Pain Catastrophizing and EEG-Alpha Asymmetry

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Running title: Catastrophizing and alpha asymmetry
Abstract

Objectives: Pain catastrophizing is thought to play a causal role in the development and maintenance of chronic pain and its negative impact on functioning. However, few studies have examined the factors that might contribute to the development and maintenance of catastrophizing. The anterior asymmetry and emotion (AAE) model hypothesizes that more activity in left anterior brain regions is associated with a tendency to engage in approach responses (often, but not always, associated with positive valance), and that more right anterior activity is associated with a tendency to engage in more withdrawal responses (often associated with negative valance). Given the consistent associations found between catastrophizing and both (1) approach vs avoidance pain coping styles and (2) affective responses to pain, the AAE model would predict that more left (versus right) anterior brain activity would prospectively predict future catastrophizing.

Methods: Anterior asymmetry measures computed using electroencephalogram data from 30 individuals with spinal cord injury were correlated with catastrophizing scores obtained two years after the EEG recording.

Results: Consistent with the AAE model, anterior asymmetry scores reflecting greater left than right anterior activity were negatively associated with subsequent catastrophizing.

Conclusions: The study findings identify a biological factor that may be associated with greater vulnerability to pain-related catastrophizing. If replicated in future research, the findings suggest new possibilities for treating catastrophizing, which may then contribute to improved pain treatment outcomes.

Keywords: pain catastrophizing, spinal cord injury, chronic pain, electroencephalography, EEG asymmetry, alpha-wave bandwidth
Introduction

Chronic pain is a significant problem worldwide that is costly for society and contributes to significant suffering and disability for many individuals\(^1^,\)\(^2\), including individuals with spinal cord injury\(^3^,\)\(^4\). To help address this problem, a variety of psychosocial treatments – many of which fall under the general rubric of cognitive-behavioral therapy (CBT) – have been developed that teach and encourage the use of strategies for more effectively managing chronic pain and its impact\(^5^\)\(^-\)\(^7\). However, even though CBT treatments have demonstrated efficacy for reducing pain and improving psychological and physical functioning for some individuals, not everyone benefits from these treatments, and even among those who benefit in the short term, not everyone is able to maintain treatment gains\(^5\).

The variability in patient response to CBT treatments strongly suggests the presence of mediators (factors that explain how treatment works) and moderators (factors that predict who treatment works for) as impacting outcome. Perhaps the factor with the strongest evidence for playing both of these roles is pain-related catastrophizing. Catastrophizing represents a tendency to focus on and exaggerate the threat value of pain\(^8^,\)\(^9\), and is consistently associated with poor outcomes, such as depression, anxiety, use of maladaptive (i.e., avoidance) coping responses. It’s role as a mechanism of psychosocial treatment outcome has been supported by research demonstrating that (1) those who evidence the best outcomes with psychosocial pain treatments are those who report the greatest pre- to post-treatment decreases in catastrophizing\(^10^\)\(^-\)\(^13\) and (2) early-treatment decreases in catastrophizing prospectively predict late-treatment improvements in outcome measures\(^14^,\)\(^15\). The role of catastrophizing as a moderator of treatment outcome is supported by research demonstrating that higher pre-treatment catastrophizing predicts poor treatment response across a variety of chronic pain treatments (e.g.,\(^16^\)\(^-\)\(^19\)).
Given the strong evidence indicating that catastrophizing is important to positive outcomes in individuals with chronic pain as well as the growing evidence supporting a role for catastrophizing as a mediator and moderator of pain treatment outcomes, it would be useful to better understand the factors that contribute to the development and maintenance of catastrophizing. To the extent that these factors are modifiable, this knowledge could inform the development of innovative treatments that could influence those factors, and potentially increase the number of individuals who respond to psychosocial pain interventions.

Evidence from a number of sources suggests the possibility that one modifiable factor influencing catastrophizing is anterior brain asymmetry; that is, the relative differences in activation measured from left versus right anterior brain regions. A great deal of research supports the existence of distinct neurophysiological systems underlying both (1) behavioral engagement (most often associated with optimism and positive affect, but sometimes with anger, labeled a Behavioral Activation System or BAS;\textsuperscript{20,21} and (2) behavioral disengagement (most often associated with negative affect, including both sadness/depression and fear, labeled a Behavioral Inhibition System or BIS;\textsuperscript{20,21}). Although the neurophysiological substrates of these two systems lie in many places in the cortex and deeper structures, neurophysiological circuits in the frontal cortex are essential to their operation (e.g.,\textsuperscript{21}). Importantly, for the purposes of this study, research from a variety of sources, including studies of individuals with brain lesions, suggest that more neuron assemblies devoted to BIS operate in the anterior right hemisphere, and a more neuron assemblies devoted to BAS operate in the anterior left hemisphere\textsuperscript{22,23}. Thus, the relative differences in the activity measured in left anterior regions versus right anterior regions (i.e., anterior asymmetry) may serve as a biological marker or proxy measure of the relative engagement of the BAS versus BIS systems\textsuperscript{24}. 

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The preponderance of research studies that has examined the correlates of anterior asymmetry has used activity in the alpha bandwidth (8 – 13 Hz) as an indication of a lack of activity in anterior brain regions, given that the presence of alpha oscillations has been shown to be inversely related to brain activation\textsuperscript{25,26}. Moreover, a substantial body of research indicates that measures of frontal alpha asymmetry (FAA; indicating relative less activity in left versus frontal brain regions) are highly internally consistent moderately stable across time, although the reliability of FAA estimates can be improved by averaging multiple measures obtained on different occasions\textsuperscript{24,27-32}. Thus, FAA is thought to have trait-like qualities, although there is evidence (see Discussion) that a number of neuromodulatory treatments can alter measures of FAA.

Consistent with a conclusion that FAA is at least moderately stable, measures of FAA have been shown to prospectively predict future emotional and behavioral responses consistent with FAA reflecting a biological substrate of BIS and BAS systems. For example, one study has shown that FAA assessed in infants was significantly associated with behavioral inhibition assessed in 4-year old toddlers over 3 years after the initial assessment\textsuperscript{33}. Similarly, FAA asymmetry measures obtained in infants who were 10 and 24 months old prospectively predicted mother-rated toddler internalizing and externalizing behaviors when the children were 30 months old\textsuperscript{34}.

To the extent that catastrophizing reflects a component of the BIS system – that is, a cognitive and emotional response associated with negative mood and a decision to become inactive and negative valance\textsuperscript{35-37}, and to the extent that greater right frontal activity and less left frontal activity reflects brain states consistent with a greater risk for passive/catastrophizing responses, an AAE model of catastrophizing would predict that FAA assessed at one point in
time in individuals with chronic pain would prospectively predict later catastrophizing. In order to test this hypothesis, we administered a measure of pain-related catastrophizing to a group of individuals who had participated two years earlier in a study to examine the effects of different neuromodulatory treatments and pain, and for whom we had multiple measures of electroencephalograph (EEG) data, from which reliable FAA measures could be obtained.  

Methods

Participants

The study participants were recruited from a pool of 30 individuals with SCI and chronic pain who had participated in a study examining the effects of a single session of four neuromodulatory pain treatments and a control treatment (sham tDCS) on EEG activity and pain. In this study, EEG data were obtained before and after each of the five procedures, which were provided to the participants at least one week apart. We were able to contact 29 of these individuals approximately two years after their participation in the previous study, and invited them to respond to the 13 items of the Pain Catastrophizing Scale (described in the Measures section). The study procedures were approved by the University of Washington Institutional Review board, and all participants provided oral consent to participate in this additional study.

Measures

Participant demographic and descriptive data. All participants were asked to provide basic demographic information (age, sex, ethnicity, education level, and marital status) for descriptive purposes. They were also seen by the study physician to diagnose pain type (as primarily neuropathic, nociceptive, or mixed) and rate their level of impairment using the American Spinal Injury Association (ASIA) impairment scale.
Pain-Related Catastrophizing. Pain-related catastrophizing was measured using the 13-item Pain Catastrophizing Scale, which assesses three domains of pain-related catastrophizing: rumination, magnification, and helplessness (PCS; \(^{41}\)). Respondents indicate the frequency with which they experience each catastrophizing thought on a 0 (“Not at all”) to 4 (“All the time”) Likert scale. As indicated previously, the PCS items were administered approximately two years after the EEG data were obtained. The PCS scales evidence a great deal reliability and validity via their association with other pain-relevant criterion measures \(^{41}\). Each of the PCS scales evidenced excellent reliability in the current sample; Cronbach’s alphas were .89, .87, .87, and .92 for the Rumination, Helplessness, Magnification, and Total scales, respectively.

EEG Recording and EEG Data Reduction. EEG data were obtained during 10-minute eyes-closed sessions on 10 occasions, once before and once again after each of the pain treatment procedures provided in the original study.\(^{38}\) However, only the pre-procedure EEG data are used in the current analyses, given the evidence that each of the procedures had an immediate impact on EEG measures.\(^{38}\) Before each of the EEG assessments, the participant’s forehead and earlobes were prepped with Nuprep (Weaver and Company, Aurora, CO). Electrode placement was accomplished using an electrode cap with pre-measured sites using the international 10/20 system.\(^{42}\) The electrode sites were then filled with Electrogel (Electro-Cap International, Eaton, OH), and prepped to ensure impedance values between 3 and 5 Kohms between each site and each ear individually, as well as between the ears. EEG data were then acquired using the WinEEG (Mitsar, St. Petersburg, Russia) software, utilizing 19 electrodes referenced to A1 and A2 (linked ear montage). Signals were amplified using a bandpass of 0.3–70 Hz and sampled at the rate of 250 Hz. Participants were monitored throughout the recording to ensure that they remained awake, and were asked to engage in standardized cognitive task during the EEG
(specifically, to "recall" a beach scene shown to them prior to the session and asking them to keep it in mind during the eyes closed task) to help control for cognitive activity that might affect the EEG measures.

Data from the EEG assessments were exported to the EureKa! Data management software and then re-montaged to the average reference montage. The data were plotted and inspected for potential artifacts (e.g., evidence of eye blinks, eye movements, body movements); entire epochs were removed if one or more channels exhibited presence of artifact. EEG spectrum was calculated from the first 2 minutes of artifact-free data with Fast Fourier Transform (FFT) using 4-second epochs with 1/32 seconds of overlapping window advancement factor. We then computed absolute and relative EEG power for the alpha bandwidth (8 – 13 Hz), and used these power estimates for all subsequent analyses.

Although FAA measures are highly reliable within a single EEG session, test-retest stability of FAA measures tend to be lower over the course of weeks and months. This lack of high test-retest reliability of FAA measures obtained from a single EEG assessment is likely due to the fact that a number of factors that can influence alpha power and asymmetry, such as time of day and montage used. Evidence indicates, fortunately, that it is possible to increase reliability by combining FAA scores from multiple individual EEG sessions into composite scores. Therefore, to ensure adequate reliability of the FAA measure used in this study, we computed a composite FAA score by averaging five FAA scores obtained from different EEG sessions that occurred at least a week apart from each other. To accomplish this, we first computed FAA scores for each of five EEG assessments (i.e., EEG assessments performed prior to each of the study procedures; post-procedure EEG were not used, given our finding that the procedures had short-term effects EEG) between three of pairs of frontal sites (FP2 – FP1, F8
– F7, F4 – F3). Prior to computing the difference scores, the power density scores were log-transformed to normalize their distribution. We then averaged the five individual FAA measures into a single composite score representing the average degree of FAA obtained over time for each pair of electrodes. A positive FAA value reflects more right hemisphere power in the alpha bandwidth (i.e., more relative right inhibition and therefore more relative left activation, hypothesized here to be associated negatively with catastrophizing scores).

**Data analyses**

We first computed descriptive statistics (ranges and means for all continuous variables, and also standard deviations and skewness for the FAA scores) and percentages for categorical variables for all of the study variables. Next, and given evidence regarding the moderating impact of the stability of asymmetry measures on the associations between such measures and criterion variables we computed Cronbach’s alphas for each composite FAA score to ensure that they were adequately reliable for the planned analyses. To test the hypothesized negative associations between the FAA and catastrophizing scores, we computed 12 correlation coefficients between the FAA scores for each of the three anterior electrode pairs (FP2-FP1, F8-F7, and F4-F3) and the four catastrophizing scale scores (assessing Helplessness, Rumination, Magnification, and Total catastrophizing). If there were no association between FAA and subsequent catastrophizing (i.e., if the correlation coefficient between these variables was 0.00 in the population), then 50% of the coefficients would be expected to be negative on average, and 5% of the coefficients would be expected to be statistically significant. We compared these expected percentages with the percentages of (1) negative coefficients and (2) significant associations that we actually found using nonparametric binomial tests.

**Results**
**Description of the study participants.** The descriptive information for the study participants are presented in Table 1.

[Insert Table 1 about here]

**Means, standard deviations, ranges, and skewness of the FAA scores.** The mean composite FP2-FP1, F8-F7, and F4-F3 FAA scores were -0.0005 (SD = 0.0265), -0.0061 (SD = 0.0696), and .0189 (SD = 0.0626), respectively. These FAA scores ranged from -0.05 to 0.06 (FP2-FP1), -0.02 to 0.09 (F8-F7), and -0.12 to 0.13 (F4-F3), and all had absolute skewness values less than 1.0 (-0.21, -0.96, and -0.30, respectively).

**Reliability of the asymmetry measures.** The Cronbach’s alphas for the three FAA composite scores (one for each electrode pair, made up of an average of the asymmetry scores from each of the five assessments) were .79 (FP2-FP1), .81 (F8-F7), and .89 (F4-F3) for FAA, indicating a high degree of internal consistency for each measure of FAA.

**Associations between asymmetry scores and subsequent catastrophizing.** The correlation coefficients used to test the study hypotheses regarding baseline FAA scores and measures of subsequent (two years later) catastrophizing are presented in Table 2. As can be seen, and generally consistent with the study hypothesis, almost all (11 of 12 coefficients, or 92%) of the correlation coefficients were negative (i.e., more relative left frontal activity is associated negatively with catastrophizing); two of these coefficients (between the PCS Magnification and Total scale scores and FAA at F4-F3) representing 16% of the coefficients computed, were statistically significant (p < .05). The number of negative associations found (92%) was statistically significantly more frequent than would be expected if there were no association between anterior asymmetry and subsequent catastrophizing in the population (50%, p = .006). However, the difference between the rate of statistically significant associations found
(16%) and the rate expected if there was no association in the population (5%) only approached significance (p = .118).

[Insert Table 2 about here]

**Discussion**

To our knowledge, this is the first time that the associations between a measure of brain activity as assessed by EEG – specifically, a measure of FAA – has been tested as prospective predictor of pain-related catastrophizing. Consistent with the study hypothesis based on the AAE model, we found that almost all of the measures reflecting greater anterior alpha asymmetry assessed at one point in time were associated negatively with subsequent catastrophizing scores obtained two years later.

Just as the AAE model does not argue that anterior brain regions are a “center” of emotional processing, the current findings should not be interpreted as supporting a conclusion that catastrophizing is necessarily caused by or depends entirely on anterior brain circuits. Catastrophizing is a complex cognitive activity that likely involves neuron assemblies and circuits spread throughout the cortex and deeper brain structures. However, a growing body of research using functional brain imaging assessment strategies indicates that activity in anterior brain areas plays a role in catastrophizing. These areas include the dorsolateral prefrontal, medial frontal, ventrolateral prefrontal, premotor, and anterior cingulate cortices. However, medial and posterior regions, such as the claustrum, posterior cingulate cortex, cerebellum, and parietal cortex have also been shown to be associated with catastrophizing. Thus, anterior regions appear to be important to catastrophizing, but the
neurophysiological substrates of catastrophizing do not appear to reside only in the frontal cortices.

Although neither the AAE model nor existing evidence support the existence of a catastrophizing “center” in the brain, the AAE model of catastrophizing does hypothesize a relative difference in neuron assembly activity in the left versus right hemispheres associated with catastrophizing, with more relative left activity associated with less catastrophizing. However, although a specific pattern of associations between reliable measures of FAA and catastrophizing is hypothesized from the AAE model – that is, a pattern of more frequency negative than positive associations – research from a number of sources suggests that these associations would tend to be weak to moderate rather than strong. First, research indicates that a number of factors unrelated to trait FAA can influence FAA scores. These include the time of day and perhaps time of year and the montage used when computing alpha power, among many other possible factors. These factors would work to limit the associations found between FAA estimates and other variables. Consistent with this idea, the associations found between FAA and other measures, while sometimes statistically significant, tend to be only moderate, and are not always statistically significant. However, the patterns of (1) mostly negative correlations and (2) negative correlations when statistically significant associations are found, has been consistent in this literature (see reviews by Harmon-Jones and Davidson). Hence, the correlation coefficients between reliable measures of alpha asymmetry and catastrophizing would not necessarily be expected to be strong. Rather, the model would predict that the majority of the correlations computed would be negative, consistent with our findings.

If our results are replicated in other samples of individuals with chronic pain, they would have important implications for enhancing the beneficial effects of treatments designed to reduce
catastrophizing. This idea is consistent with a recently proposed model of psychosocial pain treatments that hypothesizes a role for brain states in facilitating response to pain treatment\textsuperscript{55}. Specifically, the findings raise the possibility that by increasing FAA prior to CBT targeting catastrophizing, response to CBT could be enhanced. Evidence consistent with the general idea that patient response to treatments can be enhanced by procedures that prepare patients for treatment has been demonstrated using hypnosis to prepare patients for or enhance response to CBT\textsuperscript{56,57}, as well as the use of Motivational Interviewing to prepare patients with chronic pain for pain self-management training\textsuperscript{58} or physical therapy\textsuperscript{59,60}).

There are a large variety of interventions that influence brain activity which could potentially be used to enhance FAA and potentially prepare patients for responding more to CBT treatments that target catastrophizing and behavioral activation, including lateralized exercises, EEG-neurofeedback (also known as neurofeedback), brain stimulation procedures, meditation training, and imaging among others\textsuperscript{61}. Interestingly, and consistent with the AAE model, contractions of the left hand (squeezing a rubber ball) induces positive affect and positive bias judgments, respectively, and appear to have these effects via changes in FAA\textsuperscript{62}. Neurofeedback, which can result in increases or decreases in specific brain oscillations (e.g., alpha oscillations) as measured from specific scalp locations (e.g., just above the prefrontal cortices)\textsuperscript{38} could be used to decrease alpha activity as measured from electrodes placed over left anterior cortex and/or increase alpha as measured from electrodes placed over the right anterior cortex. Both transcranial direct current stimulation and repetitive transcranial magnetic stimulation, have shown some preliminary evidence for resulting in pain reduction when left anterior brain regions are stimulated\textsuperscript{61}. Some meditation procedures have also been shown to result in pain reductions\textsuperscript{61}, and recent evidence indicates that meditation training can also result in changes in FAA,
consistent with greater left hemisphere activation with meditation practice e.g., 56,57. Finally, an intervention that is designed to increase optimism (visualizing a future best possible self) – an emotional experience that is linked to the BAS system – not only resulted in the expected increase in optimism, but resulted in lower pain ratings in among healthy participants in response to a cold pressor task 65. Any of these treatments, providing they result in an increase in alpha asymmetry, could potentially result in decreases in catastrophizing via their effects on FAA, but could also enhance the efficacy of treatments that target catastrophizing, such as CBT, if provided just before those treatments. Given the urgent need to increase the overall efficacy of chronic pain treatments, including CBT, this possibility should be explored.

This study has a number of important limitations that should be considered. First, the sample size of 29 is relatively small, which can lead to both decreased reliability of the statistics obtained as well as a decreased ability to detect true associations. However, the fact that the FAA hypothesis was largely supported despite the low sample size provides some support for the reliability of the results. Still, because this is the first time, to our knowledge, that the associations between FAA and subsequent catastrophizing was assessed, replication in other samples of individuals with chronic pain is needed before we can determine the reliability and generalizability of the findings. A second issue to consider is that the study upon which these findings are based was not specifically designed to test the FAA hypothesis. The idea for assessing catastrophizing in this sample of individuals for whom EEG data were available occurred to us two years after the EEG data were collected. We were therefore not able to evaluate whether (and to what extent) FAA is associated with concurrent catastrophizing. Previous longitudinal FAA research, as well as evidence indicating that FAA is moderately stable, provides a rationale for the longitudinal design and analyses presented here. Moreover,
the fact that the predicted associations between FAA and subsequent catastrophizing were found even though the assessments were separated by two years provides some evidence for the robustness of the association. Still, research examining the concurrent associations between FAA and catastrophizing, and perhaps more importantly, the effects of interventions that alter FAA and subsequent catastrophizing, would be useful next steps.

Despite the study’s limitations, the findings identify a possible biological correlate of catastrophizing, which is consistent with a neurophysiological model of behavioral activation/inhibition. The findings also have important implications for the development and evaluation of novel approaches that might enhance the efficacy of treatments that target changes in catastrophizing. Ultimately, this understanding, and possible new interventions that emerge from this understanding, could contribute to more individuals being more able to cope adaptively with pain.

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References


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Table 1. Participant demographic and descriptive information.

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<tr>
<th>Variable</th>
<th>Range or number</th>
<th>Mean or percent</th>
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<tr>
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<tr>
<td>More than one race*</td>
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<tr>
<td>Highest education level</td>
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<tr>
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<td>Level 4</td>
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Note:  GED = Graduate Equivalency Degree
*One subject described himself as White and American Indian
**One subject’s sensation deficit was not clearly attributable to SCI, so the evaluating physician was unable to assign level by clinical exam, although the SCI diagnosis was confirmed by radiographical exam.
Table 2. Pearson correlation coefficients between the Pain Catastrophizing Scale scores and measures of alpha asymmetry at each pair of frontal electrode sites

<table>
<thead>
<tr>
<th>Electrode pairs</th>
<th>Helplessness</th>
<th>Rumination</th>
<th>Magnification</th>
<th>Total</th>
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<tr>
<td>Alpha asymmetry</td>
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<tr>
<td>FP2-FP1</td>
<td>-.11</td>
<td>-.20</td>
<td>-.30</td>
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<td>F8-F7</td>
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<td>-.27</td>
<td>-.46*</td>
<td>-.38*</td>
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</table>

†p < .10, *p < .05

Note: Because alpha power is associated with greater inhibition, a higher asymmetry score indicates greater relative left hemisphere versus right hemisphere activity; negative coefficients therefore indicate that more left versus right hemisphere activity is associated with less catastrophizing.