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Season-Long Heart-Rate Variability Tracking Reveals Autonomic Imbalance in American College Football Players

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Purpose: To track cardiac-autonomic functioning, indexed by heart-rate variability, in American college football players throughout a competitive period. ***Methods:*** Resting heart rate (RHR) and the natural logarithm root mean square of successive differences (LnRMSSD) were obtained throughout preseason and ~3 times weekly leading up to the national championship among 8 linemen and 12 nonlinemen. Seated 1-minute recordings were performed via mobile device and standardized for time of day and proximity to training. **Results:** Relative to preseason, linemen exhibited suppressed LnRMSSD during camp-style preparation for the playoffs ($P = .041$, effect size [ES] = −1.01), the week of the national semifinal ($P < .001$, ES = −1.27), and the week of the national championship ($P = .005$, ES = −1.16). As a combined group, increases in RHR ($P < .001$) were observed at the same time points (nonlinemen ES = 0.48–0.59, linemen ES = 1.03–1.10). For all linemen, RHR trended upward (positive slopes, $R^2 = .02–.77$) while LnRMSSD trended downward (negative slopes, $R^2 = .02–.62$) throughout the season. Preseason to postseason changes in RHR ($r = .50$, $P = .025$) and LnRMSSD ($r = –.68$, $P < .001$) were associated with body mass. ***Conclusions:*** Heart-rate variability tracking revealed progressive autonomic imbalance in the lineman position group, with individual players showing suppressed values by midseason. Attenuated parasympathetic activation is a hallmark of impaired recovery and may contribute to cardiovascular maladaptations reported to occur in linemen following a competitive season. Thus, a descending pattern may serve as an easily identifiable red flag requiring attention from performance and medical staff.

Keywords: parasympathetic, cardiovascular, linemen, smartphone application

American college football players are exposed to frequent high-impact collisions, intensive training regimens, and high loads of static hemodynamic stress.[1] Players are further burdened with nonphysical stressors such as travel, academics, media interactions, and social-life restrictions. Decrements in a variety of health and performance markers have been observed following a competitive season,[2–4] potentially impacting postseason competition outcomes and players' long-term risk for chronic conditions.[1] Therefore, practical monitoring protocols, which mutually serve coaching and medical staff in their efforts to preserve player health and performance, require investigation.

Chronic predominance of sympathetic activation in the resting state is associated with a variety of pathological conditions.[5] Autonomic imbalance in athletes is a manifestation of chronic physical and psychological stress and represents a physiological hallmark of training fatigue.[6,7] A noninvasive measure of autonomic function is heart rate variability (HRV), which is quantified in the time domain from successive beat-to-beat fluctuations. HRV increases as a result of vagal (ie, parasympathetic) inhibitory influence on sinoatrial depolarization.[8] Parasympathetic modulation also promotes restorative processes that facilitate recovery from physical exertion.[9] Attenuated HRV is a primary characteristic of the physiological expression of stress[5] and is frequently observed with overreaching.[6] Increased HRV from parasympathetic hyperactivity may also occur during overreaching but is generally observed in endurance athletes.[6] Increasing support for the applied utility of HRV has given way to more affordable and time-efficient acquisition methodologies.[10] These advancements provide a practical means of tracking autonomic status in a large roster of players that is feasible for season-long implementation.

Football staff are challenged with managing the preparation of a highly heterogeneous group of players due to positional demands that require unique physical and performance characteristics. Notably, body mass in excess of 136 kg (300 lb) is typical of linemen. Immense size and stature provides an advantage during repeated altercations with opposing linemen. Nonlinemen are comparatively leaner and more aerobically fit to facilitate greater running demands.[2,11] Anthropometric and lifestyle characteristics predispose linemen to a variety of health conditions[12–14] that may negatively impact autonomic regulation and their capacity to adapt to in-season demands. It has recently been demonstrated that linemen exhibit significantly slower interday HRV recovery than nonlinemen.[15,16] However, the implications of these findings are unclear as longitudinal changes in HRV throughout a competitive football season have yet to be characterized.

In addition to performance staff, season-long HRV tracking may also be relevant to sports medicine personnel. Autonomic imbalance has been implicated as a potential contributory mechanism that precedes clinically relevant cardiovascular maladaptations found to occur predominantly in larger players.[1] Thus, parasympathetic hypoactivity indexed by suppressed HRV may be an easily identifiable red flag that can be used to support interventions relevant to players' health and performance. Therefore, the purpose of this study was to prospectively monitor HRV in college football players throughout an entire competitive season. We hypothesized that linemen would experience greater decrements in HRV than nonlinemen.

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Methods

Subjects

Study volunteers (n = 32) were first- or second-string members of a Division 1 National Collegiate Athletics Association program that were Football Bowl Subdivision national champions from the previous season. Players who missed ≥ 1 competition due to injury (n = 3) or did not record at least one HRV measure per time point (n = 9) were excluded from analysis. Players meeting inclusion criteria (n = 20, 80% African American) were categorized as linemen (n = 8; height = 191.6 [5.3] cm; weight = 135.2 [5.9] kg) and nonlinemen (n = 12; 187.7 [6.1] cm, weight = 96.8 [9.6] kg). Nonlinemen consisted of running backs (n = 2), linebackers (n = 2), tight ends (n = 1), receivers (n = 4), and defensive backs (n = 3). All players provided written informed consent to participate in the study prior to data collection. Ethical approval for this investigation was obtained from the University of Alabama Institutional Review Board and complied with regulations set forth by the Declaration of Helsinki.

Competitive Season

The HRV was monitored throughout the preparatory and competitive college football period during this prospective observational study. Details of the weekly training structure have been previously described.[15] After an undefeated regular season, the team earned a playoff berth as the number-one seed and advanced to the national championship (NC). Twelve regular season competitions, 8 versus top 25 nationally ranked opponents, occurred on Saturdays throughout weeks 1 to 13. Week 9 was a bye week. The conference championship (CC) occurred 1 week following the regular season. Formal team practices did not occur for 12 days following the CC, and thus HRV data were not collected. A playoff-preparation week (PP) involving 7 consecutive days of training camp-style practice preceded the week of the national semifinal (SF). The NC occurred 9 days following the SF. Intraindividual HRV measures were averaged[7] at 18 time points for analysis as follows: the preseason (PS) to represent baseline, regular season weeks 1 to 13, CC, PP, SF, and NC.

Training Load

Players (n = 15) performed all training sessions with a microsensor (Catapult Innovations, Melbourne, Australia) affixed to their shoulder pads between the scapulae in a custom-built cartridge. These devices quantify full-body acceleration in the sagittal, frontal, and vertical plane via integration of a 100 Hz triaxial accelerometer, gyroscope, and compass. Workloads from all football training sessions throughout the season were characterized with total Player Load, calculated as the square root of the sum of the squared instantaneous rate of change in acceleration in 3 planes divided by 100. This metric is a valid and reliable marker of external workload in team-sport athletes.[17] Player Load values are displayed in Figure 1 for descriptive purposes.

Heart-Rate Variability

The HRV was approximated via photoplethysmography, enabled by an optical pulse-wave finger-sensor and mobile application (ithlete™; HRV Fit LTD, Southampton, United Kingdom). This tool has shown acceptable agreement with simultaneous electrocardiographic comparisons for determining HRV parameters.[10] Five tablet devices (iPad; Apple Inc, Cupertino, CA) with finger sensors inserted into headphone slots were distributed to players seated comfortably on an athletic training table with their backs supported against the wall. Players would insert their left index finger into the cuff, select their identification from the application, and perform a supervised measurement. During HRV acquisition, players were instructed to remain quiet, motionless, and breathe naturally. Recordings were 55 seconds in duration and were preceded by at least 60 seconds for stabilization.[15,16,18] The application automatically detects and corrects for irregular interpulse intervals using the following algorithm:

$$(\mathrm{PPn} - [\mathrm{PPn} - 1])^2 < (40 \times \mathrm{Exp}[120/\mathrm{PRavg}])^2,$$

where PRavg is the average pulse rate calculated since commencement of the recording.

After processing, the application automatically displays resting heart rate (RHR) and the natural logarithm of the root mean square of successive differences (LnRMSSD). LnRMSSD represents

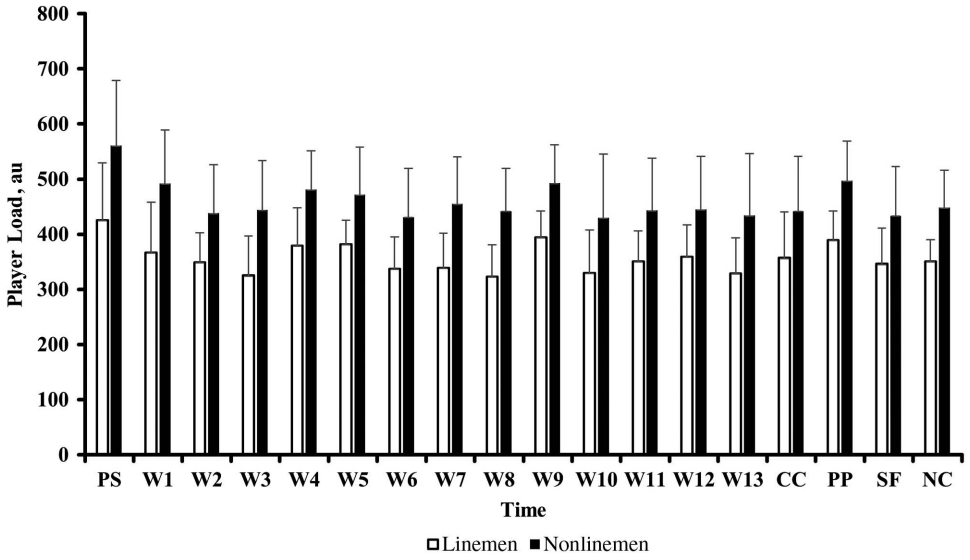

Figure 1 — Training-session-derived Player Load values for linemen and nonlinemen throughout the competitive season. CC indicates conference championship; NC, national championship; PP, playoff preparation; PS, preseason; SF, national semifinal; W, week.

parasympathetic modulation[8] and is the preferred HRV index for monitoring training adaptations in athletes.[6] Measures were consistently collected around noon before lunch to control for time of day and postprandial state. Acquisition of HRV primarily occurred Tuesday to Friday. Measures were omitted when strength and conditioning sessions were performed earlier in the day, which varied for players based on group schedules. We aimed to obtain 3 measures from each player per time point.[19]

Statistical Analyses

A total of 1311 RHR and HRV samples were collected. We obtained (mean [SD]) 18.3 (2.8) samples per player throughout PS and 2.8 (0.7) samples per player at time points thereafter (ie, week 1 – NC) for a total of 65.6 (11.2) samples per player. Normally distributed residuals for RHR and LnRMSSD were confirmed with Shapiro–Wilks tests ($P = .203$–$.775$). Linear mixed models were used to examine variation in RHR and LnRMSSD. Position (nonlinemen vs linemen) was included as a between-subjects fixed effect, time as a within-subjects repeated measure, the position × time interaction as a fixed effect, and player identification as a random effect. Tukey tests were used for post hoc analyzes. Hedges' *g* effect size (ES) ± 95% confidence interval was used to determine standardized differences.[20] All time-related comparisons were made relative to PS (ie, baseline). ES were qualitatively interpreted as small (0.20–0.59), moderate (0.60–1.19), large (1.20–1.99), and very large (≥2.00).[21] If the 95% confidence interval of the ES overlapped the trivial zone (–0.20 to 0.20), the ES was deemed unclear. Intraindividual linear regressions were performed to assess the rate of change for mean RHR and LnRMSSD across time points. Finally, changes (Δ) in RHR and LnRMSSD from PS to postregular season (ie, mean of PP, SF, NC) were calculated. Associations between Δ variables and preseason body mass were quantified with Pearson *r*. Statistical procedures were performed with JMP (version 13; SAS Institute Inc, Cary, NC). *P* values <.05 were considered statistically significant.

Results

The RHR and LnRMSSD are reported as model-adjusted least square mean (SD). A significant effect of time ($P < .0001$) was observed for RHR without an effect for position ($P = .058$) or an interaction ($P = .384$). As both groups combined (n = 20), RHR at PS (72.3 [7.9] beats·min^{-1}) was lower than PP (79.0 [8.0] beats·min^{-1}, $P = .0007$, ES = 0.79 [0.64]); SF (79.2 [7.9] beats·min^{-1}, $P = <.0001$, ES = 0.86 [0.65]); and NC (78.5 [7.9] beats·min^{-1}, $P = .0008$, ES = 0.77 [0.64]).

A position × time interaction was observed for LnRMSSD ($P = .024$). LnRMSSD at PS was greater than PP ($P = .041$, ES = –1.01 [1.05]); SF ($P < .001$, ES = –1.27 [1.07]); and NC ($P = .005$, ES = –1.16 [1.06]) for linemen. No differences were observed for nonlinemen ($P > .05$). Position × time plots for RHR and LnRMSSD are displayed in Figure 2. ES ± 95% confidence interval is presented in Table 1.

Linear trends for RHR and LnRMSSD across time were directionally homogenous, but the strength of the associations varied among players. RHR trended upward throughout the season (positive slopes) for 11/12 nonlinemen ($R^2 = .00$–$.25$) and 8/8 linemen ($R^2 = .02$–$.77$). LnRMSSD trended downward throughout the season (negative slopes) for 10/12 nonlinemen ($R^2 = .02$–$.40$) and 8/8 linemen ($R^2 = .02$–$.62$). Intraindividual slope and R^2 values from linear regressions are presented in Table 2.

Individual linemen showed varying timeframes of cardiac-parasympathetic suppression. A comparison of longitudinal LnRMSSD trends in 3 linemen are presented in Figure 3.

Preseason body mass was significantly associated with preseason to postseason changes in RHR ($r = .50$, $P = .025$) and LnRMSSD ($r = -.68$, $P < .001$) (Figure 4).

Discussion

This is the first study to document longitudinal trends in cardiac-autonomic parameters among college football players throughout an entire competitive period. In support of our hypothesis, HRV responses across time varied as a function of playing position. Unlike nonlinemen, linemen exhibited progressive autonomic imbalance throughout the latter half of the season, characterized by progressive suppression of vagal-mediated HRV.

Previous investigation into HRV responses to football training are limited to short-term training periods (≤4 wk) with aims of delineating position-based differences in day-to-day cardiac-autonomic recovery.[15,16] Linemen demonstrated progressive adaptation to football training throughout the annual cycle, reflected in progressively smaller physiological responses to recurrent training stress.[22] ES decrements in LnRMSSD observed ~20 hours post-training were qualitatively *large* during off-season spring camp,[16] *moderate* following day 1 of preseason camp,[18] and *small* following Tuesday sessions during the early competitive phase (all $P < .05$).[15] Nonlinemen showed consistently trivial to small reductions at the same time points.[15,16,18] Individual changes in LnRMSSD from pre to ~20 hours posttraining were significantly associated with body mass during spring camp ($r = -.62$, $P < .01$)[17] and the early competitive period ($r = -.39$, $P < .05$).[16] A novel finding of the current investigation is the association between body mass and longitudinal changes in LnRMSSD (Figure 4). These associations collectively indicate that anthropometric features are a determinant of both short- and long-term cardiac-autonomic reactivity to collegiate football participation. Moreover, improved interday HRV recovery demonstrated by linemen earlier in the season[15] was not maintained.

Though significant group-level reductions in LnRMSSD were not observed until postseason for linemen, some players experienced suppressed LnRMSSD at earlier time points and in the absence of intensified training (Figure 1). Exaggerated physiological responses to a recurrent stressor (eg, similar training load) or failing to turn off the response when it is no longer needed (eg, posttraining) are indicative of maladaptation.[22] Negative ES (*unclear–moderate*, Table 1) along with a descending group trend beginning in week 6 (Figure 2) correspond with the academic midterm examination period and the first of 5 consecutive conference match-ups against top 25 nationally ranked opponents. We hypothesize that the descending LnRMSSD pattern in linemen may partly be explained by a progressive increase in allostatic load. Allostatic load represents the cumulative physiological effects of stress-related factors in response to daily demands.[23] Sustained activation of the hypothalamic–pituitary–adrenal and sympathetic systems (ie, stress response mediators) characterize the allostatic state, which can be reflected in suppressed HRV.[5,23] Heightened vigilance is a short-term adaptive solution to persistent taxing of body resources in an effort to maintain normal functioning.

The list of variables contributing to allostatic load may be more extensive for linemen. Higher body and fat mass are chronic contributors,[5,23] worsened by associated sleep (disordered breathing)[24], dietary (high intake of empty calories to maintain or gain

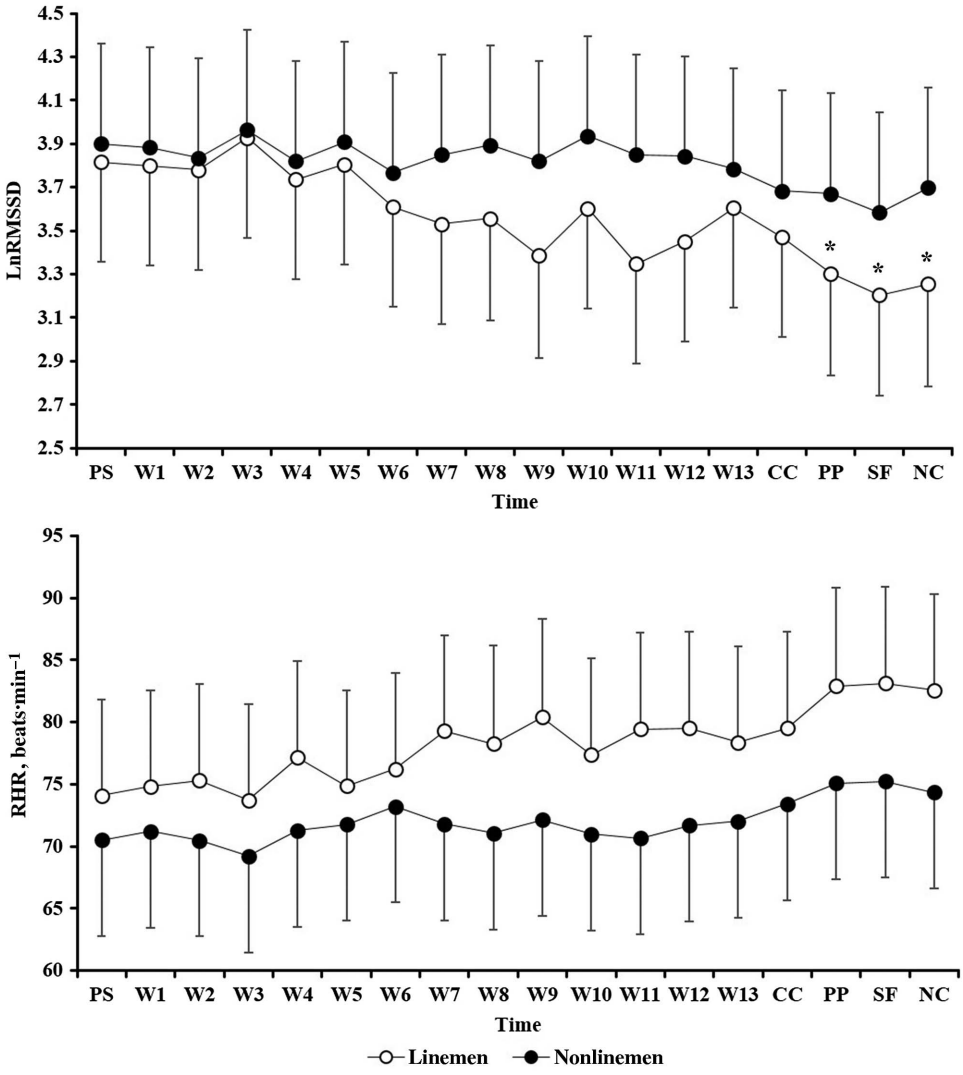

Figure 2 — Position × time plots for RHR and LnRMSSD. CC indicates conference championship; NC, national championship; LnRMSSD, natural logarithm of the root mean square of successive differences; PP, playoff preparation; PS, preseason; RHR, resting heart rate; SF, national semifinal; W, week. *Different from PS ($P < .05$).

weight)[14], and metabolic factors (visceral adiposity, dyslipidemia, hyperglycemia, and insulin resistance).[5,13,23] In addition, aerobic fitness functions as an important stress-buffering mechanism[9,25] and is generally lower in linemen versus nonlinemen[2] or may decline throughout the season. Thus, although players are subjected to matched training and academic schedules, linemen may be disadvantaged by an unmatched or more highly taxed coping capacity. Autonomic strain may be further compounded in linemen by chronic damage-associated inflammation from repetitive musculoskeletal trauma[26] and a higher exposure to subconcussive impacts that promote diffuse axonal injury and neuroinflammation.[27] It has been shown that average peak linear acceleration per impact explains 15% of the variance in preseason to postseason changes in RMSSD.[28] The physical and psychological demands of regular-season participation in a highly competitive conference may therefore be sufficient for altering autonomic regulation in some linemen.

Two linemen exhibiting suppressed LnRMSSD experienced upper-extremity injuries in the same competition (Figure 3). A precautionary and temporary cessation of training (3 sessions) for one player had a restorative effect on LnRMSSD, whereas the player with immediate return-to-play clearance maintained attenuated values and subsequently became ill. A similar LnRMSSD pattern (7-d rolling average, slope = –0.17, $R^2 = .88$) preceded nonfunctional overreaching and compromised immune functioning in an elite triathlete during an intensive preparatory period.[7] Progressive attenuation of LnRMSSD observed here and elsewhere[7] graphically resembles 1 of the 4 models of allostatic load, reflecting a prolonged response due to inadequate recovery.[22] Applying this model to the current case examples, routine training and competing may have come at a progressively greater cost for first stringers, necessitating lengthier recovery periods to restore cardiac-parasympathetic activity. Better maintenance of values in the nonstarter who received minimal or no playing time in matches attests to a high toll of competition for starting linemen. We emphasize that substantial alterations in LnRMSSD were observed in key players (Figure 3) despite no spike in Player Load (Figure 1). Thus, autonomic responses to apparently similar demands can change throughout the season, occur at varying time points among players of the same position group, and may carry infection risk

Table 1 Position × Time ES ± 95% CI for Resting Heart Rate and LnRMSSD

	Nonlinemen		Linemen	
Time	Resting heart rate	LnRMSSD	Resting heart rate	LnRMSSD
PS vs				
Week 1	0.09 ± 0.80	–0.04 ± 0.80	0.09 ± 0.98	–0.04 ± 0.98
Week 2	0.00 ± 0.80	–0.15 ± 0.80	0.15 ± 0.98	–0.08 ± 0.98
Week 3	–0.16 ± 0.80	0.13 ± 0.80	–0.05 ± 0.98	0.21 ± 0.98
Week 4	0.10 ± 0.80	–0.17 ± 0.80	0.37 ± 0.98	–0.16 ± 0.98
Week 5	0.16 ± 0.80	0.02 ± 0.80	0.09 ± 0.98	–0.04 ± 0.98
Week 6	0.34 ± 0.81	–0.27 ± 0.80	0.26 ± 0.98	–0.43 ± 0.99
Week 7	0.16 ± 0.80	–0.10 ± 0.80	0.64 ± 1.00	–0.60 ± 1.00
Week 8	0.07 ± 0.80	–0.02 ± 0.80	0.50 ± 0.99	–0.53 ± 1.00
Week 9	0.20 ± 0.80	–0.17 ± 0.80	0.76 ± 1.01	–0.88 ± 1.03[a]
Week 10	0.06 ± 0.80	0.08 ± 0.80	0.40 ± 0.99	–0.45 ± 0.99
Week 11	0.02 ± 0.80	–0.10 ± 0.80	0.65 ± 1.00	–0.97 ± 1.04[a]
Week 12	0.15 ± 0.80	–0.13 ± 0.80	0.66 ± 1.00	–0.76 ± 1.01
Week 13	0.19 ± 0.80	–0.24 ± 0.80	0.53 ± 0.99	–0.45 ± 0.99
CC	0.36 ± 0.81	–0.46 ± 0.81	0.66 ± 1.00	–0.72 ± 1.01
PP	0.57 ± 0.82	–0.48 ± 0.81	1.07 ± 1.04[a]	–1.05 ± 1.05[a]
SF	0.59 ± 0.82	–0.66 ± 0.83[a]	1.10 ± 1.05[a]	–1.27 ± 1.07[b]
NC	0.48 ± 0.81	–0.42 ± 0.81	1.03 ± 1.04[a]	–1.16 ± 1.06[a]

Abbreviations: CC, conference championship; CI, confidence interval; ES, effect size; LnRMSSD, natural logarithm of the root mean square of successive differences; NC, national championship; PP, playoff preparation; PS, preseason; SF, national semifinal. Note: Absence of superscripted lettering reflects an unclear effect.
[a] Moderate effect size. [b] Large effect size.

Table 2 Linear Regression for Mean Resting Heart Rate and Mean LnRMSSD Across All 18 Time Points

	Nonlinemen					Linemen			
	Resting heart rate, beats·min^{-1}		LnRMSSD			Resting heart rate, beats·min^{-1}		LnRMSSD	
Player	Slope	R^2	Slope	R^2	Player	Slope	R^2	Slope	R^2
1	0.10	.04	0.00	.00	1	0.14	.06	–0.02	.19
2	0.19	.12	–0.02	.16	2	0.63	.57*	–0.04	.55*
3	0.42	.20	–0.02	.22	3	0.12	.01	–0.01	.08
4	0.30	.18	–0.02	.26*	4	0.37	.41*	–0.03	.46*
5	0.01	.00	–0.01	.02	5	0.85	.50*	–0.07	.53*
6	0.17	.03	–0.01	.04	6	0.68	.40*	–0.04	.61*
7	0.16	.15	–0.01	.03	7	1.12	.77*	–0.06	.62*
8	0.29	.20	–0.02	.13	8	0.07	.02	–0.01	.02
9	0.36	.19	–0.02	.23*					
10	0.59	.25*	–0.03	.15					
11	–0.07	.01	0.01	.01					
12	0.23	.18	–0.03	.40*					

Abbreviation: LnRMSSD, natural logarithm of the root mean square of successive differences.
*Statistically significant ($P < .05$).

implications.[7] The evolving status of the autonomic nervous system therefore warrants consideration within load management strategies at the individual level.

Significant group-level reductions in LnRMSSD for linemen were first observed in response to 7 consecutive days of training camp–style practices in preparation for the College Football Playoff (PP). Typical in-season training weeks involved fewer total and full-contact training sessions relative to PP. Cardiac-autonomic responses to preseason training camp were dissimilar from responses observed postseason.[18] Despite reporting increases in perceived fatigue, maintenance or improvements in RHR and LnRMSSD following day 2 were observed throughout a 13-day camp, performed in hot and humid conditions (heat index = 38.1 °C [6.5 °C]). It was hypothesized that heat acclimation responses, related to

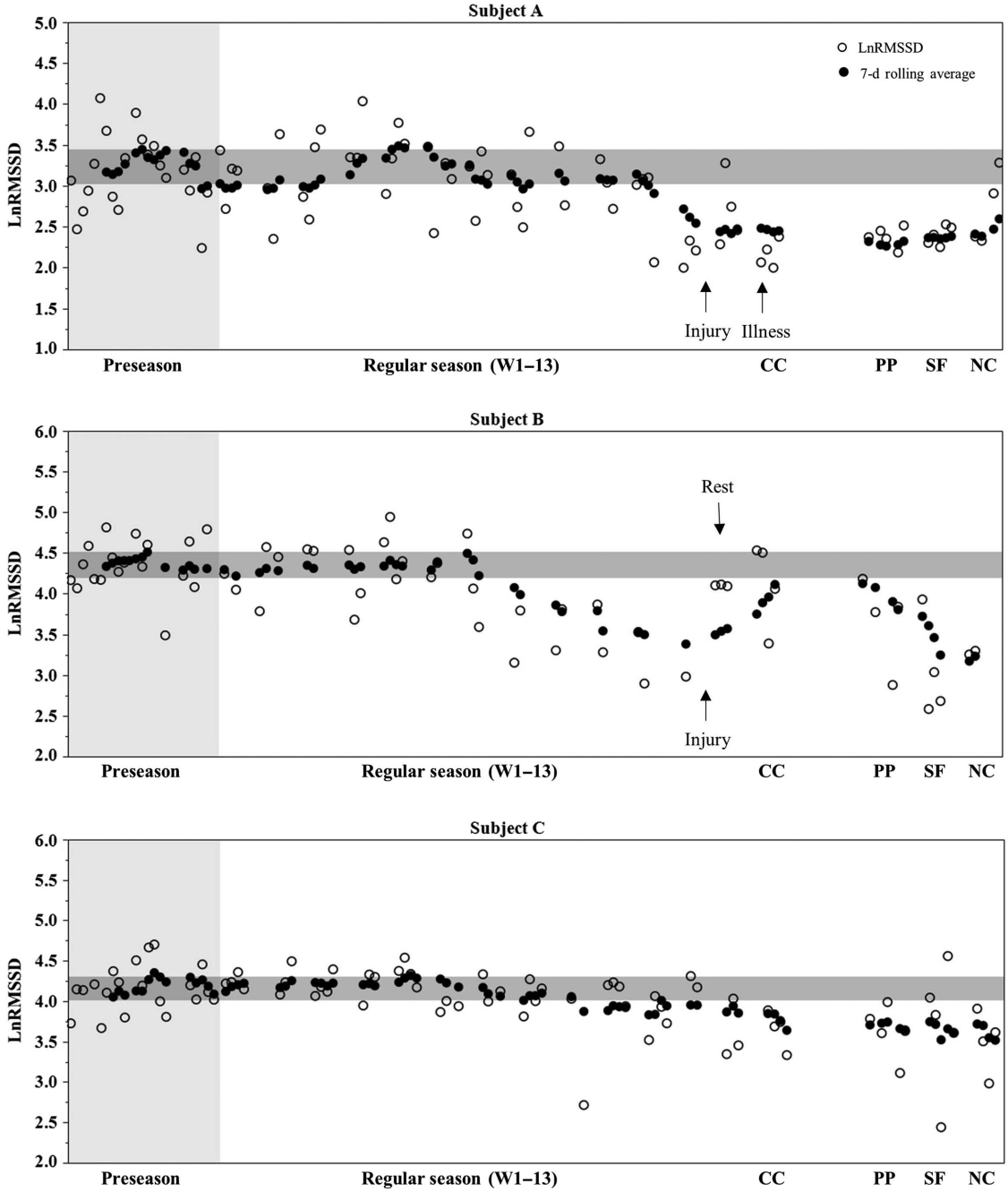

Figure 3 — Daily and 7-day rolling average LnRMSSD among offensive linemen who were first- (Subjects A and B) or second-string (subject C) players. Subjects A and B experienced upper-extremity soft-tissue injuries during the same competition. Subject B was rested for several days as a precaution while Subject A was cleared to resume full-contact participation. LnRMSSD for Subject B improved during his rest period while LnRMSSD for Subject A remained suppressed. In the subsequent week, Subject A reported an illness to the sports-medicine staff and was diagnosed with a bacterial infection by the team physician. Subject C received minimal playing time in competitions and demonstrated better maintenance of LnRMSSD throughout the regular season. Horizontal shaded area represents meaningful change thresholds, calculated as ±0.5 of intraindividual preseason SD.[7] CC indicates conference championship; NC, national championship; LnRMSSD, natural logarithm of the root mean square of successive differences; PP, playoff preparation; SF, national semifinal; W, week.

plasma volume expansion and improved aerobic fitness, preserved or improved RHR and HRV and potentially masked fatigue-related decrements.[18] Players are also fresher and healthier when entering camp-style training at preseason versus postseason, likely impacting their ability to adapt and recover. Thus, residual fatigue from the regular season along with differences in environmental conditions may explain the discrepancy in cardiac-autonomic responses observed at PP relative to preseason camp.

The LnRMSSD remained altered for linemen during weeks preceding the SF and the NC. Evidence of smaller alterations in nonlinemen were also observed (Table 1). These postseason college football matches draw intense media attention (eg, press

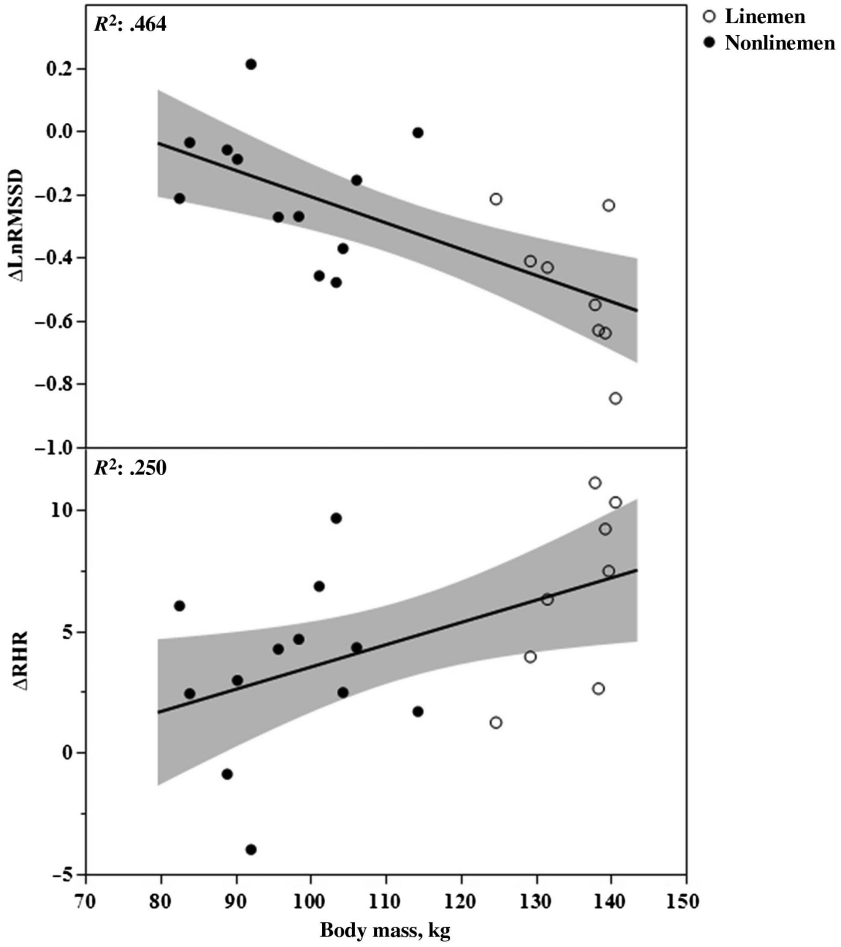

Figure 4 — Scatterplots representing the association between preseason body mass and ΔRHR and ΔLnRMSSD from pre–regular season to post–regular season. ΔLnRMSSD indicates changes in the mean natural logarithm of the root mean square of successive differences; ΔRHR, changes in mean resting heart rate.

conferences, interviews) and are broadcasted to a large international audience, increasing pressure to perform. Allostatic load encompasses mental stress and anxiety that occurs in anticipation of high resource allocation,[22,23] likely to occur in advance of high-stake matches that threaten competitive elimination. Thus, psycho-emotional factors, concurrent with physical factors described above, may have contributed to the current findings.

A consequence of chronic upregulation of the stress response resulting from allostatic load is wear-and-tear on effector organs, including the vasculature and myocardium.[5,22,23] Evidence of potential autonomic imbalance and end-organ effects among linemen following a single competitive football season have previously been observed. Relative to preseason, postseason testing revealed increases in RHR and systolic and diastolic blood pressure among first-year linemen ($n = 64$, $P < .001$).[4] Linemen also displayed increased left ventricular concentric hypertrophy with changes in ventricular mass being associated with changes in systolic blood pressure ($r = .46$, $P < .001$).[4]A subsequent investigation determined that myocardial remodeling in linemen is pathologic rather than adaptive.[29] Kim et al[3] reported significant increases in systolic blood pressure ($P < .001$) and central pulse pressure ($P = .004$) of linemen but not of nonlinemen, following a competitive season.

In nonathletic adults, attenuated HRV is associated with elevated blood pressure, and it is theorized that altered autonomic functioning precedes hypertension.[30] Thus, unabated autonomic imbalance, reflected in sustained decrements in LnRMSSD, may be one of the earliest indications of impending arterial stiffening and adverse cardiac morphology in linemen from chronic pressure overload.[4] Therefore, we propose that identifying autonomic imbalance as it occurs (eg, Figure 3) and intervening with restorative modalities may serve as a potential strategy for mitigating the cascading cardiovascular maladaptations that occur in players throughout a season. Investigation into this hypothesis is an important next step for future research.

This study is limited by sample size, lack of athletic performance assessment, and lack of clinical markers of cardiovascular health. Thus, it is unclear whether individuals with sustained reductions in LnRMSSD were overreaching, or exhibited more adverse cardiovascular or neurological changes relative to players with a more stable pattern. Data collection with a mobile application at the training facility, as opposed to postwaking, provides suboptimal conditions for HRV assessment. However, without tools that facilitated expedient data collection procedures with minimal compliance demand from the players, near-daily monitoring would have been unfeasible. HRV was not obtained on the day following matches. Thus, altered values reported in this investigation exclude acute effects of competition. Further research to elucidate causal mechanisms of autonomic imbalance in linemen is necessary to

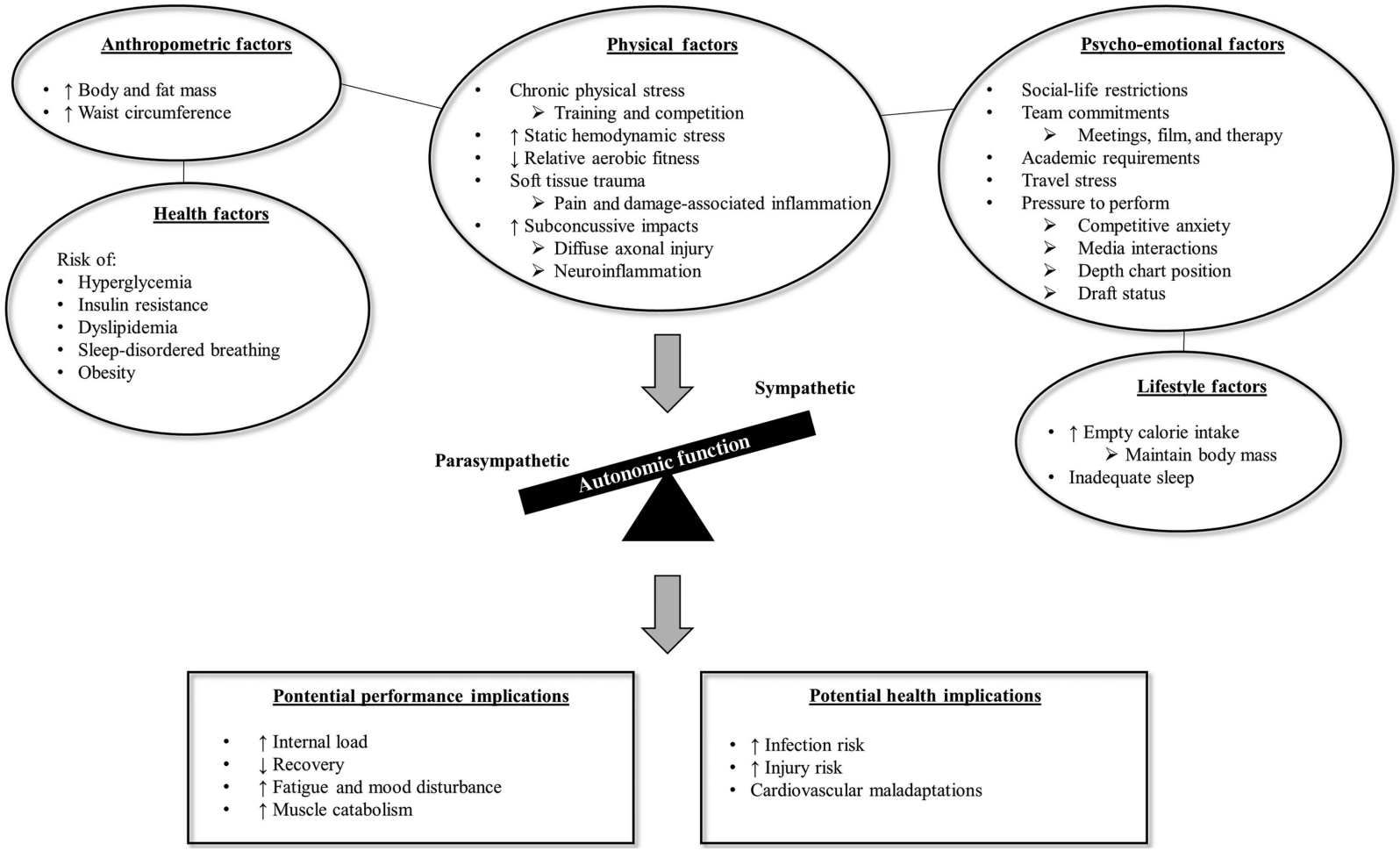

Figure 5 — Summary of factors hypothesized to contribute to autonomic imbalance in linemen throughout a competitive season and their potential implications for health and performance. The extent to which any factor contributes to alterations in heart-rate variability throughout a season is likely individual. Nonlinemen are exposed to similar physical, psychoemotional, and lifestyle factors.

develop preventative and restorative strategies. Effective interventions may involve addressing existing health conditions, emphasizing low-impact aerobic exercise in the off-season, implementing recovery modalities that stimulate parasympathetic activity, and workload management to facilitate cardiac-autonomic recovery.

Practical Applications

Serial tracking of ultrashort-term HRV with mobile devices at the training facility enabled timely detection of cardiac-parasympathetic suppression among individual players. Factors hypothesized to contribute to autonomic imbalance in linemen and their potential implications for health and performance are summarized in Figure 5. Implementation of recovery modalities and lifestyle practices that aim to maintain parasympathetic activation in linemen may limit sustained decrements throughout the season. Due to differences in health conditions, body mass, physical demands, and aerobic fitness among position groups, periodic modification of training frequency or intensity may be required to manage the season-long impact of football participation on autonomic functioning in linemen. Elevations in RHR conceivably predispose players to concomitant elevations in exercising heart rate. Thus, such players may experience increased physiological strain (ie, greater internal load) during a typical training session throughout the mid-to-late competitive period. In addition to being a hallmark of impaired recovery, parasympathetic hypoactivity may contribute to cardiovascular maladaptations reported to occur in linemen following a competitive season. Though a better understanding of the health and performance ramifications of suppressed HRV in linemen is needed, a descending pattern in daily values may serve as an easily identifiable red flag requiring attention from performance and medical staff.

Conclusions

Tracking HRV in college football players throughout a competitive season revealed progressive autonomic imbalance among the linemen position group. Although significant reductions in LnRMSSD were not observed until the postseason, individual players experienced sustained decrements at earlier time points and in the absence of intensified training. These findings highlight potential deficiencies in or greater taxation to the coping capacity of linemen during the competitive phase. Serial HRV tracking facilitated timely detection of suppressed values, indicating a potential role for mobile devices in guiding and monitoring interventions relevant to players' health and performance.

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