

$\dot{V}O_2$ decrease Before Exhaustion During Constant Load Exercise. Role of Respiratory Muscles

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ABSTRACT

Objectives: The aim of our work was to analyse the $\dot{V}O_2$ kinetic during a constant load exercise, to check the existence of a $\dot{V}O_2$ decrease at the end of this kind of exercise and finally to study the respiratory muscles strength evolution, before and after this kind of exercise. **Patients and Methods:** Eight endurance trained athletes (20.6 ± 2.7 yrs) performed three field-tests until exhaustion: firstly a maximal graded test to determine their maximal oxygen uptake ($\dot{V}O_{2max}$) and maximal aerobic velocity (MAV) and secondly two constant velocity exercises on track at 100% (t_{lim100}) and 95% of MAV (t_{lim95}) until exhaustion. **Results:** Our study outcomes revealed a $\dot{V}O_2$ decrease before the end of exercise for three subjects. The mean decrease duration was 51.3 ± 13.4 s and represented 8.3 ± 2.1 % of the total exercise duration. Maximal inspiratory and expiratory pressures (PI_{max} and PE_{max}), measured before and after exercise were considered as respiratory muscle strength indices and were not significantly different before or after the exercise. **Conclusion:** The existence of a $\dot{V}O_2$ decrease before the end of the exercise, already highlighted in the literature is confirmed. Our results indicated that respiratory muscle fatigue was not explicative for $\dot{V}O_2$ decrease. However, further studies are necessary to confirm these results.

Keywords: Continuous exercises, $\dot{V}O_2$ decrease, respiratory muscle strength, maximal respiratory pressures

BACKGROUND

During exercise, muscle and pulmonary $\dot{V}O_2$ uptake ($\dot{V}O_2$) increase approximately exponentially to a steady state until the end of exercise [1]. However, Perrey et al. [2] during a continuous exercise realized on treadmill at 95% of $\dot{V}O_{2max}$ (velocity associated with $\dot{V}O_{2max}$), observed a $\dot{V}O_2$ decrease before exhaustion for 7 of their 13 subjects (54 %). This $\dot{V}O_2$ decrease before exhaustion had been also found by Astrand

and Saltin [3], Nummela and Rusko [4] and Heubert et al. [5] during a maximal constant load exercise. Nevertheless, this decrease is merely limited to an observation in these studies. More recently, Thevenet et al. [6] during intermittent exercise (105% of maximal aerobic velocity (MAV) alternated with passive recovery) with trained adolescents, also showed a $\dot{V}O_2$ decrease before exhaustion. According to these authors, this result could be explained by a minute ventilation (\dot{V}_E) decrease. In their study, Perrey et al. [2] also suggested that ventilatory system deterioration could explain $\dot{V}O_2$ decrease before exhaustion. Unfortunately, these authors did not highlight specific characteristics for subjects with a $\dot{V}O_2$ decrease. Considering respiratory muscles fatigue as a condition in which there is a loss in the capacity for developing force of muscle, which is reversible by rest [7], we hypothesized that the respiratory muscle strength loss could be the origin of

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$\dot{V}O_2$ decrease during continuous exercise. Maximal respiratory pressures are considered a good index of respiratory muscle strength [8]. Moreover, at the end of the exercise, respiratory frequency (fr) increase could be insufficient compared to tidal volume (V_T) decrease and hence, according to the relationship $\dot{V}_E = V_T \times \text{fr}$, could be responsible for \dot{V}_E decrease [9].

OBJECTIVES

Then, the aim of our work was to analyse the $\dot{V}O_2$ kinetic during a constant load exercise, to check the existence of a $\dot{V}O_2$ decrease at the end of this kind of exercise and finally to study the respiratory muscles strength evolution, before and after this kind of exercise.

PATIENTS AND METHODS

Subjects

Eight male physical education students (mean age 20.6 ± 2.7 yrs) volunteered to participate in this study. All were from the same athletic club and regularly practised athletics for at least 3 years. Subjects were 19 to 27 years old. Their mean \pm SD for mass, height and percentage of fat were 70.5 ± 3.1 kg, 180.2 ± 6.2 cm and 12.4 ± 2.2 %, respectively. Before testing, the subjects underwent a medical examination and were fully informed of the experimental procedures and a signed consent was obtained from the subjects. The inclusion criteria required for subjects was the absence of cardiovascular diseases; pre- or diabetes risk and hypertension (i.e., blood pressure $> 140/90$ mmHg) and absence of electrocardiogram abnormalities. This study had been approved by the University of Nantes Research Ethics Committee.

Overview

Subjects performed three field-tests until exhaustion on a 400-m outdoor tartan track at the same time of the day, with at least 48h rest between each test [10]. Atmospheric conditions were checked before each test ensuring that all sessions were carried out under similar environmental conditions (wind speed lower than 2.5 m.s^{-1} ; temperature between 18 and 23°C ; humidity between 40 and 70%). Athletes first performed a maximal graded test to determine $\dot{V}O_{2\text{max}}$ and MAV. Then, they performed in a randomized order two continuous exercises until exhaustion at 100% ($t_{\text{lim}100}$) and 95% ($t_{\text{lim}95}$) of MAV. During all tests, we used the Cosmed K4b²

breath-by-breath portable metabolic system (Cosmed K4b², Rome, Italy; [11]) in order to determine \dot{V}_E , V_T , fr and $\dot{V}O_2$. Further details about the system are provided elsewhere [12]. The K4b² was calibrated before the beginning of each test according to the manufacturer's guidelines. Heart rate (HR) was continuously monitored (Polar Electro, Kempele, Finland).

Maximal Static Mouth Pressure Measurements

Maximal respiratory pressures, considered as a good index of respiratory muscle strength, can be used in order to appreciate respiratory muscle fatigue [13]. Maximal inspiratory (PI_{max}) and expiratory (PE_{max}) pressures were respectively measured at residual volume (RV) and total pulmonary capacity (TPC) with a ZAN betterflow portable device (Flowhandy ZAN 100, Messgeraete GmbH, Germany) using the technique of Black and Hyatt [14]. This measure was realized in the athletics stadium, just next to the athletics tracks, by the same experimenter at rest and 3 min after the end of the test. In each case, PI_{max} and PE_{max} were measured 5 times respectively. The highest and lowest values were rejected and the three others were averaged for data processing [15]. Maximal pressures were generated at the mouth as previously detailed [16].

Maximal Graded Test

Red cones were set at 20 m intervals along the track (inside the first line). The initial speed of the maximal graded test was 8 km.h^{-1} and was increased by 1 km.h^{-1} every 2 min [17], to determine $\dot{V}O_{2\text{max}}$, MAV, peak minute ventilation ($\dot{V}_{E\text{max}}$) and peak respiratory frequency (fr_{max}). $\dot{V}_{E\text{max}}$ and fr_{max} were determined at the corresponding time associated with $\dot{V}O_{2\text{max}}$. The determination methods of MAV and $\dot{V}O_{2\text{max}}$ have been extensively described elsewhere [6].

Constant Load Exercises and Breathing Pattern Measurement

Athletes performed two constant load exercises until exhaustion (t_{lim}): a 100% of MAV constant exercise ($t_{\text{lim}100}$) to confirm the MAV values and a constant load exercise at 95% of MAV ($t_{\text{lim}95}$) to study $\dot{V}O_2$ decrease and its link with maximal respiratory pressures. For $t_{\text{lim}95}$, \dot{V}_E , V_T and fr values were averaged over a 2s period. Then, the values were averaged over 20 periods, each corresponding to 5% of the individual t_{lim} duration. The time course of \dot{V}_E , V_T , fr are presented on figure 1 for a representative subject. We also calculated the last minute \dot{V}_E and fr values for $t_{\text{lim}95}$.

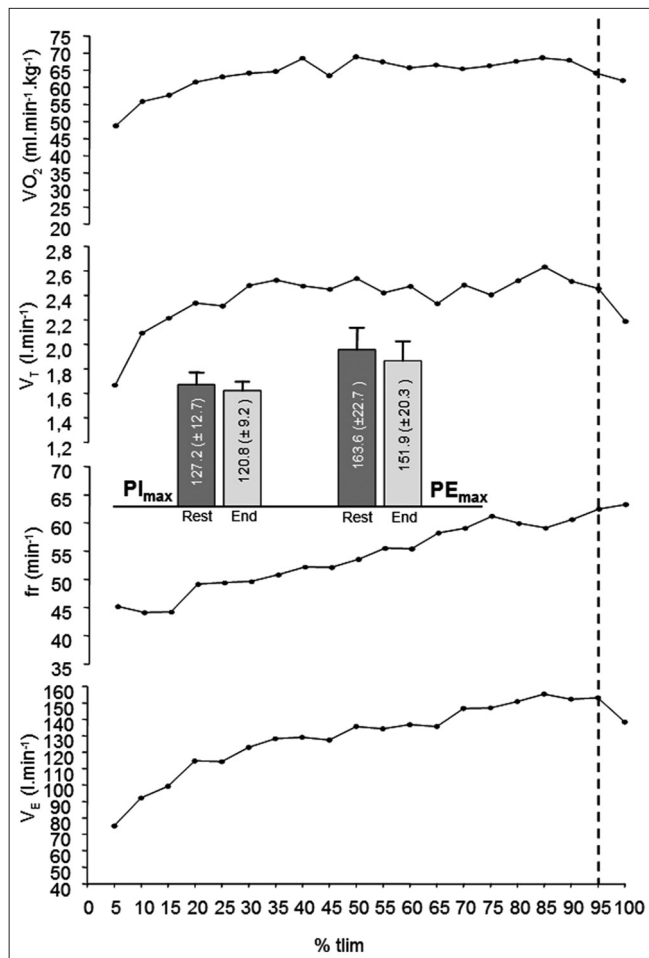


Figure 1: $\dot{V}O_2$, breathing pattern and maximal respiratory pressures for t_{lim95} in subject S2

$\dot{V}O_2$: Oxygen uptake, V_T : tidal volume, fr: respiratory frequency, \dot{V}_E : minute ventilation; PI_{max} and PE_{max} : Maximal inspiratory and expiratory pressures. The vertical line shows the \dot{V}_E decrease beginning (95% t_{lim})

$\dot{V}O_2$ Kinetic Modelling

Exercise data before recovery were analysed using Matlab® (Mathworks, Natick, MA). The cardio-respiratory values were averaged on a 2 s periods and then smoothed thanks to a gaussian sliding mean processing along a 10 s wide window. The second order model also usually called a mono-exponential function that best fits the $\dot{V}O_2$ kinetic curve obtained was identified. Finally, a Kalman filter and a algorithm of abrupt changes detection [18] were used in order to detect a local loss of adequacy between the model estimated and the measured $\dot{V}O_2$ kinetic. If such a change was detected at the end of $\dot{V}O_2$ kinetics, the algorithm computed the best linear approximation of this phase ($\dot{V}O_2$ slope), meaning the part of the curve after the change. Based on $\dot{V}O_2$ slope values, we calculated amplitude ($ml.min^{-1}.kg^{-1}$) and duration (s) values to characterize the $\dot{V}O_2$ decrease [2].

Speed Control

The tests were performed on an athletic track equipped with cones every 20 m. During both constant load exercise and maximal graded test, running speed was maintained constant thanks to an experimenter on bicycle that the subject followed. Firstly, the latter experimenter was provided with a mp3 device (located in a bag carried across his shoulder by the experimenter and connected to him by headphones) giving an imposed time signal every 20 m and took care of the subject position on a level with the aft wheel. Secondly, another experimenter also paid attention to the subject position and to lap time.

Statistical Analysis

Mean PI_{max} and PE_{max} values were compared using a paired t-test. A linear regression model was used to assess the relationship between PI_{max} , PE_{max} both measured at the end of t_{lim95} and the duration of t_{lim95} . Normal Gaussian distribution of the data was verified with the Kolmogorov-Smirnov test (with Lilliefors's correction). For all statistical analyses, the level of significance was set at $p < 0.05$. Statistical analyses for $\dot{V}O_2$, V_T , \dot{V}_E and fr were not provided since they are not consistent regarding the weak number of subjects. Effect sizes (ES) were evaluated from the Cohen's d. ES of ≤ 0.2 , 0.21-0.60, 0.61-1.20, 1.21-2.0, ≥ 2.0 were respectively considered as trivial, small, moderate, large and very large [19].

RESULTS

Maximal Graded Test

Mean values for MAV, $\dot{V}O_{2max}$, R_{max} , HR_{max} , $\dot{V}_{E,max}$ and fr_{max} were: $18.4 \pm 0.6 km.h^{-1}$, $58.1 \pm 3.5 ml.min^{-1}.kg^{-1}$, 1.2 ± 0.1 , $192.1 \pm 6.2 bpm$, $151.8 \pm 11.1 l.min^{-1}$ and $55.1 \pm 6.5 min^{-1}$ respectively (Table 1). In subjects with $\dot{V}O_2$ decrease, mean values of MAV ($18.5 \pm 0.9 km.h^{-1}$), $\dot{V}O_{2max}$ ($56.3 \pm 4.3 ml.min^{-1}.kg^{-1}$), R_{max} (1.2 ± 0.0), HR_{max} ($190.7 \pm 7.6 bpm$), $\dot{V}_{E,max}$ ($148.4 \pm 5.7 l.min^{-1}$) and fr_{max} ($57.9 \pm 5.6 min^{-1}$) did not present any particularly higher or lower values compared with mean values of MAV ($18.4 \pm 0.5 km.h^{-1}$), $\dot{V}O_{2max}$ ($59.2 \pm 2.8 ml.min^{-1}.kg^{-1}$), R_{max} (1.2 ± 0.1), HR_{max} ($193.0 \pm 5.9 bpm$), $\dot{V}_{E,max}$ ($153.9 \pm 13.6 l.min^{-1}$) and fr_{max} ($53.4 \pm 7.0 min^{-1}$) in subjects without $\dot{V}O_2$ decrease.

Constant Load Exercises

Mean values for t_{lim100} were $363.4 \pm 56.8 s$ and ensured the subjects MAV values. Mean values of t_{lim95} was

564.7 \pm 75.3 s. Table 2 shows last minute \dot{V}_E and fr values during t_{lim95} . The higher last minute \dot{V}_E (149.4 \pm 10.3 l.min⁻¹ vs 147.3 \pm 12.4 l.min⁻¹) and fr (54.4 \pm 7.9 min⁻¹ vs 55.0 \pm 6.9 min⁻¹) values were not systematically observed in subjects with $\dot{V}O_2$ decrease.

$\dot{V}O_2$ Decrease Characteristics

The model used did reveal a slope at the end of exercise for subjects 2, 3 and 5 during t_{lim95} (Table 3), illustrating a $\dot{V}O_2$ decrease for these subjects. The

mean duration of the $\dot{V}O_2$ decrease (DD) was 51.3 \pm 13.4s and corresponded in mean to 8.3 \pm 2.1% of total exercise duration.

Respiratory Muscle Fatigue

$\dot{V}O_2$, \dot{V}_E , fr and V_T expressed relatively to time to exhaustion for t_{lim95} are shown in figure 1. Only representative subject S2 is represented in this figure. This figure also shows individual values of PI_{max} and PE_{max} measured before and at the end of the exercise.

Table 1: Maximal graded test parameters

| Subjects | MAV (km.h ⁻¹) | $\dot{V}O_{2max}$ (ml.min ⁻¹ .kg-1) | R_{max} | HR _{max} (bpm) | \dot{V}_{Emax} (l.min ⁻¹) | fr _{max} (min ⁻¹) |
|---------------------|---------------------------|--|-----------|-------------------------|---|--|
| S2 | 19.0 | 61.1 | 1.2 | 196.0 | 145.1 | 61.1 |
| S3 | 17.5 | 52.9 | 1.2 | 194.0 | 145.0 | 51.5 |
| S5 | 19.0 | 54.8 | 1.2 | 182.0 | 155.0 | 61.3 |
| Mean \pm SD (n=3) | 18.5 | 56.3 | 1.2 | 190.7 | 148.4 | 57.9 |
| | 0.9 | 4.3 | 0.0 | 7.6 | 5.7 | 5.6 |
| S1 | 19.0 | 58.0 | 1.2 | 185.0 | 156.7 | 55.8 |
| S4 | 18.5 | 63.3 | 1.2 | 196.0 | 141.7 | 45.6 |
| S6 | 18.5 | 59.6 | 1.0 | 200.0 | 150.6 | 57.8 |
| S7 | 17.5 | 59.6 | 1.3 | 195.0 | 144.5 | 46.5 |
| S8 | 18.5 | 55.7 | 1.2 | 189.0 | 176.0 | 61.3 |
| Mean \pm SD (n=5) | 18.4 | 59.2 | 1.2 | 193 | 153.9 | 53.4 |
| | 0.5 | 2.8 | 0.1 | 5.9 | 13.6 | 7.0 |
| Mean \pm SD (n=8) | 18.4 | 58.1 | 1.2 | 192.1 | 151.8 | 55.1 |
| | 0.6 | 3.5 | 0.1 | 6.2 | 11.1 | 6.5 |
| ES (Cohen's d) | 0.17 | 0.83 | 0.00 | 0.37 | 0.50 | 0.69 |

MAV: maximal aerobic velocity; $\dot{V}O_{2max}$: maximal oxygen uptake; R_{max} : maximal respiratory exchange ratio; HR_{max}: maximal heart rate; \dot{V}_{Emax} : maximal minute ventilation; fr_{max}: maximal respiratory frequency. ES: effect size (Cohen's d)

Table 2: Minute ventilation and respiratory frequency values during t_{lim95}

| Subjects | t_{lim95} (s) | Last minute \dot{V}_E (l.min ⁻¹) | % \dot{V}_{Emax} | fr (min ⁻¹) | %fr _{max} |
|---------------------|-----------------|--|--------------------|-------------------------|--------------------|
| S2 | 510.0 | 151.3 | 104.3 | 61.5 | 100.7 |
| S3 | 624.0 | 138.3 | 95.4 | 45.8 | 88.9 |
| S5 | 742.0 | 158.6 | 102.3 | 55.9 | 91.2 |
| Mean \pm SD (n=3) | 625.3 | 149.4 | 100.7 | 54.4 | 93.6 |
| | 116.0 | 10.3 | 4.7 | 7.9 | 6.3 |
| S1 | 474.0 | 143.9 | 91.8 | 52.4 | 93.9 |
| S4 | 586.0 | 127.7 | 90.1 | 44.3 | 97.1 |
| S6 | 632.0 | 159.9 | 106.2 | 60.4 | 104.5 |
| S7 | 648.0 | 154.9 | 107.2 | 57.7 | 124.1 |
| S8 | 479.0 | 149.9 | 85.2 | 62.2 | 101.5 |
| Mean \pm SD (n=5) | 563.8 | 147.3 | 96.1 | 55.4 | 104.2 |
| | 82.9 | 12.4 | 9.9 | 7.2 | 11.8 |
| Mean \pm SD (n=8) | 564.7 | 148.1 | 97.8 | 55.0 | 100.2 |
| | 75.3 | 10.9 | 8.3 | 6.9 | 11.0 |
| ES (Cohen's d) | 0.82 | 0.19 | 0.55 | 0.14 | 0.96 |

\dot{V}_E : mean last minute ventilation expressed in l.min⁻¹ and relatively to \dot{V}_{Emax} (determined during maximal graded test - % \dot{V}_{Emax}); fr: mean last minute respiratory frequency expressed in min⁻¹ and relatively to fr_{max} (determined during maximal graded test - % fr_{max}). ES: effect size (Cohen's d)

Table 3: $\dot{V}O_2$ decrease characteristics during t_{lim95} for subjects S2, S3 and S5

| Subjects | t_{lim} (s) | TD (s) | DD (s) | %DD | Δ (ml.min ⁻¹ .kg ⁻¹) | % Δ |
|---------------------|---------------|---------------|--------------|------------|--|------------|
| S2 | 510.0 | 474.0 | 36.0 | 7.1 | 2.7 | 5.6 |
| S3 | 624.0 | 546.0 | 78.0 | 12.5 | 2.5 | 5.8 |
| S5 | 742.0 | 702.0 | 40.0 | 5.4 | 5.6 | 12.8 |
| Mean \pm SD (n=3) | 625.3 67.0 | 574.0 67.3 | 51.3 13.4 | 8.3 2.1 | 3.6 1.0 | 8.1 2.4 |

t_{lim95} : time to exhaustion performed at 95% of MAV; TD: time delay of $\dot{V}O_2$ decrease beginning; DD: decrease duration; %DD: percentage of decrease duration
 t_{lim95} : Δ : decrease amplitude; % Δ : percentage of $\dot{V}O_2$ decrease amplitude relatively to the total $\dot{V}O_2$ amplitude A, considered as the difference between $\dot{V}O_2$ plateau and $\dot{V}O_2$ at rest ($t=0$)

The vertical line in the figure represents the onset of \dot{V}_E decrease and is linked to the other parameters. Despite the lack of statistical analyse in the three subjects with a $\dot{V}O_2$ decrease, V_T and \dot{V}_E seemed to decrease and fr seemed to increase at the end of t_{lim95} .

Mean values of maximal inspiratory and expiratory pressures values (PI_{max} and PE_{max}) measured at rest (rest) and at the end of the exercise (end) are shown in figure 2. Statistical results did not highlight any significant evolution of PI_{max} ($d = 0.21$) and PE_{max} ($d = 0.20$) values between the rest and the end of the exercise during t_{lim95} . No significant relationship was found between PI_{max} [$=83.8 + (0.063 \times t_{lim95})$, $r^2 = 0.05$, $p=0.587$], PE_{max} [$=-6.96 + (0.27 \times t_{lim95})$, $r^2 = 0.19$, $p=0.27$], both measured at the end of t_{lim95} , and the duration of t_{lim95} .

DISCUSSION

The aim of our work was to analyse the $\dot{V}O_2$ kinetic during a constant load exercise, to check the existence of a $\dot{V}O_2$ decrease at the end of this kind of exercise and finally to study the respiratory muscles strength evolution, before and after this kind of exercise. Our study outcomes reveal a $\dot{V}O_2$ decrease before the end of exercise for three subjects. From a methodological point of view, firstly, $\dot{V}O_2$ data were averaged on a two seconds period. Data were analysed with Matlab® software and a second order model was applied to $\dot{V}O_2$ kinetics. An ad-hoc filtering process, based on Kalman filter was then used in order to detect the changes of model relatively to $\dot{V}O_2$ kinetic. When, and only when, a series of changes was detected at the end of $\dot{V}O_2$ kinetic, the software applied a linear phase. This latter was only applied after a $\dot{V}O_2$ steady state detection. As the linear phase is based on several decreasing plots, we conclude that the decrease observed is not due to measurement artefacts. Secondly, the $\dot{V}O_2$ decrease

observed in our three subjects also cannot be explained by running speed variations. Indeed, running speed was maintained constant for each intensity thanks to four controls. Therefore, according to these methodological considerations, we concluded that the $\dot{V}O_2$ decrease could be only explained by physiological process. Moreover, the proportion of subjects with a $\dot{V}O_2$ decrease (47%) are close to those reported by Perrey et al. [2].

Respiratory Muscle Fatigue

One hypothesis put forward in order to understand this decrease in $\dot{V}O_2$ before exhaustion during continuous exercise concerns respiratory muscle fatigue [2]. During short intermittent exercise (30s at 105% of MAV alternated with 30s passive recovery) we have shown [6] a $\dot{V}O_2$ decrease before exhaustion. We also suggested that this $\dot{V}O_2$ decrease was partly connected with respiratory muscle fatigue. Maximal respiratory pressures, considered as a good index of respiratory muscle strength, can be used in order to assess respiratory muscle fatigue [13]. However, according to Hayot et Matecki [20] maximal respiratory pressures used as a fatigue index should be coupled with other fatigue evaluation methods. Thus, in our study, two approaches were used to appreciate the respiratory muscle fatigue. The first was related to maximal respiratory pressures (PI_{max} and PE_{max}) measurements and the second one depended on the use of V_T , fr and \dot{V}_E kinetics [9]. Maximal respiratory pressure measurement fell within the range described by Leech et al. [21] and Chen et al. [22], and our results (Figure 2) did not present a significant difference between these two parameters at rest (rest) and at the end of the exercise (end). Since the phenomenon of a decrease in $\dot{V}O_2$ was not present in more than half of the subjects, it seems logical that there was no systematic fatigue of the respiratory muscles in the total group. But that does not necessarily mean

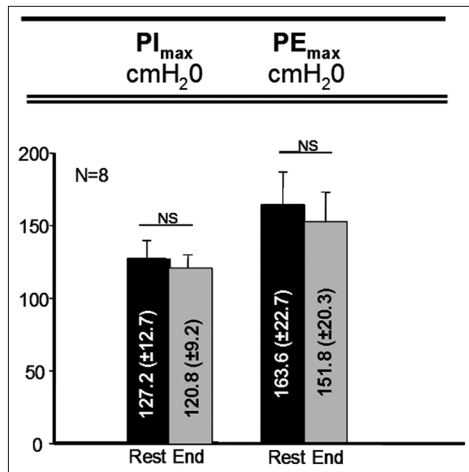


Figure 2: Mean values (\pm SD) for maximal inspiratory and expiratory pressures for the whole population during t_{lim95} . PI_{max} and PE_{max} : Maximal inspiratory and expiratory pressures. NS: no significant difference

that respiratory muscles fatigue was not the cause of the decrease in $\dot{V}O_2$ in those three subjects. Moreover, PI_{max} and PE_{max} values seemed to be lower at the end of the exercise (Figure 2). We could hypothesize that even without a significant decrease, 95% of MAV intensity seems to induce respiratory muscle fatigue. Thevenet et al. [6] during intermittent exercise observed a longer decrease (30% of time to exhaustion) during a longer exercise duration (around 35min). Whereas our mean t_{lim95} was shorter than ten minutes, mean time to exhaustion during intermittent exercise in Thevenet et al. [6] study was much longer because of the exercise modality. If decrease is linked to respiratory muscle fatigue, it could confirm results of Jonhson et al. [23] who recommended exercise intensity and duration at less of 85% of MAV and ten minutes respectively to induce respiratory muscle fatigue. We thought that 95% of MAV was an appropriate intensity in order to run longer than ten minutes, but it seems to be necessary to choose lower intensities or other exercise modality to run longer and then support the respiratory muscle fatigue hypothesis. These observations are consistent with conclusions of Romer et Polkey who suggested that exercise intensity is important but also that exercise duration plays a major role in diaphragm fatigue [24].

The second approach was based on the use of V_T , f_r and \dot{V}_E kinetics [9]. Let us first note that the filtering process used to analyse $\dot{V}O_2$ kinetics cannot be used in those cases. Indeed, \dot{V}_E and f_r kinetics do not fit a second order model. For example, no stable state is reached before exhaustion. Our results showed that

the decrease in \dot{V}_E began at around 95% of t_{lim95} . This result could be explained by a V_T decrease. Indeed, after 95% of t_{lim95} , the increase in f_r seemed to be insufficient to prevent the V_T decrease, that can be considered as an indirect sign of respiratory muscle fatigue [9] and can partly explain the $\dot{V}O_2$ decrease [6] before exhaustion. Nevertheless, the absence of significantly different results on respiratory muscle fatigue and the impossibility of statistical processing \dot{V}_E , V_T and f_r values, lead us to consider our results with caution and to partly reject our hypothesis. Another hypothesis has been advanced by Perrey et al. [2] to explain the origin of the $\dot{V}O_2$ decrease before exhaustion. This hypothesis is related to a cardiac output decrease and to an O_2 arterio-venous difference decrease. The latter hypothesis seems to be more likely during a maximal exercise and could have an influence on the locomotor muscles perfusion and $\dot{V}O_2$ for trained athletes. Indeed, during a maximal exercise, respiratory muscles O_2 consumption corresponds to 10-15% of $\dot{V}O_{2max}$. It induces a greater respiratory muscle blood flow, which could in turn induce locomotor muscle vasoconstriction. Then, it could compromise the blood flow, necessary for a good perfusion of locomotor muscles, and decrease the O_2 arterio-venous difference [25]. An exercise-induced hypoxaemia (EIH) could also explain an O_2 arterio-venous difference decrease. EIH is defined as a reduction in the arterial pressure O_2 (PaO_2) by more than 1kPa and/or a haemoglobin O_2 saturation (SaO_2) below 95% [26]. The ability to maintain a high alveolar O_2 pressure (PAO_2) is critical for blood oxygenation and this appears to be difficult in large individuals. A large lung capacity and, in turn, diffusion capacity seem to protect PaO_2 . A widening of the PAO_2 - PaO_2 difference does indicate that a diffusion limitation, a ventilation-perfusion mismatch and/or a shunt influence the transport of O_2 from alveoli to the pulmonary capillaries. A marked increase in cardiac output induces a faster transit time. When the latter is combined with diffusion limitation previously described, the O_2 transport problem is accentuated. To conclude, the existence of a $\dot{V}O_2$ decrease before the end of the exercise, already highlighted in the literature [2, 3, 4], seems to be confirmed. However, the respiratory muscle fatigue hypothesis seems to be partly rejected to explain our results. We suppose, indeed, that the exercise duration was insufficient to induce a respiratory muscle fatigue in the subjects. It could be interesting hence, to test other exercise intensities or modalities, in order to study respiratory muscle fatigue and its link with $\dot{V}O_2$ decrease over a longer period.

Limitations

Whilst this study examined only eight male physical education students, studying more high-level middle and long-distance runners is certainly warranted to get a better understanding of the nature of the associations between the $\dot{V}O_2$ decrease and the respiratory muscle fatigue.

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In memory of Delphine Thevenet.

Conflict of Interests

The authors have no conflicts of interest that are directly relevant to the content of this article.

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