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Attenuated Cardiovascular Responses to the Cold Pressor Test in Concussed Collegiate Athletes

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Context: Cardiovascular responses to the cold pressor test (CPT) provide information regarding sympathetic function.

Objective: To determine if recently concussed collegiate athletes had blunted cardiovascular responses during the CPT.

Design: Cross-sectional study.

Setting: Laboratory.

Patients or Other Participants: A total of 10 symptomatic concussed collegiate athletes (5 men, 5 women; age = 20 ± 2 years) who were within 7 days of diagnosis and 10 healthy control individuals (5 men, 5 women; age = 24 ± 4 years).

Intervention(s): The participants' right hands were submerged in agitated ice water for 120 seconds (CPT).

Main Outcome Measure(s): Heart rate and blood pressure were continuously measured and averaged at baseline and every 30 seconds during the CPT.

Results: Baseline heart rate and mean arterial pressure were not different between groups. Heart rate increased throughout 90 seconds of the CPT (peak increase at 60 seconds = 16 ± 13 beats/min; $P < .001$) in healthy control participants but remained unchanged in concussed athletes (peak increase at 60 seconds = 7 ± 10 beats/min; $P = .08$). We observed no differences between groups for the heart rate response ($P > .28$). Mean arterial pressure was elevated throughout the CPT starting at 30 seconds (5 ± 7 mm Hg; $P = .048$) in healthy control individuals (peak increase at 120 seconds = 26 ± 9 mm Hg; $P < .001$). Mean arterial pressure increased in concussed athletes at 90 seconds (8 ± 8 mm Hg; $P = .003$) and 120 seconds (12 ± 8 mm Hg; $P < .001$). Healthy control participants had a greater increase in mean arterial pressure starting at 60 seconds ($P < .001$) and throughout the CPT than concussed athletes (peak difference at 90 seconds = 25 ± 10 mm Hg and 8 ± 8 mm Hg, respectively; $P < .001$).

Conclusions: Recently concussed athletes had blunted cardiovascular responses to the CPT, which indicated sympathetic dysfunction.

Key Words: heart rate, blood pressure, mild traumatic brain injury, autonomic nervous system

Key Points

- Cardiovascular responses to the cold pressor test were blunted and delayed in recently concussed collegiate athletes.
- Cardiovascular responses to the cold pressor test were not correlated with concussion symptoms, which indicates that concussion symptoms might not reflect impaired physiology.
- Sport-related concussion caused sympathetic-cardiovascular dysfunction during a sympathoexcitatory maneuver.

Brainstem lesions and abnormalities have been found in concussed patients using neuroimaging techniques,[1] providing evidence that autonomic dysfunction could arise after a concussion. Heart rate variability, which provides information primarily on parasympathetic activity,[2] has been shown to be reduced in concussed patients at rest,[3,4] but these findings have not been consistently reported.[5–8] Heart rate variability; heart rate complexity; and cardiovascular responses to sympathoexcitatory maneuvers, such as exercise, orthostatic challenges, and the Valsalva maneuver, were blunted in concussed patients.[3,6–9] Therefore, evoking a stressor that stimulates the autonomic nervous system and increases heart rate and blood pressure might be an appropriate way to uncover autonomic dysfunction in concussed patients.

The cold pressor test (CPT) is a simple nonpharmacologic test of sympathetic activation that has been used to study the autonomic nervous system in a variety of populations.[10–16] Submerging the hand in cold water stimulates the cold-sensitive nociceptive fibers, evoking large increases in sympathetic neural activity and increasing heart rate and blood pressure in healthy participants.[17] In contrast, exercise, orthostatic challenges, and the Valsalva maneuver activate multiple pathways that influence sympathetic output (ie, central command, mechanoreflex, metaboreflex, baroreflex), so the CPT might be a simpler test that could be used to detect sympathetic dysfunction in concussed patients. Therefore, the purpose of our study was to determine if symptomatic concussed collegiate athletes exhibited abnormal cardiovascular responses to the CPT. We hypothesized that sympathetically mediated increases in heart rate and blood pressure during the CPT would be attenuated in symptomatic concussed collegiate athletes compared with healthy control individuals.

Table 1. Participant Demographics

Characteristic	Group		t_{18} Value[a]	*P* Value
	Symptomatic Concussed Athletes	Healthy Control Participants		
Sex (males/females), no.	5/5	5/5	NA	NA
	Mean ± SD			
Age, y	20 ± 2	24 ± 4	3.35	.004[b]
Height, cm	178 ± 13	177 ± 13	0.30	.77
Mass, kg	90 ± 28	77 ± 17	1.27	.22
Body mass index, kg/m^2	28 ± 5	24 ± 3	1.88	.08
Time postconcussion, d	5 ± 2	NA	NA	NA

Abbreviation: NA, not applicable.

[a] Demographic values were compared between groups using unpaired *t* tests.

[b] Indicates a difference ($P < .05$).

METHODS

Participants

Concussed athletes were referred to us from the University at Buffalo Concussion Management Clinic. Physically active healthy control participants were recruited at random from the community. Participants consisted of 10 collegiate athletes (5 men, 5 women) who were diagnosed with a sport-related concussion within the 7 days before the study and 10 healthy individuals serving as the control group (5 men, 5 women) who self-reported that they were recreationally active and had not sustained a concussion in the year before the study (Table 1). Concussed athletes had a Glasgow Coma Scale score of 15. They participated in National Collegiate Athletic Association Division I, II, or III sports (eg, basketball, football, lacrosse, rowing, volleyball, or wrestling). Our clinic avoids the use of "cocoon therapy" (ie, isolation in a dark room), and the concussed athletes were encouraged to perform light physical activity (eg, walking, stationary cycling) without exacerbating symptoms from the time of their injury to the day of the study visit. We studied recreationally active control participants to avoid any potential confounding effects of repeated nonconcussive head impacts that athletes who participate in collision sports might experience.[18–25]

We used the postinjury Symptom Evaluation from the Sport Concussion Assessment Tool, fifth edition, which is a 22-item Likert scale questionnaire, to rate the symptom severity at the time of the study in the concussed athletes (*none* = 0, *mild* = 1–2, *moderate* = 3–4, *severe* = 5–6).[26,27] The symptoms listed on the questionnaire were headache, "pressure in the head," neck pain, nausea or vomiting, dizziness, blurred vision, balance problems, sensitivity to light, sensitivity to noise, feeling slowed down, feeling like "in a fog," "don't feel right," difficulty concentrating, difficulty remembering, fatigue, confusion, drowsiness, more emotional, irritability, sadness, nervous or anxious, and trouble falling asleep.[26,28] The responses to the questionnaire were grouped into 4 symptom domains: *physical*, *cognitive*, *emotional*, and *fatigue*. The numeric sum of the responses was the symptom severity score (range = 0–132). All concussed athletes reported concussion symptoms on the day of the study visit. All participants self-reported that they were free from any cardiovascular, metabolic, neurologic, respiratory, or endocrine disease; were nonsmokers; and were not taking any medications (except birth control). All participants provided written informed consent, and the study was approved by our university's institutional review board.

Instruments

Height and weight were measured using a stadiometer and scale (Sartorius, Bohemia, NY) at the beginning of the study visit. Three-lead electrocardiography (model DA100C; Biopac Systems Inc, Goleta, CA) was used to continuously record heart rate. Beat-to-beat blood pressure was collected (ccNexfin; BMEYE BV, Amsterdam, The Netherlands, or Finometer Pro; FMS, Amsterdam, The Netherlands) using the Penaz method. It was confirmed via auscultation of the brachial artery via electrosphygmomanometry (Tango M2; SunTech, Raleigh, NC), and no corrections were necessary. Stroke volume was calculated using Modelflow.[29]

Procedures

Participants were instructed to report to the laboratory on the day of the study visit after refraining from alcohol and caffeine consumption and exercise for 12 hours and food consumption for 2 hours.[28] With participants in the supine position, instrumentation was applied and they continued to rest quietly for 10 minutes. We then collected baseline data for 5 minutes before initiating the CPT. During the CPT, the participants' right hands were submerged in agitated ice water (approximately 0°C) up to the wrist for 120 seconds. After the CPT, we collected data while the participants remained in the supine position for 60 seconds.

Data Analyses

We used a data-acquisition system (model MP150; Biopac Systems Inc) to record data continuously at 1 kHz. Data were analyzed over 3 minutes at baseline, every 30 seconds during the CPT, and at 30 and 60 seconds after the CPT. Cardiac output was calculated as the product of heart rate and stroke volume. Total peripheral resistance was calculated as the quotient of mean arterial pressure and cardiac output. The peak change from baseline was determined using the greatest change value obtained during the 30-second increments of the CPT for heart rate, mean

Table 2. Baseline Cardiovascular Values

Measure	Group, Mean ± SD		t_{18} Value[a]	*P* Value
	Symptomatic Concussed Athletes	Healthy Controls		
Heart rate, beats/min	57 ± 8	60 ± 8	1.01	.33
Stroke volume, mL	117 ± 23	106 ± 22	1.07	.30
Cardiac output, L/min	7.6 ± 2.0	6.1 ± 0.7	2.00	.06
Mean arterial pressure, mm Hg	96 ± 13	87 ± 9	1.85	.08
Systolic blood pressure, mm Hg	132 ± 19	121 ± 11	1.66	.12
Diastolic blood pressure, mm Hg	74 ± 10	67 ± 6	1.84	.08
Total peripheral resistance, mm Hg/L/min	13.4 ± 3.6	14.5 ± 2.3	0.73	.47

[a] Baseline values were compared between groups using unpaired *t* tests.

arterial pressure, systolic blood pressure, and diastolic blood pressure.

Statistical Analyses

Unpaired *t* tests were used to compare baseline variables between groups. A mixed-model repeated-measures analysis of variance with group as a between-subjects condition and time as a within-subjects condition was used to compare cardiovascular responses to the CPT. If the analysis of variance revealed an interaction or main effect, we performed the Holm-Sidak post hoc procedure to determine where differences existed. Data over time were compared with those acquired at baseline. The peak changes in heart rate, mean arterial pressure, systolic blood pressure, and diastolic blood pressure during the CPT were compared using unpaired *t* tests. A Spearman correlation (*r*) was also performed to determine if the peak cardiovascular responses (ie, heart rate, mean arterial pressure, systolic blood pressure, and diastolic blood pressure) during the CPT were correlated with the symptom severity score from the Symptom Evaluation of the Sport Concussion Assessment Tool, fifth edition, in the concussed athletes. To determine if age contributed to mean arterial pressure responses, we conducted post hoc analyses consisting of the Spearman correlation and analysis of covariance. Prism software (version 8; GraphPad Software, La Jolla, CA) was used to perform the statistical analyses. Unless otherwise indicated, data are reported as a change from baseline (mean ± standard deviation), and the α level was set a priori at .05.

RESULTS

Heart Rate, Stroke Volume, and Cardiac Output

Baseline data are presented in Table 2, and changes from the baseline data are illustrated in Figure 1. We observed no differences at baseline between concussed athletes and healthy control participants for heart rate ($t_{18} = 1.01$, $P = .33$), stroke volume ($t_{18} = 1.07$, $P = .30$), or cardiac output ($t_{18} = 2.00$, $P = .06$). Heart rate in the concussed athletes did not change throughout the CPT ($P > .082$). In the healthy control group, heart rate increased beginning at 30 seconds (14 ± 7 beats/min; $P < .001$) and remained elevated at 90 seconds of the CPT, with a peak increase at 60 seconds (16 ± 13 beats/min; $P < .001$). Despite that increase, we found no differences between the concussed athletes and healthy control participants at any point during the CPT ($P > .28$). A transient reduction in stroke volume among the concussed athletes was noted starting at 30 seconds (–12 ± 10 mL; $P < .001$) and lasting until 90 seconds (–9 ± 9 mL; $P < .007$). Stroke volume in the healthy control group was unchanged throughout the CPT ($P > .15$). We demonstrated no differences in stroke volume between groups ($P > .62$). Cardiac output did not change in the concussed athletes ($P > .99$) but increased in the healthy control individuals at 30 seconds (1.0 ± 1.2 L/min; $P = .03$) and 60 seconds (1.0 ± 1.3 L/min; $P = .03$). However, we identified no differences in cardiac output between groups ($P = .12$).

Blood Pressure and Total Peripheral Resistance

Baseline data are provided in Table 2, and the changes from baseline data are shown in Figure 2. We observed no differences at baseline between groups for mean arterial pressure ($t_{18} = 1.85$, $P = .08$), systolic blood pressure ($t_{18} = 1.66$, $P = .12$), diastolic blood pressure ($t_{18} = 1.84$, $P = .08$), or total peripheral resistance ($t_{18} = 0.73$, $P = .47$). Mean arterial pressure in the concussed athletes increased at 90 seconds (8 ± 8 mm Hg; $P = .003$) and 120 seconds (12 ± 8 mm Hg; $P < .001$). Mean arterial pressure in the healthy control participants increased throughout the CPT, starting at 30 seconds (5 ± 7 mm Hg; $P = .048$) and peaking at 120 seconds (26 ± 9 mm Hg; $P < .001$). The increase in mean arterial pressure was greater in the healthy control group at 60 seconds ($P < .001$), 90 seconds ($P < .001$), and 120 seconds ($P < .001$) of the CPT. Systolic blood pressure increased at 120 seconds in the concussed athletes (11 ± 10 mm Hg; $P = .003$) and was increased throughout the CPT, starting at 60 seconds in the healthy control individuals (17 ± 13 mm Hg; $P < .001$). The increase in systolic blood pressure was greater in the healthy control group at 60 seconds ($P = .006$), 90 seconds ($P < .001$), and 120 seconds ($P = .003$). The increase in diastolic blood pressure in the concussed athletes began at 60 seconds (6 ± 6 mm Hg; $P = .009$) and remained elevated throughout the CPT, with a peak increase at 120 seconds (10 ± 7 mm Hg; $P < .001$). The increase in diastolic blood pressure in the healthy control individuals began at 30 seconds (4 ± 6 mm Hg; $P = .03$) and remained elevated throughout the CPT, with a peak increase at 120 seconds (20 ± 6 mm Hg; $P < .001$). The increase in diastolic blood pressure was greater in the healthy control participants at 60 seconds ($P = .002$), 90 seconds ($P < .001$), and 120 seconds ($P = .001$). We observed no increases in total peripheral resistance in the concussed athletes throughout the CPT ($P > .42$). In the

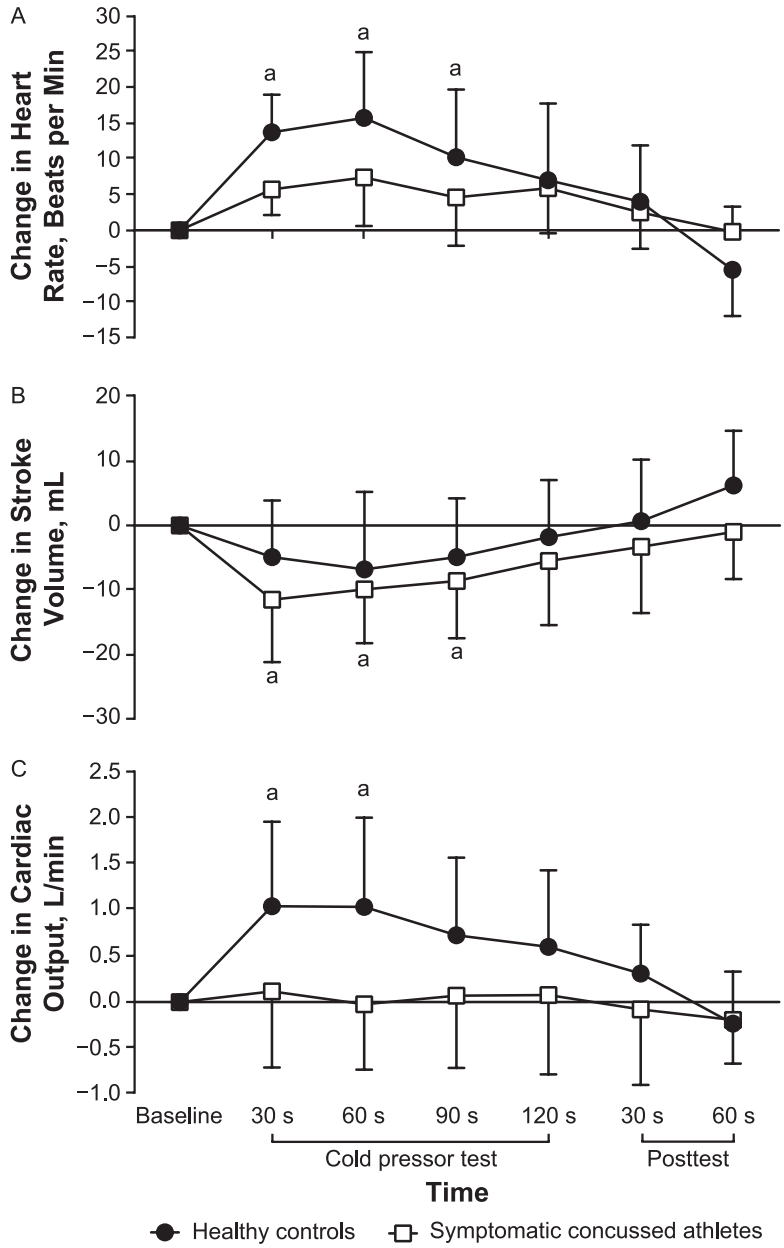

Figure 1. Changes in, A, heart rate, B, stroke volume, and C, cardiac output from baseline during 2 minutes of the cold pressor test in healthy control participants and symptomatic concussed athletes. Data were compared using a mixed-model repeated-measures analysis of variance; if an interaction or main effect was present, we conducted the Holm-Sidak post hoc procedure to determine where differences existed. Data are presented as mean ± standard deviation. [a] Different from baseline ($P < .05$).

healthy control group, an increase in total peripheral resistance began at 90 seconds (2.8 ± 3.2 mm Hg/L/min; $P = .002$) and peaked at 120 seconds (3.4 ± 3.5 mm Hg/L/min; $P < .001$). We found no differences in total peripheral resistance between groups ($P = .24$).

Peak Heart Rate and Blood Pressure Responses

The peak heart rate and blood pressure responses are presented in Figure 3. The peak change in heart rate was not different between the concussed athletes (12 ± 8 beats/min) and healthy control participants (18 ± 12 beats/min; $t_{18} = 1.23$, $P = .12$). The latter had greater peak increases in mean arterial pressure (13 ± 7 versus 27 ± 9 mm Hg, respectively; $t_{18} = 3.95$, $P < .001$), systolic blood pressure (12 ± 10 versus 30 ± 13 mm Hg, respectively; $t_{18} = 3.27$, $P = .002$), and diastolic blood pressure (11 ± 5 versus 21 ± 6 mm Hg, respectively; $t_{18} = 3.87$, $P < .001$) during the CPT than the concussed athletes.

Correlations Between the Cardiovascular Responses and Symptom Severity

The Spearman correlation r values between the peak cardiovascular responses during the CPT (ie, heart rate, mean arterial pressure, systolic blood pressure, and diastolic blood pressure) and symptom severity scores are presented in Table 3. We noted no correlations between the cardiovascular responses during the CPT and the symptom severity scores.

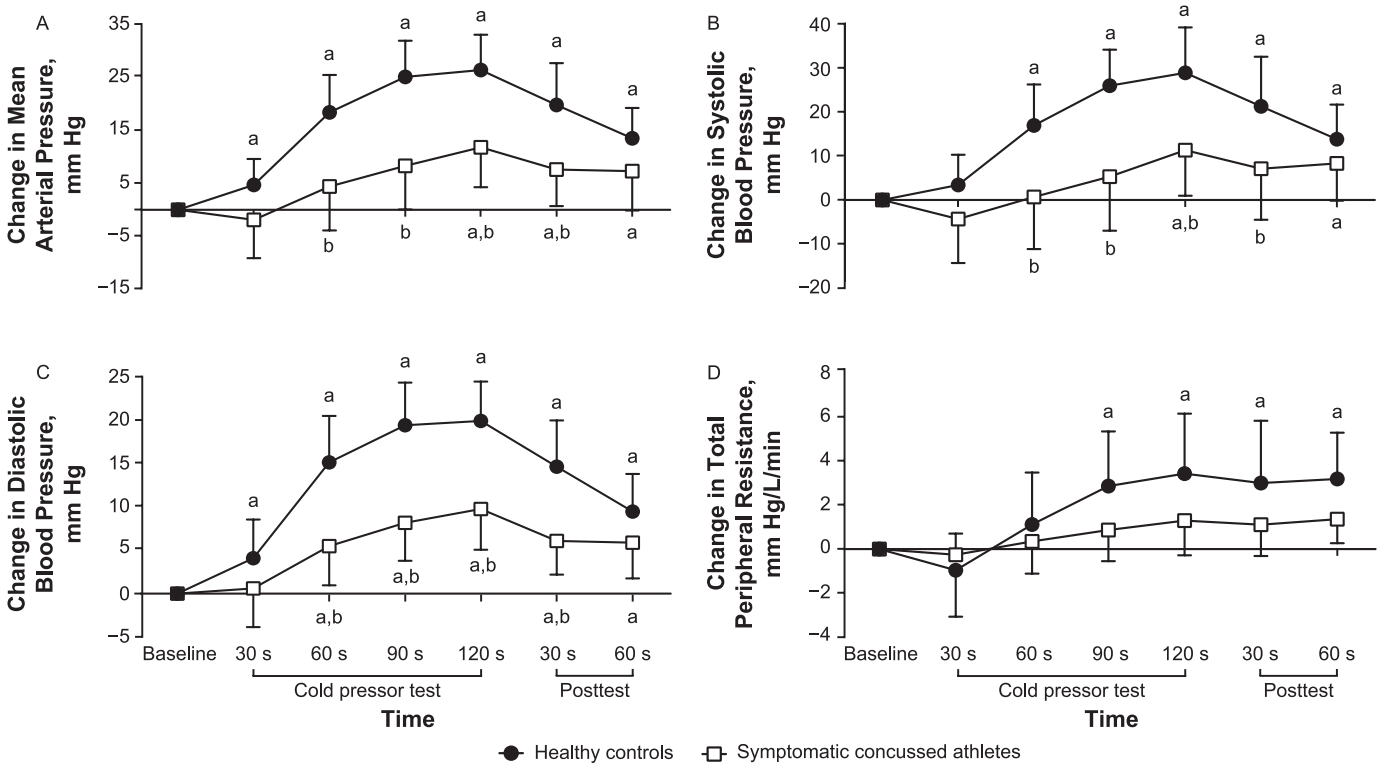

Figure 2. Changes in, A, mean arterial pressure, B, systolic blood pressure, C, diastolic blood pressure, and D, total peripheral resistance from baseline during 2 minutes of the cold pressor test in healthy control individuals and symptomatic concussed athletes. Data were compared using a mixed-model repeated-measures analysis of variance; if an interaction or main effect was present, we conducted the Holm-Sidak post hoc procedure to determine where differences existed. Data are presented as mean ± standard deviation. [a] Different from baseline ($P < .05$). [b] Different from healthy control participants ($P < .05$).

DISCUSSION

We are the first to demonstrate that symptomatic concussed collegiate athletes exhibited attenuated and delayed cardiovascular responses to the CPT compared with a healthy active control group. The blunted and delayed increases in heart rate and blood pressure in response to a sympathetic stimulus suggested that the recently concussed patients had impaired sympathetic neural activation.

During the CPT, heart rate did not increase in the concussed athletes but did increase in the healthy control individuals. The increase in heart rate during the CPT was largely due to sympathetic activation and not to withdrawal of cardiac parasympathetic activity.[30] Therefore, the lack of a rise in heart rate in the concussed athletes during the CPT suggested that cardiac sympathetic activity did not increase. However, we did not obtain direct measures of cardiac sympathetic nerve activity and therefore cannot definitively state that the absent heart rate response in the concussed athletes was due to an attenuated sympathetic response. The blunted heart rate response could also have been due to increased cardiac parasympathetic activity limiting the rise in heart rate. However, Johnson et al[28] recently demonstrated that symptomatic concussed athletes had blunted cardiac parasympathetic activation evoked by face cooling. Therefore, it is unlikely that cardiac parasympathetic activity restrained the rise in heart rate during the CPT in the concussed athletes.

In contrast to our results, other sympathoexcitatory maneuvers, such as standing,[3] the handgrip exercise,[8] and the Valsalva maneuver,[31] did not elicit blunted heart rate responses in concussed patients when compared with healthy control groups. These maneuvers activate different receptors (ie, baroreceptors, mechanoreceptors, and metaboreceptors) than the CPT (cold-sensitive nociceptive fibers). Thus, monitoring heart rate, as an index of cardiac sympathetic activation, during the CPT could better reveal sympathetic dysfunction in concussed patients than standing, the handgrip exercise, and the Valsalva maneuver. Some markers of heart rate variability, such as the root mean square of successive differences, low-frequency power, and high-frequency power, were abnormal in concussed patients at rest[31] and during standing,[3] the isometric handgrip exercise,[6,8] and submaximal whole-body aerobic exercise.[7] However, heart rate variability provides information primarily on parasympathetic activity.[2,32] Therefore, interpretations of which branch of the autonomic nervous system is dysfunctional in concussed patients during stressors that primarily activate the

Table 3. Spearman Correlations Between Peak Changes in Cardiovascular Responses and Baseline Total Symptom Severity Scores in Concussed Athletes

Variable	Symptom Severity Score	
Peak Change	*R*	*P* Value
Heart rate	−0.18	.61
Mean arterial pressure	−0.25	.48
Systolic blood pressure	−0.26	.46
Diastolic blood pressure	−0.48	.16

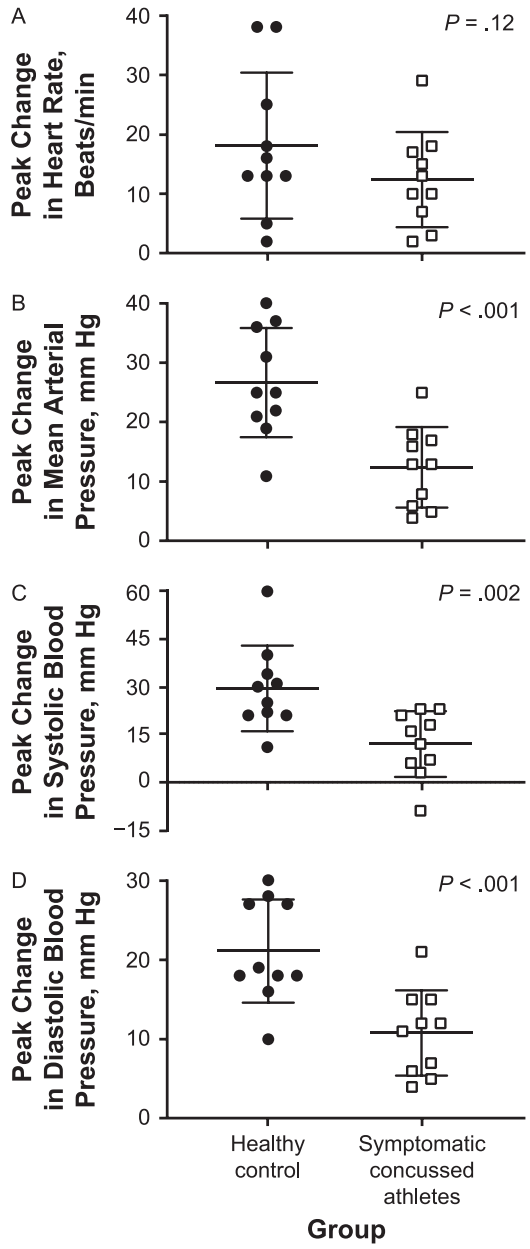

Figure 3. Peak change in, A, heart rate, B, mean arterial pressure, C, systolic blood pressure, and D, diastolic blood pressure during 2 minutes of the cold pressor test in healthy controls and symptomatic concussed athletes. Data were compared using unpaired *t* tests, and the *P* values are reported. Individual responses and the mean ± standard deviation are presented.

sympathetic nervous system based on heart rate variability should be cautious. Future research that focuses on direct measurements of sympathetic neural activity during sympathoexcitation will supply valuable information regarding autonomic activation in concussed patients.

Our concussed athletes had a delayed and blunted rise in blood pressure during the CPT. The lack of a rise in cardiac output due to no changes in heart rate and attenuated stroke volume during the first 60 seconds of the CPT in these athletes were likely responsible for most of the initial delay and blunted pressor responses. Furthermore, the absence of an increase in total peripheral resistance also appeared to be a major contributor to the blunted pressor response during the second minute of the CPT. The blunted blood pressure responses during the CPT in the concussed athletes supported our heart rate findings. Collectively, these data indicated blunted sympathetic activation in symptomatic concussed athletes. Concussed patients have a greater decrease in systolic blood pressure during standing tests, which indicates attenuated or delayed sympathetic activation during an orthostatic challenge.[9] In addition, concussed patients exhibited lower resting cardiac baroreflex sensitivity,[3,31] which might predispose them to greater decreases in blood pressure during orthostatic challenges. Our results supported the idea that sympathetic activation is attenuated in concussed patients and leads to reduced cardiovascular responsiveness. However, we did not obtain a direct measurement of sympathetic activity (eg, muscle sympathetic nerve activity) in our concussed athletes. Based on the resting heart rate and blood pressure data, it is unlikely that the concussed athletes in our study had high resting sympathetic activity that would have prohibited the rise in sympathetic activation during the CPT.

Concussion symptoms were not correlated with the peak cardiovascular responses during the CPT. Our data suggested that these symptoms might not represent physiological function, which is important because current clinical practice for return-to-play decisions is heavily reliant on subjective symptom reporting.[27] Therefore, to make informed return-to-play decisions, clinicians should account for physiological function, as well as patients' symptoms.

Limitations

Our study had several limitations. First, we did not perfectly match the concussed athletes with the healthy control participants for age, which could have contributed to the differential cardiovascular responses to the CPT. We performed post hoc analyses to determine if age contributed to our results. We found that only the mean arterial pressure and age were correlated ($r = 0.48$, $P = .03$). Next, we used an analysis of covariance with age as a covariate to statistically control for age and demonstrated that the mean arterial pressure response to the CPT was still lower in the concussed athletes after controlling for age ($P = .01$). Furthermore, the healthy control individuals reported that they were recreationally active, and both groups were young despite this small difference in age. Therefore, we do not believe any age differences contributed to the attenuated cardiovascular responses to the CPT in the concussed athletes. In this regard, previous researchers have shown that blood pressure responses during the CPT were not different between athletes and nonathletes[33] or between men and women with different levels of aerobic fitness,[34] which suggests that preinjury CPT responses in the concussed athletes would likely be similar to those of our healthy control group. We did not record physical activity in the concussed athletes from when they incurred their concussions to when they were tested; however, it is unlikely that such a short period of reduced exercise training volume and intensity contributed to reduced sympathoexcitatory responses to the CPT. Our concussed athletes were encouraged, however, to perform light exercise that did not exacerbate symptoms and avoid cocoon therapy.

Second, we found robust differences between groups for the blood pressure responses to the CPT. Yet we did not observe between-groups differences for some secondary outcome measures, likely because our study was underpowered due to the relatively small sample size.

Third, we did not assess cardiac or arterial function during the CPT. Although we cannot rule out cardiac or arterial dysfunction, the concussion itself was unlikely to have modified cardiac or arterial function in such a short period. Furthermore, any changes in cardiac or arterial function due to a presumed short period of reduced physical activity would have been unlikely to contribute to the differences in the cardiovascular responses to the CPT that we observed.[33]

Fourth, we did not use advanced imaging techniques to determine if and where cerebral abnormalities existed in either group. This approach might have given us insight into the locations of brain abnormalities and whether the injury location contributed to the blunted cardiovascular responses to the CPT that we observed in the concussed athletes.

Fifth, we did not assess perceived pain during the CPT. Although unlikely, the concussed athletes might have perceived the CPT to be less painful than the healthy control group, which may have influenced the sympathetic responses. Evidence[35] has indicated that pain threshold and pain tolerance may both be greater in athletes than in nonathletes, but the 2-minute CPT that we used was not designed to assess pain threshold or tolerance.

Sixth, we did not repeat the CPT after clinical recovery, so we do not know how long sympathetic dysfunction lasts after sustaining a concussion. We also do not know if the CPT could be used to assess recovery.

Perspectives and Importance

The subjective reporting of no concussion symptoms may lead to a false sense that the concussed athlete has recovered. Experiencing a second head injury before the brain has recovered can worsen symptoms and prolong recovery.[36–38] Therefore, identifying objective physiological biomarkers of concussion could improve both concussion diagnosis and recovery. However, an objective physiological biomarker of concussion has not been established. Some blood markers have shown promise as objective biomarkers of concussion, whereas other blood markers have not.[39] Major problems with measuring blood markers is that they require the ability to perform or enlist others to perform complex assays and the results may not be available for several days. Graded exercise stress tests have also been used to aid in concussion diagnosis by determining exercise tolerance. However, graded exercise stress tests cannot always be easily and readily conducted in most clinical settings due to the equipment and personnel requirements (eg, trained exercise physiologists). Conversely, the CPT is logistically simple and quick and does not rely on the ability of the patient to perform exercise. In this context, the CPT could possibly be used with other standard diagnostic tools soon after a suspected head injury to aid in concussion diagnosis. Further research is needed to determine the specificity and sensitivity of the CPT in aiding concussion diagnosis and to identify if cardiovascular responses to the CPT return to healthy control values after recovery.

CONCLUSIONS

Recently concussed collegiate athletes had blunted and delayed cardiovascular responses to the CPT. Our data supported the idea that sport-related concussion causes a disturbance between the sympathetic nervous system and cardiovascular function that is revealed during sympathoexcitation.

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