Eur J Appl Physiol (1990) 59:421–429

European Journal of Applied Physiology and Occupational Physiology

The effects of training on the metabolic and respiratory profile of high-intensity cycle ergometer exercise*

David C. Poole, Susan A. Ward, and Brian J. Whipp

Departments of Anesthesiology, Kinesiology and Physiology, UCLA, Los Angeles, California 90024, USA

Summary. The tolerable work duration (t) for high-intensity cycling is well described as a hyperbolic function of power (W): $W=(W'\cdot t^{-1})+W_a$, where W_a is the upper limit for sustainable power (lying between maximum W and the threshold for sustained blood [lactate] increase, Θ_{lac}), and W' is a constant which defines the amount of work which can be performed $>W_a$. As training increases the tolerable duration of high-intensity cycling, we explored whether this reflected an alteration of W_a, W' or both. Before and after a 7-week regimen of intense interval cycle-training by healthy males, we estimated ($\hat{}$) Θ_{lac} and determined maximum O_2 uptake ($\mu\dot{V}_{O_2}$); W_a; W'; and the temporal profiles of pulmonary gas exchange, blood gas, acid-base and metabolic response to constant-load cycling at and above W_a. Although training increased $\hat{\Theta}_{lac}$ (24%), $\mu\dot{V}_{O_2}$ (15%) and W_a (15%), W' was unaffected. For exercise at W_a, a steady state was attained for $\dot{V}_{O_2}$, [lactate] and pH both pre- and post-training, despite blood [norepinephrine] and [epinephrine] ([NE], [E]) and rectal temperature continuing to rise. For exercise $>W_a$, there was a progressive increase in $\dot{V}_{O_2}$ (resulting in $\mu\dot{V}_{O_2}$ at fatigue), [lactate], [NE], [E] and rectal temperature, and a progressive decrease for pH. We conclude that the increased endurance capacity for high-intensity exercise following training reflects an increased W asymptote of the $W-t$ relationship with no effect on its curvature; consequently, there is no appreciable change in the amount of work which can be performed above W_a. Furthermore, regardless of training status, W_a represents the upper power limit at which $\dot{V}_{O_2}$, blood [lactate] and blood pH can eventually be stabilized. Exercise $>W_a$, in contrast, is characterized by a steadily increasing $\dot{V}_{O_2}$ and blood [lactate], a falling blood pH and consequently, imminent fatigue.

Key words: Lactate threshold — Fatigue threshold — Oxygen uptake — Catecholamines — Blood gas and acid-base status

Introduction

The power-duration relationship (i.e., W vs t) for exhausting high-intensity exercise, first documented by Hill in 1927, has recently been characterized as a rectangular hyperbola (Monod and Scherrer 1965; Moritani et al. 1981; Whipp et al. 1982):

$$(W-W_a)\cdot t=W' \qquad \text{(a)}$$

where W_a[1] is the power asymptote, which lies above the lactate threshold[2] (Θ_{lac}) but below the maximal power attained with incremental cycle ergometry (Moritani et al. 1981; Whipp et al. 1982), and the constant W' represents a finite amount of work which can be performed above

Offprint requests to: D. C. Poole, Department of Medicine, M-023A, University of California, San Diego, La Jolla, CA 92093, USA

* Supported in part by a UCLA Graduate Division Doctoral Research Award

[1] The parameter W_a is the asymptote of power for the power-duration relationship and consequently represents a unique work rate above which the time to fatigue is predictably determined. W_a may be expressed with regard to power output (watts) or the asymptotic $\dot{V}_{O_2}$ which obtains at W_a (i.e., $W_a\{O_2\}$)

[2] The term lactate threshold (Θ_{lac}) is used to denote the $\dot{V}_{O_2}$ at which blood [lactate] starts to increase systematically during a particular form of exercise

W_a independent of its rate of performance. This relationship may be transformed to yield:

$$W = W' \cdot t^{-1} + W_a \quad \text{(b)}$$

where W' and W_a are the slope and intercept, respectively, of the linear $W-t^{-1}$ relationship.

The physiological determinants of W' and W_a remain conjectural. However, W' may usefully be regarded as an energy store which is comprised of O_2 stores, a phosphagen pool, and a source related to anaerobic glycolysis and the consequent production and accumulation of lactate (which has been considered to be equivalent to the O_2 deficit: diPrampero 1981). W_a may represent a rate of energy-pool reconstitution (which can be expressed either in terms of work rate or in terms of $\dot{V}_{O_2}$ as the O_2 cost of the exercise, i.e., $W_a\{O_2\}$) which dictates the maximum power which can be sustained without a continued and progressive anaerobic contribution. Below W_a, exercise can therefore proceed beyond the initial transient phase without further depletion of the stored resources, with the energy stores (W') being reduced but not to critical levels (Hill 1927; Monod and Scherrer 1965; Moritani et al. 1981; Whipp et al. 1982).

It is not known whether the $W-t$ relationship remains hyperbolic after training and if so which of the individual parameters (i.e., W_a, W') are altered — or if both are. We therefore chose to investigate the consequences of an intense interval-training regimen on the power-duration relationship for high-intensity exercise, with particular reference to metabolic, ventilatory, blood-gas and acid-base response profiles at and above W_a. We hypothesized that, as such training improved performance predominantly through improvements in O_2 transfer and utilization, W_a would be increased with little or no effect on the curvature constant W'.

Methods

Eight healthy men participated in this study: age ($\pm$SD) = 22.0 $\pm$ 1.9 y, mass = 75.0 $\pm$ 13.0 kg, and height = 1.76 $\pm$ 0.08 m. Prior to, and following, completion of a training program (see below), each subject performed incremental and square-wave exercise tests on an electromagnetically-braked cycle ergometer (Mijnhardt, KEM-2); although work rate is independent of pedaling frequency between 30 and 90 min^{-1} for this ergometer, the subjects maintained a frequency of $\sim$60 min^{-1} during the tests. Expired airflow was measured with a pneumotachograph (Rudolph, no. 3800) downstream of the expiratory port of a low-resistance breathing valve (Rudolph, no. 2700, dead space: 90 ml), connected to a variable-reluctance manometer (Validyne, no. MP45: $\pm$2 cm H_2O); the pneumotachograph was maintained at 37° C by a thermal feedback device and calibrated by inputting known volumes of room air at several mean flows. Rapidly-responding analyzers continuously monitored PCO_2 (Datex, no. CD101; 90% response time: 0.1 s) and PO_2 (Applied Electrochemistry, no. S3A: 90% response time: <0.08 s) in respired gas sampled from the mouthpiece; precision-analyzed gas mixtures were used for calibration. Heart rate (HR) was derived from the R-R interval of an ECG signal (Birtcher, no. 7000). The electrical signals from these devices underwent analog-to-digital conversion and computer analysis (Tektronix, no. 4052) for on-line, breath-by-breath determination of ventilation ($\dot{V}_{E,BTPS}$), CO_2 output ($\dot{V}_{CO_2,STPD}$), O_2 uptake ($\dot{V}_{O_2,STPD}$), ventilatory equivalents for CO_2 and O_2 ($\dot{V}_E/\dot{V}_{CO_2}$, $\dot{V}_E/\dot{V}_{O_2}$), respiratory exchange ratio (R), and end-tidal PCO_2 and PO_2 ($P_{ET}CO_2$, $P_{ET}O_2$).

Each subject performed an incremental exercise test (25 W/min) to the limit of tolerance (μW) for estimation ($\hat{}$) of the lactate threshold ($\hat{\Theta}_{lac}$) and the maximum $\dot{V}_{O_2}$($\mu\dot{V}_{O_2}$). $\hat{\Theta}_{lac}$ was established as the $\dot{V}_{O_2}$ at which $P_{ET}O_2$ and $\dot{V}_E/\dot{V}_{O_2}$ started to rise systematically without a simultaneous increase in $\dot{V}_E/\dot{V}_{CO_2}$ and a decrease in $P_{ET}CO_2$ (Whipp and Mahler 1980). $\mu\dot{V}_{O_2}$ was established as the highest $\dot{V}_{O_2}$ attained. To define the $W-t$ characteristic for high-intensity exercise, each subject then undertook a series of at least 5 different maximal square-wave exercise tests. These were always preceded by a 5-min baseline of unloaded pedaling, and we deliberately avoided power outputs which would induce exhaustion in less than 1 min. The time to fatigue was taken as the interval between the imposition of the load and the point at which the subject could no longer maintain the required pedaling rate. Only one test was undertaken on a given day and the sequence was randomized; the subjects were not told for how long or at what power they had exercised. Reproducibility was assessed by repeat tests assigned randomly to subjects. The time to fatigue at a given power for a given subject showed only minor variation: e.g., <5–10 s (<2–4%) for tests of $\sim$4 min duration, and <20–30 s (<4–6%) for tests of $\sim$8 min duration. The $W-t$ characteristic was linearized (eq. b), and the slope (W') and intercept (W_a) parameters extracted by least-squares linear regression techniques. While this relationship has traditionally been presented with "power" as the dependent variable there appears to be no *a priori* reason for this. However, when power is plotted as the independent variable for tests in which the correlation of P vs $1/t$ is high (i.e. >0.94) neither the intercept (W_a) nor the slope (W') are changed appreciably.

Each subject then performed at least 2 additional square-wave tests on different days at power outputs equivalent to W_a ("W_a" test) and to W_a + 5% of μW ("> W_a" test). The exercise was continued for 24 min or until the subject could no longer sustain the power. Rectal temperature was monitored (Yellow Springs Instrument Co., no. 400/44TA). Arterialized venous blood was sampled from an indwelling catheter (18 ga.) in a superficial vein on the heated dorsum of the hand (Forster et al. 1972). Samples were drawn at rest, during the steady-state of unloaded pedaling, and regularly throughout the work: at min 2 and every subsequent 4 min for lactate (2 ml); at min 4 and every subsequent 4 min for blood-gas and acid-base variables (1–2 ml); at min 8 and every subsequent 8 min for catecholamines (5–7 ml). PO_2, PCO_2, pH and $[HCO_3^-]$ were measured in duplicate using standard electrodes (Instrumentation Laboratories, no. 1303) calibrated before and after each measurement with certified standard buffers and gas mixtures; PCO_2 and pH measurements were corrected for temperature differences between measuring electrodes (37° C) and body (Kelman and Nunn 1966). Lactate concentration [lac] was

measured by a standard enzymatic technique (Hohorst 1963), and norepinephrine and epinephrine levels ([NE], [E]) by high-performance liquid chromatography (Watson 1981).

The training program consisted of supervised sessions undertaken 3 days/wk for a 7-wk period (with the exception of subjects 7 and 8 who completed 4 and 3 wk, respectively). Each session was comprised of 10 2-min exercise bouts (each separated by a 2-min rest period) performed on a cycle ergometer (Monark, no. 668) at a power output of 105% of μW. Each week, the maximal HR response attained on the 10th exercise bout was monitored; if it was more than 10 bt min^{-1} below the maximum obtained on the pre-training incremental test, the power was increased by a further 30 W. This training regimen has been demonstrated previously to cause substantial increases in both $\mu\dot{V}_{O_2}$ and Θ_{lac} (Poole and Gaesser 1985).

Differences between responses (i.e., pre- vs. post-training; W_a vs. $> W_a$ at specified times during the test) were assessed by a standard paired-comparisons t-test ($p < 0.05$). The Bonferroni correction was applied for repeated t-tests (Morrison 1976).

Results

Following training, the subjects were able to exercise to a greater μW (an average increase of 37.5 W, or 13%) and achieve a higher $\mu\dot{V}_{O_2}$ (increased, on average, by 0.58 $l \cdot min^{-1}$, or 15%) with the incremental test (Fig. 1; Tables 1 and 2). Likewise, $\dot{V}_E$ and $\dot{V}_{CO_2}$ at maximum both increased (Table 2). $\hat{\Theta}_{lac}$ was also greater after training (by 0.42 $l \cdot min^{-1}$ $\dot{V}_{O_2}$ or 24%, on average: Fig. 1; Table 1).

A representative example of the $W-t$ relationships for high-intensity cycling prior to and following the training in the same subject is given in Fig. 2 (upper panel). The excellent linear fit of the transformed $W-t^{-1}$ relationships (lower panel) indicates that the data are well described by a hyperbolic function: there was characteristically a high correlation coefficient, and this averaged 0.98 both pre- and post-training and never fell below 0.94 in any subject. W_a was significantly increased in all subjects, from an average pre-training value of 197 W to 217 W post-training and in each case W_a was substantially greater than $\hat{\Theta}_{lac}$ (i.e., averaging 164% of $\hat{\Theta}_{lac}$ pre-training and 131% post-training: Table 1). In contrast, the training had no significant effect on W'.

All subjects were able to continue the prolonged constant-load test conducted at W_a (i.e., 197 ± 12 W pre-training; 217 ± 11 W post-training) for the entire 24 min without appreciable duress. In all but one of the $> W_a$ tests (i.e., 213 ± 15 W pre-training; 240 ± 8.5 W post-training), the subjects became exhausted prior to the target duration of 24 min (average duration 17.7 ± 1.2 min pre-training; 17.1 ± 1.3 min post-training).

As we have reported previously (Poole et al. 1988), the prolonged exercise at W_a elicited a characteristic profile of metabolic and acid-base response which contrasted sharply with that found $> W_a$ (Fig. 3). The features of these responses were qualitatively similar for all subjects and were preserved following the training, though of course occurring at a higher work rate. For the W_a test, $\dot{V}_{O_2}$ increased rapidly over the first few minutes of the exercise, rising subsequently at a slower rate to attain a new steady-state within about 18 min on average (2.97 ± 0.11 $l \cdot min^{-1}$ or $79.0 \pm 3.1\%$ of $\mu\dot{V}_{O_2}$ pre-training; 3.42 ± 0.18 $l \cdot min^{-1}$ or $79.1 \pm 4.0\%$ of $\mu\dot{V}_{O_2}$ post-training: Table 1). Post-training, there was a continued, though slight, rise in the average $\dot{V}_{O_2}$ response for the W_a test. We recognize that a region of statistical and physiological uncertainty in the location

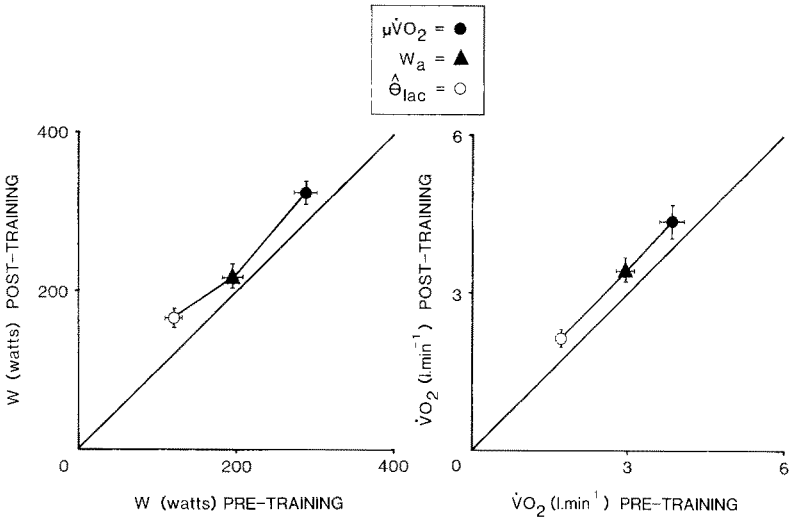

Fig. 1. Effect of training on power output (W) (*left panel*) and O_2 uptake ($\dot{V}_{O_2}$) (*right panel*) associated with the maximal $\dot{V}_{O_2}$ ($\mu\dot{V}_{O_2}$) and the estimated lactate threshold ($\hat{\Theta}_{lac}$) obtained from incremental testing, and the power asymptote of the power-duration relationship (W_a) obtained from constant-load testing ($n = 8$)

Table 1. Effect of training on estimated lactate threshold ($\hat{\Theta}_{lac}$), parameters of power-duration relationship (W_a, W'), and maximal O_2 uptake ($\mu\dot{V}_{O_2}$)

Pre-training									
	$\hat{\Theta}_{lac}$			W_a			$\mu\dot{W}$		W'
Subj	W	$\dot{V}_{O_2}$ l min^{-1}	$\%\mu\dot{V}_{O_2}$	W	$\dot{V}_{O_2}$ l min^{-1}	$\%\mu\dot{V}_{O_2}$	W	$\dot{V}_{O_2}$ l min^{-1}	kJ
1	137.0	2.04	43.8	222.0	3.52	75.5	350	4.66	12.5
2	107.0	1.59	41.1	178.5	3.02	78.0	275	3.87	24.0
3	87.0	1.42	40.2	156.5	2.84	80.5	250	3.53	11.5
4	125.0	1.79	48.1	205.0	2.72	73.1	300	3.72	17.8
5	126.0	1.75	52.9	179.0	2.64	79.8	250	3.31	11.9
6	95.0	1.52	49.5	170.5	2.70	88.0	250	3.07	14.2
7	150.0	1.72	47.8	205.0	3.30	91.7	300	3.60	13.2
8	125.0	1.93	41.1	259.0	3.07	65.6	325	4.68	11.5
Mean	120	1.72	45.6	197.3	2.97	79.0	287.5	3.81	14.6
SE	8	0.08	1.8	12.5	0.11	3.1	14.0	0.22	1.6
Post-training									
	$\hat{\Theta}_{lac}$			W_a			$\mu\dot{W}$		W'
Subj	W	$\dot{V}_{O_2}$ l min^{-1}	$\%\mu\dot{V}_{O_2}$	W	$\dot{V}_{O_2}$ l min^{-1}	$\%\mu\dot{V}_{O_2}$	W	$\dot{V}_{O_2}$ l min^{-1}	kJ
1	212.5	2.75	43.4	249.0	3.81	60.2	375	6.33	16.5
2	157.0	1.96	44.1	200.0	3.30	74.3	350	4.44	22.7
3	137.5	1.76	46.7	182.0	3.16	84.0	300	3.76	13.1
4	137.5	1.78	45.4	223.0	3.47	88.5	325	3.92	17.8
5	150.0	2.04	50.6	204.0	2.88	72.0	300	4.00	12.4
6	170.0	2.19	64.8	191.5	2.92	86.4	275	3.38	11.5
7	175.0	2.38	52.2	221.0	3.43	75.2	325	4.56	14.0
8	187.5	2.58	54.3	270.0	4.38	92.4	350	4.74	10.5
Mean	166.0	2.14	50.2	217.0	3.42	79.1	325	4.39	14.8
SE	10.0	0.12	2.7	11.0	0.18	4.0	12	0.34	1.5

$\hat{\Theta}_{lac}$ and $\mu\dot{V}_{O_2}$ determined from incremental testing; W_a and W' determined from constant-load testing. $\dot{V}_{O_2}$ at W_a is value at 24th min

of the intercept parameter (W_a) is inevitable. Hence, it is conceivable that the W_a tests may have included a small proportion in which the power actually exceeded the true W_a; however, the 95% confidence intervals associated with W_a were small (21.8 W pre-training, 16.0 W post-training). Therefore, the slight, continued rise in $\dot{V}_{O_2}$ at W_a post-training is likely the consequence of (i) a small overestimation of W_a in occasional instances and (ii) the averaging of $\dot{V}_{O_2}$ responses which approached steady-state levels with different kinetics in different subjects.

In contrast, $> W_a$ there was no evidence of $\dot{V}_{O_2}$ stabilizing in any of the individual subjects or for the group as a whole. Rather, $\dot{V}_{O_2}$ rose slowly and systematically throughout the test, achieving a value of 3.70 ± 0.47 l·min^{-1} ($97.1 \pm 5.9\%$ of $\mu\dot{V}_{O_2}$) pre-training and 4.15 ± 0.25 l·min^{-1} ($94.5 \pm 5.0\%$ of $\mu\dot{V}_{O_2}$) post-training (Tables 1 and 2). In three instances pre-training and two post-training, the end-exercise $\dot{V}_{O_2}$ exceeded $\mu\dot{V}_{O_2}$

Table 2. Maximal ventilatory and gas exchange responses for incremental exercise pre- and post-training

	Pre-training (±SE)	Post-training (±SE)	p
$\dot{V}_E$ (l min^{-1})	119.5 ± 7.7	136.5 ±12.4	0.05
$\dot{V}_{O_2}$ (l min^{-1})	3.81± 0.22	4.39± 0.34	0.01
(ml kg^{-1}min^{-1})	50.6 ± 1.8	58.8 ± 2.7	0.01
$\dot{V}_{CO_2}$ (l min^{-1})	4.97± 0.27	5.59± 0.52	0.05
R	1.31± 0.03	1.27± 0.03	N.S.
HR (bt min^{-1})	181.4 ± 2.8	184 ± 2.1	N.S.
W (watts)	287.5 ±14	325 ±12	0.005

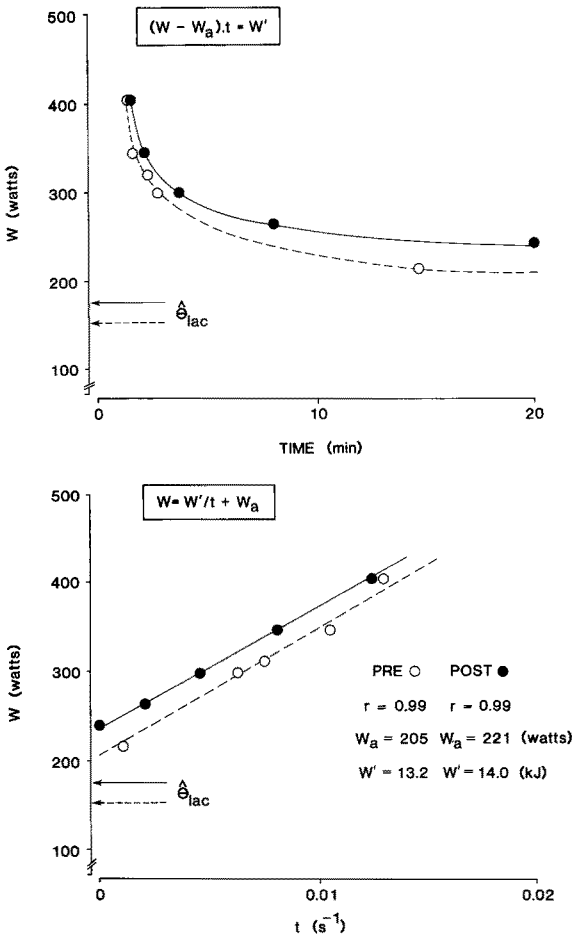

Fig. 2. Effect of training on power (W) — duration (t) relationship (*upper panel*) and its linear transformation $W-t^{-1}$ (*lower panel*) for high-intensity exercise, pre-training (*open symbols*) and post-training (*solid symbols*); subj. 7. W' and W_a represent the curvature factor and the power asymptote, respectively, of the $W-t$ relationship; $\hat{\Theta}_{lac}$ is the estimated lactate threshold

slightly; in the remaining subjects, it was either equal to or slightly less than $\mu\dot{V}_{O_2}$.

The $\dot{V}_{O_2}$ attained at the end of both the W_a and $> W_a$ tests was compared in each subject with the corresponding $\dot{V}_{O_2}$ that was predicted from the linear extrapolation of the sub-$\hat{\Theta}_{lac}$ $\dot{V}_{O_2}-W$ relationship: i.e., the predicted $\dot{V}_{O_2}=\dot{V}_{O_2}(u)+W\cdot(d\dot{V}_{O_2}\cdot dW^{-1})$, where $\dot{V}_{O_2}(u)$ is the $\dot{V}_{O_2}$ for unloaded pedaling, and $d\dot{V}_{O_2}\cdot dW^{-1}$ is the slope of the sub-$\hat{\Theta}_{lac}$ $\dot{V}_{O_2}-W$ relationship for incremental exercise. The measured $\dot{V}_{O_2}$ was found to exceed the predicted value by an average of 0.45 ± 0.10 $l\cdot min^{-1}$ for the W_a tests and by 0.67 ± 0.24 $l\cdot min^{-1}$ for the $> W_a$ tests pre-training, and by 0.40 ± 0.10 $l\cdot min^{-1}$ at W_a and 0.75 ± 0.12 $l\cdot min^{-1}$ $> W_a$ post-training.

Arterialized venous blood [lac] increased for the W_a and $> W_a$ tests both pre- and post-training (Fig. 3). In the W_a tests, [lac] plateaued towards the end of the exercise, with the final value being slightly lower after training (i.e., 5.6 $mmol\cdot l^{-1}$ pre-training vs. 4.2 $mmol\cdot l^{-1}$ post-training, on average). In contrast, for the $> W_a$ test, [lac] increased progressively until the point of fatigue; again, the final [lac] was lower after training (11.3 $mmol\cdot l^{-1}$ pre-training vs. 6.7 $mmol\cdot l^{-1}$ post-training, on average).

For both the W_a and $> W_a$ tests, the [HCO_3^-] response essentially mirrored that of [lac] (Fig. 3): at W_a, a new stable level was approached within ~20 min pre-training and ~16 min post-training; for the $> W_a$ tests, [HCO_3^-] fell systematically until fatigue. The pH fell sharply during the first 4 min for the W_a tests (both pre- and post-training) to stabilize soon thereafter at 7.32–7.35 (pre-training, there was evidence of a small, though nonsignificant, rise back towards resting levels at the end of the work) (Fig. 3). In contrast, for the $> W_a$ tests the pH continued to fall throughout the work, attaining average values at fatigue of 7.25 ± 0.04 pre-training and 7.27 ± 0.02 post-training (Fig. 3). Both prior to and following the training, arterialized venous PCO_2 fell throughout the work for both the W_a and the $> W_a$ tests; this effect was more marked at the higher power output (Fig. 3).

For both the W_a and $> W_a$ tests, there was an early marked increase in $\dot{V}_E$ which was followed by a more gradual increase (Fig. 4); this reflected an initial rapid increase in V_T (plateauing at an average value of some 2.5–2.7 l) and a more slowly-developing increase in frequency. The post-training $\dot{V}_E$ responses at W_a were somewhat higher than pre-training, consistent with the greater post-training power output; this difference was not evident $> W_a$, however.

Rectal temperature (T_{rec}) increased steadily throughout the work by $0.81\pm0.16°$ C at the end of the W_a test and by $0.77\pm0.3°$ C at the end of the $> W_a$ test prior to the training; and by $1.34\pm0.2°$ C at W_a and $0.99\pm0.10°$ C $> W_a$ post-training (Fig. 4). These end-exercise differences in T_{rec} were not significant either pre- or post-training; however, the end-exercise T_{rec} values for both the W_a tests and the $> W_a$ tests post-training were greater than the corresponding values pre-training. Blood [NE] and [E] increased steadily from exercise onset until the end of exercise, with the final values being higher $> W_a$ than at W_a; however, these values were not different pre- and post-training (Fig. 4).

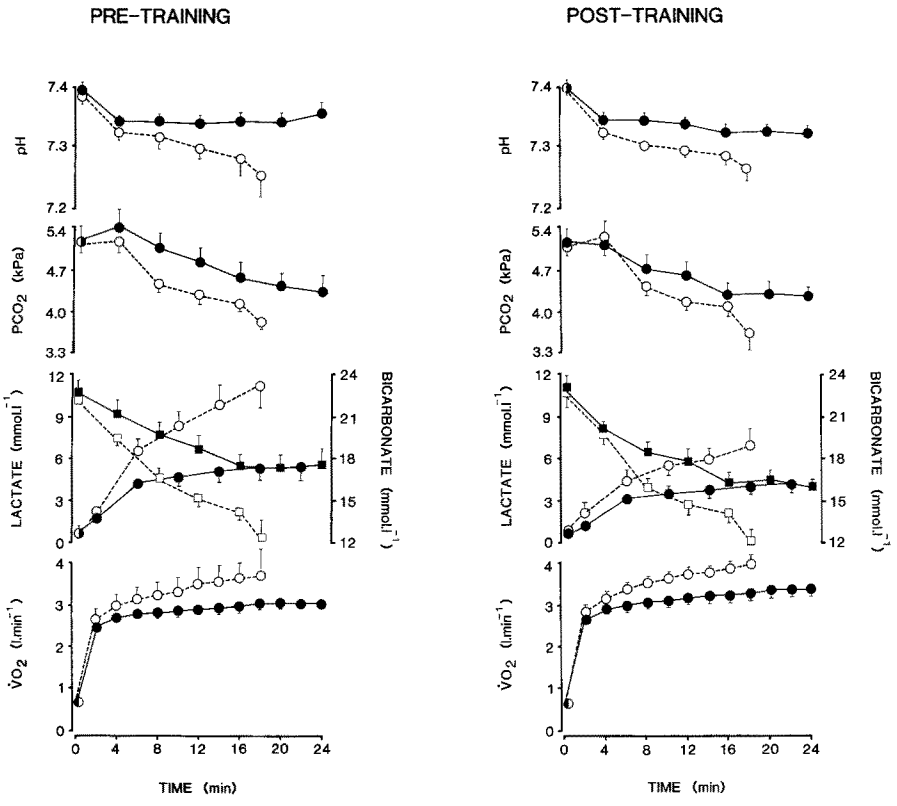

Fig. 3. Group mean responses (±SE) for arterialized-venous blood pH, PCO_2, [lactate], and bicarbonate [HCO_3^-] and for O_2 uptake ($\dot{V}_{O_2}$) during constant-load exercise at W_a (*solid symbols*) and $> W_a$ (*open symbols*). Pre-training: left panel; post-training: right panel

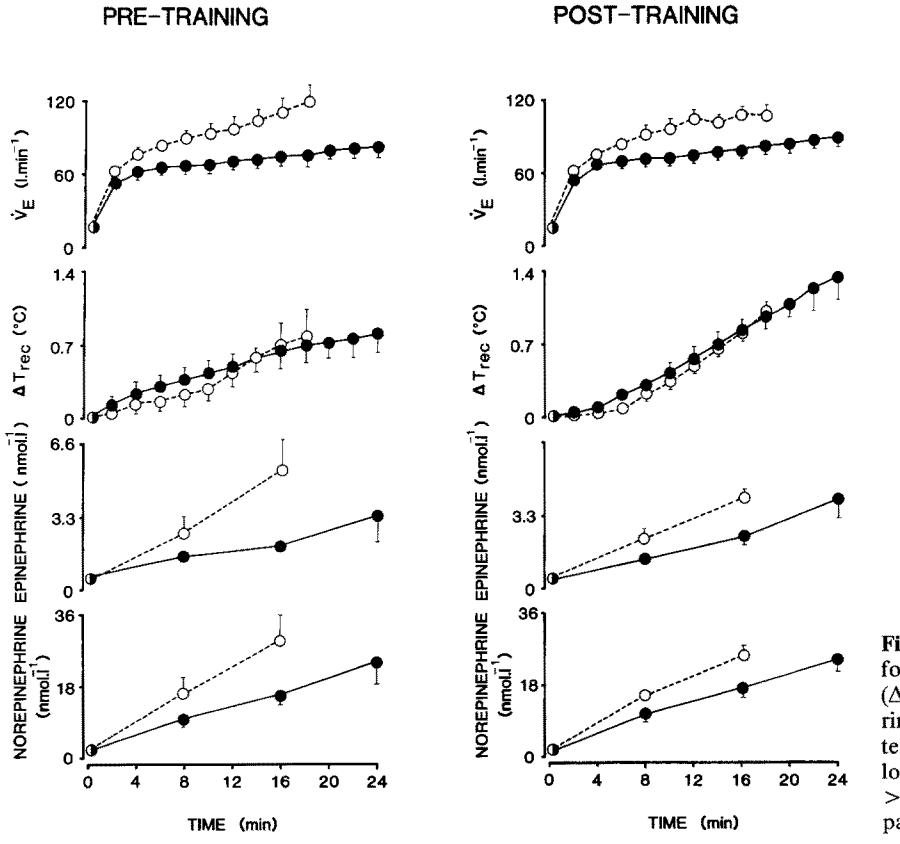

Fig. 4. Group mean responses (±SE) for ventilation ($\dot{V}_E$), rectal temperature (ΔT_{rec}), and concentrations of epinephrine [E] and norepinephrine [NE] in arterialized-venous blood during constant-load exercise at W_a (*solid symbols*) and $> W_a$ (*open symbols*). Pre-training: left panel; post-training: right panel

Discussion

The principal original findings of the present investigation are that: 1) a training regimen designed to elevate both $\mu\dot{V}_{O_2}$ and Θ_{lac} preserves the hyperbolic character of the $W-t$ relationship for high-intensity exercise, evincing increases in the power asymptote but with no significant effect on its curvature (Fig. 2; Table 1) — i.e., no change in the *amount* of work which could be performed *above* that critical power output; and 2) regardless of the training status of our subjects and thus the absolute $\dot{V}_{O_2}$ at which it occurs, W_a represents the upper limit for stability of $\dot{V}_{O_2}$, blood [lac] and pH: i.e., constant-load exercise above W_a elicited continuously rising profiles of $\dot{V}_{O_2}$ and [lac] and a falling profile of pH with (in 15 out of 16 instances) fatigue occurring prior to the target time of 24 min (Figs. 3 and 4). These observations further strengthen our contention that W_a is an important demarcator of work intensity domains, with significant implications for profiles of metabolic and acid-base response to exercise.

We have hypothesized that W_a is determined by the capacity to transport and utilize O_2, while W' represents components of an energy pool incorporating stored O_2 and phosphagens and anaerobic glycolysis. Although we cannot tell from these experiments whether the components of W' change with training, it is clear that their net contribution to energy provision remains unaltered.

The precise mechanisms which underlie the fatigue associated with high-intensity exercise remain the subject of considerable debate (reviewed by Porter an Whelan 1981). Such work rates are characterized by elevated levels of blood lactate, in contrast to lower work rates for which there is no sustained increase in [lac] and which therefore may be termed "moderate" (Wells et al. 1957; Wasserman et al. 1967; Whipp and Mahler 1980). Based upon the temporal profile of blood [lac] increase at these higher work rates, two further intensity domains have been identified: those for which [lac] is increased and (given sufficient time) may eventually stabilize may be termed "heavy", and a higher range within which [lac] continues to increase throughout the work to the point of fatigue is "severe" (Whipp and Mahler 1980).

On the basis of the hyperbolic $W-t$ relationship for high-intensity exercise that we and others have demonstrated in normal sedentary subjects (Monod and Scherrer 1965; Moritani et al. 1981; Whipp et al. 1982; Poole et al. 1988), we have proposed previously that the power asymptote W_a (and its $\dot{V}_{O_2}$ equivalent, $W_a\{O_2\}$) provides an important functional demarcation between the heavy and severe domains of exercise (Whipp and Mahler 1980; Whipp et al. 1982; Poole et al. 1988). Thus, the heavy-intensity domain extends from Θ_{lac} to W_a, providing a range of work rates for which $\dot{V}_{O_2}$, blood [lac] and blood pH are each able to stabilize at new steady-state values; hence, this allows a particular power output to be sustained for a prolonged period. Within the severe-intensity domain, which lies above W_a, not only does blood [lac] rise throughout the exercise but blood pH also falls; fatigue thus ensues more rapidly and as a hyperbolic function of W, with $\dot{V}_{O_2}$ eventually reaching $\mu\dot{V}_{O_2}$ (as determined from incremental testing). In this domain, therefore, $\dot{V}_{O_2}$ is a function of both time *and* power: W_a can therefore be taken to represent a discontinuity in the *achievable* $\dot{V}_{O_2}-W$ relationship (Poole et al. 1988).

Important initial studies of the power-duration relationship by Wilkie (1981) attempted to provide physiological correlates for the parameters i.e.

$$W=E+(A\cdot t^{-1})-E\tau(1-e^{-t/\tau})t^{-1} \qquad \text{(c)}$$

where E is the power equivalent of $\mu\dot{V}_{O_2}$, A is the anaerobic energy store and τ is the $\dot{V}_{O_2}$ time constant. While this formulation provided a reasonable fit to the data, we believe the underlying assumptions are flawed for the following reasons (see also Poole et al. 1988). The present investigation has demonstrated that all work rates $> W_a$ yield $\mu\dot{V}_{O_2}$ if continued to fatigue. Thus, in this exercise intensity domain, $\dot{V}_{O_2}$ is a function of time as well as power and cannot be characterized by a discrete value for power. For high-intensity exercise the kinetics of $\dot{V}_{O_2}$ are complex and have yet to be characterized. A single value of 10 s for τ given by Wilkie is not appropriate for man (Linnarsson 1974; Whipp and Mahler 1980). We contend that the data of Wilkie are fit by this equation only because the third term in equation c becomes disappearingly small as t becomes long compared with this assumed time constant, and the equation therefore reduces to $W=E+A\cdot t^{-1}$. This is similar in form to equation b; the physiological equivalents of the parameters are significantly different, however. It is our contention that W_a occurs at a submaximal $\dot{V}_{O_2}$ (Figs. 1 and 3; Table 1). Indeed, we are not aware of any published data which supports the notion that man can perform prolonged exercise at $\mu\dot{V}_{O_2}$, as suggested by equation c.

Hoppeler and colleagues (1985) have documented structural adaptations (mitochondrial vol-

ume, capillary number·fiber mm^{-2}) to training in human muscle. These authors concluded that the increased mitochondrial volume and capillarity in the trained muscles could account for the increased $\mu\dot{V}_{O_2}$. However, the argument was based in part on the *estimated* $\dot{V}_{O_2}$ response to high-intensity exercise. We and others have demonstrated clearly that the O_2 requirement of such exercise cannot be predicted reliably because of the slowly-developing phase of $\dot{V}_{O_2}$ (Whipp and Mahler 1980; Casaburi et al. 1987; Poole et al. 1988) (Fig. 3). In the present investigation we have determined rigorously the $\dot{V}_{O_2}$ response to the highest work rate which can be supported solely by oxidative processes (i.e. W_a) and have related these measurements to $\mu\dot{V}_{O_2}$ before and after training. The finding that the increase of $W_a\{O_2\}$ was of the same magnitude as that of $\mu\dot{V}_{O_2}$ (i.e. 0.45 and 0.58 $l\cdot min^{-1}$, respectively; Fig. 1, Tables 1 and 2) provides support for the hypothesis of Hoppeler that the training induced changes manifest at submaximal exercise are in proportion to the increased $\mu\dot{V}_{O_2}$.

The requirements for ATP production during high-intensity exercise are argued to depend on several energetic pathways, although these have not yet been considered formally within the context of the $W-t$ relationship. diPrampero (1981), for example, has described the rate of intramuscular ATP resynthesis during exercise in terms of the rates of phosphocreatine (PC) breakdown, lactate accumulation and muscle O_2 consumption:

$$\overrightarrow{[ATP]} = \overrightarrow{[PC]} + b\cdot[lac] + c\cdot\dot{V}_{O_2} \quad \text{(d)}$$

where b represents the ATP resynthesized per unit of lactate formed anaerobically, and c represents the ATP resynthesized per unit of O_2 consumed. Following a short initial phase at exercise onset in which the depletion of PC stores contributes to the energy requirement, this expression reduces to: $\overrightarrow{[ATP]} = b\cdot[lac] + c\cdot\mu\dot{V}_{O_2}$, thus predicting that blood [lac] continues to increase during the exercise only for power outputs that would elicit $\dot{V}_{O_2}$'s in excess of $\mu\dot{V}_{O_2}$.

Our results do not support this formulation as it implies that all power outputs sufficient to elicit $\mu\dot{V}_{O_2}$ or less can be sustained for prolonged periods. This is clearly not the case. We have identified a critical *submaximal* power domain (i.e., between W_a and $\mu\dot{V}_{O_2}$) in which blood [lac] and $\dot{V}_{O_2}$ continue to rise, pH continues to fall, until fatigue is manifest (Fig. 3). Thus, work rates above W_a exhibit a sustained anaerobic contribution and, therefore, the term "$c\cdot\mu\dot{V}_{O_2}$" in the diPrampero (1981) formulation should actually be "$W_a\{O_2\}$".

In such considerations, it is important to recognize that — as the pathways of lactate removal are chiefly aerobic — an elevated but stable blood [lac] is not necessarily indicative of a sustained anaerobic contribution to metabolism. Rather, it may merely reflect an initially high lactate production rate in one part of the body which is balanced by a slower aerobic removal of lactate at some other site. In contrast, a systematically rising blood [lac] — as we have observed at power outputs $> W_a$ — presumably reflects the development of a progressively greater O_2 deficit, regardless of the rate at which lactate subsequently may be removed.

Thus, exercise performed above W_a incurs a continuous depletion of energy stores (whose capacity may be represented by W') likely from within the working muscle. Fatigue ensues only when this store is depleted. And as we have predicted previously (Poole et al. 1988), should such a store become depleted monotonically with time (linearly or exponentially, for example) towards some low, limiting value during constant-load exercise, then the parameter characterizing this rate of decline (i.e., the slope or the rate constant, respectively) should be a function of W. The time required to attain this low, limiting value should consequently decrease hyperbolically as W increases.

As discussed previously, implicit in the work of Wilkie (1980) and diPrampero (1981) is the notion that man can perform prolonged exercise at $\mu\dot{V}_{O_2}$ (equations c and d). In contrast, this investigation has demonstrated that irrespective of fitness $W_a\{O_2\}$ is appreciably lower than $\mu\dot{V}_{O_2}$ (i.e. ~79% $\mu\dot{V}_{O_2}$, on average; Fig. 1, Table 1). The $W-t$ relationship has also been demonstrated to be hyperbolic for isometric exercise (i.e. tension vs. t, Monod and Scherrer 1965) and also treadmill running (Hughson et al. 1984). It is quite likely that the relationship between $W_a\{O_2\}$ and $\mu\dot{V}_{O_2}$ is different for other forms of exercise. Indeed, such data might reveal important information regarding what particular characteristics of the exercising musculature are important for determining $W_a\{O_2\}$.

A further conceptual issue which arises from the present investigation relates to the most appropriate means of expressing exercise intensity; this is most usually expressed relative to $\mu\dot{V}_{O_2}$. As our observations demonstrate, quite different profiles of pulmonary gas exchange and acid-base response and also differences in exercise tolerance

will result depending on the relationship of a particular power output to Θ_{lac} and W_a. Recognizing that W_a ranged from about 60%–90% of $\mu\dot{V}_{O_2}$ in the present investigation (Table 1), the ability to sustain a power output at 75% of $\mu\dot{V}_{O_2}$, for example, clearly should be very different for an individual whose W_a occurred at 60% of $\mu\dot{V}_{O_2}$ (e.g., subj. 1, post-training) than for one whose W_a was at 90% of $\mu\dot{V}_{O_2}$ (e.g., subj. 8, post-training). Hence, care must be taken when extrapolating the results of an incremental exercise test to sustained constant-load exercise, in that a $\mu\dot{V}_{O_2}$ could be manifest at power outputs *well below* those achieved on (or predicted from) the incremental test. Furthermore, the designation of a discrete work-rate equivalent for $\mu\dot{V}_{O_2}$ is nebulous because, for exercise performed above W_a, $\dot{V}_{O_2}$ becomes a function of time as well as power output. As a result, while differing testing protocols may produce the same $\mu\dot{V}_{O_2}$ for a given individual, this $\mu\dot{V}_{O_2}$ could occur at strikingly different power outputs (Whipp et al. 1981).

In conclusion, it is apparent that the pulmonary gas exchange and metabolic responses to, and endurance characteristics of, prolonged exercise depend crucially upon whether the exercise is performed below or above the power asymptote of the $W-t$ relationship. Furthermore, the observation that the time to fatigue increases proportionally more than $\mu\dot{V}_{O_2}$ in response to training (Holloszy and Coyle 1984) and therefore proportionally more than $W_a\{O_2\}$, is predictable from the hyperbolic power-duration relationship and our demonstration that W_a can be increased by training.

Acknowledgements. The authors wish to thank the Medical Graphics Corporation for the loan of equipment. D. C. Poole is the recipient of a National Institutes of Health N.R.S.A. award (HL 07694).

References

Boobis LH, Brooks S, Cheetham ME, Williams C (1987) Effect of sprint training on muscle metabolism during treadmill sprinting in man. J Physiol 384:31P

Casaburi R, Storer TW, Ben-Dov I, Wasserman K (1987) Effect of endurance training on possible determinants of $\dot{V}_{O_2}$ during heavy exercise. J Appl Physiol 62:199–207

diPrampero PE (1981) Energetics of muscular exercise. Rev Physiol Biochem Pharmacol 89:143–222

Forster HV, Dempsey JA, Thompson J, Vidruk E, DoPico GA (1972) Estimation of arterial PO_2, PCO_2, pH and lactate from arterialized venous blood. J Appl Physiol 32:134–137

Gollnick PD, Armstrong RB, Saltin B, Saubert CW, Sembrowich WL, Shepherd RW (1973) Effect of training on enzyme activity and fiber composition of human skeletal muscle. J Appl Physiol 34:107–111

Gollnick PD, Hermansen L (1973) Biochemical adaptations to exercise: anaerobic metabolism. Exerc Sport Sci Rev 1:1–43

Hill AV (1927) Muscular movement in man. McGraw-Hill, New York

Hohorst HL (1963) -(+)-lactate: determination with lactic dehydrogenase DPN. In: Bergmeyer HU (ed) Methods of enzymatic analysis. Academic Press, New York, pp 266–270

Holloszy JO, Coyle EF (1984) Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. J Appl Physiol 56:831–838

Hoppeler H, Howald H, Conley K, Lindstedt SL, Claassen H, Vock P, Weibel ER (1985) Endurance training in humans: aerobic capacity and structure of skeletal muscle. J Appl Physiol 59:320–327

Kelman GR, Nunn JF (1966) Nomograms for correction of blood PO_2, PCO_2, pH, and base excess for time and temperature. J Appl Physiol 21:1484–1490

Linnarsson D (1974) Dynamics of pulmonary gas exchange and heart rate changes at start and end of exercise. Acta Physiol Scand [Suppl] 415:1–68

Monod H, Scherrer J (1965) The work capacity of a synergic muscular group. Ergonomics 8:329–338

Moritani T, Nagata A, deVries HA, Muro M (1981) Critical power as a measure of physical work capacity and anaerobic threshold. Ergonomics 24:339–350

Morrison DF (1976) Multivariate statistical methods. McGraw-Hill, New York, pp 33–34

Poole DC, Gaesser GA (1985) Response of ventilatory and lactate thresholds to continuous and interval training. J Appl Physiol 58:1115–1121

Poole DC, Ward SA, Gardner G, Whipp BJ (1988) A metabolic and respiratory profile of the upper limit for prolonged exercise in man. Ergonomics 31:1265–1279

Porter R, Whelan J (1981) Human muscle fatigue: physiological mechanisms. Pitman, Bath

Wasserman K, Van Kessel AL, Burton GG (1967) Interaction of physiological mechanisms during exercise. J Appl Physiol 22:71–85

Watson E (1981) Liquid Chromatography with electrochemical detection for plasma norepinephrine and epinephrine. Life Sci 28:493–497

Wells JG, Balke B, Van Fosson BD (1957) Lactic acid accumulation during work. A suggested standardization of work classification. J Appl Physiol 10:51–55

Whipp BJ, Mahler M (1980) Dynamics of pulmonary gas exchange during exercise. In: West JB (ed) Pulmonary gas exchange, vol II. New York, Academic Press, pp 33–96

Whipp BJ, Davis JA, Torres F, Wasserman K (1981) A test to determine parameters of aerobic function during exercise. J Appl Physiol 50:217–221

Whipp BJ, Huntsman DJ, Storer T, Lamarra N, Wasserman K (1982) A constant which determines the duration of tolerance to high-intensity work. Fed Proc 41:1591

Wilkie DR (1981) Shortage of chemical fuel as a cause of fatigue: studies by nuclear magnetic resonance and bicycle ergometry. In: Porter R, Whelan J (eds) Human muscle fatigue: physiological mechanisms. Pitman, Bath, pp 102–114

Accepted June 22, 1989