Oxygen Uptake, Carbon Dioxide Production, Minute Ventilation and Heart Rate during Post Exercise Recovery in Healthy and Unmedicated Elderly and Young Men

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Abstract

This study investigated the influence of aging and effort intensity upon oxygen uptake (\(\dot{VO}_2\)), carbon-dioxide production (\(\dot{VCO}_2\)), minute ventilation (\(\dot{V}_E\)) and Heart Rate (HR) during postexercise recovery. Young (YG: n = 16; 24 ± 2 yr) and older (OG: n = 18; 63 ± 1 yr) groups performed cycle ergometer exercise bouts at 100%, 75%, and 40% of maximal work rate, preceded by 30-min resting assessment. Postexercise data were acquired during 15-min passive recovery. In all cases, postexercise recovery was best described by subtraction of exercise data were acquired during 15-min passive recovery. In all cases, postexercise recovery was best described by two vs. single exponential equation (\(\int_0^\infty x(t)dt = A/\alpha + B/\beta\)), where \(A/\alpha\) and \(B/\beta\) constants designating fast and slow components, respectively. On average, the relative contribution of \(A/\alpha\) was similar (P > 0.05) across age groups and work rates (YG vs. OG: \(\dot{VO}_2\) - 42% vs. 45%; \(\dot{VCO}_2\) - 38% vs. 39%; \(\dot{V}_E\) - 26% vs. 27%; HR - 15% vs. 16%). The relative contribution of \(B/\beta\) was similar (P > 0.05) between age groups at low work rate (P > 0.05) and increased with exercise intensity (P < 0.05) for \(\dot{VO}_2\) (YG vs. OG: Ex100% - 69% vs. 69%; Ex75% - 56% vs. 50%; Ex40% - 48% vs. 47%) and HR (YG vs. OG: Ex100% - 93% vs. 92%; Ex40% - 69% vs. 63%), but not \(\dot{VCO}_2\) and \(\dot{V}_E\). Total recovery was similar in OG and YG for all variables (P > 0.05). However, recovery curves of \(\dot{VCO}_2\), \(\dot{V}_E\) and HR lasted longer in OG than YG (P < 0.05). In conclusion, aging did not affect total postexercise recovery or relative contribution of fast or slow components. Exercise intensity but not age prolonged \(\dot{VO}_2\) recovery, while slow components of \(\dot{VCO}_2\) and \(\dot{V}_E\) were longer in OG. HR recovery increased with age and was markedly longer than respiratory variables, particularly among older individuals.

Keywords

Cardiopulmonary exercise testing, Aging, Aerobic exercise, EPOC, Exercise physiology

Introduction

Cardiorespiratory responses during exercise have been extensively described in populations with different demographic and clinical characteristics [1-5], while fewer studies focused on postexercise recovery [6]. However, more recently this “window”, defined as the time between the end of a bout of exercise and the subsequent return to what is considered a “resting” state, has been considered as a discrete phenomenon that should be given attention as an independent and valuable source of information. Indeed, the analysis of recovery from exercise may provide insights that could help optimize practical applications in terms of exercise recommendations for health and performance [7]. For instance, aerobic training prescription and control are sometimes based upon the relationship between exercise intensity and recovery duration [8], particularly within athletic fatigue/performance [9]. Furthermore, postexercise adjustments relate with homeostatic ability and health [7].

Undoubtedly, the most studied variable during postexercise recovery is the oxygen uptake (\(\dot{VO}_2\))
[8,10-13]. In the early 1920s, Hill, et al. [14,15] originally demonstrated that $\dot{V}O_2$ recovery from intense exercise involved fast and slow phases. Subsequently, Margaria, et al. [16] and Henry [17,18] proposed that components of $\dot{V}O_2$ recovery could be described by the sum of two exponential components as in the equation $X(t) = Ae^{-at} + Be^{-bt}$. The first component corresponded to aerobic replenishment of ATP and PCr stores and the second reflected lactate recovery [19]. Additional research quantified those components [20,21] and demonstrated that this mathematical model could be also applied to recovery curves of carbon dioxide production ($\dot{V}CO_2$), ventilation minute ($\dot{V}_E$), and heart rate (HR) [22,23].

Research in regard to postexercise recovery has mostly investigated young or middle-age adults [24]. However, to extend these observations to older individuals is important, since aging has long been associated with an attenuation of aerobic capacity and adrenergic stimulation during exercise [5,25,26]. For this reason, the effects of aging upon exercise performance have been compared to those of adrenergic blocking administered to young subjects [22,27]. An equivalent impact on the time and/or capacity to bring cardiorespiratory variables to resting levels would be certainly related to lower ability to recover and resume physical work, with obvious consequences to the overall work capacity in the elderly. Furthermore, limitations pertaining physical status and clinical conditions frequently preclude comparisons between healthy older and younger groups. This would be nonetheless useful, since information about expected values may help establishing goals and provide parameters to evaluate the impact of training upon cardiorespiratory markers throughout aging.

One of the few studies examining age-related effects upon respiratory recovery from different exercise intensities demonstrated that older subjects had reduced upward drift of gas exchange variables and HR during continuous submaximal exercise [22]. Overall, the time of $\dot{V}O_2$, $\dot{V}CO_2$, and $\dot{V}_E$ to return to resting levels increased with aging. However, those data referred to a relatively small sample of eight participants aged 60–72 yrs., which performed only 10 minutes of constant-load exercise consisting of pedaling at 70% of maximal work rate. Evidently, this is not enough to establish a universal recovery pattern, considering larger samples and different exercise intensities. A previous study by our group assessed the cardiorespiratory responses following exercise bouts performed at 40%, 75%, and 100% of peak $\dot{V}O_2$ in 10 young and 10 older individuals [28]. Data for $\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}_E$, and HR exhibited better adjustment to models with two vs. one exponential during 15-min recovery. Moreover, the influence of exercise intensity upon both faster and slower recovery was greater in older than young individuals. However, limitations as the small sample and lack of control of used medications warranted a replication of the protocol, to ratify those preliminary findings.

A better understanding about cardiorespiratory recovery in older vs. young unmedicated individuals would be relevant to provide parameters for exercise prescription and health evaluation in this specific population. Given the lack of data on this matter, the present study compared the $\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}_E$, and HR during postexercise recovery from aerobic exercise performed with different intensities in healthy and unmedicated older and young men. Due to expected age-related changes in central and peripheral components determining aerobic capacity, we hypothesized that healthy older individuals would have reduced $\dot{V}O_2$ drift and slower recovery in comparison with younger individuals with similar clinical status and relative fitness, affecting the kinetics of $\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}_E$, and HR return to resting levels. However, total recovery was expected to be similar across age groups.

Methods

Subjects

After advertisement, thirty-six healthy males volunteered for the study and were assigned into young (YG) ($n = 16$; age = $25 \pm 2$ yrs.; $1.8 \pm 0.5$ m; $77 \pm 6$ kg) and older (OG) ($n = 20$; age = $64 \pm 1$ yrs.; $1.7 \pm 0.2$ m; $71 \pm 8$ kg) groups. Subjects dwelled in community, exhibited normal weight and were moderately active, practicing at least 3 hours per week of light activities, as walking or jogging. Participants were non-smokers and unmedicated for any type of cardiovascular disorder (hypertension, cardiac disease etc). Additional exclusion criteria were blood pressure abnormalities, heart disease, pulmonary limitation, locomotion impairment, or other condition that could preclude exercise performance or influence cardiorespiratory responses during and after exercise. Health information was extracted from patients’ medical records with the agreement of their primary physicians. All participants provided informed consent. This experiment is part of a broader project, which gained approval from the institutional ethical committee of the University of Rio de Janeiro State (CAAE 38263114.7.0000.5259).

Procedures

Participants performed a cycle ergometer maximal Cardiopulmonary Exercise Testing (CPET) designed to elicit individual volitional exhaustion [27]. The test protocol included 3 min warming-up at 5 W, which was followed by incremental exercise beginning at 25 W with similar work rate being added every min. Test interruption followed recommendations of the American College of Sports Medicine [29]. The CPET was considered as maximal if at least two of the following criteria were observed [30]: Score 10 on Borg CR-10 scale; 90% predicted $HR_{max}$ [220 - age] or heart rate plateau ($\Delta HR$ between two consecutive work rates $\leq 4$ beats·min⁻¹); $\dot{V}O_2$ plateau ($\Delta \dot{V}O_2$ between two consecutive work rates $< 2.1$ mL·kg⁻¹·min⁻¹); respiratory exchange ratio (RER) $> 1.10$.
Table 1: Cardiorespiratory data at rest and maximal exercise in young and older groups (n = 36).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Young group (n=16)</th>
<th>Older group (n=20)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Ex100%</td>
<td>Ex40%</td>
</tr>
<tr>
<td></td>
<td>Ex75%</td>
<td>Ex100%</td>
</tr>
<tr>
<td></td>
<td>Ex40%</td>
<td>Ex75%</td>
</tr>
<tr>
<td>VO₂ (mL.kg⁻¹.min⁻¹)</td>
<td>4 ± 1</td>
<td>24 ± 6*</td>
</tr>
<tr>
<td>VCO₂ (mL.kg⁻¹.min⁻¹)</td>
<td>5 ± 1</td>
<td>32 ± 4*</td>
</tr>
<tr>
<td>V̇E (mL.kg⁻¹.min⁻¹)</td>
<td>151 ± 26</td>
<td>728 ± 86*</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>73 ± 7</td>
<td>128 ± 6*</td>
</tr>
<tr>
<td>Work rate (watts)</td>
<td>0</td>
<td>88 ± 10*</td>
</tr>
</tbody>
</table>

*: Significantly different than young group (P < 0.05); †: Significantly different than previous work rate (P < 0.05)

Table 2: Mean deviations of recovery curves from mathematical models with one or two exponents (n = 36).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Exercise intensity</th>
<th>Young group (n = 16)</th>
<th>Older group (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>x(t) = A e⁻αt</td>
<td>x(t) = A e⁻αt + B e⁻βt</td>
<td></td>
</tr>
<tr>
<td></td>
<td>YG (n = 16)</td>
<td>OG (n = 20)</td>
<td>YG (n = 16)</td>
</tr>
<tr>
<td>VO₂ (mL.kg⁻¹.min⁻¹)</td>
<td>100%</td>
<td>0.21</td>
<td>0.08</td>
</tr>
<tr>
<td></td>
<td>75%</td>
<td>0.13</td>
<td>0.57</td>
</tr>
<tr>
<td></td>
<td>40%</td>
<td>0.07</td>
<td>0.36</td>
</tr>
<tr>
<td></td>
<td>100%</td>
<td>0.04</td>
<td>0.43</td>
</tr>
<tr>
<td>VCO₂ (mL.kg⁻¹.min⁻¹)</td>
<td>75%</td>
<td>0.11</td>
<td>0.46</td>
</tr>
<tr>
<td></td>
<td>40%</td>
<td>0.08</td>
<td>0.77</td>
</tr>
<tr>
<td></td>
<td>100%</td>
<td>0.04</td>
<td>0.34</td>
</tr>
<tr>
<td>V̇E (mL.kg⁻¹.min⁻¹)</td>
<td>75%</td>
<td>0.47</td>
<td>0.58</td>
</tr>
<tr>
<td></td>
<td>40%</td>
<td>0.26</td>
<td>12.13</td>
</tr>
<tr>
<td></td>
<td>100%</td>
<td>0.10</td>
<td>9.56</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>75%</td>
<td>0.55</td>
<td>19.11</td>
</tr>
<tr>
<td></td>
<td>40%</td>
<td>0.43</td>
<td>12.81</td>
</tr>
<tr>
<td></td>
<td>100%</td>
<td>0.30</td>
<td>21.47</td>
</tr>
</tbody>
</table>

*: Significantly different vs. single-exponent model.

Subsequently, participants underwent two submaximal exercise bouts with intensity corresponding to 40% and 75% of maximal work rate, with durations of 25 min and 15 min, respectively. Submaximal bouts were interspersed with 48-72 h intervals, in a random counterbalanced order. In brief, three exercise intensities were compared: Ex100%, Ex75%, and Ex40%. Each exercise bout was preceded by 30-min baseline assessment to determine VO₂, VCO₂, V̇E, and HR at rest. Assessment of 15-min recovery was initiated immediately after the end of exercise bouts (CPET and submaximal bouts) with participants comfortably seated at the lab. A bipolar chest lead (V5) ECG was used to record HR, while ventilatory responses (VO₂, VCO₂, and V̇E) were assessed via metabolic cart (Morgan Scientific™, Haverhill, MA, USA). Ventilatory responses were averaged and recorded every 30 s. The 30 s time average provided a good compromise between removing noise from VO₂ data while maintaining the underlying trend [31]. In all cases, the ambient temperature ranged within 21-23 °C and relative humidity within 55-70%.

Statistical analysis

The Kolmogorov-Smirnov test ratified data normality, and results are expressed as mean ± standard deviation. Potential differences of cardiorespiratory responses between age groups and exercise intensities were tested by 2-way ANOVA with repeated measures, followed by Fisher LSD post hoc verification in the event of significant F ratios. The recovery pattern of each variable was tested regarding their adjustment to models including one or two exponentials. To determine deviations, the equation D = [∑d_i^2/(N_p-N_a)]^1/2 was used, where d_i: deviation from the first point, N_p: number of points; and N_a: number of adjusted constants. Values of each component of the equation were also determined. Finally, constants for components describing fast and slower recovery from exercise situations were extracted for each variable. Since total recovery of a given variable might be represented by the area under the experimental curve, the integer of two exponential components was individually calculated by the equation [S = ∫₀∞x(t)dt = A/α + B/β]. Constants obtained for exercise intensities within age groups were also compared by means of 2-way ANOVA followed by Fisher LSD verification. In all cases, calculations were made using the Statistica 7.0 software (Statsoft™, Tulsa, OK, USA) and significance level was fixed at P ≤ 0.05.

Results

Table 1 presents data obtained at rest and during exercise protocols. None of the young or older subjects showed significant ST segment depression or developed chest discomfort during the CPET or submaximal exercise...
bouts. No difference between age groups was detected at rest for any of the observed variables, but cardiorespiratory data during Ex75% and Ex100% were always lower in OG than YG (P < 0.05). Adjustments calculated for the recovery curves are depicted in Table 2. Deviations were systematically greater when applying models with two exponents. Therefore, further analyses describing postexercise recovery were performed using the following polynomial equation: x(t) = Ae-αt + Be-βt, where A: value of fast component at time zero; B: value of slow component at time zero; α: recovery rate related to the first exponential; β: recovery rate related to the second exponential; e: neperian log basis; t: time.

Table 3 presents components and constants calculated for exercise intensities within age groups. No significant differences between age groups and exercise intensities occurred for the fast component of VO2 and VCO2. In fact, the duration of this component, as estimated from 1/α, was of approximately 1 min in all experimental situations. The slow component (β) of VO2 recovery was consistently influenced by exercise intensity, but not VCO2. No age-related differences for the slow component of VO2 were detected, despite a tendency of this phase of recovery to be longer among the older individuals. However, after exercise performed with higher intensities, the slow component of VCO2 was significantly longer following Ex100% and Ex75% in OG than YG.

Similarly, the fast component of VE recovery was not different across age groups. Although the estimated duration of 1/α had also fell around 1 min in all situations, this constant tended to increase with exercise intensity in OG. Exercise intensity also influenced the slow component of VE recovery, which tended to be longer in OG vs. YG. However, due to data dispersion, in YG we could not estimate its duration after Ex40%. In contrast to respiratory variables, fast and slow components of HR recovery increased with exercise intensity, particularly in YG. Moreover, the duration of slow components of HR recovery were systematically longer in OG than YG. This finding suggests that HR recovery was somewhat dissociated of the return of respiratory variables to resting values, especially among older individuals. Noteworthy, also at

| Table 3: Components A, B, α, and β associated with recovery curves after different exercise intensities (Ae-αt + Be-βt). |
|---|---|---|---|---|
| Intensity | VO2 (mL·kg⁻¹·min⁻¹) | VCO2 (mL·kg⁻¹·min⁻¹) | VE (mL·kg⁻¹·min⁻¹) | HR (bpm) |
| 100% | A = 44.19 ± 0.68 | A = 50.02 ± 1.27 | A = 1241.80 ± 27.26 | A = 80.00 ± 1.00 |
| | B = 3.18 ± 0.39 | B = 11.94 ± 1.23 | B = 331.54 ± 18.54 | B = 29.00 ± 2.00 |
| 75% | A = 31.07 ± 0.71 | A = 25.30 ± 1.00 | A = 709.21 ± 19.03 | A = 58.00 ± 1.00 |
| | B = 2.52 ± 0.65 | B = 7.07 ± 1.05 | B = 175.06 ± 16.94 | B = 30.00 ± 2.00 |
| 40% | A = 18.37 ± 0.38 | A = 16.71 ± 0.59 | A = 392.12 ± 14.10 | A = 33.00 ± 2.00 |
| | B = 0.56 ± 0.19 | B = 1.73 ± 0.75 | B = 24.39 ± 6.05 | B = 11.00 ± 1.00 |

Table 2: Components A, B, α, and β associated with recovery curves after different exercise intensities (Ae-αt + Be-βt).
Ex40% the high dispersion of 1/β precluded the estimation of this component of HR recovery in OG.

Table 4 depicts data of areas under the recovery curves, which produced an estimation of total recovery and relative contribution of fast (A/α) and slow (B/β) components in each exercise situation. As expected, total recovery increased with exercise intensity. However, there was no difference between YG and OG for the total recovery of any variable within the different exercise bouts. The relative contribution of slow components for the total recovery of \( \dot{V}O_2 \), but not \( \dot{V}CO_2 \) and \( \dot{V}_E \), seemed to increase with exercise intensity, irrespective of the age group. In regard to HR, in both age groups the slow component accounted for almost all recovery after Ex100% and Ex75% (~90%). However, after Ex40% the relative contribution of recovery components to HR normalization was closer to that observed for the respiratory variables (30% vs. 70%, respectively).

**Discussion**

The present study described and compared cardiopulmonary recovery curves after different exercise intensities in healthy and unmedicated young and older individuals. As expected, irrespective of age the recovery curves for \( \dot{V}O_2 \), \( \dot{V}CO_2 \), \( \dot{V}_E \) and HR were better described by a model including two exponents rather than one (fast and slow components, respectively). Age seemed not to be a major determinant of \( \dot{V}O_2 \) and \( \dot{V}CO_2 \) recovery, and exercise intensity influenced the slow, but not the fast component. The fast component lasted approximately 1 min for \( \dot{V}O_2 \), \( \dot{V}CO_2 \), and \( \dot{V}_E \), regardless the age group or exercise intensity. Both fast and slow components of HR recovery were always longer among the older individuals, significantly increasing with exercise intensity. Incidentally, postexercise recovery was longer for HR than any other variable, particularly in OG.

As mentioned above, fast components of \( \dot{V}O_2 \) and \( \dot{V}CO_2 \) recovery lasted approximately 1 min and were similar between exercise intensities and age groups (Table 2 and Table 3). It is therefore feasible to think that a common constant for this component exists, which would be independent of exercise intensity or aging. On the other hand, the slow component of \( \dot{V}O_2 \) recovery systematically increased with exercise intensity (Table 3). Interestingly, this was not observed for \( \dot{V}CO_2 \), particularly in YG. These data suggest that exercise intensity might influence the relative contribution of slow recovery to reestablish resting gas exchange values and that this would be more evident for \( \dot{V}O_2 \) than \( \dot{V}CO_2 \).
In fact, there is a growing body of evidence showing that VO₂ recovery, for example, increases exponentially as a function of exercise intensity, whereas linearly as a function of exercise duration [see 32 for a review]. Hagberg, et al. [33] investigated the effects of exercise intensity and duration on the time course and magnitude of VO₂ recovery following short (5 min) and prolonged (20 min) bouts of cycling at 50%, 65% and 80% VO₂peak in eighteen healthy men (aged 20-33 yr). The magnitude of the fast component of VO₂ recovery relied on the exercise intensity, irrespective of its duration (5 vs. 20 min). In which concerned the slow component of VO₂ recovery, either duration or intensity of exercise bouts have not affected the magnitude of those responses at 50% or 65% VO₂peak. However, at 80% VO₂peak the magnitude of the slow component of VO₂ recovery was nearly 5 times greater (P < 0.01) after the 20 min vs. 5 min exercise bout. In other words, the time course of the slow component of VO₂ recovery seemed to increase in exercise bouts with higher intensities (≥ 65% VO₂peak) and longer durations (i.e. ≥ 20 min).

Our data partially agree with these findings [33]; for example, the fast component (A/α) was indeed similar across all exercise intensities and age groups, but the slow component (B/β) was significantly longer after exercise performed with higher intensities (Table 3). Thus, age seemed to be a secondary factor to determine this phase of VO₂ recovery. This finding contradicts the premise that there would be an age-related effect upon the slow component of recovery, as previously suggested [22]. Although aerobic power declines with aging [5] our data suggest that the contribution of the different components of total recovery would be proportional to the relative exercise intensity, and not to maximal cardiorespiratory capacity. Noteworthy, these findings also disagree with a prior study by our group, suggesting that the influence of exercise intensity upon the slow component of VO₂ and VCO₂ recoveries would be greater in older than young individuals [28].

In the present study, total CO₂ production was similar in OG and YG at all given exercise intensities, as well the relative contribution of both components for the overall recovery (Table 4). However, the slow component was longer in OG than YG at Ex100% and Ex75%, suggesting a less effective CO₂ buffering in comparison with YG (Table 3). Concisely, elderly and young adults seem to produce similar amounts of CO₂ during equivalent exercise. Nevertheless, older individuals probably need more time to bring VCO₂ to resting levels. These observations corroborate findings of a prior research [34], claiming that VCO₂ recovery would be longer in older than young adults. However, this same study suggested that VCO₂ recovery curve would be closely related to - this premise has been not confirmed by our data, since the kinetics of VCO₂ recovery appeared to be shorter and somewhat dissociated of VO₂. In this case, it could be argued that CO₂ influence upon central receptors might not be the sole determinant of VO₂ during post exercise recovery. Alternative pathways could be also implicated, as cortical command or afferent reflex from exercised skeletal muscles [35]. Evidently, additional investigations are warranted to confirm this possibility.

In both age groups, the slow component accounted for most of VO₂ recovery (approximately 70-80% of total) (Table 4). Moreover, exercise intensity seemed to influence both fast and slow components of VO₂ recovery, at least in OG (Table 3). These findings are consistent with data observed for VCO₂. Since VCO₂ recovery was in OG than YG, VO₂ was also expected to remain above resting values for a longer period, which was confirmed by 1/B values. Some factors could be at the origin of this difference between age groups, as slower aerobic power, less efficient respiratory buffering, and poorer thermoregulation among the elderly [22]. Another potential explanation for the delayed recovery of gas exchange variables following vigorous exercise among older individuals would be an impaired elimination of CO₂ due to reduced CO₂ chemosensitivity [36]. It has been shown that the rate of elimination of CO₂ after an acute increase in body CO₂ stores induced by rebreathing, would relate with the slope of ventilatory responses to CO₂ but not with the magnitude of hypercapnia [37]. Therefore, a diminished CO₂ chemosensitivity could help to explain age-related delays in VO₂, VCO₂ and VO₂ recovery patterns.

Evidences concerning HR recovery are mixed. Williams & Horvath [6] have proposed that after 30-40 min of moderate exercise, VO₂, VCO₂ and were observed for VO₂, VCO₂ and VO₂, VO₂, VO₂ and VO₂ in OG would return to resting values within 10 min of recovery, whereas HR might remain elevated for 30 min. On the contrary, Chick, et al. [22] described respiratory and cardiac responses following exercise performed at 70% maximal capacity, suggesting that 10 min of recovery would be enough to normalize VO₂, VCO₂ and HR in a small sample of older individuals. Our findings confirmed the idea that HR and respiratory recovery would somewhat dissociated. Although there was an evident influence of exercise intensity upon HR recovery (similarly to data observed for VO₂, VCO₂ and VCO₂), the slow component of HR recovery was systematically longer vs. all respiratory variables. Moreover, HR recovery was always longer in OG than YG (Table 3). This finding did not agree with our previous study, which suggested that the influence of exercise intensity on HR recovery would be more evident in YG than OG, due to the lower ability of older individuals to increase HR during maximal exercise [28]. On the other hand, there was no difference between age groups for the total HR recovery or relative contribution of fast and slow components (Table 4).

The fact that HR suffered the influence of other factors than solely VO₂ may help explaining its longer recovery in comparison with respiratory variables - for
instance, differences in lactate metabolism, body temperature, ventilatory rate, or circulating concentrations of catecholamines [38] may also account for a longer recovery HR recovery. This is an important finding for exercise prescription, since overall recovery from exercise bouts is often assessed by means of the return of HR to preexercise levels. This may correspond to the actual recovery of the respiratory variables in the fast recovery phase (e.g., first 2 min), but not during the slow phase, whose duration for HR may be up to three times that observed for respiratory variables, particularly among older individuals.

A potential limitation of the present study was that resting values of \( \dot{V}O_2 \), \( \dot{V}CO_2 \), \( \dot{V}_e \) and HR could not be assessed on a separate day (i.e. non-exercise control day), under identical experimental conditions applied in exercise days. Similar to previous studies, we have defined resting values as the average values of \( \dot{V}O_2 \), \( \dot{V}CO_2 \), \( \dot{V}_e \) and HR measured during the last 5 min of a 30-min resting period in a seated position. This strategy was criticized in a systematic review [39], since it can lead to falsely high resting values that compromise the accuracy of recovery rate assessment after exercise. Moreover, the participants were not well matched for their baseline fitness. Our polynomial equations were fit to model few data, which is always problematic. In short, it is difficult to determine linear regressions with, so few participants and the accuracy of recovery curves could be improved with more participants. However, to find older individuals completely unmedicated for cardiovascular features is not a simple task. Although acknowledging the limitation due to the relatively small sample, our findings were consistent enough to establish recovery patterns that probably correspond to the reality, as well as differences between age groups. Finally, the external validity of our findings is restricted to healthy and unmedicated individuals. Although this information is important to address physiological differences between age groups, we must acknowledge that this is frequently not the case of older individuals.

**Conclusions**

In conclusion, total recovery of gas exchange variables and HR after exercise bouts performed with similar relative intensity did not differ between young and older healthy and unmedicated individuals, as well as the relative contribution of fast and slow recovery components. Exercise intensity but not age, increased the duration of fast and slow components of \( \dot{V}O_2 \) recovery. However, the slow components of \( \dot{V}CO_2 \) and \( \dot{V}_e \) tended to be longer in older than young individuals, perhaps reflecting an age-related reduction in buffering capacity. The duration of HR recovery was longer than respiratory variables, increasing with exercise intensity and age. This is suggestive of dissociation between HR and respiratory recoveries, particularly in older individuals.

In practical terms, total recovery and kinetics of \( \dot{V}O_2 \), \( \dot{V}CO_2 \), \( \dot{V}_e \) and HR were similar across age groups, following exercise bouts of equivalent intensity. However, since the slow component of \( \dot{V}CO_2 \), \( \dot{V}_e \) and HR were prolonged in older participants, full recovery might take longer in this group, particularly after vigorous exercise. Lastly, HR recovery may not reflect the return of \( \dot{V}O_2 \), \( \dot{V}CO_2 \) or \( \dot{V}_e \) to pre-exercise levels, particularly in older individuals.

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**Ethical Statement**

This study gained approval from institutional ethics committee (CAAE 38263114.7.0000.5259) and complied with ethical standards laid down in the Declaration of Helsinki. The authors do not have conflicts of interest to declare.

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