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Meaningful Nonsense: Invented Words Reveal Characteristics of Emotional Stimuli

Emil G. Moldovan
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Pain Perception and Perspective Taking in Spinal Cord Injury Patients

Caitlin J. Duckett

Williamsburg, Virginia

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Caitlin J. Duckett

Approved by the Committee, May, 2014

Committee Chair
Jennifer A. Stevens, PhD
Department of Psychology

Paul D. Kieffaber, PhD
Department of Psychology

Meghan M. Sinton, PhD
Department of Psychology

COMPLIANCE PAGE

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ABSTRACT

The mirror neuron system and the 8-12 Hz activity associated with it is implicated as a systematic response important for empathy. Previous research has demonstrated that this activity is suppressed when participants observe painful stimuli. Perspective taking, such as taking the perspective of a stranger, the self, or a close other has been shown to activate different areas of the brain in response to these same stimuli. The goal of the current investigation is to determine whether the mirror neuron system activity is modulated for spinal cord injury patients whose injuries have resulted in paraplegia when taking each of the three aforementioned perspectives. EEG recordings were conducted while participants observed painful images of the upper and lower extremities. It is hypothesized that the mirror neuron system activity response will be modulated for the spinal cord injury patients because they cannot feel pain in their lower extremities, but they should show the typical response for the upper extremities. This hypothesis stems from research on amputee patients who show cortical reorganization as a result of loss of limb, and we are interested in determining whether spinal cord injury patients show a similar reorganization. Our hypothesis for this experiment was not supported due to the finding that spinal cord injury patients showed similar patterns of mu rhythm suppression in response to painful stimuli in the lower extremities

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Pain Perception and Perspective Taking in Spinal Cord Injury Patients

The mirror neuron system was first identified using single cell recordings in macaque monkeys. Researchers found activation in inferior premotor area F5 when the monkeys reached for food but also when they observed others doing the same in a different condition (di Pellegrino, Fadiga, Fogassi, Gallese & Rizzolatti, 1992). The coupling of actor and action is crucial; observation of the experimenter alone or the object alone is not enough to elicit mirror neuron system activity (Gallese, Fadiga, Fogassi, & Rizzolatti, 1996). Moreover, this information can be presented in different sensory modalities. For example, activation will occur if context provides enough information about a hidden action. Mirror neurons selectively discriminate actions by sound when it is paired with an appropriate sound. In a study by Kohler and colleagues (2002), mirror neuron system activity was elicited when monkeys heard a noisy piece of paper being ripped. This activation was elicited both when the monkeys could see the paper being ripped and when they could not see it. Similar patterns of activation were also seen when a peanut shell was broken. However, noises such as white noise that does not have an action to pair it to did not elicit mirror neuron system activity. In studies like this, it is thought that the monkeys create their own mental representation of the action (Umiltà et al., 2001). In general, the evidence supports the idea that the mirror neuron system allows

representation and evaluation of the actions of others (Rizzolatti, Fadiga, Gallese, & Fogassi, 1996).

The Human Mirror Neuron System

When mirror neurons were discovered, the question of whether there was a similar system in the human quickly became important. Single cell recordings are not used with humans, so other methodologies such fMRI, EEG, and MEG were utilized. Overall, a similar pattern of activation during observation/execution situation occurs. Patterns of motor evoked potentials have been shown to match in conditions where human participants observe a motor action as well as perform the action themselves (Fadiga, Fogassi, Pavesi, & Rizzolatti, 1995; Watkins, Strafella & Paus, 2003; Buccino, Binkofski & Riggio, 2004).

Early research on the mirror neuron system in humans focused on motor representation and action, but more recent research has expanded the focus to other areas of human cognition. Research has been conducted on the importance of the mirror neuron system in similarities and differences between adults and babies in perception (Marshall & Meltzoff, 2011), the mirror neuron system's dysfunction in autism (Oberman, Ramachandran, & Pineda, 2008) and the role of the mirror neuron system in language (Gallese, 2008). Consistent with the primate research, the mirror neuron system has also been shown to be active for auditory stimuli in addition to visual stimuli

(Gazzola, Aziz-Zadeh, & Keysers, 2006). Evidence for a shared cortical network between execution and observation of action has also been demonstrated using quantified electroencephalography (Cochin, Barthelemy, Roux & Martineau, 1999). An extensive literature review conducted by Decety and Jeannerod (1995) of experimental psychology procedures also has demonstrated a shared cortical network between motor imagery and motor execution using both typical populations and neurological patients, suggesting that there is a cortical area in the human brain for representing the actions of other people. The current investigation is interested in the mirror neuron system's role in empathy and pain perception in both typical and paraplegic participants while engaging in a perspective taking task. The current investigation utilizes EEG to test difference in mu rhythm activity.

Empathy and Mirror Neuron System

The hallmark mirror neuron system activity that is recorded by EEG is called mu rhythm. Mu rhythm is a cortical oscillatory activity which occurs in the 8 – 12 Hz frequency band over the sensorimotor cortex (Hari & Salmelin, 1997; Rizzolatti & Fabbri-Destro, 2008). High mu rhythm activity represents an idling state in the brain; thus, mu rhythm activity is greatest when the body is at rest and desynchronization of it reflects activation of the mirror neuron system (Muthukumaraswamy & Johnson, 2004). Alpha rhythm also occurs in this frequency band, but it is located over the occipital region of the brain.

Modulation of this activity has been shown to reflect visual attentional processing as opposed to mirror neuron system activity (Pineda, 2005; Perry, Stein & Bentin, 2011).

In response to the actual experience of pain, oscillatory activity like mu rhythm has been shown to be suppressed globally over the cortex (Mancini, Longo, Canzoneri, Vallar & Haggard, 2005; Ploner, Gross, Timmerman, Pollock & Schnitzler, 2006). Suppression of mu rhythm activity has also been implicated in empathic processing in the observation of others experiencing painful stimuli. Mu rhythm is measured as the activity over the sensorimotor cortex and activation in the somatosensory cortex has been shown to occur while observing others in pain using fMRI. This activation in the brain causes a suppression of mu rhythm activity. This suppression occurs in both nonpainful and painful situations, but significantly more so for the painful situations (Cheng, Yang, Lin, Lee, & Decety, 2008).

In another study using EEG, Perry, Bentin, Bartal, Lamm, and Decety (2010) demonstrated that significant mu rhythm suppression occurs when participants see a hand being pricked by a needle as compared to seeing a hand being prodded by a cotton swab. Most interestingly, one group of participants were told that nonpainful stimuli were actually painful to the person in the picture who as suffering from a neurological condition. These participants also showed significant mu rhythm suppression, just as they did

to the stimuli that they found to be personally painful. This study suggests that the mirror neuron system is crucial for representing how others feel and their experiences, and that it can be active in situations that are not familiar to the participant. The use of multiple methodologies to support this finding provides even stronger evidence that the mirror neuron system mediates the representation of others.

Behavioral Correlates of Empathy

One issue raised in the midst of mirror neuron system research relates to how its activity may be associated with empathy, and not merely a form of sensory processing. In support of its relation to empathy, studies using facial expressions and instructions to take an empathetic perspective have been conducted. When participants are shown faces of people receiving medical treatment making expressions as if they are in pain, fMRI reveals a graded activation response in the middle insula, anterior medial cingulate cortex, medial and lateral premotor areas, and selectively in left and right parietal cortices occurs between when participants are told to imagine that the person in the picture is themselves or someone else (Lamm, Batson, & Decety, 2007).

In other studies using fMRI, it has been demonstrated that brain activity is modulated in taking the perspective of a loved one, a stranger or the self. In a study in which participants watched hands and feet in painful

situations, taking the perspective of a stranger caused increased activation in the right temporo-parietal junction (TPJ) while all three perspectives showed similar activation in sensory processing networks that have been associated with pain processing in many previous studies. However, as participants rated their relationship with their loved one as closer and more personal, this activation in the right TPJ was attenuated (Cheng, Chen, Lin, Chou & Decety, 2010). In another study utilizing facial expressions, participants were asked to evaluate their own reaction to the facial expression being made which acted as a self condition, or to evaluate the emotion being expressed, which acted as the other condition. In this self task, the medial prefrontal cortex, the posterior cingulate cortex and the TPJ in both hemispheres were differentially activated. This study demonstrated activation in a common network as well which included the temporal poles and the bilateral inferior frontal cortices, as with the aforementioned study (Schulte- Rüther, Markowitsch, Fink, & Piefke, 2007). These studies reflect modulated brain activity in perspective-taking. Brain activity also appears to be modulated based on the perceived closeness of the relationship of the person whose perspective they are taking, suggesting an emotional component in these effects. This emotional component is likely indicative of empathy as opposed to a basic sensory process.

Even when perspective-taking is not asked of the participants and they passively view facial expressions, significant activation in the right ventral premotor area occurs. When these participants were asked to imitate these facial expressions, bilateral activation in this area occurred (Leslie, Johnson-Frey, & Grafton, 2004). Taken together, these studies suggest that observation and execution share a cortical network. Taken further, imagining the emotions of others requires cortical structures that are required to experience one's own emotions, but perspective taking does change neural activity in order to take an empathetic perspective for strangers and loved ones.

In a study using happy and disgusted faces, more mu rhythm suppression in the mirror neuron system has been shown than for someone who is taking the perspective of a neutral face. Importantly, the degree of mu rhythm suppression correlated positively with how well the participants reported being able to take the perspective of the person in the picture (Moore, Gorodnitsky, & Pineda, 2012). Similarly, mu suppression as a result of observed motor action has been positively correlated with greater scores on the perspective-taking subscale of the Interpersonal Reactivity Index (IRI) (Woodruff, Martin, & Bilyk, 2011) as well as the personal distress subscale (Cheng et al., 2008). The Interpersonal Reactivity Index is a questionnaire consisting of four subscales that has been shown to accurately assess empathetic capabilities. Questions for the perspective-taking subscale

include, "I sometimes try to understand my friends better by imagining how things look from their perspective" and questions from the personal distress subscale include, "Being in a tense emotional situation scares me" (Davis, 1980, 1983). Given these correlational findings of mu rhythm to the IRI, it is logical to infer that mu rhythm suppression represents empathic activity in the brain.

Theory of Mind

Theory of mind abilities are also necessary to take an empathetic perspective. Theory of mind is defined as the ability to understand the mental states of others whereas empathy refers to the ability to infer others' emotional experiences (Völlm et al., 2006). Simulation theory suggests that it is necessary to experience another person's mental state in order to understand it by taking their perspective, tying theory of mind abilities and empathy together (Gallese & Goldman, 1998). Thus, the hypothesis that empathy and theory of mind are inextricably linked has been put forth.

Tager-Fulsberg and Sullivan (2000) have postulated that theory of mind may be separated into two components: social-cognitive and social perceptual. The social cognitive component entails attributing beliefs to others and theory-building. The social perceptual component is thought to involve attributions of emotions and also involves facial and body expressions. The two components most likely work in concert with each other, and the social

perceptual component appears to map on strongly with the construct of empathy.

Both empathy and theory of mind have also been associated with the mirror neuron system and mu rhythm activity. Significant mu rhythm suppression has been shown in a social-perceptual task requiring participants to judge facial expressions by only looking at the eyes, but significance was not seen in the social-cognitive task where participants were asked to declare the intentions of a cartoon strip that they were shown. This same experiment also tested a theory of mind control task in which participants were instructed to make attributions about cartoon characters using objects. Interestingly, in this task accuracy was correlated with mu rhythm suppression (Pineda & Heicht, 2009). This experiment illustrates that the mirror neuron system is active while judging emotions and attempting to infer what others are doing with objects, but addition resources may be required for attributing beliefs. However, as noted previously, the social-perceptual component appears to map on most strongly to empathy which is of particular interest in the current investigation.

The Current Investigation

The current investigation uses EEG to ascertain if spinal cord injury patients whose injuries have resulted in paraplegia will show a different empathetic response in the mirror neuron system to pain in the lower

extremities. Their empathetic response to upper extremities is hypothesized to be typical. We can expect this mirror neuron system response to pain based on the wealth of prior research demonstrating this finding by using EEG and measuring mu rhythm suppression (Lamm, Batson, & Decety, 2007; Cheng, Yang, Lin, Lee, & Decety, 2008; Perry, Bentin, Bartal, Lamm & Decety, 2010). We hypothesize that lower extremities will show less suppression due to the loss of feeling and function in these extremities. This hypothesis is based on prior research concerning cortical reorganization due to injury. For amputee patients, sensorimotor cortex reorganization has been systematically demonstrated after loss of both upper and lower extremities in both humans and macaque monkeys (Pons et al., 1991; Chen, Corwell, Yaseen, Hallett & Cohen, 1998; Karl, Birbaumer, Lutzenburger, Cohen, & Flor, 2001). This experiment will help to elucidate whether complete loss of limb is required for this cortical reorganization, or is loss of feeling and function will suffice.

In addition, we hypothesize that all participants will show greater mu rhythm suppression for the self perspective. The close other perspective will be similar and stranger response will be much less, given prior fMRI research showing a graded response in the mirror neuron system structures as mentioned previously. If the hypothesized cortical reorganization has occurred, spinal cord injury patients should not show a response for the lower extremities. If they do show any sort of response in the lower extremities it

would likely be for the self perspective because historically that has shown to have the most robust empathetic response as measured by mu rhythm suppression.

Method

Participants

Two paraplegic males were recruited for this study whose injuries have resulted in loss of feeling and function in the lower extremities. Their ages were 22 and 33 years old with a time since injury of 1.3 and 2.6 years, respectively. The 22 year old participant sustained a fraction dislocation of the T12 vertebra after a fall from a roof. The 33 year old participant sustained a complete break of the T10 vertebra after being in a car accident. These participants did not report any other incidences resulting in neurological trauma. They also did not report any neurological or psychiatric conditions, including neuropathy and other pain-related disorders sometimes seen after spinal cord injuries.

In addition to the paraplegic participants, 10 undergraduates were also recruited for course credit. One male participant reported sustaining several concussions in the past, and was excluded from analysis. Therefore EEG data was analyzed from 3 females and 6 males. Their average age was 20.6 years, SD = 1.6 years. All participants included in analysis did not report any

previous neurological injury or disease, or any psychiatric conditions at the time of testing.

Apparatus

For data acquisition, continuous EEG data was recorded using a DBPA-1 Sensorium bio-amplifier (Sensorium Inc., Charlotte, VT). We used a 10-5 cap system with 74 AgCl electrodes (Electrode Arrays, El Paso, TX). A ground electrode was placed in the center of the forehead and the reference electrode was placed on the right side of the nose. Electrodes were placed both above and below each eye as well as on the corners of the eyes to monitor for ocular artifact. Data was recorded continuously at 500 Hz and impedances were kept below 12 kΩ for all electrodes.

Stimuli

Stimuli were presented to participants using PsychoPy (Peirce, 2009). Stimuli consisted of 40 color photographs of upper and lower extremities in both painful and nonpainful conditions. Painful stimuli consisted of needle injection while non-painful stimuli consisted of prods with a Q-tip in ten different locations on the lower extremities ranging from the knee to the bottom of the foot for the lower extremities and ten different locations ranging from the tips of the fingers to the inner portion of the elbow for upper extremities. Figure 1 shows an example of upper and lower extremities in both painful and nonpainful conditions. This method was adapted from Perry et al, 2010. Examples of painful and nonpainful stimuli are shown in Figure 1.

Participants observed the stimuli in three blocks consisting of 80 trials, resulting in a total of 240 trials per participant. In each block, all stimuli were presented randomly.

Design

Participants were instructed to observe the painful and nonpainful stimuli while taking the perspective of a stranger, of the self or of a close other. All participants did the perspective taking in this order. For the close other perspective, participants were instructed to choose a person with whom they closely identify. Researchers suggested either their mother or a significant other, but some participants reported choosing their best friend or their child.

For each trial, a fixation cross appeared on the screen for 500 milliseconds, then participants saw the painful or nonpainful stimuli for 1250 milliseconds. Each trial consisted of two needle injections or two prods with the Q-tip for both the upper and lower extremities. The intertrial interval occurred for a random period of time between 3 and 5 seconds. Participants were given a short break in between each block of trials. Demographic information and screening for previous injuries or conditions was collected prior to the EEG recording. During this screening, participants were also asked to rate their own feelings about injections and to report fear of injections in order to exclude any participants with phobic responses to injections.

Data Analysis

Continuous EEG data recording was conducted at 72 channels and downsampled to 250 Hz for offline analysis. Data analysis was conducted using EEGLAB for MATLAB. Data were visually inspected for extreme artifact such as muscle movement and ocular artifact was corrected using an ICA algorithm ran through EEGLAB. Data were band-pass filtered from 1 to 30 Hz. The data were epoched into 1750 millisecond epochs from the timelocking event with a 200 millisecond baseline period. Data epochs with significantly high kurtosis were rejected as artifact.

For each clean data epoch, Fast Fourier transforms were conducted to attain absolute power values for spectrum analysis. Due to our interest in the mirror neuron system, electrodes C3, CZ and C4 were selected for statistical analysis of mu rhythm power. In the 8 – 12 Hz range, data were segmented into 0.5 Hz ranges for a more precise analysis. Paired samples t-tests were performed between the nonpainful and painful condition for each block at each electrode of interest to test for mu rhythm suppression in each condition. Difference scores greater than zero indicate mu rhythm suppression to painful stimuli as compared to nonpainful. These difference scores were then entered into a 2 (patient, typical) x 2 (upper extremity, lower extremity) x 3 (stranger, self, close other) mixed-model ANOVA with repeated measures for the last two variables at each electrode of interest.

Results

Independent samples t-tests were conducted to assess differences in sensitivity to injections based on the ratings collected prior to EEG recording. Fear of injections was not significant between typicals ($M = 1.44$, $SD = 0.53$) and patients ($M = 2.00$, $SD = 0$), $t = -1.43$, $p = 0.19$. However, discomfort from watching others receive injections did differ between typicals ($M = 1.22$, $SD = 0.44$) and patients ($M = 2.00$, $SD = 0$), $t = -2.93$, $p = 0.04$. This indicates that while the two groups do not significantly differ in sensitivity to personally receiving injections, they do differ on observing others receiving injections. Patients reported experiencing significantly more discomfort from observation of injections.

Paired-Samples t-tests at the Lower Extremities

In the stranger condition, paired-samples t-tests revealed no significant differences in mu rhythm suppression for either patients or typical participants for painful stimuli compared to nonpainful stimuli at each electrode of interest. The majority of frequency ranges at each electrode had positive suppression scores indicating suppression for painful stimuli, but none of these differences reached significance. The values for patients are presented in Table 1 and values for typicals are presented in Table 2.

In the self condition, paired samples t-tests revealed significant differences for both patients at electrode CZ and typical participants at

electrodes CZ and C4 for painful stimuli compared to nonpainful stimuli. The values for patients are presented in Table 3 and the values for typicals are presented in Table 4.

In the close other condition, paired samples t-test revealed significant differences for only the patient group at electrode C3. These values are presented in Table 5 for patients and Table 6 for typicals.

Paired-Samples t-tests at the Upper Extremities

In the stranger condition, paired-samples t-tests revealed no significant differences in mu rhythm suppression for either patients or typical participants for painful stimuli compared to nonpainful stimuli. The values for patients are presented in Table 7 and values for typicals are presented in Table 8. In the self condition, paired samples t-tests revealed significant differences only for the typical participants at electrodes CZ and C4 for painful stimuli compared to nonpainful stimuli. The values for patients are presented in Table 9 and the values for typicals are presented in Table 10. In the close other condition, paired samples t-test revealed significant differences in the upper conditions for both patients at electrodes C3 and CZ and typical participants at all three electrodes of interest. These values are presented in Table 11 for patients and Table 12 for typicals.

Analysis of the Difference Scores Between Painful and Nonpainful Stimuli

A mixed-model ANOVA of the difference scores between nonpainful and painful stimuli was conducted to assess differences among perspective, neurological status and extremity at each of the electrodes of interest.

Extremity

Extremity had a significant main effect at electrode C4 in the 10.0 – 10.5 Hz frequency range, $F = 11.34$, $p < 0.01$. This effect indicated that mu rhythm suppression was higher for the lower extremities than the upper extremities (mean difference = 0.98). This effect was qualified by an extremity x neurological status interaction showing that patients had higher mu rhythm suppression in the lower extremities than for the upper extremities. The plot of this interaction is depicted in Figure 2.

Extremity also had a significant main effect at electrode C4 in the 11.5 – 12.0 Hz frequency range, $F = 20.65$, $p < 0.01$. At this frequency range, the mu rhythm suppression was greater for the upper extremities than the lower extremities (mean difference = 1.05). Again, this effect was qualified by an extremity x neurological status interaction in this range, $F = 22.32$, $p < 0.01$. This interaction showed this effect arose from patients having greater mu rhythm suppression in the upper extremities than the lower extremities. This interaction is plotted in Figure 3.

The extremity x neurological status interaction was also significant at electrode CZ in the 10.0 – 10.5 Hz frequency range, $F = 5.59$, $p = 0.05$. This

interaction showed that patients had greater mu rhythm suppression for the lower extremities than for the upper extremities. The plot of the interaction is shown in Figure 4.

Perspective

Electrode C4 in the 11.5 – 12.0 Hz frequency range showed a main effect for perspective, $F = 4.76$, $p = 0.02$. Pairwise comparisons showed that the close other condition was significantly lower in mu rhythm suppression than both the self condition (mean difference = 0.87, $p = 0.03$) and the stranger condition (mean difference = 1.54, $p = 0.02$).

Discussion

While participants in this study did not show significant mu rhythm suppression for the stranger condition, significant effects were seen in both the self and close other conditions when these conditions were analyzed separately using paired-samples t-tests. Given this lack of significance in the stranger condition, it stands to reason that empathy was the mediating role in these differences, especially since the stranger condition was presented first to all participants.

As stated in the introduction, mirror neuron system activity analysis of perspective-taking has previously yielded significantly less suppression in the stranger condition compared to the self and close other conditions. For

analysis across the difference scores from each condition, the stranger condition did yield significantly more suppression than the close other condition electrode C4 in the 11.5 – 12.0 Hz frequency range. This may be attributable to a habituation effect given that the close other perspective was always presented last and the stranger condition was always presented first. Given the small sample size of this study particularly in the patient group, effective counterbalancing was not conceivable. This limitation should be addressed in future research to control for potential habituation effects to the painful stimuli. Although the difference scores did not present any other significant effects for perspective, the paired-samples t-tests show a clear trend for more significant suppression in the self and close other conditions as compared to the stranger condition.

The self condition showed differential suppression in that patients displayed significance in the lower condition but not the upper, where typicals showed suppression in both. The fact that suppression was found in the lower extremities for the patients directly contradicts the hypothesis of cortical reorganization. A lack of mu rhythm suppression in the upper condition may be attributable to their status as a patient. Because fear of injections scores did not differ between the two groups, the patients in this study may have had more experience with needles due to their injury and are thus less likely to show the predicted mu rhythm suppression. Considering they did show it in

the close other perspective, it is likely that an emotional component elicited that suppression where the self condition did not.

The finding of lower suppression in the close other perspective for patients also argues against the hypothesis of cortical reorganization. The lack of mu rhythm suppression for the typicals in the lower condition is interesting in light of an effect for the upper condition. Ratings for personal feelings on injections were collected, but we did not collect information on how the person whose perspective they took feels about injections. It is possible that the typical participants took the perspective of a loved one who fears injections and this may have caused an interference effect with the lower extremities, given that they were presented randomly all in the same block. Future investigations may split the blocks between extremities to test for this possibility. This interference effect is likely due to the contextual nature of syringes as tools typically used on the upper extremities rather than the lower ones, and a fairly common phobia to them. The interference effect would not be expected in the self condition due to low ratings of fear of injections for both typicals and patients, and the stranger condition may not be specific enough to elicit an interference effect. Participants may not believe that the average person has a phobia of needles and thus would not elicit the effect.

The extremity x neurological status interactions seen among the difference scores in this experiment are mixed. While patients did show greater mu rhythm suppression for the upper extremities as compared to the lower extremities at the 11.5 – 12.0 range at electrode C4, the 10.0 – 10.5 range at both electrodes C4 and CZ suggest that mu rhythm suppression was actually greater for the lower extremities. This finding of an increase in lower extremity suppression compared to upper extremity suppression argues against our hypothesis of reduced mu rhythm suppression in the patient group.

Conclusions and Future Directions

In this research it is important to note both painful sensations and nonpainful sensations that may arise from these types of injuries. As with cortical reorganization, phantom pain sensations have been extensively documented in the literature and occur in 60-80% of amputees with severe cases accounting for approximately 5-10% (Nikolajsen & Jensen, 2000). In fact, the magnitude of cortical reorganization has been shown to be correlated to amount of phantom pain sensations - but not nonpainful sensations - reported by amputee patients (Flor et al., 1995). While cortical reorganization is not the only mechanism that is thought to contribute to these phantom sensations, they are nevertheless important to consider in research and treatment practices for these patients (Foell & Flor, 2013).

Comparison of this phantom limb pain research to paraplegic neuropathy would be a worthwhile endeavor in future investigations into spinal cord injury research. Paraplegic neuropathy can occur in these patients either at or below the level of injury. Types of pain included in paraplegic neuropathy are burning pain, squeezing pain, and/or pins and needle sensations (Attal et al., 2008). This chronic pain presents in about 40% of spinal cord injury patients (Baastrup & Finnerup, 2008). In our study, neither paraplegic participant reported neuropathic pain in the lower extremities. It is very difficult to find the correct medication for treating this problem and it causes drastic quality of life decreases in the patients who experience it (Henwood & Ellis, 2004). While research has been conducted on this phenomenon, the specific causes and their mechanisms have yet to be elucidated (Finnerup, Baastrup & Jensen, 2009). Cortical reorganization may be a strong candidate for further research in conjunction with other factors, considering its effects on amputee patients. Comparing spinal cord injury patients with neuropathic pain to those who do not experience may show similar results to those that have found the magnitude of cortical reorganization is indicative of the level of phantom limb pain, as cited previously in this work. The findings in our study suggesting that paraplegic participants show similar mu rhythm suppression to painful stimuli in the lower extremities and who do not report paraplegic neuropathy further strengthens the need for this research.

Given the contextual nature of injections with a syringe discussed in the discussion section, future studies should also consider different types of pain, both mechanical and otherwise. If seen as a medical tool, empathy for pain from injections may be attenuated by the perceived benefit that the person being injected is receiving. On the other hand, if the participant is taking the perspective of someone who fears injections significantly more than they personally do, differential effects may be seen there as well, potentially as with the current study. Other possible types of pain could include simulating cuts on the arms and legs, or thermal pain on these extremities. Analysis of differences among these types of pain would be intriguing for future research as well.

Both of the paraplegic participants sustained their injuries on the thoracic spine. Thus, comparing different levels of injury would be a logical next step in future research. It is plausible that cortical reorganization may be affected by which nerves were damaged and this analysis would help to better understand if cortical reorganization occurs and to what magnitude.

The time elapsed since injury was also very similar in our two paraplegic participants and testing was conducted in a relatively short time since the injury. Longitudinal studies or between-subjects designs including people with very recent injuries and those who have had their injuries for a longer time would also be a logical next step in this research. Understanding

when these changes occur can be applications to the paraplegic neuropathy work discussed earlier.

The current study has shown significant effects to painful stimuli for spinal cord injury patients for the lower extremities. This suggests that their sensorimotor cortex is not significantly affected relative to a neurotypical control group. Increasing sample sizes and considering the limitations posed here can add to this interesting research to better understand the brain and mirror neuron system response to spinal cord injuries.

References

Attal N., Fermanian C., Fermanian J., Lanteri-Minet M., Alchaar H., & Bouhassira, D. (2008). Neuropathic pain: are there distinct subtypes depending on the etiology or anatomical lesion? *Pain, 138*, 343 – 53.

Baastrup, C., & Finnerup, N. B. (2008). Pharmacological management of neuropathic pain following spinal cord injury. *CNS Drugs*, *22*(6), 455–475.

Buccino, G., Binkofski, F., & Riggio, L. (2004). The mirror neuron system and action recognition. *Brain and Language*, *89*(2), 370–376. doi:10.1016/S0093-934X(03)00356-0

Cochin, S., Barthelemy, C., Roux, S., & Martineau, J. (1999). Observation and execution of movement: similarities demonstrated by quantified electroencephalography. *European Journal of Neuroscience*, *11*(5), 1839 –1842. doi:10.1046/j.1460-9568.1999.00598.

Chen, R., Corwell, B., Yaseen, Z., Hallett, M., & Cohen, L. G. (1998). Mechanisms of Cortical Reorganization in Lower-Limb Amputees. *The Journal of Neuroscience*, *18*(9), 3443–3450.

Cheng, Y., Chen, C., Lin, C., Chou, K., & Decety, J. (2010). Love hurts: an fmri study. *Neuroimage*, *51* (1), 923-29.

Cheng, Y., Lee, P.-L., Yang, C.-Y., Lin, C.-P., Hung, D., & Decety, J. (2008). Gender Differences in the Mu Rhythm of the Human Mirror-Neuron System. *PLoS ONE*, *3*(5), e2113. doi:10.1371/journal.pone.0002113

Cheng, Y., Yang, C.-Y., Lin, C.-P., Lee, P.-L., & Decety, J. (2008). The perception of pain in others suppresses somatosensory oscillations: a magnetoencephalography study. *NeuroImage*, *40*(4), 1833–1840. doi:10.1016/j.neuroimage.2008.01.064

Davis, M. H. (1980). A multidimensional approach to individual differences in empathy. *JSAS Catalog of Selected Documents in Psychology, 10*, 85.

Davis, M. H. (1983). Measuring individual differences in empathy: Evidence for a multidimensional approach. *Journal of Personality and Social Psychology, 44*, 113-126.

di Pellegrino, G., Fadiga, L., Fogassi, L., Gallese, V., & Rizzolatti, G. (1992). Understanding motor events: a neurophysiological study. *Experimental Brain Research*, *91*, 176-180.

Decety, J., & Jeannerod, M. (1995). [Imagery and its neurological substrate]. *Revue neurologique*, *151*(8-9), 474–479.

Fadiga, L., Fogassi, L., Pavesi, G., & Rizzolatti, G. (1995). Motor facilitation during action observation: a magnetic stimulation study. *Journal of Neurophysiology*, *73*(6), 2608–2611.

Finnerup, N. B., Baastrup, C., & Jensen, T. S. (2009). Neuropathic pain following spinal cord injury pain: mechanisms and treatment. *Scandinavian Journal of Pain*, *1*(51), 53-61.

Flor, H., Elbert, T., Knecht, S., Wienbruch, C., Pantev, C., Birbaumer, N., Larbig, W., & Taub, E. (1995). Phantom-limb pain as a perceptual

correlate of cortical reorganization following arm amputation. *Nature*, *375*(6531), 482-4.

Foell, J., & Flor, H. (2013). Phantom Limb Pain. In R. J. Moore (Ed.), *Handbook of Pain and Palliative Care* (pp. 417–430). Springer New York. Retrieved from http://link.springer.com/chapter/10.1007/978-1-4419-1651-8_25

Gallese, V. (2008). Mirror neurons and the social nature of language: The neural exploitation hypothesis. *Social Neuroscience*, *3*(3-4), 317–333. doi:10.1080/17470910701563608

Gallese, V., Fadiga, L., Fogassi, L., & Rizzolatti, G. (1996). Action recognition in the premotor cortex. *Brain*, *119*(2), 593–609. doi:10.1093/brain/119.2.593

Gallese, V., & Goldman, A. (1998). Mirror neurons and the simulation theory of mind-reading. *Trends in Cognitive Sciences*, *2*(12), 493–501. doi:10.1016/S1364-6613(98)01262-5

Gazzola, V., Aziz-Zadeh, L., & Keysers, C. (2006). Empathy and the somatotopic auditory mirror system in humans. *Current Biology: CB*, *16*(18), 1824–1829. doi:10.1016/j.cub.2006.07.072

Hari, R. & Salmelin, R. (1997). Human cortical oscillations: a neuromagnetic view through the skull. *Trends in Neurosceince, 20* (1), 44-49.

Henwood, P., & Ellis, J. A. (2004). Chronic neuropathic pain in spinal cord injury: the patient's perspective. *Pain Research & Management: The Journal of the Canadian Pain Society*, *9*(1), 39–45.

Karl, A., Birbaumer, N., Lutzenburger, W., Cohen, L., & Flor, H. (2001). Reorganization of motor and somatosensory cortex in upper extremity amputees with phantom limb pain. *The Journal of Neuroscience* , *21* (10), 3609-18.

Kohler, E., Keysers, C., Umiltà, M. A., Fogassi, L., Gallese, V., & Rizzolatti, G. (2002). Hearing Sounds, Understanding Actions: Action Representation in Mirror Neurons. *Science*, *297*(5582), 846–848. doi:10.1126/science.1070311

Lamm, C., Batson, C., & Decety, J. (2007). The neural substrate of human empathy: Effects of perspective-taking and cognitive appraisal. *Journal of Cognitive Neuroscience*, *19* (1), 42-58.

Leslie, K. R., Johnson-Frey, S. H., & Grafton, S. T. (2004). Functional imaging of face and hand imitation: towards a motor theory of empathy. *NeuroImage*, *21*(2), 601–607. doi:10.1016/j.neuroimage.2003.09.038.

Mancini, F., Longo, M., Canzoneri, E., Vallar, G., & Haggard, P. (2005). Changes in cortical oscillations linked to multisensory modulation of nociception. *European Journal of Neuroscience*, *37*, 768-76.

Marshall, P., & Meltzoff, A. (2011). Neural mirroring systems: Exploring the eeg mu rhythm in human infancy. *Developmental Cognitive Neuroscience*, 1, 110-123.

Moore, A., Gorodnitsky, I., & Pineda, J. (2012). EEG mu component responses to viewing emotional faces. *Behavioural Brain Research*, *226*(1), 309–316. doi:10.1016/j.bbr.2011.07.048

Muthukumaraswamy, S. D., & Johnson, B. W. (2004). Changes in rolandic mu rhythm during observation of a precision grip. *Psychophysiology*, *41*(1), 152–156.

Nikolajsen, L., & Jensen, T. S. (2000). Phantom limb pain. *Current Review of Pain*, *4*(2), 166–170. doi:10.1007/s11916-000-0052-0

Oberman, L., Ramachandran, V., & Pineda, J. (2008). Modulation of mu suppression in children with autism spectrum disorders in response to familiar or unfamiliar stimuli: The mirror neuron hypothesis. *Neuropsychologia*, *46*. 1558–1565.

Peirce, JW (2009). Generating stimuli for neuroscience using PsychoPy. ` Front. Neuroinform. 2:10. doi:10.3389/neuro.11.010.200

Perry, A., Bentin, S., Bartal, I. B.-A., Lamm, C., & Decety, J. (2010). "Feeling" the pain of those who are different from us: Modulation of EEG in the mu/alpha range. *Cognitive, Affective & Behavioral Neuroscience*, *10* (4), 493–504. doi:10.3758/CABN.10.4.493

Perry, A., Stein, L., & Bentin, S. (2011). Motor and attentional mechanisms involved in social interaction--evidence from mu and alpha EEG suppression. *NeuroImage*, *58* (3), 895–904. :10.1016/j.neuroimage.2011.06.060

Pineda, J. (2005). The functional significance of mu rhythms: Translating "seeing" and "hearing" into "doing". *Brain Research Reviews*, *50*, 57-68.

Pineda, J. A., & Heicht, E. (2009). Mirroring and mu rhythm involvement in social cognition: Are there dissociable subcomponents of theory of mind?. *Biological Psychology*, *80*(3), 306-14.

Ploner, M., Gross, J., Timmerman, L., Pollock, B., & Schnitzler, A. (2006). Pain suppresses spontaneous brain rhythms. *Cerebral Cortex*, *16* (4), 537-40.

Pons, T.P., Garraghty, P.E, Ommaya, A.K, Kaas, J.H, Taub, E., & Mishkin, M.M. (1991). Massive cortical reorganization after sensory deafferentation in adult macaques. *Science*, *252*, 1857–1860

Rizzolatti, G., & Fabbri-Destro, M. (2008). The mirror system and its role in social cognition. *Current Opinion in Neurobiology*, *18* (1), 84.

Rizzolatti, G., Fadiga, L., Gallese, V., & Fogassi, L. (1996). Premotor cortex and the recognition of motor actions . *Cognitive Brain Research*, *3*, 131-141.

Schulte-Rüther, M., Markowitsch, H. J., Fink, G. R., & Piefke, M. (2007). Mirror Neuron and Theory of Mind Mechanisms Involved in Face-to-Face Interactions: A Functional Magnetic Resonance Imaging Approach to Empathy. *Journal of Cognitive Neuroscience*, *19*(8), 1354–1372. doi:10.1162/jocn.2007.19.8.1354

Tager-Flusberg, H., & Sullivan, K. (2000). A componential view of theory of mind: evidence from Williams syndrome. *Cognition*, *76*(1), 59–90.s

Umiltà, M. A., Kohler, E., Gallese, V., Fogassi, L., Fadiga, L., Keysers, C., & Rizzolatti, G. (2001). I Know What You Are Doing: A Neurophysiological Study. *Neuron*, *31*(1), 155– 165. doi:10.1016/S0896-6273(01)00337-3

Watkins, K. E., Strafella, A. P., & Paus, T. (2003). Seeing and hearing speech excites the motor system involved in speech production. *Neuropsychologia*, *41*(8), 989–994. doi:10.1016/S0028-3932(02)00316-0

Woodruff, C. C., Martin, T., & Bilyk, N. (2011). Differences in self- and other-induced Mu suppression are correlated with empathic abilities. *Brain Research*, *1405*, 69–76. doi:10.1016/j.brainres.2011.05.046

Völlm, B. A., Taylor, A. N. W., Richardson, P., Corcoran, R., Stirling, J., McKie, S., Deacon, J.F., & Elliott, R. (2006). Neuronal correlates of theory of mind and empathy: A functional magnetic resonance imaging study in

a nonverbal task. *NeuroImage*, *29*(1), 90–98.

doi:10.1016/j.neuroimage.2005.07.022

Table Captions

Table 1 Suppression scores for the patient group in the stranger lower condition. Positive scores indicate suppression to painful stimuli. Significant scores are in bold.

Table 2 Suppression scores for the typical group in the stranger lower condition. Positive scores indicate suppression to painful stimuli. Significant scores are in bold.

Table 3 Suppression scores for the patient group in the self lower condition. Positive scores indicate suppression to painful stimuli. Significant scores are in bold.

Table 4 Suppression scores for the typical group in the self lower condition. Positive scores indicate suppression to painful stimuli. Significant scores are in bold.

Table 5 Suppression scores for the patient group in the close other lower condition. Positive scores indicate suppression to painful stimuli. Significant scores are in bold.

Table 6 Suppression scores for the typical group in the close other lower condition. Positive scores indicate suppression to painful stimuli. Significant scores are in bold.

Table 7 Suppression scores for the patient group in the stranger upper condition. Positive scores indicate suppression to painful stimuli. Significant scores are in bold.

Table 8 Suppression scores for the typical group in the stranger upper condition. Positive scores indicate suppression to painful stimuli. Significant scores are in bold.

Table 9 Suppression scores for the patient group in the self upper condition. Positive scores indicate suppression to painful stimuli. Significant scores are in bold.

Table 10 Suppression scores for the typical group in the self upper condition. Positive scores indicate suppression to painful stimuli. Significant scores are in bold.

Table 11 Suppression scores for the patient group in the close other upper condition. Positive scores indicate suppression to painful stimuli. Significant scores are in bold.

Table 12 Suppression scores for the typical group in the close other upper condition. Positive scores indicate suppression to painful stimuli. Significant scores are in bold.

Table 1 stranger lower: patients

electrode	frequency range	mean	SD	t	p
C3	8.0 - 8.5 Hz	1.15	0.36	4.49	0.14
	8.5 - 9.0 Hz	1.91	1.29	2.09	0.28
	9.0 - 9.5 Hz	2.15	2.22	1.37	0.40
	9.5 - 10.0 Hz	1.28	2.21	0.82	0.56
	10.0 - 10.5 Hz	-0.16	0.86	-0.26	0.84
	10.5 - 11.0 Hz	-0.10	0.05	-2.66	0.23
	11.0 - 11.5 Hz	-0.48	0.98	-0.69	0.61
	11.5 - 12.0 Hz	-0.79	0.15	-7.46	0.08
CZ	8.0 - 8.5 Hz	0.42	0.19	3.24	0.19
	8.5 - 9.0 Hz	1.40	0.32	6.15	0.10
	9.0 - 9.5 Hz	1.66	1.60	1.47	0.38
	9.5 - 10.0 Hz	1.22	1.98	0.88	0.54
	10.0 - 10.5 Hz	0.22	0.85	0.36	0.78
	10.5 - 11.0 Hz	0.30	0.07	6.28	0.10
	11.0 - 11.5 Hz	-0.48	0.49	-1.41	0.39
	11.5 - 12.0 Hz	-0.98	0.43	-3.25	0.19
C4	8.0 - 8.5 Hz	0.40	0.99	0.57	0.67
	8.5 - 9.0 Hz	1.23	0.39	4.48	0.14
	9.0 - 9.5 Hz	1.44	1.34	1.52	0.37
	9.5 - 10.0 Hz	1.21	1.98	0.87	0.54
	10.0 - 10.5 Hz	0.63	1.16	0.77	0.58
	10.5 - 11.0 Hz	1.28	1.11	1.63	0.35
	11.0 - 11.5 Hz	1.02	2.17	0.67	0.63
	11.5 - 12.0 Hz	-0.02	2.30	-0.02	0.99

Table 2 stranger lower: typicals

electrode	frequency range	mean	SD	t	p
C3	8.0 - 8.5 Hz	0.20	0.76	0.78	0.46
	8.5 - 9.0 Hz	0.17	1.18	0.44	0.67
	9.0 - 9.5 Hz	0.13	2.10	0.19	0.85
	9.5 - 10.0 Hz	0.47	1.57	0.90	0.40
	10.0 - 10.5 Hz	0.76	1.23	1.87	0.10
	10.5 - 11.0 Hz	0.56	1.54	1.09	0.31
	11.0 - 11.5 Hz	0.34	1.65	0.61	0.56
	11.5 - 12.0 Hz	0.67	1.66	1.20	0.26
CZ	8.0 - 8.5 Hz	-0.01	0.76	-0.05	0.96
	8.5 - 9.0 Hz	0.09	1.13	0.23	0.83
	9.0 - 9.5 Hz	0.05	1.46	0.11	0.91
	9.5 - 10.0 Hz	0.38	0.91	1.26	0.24
	10.0 - 10.5 Hz	0.46	1.21	1.15	0.28
	10.5 - 11.0 Hz	0.35	1.46	0.72	0.49
	11.0 - 11.5 Hz	0.26	1.59	0.49	0.64
	11.5 - 12.0 Hz	0.52	1.70	0.91	0.39
C4	8.0 - 8.5 Hz	-0.18	0.67	-0.80	0.45
	8.5 - 9.0 Hz	-0.07	1.25	-0.18	0.86
	9.0 - 9.5 Hz	0.11	1.79	0.19	0.85
	9.5 - 10.0 Hz	0.58	1.12	1.56	0.16
	10.0 - 10.5 Hz	0.75	1.47	1.52	0.17
	10.5 - 11.0 Hz	0.38	2.43	0.47	0.65
	11.0 - 11.5 Hz	0.44	1.63	0.81	0.44
	11.5 - 12.0 Hz	0.96	1.43	2.03	0.08

Table 3 self lower: patients

electrode	frequency range	mean	SD	t	p
C3	8.0 - 8.5 Hz	0.26	0.39	0.93	0.52
	8.5 - 9.0 Hz	-0.22	1.11	-0.28	0.82
	9.0 - 9.5 Hz	-1.01	2.05	-0.69	0.61
	9.5 - 10.0 Hz	-1.78	0.67	-3.75	0.17
	10.0 - 10.5 Hz	-2.00	0.92	-3.08	0.20
	10.5 - 11.0 Hz	-1.36	1.44	-1.33	0.41
	11.0 - 11.5 Hz	-0.25	0.54	-0.66	0.63
	11.5 - 12.0 Hz	2.93	2.89	1.43	0.39
CZ	8.0 - 8.5 Hz	-0.32	1.09	-0.41	0.75
	8.5 - 9.0 Hz	-0.72	1.73	-0.59	0.66
	9.0 - 9.5 Hz	-0.62	2.86	-0.31	0.81
	9.5 - 10.0 Hz	-0.34	2.48	-0.19	0.88
	10.0 - 10.5 Hz	0.21	4.86	0.06	0.96
	10.5 - 11.0 Hz	1.15	3.22	0.50	0.70
	11.0 - 11.5 Hz	**0.82**	**0.02**	**711.61**	**0.001**
	11.5 - 12.0 Hz	-0.43	2.48	-0.25	0.85
C4	8.0 - 8.5 Hz	0.41	1.35	0.43	0.74
	8.5 - 9.0 Hz	-0.35	2.04	-0.24	0.85
	9.0 - 9.5 Hz	-0.84	3.09	-0.38	0.77
	9.5 - 10.0 Hz	-0.22	2.55	-0.12	0.92
	10.0 - 10.5 Hz	0.31	3.76	0.12	0.93
	10.5 - 11.0 Hz	1.07	2.11	0.72	0.60
	11.0 - 11.5 Hz	0.83	1.04	1.13	0.46
	11.5 - 12.0 Hz	-0.50	1.58	-0.45	0.73

Table 4 self lower: typicals

electrode	frequency range	mean	SD	t	p
C3	8.0 - 8.5 Hz	0.42	2.20	0.57	0.58
	8.5 - 9.0 Hz	-0.05	2.30	-0.07	0.94
	9.0 - 9.5 Hz	-0.20	2.16	-0.27	0.79
	9.5 - 10.0 Hz	0.25	2.50	0.31	0.77
	10.0 - 10.5 Hz	0.77	2.68	0.86	0.42
	10.5 - 11.0 Hz	0.85	3.05	0.84	0.43
	11.0 - 11.5 Hz	1.12	2.55	1.31	0.23
	11.5 - 12.0 Hz	0.88	2.09	1.26	0.24
CZ	8.0 - 8.5 Hz	0.58	1.79	0.97	0.36
	8.5 - 9.0 Hz	-0.02	1.85	-0.04	0.97
	9.0 - 9.5 Hz	-0.48	1.80	-0.80	0.45
	9.5 - 10.0 Hz	0.03	2.36	0.03	0.97
	10.0 - 10.5 Hz	0.60	2.70	0.67	0.52
	10.5 - 11.0 Hz	0.89	2.89	0.93	0.38
	11.0 - 11.5 Hz	1.36	2.43	1.68	0.13
	11.5 - 12.0 Hz	**1.25**	**1.53**	**2.44**	**0.04**
C4	**8.0 - 8.5 Hz**	**1.14**	**1.50**	**2.28**	**0.05**
	8.5 - 9.0 Hz	0.16	2.10	0.22	0.83
	9.0 - 9.5 Hz	-0.27	2.24	-0.36	0.73
	9.5 - 10.0 Hz	0.40	2.62	0.45	0.66
	10.0 - 10.5 Hz	1.19	2.74	1.30	0.23
	10.5 - 11.0 Hz	1.47	2.66	1.65	0.14
	11.0 - 11.5 Hz	1.37	2.73	1.50	0.17
	11.5 - 12.0 Hz	1.11	1.79	1.87	0.10

Table 5 close other lower: patients

electrode	frequency range	mean	SD	t	p
C3	8.0 – 8.5 Hz	0.01	0.78	0.02	0.99
	8.5 – 9.0 Hz	0.05	2.57	0.03	0.98
	9.0 – 9.5 Hz	-0.64	2.32	-0.39	0.76
	9.5 – 10.0 Hz	-0.01	1.43	-0.01	0.99
	10.0 – 10.5 Hz	-0.45	0.58	-1.09	0.47
	10.5 – 11.0 Hz	**0.31**	**0.01**	**51.08**	**0.01**
	11.0 – 11.5 Hz	0.61	1.71	0.50	0.70
	11.5 – 12.0 Hz	-0.89	0.32	-3.90	0.16
CZ	8.0 – 8.5 Hz	-0.49	0.20	-3.48	0.18
	8.5 – 9.0 Hz	-1.39	1.57	-1.25	0.43
	9.0 – 9.5 Hz	-2.37	1.74	-1.93	0.30
	9.5 – 10.0 Hz	-0.77	0.91	-1.20	0.44
	10.0 – 10.5 Hz	-0.11	1.86	-0.08	0.95
	10.5 – 11.0 Hz	0.22	2.31	0.14	0.91
	11.0 – 11.5 Hz	-0.21	1.30	-0.23	0.85
	11.5 – 12.0 Hz	-1.28	1.31	-1.37	0.40
C4	8.0 – 8.5 Hz	-0.28	0.78	-0.50	0.70
	8.5 – 9.0 Hz	-1.76	1.07	-2.33	0.26
	9.0 – 9.5 Hz	-3.03	0.58	-7.37	0.09
	9.5 – 10.0 Hz	-1.39	0.35	-5.58	0.11
	10.0 – 10.5 Hz	-0.20	1.41	-0.20	0.87
	10.5 – 11.0 Hz	0.06	2.12	0.04	0.98
	11.0 – 11.5 Hz	-1.20	1.10	-1.54	0.37
	11.5 – 12.0 Hz	-1.63	1.84	-1.25	0.43

Table 6 close other lower: typicals

electrode	frequency range	mean	SD	t	p
C3	8.0 – 8.5 Hz	-0.13	2.10	-0.19	0.86
	8.5 – 9.0 Hz	0.17	1.80	0.29	0.78
	9.0 – 9.5 Hz	0.17	1.63	0.32	0.76
	9.5 – 10.0 Hz	0.10	1.13	0.28	0.79
	10.0 – 10.5 Hz	-0.23	1.03	-0.67	0.52
	10.5 – 11.0 Hz	-0.49	0.86	-1.72	0.12
	11.0 – 11.5 Hz	-0.27	0.46	-1.75	0.12
	11.5 – 12.0 Hz	-0.46	1.23	-1.12	0.30
CZ	8.0 – 8.5 Hz	0.32	2.29	0.42	0.69
	8.5 – 9.0 Hz	0.37	1.88	0.58	0.57
	9.0 – 9.5 Hz	-0.22	2.01	-0.32	0.75
	9.5 – 10.0 Hz	-0.20	1.11	-0.53	0.61
	10.0 – 10.5 Hz	-0.31	1.13	-0.82	0.44
	10.5 – 11.0 Hz	-0.57	1.41	-1.21	0.26
	11.0 – 11.5 Hz	-0.52	0.94	-1.65	0.14
	11.5 – 12.0 Hz	-0.59	0.92	-1.90	0.09
C4	8.0 – 8.5 Hz	0.12	2.12	0.17	0.87
	8.5 – 9.0 Hz	0.34	2.00	0.51	0.62
	9.0 – 9.5 Hz	-0.04	2.02	-0.07	0.95
	9.5 – 10.0 Hz	-0.21	1.09	-0.59	0.57
	10.0 – 10.5 Hz	-0.29	1.31	-0.66	0.53
	10.5 – 11.0 Hz	-0.41	1.81	-0.67	0.52
	11.0 – 11.5 Hz	-0.61	1.25	-1.46	0.18
	11.5 – 12.0 Hz	-0.60	1.11	-1.61	0.15

Table 7 stranger upper: patients

electrode	frequency range	mean	SD	t	p
C3	8.0 - 8.5 Hz	0.03	1.93	0.02	0.99
	8.5 - 9.0 Hz	0.15	1.36	0.16	0.90
	9.0 - 9.5 Hz	-0.61	1.32	-0.65	0.63
	9.5 - 10.0 Hz	-1.78	0.85	-2.98	0.21
	10.0 - 10.5 Hz	-1.71	0.56	-4.27	0.15
	10.5 - 11.0 Hz	0.39	1.53	0.36	0.78
	11.0 - 11.5 Hz	1.56	3.52	0.63	0.64
	11.5 - 12.0 Hz	1.63	2.96	0.78	0.58
CZ	8.0 - 8.5 Hz	-0.23	2.35	-0.14	0.91
	8.5 - 9.0 Hz	-0.14	2.37	-0.08	0.95
	9.0 - 9.5 Hz	-0.86	1.00	-1.21	0.44
	9.5 - 10.0 Hz	-1.26	0.81	-2.22	0.27
	10.0 - 10.5 Hz	-0.98	0.55	-2.54	0.24
	10.5 - 11.0 Hz	-0.43	0.11	-5.45	0.12
	11.0 - 11.5 Hz	1.18	2.50	0.67	0.63
	11.5 - 12.0 Hz	1.71	2.38	1.01	0.50
C4	8.0 - 8.5 Hz	0.75	0.49	2.16	0.28
	8.5 - 9.0 Hz	0.40	0.43	1.32	0.41
	9.0 - 9.5 Hz	-0.34	0.17	-2.84	0.22
	9.5 - 10.0 Hz	-0.50	0.45	-1.56	0.36
	10.0 - 10.5 Hz	0.11	0.58	0.26	0.84
	10.5 - 11.0 Hz	0.19	1.23	0.22	0.86
	11.0 - 11.5 Hz	0.70	1.15	0.87	0.55
	11.5 - 12.0 Hz	0.95	1.64	0.82	0.56

Table 8 stranger upper: typicals

electrode	frequency range	mean	SD	t	p
C3	8.0 - 8.5 Hz	0.86	1.22	2.12	0.07
	8.5 - 9.0 Hz	1.11	1.57	2.12	0.07
	9.0 - 9.5 Hz	0.43	1.38	0.92	0.38
	9.5 - 10.0 Hz	0.32	1.32	0.73	0.49
	10.0 - 10.5 Hz	0.10	1.48	0.20	0.85
	10.5 - 11.0 Hz	-0.29	1.50	-0.58	0.58
	11.0 - 11.5 Hz	-0.35	1.42	-0.74	0.48
	11.5 - 12.0 Hz	0.29	1.53	0.56	0.59
CZ	8.0 - 8.5 Hz	0.37	0.87	1.30	0.23
	8.5 - 9.0 Hz	0.50	1.17	1.28	0.24
	9.0 - 9.5 Hz	-0.26	1.13	-0.69	0.51
	9.5 - 10.0 Hz	-0.27	1.11	-0.74	0.48
	10.0 - 10.5 Hz	-0.32	1.08	-0.89	0.40
	10.5 - 11.0 Hz	-0.38	1.24	-0.92	0.39
	11.0 - 11.5 Hz	-0.54	1.59	-1.01	0.34
	11.5 - 12.0 Hz	-0.19	1.36	-0.42	0.69
C4	8.0 - 8.5 Hz	0.53	0.90	1.77	0.11
	8.5 - 9.0 Hz	0.47	1.39	1.02	0.34
	9.0 - 9.5 Hz	-0.25	1.63	-0.46	0.66
	9.5 - 10.0 Hz	-0.32	1.26	-0.76	0.47
	10.0 - 10.5 Hz	-0.31	1.19	-0.77	0.46
	10.5 - 11.0 Hz	-0.10	1.35	-0.22	0.83
	11.0 - 11.5 Hz	-0.12	1.66	-0.21	0.84
	11.5 - 12.0 Hz	0.04	1.02	0.13	0.90

Table 9 self upper: patients

electrode	frequency range	mean	SD	t	p
C3	8.0 - 8.5 Hz	-0.12	1.07	-0.16	0.90
	8.5 - 9.0 Hz	0.83	0.32	3.65	0.17
	9.0 - 9.5 Hz	0.57	0.51	1.58	0.36
	9.5 - 10.0 Hz	-0.16	0.23	-1.01	0.50
	10.0 - 10.5 Hz	0.04	0.24	0.22	0.86
	10.5 - 11.0 Hz	0.24	0.75	0.46	0.73
	11.0 - 11.5 Hz	-0.59	0.99	-0.83	0.56
	11.5 - 12.0 Hz	-0.21	1.23	-0.24	0.85
CZ	8.0 - 8.5 Hz	0.38	0.35	1.51	0.37
	8.5 - 9.0 Hz	0.15	0.70	0.31	0.81
	9.0 - 9.5 Hz	-0.35	1.45	-0.34	0.79
	9.5 - 10.0 Hz	-1.02	0.84	-1.70	0.34
	10.0 - 10.5 Hz	-1.10	0.27	-5.68	0.11
	10.5 - 11.0 Hz	-0.49	0.93	-0.73	0.60
	11.0 - 11.5 Hz	-0.53	1.14	-0.66	0.63
	11.5 - 12.0 Hz	-0.03	0.63	-0.07	0.95
C4	8.0 - 8.5 Hz	0.26	0.39	0.93	0.52
	8.5 - 9.0 Hz	-0.22	1.11	-0.28	0.82
	9.0 - 9.5 Hz	-1.01	2.05	-0.69	0.61
	9.5 - 10.0 Hz	-1.78	0.67	-3.75	0.17
	10.0 - 10.5 Hz	-2.00	0.92	-3.08	0.20
	10.5 - 11.0 Hz	-1.36	1.44	-1.33	0.41
	11.0 - 11.5 Hz	-0.25	0.54	-0.66	0.63
	11.5 - 12.0 Hz	2.93	2.89	1.43	0.39

Table 10 self upper: typicals

electrode	frequency range	mean	SD	t	p
C3	8.0 - 8.5 Hz	0.57	1.13	1.51	0.17
	8.5 - 9.0 Hz	0.55	1.24	1.32	0.22
	9.0 - 9.5 Hz	0.61	1.42	1.30	0.23
	9.5 - 10.0 Hz	0.09	1.26	0.21	0.84
	10.0 - 10.5 Hz	-0.08	1.63	-0.14	0.89
	10.5 - 11.0 Hz	0.62	1.92	0.97	0.36
	11.0 - 11.5 Hz	1.02	1.53	1.99	0.08
	11.5 - 12.0 Hz	0.48	2.46	0.58	0.58
CZ	**8.0 - 8.5 Hz**	**0.84**	**0.99**	**2.56**	**0.03**
	8.5 - 9.0 Hz	**0.64**	**0.73**	**2.65**	**0.03**
	9.0 - 9.5 Hz	0.56	1.29	1.31	0.23
	9.5 - 10.0 Hz	0.16	0.92	0.52	0.61
	10.0 - 10.5 Hz	0.09	0.97	0.28	0.78
	10.5 - 11.0 Hz	**0.87**	**0.98**	**2.65**	**0.03**
	11.0 - 11.5 Hz	**1.50**	**1.11**	**4.05**	**0.01**
	11.5 - 12.0 Hz	1.01	1.64	1.85	0.10
C4	8.0 - 8.5 Hz	0.89	1.22	2.19	0.06
	8.5 - 9.0 Hz	**0.82**	**0.81**	**3.04**	**0.02**
	9.0 - 9.5 Hz	0.66	1.37	1.45	0.18
	9.5 - 10.0 Hz	0.28	1.18	0.72	0.49
	10.0 - 10.5 Hz	0.03	1.22	0.07	0.95
	10.5 - 11.0 Hz	0.59	1.27	1.40	0.20
	11.0 - 11.5 Hz	**1.07**	**1.38**	**2.34**	**0.05**
	11.5 - 12.0 Hz	1.06	1.59	1.99	0.08

Table 11 close other upper: patients

electrode	frequency range	mean	SD	t	p
C3	8.0 – 8.5 Hz	0.26	1.75	0.21	0.87
	8.5 – 9.0 Hz	0.17	0.46	0.51	0.70
	9.0 – 9.5 Hz	**0.23**	**0.01**	**72.74**	**0.01**
	9.5 – 10.0 Hz	0.16	2.30	0.10	0.94
	10.0 – 10.5 Hz	-0.12	2.80	-0.06	0.96
	10.5 – 11.0 Hz	-0.05	1.57	-0.05	0.97
	11.0 – 11.5 Hz	0.59	0.86	0.98	0.51
	11.5 – 12.0 Hz	-0.18	0.09	-2.74	0.22
CZ	**8.0 – 8.5 Hz**	**1.46**	**0.02**	**87.94**	**0.01**
	8.5 – 9.0 Hz	0.67	1.49	0.63	0.64
	9.0 – 9.5 Hz	0.26	1.10	0.34	0.79
	9.5 – 10.0 Hz	-0.46	2.38	-0.27	0.83
	10.0 – 10.5 Hz	-2.10	2.32	-1.28	0.42
	10.5 – 11.0 Hz	-2.30	1.51	-2.15	0.28
	11.0 – 11.5 Hz	-0.48	0.70	-0.96	0.51
	11.5 – 12.0 Hz	-0.16	0.68	-0.34	0.79
C4	8.0 – 8.5 Hz	0.68	1.98	0.49	0.71
	8.5 – 9.0 Hz	-0.27	2.27	-0.17	0.90
	9.0 – 9.5 Hz	-0.06	1.50	-0.06	0.96
	9.5 – 10.0 Hz	-0.86	2.22	-0.54	0.68
	10.0 – 10.5 Hz	-2.41	2.45	-1.39	0.40
	10.5 – 11.0 Hz	-2.19	0.64	-4.83	0.13
	11.0 – 11.5 Hz	0.06	0.81	0.10	0.93
	11.5 – 12.0 Hz	0.41	0.54	1.07	0.48

Table 12 close other upper: typicals

electrode	frequency range	mean	SD	t	p
C3	8.0 – 8.5 Hz	0.45	1.54	0.87	0.41
	8.5 – 9.0 Hz	0.72	1.39	1.56	0.16
	9.0 – 9.5 Hz	0.45	1.92	0.71	0.50
	9.5 – 10.0 Hz	1.16	1.61	2.15	0.06
	10.0 – 10.5 Hz	**1.60**	**1.53**	**3.14**	**0.01**
	10.5 – 11.0 Hz	**1.05**	**1.29**	**2.43**	**0.04**
	11.0 – 11.5 Hz	**0.87**	**1.02**	**2.56**	**0.03**
	11.5 – 12.0 Hz	0.38	0.92	1.25	0.25
CZ	8.0 – 8.5 Hz	0.33	1.47	0.68	0.51
	8.5 – 9.0 Hz	0.66	1.20	1.65	0.14
	9.0 – 9.5 Hz	0.49	1.10	1.34	0.22
	9.5 – 10.0 Hz	**1.22**	**1.29**	**2.82**	**0.02**
	10.0 – 10.5 Hz	**1.38**	**1.18**	**3.52**	**0.01**
	10.5 – 11.0 Hz	0.78	1.33	1.76	0.12
	11.0 – 11.5 Hz	0.83	1.28	1.96	0.09
	11.5 – 12.0 Hz	0.28	0.87	0.98	0.35
C4	8.0 – 8.5 Hz	0.07	1.45	0.15	0.88
	8.5 – 9.0 Hz	0.51	1.17	1.31	0.23
	9.0 – 9.5 Hz	0.59	1.11	1.60	0.15
	9.5 – 10.0 Hz	**1.14**	**1.26**	**2.70**	**0.03**
	10.0 – 10.5 Hz	**1.16**	**1.41**	**2.46**	**0.04**
	10.5 – 11.0 Hz	0.37	2.37	0.46	0.66
	11.0 – 11.5 Hz	**0.92**	**1.17**	**2.35**	**0.05**
	11.5 – 12.0 Hz	0.26	0.96	0.79	0.45

Figure Captions

Figure 1 Examples of stimuli. On the right, the top picture is lower painful stimuli, the bottom lower nonpainful. On the left, the top picture is upper painful stimuli, the bottom upper nonpainful.

Figure 2 Depiction of the Extremity x neurological status interaction at electrode C4, 10.0 – 10.5 Hz. Blue represents the lower extremities, green the upper extremities

Figure 3 Depiction of the extremity x neurological status interaction at electrode C4, 11.5 – 12.0 Hz. Blue represents the lower extremities, green line upper extremities.

Figure 4 Depiction of the extremity x neurological status interaction at electrode CZ, 10.0 – 10.5 Hz. Blue represents the lower extremities, green the upper extremities

Figure 1

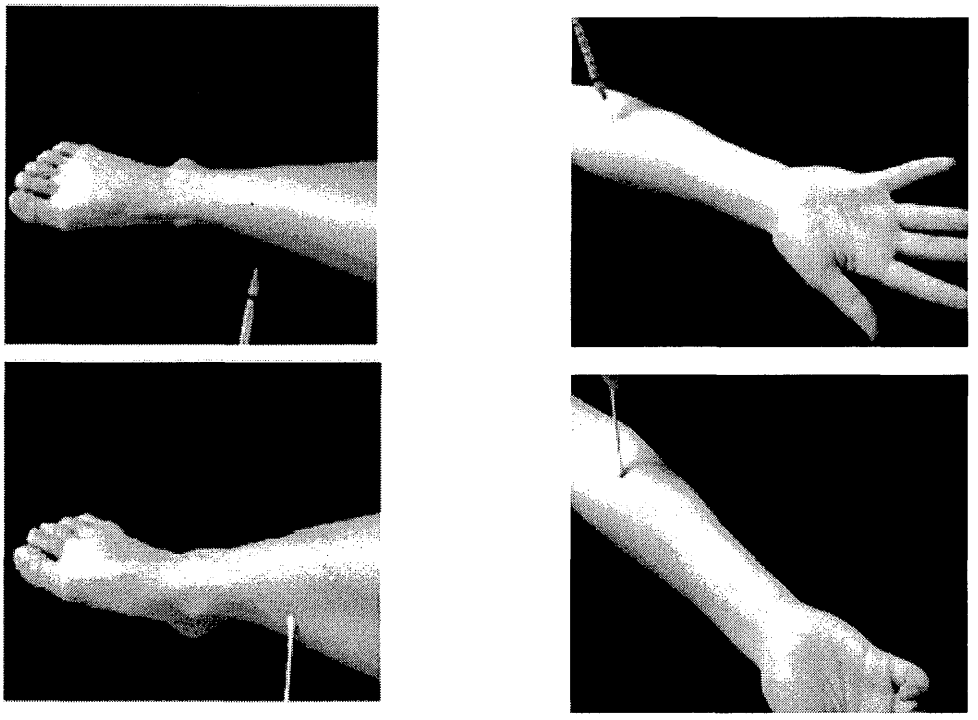


Figure 2

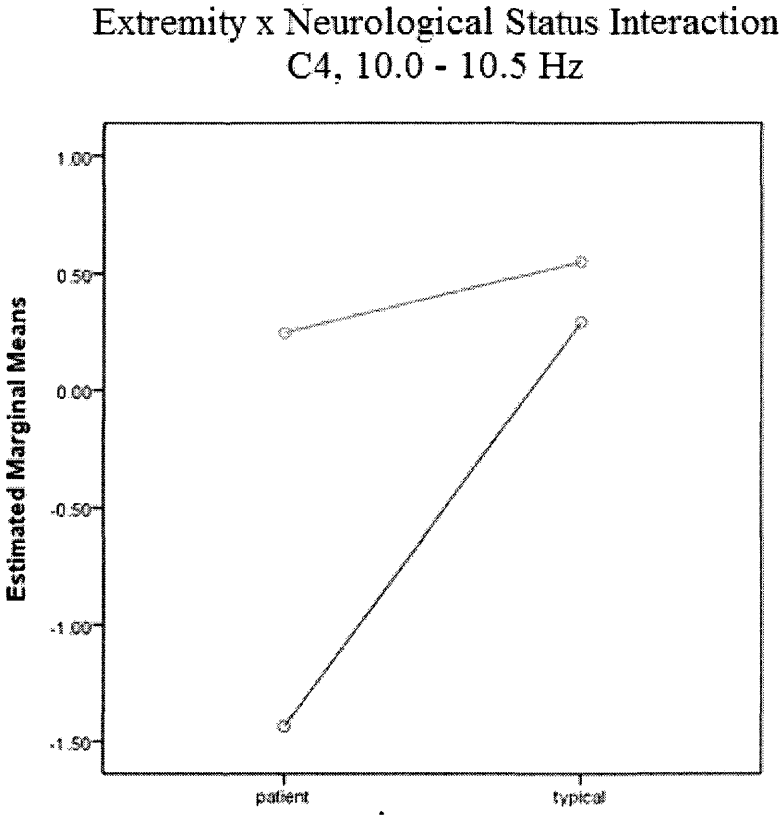

Extremity x Neurological Status Interaction
C4, 10.0 - 10.5 Hz
Estimated Marginal Means
1.00
0.50
0.00
-0.50
-1.00
-1.50
patient
typical

Figure 3

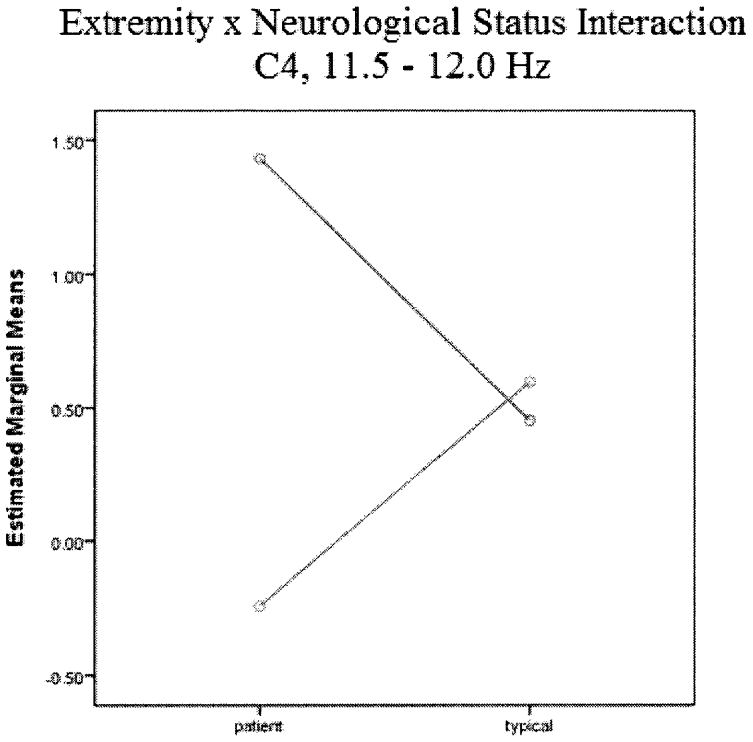
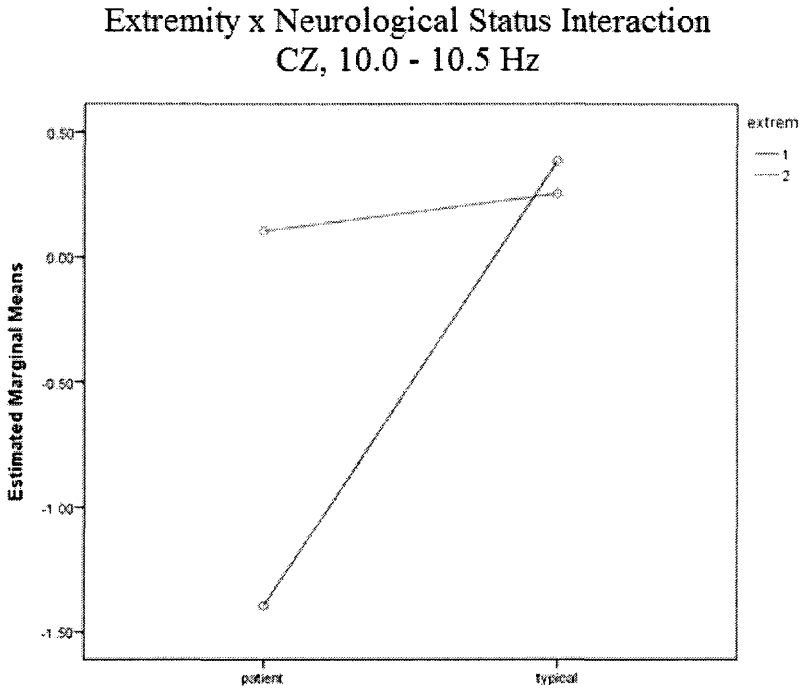

Extremity x Neurological Status Interaction
C4, 11.5 - 12.0 Hz
Estimated Marginal Means
1.50
1.00
0.50
0.00
-0.50
patient
typical

Figure 4


Extremity x Neurological Status Interaction
CZ, 10.0 - 10.5 Hz
Estimated Marginal Means
0.50
0.00
-0.50
-1.00
-1.50
patient
typical
extrem
1
2