Effect of Exercise Intensity on Relationship between $\dot{V}O_{2max}$ and Cardiac Output

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ABSTRACT

LEPRETRE, P.-M., J.-P. KORALSZTEIN, and V. L. BILLAT. Effect of Exercise Intensity on Relationship between $\dot{V}O_{2max}$ and Cardiac Output. *Med. Sci. Sports Exerc.*, Vol. 36, No. 8, pp. 1357–1363, 2004. **Purpose:** The purpose of this study was to determine whether the maximal oxygen uptake ($\dot{V}O_{2max}$) is attained with the same central and peripheral factors according to the exercise intensity. **Methods:** Nine well-trained males performed an incremental exercise test on a cycle ergometer to determine the maximal power associated with $\dot{V}O_{2max}$ ($p\dot{V}O_{2max}$) and maximal cardiac output ($\dot{Q}_{max}$). Two days later, they performed two continuous cycling exercises at 100% (tlim100 = 5 min 12 s ± 2 min 25 s) and at an intermediate work rate between the lactate threshold and $p\dot{V}O_{2max}$ (tlimΔ50 = 12 min 6 s ± 3 min 5 s). Heart rate and stroke volume (SV) were measured (by impedance) continuously during all tests. Cardiac output ($\dot{Q}$) and arterial-venous O_2 difference (a-vO_2 diff) were calculated using standard equations. **Results:** Repeated measures ANOVA indicated that: 1) maximal heart rate, $\dot{V}E$, blood lactate, and $\dot{V}O_2$ ($\dot{V}O_{2max}$) were not different between the three exercises but $\dot{Q}$ was lower in tlimΔ50 than in the incremental test (24.4 ± 3.6 L·min^{-1} vs 28.4 ± 4.1 L·min^{-1}; $P < 0.05$) due to a lower SV (143 ± 27 mL·beat^{-1} vs 179 ± 34 mL·beat^{-1}; $P < 0.05$), and 2) maximal values of a-vO_2 diff were not significantly different between all the exercise protocols but reduced later in tlimΔ50 compared with tlim100 (6 min 58 s ± 4 min 29 s vs 3 min 6 s± 1 min 3 s, $P = 0.05$). This reduction in a-vO_2 diff was correlated with the arterial oxygen desaturation (SaO_2 = −15.3 ± 3.9%) in tlimΔ50 ($r = -0.74$, $P = 0.05$). **Conclusion:** $\dot{V}O_{2max}$ was not attained with the same central and peripheral factors in exhaustive exercises, and tlimΔ50 did not elicit the maximal $\dot{Q}$. This might be taken into account if the training aim is to enhance the central factors of $\dot{V}O_{2max}$ using exercise intensities eliciting $\dot{V}O_{2max}$ but not necessarily $\dot{Q}_{max}$. **Key Words:** STROKE VOLUME, ARTERIAL-VENOUS DIFFERENCE, CYCLING, HYPOXEMIA

Exercise workloads between 85 and 100% of the power associated with $\dot{V}O_{2max}$ in an incremental test ($p\dot{V}O_{2max}$) will elicit $\dot{V}O_{2max}$, provided they are performed for a sufficient time duration (5–15min according to the exercise intensity) (6). However, the respective contribution of central (the cardiac output: $\dot{Q}$) and peripheral factors (the arterial-venous oxygen difference: a-vO_2 diff) in the attainment of $\dot{V}O_{2max}$ according to the workload is not clear. McCole et al. (21) have reported that an incremental test of a total duration of 12 min (with four stages of 3-min duration) elicited $\dot{V}O_{2max}$ with a lower cardiac output (due to lower value of stroke volume) compared with an incremental protocol lasting 6 min only (including six stages of 1 min). The authors hypothesized that, in contrast with the longer duration test, the temperature necessary to elicit cutaneous vasodilatation was not reached during the shorter test, preventing the increased competition for the distribution of $\dot{Q}$ (21).

However, the 1-min stage duration protocol allowed reaching almost a twice higher treadmill slope than the 3-min stage duration (14% vs 8%). This could induce a larger muscular mass recruitment during shorter than longer progressive exercises, and it is well known than the stroke volume is influenced by the muscle mass recruited which itself influences the muscle pump and hence the venous return (20). The large difference in power output (as 2% represents 1 km·h^{-1} of speed increment, i.e., 1 MET: 3.5 mL·kg^{-1}·min^{-1}) and the absence of $\dot{V}O_2$ steady states makes it difficult to make conclusions about the disassociation between maximal $\dot{V}O_2$ and $\dot{Q}$ during exercise. Furthermore, it has been reported that the time course of the stroke volume was affected by the exercise mode (22) and that cardiac output declines more during cycling versus running exercise. This is due to the development of high intramuscular forces in cycling that would compress or even partially occlude venous blood return from the leg (22). A notable difference was also reported with the time course of oxygen uptake between running and cycling at the same relative supra-threshold intensity in well-trained triathletes (5).

Indeed, few studies have reported the time course of these central and peripheral factors during supra-lactate threshold exercises and have examined subthreshold exercises. Cheatham et al. (11) have shown that the increase in a-vO_2 diff was associated with that of $\dot{V}O_2$ during 40 min of an exercise performed at 64.5% of $\dot{V}O_{2max}$. They interpreted

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this as a manifestation of the Böhr effect rather than a further decrease in end capillary oxygen pressure and the exercise lactic acidosis might cause different responses in tissue oxygen extraction (11,17).

However, if the Böhr effect contributes to enhancing a-vO_2 diff by decreasing venous O_2 concentration (CvO_2) in supra-threshold exercises, these intensive exercises may induce hypoxemia, especially in highly fit endurance subjects. Indeed, Billat et al. (8) have reported a hypoxemia in exhaustive constant speed runs performed at 90, 100, and 105% of the $p\dot{V}O_{2max}$, and the time to exhaustion at 90% of $\dot{V}O_{2max}$ was inversely correlated with the degree of hypoxemia: a longer exercise duration was associated with a more pronounced hypoxemia, that is, a larger decrease in arterial oxygen de-saturation (SaO_2). Therefore, the drop in CvO_2 could be balanced out by the decrease in CaO_2 (15). Given that the oxygen pulse (SV $\times$ a-vO_2 diff) has been reported to be constant in exhaustive running (10 min) at 95% of $p\dot{V}O_{2max}$ (18), this raises the question of the balance between the peripheral and the central factors of $\dot{V}O_2$ during exhaustive supra-threshold exercises eliciting $\dot{V}O_{2max}$. Because very heavy exercise is currently and mostly used during interval training with the goal of improving $\dot{V}O_{2max}$ (14), it is of interest to check whether exercise performed at the low and high range of this heavy-intensity domain elicits the same value of central and peripheral factors of $\dot{V}O_{2max}$. However, behind this (similar) maximal $\dot{V}O_2$ elicited, we hypothesized that the maximal value of $\dot{Q}$ is not systematically reached and sustained.

Therefore, the purpose of this study was to determine in endurance trained subjects and in cycling exercise whether the maximal oxygen uptake ($\dot{V}O_{2max}$) is attained with the same central and peripheral factors in exhaustive constant load exercises performed at 100% of the power associated with $\dot{V}O_{2max}$ in an incremental test ($p\dot{V}O_{2max}$) and at pΔ50, which was defined as the intensity midway between the power associated with the lactate threshold (pLT) and $p\dot{V}O_{2max}$.

METHODS

Nine healthy male triathletes participated in this study after giving their written voluntary informed consent in accordance with the guidelines of the University of Evry–Val d'Essonne. All subjects were free of cardiac and pulmonary disease. Their physical characteristics are reported in Table 1.

Protocol. Three tests (one incremental and two constant work exercises) were performed at 1-wk intervals, at the same time of the day and 2–3 h after a light breakfast in an air-conditioned room (Fig. 1). All tests were performed with the subjects in the upright position on an electronically braked ergometer (ERGOLINE 900, Hellige, Markett). Seat and handlebar heights were set for each subject and kept constant for all the tests. The pedaling frequency selected by each subject between 70 and 110 rev·min^{-1}.

Incremental exercise. After a 3-min warm-up at 80 W, each subject performed a 3-min stage incremental exercise test to exhaustion with 40 W work increment for total exercise duration not exceeding 20 min. In this incremental protocol, $\dot{V}O_{2max}$ was defined as the highest 30-s oxygen uptake value reached during exercise with a respiratory exchange ratio greater than 1.1, blood lactate greater than 8 mM, and a peak heart rate at least equal to 90% of the age-predicted maximum. A plateau of $\dot{V}O_2$ was identified if the $\dot{V}O_2$ of the least stage was not greater than the previous one by 1.75 mL·kg^{-1}·min^{-1}. The $p\dot{V}O_{2max}$ was determined as the lowest power eliciting $\dot{V}O_{2max}$ (7). If during the last stage, a subject achieved $\dot{V}O_{2max}$ without completing the 3-min stage, $p\dot{V}O_{2max}$ was calculated as following:

$$p\dot{V}O_{2max} = pF + [(t/180) \times 40] \quad [1]$$

where pF was the power of the last complete stage (W), t was the time the last workload was maintained (s), and 40 W was the power output increment between the last of two stages.

Constant-load exercises. In randomized order, each subject completed two severe constant workload exercises until exhaustion separated by at least 48 h. Time limit (tlim) was the time at which the subject was no longer able to pedal at the given power output. After a 15-min warm-up at 30% of $p\dot{V}O_{2max}$ (which was below the lactate threshold for all subjects) and 5 min at rest, the work rate was increased within 30 s to pΔ50 or $p\dot{V}O_{2max}$. The power pΔ50 was defined as the intensity midway between the power associated with the lactate threshold (pLT) and the work requiring 100% of $p\dot{V}O_{2max}$.

Measurements of gas exchange. The minute ventilation ($\dot{V}E$) and gas exchange parameters ($\dot{V}O_2$, $\dot{V}CO_2$) were measured breath-by-breath by an open-circuit metabolic cart with rapid O_2 and CO_2 analyzers (CPX, MedGraphics Cardiorespiratory Systems, Medical Graphic Corporation, Wisconsin). Before each individual exercise test, the pneumotachograph was calibrated using a 3-L calibration syringe (Hans Rudolph), and the gas analyzers were calibrated using reference gases with known O_2 (16.0%) and CO2 (5.0%) concentrations. During all tests, the breath-by-breath data were smoothed and averaged every 5 s.

Measurement of blood lactates. During both exercise tests, 10-μL ear blood samples were collected and immediately analyzed for blood lactate concentration (YSI 1500, Sport L-lactate Analyzer, Bioanalytical Products, Yellow Springs, OH). The samples were taken at rest, every 3 min during the all-out exercise, at the end of cycling, and at 2 and 4 min during recovery after the exercise test. For the incremental test, the lactate threshold (LT) was defined as

TABLE 1. Physical characteristics of the subjects.

Subjects (N = 9)	Age (yr)	Mass (kg)	Height (cm)	Body Fat (%)	Training Time (h·wk^{-1})
Mean ± SD	33 ± 7	71 ± 4	176 ± 4	11.4 ± 2.4	13 ± 4

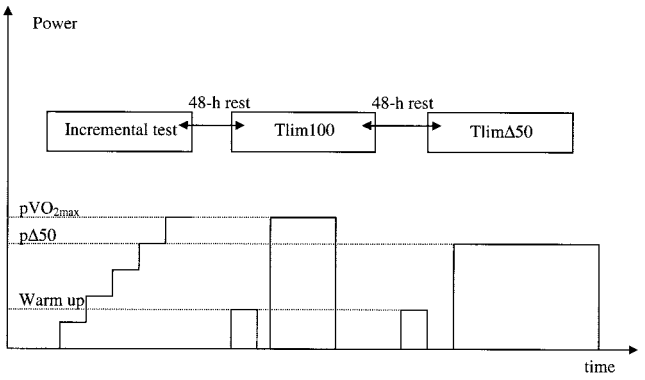

FIGURE 1—Experimental design.

the $\dot{V}O_2$ corresponding to the starting point of an accelerated lactate accumulation of ~ 4 mM and expressed in percent of $\dot{V}O_{2max}$ (2).

Arterial oxygen saturation measurements. SaO_2 was recorded pulse-to-pulse using a noninvasive external device (Ohmeda 3800 oximeter, Datex-Ohmeda, Louisville, KY) and then averaged every 5 s. The ear pulse oxymeter was calibrated using an internal protocol before each test, according to the manufacturer's instructions. The pulse oximeter used an exclusive algorithm (Trutrak$^{+®}$, Oxymetry technology, Datex-Ohmeda), which identified, quantified, and corrected the clinical movement of the subject. Pulse oximetry has been reported to be accurate across a broad range of SaO2 (from 57.2 to 97.6%) and provides an arterial O_2 saturation estimate with mean error of 2% (3). According to Dempsey and Wagner (15), arterial hypoxemia induced by exercise was defined as an arterial O_2 saturation $< 88\%$ (or SaO_2rest − end of exercise greater than 10%).

Measurements of cardiac parameters (heart rate, stroke volume) and calculation of $\dot{Q}$, a-vO_2 diff. We used a bioimpedance determination for stroke volume and heart rate (Physioflow, Manatec Type PF05L1, France). The theoretical basis for this technique and its application and validity for exercise testing have been previously described (4,10). The physioflow device measures impedance changes (dZ) in responses to a small-administered electrical current. Two sets of electrodes (Ag/AgCl, Hewlett Packard 40493 E), one electrode transmitting and the other sensing, respectively, were applied above the supra-clavicular fossa at the left base of the neck and along the xiphoid. A further set of two electrodes was used to monitor a single ECG (CM5 position). With this impedance device, a first evaluation of stroke volume index (SVi) is calculated during a calibration procedure based on 24 consecutive heartbeats recorded in the resting condition (SVi_{cal}). This evaluation keeps the largest impedance variation during the systole ($Z_{max} - Z_{min}$) and the largest rate of variation of the impedance signal (dZ/dt_{max}, called the contractility index). The SVi calculation also depends on the thoracic flow inversion time (TFIT, in $m \cdot s^{-1}$) measured on the first mathematical derivative of the impedance signal. TFIT is the time interval between the first zero value after the beginning of the cardiac cycle (beginning of the ECG's QRS complex) and the first nadir after the peak of the ejection velocity (dZ/dt_{max}). Afterward, TFIT is weighted using a specific algorithm. During the data acquisition phase, the variation of parameters were analyzed and compared with those obtained during the calibration procedure. So, the stroke volume (SV) was the product between the electrical physical volume of thorax (VEPT), the ventricular ejection time (VET), and the maximum rate of the impedance change during the systolic upstroke [(dZ/t)max] divided by the basal thoracic impedance (Z0). Each displayed stroke volume (SV) value represents the mean of 15 successive artifact-free beats. For this experiment, SV and heart rate were measured continuously during each test with beat-to-beat data smoothed by a 5-s moving averaging algorithm. $\dot{Q}$ calculation by the device is based on the following formula:

$$\dot{Q} = HR \times SVi \times BSA \quad [2]$$

where $\dot{Q}$ is expressed in liters per minute, HR is the heart rate based on the R-R interval measurement, determined on the ECG first derivative, dECG/dt, which provides a more stable signal than the ECG signal itself, SVi ($mL \cdot m^{-2}$), and BSA was the body surface area calculated according to the formula of Haycock ($BSA = 0.024265 \times BM^{0.5378} \times H^{0.3964}$, where BM is body mass in kilograms and H is height in centimeters). In a previous study, Richard et al. (24) showed that the mean difference between the $\dot{Q}$ obtained using direct Fick and the impedance method was not significant at rest ($0.07\ L \cdot min^{-1}$, i.e., 1%) and during steady state exercise ($0.26\ L \cdot min^{-1}$, i.e., 2%) (10). In addition, they also reported that the direct Fick method was highly correlated with the impedance method during steady state exercise ($r^2 = 0.79$, $P < 0.05$, $N = 40$) (10) and during incremental testing ($r = 0.94$, $P < 0.01$, $N = 50$) (24). Previously, highly significant correlations were obtained in the stroke volume ($r = 0.84$, $P < 0.001$) and the cardiac output values ($r = 0.98$, $P < 0.001$) between the direct Fick and impedance cardiography methods in six healthy male subjects during maximal cycling exercise tests (28). Therefore, it seems that the impedance cardiography provides accurate $\dot{Q}$ measurements during exercise. When associated with $\dot{V}O_2$ measurements, it allows the estimation of the arterial-venous difference by the Fick equation:

$$a\text{-}vO_2diff = \dot{V}O_2/\dot{Q}. \quad [3]$$

Data kinetics modeling. The time course of a-vO_2 diff was described by exponential functions fitted to the data with nonlinear regression techniques (Sigma Plot2000, Jandel, Chicago, IL). The calculation of the best-fit parameters was chosen by the program so as to minimize the sum of the squared differences between the fitted function and the observed response. Two exponential models were used to describe a-vO_2 diff for each work rate. According to the both following equations, oxygen uptake kinetics fit by either mono-exponential [4] or mono-exponential plus drop [5] function:

$$a\text{-}vO_2diff(t) = a\text{-}vO_2diff(b) + A1*(1 - e^{-(t-TD_1)/\tau_1}) \quad [4]$$

$$a\text{-}vO_2diff(t) = a\text{-}vO_2diff(b) + A1*(1 - e^{-(t-TD_1)/\tau_1} - A2*(t - TD2)/\tau 2) \quad [5]$$

where a-vO_2 diff(t) represents arterial venous difference at time (t), a-vO_2 diff(b) is the baseline of the value at the end of the warm-up, A1 was the asymptotic amplitude for the second exponential of oxygen uptake kinetics, τ1 was the time constants, and TD1 was the time delay of each exponential. In the second model to describe the a-vO_2 diff kinetics, A2 was the slope of the linear regression, τ2 was the time constant of the drop, and TD2 was the time delay for the linear regression. Each curve fitting used an iterative least-squares approach. The rationale for selection of the appropriate model was based on analysis of the residuals around the line of best fit (8).

Statistical methods. Descriptive statistics are expressed as mean and SD. According to the data, the normality distribution of the population was analyzed by variance comparison using of the Fisher Snedecor test. Statistical comparisons over time of the physiological variables ($\dot{V}O_2$, $\dot{Q}$, HR, SV, and a-vO_2 diff) were made using a one-way repeated measures ANOVA. Paired *t*-tests were used to compare a-vO_2 diff kinetics responses. Scheffe *post hoc* tests were carried out when appropriate. All significant differences are at the $P < 0.05$ level unless stated otherwise.

RESULTS

Maximal values of the peripheral and central factors of $\dot{V}O_{2max}$ during the incremental exercise. The maximal values measured during the incremental test are in Table 2. All subjects reached a plateau of $\dot{V}O_2$. Heart rate reached its maximal value at $\dot{V}O_{2max}$ (Fig. 2). Stroke volume increased until 93.0 ± 6.0% of $\dot{V}O_{2max}$ corresponding to 89.2 ± 5.3% of p$\dot{V}O_{2max}$, then SV_{max} remained constant until the attainment of $\dot{V}O_{2max}$ and p$\dot{V}O_{2max}$. Therefore, $\dot{Q}_{max}$ was reached with the attainment of maximal heart rate. The arterial-venous O_2 difference increased up to 61.2 ± 23.3% of p$\dot{V}O_{2max}$ corresponding to 63.8 ± 26.4% of $\dot{V}O_{2max}$. So, $\dot{V}O_{2max}$ was reached with $\dot{Q}_{max}$ and a-vO_2 $diff_{max}$ in the incremental test. This was not the case for the constant-load exercises at work rates of pΔ50 (88.0 ± 1.5% of p$\dot{V}O_{2max}$) or 100% of p$\dot{V}O_{2max}$.

Comparison between the maximal values of the peripheral and central factors of $\dot{V}O_{2max}$ between constant-load and incremental exercises. Figures 3–5 show an example of the responses of cardiorespiratory parameters ($\dot{V}O_2$, $\dot{Q}$, HR, SV, and a-vO_2 diff) at both different intensities and time to fatigue (tlim100 and tlimΔ50, respectively). In all subjects, the time to exhaustion was significantly longer at pΔ50 than at p$\dot{V}O_{2max}$ (726 ± 195 s vs 312 ± 145 s, $P < 0.01$). Peak lactate was not significantly different when comparing the three tests (Table 2). $\dot{V}E$, $\dot{V}O_{2max}$, and HR_{max} were reached in the constant-load exercises whatever their intensities (Table 3). During the incremental test, SV reached its maximal value at higher work rates (89.2 ± 5.3% of p$\dot{V}O_{2max}$) compared with pΔ50 (88.0 ± 1.5% of p$\dot{V}O_{2max}$, $P < 0.01$). Furthermore, SV was lower in the tlimΔ50 test (143 ± 27 mL·beat^{-1}) compared with the incremental test (179 ± 34 mL·beat^{-1}; $P < 0.05$). Therefore, $\dot{Q}$ was also lower in tlimΔ50 compared with the incremental test (24.4 ± 3.6 L·min^{-1} vs 28.4 ± 4.1 L·min^{-1}; $P = 0.04$). Therefore, maximal value of a-vO_2 diff was not significantly different when comparing all the ex-

TABLE 2. Time to exhaustion, maximal power output and maximal blood lactate determined during incremental, tlimΔ50 (pΔ50), and tlim100 (p$\dot{V}O_{2max}$) exercises.

Subjects (N = 9)	Incremental Test	tlimΔ50	tlim100
Time to fatigue (s)	1240 ± 153	726 ± 195*	312 ± 145*§
Power (W)	344 ± 37	303 ± 38	344 ± 37
Power (% of p$\dot{V}O_{2max}$)	100.0 ± 0.0	88.0 ± 1.5	100.0 ± 0.0
Maximal blood lactate (mmol·L^{-1})	10.7 ± 1.9	11.6 ± 2.9	11.2 ± 2.8

* $P < 0.05$, level of significance between incremental and constant work rate exercises.
§ $P < 0.05$, level of significance between time to exhaustion at p$\dot{V}O_{2max}$ and pΔ50.

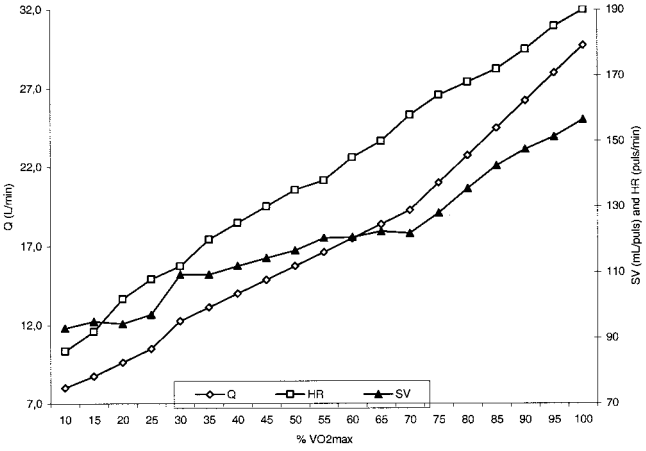

FIGURE 2—Example of the evolution of heart rate (HR), stroke volume (SV), and cardiac output ($\dot{Q}$) according to the percentage of maximal oxygen uptake (%$\dot{V}O_{2max}$) during the incremental test in subject 7. *Squares* represent heart rate kinetics during tlim100; *empty lozenges* represent $\dot{Q}$ kinetics during tlim100; *full triangles* represent SV kinetics during tlim100.

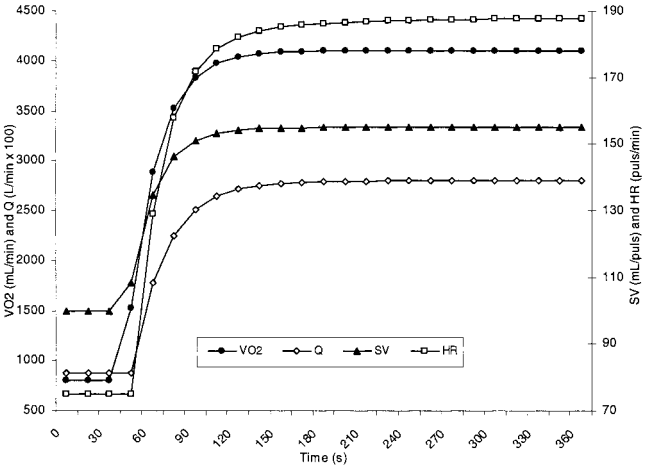

FIGURE 3—Example of modeling kinetics of heart rate (HR), stroke volume (SV), cardiac output ($\dot{Q}$), and oxygen uptake ($\dot{V}O_2$) responses during the workload at p$\dot{V}O_{2max}$ (tlim100) in subject 7. *Circles* $\dot{V}O_2$ kinetics during tlim100; *squares* represent heart rate kinetics during tlim100; *empty lozenges* represent $\dot{Q}$ kinetics during tlim100; *full triangles* represent SV kinetics during tlim100.

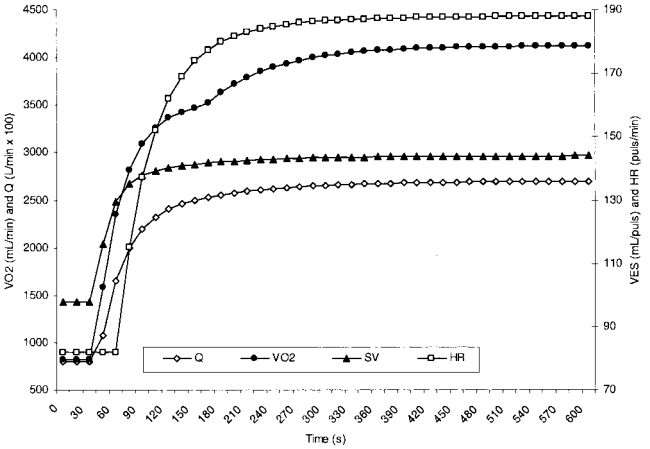

FIGURE 4—Example of modeling kinetics of heart rate (HR), stroke volume (SV), cardiac output ($\dot{Q}$), and oxygen uptake ($\dot{V}O_2$) responses during the workload at pΔ50 (tlimΔ50) in subject 7. *Circles* represent $\dot{V}O_2$ kinetics during tlim100; *squares* represent heart rate kinetics during tlim100; *empty lozenges* represent $\dot{Q}$ kinetics during tlim100; *full triangles* represent SV kinetics during tlim100.

ercises (Table 3 and Fig. 5), but it decreased later in tlimΔ50 compared with tlim100 (6 min 58 s ± 4 min 29 s vs 3 min 6 s ± 1 min 3 s, $P < 0.05$, i.e., 43.0 ± 22.5% and 48.3 ± 18.2% of the time limit, NS). This narrowing in a-vO_2 diff was correlated with the arterial oxygen desaturation that appeared in tlimΔ50 ($SaO_{2rest\ -\ end\ of\ exercise}$ = 15.3 ± 3.9%) ($r = -0.74$, $P < 0.05$) but not in tlim100 ($SaO_{2rest\ -\ end\ of\ exercise}$ = 11.9 ± 2.8%) or in the incremental test (SaO_2 = 8.8 ± 2.8%, $P < 0.05$).

DISCUSSION

The focus of this study was to examine whether the maximal oxygen uptake ($\dot{V}O_{2max}$) was reached with the same central and peripheral factors in exhaustive constant load exercises performed at p$\dot{V}O_{2max}$ and pΔ50 compared

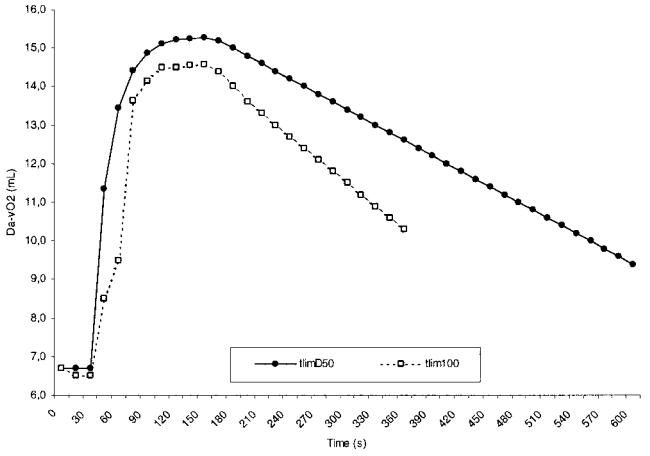

FIGURE 5—Example of arterial-venous difference kinetic (a-vO_2 diff) during exercise at p$\dot{V}O_{2max}$ (tlim100) and at pΔ50 (tlimΔ50) in subject 7. *Squares* and *dashed line* represent a-vO_2 diff values and its mathematical model during time to exhaustion at p$\dot{V}O_{2max}$ (tlim100); *circles* and *solid line* represent a-vO_2 diff values and its mathematical model during time to exhaustion at pΔ50 (tlimΔ50).

TABLE 3. Maximal values during incremental exercise and constant workload at pΔ50 (tlimΔ50) and p$\dot{V}O_{2max}$ (tlim100).

Subjects (N = 9)	Incremental Test	tlimΔ50	tlim100
$\dot{V}O_{2max}$ (mL·min^{-1})	4463 ± 452	4363 ± 453	4545 ± 500
HR_{max} (beat·min^{-1})	171 ± 14	175 ± 13	173 ± 14
SV_{max} (mL·beat^{-1})	179 ± 34	143 ± 27*	153 ± 27
$\dot{Q}_{max}$ (L·min^{-1})	28.4 ± 4.8	24.4 ± 3.6*	25.8 ± 4.1
a-vO_2 diff (mL)	13.2 ± 1.4	13.1 ± 2.1	13.3 ± 0.9
$\dot{V}_{Emax}$ (L·min^{-1})	163.4 ± 26.3	167.1 ± 27.7	161.6 ± 35.2
ΔSaO_2 (%)	8.8 ± 2.8	15.3 ± 3.9	11.9 ± 4.8

* $P < 0.05$, level of significance between incremental and constant work rate exercises.
§ $P < 0.05$, level of significance between time to exhaustion at p$\dot{V}O_{2max}$ and pΔ50.

with the reference values of a classic incremental test (with 3-min stages). The main finding from this study was that $\dot{V}O_{2max}$ was attained with a lower value of cardiac output due a lower SV in tlimΔ50 than in the incremental test. The maximal arterial-venous O_2 difference was not significantly different according the exercise intensity but decreased more slowly in the tlimΔ50 compared with tlim100 test. First, we are going to discuss the value of power output at which the stroke volume reaches its maximum.

Maximal values of the peripheral and central factors of $\dot{V}O_{2max}$ during the incremental exercise. In agreement with recent studies performed with trained subjects (9,16), SV reached its maximal value almost until $\dot{V}O_{2max}$ (93 ± 6% of $\dot{V}O_{2max}$). Indeed, SV did not plateau at 40–60% of $\dot{V}O_{2max}$ as reported in nonactive subjects (1). This continuous increase in SV until exhaustion could be explained by a greater left ventricular filling in well-trained males (16,30). In the present study, the heart rate increase until the attainment of p$\dot{V}O_{2max}$ was responsible of the attainment $\dot{Q}_{max}$ at the end of the incremental exercise test. In agreement with a previous investigation by Grassi et al. (17), who observed an increase in muscle oxygenation ([O_2Hb − HHb]) until 60–65% of $\dot{V}O_{2peak}$, our results also showed an increase in a-vO_2 diff until 63.8 ± 26.4% of $\dot{V}O_{2max}$. Therefore, $\dot{V}O_{2max}$ was reached with the maximal value of $\dot{Q}$ and a-vO_2 diff during the incremental test.

$\dot{Q}$-$\dot{V}O_2$ responses during two severe exercises eliciting $\dot{V}O_{2max}$. Despite the fact than tlimΔ50 elicited $\dot{V}O_{2max}$ (6,14), the maximal stroke volume was not reached. In a pilot study, Billat et al. (9) have reported that during a constant load exercise at 90% of p$\dot{V}O_{2max}$ (11 min 42 s ± 4 min 7 s), both SV and $\dot{V}O_2$ increased until exhaustion reaching the maximal value measured in an incremental test. The SV increase with time in constant supra-threshold exercise was not observed in the present study. This is probably due to the fact that the work rate was not sufficient because the intensity that elicited SV_{max} in the increment test was higher than pΔ50 (89.2 ± 5.3 vs 88.0 ± 1.5% of p$\dot{V}O_{2max}$, $P < 0.01$). Also, the exercise duration was put forward as being a major factor for the achievement of $\dot{V}O_{2max}$ without $\dot{Q}_{max}$ attainment. This "$\dot{Q}$–$\dot{V}O_2$ disassociation" has been attributed to the lower stroke volume in an incremental test lasting 12 min versus 6 min (21). In spite of similar maximal values of oxygen uptake and heart rate, maximal stroke volume and maximal cardiac output values were lower in the 12-min than 6-min test. A longer exercise

duration could induce a vasodilatation and an increased vascular conductance via the muscle pump in skeletal muscle considered as a determinants of sustained exercise hyperemia in skeletal muscle (13).

However, the duration of exercise, even in exercise eliciting $\dot{V}O_{2max}$ (so-called "heavy exercise," (29)) is not sufficient to elicit SV_{max}. It seems that in trained subjects, 90% of $p\dot{V}O_{2max}$ is an intensity threshold for the attainment of maximal SV both in an incremental test (with 3-min stages) and constant-load exercise. This threshold intensity to reach the maximal stroke volume attainment could depend on the quadriceps muscle mass involved in the cycling exercise, which might be higher in tlim100 compared with tlimΔ50. At the onset of dynamic exercise, the mechanical effects of the muscle pump increased the arterial-venous pressure gradient (12). Therefore, the pumping action of contracting skeletal muscle appeared to form an important regulator of increasing blood flow. The rate of increase in blood flow during exercise is closely coupled to motor unit recruitment with dilation beginning at the first contraction (26). Indeed, Nassis and Geladas (22) have recently demonstrated that the decline in stroke volume during an exercise at 60% of $p\dot{V}O_{2max}$ was higher in cycling than in running. They showed a moderate correlation between the one-leg quadriceps muscle mass and SV decline in cycling, implying that individuals with high thigh muscle mass would present a smaller SV decline in cycling compared with running (22). In the present study, we have one exercise mode (cycling) but two intensities that have a difference of 40 W, i.e., 4.44 kgF (at a rate of pedaling equal to 90 rpm). This difference was sufficient to reach or not reach the maximal stroke volume at the onset of the dynamic exercise. The quicker recruitment of a large muscle mass at the onset of exercise at the higher intensity was responsible to the attainment of maximal value of SV. Alternatively, the progressive recruitment of new unit motors associated with respiratory muscles (19,23,25) could be responsible for the attainment of $\dot{V}O_{2max}$ without the $\dot{Q}_{max}$ during tlimΔ50.

Arterial-venous O_2 difference responses during two severe exercises eliciting $\dot{V}O_{2max}$. Our results showed that a-vO_2 diff was not significantly different between the three exercises. However, a-vO_2 diff narrows less quickly in the lower work rate test (tlimΔ50). McCole et al. (21) postulated that the lower stroke volume during 12-min versus the 6-min incremental test could be in part compensated by the increased of arterial-venous O_2 difference (a-vO_2 diff). The time course of a-vO_2 diff may be the result of a rise of lactic acidosis and a subsequent decrease in pH above lactate threshold (27). In our study, the blood lactate level was not different between the three exercises. Previously, Demarie et al. (14) found a correlation between the vastus lateralis deoxygenation changes with the oxygen uptake changes and the blood lactate increase between the third minute of exercise and the time to fatigue. Furthermore, the decrease in a-vO_2 diff was associated with a hypoxemia induced by exercise ($SaO_{2rest\ -\ end\ of\ exercise}$), which was also more pronounced during tlimΔ50 than in incremental test. Thus, muscle de-oxygenation from 60-65% of $\dot{V}O_{2peak}$ could be attributed to capillary-venular hemoglobin desaturation, as a consequence of a rightward shift of the oxy-hemoglobin dissociation curve determined by the onset of lactic acidosis (17). On the other hand, during severe exercise below $p\dot{V}O_{2max}$, the right shift of the oxy-hemoglobin dissociation curve on tissue oxygenation would be responsible of the attainment of maximal value of $\dot{V}O_2$. On the contrary, the longer duration of the exercise would be responsible for a more marked hypoxemia which would counterbalance the Böhr effect and would be responsible for ending the exercise. Hence, we can define $p\dot{V}O_{2max}$ as the work rate allowing matching $\dot{Q}_{max}$ with maximal value of a-vO_2 diff, both in incremental and constant work rate. This work rate ($p\dot{V}O_{2max}$) has as also been reported as being the work rate eliciting $\dot{V}O_{2max}$ for the longest duration in constant load exercise (6).

CONCLUSION

Despite there being no difference in the maximal value of $\dot{V}O_2$, the $\dot{V}O_{2max}$ reached during the incremental test resulted in a greater maximal $\dot{Q}$ compared with the constant pΔ50 workload exercise. The lower SV seems to be the main contributor of the nonachievement of $\dot{Q}_{max}$ during time to exhaustion at pΔ50 whereas $\dot{V}O_2$ reached its maximal value in the both cases. So, $\dot{V}O_{2max}$ was not reached with the same central and peripheral factors in exhaustive exercises performed at 88.0 $\pm$ 1.5 or 100% of $p\dot{V}O_{2max}$. This might be taken into account for training at $\dot{V}O_{2max}$ using different work rates (90–100% $p\dot{V}O_{2max}$).

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