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Changes in breathing pattern over a season's training in top class cyclists

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ABSTRACT

Aim. To examine the changes in breathing pattern variables (some indicative of the action of central regulation mechanisms) over a season's training in top class cyclists. **Material and methods.** Using a cycloergometer, 10 top class cyclists were subjected to a maximum exercise test in November (preparatory period), February (pre-competition period) and June (competition period). These three moments mark the beginning and end of the two main training periods, during which the volume and intensity of training was recorded. During each test the load (W) and heart rate (HR) were measured at the maximum VO_2 and at each of the ventilatory thresholds. The behaviour of the tidal volume (V_T) and inspiratory time (t_I) were determined over the following V_T/t_I ranges: 1) V_T/t_I <2; 2) V_T/t_I 2-4; and 3) V_T/t_I >4. **Results.** The volume and intensity of training increased significantly in the second period. However, neither the VO_{2max} nor the ventilatory thresholds improved. V_T did not change in any of the studied V_T/t_I ranges at any time during the season. The only difference seen was for t_I between the first and second tests when V_T/t_I was <2. The ratio V_T/t_I did not change over the season. **Conclusions.** Training did not change the variables measured in these highly trained athletes. The changes seen in VO_2 and the ventilatory thresholds after the first test cannot be attributed to changes in the studied breathing pattern variables.

Key words. Breathing pattern. Training. Ventilatory thresholds. V_T/t_I relationship. Respiratory control.

Evolución del patrón respiratorio a lo largo de una temporada de entrenamiento en ciclistas de élite

RESUMEN

Objetivo. *Analizar la evolución de los parámetros del patrón respiratorio (algunos de los cuales son representativos de los mecanismos centrales de regulación) en ciclistas con un alto nivel de entrenamiento a lo largo de una temporada.* ***Material y métodos.*** *Participaron en el estudio diez ciclistas de élite, a los que se les realizaron tres pruebas de esfuerzo máximas en cicloergómetro, coincidentes con los meses de noviembre (periodo preparatorio), febrero (periodo precompetitivo) y junio (periodo competitivo). Estos tres momentos determinan el principio y el final de los dos periodos principales de entrenamiento, durante los cuales se controló el volumen y la intensidad del mismo. Se determinó la carga (W), la frecuencia cardiaca (HR) y el consumo de oxígeno (VO_2) en tres instantes, correspondientes con el VO_2 máximo y cada uno de los dos Umbrales Ventilatorios. En dichas pruebas se estudió la relación del Volumen Corriente con el Tiempo Inspitatorio (V_T/t_I) en tres zonas: 1) relación V_T/t_I se encuentra por debajo de 2; 2) relación V_T/t_I se encuentra entre 2 y 4; y 3) relación V_T/t_I se encuentra por encima de 4.* ***Resultados.*** *El volumen e intensidad de entrenamiento aumentaron significativamente en el segundo periodo, pese a ello, tanto el VO_2 como los Umbrales Ventilatorios no mejoraron tras la segunda visita al laboratorio. El V_T no se modificó en ninguna de las zonas estudiadas para cada momento de la temporada. Sólo se observaron diferencias en t_I, entre la primera y segunda visitas, para el primero de los rangos estudiados. La relación V_T/t_I no se modificó a lo largo de la temporada para ninguna de las tres zonas propuestas.* ***Conclusiones.*** *No hay diferencias debidas al entrenamiento en las variables medidas en sujetos altamente entrenados. Los cambios en el VO_2 y los Umbrales Ventilatorios, tras la primera visita, no pueden ser atribuidos a cambios en los parámetros estudiados del patrón respiratorio de estos sujetos.*

Palabras clave. *Patrón respiratorio. Entrenamiento. Umbrales Ventilatorios. Relación V_T/t_I. Control respiratorio.*

INTRODUCTION

The adaptation of the breathing system to exercise in healthy, active people remains little understood. Studies in rats 'trained' at different intensities and using different systems (continuous and by intervals) have shown that intense training leads to changes in the enzymatic activity of the diaphragm.[1-3] The results of other animal studies suggest that for any adaptation of the respiratory musculature to occur there must be a stimulus provided by a sufficiently intense and long period of training.[4]

Studies in humans comparing the ventilatory performance of highly trained and out-of-form cyclists have shown that resistance training leads to an increase in the power of the respiratory musculature.[5-7] However, this improvement could be due to central mechanisms of breathing control bringing about greater respiratory efficicency[4] more than to changes in the respiratory musculature itself.

Indirect means of determining breathing adaptation, such as the monitoring of voluntary isocapnic hyperpnoea[8-11] have revealed improvements in the resistance of the respiratory musculature in trained athletes and persons with chronic obstructive pulmonary disease (COPD). Methods such as measuring inspiration against resistance, however, have provided more conflicting results. Some authors report improvements when this technique is used[12-15] while others report no such improvement.

The study of the breathing pattern has served to improve our understanding of the nervous mechanisms that control the depth and frequency of breathing. The tidal volume (V_T) is determined by two nervous mechanisms. The first, which establishes the range of the increase in the pulmonary volume, is known as the central inspiratory generator (CIG);[16] this represents the afferent signal of the spinal motor neurones. The second, known as the inspiratory disconnector (ID), controls the inspiration time t_I. According to this model, the pulmonary volume increases at a rate that depends on the respiratory drive until the volume reaches the Hering-Breuer point. Since the expiratory time (t_E) is in some way linked to t_I, the Hering-Breuer point represents a key moment in the control of the respiratory cycle. Thus, the CIG and ID determine the V_T, t_I and t_E for each breath.[16,17]

The literature lacks longitudinal studies of the above variables. The aim of the present work was therefore to examine the changes in the variables associated with the breathing pattern (some representative of central regulation mechanisms) in highly trained cyclists over a season. Unlike in studies involving patients with respiratory system disease, the initial hypothesis was that, in highly trained athletes, changes in the breathing pattern should not be detected over the season. The practical application of this study lies in knowing the limits of adaptation of the respiratory variables representative of the central regulation mechanisms.

MATERIAL AND METHODS

Study Subjects

The study subjects were 10 top class, male cyclists (age 20 ± 1.9 years, height 176.8 ± 1.9 cm, weight 68.6 ± 1.7 kg), all members of elite under-23 teams. All were familiar with ergospirometric testing.

All subjects underwent a medical examination including anamnesis, a physical examination, spirometry and electrocardiogram (ECG) before the study. In accordance with the Helsinki Declaration regarding experimentation involving human subjects, all were explained the nature of the study and all provided signed consent to be included. The study was approved by the appropriate ethics committee (*Comisión de Ética de Actividades de I+D+i*) of the *Universidad Politécnica de Madrid*).

Study protocol

The subjects underwent a maximum exercise ergospirometric test at three points over a single season: in November (preparatory period), in February (pre-competition period) and in June (competition period). These three points mark the beginning and end of the two main training periods.

As in other studies involving cyclists,[18] in the periods between the ergospirometric tests (i.e., the two training periods) the subjects monitored their training volume using diaries, recording the hours spent in this activity below the first ventilatory threshold (VT_1) (*zone 1*), between VT_1 and the second ventilatory threshold (VT_2) (*zone 2*), and above VT_2 (*zone 3*). In addition, a pulsometer (Polar S720i, Polar Electro OY, Kempele, Finland) was used to record their heart rate every 5 s during each training session.

Maximum effort tests

Maximum effort tests were performed using a Jaeger ER800® cycloergometer (Erich Jaeger, Hoechberg, Germany). The exercise protocol involved one minute at rest sat on the cycloergometer, followed by a 3 min warm-up period at 50 W, and an incremental phase involving an increase of 5 W every 12 s. The tests were ended when the subject so requested, or when the pedalling cadence fell below 70 rpm.

This period was followed by a 2 min active recovery period at 50 W (70 rpm) and then 3 min of complete rest sat on the cycloergometer. The pedalling cadence was set at 70-90 rpm. The criteria for deeming that maximum effort had been used were: the plateauing of oxygen consumption, a respiratory quotient of >1.10, and a maximum heart rate above the theoretical maximum. At least two of these criteria had to be met in order to consider maximum oxygen consumption (VO_{2max}; defined as the mean of maximum consumption at the time of greatest exercise intensity)[19] to have been reached.

Measurement of respiratory exchange

Gas exchange variables were measured breath-by-breath and the means calculated for every 15 s. The composition of the exhaled air was determined using a previously validated Jaeger Oxicon Pro® spirometer (Erich Jaeger, Hoechberg, Germany)[20-22] with a low dead space and low resistance bidirectional digital turbine (Triple V®) according to the standards of the American Thoracic Society and European Respiratory Society.[23,24]

Determination of the ventilatory thresholds

The ventilatory thresholds were determined using the method of Beaver, Davis and Gaskill.[25-27] Two operators determined the ventilatory thresholds in each test. When there was disagreement between their findings a third operator was called in.

Behaviour of breathing variables

The behaviour of V_T and t_I were determined over three V_T/t_I ranges: 1) $V_T/t_I < 2$; 2) V_T/t_I 2-4; and 3) $V_T/t_I > 4$.

Statistical analysis

The normality of the distribution of the values of the studied variables was checked. One way ANOVA was used to determine whether the moment of the season affected the position of the ventilatory thresholds and the value of VO_{2max}. Differences were compared using a *post hoc* Scheffé test. Two-way ANOVA with repeated measures for one factor (moment during season in each V_T/t_I range) was used to examine differences in the variables of the breathing pattern. Significance was set at $p < 0.05$ in all cases.

RESULTS

Table 1 shows the volume and intensity of training recorded for each of the season's training periods. Significant differences were seen between the training volume, except in *zone 2* (between VT_1 and VT_2), between the two training periods.

Table 2 shows values of the physiological variables measured at the different test times. Compared to the first test in November, the VO_{2max}, VO_2 and ventilation (VE) had improved significantly for both ventilatory thresholds in the second (February) and third (June) tests. The maximum load (W_{max}) improved only in the second test. The positions of the ventilatory thresholds (measured as $\%VO_{2max}$ and W) improved in the second and third tests compared to the first. No significant differences were seen in the heart rate during any of the tests.

Table 3 and figure 1 show the values of the breathing pattern variables at each test. Between the first and second tests, significant differences were seen between t_I at low loads (when $V_T/t_I < 2$). No significant differences were seen between V_T, nor

Table 1. Volume and intensity of training in the two training periods.

Variables	Period 1 i.e., between the 1st and 2nd test	Period 2 i.e., between the 2nd and 3rd test
Volume (hrs)	211.3 ± 0.6	260.3 ± 1.0[a]
Volume in zone 1 (%)	77.7 ± 0.3	69.9 ± 0.5[a]
Volume in zone 2 (%)	19.7 ± 0.6	22.1 ± 0.4
Volume in zone 3 (%)	2.4 ± 0.3	8.1 ± 0.2[a]

Data are means ± standard deviation. [a] Significant difference ($p < 0.05$) between training periods.

Table 2. Physiological variables measured in the exercise tests.

	1st test	2nd test	3rd test
VE ($l \cdot min^{-1}$)	185 ± 6.9	180 ± 4.0	189 ± 6.2
V_T (L)	3 ± 0.1	3.2 ± 0.1	3.3 ± 0.2
FR (respiraciones·min^{-1})	61 ± 3	56 ± 3	57 ± 1
W_{max} (W)	429 ± 12.5	463 ± 13.4^a	448 ± 18.2
VO_{2max} ($ml \cdot kg^{-1} \cdot min^{-1}$)	73.1 ± 1.8	77.6 ± 1.4^a	80.5 ± 1.8^a
HR_{VO2max} (lat·min^{-1})	196 ± 3	199 ± 4	191 ± 4
VE_{VT1} ($l \cdot min^{-1}$)	60.0 ± 2.7	74.4 ± 2.8^a	77.8 ± 3.7^a
V_{T_VT1} (L)	2.3 ± 0.1	2.3 ± 0.1	2.5 ± 0.2
FR_{VT1} (respiraciones·min^{-1})	27 ± 1	32 ± 2	31 ± 2
W_{VT1} (W)	206 ± 11.2	252 ± 10.8^a	254 ± 13.5^a
VO_{2VT1} ($ml \cdot kg^{-1} \cdot min^{-1}$)	38.1 ± 1.7	47.2 ± 1.5^a	50.4 ± 1.9^a
VO_{2VT1} (%VO_{2max})	52 ± 2.1	60 ± 1.8^a	62 ± 1.7^a
HR_{VT1} (lat·min^{-1})	141 ± 3	144 ± 4	142 ± 4
VE_{VT2} ($l \cdot min^{-1}$)	111.9 ± 4.6	127.9 ± 3.6^a	139.3 ± 4.5^a
V_{T_VT2} (L)	3.2 ± 0.1	3.0 ± 0.02	3.3 ± 0.2
FR_{VT2} (respiraciones·min^{-1})	35 ± 3	43 ± 2	42 ± 3
W_{VT2} (W)	339 ± 11.1	378 ± 8.7^a	377 ± 11.7^a
VO_{2VT2} ($ml \cdot kg^{-1} \cdot min^{-1}$)	60.3 ± 1.8	68.5 ± 1.9^a	72.9 ± 2.3^a
VO_{2VT2} (%VO_2 max)	82.5 ± 1.7	87.9 ± 1.0^a	90.1 ± 1.4^a
HR_{VT2} (lat·min^{-1})	179 ± 3	179 ± 3	177 ± 4

Data are means ± standard error.[a] Significant differences ($p < 0.05$) between the 1st and 2nd/3rd tests. VE: ventilation; V_T: tidal volume. FR: respiratory frequency. W_{max}: maximum load. VO_{2max}: maximum oxygen consumption. HR_{VO2max}: heart rate at maximum oxygen consumption. VT1: first ventilatory threshold. VT2: second ventilatory threshold.

V_T/t_I, with respect to load, at any time in any of the V_T/t_I ranges.

DISCUSSION

The results of this study shows that the activity of the CIG, assessed in terms of V_T/t_I, does not change over a season's training in highly trained cyclists. This suggests that an improvement in breathing efficiency cannot be attributed to the adaptation of the central breathing control mechanisms.

The V_T increased with the load in the present tests. As in other studies[28] a stabilization in V_T was seen at high loads (> 350 W) during the third test (Figure 1A) while an increase was seen in the first and second tests. However, these differences were not significant when compared among themselves. It might be thought that, in the third test, the tidal volume regulation mechanisms (reflex and central) have reached their limits. However, no significant reduction was seen in t_I at high loads in the three tests (Figure 1B). Thus, despite the fact that training increased significantly over time, both in terms of volume and intensity (Table 1), the activity of the CIG (determined by V_T/t_I) showed no significant differences at any moment (Figure 1C).

The establishment of the V_T/t_I ranges differed with respect to those used by Lind and Hesser,[29] who instead established the following: V_T < 1.4 L, 1.4-2.4 L and > 2.4 L, corresponding to loads of < 40 W, 80-180 W and > 180 W respectively. In the present study, the values of V_T were considerably higher (approximately < 2 L, 2-2.75 L and > 2.75 L; Figure 1A). These ranges reflect the difference in the physical condition of the subjects examined; the present subjects were highly trained, as shown by the maximum power recorded by the cycloergo-

Table 3. Breathing pattern variables studied.

	V_T/t_I < 2			2 > V_T/t_I < 4			V_T/t_I > 4		
Visita	1	2	3	1	2	3	1	2	3
V_T (L)	1.6 ± 0.4	1.4 ± 0.4	1.4 ± 0.3	2.5 ± 0.4	2.4 ± 0.4	2.4 ± 0.4	3.1 ± 0.2	3.0 ± 0.2	3.1 ± 0.4
t_I (s)	1.2 ± 0.4	1.0 ± 0.2*	1.1 ± 0.3	0.9 ± 0.1	0.9 ± 0.1	0.9 ± 0.2	0.6 ± 0.7	0.6 ± 0.1	0.6 ± 0.1
V_T/t_I ($L \cdot s^{-1}$)	1.4 ± 0.3	1.3 ± 0.4	1.3 ± 0.4	3.0 ± 0.6	2.9 ± 0.6	2.9 ± 0.6	5.4 ± 0.7	5.4 ± 0.8	5.5 ± 0.9

Data are means ± standard deviation. Tidal volume (V_T), inspiration time (t_I) and V_T/t_I were recorded in each test. * Significantly different to test 1 value ($p < 0.05$).

meter (463 W). In a second study by Lind and Hesser,[28] which involved a maximum exercise protocol, the responses of V_T and t_I were similar to those seen in the present work: t_I fell linearly as V_T increased with loads of < 240W, while with high loads t_I fell but V_T did not change.

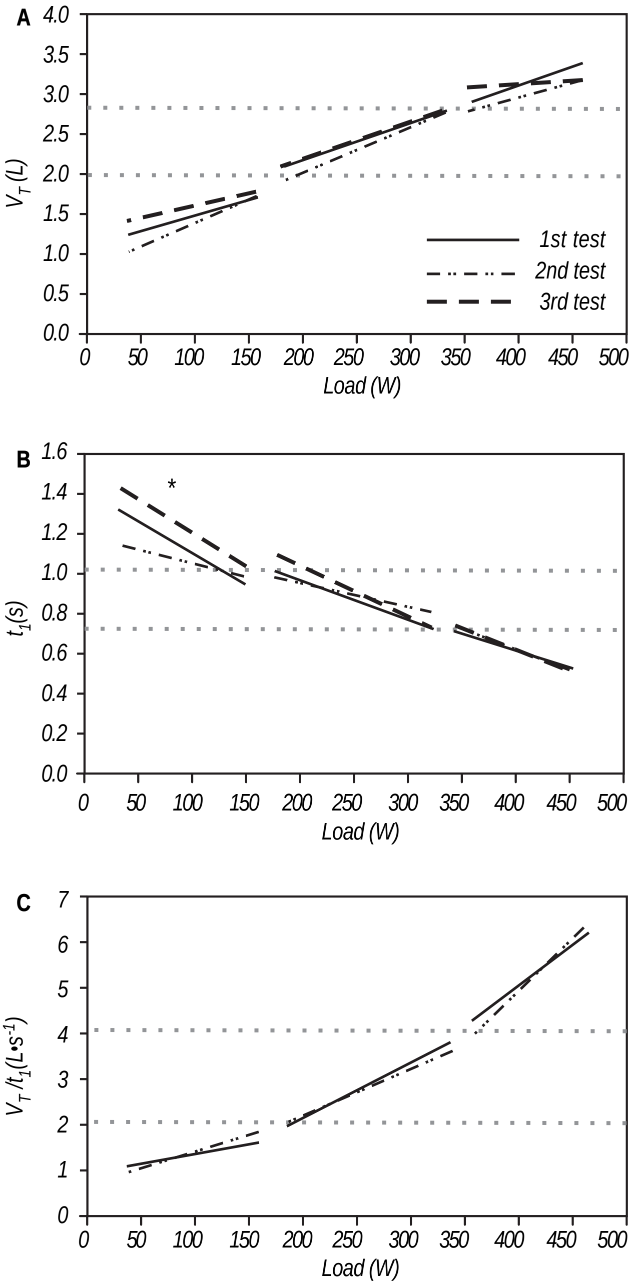

Figure 1. *Relations between* t_I ***A.*** V_T ***B.*** V_T/t_I ***C.*** *at different loads in each in each test. The discontinuous lines indicate* V_T/t_I *2, ratio of < 2, 2 2-4 and > 4.* Indicates significant differenbces (p < 0.05) between the first an second test.*

The absence of differences in V_T/t_I over the season suggests that the activity of the CIG did not change with resistance training. Other studies[30] have also reported that training does not affect V_T/t_I in patients with COPD. However, unlike in patients with respiratory disease following a program to train their respiratory musculature, the work performed by this musculature in the present cyclists was very great. Ito, *et al.*[31] reported a prolongation of t_E with training of the respiratory musculature in patients with COPD. However, the process of adaptation in these patients was assessed at rest and not under stress as in the present study. Finally, Holm, *et al.*[32] reported improvements in performance to be achieved through the training of the respiratory musculature in young cyclists, and concluded that such training allowed tolerance to a greater respiratory response without an increased sensation of dyspnoea. However, these authors themselves concluded that the perception of best effort on the part of athletes cannot guarantee better performance. The results obtained by these authors do not contradict those obtained in the present work owing to the differences in the intensity of respiratory musculature training undertaken (the work of Holm et al. involving sessions of hyperpnoea). In addition, the latter authors only analysed ventilation, and not inspiratory drive.

The maximum values of load, VO_2 and relative load (≈ 450 W, ≈ 78 $ml \cdot kg^{-1} \cdot min^{-1}$, ≈ 6.9 $W \cdot kg^{-1}$ respectively) recorded for the cyclists give an idea of their high aerobic capacity. Changes in the VO_{2max}, a variable that reflects respiratory, cardiovascular and oxygen transport functions,[33] were seen only between the first and second, and first and third, tests; no difference was seen between the second and third test (times when the cyclists were in top cardiorespiratory condition) (Table 2). Since no differences were seen between V_T/t_I between the first and second/third tests, the improvement seen in VO_{2max} cannot be attributed to central mechanisms regulating the breathing pattern. In addition, the absence of differences in the heart rate might indicate an absence of any change at the cardiovascular level. Similarly, the ventilatory thresholds increased after the first training period. After the first test this might have been due to the training undertaken in the time between the first and second tests (≈ 211 h), but no further improvement was seen between the second and third tests despite the great volume of training this period involved (≈ 260 h). Some authors[34] indicate this may be due to the reaching of a physiological limit after which no improvement is possible.

A possible limitation of the present work is the non-inclusion of measurements of the expiratory drive (V_T/t_E); it is likely that training would cause changes in this variable. The expiratory drive can strongly influence the breathing pattern at work loads close to the maximum. Lind and Hesser[28,29] found differences in the increase of V_T/t_I and V_T/t_E at such work loads.

In conclusion, training led to no differences in the breathing pattern in the highly trained athletes studied. The values for the breathing pattern variables presented might represent the maximum limits of respiratory adaptation. Finally, the changes in VO_2 and the ventilatory thresholds observed after the first test cannot be attributed to changes in the breathing pattern. If it is assumed that there was no change in cardiac function, these changes could be the result of peripheral adaptation mechanisms.

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