Pain catastrophizing in athletes correlates with pain and cardiovascular changes during a painful cold pressor test

Lentini Matylda¹, Scalia Joseph¹, Berger Lebel Frédérique BSc, MSc candidate, CAT(C)¹, Touma Fadi BSc¹, Jhajj Aneet¹, Peter J. Darlington PhD¹, Geoffrey Dover PhD, CAT(C), ATC¹,²

¹ Concordia University, PERFORM Centre, Department of Health, Kinesiology, and Applied Physiology, Loyola Campus SP165.41 - 7141 Sherbrooke Street W. Montreal, Quebec, Canada H4B 1R6
² Centre de recherche interdisciplinaire en réadaptation du Montréal Métropolitain, Constance Lethbridge centre, CIUSSS du Centre-Ouest-de-l’Île-de-Montréal, 7005 boul. de Maisonneuve Ouest, Montréal QC H4B 1T3

M_lentin@live.concordia.ca
Joseph.scalia@mail.concordia.ca
Frederike.bergerlebel@mail.concordia.ca
Fadi.touma@mail.concordia.ca
Aneet.jhajj@mail.concordia.ca
Peter.darlington@concordia.ca
Geoffrey.dover@concordia.ca

Corresponding author
Geoffrey Dover
Tel: 514-848-2424 #3304
Fax: (514) 848-8681
Richard J. Renaud Science Complex, Loyola Campus
7141 Sherbrooke Street West
Montréal, Québec, Canada
H4B 1R6
Office: SP 165.19
geoffrey.dover@concordia.ca

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Pain catastrophizing in athletes correlates with pain and cardiovascular changes during a painful cold pressor test

**Context:** Athletes are often exposed to pain due to injury and competition. There is preliminary evidence that cardiovascular measures could be an objective measure of pain, but the cardiovascular response can be influenced by psychological factors such as catastrophizing. **Objectives:** The purpose of our study was to use a painful cold pressor test to measure the relationship between catastrophizing, pain, and cardiovascular variables in athletes. **Design:** Pre-post test. **Setting:** We completed all measures in a laboratory setting. **Participants:** Thirty-six male rugby athletes participated in the study. **Main outcome measures:** We measured catastrophizing with the Pain Catastrophizing Scale and pain with a Numeric Pain Rating Scale. Cardiovascular measures included heart rate, systolic, and diastolic blood pressure, and heart rate variability. **Results:** During the cold pressor test, participants experienced a significant increase in pain (0 to 4.1±2.2), systolic blood pressure (126.7±16.5mm Hg to 149.7±23.4mm Hg), diastolic blood pressure (76.9±8.3mm Hg to 91.9±11.5mm Hg) and heart rate variability (from 0.0164ms±0.0121 to 0.0400ms±0.0323) (all p<.001). In addition, there was a significant decrease in heart rate after the cold pressor test (p=0.04). There was a significant correlation between athlete’s pain catastrophizing to both pain intensity and change in heart rate during the cold pressor test (p=.017 and p=.003 respectively). A significant linear regression indicated pain and catastrophizing explained 29% of the variance of the change in heart rate (p=.003). **Conclusion:** Athletes who have catastrophizing thoughts are 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 alternative for an objective measure of pain in athletes in the future.

**Key Words:** Pain related fear, heart rate, blood pressure, sport
Introduction

Athletes have a complex relationship with pain\textsuperscript{1,2} since they are often exposed to potentially painful stimuli.\textsuperscript{3} Sports participation and training can strain the body, potentially exposing athletes to pain.\textsuperscript{1-4} In many contact sports such as rugby, football or boxing, the ability to withstand pain appears to be essential to success and performance. Athletes have been reported to have an increased capacity to endure pain compared to both sedentary populations and regularly active populations.\textsuperscript{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.\textsuperscript{4} Most athletes accept pain to be “part of the game”, making them inclined to play through the pain by using coping mechanisms.\textsuperscript{2,4} There is some evidence reported by Finan, Goodin & Smith suggesting that pain is not necessarily proportional to the extent of an athlete’s injury.\textsuperscript{7}

Recently there has been more studies evaluating cardiovascular measures during experimentally induced pain however these studies have rarely examined athletes. Instead of using a verbal pain rating, some studies suggest that measuring cardiovascular changes can be an objective measure of pain during a cold pressor test for example.\textsuperscript{8,9} The Cold Pressor Test (CPT) is a non-invasive, pain stimulating test used frequently to study pain.\textsuperscript{10,11} Exposure of a body part to cold induces a nociceptive stimulus and stress response, causing a change in cardiovascular activity through the activation of the sympathetic nervous system.\textsuperscript{10} However, during the cold pressor test, the pain response and resulting cardiovascular change is variable in the general population. When measuring heart rate, most studies report an increase\textsuperscript{8,9,12} while one reported a decrease\textsuperscript{13} and one study reported no change in heart rate.\textsuperscript{14} There is evidence of a correlation between subjective pain rating and change in heart rate.\textsuperscript{9} In regards to blood pressure, previous studies have demonstrated an increase blood pressure response during the cold pressor test followed by a decrease in blood pressure response after the cold pressor test.\textsuperscript{8,12} The variability in responses in previous studies indicates that something else is influencing the cardiovascular changes and reported pain levels.\textsuperscript{8,14} Moreover, during a cold pressor test on non-athlete individuals, anxiety was an individual predictor for
systolic blood pressure reactivity.\textsuperscript{15} If athletes experience pain differently that in the general population it is unknown if the cardiovascular response to experimentally induced pain would be similar.

Another interesting cardiovascular measure that could be used is heart rate variability. Heart rate variability is defined as the change in the time intervals between adjacent heartbeats and is part of regulatory systems in order to adapt to environmental and psychological challenges.\textsuperscript{16} A common method for calculating heart rate variability is by calculating the standard deviation of the inter-beat interval (SDIBI).\textsuperscript{17,18} While this is a new measure in the pain and athlete field, many studies use the resting HRV or SDIBI in order to measure the reaction for various conditions including stress, depression, anxiety, and PTSD. For example, a previous study determined that heart rate variability measured through SDIBI was significantly lower in depressed individuals than in healthy controls in a large adult sample.\textsuperscript{19} Another study analyzed heart rate variability in the context of Panic Disorder and Generalized Anxiety Disorder.\textsuperscript{20} So while this measure of heart rate variability is used in other stress related studies, it is a novel measure for the cardiovascular response of an athlete to pain which is why we wanted to use it in our experiment.

Psychological factors including pain catastrophizing may explain athletes’ higher pain tolerance and the varied pain response to a painful stimulus. In a study by Geva & Defrin, compared with non-athletes, athletes display reduced pain catastrophizing.\textsuperscript{3,5} Pain catastrophizing is a negative and exaggerated psychological response to pain.\textsuperscript{3,21} Rumination, pain magnification, and helplessness are components of catastrophizing and hinder the ability to distract oneself from pain-related thoughts while experiencing pain.\textsuperscript{21} Higher scores on the Pain Catastrophizing Scale are associated with a higher pain intensity following injury or surgery, which are common in an athletic population.\textsuperscript{2,3,15,21,22} It has been proposed that anxiety and pain catastrophizing are relevant psychological qualities and leaders to development of overvalued pain perception.\textsuperscript{22} Individuals who engaged in catastrophic thinking reported the highest levels of pain during pain inducing experiments such as the cold pressor test\textsuperscript{11,23}, but this has not been measured in athletes.

Psychological factors during a cold pressor test can affect how one experiences pain but it is unclear if this relationship exists in athletes. If catastrophizing can influence the amount of pain felt by an
athlete it would have a significant impact during the clinical assessment and rehabilitation of an athlete.

So far, no study has investigated the relationship between pain catastrophizing and variables such as pain and cardiovascular changes during a cold pressor test in an athletic population, moreover, playing a contact sport. Therefore, the purpose of our study was to investigate pain levels during a cold pressor test in athletes and measure the relationship between pain levels, catastrophizing, and cardiovascular variables. We hypothesized that the cold pressor test would result in an increase in heart rate, systolic blood pressure, diastolic blood pressure, pain and standard deviation of inter-beat interval. We hypothesized a correlation between scores in the Pain Catastrophizing Scale and subjective pain ratings recorded throughout the test.

**Material and Methods**

**Participants**

The study protocol was approved by the University ethics committee (certificate# XXX). All participants signed an informed consent form prior to beginning their testing. For confidentiality purposes, we used a subject number for identification purposes during data analysis. A convenience sample of male rugby athletes volunteered to participate in the study. During the initial screening period, participants were excluded if they were smokers, currently taking medication that alters cardiovascular function, diagnosed with Reynaud’s Syndrome, or had elevated blood pressure (greater than 140 mmHg systolic and over 90 mmHg diastolic). In order to be included in the study, we asked participants to not participate in physical activity that day, not consume alcohol or caffeine for 12 hours prior to the study, and not to consume food at least two hours before the study.

**Pain, anxiety, and catastrophizing measures**

**Pain**

To measure pain intensity, we used a self-reported 0-10 Numeric Pain Rating Scale, ranging from “0” meaning no pain at all to “10”, representing the worst pain imaginable. The Numeric Rating Scale is a
valid, reliable, and responsive tool to measure pain. The numeric pain rating scale is the most common method of measuring pain and well established clinical measure.

**Pain Catastrophizing**

We used the Pain Catastrophizing Scale (PCS)\(^{21}\) to measure catastrophizing, which is a 13-item assessment of the frequency of catastrophic thoughts about pain experience. The Pain Catastrophizing Scale examines rumination (e.g., “I can’t seem to keep it out of my mind”), helplessness (e.g., “There is nothing I can do to reduce the intensity of the pain.”), and magnification (e.g., “I become afraid that the pain may become worse.”). Participants rate the statements with regards to how often catastrophic thoughts occur on a Likert scale ranging from 0 (not at all) to 4 (all the time). Total scores range from 0–52 with higher scores indicating more frequent occurrence of catastrophic thoughts. In chronic pain patients, a total PCS score of 30 or more represents a clinically high level of catastrophizing.\(^{21}\) There are no established cut-off scores for athletes since measuring catastrophizing in athletes is still a relatively novel measure.\(^{3}\) The PCS is easy to administer, has been widely used when treating chronic pain patients, has excellent psychometric properties, and has correlated to pain scores in previous studies.\(^{8,21}\)

**State and Trait Anxiety**

State-Trait Anxiety Inventory (STAI)\(^{25}\) is a self-reported instrument designed to assess levels of state anxiety and trait anxiety, through two 20-item questionnaires scored by a 4-point Likert-scale. State anxiety can be defined as a transient momentary emotional status that results from situational stress. The state scale of the State-Trait Anxiety Inventory asks people to describe how they feel at a particular moment in time (e.g., calm, tense) rated on a 4-point intensity scale ranging from “not at all” to “very much so”. Trait anxiety represents a predisposition to react with anxiety in stressful situations. The trait scale of the State-Trait Anxiety Inventory consists of 20 statements describing how people generally feel (e.g., confident) rated on a 4-point frequency scale ranging from “almost never” to “almost always”. Range of scores for STAI for each subtest is 20–80, the higher score indicating greater anxiety. A cut
point of 39–40 has been suggested to detect clinically significant symptoms for the State-Anxiety scale.\textsuperscript{26,27}

\textbf{Cardiovascular measures}

For the initial screening period we measured blood pressure using the Accutorr Plus V (Mindray, Mahwah, USA). During the rest of the procedure we used the Nexfin\textsuperscript{®}-System (BMEYE, Amsterdam, The Netherlands) to continuously measure heart rate, systolic blood pressure, diastolic blood pressure, and inter-beat interval during the testing. The Nexfin\textsuperscript{®}-System is a waveform analysis using a finger pressure cuff attached to the middle finger of the contralateral hand and calibrated with a heart-level sensor. The Nexfin\textsuperscript{®} system records values in thirty second intervals. According to previous studies, measuring HR over a time period is important due to the minute by minute variability of the heart rate.\textsuperscript{28,29} Previous authors indicate that the preferred method of measuring heart rate is over a longer duration of recording and averages over time.\textsuperscript{28,29}

\textbf{Cold Pressor Test}

We used the cold pressor test to induce pain and participants were aware of this procedure prior to testing. For our cold pressor test, we lined a plastic cooler with ice packs and filled approximately two thirds with water. We added crushed ice to maintain water temperature between 2°C and 4°C during the test and we recorded temperature at the same time points as pain to ensure that the water temperature remained between 2°C and 4°C. Throughout the duration of the test, participants were in a seated position. When instructed to do so, participants submerged their hand in the water, making sure to submerge up to 1 cm above their wrist. We instructed the participants to keep their hands open in the water and not to touch any of the ice packs surrounding the cooler. The cold pressor test lasts 3 minutes. We encouraged participants to remain in the cold water for the duration of the test, however they were informed that they could remove their hand if they felt uncomfortable, in too much pain or felt unwell. A previous study suggested that 90 to 120 seconds of stimulation should be sufficient to achieve a true peak response.\textsuperscript{9}
Participants who completed the 3-minute test remained seated during a 10-minute post-CPT period following the submersion with their hands out of the water.

**Experimental Protocol**

First, during the initial screening period, we met with participants to obtain informed, signed consent, record cardiovascular values to screen for abnormalities, obtain participant demographic information and filling of questionnaires. The initial screening period was completed prior to the cold pressor test procedures in a consultation room, separate from the test location. We recorded demographic data such as age, weight, height, ethnicity, country they grew up in and whether or not they have used cryotherapy before. If so, we recorded the frequency and body parts exposed to ice. We also asked participants to list sports they perform and how many years they have been playing. Participants then filled out the Pain Catastrophizing Scale, and the State-Trait Anxiety Inventory. Next, a research assistant measured the following values; sub-lingual temperature using a thermometer, heart rate (over 15 seconds) and blood pressure using the Accutorr Plus V. We measured blood pressure after having the participant seated for the duration of the initial screening period. This first initial screening heart rate and blood pressure measure was completed by the research assistant to identify any heart abnormalities that would exclude the participant including having an elevated systolic blood pressure over 140mmHg and diastolic over 90 mmHg. In addition, it gave us an idea of what their resting HR and blood pressure would be. Also, the research assistant would ask about exclusion criteria. After the measures were completed in the consultation room, the participant was brought to the room where we completed the cold pressor test procedures and where the heart rate measures used in the analysis were collected by the machine Nexfin®-System (BMEYE, Amsterdam, The Netherlands) over the course of the 15 minutes baseline period as part of the total 30 minutes procedure. The cold pressor testing procedure involved a 15-minute baseline, 2-minute anticipation for test, 3-minute cold pressor test, and a 10-minute post-CPT period for a total of 30 minutes (see Figure 1). After the 15-minute baseline, we brought the water bath into the room and we allowed two minutes to elapse before
commencing the hand immersion in order to minimize the effects of anticipation. Not using the 2 min prior to the test in any baseline calculations was our attempt to ensure the baseline HR and blood pressure measurements were true baseline measures and not prematurely elevated due to the impending cold pressor test. However, we acknowledge that it is challenging to get a true baseline HR and blood pressure measure prior to a painful test. At seventeen minutes, the participant immersed their right hand for three minutes. We started the cold pressor test at minute 17 and ended at minute 20. Following the cold immersion, the participant removed their hand from the water and waited during the 10-minute post-CPT period from minute 20 to minute 30. The participant reported their pain at multiple points during the procedure. We averaged the pain scores during the baseline and during the post-CPT period and used those values in the analysis. During the CPT, we used the highest pain value reported in the analysis as well. We monitored the variables such as body temperature, water temperature, pain levels (scored 0 to 10), while the Nexfin®-Monitoring System recorded the heart rate, systolic blood pressure, diastolic blood pressure, and inter-beat interval in thirty second intervals throughout the entirety of the 30 minutes. We averaged all the heart rate values, systolic blood pressure, diastolic blood pressure, and inter-beat interval during the 15 min baseline and used those values in the analysis. We repeated this for the period during CPT referred to as CPT1 and CPT2 on Figure 1. CPT1 refers to as the average of cardiovascular variables recorded at minute 17, 17.5 and 18. CPT2 refers to as the average of cardiovascular variables recorded at minute 18.5, 19 and 19.5. We averaged the cardiovascular values for the post-CPT period. The timeline of the cold pressor test and measurements are represented in Figure 1.

Calculation of Heart Rate Variability (HRV): Standard Deviation Inter-Beat Interval (SDIBI)

Heart rate variability is defined as the change in the time intervals between adjacent heartbeats and is part of regulatory systems in order to adapt to environmental and psychological challenges.\textsuperscript{16} The oscillations of a healthy heart are complex and constantly changing, which allow the cardiovascular system to rapidly adjust to sudden physical and psychological challenges.\textsuperscript{28,29} In addition, the HRV reflects regulation of autonomic balance, blood pressure, gas exchange, and heart. We measured heart rate variability by calculating the standard deviation of the inter-beat interval (SDIBI).\textsuperscript{17,18} We collected inter-beat interval
values at the same time as heart rate, systolic and diastolic blood pressure which was every 30 seconds. Then we averaged the standard deviations for the whole fifteen minutes baseline period to calculate the standard deviation inter-beat interval from minute 0 to 15. For the standard deviation inter-beat interval during cold pressor test, we used the standard deviation of the recording from minute 17 to 19.5. For standard deviation inter-beat interval in post-CPT, we calculated the standard deviation inter-beat interval from minute 20 to 30 (see Figure 1).

Statistical Analysis

The cardiovascular values recorded at thirty second intervals by Nexfin were averaged for each participant into three time points including baseline (minute 0 to 15), during CPT (minute 17 to 20), and post-CPT (minute 20 to 30). We did not include recordings from the 2-minute anticipation period (minute 15 to minute 17) since we thought that the 2 minutes right before the start of the CPT would be the most "stressful" for the participant. So, we did not want to include the HR, BP, SDIBI measures during this time as part of the baseline measures. We calculated the average values for the heart rate, systolic blood pressure, diastolic blood pressure, and standard deviation inter-beat interval for the three time periods of baseline, during CPT, and post-CPT. We conducted separate one-way ANOVA's to identify any significant changes to heart rate, systolic blood pressure, diastolic blood pressure, pain and standard deviation inter-beat interval over the three time periods of baseline, during CPT, and post-CPT. We also performed Pearson correlations (at baseline, during the cold pressor test and post-CPT) to screen for relationships among outcome variables in scores on Pain Catastrophizing Scale, State-Trait Anxiety Inventory-Trait and State scales with the cardiovascular measures (heart rate, systolic blood pressure, diastolic blood pressure, standard deviation inter-beat interval). Lastly, we ran a linear regression using change in heart rate as the dependent variable with any significant correlations that were identified with the Pearson correlations. Moreover, we used any significant correlations in a linear regression to identify the significant contribution of each variable. Significance was set at 0.05 and effect sizes (Cohen's
d) were reported for any significant findings, while a p-values between 0.05-0.1 were considered a trend. We completed all analyses using the statistics software SPSS version 24 (IBM).

**Results**

Thirty-seven male rugby athletes (age 24.0±4.6) participated in the study. Of these individuals, one was unable to complete the 3-minute cold pressor test and he withdrew his hand stating that his subjective pain rating was a 10/10. Therefore 36 subjects were analyzed. Initial screening measures of demographics and cardiovascular values are described in Table 1.

**Pain Outcomes**

Participants experienced a significant increase in pain from baseline to during the cold pressor test (0.0 to 4.1 ±2.2, p<.001, d=2.636) followed by a significant decrease in pain post-cold pressor test (4.1 ±2.2 to 0.3 ±0.7, p<.001, d=-2.328). Figure 2 represents mean changes in pain levels over time.

**Cardiovascular Outcomes**

Heart rate increased from baseline to during the CPT but it was not statistically significant (baseline 67.2bpm ±9.8 to 70.1bpm ±11.0, d=0.278). However, the heart rate decreased significantly from during the CPT to post-CPT (70.1bpm ±11.0 to 64.1 bpm ±9.0, p=0.040, d=-0.597) see figure 3. There was a significant increase in systolic blood pressure during the CPT (126.7 mmHg ±16.5 to 149.7 mmHg ±23.4, p<.001, d=1.136) and a significant decrease in systolic blood pressure post-CPT (149.7 mmHg ±23.4 to 137.1 mmHg ±18.8 after the test, p<.001, d=-0.594). Figure 4 represents systolic blood pressure changes over time. In addition, a similar response was noted for diastolic blood pressure, with a significant increase during CPT (77.0 mmHg±8.3 to 92.0 mmHg±11.5, p<.001, d=1.496) followed by a significant decrease to 82.1 mmHg±9.3 in post-CPT (p<.001, d=-0.947).

Heart rate variability as measured by the standard deviation of the inter-beat interval significantly increased during the CPT from 0.0164ms±0.0121 to 0.0400ms±0.0323 (p<.001, d=0.968) before
significantly decreasing to 0.0175ms±0.0122 (p<.001, d=-0.922). Figure 5 represents mean changes in
inter-beat interval over time.

**Relationship between psychological variables and cardiovascular measures**

Significant correlations were identified between Pain Catastrophizing Score and peak pain (0.397, p=.017). In addition, there was a significant relationship between heart rate change and peak pain (0.465, p=0.004). Table 2 represents correlations between outcomes. Figure 6 represents the relationship between Pain Catastrophizing Scale scores and peak pain. Figure 7 represents the relationship between Pain Catastrophizing Scale scores and heart rate.

**Regression analysis**

We originally ran a regression analysis with HR change as a dependent variable compared to pain, PCS, and SDIBI while controlling for age since peak pain, catastrophizing (PCS) and SDIBI were all significant correlations to HR change. However, upon further analysis, the SDIBI is too related to HR change since IBI is generated by the time difference between beats (violated collinearity). Therefore, the final regression was used where HR change is the dependent variable versus peak pain, and catastrophizing (PCS) while controlling for age was the final model. Our results indicated a significant linear regression indicating that peak pain, catastrophizing, and age significantly contributed to 29.2% of the variance in heart rate change during the cold pressor test (p=.011). While the overall model was significant, the coefficient for Pain Catastrophizing was found to be trend toward being a contributor to the model (p=.074). Age was not a predictor for change in heart rate (p=.903). Table 3 represents individual coefficients for the predictors of heart rate change. Figure 7 represents the correlation of Pain Catastrophizing Scale scores with change heart rate in rugby athletes during a CPT (0.437, p=0.008). Our data indicated that high pain catastrophizing is significantly associated with a larger increase in heart rate during a cold pressor test. There was no relationship between change in blood pressure (systolic or diastolic) and scores in Pain Catastrophizing Scale.
Discussion

The purpose of our study was to observe changes in cardiovascular responses of male rugby athletes completing the cold pressor test and the relationship of cardiovascular variables to Pain Catastrophizing and subjective pain ratings. Our results indicate that the cold pressor test, used to induce pain, causes a significant change in systolic blood pressure, diastolic blood pressure, heart rate, and heart rate variability as measured by inter-beat interval in athletes which was related to pain catastrophizing levels in athletes.

To explain the change in cardiovascular variables during a noxious stimuli, several studies of healthy young adults suggest that catastrophizing predicted increased systolic blood pressure reactivity to pain\textsuperscript{8,30,31} and enhanced myocardial contractility for a prolonged period of time following a cold pressor test.\textsuperscript{31} Catastrophizing appears to influence the relationship among muscle tension, cardiovascular stress, and pain response\textsuperscript{30,31} as well as temporal summation of pain, which is frequently a studied index of central pain facilitation.\textsuperscript{5,32} This could explain the lack of correlation between blood pressure and catastrophizing noted in the current study. Changes in blood pressure are stimulated by both pain and cold, however the change in heart rate is considered to be solely stimulated by pain. These assumptions potentially explain the results in the current study; a relationship between heart rate change, pain, and Pain Catastrophizing scores and not with other cardiovascular variables, although cardiovascular variables do respond to the nociceptive stimuli. Moreover, explaining the mechanism linking pain and cardiovascular responses, some studies reported a correlation between reduced gray matter in some regions of the brain and the duration or intensity of pain.\textsuperscript{21,33} A previous study\textsuperscript{33} conducted an 11-week cognitive behavioural therapy intervention for coping pain in order to increase the grey matter volume found in participants suffering from chronic pain. The study resulted in increased gray matter volume in prefrontal and somatosensory brain regions, as well as increased dorsolateral prefrontal volume associated with reduced pain catastrophizing. Increased grey matter through cognitive behavioural therapy reflects greater control over pain, cognitive reappraisal of pain and reduction of catastrophizing.
There have been a few studies that have measured experimentally induced pain in athletes. A previous study by Geva & Defrin compared pain threshold, tolerance, intensity and catastrophizing between a group of triathletes and participants in amateur sports during hand immersion in a 12°C water bath for 60 seconds. At baseline, catastrophizing in triathletes was lower than controls, however, the difference was not significant (16.5±9 vs 20.8±12, p=.053). The pain ratings of triathletes were significantly lower than those of the control group at 1 second 0.8±1.5 versus 4.6±3.8, respectively (P<.001); at 20 seconds 2.3±2.5 vs 6.4±3.5, respectively (P<.001); at 60 seconds 5.5±2.8 vs 8.3±2.6, respectively (P=.01). Both triathletes and non-athletes experienced a similar overall increase in pain intensity (3.7±3.0 and 4.7±3.5 VAS units, respectively). Triathletes and non-athletes appear similarly able to differentiate between painful and non-painful events, meaning they had the same pain threshold, however, the difference in pain may be explained by the difference in pain catastrophizing levels in triathletes compared to the non-athletes. This however was not further explored. Knowing that pain tolerance is the ability and/or willingness to endure pain, triathletes appeared to have greater ability and/or motivation to endure pain in the experimental setup. The difference between catastrophizing in triathletes and non-athletes was not statically significant, however the level of fear of pain of triathletes was significantly lower than that of controls (71.7 ± 14.9 vs 81.0 ± 17.1, P=.05). Higher pain tolerance may stem from lack of fear of the stimulus or its consequence. In the previous study, homogeneity of the group prevented the correlation analysis to support the link between fear of pain and pain tolerance. Of note, the overall increase in pain values in our study (4.1 ±2.2) were similar to the previous study (Triathletes: 3.7±3.0, Non-athletes: 4.7±3.5) even though the water was significantly colder in our study and our cold pressor test lasted 3 minutes. In addition, our catastrophizing average was also lower (12.4 ± 6.2) than triathletes: 16.5±9 and non-athletes: 20.8±12, p=.053 in the previous study, which may also explain the difference in our pain results with a colder and longer cold pressor test compared to this previous study.

In another study by Manning & Fillingim, a cold pressor test in a 1°C water bath was performed on intercollegiate athletes. Participants were asked to report when sensations in the immersed hand first became painful (pain threshold), when the sensations became intolerable (pain tolerance) and to report
pain intensity and unpleasantness at both threshold and tolerance time. Athletes demonstrated higher pain
threshold for cold pain than non-athletes (athletic women 3.07±2.65; athletic men 2.03±1.93; nonathletic
women 4.10±1.98; nonathletic men 3.08±2.04). Athletes demonstrated higher tolerance to cold pain than
non-athletes (athletic women 6.00±3.21; athletic men 4.89±3.37; nonathletic women 6.62±1.36;
nonathletic men 6.37±2.00). Moreover, men exhibited higher pain thresholds and tolerance for cold pain
than women (P=.05). Of all groups, nonathletic women rated pain significantly higher for threshold and
tolerance (P=.05). Similarly, in our study we measure peak pain which can also be referenced to as pain
tolerance. Our study included male rugby athletes and their peak pain during cold pressor test was 4.1
±2.2. Moreover, in the previous study, pain self-efficacy was measured and represents the ability to
directly control the pain experience. Pain Self-Efficacy in athletic activity (positive) was significantly
correlated to pain tolerance intensity (0.309, p=.05). Pain self-efficacy and pain catastrophizing are two
psychosocial factors contributing to the perception, emotional, and physical impact, and responses to
pain. Thus, the correlation between pain self-efficacy and pain tolerance is similar to our results
showing a significant correlation between Pain Catastrophizing Score and peak pain (0.397, p=.017).

In our study, the Tukey Post Hoc test revealed that the heart rate decreased significantly from during the
CPT to after the CPT (70.1bpm ±11.0 to 64.1 bpm ±9.0, p<.001) and there was a trend towards HR
significantly increasing during the CPT test (baseline 67.2bpm ±9.8 to 70.1bpm ±11.0). Similar to a
previous study by Etherton et al. that examined a non-athletic population, our results suggest that the
pain induced by the cold pressor test causes an elevation of cardiovascular measures followed by a
decline after the completion of the nociceptive stimulus. Of note, the methods of our study were similar to
the Etherton et al. study with a similar timeframe of the CPT test and the temperature of water. What was
different is that their study revealed a main effect of gender where females had higher heart rate over the
course of the experiment relative to males (F(1,35) = 9.12, p=.01). Also, a main effect of time reflected
the increase in heart rate during the CPT test for both males and females, and then a decrease after CPT
period (F(2,70) = 27.96, p<.001). However, there was no interaction between time and gender meaning
that HR response for males and females were similar over time. Moreover, there were no differences in
pain catastrophizing between males and females who completed the cold pressor test (22.0 for females
versus 21.8 for males on the PCS) in the study by Etherton et al. The participants who completed CPT
and non-completers did not differ in terms of PCS scores. Mean subjective pain ratings during the CPT
correlated with PCS (r = .403, p = .01), and the PCS scores were correlated with HR or BP indices at
baseline, CPT, or post-CPT. Pain catastrophizing can be correlated to subjective pain ratings during a
painful test. Our results indicate there was a correlation between the change in heart rate and peak pain
and a trend between the change in heart rate and Pain Catastrophizing Scale score. However, Etherton et
al. found no correlation at all 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’s sample
groups consisted of 17 males and 23 females and they found gender differences in cardiovascular
responses during the cold pressor test. Their small sample sizes were a limitation to determine possible
correlations between cardiovascular and pain variables. In contrast to this, another study by Peckerman et
al. suggest that cardiovascular changes are related to both painful and non-painful stimuli. Their results
suggest that changes in blood pressure were caused by non-painful stimulations which led to
vasoconstriction, consequently increasing blood pressure. Their results also proposed that pain induction
was capable of activating both vasomotor and cardiac mechanisms for blood pressure, thus 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. However, a systematic review examining blood pressure in athletes found
that strength-trained athletes have slightly higher blood pressure than endurance-trained athletes. This
larger review paper included 138,390 men and women aged mostly between 18 and 40 years, from varied
sports disciplines. Mean systolic BP varied from 109±11 to 138±7 mm Hg and mean diastolic BP from
57±12 to 92±10 mm Hg. Strength-trained athletes had higher BP than endurance-trained athletes (131.3
±5.3/77.3±1.4 vs 118.6±2.8/71.8±1.2 mm Hg, p< 0.05), and there was a trend towards a higher BP in
athletes training ≥10 hours per week compared with others (121.8 ±3.8/73.8±2.5 vs 117.6±3.3/66.8±6.9, p=0.058), but overall there was no significant difference in BP between athletes and controls.\textsuperscript{35} Therefore, it seems the resting SBP in our athletes was similar to previous studies.

It is possible that previous experience with cryotherapy or cold exposure could affect the results of a cold pressor test. We asked all participants about their experience with ice application and full body cold immersion, and not surprisingly all of our athletes use ice regularly for minor injuries. There are no previous studies we can find that indicate previous experience using local cryotherapy can influence the CPT results. We did collect information about previous use of cold tubs or full body cold immersion. Eight athletes indicated they have used one in the past year usually during tournaments but no more than one a year. There are some studies that suggest whole body exposure to cold can influence the CPT response.\textsuperscript{36,37} However due to the limited use of cold tubs in our athletes, we do not feel like it affected our results. More studies are needed to see if previous local cryotherapy applications can affect the results of a cold pressor test.

Psychological factors such as pain catastrophizing and anxiety may explain athletes’ higher pain tolerance and the varied pain response to a painful stimulus. However, the results of our study indicate catastrophizing is significantly correlated to pain while anxiety was not. We wanted to compare the scores on the PCS and STAI in our athletes to other studies. Our anxiety scores including the 46.9±3.2 for STAI State score and 46.7±3.1 for Trait Score indicating the state and trait anxiety in our athletes was slightly higher than normal.\textsuperscript{26,27} But this elevated level of anxiety was not correlated to the amount of pain experienced during the CPT test. Anxiety is often suggested as a reason for athletes who experienced increased pain, however our results do not support this notion. With regards to pain catastrophizing, total scores range from 0–52 with higher scores indicating more frequent occurrence of catastrophic thoughts.\textsuperscript{21} Our athletes averaged a 12.4±6.2 on the pain catastrophizing scale which on average is lower than some
other studies who obtained a score of 22.0 for females versus 21.8 for males, and a score of 16.5±9 for triathletes versus 20.8±12 for non-athletes. A previous study at the University Centre for Research on Pain and Disability indicated that a total PCS score of 30 represents clinically high level of catastrophizing which the participants in this study are lower than. However, we caution the use of cut off scores for these scales. The PCS was not designed to be sensitive enough to separate from someone with a 31 versus a 29 score for example. Using the proposed cut off would indicate that the person with the 31 is considered a high catastrophizer and the person with a 29 is considered a low catastrophizer could be due to a change to one response on the scale. The scale not being sensitive enough for a change of one or two numbers is also part of the reason why we did not split up the group into a low and high catastrophizing group for further analysis.

Limitations

Interestingly, with a controlled stimulus, while most subjective peak pain ratings hovered near the average, two participants claimed to feel no pain and one participant could not complete the test due to a 10 on 10 pain rating. Our results are consistent with findings that demonstrate the variability of pain. As mentioned earlier, the cold pressor test has been shown to be an effective inducer of pain. However, a limitation and a reason in our study explaining the failure at causing pain in some participants might be that the male athletes were reluctant to report their true pain levels, which could have influenced the average. We would also like to acknowledge the potential benefit from future studies including a path analysis to determine the mediation effect of catastrophizing on induced pain. We were not able to conduct a path analysis with this data since we violated an assumption due to the complex relationship between pain and pain catastrophizing. Future analyses and studies are needed to examine this bi-directional relationship between pain and pain catastrophizing. Other limitations may include the amount of sleep the participants got the night preceding the testing day, which was not controlled. As suggested by Finan et al, poor sleep may lead to a stronger pain reaction. The time of day of the testing, which ranged between 8:00 am and 6:00 pm may have also had an effect on the results. Lastly, we only tested
male athletes since sex may influence cardiovascular responses to pain, so future studies are needed to examine the influence of catastrophizing on female athletes’ cardiovascular response to pain.

**Clinical Implications**

The clinical implications of our study are that athletes with high catastrophizing feel more pain and have a stronger cardiovascular response to pain during the same painful stimulus. This has several clinical implications regarding assessment and rehabilitation of athletes. Pain is quite variable between athletes even with similar injuries. It has been suggested that pain catastrophizing can explain important differences in sport-related pain behaviour, more than the pain intensity itself. While our results were using experimentally induced pain it seems like catastrophizing may explain some of the clinical variability in pain while assessing injured athletes. In this study, anxiety was not correlated to any other variables including pain, contrarily to other studies who found that anxiety was a predictor for systolic blood pressure reactivity to painful stimuli and proposed that anxiety and pain catastrophizing are relevant in development of exaggerated pain perception. If an athlete experiences an elevated pain response over time that could be problematic. Previous studies have suggested that an elevated pain response can lead to the development of chronic pain. The fear avoidance model has indicated that in the general population, those that have higher pain catastrophizing are more likely to develop chronic pain and increased disability. An increase in chronic pain could prolong the rehabilitation of some athletes. Future studies are also needed for potential individual pain management strategies for athletes based on their psychological and possibly cardiovascular response to pain, which may help athletes return to play more quickly. In addition, more studies are needed to identify a more objective or physiological measure of pain that can reduce the subjective nature of pain measurement making the pain assessment more accurate.

**Conclusion**
Our results indicated a significant relationship between catastrophizing and pain experienced during a cold pressor test in athletes. In addition, the amount of pain experienced by the athletes was correlated to the amount of heart rate change during the cold pressor test. This means that athletes with higher catastrophizing felt more pain and experienced a bigger cardiovascular response during the same painful stimulus as other athletes. A linear regression indicating that peak pain, catastrophizing, and age significantly contributed to 29.2% of the variance in heart rate change during the cold pressor test (p=.011). While cardiovascular measures may be a potential objective measure of pain in the future, our data indicates that psychological constructs such as catastrophizing will need to be taken into consideration for any potential cardiovascular measures of pain. With further research, heart rate change may be an interesting option to obtain a more objective measure for pain. The relationship between pain and cardiovascular changes helps 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 it is possible that administering the Pain Catastrophizing Scale as a screening tool for athletic trainers and athletic therapists working with athletes may be of use in interpreting the clinical pain presentation during an injury, as well as identifying a factor that could prolong rehabilitation.
Figure 1. Timeline and measurements during the Initial Screening Period and the Cold Pressor Test procedures

Initial Screening Period prior to the start of the Cold Pressor Test Procedures where we obtained signed consent, recorded cardiovascular values to screen for abnormalities, obtained participant demographic information and filling of questionnaires. The cold pressor testing procedure involved a 15-minute baseline, 2-minute anticipation for test, 3-minute cold pressor test, and a 10-minute post-CPT period. Baseline timeframe is from minute 0 to minute 15, CPT timeframe is from minute 17 to minute 20 and post-CPT timeframe is from minute 20 to minute 30. Cardiovascular measures including: Heart rate, systolic blood pressure, diastolic blood pressure and inter-beat interval were recorded at every 30-second interval throughout the entire 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 corresponds to the average of all measurements taken from 0 to P1. Peak pain between P2, P3 and P4 was determined as pain during CPT. Cardiovascular values during CPT were averaged from recordings at minute 17, 17.5 and 18 (CPT1) and from minute 18.5, 19 and 19.5 (CPT2). We averaged pain measured at P5 and P6 to obtain the pain for post-CPT. Cardiovascular value at post-CPT corresponds to the average of all measurements taken from minute 20 to 30.

CPT: Cold Pressor Test

Table 1. Initial Screening measures of all participants.

| Measure            | Mean   | SD     |
|--------------------|--------|--------|
| BMI                |        |        |
| PCS                |        |        |
| HR                 |        |        |
| SBP                |        |        |
| DBP                |        |        |

Values are the mean and standard deviation unless otherwise indicated. BMI Body Mass Index, PCS Pain Catastrophizing Scale, HR Heart Rate, SBP Systolic Blood Pressure, DBP Diastolic Blood Pressure
Figure 2. Significant increase in pain from baseline to during cold pressor test (p<0.001, d=2.636) followed by a significant decrease in pain after a cold pressor test (p<0.001, d=-2.328) in rugby athletes.

CPT Cold Pressor Test

*P < 0.05
** P < 0.001

Figure 3. Significant decrease in mean heart rate after a cold pressor test in rugby athletes. (p=0.040, d=-0.597)

CPT Cold Pressor Test

*P < 0.05
** P < 0.001

Figure 4. Significant increase in mean systolic blood pressure from baseline to during cold pressor test (p<0.001, d=1.136) followed by a significant decrease in mean systolic blood pressure after a cold pressor test (p<0.001, d=-0.594) in rugby athletes.

CPT Cold Pressor Test

*P < 0.05
** P < 0.001

Figure 5. Significant increase in mean standard deviation of the inter-beat interval from baseline to during cold pressor test (p<0.001, d=0.968) followed by a significant decrease in mean standard deviation of the inter-beat interval after a cold pressor test (p<0.001, d=-0.922) in rugby athletes.

CPT Cold Pressor Test

*P < 0.05
** P < 0.001
Table 2. Correlations between pain, state and trait anxiety, catastrophizing, and cardiovascular measurements during a cold pressor test in rugby athletes

* P < 0.05

** P < 0.001

*HR* Heart Rate, *SBP* Systolic Blood Pressure, *DBP* Diastolic Blood Pressure, *Avg* Average, *CPT* Cold Pressor Test, *PCS* Pain Catastrophizing Scale, *SDIBI* Standard Deviation Inter-Beat Interval

Figure 6. Correlation of Pain Catastrophizing Scale scores with Peak Pain experienced during a CPT in rugby athletes (0.397, p= 0.017).

PCS Pain Catastrophizing Scale

Figure 7. Correlation of Pain Catastrophizing Scale scores with change heart rate in rugby athletes during a CPT (0.437, p=0.008).

*HR* Heart Rate, *PCS* Pain Catastrophizing Scale, *CPT* Cold Pressor Test

Table 3. Coefficients of linear regression for the heart rate change in rugby athletes during a cold pressor.

* P < 0.05

** P < 0.001

*HR* Heart Rate
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## Table 1. Baseline measures of all participants.

Values are the mean and standard deviation unless otherwise indicated. **BMI** Body Mass Index, **PCS** Pain Catastrophizing Scale, **HR** Heart Rate, **SBP** Systolic Blood Pressure, **DBP** Diastolic Blood Pressure.
Table 2. Correlations between pain, state and trait anxiety, catastrophizing, and cardiovascular measurements during a cold pressor test in rugby athletes

|                | HR change | SBP change | DBP change | Avg Pain Post CPT | Peak Pain | PCS | STATE | TRAIT | SDIBI change |
|----------------|-----------|------------|------------|-------------------|-----------|-----|-------|-------|--------------|
| HR change      | 1         | .161       | .186       | .291              | .465*     |    |       |       |              |
| SBP change     | 1         | .887**     | .202       | .286              | .437*     | -.011| -.002 | .444*  |              |
| DBP change     | 1         |            | .208       | .309              | .088      | -.069| .195  | -.050  |              |
| Avg Pain Post CPT | 1   | .180       | .491*      | .214              | .063      | .153 |       |       |              |
| Peak Pain      | 1         | .397*      |            | .132              | .103      | .205 |       |       |              |
| PCS            | 1         |            |            | -.196             | -.279     | -.012|       |       | .219         |
| STATE          | 1         |            |            | .172              | .031      |     |       |       |              |
| TRAIT          | 1         |            |            |                   |           |     |       |       |              |
| SDIBI change   |           |            |            |                   |           |     |       |       | 1            |

* P < 0.05
** P < 0.001

*HR* Heart Rate, *SBP* Systolic Blood Pressure, *DBP* Diastolic Blood Pressure, *Avg* Average, *CPT* Cold Pressor Test, *PCS* Pain Catastrophizing Scale, *SDIBI* Standard Deviation Inter-Beat Interval
Table 3. Coefficients of linear regression for the heart rate change in rugby athletes during a cold pressor.

* P < 0.05
** P < 0.001

HR Heart Rate
CPT
- CPT Average Heart Rate, Systolic Blood Pressure, Diastolic Blood Pressure & Inter-Beat Interval
  CPT1: Average of minute 17, 17.5 and 18
  CPT2: Average of minute 18.5, 19 and 19.5
- CPT Peak Pain from recordings P2, P3, P4

BASELINE
- Baseline Average Heart Rate, Systolic Blood Pressure, Diastolic Blood Pressure & Inter-Beat Interval
- Baseline Pain from recording P1

POST-CPT
- Post-CPT Average Heart Rate, Systolic Blood Pressure, Diastolic Blood Pressure & Inter-Beat Interval
- Post-CPT Average Pain from recordings P5, P6

Initial Screening Period
- Change from consultation room to test room

Start CPT

Finish CPT
Initial Screening Period prior to the start of the Cold Pressor Test Procedures where we obtained signed consent, recorded cardiovascular values to screen for abnormalities, obtained participant demographic information and filling of questionnaires. The cold pressor testing procedure involved a 15-minute baseline, 2-minute anticipation for test, 3-minute cold pressor test, and a 10-minute recovery period. Baseline timeframe is from minute 0 to minute 15, CPT timeframe is from minute 17 to minute 20 and post-CPT timeframe is from minute 20 to minute 30. Cardiovascular measures including: Heart rate, systolic blood pressure, diastolic blood pressure and inter-beat interval were recorded at every 30-second interval throughout the entire 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 corresponds to the average of all measurements taken from 0 to P1. Peak pain between P2, P3 and P4 was determined as pain during CPT. Cardiovascular values during CPT were averaged from recordings at minute 17, 17.5 and 18 (CPT1) and from minute 18.5, 19 and 19.5 (CPT2). We averaged pain measured at P5 and P6 to obtain the pain for post-CPT. Cardiovascular value at post-CPT corresponds to the average of all measurements taken from minute 20 to 30.

*CPT*: Cold Pressor Test
Figure 2. Significant increase in pain from baseline to during cold pressor test (p<0.001, d=2.636) followed by a significant decrease in pain after a cold pressor test (p<0.001, d=-2.328) in rugby athletes.

*CPT Cold Pressor Test*

*P < 0.05

**P < 0.001
Figure 3. Significant decrease in mean heart rate after a cold pressor test in rugby athletes.

\( p = 0.040, \ d = -0.597 \)

*CPT* Cold Pressor Test

\* \( P < 0.05 \)

\** \( P < 0.001 \)
Figure 4. Significant increase in mean systolic blood pressure from baseline to during cold pressor test (p<0.001, d=1.136) followed by a significant decrease in mean systolic blood pressure after a cold pressor test (p<0.001, d=-0.594) in rugby athletes.

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*P < 0.05

** P < 0.001
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Figure 7. Correlation of Pain Catastrophizing Scale scores with change heart rate in rugby athletes during a CPT (0.437, p=0.008).

*HR Heart Rate, PCS Pain Catastrophizing Scale, CPT Cold pressor test