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ORIGINAL RESEARCH

Concussions Are Associated With Increases in Blood Pressure and Cardiovascular Risk in American-Style Football Athletes

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ABSTRACT

BACKGROUND Concussions sustained during American-style football (ASF) participation are common. Whether concussions are associated with cardiovascular risk is unknown.

OBJECTIVES The authors sought to determine whether concussions sustained during collegiate ASF participation lead to increases in systolic blood pressure (SBP) and acquired maladaptive cardiovascular phenotypes.

METHODS In a longitudinal and case-controlled analysis, ASF athletes at 2 National Collegiate Athletic Association Division-I programs were followed for up to 2 years with cardiovascular assessments including BP, echocardiography, and vascular applanation tonometry. ASF athletes who sustained clinically diagnosed concussions were compared to weight and player position-matched controls without concussions.

RESULTS Thirty-nine ASF athletes who sustained concussions (preconcussion baseline: [SBP] 129.6 ± 13.6 mm Hg) and 39 weight and player position-matched controls (preconcussion baseline: [SBP] 131.6 ± 13.1 mm Hg) were followed for 1 season postconcussion; 14 of these pairs were followed through 2 seasons. After 1 season, increased pulse wave velocity (PWV) (case $\Delta = 0.4$ [0.2] m/s, $P = 0.036$; control $\Delta = -0.1$ [0.1] m/s, $P = 0.38$) was observed among cases. Among the case and control pairs followed over 2 seasons, increased SBP (case $\Delta = 10.1$ [3.6] mm Hg, $P < 0.01$; control $\Delta = 0.1$ [3.2] mm Hg, $P = 0.97$) and diastolic blood pressure (case $\Delta = 8.2$ [2.9] mm Hg, $P < 0.01$; control $\Delta = -4.1$ [4.0] mm Hg, $P = 0.30$) and decreased É (case $\Delta = -2.8$ [0.8] cm/s, $P = 0.001$; control $\Delta = -1.0$ [0.8] cm/s, $P = 0.21$) were observed among cases. Over 1 season, concussions were associated with higher PWV ($\beta = 0.33$ [95% CI: 0.09-0.56], $P = 0.007$). Among athletes followed over 2 seasons, concussions were associated with higher PWV ($\beta = 0.42$ [95% CI: 0.05-0.78], $P = 0.03$), diastolic blood pressure ($\beta = 5.89$ [95% CI: 1.23-10.54], $P = 0.01$), LV mass index ($\beta = 11.01$ [95% CI: 6.13-15.90], $P < 0.001$), and lower É ($\beta = -2.11$ [95% CI: −3.27 to −0.95], $P < 0.001$).

CONCLUSIONS Concussions sustained during collegiate ASF participation are independently associated with markers of cardiovascular risk and acquired maladaptive cardiovascular phenotypes. Clinical ASF concussion management strategies inclusive of careful BP surveillance may lead to early identification of hypertension. (JACC Adv. 2025;4:101717)

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ABBREVIATIONS AND ACRONYMS

ASF = American-style football

DBP = diastolic blood pressure

É = tissue-Doppler É velocity

LM = lineman

LVH = left ventricular hypertrophy

NCAA = National Collegiate Athletic Association

NFL = National Football League

NSAID = nonsteroidal anti-inflammatory drug

PED = performance-enhancing drug

PWV = pulse wave velocity

SBP = systolic blood pressure

SCAT5 = Sports Concussion Assessment Tool 5th edition

TBI = traumatic brain injury

Concussions are common during competitive American-style football (ASF) participation.[1,2] Clinical concerns among former professional ASF athletes, as a consequence of prior sustained concussions, include primary neurocognitive pathology including amyotrophic lateral sclerosis, Parkinson disease, and chronic traumatic encephalopathy.[1,3-6] However, alternative disease processes with established impact on cognitive function, particularly hypertension, are also enriched among active professional and collegiate ASF athletes.[7,8]

Among collegiate ASF athletes, prior longitudinal data demonstrate an increased risk of hypertension and the development of maladaptive cardiovascular phenotypes including arterial stiffening,[9] concentric left ventricular hypertrophy (LVH),[10] and reductions in systolic and diastolic function.[11-14] Explanatory and complementary mechanisms, which include deliberate and rapid weight gain,[11] sleep apnea,[15] and nonsteroidal anti-inflammatory drug (NSAID) overuse,[16] have been proposed. Among older, former professional ASF athletes, retrospectively derived concussion symptom scores predict increased later-life hypertension prevalence and other postcareer atherogenic cardiovascular risk profiles in a dose-dependent manner.[17,18] These findings raise the possibility that ASF-sustained concussions may represent a novel cardiovascular risk factor. However, to date, the relationship between concussions, blood pressure, and the corollary development of maladaptive cardiovascular phenotypes among active ASF athletes has not been explored in rigorous, prospective fashion.

We hypothesized that collegiate ASF athletes who sustained a concussion would demonstrate subsequent arterial stiffening followed by more pronounced increases in blood pressure compared to those without concussions and that concussion exposure would independently predict the development of maladaptive cardiovascular phenotypes including increased arterial stiffness, LVH, and decreased LV diastolic function. To test this hypothesis, we performed a rigorous, longitudinal case-control analysis using data from an ongoing prospective collegiate athlete cardiovascular registry to determine whether concussions sustained during collegiate ASF participation are associated with increased cardiovascular risk and acquired maladaptive cardiovascular phenotypes.

METHODS

GEORGIA INSTITUTE OF TECHNOLOGY, FURMAN UNIVERSITY, AND EMORY UNIVERSITY STUDENT-ATHLETE CARDIOVASCULAR REGISTRY (INCLUDING ASF ATHLETES). Since 2014, the primary investigative team, in collaboration with the National Collegiate Athletic Association (NCAA) Division-I athletic programs at Georgia Institute of Technology (Atlanta, GA) and Furman University (Greenville, SC), and Division-III athletic program at Emory University (Atlanta, GA), has longitudinally enrolled student-athletes participating in numerous sporting disciplines in a registry inclusive of comprehensive cardiovascular measurements. Demographics, clinical characteristics, and anthropometric, 2-dimensional echocardiographic, and vascular applanation tonometry measurements have been prospectively recorded for all registry participants. For this study, newly enrolled freshman ASF athletes participating at Georgia Tech and Furman were eligible for inclusion with longitudinal assessments through one or 2 years of collegiate ASF participation. Registry study assessments occurred at specific longitudinal time points: baseline (preseason freshman year at college matriculation), postseason freshman year (at the conclusion of the ASF competitive season, ~6 months later), and postseason

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

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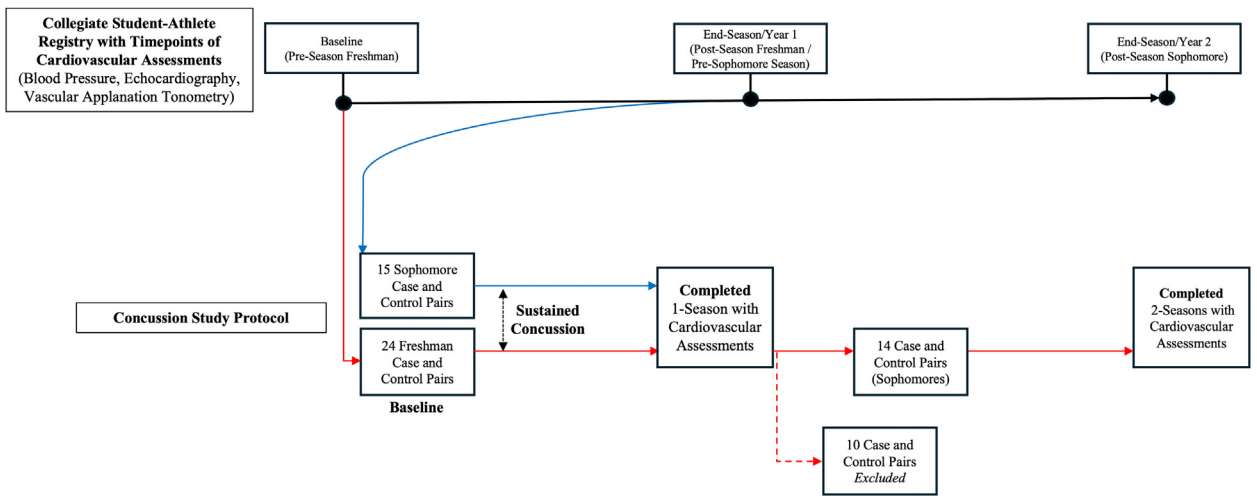
FIGURE 1 Overview of the Collegiate Student-Athlete Cardiovascular Registry and the Concussion Study Protocol



sophomore year (conclusion of the season, 1 year after postseason freshman year) (Figure 1). The Emory Institutional Review Board approved all aspects of this study, and subjects provided written informed consent.

ASF ATHLETE CASE-CONTROL DEFINITIONS AND CONCUSSION STUDY PROTOCOL. After study enrollment as freshman ASF athletes, inclusion criteria for cases were: 1) at least one clinically diagnosed concussion (defined below) during the freshman or sophomore-year season; and 2) at least one complete study assessment (clinical measurements, echocardiography, applanation tonometry) after a clinically diagnosed concussion at a longitudinal registry study time point. Any athlete with a diagnosed concussion but no subsequent complete study assessment after the concussion was not eligible for final analysis. ASF athletes without a clinically diagnosed concussion during their freshman and sophomore season and who also had complete registry study assessments were eligible as controls. Eligible cases were randomly matched to controls by weight (≤5 kg difference between case and control pair) and player position (nonlineman or lineman [LM]). Nonlineman and LM player positions were defined as previously proposed.[19] Cases and controls were matched at the registry assessment time point prior to a first sustained concussion (preseason freshman year if a concussion was sustained during the freshman season or postseason freshman year if a first concussion was sustained during the sophomore season). Prospectively recorded, comprehensive cardiovascular measurements were then compared between the matched cases and controls over one or 2 full seasons within this 2-year longitudinal time frame (Figure 1).

We also performed a sensitivity analysis comparing the matched case and control pairs through the chronological progression of the first 2 years of collegiate ASF participation that paralleled the registry study time point assessments (Figure 1).

All cases and controls tested negative for performance-enhancing drugs (PEDs) as per NCAA testing protocols. Blood pressure was measured after ≥15 minutes of rest using a manual aneroid sphygmomanometer and an appropriately sized cuff, recorded as the average of 2 measurements, and categorized in accordance with contemporary guidelines.[20]

ASF ATHLETE MEDICATION AND NSAID USE. All study participants detailed prescription medication use at the time of freshman year matriculation. Additionally, in a prior study, separate questionnaires were administered prospectively to freshman ASF athletes to qualitatively assess NSAID use throughout the freshman season.[21] For those ASF athletes who participated in this prior study and were included in the present analysis, participants qualitatively reported NSAID use as either frequent (weekly or daily) or infrequent (never or no more than monthly).[21]

CLINICAL DIAGNOSIS OF CONCUSSION. ASF athletes with a clinical diagnosis of concussion were identified after review of official medical records maintained in the respective athletic departments. The official date of concussion was prospectively recorded at the time of diagnosis by team medical staffs. Consistent with clinical standards of care,

formal concussion evaluations were triggered by team physicians and/or athletic trainers following a witnessed mechanism of injury (eg, collision with or without visualized head impact that was accompanied by balance/gait difficulties, motor incoordination, and/or inability to resume standing body position during competitive games or practice, including spring football). In addition, athletes who reported symptoms including headache, neck pain/stiffness, diplopia, loss of mental acuity, or weakness in the extremities were formally assessed for concussion and cervical spine injury. A complete concussion evaluation was performed for all cases, including those who generated immediate clinical concern and those who generated later concern due to delayed symptom reporting. In conjunction with the team physician's clinical judgment, the 5th edition of the Sports Concussion Assessment Tool (SCAT5), a validated method to assist in the diagnosis of concussion using the definition of concussion from the international consensus conference, was used to establish a concussion diagnosis.[22,23] SCAT5 includes a memory assessment, Glasgow Coma Scale, symptom evaluation, Standardized Assessment of Concussion, concentration assessments, delayed recall, and other neurological screens. Final clinical adjudication was made by the respective treating team physician(s) at Georgia Tech and Furman. Throughout this study, team physicians and ASF athletic trainers at both institutions were unchanged.

TWO-DIMENSIONAL TRANSTHORACIC ECHOCARDIOGRAPHY. Transthoracic echocardiography was performed using a commercially available system (Vivid-I, GE Healthcare). Two-dimensional and tissue-Doppler imaging from standard parasternal and apical positions were performed by experienced sonographers, who were consistent throughout the study. Frame rates were individualized per study for optimal image quality between 60 and 100 Hz. All information was stored digitally, and poststudy offline data analysis (EchoPAC version 7, GE Healthcare) was performed by study investigators (A.J.R., J.H.K.). Definitions of normality for cardiac structure and function were adopted from the most recent guidelines.[24] LV mass was calculated using the area-length method (accounts for LV morphology in both the short and long axis) and indexed to body surface area, and LV ejection fraction was calculated using the modified biplane technique. Comprehensive assessment of cardiac diastolic function using tissue-Doppler imaging was performed, and tissue velocities (E′, A′, and S′) were measured from color-coded images at the lateral and septal mitral annulus. E′ was then reported as the average value between the 2 measurements.

VASCULAR APPLANATION TONOMETRY. Arterial stiffness was measured using high-fidelity applanation tonometry (SphygmoCor, Atcor Medical), which records sequential high-quality pressure waveforms at peripheral pulse sites. The primary measure of arterial function was the carotid-femoral pulse wave velocity (PWV), which is the gold standard index of arterial stiffness and an established independent predictor and risk factor for adverse cardiovascular outcomes.[25,26] PWV was measured as previously described.[9,11,26]

STATISTICAL ANALYSIS. Continuous variables are presented as mean ± SD or SE, and categorical variables as percentages. For matched cases and controls, variables were compared using 2-sample *t*-tests for continuous variables. For categorical variables, chi-square or Fisher's exact test was used. Mixed-effects linear regression models, incorporating within-participant correlation and adjusted for player position, were constructed to evaluate for longitudinal changes at each time point (preconcussion baseline, postconcussion year 1, postconcussion year 2 [for the case/control pairs analyzed over 2 years]) in select clinical and cardiovascular measurements from baseline with time being the categorical independent variable. Concussion was coded as a cumulative lasting effect, consistent with prior characterization of concussion exposure.[27-31] In the sensitivity analysis, these models were similar with the exception that time points were based on chronological progression throughout collegiate ASF participation (preseason freshman year, postseason freshman year, and postseason sophomore year). To assess the association between concussion and our primary outcome measures of systolic blood pressure (SBP), diastolic blood pressure (DBP), tissue-Doppler E¢, PWV, and LV mass index, univariate and multivariable mixed-effects linear regression models (adjusting for weight, player position, and self-identified race) were constructed and accounted for within-participant correlation. All statistical analyses were performed using SAS software (version 9.4, SAS Institute). A *P* value of ≤0.05 was considered significant.

RESULTS

BASELINE COMPARISON OF CASES VS CONTROLS. Study participants were enrolled between 2014 to 2019 and 2021 to 2022 (data collection was suspended between 2020 and 2021 due to the COVID-19

TABLE 1 Baseline ASF Athlete Characteristics Prior to Concussion Exposure for Cases

	No Concussion (n = 39)	Concussion (n = 39)	*P* Value
Clinical characteristics			
Weight (kg)	102.0 ± 22.0	101.8 ± 22.4	0.96
SBP (mm Hg)	131.6 ± 13.1	129.6 ± 13.6	0.50
DBP (mm Hg)	76.9 ± 10.2	74.5 ± 11.3	0.33
Prescription stimulant use	4 (10.2)	3 (7.7)	1.00
Position			
Linemen	17 (44)	17 (44)	N/A
Nonlinemen	22 (56)	22 (56)	
Self-identified race			
White	19 (49)	17 (44)	0.65
Black	20 (51)	22 (56)	
Cardiovascular measurements			
LVIDd (mm)	53.3 ± 4.9	52.7 ± 4.3	0.58
Average wall thickness (mm)	9.3 ± 1.2	9.4 ± 1.0	0.56
Ejection fraction (%)	61.6 ± 4.6	61.7 ± 4.9	0.96
PWV (m/s)	5.3 ± 0.7	5.1 ± 0.7	0.24
LV mass index (g/m^2)	94.4 ± 11.2	97.0 ± 12.5	0.34
E wave velocity (cm/s)	84.2 ± 12.7	82.6 ± 15.5	0.63
A wave velocity (cm/s)	41.2 ± 7.0	43.2 ± 10.8	0.34
E-wave/A-wave velocity	2.1 ± 0.5	2.0 ± 0.5	0.19
Tissue Doppler É velocity (cm/s)[a]	15.8 ± 2.6	15.3 ± 2.9	0.41

Values are n (%) or mean ± SD. [a]Mean of septal and lateral wall tissue Doppler É velocities.

ASF = American-style football; DBP = diastolic blood pressure; LV = left ventricular; LVIDd = left ventricular internal diameter end diastole; PWV = pulse wave velocity; SBP = systolic blood pressure.

pandemic). Across the first 2 years of collegiate ASF participation, 39 ASF athletes sustained at least 1 concussion (n = 24 sustained a first concussion during their freshman season and N = 15 sustained a first concussion during their sophomore season) and fulfilled inclusion criteria (Figure 1). One case sustained 2 concussions during their freshman season and one case sustained a concussion in both their freshman and sophomore seasons. These 39 cases were randomly matched to 39 controls (randomly matched out of 297 ASF athletes without clinically diagnosed concussions over the first 2 years of collegiate ASF participation). All 39 cases completed 1 year of longitudinal assessment. Among the 24 athletes who sustained at least one concussion during their freshman season, 14 completed 2 seasons of longitudinal assessments (Figure 1). The 10 freshman cases lost to follow-up were due to cessation of the collegiate ASF career (none because of postconcussive symptoms and/or disability) or participation in the NCAA transfer portal. Baseline demographics and clinical characteristics for the cases and controls are shown in Table 1. Cases and controls were also similar by self-identified race (44% [White]/56% [Black] vs 49% [White]/51% [Black], $P = 0.65$) and SBP (129.6 ± 13.6 mm Hg vs 131.6 ± 13.1 mm Hg, $P = 0.50$), respectively. Prescription stimulant use was uncommon and similar between cases and controls (7.7% vs 10.2%, $P = 1.00$, respectively).

Qualitative and prospectively reported NSAID use was available for cases (n = 12/24, 50%) and controls (n = 16/24, 67%) during the freshman season.[16] Frequent NSAID use reported as weekly or daily was nearly identical between these cases and controls (41.7% vs 43.7%, $P = 1.00$, respectively).

In our sensitivity analysis of the 39 cases, 29 cases completed 2 years of collegiate ASF through their sophomore season (14 sustained concussion during their freshman season and 15 sustained concussion during their sophomore season). All baseline preseason freshman year measurements were similar to their matched controls (Supplemental Table 1).

LONGITUDINAL CHANGES IN CLINICAL AND CARDIOVASCULAR PHENOTYPES IN CASES AND CONTROLS. For the 39 case and control pairs assessed over one season after concussion exposure, similar increases in weight (case Δ = 1.3 [0.6] kg, $P = 0.05$; control Δ = 1.4 [0.6] kg, $P = 0.03$) and LV mass index (case Δ = 5.6 [1.9] gm/m^2, control Δ = 7.2 [1.9] gm/m^2, $P < 0.01$ for both) were observed (Table 2). However, significant increases in PWV (case Δ = 0.4 [0.2] m/s, $P = 0.036$; control Δ = −0.1 [0.1] m/s, $P = 0.38$) were observed only among cases (Table 3). Among the 14 case and control pairs followed over 2 seasons after concussion exposure, significant increases in SBP (case Δ = 10.1 [3.6] mm Hg, $P < 0.01$; control Δ = 0.1 [3.2] mm Hg, $P = 0.97$) and DBP (case Δ = 8.2 [2.9] mm Hg, $P < 0.01$; control Δ = −4.1 [4.0] mm Hg, $P = 0.30$) and decreases in É (case Δ = −2.8 [0.8], $P = 0.001$; control Δ = −1.0 [0.8], $P = 0.21$) were observed only among cases (Table 3, Figure 2).

In our sensitivity analysis, results were similar (see Supplemental Table 2). For the 39 cases and control pairs compared by chronological progression through the sophomore year of collegiate ASF participation, significant increases in SBP (case Δ = 8.5 [2.4] mm Hg, $P < 0.001$, control Δ = 0.5 [2.0] mm Hg, $P = 0.80$) and PWV (case Δ = 0.5 [0.2] m/s, $P = 0.004$; control Δ = 0.1 [0.1] m/s, $P = 0.39$) were observed only among cases.

COMPARISON OF LONGITUDINAL CHANGES IN SBP AND PWV IN CASES VS CONTROLS. For the 39 case and control pairs assessed over one season after concussion exposure, when comparing longitudinal changes in cases vs controls, differences for ΔPWV (Δ0.45 [95% CI: 0.06-0.83] m/s, $P = 0.025$) were

TABLE 2 Longitudinal Changes in Select Measurements in Cases (n = 39) and Controls (n = 39) After 1 Complete Collegiate American-Style Football Season

	Preconcussion Baseline (Measured Preseason)	Postconcussion Year 1 (Measured Postseason)	Δ From Baseline	*P* Value[a]
Anthropometric measures				
Weight (kg)				
Control	102.0 (2.4)	103.5 (2.4)	1.4 (0.6)	**0.03**
Case	101.8 (2.4)	103.1 (2.4)	1.3 (0.6)	**0.05**
Blood pressure				
SBP (mm Hg)				
Control	131.6 (1.9)	132.7 (1.9)	1.1 (1.9)	0.58
Case	129.6 (2.1)	131.7 (2.1)	2.1 (2.2)	0.35
DBP (mm Hg)				
Control	76.9 (1.7)	74.9 (1.7)	−2.0 (2.1)	0.35
Case	74.5 (1.6)	76.6 (1.6)	2.1 (1.8)	0.26
Cardiac structure and function				
LV mass index (g/m^2)				
Control	94.4 (1.7)	101.6 (1.7)	7.2 (1.9)	**<0.001**
Case	97.2 (2.0)	102.8 (2.0)	5.6 (1.9)	**0.006**
Tissue Doppler É velocity (cm/s)				
Control	15.8 (0.4)	15.3 (0.4)	−0.4 (0.3)	0.19
Case	15.3 (0.4)	14.5 (0.4)	−0.7 (0.5)	0.18
PWV (m/s)				
Control	5.3 (0.1)	5.2 (0.1)	−0.1 (0.1)	0.38
Case	5.1 (0.1)	5.5 (0.1)	0.4 (0.2)	**0.036**

Bold indicates $P \leq 0.05$ is significant. [a]*P* value notes significant change from baseline.
Abbreviations as in Table 1.

significantly higher for cases. Among the 14 case and control pairs followed over 2 seasons after concussion exposure, differences in ΔSBP (Δ9.93 [95% CI: 0.42-19.44] mm Hg, $P = 0.046$) and ΔDBP (Δ12.36 [95% CI: 2.70-22.01] mm Hg, $P = 0.015$) were also significantly higher for cases. In addition, the prevalence of combined stage 1 and 2 hypertension[20] among cases in the 2-year cohort increased over time (36% at preseason freshman vs 86% at postseason sophomore year, $P = 0.02$) compared to no change among controls (71% at preseason freshman vs 57% at postseason sophomore year, $P = 0.69$).

In our sensitivity analysis, when comparing longitudinal changes for the 39 case and control pairs by chronological progression through 2-years of collegiate ASF participation, differences for ΔSBP (Δ8.00 [95% CI: 1.98-14.03] mm Hg, $P = 0.01$) were significantly higher for cases.

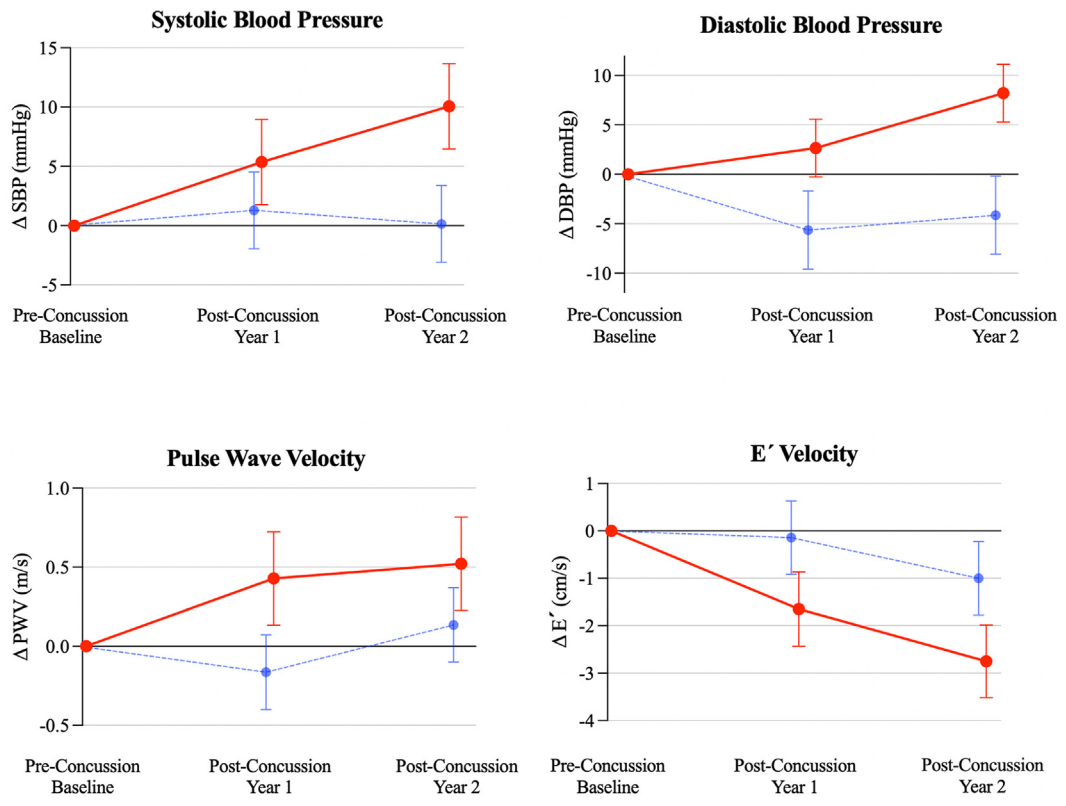
CONCUSSIONS ARE ASSOCIATED WITH ACQUIRED MALADAPTIVE CARDIOVASCULAR PHENOTYPES. In multivariable analyses of the 1-year case and control cohort adjusting for weight, player position, and race, sustained concussions were independently associated with higher PWV (β = 0.33 [95% CI: 0.09-0.56], $P = 0.007$) (Table 4). In select univariate analyses, sustained concussions were associated with higher PWV [β = 0.36 (95% CI: 0.12-0.60), $P = 0.003$] and LV

TABLE 3 Longitudinal Changes in Select Measurements in Cases (n = 14) and Controls (n = 14) After 2 Complete Collegiate American-Style Football Season

	Preconcussion Baseline (Measured Preseason)	Postconcussion Year 1	Δ from Baseline	*P* Value[a]	Postconcussion Year 2	Δ From Baseline	*P* Value[a]
Anthropometric measures							
Weight (kg)							
Control	99.2 (3.1)	100.4 (3.1)	1.2 (1.0)	0.21	101.7 (3.1)	2.4 (1.0)	**0.02**
Case	98.7 (3.5)	100.7 (3.5)	2.0 (1.2)	0.11	102.6 (3.5)	3.9 (1.2)	**0.003**
Blood pressure							
SBP (mm Hg)							
Control	133.2 (3.3)	134.5 (3.3)	1.3 (3.2)	0.70	133.4 (3.3)	0.1 (3.2)	0.97
Case	125.1 (3.5)	130.4 (3.5)	5.4 (3.6)	0.15	135.1 (3.5)	10.1 (3.6)	**<0.01**
DBP (mm Hg)							
Control	77.1 (2.8)	71.5 (2.8)	−5.6 (4.0)	0.17	73.0 (2.8)	−4.1 (4.0)	0.30
Case	74.9 (2.6)	77.6 (2.6)	2.6 (2.9)	0.38	83.1 (2.6)	8.2 (2.9)	**<0.01**
Cardiac structure and function							
LV mass index (g/m^2)							
Control	89.9 (2.5)	99.9 (2.5)	10.0 (2.9)	**0.002**	102.1 (2.5)	12.3 (2.9)	**<0.001**
Case	92.9 (2.9)	104.2 (2.9)	11.3 (3.1)	**0.001**	110.2 (2.9)	17.3 (3.1)	**<0.001**
Tissue Doppler É velocity (cm/s)							
Control	16.8 (0.8)	16.6 (0.8)	−0.1 (0.8)	0.86	15.8 (0.8)	−1.0 (0.8)	0.21
Case	15.8 (0.7)	14.2 (0.7)	−1.6 (0.8)	**0.05**	13.1 (0.7)	−2.8 (0.8)	**0.001**
PWV (m/s)							
Control	5.2 (0.2)	5.0 (0.2)	−0.2 (0.2)	0.49	5.3 (0.2)	0.1 (0.2)	0.57
Case	5.2 (0.3)	5.6 (0.3)	0.4 (0.3)	0.16	5.7 (0.3)	0.5 (0.3)	0.09

Bold indicates $P \leq 0.05$ is significant. [a]*P* value notes significant change from baseline.
Abbreviations as in Table 1.

FIGURE 2 Comparison of Changes in Primary Outcome Measures From Baseline Over 2 Complete Collegiate American-Style Football Seasons



Concussion = red line; no concussion = blue Line. DBP = diastolic blood pressure; PWV = pulse wave velocity; SBP = systolic blood pressure.

mass index (β = 5.31 [95% CI: 1.64-8.97], $P = 0.005$) and lower É (β = −0.80 [95% CI: −1.59 to −0.01], $P = 0.047$). In multivariable analyses of the 2-year case and control cohort, sustained concussions were independently associated with higher PWV (β = 0.42 [95% CI: 0.05-0.78], $P = 0.03$), DBP (β = 5.89 [95% CI: 1.23-10.54], $P = 0.01$), and LV mass index (β = 11.01 [95% CI: 6.13-15.90], $P < 0.001$), and lower É (β = −2.11 [95% CI: −3.27 to −0.95], $P < 0.001$, **Table 3**).

In our sensitivity analysis, multivariable analyses were similar (see Supplemental Table 3). Incorporating the total case and control cohort and adjusting for weight, player position, and race, sustained concussions were independently associated with higher PWV (β = 0.35 [95% CI: 0.16-0.54], $P < 0.001$) and LV mass index (β = 8.12 [95% CI: 4.75-11.50], $P < 0.001$) and lower É (β = −1.25 [95% CI: −1.97 to −0.54], $P < 0.001$).

DISCUSSION

To our knowledge, this is the first longitudinal analysis characterizing the chronic and persistent cardiovascular sequelae associated with concussions sustained during ASF participation. Key findings from this study are as follows. First, compared to matched ASF athlete controls, collegiate ASF athletes who sustained concussion experienced early arterial stiffening, an established independent cardiovascular risk factor and primary mechanistic precursor to overt hypertension,[32] followed temporally by significant increases in blood pressure. Second, concussions were associated with a constellation of acquired maladaptive cardiovascular phenotypes including arterial stiffening, LVH, and reductions in diastolic function. Critically, these maladaptive changes were independent of previously established ASF-specific

TABLE 4 Multivariable Analyses[a] of Primary Outcome Measures and Sustained Concussions in the Total Cohort of 39 Case and Control Pairs Followed for 1 Complete Season and Subcohort of 14 Case and Control Pairs Followed for 2 Complete Seasons

	1-Year Cohort		2-Year Cohort	
	Estimate (95% CI)	*P* Value	Estimate (95% CI)	*P* Value
	Systolic Blood Pressure (mm Hg)		**Systolic Blood Pressure (mm Hg)**	
Concussion	0.43 (−3.17 to 4.02)	0.81	3.49 (−1.53 to 8.50)	0.17
Position	−1.82 (−8.51 to 4.86)	0.59	−0.21 (−11.89 to 11.47)	0.97
Black race	−0.95 (−5.9 to 3.99)	0.70	−0.16 (−8.85 to 8.53)	0.97
Weight (kg)	0.32 (0.16-0.48)	**<0.001**	0.36 (0.04-0.69)	**0.03**
	Diastolic Blood Pressure (mm Hg)		**Diastolic Blood Pressure (mm Hg)**	
Concussion	1.34 (−2.01 to 4.69)	0.43	5.89 (1.23-10.54)	**0.01**
Position	0.89 (−4.57 to 6.36)	0.75	6.67 (−1.76 to 15.10)	0.12
Black race	−0.77 (−4.80 to 3.26)	0.71	−3.03 (−9.16 to 3.11)	0.33
Weight (kg)	0.13 (0.002-0.26)	**0.05**	−0.11 (−0.35 to 0.13)	0.37
	É Velocity (cm/s)		**É Velocity (cm/s)**	
Concussion	−0.76 (−1.54 to 0.03)	0.06	−2.11 (−3.27 to −0.95)	**<0.001**
Position	0.21 (−1.29 to 1.70)	0.78	−0.96 (−3.57 to 1.66)	0.47
Black race	−0.36 (−1.47 to 0.75)	0.52	−0.15 (−2.09 to 1.80)	0.88
Weight (kg)	−0.04 (−0.07 to −0.003)	**0.03**	−0.01 (−0.08 to 0.06)	0.77
	Pulse Wave Velocity (m/s)		**Pulse Wave Velocity (m/s)**	
Concussion	0.33 (0.09-0.56)	**0.007**	0.42 (0.05-0.78)	**0.03**
Position	0.08 (−0.33 to 0.49)	0.69	0.29 (−0.47 to 1.04)	0.45
Black race	−0.18 (−0.48 to 0.12)	0.24	−0.44 (−0.99 to 0.12)	0.12
Weight (kg)	0.01 (0.004-0.02)	**0.01**	0.01 (−0.02 to 0.03)	0.56
	LV Mass Index (g/m^2)		**LV Mass Index (g/m^2)**	
Concussion	5.18 (1.5-8.87)	**0.007**	11.01 (6.13-15.90)	**<0.001**
Position	−1.00 (−7.67 to 5.68)	0.77	−4.31 (−13.75 to 5.13)	0.36
Black race	1.55 (−3.37 to 6.48)	0.53	2.91 (−4.0 to 9.81)	0.40
Weight (kg)	0.08 (−0.08 to 0.24)	0.33	0.10 (−0.17 to 0.36)	0.47

Bold indicates $P \leq 0.05$ is significant. [a]Multivariable analyses adjusted for weight, player position, and race. Abbreviation as in Table 1.

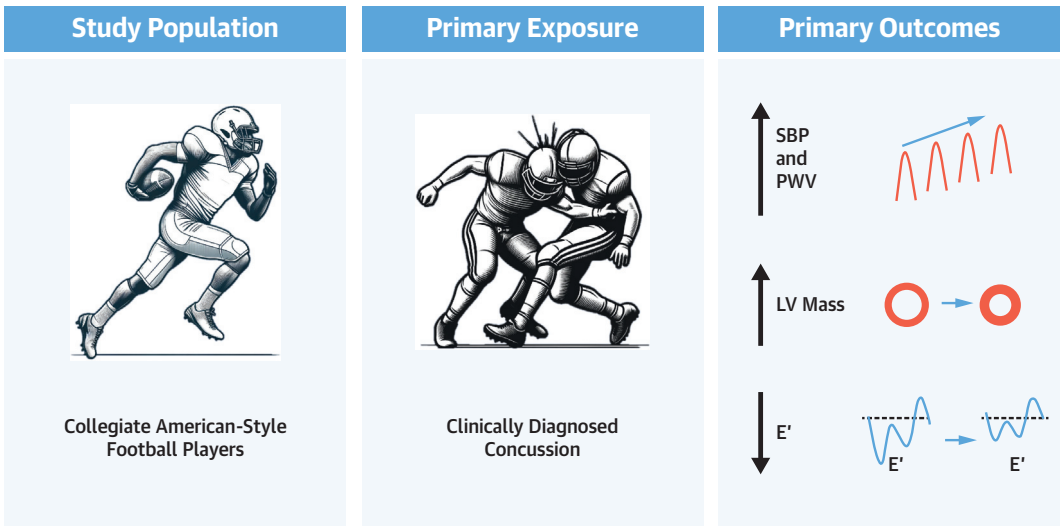
cardiovascular risk factors that include weight gain and the LM player position.[11] Taken together, our findings suggest that concussion may represent a novel, independent cardiovascular risk factor among competitive ASF athletes (**Central Illustration**).

Traumatic brain injury (TBI) is associated with the development of hypertension and other cardiometabolic diseases that include hyperlipidemia, diabetes, coronary artery disease, and heart failure.[27,33-36] While sport-related concussions are considered distinct from TBI, prior retrospective studies suggest an association between ASF-sustained concussions and acquired cardiovascular risk. In a study of former National Football League (NFL) athletes, higher concussion symptom scores, generated from retrospective recall of ASF playing-time concussion burden, were associated with a higher prevalence of hypertension decades after retirement.[17] More recently, in a cross-sectional analysis of 4,080 former NFL players, recalled higher concussion symptom scores were associated with higher cardiovascular risk profiles, particularly among former players currently ≤40 years of age.[18] Finally, in another retrospective study, higher odds of ischemic stroke were observed among former NFL athletes who recalled ≥10 concussions during their playing career.[37] However, notable limitations of these prior studies include the retrospective ascertainment of concussion exposure and significant temporal delay in between recalled concussions and the assessment of cardiovascular risk profiles.

Findings from this study, utilizing prospective data collection of both concussion exposure and cardiovascular physiology among active collegiate ASF athletes, provide crucial insights and suggest a causal relationship between sustained concussions and acquired cardiovascular risk. Critically, our findings not only support prior retrospective data, suggesting an enduring intensification of cardiovascular risk throughout the lifespan of the ASF athlete[17,37] but also establish that vascular dysfunction, acquired hypertensive blood pressures, and cardiovascular maladaptation occur in temporal proximity following a sustained concussion. An additional strength of this study comes from our sensitivity analysis in which similar trends were observed when case and control pairs were analyzed in chronological progression through 2 years of ASF participation. Proposed mechanisms underlying increased cardiovascular risk after TBI, which may also be relevant for ASF-sustained concussions, include indirect considerations such as sedentary lifestyle choices after significant head trauma and direct pathologic neurophysiologic mechanisms including autonomic dysfunction and neuroinflammation.[35,38,39] The connection between the brain and heart after a sport-related concussion has previously been recognized with potential cardiovascular physiologic sequelae extending past complete neurologic recovery.[40]

Previous longitudinal data, absent consideration of the impact of concussions, have established that collegiate ASF athletes are at risk for hypertension and the development of maladaptive cardiovascular phenotypes that include arterial stiffening,[9] concentric LV hypertrophy,[10] and reduced LV diastolic function.[11-14,41] Rapid weight gain,[11] and other pathologic mediators that include sleep apnea[42] and NSAID overuse,[16] likely represent key and multifactorial mechanistic factors underlying these observations. Our findings add to the complex mechanistic underpinnings of elevated early life cardiovascular risk profiles observed among ASF athletes. It is noteworthy that the association between concussions and our measures of increased cardiovascular risk were

CENTRAL ILLUSTRATION **Concussion and Cardiovascular Risk in American-Style Football Athletes**



Rim AJ, et al. JACC Adv. 2025;4(5):101717.

LV = left ventricular; other abbreviations as in Figure 2.

generally independent of significant weight gain, which has been established by prior studies as the most potent predictor of early cardiovascular risk among ASF athletes.[11,41] Future longitudinal analyses of active and former ASF athletes with and without prior concussions, inclusive of the methodology employed in this study along with detailed mechanistic analyses and long-term clinical outcomes, are warranted.

There are clinical implications of our findings that warrant reconsideration of recommended medical algorithms in the management of the concussed athlete and for determination of return-to-play.[43] Currently, a brief period (24-48 hours) of relative cognitive and physical rest with a gradual return to sports activity represents the gold standard of medical management after ASF-sustained concussions.[23,44,45] Persistent physiological dysfunction, however, has been found among athletes who sustain sports-related concussion.[46] Consistent with these prior findings, our data suggest that the inclusion of longitudinal surveillance for incident hypertension, even after complete clinical neurologic recovery, should be considered as an adjunct to the standard clinical paradigm of postconcussion care. For ASF athletes with confirmed hypertension, guideline-based interventions, which include lifestyle modifications and initiation of antihypertensive pharmacotherapy, if appropriate, should be considered. Significant clinical implications for former ASF athletes arise with unrecognized hypertension in the context of prior ASF-sustained concussions in which earlier recognition, diligent surveillance, and appropriate therapeutic interventions could alter and likely improve both later-life cardiovascular and neurocognitive outcomes. Hypertension and other treatable cardiovascular conditions should be studied as potential mediators or modifiers of the association between concussion and long-term cognitive dysfunction.

STUDY LIMITATIONS. We acknowledge several limitations in this study. First, prevalent NSAID use[16,21]; sleep apnea; alcohol intake; prior exercise-training exposures; and stimulant, recreational drug, and PED use all represent potential confounders. However, there were no differences in prescription stimulant use or prospectively reported NSAID use among the case and control participants who were previously queried,[16] and there were no positive PED tests within this entire cohort. In addition, random matching of the cases and controls by player position and weight likely ensured that the prevalence of sleep apnea and dietary intake habits (including alcohol and recreational drugs) were similar between the case and control cohorts. Second, the effects of concussions sustained prior to college matriculation are

unknown. However, the distribution of prior concussions in cases and controls was likely random, and consideration of selection bias would imply a higher-risk of recurrent concussions among those athletes with previously sustained concussions.[29] Moreover, concussions more often occur during collegiate ASF participation compared to high school ASF.[47] Third, although we relied on the clinical diagnosis of concussion by experienced sports medicine providers, additional granular clinical details, including specifics of the mechanism of concussion, concussion symptomatology, qualitative severity of symptoms (including loss of consciousness), and total time away from training and competition were not available, thus limiting our ability to assess the association between these descriptive factors and our primary outcome measures. Fourth, while both institutions in this study employed similar clinical protocols adapted from SCAT5 to diagnose concussion[22] and the medical and athletic training personnel at both schools were consistent throughout the study period, the clinical evaluations for concussion may not have been identical across sites. Fifth, while not extensive, we experienced case subject attrition over the study period. Finally, this study was focused only on collegiate ASF athletes and therefore not fully generalizable to other levels of ASF play or sports with risks of head injury and concussion.

CONCLUSIONS

Sustained concussions during collegiate ASF participation are associated with arterial stiffening, significant increases in blood pressure, and other acquired, clinically relevant maladaptive cardiovascular phenotypes. As such, ASF-sustained concussions appear to represent a novel cardiovascular risk factor among active ASF athletes. Our findings add further credence to the importance of cardiac surveillance, prevention, and treatment among ASF athletes, particularly those who experience concussion, with the potential to improve later-life cardiovascular and cognitive outcomes.

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PERSPECTIVES

COMPETENCY IN MEDICAL KNOWLEDGE: Sport-related concussion may represent a novel cardiovascular risk factor for active ASF athletes associated with increases in SBP and maladaptive cardiovascular phenotypes. As such, surveillance for incident hypertension during the recovery process after a sustained concussion may be an important additional consideration in the clinical management of ASF athletes.

TRANSLATIONAL OUTLOOK: Future longitudinal research efforts are needed to evaluate the diagnostic yield of serial blood pressure monitoring after ASF-associated concussions and impact of more intensive blood pressure surveillance and management on long-term clinical cardiovascular outcomes among ASF athletes who sustain concussions.

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KEY WORDS **American-style football, arterial stiffness, athlete, concussion, hypertension, sports**

APPENDIX **For supplemental tables, please see the online version of this paper.**