The iEMG-velocity relationships were strongly fitted by a second-order polynomial function for data taken at both the start ( $r = 0.98$ ) and the end ( $r = 0.98$ ) of the stage. Based on the stability of the 50%IMVC-iEMG relationship noted between stages, the start-iEMG has been identified as expressing the iEMG-velocity relationship without fatigue. The stage after which end-iEMG increased significantly more steeply than start-iEMG was considered as the iEMG threshold and was simultaneous with the ventilatory equivalent for carbon dioxide threshold. The parallel changes of minute ventilation and iEMG would suggest the existence of common regulation stimuli linked either to effort intensity and/or to meta-

bolic conditions. The fall in intracellular  $[K^+]$  has been discussed as being one of the main factors in regulating ventilation.

**Key words** Lactate threshold · Ventilation threshold · Surface electromyography threshold · Treadmill running · Isometric exercise

### Introduction

The transition from aerobic to anaerobic metabolism has been a subject for special focus over the last few decades. During an exercise protocol of incrementing intensity the relationships between exercise intensity and most of the parameters involved [heart rate (HR), expiratory flow ( $\dot{V}_E$ ), oxygen uptake ( $\dot{V}O_2$ ), lactacidaemia] are linear until a breakpoint which has been considered as an indicator of metabolic change. Thus, Conconi et al. (1982), Davis et al. (1983) and Wasserman (1984) have used HR, lactacidaemia and  $\dot{V}_E - \dot{V}O_2$  breakpoints, respectively to determine this metabolic change.

During the last few years a changing pattern of electromyographic (EMG) activity has been associated with this assessment of threshold. These experiments have analysed the integrated EMG (iEMG) and/or the mean power frequency (MPF) of the electromyographic signal. These two parameters are indicators of neuromuscular fatigue. It has been shown that the increase in iEMG reflects a greater motor unit recruitment to maintain the required force level (Enoka and Stuart 1992). The shift in MPF to lower frequencies has been attributed to a decrease in action potential conduction velocity over the muscle (attributable to increased acidity; Hägg 1992).

The EMG threshold has sometimes been detected during the aerobic-anaerobic transition phase at similar intensities to that of the lactate threshold (Viitasalo et al. 1985; Sihvonen et al. 1988). For these authors, the ventilatory threshold was identified at these same exercise power values.

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However, the thresholds for EMG and  $\dot{V}_E$  have more often been detected later than that for lactate. When this has been the case, the EMG threshold has occurred either at the same time as (Taylor and Bronks 1994; Mateika and Duffin 1994) or later than (Nagata et al. 1981; Moritani et al. 1993) that for  $\dot{V}_E$ . Thus, there is a discrepancy in the results although the same muscle has been studied, all authors having chosen to investigate the vastus lateralis muscle (VL). This choice was the same whether the activity tested was cycling or running. This choice has been justified because fatigue thresholds in the quadriceps and triceps surae muscles are not significantly different in both activities (De Vries et al. 1990). The discrepancies in the results obtained by these authors have undoubtedly been due to the variety of experimental protocols (continuous – Nagata et al. 1981; or discontinuous – Moritani et al. 1993) and the diversity of the methods used to determine the different thresholds. Some have used a visual method (Nagata et al. 1981); others have compared slopes which have been calculated using the EMG-exercise intensity relationship (Héjal et al. 1987). Different methods of determination have sometimes been used by the same authors (Taylor and Bronk 1994). Most of the time the detection of thresholds has been from individual data, and as a result the authors have noted problems in detecting a breakpoint (Taylor and Bronks 1994). Authors have also mentioned technical problems encountered in the collection and analysis of the EMG signal (Taylor and Bronks 1994; Héjal et al. 1987).

In spite of these difficulties, the EMG threshold has often been determined during an incremental exercise intensity test on the basis of the change in iEMG. However, this parameter can increase in relation to the level of force produced without any metabolic change occurring. Lippold (1952) was the first to identify this phenomenon during isometric contractions. Since then Taylor and Bronks (1995) have also obtained the same results during dynamic contractions. This increase in neural activity occurs during exercise of incremental intensity in response to the need to recruit progressively additional motor units.

However, the relationship between EMG and the force level does not always seem to be linear: a breakpoint has been detected. This can be explained by several different phenomena. Miyashita et al. (1981) have pointed out that muscle groups are involved in different ways according to the running velocity. It should be underlined that velocity increase has been shown to be managed differently by runners who increase the frequency and amplitude of their stride by greater or lesser amounts (Nilsson and Thorstensson 1987). Moreover, during an incremental exercise test, the succession of stages leads progressively to a state of fatigue, which is all the more inevitable since type II fibres are progressively recruited. It has been found that a failure in the fast oxidative glycolytic and fast glycolytic fibres at a certain stage causes an additional recruitment of fibres

to meet the level of power required for the test (Enoka and Stuart 1992). The additional fibre recruitment induced by fatigue can also cause a breakpoint in the EMG-exercise intensity relationship.

For all these reasons, an iEMG breakpoint observed during an incremental running test may be due either to the increase in exercise intensity and/or to the onset of neuromuscular fatigue.

The aim of this experiment was to test the subjects in such a way as to detect the effect of fatigue on the EMG signal independently of the effects due to the increase in exercise intensity. That is why an isometric effort (of identical intensity, i.e. 50% isometric maximal voluntary contraction, IMVC) between each stage was introduced. An increase in this signal would then be considered to be the onset of neuromuscular fatigue. The method used allowed us to determine an EMG breakpoint not for each individual but for a given group of subjects. The breakpoint was detected using a statistical method to minimize the effects of interindividual variations. This same method was used to determine lactate and ventilatory thresholds so that the processing of results could be the same, thus achieving the same sensitivity in the detection of all the thresholds studied.

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## Methods

### Subjects

Eight well-trained male subjects took part in this study. They were training regularly five times a week. They had previously been accustomed to treadmill running tests. Their mean age, mass and height were 24.7 (SD 3.8) years, 64.1 (SD 6.4) kg and 175.2 (SD 5.5) cm respectively. Their mean peak  $\dot{V}O_2$  was 72.3 (SD 5.1)  $\text{ml} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$  and ranged from 63.6 to 78.5  $\text{ml} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$ .

The subjects were informed of the experiment procedures and gave their written consent before the tests. Prior to the experiment, each subject underwent a general medical checkup.

### Experiment protocol

The test included two types of exercise (Fig. 1):

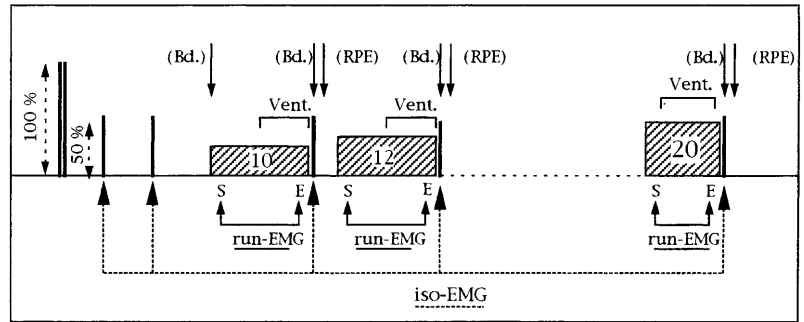
1. A running protocol performed on a treadmill, consisting of 4-min stages separated by 1-min rest periods.
2. An isometric protocol consisting of isometric voluntary contractions of the leg extensors performed at the start of the experiment and after each stage of the running protocol.

The ventilatory measurements were made during the last 2 min of each stage of the running protocol while the blood samples were collected after the isometric contractions. The EMG was recorded throughout the experiment (running and isometric protocols).

### Isometric protocol

Isometric leg extensions were performed using a dynamometer (leg extension machine). The subjects were seated in an upright position (angle of hip of 90°). Great care was taken to align the flexion-extension axis of the knee joint with the axis of the dynamometer. A belt fastened around the pelvis was used to secure the subject in the seat. The movement of the mechanical lever arm was blocked

**Fig. 1** Design of the experiment. Running protocol: vertical bars; isometric protocol: hatched area *Bd* blood samples; *Vent.* ventilatory samples; *RPE* rate of perceived exertion (Borg 1970); *EMG* electromyogram; *S*, *E* start and end of stages



by installing a metal rod that linked this mechanical lever arm to the frame of the dynamometer. No movement was possible and the knee angular position was fixed at  $120^\circ$  of knee extension ( $180^\circ$  = complete knee extension). Force exerted on the mechanical arm was measured using of a strain gauge mounted on the metal rod.

Before the running protocol the subjects performed two IMVCs of the right knee extensors at an interval of 2 min. Each contraction was maintained for 3 s. The isometric effort used subsequently consisted of 50% of the better of the two scores of the IMVC (50%IMVC). The subjects were given visual feedback of the torque produced on an oscilloscope and were instructed to maintain the 50%IMVC for 10 s. This isometric task was first performed before the running protocol, with 3-min rest between the two protocols, and then at the end of each stage of the running protocol. The mean time elapsed between the end of the stages and the 50%IMVC was less than 10 s.

#### Running protocol

The incremental exercise intensity tests were conducted on a motorized treadmill (3% gradient). The velocity was controlled by an electronic cell placed on the treadmill belt and was increased by  $2 \text{ km} \cdot \text{h}^{-1}$  in stages of 4 min until the subject felt exhausted ( $20 \text{ km} \cdot \text{h}^{-1}$  for all subjects). At the end of each stage the exercise was interrupted for a 1-min rest.

The tests began at a treadmill velocity of  $10 \text{ km} \cdot \text{h}^{-1}$  [corresponding to 57% maximal oxygen uptake ( $\dot{V}O_{2\text{max}}$ ) for this group]. Before the tests the subjects had to run for 10 min at a treadmill velocity of  $9 \text{ km} \cdot \text{h}^{-1}$  to warm up.

#### Measurements of respiratory parameters

The subjects' expired gases were collected on-line during the last 2 min of each stage and analysed using a metabolic measurement cart (CPX, Medical Graphics). The apparatus was calibrated before and after each subject's session by using gases of known concentration. The data were averaged every 10 s throughout the test. Only data obtained during the last 30 s of each stage were processed.

In addition to  $\dot{V}_E$ , the respiratory equivalents of oxygen ( $\dot{V}_E/\dot{V}O_2$ ) and carbon dioxide ( $\dot{V}_E/\dot{V}CO_2$ ) were determined as has been recommended by Davis (1985).

#### Blood sampling and lactate analysis

Immediately at the end of each stage of the treadmill session, a 50- $\mu\text{l}$  blood sample was withdrawn from the earlobe to determine lactate concentration using an automatic enzymatic method (Microzym-L, Inceltech).

#### EMG sampling and analysis

The EMG signal was picked up from the belly of VL using a bipolar configuration. The skin was scraped to ensure that skin

impedance was kept below  $1000 \Omega$ . A pair of electrodes (10 mm, Ag-AgCl, Numeris) was placed parallel to the muscle fibres. The centre-to-centre distance was 20 mm. The differential electrodes were referenced to an earth lead on the tibial tuberosity. The EMG signal was recorded continuously throughout each test, amplified ( $\times 600$ ), filtered (bandwidth 6/1500 Hz, Mazet Electronic) and stored on cassette using a digital audio tape recorder (KMT D-8 Mini type, Sony). The sampling rate was 12 kHz per channel with 12-bit data resolution. Off-line EMG recorded samples were digitized using a 1 kHz sampling rate and stored on computer disk. The EMG was then processed.

#### Iso-electromyogram

The EMG signal corresponding to 50%IMVC was analysed from the 2.5 to 9.5 s (i.e. over 7 s). The first 2.5 and the last 0.5 s were excluded to eliminate start and stop artefacts in the subject and the instrument (Fig. 2).

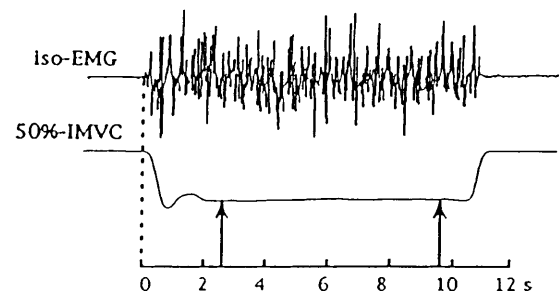
This signal was integrated and the value at each second was individually noted. This value was related to iso-iEMG data obtained for the 1st recorded during the first 50%IMVC performed 3 min before the running protocol.

A 7-s iso-EMG spectrum analysis (fast Fourier transform, 512 points) was performed on the first 13 windows. From each spectrum, the MPF was calculated.

#### Run-integrated electromyogram

The EMG signal recorded during the different running stages was analysed at the start (S) and at the end (E) of each 4-min stage (i.e. five consecutive running bursts were integrated at 20 s and 3 min 40 s, respectively). The run-iEMG values were expressed with regard to the burst duration (iEMG/burst duration) and were considered as the measures of muscle activity.

The mean value of the five running bursts was then expressed relative to those obtained at the start of the first stage.



**Fig. 2** Electromyogram and force signals recorded during a 50% isometric maximal voluntary contraction (IMVC). The arrows delimit the window where the EMG data were analysed

### Perceived exertion

Immediately after each isometric contraction, the subjects were asked to give a rating of perceived exertion (RPE) using Borg's 6/20 point scale (Borg 1970).

### Statistics and threshold determination

Changes in the data compared to changes in treadmill velocity were shown by an analysis of variance for repeated measures. The lactate, ventilatory, RPE and iso-EMG thresholds were assessed from the results of paired Student's *t*-tests including the results of two successive stages (i.e. 10 km · h<sup>-1</sup> vs 12, 12 vs 14, ... 18 vs 20). The last stage at which there was no significant difference between that and the previous one was considered as the threshold.

The run-iEMG threshold was determined by using paired Student's *t*-tests comparing the run-iEMG values between S and E of each successive stage. The last stage where no difference was detected was considered as the run-iEMG threshold.

The changes at  $P < 0.05$  were considered as significant.

## Results

### Lactate concentration

Figure 3 shows that the last non-significant interstage difference observed in analysing lactacidaemia change during the treadmill session appeared between 12 and 14 km · h<sup>-1</sup> [2.05 (SD 0.3) vs 2.6 (SD 0.38) mmol · l<sup>-1</sup>]. Therefore, the threshold was determined as being 14 km · h<sup>-1</sup>. For this group, the 4-mmol · l<sup>-1</sup> threshold (see Mader et al. 1976) was detected at 15.71 km · h<sup>-1</sup>.

### Ventilatory parameters

1.  $\dot{V}_E$  – No steady state was detected for this parameter. The 10 versus 12 km · h<sup>-1</sup> paired Student's *t*-test showed a significant difference. A second-order function ( $y = 48.7 - 3.1x + 0.4x^2$ ), where  $y = \dot{V}_E$  and  $x = \text{velocity}$  was significantly correlated ( $r = 0.99$ ).

2. Figure 3 shows  $\dot{V}_E/\dot{V}O_2$  was unchanged during the first three stages. The last non-significant difference appeared between 12 and 14 km · h<sup>-1</sup>, the  $\dot{V}_E/\dot{V}O_2$  threshold therefore being determined at 14 km · h<sup>-1</sup>.

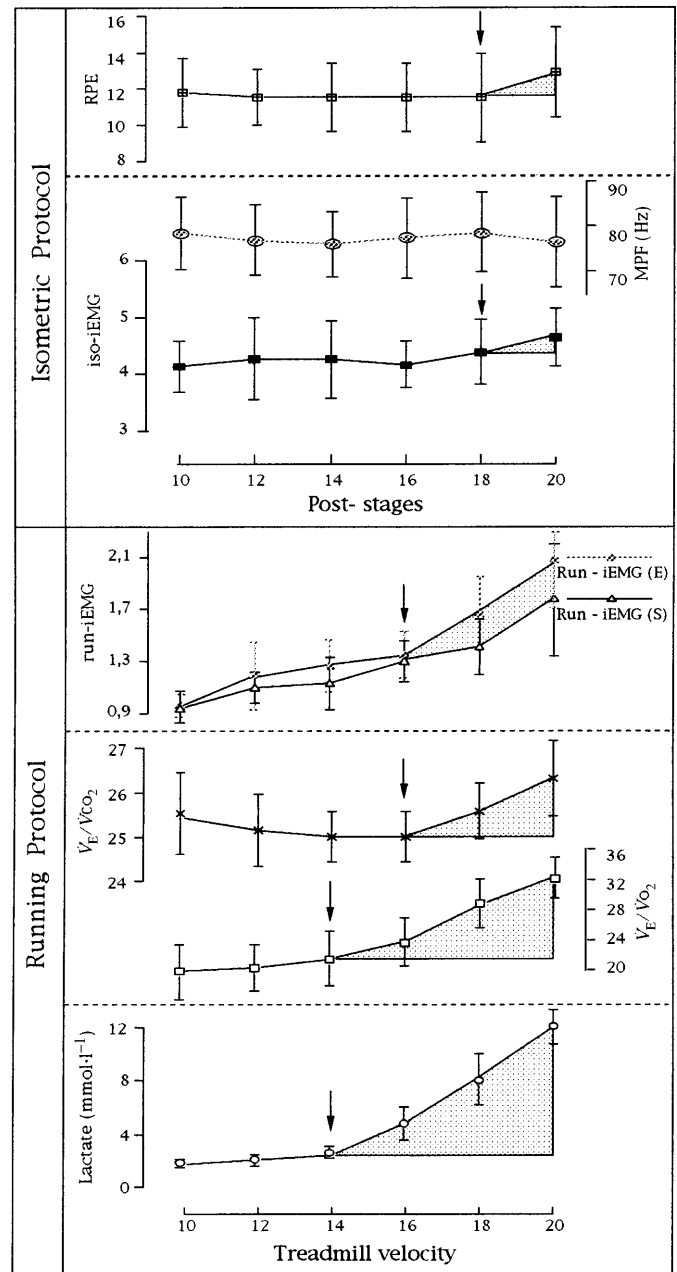
3. Figure 3 shows for  $\dot{V}_E/\dot{V}CO_2$  that the last non-significant difference appeared between 14 and 16 km · h<sup>-1</sup>, the threshold being determined at 16 km · h<sup>-1</sup>.

### EMG parameters

#### Iso-electromyogram

*Iso-integrated electromyogram.* The data collected during the 50%IMVC were analysed in two ways:

1. Intra-contraction: changes in iso-EMG during a contraction were studied to identify the initial sequence of the contraction during which the EMG



**Fig. 3** Threshold chronology observed for metabolic [lactacidaemia, ventilatory equivalents ( $\dot{V}_E/\dot{V}O_2$ ,  $\dot{V}_E/\dot{V}CO_2$ )], myoelectrical and psychological (rate of perceived exertion, *RPE*) parameters during running and isometric protocols (arrows indicate the thresholds).  $\dot{V}_E$  Minute ventilation;  $\dot{V}O_2$  oxygen uptake;  $\dot{V}CO_2$  carbon dioxide production; *iEMG* integrated electromyogram (run-iEMG values were expressed with regard to those obtained at the start of the first stage; iso-iEMG values were related to the iso-EMG obtained for the 1st s 50%IMVC performed before the running test); *MPF* mean power frequency; *S*, *E* start and end of stages

could be considered as stable and unaffected by the duration of the contraction itself.

2. Inter-contraction: the mean iso-EMG values noted during the different post-stage contractions were compared. For this analysis only sequences considered as stable by the intra-contraction analysis were used.

**Intra-contraction data.** The results of processing these data are given in Table 1. The intra-contraction comparisons for each of the iso-iEMG data showed significant differences between S and E of the contraction. For example, the iso-iEMG noted at the 7th s after the 20 km · h<sup>-1</sup> run was significantly higher than the one observed during the first 5 s. According to paired Student's *t*-tests a significant increase was usually noted from the 5th s after the 14, 18 and 20 km · h<sup>-1</sup> running stages. These results suggest that the iso-iEMG for the first 4 s of each contraction could be considered as stable. This was not the case for two points in the analysis after the 10 km · h<sup>-1</sup> stage, 1st versus 4th s and after the 18 km · h<sup>-1</sup> stages, 2nd versus 3rd s but these increases in the iso-iEMG were not maintained: in each case a decrease was observed the second after.

**Inter-contraction data.** Figure 3 shows that the iso-iEMG obtained during successive holds did not change in any significant way until after the highest intensity run (20 km · h<sup>-1</sup>): the mean value was then greater than after the 18 km · h<sup>-1</sup> stage. The iso-iEMG threshold was therefore localized at post-stage 18.

From Fig. 3 it can be noted that no intra-contraction variation was observed in iso-MPF. Accordingly an inter-contraction comparison made with the values obtained from the all of the processed signals. The mean MPF were calculated from the 13 samples processed for each isometric contraction. No significant difference was noted during this protocol although a slight decrease in MPF was observed during the last isometric contraction

[values after 18 and 20 km · h<sup>-1</sup> running stages: 74.92 (SD 9.5) Hz and 72.53 (SD 10.5) Hz, respectively].

#### Run-integrated electromyogram

The EMG data collected during the different running stages were analysed in order to study changes in the iEMG parameter between treadmill sessions (inter-stage analysis) and during each session (intra-stage analysis) (Fig. 3).

**Inter-stage analysis.** This analysis was done by comparing the results obtained during the successive stages. These comparisons were made by using the data collected at S or E of each stage.

For both S and E we noted an increase in the run-iEMG in relation to treadmill velocity. [This could be considered as a linear relationship: the correlation coefficients (*r*) were 0.95 and 0.96 for S and E respectively]. However, this increase was not consistent according to paired Student's *t*-test: the first inter-stage differences for data recorded occurred between 14 and 16 and between 10 and 12 km · h<sup>-1</sup> for the results obtained at S and E of the stages, respectively (Table 2).

A systematic significant inter-stage increase detected between the last stages (16 vs 18 km · h<sup>-1</sup> – E) and (18 vs 20 km · h<sup>-1</sup> – S and E) involved a greater increase in the run-iEMG at the end of the incremental test. Therefore the run-iEMG (S and E)-velocity relationships were strongly fitted by a second-order polynomial function (S,  $y = 1.56$

**Table 1** Integrated myoelectrical signal (iEMG) observed during the isometric protocol for each post-stage standard test (PSST) (50% isometric maximal voluntary contraction). iEMG values were related to iso-EMG obtained for the 1st s 50%IMVC performed before the running test

		Time of isometric contraction (s)						
		1st	2nd	3rd	4th	5th	6th	7th
PSST 10	mean	1.09	1.06	1.1	1.13	1.08	1.11	1.17
	SD	0.12	0.15	0.13	0.12	0.13	0.19	0.17
					<i>*1</i>			<i>*1,2,5</i>
PSST 12	mean	1.09	1.1	1.06	1.12	1.08	1.25	1.27
	SD	0.16	0.15	0.09	0.18	0.19	0.31	0.45
							<i>*1,2,5</i>	<i>*1,2</i>
PSST 14	mean	1.09	1.1	1.07	1.06	1.12	1.16	1.11
	SD	0.17	0.19	0.2	0.12	0.14	0.2	0.17
							<i>*4</i>	<i>*2,3,4</i>
PSST 16	mean	1.07	1.11	1.09	1.1	1.17	1.15	1.21
	SD	0.1	0.11	0.08	0.12	0.17	0.21	0.18
								<i>*3</i>
PSST 18	mean	1.12	1.09	1.16	1.14	1.21	1.21	1.24
	SD	0.14	0.15	0.14	0.17	0.19	0.24	0.1
						<i>*2</i>		<i>*1,2,4</i>
PSST 20	mean	1.19	1.16	1.19	1.21	1.24	1.26	1.33
	SD	0.15	0.11	0.07	0.08	0.1	0.1	0.1
						<i>*2</i>	<i>*2</i>	<i>*1,2,3,4,5</i>

\* Significant difference  $P < 0.05$  (paired Student *t*-test) between iEMG in the column and those at the time indicated in *italic*

**Table 2** Integrated electromyographic signal observed during the incremental running protocol (run-iEMG) and the results of statistical comparisons. The data are expressed as the quotient iEMG/burst duration. S, E data recorded at the start (S) and the end (E) of each stage. (*Thres* Threshold, *ns* not significant)

Run-iEMG (S)						
Mean	1.01	1.17	1.2	1.38	1.49	1.88
SD	0.12	0.12	0.2	0.16	0.22	0.46
10		ns	*	*	*	*
12			Thres	*	*	*
14				*	*	*
16					ns	*
18						*
Run-iEMG (E)						
Mean	1	1.26	1.34	1.43	1.76	2.17
SD	0.08	0.27	0.21	0.18	0.28	0.41
10		*	*	*	*	*
12			ns	ns	*	*
14				ns	*	*
16					*	*
18						*
Run-iEMG (S vs E)						
	ns	ns	ns	ns	*	*
	10	12	14	16	18	20
	Treadmill velocity (km · h <sup>-1</sup> )					

\* Significant difference  $P < 0.05$  (paired Student *t*-test)

$-0.11x + 0.006x^2$ ,  $r = 0.98$ ; E,  $y = 1.55 - 0.12x^2$ ,  $r = 0.98$ , where  $y = \text{run-iEMG}$  and  $x = \text{velocity}$ ).

**Intra-stage analysis.** The intra-stage analysis consisted in comparing the run-iEMG observed at S and E of the same stage. This paired Student *t*-test (Table 2; run-iEMG S vs E) showed that run-iEMG remained unchanged for the first four stages. The only significant increases were noted during the 18 and 20 km · h<sup>-1</sup> stages. Therefore, it was considered that the run-iEMG threshold occurred at 16 km · h<sup>-1</sup> (Fig. 3).

#### Rating of perceived exertion

According to the statistical analysis, RPE increased significantly in the last isometric contraction (i.e. post-stage 20 km · h<sup>-1</sup>). The RPE threshold was determined at 18 km · h<sup>-1</sup> (Fig. 3).

#### Threshold chronology

A threshold chronology can be established (Fig. 3): the lactate and respiratory equivalent for oxygen thresholds were the first to be detected after a phase where the different parameters considered were unchanged (from 10 to 14 km · h<sup>-1</sup>). The respiratory equivalent for carbon dioxide threshold appeared at 16 km · h<sup>-1</sup> and co-

incided with the run-iEMG threshold. The iso-iEMG was later (post-stage 18) and occurred at the same time as the RPE threshold for the isometric effort.

However, the parallel change of  $\dot{V}_E$  and run-iEMG relative to velocity noted during the incremental test must be underlined. (This is also expressed by a significant linear relationship between these parameters:  $r = 0.95$  and  $0.97$  for S- and E-run-iEMG/ $\dot{V}_E$ , respectively).

## Discussion

Even before any change in metabolism appeared (the first three stages) the run-EMG and the  $\dot{V}_E$  increased in relation to running velocity. These changes were not related either to changes in blood lactate concentration or to the appearance of the respiratory equivalent thresholds: no significant change in lactate concentration and in respiratory equivalent appeared before 14 km · h<sup>-1</sup> [changes in  $\dot{V}_E$  and EMG (E-run-iEMG) were noted as early as the comparison between the 10 and 12 km · h<sup>-1</sup> stages]. Therefore, the increase in  $\dot{V}_E$  observed during these first stages would not seem to be linked to a humoral type regulation but to a central or reflex neurogenic regulation (reflex triggered by muscular mechanoreceptor stimulation) involving both ventilatory and muscle activities.

The problem, then, is to identify the effects of regulation linked to an increase in exercise intensity and those arising from a change in metabolic conditions during an incremental running test.

In the case of ventilatory parameters, the changes in ventilatory equivalents ( $\dot{V}_E/\dot{V}O_2$  and  $\dot{V}_E/\dot{V}CO_2$ ) can be considered as indications of metabolic changes which have been interpreted as evidence of acidaemia (Wasserman et al. 1990) or as a sign of K<sup>+</sup> accumulation (Busse et al. 1991). The results of the statistical analyses enable us to pinpoint the  $\dot{V}_E/\dot{V}O_2$  and  $\dot{V}_E/\dot{V}CO_2$  thresholds at 14 and 16 km · h<sup>-1</sup> respectively.

Considering the EMG breakpoint, it is difficult to know how to discriminate the role played by an increase in exercise intensity from the one played by fatigue (see Viitasalo and Komi 1978). The detection of an EMG breakpoint has often been interpreted as a sign of type II fibre recruitment during an incremental test (Hélal et al. 1987; Mateika and Duffin 1994; Nagata et al. 1981). Nevertheless, this breakpoint may appear:

1. With no state of fatigue: the action potentials in type II fibres being more important, contributing to the relative increase in the EMG signal (see Tanji and Kato 1973).
2. In a state of fatigue: the newly recruited fibres for stages requiring a great deal of power become fatigued quickly (fast glycolytic fibres). This great fatigability can cause the recruitment of additional motor units in order to maintain the required power at the same stage.

However, a breakpoint in the EMG-exercise intensity relationship has not always been detected, whether it be with no influence of fatigue (Goto et al. 1976), or under the effect of fatigue during an incremental exercise test leading to exhaustion (Taylor and Bronks 1995).

The contradiction in these observations has led us to be cautious in the conclusions relating the type of fibres recruited with respect to the characteristics of the EMG-exercise intensity relationship.

Conversely, for a given stage, it can be stated that an increase in EMG is the result of the additional recruitment of fibres. However, this does not imply that this breakpoint indicates the activation threshold of those type II fibres. Some of these type II fibres may already have been activated at one or several earlier stages although they have shown no sign of fatigue at that time. This phenomenon is even more likely concerning our group of subjects since they were well trained and were able to develop fully the oxidative potential of their type II fibres (Henriksson and Reitman 1977).

Because of all these reasons we did not choose to make a direct analysis of the changes in the EMG signal in relation to velocity to detect fatigue onset. Our method was based on observations by Miyashita et al. (1981), who have found that the EMG was constant during the low or medium power stages of a 10-min fixed-rate intensity test. However, the EMG increased at higher power outputs, from the first minutes of exercise, indicating neuromuscular fatigue.

To determine the EMG threshold, the first occurrence of an increase in the level of electrical activity must be identified. The duration of our stages (4 min) was chosen so as to be long enough to come over the ventilatory adaptation inertia phenomena and to observe the increase in the activity level (see Miyashita et al. 1981).

According to these analyses the run-iEMG remained constant during the first four stages (10–16 km · h<sup>-1</sup>, i.e. 62%–83% ( $\dot{V}O_{2\max}$ )). The first stage (18 km · h<sup>-1</sup>) where the run-iEMG increased significantly during the 4-min run corresponded to the one where the  $\dot{V}_E/\dot{V}CO_2$  value also increased. These different increases were confirmed at the 20 km · h<sup>-1</sup> stage.

To avoid muscle activation processes related to an increase in exercise power a standard isometric contraction of the knee extensors was performed just after each running stage. These standard tests were carried out at 50% of IMVC, supposing this contraction level to be high enough to be sensitive to prior run exertion and low enough to be maintained for 10 s throughout the experiment in accordance with the findings of Arendt-Nielsen and Mills (1988), who have demonstrated that this contraction level can be maintained for 50 s without decline. The 50%IMVC is also, according to Duchêne and Goubel (1993), the force level from which the changes in EMG signal spectrum parameter can be more systematically observed.

For the standard tests, the changes in the EMG signal were later than all those discussed above. Only a significant increase in iso-iEMG appeared during the test

carried out after the last stage reached by the subjects (iso-iEMG threshold: 18 km · h<sup>-1</sup>) while the spectrum parameters showed no significant changes.

The stability in EMG noted during the isometric contraction, and during the period averaging about 15 s following the end of stages 10–18 km · h<sup>-1</sup> of the running protocol, would imply all the more so that it would also have been stable if tested 45 s later (i.e. at the end of the inter-stage rest time). Thus the iEMG noted at S of each stage may be considered to have been determined by exercise intensity and not affected by fatigue. On the basis of these results the iEMG-treadmill velocity relationship obtained at S of the stage represents the strict EMG-intensity relationship. Consequently, the increase in the iEMG noted during the same stage can be seen as a sign of local neuromuscular fatigue. The analyses of EMG changes recorded during the running and isometric protocols can therefore be considered as complementary.

The main purpose of the method applied in this study was to identify a relative instability not only in the EMG parameters but also in the ventilatory ones by taking into account variations in respiratory equivalents. We have seen that different methods have been used to identify these breakpoints. These differences in the methods used in the same experiment can lead to distortion because the different tests have different sensitivities. To solve this problem all our data were processed in the same way, based on variations observed at group level rather than individually and the same method was applied whatever the nature of the parameter to be analysed. This procedure requires a homogeneous experimental group, which explains why we chose well-trained athletes of similar ability: all the subjects were able to finish the incremental test at the same stage (20 km · h<sup>-1</sup>) with limited inter-individual variation in ( $\dot{V}O_{2\max}$ ) [72.3 (SD 5) ml · min<sup>-1</sup> · kg<sup>-1</sup>]. However, as Taylor and Bronks (1995) have found, there was a greater inter-individual variability in the EMG parameters. The run-iEMG values reached at the last stage were 1.88 (SD 0.47) and 2.17 (SD 0.42) at S and E of the stage, respectively (values normalized relative to the first stage).

The results obtained and shown in an exercise intensity continuum revealed that the lactacidaemia and  $\dot{V}_E/\dot{V}O_2$  thresholds both occurred at 14 km · h<sup>-1</sup>. The 14 km · h<sup>-1</sup> stage, where the level of lactacidaemia reached 2.6 (SD 0.3) mmol · l<sup>-1</sup> while the  $\dot{V}_E/\dot{V}O_2$  was still constant, would correspond to the limit of the aerobic phase. The intra-stage constancy of the run-iEMG signal could be considered as a sign of metabolic equilibrium. The 16 km · h<sup>-1</sup> stage, where the respiratory equivalent for  $\dot{V}_E/\dot{V}O_2$  increased significantly while the respiratory equivalent for the  $\dot{V}_E/\dot{V}CO_2$  was still constant, would correspond to the isocapnic buffering allowing suitable metabolic conditions for muscular contractions to be maintained. An intra-stage constancy in run-iEMG was noted for this stage where the lactacidaemia was close to 4 mmol · l<sup>-1</sup>. Above this stage,



(from 18 to 20 km · h<sup>-1</sup>), the buffering system would have been weakened leading to a deterioration in the metabolic conditions for contractions. These phenomena were simultaneous with the intra-stage increase in the run-iEMG signal.

$\dot{V}_E/\dot{V}O_2$	---	78.3%	---	87.6%	---	100%
		Lactacidaemia		Run-iEMG		
		$\dot{V}_E/\dot{V}O_2$		$\dot{V}_E/\dot{V}CO_2$		

These results show a later iEMG threshold than has been noted by Viitasalo et al. (1985) or Sihvonen et al. (1988). For these authors, the iEMG threshold during a cycle ergometer incremental exercise test was simultaneous with the lactic acid and ventilatory thresholds (respiratory equivalent for  $\dot{V}_E/\dot{V}O_2$ ). Mateika and Duffin (1994) have recorded an iEMG threshold appearing earlier than the first ventilatory threshold. However, for other authors such as Nagata et al. (1981), Moritani et al. (1993) and Taylor and Bronks (1994), the iEMG threshold has occurred after the first ventilatory threshold.

The distinctive feature of our results, i.e. the late occurrence of the iEMG threshold, can be explained by the method chosen, which allowed us to minimize the effect of the exercise power. The fact that the iEMG threshold appeared later on is perhaps, within the framework of our experiment, due to the characteristics of the subjects studied. They were well-trained athletes specializing in the activity in which they were evaluated (recall their high  $\dot{V}O_{2\max}$  values). Their level of training provided them with good respiratory control and they were able to adapt well both from a general and local viewpoint.

These observations, made after having minimized the effect of the increase in exercise power, confirm the fact that humoral factors influence both ventilation mechanisms and muscle contraction processes. The main factors are probably "a decrease in muscle pH and elevated extracellular potassium concentration" as stated by Vogiatzis et al. (1996). The fall in muscle pH causing inhibition in metabolic regulation as well as a decrease in the excitability of the muscle cell leads to a decrease in the mechanical effect of the contraction. Thus, to maintain a particular force level more motor units must be recruited. This fall in muscle pH, leading to a fall in blood pH also acts indirectly on the arterial chemoreceptors linked to ventilation regulating mechanisms. The intra-cellular acidosis has also been favoured by a fall in intercellular  $[K^+]$  that can reach 10–20% in VL after an exhausting exercise (Sjogaard 1986). Moreover, the variations in  $[K^+]$  themselves represent a stimulus. The cellular variations in  $[K^+]$  stimulate muscular chemosensitive groups III and IV nerve fibres, and similarly have been shown to take part in ventilation regulation (Vogiatzis et al. 1996). It has been suggested that this action is probably strengthened by the stimulation of arterial chemoreceptors (Paterson and Nye 1991). Thus, the variations in  $[K^+]$  are ventilation regulating stimuli by reflex loops different to those responding to pH. This

suggests a strong relationship between  $\dot{V}_E$  and  $[K^+]$ , as has been reported by Busse et al. (1991).

However, the conclusion about the comparative time-courses of the different parameters must be regarded with caution. It is important to underline the difference in the measurement frequency of these parameters (see Mateika and Duffin 1994) and the different diffusion speeds and the delays between the intra- and extra-cellular phenomena. Finally, as in most other experiments of this nature, our results are based on EMG of VL. It seems necessary to check these results against muscles recruited differently during a running protocol to have a more general view of the neuromuscular fatigue threshold.

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