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Original research

Effects of head impact exposure on in-game heart rate dynamics in Canadian varsity football athletes

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ABSTRACT

Objectives: To characterize the association between head impact exposure and heart rate dynamics during varsity football games.
Design: Prospective observational study following a convenience sample of 72 athletes over two seasons.
Methods: Athletes wore instrumented mouthguards and chest-strap sensors to record head impact exposure, heart rate, and velocity. After filtering for physiologically relevant recovery segments ($n = 7693$), linear mixed-effects models assessed associations between head impact exposure and heart rate dynamics, controlling for physical exertion and inter-individual variability. Exploratory non-linear associations were modeled using generalized additive mixed models.
Results: Greater acute impact magnitude was associated with elevated heart rate and delayed time to peak heart rate recovery ($p < 0.001$), independent of running speed. Random slopes analysis further indicated substantial inter-individual variability in the immediate cardiac response to impacts. Higher accumulated session-wide impact exposure demonstrated a non-linear relationship with heart rate recovery speed; recovery dynamics remained preserved at lower exposure levels (i.e., ≤ 200 g) but were progressively attenuated at higher cumulative loads (i.e., > 200 g).
Conclusions: Head impact exposure is associated with systematic alterations in heart rate recovery dynamics during live sport participation. These findings identify cardiovascular recovery as a sensitive, non-invasive physiological signal linked to head impacts, independent of measured locomotor workload variables. While clinical outcomes were not assessed, the results demonstrate the feasibility of integrated wearable monitoring to characterize physiological strain and motivate future studies linking impact history with functional and clinical endpoints.

Practical implications

- Harder head impacts during games were linked with athletes' heart rates staying higher for longer after intense efforts.
- As head impacts accumulated across a game, athletes' heart rates recovered more slowly, especially after higher overall contact loads.
- Wearable sensors used during games were able to detect these changes, demonstrating a practical way to track how athletes are responding to repeated contact in games.

1. Introduction

Contact sports such as American football pose significant challenges with regard to health benefits versus injury risk.[1] Head impacts occur on a regular basis during practices and games and can lead to short- and long-term brain dysfunctions. Evidence suggests that sport-related head impact exposure (HIE) induces graded neurometabolic and vascular perturbations.[2] While the hallmark neurometabolic cascade is most acutely observed in clinical concussions, repetitive sub-clinical loading is thought to exert a cumulative strain on cellular metabolism and neural networks, where the physiological response scales with both the magnitude and temporal density of the impacts.[3] Consequently,

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changes in cerebral perfusion, functional connectivity, and white matter integrity – as revealed by advanced neuroimaging techniques – have been shown to correlate with impact magnitude.[4,5] Consistent with these findings, acute changes in brain function have also been observed following head impacts sustained during a single football game, including altered cortical excitability in non-concussed athletes relative to non-impact controls.[6]

These studies, which aim to distinguish the effects of HIE from those of clinically diagnosed concussion, have primarily centered on brain-specific outcomes. However, the potential interaction between HIE and other physiological systems has received comparatively less attention. The cardiovascular system, for example, is intricately linked to cerebral function through the autonomic regulation of blood pressure. The autonomic nervous system (ANS), consisting of the sympathetic and parasympathetic branches, is essential for maintaining homeostasis by modulating physiological responses to internal and external stimuli.[7] The ANS innervates cardiac muscle and regulates peripheral arterial stiffness, thereby playing a crucial role in sustaining cerebral perfusion.[7] A reduction in cerebral blood flow is a defining feature of the neurometabolic cascade and has been associated with persistent post concussive symptoms.[7] Nevertheless, the role of the ANS is often underrepresented in studies investigating the effects of repeated head impacts. This stands in contrast to the broader literature on mild traumatic brain injury (mTBI), which consistently highlights autonomic dysregulation as a consequence of concussive injuries.

Autonomic nervous system dysfunction following concussion has been documented in over 30 studies using assessments such as heart rate variability (HRV), the Valsalva maneuver, and head-up tilt testing and is considered a "likely" consequence of concussion according to American Academy of Neurology guidelines.[7] In athletic populations, concussions are commonly associated with reductions in HRV, consistent with parasympathetic withdrawal, particularly during exertional or challenge-based tasks rather than at rest.[8–10] Although HRV is widely used to assess autonomic regulation, it is strongly influenced by absolute heart rate (HR), which can complicate interpretation of parasympathetic tone and contribute to inconsistent findings across studies. Indeed, HRV does not consistently distinguish concussed from non-concussed athletes, highlighting the need for complementary autonomic markers that are less sensitive to baseline heart rate and more responsive to dynamic physiological stressors.[10,11]

Heart rate recovery (HRR) has emerged as a robust indicator of parasympathetic (vagal) branch reactivation and is closely associated with independent measures of aerobic fitness, including maximal oxygen uptake and anaerobic threshold.[12] The post-exercise HRR period reflects a complex interplay between the re-engagement of parasympathetic influence and the withdrawal of sympathetic activity.[13] As such, HRR characteristics are widely used to assess exercise intensity, training load, and fatigue.[14,15] For instance, lower absolute HR values and faster HRR are indicative of higher exercise capacity and training status, whereas slower recovery and elevated post-exercise HR suggest detraining or impaired vagal tone.[10,12,16] In athletic populations, HRR analyses are well suited for real-time assessment of physiological responses to acute stressors, including head impacts. Alterations in HRR dynamics following impacts may be consistent with changes in autonomic regulation, suggesting a potential marker of early physiological perturbation prior to clinical symptom onset.

Among contact sports, football offers a particularly relevant model for investigating the brain–heart interaction, as it combines high-frequency HIE with intense cardiovascular demands. In Canadian football, players sustain an average of 42 head impacts per game, with a mean impact magnitude of approximately 20g.[17] The sport's structure – characterized by repeated bursts of high-intensity activity, such as sprints during plays – places substantial demands on the cardiorespiratory system, necessitating rapid HR adjustments during both exertion and recovery phases. Consequently, the analysis of HR dynamics during post-play recovery periods may serve as a sensitive and ecologically valid marker of ANS function in this athletic context. However, accurately assessing the "dose" of HIE that triggers these autonomic shifts requires accounting for the temporal distribution of hits. While established biomechanical standards like the Head Impact Criterion (HIC)[18] or Impact Density[19] recognize that injury risk is time-dependent, they often focus on isolated kinematic windows. There remains a critical need to account for cumulative "burden" across multiple biological time scales, similar to neuromuscular fatigue frameworks, where the systemic response is governed not just by the magnitude of a stimulus, but by the residual load remaining from previous insults.[20,21]

The objective of this study was to examine the extent to which HIE during Canadian university-level varsity football games influences HR dynamics, specifically seeking to determine if a dose–response relationship exists between impact exposure and autonomic regulation. Our a-priori hypothesis was that increased acute and cumulative temporal density of hits would be associated with impaired recovery dynamics, characterized by elevated HR at recovery onset and slower HRR. We further explored whether the accumulated magnitude of impact exposure over a session may lead to a disproportionate collapse in parasympathetic reactivation, resulting in a "tipping point" beyond which HRR is compromised. By distinguishing between linear trends and non-linear thresholds, this approach identifies how the temporal density of HIE, rather than just isolated kinematic magnitudes, governs the transition from physiological strain to autonomic dysfunction.

2. Data & methods

2.1. Study design and participants

This prospective observational study was designed to investigate the effect of HIE on cardiovascular kinetics in varsity Canadian football players. A convenience sample of 72 university-level male athletes who agreed to participate was recruited from three teams in the Canadian Varsity Football League during the 2021 and 2024 seasons. This recruitment was part of a research program focused on implementing multimodal monitoring of biomechanics and physiological data in collision sports. Athletes received no particular guidance on their caffeine consumption but adhered to the hydration and nutrition protocols established by their team nutritionist. The study was approved by the local ethics committee and written informed consent from individual participants and the football clubs was obtained prior to data collection. The research adhered to the ethical standard outlined in the Declaration of Helsinki.[22]

2.2. Head impact exposure and heart rate monitoring

Measurements were conducted over the course of 32 games of the 2021 and 2024 seasons from August to November of each year. Game duration (between official kickoff and end of game) ranged between 143 min and 211 min (mean 169 min, SD 27 min). Each athlete wore two wearable sensors simultaneously: (1) a Catapult Vector S6 sensor (Catapult, Australia) integrated into a customized chest strap for recording HR, rigid-body accelerations, velocity, and position as time series; and (2) a Vector (Athlete Intelligence(©), USA)[23] or a Prevent (Prevent Biometrics(©), USA)[24] model instrumented mouthguard to record impact events. Mouthguards were individually fitted using a standard boil-and-bite process and specifically engineered to allow for unobstructed speech and respiration.

Regarding the Catapult system, chest-based electrodes, similar to ECG, were used to capture the heart's electrical activity and determine heart rate in beats per minute.[25] HR and speed (from GPS) were recorded with a sampling frequency of 10 Hz using the OpenField version 3.3.1.6805 software manager (device firmware version: 8.01). Head impact data were collected using the mouthguard, which continuously monitored acceleration and orientation parameters. Impact events were automatically triggered and recorded when linear acceleration exceeded a threshold of 10g. Medical records and team injury reports were reviewed to identify any clinical diagnoses of concussion during the study period. No

athletes included in the current analysis were diagnosed with a concussion or acute head injury by team medical staff during the games monitored.

2.3. Head impact filtering

The initial data processing phase involved a comprehensive screening of recorded head impacts, utilizing GPS positioning data to verify the spatial context of each event (Fig. 1). To ensure the integrity of the dataset, a tiered spatial-kinematic filter was applied based on the athlete's proximity to the field boundaries. Impacts occurring within the "inner field" (defined as the area greater than 2 m from any boundary) were automatically retained in the study. For events occurring outside of the inner field (±2 m of the field perimeter or beyond the field lines), a secondary verification process was triggered to mitigate the effects of the GPS horizontal error, which typically remains under 2 m for 10 Hz units.[26] These peripheral impacts were only included if the athlete was positioned on the field and maintained a velocity exceeding 10 km/h within the 5 s preceding the impact event. This specific speed threshold corresponds to low-to-medium intensity running in Canadian football,[27] serving as a reliable indicator of active movement intent, while the five-second window aligns with the average duration of a collegiate football play.[28] This dual-layered verification scheme preserves the inclusion of low-velocity events in the inner field, such as line-of-scrimmage pile-ups, while filtering out sideline activity without excluding impacts involving actively moving athletes near the boundaries. Due to the lack of available full-game footage, no video verification was performed.

2.4. Quantifying head impact exposure

To characterize head impact exposure across multiple physiological timescales, we developed two complementary impact exposure metrics based on recursive exponential decay (Fig. 1). Rather than relying on discrete impact counts or unweighted cumulative sums, this formulation approximates a temporal summation effect where physiological responses may depend on recent exposure history. The selected decay time constants were defined to reflect competition-relevant temporal windows for weighting prior impact events.

1. Play-scale HIE (HIE_P): time constant of $\tau = 60$ s was selected to capture high-density clusters of head impacts occurring over short intervals, such as those arising during consecutive plays separated by typical rest periods of approximately 36.1–46.9 s.[28] This timescale aligns with the rapid phase of post-exertional autonomic recovery, during which transient parasympathetic reactivation is observed,[29] and thus also provides a physiologically relevant window for aggregating closely spaced impact events.
2. Session-scale HIE (HIE_S): A longer time constant ($\tau = 3$ h) was selected to represent session-scale accumulation of head impact exposure across the duration of a game. Unlike a simple cumulative sum, which weights all impacts equally regardless of timing, the recursive decay formulation progressively downweights earlier impacts while prioritizing more recent exposure. This approach distinguishes between early- and late-game exposure profiles that would otherwise appear equivalent under a simple cumulative count. It allows earlier impacts to retain partial influence over extended periods, consistent with evidence that physiological and neurological responses to repeated head impacts may evolve over the course of a game rather than fully resolving between isolated events.[3] In contrast to the HIE_P, which emphasizes short-term clustering, HIE_S captures the integrated history of impact exposure as play progresses.

Both metrics were calculated using the recursive formula:

$$HIE_{\text{current}} = \left(HIE_{\text{prior}} \cdot e^{-\Delta t/\tau}\right) + \text{Magnitude}_{\text{new}}$$

where:

- HIE_{current} is the updated impact exposure at the time of the latest event;
- HIE_{prior} is the exposure value calculated at the time of the previous event;
- Δt is the time interval elapsed between the prior and current impacts;
- τ is the timescale-specific decay constant (60 s for HIE_P; 3 h for HIE_S);
- $\text{Magnitude}_{\text{new}}$ is the peak linear acceleration (PLA) of the current impact.

2.4.1. Magnitude selection and data integrity

While rotational kinematics are a recognized factor in head injury biomechanics, this study utilized peak linear acceleration (PLA) to represent impact magnitude. This choice was informed by considerations of longitudinal data consistency across multiple hardware versions. Specifically, analysis of our longitudinal dataset revealed that while mean magnitudes for both linear and rotational metrics were consistent

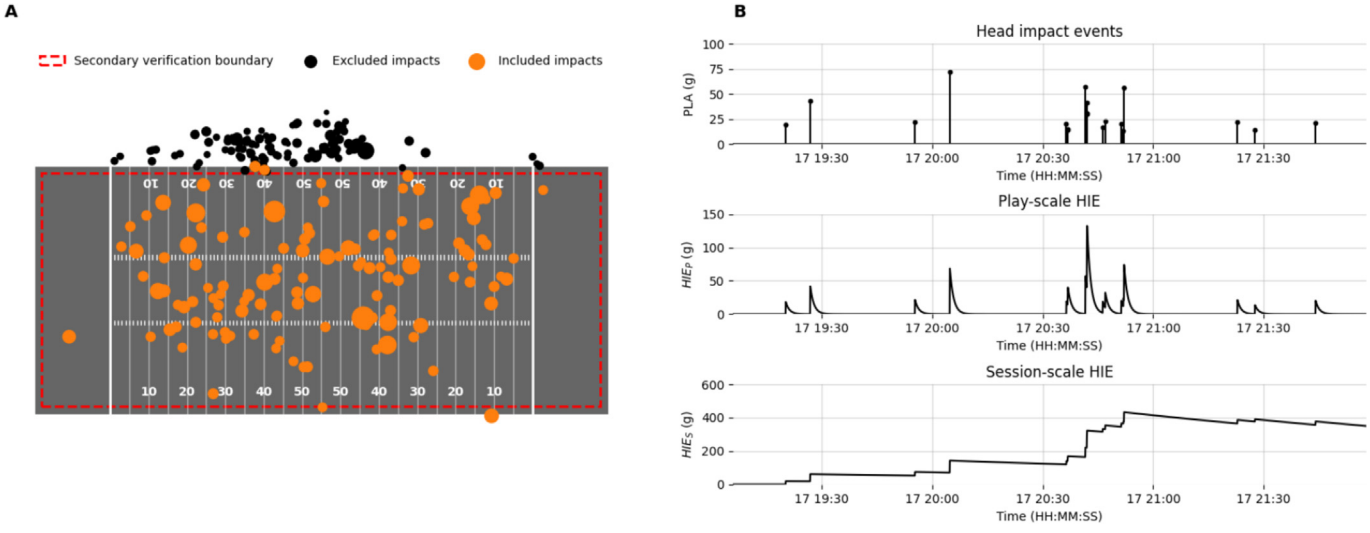

Fig. 1. Head impact event processing and continuous HIE profiling. (A) Impact events underwent a tiered spatial filtering process: impacts occurring within the 2 m boundary inside the field periphery (indicated by red dashed lines) were automatically included, while those beyond the boundary were subjected to secondary verification of active play participation. (B) Continuous HIE metrics were derived via recursive exponential decay, generating play-scale ($\tau = 60$ s) and session-scale ($\tau = 3$ h) impact exposure profiles. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

across seasons, the standard deviation of rotational acceleration exhibited a 39.6% reduction following hardware updates (Vector to Prevent mouthguards), compared to a relatively stable 12.4% change in PLA. Given the sensitivity of rotational kinematics to sensor coupling and coordinate transformation algorithms, PLA was prioritized as the more robustly captured signal. This approach was intended to minimize the propagation of measurement-specific variance into the physiological models and ensure that observed associations with autonomic regulation were reflective of impact exposure rather than sensor-related noise.

2.5. Heart rate recovery period detection and processing

The heart rate signal was processed using a zero-phase, second-order low-pass Butterworth filter with a cut-off frequency of 1/60 Hz.[30] A heart rate recovery period was defined as the interval from recovery onset, identified as the point at which filtered HR began a sustained decline, to HR subsequently beginning to increase. Instantaneous HRR speed values were computed as the first derivative (slope) of the filtered HR signal using centered differences. Maximum HR for each athlete was defined as the highest HR value recorded across all games.[31] HRR periods were excluded from analysis if their duration was less than 15 s or if the athlete's HR at the start of recovery was less than 70% of their season-wide maximum HR, consistent with previous studies ensuring adequate exertion for HRR analysis.[29,32] For each period, the following HR, impact exposure and performance metrics were extracted:

- HR dynamics (Fig. 2): Absolute HR at recovery onset (HR_{start}), as well as the peak HRR speed (HRR_{peak}, bpm/s) and time to the peak HRR speed ($tHRR_{peak}$) within the 60 s following recovery onset;
- Impact exposure: HIE_P and HIE_S values at recovery onset;
- Movement intensity: Average speed in the 60 s windows immediately preceding ($Speed_{pre}$) and following ($Speed_{post}$) recovery onset;
- Session workload: Total time played (*TP*) and work rate (*WR*, meters covered per minute played — m/min) since the start of the game.

2.6. Statistical analyses

Linear mixed-effects models (LMEMs) were used to account for the hierarchical and repeated-measures structure of the data, with multiple HRR periods observed per athlete across multiple matches. Three primary LMEMs were constructed to evaluate the association between HIE and different aspects of HR dynamics: HR_{start} (absolute HR at recovery onset), HRR_{peak} (peak HRR speed), and $tHRR_{peak}$ (time to the peak HRR speed).

In each model, head impact exposure metrics (HIE_P and HIE_S) were included as fixed effects alongside several control variables: movement intensity ($Speed_{pre}$ and $Speed_{post}$), session workload (*TP* and *WR*), and athlete characteristics (body mass index, *BMI*, and positional group: Linemen or Non-Linemen). For the HRR_{peak} and $tHRR_{peak}$ models, HR_{start} was additionally included as a covariate to account for differences in starting exertional state prior to recovery.

All models included crossed random intercepts for individual athletes and games to control for between-athlete variability and contextual differences across games. For the HR_{start} LMEM, a random slope for HIE_P was included for each subject ($1 + HIE_P|\text{athlete}$), accounting for individual differences in acute cardiovascular responses to mechanical loading.

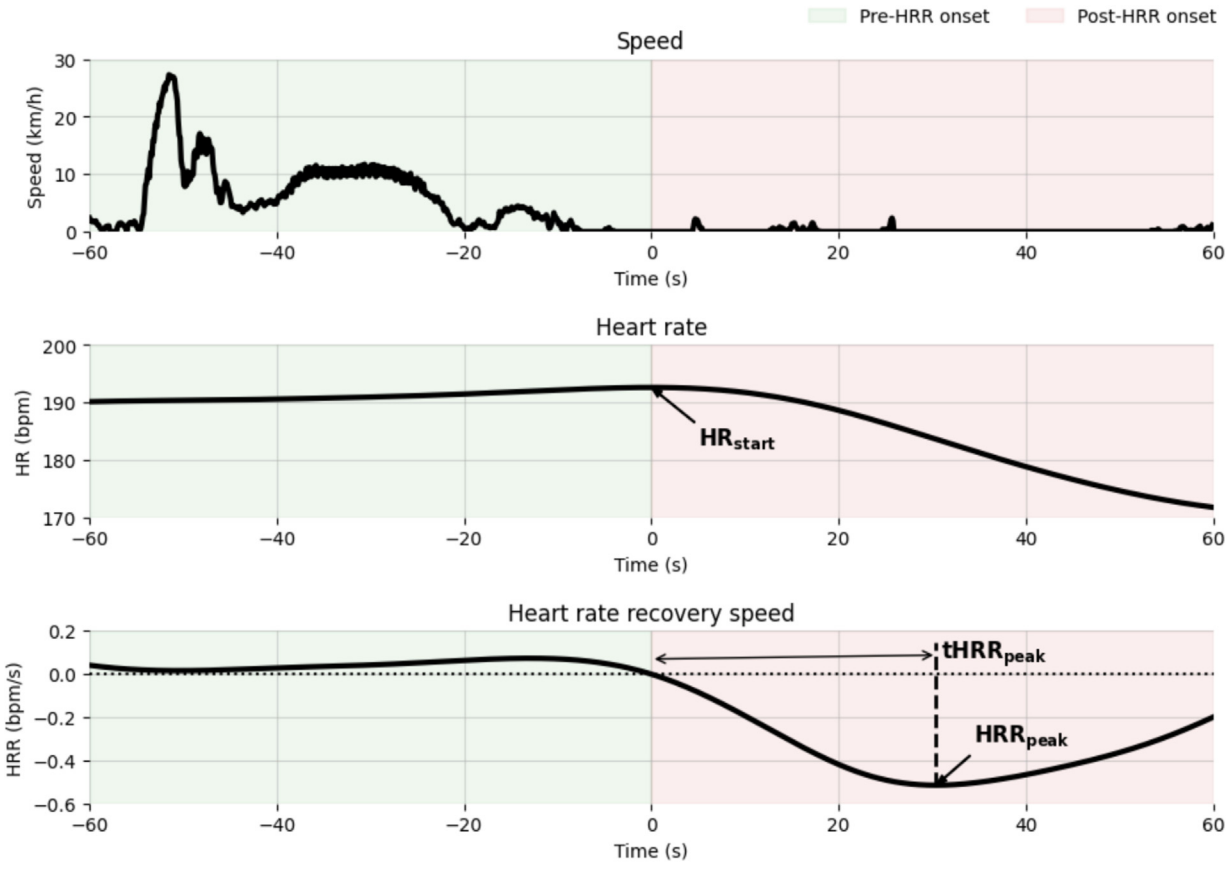

Fig. 2. Heart rate recovery period and metric extraction. Representative traces for speed, HR, and HRR speed are shown for a single recovery window. The 60 s window immediately preceding recovery onset (green shaded region) and the 60 s post-onset period (red shaded region) are highlighted. Key extracted features are indicated, including HR at recovery onset (HR_{start}), peak HRR speed (HRR_{peak}), and the time elapsed from onset to peak recovery ($tHRR_{peak}$). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

To explore potential non-linearity in the relationship between sustained head impact exposure and HRR_{peak}, a generalized additive mixed model (GAMM) was fit using a scaled-t response distribution. This approach was employed to improve robustness of the estimated fit to extreme physiological responses while allowing flexible characterization of exposure–response shape under high cumulative exposure. The HIE_S variable was modeled using a thin-plate regression spline ($k = 5$), while all additional covariates were included as linear terms. Random effects for athlete and match were incorporated as random intercepts to maintain a hierarchical and repeated-measures structure consistent with the primary LMEMs.

Both HIE_P and HIE_S variables were log-transformed ($\ln(x+1)$) prior to analysis to address the strong right-skew of impact exposure distributions.[33] Furthermore, to facilitate the comparison of effect sizes across variables with different units, all continuous predictors were Z-score standardized (scaled to mean = 0, SD = 1). Consequently, model estimates are reported as standardized coefficients (β).

Significance levels for all statistical tests were initially set at $p < 0.05$. To control for the family-wise error rate across our three related heart rate outcomes (HR_{start}, HRR_{peak}, and $tHRR_{peak}$), a Bonferroni correction was applied, resulting in a threshold of $p < 0.017$ for significance. All statistical analyses were performed using R Statistical Software (v4.1.2).

3. Results

3.1. Participants and identified heart rate recovery periods

A total of 72 athletes were initially enrolled in the study. Of these, 62 athletes (86.1%; Linemen: n = 17, Non-Linemen: n = 45) were included in the final analytical sample after excluding cases of hardware non-compliance and HRR periods that did not meet predefined physiological inclusion criteria: heart rate at recovery onset ≥ 70% of season-wide HR_{max} and minimum HRR duration of 15 s.[29,31,32]

The final longitudinal dataset comprised 7693 valid HRR periods associated with a cumulative total of 3249 recorded head impacts over the two competitive seasons. On average, players completed 7 (SD 2.2) games per season. Comprehensive participant demographics and session-wide mechanical load characteristics are summarized in Table 1.

3.2. HR response to impact exposure

Standardized LMEM coefficients for heart rate metrics are summarized in Table 2. Play-scale impact exposure (HIE_P) was a robust predictor of heart rate at recovery onset (HR_{start}), with each 1 − SD increase in log-transformed impact exposure associated with a 2.26 bpm increase in heart rate ($\beta = 2.26, p < 0.001$). The inclusion of random slopes for HIE_P improved model fit for HR_{start} ($\Delta AIC = 27.09$), indicating substantial inter-individual variability in the immediate cardiac response to impacts. Estimated individual slopes for the effect of HIE_P on HR_{start} ranged from 0.87 to 3.93 bpm/SD (Fig. 3A). Furthermore, acute exposure significantly influenced the timing of recovery ($\beta = 0.41, p < 0.001$), with higher-magnitude impacts associated with a delayed time to peak recovery rate (Fig. 3B).

Table 1
Participant characteristics and descriptive exposure and physiological metrics. Values are presented as mean (SD) unless otherwise specified.

Metric	Total cohort (n = 62)	Linemen (n = 17)	Non-Linemen (n = 45)
Demographics			
Age (years)	23.2 (1.5)	23.4 (1.5)	23.2 (1.6)
BMI ($kg \cdot m^{-2}$)	29.3 (5.2)	35.5 (4.4)	27.0 (3.1)
Participation and impact exposure			
Games per season	7.0 (2.2)	7.2 (1.9)	6.9 (2.3)
Total impacts (n)	3249	1022	2227
Mean PLA (g)	13.0 (6.4)	13.0 (6.6)	13.0 (6.5)
Physiology			
Total of HRR periods (n)	7693	1697	5996
Season-wide HR_{max} (bpm)	190.6 (6.8)	189.2 (9.5)	191.1 (5.5)
Mean HR_{start} (bpm)	156.5 (8.6)	158.5 (9.5)	155.7 (8.2)
Mean HR_{start} (% HR_{max})	82.1 (2.9)	83.8 (2.2)	81.5 (2.9)

Table 2
β represents standardized coefficients of log-transformed HIE variables. All models were adjusted for athlete group (Lineman/Non-Linemen), BMI, time played, on-field work rate, and pre/post-recovery running speed. The *HRR* models were additionally adjusted for *HR* at recovery onset.

Outcome variable	Predictor	β	95% CI	p
HR_{start}	HIE_P	2.26	[1.79, 2.69]	**<0.001***
	HIE_S	−0.34	[−0.74, 0.06]	0.097
	Model fit	$R^2_m = 0.49$	$R^2_c = 0.66$	
HRR_{peak}	HIE_P	−0.004	[−0.014, 0.005]	0.374
	HIE_S	−0.014	[−0.029, 0.001]	0.066
	Model fit	$R^2_m = 0.06$	$R^2_c = 0.28$	
$tHRR_{peak}$	HIE_P	0.41	[0.18, 0.65]	**<0.001***
	HIE_S	−0.06	[−0.39, 0.25]	0.705
	Model fit	$R^2_m = 0.26$	$R^2_c = 0.29$	

* denotes $p < 0.05$.

In contrast, no global linear relationship was observed between cumulative session strain (HIE_S) and HR_{start} ($p = 0.097$), HRR_{peak} ($p = 0.066$), or $tHRR_{peak}$ ($p = 0.705$). However, the low marginal R^2 for the linear HRR_{peak} model ($R^2_m = 0.06$) compared to the conditional R^2 ($R^2_c = 0.28$) suggests that individual variability and non-linear factors (further explored via GAMMs) play a more prominent role in recovery depth than simple linear accumulation.

Across all models, pre-recovery running speed was the strongest predictor of heart rate metrics (HR_{start}: $\beta = 11.7$; HRR_{peak}: $\beta = 0.097$; $tHRR_{peak}$: $\beta = 3.8$; all $p < 0.017$), consistent with expected metabolic demands. Post-recovery speed and time played were also significant predictors (all $p < 0.017$), indicating that both immediate movement and cumulative match duration influenced heart rate dynamics. Athlete BMI was significant only for HR_{start} ($\beta = 3.2, p < 0.017$), while athlete group and work rate did not significantly influence any metric ($p > 0.017$). For all HRR outcomes, HR_{start} served as a significant predictor ($p < 0.001$). Importantly, the reported effects of head impact exposure remained significant after adjusting for these physical workload variables.

3.3. Non-linear relationship between session HIE and HRR

To account for potential threshold effects that linear models might omit, a generalized additive mixed model (GAMM) was utilized to examine the relationship between session-scale HIE and HRR_{peak}. The GAMM revealed a significant non-linear effect of HIE_S on peak recovery speed ($edf = 2.40$, $\chi^2 = 11.15, p = 0.015$; Fig. 4).

Several parametric covariates significantly influenced recovery depth. Higher peak heart rates ($\beta = 0.045, p < 0.001$) and greater pre-recovery running velocities ($\beta = 0.110, p < 0.001$) were associated with higher HRR_{peak}, while active movement during the recovery window (higher post-recovery velocities) was the strongest parametric predictor of recovery speed ($\beta = -0.122, p < 0.001$). In contrast to HIE_S, play-scale impact exposure (HIE_P) had only a marginal effect on recovery rate ($\beta = -0.005, p = 0.043$). Notably, after accounting for these physical workloads and individual variability (subject and match random effects), the non-linear effect of session-scale head impact exposure remained a significant predictor of slowed autonomic recovery.

4. Discussion

This study used commercially available wearable sensors to monitor HIE and HRR dynamics during a football season in 72 male varsity athletes. A criterion-based selection of physiologically relevant HRR periods

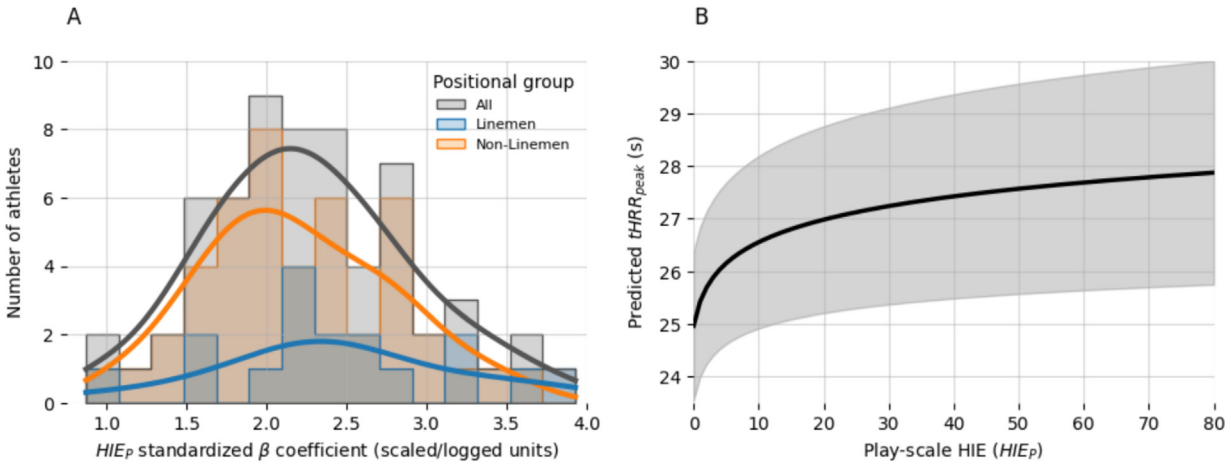

Fig. 3. Play-scale HIE effects on HR response. (A) Distribution of individual athlete random slopes (altogether and by positional group) for the effect of play-scale HIE (HIE_P) on HR_{start}, demonstrating inter-individual variability in HR sensitivity to acute impact exposure. (B) Marginal effect of HIE_P on the time to peak HR recovery ($tHRR_{peak}$) magnitude. The solid line represents the population-level prediction with 95% confidence intervals (shaded).

was used to characterize the influence of the magnitude of head impact PLAs on HR profiles during HRR. Analysis of a filtered dataset comprising 7693 HRR periods revealed a significant increase in HR and a delayed time to peak HRR following acute impact exposure. Notably, this effect remained after accounting for physical exertion and athlete characteristics. Further analysis revealed a non-linear association between accumulated session-wide impact exposure and peak HRR speed. Specifically, HRR remained largely preserved during low exposure (fewer than 200 g accumulated), followed by a non-linear attenuation of recovery speed observed at higher session-wide exposure levels. These findings contribute to a growing body of evidence indicating that subconcussive impacts may serve as a relevant predisposing factor for injury.[34,35] Specifically, they are associated with alterations in cardiovascular recovery dynamics that are consistent with changes in autonomic regulation following concussion, including reduced cerebral blood flow (CBF) and altered metabolic demands.

The autonomic nervous system is responsible for maintaining homeostasis by adapting responses to changes in the external and internal environment using sympathetic and parasympathetic influences.[7] It innervates the cardiac muscle and regulates peripheral artery stiffness so that sympathetic activation will increase atrial muscle contractility thereby increasing HR while, on the other hand, parasympathetic activation will decrease HR through the vagus nerve. Acutely, the observed delay in recovery initiation following high-magnitude impacts may reflect a transient suppression of this vagal reactivation or a heightened immediate sympathetic surge in response to the mechanical stimulus. Such autonomic perturbations have been shown to influence exercise capacity, as demonstrated in animal studies where silencing brainstem vagal preganglionic neurons — thereby reducing parasympathetic activity — led to a dramatic impairment in performance.[12] Since HR dynamics are primarily regulated by the ANS, the present findings demonstrating systematic alterations in HR and HRR are consistent with subtle perturbations in autonomic regulation, even in the absence of a diagnosed concussion. Importantly, these observations do not provide direct evidence of autonomic dysfunction but rather suggest that cardiovascular recovery dynamics may be sensitive to impact-related physiological stressors.

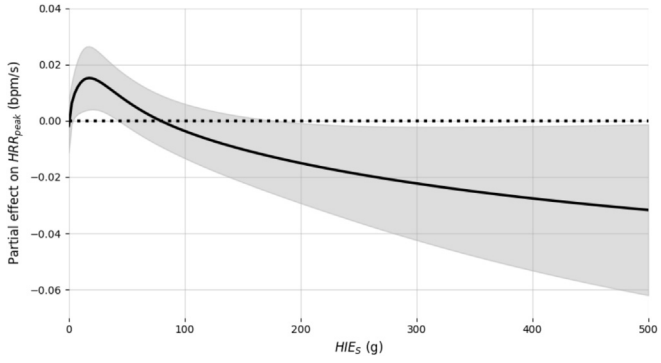

Fig. 4. Session-scale HIE effects on HR recovery speed. GAMM-derived relationship between session-scale HIE (HIE_S) and peak HRR speed (HRR_{peak}). The solid line represents the population-level smooth estimate with a 95% confidence interval (shaded).

The reduced HRR speed observed at higher accumulated head impact loads may reflect altered physiological regulation in response to cumulative impact exposure. Several candidate mechanisms may plausibly underlie these observations, although none were directly assessed in the present study. Prior work in mTBI populations suggests that diffuse axonal injury, altered cerebral perfusion, and disruptions to autonomic control circuits may co-occur following traumatic head impacts.[10] While we observed cumulative slowing of HRR, the acute elevation in heart rate following high-magnitude hits may represent the immediate physiological manifestation of these neural disruptions. In terms of CBF, reduced perfusion was observed in the right dorsal mid-insular cortex of collegiate athletes following mTBI, as measured by arterial spin labeling, when compared to controls.[36] The reduction in CBF was transient, normalizing within one month post-injury, and appeared to predict the rate of HRR. However, as autonomic neurotransmitters and CBF were not directly measured in this study, these physiological pathways remain speculative. Further exploration is needed to establish the relationship between ANS dysfunction and alterations in CBF.

In parallel, the slowing of HRR speed observed following cumulative impact load may also be attributable to sustained elevation in physiological stress induced by repeated impacts. This stress response likely involves increased circulating cortisol, a glucocorticoid hormone produced via the hypothalamic–pituitary adrenal axis, which plays a key role in maintaining homeostasis under stress.[37] Given cortisol's regulatory influence on the cardiovascular system, impact-induced elevations in cortisol may contribute to the observed changes in HRR. Testing this hypothesis would require direct monitoring of cortisol levels, potentially through non-invasive methods such as eccrine sweat analysis.[38]

The present work contributes to the growing body of evidence supporting autonomic dysfunction following mild traumatic brain injury,[39] while focusing specifically on the relationship between impact magnitude and heart rate recovery dynamics. Beyond population-

averaged effects, the inclusion of subject-specific slopes for acute HIE revealed substantial inter-individual variability in HR at the onset of recovery, indicating that athletes differ in how strongly their cardiovascular systems respond to comparable impact loads.

This variability raises the question of whether autonomic shifts are merely markers of neurological strain or active contributors to post-impact symptoms. While this study did not assess clinical outcomes, a bidirectional relationship has been proposed in prior work, such that altered HR dynamics could theoretically contribute to physiological symptoms rather than solely acting as a downstream consequence of neural injury.[40] Although previous research has linked autonomic dysfunction to a "vulnerability window",[39] our study focuses on these physiological associations without assessing performance metrics or injury outcomes. It remains to be determined whether these cardiovascular alterations directly increase an athlete's susceptibility to subsequent impacts or clinical injury. Future research integrating HIE monitoring with objective performance data (e.g., GPS-derived velocity or reaction time) is necessary to establish the functional consequences of these heart rate recovery patterns.

4.1. Methodological considerations and limitations

While the mathematical formulation of impact exposure provides a principled approach for continuous exposure metric, the decay-weighted accumulated HIE necessarily assumes a common temporary recovery profile across athletes. Although this formulation captures the relative timing and magnitude of impacts across multiple timescales, it may not fully reflect inter-individual differences in physiological recovery or adaptation. Furthermore, the non-linear spline-based observations are exploratory, and while they indicate population-level trends in HRR_{peak} attenuation, the specific inflection points should not be overinterpreted as universal clinical thresholds.

The use of two different instrumented mouthguard systems also introduces potential sensor heterogeneity. To mitigate this, peak linear acceleration was selected as the primary exposure metric due to its relatively greater cross-device stability compared to rotational kinematics. Nevertheless, device-specific calibration procedures and inter-device reliability were not explicitly modeled, thus the direction and magnitude of potential device-related bias remain unknown. Furthermore, the use of a convenience sample of male varsity athletes may limit generalizability, as participating athletes may differ from the broader population in training status, play style, or exposure profiles.

Several additional factors that may influence heart rate dynamics were not directly accounted for in the statistical models, including hydration status, sleep quality, prior concussion history, and psychological stress. Although running speed, time played, and work rate were included to partially control for physical exertion, American football is a highly dynamic environment in which high-magnitude impacts frequently coincide with emotionally salient game events that may independently modulate heart rate through adrenergic or cognitive mechanisms. As such, the present findings should be interpreted as associations between head impact exposure and cardiovascular recovery dynamics rather than as evidence of isolated central nervous system effects. Future research integrating head impact monitoring with autonomic, hypothalamic–pituitary–adrenal, and immunological measures will be essential for a more complete understanding of subconcussive impacts.[7]

5. Conclusion

The present study demonstrates that head impact exposure is associated with systematic alterations in heart rate and heart rate recovery dynamics following intense physical effort in collegiate football athletes. Head impact exposure effects were observed across both acute and cumulative timescales: higher acute exposure was associated with elevated heart rate at recovery onset and prolonged time to peak HRR speed, while increased cumulative exposure was linked to a slowing of the peak HRR speed itself. These findings indicate that cardiovascular recovery dynamics represent a measurable physiological signal that is sensitive to head impact exposure during live sport participation. Importantly, this study did not assess clinical injury outcomes or performance metrics and therefore cannot determine whether the observed cardiovascular alterations reflect increased susceptibility to injury or functional impairment. Rather, the results highlight heart rate recovery as a promising, non-invasive physiological marker that may complement existing biomechanical exposure metrics in future multimodal investigations. Given the scalability of wearable telemetry, this approach provides a practical and versatile framework for future multimodal investigations aimed at linking physiological markers with long-term clinical and performance outcomes in contact sports.

CRediT authorship contribution statement

Abdullah Zafar: Methodology, writing — original draft preparation, and visualization. Géraldine Martens: Writing — original draft preparation. Samuel Guay: Data curation. Sophie-Andrée Vinet: Data curation. Eric Wagnac: Conceptualization. Laurie-Ann Corbin-Berrigan: Data curation and writing — reviewing and editing. François Prince: Writing — reviewing and editing. Jaden Pantazis: Data curation. Simon Prince: Data curation. Louis De Beaumont: Conceptualization and writing — reviewing and editing.

Consent to participate

Written consent from individual participants and the football clubs was obtained prior to data collection.

Confirmation of ethical compliance

The study was approved by the Comité d'Éthique de la Recherche vieillissement-neuroimagerie CIUSSS — Nord-de-l'Île-de-Montréal ethics committee with attributed number CER VN 19-20-22.

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Declaration of interest statement

The authors report no conflict of interest.

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Data availability

De-identified data from the study will be made available by the lead author based on a reasoned request.

References

1 Barden C, Quarrie KL, McKay C et al. Employing standardised methods to compare injury risk across seven youth team sports. *Int J Sports Med* 2021;42:1019-1026.

2 Giza CC, Hovda DA. The new neurometabolic cascade of concussion. *Neurosurgery* 2014;75:S24.
3 Bailes JE, Petraglia AL, Omalu BI et al. Role of subconcussion in repetitive mild traumatic brain injury. *J Neurosurg* 2013. doi:10.3171/2013.7.JNS121822.
4 Johnson B, Neuberger T, Gay M et al. Effects of subconcussive head trauma on the default mode network of the brain. *J Neurotrauma* 2014;31:1907-1913.
5 McAllister TW et al. Effect of head impacts on diffusivity measures in a cohort of collegiate contact sport athletes. *Neurology* 2014;82:63-69.
6 Vinet S-A et al. Short-term changes in the physiology of the primary motor cortex following head impact exposure during a Canadian football game. *J Neurosurg* 2024;141:436-444.
7 Pertab JL et al. Concussion and the autonomic nervous system: an introduction to the field and the results of a systematic review. *NeuroRehabil Int Interdiscipl J* 2018;42:397-427.
8 Abaji JP, Curnier D, Moore RD et al. Persisting effects of concussion on heart rate variability during physical exertion. *J Neurotrauma* 2016;33:811-817.
9 Hutchison MG et al. Psychological and physiological markers of stress in concussed athletes across recovery milestones. *J Head Trauma Rehabil* 2017;32:E38-E48.
10 Mercier LJ et al. Autonomic dysfunction in adults following mild traumatic brain injury: a systematic review. *NeuroRehabilitation* 2022;50:3-32.
11 Charron J, Comtois AS. Unclear outcomes of heart rate variability following a concussion: a systematic review. *Brain Inj* 2021;35:987-1000.
12 Machhada A et al. Vagal determinants of exercise capacity. *Nat Commun* 2017;8:15097.
13 Borresen J, Lambert MI. Autonomic control of heart rate during and after exercise. *Sports Med* 2008;38:633-646.
14 Heffernan KS, Fahs CA, Shinsako KK et al. Heart rate recovery and heart rate complexity following resistance exercise training and detraining in young men. *Am J Physiol Heart Circ Physiol* 2007;293:H3180-H3186.
15 Storniolo JL, Esposti R, Cavallari P. Heart rate kinetics and sympatho-vagal balance accompanying a maximal sprint test. *Front Psychol* 2020;10.
16 Imai K et al. Vagally mediated heart rate recovery after exercise is accelerated in athletes but blunted in patients with chronic heart failure. *J Am Coll Cardiol* 1994;24:1529-1535.
17. Corbin-Berrigan L-A et al. Head impacts in Canadian varsity football: an exploratory study. *Concussion* 2021;6.
18. Versace J. A review of the severity index. https://saemobilus.sae.org/papers/a-review-severity-index-710881 1971.
19 Broglio SP, Lapointe A, O'Connor KL et al. Head impact density: a model to explain the elusive concussion threshold. *J Neurotrauma* 2017;34:2675-2683.
20 Banister EW, Calvert TW, Savage MV et al. A systems model of training for athletic performance. *Aust J Sports Med* 1975;7:5761.
21. De Beaumont L, Lassonde M, Leclerc S et al. Long-term and cumulative effects of sports concussion on motor cortex inhibition. *Neurosurgery* 2007;61:329.
22. World Medical Association Declaration of HELSINKI: ethical principles for medical research involving human subjects. *JAMA* 2013;310:2191-2194.
23 Camarillo DB, Shull PB, Mattson J et al. An instrumented mouthguard for measuring linear and angular head impact kinematics in American football. *Ann Biomed Eng* 2013;41:1939-1949.
24 Jones B et al. Ready for impact? A validity and feasibility study of instrumented mouthguards (iMGs). *Br J Sports Med* 2022;56:1171-1179.
25. *Vector Elite Vest 2.3 — Design Improvements*, 2023.
26 Linke D, Link D, Lames M. Validation of electronic performance and tracking systems EPTS under field conditions. *PloS One* 2018;13:e0199519.
27 Zafar A et al. Characterization of running intensity in Canadian football based on tactical position. *Sensors (Basel)* 2024;24.
28 Iosia MF, Bishop PA. Analysis of exercise-to-rest ratios during division IA televised football competition. *J Strength Cond Res* 2008;22:332-340.
29 Arduini A, Gomez-Cabrera M-C, Romagnoli M. Reliability of different models to assess heart rate recovery after submaximal bicycle exercise. *J Sci Med Sport* 2011;14:352-357.
30 Zafar A et al. Heart rate dynamics and quantifying physical fatigue in Canadian football. *Appl Sci (Basel)* 2024;14:5340.
31. Semin K et al. Discrepancy between training, competition and laboratory measures of maximum heart rate in NCAA division 2 distance runners. *J Sports Sci Med* 2008;7:455-460.
32 Ostojic SM, Calleja-Gonzalez J. Aerobic capacity and ultra short-term heart rate recovery after maximal exercise in sportswomen. *Sci Sports* 2010;25:267-271.
33 Crisco JJ et al. Head impact exposure in collegiate football players. *J Biomech* 2011;44:2673-2678.
34 Lynall RC, Mauntel TC, Padua DA et al. Acute lower extremity injury rates increase after concussion in college athletes. *Med Sci Sports Exerc* 2015;47:2487.
35 Chmielewski TL et al. Impaired motor control after sport-related concussion could increase risk for musculoskeletal injury: implications for clinical management and rehabilitation. *J Sport Health Sci* 2021;10:154-161.
36 Meier TB et al. Recovery of cerebral blood flow following sports-related concussion. *JAMA Neurol* 2015;72:530-538.
37 Jia M, Chew WM, Feinstein Y et al. Quantification of cortisol in human eccrine sweat by liquid chromatography tandem mass spectrometry. *Analyst* 2016;141:2053-2060.
38 Parlak O, Keene ST, Marais A et al. Molecularly selective nanoporous membrane-based wearable organic electrochemical device for noninvasive cortisol sensing. *Sci Adv* 2018;4:eaar2904.
39 Flatt AA, Allen JR, Keith CM et al. Season-long heart-rate variability tracking reveals autonomic imbalance in American college football players. *Int J Sports Physiol Perform* 2021. doi:10.1123/ijspp.2020-0801.
40 Kibria F et al. Bidirectional interplay between traumatic brain injury and cardiovascular dysfunction in athletes. *J Clin Med* 2025;14.