Postexercise heart rate recovery in children: relationship with power output, blood pH, and lactate

Martin Buchheit, Pascale Duché, Paul B. Laursen, and Sébastien Ratel

Abstract: The aim of the present study was to determine whether differences in age-related heart rate recovery (HRR) kinetics were associated with differences in power output, blood lactate concentration ($[La]_b$), and acidosis among children, adolescents, and adults. Ten prepubertal boys (aged 9.6 ± 0.7 years), 6 pubertal boys (aged 15.2 ± 0.8 years), and 7 men (aged 20.4 ± 1.0 years) performed 10 repeated 10-s all-out cycling sprints, interspersed with 5-min passive recovery intervals. Mean power output (MPO) was measured during each sprint, and HRR, $[La]_b$, and acidosis (pH_b) were determined immediately after each sprint. Children displayed a shorter time constant of the primary component of HRR than adolescents and adults (17.5 ± 4.1 vs. 38.0 ± 5.3 and 36.9 ± 4.9 s, $p < 0.001$ for both), but no difference was observed between adolescents and adults ($p = 1.00$). MPO, $[La]_b$, and pH_b were also lower in children compared with the other 2 groups ($p < 0.001$ for both). When data were pooled, HRR was significantly correlated with MPO ($r = 0.48$, $p < 0.001$), $[La]_b$ ($r = 0.58$, $p < 0.001$), and pH_b ($r = –0.60$, $p < 0.001$). Covarying for MPO, $[La]_b$, or pH_b abolished the between-group differences in HRR ($p = 0.42$, $p = 0.19$, and $p = 0.16$, respectively). Anaerobic glycolytic contribution and power output explained a significant portion of the HRR variance following high-intensity intermittent exercise. The faster HRR kinetic observed in children appears to be related, at least in part, to their lower work rate and inherent lack of anaerobic metabolic capacity.

Key words: parasympathetic activity, intermittent exercise, repeated sprints, maturation, pH.

Résumé : Cette étude se propose de vérifier si les différences de cinétique de la récupération du rythme cardiaque selon l'âge (« HRR ») sont associées aux différences de production de puissance, de concentration de lactate sanguin et d'acidose chez des enfants, des adolescents et des adultes. Dix garçons prépubères (âgé 9,6 ± 0,7 ans), 6 garçons pubères (âgé 15,2 ± 0,8 ans) et 7 hommes (âgé 20,4 ± 1,0 ans) participent à des séances de sprint répété à plein régime sur un vélo, d'une durée de 10 s et entrecoupé de périodes de repos passif d'une durée de 5 min. À chaque sprint, on évalue la production moyenne de puissance (« MPO ») et, après chaque sprint, on évalue l'HRR, la concentration sanguine de lactate ($[La]_b$) et le degré sanguin d'acidose (pH_b). Chez les enfants, on observe une plus petite constante de temps de la composante primaire de HRR que chez les adolescents et les adultes (17,5 ± 4,1 comparativement à 38,0 ± 5,3 et à 36,9 ± 4,9 s, $p < 0{,}001$ pour les deux), mais on n'observe pas de différences entre les adolescents et les adultes ($p = 1{,}00$). Comparativement aux 2 autres groupes, les valeurs de MPO, $[La]_b$ et de pH_b sont aussi plus faibles chez les enfants ($p < 0{,}001$ pour les deux). Après regroupement des données, on observe une corrélation significative entre la MPO ($r = 0{,}48$, $p < 0{,}001$), la $[La]_b$ ($r = 0{,}58$, $p < 0{,}001$) et le pH_b ($r = –0{,}60$, $p < 0{,}001$). La covariation des valeurs de MPO, de $[La]_b$ et de pH_b abolit les différences de HRR entre les groupes ($p = 0{,}42$, $p = 0{,}19$ et $0{,}16$, respectivement). La contribution de la glycolyse anaérobie et la production de puissance expliquent une part importante de la variance de HRR à la suite de l'effort intermittent de haute intensité. La cinétique plus rapide de récupération du rythme cardiaque observée chez les enfants est probablement le résultat, du moins en partie, de leur plus faible régime de travail et de leur déficience inhérente en matière de capacité anaérobie du métabolisme.

Mots-clés : activité parasympathique, exercice physique intermittent, sprints répétés, maturation, pH.

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Introduction

After a set bout of exercise, the recovery of physiological functions, including heart rate (HR), ventilation, oxygen uptake, and carbon dioxide production, tends to be faster in children compared with adults (for review, see Ratel et al.

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2006). For example, HR recovery (HRR) in children, as assessed by the time to recover 50% of its initial value, has been reported as being about 2-fold faster than adult values after cycling all-out for 30 s (Hebestreit et al. 1993) or for 1 min at the power output associated with maximal oxygen uptake ($\dot{V}O_{2\ peak}$) (Baraldi et al. 1991). Nevertheless, despite growing interest in mechanisms governing the recovery process in children, it is unclear why children recover HR faster than their adult counterparts (Ratel et al. 2006).

HR deceleration following exercise is controlled by complex interactions between neural and humoral factors (Buchheit et al. 2007*b*). Although probably the strongest determinant of HRR is parasympathetic reactivation (Imai et al. 1994; Buchheit et al. 2007*b*), the progressive withdrawal of sympathetic nerve activity is also important (Savin et al. 1982; Kannankeril et al. 2004). Together with the neural sympathoexcitation inherent to exercise (Franke et al. 2000), system stress metabolite accumulation during postexercise recovery (e.g., plasma epinephrine, lactate, proton, inorganic phosphate (Vissing 2000; Kaufman and Hayes 2002)) might promote sympathetic activity via the chemoreflex control of HR (Rowell and O'Leary 1990). This centrally generated cardiovascular control is modulated by chemosensitive and mechanosensitive afferent nerve fibres in the active muscle and by the mechanosensitive afferents within the carotid sinuses and aortic arch (Rowell and O'Leary 1990). It is not surprising, therefore, that significant relationships have been observed in adults between HRR and postexercise plasma epinephrine (Perini et al. 1989), blood lactate concentration ($[La]_b$) (Buchheit et al. 2007*a*), blood acidosis, and arterial oxygenation (Ba et al. 2009).

In children, using a multivariable linear regression model, age, gender, body mass index, and baseline HR were recently shown to be significant predictors of 1-min HRR, explaining 39% of HRR variance (Singh et al. 2001). In agreement with HRR determinants in adults, the faster HRR observed in children appears to be positively related to their high-functioning parasympathetic system (Ohuchi et al. 2000). It is also possible that the central command for sympathetic drive, thought to be related to muscle mass recruitment (Franke et al. 2000), is less activated in children, who have a significantly lower work rate than their older counterparts (Ratel et al. 2006). However, whether the autonomic sympathetic system is effectively less activated at the end of maximal exercise in children, or whether the inherently lower level of muscle byproducts (e.g., lactate and H^+ ions) that children experience after intense exercise explains their faster HRR, is still debated. Glycolytic activity, known to be maturity dependant, is generally lower in young children than in adults (Eriksson et al. 1973). This is based on lower muscle lactate concentrations measured after maximal exercise in 13.6-year-old-boys compared with young adults, and on significant positive relationships shown between testicular volume index and postexercise muscle lactate concentrations (Eriksson et al. 1973). Furthermore, at rest, a lower activity level of muscle phosphofructokinase has been shown in 11- to 13-year-old boys, compared with young adults (Eriksson et al. 1973). The clearance of lactate and H^+ ions within muscles might also be faster in children (Dotan et al. 2003; Ratel et al. 2006). This latter finding might, in turn, lead to lower accumulation of system stress metabolites and decreased muscle metaboreflex activation, thus evolving as a faster HRR in children (Baraldi et al. 1991). Conversely, other centrally located chemoreceptors (i.e., carotid) with a higher sensitivity to these metabolites compared with those of adults (Turley 2005) could potentially trigger the metaboreflex, resulting in a slower HRR. A similar rise in epinephrine levels in boys and men at maximal exercise has also been reported (Rowland and von Duvillard 1990). Because HRR was not measured in this study, the effective link among sympathetic activity, postexercise blood metabolite accumulation, and HRR is not clear. To the best of our knowledge, HRR in children has never been examined while also taking into account the power outputs and level of anaerobic system participation.

To determine whether HRR kinetics could be explained partly by differences in power output, blood metabolites, or acidosis, we examined postexercise HRR, power output, ($[La]_b$) accumulation, and blood pH (pH_b) in children, adolescents, and adults during repeated all-out cycling sprints. We expected that differences in HRR among the 3 age groups would be related to age-related differences in power output and blood-borne muscle byproducts. We hypothesized that the age- or pubertal-related differences in HRR would thus be weakened or eventually abolished when statistically accounting for sprint performance, pH_b, and lactate.

Materials and methods

Subjects

Based on the assumption that a 10 ± 5 beats·min^{-1} difference in $HRR_{60\ s}$ (i.e., absolute difference between the final HR at exercise end and the HR recorded 60 s later) is meaningful (Baraldi et al. 1991; Zafeiridis et al. 2005; Ba et al. 2009), we used Minitab 14.1 (Minitab Inc., Paris, France) to determine that a sample size of at least 6 participants per group would provide a statistical power of 0.8% at an α level of 0.05. To further increase the statistical power of the study, we eventually recruited 10 prepubescent boys, 6 pubescent boys, and 7 men for this study. The physical characteristics of the participants are presented in Table 1. All participants were involved in a variety of physical activities, including ice hockey and swimming. The study protocol was approved by the ethical committee of Auvergne University (Clermont-Ferrand, Auvergne, France), and written informed consent to participate in the study was signed by each participant or his or her parents prior to commencement.

Study overview

Each participant attended the laboratory for 2 sessions, separated by more than 48 h. The preliminary session was used to gather the participants' physical characteristics and for habituation with the testing procedures. The second session was dedicated to the experimental test.

Preliminary session

During the first visit, tricipital and subscapular skinfold thicknesses were obtained to estimate body fat percentage (Slaughter et al. 1988) and lean body mass (LBM). Pubescent stage was determined according to pubic hair and gonadal development (Tanner and Davies 1985). All participants performed a force–velocity test on a cycle ergometer (Ergo-

Table 1. Participant characteristics.

Variables	Children (n = 10)	Adolescents (n = 6)	Adults (n=7)
Age (y)	9.6±0.7	15.2±0.8*	20.4±1.0*,†
Tanner stage	I: 10	III: 3 and IV:3	V: 7
Stature (cm)	136.9±6.3	170.3±2.6*	181.3±4.5*,†
Body mass (kg)	31.9±5.4	59.5±3.7*	73.9±7.3*,†
LBM (kg)	27.2±3.9	53.7±4.0*	66.3±8.0*,†
Training loads ($h \cdot wk^{-1}$)	3.1±1.6	5.4±3.1	10.6±4.1*,†
PPO (W)	298±52	800±154*	1097±179*,†
$\dot{V}O_{2\ peak}$ ($L \cdot min^{-1}$)	1.57±0.33	3.42±0.44*	4.11±0.59*,†
$\dot{V}O_{2\ peak}$ ($mL \cdot min^{-1} \cdot kg^{-1}$)	49.4±7.2	57.5±6.8*	55.5±5.7
HR_{max} ($beats \cdot min^{-1}$)	197±11	189±8	191±10
$[La]_{b\ peak}$ ($mmol \cdot L^{-1}$)	6.6±1.7	11.6±2.3*	10.2±2.4*
RER	1.08±0.07	1.13±1.12	1.19±0.06*

Note: Values are means ± SD. Traits include anthropometric characteristics (lean body mass), peak power output, and cardiorespiratory parameters (maximal oxygen uptake, expressed in absolute and in relative terms; maximal heart rate; maximal blood lactate; respiratory exchange ratio) of participants in the 3 groups. LBM, lean body mass; PPO, peak power output; $\dot{V}O_{2\ peak}$, maximal oxygen uptake; HR_{max}, maximal heart rate; $[La]_{b\ peak}$, maximal blood lactate; RER, respiratory exchange ratio.

*Significant difference vs. children ($p < 0.05$).

†Significant difference vs. adolescents ($p < 0.05$).

meca Sorem, Toulon, France) to determine peak power output (PPO) and corresponding optimal values of force (Fopt) and velocity. The methodology of this test has been described in detail elsewhere (Doré et al. 2000). After at least a 30-min recovery period, $\dot{V}O_{2\ peak}$ was determined by direct methods (CPX Medical Graphics, Saint Paul, Minn.) using a graded cycling test. The first stage of the test lasted 3 min, and its initial power output was 30 W for children and 60 to 75 W for both adolescents and adults. Power output was then increased by 15 $W \cdot min^{-1}$ in children, by 30 W every 90 s in adolescents, and by 30 W every 2 min in adults. Pedaling rate was maintained at 60 $r \cdot min^{-1}$, and tests continued until volitional exhaustion. Tests were considered maximal when the participants' maximal HR (HR_{max}) was near their age-predicted HR_{max} (i.e., 220 – age ± 10 $beats \cdot min^{-1}$), when $[La]_b$ was higher than 5 $mmol \cdot L^{-1}$ in children (Armstrong et al. 1995) and 8 $mmol \cdot L^{-1}$ in adolescents and adults, and when the respiratory exchange ratio was greater than 1.10.

Exercise session

Participants performed a 6-min warm-up on a calibrated friction-loaded cycle ergometer (Ergomeca Sorem) at the power output that elicited HR to be 140 to 150, 130 to 140, and 120 to 130 $beats \cdot min^{-1}$ in children, adolescents, and adults, respectively. After a 5-min resting period, participants performed 10 repeated all-out 10-s cycling sprints, interspersed by 5-min passive recovery intervals (Ratel et al. 2006). Each sprint was performed against a friction load corresponding to 50% of the Fopt previously determined in the force–velocity test (Doré et al. 2000). Before each sprint, the starting position was standardized with the crank of the left leg 45° forward of top dead center. At the signal, participants were told to remain seated and to pedal as fast as possible. Each participant was encouraged verbally to give a maximal effort throughout the repeated 10-s sprints. During each postexercise recovery period, the subjects remained sitting on the cycle ergometer. The mean power output (MPO) was calculated over each sprint for each participant, and the percentage sprint decrement (%Dec) was determined as follows:

$$\%\text{Dec} = \frac{\text{mean power}}{\text{best power}} \times 100$$

(Glaister et al. 2004)

Measurements

Blood samples

Capillary arterialized blood samples (150 μL) were drawn from the earlobe at rest, before the 1st sprint, and after the 2nd, 4th, 6th, 8th, and 10th sprint, to determine the time course of H^+ ion concentration ($[H^+]$). These values were measured immediately after collection using a blood-gas analyzer (model IL Synthesis 1710, Instrumentation Laboratory). Additional capillary blood samples (10 μL) were collected at rest, before, and after the 1st sprint, and after the 3rd, 5th, 7th, 9th, and 10th sprint to measure the time course of $[La]_b$. Capillary tubes were frozen at –20 °C. $[La]_b$ values were measured by an Analox GM7 GB analyzer (Analox Instruments, London, UK) using L-lactate O_2 oxide reductase, which catalyzes the oxidation of L-lactate to pyruvate and hydrogen peroxide. Capillary blood samples were drawn within the first 1 min of recovery.

HR measurement and analysis

HR was measured continuously during the repeated test session using 12 electrodes and was visualized on a cardioscope (cardiovit CS-6/12, Schiller, Baar, Switzerland). HR data were then automatically averaged on a 1-s basis. Postexercise HRR was assessed following each sprint by (*i*) taking the $HRR_{60\ s}$, and (*ii*) taking the time constant of the HR

decay obtained by fitting the 5-min postexercise HRR into a first-order exponential decay curve (HRRτ) (Buchheit et al. 2007*a*, 2007*b*). Other polynomial regressions were rejected on the basis of increasingly higher residuals. Finally, HRR data after each sprint were computed for each subject, so that the total analyzed data set for either $HRR_{60\ s}$ or HRRτ was 10 × 10 for prepubescent boys + 6 × 10 for pubescent boys + 7 × 10 for men = 230. Both indices have previously shown good reliability: for $HRR_{60\ s}$, calculated values (test–retest) were *t* test $p = 0.56$, intraclass correlation coefficient (ICC) = 0.70, and SEM = 10.1 beats·min^{-1}; for HRRs, *t* test $p = 0.84$, ICC = 0.86, and SEM = 7.0 s (Buchheit et al. 2008).

Temperature measurement

Tympanic temperature, which has been shown to provide a valid and reliable estimate of rectal temperature (Pandey et al. 2006), was measured at rest and 1, 3, 5, and 10 min after the end of the last sprint using a tympanic thermometer (Kendall, GENIUS Model 3000A).

Statistical analysis

The distribution of each variable was examined with the Shapiro–Wilk normality test. Homogeneity of variance was verified by a Levene's test. When data were skewed or heteroscedastic (i.e., MPO), they were transformed by taking the natural logarithm to allow parametric statistical comparisons that assume a normal distribution. For the sake of clarity, however, values presented in the text are nontransformed. Changes in HRR, MPO, $[La]_b$, and pH_b values throughout the 10 repeated 10-s sprints in the 3 age groups were analyzed using a 2-way repeated-measures analysis of variance (ANOVA), with a within factor (i.e., sprint) and a between factor (i.e., age), the analysis being performed with or without MPO, $[La]_b$, or pH_b used as covariates to account for MPO, $[La]_b$, or pH_b effects on HRR distribution among the groups. If a significant interaction was identified, a Bonferroni's post hoc test was used to further delineate differences between age and (or) sprint. Linear regressions with Pearson's coefficients (95% CI) were used to establish relationships among age, Tanner stage, body size, LBM, HRR, MPO, $[La]_b$, and pH_b. Other polynomial regressions were rejected on the basis of increasingly higher residuals. Because $[La]_b$ or pH_b values were only measured every 2 sprints, correlations were performed on paired values (i.e., HRR after the 2nd, 4th, 6th, 8th and 10th sprint were plotted against the corresponding pH_b values, and HRR after the 1st, 3rd, 5th, 7th and 9th sprint were plotted against $[La]_b$ values). Semipartial correlations were used to adjust the relationship between HRR and age, Tanner stage, body size, and LBM on MPO, $[La]_b$, or pH_b. For all analyses, the level of significance was set at $p < 0.05$. All statistical analyses were carried out using Minitab 14.1 (Minitab Inc.). Data are presented as means ± SD, except for HRR data, which are expressed as means ± SE as a consequence of the statistical adjustments (i.e., least-squares means).

Results

Subjects' characteristics, training loads, and performances

Differences in participants' characteristics, training loads, and performances are illustrated in Table 1. The training load for children and adolescents was similar ($p = 0.31$) but was lower than that of adults (both $p < 0.01$). PPO and absolute $\dot{V}O_{2\ peak}$ values were different among the 3 groups, with adults having the highest values, followed by adolescents and then children (all $p < 0.001$). However, relative $\dot{V}O_{2\ peak}$ values were only different between children and adolescents ($p = 0.04$).

MPO profiles

An age effect was observed for MPO during the repeated-sprint exercise ($p < 0.001$) and MPO was different for each group (211 ± 37, 533 ± 64, and 702 ± 134 W for children, adolescents, and adults, respectively, all $p < 0.001$). There was, however, no difference shown between adolescents and adults when considering the relative values (8.9 ± 0.6 vs. 9.4 ± 0.2 W·kg^{-1} body mass, $p = 0.82$), but children presented with lower values (6.7 ± 0.8 W·kg^{-1} body mass, both $p < 0.001$). There was also no difference in %Dec among the 3 groups (5.6 ± 1.5%, 6.2 ± 4.5%, and 4.9 ± 1.4% for children, adolescents, and adults, respectively, $p = 0.64$). Neither a sprint effect ($p = 0.59$), nor an age × sprint interaction was detected ($p = 0.49$).

Tympanic temperature

There was neither an age ($p = 0.13$) nor a sprint ($p = 0.81$) effect on tympanic temperature, nor an interaction effect ($p = 0.13$) shown over the entire protocol. Mean values for children, adolescents, and adults were 36.5 ± 0.4, 36.6 ± 0.3, and 36.8 ± 0.6 °C, respectively.

HRR profiles

Figure 1 illustrates the mean HR profiles over the 10 repeated 10-s cycling sprints in the 3 age groups. Mean peak HR values reached over the 10 repeated sprints were higher in adolescents (82.2 ± 4.5% HR_{max}) compared with children and adults (77.8 ± 6.2% HR_{max} and 77.6 ± 6.1% HR_{max}, respectively, $p < 0.001$). In contrast, there was no difference between children and adults ($p = 0.96$). The ANOVA revealed a trend towards a sprint effect ($p = 0.06$), but without an age × sprint interaction ($p = 0.99$) for peak HR profiles during the 10 repeated sprints.

For $HRR_{60\ s}$, there was an age effect ($p < 0.001$). However, neither a sprint ($p = 0.09$) nor an age × sprint interaction was detected ($p = 0.99$). Children displayed faster $HRR_{60\ s}$ values than did adolescents and adults (50 ± 1 vs. 37 ± 1 and 39 ± 1 beats·min^{-1}, respectively, $p < 0.001$ for both). No difference was observed between adolescents and adults ($p = 0.61$).

Similarly, for HRRτ, there was an age effect ($p < 0.001$) without a sprint ($p = 0.99$) or age × sprint interaction ($p = 0.89$). As shown in Fig. 2 (left panel), children displayed faster HRRτ values compared with adolescents and adults ($p < 0.001$ for both); no difference was found between adolescents and adults for HRRτ ($p = 0.92$). Nonadjusted linear correlations between the 2 HR-derived HRR indices and

Fig. 1. Mean heart rate (HR) profiles during the 10 consecutive sprints and corresponding 5-min recovery periods in children, adolescents, and adults. For figure clarity, errors bars have not been added.

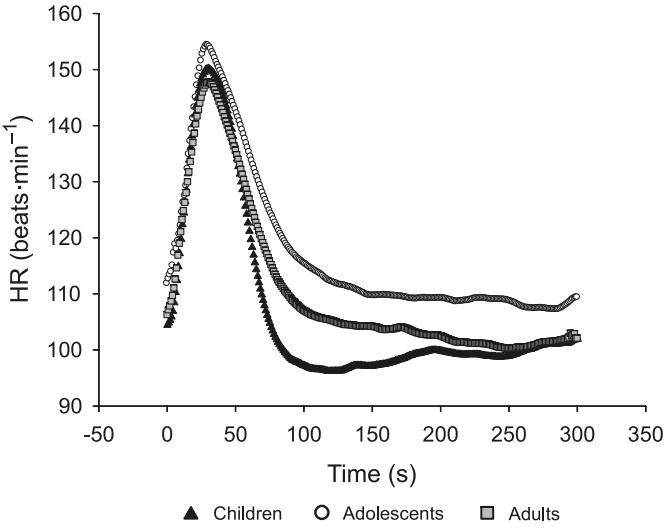
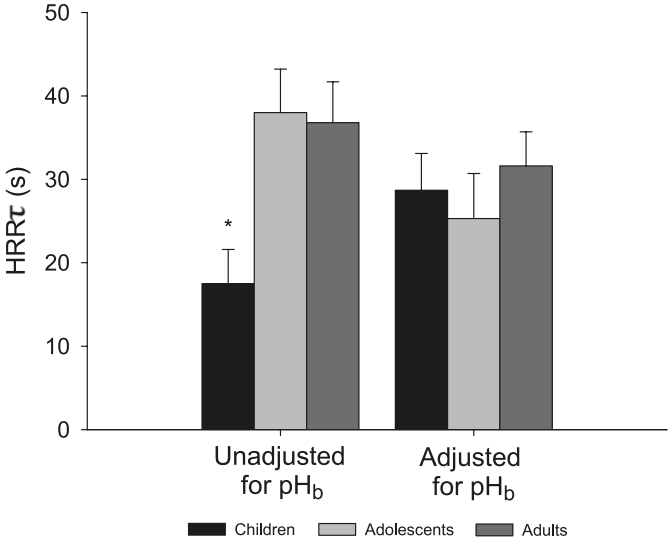


Fig. 2. Mean time constant of postsprint heart rate recovery (HRRτ) in children, adolescents, and adults before and after adjusting for mean postexercise blood pH (pH_b) (unadjusted and adjusted, respectively). Values are least-squares means ± SE. *, Significantly different from adolescents and adults ($p < 0.001$).



age, pubertal status, and anthropometric attributes were all moderate and significant (all $p < 0.001$) (Table 2).

$[La]_b$ and pH

Resting $[La]_b$ and pH_b values were 1.4 ± 0.3, 1.3 ± 0.4, and 1.5 ± 0.4 mmol·L^{-1} and 7.44 ± 0.02, 7.42 ± 0.01, and 7.41 ± 0.01, for children, adolescents, and adults, respectively. No between-group difference was noted for any of the resting blood variables (all $p > 0.09$). Post-warm-up $[La]_b$ and pH_b values were 1.4 ± 0.4, 1.7 ± 0.6, and 1.5 ± 0.3 mmol·L^{-1} and 7.43 ± 0.02, 7.42 ± 0.01, and 7.42 ± 0.01 mmol·L^{-1}, for children, adolescents, and adults, respectively. Similarly, no between-group differences were noted for any of the blood variables (all $p > 0.09$). For both $[La]_b$ and pH_b, there were age ($p < 0.001$ for both) and sprint ($p < 0.001$ and $p = 0.05$) effects, respectively. Nevertheless, there was no age × sprint interaction ($p = 0.10$ and 0.81 for $[La]_b$ and pH_b, respectively). Mean $[La]_b$ values throughout the 10 repeated 10-s sprints were lower in children (5.4 ± 1.9 mmol·L^{-1}) compared with the other 2 groups (7.9 ± 2.4 and 7.8 ± 20 mmol·L^{-1}, $p < 0.001$ for both), which were not different ($p = 1.00$). Mean pH_b values throughout the 10 sprints were higher in children (7.38 ± 0.02 mmol·L^{-1}) compared with the other 2 groups (7.31 ± 0.04 and 7.33 ± 0.01 mmol·L^{-1}, $p < 0.001$ for both). In contrast to the $[La]_b$ findings, there was a difference shown between adolescents and adults for pH_b ($p = 0.01$).

Relationships among HRR indices, MPO, pH_b, and $[La]_b$

Both $HRR_{60\ s}$ ($n = 230$; $r = –0.45$ (–0.58; –0.29); $p < 0.001$) and HRRτ ($n = 230$; $r = 0.48$ (0.37; 0.57); $p < 0.001$) were significantly correlated with relative MPO (W·kg^{-1} body mass). Except for sprint 1, HR-derived HRR indices were significantly related to $[La]_b$ and pH_b values ($p < 0.05$ for all). For example, the relationships shown between $HRR_{60\ s}$ and $[La]_b$ measured after sprints 3, 5, 7, 9, and 10 were moderate to strong ($n = 115$; $r = –0.67$ (–0.76; –0.55); $p < 0.001$) (Fig. 3*b*). Similarly, HRRτ showed moderate-to-strong correlations with $[La]_b$ ($n = 115$; $r = 0.58$ (0.69; 0.44); $p < 0.001$). There was also a positive relationship shown between $HRR_{60\ s}$ and pH_b values measured after sprints 2, 4, 6, 8, and 10 ($n = 115$; $r = 0.62$ (0.49; 0.72); $p < 0.001$) (Fig. 3*a*), as well as a negative correlation between the HRRτ and pH_b values ($n = 115$; $r = –0.60$ (–0.71; –0.47); $p < 0.001$). Finally, relative MPO (W·kg^{-1}) was correlated with $[La]_b$ ($n = 115$; $r = 0.44$ (0.28; 0.58); $p < 0.001$) and pH_b ($n = 115$; $r = –0.68$ (–0.77; –0.57); $p < 0.001$).

MPO contribution to the age-related differences in HRR

Covarying for relative MPO (W·kg^{-1}) abolished the between-group differences in $HRR_{60\ s}$ and HRRτ ($p = 0.21$ and $p = 0.42$, respectively). Least-squares means for $HRR_{60\ s}$ and HRRτ were 48 ± 4, 38 ± 4, and 41 ± 4 beats·min^{-1} and 20.4 ± 6.7, 36.3 ± 6.2, and 34.2 ± 7.0 s in children, adolescents, and adults, respectively. As presented in Table 2, relationships between HRR indices and age, maturational status, and anthropometrical attributes were no more significant when adjusting for relative MPO (all $p > 0.45$).

pH_b and $[La]_b$ contributions to the age-related differences in HRR

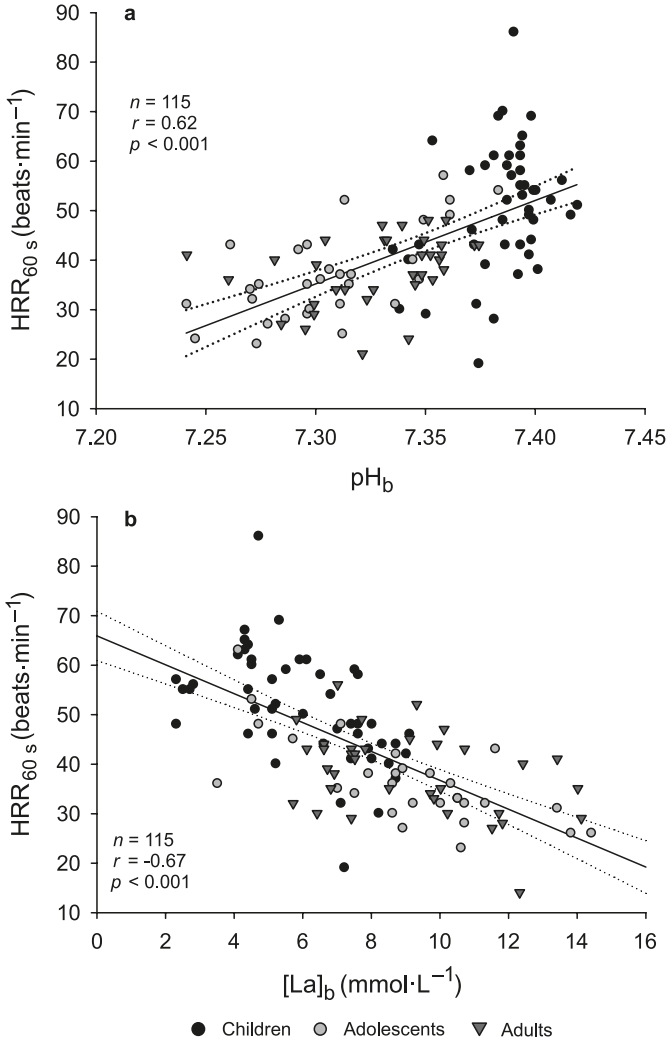
Covarying for $[La]_b$ abolished the between-group differences in $HRR_{60\ s}$ and HRRτ ($p = 0.18$ and $p = 0.19$, respectively). Least-squares means for $HRR_{60\ s}$ and HRRτ were 46 ± 2, 40 ± 2, and 42 ± 2 beats·min^{-1} and 23.5 ± 3.4, 33.1 ± 4.1, and 32.4 ± 3.8 s in children, adolescents, and adults, respectively. When adjusting for the effect of pH_b on HRR, the age effect on $HRR_{60\ s}$ was no more significant ($p = 0.29$), with $HRR_{60\ s}$ least-squares means of 45 ± 3, 42 ± 4, and 40 ± 3 beats·min^{-1} in children, adolescents, and adults, respectively. When adjusting HRRτ for pH_b, the same results were found ($p = 0.16$) (Fig. 2, right panel). As presented in Table 2, relationships between HRR indices and

Table 2. Relationships between HRR indices and age, maturation status, and anthropometrical attributes.

	$HRR_{60\ s}$				HRRτ				
Variables	Unadjusted	Adjusted for pH_b	Adjusted for $[La]_b$	Adjusted for MPO ($W{\cdot}kg^{-1}$)	Unadjusted	Adjusted for pH_b	Adjusted for $[La]_b$	Adjusted for MPO ($W{\cdot}kg^{-1}$)
Age	–0.43	–0.11	–0.20	–0.01	0.42	0.12	0.14	–0.01
	(–0.57 to –0.27)	(–0.29 to 0.07)	(–0.37 to –0.02)	(–0.14 to 0.12)	(0.26 to 0.56)	(–0.06 to 0.30)	(–0.04 to 0.32)	(–0.14 to 0.12)
	($p < 0.001$)	($p = 0.28$)	($p = 0.04$)	($p = 0.85$)	($p < 0.001$)	($p = 0.23$)	($p = 0.13$)	($p = 0.92$)
Tanner	–0.45	–0.12	–0.24	–0.04	0.46	0.14	0.22	0.05
	(–0.58 to –0.29)	(–0.30 to 0.06)	(–0.41 to 0.06)	(–0.17 to 0.06)	(0.30 to 0.59)	(–0.04 to 0.32)	(–0.04 to 0.39)	(–0.08 to 0.18)
	($p < 0.001$)	($p = 0.23$)	($p = 0.01$)	($p = 0.53$)	($p < 0.001$)	($p = 0.15$)	($p = 0.02$)	($p = 0.50$)
Body size	–0.47	–0.08	–0.22	–0.05	0.48	0.09	0.22	0.05
	(–0.60 to –0.31)	(–0.26 to 0.10)	(–0.39 to –0.04)	(–0.18 to 0.08)	(0.33 to 0.61)	(–0.09 to 0.27)	(–0.04 to 0.39)	(–0.08 to 0.18)
	($p < 0.001$)	($p = 0.43$)	($p = 0.02$)	($p = 0.45$)	($p < 0.001$)	($p = 0.33$)	($p = 0.02$)	($p = 0.44$)
LBM	–0.42	–0.07	–0.18	0.02	0.46	0.12	0.20	0.01
	(–0.56 to –0.26)	(–0.25 to 0.11)	(–0.35 to 0.00)	(–0.11 to 0.15)	(0.30 to 0.59)	(–0.06 to 0.30)	(–0.02 to 0.37)	(–0.12 to 0.14)
	($p < 0.001$)	($p = 0.50$)	($p = 0.06$)	($p = 0.80$)	($p < 0.001$)	($p = 0.20$)	($p = 0.03$)	($p = 0.91$)

Note: Correlation coefficients (95% CI) obtained from linear regression between heart rate recovery indices and age, pubertal status (Tanner), and anthropometric attributes (body size and lean body mass), with or without adjustments for blood lactate, pH, or mean power output. $HRR_{60\ s}$, absolute difference between the final heart rate at exercise end and the heart rate recorded 60 s later; HRRτ, mean time constant of postprint HRR; pH_b, blood pH; $[La]_b$, blood lactate concentration; MPO, mean power output; LBM, lean body mass.

Fig. 3. Heart rate recovery ($HRR_{60\ s}$) in children, adolescents, and adults as a function of blood pH (pH_b) (*a*) and blood lactate ($[La]_b$) (*b*) measured immediately after sprints 2, 4, 6, 8, and 10, and 3, 5, 7, 9, and 10 for pH_b and $[La]_b$, respectively.



age, maturational status, and anthropometrical attributes were no more significant when adjusting for pH_b (all $p > 0.15$). Removing the effects of $[La]_b$ reduced the level of variance in HRR explained by age, maturational status, and anthropometric attributes by a ratio of 0.5 (Table 2).

Discussion

The present study compared, for the first time, the post-exercise HRR response among children, adolescents, and adults, while also taking into account power output and the postexercise $[La]_b$ and acidosis. Our results confirm that children experience significantly faster HRR compared with their adolescent and adult counterparts. This apparent maturation-related difference in HRR was associated with lower values of relative cycling power output, $[La]_b$, and $[H^+]$ in children as compared with adolescents and adults. HRR was also well correlated with sprint performance and blood acidosis, and covarying for cycling power output, $[La]_b$, or pH abolished the between-group differences in HRR. These findings reveal that the maturity (or at least the

participation) of anaerobic metabolism, which likely influences both muscle power production and associated postexercise blood metabolite accumulation, may explain a significant portion of the variance in HRR during high-intensity intermittent exercise. Although the correlations shown in the present study do not establish a cause-and-effect relationship among the observed variables, results do suggest that the faster HRR kinetics in children might be partly related to their immature capacity for anaerobic metabolism.

Repeated-sprint exercise as a means of assessing HRR in children

The choice to use a repeated-sprint exercise in the present study was motivated by several physiological factors. First, such all-out exercise has been shown to be well tolerated and even more appropriate than longer continuous efforts in young participants (Ratel et al. 2006). Second, such exercise has already been shown to induce a marked increase in HR (Buchheit et al. 2007*a*) (up to about 75%–80% of HR_{max} in the present study) and $[La]_b$ (Ratel et al. 2006; Buchheit et al. 2007*a*), as well as a sharp decline in pH_b (Ratel et al. 2006). Third, the repeated nature of the sprints enabled us to obtain a number of measurements per subject over a wide range of HRR, power output, $[La]_b$, and pH_b values, thereby increasing the statistical power of our analysis. Fourth, age-related differences in HRR are known to be more apparent following high- vs. moderate-intensity exercise (Baraldi et al. 1991; Hebestreit et al. 1993). Fifth, because children might employ a different thermoregulatory strategy than do adults (Ratel et al. 2006), and because body temperature is likely to influence HRR (Buchheit and Laursen 2009), we employed 5-min recovery periods between sprints, which appeared adequate for preventing a confounding rise in core temperature that might have occurred throughout a longer and (or) continuous effort. Finally, the time course of postsprint HRR was well modeled via monoexponential curve fitting (Fig. 1), as previously reported (Perini et al. 1989; Buchheit and Gindre 2006; Buchheit et al. 2007*a*, 2008).

Postexercise HRR according to age and pubertal development

To quantify the rate of HRR, various methods have been used in the literature, so comparisons among studies is difficult. However, time constants for HRR (i.e., HRRτ) calculated here, as well as absolute heart beats recovered within the first minute after each sprint (i.e., $HRR_{60\ s}$), are in the range of those previously described in children (Baraldi et al. 1991; Hebestreit et al. 1993; Ohuchi et al. 2000; Zafeiridis et al. 2005), adolescents (Zafeiridis et al. 2005; Buchheit et al. 2008), and adults (Zafeiridis et al. 2005; Buchheit and Gindre 2006). As expected, HRR was moderately related to age and pubertal development (i.e., Tanner stages), but was also related to age-related anthropometric factors (i.e., body size or LBM) (Table 2). When averaging HRR values by group, children displayed a significantly faster HRR compared with adolescents and adults (Fig. 2, left panel). Although this is not a new finding (Baraldi et al. 1991; Hebestreit et al. 1993; Ohuchi et al. 2000; Zafeiridis et al. 2005), taken as they are (i.e., independently of blood acidosis, see later), our data confirm the body of evidence showing that HRR is, at least in part, age or maturation dependent (Singh et al. 2001). Nevertheless, we found no difference in HRR between adolescents and adults, despite a significant difference in the maturational status (i.e., Tanner stage III and IV for adolescents vs. stage V for adults). This lack of difference in HR-related indices, which was already highlighted by Zafeiridis et al. (2005) after 2×60 s of maximal knee extensions, suggests that HRR might not be entirely maturity-level dependent. Although the higher parasympathetic cholinergic modulation in children might account, in part, for their higher HRR (i.e., $\approx$36% (Ohuchi et al. 2000)), and even if vagal activity is known to decrease with aging (Craft and Schwartz 1995), there is no evidence yet presented for a lower vagal activity in pubertal adolescents compared with children (Garet et al. 2004; Perini et al. 2006; Buchheit et al. 2007*c*). Along the same line of thinking, it is not known whether the DNA sequence in the *CHRM2* gene locus of the heart's muscarinic receptors, shown to significantly influence HRR (Hautala et al. 2006), is affected differently during growth. It is instead more likely that other factors, probably nonautonomic and nonstructural, are involved in the faster HRR in children. A possible explanation for the faster HRR in children could be their faster clearance of sympathetic-related blood metabolites (Dotan et al. 2003; Ratel et al. 2006), related to a presumably higher muscle blood flow (Koch 1978), which has been shown to indirectly affect HRR via chemoreflex stimulation (Rowell and O'Leary 1990) or vagal inhibition (Miyamoto et al. 2003). Nevertheless, there is no consensus as to whether children effectively present lower postexercise epinephrine levels than do adults (Rowland and von Duvillard 1990; Baraldi et al. 1991), so this hypothesis has been impossible to verify previously. The data we have provided on $[La]_b$, acidosis, and HRR assist in answering this question.

Age-related postexercise $[La]_b$ and acidosis

As previously reported (Ratel et al. 2006), children displayed lower $[La]_b$ accumulation and maintained a higher pH_b throughout exercise, compared with their older counterparts. This was also consistent with the peak $[La]_b$ values measured during the incremental test (Table 1). This lower $[La]_b$ accumulation in children has been hypothesized to be related to both a lower anaerobic system participation and a higher rate of lactate and H^+ ion removal in children (Ratel et al. 2006). As reported previously (Zafeiridis et al. 2005), adolescents and adults displayed similar sprint performance and $[La]_b$ levels, suggesting that anaerobic metabolic capacity was already fully developed in the adolescents.

Anaerobic metabolic contribution to HRR

The significant relationships shown between HR-derived HRR indices and MPO, $[La]_b$, and acidosis (Fig. 3) indicate that anaerobic metabolism (or at least its level of maturation or participation) might explain a significant part of the variance in HRR after exercise (i.e., $\approx$40% for $HRR_{60\ s}$ vs. pH_b) (Perini et al. 1989; Buchheit et al. 2007*a*; Ba et al. 2009). Although pH_b and lactate are not the only chemical mediators of the chemoreflex control of HR (Rowell and O'Leary 1990; Kaufman and Hayes 2002), neural sympathetic activity may remain accentuated in individuals with

higher postsprint blood acidosis (i.e., adolescents and adults), which likely slows HRR. Indeed, adjusting for sprint power output, $[La]_b$ or acidosis reduced (Table 2), and in some cases completely abolished, the age- or maturational-associated differences in HRR (Fig. 2, right panel). These findings provide, for the first time, experimental evidence that HR deceleration may be related, at least partly, to muscle power output and (or) the associated blood acidosis and lactate accumulation in children after intense exercise.

Future research is needed to examine the respective impact of other mediators of the chemoreflex (e.g., potassium, ammonia, inorganic phosphate, products of purine metabolism, and nitric oxide (Vissing 2000; Kaufman and Hayes 2002)) on HRR. Although the higher chemoreceptor sensitivity of children compared with adults (Springer et al. 1988; Turley 2005) could have accentuated the sympathetic response and thus delayed HRR in the youngest subjects (abolishing between-group differences), the results of the present study suggest that the lower blood acidosis–lactate may have a greater impact on HRR and probably overpowered age-related chemoreflex differences. The lower muscle mass, as well as the lower associated maximal power output of children, may also explain their faster HRR, because it is thought that the contribution of the muscle metaboreflex to the cardiorespiratory response is inversely related to muscle mass and (isometric) contraction intensity (Iellamo et al. 1999). Irrespective of the blood acid-base status, the central command for sympathetic drive is also thought to be related to muscle mass recruitment (Franke et al. 2000). However, inference from static (Iellamo et al. 1999; Franke et al. 2000) to dynamic exercise (present study) needs to be viewed with caution. Because our experimental approach was based only on correlations, and because our measures were restricted to cycling power output, blood metabolites, and acidosis, the respective contribution of muscle power vs. the metaboreflex or vs. those discussed above (e.g., autonomic control of the heart) could not have been determined. Further studies in participants of various maturational levels using simultaneous measurements of HR variability (Ohuchi et al. 2000) may help improve our understanding of the physiological background of HRR throughout human development. Finally, it should be acknowledged that between-group differences in habitual training loads, likely to affect HRR (Buchheit and Gindre 2006), were not taken into account in the present analysis. However, age-related differences in HRR would have been even greater with highly trained children, so it is likely that the consistently faster HRRs observed here in moderately active children reflect true differences in the regulatory mechanisms of HRR among the 3 age groups.

Conclusions

The present study compared, to our knowledge for the first time, the postexercise HRR response in children, adolescents, and adults, while also taking into account muscle power output, postexercise $[La]_b$, and acidosis. The present results confirm that anaerobic system participation (and (or) its maturation) explains a significant portion of the variance in HRR during high-intensity intermittent exercise. Even if the correlations cannot determine a cause-and-effect link, these results suggest that the faster HRR kinetic observed in children during successive sprint exercises could be related partly to their lower muscle power output and immature anaerobic metabolic capacity. Maturational status, muscle power production, and (or) postexercise blood acidosis should be taken into account when evaluating HRR in (young) people.

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