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Association Between Pain Catastrophizing and Pain and Cardiovascular Changes During a Cold-Pressor Test in Athletes

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Context: Athletes are often exposed to pain due to injury and competition. Using preliminary evidence, researchers have shown that cardiovascular measures could be an objective measure of pain, but the cardiovascular response can be influenced by psychological factors, such as catastrophizing.

Objective: To use a painful cold-pressor test (CPT) to measure the relationship among catastrophizing, pain, and cardiovascular variables in athletes.

Design: Cohort study.

Setting: Laboratory.

Patients or Other Participants: A total of 36 male rugby athletes (age = 24.0 ± 4.6 years, height = 180.0 ± 6.1 cm, mass = 90.5 ± 13.8 kg).

Main Outcome Measure(s): We measured catastrophizing using the Pain Catastrophizing Scale and pain using a numeric pain rating scale. Cardiovascular measures were heart rate, systolic and diastolic blood pressure, and heart rate variability.

Results: During the CPT, participants experienced increases in pain (from 0 to 4.1 ± 2.2), systolic blood pressure (from 126.7 ± 16.5 to 149.7 ± 23.4 mm Hg), diastolic blood pressure (from 76.9 ± 8.3 to 91.9 ± 11.5 mm Hg), and heart rate variability (from 0.0164 ± 0.0121 to 0.0400 ± 0.0323 milliseconds; all P values $< .001$). In addition, we observed a decrease in heart rate after the CPT ($P = .04$). We found a correlation between athletes' pain catastrophizing and both pain intensity and change in heart rate during the CPT ($P = .02$ and $P = .003$, respectively). Linear regression indicated that pain and catastrophizing explained 29% of the variance in the change in heart rate ($P = .003$).

Conclusions: Athletes who had catastrophizing thoughts were more likely to experience higher levels of pain and a greater cardiovascular response during a painful stimulus. The change in cardiovascular variables may be a good objective measure of pain in athletes in the future.

Key Words: pain-related fear, heart rate, blood pressure, sport

Key Points

- Catastrophizing was related to pain experienced during a cold-pressor test (CPT) in athletes.
- The amount of pain experienced by the athletes and pain catastrophizing were correlated with the heart rate change during the CPT.
- Peak pain, catastrophizing, and age contributed to 29.2% of the variance in heart rate changes during the CPT.
- The relationship between pain and cardiovascular changes helps to provide a link between the sympathetic nervous system and psychological pain perception and may explain an increased response to pain.
- Administering the Pain Catastrophizing Scale as a screening tool for athletes may help athletic trainers and athletic therapists interpret the clinical pain presentation during an injury and identify a factor that could prolong rehabilitation.

Athletes have a complex relationship with pain[1,2] because they are often exposed to possibly painful stimuli.[3] Sport participation and training can strain the body, potentially exposing athletes to pain.[1–4] In many contact sports, such as rugby, football, and boxing, the ability to withstand pain appears to be essential to performance and success. Athletes have been reported to have an increased capacity to endure pain compared with both sedentary and regularly active populations.[3–6] Athletes who choose to participate in contact sports are aware that they will experience pain due to contact with opponents, injuries, and exertion; however, they still engage in the activity.[4] Most athletes accept pain as "part of the game," making them inclined to play through the pain by using coping mechanisms.[2,4] Evidence reported by Finan et al[7] suggested that pain was not

necessarily proportional to the extent of an athlete's injury.

Researchers have evaluated cardiovascular measures during experimentally induced pain but rarely among athletes. Instead of using a subjective pain rating, some investigators[8,9] have proposed that cardiovascular changes can be an objective measure of pain during a cold-pressor test (CPT), for example. A CPT is a noninvasive, pain-stimulating test frequently used to study pain.[10,11] Exposing a body part to cold induces a nociceptive stimulus and stress response, causing a change in cardiovascular activity via activation of the sympathetic nervous system.[10] However, during a CPT, the pain response and resulting cardiovascular change vary in the general population. When measuring heart rate, most researchers[8,9,12] have reported an increase; yet Anwar et al[13] observed a decrease, and Atterhog et al[14] found no change. Authors[9] have supplied evidence of a correlation between subjective pain ratings and changes in heart rate. Blood pressure increased during a CPT, followed by a decrease post-CPT.[8,12] These variable responses indicate that something else is influencing the cardiovascular changes and reported pain levels.[8,14] Moreover, during a CPT in nonathletes, anxiety was an individual predictor of systolic blood pressure reactivity.[15] If athletes experience pain differently than the general population, their cardiovascular response to experimentally induced pain might also be different.

Another interesting cardiovascular measure that could be used is heart rate variability. *Heart rate variability* has been defined as the change in the time intervals between adjacent heartbeats and is part of the regulatory system for adapting to environmental and psychological challenges.[16] A common method of measuring heart rate variability is calculating the SD of the interbeat interval (SDIBI).[17,18] Although the SDIBI is a new measure in the area of athletes' pain, many researchers have used resting heart rate variability or SDIBI to explore the reaction to various conditions, including stress, depression, anxiety, and posttraumatic stress disorder. For instance, among a large adult sample, Licht et al[19] determined that heart rate variability measured using SDIBI was lower in individuals who were depressed than in a healthy control group. Friedman and Thayer[20] analyzed heart rate variability in the context of panic disorder and generalized anxiety disorder. Therefore, although heart rate variability has been examined in stress-related studies, it is a novel measure for the cardiovascular response of an athlete to pain, which is why we chose to use it in our experiment.

Psychological factors, including pain catastrophizing, may explain athletes' higher pain tolerance and varied responses to a painful stimulus. Geva and Defrin[5] reported that, compared with nonathletes, athletes displayed reduced pain catastrophizing. *Pain catastrophizing* is a negative and exaggerated psychological response to pain.[3,21] Rumination, pain magnification, and helplessness are components of catastrophizing and hinder one's ability to distract oneself from pain-related thoughts while experiencing pain.[21] Higher scores on the Pain Catastrophizing Scale (PCS) were associated with greater pain intensity after injury or surgery, which is common in an athletic population.[2,3,15,21,22] Investigators[22] have proposed that anxiety and pain catastrophizing are relevant psychological qualities and lead to overvalued pain perception. Individuals who engaged in catastrophic thinking described the highest levels of pain during pain-inducing experiments, such as a CPT,[11,23] but this variable has not been measured in athletes.

Psychological factors during a CPT can affect how one experiences pain, but it is unclear if this relationship exists in athletes. If catastrophizing can influence the amount of pain perceived by an athlete, it would have an important effect during clinical assessment and rehabilitation. So far, no one has studied the relationship between pain catastrophizing and variables such as pain and cardiovascular changes during a CPT in an athletic population playing a contact sport. Therefore, the purpose of our study was to assess pain levels in athletes during a CPT and the relationships among pain levels, catastrophizing, and cardiovascular variables. We hypothesized that the CPT would result in increased heart rate, systolic blood pressure, diastolic blood pressure, pain, and SDIBI. We also hypothesized that scores on the PCS and subjective pain ratings would be correlated throughout the test.

METHODS

Participants

A convenience sample of male rugby athletes volunteered to participate in the study. During the initial screening period, volunteers were excluded if they were smokers, were currently taking medication that alters cardiovascular function, had a diagnosis of Raynaud syndrome, or had elevated blood pressure ($>$140 mm Hg systolic or $>$90 mm Hg diastolic). To be included in the study, individuals could not participate in physical activity on the day of testing, consume alcohol or caffeine for 12 hours before testing, or consume food at least 2 hours before testing. For confidentiality purposes, we used participant numbers for identification during data analysis. All participants provided written informed consent, and the study was approved by the University Human Research Ethics Committee of Concordia University (certificate no. 30004539).

Pain, Catastrophizing, and Anxiety Measures

Pain. To measure pain intensity, we used a self-reported 11-point numeric pain rating scale that ranges from 0 (*no pain at all*) to 10 (*worst pain imaginable*). The numeric pain rating scale is a valid, reliable, and responsive tool for measuring pain.[24] It is the most common method of measuring pain and a well-established clinical measure.[24]

Pain Catastrophizing. To measure catastrophizing, we used the PCS,[21] which is a 13-item assessment of the frequency of catastrophic thoughts about a pain experience. The PCS allowed us to examine rumination (eg, "I can't seem to keep it out of my mind."), helplessness (eg, "There is nothing I can do to reduce the intensity of the pain."), and magnification (eg, "I become afraid that the pain may become worse."). Participants rate the statements with regard to how often catastrophic thoughts occur using a Likert scale, ranging from 0 (*not at all*) to 4 (*all the time*). Total scores range from 0 to 52, with higher scores indicating more frequent catastrophic thoughts. In patients with chronic pain, a total PCS score of $\geq$30 represents a clinically high level of catastrophizing.[21] No established

cutoff scores exist for athletes because measuring catastrophizing in athletes is still relatively novel.[3] The PCS is easy to administer, has been widely used when treating patients with chronic pain, has excellent psychometric properties, and has been correlated with pain scores in previous studies.[8,21]

State and Trait Anxiety. The State-Trait Anxiety Inventory (STAI)[25] is a self-reported instrument designed to assess levels of state anxiety and trait anxiety via two 20-item questionnaires scored on a 4-point Likert scale. *State anxiety* can be defined as a transient momentary emotional state that results from situational stress. The state scale of the STAI asks people to describe how they feel at a particular moment in time (eg, calm or tense) using a 4-point intensity scale that ranges from 0 (*not at all*) to 3 (*very much so*). *Trait anxiety* represents a predisposition to react with anxiety in stressful situations. The trait scale of the STAI consists of 20 statements that describe how people generally feel (eg, confident) that are rated using a 4-point frequency scale, ranging from 0 (*almost never*) to 3 (*almost always*). The STAI scores for each scale range from 20 to 80, with a higher score indicating greater anxiety. A cut point of 39 to 40 has been suggested for detecting clinically important symptoms on the state anxiety scale.[26,27]

Cardiovascular Measures. During the initial screening period, we measured blood pressure using the Accutorr Plus V instrument (Mindray). During the rest of the procedure, we used the Nexfin system (BMEYE) to continuously measure heart rate, systolic blood pressure, diastolic blood pressure, and IBI during testing. The Nexfin system is a waveform analysis that uses a finger pressure cuff attached to the middle finger of the contralateral hand and is calibrated via a heart-level sensor. The Nexfin system records values in 30-second intervals. According to previous authors,[28,29] measuring heart rate over time is important because of the minute-by-minute variability of heart rate. The preferred method of measuring heart rate is over a longer duration of recording and using averages over time.[28,29]

Cold-Pressor Test. We used a CPT to induce pain, and participants were aware of this procedure before testing. For our CPT, we lined a plastic cooler with ice packs and filled it approximately two-thirds full of water. We added crushed ice to maintain the water temperature between 2°C and 4°C during the test and recorded temperature when we recorded pain to ensure that the water temperature remained in this range. Throughout the test, participants were seated. When instructed to do so, they submerged their hand in the water up to 1 cm above the wrist. We instructed them to keep the hand open in the water and not to touch any of the ice packs surrounding the cooler. The CPT lasted 3 minutes. We encouraged participants to keep their hand in the cold water for the entire test; however, they were informed that they could remove it if they were uncomfortable, in too much pain, or unwell. Researchers[9] have suggested that 90 to 120 seconds of stimulation should be sufficient to achieve a true peak response. Individuals who completed the 3-minute test remained seated during a 10-minute post-CPT period with their hand out of the water.

Experimental Protocol

During the initial screening period, we recorded participants' cardiovascular values and screened them for abnormalities, obtained demographic information, and asked them to complete the questionnaires. The initial screening was finished before the CPT procedures were conducted in a separate consultation room. We recorded demographic data (ie, age, height, mass, ethnicity, country in which they grew up) and whether they had used cryotherapy in the past. If they had used cryotherapy, we noted the frequency and body parts exposed to ice. We also instructed participants to list the sports they performed and the number of years played. They then filled out the PCS and STAI. Next, a research assistant measured the following values: sublingual temperature using a thermometer (H-B instrument 20592; Thomas Scientific) and heart rate (over 15 seconds) and blood pressure using the Accutorr Plus V. Blood pressure was measured after the participant had been seated during the initial screening period. The assistant obtained these measures to identify any heart abnormalities that would exclude the participant, including having a systolic blood pressure of >140 mm Hg or diastolic blood pressure >90 mm Hg. In addition, it gave us an indication of what their resting heart rate and blood pressure would be. The assistant also asked participants about the exclusion criteria. After the measures were completed in the consultation room, participants were brought to another room for the CPT procedures and collection of heart rate measures using the Nexfin system over the 15-minute baseline period as part of the total 30-minute procedure.

The 30-minute CPT procedure involved a 15-minute baseline, a 2-minute anticipatory period, a 3-minute CPT, and a 10-minute post-CPT period (Figure 1). After the 15-minute baseline, we brought the water bath into the room and allowed 2 minutes to elapse to minimize the effects of anticipation before starting the hand immersion. The 2 minutes before the test constituted our attempt to ensure that the baseline heart rate and blood pressure measures reflected the true baseline and were not prematurely elevated due to the impending CPT. However, we acknowledge that it is challenging to obtain true baseline heart rate and blood pressure measures before a painful test. At 17 minutes, participants immersed their right hand for 3 minutes. We started the CPT at minute 17 and ended it at minute 20. Participants then removed their hand from the water and waited during the 10-minute post-CPT period from minute 20 to minute 30. They reported their pain at multiple points during the procedure. We averaged the pain scores at baseline and during the post-CPT period and used those values in the analysis. The highest pain value reported during the CPT was used in the analysis. We monitored body temperature, water temperature, and pain level (range = 0–10) while the Nexfin system recorded heart rate, systolic blood pressure, diastolic blood pressure, and IBI in 30-second intervals throughout the 30 minutes. The heart rate, systolic blood pressure, diastolic blood pressure, and IBI values during the 15-minute baseline were averaged for analysis. This was repeated for the periods during CPT referred to as *CPT1* and *CPT2* in Figure 1. The *CPT1* refers to the average of cardiovascular variables recorded at minutes 17, 17.5, and 18; *CPT2* refers to the average of cardiovascular variables recorded at minutes 18.5, 19, and 19.5. We averaged the cardiovascular values for the post-CPT period. The timeline of the CPT and measurements is represented in Figure 1.

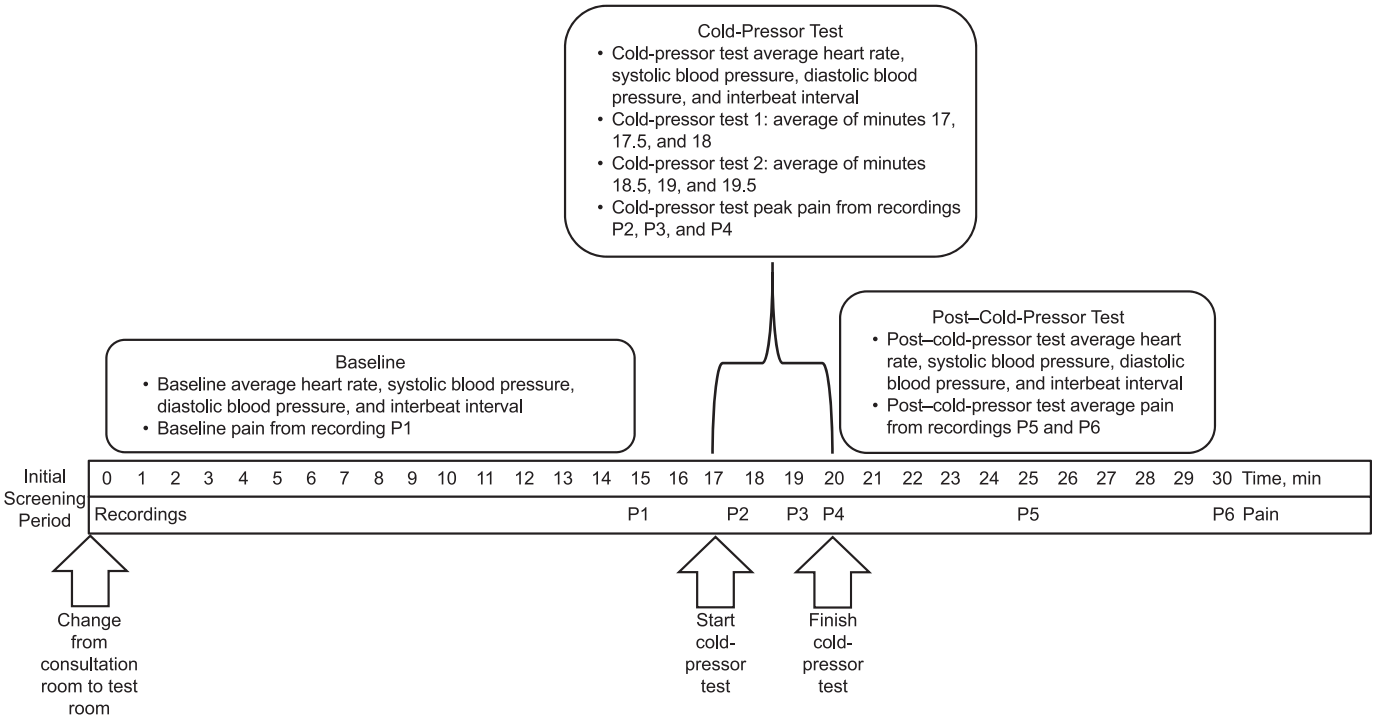

Figure 1. Timeline and measurements during the initial screening period and the cold-pressor test (CPT) procedure. During the initial screening before the CPT procedure, we obtained written consent, recorded cardiovascular values to screen for abnormalities, acquired participant demographic information, and instructed participants to complete the questionnaires. The CPT procedure involved a 15-minute baseline period, 2 minutes of anticipation before the test, a 3-minute CPT, and 10 minutes post-CPT. The baseline timeframe was from minutes 0 to 15, the CPT timeframe was from minutes 17 to 20, and the post-CPT timeframe was from minutes 20 to 30. Cardiovascular measures of heart rate, systolic blood pressure, diastolic blood pressure, and interbeat interval were recorded every 30 seconds throughout the 30 minutes. Participants rated their pain at P1 (minute 15), P2 (minute 17.5), P3 (minute 19), P4 (minute 20), P5 (minute 25), and P6 (minute 30). Baseline pain corresponds to P1. Cardiovascular values at baseline correspond to the average of all measurents taken from minute 0 to P1. Peak pain among P2, P3, and P4 was determined as pain during the CPT. Cardiovascular values during the CPT were averaged from recordings at minutes 17, 17.5, and 18 (CPT1) and at minutes 18.5, 19, and 19.5 (CPT2). We averaged pain measured at P5 and P6 to obtain the pain post-CPT. The cardiovascular value at post-CPT corresponds to the average of all measurements taken from minutes 20 to 30.

Calculation of Heart Rate Variability: SDIBI

Heart rate variability has been used to measure cardiovascular responses to stress.[16] The oscillations of a healthy heart are complex and constantly changing, allowing the cardiovascular system to rapidly adjust to sudden physical and psychological challenges.[28,29] In addition, heart rate variability reflects the regulation of autonomic balance, blood pressure, gas exchange, and the heart. We measured heart rate variability by calculating the SDIBI.[17,18] We collected IBI values at the same time as heart rate and systolic and diastolic blood pressure, every 30 seconds. Next, we averaged the SDs for the 15-minute baseline period to calculate the SDIBI from minutes 0 to 15. For the SDIBI during CPT, we used the SD of the recording from minutes 17 to 19.5. For SDIBI at post-CPT, we calculated the SDIBI from minutes 20 to 30 (Figure 1).

Statistical Analysis

The cardiovascular values recorded at 30-second intervals were averaged for each participant for 3 times: baseline (minutes 0–15), during CPT (minutes 17–20), and post-CPT (minutes 20–30). We did not include measures from the 2-minute anticipatory period (minutes 15–17) because we thought that directly before the start of the CPT would be the most stressful time for the participants. Therefore, we did not want to include the heart rate, blood pressure, and SDIBI measures during this time as part of the baseline measures. We calculated the average values for heart rate, systolic blood pressure, diastolic blood pressure, and SDIBI at baseline, during the CPT, and post-CPT. Separate 1-way analyses of variance were conducted to identify any changes to heart rate, systolic blood pressure, diastolic blood pressure, pain, and SDIBI at baseline, during CPT, and post-CPT. When the analyses of variance indicated a difference, we used the Tukey test to identify the difference among means. We also performed Pearson product moment correlations (at baseline, during CPT, and post-CPT) to screen for relationships between scores on the PCS and STAI and the cardiovascular measures (heart rate, systolic blood pressure, diastolic blood pressure, and SDIBI). Lastly, we calculated a linear regression by using the change in heart rate as the dependent variable with any identified Pearson correlations. Moreover, we used any correlations in a linear regression to identify the contribution of each variable. We set the α level at .05, and Cohen d effect sizes were reported for any findings that were different. Any *P* values between .05 and .10 were considered a trend. We conducted all analyses using SPSS (version 24; IBM Corp).

Table 1. Baseline Measures of All Participants (N = 36)

Measure	Mean ± SD
Age, y	24.0 ± 4.6
Height, cm	180.0 ± 6.1
Mass, kg	90.5 ± 13.8
Body mass index	27.9 ± 4.2
Rugby experience, y	7.7 ± 4.7
Pain Catastrophizing Scale score	12.4 ± 6.2
State-Trait Anxiety Inventory	
State	46.9 ± 3.2
Trait	46.7 ± 3.1
Baseline	
Heart rate, beats/min	62.6 ± 9.7
Systolic blood pressure, mm Hg	129.3 ± 11.5
Diastolic blood pressure, mm Hg	70.8 ± 8.6

RESULTS

A total of 37 male rugby athletes (age = 24.0 ± 4.6 years, height = 180.0 ± 6.1 cm, mass = 90.5 ± 13.8 kg) participated in the study. Of these individuals, 1 was unable to complete the 3-minute CPT and withdrew his hand, stating that his subjective pain rating was a 10 out of 10. Therefore, data from 36 participants were analyzed. The initial screening measures of demographic, PCS, STAI, and cardiovascular values are described in Table 1.

Pain Outcomes

Participants experienced an increase in pain from baseline during the CPT (0.0 to 4.1 ± 2.2; $P < .001$; d = 2.636), followed by a decrease in pain post-CPT (0.3 ± 0.7; $P < .001$; d = –2.328). Figure 2 represents the mean changes in pain levels over time.

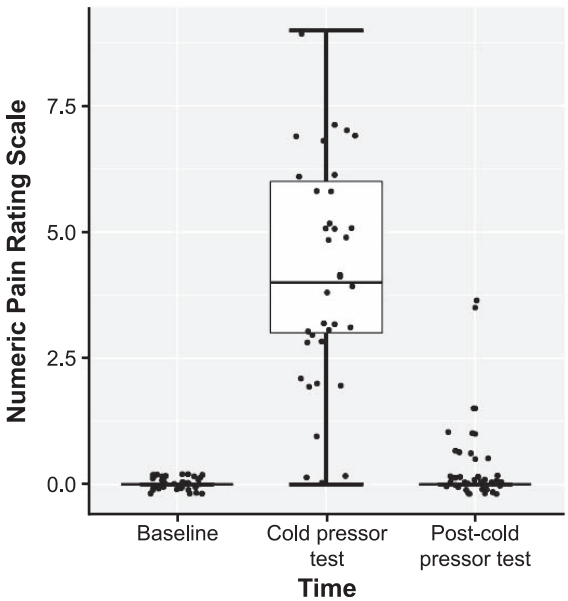

Figure 2. Pain at baseline, during the cold-pressor test (CPT), and post-CPT in rugby athletes. Pain increased from baseline to during CPT ($P < .001$; d = 2.636) and then decreased post-CPT ($P < .001$; d = –2.328). The dots indicate the individual data points, the boxes indicate the 25th to 75th interquartile range, the line within the boxes indicates the median, and the whiskers indicate the smallest and largest values within 1.5 times the interquartile range below and above the 25th and 75th percentile, respectively. [a] $P < .001$.

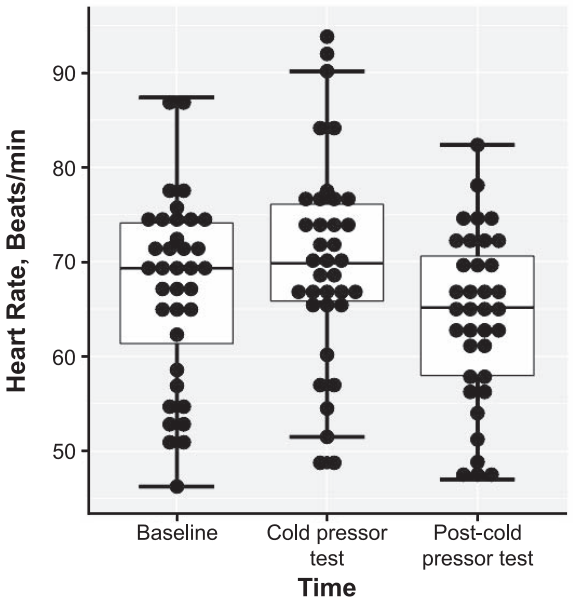

Figure 3. Heart rate at baseline, during the cold-pressor test (CPT) and post-CPT. Median heart rate decreased post-CPT ($P = .040$; d = –0.597). The dots indicate the individual data points, the boxes indicate the 25th to 75th interquartile range, the line within the boxes indicates the median, and the whiskers indicate the smallest and largest values within 1.5 times the interquartile range below and above the 25th and 75th percentile, respectively. [a] $P < .05$.

Cardiovascular Outcomes

During the CPT, heart rate increased from 67.2 ± 9.8 beats/min at baseline to 70.1 ± 11.0 beat/min during the CPT, but the increase was not different ($P > .001$). However, heart rate decreased to 64.1 ± 9.0 beats/min post-CPT ($P = .040$; d = –0.597; Figure 3).

We observed an increase in systolic blood pressure from 126.7 ± 16.5 mm Hg at baseline to 149.7 ± 23.4 mm Hg during the CPT ($P < .001$; d = 1.136) and a decrease in systolic blood pressure to 137.1 ± 18.8 mm Hg post-CPT ($P < .001$; d = –0.594). Systolic blood pressure changes over time are shown in Figure 4. A similar response was noted for diastolic blood pressure, with an increase from 76.9 ± 8.3 mm Hg at baseline to 91.9 ± 11.5 mm Hg during the CPT ($P < .001$; d = 1.496), followed by a decrease to 82.1 ± 9.3 mm Hg post-CPT ($P < .001$; d = –0.947).

Heart rate variability measured using the SDIBI increased from 0.0164 ± 0.0121 milliseconds at baseline to 0.0400 ± 0.0323 milliseconds during the CPT ($P < .001$; d = 0.968) before decreasing to 0.0175 ± 0.0122 milliseconds post-CPT ($P < .001$; d = –0.922). Mean changes in IBI over time are illustrated in Figure 5.

Relationships Between Psychological Variables and Cardiovascular Measures

Correlations were identified between the PCS score and peak pain ($r = 0.397$, $P = .02$). In addition, we observed a relationship between heart rate change and peak pain ($r = 0.465$, $P = .004$). Table 2 shows correlations between outcomes, Figure 6 displays the relationship between PCS scores and peak pain, and Figure 7 describes the relationship between PCS scores and heart rate.

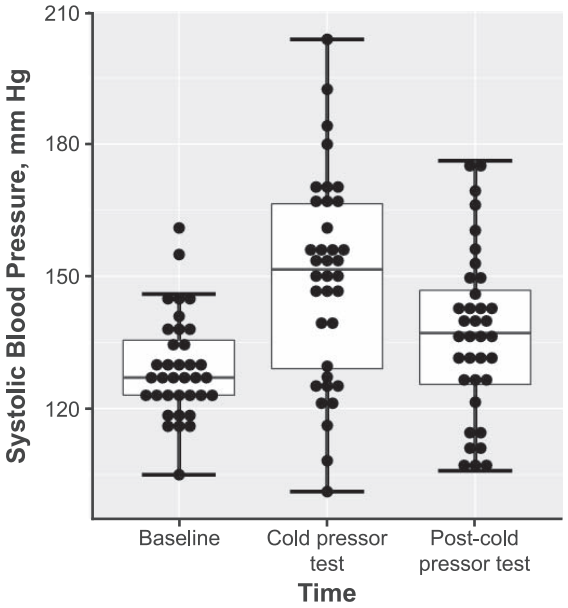

Figure 4. Systolic blood pressure at baseline, during the cold pressor test (CPT), and post-CPT. Median systolic blood pressure increased from baseline to during CPT ($P < .001$; d = 1.136) and then decreased post-CPT ($P < .001$; d = –0.594). The dots indicate the individual data points, the boxes indicate the 25th to 75th interquartile range, the line within the boxes indicates the median, and the whiskers indicate the smallest and largest values within 1.5 times the interquartile range below and above the 25th and 75th percentile, respectively. [a] $P < .001$.

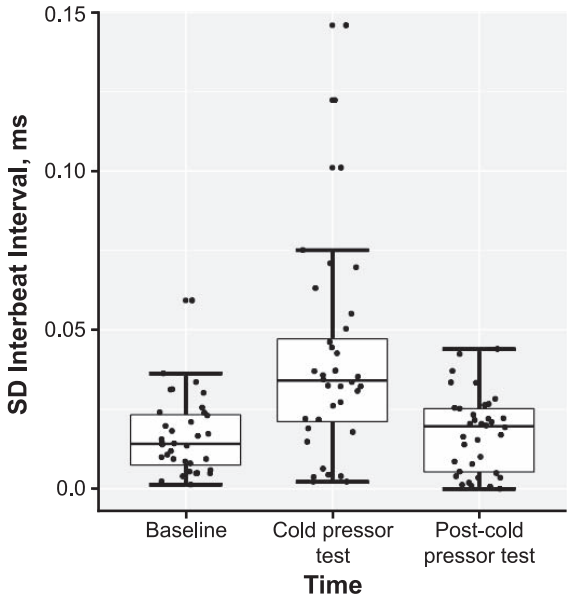

Figure 5. The SD of the interbeat interval at baseline, during the cold-pressor test (CPT), and post-CPT in rugby athletes. Median SD of the interbeat interval increased from baseline to during CPT ($P < .001$; d = 0.968) and then decreased post-CPT ($P < .001$; d = –0.922). The dots indicate the individual data points, the boxes indicate the 25th to 75th interquartile range, the line within the boxes indicates the median, and the whiskers indicate the smallest and largest values within 1.5 times the interquartile range below and above the 25th and 75th percentile, respectively. [a] $P < .001$.

Regression Analysis

We originally ran a regression analysis with heart rate change as a dependent variable compared with pain, PCS score, and SDIBI while controlling for age because peak pain, catastrophizing (PCS score), and SDIBI were all correlated with heart rate change. However, after further analysis, it was evident that the SDIBI was also related to heart rate change because IBI is generated by the time difference between beats (a violation of collinearity). Therefore, the final model consisted of heart rate change as the dependent variable versus peak pain and catastrophizing (PCS score) while controlling for age. Linear regression indicated that peak pain, catastrophizing, and age contributed to 29.2% of the variance in heart rate change during the CPT ($r = 0.540$, $r^2 = 0.292$, $P = .01$). Although the overall model was different, the coefficient for pain catastrophizing trended toward being a contributor to the model ($P = .07$). Age was not a predictor for change in heart rate ($P = .90$). Individual coefficients for the predictors of heart rate change are shown in Table 3. The correlation between PCS score and the change in heart rate during the CPT ($r = 0.437$, $P = .008$) appears in Figure 7. A high level of pain catastrophizing was associated with a

Table 2. Correlations Among Pain, State-Trait Anxiety Inventory State, State-Trait Anxiety Inventory Trait, Catastrophizing, and Cardiovascular Measurements During a Cold-Pressor Test (CPT) in Rugby Athletes

	Δ						State-Trait Anxiety Inventory Score		
Variable	Heart Rate	Systolic Blood Pressure	Diastolic Blood Pressure	Average Pain Post–CPT	Peak Pain	Pain Catastrophizing Scale Score	State	Trait	SD Interbeat Interval Change
Δ									
Heart rate	1	0.161	0.186	0.291	0.465[a]	0.437[a]	–0.011	–0.002	0.444[a]
Systolic blood pressure		1	0.887[b]	0.202	0.286	0.138	–0.170	0.177	0.000
Diastolic blood pressure			1	0.208	0.309	0.088	–0.069	0.195	–0.050
Average pain post–CPT				1	0.180	0.491[a]	0.214	0.063	0.153
Peak pain					1	0.397[a]	0.132	0.103	0.205
Pain Catastrophizing Scale score						1	–0.196	–0.279	–0.012
State-Trait Anxiety Inventory score									
State							1	0.172	0.031
Trait								1	0.219
SD interbeat interval change									1

[a] $P < .05$.
[b] $P < .001$.

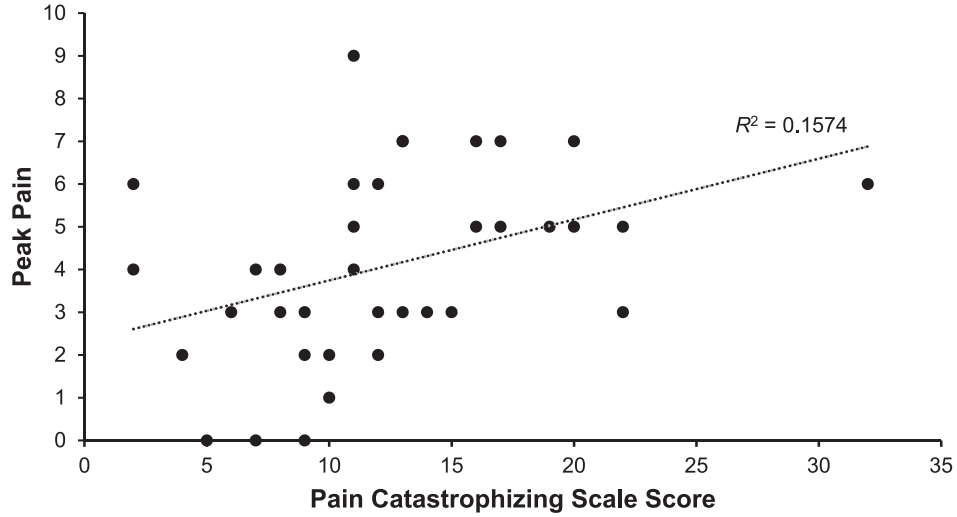

Figure 6. Correlation of Pain Catastrophizing Scale scores with peak pain experienced during the cold-pressor test ($r = 0.397$, $P = .02$).

larger increase in heart rate during the CPT. We observed no relationship between changes in blood pressure (systolic or diastolic) and PCS scores.

DISCUSSION

The purpose of our study was to assess changes in the cardiovascular responses of male rugby athletes completing a CPT and the relationship of the cardiovascular variables to pain catastrophizing and subjective pain ratings. The CPT used to induce pain caused changes in heart rate, systolic blood pressure, diastolic blood pressure, and heart rate variability (the last as measured using the IBI, which is related to pain catastrophizing in athletes).

To explain the changes in cardiovascular variables during a noxious stimulus, several groups studying healthy young adults have suggested that catastrophizing predicts increased systolic blood pressure reactivity to pain[8,30,31] and enhances myocardial contractility for a prolonged period after a CPT.[31] Catastrophizing appears to influence the relationship among muscle tension, cardiovascular stress, and pain response,[30,31] as well as temporal summation of pain, a frequently studied index of central pain facilitation.[5,32] This could explain the lack of correlation between blood pressure and catastrophizing noted in our study. Changes in blood pressure are stimulated by both pain and cold; however, the change in heart rate is considered to be stimulated solely by pain. These assumptions may explain our results: a relationship among heart rate change, pain, and PCS scores but not with other cardiovascular variables, although cardiovascular variables do respond to nociceptive stimuli. Moreover, in explaining the mechanism linking pain and cardiovascular responses, researchers[21,33] have reported a correlation between reduced gray matter in certain regions of the brain and the duration or intensity of pain. Seminowicz et al[33] conducted an 11-week cognitive behavioral therapy intervention for individuals coping with chronic pain to increase grey matter volume. They found increased gray matter volume in the prefrontal and somatosensory brain regions, as well as increased dorsolateral prefrontal volume associated with reduced pain catastrophizing. Increased grey matter using cognitive behavioral therapy reflects greater control over pain, cognitive reappraisal of pain, and reduced catastrophizing.

In a few studies, investigators measured experimentally induced pain in athletes. Geva and Defrin[5] compared pain threshold, tolerance, intensity, and catastrophizing between triathletes and participants in amateur sports (nonathletes)

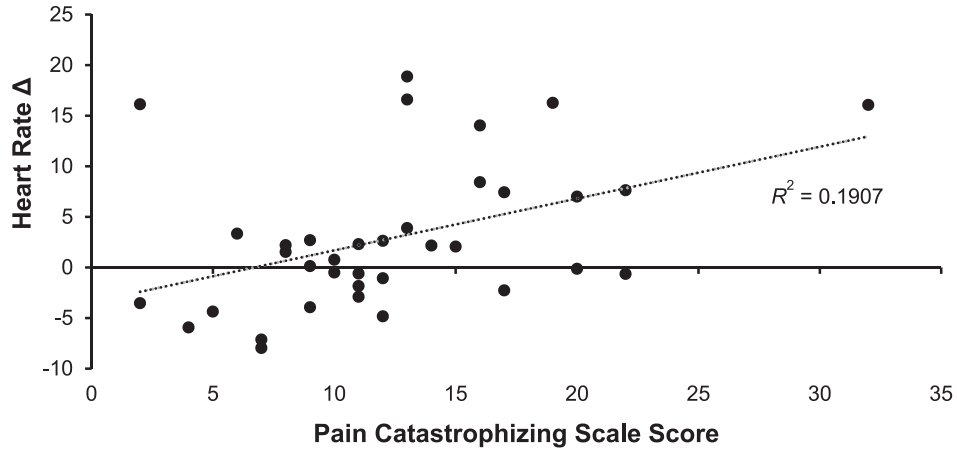

Figure 7. Correlation of Pain Catastrophizing Scale scores with change in heart rate during the cold-pressor test ($r = 0.437$, $P = .008$).

Table 3. Coefficients of Linear Regression for Heart Rate Change During the Cold-Pressor Test

Variable	R^2	B	SE B	β	*P* Value
Heart rate Δ	0.29				.01[a]
Age		–0.029	0.237	–0.018	.90
Peak pain		1.128	0.529	0.345	.041[a]
Catastrophizing		0.354	0.192	0.302	.07

[a] $P < .05$.

during hand immersion in a 12°C water bath for 60 seconds. At baseline, catastrophizing in triathletes was less than in nonathletes; however, this finding was not different (16.5 ± 9 versus 20.8 ± 12; $P = .053$). The pain ratings on a visual analog scale during a CPT in triathletes were lower than those of nonathletes at 1 second (0.8 ± 1.5 versus 4.6 ± 3.8; $P < .001$), 20 seconds (2.3 ± 2.5 versus 6.4 ± 3.5; $P < .001$), and 60 seconds (5.5 ± 2.8 versus 8.3 ± 2.6; $P = .01$). Both groups experienced similar overall increases in pain intensity (3.7 ± 3.0 [triathletes] and 4.7 ± 3.5 [nonathletes]). They also appeared similarly able to differentiate between painful and nonpainful events, meaning they had the same pain threshold; yet the difference in pain may be explained by the difference in pain-catastrophizing levels in triathletes compared with nonathletes. This difference, however, was not further explored. Knowing that *pain tolerance* is the ability or willingness to endure pain, the triathletes appeared to have a greater ability or motivation to endure pain from the experimental setup. Catastrophizing in triathletes and nonathletes was not different, but the fear of pain in triathletes was less than in nonathletes (71.7 ± 14.9 versus 81.0 ± 17.1, respectively; $P = .05$). A higher pain tolerance may stem from a lack of fear of the stimulus or its consequence. In the previous study,[5] homogeneity of the group prevented the correlation analysis from supporting the link between fear of pain and pain tolerance. Of note, the overall increase in pain values in our work (4.1 ± 2.2) was similar to the earlier study (triathletes = 3.7 ± 3.0, nonathletes = 4.7 ± 3.5), although our water was colder and our CPT lasted 3 minutes. In addition, the catastrophizing average of our participants was also lower (12.4 ± 6.2) than that of the triathletes (16.5 ± 9) and nonathletes (20.8 ± 12; $P = .053$) in the earlier study, which may also explain the different pain results in our study with a colder and longer CPT.

Manning and Fillingim[6] performed a CPT on intercollegiate athletes in a 1°C water bath. Participants indicated when sensations in the immersed hand first became painful (pain threshold) and then intolerable (pain tolerance) and reported pain intensity and unpleasantness at both times. Athletes demonstrated a higher pain threshold for cold pain than nonathletes (athletic women = 3.07 ± 2.65 versus nonathletic women = 4.10 ± 1.98; athletic men = 2.03 ± 1.93 versus nonathletic men = 3.08 ± 2.04). Athletes demonstrated a higher tolerance to cold pain than nonathletes (athletic women = 6.00 ± 3.21 versus nonathletic women = 6.62 ± 1.36; athletic men = 4.89 ± 3.37 versus nonathletic men = 6.37 ± 2.00). Moreover, men exhibited higher pain thresholds and tolerance for cold pain than women ($P = .05$). Among all groups, nonathletic women demonstrated a higher pain threshold and tolerance ($P = .05$). Similarly, we measured *peak pain*, which can also be referred to as pain tolerance. We evaluated male rugby athletes, and their peak pain during the CPT was 4.1 ± 2.2. Furthermore, Manning and Fillingim[6] measured *pain self-efficacy*, which represents the ability to directly control the pain experience. Pain self-efficacy in athletic activity (positive) was correlated with pain tolerance intensity ($r^2 = 0.309$; $P = .05$).[6] Pain self-efficacy and pain catastrophizing are 2 psychosocial factors contributing to the perception of pain and the emotional and physical effects and responses to pain.[34] Therefore, the correlation between pain self-efficacy and pain tolerance was similar to our finding of a correlation between PCS score and peak pain ($r = 0.397$; $P = .02$).

In our study, heart rate decreased from during the CPT to post-CPT and trended toward increasing during the CPT. Consistent with the results of Etherton et al,[8] who examined a nonathletic population, we found that the pain induced by the CPT caused an elevation in cardiovascular measures, followed by a decline after the nociceptive stimulus was removed. Of note, our methods were similar to those of Etherton et al,[8] with a similar CPT timeframe and water temperature. Unlike us, they identified a main effect of sex: female participants had a higher heart rate over the course of the experiment relative to male participants ($F_{1,35} = 9.12$, $P = .01$).[8] A main effect of time also reflected the increased heart rate during the CPT test for both male and female participants and then a decrease after the CPT ($F_{2,70} = 27.96$, $P < .001$). However, they found no interaction between time and sex, meaning that heart rate responses for male and female participants were consistent over time. In addition, they observed no differences in pain catastrophizing between male and female participants who completed the CPT (21.8 versus 22.0 on the PCS). The PCS scores did not differ between participants who completed and those who did not complete the CPT. Mean subjective pain ratings during the CPT were correlated with PCS scores ($r = 0.403$, $P = .01$), and PCS scores were correlated with heart rate and blood pressure indices at baseline, during the CPT, and post-CPT. Pain catastrophizing has been correlated with subjective pain ratings during a painful test.[8] Our findings demonstrated a correlation between the change in heart rate and peak pain and a trend between the change in heart rate and PCS score. Nevertheless, Etherton et al[8] noted no correlation between pain catastrophizing and cardiovascular reactivity. Heart rate and blood pressure may indicate a response to pain without being an index of pain severity. Etherton et al[8] studied 17 male and 23 female participants and characterized sex differences in cardiovascular responses during the CPT. Yet their small sample sizes were a limitation in determining possible correlations between cardiovascular and pain variables. In contrast, Peckerman et al[9] suggested that cardiovascular changes were related to both painful and nonpainful stimuli. Their results suggested that changes in blood pressure were caused by nonpainful stimulations that led to vasoconstriction, consequently increasing blood pressure. They also proposed that pain induction could activate both vasomotor and cardiac mechanisms for blood pressure, thereby increasing heart rate.

During the initial screening period, we noted what we initially thought was an elevated mean systolic blood pressure of 129.3 ± 11.5 mm Hg. However, in a systematic review examining blood pressure in athletes, Berge et al[35]

observed that strength-trained athletes had slightly higher blood pressure than endurance-trained athletes. This larger review consisted of 138 390 men and women; most were aged 18 to 40 years, and they represented various sport disciplines. Mean systolic blood pressure varied from 109 ± 11 mm Hg to 138 ± 7 mm Hg, and mean diastolic blood pressure varied from 57 ± 12 mm Hg to 92 ± 10 mm Hg. Strength-trained athletes had higher blood pressure than endurance-trained athletes ([systolic/diastolic] 131.3 ± 5.3 mm Hg/77.3 ± 1.4 mm Hg versus 118.6 ± 2.8 mm Hg/71.8 ± 1.2 mm Hg; $P < .05$) and a trend toward higher blood pressure was evident in athletes training ≥10 h/wk compared with others (121.8 ± 3.8 mm Hg/73.8 ± 2.5 mm Hg versus 117.6 ± 3.3 mm Hg/66.8 ± 6.9 mm Hg, respectively; $P = .058$). Still, overall, they reported no difference in blood pressure between athletes and control individuals.[35] Therefore, it seems that the resting systolic blood pressure in our athletes was similar to that in previous studies.

Previous experience with cryotherapy or cold exposure could affect the results of a CPT. We asked all participants about their experience with ice application and full-body cold immersion, and not surprisingly, all our athletes described using ice regularly for minor injuries. We located no studies indicating that previous experience of athletes using local cryotherapy could influence CPT results. We did collect information about the previous use of cold tubs or full-body cold immersion. Eight athletes stated they had used no more than 1 modality in the year before the study, usually during tournaments. Researchers[36,37] have suggested that whole-body exposure to cold can influence the CPT response. However, given the limited use of cold tubs by our athletes, we do not believe it affected our results. More studies are needed to see if previous local cryotherapy applications can affect the results of a CPT.

Psychological factors, such as pain catastrophizing and anxiety, may explain athletes' higher pain tolerance and the varied responses to a painful stimulus. Yet our results indicated that catastrophizing was correlated with pain whereas anxiety was not. We wanted to compare our athletes' PCS and STAI scores with those in other investigations. Our participants' anxiety scores, 46.9 ± 3.2 for the STAI state score and 46.7 ± 3.1 for the trait score, were slightly higher than normal,[26,27] but this elevated level of anxiety was not correlated with the amount of pain experienced during the CPT. Anxiety is often suggested as a reason for athletes experiencing increased pain, but our work did not support this notion. With regard to pain catastrophizing, total scores range from 0 to 52, with higher scores indicating a more frequent occurrence of catastrophic thoughts.[21] Our athletes had an average score of 12.4 ± 6.2 on the PCS, which was lower than the scores reported in other studies: 22.0 for female participants versus 21.8 for male participants[8] and 16.5 ± 9 for triathletes versus 20.8 ± 12 for nonathletes.[5] Authors[21] at the University Centre for Research on Pain and Disability demonstrated that a total PCS score of 30 represented a clinically high level of catastrophizing, which was higher than our participants' score. However, we caution against the use of cutoff scores for these scales. The PCS was not designed to be sufficiently sensitive to separate a score of 31 from a score of 29, for example. Use of the proposed cutoff would indicate that a change in 1 response on the scale could result in a person with a score of 31 being considered a high catastrophizer and a person with a score of 29 being considered a low catastrophizer. The scale's lack of sensitivity to a change in 1 or 2 numbers is also part of the reason we did not split the participants into low- and high-catastrophizing groups for further analysis.

Limitations

Interestingly, with a controlled stimulus, although most subjective peak pain ratings hovered near the average, 2 participants claimed to perceive no pain, and 1 participant could not complete the test because of a 10 out of 10 pain rating. Our results are consistent with earlier findings that showed variability in pain. As mentioned, the CPT is an effective inducer of pain. Nonetheless, a limitation and possible reason for the failure to cause pain in some participants might have been that the male athletes were reluctant to report their true pain levels, which could have influenced the average. We also acknowledge the potential benefit of future studies that include a path analysis to determine the mediation effect of catastrophizing on induced pain. We were not able to conduct a path analysis with these data because we violated an assumption due to the complex relationship between pain and pain catastrophizing. Future researchers should examine this bidirectional relationship between pain and pain catastrophizing. Other limitations may include the amount of sleep the participants had the night before the testing day, which was not controlled. As suggested by Finan et al,[7] poor sleep may lead to a stronger pain reaction. The time of day of the testing, which ranged from 8 AM to 6 PM may have also affected the results. Lastly, we tested only male athletes because sex may influence cardiovascular responses to pain; future investigators should examine the effect of catastrophizing on female athletes' cardiovascular response to pain.

Clinical Implications

The clinical implications of our study are that during a painful stimulus, athletes with a high level of catastrophizing perceived more pain and had a stronger cardiovascular response to pain than those with a low level of catastrophizing. This has several clinical implications regarding the assessment and rehabilitation of athletes. Pain is quite variable between athletes, even those with similar injuries. Deroche et al[2] suggested that pain catastrophizing can explain important differences in sport-related pain behavior more than pain intensity itself. Although we experimentally induced pain, it seems that catastrophizing may explain some of the clinical variability in pain among injured athletes. Anxiety was not correlated with any other variables, including pain, which was contrary to other findings that anxiety was a predictor for systolic blood pressure reactivity to painful stimuli[15] and that anxiety and pain catastrophizing may be relevant in developing exaggerated pain perception.[22] If an athlete experiences an elevated pain response over time, it could be problematic. Previous investigators[3,6,7,21,22,32,38] indicated that an elevated pain response can lead to the development of chronic pain. According to the fear-avoidance model, in the general population, those who have a higher level of pain catastrophizing are more likely to develop chronic

pain and increased disability.[38] An increase in chronic pain could prolong the rehabilitation of some athletes. Future researchers should study potential individual pain-management strategies for athletes based on their psychological and possibly cardiovascular responses to pain, which may help them return to play more quickly. In addition, authors should identify a more objective or physiological measure of pain that can reduce the subjective nature of pain measurement, which would make pain assessments more accurate.

CONCLUSIONS

Our results indicated a relationship in athletes between catastrophizing and pain experienced during a CPT. In addition, the amount of pain experienced by the athletes was correlated with the amount of heart rate change during the CPT. Thus, athletes with a higher level of catastrophizing perceived more pain and experienced a greater cardiovascular response than other athletes during the same painful stimulus. Peak pain, catastrophizing, and age contributed 29.2% of the variance in heart rate change during the CPT. Although cardiovascular measures may be an objective measure of pain in the future, our data demonstrated that psychological constructs, such as catastrophizing, will need to be considered for any potential cardiovascular measures of pain. Further exploration may characterize heart rate change as an interesting option for obtaining a more objective measure for pain. The relationship between pain and cardiovascular changes helps to provide a link between the sympathetic nervous system and psychological pain perception and may explain an increased response to pain. Lastly, future studies are needed, but athletic trainers and athletic therapists may wish to administer the PCS as a screening tool for interpreting the clinical pain presentation during an injury, as well as revealing a factor that could prolong rehabilitation.

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