Effect of maturation on parasympathetic modulation during exercise and recovery

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Abstract

Objective: This study examined the effect of maturation on parasympathetic nervous system (PNS) modulation of heart rate (HR) from rest to light- to moderate-intensity exercise and recovery from maximal exercise in prepubertal (n = 10; maturity offset = −0.6 ± 1.2 years; age = 13.7 ± 1.0 years) and postpubertal (n = 10; maturity offset = 1.9 ± 0.6 years; age = 15.6 ± 1.2 years) boys and men (n = 10; age = 24.1 ± 2.0 years).

Design: Participants completed seated rest, light-intensity exercise (50% HRmax), and moderate-intensity exercise (65% HRmax). Following moderate-intensity exercise, intensity was ramped to elicit maximal HR and followed by 25 min of seated recovery. Log transformed values for root mean square of successive differences (lnRMSSD), high-frequency power (lnHF) and normalized HF power (lnHFnHu) were assessed PNS modulation during 3 min of rest, light-intensity exercise, moderate-intensity exercise, and 3-min epochs throughout recovery.

Results: During light-intensity exercise, lnRMSSD and lnHF were greater in prepubertal (lnRMSSD = 3.4 ± 0.3 ms2; lnHF = 5.4 ± 0.7 ms2) compared to men (lnRMSSD = 2.8 ± 0.5 ms2; lnHF = 4.0 ± 0.9 ms2). During moderate-intensity exercise, lnHF differed between prepubertal and men (2.8 ± 1.0 vs. 1.4 ± 1.0 ms2). During recovery, HRV variables were greater in prepubertal compared to postpubertal and men.

Conclusions: Prepubertal boys have reduced PNS withdrawal during light-intensity exercise and greater PNS reactivation following exercise.

Introduction

Heart rate (HR) is primarily controlled by the two complementary branches of the autonomic nervous system, the parasympathetic nervous system (PNS), and the sympathetic nervous system (SNS). At rest, PNS activity is dominant, resulting in a low HR [1]. During the transition from rest to light-intensity exercise, the PNS gradually withdraws to increase HR. As exercise intensity increases, the PNS continues to withdraw and the SNS activates to further increase HR linearly up to maximal exercise [2]. The point at which the PNS influence is mostly withdrawn and the SNS begins to dominate occurs during moderate-intensity exercise (~50–60% of VO2max) [3]. Once maximal intensity exercise ceases, the PNS quickly reactivates resulting in a large decrease in HR early in recovery [4]. Later in recovery, PNS modulation continues to increase concomitantly with SNS withdrawal, resulting in a slower decrease in HR toward the resting value [5,6].

PNS regulation of HR can be measured non-invasively using heart rate variability (HRV), a measurement of the deviation in time between normal successive heartbeats [7]. Specifically, root mean square of successive differences (RMSSD), a time-domain measurement of HRV, and high-frequency (HF) power in the spectral domain represent PNS modulation [7,8]. Heightened PNS modulation, as measured by HRV, is considered an indicator of good cardiac health, and depressed PNS modulation is associated with certain diseases and can predict future disease [7,9]. In children, decreased resting PNS modulation has been associated with cardiovascular disease risk factors [8,10].

Although resting measures can offer insights into PNS dysfunction, they may not reveal alterations in PNS regulation of HR in response to stress. In adults, it is proposed that the PNS response to exercise or recovery from exercise may be a better predictor of certain cardiovascular diseases [3]. A study by Dewey et al. [11] not only found that resting HRV was a poor predictor of PNS responsiveness during exercise but also a blunted PNS response to exercise stress is associated with mortality in adults. Additionally, attenuated PNS reactivation following maximal-intensity exercise is a powerful clinical indicator of cardiometabolic disease [12] and all-cause mortality [13].
While it is becoming clear that the PNS response to exercise is an important clinical measure in adults, the PNS response to exercise and during recovery in children has been studied to a lesser extent. In obese children, the PNS response to exercise stress was reported as a better indicator of PNS dysfunction than when measured at rest [14]. Furthermore, evidence suggests a relationship between slowed PNS reactivation and cardiometabolic risk factors in children and adolescents [15]. These studies suggest PNS response to exercise and during recovery could be important clinically in children, similar to the conclusions in adults. This association between a responsive PNS and cardiometabolic health demonstrates the importance of understanding the entire PNS profile from rest to exercise to recovery, even in apparently healthy children.

One important aspect of the PNS profile that is currently lacking is the understanding of the changes that occur due to normal maturation. At rest, PNS modulation changes with chronological age [16], but these studies do not account for the maturational influence on resting PNS modulation, independent of chronological age. Currently, there appears to be a paucity of data regarding PNS response to dynamic exercise in children at different stages of maturation. During recovery from high-intensity exercise, younger, less mature children have greater PNS reactivation compared to older, more mature children and adults [17,18]. However, these studies were unable to discern the point during maturation when PNS reactivation during recovery begins to slow and decrease in magnitude. Therefore, the maturational influence on the full profile of the PNS response at rest, during light-intensity exercise and moderate-intensity exercise, and recovery from intense exercise is currently unknown.

Examining the PNS profile from rest to exercise to recovery from exercise in children could help identify the normal maturational changes in PNS regulation in response to exercise stress and guide preventative strategies in children and adolescents to avoid the clinical complications associated with PNS dysfunction. More specifically, maturational changes in the PNS response to exercise stress are important to understand in healthy children before examining clinical populations because it can serve as a baseline of normal developmental changes through childhood. The purpose of this study was to examine the influence of maturation on PNS modulation, as measured by HRV, at rest, during light-intensity exercise and moderate-intensity exercise, and during recovery from high intensity/maximal exercise.

All participants reported to the exercise testing laboratory on two occasions. During the first visit, height (cm) and mass (kg) were measured and the maturity offset was calculated for child subjects to estimate the child’s maturational status. The maturity offset was estimated as the age difference from the age at peak height velocity using the equation developed by Mirwald et al. [19]. Based on the maturity offset, the boys were classified as prepubertal (>1 year prior to peak height velocity), midpubertal (≤1 year prior to and ≤1-year past peak height velocity), and postpubertal (>1-year past PHV). This approach has been used by others [17,20]. Maturity offset was not determined on the adult men and they were placed in a fourth group (men).

**Exercise procedures**

During the first visit, participants completed three different levels of submaximal exercise on a cycle ergometer while HR and oxygen consumption (VO₂) were measured. This served to familiarize participants to the cycle ergometer and mouthpiece breathing valve and develop an individualized HR/work rate relationship, in order to estimate work rates in the next visit. Following a short rest (5–10 min), boys performed the McMaster All-Out Progressive Continuous Cycling Test [21] and adults completed a modified version of the McMaster All-Out Progressive Continuous Cycling Test that commenced at 50 W and increased by the prescribed increment of the tallest child. As all participants approached a maximal effort, half of the initial increment in the work rate was applied every minute until a maximal voluntary effort was reached [22]. HR was recorded from a Polar HR monitor every 30 s and OMNI (0–10) ratings of perceived exertion (RPE) were obtained at the end of each stage. Gas exchange measurements were collected continuously throughout the test using standard open-spirometry techniques to determine maximal VO₂ (VO₂max). In lieu of a plateau in VO₂, the following criteria were used to establish a maximal effort: 1) failure to maintain a pedal rate >50 rpm, 2) a respiratory exchange ratio ≥1.00 for boys and 1.10 for men, 3) OMNI RPE ≥8 and 4) peak HR ≥95% of age-predicted maximal (208 – 0.7 × age) [23]. If 2 out of the 4 criteria were achieved, the effort was considered maximal. VO₂max and maximal HR (HRmax) were the highest values observed during the test.

The second visit served as the experimental trial and took place between 2 and 7 pm. The participants were asked to refrain from eating food for at least 3 h prior to the visit and to refrain from alcohol, tobacco, and caffeine consumption, and vigorous physical activity on the day of the visit [7]. Upon arrival to the laboratory, participants and/or parents and guardians verbally confirmed adherence to all pre-visit requirements.

Participants sat quietly for 10 min (rest), then performed two submaximal exercise bouts at 50% (light-intensity exercise) and 65% (moderate-intensity exercise) of HRmax (±5%). The initial work rate was estimated from the HR/work rate relationship, determined from the previous visit. HR during the exercise trial was recorded in real-time using

**Materials and methods**

**Participants**

Recreationally active, apparently healthy boys (7–17 years) and men (18–29 years) were recruited for this study. Prior to data collection, adult men and parents/guardians of the boys gave written consent and the boys gave written assent to participate in the study. This study was approved by the Institutional Review Board at Ball State University.
a Polar HR monitor. If the desired HR was not achieved within 2.5 min of the start of the exercise stage, the work rate was adjusted to elicit the desired HR response. Each participant exercised for 4 min after a steady-state HR (ΔHR < 5 bpm) was achieved at each intensity. Immediately after the second submaximal exercise bout, the work rate was increased in a ramp-like fashion until 100% of HRmax (±5%) (maximal exercise) was achieved. HRmax was achieved within ~6–8 min. Within 30 s of the achievement of HRmax, the participants assumed a seated position and remained seated for 25 min, avoiding talking and excessive movement.

Heart rate variability

An electrocardiogram (ECG) recorded continuously throughout the rest, exercise, and recovery at a sampling frequency of 1000 Hz using a Biopac MP35 recording system, under free-breathing conditions. To ensure stationarity requirements were met for spectral analysis, HRV was analyzed during the final three minutes of rest, and each intensity of submaximal exercise [7]. During recovery, HRV was analyzed at 7–10 min (Rec10), 12–15 min (Rec15), 17–20 min (Rec20), and 22–25 min (Rec25). The WinCPRS software program was employed to assess HRV. The ECG was manually inspected to ensure all R-waves were included in the analysis and to identify ectopic heartbeats, which were corrected using the interpolation method [24].

In the time domain, RMSSD was the only variable reported because it characterizes PNS modulation at rest, during exercise, and recovery from exercise [25,26]. The fast Fourier transformation was used to determine the power spectrum of HRV. The power spectrum was analyzed for total power (TP) (0.0–0.4 Hz), LF (0.04–0.15 Hz), and HF (0.15–0.4 Hz). Of these variables, HF and HFnu indicate PNS modulation in the frequency domain and are the only frequency domain variables reported [7].

Heart rate data processing

Target HR for light-intensity exercise, moderate-intensity exercise, and maximal exercise in the second visit was calculated from HRmax recorded by the Polar HR monitor during the graded exercise test. While the Polar HR monitors were used in real-time to aid in the determination of the proper exercise intensity, the reported HR during the trial was analyzed post-hoc from the single-lead ECG. This was performed so the reported HR was from the same epochs as the HRV analysis. At rest, HR was the average HR over the final three mins of the seated period. HR during light-intensity exercise and moderate-intensity exercise was the average HR over the final 3 mins of each stage. The HR at maximal exercise was the average HR over the final 30 s of the exercise trial from the ECG recording. During recovery, HR was the average over the 3-min epoch analyzed for HRV.

Statistics

The physical characteristics and the maximal exercise responses were compared between groups using a one-way ANOVA or a Kruskal–Wallis analysis in instances where normality was violated. As a result of a significant one-way ANOVA outcome, a Bonferroni (homogeneity of variance assumed) or a Dunnett T3 (homogeneity of variance not assumed) post-hoc test was performed. Two variables, height, and weight, showing a violation of the homogeneity of variance, were examined with a Welch F test to further confirm the ANOVA finding. The percentage of HRmax during light-intensity exercise and moderate-intensity exercise and at maximal exercise also was analyzed with a one-way ANOVA or Kruskal–Wallis analysis. A two-way (group by time) ANOVA was used to analyze the HR responses from rest to exercise to recovery between groups, with Bonferroni post-hoc testing when necessary. LnRMSSD and lnHF were analyzed using a 2-way (group-by-time) ANOVA, with Bonferroni post-hoc testing as warranted. HRV measurements were not normally distributed and each variable was log-transformed (e.g. lnRMSSD, lnHF, and lnHFnu) to compensate. It should be noted that HR at rest was not normally distributed and despite log transformation of the HRV variables, there were 1–2 instances for each HRV variable where the data within a group were not normally distributed. Because the ANOVA is robust to normality violations [27] and the HRV variables were already transformed, no further alterations to any of these variables were considered. Due to lnHFnu values being negative, HFnu values were reported but the comparisons were made with the lnHFnu values. Significance was set at \( p \leq .05 \).

Results

Physical characteristics

There were 39 total participants who completed this study. Based on their maturity offset, there were 10 boys considered prepubertal, 9 boys considered midpubertal, and 10 boys considered postpubertal. Additionally, 10 men were in the men group. Table 1 shows the physical characteristics of the participants. As expected, there were significant group effects for age, weight, height, and body mass index (BMI). Post-hoc testing revealed that age in prepubertal was significantly different from the other groups; midpubertal and postpubertal were also significantly different from men. For weight, prepubertal was different from postpubertal and men; the weight in midpubertal was also different than men (\( p \leq .05 \)). For height, prepubertal was significantly different

| Age (years) | MO (years) | Weight (kg) | Height (cm) | BMI |
|------------|------------|-------------|-------------|-----|
| Pre 10.1 ± 1.9abc | 3.0 ± 1.2ab | 34.3 ± 6.4abc | 141.0 ± 1.08abc | 17.2 ± 1.8abc |
| Mid 13.7 ± 1.0 | −0.1 ± 0.6 | 51.9 ± 9.5c | 164.7 ± 3.1b | 19.2 ± 3.8c |
| Post 15.6 ± 1.2 | 1.9 ± 0.6 | 75.3 ± 15.8 | 173.8 ± 6.4 | 25.0 ± 5.3 |
| Adult 24.1 ± 2.0 | − | 78.4 ± 10.7 | 178.8 ± 7.5 | 24.5 ± 3.5 |

a: different from mid; b: different from post; c: different from adult; significance at \( p \leq .05 \).

Table 1. Physical characteristics.
compared to the other groups; midpubertal also significantly different from postpubertal and men. Prepubertal BMI was different from postpubertal and men ($p \leq .05$); midpubertal BMI was also significantly different compared to BMI for men. For the maturity offset comparison, all three groups differed from one another, as was planned in the design of the study ($p \leq .05$).

**Maximal exercise responses**

Table 2 shows the physiological responses at maximal exercise. All participants achieved at least 2 of the 4 criteria for a maximal response. All groups had a significantly different absolute VO$_2$ (L-min$^{-1}$). The only other group difference was for RER, in which prepubertal was significantly different compared to postpubertal and men.

**HR responses**

HR responses at rest, during exercise, and recovery are shown in Table 3. There was a significant group-by-time interaction in the HR responses. Post-hoc testing showed the only significant group difference was HR during rest between prepubertal and adult. However, there were significant time effects within groups. In each group, HR during rest, light-intensity exercise, moderate-intensity exercise, and maximal exercise were significantly different from each other, and HR during moderate-intensity exercise and maximal exercise were significantly different from all HRs during recovery. There also were differences within each group during recovery. HR at Rec10 and Rec25 were significantly different in prepubertal. In Mid, HRs at Rec10, and Rec15 were both significantly different from Rec20 and Rec25. For postpubertal, HR at Rec10 was significantly different compared to HRs at light-intensity exercise, Rec20, and Rec25; HR at Rec15 was also significantly different from Rec25. For men, HR at Rec 10 was significantly different compared to other recovery responses; Rec15 and Rec20 HRs were significantly different than Rec25. For each group, all recovery values were significantly different than resting HR.

### Table 2. Maximal exercise responses.

|            | VO$_2$ (L-min$^{-1}$) | VO$_2$ (ml kg$^{-1}$ min$^{-1}$) | HR (beats min$^{-1}$) | RER | RPE (0–10) |
|------------|----------------------|-------------------------------|------------------------|-----|-------------|
| Pre        | 1.70 ± 0.38$^{a,b,c}$ | 49.6 ± 5.8                    | 193.1 ± 10.8           | 1.13 ± 0.05$^{a,b,c}$ | 9.8 ± 0.6 |
| Mid        | 2.56 ± 0.46$^{a,b,c}$ | 49.9 ± 8.2                    | 191.4 ± 6.6            | 1.19 ± 0.06$^{a,b,c}$ | 10 ± 0.0  |
| Post       | 3.16 ± 0.38$^{a,b,c}$ | 43.2 ± 8.7                    | 193.9 ± 11.6           | 1.22 ± 0.04$^{a,b,c}$ | 9.5 ± 0.7 |
| Adult      | 3.75 ± 0.43          | 48.5 ± 7.4                    | 190.5 ± 9.4            | 1.23 ± 0.06$^{a,b,c}$ | 9.8 ± 0.4 |

- $^{a}$: different from mid; $^{b}$: different from post; $^{c}$: different from Adult; significance at $p \leq .05$.

### Table 3. HR responses during exercise and recovery.

|               | Rest (bpm) | Light-Intensity Exercise (bpm) | Moderate-Intensity Exercise (bpm) | MAX (bpm) | Rec 10 (bpm) | Rec 15 (bpm) | Rec 20 (bpm) | Rec 25 (bpm) |
|---------------|------------|-------------------------------|----------------------------------|-----------|--------------|--------------|--------------|--------------|
| Pre           | 77.0 ± 10.2$^{a,b}$ | 98.0 ± 4.9                   | 124.2 ± 6.3                      | 191.6 ± 9.3 | 97.1 ± 8.5   | 93.4 ± 10.2  | 91.2 ± 11.4  | 89.6 ± 9.2   |
| Mid           | 67.9 ± 8.7   | 94.3 ± 4.5                    | 123.1 ± 5.1                      | 189.1 ± 7.6 | 98.7 ± 7.6   | 93.7 ± 8.4   | 88.6 ± 8.6   | 86.4 ± 8.6   |
| Post          | 71.1 ± 7.8   | 96.1 ± 6.1                    | 122.9 ± 6.1                      | 193.3 ± 6.9 | 106.0 ± 7.4  | 101.7 ± 9.2  | 97.7 ± 9.9   | 95.9 ± 9.2   |
| Adult         | 63.1 ± 12.4  | 95.3 ± 4.4                    | 125.7 ± 7.6                      | 186.4 ± 9.9 | 101.9 ± 12.6 | 97.0 ± 8.7   | 94.1 ± 8.2   | 90.6 ± 8.4   |

- $^{a}$: different from adult; significance at $p \leq .05$.

The relative HRs during light-intensity exercise for prepubertal, midpubertal, postpubertal, and men were $50.8 ± 1.6$, $49.2 ± 1.4$, $49.6 ± 1.1$, and $50.1 ± 2.4%$, respectively. During moderate-intensity exercise, the relative HRs for prepubertal, midpubertal, postpubertal, and men were $64.3 ± 1.0$, $64.3 ± 1.9$, $63.5 ± 1.4$, and $66.0 ± 2.2%$, respectively. The relative HRs at maximal exercise for prepubertal, midpubertal, postpubertal, and men were $99.2 ± 2.8$, $98.7 ± 1.6$, $99.8 ± 3.2$, and $97.8 ± 2.1%$, respectively. The only significant difference for relative HR between groups was observed during moderate-intensity exercise between postpubertal and men.

**HRV responses**

Figure 1 shows the lnRMSSD response to rest, exercise, and recovery. There was a significant group-by-time interaction effect. Post-hoc testing revealed lnRMSSD during light-intensity exercise was significantly different in prepubertal compared to men. Throughout recovery, lnRMSSD for prepubertal was significantly different from postpubertal and men. The only other significant group difference for lnRMSSD was between midpubertal and postpubertal at Rec15. Post-hoc testing also showed significant time effects within groups. For each group, lnRMSSD during rest, light-intensity exercise, and moderate-intensity exercise were all significantly different. For midpubertal, postpubertal, and adult, lnRMSSD at rest was significantly different from all recovery points; for prepubertal, lnRMSSD at rest was only different from Rec10, Rec15, and Rec20. For prepubertal, lnRMSSD at moderate-intensity exercise was significantly different compared to all recovery points; Rec10 and Rec15 were also significantly different compared to Rec25. For midpubertal, lnRMSSD at moderate-intensity exercise and Rec10 were both significantly different from Rec15, Rec20, and Rec25. For postpubertal, lnRMSSD at light-intensity exercise was different from Rec10 and Rec15; moderate-intensity exercise was also different from Rec25. During recovery for postpubertal, lnRMSSD at Rec10 was significantly different from Rec20 and Rec25; Rec15 was also significantly different from Rec25. For adult, lnRMSSD at light-intensity exercise was different from Rec10. Additionally, lnRMSSD at moderate-intensity exercise and Rec10 were each significantly different from Rec20 and Rec25; Rec15 was also significantly different from Rec25. Figure 2 shows the lnHF response to rest, exercise, and recovery. There was a significant group-by-time interaction. Post-hoc testing revealed significant differences between prepubertal and men at light-intensity exercise and moderate-intensity exercise ($p = .051$). Throughout recovery, lnHF was...
significantly different in prepubertal compared to postpubertal and men. The only other significant group differences for lnHF were between midpubertal and postpubertal at Rec20. There were also significant time effects within groups. For each group, lnHF during rest, light-intensity exercise, and moderate-intensity exercise were all significantly different. For midpubertal, postpubertal, and men, lnHF at rest significantly different from all recovery points, but for prepubertal, rest was only significantly different from Rec10, Rec15, and Rec20. For prepubertal, lnHF at moderate-intensity exercise was significantly different from all recovery points and lnHF at Rec10 was significantly different compared to Rec25. For midpubertal, lnHF at moderate-intensity exercise was significantly different from Rec15, Rec20, and Rec25; Rec10 was also significantly different from Rec20 and Rec25. For postpubertal, lnHF at light-intensity exercise was different from Rec10 and Rec15; moderate-intensity exercise was also different from Rec25.
During recovery for postpubertal, lnHF at Rec10 was significantly different from Rec20 and Rec25. For men, lnHF at moderate-intensity exercise was significantly different from Rec15, Rec20, and Rec25; Rec10 was also significantly different from Rec20.

As noted previously, HFnu was log-transformed and these data were analyzed; however, since these values were negative, the non-log transformed HFnu response to rest, exercise, and recovery are illustrated in Figure 3. There was a significant group-by-time interaction. Post-hoc testing revealed significant group effects, but the only differences between groups were during recovery. There were significant differences in lnHFnu between prepubertal and postpubertal and men at Rec15, Rec20, and Rec25; differences between midpubertal and men were also observed at Rec20 and Rec25. Additionally, post-hoc testing revealed there were significant time effects. Within prepubertal, there were only differences in lnHFnu between rest and moderate-intensity exercise, and moderate-intensity exercise and Rec25. Within midpubertal, lnHFnu at rest was different from moderate-intensity exercise, Rec10, and Rec15. Within postpubertal, lnHFnu during rest was different from all points during recovery. Additionally, lnHFnu during light-intensity exercise was different from Rec10 and Rec15 within postpubertal. In adults, lnHFnu at rest was significantly different from Rec15, Rec20, and Rec25; lnHFnu during light-intensity exercise was also different from Rec 25.

**Discussion**

The purpose of this study was to examine the effect of maturation on changes in PNS responses from rest to exercise and during recovery from maximal exercise. This study found maturational differences in PNS response from rest to light-intensity exercise and during recovery from maximal exercise. Specifically, lnRMSSD and lnHF indicated PNS withdrawal was greater in men compared to prepubertal boys at light-intensity exercise, despite a similar relative HR (i.e. % of HRmax). Furthermore, prepubertal boys had greater lnRMSSD and lnHF during recovery from maximal exercise compared to postpubertal boys and men. While there were no differences in lnHFnu during exercise, responses during recovery supported the results of lnRMSSD and lnHF. Prepubertal boys had greater lnHFnu after 15 min of recovery compared to postpubertal and men. Additionally, midpubertal had greater lnHFnu response compared to men after 20 min of recovery. Based on lnRMSSD and lnHF, the only group to fully recover to the resting level at 25 min post-exercise was the pubertal boys; however, full recovery to resting level was apparent in prepubertal boys and midpubertal boys based on lnHFnu. Overall, it appears changes occur as a result of growth and maturation in the PNS regulation of HR during exercise and recovery.

There were no maturational differences in PNS modulation at rest. Similarly, Buchheit et al. [17] found no differences in resting PNS modulation between pre-, circum-, and post-peak height velocity male soccer players. Other studies found similar PNS modulation at rest between pre- and post-peak height velocity boys [20] and boys and men [18]. Conversely, Finley et al. [16] found PNS modulation decreased from age 5 to 24 years, whereas Pikkujäämsä et al. [28] found increasing PNS modulation at rest with increasing chronological age through childhood. The difference in results among these studies could be due to variations in methodology, such as differences in the time of day, use of controlled breathing, and duration of epoch analyzed. Moreover, aerobic fitness and habitual physical activity
could have contributed to the different findings between studies. In the study by Buchheit et al. [17] and this study, VO\textsubscript{2\textmax} was relatively high in all groups (range of both studies: 43.2–60.6 ml·kg\textsuperscript{-1}·min\textsuperscript{-1}). Furthermore, there were no differences in VO\textsubscript{2\textmax} between groups in this study. Aerobic fitness could exert a greater influence on PNS modulation at rest than maturation, although more research is needed to confirm this assertion.

In contrast to rest, there were maturational differences in PNS modulation during exercise. During light-intensity exercise, lnHF and lnRMSSD were greater in prepubertal boys compared to men, but lnHFnu was similar. As PNS modulation was similar at rest, lnHF and lnRMSSD responses suggest prepubertal boys had less PNS withdrawal, or less of a decline in PNS modulation from rest to light-intensity exercise, compared to men. During moderate-intensity exercise, PNS modulation tended to remain greater in prepubertal boys compared to men, although the differences in PNS modulation were less clear at this higher intensity. Prepubertal boys had greater lnHF compared to men, but lnRMSSD and lnHFnu were not different between the groups. As each of these variables can give insight into PNS modulation, the contrasting results make it difficult to have a clear understanding of PNS modulation at moderate-intensity exercise [7]. Moreover, absolute HR and the percent of HR\textsubscript{max} were similar between all groups at light-intensity exercise and moderate-intensity exercise, but prepubertal boys had a smaller change in HR from rest to light-intensity exercise and rest to moderate-intensity exercise because resting HR was greater than all other groups. The smaller change in HR in prepubertal boys could be due to lesser withdrawal in PNS from rest to exercise [5]. Regardless, it appears that prepubertal boys may be able to elevate HR during light-intensity exercise with less PNS withdrawal compared to men, but as children approach puberty these differences begin to abate. Although PNS responses were not significantly different between prepubertal boys and midpubertal boys, there appears to be a transition toward a more adult-like pattern (i.e. greater PNS withdrawal) that occurs concomitantly with puberty. However, the underlying basis for these maturational differences is unclear and was not addressed in this study.

In this study, lnHF and lnRMSSD variables followed the expected pattern, that is as exercise intensity increases, PNS withdrawal increases (PNS modulation abates) from rest to light-intensity exercise to moderate-intensity exercise [29]. On the other hand, HF\textsubscript{nu} may not respond in a manner similar to rest. In this study, lnHF and lnRMSSD responses were not significantly different between prepubertal boys and men, but lnHFnu was similar. As PNS modulation was similar at rest, lnHF and lnRMSSD were greater in prepubertal boys compared to men, but lnHFnu was similar. lnHFnu was not different from exercise at any point in midpubertal boys. This study measured PNS modulation during a fully activated state (i.e. rest) and a withdrawn state (i.e. moderate-intensity exercise) and used those time points as baselines to understand the relative magnitude of the response [3]. Additionally, multiple time points were measured throughout an extended recovery to understand the speed and timing of PNS recovery from maximal exercise [2,3]. In this context, prepubertal boys were the only group to increase PNS activity from moderate-intensity exercise in the first 10 min of recovery. After the increase in PNS modulation early in the recovery, PNS reactivation tapered and PNS activity did not increase again until 25 min post-exercise. However, each HRV variable showed PNS modulation fully recovered to the resting value after 25 min. Contrary to prepubertal boys, the other groups had a more delayed recovery of PNS reactivation. For, postpubertal boys, and men, HRV variables did not increase from moderate-intensity exercise until 15 or 20 min into recovery and did not fully recover PNS modulation to the resting level within 25 min post-exercise. For midpubertal, the lnHFnu response was not consistent with lnRMSSD and lnHF; lnHFnu was not different from exercise at any point in recovery and returned the resting value within the recovery period. These findings suggest that prepubertal boys had a large PNS response early in the post-exercise recovery and the magnitude of PNS response allowed for the PNS to fully
recover within 25 min post-exercise. In the more mature groups, there was a slower and reduced PNS response early in the post-exercise recovery and PNS modulation did not fully recover to their resting value within 25 min. This appears to be the first study to show PNS modulation in prepubertal boys returned to baseline within 25 min following maximal exercise.

In adults, it has been proposed that examining PNS response to and following exercise stressors could be a better method to diagnose or predict cardiometabolic disease [3]. In this context, the PNS response is thought to be “cardioprotective” [36] and greater PNS activity during exercise and a greater response following exercise is associated with cardio-metabolic health [7,9]. On the other hand, a greater reduction in PNS modulation during exercise and smaller increases in PNS modulation after exercise suggest a less responsive PNS [3]. While it is becoming clear that the PNS response to exercise and recovery is an important clinical measure in adults, it has been studied to a lesser extent in children. A study reported PNS response to exercise stress was a better indicator of PNS dysfunction than when measured at rest in obese boys [14]. Furthermore, evidence has suggested a relationship between slowed PNS re-activation and cardiometabolic risk factors in children and adolescents [15]. This association between a responsive PNS and cardiometabolic health demonstrates the importance of understanding the entire PNS profile from rest to exercise to recovery. Additionally, changes in cardiorespiratory fitness through training affects the reactivity of the PNS from rest to exercise to recovery, in that training-induced improvements in cardiorespiratory fitness would lead to greater PNS modulation during exercise and recovery [37]. Moreover, the adaptations of the PNS as a result of training have become a popular means to monitor athlete training [38,39], although current research in children is limited. These results suggest that maturation should be a consideration when utilizing HRV and PNS modulation to monitor youth training adaptations. While this study did not examine the clinical or sport implications of maturational-dependent decreases in PNS responses, future studies, clinicians, and sport scientists should consider maturational changes in PNS regulation of HR during exercise and prolonged recovery from exercise.

While it appears that maturation does have some effect on PNS modulation, it must be noted there are numerous factors that can affect PNS modulation. One major factor that affects PNS modulation during exercise and recovery is exercise intensity [8,40]. This study carefully controlled exercise intensity to a relative percentage of HRmax. The intensities for light-intensity exercise and moderate-intensity exercise were specifically chosen because the regulation of HR at these intensities is primarily controlled by the withdrawal of PNS influence [2,6]. Furthermore, maximal exercise was chosen because recovery from maximal exercise is strongly related to clinical outcomes in adults [13] and children [15]. For the most part, all groups exercised at a relative HR that was similar to the targeted relative HR (e.g. 50, 65, and 100% of HRmax). The only difference between groups in relative HR occurred during moderate-intensity exercise between postpubertal boys and men, but there was no difference between groups in absolute HR. Therefore, all groups had a similar exercise stressor for the PNS system to respond.

Another factor that can affect PNS modulation during exercise is the duration of the bout of exercise [3,41]. In this study, the duration of each exercise stage was not controlled. Because exercise intensity was closely controlled and HR during exercise has some daily variations, some participants needed adjustments in the work rate during exercise stages. Following adjustments in the work rate, the participant needed to reach a steady-state HR to ensure stationarity before the HRV measurement could commence [7]. Therefore, the duration of each participant varied slightly. Few studies have examined the effect of exercise duration on HRV during exercise or recovery [41] and it appears no studies have been conducted with children. However, exercise intensity has a more pronounced effect on HRV than exercise duration [3]. In this study, differences in exercise duration were necessary to control exercise intensity. Lastly, non-neural factors have shown to influence HF power, especially during exercise [35]. As these influences were not assessed in this study, it is unclear how these factors impacted the findings.

This study suggests that there are maturational differences in magnitude and pattern of PNS reactivity during light-intensity exercise and recovery from high-intensity exercise. Specifically, prepubertal boys had less PNS withdrawal to reach the same relative HR as men. During recovery, prepubertal boys had a greater PNS response early in recovery compared to postpubertal boys and men. Moreover, prepubertal boys had the largest response in PNS modulation during recovery, as they were the only group to fully recover PNS modulation after maximal-intensity exercise. These findings suggest that at some point near puberty there is a transition in PNS regulation of HR toward a more adult-like pattern, although the exact timing or the underlying basis for these changes is unclear and were not addressed in this study. Future research is needed to understand the specific mechanism of puberty and the effects of maturation on the PNS during exercise and recovery. However, these findings suggest that maturation should be considered when interpreting PNS modulation during exercise and recovery in children and could be a factor in guiding pointed preventative strategies in children and adolescents to avoid the clinical complications associated with PNS dysfunction.

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