Eccentric Cycle Exercise: Training Application of Specific Circulatory Adjustments

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ABSTRACT

DUFOUR, S. P., E. LAMPERT, S. DOUTRELEAU, E. LONSDORFER-WOLF, V. L. BILLAT, F. PIQUARD, and R. RICHARD. Eccentric Cycle Exercise: Training Application of Specific Circulatory Adjustments. Med. Sci. Sports Exerc., Vol. 36, No. 11, pp. 1900–1906, 2004. Purpose: Despite identical oxygen uptake (VO2) responses have been reported in eccentric (ECC) versus concentric (CON) cycle exercise, the aim of this study was to describe the specific circulatory adjustments (HR and stroke volume (SV)) to incremental ECC cycle exercise in order to: 1) determine the HR values leading to identical VO2 in ECC and CON cycling; and 2) estimate the interindividual variability of this HR correspondence between the two exercise modes, with emphasis upon rehabilitation and training purposes. Methods: Eight healthy male subjects (age, 28 ± 2 yr) participated in this study. They performed CON and ECC cycle incremental exercises (power output increases of 50 W every 3 min). Breath-by-breath gas exchange analysis and beat-by-beat thoracic impedancemetry were used to determine VO2 and Q, respectively. Results: At the same metabolic power (VO2 of 1.08 ± 0.05 L·min⁻¹ in CON vs 1.04 ± 0.06 L·min⁻¹ in ECC), SV was not different, but HR was 17% higher in ECC (P < 0.01), leading to a 27% enhanced Q (P < 0.01). Q and HR net adjustments (exercise minus resting values) in ECC versus CON muscle involvement demonstrated important interindividual variability with coefficients of variation amounting to 32% and 30%, respectively. Conclusion. In practice, if a given level of VO2 is to be reached, ECC HR has to be set above the CON one. Taking into account the interindividual variability of the circulatory adjustments in ECC versus CON muscle involvement, a precise HR correspondence can be established individually from the VO2/HR relationship obtained using ECC incremental testing, allowing prescription of accurate target HR for rehabilitation or training purposes. Key Words: CARDIOVASCULAR ADAPTATIONS, MUSCLE ACTION TYPES, IMPEDANCE CARDIOGRAPHY, INCREMENTAL EXERCISE TESTING

In conventional cycle ergometry, muscles mainly shorten during activation, therefore performing under concentric (CON) involvement. However, cycle ergometry can also be adapted to impose muscle lengthening during pedaling movements, that is, eccentric actions (ECC) (19,20,23). If the concept of ECC cycle ergometry is already an old issue (1), its exercise-induced metabolic and circulatory adaptations are by far less well understood than for conventional CON cycle exercise.

Compared with CON, early studies on the physiological response to ECC cycling established that its oxygen cost is considerably lower (about 1/5 of the CON one) (25) and that, for a given level of VO2 (higher than 1 L·min⁻¹), both cardiac output (Q) and heart rate (HR) are significantly enhanced (25,29). As a consequence, studies using multiple constant-load exercises established that the ECC VO2/HR relationship is steeper than for CON cycling (14,17,29). Based on this specific feature of the ECC circulatory response, a direct application for training and rehabilitation purposes is that a similar HR value in both exercise modes does not mean identical VO2.

However, recent promising results on ECC cycle training did not have taken into account this particular adaptation, because the experimental groups exercised at the same expected VO2, set as a percentage of peak HR whatever the exercise mode (19,23). As ECC cycle intensity is currently based upon exercise HR (19,20) with wide potential applications in rehabilitation and training fields (18–20,22,23,27,28), possible differences (that is, magnitude and interindividual variability) between ECC and CON cycling circulatory adaptations need to be clearly identified.

In CON cycling, the individual VO2/HR relationship is commonly determined using a single session of incremental exercise testing (IET) (3). Therefore, VO2 or energy expenditure could be indirectly estimated based on a given value of exercise HR, allowing many of the current training pre-
scriptions to be based upon the attainment of target HR values (2,5). Moreover, this type of exercise testing is repeated regularly in order to adjust the training intensities to the improved physical fitness. Apparently, only one study has investigated the physiological response to ECC IET. Nevertheless, the experimental design was limited to low power output levels (up to 147 W) (14), which are far from the levels employed in current ECC training studies (that is, higher than 250 W). As VO2 and HR have been reported to adjust more rapidly to a given magnitude of ECC rather than CON stepwise power output transition, one could assume that the achievement of the individual ECC VO2/HR relationship could also be established with an IET model. Such a methodology would enable practical information to be obtained for training and rehabilitation purposes, in order to both individualize and optimize ECC exercise prescriptions.

In this context, the aim of this study was to describe the specific circulatory adjustments (HR and stroke volume (SV)) to ECC cycle exercise in order to: 1) determine the respective HR values leading to identical VO2 in ECC and CON cycling, and 2) estimate the interindividual variability of this HR correspondence between both exercise modes.

**METHODS**

**Subjects**

Eight healthy well-motivated men were included in this study. Subjects were free of drug therapy and did not report any history of muscle, tendinous, or articular problems. After they were informed of the nature of the experiment and the possible risks, each subject gave an informed consent document consistent with the Declaration of Helsinki and approved by our institutional review board.

**Experimental Design**

**Habituation period.** ECC muscle actions have been reported to induce delayed onset muscle soreness (DOMS) (9), especially at high muscle tension. Nevertheless, recent studies have shown that regular and progressive exposition to ECC constraints significantly reduce DOMS and prevent the appearance any adverse side effect (that is, repeated bout effect) (19,20). During the 2 wk preceding the study, all the subjects underwent learning sessions to acquire the specific coordination of ECC pedaling and minimize muscle, tendinous, or articular problems (25). This learning period comprised 3 or 4 exercise days separated by a minimum of 2–3 d of recovery. Each session consisted of 20 min of exercise, beginning with a 5-min CON warm-up at a self-selected intensity, followed by 10 min of ECC pedaling, and ending with 5 min of CON recovery at a self-selected intensity. The intensity of the 10-min ECC pedaling was progressively increased from 100 W at the first session to 200 W in the last session, which was performed at least 3 d before the start of the experimental protocol. After this habituation period, the subjects entered the experimental protocol if they reported no residual muscle soreness.

**Exercise testing.** The learning exercises and the two tests were performed on a specific half-sitting position ergometer developed on the basis of a commercially available ergometer (Recline XT, TechnoGym, Gambettola, Italy) that was modified with a 2.2-kW asynchronous electric motor with a precision of ± 1% on the nominal power (MasterDrive Simover Vector Control, Siemens, Erlangen, Germany). This motor allows the pedal to be driven in a reverse direction from normal pedaling in ECC mode and also permits setting resistances in CON mode. The mechanical power measured was controlled in both exercise modes independently using a calibration device comprising a torque meter and a tachymeter. The accuracy of the power was shown to be ± 5 W between 0 and 1000 W. Specially developed, this ergometer was commanded by a computer and specific software (Labview FDS 5.1, National Instruments, Austin, TX) to permit CON and ECC pedaling exercises. It should be emphasized that this ergometer: 1) allowed setting the level of mechanical power the subject had to overcome in the CON condition; and 2) measured the mechanical power that the subject developed in the ECC exercise, which had to be self-adjusted to reach the desired level of mechanical power. The seat and handlebar positions were adjusted to each subject’s morphology and kept constant for the ECC and CON tests.

The experimental protocol consisted of two test sessions on the specific ergometer with either CON or ECC leg pedaling at 80 rpm. Each exercise test was preceded by 10 min of resting recordings with the subject half-seated on the ergometer. The first exercise test was always realized with CON muscle involvement. The initial work rate of 50 W lasting 3 min was increased by 50 W every 3 min until exhaustion. Maximal effort was attested as the subjects always reached at least three of four of the following criteria: 1) plateau of VO2 despite further increase in mechanical power output, 2) respiratory exchange ratio (RER) greater than 1.15, 3) blood lactate level higher than 8 mmol·L−1, and 4) maximal HR higher than 90% of the theoretical maximal value (16). For the ECC test, we used the same incremental workloads as for the CON test. Because the purpose of this study was to compare the ECC and CON circulatory responses with identical mechanical power and with equivalent VO2, the subjects did not perform ECC exercise beyond the peak mechanical power output reached during the CON test. Therefore, end-exercise ECC (EE ECC) values correspond to the highest values attained during the ECC test but do not represent true maximal ECC data.

**Measurements**

**Breath-by-breath gas exchange.** Gas exchange (VO2, carbon dioxide output (VCO2)) data were collected breath-by-breath with an open-circuit metabolic cart with rapid O2 and CO2 analyzers (Ergocard with Exp’Air software version 1.26.35, Medi-Soft, Dinant, Belgium). Before each exercise test, the pneumotachograph was calibrated with a 3-L calibration syringe (model 5530, Hans Rudolph, Kansas City, MO). The gas analyzers were calibrated with
reference gases of known O₂ and CO₂ concentrations (12% O₂ and 5% CO₂).

**Cardiac output measurements.** We used a bioimpedance device to measure the SV and obtained an electrocardiogram (ECG) to measure HR concomitantly (Physio Flow, Manatec type PF05L1, Paris, France) (6,26). HR was based on the R-R interval duration, determined on the ECG first derivative dECG/dt, which provides a more stable signal than the ECG signal itself. Q determination by the apparatus is based on the following formula:

\[ Q = HR \times SV_i \times BSA \]

where \( Q \) is expressed in liters per minute, SV\( i \) is the SV index, and BSA is the body surface area calculated according to the Haycock et al. formula (10). With this impedance device, a first evaluation of SV\( i \), called SV\( \text{i}_{\text{cal}} \), is computed during a calibration procedure based on 24 consecutive heartbeats recorded with the subject at rest in the half-seated position on the ergometer (6).

**Blood parameters.** Venous blood samples were drawn at rest and at the end of each workload. Whole-blood samples were immediately analyzed for total plasma lactate concentration ([La], Chiron-Diagnostics Serie 800, Bayer, Puteau, France).

**Data Analysis**

**Signal treatment.** Gas exchange and hemodynamic data, initially obtained breath-by-breath and beat-to-beat, respectively, were later reduced to 15-s averages for all the tests. For each parameter, the start of the test was stamped while recording. These reference points permitted to obtain a single synchronous database for both gas exchange and hemodynamic variables. Therefore, as \( \dot{V}O_2 \) and \( Q \) were determined by two independent methods (gas exchange analysis and bioimpedance, respectively), we were able to estimate the arteriovenous O₂ difference (A-\( \dot{V}O_2\)imp), by dividing \( \dot{V}O_2 \) by \( Q \) values averaged over the corresponding time interval. To further emphasize the accuracy of our impedance-derived data, we also calculated theoretical \( Q \) values during the CON test, based upon the formulas of Astrand et al. (4).

**Statistics.** The variability of the individual response to ECC and CON pedaling was computed from the net physiological values, calculated as exercise minus resting values for each parameter in each condition. The individual differences in ECC versus CON values were calculated as net ECC minus net CON values for each investigated parameter and referred to as net delta values. Coefficients of variation (CV) for the net delta values were calculated as the standard deviation of an individual’s measurement (net ECC vs net CON), expressed as a percentage of his individual mean test score (15). Within a given physiological parameter, the CV was, therefore, calculated as the mean ± SEM of the corresponding individual’s CV.

Statistical analyses were performed using Sigma Stat for Windows (version 2.0, SPSS Inc., Chicago, IL). After testing for data distribution normality and variance homogeneity, paired \( t \)-tests were employed to assess significant differences for a given condition (peak values or equal metabolic power) between the two types of muscle action. Two-way ANOVA for repeated measures was used to assess the global effects of mechanical power and type of muscle action in each relation investigated. This procedure was followed by Newman-Keuls post hoc tests. \( P < 0.05 \) was considered as statistically significant and all results are expressed as means ± SEM.

**RESULTS**

**Habituation Period**

None of the subjects complained of DOMS after the initial training sessions. Moreover, all the subjects acquired good ECC pedaling skills after three or four training sessions and were thereafter able to realize adequately the incremental ECC test.

Mean age, height, body mass, peak [La], peak \( \dot{V}O_2 \), and RER at peak exercise for all the subjects are presented in Table 1.

**TABLE 1. Physical characteristics and maximal metabolic capacity of the subjects obtained during the CON exercise test.**

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age (yr)</th>
<th>Height (cm)</th>
<th>Body Mass (kg)</th>
<th>Peak [La] (mmol·L(^{-1}))</th>
<th>Peak ( \dot{V}O_2 ) (mL·min(^{-1})·kg(^{-1}))</th>
<th>RER (At Peak Exercise)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>26</td>
<td>178</td>
<td>78</td>
<td>12.6</td>
<td>43.8</td>
<td>1.21</td>
</tr>
<tr>
<td>2</td>
<td>27</td>
<td>173</td>
<td>67</td>
<td>4.3</td>
<td>61.0</td>
<td>1.16</td>
</tr>
<tr>
<td>3</td>
<td>22</td>
<td>191</td>
<td>72</td>
<td>9.4</td>
<td>49.1</td>
<td>1.26</td>
</tr>
<tr>
<td>4</td>
<td>26</td>
<td>175</td>
<td>63</td>
<td>8.6</td>
<td>62.0</td>
<td>1.18</td>
</tr>
<tr>
<td>5</td>
<td>23</td>
<td>182</td>
<td>79</td>
<td>12.1</td>
<td>52.2</td>
<td>1.27</td>
</tr>
<tr>
<td>6</td>
<td>35</td>
<td>181</td>
<td>67</td>
<td>12.4</td>
<td>60.1</td>
<td>1.27</td>
</tr>
<tr>
<td>7</td>
<td>42</td>
<td>178</td>
<td>79</td>
<td>5.7</td>
<td>35.9</td>
<td>1.30</td>
</tr>
<tr>
<td>8</td>
<td>24</td>
<td>180</td>
<td>62</td>
<td>11.8</td>
<td>65.5</td>
<td>1.24</td>
</tr>
<tr>
<td>Mean ± SEM</td>
<td>28 ± 2</td>
<td>180 ± 2</td>
<td>71 ± 3</td>
<td>9.6 ± 1.1</td>
<td>53.7 ± 3.6</td>
<td>1.24 ± 0.02</td>
</tr>
</tbody>
</table>

RER, respiratory exchange ratio; [La], plasma lactate concentration.

**TABLE 2. Peak CON and end-exercise ECC values at the same mechanical power (287 ± 16 W).**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Peak CON*</th>
<th>EE ECC*</th>
<th>EE ECC (% Peak CON)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \dot{V}O_2 ) (mL·min(^{-1})·kg(^{-1}))</td>
<td>53.7 ± 3.6</td>
<td>19.1 ± 1.5*</td>
<td>37 ± 4</td>
</tr>
<tr>
<td>( Q ) (L·min(^{-1}))</td>
<td>22.8 ± 1.1</td>
<td>14.0 ± 0.9*</td>
<td>61 ± 3</td>
</tr>
<tr>
<td>HR (beats·min(^{-1}))</td>
<td>180 ± 6</td>
<td>120 ± 8*</td>
<td>66 ± 3</td>
</tr>
<tr>
<td>SV (mL)</td>
<td>127 ± 6</td>
<td>120 ± 9</td>
<td>94 ± 4</td>
</tr>
<tr>
<td>(A - ( \dot{V}O_2)imp) (mL·100 mL(^{-1}))</td>
<td>16.6 ± 0.9</td>
<td>9.7 ± 0.5*</td>
<td>59 ± 5</td>
</tr>
<tr>
<td>[La] (mmol·L(^{-1}))</td>
<td>9.6 ± 1.1</td>
<td>1.2 ± 0.3*</td>
<td>13 ± 2</td>
</tr>
</tbody>
</table>

Values are means ± SEM; \( N = 8 \) subjects; EE ECC, end-exercise eccentric.

* Peak data recorded during the CON test.

\( \dot{V}O_2 \), oxygen consumption; Q, cardiac output; HR, heart rate; SV, stroke volume; (A - \( \dot{V}O_2\)imp), arteriovenous oxygen difference determined with Q measured by impedance; [La], plasma lactate concentration.

* Significant difference \( P < 0.001 \) between ECC and CON at peak exercise.
ECC versus CON Cycling: Magnitude of the Metabolic and Circulatory Responses

Same mechanical power. Peak CON and EE ECC values were observed at the same mechanical power corresponding to 287 ± 16 W (Table 2). Despite high values of mechanical power output, it should be brought to mind that EE ECC values do not represent maximal ECC values but rather correspond to the highest values obtained during the ECC IET and therefore remain submaximal. In this context, the EE ECC value for VO₂ was significantly lower than the one at peak CON (P < 0.001). This observation was associated with significantly lower Q and HR (P < 0.001) but not different SV (Table 2). The Δ VO₂imp was also reduced at EE ECC compared with peak CON (P < 0.001).

The differences reported between peak CON and EE ECC parameters occurred over the totality of the work intensity span (50–300 W) for metabolic (Fig. 1) as well as for cardiovascular variables (Fig. 2). In contrast, the type of muscle action did not induce significant difference in SV for any of the mechanical workloads considered. Theoretical and measured Q values were not different throughout the CON test (Fig. 2).

Same metabolic power. Parameters of the circulatory response as a function of VO₂ are reproduced in Figure 3. During the course of both IET, the same VO₂ of about 1 L·min⁻¹ reached at 50 W in CON was elicited by a mechanical power output of 256 ± 20 W in ECC. The oxygen uptake corresponded to absolute values of 1070 ± 43 mL·min⁻¹ in CON versus 1035 ± 62 mL·min⁻¹ in ECC, leading to an ECC oxygen cost ratio of about 1/5 that of the CON one.

In this condition, A-VO₂imp was lower (P < 0.001) but offset by higher Q in ECC compared with CON exercise (P < 0.001). As shown in Table 3, the higher Q in ECC muscle involvement was associated with higher HR (P < 0.001) but not a statistically significant difference in SV.

ECC versus CON Cycling: Interindividual Variability of the Circulatory Response

Because no difference appeared in the basal VO₂ (260 ± 17 mL·min⁻¹ in CON vs 249 ± 13 mL·min⁻¹ in ECC), net VO₂ values (exercise minus resting value) were also the same, amounting to 810 ± 46 mL·min⁻¹ in CON and 790
individual response to ECC cycle exercise, owing to optimize the calibration of ECC exercise intensity according to target training HR.

**ECC versus CON cycling: magnitude of the metabolic and circulatory responses.** All metabolic ($V_{O2}$, $[La]$, A-$\overline{V_{O2}}_{imp}$) and cardiovascular parameters (HR and $Q$) demonstrated lower values in ECC than in CON muscle involvement whatever the mechanical workload. Significant physiological differences appeared as soon as the first workload during the incremental test (50 W) and persisted over the entire intensity span (50–300 W). These observations confirm the data on cardiovascular constraint and subjective perception of effort, which have also been reported to be lower in isolated constant load ECC exercises (11,29).

Moreover, we found that $[La]$ increased only slightly with ECC mechanical power (EE ECC $[La]$ was not different from basal values $1.22 \pm 0.25$ vs $0.79 \pm 0.07$ mmol L$^{-1}$, respectively, data not shown). Therefore, in agreement with Perrey et al. (25), no blood lactate accumulation appeared until 300 W (Fig. 1), preventing us localizing any potential ECC lactate threshold and confirming the low energy cost of ECC cycle exercise. As a consequence, the physiological values obtained at the EE ECC power output should not be interpreted as maximum ECC values as long as the ECC IET remains submaximal.

According to Coyle (7), we compared the physiological responses to CON and ECC cycle exercises at equivalent oxygen uptake ($1.07 \pm 0.04$ vs $1.04 \pm 0.06$ L min$^{-1}$, respectively). This metabolic level, corresponding to $28 \pm 3\%$ of the subject’s peak CON $V_{O2}$, was obtained at 50 W for all the subjects in the CON test. In contrast, this $V_{O2}$ corresponded to a mean power output of $250 \pm 21$ W in ECC cycling. Despite high values of ECC mechanical power, it should be kept in mind that this intensity remains submaximal. Even at this submaximal intensity and despite similar metabolic power, we observed a significantly reduced A-$\overline{V_{O2}}_{imp}$ in ECC compared with CON exercise. This $22\%$ lower A-$\overline{V_{O2}}_{imp}$ was associated with a $27\%$ higher $Q$ for a given oxygen uptake during ECC exercise. This difference also appears when ECC $Q$ values are expressed relative to theoretical CON $Q$ values ($+31\%$). The physiological mechanisms underlying this compensatory adapta-

**DISCUSSION**

The main findings of this study are that: 1) the greater $Q$ response to ECC versus CON exercise of equivalent metabolic power is mainly the result of an enhanced HR with no difference in SV, 2) HR values leading to identical $V_{O2}$ could be determined from ECC and CON cycle IET, and 3) HR correspondence between the two exercise modes demonstrated some interindividual variability. Therefore, absolute ECC HR values need to be interpreted cautiously to manage ECC exercise intensities. In practice, incremental ECC testing provides an accurate determination of the individual variation. The net delta $Q$ showed a CV amounting to $32 \pm 8\%$, with individual values in the range of $0.1$ to $6.3$ L min$^{-1}$. For HR, all net delta values lay between $1$ to $40$ beats min$^{-1}$ with a CV of $30 \pm 8\%$.

**TABLE 3.** CON and ECC values corresponding to a $V_{O2}$ of 1 L min$^{-1}$.

<table>
<thead>
<tr>
<th>Variable</th>
<th>CON</th>
<th>ECC</th>
<th>ECC (% CON)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mechanical power (W)</td>
<td>50 ± 0</td>
<td>250 ± 21</td>
<td>500 ± 38</td>
</tr>
<tr>
<td>$V_{O2}$ (L min$^{-1}$)</td>
<td>1.07 ± 0.05</td>
<td>1.04 ± 0.06</td>
<td>97 ± 4</td>
</tr>
<tr>
<td>$Q$ (L min$^{-1}$)</td>
<td>10.0 ± 0.5</td>
<td>12.5 ± 0.5</td>
<td>127 ± 7</td>
</tr>
<tr>
<td>HR (beats·min$^{-1}$)</td>
<td>95 ± 5</td>
<td>110 ± 8*</td>
<td>117 ± 4</td>
</tr>
<tr>
<td>SV (mL)</td>
<td>107 ± 7</td>
<td>116 ± 8</td>
<td>109 ± 4</td>
</tr>
<tr>
<td>$A-\overline{V_{O2}}_{imp}$ (mL·100 mL$^{-1}$)</td>
<td>10.9 ± 0.6</td>
<td>8.3 ± 0.4*</td>
<td>78 ± 5</td>
</tr>
<tr>
<td>$[La]$ (mmol·L$^{-1}$)</td>
<td>1.0 ± 0.1</td>
<td>1.0 ± 0.1</td>
<td>96 ± 8</td>
</tr>
</tbody>
</table>

Values are means ± SEM; N = 8 subjects; CON, concentric data corresponding to an oxygen uptake of 1 L min$^{-1}$; ECC, eccentric data corresponding to an oxygen uptake of 1 L min$^{-1}$; *Eccentric values expressed as a percentage of the concentric values. $V_{O2}$, oxygen uptake; $Q$, cardiac output; HR, heart rate; SV, stroke volume; $A-\overline{V_{O2}}_{imp}$, arteriovenous oxygen difference determined with $Q$ measured by impedance; $[La]$, plasma lactate concentration. * Significant difference ($P < 0.001$) between ECC and CON at 1 L min$^{-1}$ $V_{O2}$. 

**FIGURE 3.** Evolution of the circulatory parameters as a function of metabolic power during CON and ECC tests. Values are means ± SEM of the last 30 s of each workload. $Q$, cardiac output (top); SV, stroke volume (middle); HR, heart rate (bottom); □, CON measured; ■, ECC.
tion are presently unclear. An elevated Q could direct more blood flow toward inactive tissues (that is, the cutaneous level). Because these territories extract less oxygen than exercising muscles, the returning blood flow to the heart could be more oxygenated (30). Thus, the circulation during ECC exercise may not be governed only by metabolic requirements but other regulations could play a major role (that is, thermoregulatory or muscle tension induced adaptations) (21). Recent results obtained in rats with a downhill versus level locomotion model showed that the distribution of muscle blood flow in active muscles is muscle action type dependent. Delp et al. (8) observed a lower total leg blood flow during ECC exercise but reported that this variable did not decrease uniformly in all active muscles. Lower or equal muscle blood flows were reported in some eccentrically solicited ankle flexor muscles (8). These different patterns of muscle blood flow adjustment, together with reduced whole-body VO$_2$ and [La] for a given speed of downhill versus level running, are consistent with our observed dissociated metabolic and cardiovascular responses to CON and ECC cycle exercise.

Because no significant difference was observed for SV, the higher Q response in ECC cycle exercise was mainly the result of a 17% higher HR. This higher ECC HR could originate principally from two factors. First, ECC work has been shown to induce a specific elevated muscle temperature with no change in core temperature compared with CON work at the same metabolic power (24). This characteristic could have induced an HR-mediated elevated Q either by thermoregulation activation (29) or by thermal response of group III and IV muscle afferents (13). Second, CON and ECC cycle exercises, although identical from a metabolic standpoint, require a large difference in mechanical power (50 vs 256 W, respectively). This difference induces greater muscle tension during ECC exercise and could influence cardiovascular response in this way. Previous research has shown that muscle tension is an important factor for HR control (21) mainly via the mechanically sensitive group III and IV muscle nerve afferents, the free nerve endings of which reside within the interstitium of the exercising muscles (12).

**ECC versus CON cycling: interindividual variability of the circulatory response.** As they contribute to the muscle sympathetic nerve activity, thermal and mechanical hypotheses of the greater circulatory response to ECC cycle exercise could also be involved in the interindividual variability observed (CV = 30% for HR and 32% for Q). Their respective individual sensitivity could at least partly account for the interindividual variability of the ECC versus CON HR and Q exercise response. To the best of our knowledge, the individual variability of the muscle tension or temperature threshold that leads to increased sympathetic outflow from the exercising muscle is not well established. For instance, one can postulate that those individuals presenting the wider HR difference between both exercise modes at a given VO$_2$ also possess the lower tension threshold to activate their muscle mechanoreceptors and then increase their sympathetic drive to HR and Q. In this case, differential circulatory adjustments as a function of muscle action type would be intimately related to muscle innervation properties. However, the results of the present study do not allow us to investigate these mechanisms further.

**Training application.** Given its metabolic and cardiovascular properties, ECC cycle exercise is currently proposed as an interesting way of allowing high muscle tension development with little oxygen and circulatory requirements (19,20,22). Recently, two studies investigated the physiological benefits of an 8-wk ECC cycle training program with healthy subjects (19,20). There were reported significant improvements in isometric leg strength (+30%) associated with increased fiber cross sectional area (+52%) and capillary-to-fiber ratio (+47%). In comparison, a control group exercising concentrically on a classic cycle ergometer demonstrated no significant alterations despite similar training HR. Using an identical approach, Meyer et al. (23) also reported that high force ECC cycle training is well-tolerated and implies no adverse effects either on central hemodynamic or metabolic responses. Therefore, as muscle strength is recognized as an important performance factor in daily living as well as in many sporting events, these promising results suggest that ECC cycle training could be beneficial for a broad population from the most severely centrally-limited patients to high-level athletes (22).

However, because of the specific circulatory adjustments of ECC cycle exercise, ECC HR values need to be interpreted cautiously. Despite equivalent metabolic power, HR becomes significantly elevated in ECC compared with CON cycle exercises, even at moderate ECC exercise level (250 W). As the VO$_2$/HR relationship appears to be steeper in ECC cycling, one can expect that higher ECC metabolic intensities (that is, greater than 1 L·min$^{-1}$) would be associated with even wider absolute HR differences. A direct consequence is that a given HR value in both exercise modes corresponds to a lower VO$_2$ in ECC than in CON cycling. Therefore, because it underestimate ECC exercise intensity, equalizing exercise HR no matter what the exercise mode should no longer be interpreted as a valid procedure to establish identical metabolic intensity during training programs. Because this method has been used in recent ECC training studies in normal and coronary subjects (19,20,23,28), we can hypothesize that the promising results reported were also underestimated. Moreover, all the subjects did not respond similarly to ECC cycle exercise. Indeed, the amplitude of the HR difference leading to identical oxygen uptake for both types of muscle involvement demonstrates variations among subjects that are required to be individually determined.

The present results were obtained with healthy subjects and their direct applicability to patients that are candidates for rehabilitation should be considered with caution. Whether such a different population demonstrates the same features in its metabolic and/or circulatory response to ECC cycling is an important issue that requires further attention. Nevertheless, independently of the type of population, the performance of an ECC IET enables the specific VO$_2$/HR
relationship to be individually established, owing to the
determination of target ECC training HR values that lead to
a desired VO₂.

CONCLUSION

Provided a given level of VO₂ is considered (higher than
1 L-min⁻¹), ECC exercises are characterized by signifi-
cantly elevated HR leading to higher Q but reduced
A-V̇O₂imp compared with CON exercises. Therefore, an
important application for ECC, as opposed to CON cycling,
is to consider higher exercise HR to reach comparable level
of oxygen uptake. Moreover, this greater circulatory re-
sponse demonstrates interindividual variability that can be
established from ECC IET to allow accurate determination
of the individual HR training target. Nonetheless, the pre-

cise mechanisms underlying the particular circulatory re-
sponse to ECC cycle exercise need to be explored further.
Additionally, it remains to be determined if such HR
correspondences depend on a subject’s fitness level, and
particularly if they can be applied for candidates for
rehabilitation. Also, whether these HR differences are
modified by training is an important issue that warrants
further investigations.

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REFERENCES

1. Abbott, B. C., B. Bigland, and J. M. Ritchie. The physiological
2. Achten, J., and A. E. Jeukendrup. Heart rate monitoring: appli-
3. Arts, F. J., and H. Kuijpers. The relation between power output,
4. Astrand, P. O., T. E. Cuddy, B. Saltin, and J. Stenberg. Cardiac
output during submaximal and maximal work. J. Appl. Physiol.
Monitoring high-intensity endurance exercise with heart rate and
impedance cardiograph device for the non-invasive evaluation
of cardiac output at rest and during exercise: comparison with the “direct” Fick method. Eur. J. Appl. Physiol. 82:313–320,
2000.
hindlimb muscle blood flow during level and downhill locomot-
method for measuring body surface area: a height-weight formula
11. Henniksson, J., H. G. Knuttgen, and F. Bonde-Petersen. Per-
ceived exertion during exercise with concentric and eccentric
Characteristics of the muscle mechanoreflex during quadriceps
and group III muscle afferents to thermal stimuli. Brain Res. 113:201–205,
1976.
14. Hesser, C. M., D. Linnarsson, and H. Bjurstedt. Cardiorespira-
tory and metabolic responses to positive, negative and minimum-
15. Hopkins, W. G., E. J. Schabert, and J. A. Hawley. Reliability of
and heart rate responses to exercise performed with concentric and
Exerc. 34 (Suppl. 1):6, 2002.
Lindstedt. Eccentric ergometry: increases in locomotor muscle
Lindstedt. Chronic eccentric exercise: improvements in muscle
strength can occur with little demand for oxygen. Am. J. Physiol.
of muscle tension variations and energy absorption on oxygen
consumption, heart rate, and cardiac output during negative work.
22. Lindstedt, S. L., P. C. Lastayo, and T. E. Reich. When active
muscles lengthen: properties and consequences of eccentric con-
coronary patients: central hemodynamic and metabolic responses.
Comparison of oxygen uptake kinetics during concentric and eccen-
invasive cardiac output evaluation during a maximal progressive
Eccentric exercise training in patients with chronic obstructive pul-
28. Steiner, R., K. Meyer, K. Lippsner, J. P. Schim11, H. Saner, and
H. Hoppe11er. Eccentric endurance training in subjects with coro-
nary artery disease: a novel exercise paradigm in cardiac rehabili-
29. Thomson, D. A. Cardiac output during positive and negative work.
George, and A. G. Fish11er. Stroke volume does not plateau during