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Original Research

Contribution of the Autonomic Nervous System to Recovery in Firefighters

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Context: Sudden cardiac deaths (SCDs) have accounted for nearly half of the line-of-duty deaths among US firefighters over the past 10 years. In 2018, 33% of all SCDs occurred after the end of a fire service call. Researchers have suggested that an imbalance in autonomic nervous system (ANS) regulation of heart rate postcall may interfere with recovery in firefighters.

Objective: To use heart-rate recovery (HRR) and heart-rate variability (HRV), 2 noninvasive markers of ANS function, to examine the ANS recovery profiles of firefighters.

Design: Cross-sectional study.

Setting: Firehouse and research laboratory.

Patients or Other Participants: Thirty-seven male career active-duty firefighters (age = 39 ± 9 years, height = 178.8 ± 5.4 cm, mass = 87.9 ± 11.2 kg).

Main Outcome Measure(s): Percentage of maximal HR (%MHR) and HRV (natural log of the square root of the mean sum of the squared differences [lnRMSSD]) were collected after both submaximal and maximal exercise protocols during a 10-minute seated recovery. The HRR profiles were examined by calculating the asymptote, amplitude, and decay parameters of the monoexponential HRR curve for each participant.

Results: Differences in HRR parameters after 10 minutes of seated recovery were identified after submaximal versus maximal exercise ($P < .001$). In addition, although ANS was more suppressed after maximal exercise, HRV indicated incomplete recovery, and regardless of the test, recovery %MHR and lnRMSSD values did not return to pretest %MHR and lnRMSSD values.

Conclusions: Our results suggest that the ANS contributions to recovery in active-duty firefighters are exercise-intensity specific, and this is likely an important factor when establishing best-practice recovery guidelines.

Key Words: heart-rate recovery, heart-rate variability, blood lactate, sympathetic nervous system, vagal reactivation

Key Points

- The contribution of the autonomic nervous system to recovery in active-duty firefighters is exercise-intensity specific.
- The exercise-intensity–specific contribution of the autonomic nervous system is likely an important factor when establishing best-practice recovery guidelines.
- Heart-rate variability may be an important measure for monitoring recovery in firefighters.

Firefighting involves aerobic and anaerobic physical activities[1] that result in heart rates (HRs) ranging from submaximal to exceeding maximal.[2] These varying demands occur with each call to which firefighters respond during a shift, placing both acute and cumulative cardiovascular strain on them.[1,2] Therefore, it is not surprising that sudden cardiac deaths (SCDs) have accounted for nearly half of the line-of-duty deaths among US firefighters over the past 10 years.[3] Most of these SCDs occurred during or shortly after fire-suppression activities,[4] and investigators[1] have demonstrated that the odds of a firefighter experiencing an SCD after a call remain 2.2 to 10.5 times higher than during nonemergency duties. This extended elevated risk of SCD has also prompted the passage of the Hometown Heroes Survivors Benefit Act,[5] which considers the cardiac-related deaths of public safety officers occurring within 24 hours of stressful occupational activity to be line-of-duty incidents. In 2018, 12 of the 37 firefighters who died due to stress or overexertion[5] were designated "Hometown Heroes" by the Federal Emergency Management Agency.[3] Although the risk of postactivity SCD is acknowledged in the fire service, research examining the physiological recovery of firefighters is lacking. An understanding of the autonomic nervous system (ANS) response during recovery may provide insight that will enable us to better address the SCD risk in firefighters.

The HR transition from rest to exercise to recovery is accomplished through an interplay between the parasympathetic nervous system (PSNS) and sympathetic nervous system (SNS) branches of the ANS. Increases in HR from rest to exercise occur by decreasing PSNS influence and increasing SNS influence, whereas postexercise restoration of HR is attributed to a reversal of these ANS regulatory actions.[6,7] Based on the high HR response achieved during fire-suppression activities, Smith et al[2] hypothesized that elevated SNS activation during recovery may contribute to

this elevated risk of SCD. Authors[8] have also reported that the intensity of an activity may be related to blood lactate (La^-) levels during recovery. However, this invasive measure does not offer insight into the potential contributions of the PSNS and SNS. Researchers[9] have used noninvasive methods, such as HR recovery (HRR) and, more recently, HR variability (HRV), to assess the contribution of the ANS to cardiac recovery.

The HRR quantifies the HR response across time after exercise. In a review, Peçanha et al[9] described HRR as a monoexponential curve, consisting of fast and slow phases. The fast phase represents the initial deceleration of HR due to PSNS reactivation. In the subsequent slow phase of recovery, HR continues to decrease toward pre-exercise levels and is the product of PSNS reactivation and SNS withdrawal. An HRR threshold exists for which HR decreases of $\leq$18 beats per minute (bpm) at 1-minute postexercise are associated with a higher risk for myocardial injuries.[7] In athletes, HRR is used to monitor training load and indicate fitness.[10] Therefore, the fast and slow phases of HRR have meaningful influences in both clinical and athletic populations.

Heart-rate variability is a noninvasive marker of ANS function based on the beat-to-beat variation in HR measured by the duration of the R-R interval (ie, time between beats, which is used to calculate HR).[11] In general, higher HRV is thought to be predominately under greater PSNS control.[11] After exercise, HRV is expected to increase across time due to greater PSNS activity decreasing the cardiovascular demand.[9,12,13] The HRV has been used in clinical populations, such as those with diabetes, or to generally assess those at risk for coronary heart disease.[6] In active populations, HRV has been used to prescribe and monitor training in elite to recreational athletes.[10,14,15]

After maximal exercise, SNS withdrawal may have a greater influence on the early recovery period rather than a predominate restoration of PSNS followed by SNS withdrawal.[12,16] However, this has not been examined in the firefighter population. Monitoring HRR and HRV during recovery from exercise could elucidate the contributions of the PSNS and SNS to HR control during recovery from 2 intensities, as not all fire calls require maximal intensity. Therefore, the purpose of our study was to investigate the postexercise ANS recovery of active-duty firefighters by (1) descriptively examining the ability of HR to recover after submaximal and maximal protocols, (2) examining differences between the submaximal and maximal HRR profiles, and (3) using measures of HRV and La^- to further delineate ANS activity after submaximal and maximal exercise. We hypothesized that a descriptive examination of the HRR profiles would determine the relative recovery status after submaximal and maximal tests and that HRR, HRV, and La^- would differ between the 2 testing intensities.

METHODS

Participants

A total of 37 male career active-duty firefighters (age = 39 $\pm$ 9 years, height = 178.8 $\pm$ 5.4 cm, mass = 87.9 $\pm$ 11.2 kg, body mass index = 27.7 $\pm$ 3.9 kg/m^2) from the same department volunteered for this study. Participants were cleared for full active-duty service; free of cardiopulmonary, metabolic, renal, and musculoskeletal conditions; and not taking medications that could influence HR. All participants gave written informed consent, and the study was approved by the Institutional Review Board of the University of Wisconsin–Milwaukee.

Procedures

All participants completed 2 testing sessions. The submaximal test was completed at a fire station, and the maximal test was completed in the Human Performance and Sport Physiology Laboratory at the University of Wisconsin–Milwaukee. Testing sessions were separated by at least 24 hours but no longer than 96 hours. Participants wore exercise clothing during both testing sessions. To ensure consistency, all data were collected by the same researcher (R.J.F.).

Submaximal Exercise Protocol. The Queens College Step Test was used as a submaximal exercise test,[17] which is consistent with other submaximal step tests performed within the firefighter population.[18,19] This test requires participants to step up a 16.25-in (40.3-cm) step for 3 minutes to a cadence of 96 steps per minute.[17] After completing the test, participants immediately transitioned to a seated position for the 10-minute recovery period.

Maximal Exercise Protocol. Participants also completed a maximal graded treadmill (model RTM 600; Biodex Medical Systems, Shirley, NY) exercise test.[20] The protocol began with a 3-minute walking warm-up at 3.0 mi/h (4.83 km/h) and 0% grade, followed by an increase to 4.5 mi/h (1.24 km/h) for 1 minute. The remainder of the protocol involved alternating increases of 2% grade and 0.5 mi/h (0.80 km/h) every minute until maximal effort was exerted. After the test, participants were immediately transitioned to a seated position for the 10-minute recovery period. Effort during the treadmill test was considered *maximal* when participants achieved an HR within 10 bpm of their estimated maximal HR (220 – age).

Heart-Rate Recovery Measures

The HR (in bpm) and R-R interval (in milliseconds) data were collected using the Polar V800 watch and H7 monitor (250-Hz sampling rate; Polar Electro, Kempele, Finland), which has demonstrated acceptable criterion validity in reference to electrocardiography when collecting HR and R-R interval data during resting and activity states.[21,22] Data were transferred from the watch to a desktop computer via software (Polar Electro) for subsequent analysis.

Percentage Maximal HR Data. Resting HR (HR_{Rest}) was averaged during a 5-minute pretest period. The HR immediately after cessation of exercise was identified (HR_0), and all remaining HRR measures were averaged as 30-second epochs (eg, HR_{30}, HR_{60}, and HR_{90}) for a total of 21 HRR measures during the 10-minute recovery period. The HR data were normalized to each participant's age-predicted HR maximum and expressed as a percentage of maximal HR (%MHR).

To describe the HRR profiles of each participant, we fitted the 21 HRR measures within both the submaximal and maximal protocols of each participant to a monoexponential curve[13] (Figure 1) using the nlinfit function in MATLAB (version R2018a; The MathWorks, Inc, Natick, MA):

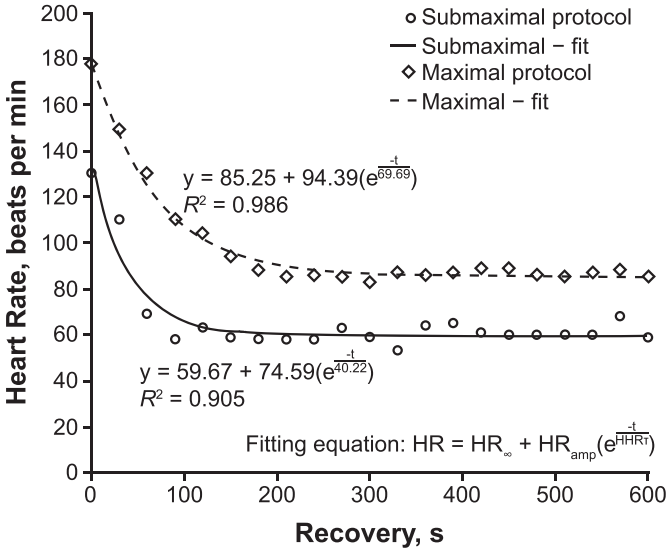

Figure 1. Example of the monoexponential curve-fitting technique with the heart-rate (HR) data collected from a representative participant during the 10-min recovery period. Abbreviations: HR_{∞}, asymptotic value of HR; HR_{amp}, difference between HR_0 (immediately after cessation of exercise) and HR_{∞}; t, time (in seconds); HRR_t, decay constant.

$$HR = HR_{\infty} + HR_{amp}\left(e^{\frac{-t}{HRR_{\tau}}}\right),$$

where HR_{∞} is the asymptotic value of HR, HR_{amp} is the difference between HR_0 and HR_{∞}, t is time (in seconds), and HRR_{τ} is the decay constant.

Heart-Rate Variability Data. The R-R interval data were analyzed using HRV standard software (version 3.0; Kubios, Kuopio, Finland) by applying a low-artifact correction to remove ectopic beats and a time-varying high-pass filter (smoothing priors) to detrend the data.[11] After filtering, we calculated the square root of the mean sum of the squared differences (RMSSD; in milliseconds) between R-R intervals to quantify the time domain measure of HRV,[11] which is considered more reflective (versus frequency domain) of vagal tone and is not influenced by breathing rate.[23] To remove skewness, RMSSD data were transformed using a natural logarithm transformation (lnRMSSD).[14,15]

Similar to %MHR measures, HRV was calculated during the 5-minute pretest period ($lnRMSSD_{Rest}$) and in 30-second epochs (eg, $lnRMSSD_{30}$, $lnRMSSD_{60}$, and $lnRMSSD_{90}$) for a total of 20 time points during the 10-minute recovery period. We chose this 30-second epoch window because it is the smallest period for analysis that can capture levels of PSNS activity and has been used to examine postexercise vagal reactivation.[24]

Blood La^- Measures. The La^- samples were collected from a finger sample (~0.7 μL) using the Lactate Plus device (Nova Biomedical, Waltham, MA). The Lactate Plus device has been demonstrated to have adequate criterion validity[25] ($r = 0.936$) and was calibrated before data collection using 2 known control solutions (low = 1.0–1.6 mmol/L, high = 4.0–5.4 mmol/L). Consistent with HRR and HRV measures, the examiner collected La^- samples during the pretest period (La^-_{Rest}), immediately after the exercise (La^-_0), and after the 10-minute recovery period (La^-_{600}). The La^- data were not collected from 5 participants after the submaximal exercise protocol, so data from only 32 participants were included in the La^- statistical analyses of the submaximal protocol.

Statistical Analysis

To descriptively examine the ability of HR to recover after submaximal and maximal protocols, we calculated the mean %MHR and 95% confidence intervals (CIs) at each HRR measure and subsequently compared them with the mean percentage of maximal heart rate [$\%MHR_{Rest}$]. Any 95% CIs for the mean %MHR data that did not overlap with the mean $\%MHR_{Rest}$ 95% CIs were considered different and meaningfully different from rest.

Parametric repeated-measures analyses of covariance were used to identify differences among the mean HR_{∞}, HR_{amp}, and HRR_{τ} parameters associated with each protocol (submaximal and maximal). Given the potential influence of obesity on vagal tone,[26] body mass index was used as a covariate in the model. We calculated partial η^2 (η^2_p) effect sizes to determine the effect of the exercise protocol on these HRR parameters.

To descriptively examine the ability of HRV to recover after submaximal and maximal protocols, we computed the mean lnRMSSD and 95% CIs at each HRR measure and subsequently compared them with the mean $lnRMSSD_{Rest}$ data. Any 95% CIs of the mean lnRMSSD data that did not overlap with the mean $lnRMSSD_{Rest}$ 95% CIs were considered different and, thus, meaningfully different from rest.

Finally, given that La^- data demonstrated a nonnormal distribution ($W = 0.901$, $P < .001$), 2 Friedman tests and a post hoc follow-up Wilcoxon signed rank test were used to examine differences in La^- concentration at La^-_0 and La^-_{600} compared with La^-_{Rest} in the submaximal and maximal protocols.

All statistical analyses were performed using SPSS (version 25; IBM Corp, Armonk, NY). The α level was set at .05 for all parametric repeated-measures analyses of covariance and all nonparametric Friedman and Wilcoxon signed rank tests. We interpreted η^2_p effect sizes using the following criteria: $\eta^2_p < 0.06$ (*small*), $0.06 \leq \eta^2_p < 0.14$ (*medium*), or $\eta^2_p \geq 0.14$ (*large*).[27]

RESULTS

All participants successfully completed the 3-minute submaximal step test, and the average time to completion for the maximal treadmill test was 11.7 ± 1.9 minutes. The HR_0 was 140.0 ± 14.5 bpm and 181.5 ± 10.8 bpm after the submaximal and maximal test protocols, respectively. In addition, HR decreased 43.6 ± 12.1 bpm and 33.8 ± 8.2 bpm 1 minute after the submaximal and maximal tests, respectively. Based on the 95% CIs associated with the mean %MHR data, each HRR measure was different from the $\%MHR_{Rest}$ measure in both the submaximal (Figure 2A) and maximal (Figure 2B) protocols. Therefore, HR remained elevated from resting values, even after a 10-minute recovery period, following submaximal (35.4% ± 4.4% versus 43.7% ± 6.7%, respectively) and maximal (37.6% ± 5.5% versus 56.5% ± 6.3%, respectively) exercise. After controlling for the influence of body mass index, we observed differences between the submaximal and maximal protocols in the mean HR_{∞} ($F_{1,35} = 4.878$, $P =$

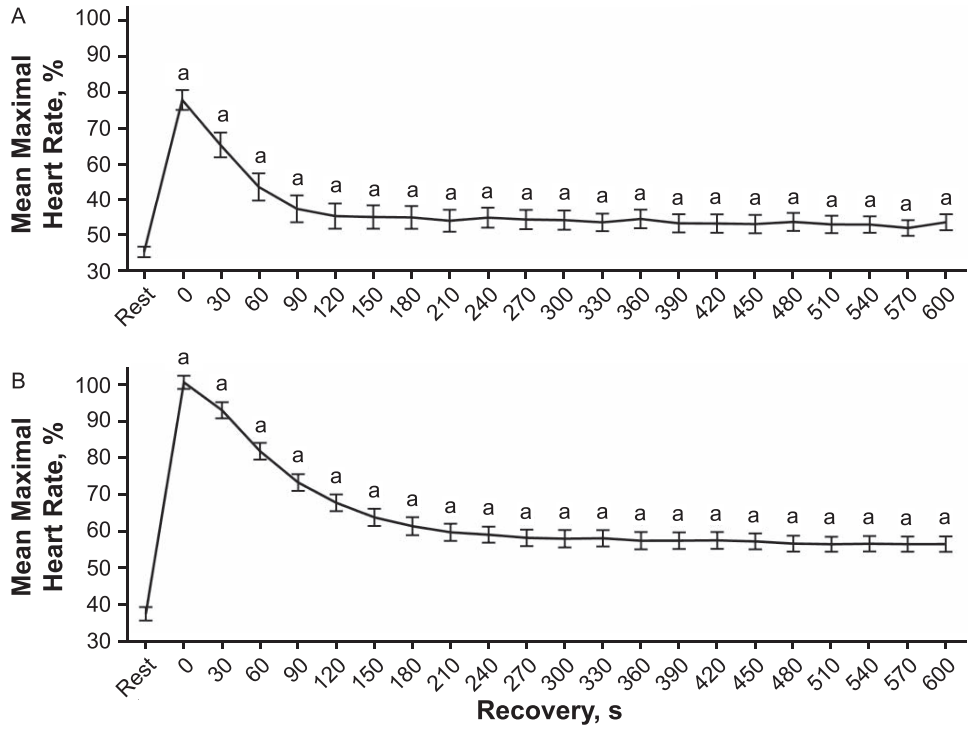

Figure 2. Changes in the percentage of maximal heart rate (%MHR) data during the 10-minute recovery window after completing, A, submaximal and, B, maximal exercise. Data are expressed as mean ± 95% confidence interval. [a] Indicates difference based on 95% confidence interval.

.034, $\eta^2_p = 0.122$), HR_{amp} ($F_{1,35} = 10.790$, $P = .002$, $\eta_p^2 = 0.236$), and HRR_τ ($F_{1,35} = 8.023$, $P = .008$, $\eta_p^2 = 0.186$), with a medium to large effect of protocol on all parameters (Table 1).

Based on the 95% CIs associated with the mean lnRMSSD data within the submaximal protocol, we found that $lnRMSSD_{30}$ and $lnRMSSD_{60}$ were different from $lnRMSSD_{Rest}$, but $lnRMSSD_{90}$, $lnRMSSD_{120}$, $lnRMSSD_{150}$, and $lnRMSSD_{180}$ were not different from $lnRMSSD_{Rest}$ (Figure 3A). Although the remaining lnRMSSD data ($lnRMSSD_{210}$ through $lnRMSSD_{600}$) were subsequently different from $lnRMSSD_{Rest}$, these results indicated that a level of ANS recovery occurred after participants completed the submaximal exercise. In contrast, based on the 95% CIs associated with the mean lnRMSSD data in the maximal protocol, each HRV recovery measure was lower than $lnRMSSD_{Rest}$ throughout the entire 10-minute recovery period (Figure 3B).

Finally, changes in La^- were identified between La^-_{Rest} and the La^-_0 and La^-_{600} measures in both the submaximal ($\chi^2 = 52.079$, $P < .001$) and maximal protocols ($\chi^2 = 61.459$, $P < .001$). Post hoc follow-up analyses indicated that the La^-_0 and La^-_{600} measures were greater than the La^-_{Rest} measures in both the submaximal ($Z = 4.872$, $P < .001$ and $Z = 4.117$, $P < .001$, respectively) and maximal ($Z = 5.304$, $P < .001$ and $Z = 5.304$, $P < .001$, respectively) protocols, demonstrating that La^- measures remained elevated after submaximal and maximal exercise (Table 2).

Table 1. Submaximal and Maximal Heart Rate Recovery Parameters[a]

Variable	Exercise Protocol, beats/min	
	Submaximal	Maximal
Resting heart rate	63.9 ± 7.4	67.9 ± 9.3
Heart-rate asymptote[b]	77.8 ± 12.4	101.6 ± 12.0
Heart-rate amplitude[b]	63.8 ± 9.3	84.2 ± 11.3
Heart-rate decay[b]	54.9 ± 26.1	91.8 ± 15.6

[a] Values are mean ± SD.
[b] Indicates difference between submaximal and maximal protocols ($P < .001$).

DISCUSSION

Our results suggest that the recovery patterns of active-duty firefighters differed after submaximal versus maximal exercise. The difference between the respective submaximal and maximal HR_{amp} and HR_∞ parameters indicated that, although participants demonstrated a greater change in %MHR during the 10-minute recovery after the maximal exercise protocol, they were still less recovered after this recovery period, as HR remained higher during the maximal protocol than during the submaximal protocol after 10 minutes of recovery (ie, HR_∞; Table 1). Furthermore, the difference between the exercise protocols for the HRR_τ parameters suggested that the exponential decay in HR across time was faster after submaximal than after maximal exercise. Unlike the HRR results, the HRV results reflected a dissociation in recovery between the submaximal and maximal tests: lnRMSSD increased after the submaximal protocol and remained decreased after the maximal protocol. These results suggest that the ANS contribution to recovery differed between the submaximal and maximal tests.

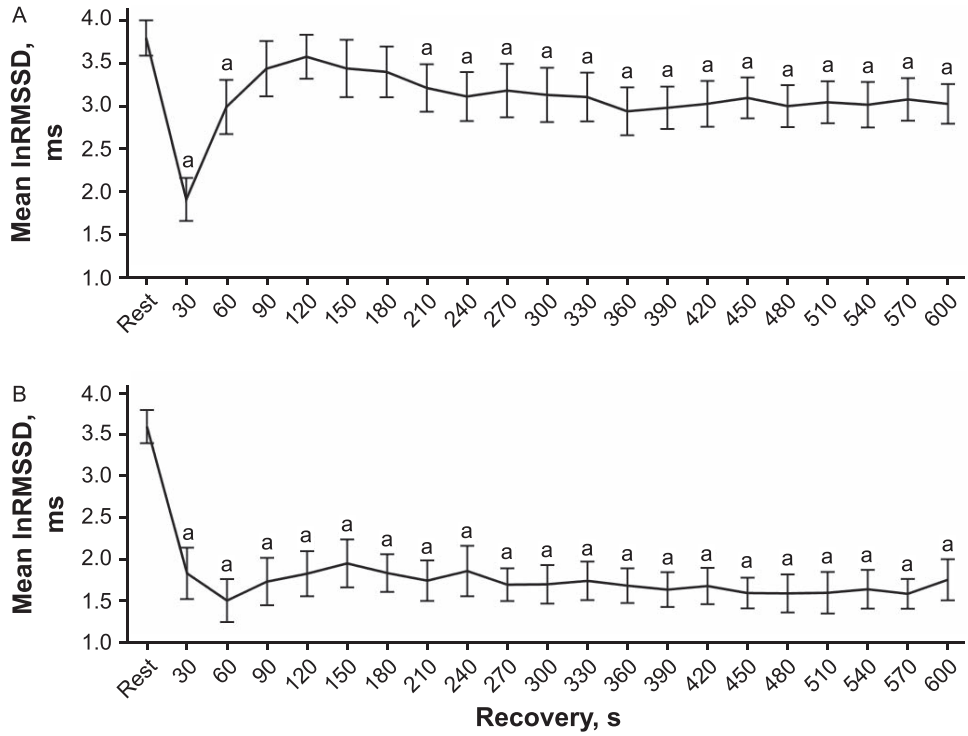

Figure 3. Changes in the natural logarithm of the square root of the mean sum of the squared differences (lnRMSSD) between R-R intervals (ie, time between beats, which is used to calculate heart rate) data during the 10-minute recovery window after completing, A, submaximal and, B, maximal exercise. Data are expressed as mean ± 95% confidence interval. [a] Indicates difference based on 95% confidence interval.

Heart-Rate Recovery and Heart-Rate Variability After Submaximal Exercise

The HR_0 during the submaximal protocol (140.0 ± 14.5 bpm) was consistent with that reported by researchers in previous studies of submaximal exercise in a similar population.[8,16,28,29] The submaximal HRR profiles demonstrated a rapid initial decrease in HR, followed by no change in %MHR after approximately 150 seconds (Figure 2A). After 10 minutes of recovery, the $\%MHR_{600}$ was still greater than %MHR at rest by 23%. The HRV results indicated that lnRMSSD decreased from $lnRMSSD_{30}$ to $lnRMSSD_{60}$ but then increased from $lnRMSSD_{90}$ to $lnRMSSD_{180}$ such that it was not different than $lnRMSSD_{Rest}$ (Figure 3A). However, lnRMSSD once again decreased from $lnRMSSD_{210}$ through $lnRMSSD_{600}$, with each time point being different than $lnRMSSD_{Rest}$. Collectively, these findings indicate that recovery from the submaximal test was initially a product of PSNS reactivation, but after 210 seconds, the incomplete recovery was likely due to the lack of complete SNS withdrawal.[9,12]

Table 2. Changes in Blood Lactate From Rest to Recovery[a]

	Blood Lactate, mmol/L		
Exercise Protocol	Pretest	Immediately Postexercise	After 10-min Recovery
Submaximal (n = 32)	2.1 ± 1.3	5.6 ± 1.9[b]	4.3 ± 2.3[b]
Maximal (n = 37)	2.0 ± 1.2	13.3 ± 2.3[c]	11.7 ± 2.3[c]

[a] Values are mean ± SD.
[b] Different from submaximal resting blood lactate measure ($P < .001$).
[c] Different from maximal resting blood lactate measure ($P < .001$).

Researchers[12,16] have reported fast and slow phases of HRR. The initial decline in HR was a function of PSNS reactivation and the later and slower changes in HR were a function of SNS withdrawal. In addition, elevated La^- during recovery has been suggested to prompt an extended slow phase.[9,12,28] The La^-_0 response in this study (5.6 ± 1.9 mmol/L) differed from that of Gladwell et al[8] (2.9 ± 0.5 mmol/L), perhaps because of differences in exercise (step test versus supine cycle ergometer) or duration (3 versus 20 minutes) or both. Nonetheless, our step test resulted in moderately elevated La^- levels that were greater at La^-_{600} (4.3 ± 2.3 mmol/L) than at La^-_{Rest} (2.1 ± 1.3 mmol/L), reflecting that complete SNS withdrawal may not have been possible, as supported by the lnRMSSD results, and, thus, explaining why HR_∞ was greater than $\%MHR_{Rest}$ after 10 minutes of recovery.

The pattern of lnRMSSD during recovery may also provide insight into the ANS imbalance at the end of recovery. Researchers[12,30] have postulated that HRR and HRV represent independent aspects of the PSNS contribution to recovery: HRR represents vagal (ie, PSNS) tone and HRV represents vagal (ie, PSNS) modulation. Specifically, the initial decrease in HR is a product of vagal tone, but the lack of change in lnRMSSD is a function of vagal modulation.[16,29] In our study, the decrease in HR and initial increase in lnRMSSD after the submaximal test demonstrated the onset of vagal tone, but the subsequent decrease in lnRMSSD showed that vagal modulation was unable to overcome the lack of SNS withdrawal, leading to incomplete ANS recovery, as exhibited by suppressed lnRMSSD and elevated HR_∞.

Heart-Rate Recovery and Heart-Rate Variability After Maximal Exercise

In this study, the immediate HR after maximal exercise (181.5 ± 10.8 bpm) was consistent with previous reports[16,20,24] of maximal exercise in a similar population. Although the HRR after maximal exercise displayed a greater overall change in HR than that with submaximal exercise, the elevated HR_{∞} indicated that the HR from 180 seconds to the end of the 10-minute recovery was at a higher level than the submaximal response (101.6 ± 12.0 bpm versus 77.8 ± 12.4 bpm; Table 1). Furthermore, unlike after submaximal exercise (Figure 3A), the posttest HRV ($lnRMSSD_0$) after maximal exercise remained suppressed and different from $lnRMSSD_{Rest}$ through the entire recovery period (Figure 3B). It is possible that our findings indicated a lack of PSNS reactivation, perhaps associated with elevated La^- levels.

As observed in the submaximal recovery results, the reduction in HR in the presence of depressed HRV may signify that HRR and HRV represent independent measures of cardiac ANS function.[12,16] As such, after maximal-intensity exercise, vagal tone recovery (ie, HRR) occurs without vagal modulation recovery (ie, HRV) and could explain the impaired HRV throughout the 10 minutes of recovery seen in this study and others[12,16,24] despite the reduction in HR. It is possible that, after exercise, the removal of central nervous system command reduces the SNS stimulation to the heart, which is no longer needed because the task has ended. Therefore, the re-established vagal tone (HRR) reduces HR without vagal modulation (HRV) and SNS withdrawal, suppressing lnRMSSD during the entire recovery period.

Suppressed vagal reactivation has been previously associated with an increase in metabolite accumulation, such as La^-.[8,12,28] In our investigation, La^-_0 (13.3 ± 2.3 mmol/L) and La^-_{600} (11.7 ± 2.3 mmol/L) were both greater than La^-_{Rest} (2.0 ± 1.2 mmol/L), and La^-_0 was similar to that after fire-suppression tasks (13.0 ± 3.0 mmol/L).[31] The elevated La^- may support a greater SNS influence on regulation of HR during recovery and interfere with a full transition to PSNS regulation of HR,[12] with a particularly strong influence on the slow phase of HRR.[9,23] The suppressed lnRMSSD during recovery that we identified suggests that the PSNS may not have reactivated during the 10-minute window. Furthermore, the HRR profiles revealed differences in the HR_{∞} for submaximal (77.8 ± 12.4 bpm) and maximal (101.6 ± 12.0 bpm) exercise. Freeman et al[6] proposed that an absence of PSNS tone, without the influence of a pathologic condition, results in an HR_{Rest} between 100 and 110 bpm. Therefore, the collective interpretation of the decreased HR that is still >100 bpm in the presence of suppressed lnRMSSD and elevated La^- implies that the influence of the SNS is greater than the ability of the PSNS to re-engage in HR modulation.

Practical Applications for Athletic Trainers

As more athletic trainers are employed in the fire service and working with occupational athletes,[32] their expertise will increasingly be needed to help refine postfire-recovery protocols both at the scene and after return to the firehouse. The current National Fire Protection Association[33] recommendations for recovery at an incident scene indicate that a minimum of 10 minutes of recovery, if practical, should be provided after exiting a fire for the first time and a minimum of 20 minutes should be provided after a second self-contained breathing apparatus cylinder is used. Based on the current results, regardless of exercise intensity, 10 minutes of seated recovery may not be adequate to reduce the long-term ANS risk factors for later cardiac injury. It is unclear how the ANS would respond to a repeated submaximal or maximal test, and future researchers should determine if repeated exposure to fire-suppression activities has a compound effect on ANS recovery. This information will help investigators develop best practices for facilitating ANS recovery and reduce ANS factors linked to a cardiac event after a fire call.

Related to recovery protocol development is the information used to make decisions about recovery status. Although current practices (eg, vital signs) in emergency scene management remain relevant, technological advancements allow for immediate access to information such as HRR and HRV. Our results demonstrated that, although measuring HRR is noninvasive, this information does not represent the complete story of recovery, and therefore, mismanagement of recovery may occur. For example, our maximal-exercise test showed that all participants recovered approximately 33.8 bpm during the first minute, which was within the suggested HRR values after maximal exercise.[7] However, the HRV results indicated the participants were not fully recovered from an ANS perspective. This could be a concern during actual fire-service calls in which a firefighter performs multiple bouts of fire suppression while at a location and, thus, may further exacerbate an already impaired PSNS modulation. This may not have an immediate effect, but it could become an important component of the SCD incidences that occur after a firefighter returns from a fire service call and has an ANS profile that does not favor PSNS regulation at rest. Future authors should identify methods that allow athletic trainers access to such information as part of managing recovery in firefighters and explore the need for implementing recovery protocols after return to the firehouse.

Finally, our results provide insight to support the development of recovery strategies to mitigate the risk for cardiac injury after a call. The current data are consistent with the existing literature, indicating that the greater the intensity, the greater the impairment of PSNS reactivation.[29] From a practical perspective, the firefighter's recovery may be specific to the type of service call. Kaikkonen et al[34] reported that HRV was lower after fire and rescue calls than after medical calls, which implies that firefighters may be under more SNS influences after fire calls. By acknowledging that different service calls result in different responses, recovery protocols can be adapted to match the type of call.

Limitations

The design constraints of the study did not allow us to include a control group; therefore, the results are relevant only to the firefighter population. Also, our tools did not permit examination of respiration rate and direct assessment of the SNS. However, the tools represent methods likely to be used in the field by athletic trainers or other

health care professionals. The 10-minute seated recovery period was chosen to be comparable with previous studies and is consistent with the minimum recommendations of the National Fire Protection Association. It is possible that ANS recovery would have been observed during a longer recovery period. We used a controlled laboratory approach to maximize the opportunity to understand how HRR and HRV might respond in this population. Nonetheless, researchers should examine these measures in response to actual fire-suppression activity. Lastly, all participants were male career structural firefighters, and their responses may not represent the responses of female, volunteer, or wildland firefighters.

CONCLUSIONS

Collectively, our findings suggest that the contributions of the PSNS and SNS to recovery in firefighters are likely exercise-intensity specific and could play an important role in establishing best-practice recovery guidelines. The HRR and HRV were different after activities of various intensities and illustrated that 10 minutes were not enough for firefighters to fully recover from either submaximal or maximal exercise. In addition, our results contribute to the hypothesis that SNS withdrawal after maximal exercise may be more influential during recovery than previously thought. Furthermore, our findings provide evidence that HRR and HRV represent independent measures of cardiac autonomic regulation. That is, HRR represents PSNS tone and the frequency by which the vagus nerve is stimulated, and HRV represents PSNS modulation and the balance between the PSNS and SNS influences on the heart.

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