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Negative Emotion and Joint-Stiffness Regulation Strategies After Anterior Cruciate Ligament Injury

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Context: Fear of reinjury after an anterior cruciate ligament (ACL) reconstruction (ACLR) may be associated with persistent deficits in knee function and subsequent injury. However, the effects of negative emotion on neuromuscular-control strategies after an ACL injury have remained unclear.

Objective: To identify how negative emotional stimuli affect neural processing in the brain and muscle coordination in patients after anterior cruciate ligament reconstruction compared with healthy control participants.

Design: Case-control study.

Setting: Neuromechanics laboratory.

Patients or Other Participants: Twenty patients after unilateral anterior cruciate ligament reconstruction and 20 healthy recruits.

Main Outcome Measure(s): Electrocortical θ (4–8 Hz) activity (event-related synchronization, % increased power relative to a nonactive baseline) at selected electrodes placed at the frontal (F3, Fz, F4) and parietal (P3, Pz, P4) cortices using electroencephalography, neurophysiological cardiac changes (beats/min), and subjective fear perceptions were measured, along with joint stiffness (Nm/°/kg) with and without an acoustic stimulus in response to 3 types of emotionally evocative images (neutral, fearful, and knee-injury pictures).

Results: Both groups had greater frontoparietal θ power with fearful pictures (Fz: 35.9% ± 29.4%; Pz: 81.4% ± 66.8%) than neutral pictures (Fz: 24.8% ± 29.7%, $P = .002$; Pz: 64.2 ± 54.7%, $P = .024$). The control group had greater heart-rate deceleration with fearful (–4.6 ± 1.4 beats/min) than neutral (–3.6 ± 1.3 beats/min, $P < .001$) pictures, whereas the ACLR group exhibited decreased heart rates with both the fearful (–4.6 ± 1.3 beats/min) and injury-related (–4.4 ± 1.5 beats/min) pictures compared with neutral pictures (–3.4 ± 1.4 beats/min, $P < .001$). Furthermore, during the acoustic startle condition, fearful pictures increased joint stiffness (Nm/°/kg) in the ACLR group at the midrange (0°–20°: 0.027 ± 0.02) and long range (0°–40°: 0.050 ± 0.02) compared with the neutral pictures (0°–20°: 0.017 ± 0.01, $P = .024$; 0°–40°: 0.043 ± 0.02, $P = .014$).

Conclusions: Negative visual stimuli simultaneously altered neural processing in the frontoparietal cortices and joint-stiffness regulation strategies in response to a sudden perturbation. The adverse effects of fear on neuromuscular control may indicate that psychological interventions should be incorporated in neuromuscular-control exercise programs after ACL injury.

Key Words: functional joint instability, neuroplasticity, electroencephalography, fear of reinjury, neurocognition

Key Points

- Unpleasant visual stimuli may increase neural processing in several regions of the brain that are highly associated with the goal-directed motor-planning processes required for muscle coordination.
- Fear of reinjury may interrupt the normal cascade of neurocognitive processing and cause joint-stiffness dysregulation in patients after anterior cruciate ligament (ACL) injury.
- Fear of reinjury should be assessed after an ACL injury because it can lead to neuromuscular-control deficits and reinjury, even among patients who have been cleared to participate at their preinjury level of physical activity.
- Psychological interventions should be incorporated with neuromuscular-control exercises after ACL injury.
- Further research is needed to assess the effectiveness of psychological interventions used in conjunction with traditional neuromuscular-control exercise programs in improving joint-stiffness regulation strategies in patients after ACL injury.

Neuromuscular-control dysfunction, particularly when an unanticipated event occurs during high-speed functional movements, is the leading cause of damage to the anterior cruciate ligament (ACL), especially in physically active and athletic populations.[1] Persistent neuromuscular-control deficits after an ACL injury can also lead to experiences of the joint "giving way," known as *functional joint instability*,[2] decreased quality of physical or athletic performance, subsequent ACL tears, or early development of knee osteoarthritis.[3] Therefore, appropriate neuromuscular control is critical not only for injury prevention but also for maintaining

Table 1. Participants' Demographic Data[a]

Characteristic	Participant Group (Mean ± SD)		P Value[b]	95% Confidence Interval for Group Difference
	Healthy Control (n = 20)	Anterior Cruciate Ligament Reconstruction (n = 20)		
Age, y	23.90 ± 4.78	21.90 ± 3.51	.102	−0.68, 4.68
Height, cm	166.62 ± 9.20	165.86 ± 10.42	.808	−5.53, 7.05
Mass, kg	62.52 ± 12.47	71.76 ± 25.15	.150	−21.94, 3.48

[a] No difference was present in the demographic data between groups (P values > .05).
[b] Difference between groups.

functional joint stability and joint health after knee injury.[4]

To regulate neuromuscular control properly, the central nervous system (CNS) must be able to simultaneously and precisely prepare for and react to sudden events. These preparatory (feed-forward) and reactive (feedback) pathways of the dynamic-restraint system contribute to functional joint stability by optimizing muscle-stiffness regulation strategies during high-velocity physical maneuvers.[2,5] The control of muscle excitation and inhibition through both feed-forward and feedback pathways is highly associated with cognitive processing in the brain, including current and learned proprioceptive information, which may be altered after an ACL rupture.[4,6] Growing evidence[7,8] supports the contribution of altered neural networks related to muscle coordination to neuromuscular deficits and subsequent functional joint instability after ACL injury.

Furthermore, several psychological factors, such as greater fear of movement after an ACL injury, are thought to be associated with diminished knee function.[8,9] Anterior cruciate ligament-injured patients with decreased knee function reported greater fear of reinjury and movement compared with those who were able to cope and return to preinjury levels of physical activity.[9] Fear-evoking stimuli can increase cortical activation in several frontal regions in the brain as a part of emotional regulation.[10,11] For example, the anterior cingulate and prefrontal cortices perform executive functions that help regulate emotions but are also responsible for the preparation of voluntary movements.[10,11] Because fear is a potent cognitive and emotional response to a perceived threat or noxious stimuli, emotional regulatory neural circuits in the brain may instantly demand greater cognitive processing to manage increased attentional resources.[12] As both emotional regulation and muscle coordination require precise and accurate neurocognitive strategies, more fear during physical activity after ACL injury may disrupt the simultaneous neurocognitive processing necessary for regulating both aversive feelings and neuromuscular control, thereby resulting in knee-stiffness dysregulation and functional joint instability.[2,4,13] However, it remains unclear how negative emotional stimuli after ACL injury may alter critical neural processing in the brain related to cognitive processing or muscle-stiffness regulation strategies, which could predispose patients after ACL reconstruction (ACLR) to experience persistent functional joint instability.

Because greater fear of reinjury or movement can interrupt goal-directed decision making and the cognitive processing related to muscle coordination, we hypothesized that increased frontoparietal cortex activation in the θ frequency band (4–8 Hz), representing higher neural processing in the cognitive control network,[14] and greater alteration in joint-stiffness regulation strategies in response to general fearful or sport knee-injury–related pictures may be observed when compared with neutral emotional stimuli. Therefore, the aim of our study was to compare cortical activation and joint-stiffness regulation strategies in response to general or specific situation-related fearful visual stimuli between patients after ACLR and healthy control participants. The findings may illustrate the importance of psychological factors and cognitive function skills as critical components of rehabilitation strategies and help to improve current ACL rehabilitation practice to optimize patient outcomes.

METHODS

Participants

Forty individuals (20 ACLR patients, 20 healthy control participants) between the ages of 18 and 45 years volunteered (Table 1). The ACLR group consisted of patients who had undergone ACLR for 1 or more unilateral ACL ruptures more than 6 months earlier and were cleared to return to participation at their preinjury level of physical activity. Healthy control participants were physically active with no history of ACL rupture. Participants were excluded if they had a history of lower extremity fracture or surgery in the prior 6 months or a medical condition that could interfere with electrocardiography (ECG) or electroencephalography (EEG) data acquisition, such as metal implants in the head, face, or chest or neurologic problems. Additionally, participants were excluded if they had a history of a hearing impairment due to the inclusion of an acoustic-startle stiffness condition in the study protocol. All participants signed an informed consent form that was approved by the University of Delaware Institutional Review Board, which also approved the study.

Protocol

This case-control study involved measurements of emotional responses followed by joint-stiffness regulation in response to emotion-related visual stimuli. All testing was performed in a single testing session of approximately 2 hours (Figure 1). Participants were asked to dress in athletic shorts and a T-shirt, avoid the use of hair and cosmetic products, and not wear metal earrings, which can interfere with measures of joint stiffness and electrocortical activity.

Emotional Responses. To induce targeted neutral and fearful emotions, we selected 62 neutral and 60 fear-related

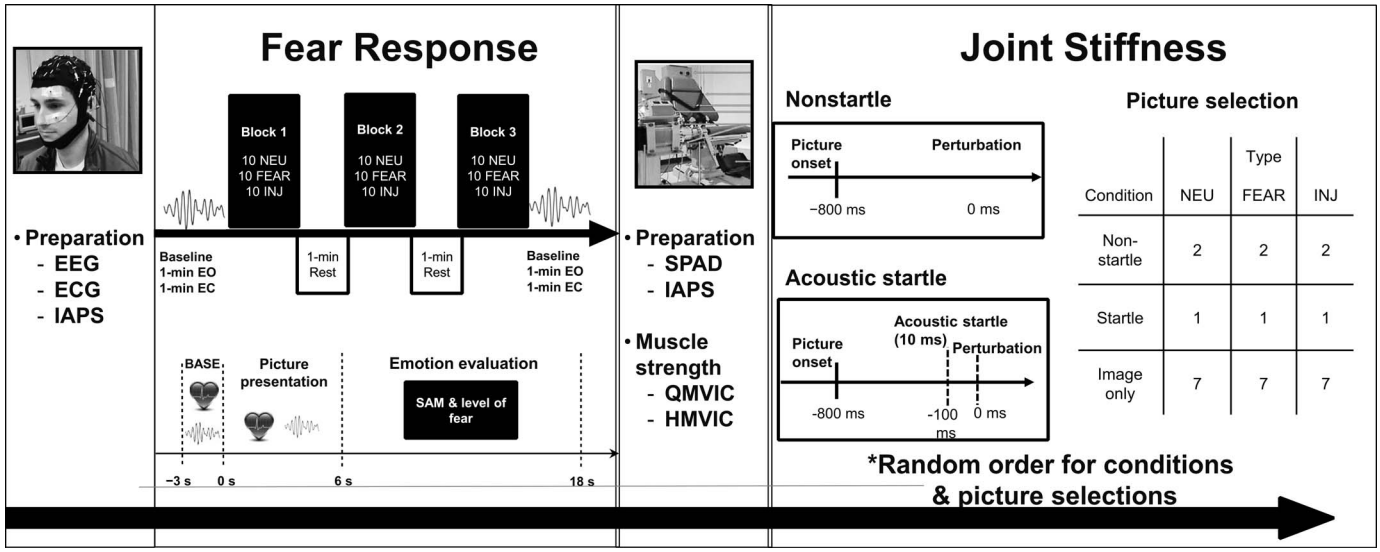

Figure 1. Research protocol for measures of emotional response and joint stiffness. Nonstartle: A 40° knee-flexion perturbation at 800 milliseconds after picture presentation. Startle: An acoustic sound at 100 milliseconds before the perturbation. Abbreviations: BASE, 3 seconds before picture onset; EC, eyes closed; ECG, electrocardiogram; EEG, electroencephalogram; EO, eyes opened; FEAR, fearful pictures; HMVIC, hamstrings maximum voluntary isometric contraction; IAPS, International Affective Picture System; INJ, sport knee-injury–related pictures; NEU, neutral pictures; QMVIC, quadriceps maximum voluntary isometric contraction; SAM, Self-Assessment Manikin.

pictures from the International Affective Picture System (IAPS), which was developed to provoke a variety of emotions in 2 major dimensions of the Self-Assessment Manikin (SAM): affective valence and arousal.[15] The valence dimension ranges from 1 = *very unhappy* to 9 = *very happy*, whereas the arousal dimension ranges from 1 = *very calm* to 9 = *very aroused*. The neutral pictures, which consist of objects such as plants, office supplies, or neutral human images, were chosen from a range of valence (4.03–5.20) and arousal (1.72–3.46) values, whereas the fear-related pictures, such as severely injured animals or humans, attacks by animals, threatening images of other people, or accident-related images, were chosen from a different range of valence (1.31–4.32) and arousal (5.9–7.15) values.[15] These valence and arousal ranges for both neutral and fear-related pictures were based on standard protocols from the scientific literature.[16] Additionally, 60 knee-injury–related pictures from an online search were added to determine the effects on neurophysiological responses, SAM scores, level of fear, brain activity, and joint-stiffness regulation strategies compared with the selected pictures from the IAPS. The knee-injury–related images were included if they were sport related and showed either noncontact or contact mechanisms of ACL injuries, such as pivoting or twisting movements. Sport selection was categorized into 9 types according to ACL incidence rates: basketball, cycling, football, gymnastics, handball, soccer, ski, tennis, and wresting.[17] A picture was excluded if its resolution was lower than 1024 × 768 pixels.[16] Six presentation blocks were constructed, and each block contained 30 pictures (10 neutral, 10 fear related, 10 knee-injury related). The order of block presentation was counterbalanced using a Latin square and pictures in each block were randomized across participants.

Emotional responses were evaluated using measures of heart rate, cortical activation, and the SAM, as well as the level of fear on 9-point Likert scales (Figure 2).[18] A custom-built single-channel surface ECG machine was used, and Ag/ACI bipolar self-adhesive ECG electrodes were attached to both sides of the shoulders with the hip as a reference location for recording heart rate. Cortical activation related to each targeted emotional response was measured using a 32-channel EEG system (Compumedics Neuroscan, Charlotte, NC) in compliance with the international 10 : 20 system.[19]

Three testing blocks were performed, and each block was composed of 30 randomly ordered trials. Each trial included a black screen before the picture (baseline = 6 seconds), the picture presentation (6 seconds), a black screen (postbaseline = 3 seconds), and an emotional rating interval (12 seconds) in which the picture was not displayed. Participants rated valence, arousal, and level of fear regarding the selected picture, which was presented on a 21-in (53-cm) liquid crystal display monitor (43 × 32 cm), approximately 100 cm away.[20] Continuous heart rate was collected during baseline and the picture presentation and after baseline and synchronized with EEG data via a custom LabVIEW program (National Instruments, Austin, TX); emotional rating scores were reported separately for each trial. Participants were asked to keep their eyes open and looking at the screen during testing while blinking comfortably as needed. They were monitored and encouraged to minimize body or facial muscle movements to limit impedance and artifact. Digital triggers from a custom IAPS LabVIEW program were sent to Scan software (version 4.5; Compumedics Neuroscan) to synchronize the picture onset and heart-rate data with brain activation.

Joint-Stiffness Strategy. Joint stiffness in response to visual stimuli was measured using a custom-built stiffness- and proprioception-assessment device (Figure 2).[1] We followed established joint-stiffness protocols[1] that require maximum knee-extension efforts in response to a rapid, unanticipated perturbation, which occurred through a 40°

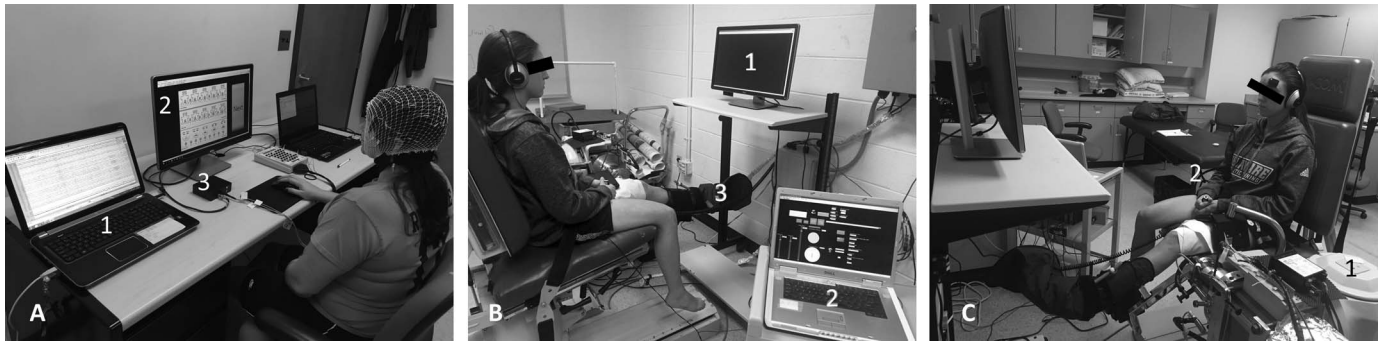

Figure 2. A, Setup for measure of emotional response. A1, Electroencephalograph; A2, Self-Assessment Manikin; A3, Electrocardiogram. B and C, Setup for stiffness testing in response to emotionally evocative pictures. B1, Picture presentation screen; B2, SPAD control computer; B3, Vacuum splint. C1, SPAD machine; C2, Safety switch. SPAD, a custom-built stiffness- and proprioception-assessment device.

flexion arc from 30°–70°, with an acceleration of 1000°/s^2 to a velocity of 100°/s. Stiffness trials consisted of 2 conditions: a nonstartle trial and an acoustic-startle trial. The nonstartle trial involved a picture presentation delivered 800 milliseconds before the perturbation, whereas the acoustic-startle trial used a startle noise (100 dB for 10 milliseconds) supplied through headphones, which was delivered 100 milliseconds before the perturbation via the customized LabVIEW program.[1] Participants underwent 2 nonstartle trials and 1 acoustic-startle trial for each category of the selected block, which was not previously used, whereas other pictures were displayed for 6 seconds without the perturbation. The orders of stiffness trials and picture selections were randomized to avoid a learning effect.

Signal Processing

For the cerebral cortex fear responses, after ocular artifact reduction, only artifact-free EEG trials synchronized with picture onset and heart rate were cut into 4000-millisecond epochs from 2000 milliseconds before to 2000 milliseconds after picture onset. Averaged event-related desynchronization/synchronization in the θ frequency band (4–8 Hz) during the first 1000 milliseconds of picture presentation (EEG, 0 to 1000 milliseconds from the picture presentation) compared with baseline (–2000 to –1000 milliseconds before picture onset) was calculated for selected electrodes of the frontal (F3, Fz, F4) and parietal (P3, Pz, P4) cortices (Table 2). Positive values reflect a decreased percentage of θ power (event-related desynchronization), indicating less attention, whereas negative values represent an increased percentage of θ power (event-related synchronization), indicating more mental effort.

Table 2. Abbreviation Key

Abbreviation	Definition
θ Power	A neural oscillatory pattern with a frequency range of 4–8 Hz
EEG	Electroencephalography: an electrophysiological method for monitoring and recording electrical activity of the brain
ERS	Event-related synchronization: % increased electrocortical power relative to nonloading baseline period
ERD	Event-related desynchronization: % decreased electrocortical power relative to nonloading baseline period
F3, Fz, F4, P3, Pz, P4	Electrode locations: Odd numbers are located in the left hemisphere, and even numbers are located in the right hemisphere. F represents the frontal lobe, P reflects the parietal lobe, and z indicates the center. (ie, Fz is the electrode at the centrofrontal lobe).
MHRD	Maximum heart-rate deceleration: changes in heart rate relative to nonevent baseline period

For neurophysiological fear responses to emotionally evocative pictures, interbeat R-wave intervals were detected to the nearest millisecond, and 500-millisecond intervals were calculated for heart rate in beats/min.[20] The maximum heart-rate deceleration was calculated as the difference between the minimum heart rate during the first 3 seconds of picture presentation and the average heart rate of the 3-second baseline.[21] We used each picture's scores for valence, arousal, and the level of fear, which ranged from 1 = *not at all fearful* to 9 = *very fearful*, to determine the level of subjective fear perception of the picture.[22]

For joint stiffness, raw torque and position signals were band-pass filtered at 20 to 400 Hz, rectified, and low-pass filtered at 5 Hz. Stiffness values were calculated as the Δ torque (N·m)/Δ displacement (°) and normalized to body mass (Nm/°/kg). Normalized joint-stiffness values were also corrected for gravity and calculated from 0° to 4° (short range), 0° to 20° (midrange), and 0° to 40° (long range) during knee-flexion perturbations.

Statistical Analysis

The effects of the specific picture type on subjective (valence, arousal, level of fear), neurophysiological (beats/min), and cortical (event-related desynchronization/synchronization) emotional responses were assessed using separate 2-way repeated-measures analyses of variance (ANOVAs) with 1 within-subject factor (category, 3 levels) and 1 between-subjects factor (group, 2 levels). Effects of picture types on stiffness between groups were evaluated by conducting separate 2-way repeated-measures ANOVAs with 1 within-subject factor (type, 3 levels) and 1 between-subjects factor (group, 2 levels) for each stiffness condition. Additionally, the effects of specific picture type and condition on stiffness were determined using separate 2-way repeated-measures ANOVAs with 2 within-subject factors (category, 3 levels; condition, 2 levels) for each

Table 3. Emotional Responses to 3 Picture Types

Group	Picture Type, Mean ± SD (95% Confidence Interval)			Variable(s): *P* Value
	Neutral	Fearful	Sport Knee-Injury Related	
Maximum heart-rate deceleration, beats/min				
Control	−3.67 ± 1.30 (−4.00, −3.34)	−4.62 ± 1.43[d] (−4.97, −4.28)	−4.18 ± 1.40 (−4.54, −3.83)	Group × type: .028
ACLR	−3.37 ± 1.36 (−3.72, −3.02)	−4.58 ± 1.31[d] (−4.94, −4.22)	−4.43 ± 1.46[d] (−4.80, −4.05)	
Total	−3.53 ± 1.33 (−3.76, −3.28)	−4.60 ± 1.37 (−4.85, −4.35)	−4.30 ± 1.43 (−4.57, −4.05)	
Valence[a] (SAM)				
Control	4.99 ± 0.22 (4.92, 5.07)	2.36 ± 0.94 (2.11, 2.62)	4.00 ± 0.69 (3.80, 4.21)	
ACLR	5.07 ± 0.35 (4.99, 5.14)	2.55 ± 1.13 (2.29, 2.82)	3.85 ± 0.92 (3.64, 4.06)	
Total	5.03 ± 0.29 (4.98, 5.08)	2.46 ± 1.04[e,f] (2.27, 2.64)	3.93 ± 0.81[e] (3.78, 4.07)	Type: <.001
Arousal[b] (SAM)				
Control	1.49 ± 0.99 (1.29, 1.68)	5.63 ± 1.96 (5.10, 6.16)	3.29 ± 1.48 (2.86, 3.71)	Group × type: .012
ACLR	1.31 ± 0.46 (1.11, 1.51)	5.50 ± 2.23 (4.96, 6.05)	3.97 ± 1.93[g] (3.54, 4.41)	
Total	1.40 ± 0.78 (1.26, 1.54)	5.57 ± 2.12[e,f] (5.19, 5.95)	3.62 ± 1.74[e] (3.32, 3.94)	Type: <.001
Level of fear[c]				
Control	1.14 ± 0.28 (1.06, 1.23)	5.83 ± 1.92 (5.28, 6.38)	2.92 ± 1.51 (2.49, 3.35)	Group × type: .003
ACLR	1.23 ± 0.38 (1.14, 1.31)	5.63 ± 2.48 (5.07, 6.20)	3.83 ± 1.93[g] (3.39, 4.27)	
Total	1.18 ± 0.33 (1.13, 1.24)	5.73 ± 2.20[e,f] (5.34, 6.13)	3.37 ± 1.78[e] (3.07, 3.69)	Type: <.001

Abbreviations: ACLR, anterior cruciate ligament reconstruction; SAM, the Self-Assessment Manikin, including 2 valence and arousal domains.

[a] Level of happiness ranged from 1 = *very unhappy* to 9 = *very happy*.
[b] Arousal level ranged from 1 = *very calm* to 9 = *very aroused*.
[c] Ranges from 1 = *not fearful at all* to 9 = *very fearful*.
[d] Greater heart-rate deceleration than for the neutral pictures ($P < .05$).
[e] Difference from the neutral pictures ($P < .05$).
[f] Difference from the injury pictures ($P < .05$).
[g] Difference between groups ($P < .05$).

group. We also reported mean differences and associated 95% confidence intervals (CIs) to establish the clinical implications. The α level was set a priori at .05.

RESULTS

Emotional Responses

Significant picture type × group interaction effects were observed for the arousal dimension ($F_{1.631,197.325} = 4.991$, $P = .012$, effect size: $\eta^2 = 0.04$) and level of fear ($F_{1.732,209.590} = 6.353$, $P = .003$, effect size: $\eta^2 = 0.05$; Table 3). Compared with the control group, the ACLR group displayed greater arousal (mean difference = 0.69, 95% CI = 0.08, 1.30; $P = .028$; effect size: d = 0.40) and fear (mean difference = 0.91, 95% CI = 0.29, 1.53; $P = .004$; effect size: d = 0.53) scores in response to injury-related pictures. Also, significant picture-type main effects were noted for the valence ($F_{1.846,223.341} = 490.772$, $P < .001$), arousal ($F_{1.631,197.325} = 368.135$, $P < .001$), and fear ($F_{1.732,209.590} = 360.603$, $P < .001$) components. Neutral pictures resulted in higher valence scores than both the fearful and injury-related picture types (mean difference with fearful pictures = 2.57, 95% CI = 2.34, 2.80; effect size: d = 3.37; mean difference with injury-related pictures = 1.11, 95% CI = 0.92, 1.29; effect size: d = 1.81; $P < .001$). Injury-related pictures also produced higher valence scores than fearful pictures (mean difference = 1.47, 95% CI = 1.29, 1.65; $P < .001$; effect size: d = 1.58). Conversely, both arousal dimension (fearful pictures mean = 5.57 ± 2.12, 95% CI = 5.19, 5.95; injury-related pictures mean = 3.63 ± 1.74, 95% CI = 3.32, 3.94; neutral pictures mean = 1.40 ± 0.78, 95% CI = 1.26, 1.54; effect size: $\eta^2 = 0.75$) and level of fear (fearful pictures mean = 5.73 ± 2.20, 95% CI = 5.34, 6.13; injury-related pictures mean = 3.37 ± 1.78, 95% CI = 3.07, 3.69; neutral pictures mean = 1.18 ± 0.33, 95% CI = 1.13, 1.24; effect size: $\eta^2 = 0.77$) showed differences among all picture types ($P < .001$): fearful pictures produced the highest score and neutral pictures produced the lowest score.

A significant picture type × group interaction effect was observed for maximum heart-rate deceleration ($F_{2,236} = 3.236$, $P = .028$, effect size: $\eta^2 = 0.03$; Table 3). The control group had greater heart-rate deceleration with the fearful than neutral pictures (mean difference = –0.96, 95% CI = –1.52, –0.40; $P < .001$; effect size: d = –0.70), whereas the ACLR group's heart rate decreased more with both the fearful (mean difference = –1.21, 95% CI = –1.82, –0.60; $P < .001$; effect size: d = –0.91) and injury-related (mean difference = –1.06, 95% CI = –1.67, –0.45; $P < .001$; effect size: d = –0.75) pictures compared with the neutral pictures (Figure 3).

Significant picture-type main effects in electrocortical activity were found for the F3 ($F_{2,76} = 3.762$, $P = .028$, effect size: $\eta^2 = 0.09$), Fz ($F_{1.625,61.763} = 3.470$, $P = .046$, effect size: $\eta^2 = 0.08$), P3 ($F_{1.674,63.622} = 23.975$, $P < .001$, effect size: $\eta^2 = 0.39$), Pz ($F_{1.826,69.383} = 24.043$, $P < .001$, effect size: $\eta^2 = 0.39$), and P4 ($F_{1.532,58.228} = 26.662$, $P < .001$, effect size: $\eta^2 = 0.41$) locations (Table 4). Pairwise comparisons revealed greater θ event-related synchronization with fearful versus neutral pictures for F3 (mean difference = –13.14, 95% CI = –22.85, –3.42; $P = .005$; effect size: d = –0.43), Fz (mean difference = –11.14, 95% CI = –18.77, –3.51; $P = .002$; effect size: d = –0.38), P3 (mean difference = –14.66, 95% CI = –26.49, –2.82; $P =$

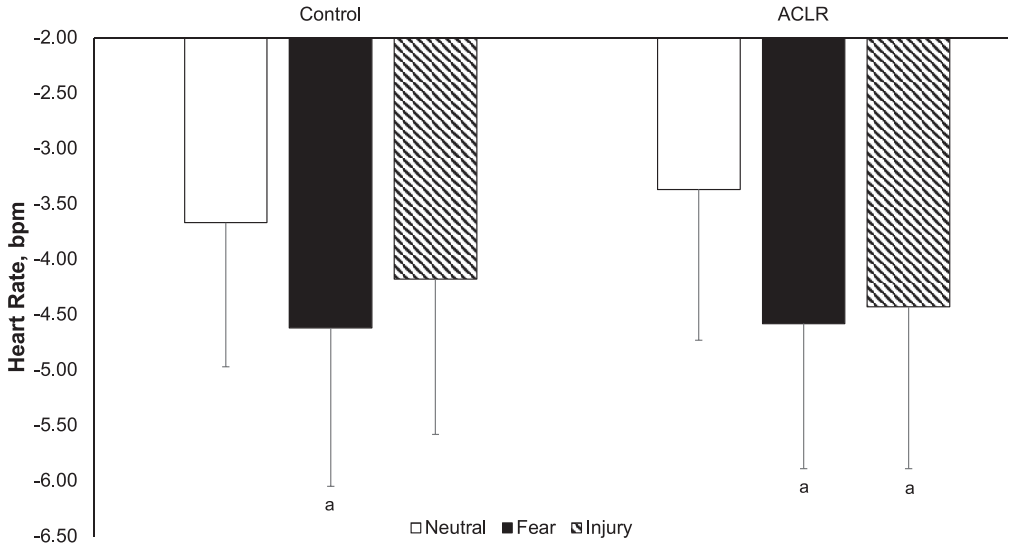

Figure 3. Maximum heart-rate deceleration between groups across picture types. Control indicates healthy controls; neutral, neutral pictures; fear, fearful pictures; injury, sports knee-injury–related pictures. Abbreviation: ACLR, anterior cruciate ligament reconstructed patients. [a] Greater heart rate deceleration than neutral ($P < .05$).

.011; effect size: d = –0.38), and Pz (mean difference = –17.27, 95% CI = –32.72, –1.82, $P = .024$; effect size: d = –0.28). Specific knee-injury–related pictures showed greater θ event-related synchronization compared with both neutral and fearful pictures in the P3 ($P < .001$, mean difference with neutral pictures = –46.18, 95% CI = –65.47, –26.89; effect size: d = –0.90; mean difference with fearful pictures = –31.52, 95% CI = –50.55, –12.49; effect size: d = –0.61), Pz (mean difference with neutral pictures = –52.80, 95% CI = –75.38, –30.22; effect size: d = –0.75; mean difference with fearful pictures = –35.53, 95% CI = –55.18, –15.89; $P < .001$, effect size: d = –0.47), and P4 (mean difference with neutral pictures = –63.89, 95% CI = –92.59, –35.19; effect size: d = –0.92; mean difference with fearful pictures = –54.70, 95% CI = –79.71, –29.70; $P < .001$; effect size: d = –0.77) locations (Figure 4).

Joint Stiffness

The ACLR group showed significant type × condition interaction effects for midrange ($F_{2,34} = 6.659$, $P = .004$, effect size: $\eta^2 = 0.28$) and long-range ($F_{2,36} = 4.499$, $P =$

Table 4. Electrocortical Responses to Emotional Evocative Pictures

	Theta (4–8 Hz) ERD/ERS, %[a]					
	Frontal Cortex Area			Parietal Cortex Area		
Pictures Group	F3	Fz	F4	P3	Pz	P4
Neutral						
Control	−25.47 ± 30.82	−27.36 ± 30.62	−33.25 ± 30.27	−29.92 ± 31.49	−62.87 ± 63.51	−56.00 ± 48.86
ACLR	−22.64 ± 26.78	−22.19 ± 29.30	−27.37 ± 31.57	−35.32 ± 43.33	−65.45 ± 45.95	−63.64 ± 39.92
Total	−24.05 ± 28.54	−24.78 ± 29.69	−30.31 ± 30.67	−32.62 ± 37.49	−64.16 ± 54.73	−59.82 ± 44.21
95% CI	−33.30, −14.81	−34.37, −15.18	−40.21, −20.41	−44.74, −20.50	−81.90, −46.41	−74.10, −45.54
Fearful						
Control	−34.39 ± 27.15	−37.25 ± 29.74	−30.76 ± 24.71	−38.09 ± 34.96	−70.17 ± 72.62	−54.77 ± 50.41
ACLR	−39.99 ± 37.91	−34.58 ± 29.79	−37.38 ± 37.95	−56.46 ± 41.28	−92.67 ± 60.12	−83.25 ± 42.21
Total	−37.19 ± 32.67[b]	−35.92 ± 29.41[b]	−34.07 ± 31.79	−47.28 ± 38.89[b]	−81.42 ± 66.78[b]	−69.01 ± 48.11
95% CI	−47.75, −26.64	−45.44, −26.39	−44.32, −23.82	−59.52, −35.03	−102.76, −60.09	−83.89, −54.13
Knee-injury related						
Control	−27.00 ± 26.00	−33.04 ± 30.87	−31.69 ± 29.21	−75.40 ± 50.34	−111.82 ± 95.61	−107.96 ± 75.65
ACLR	−29.64 ± 19.14	−35.06 ± 16.04	−42.80 ± 40.63	−82.20 ± 72.84	−122.09 ± 71.40	−139.46 ± 95.51
Total	−28.32 ± 22.58	−34.05 ± 24.30	−37.24 ± 35.38	−78.80 ± 61.90[b,c]	−116.96 ± 83.45[b,c]	−123.71 ± 86.53[b,c]
95% CI	−35.63, −21.01	−41.92, −26.18	−48.57, −25.82	−98.84, −58.75	−143.97, −89.95	−151.28, −96.13
P value	.028	.046	.339	<.001	<.001	<.001

Abbreviations: ACLR, anterior cruciate ligament reconstruction; CI, confidence interval; ERD, event-related desynchronization (% decreased power relative to nonloading baseline, positive [+]); ERS, event-related synchronization (% increased power relative to nonloading baseline, negative [–]).

[a] Values are mean ± SD except for the rows labeled "95% CI" and "*P* Value."
[b] Greater θ ERS than for the neutral pictures ($P < .05$).
[c] Greater θ ERS than for the fearful pictures ($P < .05$).

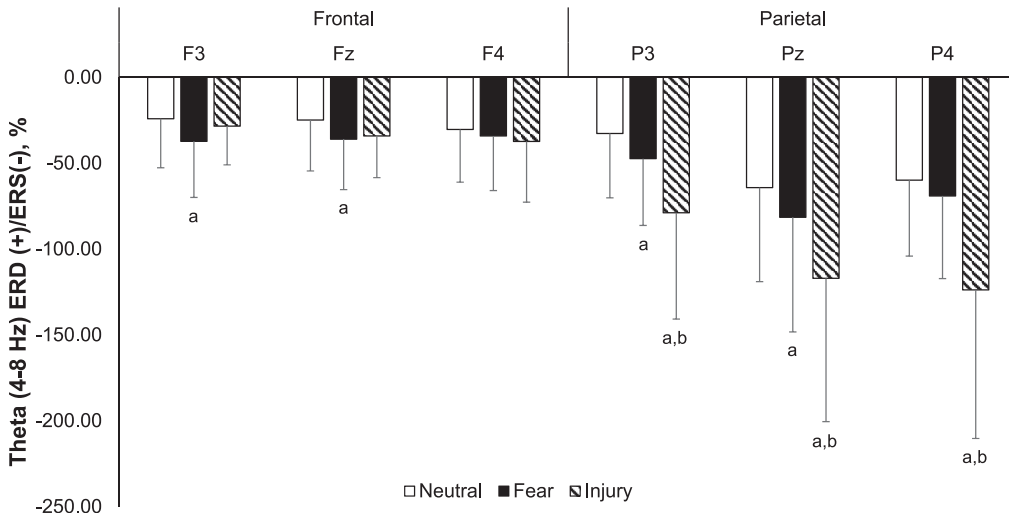

Figure 4. Frontoparietal ERD/ERS in the θ frequency (4–8 Hz) across picture types during first second of picture presentation. Neutral indicates neutral pictures; fear indicates fearful pictures; injury, sports knee-injury–related pictures. Abbreviations: ERD, event-related desynchronization (% decreased power relative to nonloading baseline, positive [+]); ERS, event-related synchronization (% increased power relative to nonloading baseline, negative [–]). [a] Greater θ ERS than neutral ($P < .05$). [b] Significantly greater θ ERS than fear ($P < .05$).

.018, effect size: $\eta^2 = 0.20$) stiffness (Table 5 and Figure 5). When an acoustic noise was delivered before the perturbation in the ACLR group, both fearful and injury-related pictures created greater midrange stiffness values (mean difference with fearful pictures = 0.010, 95% CI = 0.001, 0.018, $P = .024$; effect size: d = 0.58; mean difference with injury-related pictures = 0.013, 95% CI = 0.003, 0.023; $P = .017$; effect size: d = 0.56) than neutral pictures, but no difference occurred between the fearful and injury-related pictures (mean difference = –0.001, 95% CI = –0.009, 0.007, $P = .751$; effect size: d = –0.05). The ACLR group also had increased long-range stiffness values under the acoustic-startle condition in response to both the fearful (mean difference = 0.010, 95% CI = 0.002, 0.017; $P = .014$; effect size: d = 0.69) and injury-related (mean difference = 0.006, 95% CI = 0.001, 0.011; $P = .031$; effect size: d = 0.35) pictures versus the nonstartle condition, whereas no changes were present in response to the neutral pictures (mean difference = –0.002, 95% CI = –0.010, 0.006; $P = .633$; effect size: d = –0.13).

DISCUSSION

Our primary findings were that the emotional stimuli (neutral, fearful, and injury-related pictures) provoked different electrocortical and neurophysiological activation and fear perceptions. Furthermore, negative emotional pictures (fearful or knee-injury–related pictures) altered joint stiffness in the ACLR group, particularly when unanticipated acoustic stimuli were delivered before the knee-flexion perturbation. To our knowledge, we are the first to provide evidence of neuromechanical coupling between an emotion, such as fear, and joint-stiffness regulation strategies that are critical to dynamic restraint

Table 5. Normalized Joint-Stiffness Values (Nm/°/kg) in Response to Emotionally Evocative Pictures (Mean ± SD)

Group Range	Condition	Pictures			Variable(s): *P* Value
		Neutral	Fearful	Knee-Injury Related	
Control					
Short (0°–4°)	Nonstartle[a]	0.054 ± 0.006	0.052 ± 0.006	0.052 ± 0.006	
	Startle[b]	0.057 ± 0.010[c]	0.055 ± 0.009[c]	0.058 ± 0.012[c]	Condition: .013
Mid (0°–20°)	Nonstartle[a]	0.008 ± 0.004	0.009 ± 0.006	0.009 ± 0.005	
	Startle[b]	0.022 ± 0.012[c]	0.031 ± 0.022[c]	0.025 ± 0.019[c]	Condition: .001
Long (0°–40°)	Nonstartle[a]	0.041 ± 0.010	0.039 ± 0.013	0.040 ± 0.011	
	Startle[b]	0.048 ± 0.010[c]	0.049 ± 0.016[c]	0.045 ± 0.013[c]	Condition: .003
Anterior cruciate ligament reconstruction					
Short (0°–4°)	Nonstartle[a]	0.049 ± 0.009	0.049 ± 0.010	0.050 ± 0.010	
	Startle[b]	0.054 ± 0.014	0.048 ± 0.011	0.052 ± 0.014	
Mid (0°–20°)	Nonstartle[a]	0.009 ± 0.008	0.008 ± 0.006	0.007 ± 0.007	Condition × type: .004
	Startle[b]	0.017 ± 0.014[c]	0.027 ± 0.020[c,d]	0.030 ± 0.025[c,d]	Condition: .001
Long (0°–40°)	Nonstartle[a]	0.045 ± 0.014	0.039 ± 0.016	0.040 ± 0.015	Condition × type: .018
	Startle[b]	0.043 ± 0.016	0.050 ± 0.016[d]	0.046 ± 0.019[d]	

[a] A 40° knee-flexion perturbation at 800 milliseconds after presentation of picture.
[b] An acoustic sound at 100 milliseconds before the perturbation.
[c] Difference between stiffness conditions ($P < .05$).
[d] Difference from neutral pictures ($P < .05$).

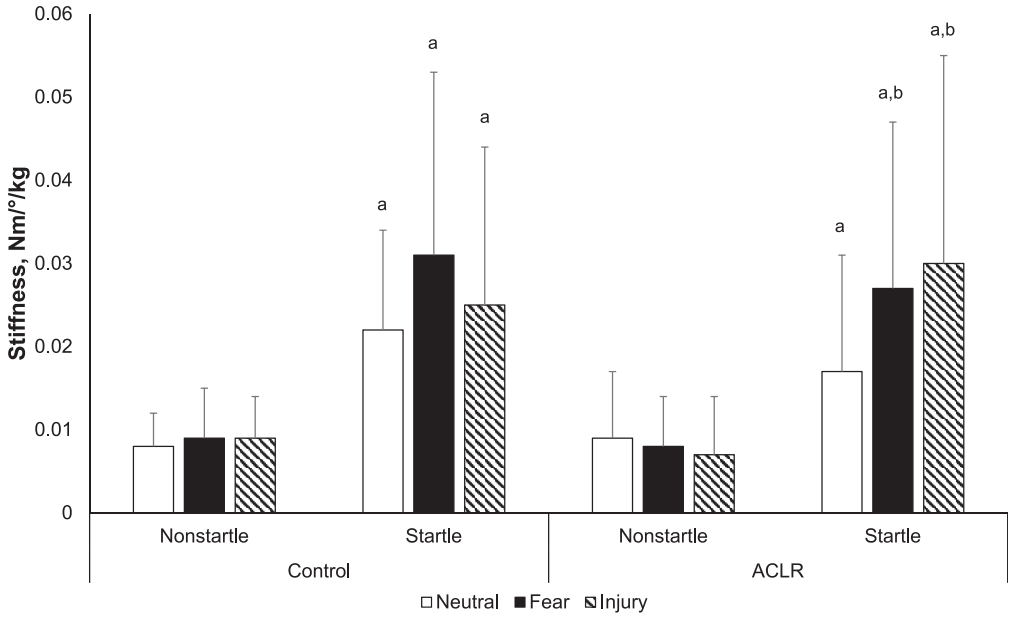

Figure 5. Normalized midrange stiffness (0°–20°) between groups by picture types. Control indicates healthy controls; neutral, neutral pictures; fear, fearful pictures; injury, sport knee-injury–related pictures. Nonstartle: A 40° knee-flexion perturbation at 800 milliseconds after picture presentation. Startle: An acoustic sound at 100 milliseconds before the perturbation. Abbreviation: ACLR, anterior cruciate ligament reconstruction. [a] Difference between stiffness conditions ($P < .05$). [b] Difference from neutral ($P < .05$).

and functional joint stability. Our results indicated that negative emotional visual stimuli altered neurocognitive processing and that this effect was different in the ACLR group. Furthermore, these negative emotional stimuli appeared to exacerbate the ACLR group's neuromuscular responses to sudden, unanticipated events such as a knee perturbation, which led to momentary stiffness dysregulation and the potential loss of functional joint stability.

Emotional Responses: Subjective, Neurophysiological, and Electrocortical Responses

The IAPS is one of the tools used most commonly to induce a variety of emotional responses via visual stimuli such as neutral, pleasant, and unpleasant pictures.[15] Different types of emotions have been evaluated in 2 subjective domains: valence and arousal.[22] Our results demonstrated that fearful and injury-related pictures resulted in lower valence (ie, more sadness), higher arousal values, and increased fear compared with neutral pictures; the effect was greater in patients after ACLR. These findings support previous research[18] that showed individuals produced even greater negative feelings in response to fearful and traumatic knee-injury–related pictures.

Emotional stimuli, in general, alter cardiovascular reaction and electrocortical activation. These physiological and neurologic responses are known to reflect homeostatic emotional regulation in the CNS.[10] Investigators[23] who used the IAPS suggested that unpleasant pictures provoked greater, rapid heart-rate deceleration and electrocortical activation in the frontal and parietal cortex areas than neutral stimuli. The initial decrease in heart rate is primarily associated with the parasympathetic nervous system, which quickly suppresses the targeted cardiac outputs.[20,23] This neurophysiological inhibition is concerned with early defensive behavior by promoting neural processing of aversive visual stimuli.[21] In our study, fearful pictures also caused greater heart-rate deceleration than neutral pictures during the early picture presentations, which supports previous results.

These cardiac responses to emotional stimuli have also been associated with neural activation in the CNS.[24] Fearful stimuli can increase cortical activation in the prefrontal cortex; such heightened prefrontal cortex activity implies increased cognitive processing is required to sufficiently regulate fearful stimuli.[25,26] Our EEG data indicated that fearful and injury-related pictures increased the θ frequency band power in the frontal and parietal regions during the first second compared with neutral pictures. Furthermore, the injury-related pictures caused not only increased θ power in the parietal cortex regions versus the neutral pictures but even more activation than the fearful pictures. Although the θ power in the frontal areas is known to be associated with cognitive fear-regulation processing, parietal θ activation is thought to be linked to situational awareness of visual cues.[24–27] Moreover, sport knee-injury–related pictures induced greater heart-rate deceleration and parietal θ power in the ACLR group than the general fear-related pictures. This population-specific response to unpleasant visual scenes, related to previous traumatic experiences, may exacerbate emotional responses, as vigorous negative stimuli can facilitate defensive behavior processing through the parasympathetic nervous system, as well as cortical activation related to cognitive emotional-regulation management.[23] These data imply that both general and specific situational fearful stimuli may elicit potent cortical activation in the frontal and parietal cortex areas, in addition to greater heart-rate deceleration, which may indicate increased internal cognitive-processing demands on the fear network.[22] Because the frontal and parietal cortex areas are also crucial for cognitive processing related to task-specific muscle coordination,[14] certain visual cues, such as injury-related or fearful pictures,

disrupt a person's situational awareness simply because they immediately occupy his or her attention, which may demand important cognitive resources or delay reactions to other critical events. Therefore, in patients after ACLR, vigorous negative emotional stimuli during dynamic movements may disrupt the normal cognitive motor planning needed for sufficient regulation of neuromuscular control. Although minimum clinical differences for subjective, neurophysiological, and electrocortical measurements have not yet been determined, our findings demonstrating 95% CIs of mean differences with effect-size values for outcomes displayed significant effects of negative stimuli on emotional responses, pointing to the need for clinicians to consider psychological factors during ACLR rehabilitation of patients with a greater fear of reinjury who experience diminished knee function.[9]

Fear and Stiffness Dysregulation

Task-specific neuromuscular-control strategies are critical for protecting the knee during rapid and intense physical activities because passive joint structures alone may not sufficiently maintain joint stability.[2] Therefore, after ACLR, patients must be able to appropriately prepare for and react to external loading by regulating muscle contractions surrounding the knee in order to absorb high forces and prevent excessive strain on the ACL.[2,6] However, because up to 80% of all ACL injuries occur from noncontact mechanisms, a cognitive concern may account for the failure to anticipate or the adverse reaction to sudden perturbations, which could lead to ACL tears.[13] These preparatory (feed-forward) and reactive (feedback) joint-stiffness regulation strategies are controlled by the CNS, as the brain must simultaneously predict oncoming loads and monitor afferent proprioceptive inputs to optimize the task-specific level of joint stiffness needed for both stability and performance.[2,5] Joint-stiffness regulation strategies in response to rapid joint loading have been measured to explore neuromechanical coupling and learn how altered dynamic-restraint mechanisms may predispose individuals to a heightened risk of peripheral ligamentous injury.[1,4] Previous authors[1] found the startle response during the brief preparatory period before knee perturbations altered joint-stiffness and muscle-contraction patterns. Because the startle condition replicates unanticipated events, this may imply that cognitive motor planning is interrupted and causes joint-stiffness dysregulation during sudden, high-velocity athletic maneuvers. Several regions of the brain that are critical sites for motor planning also mediate emotional responses.[1,10,11] However, no researchers have investigated the effects of negative emotion on knee-stiffness regulation strategies.

In our study, the startle condition increased midrange and long-range stiffness in both the ACLR group and healthy control participants, whereas short-range stiffness values increased only in the latter, regardless of the type of emotional stimuli. In general, short-range stiffness is concerned with passive mechanical resistance, provided mainly by the involuntary reversal of existing cross-bridges within the muscle fibers during a brief period after load onset.[28] Long-range stiffness represents the continuous voluntary eccentric contraction of muscles throughout the knee-excursion range of motion during knee perturbations.[28] As increased internal tension on the ACL occurs between nearly full extension and 45° of knee flexion and can damage ligaments,[3] we also employed midrange stiffness (30°–50° of knee flexion), which may include not only passive contractile components but reflexive muscular contractions in addition.[28] Our increased stiffness values with respect to the acoustic startle support earlier findings[1,8] that suggest an unanticipated event can disturb neuromuscular control, possibly due to the sudden attentional demands. This may compromise the cognitive processing associated with both feed-forward and feedback neural circuits in the brain.[1,8]

The CNS can quickly detect negative stimuli that initiate early, strong, and prolonged cortical activation in the fear network between the prefrontal and parietal cortices.[29] An ACL rupture may cause neural adaptations in the CNS responsible for perceiving proprioceptive inputs, as well as goal-directed motor behavior.[7,8] This may indicate that the increased cerebral cortex activity, as a result of a fearful stimulus, may limit the available neural resources needed for optimal joint-stiffness regulation strategies. This also agrees with previous research[30] demonstrating that several types of cognitive loading altered knee-joint stiffness regulation strategies. Our ACLR group had increased midrange and long-range stiffness during an unanticipated startle condition in response to both fearful and injury-related pictures, with greatly increased cortical activation in the frontal and parietal cortices. Although both fearful and injury-related pictures also increased frontoparietal cortical activation in the healthy control participants, no stiffness differences were observed for the 3 picture types. Because emotion-provoking pictures were presented 700 milliseconds before the acoustic stimulus, healthy participants may have been able to stiffen the knee joint, regardless of picture type. However, the combined negative stimuli and possibly reorganized sensorimotor system after an ACL injury may have exceeded the neural capability of goal-directed motor behavior in patients. Our findings with the 95% CIs for joint stiffness in response to both fearful and injury-related pictures provide clinical evidence that the increased neural demands produced by noxious visual cues may impair preparatory or reactive (or both) dynamic-restraint mechanisms and ultimately lead to joint-stiffness dysregulation.[8,27]

Limitations and Future Directions

This study had several limitations. First, to induce a variety of emotions, we used the IAPS, which is common in psychological investigations and evaluated using 2 subjective valence and arousal domains in addition to a 9-point Likert scale, heart-rate changes, and electrocortical responses measured through EEG. Although 60 neutral and fearful pictures were included based on previous norm value ranges of valence and arousal domains, we also included 60 sport knee-injury–related pictures chosen from an online search. The sport-injury pictures resulted in significant negative effects compared with the neutral pictures, but these were not as strong as general fearful pictures. Second, this was the first study, to our knowledge, to examine emotional responses in patients after ACLR compared with healthy control

participants. Although the differences in emotional responses between neutral and fearful contents have been well defined in psychological studies,[15,18,22] minimum clinical differences for these measures, particularly in patients after ACLR, have not yet been established. Our results suggest the need to determine the validity and reliability of these measures in order to determine clinically meaningful ranges. Furthermore, the neurophysiological and electrocortical emotional responses were not directly accessed during joint-stiffness regulation testing due to movement and wire artifact, which could have altered heart-rate and EEG data. Future researchers may investigate real-time measures of these emotional responses during measures of joint-stiffness regulation testing. Additionally, the orders of emotion types and stiffness condition were randomized to reduce practice effects, and the picture presentation and acoustic startle were provided at 800 and 100 milliseconds, respectively, before the perturbation. This may have allowed participants to anticipate these events. Future authors may provide random picture onsets and timings of the acoustic startle to minimize participants' anticipation.

CONCLUSIONS

This study is the first, to our knowledge, to demonstrate that negative emotional stimuli, in response to a sudden event (ie, acoustic startle) that disrupted the anticipation of joint loading, interfered with goal-directed cognitive motor-planning strategies. Such disrupted neurocognitive processing may be insufficient to prepare for and react to unanticipated, high-velocity movement tasks. The resulting knee-stiffness dysregulation may fail to maintain task-specific functional joint-stability demands, thereby placing the ACL in a vulnerable state. The adverse effects of fear on neuromuscular control may indicate that psychological interventions should be incorporated into neuromuscular-control exercise programs after ACL injury to minimize risk and optimize patient outcomes.

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