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Research article

Influence of Acute Normobaric Hypoxia on Physiological Variables and Lactate Turn Point Determination in Trained Men

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Abstract

The goal of this study is to evaluate the response of physiological variables to acute normobaric hypoxia compared to normoxia and its influence on the lactate turn point determination according to the three-phase model of energy supply (Phase I: metabolically balanced at muscular level; Phase II: metabolically balanced at systemic level; Phase III: not metabolically balanced) during maximal incremental exercise. Ten physically active (VO_2max 3.9 [0.49] l·min^{-1}), healthy men (mean age [SD]: 25.3 [4.6] yrs.), participated in the study. All participants performed two maximal cycle ergometric exercise tests under normoxic as well as hypoxic conditions (FiO_2 = 14%). Blood lactate concentration, heart rate, gas exchange data, and power output at maximum and the first and the second lactate turn point (LTP_1, LTP_2), the heart rate turn point (HRTP) and the first and the second ventilatory turn point (V_ETP_1, V_ETP_2) were determined. Since in normobaric hypoxia absolute power output (P) was reduced at all reference points (max: 314 / 274 W; LTP_2: 218 / 184 W; LTP_1: 110 / 96 W), as well as VO_{2max} (max: 3.90 / 3.23 l·min^{-1}; LTP_2: 2.90 / 2.43 l·min^{-1}; LTP_1: 1.66 / 1. 52 l·min^{-1}), percentages of P_{max} at LTP_1, LTP_2, HRTP and V_ETP_1, V_ETP_2 were almost identical for hypoxic as well as normoxic conditions. Heart rate was significantly reduced at P_{max} in hypoxia (max: 190 / 185 bpm), but no significant differences were found at submaximal control points. Blood lactate concentration was not different at maximum, and all reference points in both conditions. Respiratory exchange ratio (RER) (max: 1.28 / 1.08; LTP_2: 1.13 / 0.98) and ventilatory equivalents for O_2 (max: 43.4 / 34.0; LTP_2: 32.1 / 25.4) and CO_2 (max: 34.1 / 31.6; LTP_2: 29.1 / 26.1) were significantly higher at some reference points in hypoxia. Significant correlations were found between LTP_1 and V_ETP_1 ($r = 0.778$; $p < 0.01$), LTP_2 and HRTP ($r = 0.828$; $p < 0.01$) and V_ETP_2 ($r = 0.948$; $p < 0.01$) for power output for both conditions. We conclude that the lactate turn point determination according to the three-phase-model of energy supply is valid in normobaric, normoxic as well as hypoxic conditions. The turn points for La, HR, and V_E were reproducible among both conditions, but shifted left to lower workloads. The lactate turn point determination may therefore be used for the prescription of exercise performance in both environments.

Key words: Hypoxia, threshold determination, performance, heart rate, spiroergometry.

Introduction

Hypobaric (HH) as well as normobaric hypoxia (NH) are well-described situations and several authors have shown this impact on exercise performance and related physiological variables (Angermann et al., 2006; Friedmann et al., 2007; Petrassi et al., 2012). Differences in physiological responses of HH and NH are reported in an actual paper (Girard et al., 2012). Some papers showed a linear reduction of exercise performance and oxygen uptake with increasing altitude (Benoit et al., 1997; Grataloup et al., 2007; Mollard et al., 2007a). The decrease in maximal exercise performance under hypoxic conditions was shown to be dependent on maximal exercise performance in normoxia even this relationship is not always reported as linear (Faiss et al., 2014; Wehrlin and Hallen, 2006). Performance at submaximal parameters such as the anaerobic threshold was shown to be significantly reduced by hypoxia, but the reliability and validity of different thresholds in normoxia and normobaric hypoxia have not been investigated yet.

Additionally, the impact of chronic hypoxia on the human body and its adaptation processes have been investigated extensively; however, only a few papers were found about the impact of acute normobaric hypoxia (Benoit et al., 2003; Calbet et al., 2003; Fukuda et al., 2010; Gallagher et al., 2014; Richard and Koehle, 2012; Soroko et al., 2012).

A better understanding of the typical acute responses in these conditions could be essential for training supervision as well as for medical purposes (McKenzie, 2012). Skinner and McLellan had already described a three-phase behaviour of selected physiological variables during incremental exercise in 1980 (Hofmann et al., 1997; Skinner and McLellan, 1980; Smekal et al., 2012). This concept has been adapted and may be seen as the standard model to describe exercise performance (Hofmann and Tschakert, 2011). The concept is based on the lactate shuttle theory (Brooks, 1986; 2009) which implies three specific phases of energy supply and consequently two turn points namely the first (LTP_1) and the second (LTP_2) lactate turn point (Hofmann et al., 1994a; 1997; 2001). They were shown to be objective markers of performance to describe defined metabolic conditions and the maximal lactate steady state (Hofmann et al., 1994a; Smekal et al., 2012; Wonisch et al., 2002). However, no studies were found to investigate these lactate turn points in hypoxic conditions.

Several authors showed contrary outcomes for the

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impact of acute hypoxia on selected physiological variables and thresholds. One of the reasons for these differences could be the use of different test protocols and the application of different fractions of oxygen in the air (FiO_2) (Angermann et al., 2006; Calbet et al., 2003; Friedmann et al., 2004). Some authors reported a loss of power output and a reduced maximal heart rate in acute hypoxia (Angermann et al., 2006; Grataloup et al., 2007; Mollard et al., 2007b). In addition, some authors reported a higher reduction in power output in trained athletes compared to untrained participants. However, other authors reported no reduction of maximal heart rate in hypoxia (Fukuda et al., 2010; Lawler et al., 1988; Peltonen et al., 2001). Some authors showed that the maximal lactate concentration in both hypoxia and normoxia was the same (Angermann et al., 2006; Benoit et al., 1997; Mollard et al., 2007b) while Marees reported higher lactate concentration in hypoxia than in normoxia at the same submaximal power output (Marees, 2003). This author also reported an increase of the tidal volume in hypoxia whereas no differences were found by Mollard et.al. (2007b). Rathat et.al suggested a test to identify high risk subjects for high altitude diseases (Rathat et al., 1992). Discrepancies were also shown for minute ventilation, where Marees (2003) claimed an increasing minute ventilation volume in hypoxia while other authors reported no differences (Marees, 2003; Grataloup et al., 2007; Orhan et al., 2010). All authors agreed that the VO_{2max} is clearly lower in hypoxia while the O_2-uptake at rest was higher in hypoxia compared to normoxia (Hofmann et al., 1994b; Mollard et al., 2007b). These effects were more pronounced in trained than in untrained participants. Furthermore, Friedmann et al. reported a higher maximal respiratory exchange ratio (RER) in hypoxia, while Fukada et al. could not find RER differences in hypoxic conditions (Friedmann et al., 2004; Fukuda et al., 2010). Additionally, the so-called lactate paradox at high altitudes was discussed by several authors (West, 2007; van Hall, 2007). However, comparison of data is difficult as most authors applied a different O_2-concentration (FIO_2 8-17%) and pressure mode (normobaric vs. hypobaric).

The aim of this study is therefore to determine and compare the lactate turn points to standard ventilatory turn points and the heart rate turn point in normoxia and normobaric hypoxia. According to our hypothesis, the maximal and the turn point variables were suggested to be not significantly different in relative and absolute terms independent of the environmental conditions.

Methods

The study was designed as a single-blinded randomized case control study. Ten moderately trained participants underwent two maximal incremental exercise tests under normoxic and normobaric hypoxic conditions (FIO_2 = 0.14 O_2 according to an altitude of 3500 m above sea level) in a hypoxic chamber. Air temperature and humidity were the same in both conditions. For inclusion, participants had to be at the age of 20-35, athletically active, healthy, and without any medication. The study was approved by the local ethics committee and meets the requirements of ICH-GCP as well as the requirements of the actual Declaration of Helsinki.

Both incremental exercise tests were performed on an electronically braked cycle ergometer (Ergoline ergometrics 800s, Ergoline, Germany) in a random order applying the following protocol. After 3 minutes without physical workload in a sitting position (R1) on the cycle ergometer, participants started to cycle at a workload of 40 watt for additional 3 minutes (W1), followed by an increase of 20 watt per minute up to voluntary exhaustion. The cycling cadence was set at 70 rpm. After completion of the maximal work load (P_{max}), participants worked at 40 W for 3 min. again (W2) and subsequently passively rested for additional 3 min. sitting on the cycle ergometer (R2). Between the first and the second exercise test participants had a break of 7 days for optimal recovery. The tests were performed by all participants at the same day and time. All participants were instructed to avoid any strenuous exercise the day before the tests and were allowed to pre-acclimatize in the altitude chamber 30 min before performing the test in both conditions. N_2-generators were running in both conditions to keep participants blinded for hypoxia. Submaximal turn points for blood lactate concentration (LTP_1, LTP_2), ventilation ($VETP_1$, $VETP_2$), and heart rate (HRTP) were calculated by means of linear regression break point analysis as shown previously (Binder et al., 2008; Hofmann et al., 1994b; Hofmann and Tschakert, 2011). The determination was performed within the same defined regions of interest for all selected variables.

Blood lactate concentration (La) was determined from capillary blood samples (Biosen S_line, EKF-diagnostic, Germany) taken from the hyperemized earlobe at rest, after warm-up, after every workload step and after 3 and 6 minutes of recovery.

Heart rate (HR) was measured continuously by means of a 12-lead ECG (Cardiovit AT-10, Schiller, Switzerland) and by means of a heart rate monitors (Polar PE 4000, Polar Electro, Finland). Data were stored in 5 second intervals for further analysis. Gas exchange variables as well as ventilation measured were assessed by breath-by-breath mode (intra-breath) and stored in 10 s intervals for further analysis (Cortex Metamax, Cortex Biophysik, Germany). The analyzers were calibrated before each test according to the manufacturers' guidelines. Ventilation was calibrated by a 1 L pump syringe, accordingly.

Statistical analysis

For statistical analysis, the software SPSS (IBM, Germany) was used. To test data for normal distribution, the Kolmogoroff-Smirnov test was applied. Data are presented as means ± standard deviation (SD). A two-sided paired t-test was used to detect significant differences between normoxia and hypoxia. Relationship between variables was detected by means of Pearson's product-moment correlation coefficient. Comparison between turn points was determined by ANOVA and Tukey's post-hoc test. A p-value of 0.05 was considered as significant.

Results

Participants were 25.3 (±4.6) years of age, 1.82 (±0.07) m of height and had a body mass of 75.9 (±6.7) kg. Table 1 represents maximal and submaximal performance and gas exchange variables in normoxia and normobaric hypoxia.

Maximal and threshold power output were significantly reduced in hypoxia ($p < 0.01$) and the decrease in power output was significantly related to normoxic P_{max} ($r = -0.91$) and P_{LTP2} ($r = -0.97$).

The variables significantly influenced by normobaric hypoxia were maximal oxygen uptake (VO_{2max}) and related variables (EQO_2, $EQCO_2$, RER), and maximal heart rate (Figure 1 and 2) ($p < 0.05$).

Table 1. Measured parameters in normoxia and hypoxia. Data are mean (±SD).

	rest		LTP_1		LTP_2		P_{max}	
	N	H	N	H	N	H	N	H
P (W)	0 (00)	0 (00)	110 (20)	96 (14)***	218 (40)	184 (22)***	314 (50)	274 (30)***
HR (bpm)	86 (17)	86 (14)	120 (10)	126 (9)	165 (9)	163 (8)	190 (8)	185 (7)**
La [mmol/l]	1.1 (.2)	1.0 (.4)	1.4 (.2)	1.4 (.4)	4.1 (.9)	3.8 (.9)	11.4 (2.3)	10.4 (2.3)
VO_2 ($l \cdot min^{-1}$)	.43 (.12)	.46 (.09)	1.66 (.26)	1.52 (.19)	2.90 (.34)	2.43 (.43)*	3.90 (.49)	3.23 (.48)*
VCO_2 ($l \cdot min^{-1}$)	.37 (.11)	.42 (0.09)	1.44 (.22)	1.47 (.11)	2.83 (.32)	2.67 (.35)	4.20 (.51)	4.08 (.49)
V_E [($l \cdot min^{-1}$)	12.5 (3.2)	15.3 (4.7)	37.8 (5.2)	42.3 (5.3)*	73.4 (12.3)	76.9 (10.8)	132.1 (24.8)	138.2 (19.7)
EQO_2	28.7 (5.2)	32.8 (6.0)	23.1 (3.7)	28.1 (4.3)*	25.4 (4.1)	32.1 (4.0)**	34.0 (5.7)	43.4 (7.1)**
$EQCO_2$	33.5 (6.1)	35.8 (10.4)	26.7 (4.2)	29.1 (4.3)*	26.1 (4.6)	29.0 (4.3)**	31.6 (5.3)	34.1 (4.5)*
RER	.86 (.07)	.91 (.14)	.87 (.04)	.98 (.16)	.98 (.04)	1.13 (.20)*	1.08 (.05)	1.28 (.20)**

LTP: Lactate Turn Point; P_{max}: maximal power; N: normoxia; H: hypoxia; P: power; HR: heart rate; La: lactate concentration; VO_2: oxygen uptake; VCO_2 CO_2 release; V_E: ventilation; EQO_2:ventilatory equivalent for O_2; $EQCO_2$: ventilatory equivalent for CO_2; RER: respiratory exchange ratio. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

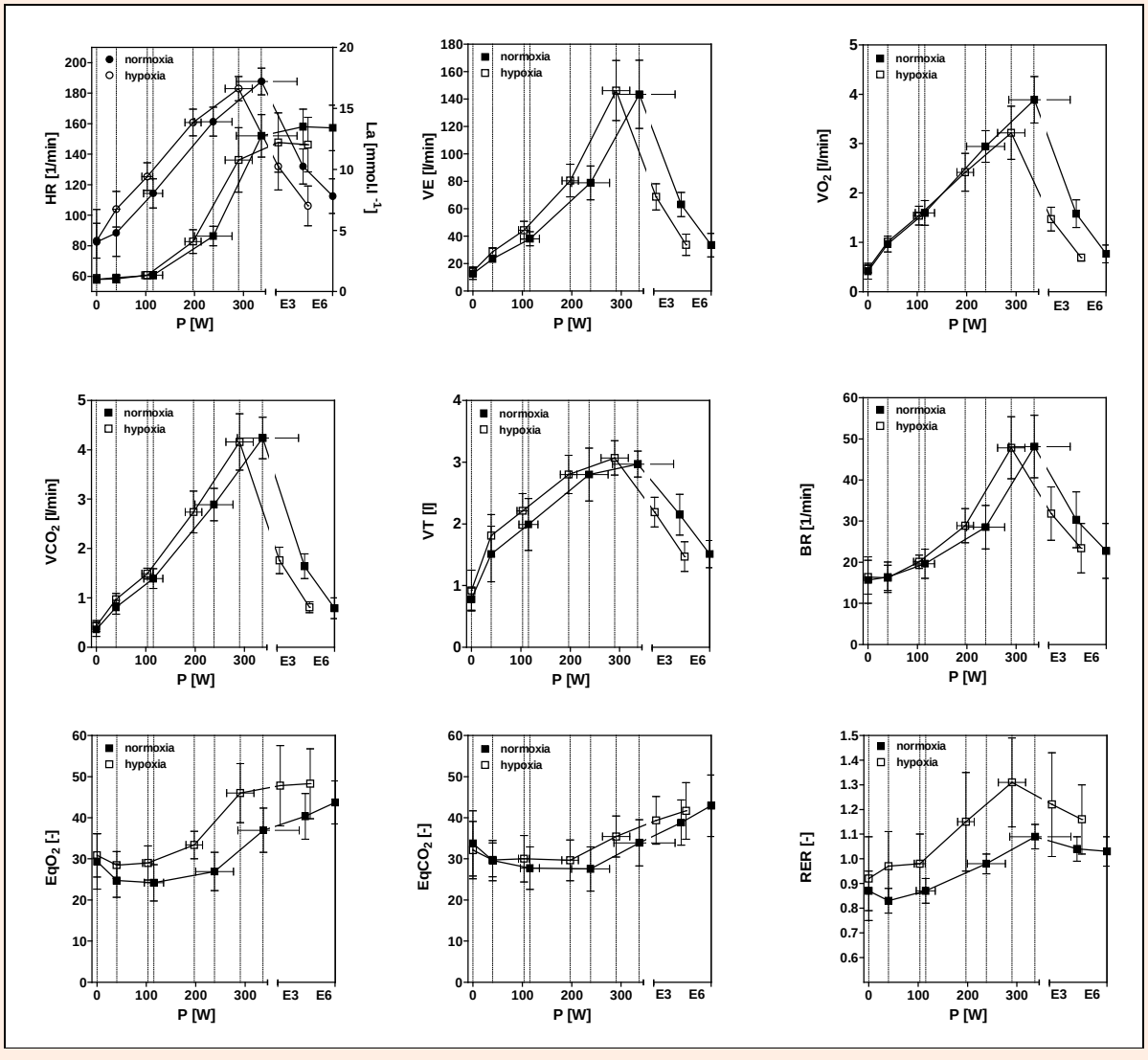

Figure 1. Absolute values of measured parameters in normoxia (N) and hypoxia (H). P: power; HR: heart rate; La: lactate concentration; V_E: ventilation; VO_2: oxygen uptake; VCO_2 CO_2 release; tidal volume (VT); breathing rate (BR); EQO_2: ventilatory equivalent for O_2; $EQCO_2$: ventilatory equivalent for CO_2; RER: respiratory exchange ratio.

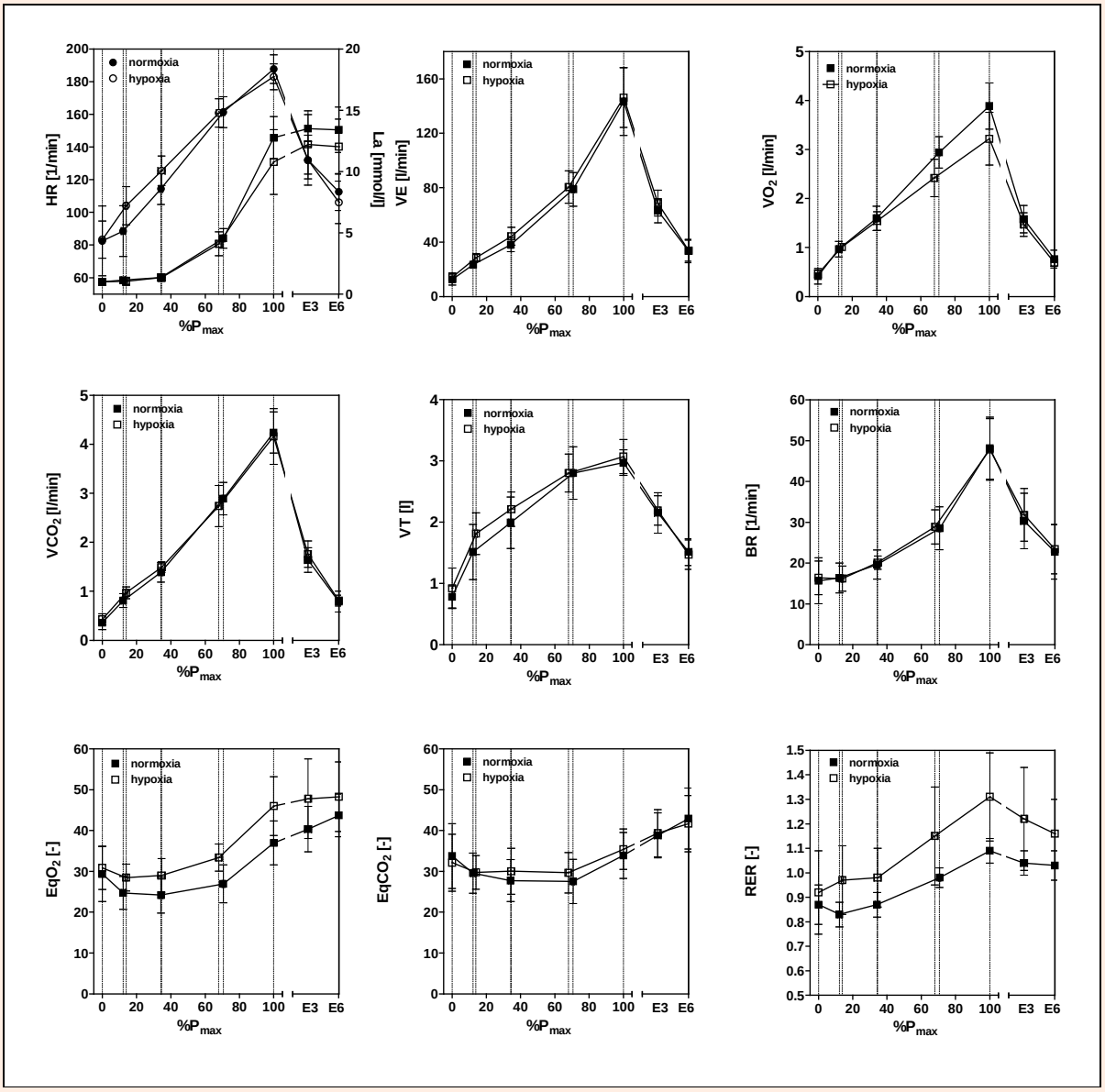

Figure 2. Measured parameters in relative terms in normoxia and hypoxia. P: power; HR: heart rate; La: lactate concentration; V_E: ventilation; VO_2: oxygen uptake; VCO_2 CO_2 release; tidal volume (VT); breathing rate (BR); EQO_2: ventilatory equivalent for O_2; $EQCO_2$: ventilatory equivalent for CO_2; RER: respiratory exchange ratio.

Although the lactate-power output curve was left shifted, we found completely the same pattern of the curve and no significant difference in lactate concentration between normoxia and hypoxia in relative terms (Table 1). Concerning net lactate production, the same lactate concentration per watt was found in normoxia and normobaric hypoxia.

Power output at V_ETP_1 (N: 119.6 [±29.1] W; H: 83.5 [±24.0] W) was not significantly different from P LTP_1 in normoxia but significantly different in hypoxia ($p < 0.05$). Power output at V_ETP_2 (N: 217.9 [±48.4] W; H: 186.8 [±27.2] W) was not significantly different from P HRTP (N: 216.0 [±35.6] W; H: 178.5 [±28.0] W) and P LTP_2 (Table 1) in normoxia and hypoxia.

A significant linear correlation was found for power output at LTP_1 and V_ETP_1 ($r = 0.778$, $p < 0.01$), as well as for the second lactate turn point (LTP_2) and V_ETP_2 ($r = 0.948$; $p < 0.01$) and LTP_2 and the HRTP ($r = 0.828$; $p < 0.01$). This relationship was present, and similar in both normoxic and hypoxic conditions. There was no significant difference in both environmental milieus for breathing rate and tidal volume at all reference points, although there was a tendency for higher values in hypoxia. The ventilation was significantly higher in hypoxia at W1, LTP_1 and R2 ($p < 0.05$), and there was a tendency for higher values at the other reference points. In our participants the O_2-uptake was significantly reduced in hypoxia from the average of 150 W upwards. Participants with higher VO_{2max} in normoxia, demonstrated a more pronounced reduction of this parameter in hypoxia in absolute terms as well as relative to P_{max} ($r = -0.76$, $p < 0.05$). Oxygen uptake per watt was the same in normoxia and hypoxia at maximum and at the second reference points (LTP_2, HRTP, V_ETP_2). There was a trend for higher val-

ues in CO_2 release in hypoxia which was significant only for W2 ($p < 0.05$). The RER was significantly higher in hypoxia at almost all reference points ($p < 0.05$) except LTP_1 ($p = 0.054$). The ventilatory equivalent for O_2 was significantly higher ($p < 0.05 – p < 0.01$) in hypoxia at all reference points except at R1 and R2 as well as the ventilatory equivalent for CO_2 which was higher in hypoxia at LTP_1, LTP_2, and P_{max} ($p < 0.05$) in absolute and relative terms. The changes in oxygen uptake may be explained by the reduction in submaximal and maximal power output and related variables. All other variables remained the same in absolute terms at maximum and in absolute and relative terms at the thresholds, which were consistent throughout conditions, except HR_{max}.

Discussion

Our study confirms the consistency of the three-phase and two turn point concept for the blood lactate concentration theoretically founded by the lactate shuttle theory for normoxic as well as hypoxic conditions (Brooks, 1986; 2009). The turn points in La, HR, and V_E were stable among conditions, but exercise performance was reduced.

Koistinnen et al. (1995) described a correlation of the lactate threshold in hypoxia and normoxia recently but they used a different model for calculation of the metabolic phases. The principal pattern of all variables relative to maximum power output was unchanged due to hypoxia, the turn points were reproducible in both conditions and not significantly different. This allows the assumption that the two turn point and three-phase concept of lactate concentration may be seen as the standard model of energy contribution during incremental exercise as long as a certain protocol is applied independently of environmental O_2-concentration. In general, the well-described significant reduction of maximal oxygen uptake and maximal power output was found in acute normobaric hypoxia in healthy trained participants. All other variables measured did not change significantly in relative terms except HR_{max}, which suggest that only the reduction in oxygen availability in normobaric hypoxia shifted the work performance curve to the left but without any changes in relative terms.

A correlation of the absolute and relative hypoxia decrease of P_{max} related to normoxic P_{max} as found confirms the results of other authors (Fukuda et al., 2010; Mollard et al., 2007a). We suggest that the higher the VO_{2max} of a subject is, the stronger is the influence of a reduction of oxygen availability in ambient air. As expected, power output at LTP_1 and LTP_2 was also significantly reduced in absolute terms but was identical with respect to relative terms (% P_{max}), which is a novel result. Additionally, lactate turn points (LTP_1, LTP_2) were not significantly different from the ventilatory turn points (V_ETP_1, V_ETP_2) and the heart rate turn point (HRTP) in both conditions, suggesting the turn point determination for lactate to be a valid and reliable method which is independent from oxygen availability. This is clearly in line with the lactate shuttle theory concept, which implies three distinct phases of energy supply also for hypoxic conditions (Hofmann et al., 1997; Smekal et al., 2012).

Maximal lactate concentration was the same in both conditions, which supports the findings of previous research (Angermann et al., 2006; Benoit et al., 2003; Mollard et al., 2007a). Lactate concentration at LTP_1 and LTP_2 was identical in both conditions, but the lactate power curve was left shifted. Lactate concentration with respect to relative power output (% P_{max}) was identical, which suggests the same anaerobic energy contribution in both conditions. The decrease in absolute power may be explained solely by the limitations in the aerobic capacity namely oxygen availability which could not be compensated. As maximal lactate concentrations were similar in both conditions, it is suggested that the anaerobic capacity was utilized to the same extent. Expectedly, the VO_{2max} was significantly reduced in hypoxia. The effects of O_2-delivery deficiency were significantly correlated to exercise performance where the higher trained participants presented a stronger decrease. This was already reported by several authors and has been explained by different mechanisms, i.e. ventilation/perfusion inequality or alveolar-pulmonary capillary diffusion limitation. (Benoit et al., 2003; Mollard et al., 2007a; Martin and O'Kroy, 1993). Our results are according to the results by Hopkins et al., who reported that the resting O_2-uptake was identical in both conditions and disagrees with Friedmann et al. and their findings of higher values for O_2-uptake in hypoxia (Friedmann et al., 2004; Hopkins et al., 2003).

An interesting finding was the fact that O_2-uptake was found identical up to a power output of about 150 watt in our participants. This might be explained that up to this point O_2-saturation of the participants' was adequate and metabolism was primarily aerobic. These findings support the report of Ibanez et al. who found O_2-uptake not to be reduced below a work load of 125 watt (Ibanez et al., 1993). However, it disagrees with Benoit et al. (1997) and Brooks et al. (1998) showing higher O_2-uptake from rest to P_{max} . No significant differences were found for breathing rate (BR), tidal volume (VT), and minute ventilation (V_E), but a tendency for higher values in hypoxia was observed that supports findings of previous studies (Angermann et al., 2006; Benoit et al., 2003; Calbet and Lundby, 2009). The evaluation of the RER presented identical values in R1 and significantly higher values at all other reference points in hypoxia. These findings were in contrast to Friedmann et al. (2004) and Fukada et al. (2010), but we suggest that our consistent threshold determination explains these discrepancies for both conditions (Friedmann et al., 2004; Fukuda et al., 2010). The ventilatory equivalent for O_2 and CO_2 was increased in hypoxia which is in agreement with previous studies and may be explained by a clearly reduced breathing efficiency (Calbet and Lundby, 2009; Mollard et al., 2007a).

The heart rate at rest was identical in both study conditions which contrasts the findings of Kato et.al. (2004). We measured HR during 3 minutes of resting conditions; before testing participants may have been nervous. In agreement with other authors we found the maximal heart rate significantly reduced in hypoxia (Mollard et al., 2007b); however no correlation for athletes' performance was found. Several factors were de-

scribed to explain the hypoxia-induced reduction in exercise performance. Reduced muscle O_2-delivery and exercise performance may result from impairments in pulmonary gas exchange such as alveolar-capillary O_2-diffusion limitation (Verges et al., 2012). Under the assumption that all athletes were motivated and performed the tests up to maximal possible individual power output, which was supported by the fact of the same measured maximal ventilation in normoxia and hypoxia, the cause of the reduced HR_{max} may be explained by a protecting mechanism such as a reduced central drive. Verges et al. suggested an accelerated rise in central motor drive in hypoxia to compensate for increasing muscle contractile fatigue (Verges et al., 2012). An accelerated development of muscle fatigue in moderate hypoxia may therefore be responsible for increased inhibitory afferent signals to the central nervous system leading to impaired central drive. Obviously, the organism shortly adapts its functions to the reduced oxygen availability and shifts to lower power output without changing the principal patterns of physiological functions. The variable significantly reduced was oxygen uptake. The similarity of patterns can be clearly seen in the high reproducibility of the turn points, and the consistency of turn points in both normoxia and normobaric hypoxia for the different variables applied in turn point determination.

Limitations of the study

It has to be noted that the interpretation of our results is limited to an oxygen content of 14% O_2 in normobaric air conditions and results may be different in real high altitude hypobaric hypoxia of 3500 m above sea level.

Conclusion

We conclude that the lactate turn point determination is valid in normobaric normoxic as well as in hypoxic conditions and the turn points in La, HR, and V_E were stable among conditions, but exercise performance was reduced. The lactate turn point concept may therefore be used for exercise performance testing in both conditions.

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Key points

- The lactate turn point concept can be used for performance testing in normoxic and hypoxic conditions
- The better the performance of the athletes the higher is the effect of hypoxia
- The HRTP and LTP_2 are strongly correlated that allows a simple performance testing using heart rate measures only.

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