Anaerobic energy provision does not limit Wingate exercise performance in endurance-trained cyclists

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35017 Las Palmas de Gran Canaria, Canary Islands;* [2]*Instituto Nacional de
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Calbet, J. A. L., J. A. De Paz, N. Garatachea, S. Cabeza de Vaca, and J. Chavarren. Anaerobic energy provision does not limit Wingate exercise performance in endurance-trained cyclists. *J Appl Physiol* 94: 668–676, 2003. First published October 4, 2002; 10.1152/japplphysiol.00128. 2002.—The aim of this study was to evaluate the effects of severe acute hypoxia on exercise performance and metabolism during 30-s Wingate tests. Five endurance- (E) and five sprint- (S) trained track cyclists from the Spanish National Team performed 30-s Wingate tests in normoxia and hypoxia (inspired O_2 fraction = 0.10). Oxygen deficit was estimated from submaximal cycling economy tests by use of a nonlinear model. E cyclists showed higher maximal O_2 uptake than S (72 ± 1 and 62 ± 2 ml·kg^{-1}·min^{-1}, $P < 0.05$). S cyclists achieved higher peak and mean power output, and 33% larger oxygen deficit than E ($P < 0.05$). During the Wingate test in normoxia, S relied more on anaerobic energy sources than E ($P < 0.05$); however, S showed a larger fatigue index in both conditions ($P < 0.05$). Compared with normoxia, hypoxia lowered O_2 uptake by 16% in E and S ($P < 0.05$). Peak power output, fatigue index, and exercise femoral vein blood lactate concentration were not altered by hypoxia in any group. Endurance cyclists, unlike S, maintained their mean power output in hypoxia by increasing their anaerobic energy production, as shown by 7% greater oxygen deficit and 11% higher postexercise lactate concentration. In conclusion, performance during 30-s Wingate tests in severe acute hypoxia is maintained or barely reduced owing to the enhancement of the anaerobic energy release. The effect of severe acute hypoxia on supramaximal exercise performance depends on training background.

fatigue; anaerobic power; anaerobic capacity; lactate

THE IMPACT THAT ACUTE SEVERE hypoxia has on aerobic and anaerobic energy yield during the Wingate test remains unknown. Although moderate acute hypoxia [inspired O_2 fraction (F_{IO_2}) = 0.13] has no effect on either O_2 uptake ($\dot{V}O_2$) or performance during supramaximal exercise of a duration up to 30 s (39), conflicting results have been reported about the influence that

higher levels of hypoxia might have on performance and metabolism (22, 23). McLellan et al. (23) observed unchanged mean power output (Pmean) and $\dot{V}O_2$ during a 30-s Wingate test performed with a F_{IO_2} ~0.11. Prolonging the Wingate test duration until 45 s consistently resulted in lower $\dot{V}O_2$ with hypoxia (22, 23) whereas, compared with normoxia, Pmean during the 45-s Wingate test was not affected by acute hypoxia in one study (23) and slightly reduced (~3% less) in another investigation (23). Small differences in the F_{IO_2} and time exposure to hypoxia before the start of the Wingate tests could explain the apparently contradictory results reported by McLellan et al. (22, 23). It was clearly shown, on the other hand, that during all-out exercise in acute hypoxia lasting 30 or 45 s, muscle lactate accumulation is markedly increased, indicating a greater anaerobic energy release with acute hypoxia (23). Furthermore, some pieces of evidence suggest that during supramaximal exercise producing exhaustion between 30 and 60 s the contribution of the anaerobic energy sources is increased in acute moderate hypoxia (22, 39). Whether a higher degree of hypoxia could enhance anaerobic energy release during 30-s Wingate tests is not known.

It has been estimated that, in general, anaerobic energy sources provide 70–80% of energy utilized throughout the Wingate test (6, 28, 33, 40). However, compared with sprint specialists, endurance-trained athletes have greater mean $\dot{V}O_2$ during the Wingate tests (12) and obtain a higher fraction of the energy from oxidative metabolism. In turn, sprint-trained athletes obtain a greater fraction of energy through the anaerobic pathways (6). Thus it can be hypothesized that because endurance-trained athletes rely more on aerobic energy sources to perform all-out supramaximal exercise they will experience a relatively greater impairment of performance than sprint-trained athletes with acute hypoxia, unless they compensate for the reduction in $\dot{V}O_2$ by enhancing the anaerobic energy release.

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Therefore, the primary aim of this study was to find out whether the reduction in O_2 supply elicited by severe acute hypoxia can be counteracted by enhancing anaerobic energy production during 30-s Wingate tests. Another purpose was to verify whether endurance-trained elite track cyclists would experience superior deterioration of performance than their sprint-trained counterparts during 30-s Wingate tests in acute hypoxia. To unravel more easily the effect of hypoxia, the F_{IO_2} was reduced to 0.104, which is equivalent to an altitude of ~5,300 m. This level of hypoxia is close to the limit that a healthy nonacclimatized human can tolerate acutely during upright exercise (4, 32, 36).

METHODS

Subjects

This study was approved by the ethics committee at the University of Las Palmas de Gran Canaria. The subjects were fully informed of the purpose and possible risks and benefits of their participation in the study before giving their written consent. Ten track cyclists from the Spanish National Team participated in this study. All them had qualified more than once among the first three finishers in the Spanish National Championship. The group included several National Champions and a silver medalist in the last World Championship, who was a sprint specialist. Five cyclists were sprint specialists, and the other five were endurance-trained cyclists. The mean ± SE ages, heights, weights, and percentages of body fat were 19.0 ± 0.7 vs. 18.8 ± 0.4 yr, 176 ± 2 vs. 179 ± 0.7 cm, 74.7 ± 3.1 vs. 65.8 ± 1.0 kg, and 13.2 ± 1.9 vs. 9.3 ± 0.5% in the sprint and endurance cyclists, respectively. Subjects were requested to follow similar diets and lessen physical activities during the 48 h before the experiments. They were also instructed to refrain from any food for at least 4 h before each test session.

Experimental Protocol and Procedures

The percentage of body fat was estimated anthropometrically by using a population-specific equation, which was developed and validated in our laboratory by using dual X-ray absorptiometry as a gold standard (19). Subjects completed four test sessions in separate days. First, their maximal $\dot{V}O_2$ ($\dot{V}O_{2\,max}$) and maximal power output (Wmax) were assessed via an incremental exercise test to exhaustion (25 W/min, at 90 rpm). During the next 2 testing days, the relationship between $\dot{V}O_2$ and intensity (or cycling economy) was determined. Then, during the last test session, Wingate tests were carried out while the subjects were breathing either room air (normoxia) or air from a Douglas bag containing 10.4% O_2 in N_2 (hypoxia), equivalent to an altitude of ~5,300 m. This level of acute hypoxia was chosen because it reduces $\dot{V}O_{2\,max}$ by nearly 50% (10, 36), creating, therefore, a condition in which a potential limitation to aerobic energy supply would be present during the Wingate tests performed in severe hypoxia. Normoxic and hypoxic Wingate tests were performed in random order and separated by a recovery period of at least 1 h, which is enough to allow for a complete recovery (6).

Cycling economy tests. Cycling economy was determined by using 12 submaximal workloads at intensities of between 60 and 90% $\dot{V}O_{2\,max}$, at four different pedaling rates: 60, 80, 100, and 120 rpm. Exercise intensities and pedaling rates were administered in random order, separated by rest periods of 3–5 min. To reduce thermal stress and minimize water losses due to sweating, subjects were fan cooled and ingested in total 600 ml of water during the resting periods. Tests were performed at 18–24°C, 60–80% relative humidity, and 750–770 mmHg atmospheric pressure. The duration of each submaximal bout was set at 6 min. The mean $\dot{V}O_2$ registered during the last 2 min was taken as representative of each submaximal exercise intensity. To relate $\dot{V}O_2$ to power, individual nonlinear regression equations were calculated by least-square linear fit, applying the following nonlinear model: $\dot{V}O_2 = a + b \cdot W + c \cdot rpm + d \cdot rpm^2$, which gave a standard error of estimate that was always lower than 100 ml/min of O_2, where a, b, c, and d are the constants to be determined by the nonlinear regression equation; W is the exercise intensity; and rpm is the pedaling rate.

Wingate tests. To determine whole blood lactate concentration ([La]) in the femoral vein, a 20-gauge catheter (Hydrocath, Ohmeda, Swindon, UK) was inserted percutaneously by use of the Seldinger technique into the right femoral vein under local anesthesia (2% lidocaine) as previously reported (29). The catheter was inserted 2 cm below the inguinal ligament and was advanced 12 cm toward the knee to avoid contamination of the blood coming from the deep quadriceps muscle veins with saphenous vein blood. Once in place, the catheter was sutured to the skin to minimize the risk of movement or creasing, and the outlet of the catheter was connected to a three-way stopcock. After a resting period of ~5 min, subjects carried out a standardized warm-up consisting of 10 min of continuous cycling at an intensity close to 60% $\dot{V}O_{2\,max}$ followed by five maximal accelerations lasting for no more than 5–6 s. Then the subjects rested for 10 min and were randomly assigned to a normoxic or hyperoxic Wingate test. To minimize the risk of hypotension and respiratory alkalosis during the hypoxic Wingate tests, the subjects commenced to breathe from the hypoxic gas bag only 3 min before the onset of the exercise (36). During the Wingate tests, femoral vein blood samples were drawn continuously for periods of 5 s, given a total of six exercise samples. An hour later, the Wingate test was repeated in the other condition. After both Wingate tests, the subjects recovered in normoxia while lying on a bed, and additional blood samples were withdrawn from the femoral vein 3, 5, 7, and 10 min after the start of the recovery period. Braking forces equivalent to 0.11 kp per kg of body mass were used in all cyclists.

All cycling tests were carried out on a Monark cycle ergometer (Monark 818E). During the Wingate tests double-toe stirrups and straps were used to tightly fix the feet to the pedals. To record the pedal velocity every 2 ms, a disk with 250 slots was connected through a gear to the chain (6). The slotted disk was turning in front of a photoelectric cell interfaced with a computer. The work performed on the Monark cycle ergometer was corrected for the work required to accelerate the flywheel, as reported elsewhere (6). Briefly, the load on the cycle ergometer was considered as the sum of the frictional load (braking force applied through the belt to the flywheel) and the load that would be required at any instant to stop the subject from accelerating the flywheel, which depends on the flywheel's moment of inertia and other frictional forces acting on the flywheel. To calculate this load, we generated a series of deceleration curves using different frictional loads. First, the flywheel was set in motion at a constant speed of 120 rpm by pedaling. Second, after achieving a steady speed, pedaling was suddenly interrupted and the time elapsed between 105 and 0 rpm was used to calculate the deceleration for each braking force. Lastly, a regres-

sion equation was derived by least-square linear fit between flywheel deceleration and load.

The anaerobic energy yield during the Wingate tests was calculated as the O_2 deficit as previously reported (6). First, the O_2 demand was estimated individually by extrapolating the nonlinear relationship between $\dot{V}O_2$ and power output measured at submaximal loads. Then the O_2 deficit was computed as the difference between the O_2 demand and the O_2 consumed during the supramaximal bouts (25, 39).

Respiratory variables. Ventilatory and gas exchange variables were monitored breath-by-breath by an open-circuit sampling system (CPX, Medical Graphics, St. Paul, MN) and averaged every 15 s during the incremental exercise tests and every 5 s during the Wingate tests. The metabolic cart was calibrated with calibration gas mixtures of known O_2 and CO_2 concentrations (accuracy 0.01%), which were provided by the manufacturer (CPX, Medical Graphics). In our laboratory, $\dot{V}O_2$ and CO_2 production during submaximal cycling has been assessed with a coefficient of variation lower than 5%, as well as with an intraclass reliability coefficient higher than 0.98, as determined in six physical education students at four different intensities on 4 different days. The highest $\dot{V}O_2$ value attained during the incremental exercise tests was taken as the $\dot{V}O_{2 \, max}$, whereas the intensity attained just before exhaustion is referred to as Wmax. The Wmax was adjusted by extrapolation depending on the duration of the last step (18). To determine the kinetics of the $\dot{V}O_2$ on-response, breath-by-breath data were averaged every 5 s and fit to a curve by using an exponential model, by means of the least-squares error approach. The curve-fitting procedure was iterated until any further changes in the parameters for the model did not result in a reduction in the mean squared error between the curve obtain from the model and the original data set. The model used to fit the $\dot{V}O_2$ on-data had a constant, which corresponds to resting $\dot{V}O_2$, an amplitude term (b), and a time constant (c) as follows

$$\dot{V}O_2(t) = a + b(1 - e^{-ct})$$

where t is the time in seconds and $\dot{V}O_2(t)$ is the time-dependent variation in $\dot{V}O_2$.

Blood lactate. [La] was determined in whole blood by using a lactate analyzer (YSI 1500 Sport, Yellow Springs, CO) provided with hemolyzing agent (Triton X-100). With this instrument, we obtained a coefficient of variation for whole blood [La] assessment lower than 1%, for [La] between 1 and 26 mM. Recovery blood [La] curves were integrated over time and expressed as millimolar·seconds. Reported values correspond to the net area under curve, i.e., the area under the curve after accounting for the resting blood [La] immediately before the start of the Wingate tests.

Statistical Analysis

Descriptive statistics were performed on each variable to confirm the assumptions of normality and homocedasticity. The effect of O_2 inspired fraction on femoral blood lactate during the Wingate tests was determined using a two-way repeated-measures analysis of variance with cyclists specialty as a two-level factor. In the case of a significant F value, planned comparisons were carried out by using a Student's paired or unpaired (as appropriate) t-test with Bonferroni correction to account for multiple comparisons. The significance level was set at $P < 0.05$. Data are expressed as means ± SE.

RESULTS

Differences Between Endurance- and Sprint-Trained Cyclists

Endurance cyclists showed higher $\dot{V}O_{2 \, max}$ than the sprint-trained cyclists (72 ± 1 and 62 ± 2 ml·kg^{-1}·min^{-1}, $P < 0.05$). As depicted in Fig. 1A and Table 1, absolute and relative peak power output (Pmax) and Pmean were greater in the sprint than in the endurance cyclists. Accordingly, sprint cyclists reached greater maximal and mean pedaling rates than endurance-trained cyclists (Fig. 1B). Sprint cyclists, however, fatigued faster than the endurance cyclists, as indicated by the fatigue index during the Wingate test, which was 0.46 ± 0.12 and 0.32 ± 0.12 W·s^{-1}·kg^{-1} body mass in the sprint and endurance cyclists, respectively (Fig. 2A; $P < 0.05$). In consequence, the sprint cyclists' superiority in power output was reduced progressively during the second half of the Wingate tests, developing in both groups almost similar power output values per kilogram of body mass during the last 15 s (Fig. 1A). The sprint cyclists incurred a greater O_2 demand during the first 10–15 s of the Wingate test (Fig. 1, C and D). Likely because of their greater $\dot{V}O_{2 \, max}$, the endurance cyclists were able to consume 26% more O_2 per kilogram of body mass during the Wingate tests than the sprint cyclists ($P < 0.05$), because both groups utilized a similar percentage of their $\dot{V}O_{2 \, max}$ during the Wingate tests in normoxia (Fig. 2B). In fact, a close correlation was observed between the mean $\dot{V}O_2$ during the Wingate test and $\dot{V}O_{2 \, max}$ (Fig. 2C; $r = 0.86$, $P < 0.001$). With greater O_2 demand and lower $\dot{V}O_2$, O_2 deficit per kilogram of body mass resulted 33% higher in the sprint than the endurance cyclists (Fig. 1H). The difference between 26% superior aerobic energy yield in the endurance cyclists and 33% greater O_2 deficit in the sprint cyclists leads to the ~8% superior Pmean developed during the Wingate by the sprint specialists. Despite these remarkable differences in anaerobic energy yield during the Wingate tests, the rate of femoral vein blood [La] accumulation during exercise was similar in the endurance and sprint cyclists. In both groups, femoral blood [La] did not change during the first 15 s of exercise, but thereafter it augmented, describing a parabola as exercise time passed (in all conditions, $r = 0.99$, $P < 0.001$; Fig. 1G). During the first 10 min of the recovery period, the sprint cyclists accumulated 27% more lactate in the femoral vein than their endurance counterparts ($P < 0.05$; Fig. 3).

As depicted in Fig. 4 the anaerobic energy contribution to the overall energy expended during the Wingate test decreased following a parabolic pattern ($r > 0.98$, $P < 0.01$). The sprint cyclists obtained a slightly greater proportion of energy through the anaerobic pathways (Table 1). Extrapolation of the curves depicted in Fig. 3 to 0% anaerobic energy contribution permits us to predict the duration that an all-out test should have to enable a complete utilization of all the
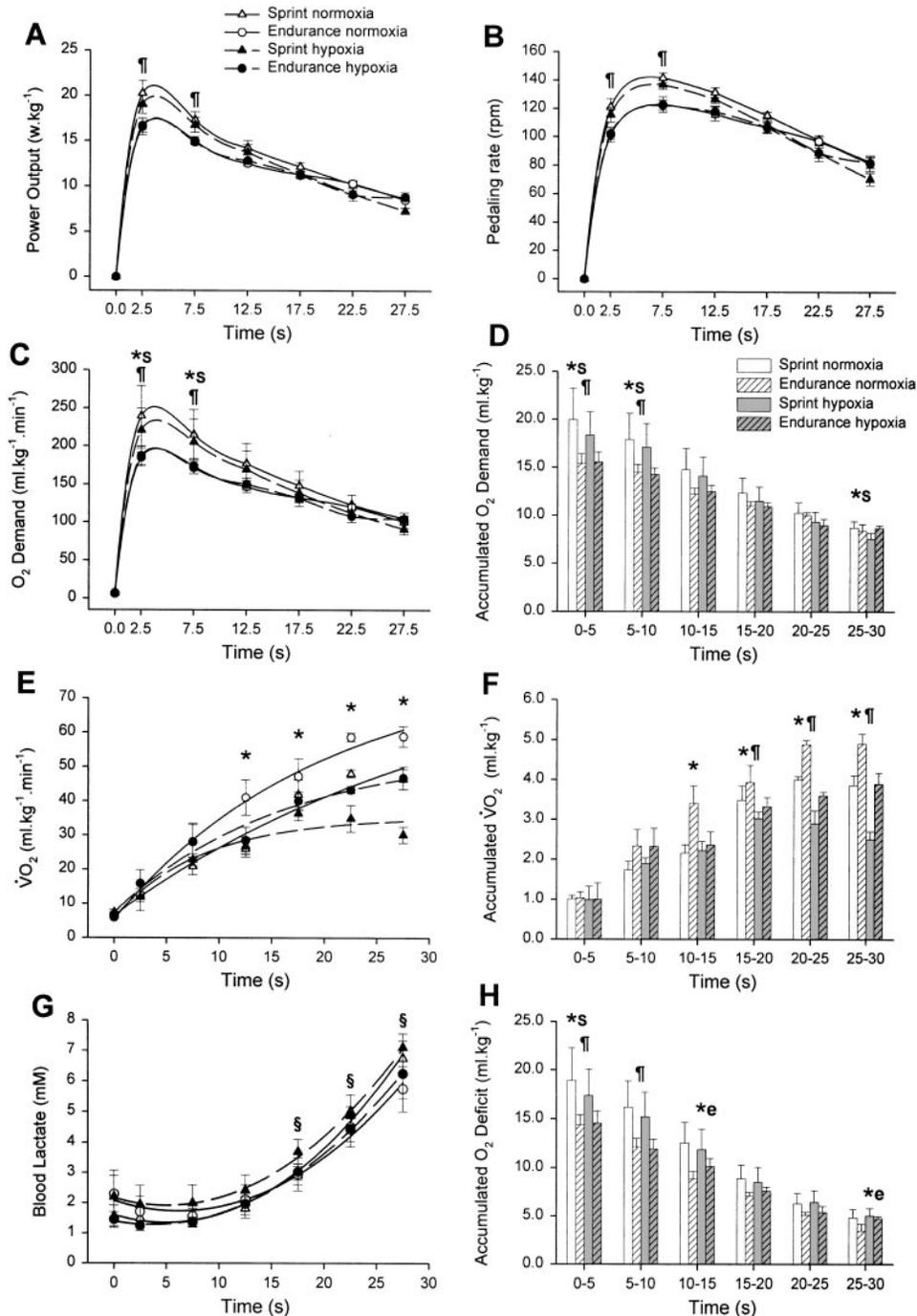
Fig. 1. Time course of power output (*A*), pedaling rate (*B*), O_2 demand (*C*), accumulated O_2 demand (*D*), O_2 uptake ($\dot{V}O_2$; *E*), accumulated $\dot{V}O_2$ (*F*), femoral blood lactate concentration (*G*), and O_2 deficit (*H*) during 30-s Wingate tests in normoxia and acute severe hypoxia. *$P < 0.05$ normoxia vs. hypoxia; ¶$P < 0.05$ sprint vs. endurance-trained cyclists; §$P < 0.05$ compared with *time 0*; *S$P < 0.05$ normoxia vs. hypoxia, but only for the sprint cyclists; *e$P < 0.05$ normoxia vs. hypoxia, but only for the endurance cyclists.

anaerobic energy potential (Fig. 4). Because the sprint cyclists had larger O_2 deficit and obtained a greater fraction of energy through the anaerobic pathways, the rate of anaerobic energy release was much higher in the sprint than in the endurance cyclists. Despite this fast recruitment of their anaerobic capacity, the sprint cyclists needed 4–5 s more than the endurance cyclists to fully express their anaerobic capacity.

Effect of Severe Acute Hypoxia

The time course of power output, O_2 demand, $\dot{V}O_2$, O_2 deficit, and blood [La] during the normoxic and hypoxic Wingate tests are depicted in Fig. 1. Although Pmax was not affected by hypoxia in any group, Pmean and pedaling rate were reduced by 6–7% in the sprint cyclists ($P < 0.05$). In the endurance cyclists, however, Pmean and pedaling rate remained at same level as in normoxia. With a lower Pmean, O_2 demand was diminished in the sprint cyclists exercising in hypoxia ($P < 0.05$). In both groups, hypoxia resulted in a 16% lower mean $\dot{V}O_2$ ($P < 0.01$; Fig. 1*E*). The divergence between normoxic and hypoxic $\dot{V}O_2$ during the Wingate test began 10 s after the start of the test and became more accentuated as the exercise progressed ($P < 0.05$).

Table 1. *Differences between sprint- and endurance-trained cyclists*

	Normoxia		Hypoxia	
	Sprint	Endurance	Sprint	Endurance
Pmax	$1,547 \pm 128$	$1,122 \pm 65$†	$1,490 \pm 153$	$1,128 \pm 54$†
Pmax, W/kg	20.6 ± 1.3	17.0 ± 0.9†	19.8 ± 1.3	17.1 ± 0.7†
RPMmax, rpm	156.5 ± 3.4	134.3 ± 5.7†	153.3 ± 4.5	134.0 ± 3.9†
TRPMmax, s	5.89 ± 0.36	6.57 ± 0.53	6.41 ± 0.49	6.76 ± 0.20
Pmean, W	$1,030 \pm 52$	813 ± 22†	962 ± 51*	805 ± 20†
Pmean, W/kg	13.8 ± 0.6	12.4 ± 0.3†	12.9 ± 0.6*	12.2 ± 0.2†
RPMmean, rpm	115.1 ± 3.0	105.5 ± 4.0†	107.9 ± 3.2*	104.5 ± 3.0
O_2Dem, ml/min	$6,218 \pm 801$	$4,715 \pm 250$†	$5,780 \pm 682$*	$4,680 \pm 275$†
$\dot{V}O_2$, ml	$1,207 \pm 31$	$1,350 \pm 78$	$1,010 \pm 87$	$1,127 \pm 60$
$\dot{V}O_2$, ml/kg	16.3 ± 0.8	20.5 ± 1.1†	13.6 ± 1.2*	17.2 ± 1.0*†
O_2D, ml	$5,011 \pm 811$	$3,365 \pm 219$†	$4,770 \pm 761$*	$3,591 \pm 269$†
O_2D, ml/kg	67.7 ± 11.4	51.0 ± 2.6†	64.4 ± 10.7*	54.4 ± 3.3†
Anaerobic, %	79.3 ± 2.7	71.2 ± 1.6†	81.0 ± 3.4	76.5 ± 1.8
[La], mM	3.2 ± 0.3	3.1 ± 0.5	3.7 ± 0.3	3.0 ± 0.3

Values are means $\pm$ SE. Pmax, peak pace; RPMmax, maximal pedaling rate; TRPMmax, time for pedaling rate to peak; Pmean, mean power output; RPMmean, mean pedaling rate; O_2Dem, O_2 demand; $\dot{V}O_2$, O_2 uptake; O_2D, O_2 deficit; Anaerobic, percentage of energy yield provided by the anaerobic metabolism; [La], blood lactate concentration in the femoral vein. *Significant difference from normoxia to hypoxia; †significant difference between sprint- and endurance-trained cyclists.

Hypoxia had opposed effects on O_2 deficit in sprint and endurance cyclists, inasmuch as, compared with normoxia, hypoxia resulted in a 5% lower O_2 deficit in the sprint cyclists ($P < 0.05$), whereas it promoted a 7% greater value in the endurance cyclists ($P < 0.05$). This different effect was further sustained by a significant interaction effect in the ANOVA analysis. In agreement, the endurance cyclists achieved an 11% greater area under the blood [La] curve during the recovery period after the hypoxic Wingate tests ($P < 0.05$; Fig. 3), whereas recovery [La] was unaffected by hypoxia in the sprint cyclists. During the 30-s exercise period, however, the rate of blood lactate accumulation was similar in normoxia and hypoxia (Fig. 1*G*). Fatigue index was also similar in normoxia and hypoxia (Fig. 2*A*). The anaerobic contribution to the energy expenditure in hypoxia was greater than in normoxia. However, this effect was significant only in the endurance cyclists ($P < 0.05$).

DISCUSSION

The effect of severe acute hypoxia, equivalent to an altitude of ~5,300 m, on exercise metabolism and performance has been studied in elite endurance and sprint-trained track cyclists. This study demonstrates that severe acute hypoxia does not alter peak power output and fatigue index during 30-s Wingate test. In contrast, Pmean is reduced with acute severe hypoxia in sprint but not in endurance-trained cyclists, despite the fact that in both groups mean $\dot{V}O_2$ was reduced by 16% during the Wingate tests in hypoxia. In addition, it has been shown that, compared with sprint, endurance-trained cyclists display superior maximal aerobic power, attain lower Ppeak and Pmean, achieve a lower O_2 deficit, have a lower fatigue index, obtain a greater fraction of the energy expended via oxidative pathways during a 30-s Wingate test, and show a lower femoral venous blood [La] after the Wingate test.

Effect of Severe Acute Hypoxia

In contrast to our hypothesis, Wingate test performance was only reduced with severe acute hypoxia in the elite sprint cyclists whereas it was maintained in the endurance cyclists. In both groups, $\dot{V}O_2$ was lowered in the same proportion (i.e., 16%), but because $\dot{V}O_2$ per kilogram of body mass was larger in the endurance cyclists, the absolute reduction of $\dot{V}O_2$ was slightly greater in the endurance than the sprint cyclists. If we assume similar cycling efficiencies in normoxia and acute hypoxia, the only possible explanation for these findings is superior anaerobic energy release in hypoxia (39). The fact that O_2 deficit increased with hypoxia in the endurance but not in the sprint cyclists supports this concept. Several metabolic pathways are stimulated to supplement energy production when aerobic metabolism is not capable of matching aerobic ATP production to consumption, especially the splitting of phosphocreatine and glycolysis. To our knowledge only McLellan et al. (23) have investigated the effect of moderate acute hypoxia on muscle metabolism during the Wingate test. They reported that, compared with normoxia, muscle [La] doubles when Wingate tests are performed in acute hypoxia. Because the glycolytic branch of the anaerobic metabolism represents ~3/4 of the anaerobic capacity (1, 3, 33), an increase in muscle lactate accumulation of the magnitude reported by McLellan et al. (23) would account for most of the reduction in aerobic ATP generation with hypoxia in the present study.

It has been shown that during all-out bicycling lactate production commences almost at the onset of muscular contractions reaching concentrations up to ~7 (11), ~11 (16), and ~17–29 mM (3, 16, 23, 28) in 6, 10, and 30 s, respectively. Despite the superior muscle lactate accumulation with hypoxia, recovery blood [La] (measured in a forearm vein) was found to be slightly lower in hypoxia in one study (23) and similar to the
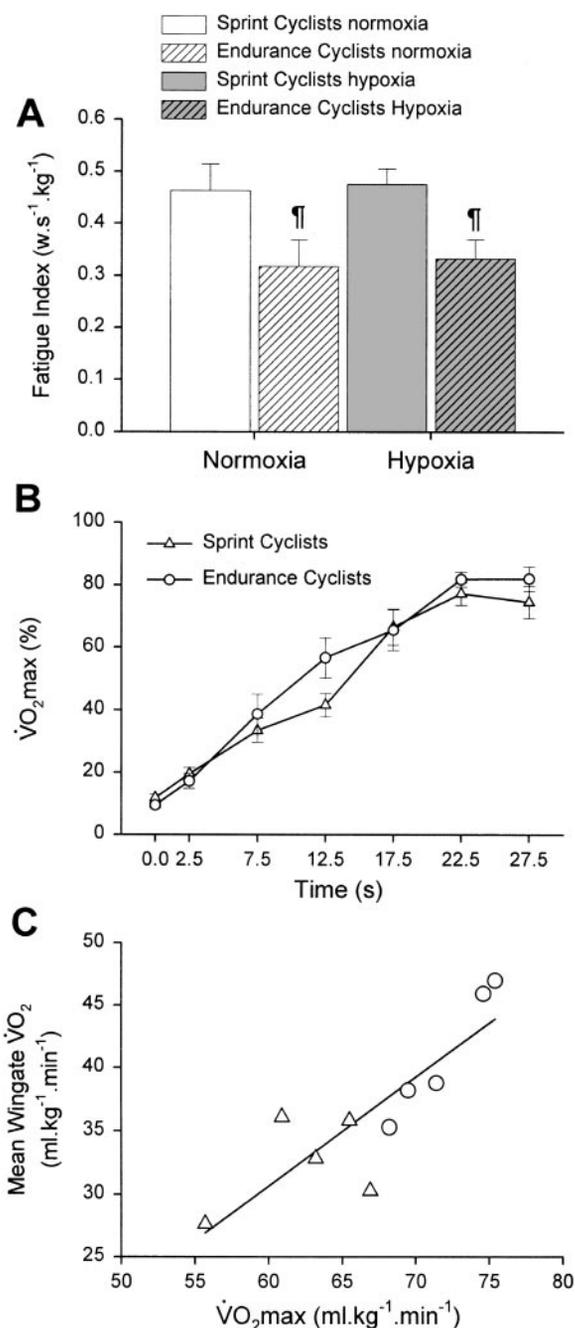
Fig. 2. *A*: fatigue index during 30-s Wingate tests. *B*: percentage of utilization of maximal V̇o₂ (Vo₂ₘₐₓ) during the Wingate tests performed in normoxia. *C*: relationship between mean V̇o₂ during the Wingate test in normoxia and Vo₂ₘₐₓ. ¶$P < 0.05$ sprint vs. endurance-trained cyclists.

normoxic conditions in another study from the same group (22). In a novel procedure, in the present study blood was sampled from the vicinity of the contracting muscle because systemic and local arm metabolism may interfere with forearm [La]. Moreover, by sampling blood every 5 s we have been able to measure for the first time the kinetics of femoral vein blood [La] accumulation during Wingate tests. Our data demonstrate that the kinetics of femoral venous blood lactate accumulation is independent of $F_{I_{O_2}}$ during 30-s all-out

exercise. In both conditions there was a 15-s lag between the onset of exercise and the beginning of blood lactate accumulation, indicating that most of the lactate produced at the onset of exercise is retained inside the muscle. No clue is given by the present study to explain why lactate is not released during the first 15 s of exercise. If the principal motor driving lactate net
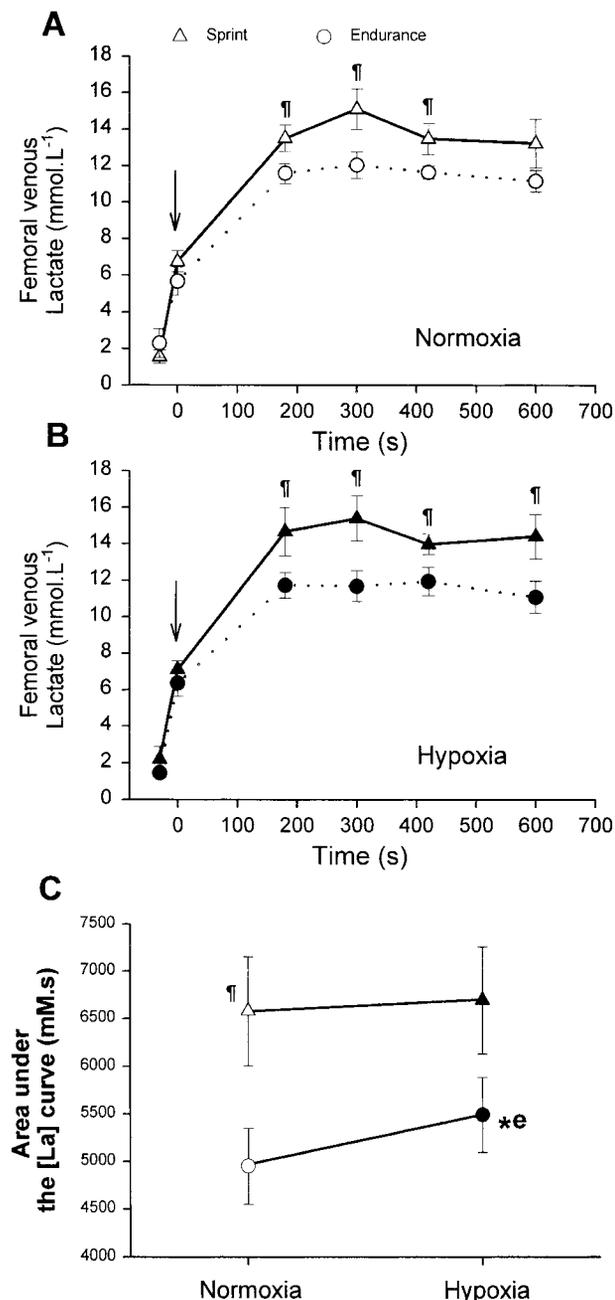
Fig. 3. *A*: recovery of femoral blood lactate concentrations ([La]) after the Wingate test in normoxia. *B*: recovery of femoral blood lactate concentrations after the Wingate test in hypoxia. *C*: area under the curve of blood [La] in the femoral vein during the first 10 min of the recovery period (basal values discounted). ¶$P < 0.05$ sprint- vs. endurance-trained cyclists. *e$P < 0.05$ normoxia vs. hypoxia, but only for the endurance cyclists. The vertical arrow indicates the time point corresponding to the end of the Wingate test (*time 0* of the recovery curve).
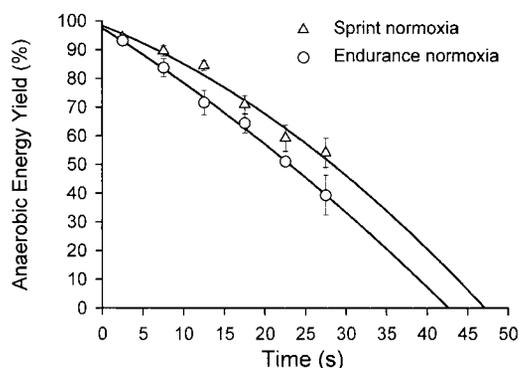
Fig. 4. Fraction of energy provided by anaerobic sources during 30-s Wingate tests performed in normoxia. A parabola was fitted to each set of data points ($r = 0.99$, $P < 0.01$) and extrapolated to 0% anaerobic contribution to estimate the time required to utilize the totality of the anaerobic capacity during an all-out test performed in normoxia.

release is muscle pH (30), the fact that muscle pH is barely changed at the onset of exercise could facilitate muscle lactate accumulation, or perhaps the muscle lactate transporters must first be activated.

A number of studies have shown that the total anaerobic capacity cannot be used up in 30 s (6, 7, 25, 26, 31, 33, 35). Hypoxia appears to stimulate additional utilization of the anaerobic capacity to compensate for the reduction in aerobic ATP production. This effect was clear in the endurance cyclists, whereas the sprint cyclists showed a small reduction in their O$_2$ deficit, which, combined with the lower $\dot{V}$O$_2$, led to decreased Pmean. The critical question is what limits performance during the Wingate test? Our findings contrast with the prevailing paradigm, which proposes that performance during the Wingate test is limited by the rate of anaerobic energy release (2, 7), which, in turn, depends on substrate availability and enzymatic control (7, 28, 35). It is well known that performance during the Wingate test is not limited by glycogen availability (16, 17, 23, 26). Our study novelly shows that neither the availability of anaerobic energy nor the rate of anaerobic energy release limits performance during the Wingate test in the elite endurance cyclists. Our results point to the rate of ATP utilization as the performance-limiting factor during the traditional Wingate test. According to our results, the factors reducing the rate of ATP hydrolysis should be common to the aerobic and anaerobic pathways. This requirement is fulfilled by ADP and P$_i$, which accumulate during all-out exercise and thus could set the upper limit of ATP utilization throughout their inhibitory effects on muscle contraction (9, 38). In contrast, according to our results, performance in sprint cyclists appears to be substrate limited in hypoxia, as reported in normoxia in nontrained (35) and physically active subjects (7).

It should be highlighted that the level of hypoxia used in our study is very close to the limit that a human can tolerate acutely. During incremental exercise to exhaustion under these conditions, arterial Po$_2$

values at exhaustion approached 30–35 mmHg (36). This level of hypoxia is very close to the limit that can be tolerated for a short time by an unacclimatized human and is similar to that reported in altitude-acclimatized subjects at the summit of Mt. Everest simulated in Operation Everest II (34). Severe hypoxemia, in turn, could have elicited central fatigue (27) or reduced peak leg blood flow (5). Although we cannot rule out a central component in fatigue appearance, in agreement with previous investigations performed with lower levels of hypoxia (22, 23), no effect of severe acute hypoxia on fatigue index or peak power output was found in this study. Furthermore, clinical signs of fatigue and the ventilatory response during the hypoxic Wingate tests (data not shown) were similar in both groups of cyclists. It does not seem, therefore, that differences in central fatigue mechanisms could explain the reduction of performance with hypoxia in the sprint cyclists.

Oxygen deficit, as measured in this study, is not a pure estimation of the anaerobic energy utilized because the O$_2$ consumed from the O$_2$ stores [especially O$_2$ bound to myoglobin, which has been estimated to be 2 mmol/kg wet wt (14), and O$_2$ bound to hemoglobin] at the onset of the exercise is computed as the O$_2$ deficit, leading to an overestimation of the anaerobic energy production and, conversely, an underestimation of the real $\dot{V}$O$_2$ (25). This intrinsic error of the deficit method is, however, rather small because of the comparatively low amount of O$_2$ stored in the skeletal muscle in relation to the magnitude of the O$_2$ deficit. If exercise bouts are performed in normoxia and hypoxia, the O$_2$ stores at the beginning of the exercise would be lower in the hypoxic condition, reducing the overestimation of the O$_2$ deficit. Resting myoglobin saturation, however, was likely very similar in normoxia and hypoxia, owing to the especial characteristics the O$_2$ dissociation myoglobin curve, which has an O$_2$ pressure at which myoglobin saturation is 50% that is close to 3 mmHg (30). With the level of hypoxia used in this study, a resting arterial Po$_2$ of 45–50 mmHg and saturation of ~80% have been reported (32). Consequently, the amount of O$_2$ stored as O$_2$ bound to hemoglobin was probably 20% lower at the onset of the hypoxic Wingate test. If this was actually the case, the hypoxia-normoxia O$_2$ deficit difference could have been even higher than the 7% calculated for the endurance cyclists, and perhaps no reduction in O$_2$ deficit with hypoxia would have been observed in the sprint cyclists. Assuming that both groups had a similar reduction in their O$_2$ stores with hypoxia, our data demonstrate that endurance cyclists have a greater capacity to enhance the anaerobic energy production in response to hypoxia than the sprint cyclists. Perhaps the sprint cyclists are already using their anaerobic energy at a rate close to the maximum in normoxia, and, thus, they are not able to compensate for a reduction in aerobic ATP production with hypoxia via an enhancement of rate of anaerobic energy release.

Differences Between Endurance and Sprint Specialists

One singularity of this study is the outstanding level of the cyclists examined, who were among the best in Spain with several very successful achievements in international competitions in both groups. In the case of the sprint cyclists, Ppeak and Pmean were among the highest reported in literature (8, 12, 22, 23, 40). It should be mentioned that the Pmax obtained in the Monark cycle ergometer was similar to and closely correlated with the Pmean measured during the first 30 s of a 500 m track sprint (measured in the 10 cyclists here studied with a SRM ergometer, data not shown; Ref. 8). The Pmean developed by these cyclists was far beyond the maximal aerobic power output; accordingly, O_2 deficit was also higher than reported in previous studies for cyclists (8, 13, 40). As expected, sprint cyclists had lower $\dot{V}O_{2\,max}$ but much larger O_2 deficit than the endurance specialists (8, 24). Assuming that the sprint cyclists utilized 80–90% of their anaerobic capacity in 30 s (6, 25, 31, 33), their actual maximal accumulated O_2 deficit can be estimated to lie in between 75 and 85 ml/kg. In fact, it has been suggested that world class sprint athletes may reach even higher O_2 deficits (8, 25, 31). The superiority of sprint cyclists in anaerobic power and capacity is probably the result of an increased percentage of type II fibers, more appropriate enzymatic machinery to produce ATP through anaerobic pathways, and enhanced buffer capacity (8, 13, 20, 21). However, fatigue index was markedly higher in the sprint than the endurance cyclists. Because sprint cyclists have a higher proportion of type II fibers than the endurance cyclists (21), they should choose a faster pedaling rate, because type II fibers are more efficient at a faster contraction speed (15). The drawback of this strategy is that type II fibers are less resistant to fatigue (15). The election of high pedaling frequencies may be advantageous for speed in track competitions (37). In an interesting experiment using an isokinetic cycle ergometer, Jones et al. (17) demonstrated that the same Pmean is developed at 60 and 140 rpm, but peak power output is considerably higher at 140 rpm, at the price, however, of an increased fatigue index.

Withers et al. (40) reported that endurance-trained cyclists can utilize 94% of their anaerobic capacity in a 45-s all-out test. Using a different approach, we have estimated that the endurance cyclists might utilize the totality of their anaerobic capacity in 43 s whereas the sprint cyclists would need 47 s (see Fig. 3).

Limitations of the O_2 Deficit as a Measure of the Anaerobic Energy Yield

Despite the limitations of O_2 deficit as a method to estimate the anaerobic energy yield, we should emphasize that our estimations on the partitioning between aerobic and anaerobic energy sources during the Wingate test agree amazingly well with the ATP turnover rates reported by Parolin et al. (28). These authors measured the aerobic and anaerobic ATP turnover rate in muscle biopsies obtained at three different time points during an isokinetic Wingate test at 100 rpm. During the last 15 s of the Wingate test, the mean aerobic contribution to the energy expenditure was 54% in Parolin et al.'s work and 48% in our endurance cyclists. The small difference between our estimations and the biopsy data of Parolin et al. probably reflects the fact that lactate release from the muscle was not accounted for in Parolin et al.'s work. Thus they probably underestimated slightly the anaerobic contribution during the last 15 s of the Wingate test, a period in which we have shown an increase in femoral venous lactate.

The real values of O_2 deficit could be lower or higher than reported here, but if we assume that mechanical efficiency during the Wingate tests was similar in normoxia and acute hypoxia, as has been demonstrated for submaximal exercise, then our conclusion that anaerobic energy production is increased during 30-s all-out exercise in hypoxia to account for the reduction in $\dot{V}O_2$ is irrefutable.

In summary, the effects of severe acute hypoxia, equivalent to an altitude of ~5,300 m, on exercise metabolism and performance have been studied in elite endurance- and sprint-trained track cyclists. We have demonstrated that peak power output and fatigue index are not altered by severe acute hypoxia, whereas mean $\dot{V}O_2$ is reduced by 16% in endurance- and sprint-trained cyclists. Interestingly, despite this marked reduction in $\dot{V}O_2$, only endurance-trained cyclists are able to maintain Pmean by increasing their anaerobic energy production, which shows that neither anaerobic capacity nor the rate of anaerobic energy release limits Wingate test performance in endurance cyclists. Conversely, a small decrease, inferior to that expected from the reduction in $\dot{V}O_2$, of Pmean with hypoxia is observed in the sprint-trained cyclists. Endurance-trained cyclists, on the other hand, possess superior maximal aerobic power and obtain a greater fraction of the energy expended during a Wingate test via oxidative pathways than sprint-trained cyclists. In turn, sprint-trained cyclists display larger O_2 deficit (+33%), rely more on anaerobic energy sources, and achieve higher peak and Pmean during a Wingate tests. Sprint-trained cyclists, however, develop fatigue at a faster rate than endurance-trained cyclists during supramaximal all-out exercise. In contrast to the prevailing paradigm, this study shows for the first time that performance during the traditional Wingate test is not limited by anaerobic energy supply in endurance cyclists.

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