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REFLEX SYMPATHETIC DYSTROPHY: A TASK-RELATED  
ELECTROENCEPHALOGRAPHIC ANALYSIS  
IN CHRONIC PAIN PATIENTS

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A Thesis  
Presented to the  
Faculty of  
California State University,  
San Bernardino

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In Partial Fulfillment  
of the Requirements for the Degree  
Master of Arts  
in  
Psychology

---

by  
Mark Alan Clair  
December 1996

REFLEX SYMPATHETIC DYSTROPHY: A TASK-RELATED  
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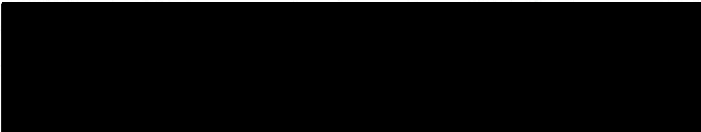
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A Thesis  
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by  
Mark Alan Clair  
December 1996

Approved by:

  
Frederick A. Newton Ph.D., Chair, Psychology

  
Kurt Bickford Ph.D., Clinical Psychologist

  
Hideya Koshino Ph.D., Psychology

11/1/96  
Date

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## **ABSTRACT**

The purpose of this study was to assess the EEG correlates of chronic pain. A total of twenty-six adults were randomly assigned to either a no-pain group, a chronic pain group, or a Reflex Sympathetic Dystrophy (RSD) group. EEG's were recorded from 19 scalp electrodes during eyes-open and eyes-closed baselines, two cognitive tasks, and a Cold Water Pressor Task (CWPT). Alpha and beta amplitudes were used as the dependent measure. As expected, eyes-closed alpha amplitude was significantly lower in the pain groups as compared to the control group. The differences in alpha amplitude between the groups and the hemispheric differences observed during tasks, were discussed in terms of use for possible diagnosis and treatment.

## **ACKNOWLEDGMENTS**

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## INTRODUCTION

### Reflex Sympathetic Dystrophy: A Task-Related Electroencephalographic Analysis in Chronic Pain Patients

As humans we all experience pain to some extent. However, for some, intense chronic pain is part of everyday life. Chronic pain is multifarious in form and etiology, making it extremely difficult to study and examine. One such example of the multifaceted nature of chronic pain was discovered by Anzio Beachhead in World War II (Beecher, 1956). He found that injured soldiers, when questioned about their pain, responded in an unexpectedly calm manner. Amazingly, a relatively small percentage of these soldiers asked for medication to relieve their pain. Whereas, patients in non-war settings with similar injuries when questioned about their pain, responded that it was severe and many requested medication (Suter, 1986). Beachhead proposed that the soldiers' calmness was the result of being removed from the battle field producing such a relief that the pain was compensated for. This interpretation was named the "Anzio Effect" and clearly demonstrated that the connection between tissue damage and pain is not rigid and one-dimensional but may in fact involve psychological factors (Suter, 1986).

Melzack and Casey (1968) view pain as a phenomenon that consists of characteristics such as cognition, motivation, and arousal. Some believe pain to be intertwined between sensory and affective processing, causing the painful experience to also be an emotional one (Chapman & Gavrin, 1993). The characteristics of pain may become worse when the duration of pain extends for a surmountable time and the pain is no longer the result of a specific injury. Under these conditions, psychological attributes may result from the pain (Nursing 84 Books, 1984).

The treatment of chronic pain is a vexing problem because there appears to be many different cures, the symptoms sometimes occur many years after the initial injury, and placebo studies often show to be as effective as medication (Suter, 1986). Dorsel (1989) effectively revealed the complicated nature of chronic pain when he stated, "Indeed, the reality of the pain is not the issue. The issue is the source of the pain, its etiology, how to relieve it, and how to prevent it from recurring" (p. 783).

Dorsel (1989) has developed a theoretical model to explain the etiology of chronic pain and he suggests that there is a chronic pain behavior pattern. Initially, he explains that individuals who live with chronic pain seem to be those who have lived painful lives. These individuals



seem to be unassertive, dependent, afraid, helpless and unskilled (Dorsel, 1989). Therefore, the pain sufferer, like many individuals, is burdened by painful events in their lives. However, for them it seems as though they are incapable of releasing their pain due to the personality traits previously described. According to Dorsel's (1989) theory, this psychological pain that accumulates may release itself as a physical injury, thereby providing an acceptable escape for the pain. In summary, the theory suggests that after a minor injury occurs, the sympathetic nervous system responds in a heightened degree and therefore cannot be inhibited because of the years of defensive energy that bursts out as an overreaction to the injury (Dorsel, 1989). According to this model, this may be the reaction seen with those experiencing a type of chronic pain known as Reflex Sympathetic Dystrophy (RSD).

Mainly affecting the extremities, RSD can become debilitating. It is characterized by causalgia (Buchta, 1983), edema, vasomotor instability, dystrophic skin changes, limitation of movement, allodynia (Price, Long, & Huitt, 1992), and dystonic muscle spasms (Gainer, 1992 ; Schwartzman & McLellan, 1987).

One psychological view has focused on a possible relationship between RSD and helplessness. Houdenhove and

Vasquez (1993) suggest that helplessness, in susceptible patients, may cause an overreaction of the sympathetic system and cause the symptoms of RSD to be maintained. Furthermore, helplessness may initiate certain coping strategies leading to the disuse of an effected limb, or the RSD symptoms may be a result of the helplessness. These biopsychosocial approaches are in their infancy, although they may add to the multidisciplinary approach of helping chronic pain sufferers.

Gainer (1992) has shown that hypnosis is a valuable tool in relieving the pain associated with RSD. He demonstrated, by using hypnotic interventions on three individuals, that all three could lower the discharge of the sympathetic system causing a vasodilation of the infected area and resulting in the alleviation of pain. This leads to a more symptomatic treatment for RSD such as biofeedback. RSD sufferers, such as the patient discussed by Blanchard (1979), first undergo thermal biofeedback training directed to increase the temperature of the infected appendage. This increase in the temperature of the affected area was shown to be effective for attenuating the pain associated with RSD. However, this was only a one-patient case study and an experimental design was not implemented.

Hooshmand (1993) stresses that individuals with RSD must be diagnosed early, and the key to successfully treating RSD is to place the patient in a strict, physiotherapy regimen as early as possible. This therapy should consist of encouraging the patient to become active and to overcome the fear of exercising the painful appendage. Physiotherapy, when used frequently, can substantially increase movement of the ailing appendage for the RSD sufferer. However, the patient's foremost concern appears to be the persistent, burning pain and understanding why it is occurring.

Painful stimuli are transmitted through more than one pathway and because of this there appears to be different types and intensities of pain. Hooshmand (1993) emphasizes two very different pathways for pain. First, the lateral afferent system is responsible for somatic pain. Secondly, the medial afferent system is responsible for sympathetic pain. The somatic system, using large A and C fibers, appears to be focalized, noticed as a sharp pain of short duration, and terminates in the contralateral sensory cortex. In contrast, the sympathetic system, using small C fibers, appears to be very wide in range (i.e. referred pain), noticed as a dull pain of long duration, and

terminates in the bilateral prefrontal and limbic areas (Hooshmand, 1993).

Because RSD shares many of the characteristics of sympathetic pain, it is categorized as a sympathetically maintained pain (SMP). Some researchers believe that sympathetic pain is any class of pain that can be sympathetically blocked and therefore attenuated (Treede et al., 1991). Many surgical procedures have been performed in order to relieve SMP. Some of these procedures include ganglion blocks, stellate blocks, and sympathectomies. Calcium channel blockers as well as central and peripheral  $\alpha$ -receptor blockers have been successful in lessening pain (Hooshmand, 1993). However, repeated blockades using these drugs must be performed in most cases and often the relief is only temporary. Contrary to the benefits of sympathetic blockades, some research has suggested that these blockades do not produce pain relief. For example, Clonidine, which interrupts sympathetic transmission, has shown not to affect sympathetic pain (Glynn & Jones, 1990). Verdugo and Ochoa (1994) discovered that administering phentolamine resulted in 9.2% of the patients reporting a relief of pain; whereas the administration of a placebo resulted in 28.9% of the patients reporting diminished pain. In the second experiment, Verdugo and Ochoa (1994) found that by using

three experimental groups, one receiving phentolamine, one receiving phenylephrine, and one receiving a placebo, the placebo and the phenylephrine groups were identical resulting in 17.3% of the patients reporting an alleviation of pain. However, only 8.7% of the phentolamine group reported diminished pain. These studies demonstrate that the placebo effect is very prominent when it comes to drugs that are used to control sympathetic pain.

In summary, some sympathetic nerve blocks initially relieve pain but the effect is often short-lived. King and Nuss (1993) investigated one such incident in which a 32-year-old woman with RSD, who was no longer being aided by sympathetic blocks, underwent electroconvulsive therapy (ECT). Surprisingly, the patient was relieved of her pain completely. This finding brought about a new scientific perspective in regard to RSD. Traditionally, it was thought that the dysfunctioning responsible for RSD occurred between the nerve injury and the spinal cord, including the sensory and autonomic reflex pathway loops (King & Nuss, 1993). However, in using bilateral ECT, electrical impulses are passed through the diencephalon, containing the thalamus and hypothalamus, (Abrams & Taylor, 1976) and higher cortical areas such as the frontoparietal cortex (Canavero et al. 1993). All of these areas are penetrated by pathways

responsible for the sensation and perception of pain.

Because the ECT seems to involve higher cortical areas than were believed to be applicable for RSD, it may be that the perception of pain is being exaggerated on the cortical level.

Pain has been extremely difficult to study at the cortical level because it appears that there are many pain pathways. Melzack's (1991) "neuromatrix" concept lists three neural pathways in which pain appears to travel. One such pathway passes through the thalamus to the somatosensory cortex. The second pathway passes through the reticular formation of the brain stem to the limbic system, and a third seems to be a neural pathway responsible for relaying the information to the higher cortical regions. According to Melzack (1991), these pathways each have a distinctive component in relation to pain. The pathway projecting to the somatosensory cortex has sensory-discriminating characteristics. It is suggested that the intensity and duration of the nociceptive stimulus is analyzed in this region (Guilbaud, Bernard, & Besson, 1994). The motivational component seems to be linked with the pathway involving the limbic system. The last pathway appears to add a cognitive component in that attention and past experiences are analyzed in the frontal cortex

(Guilbaud, Bernard, & Besson, 1994). These researchers conclude that these differing pain pathways may be the reason for ineffective neurosurgical lesions. If neurosurgical lesions are created in one of these pathways, the pain may not be abated due to the possibility of pain continuing to be transmitted via alternate pathways.

Kolb and Whishaw (1995) have studied the role of the sensory pathway for pain which leads to the somatosensory cortex. Nociceptors which are sensitive to mechanical and thermal stimuli ascend to the dorsal horn of the spinal cord. The nerve cells in this area then contralaterally traverse to the spinal cord to form the spinothalamic tract which terminates in the ventrobasal thalamus and posterior thalamus. These nuclei project to the somatosensory cortex which is divided into specific areas that are represented by particular types of body receptors.

Using the concept of this neural pain pathway, researchers have been investigating the nature of neuropathic pain. Bennett and Xie (1988), using ligatures applied around the sciatic nerve of a rat, were successful in producing a peripheral unilateral mononeuropathy. Behaviors such as hyperalgesia and allodynia were found to result from thermal and mechanical stimuli after this procedure was performed. Specifically, two very noticeable



factors were discovered. Firstly, neuronal responses to mechanical and thermal stimuli were intensified in the thalamus and cortical regions. Secondly, the somatic inputs to the somatosensory cortex appeared to become reorganized with an increasing abundance of cutaneous inputs and joint inputs being developed which has been confirmed by many researchers (Guilbaud, Benoist, Jazat, & Gautron, 1990; Guilbaud, Benoist, Levante, Gautron, & Willer, 1992).

According to current research, (Hooshmand, 1993) if pathological pain such as RSD is the result of nerve trauma causing scar formation and the disruption of the insulating effects of myelin, C fibers may be involved in nonspecific mechanosensitive ectopic discharges which result in ephaptic cross-talk. This cross-talk may be representative of an electrical short between the small C fibers of the medial pain pathway and the large A fibers of the lateral pain pathway resulting in an exaggeration of the pain, vasoconstriction, and a noticeable increase in bioelectric activity in the somatosensory cortex. In addition, the factors of C fibers backfiring (Hooshmand, 1993) as well as the chemical change in the environment around the nociceptors, which may cause them to respond to normally inoffensive stimuli (Chapman & Gavrin, 1993), also cause an exaggeration of pain. Because the large A fibers are now

seemingly intertwined with the C fibers, there appears to be a diffuse stimulation of pain inputs into the spinal cord which Hooshmand (1993) believes to be the cause of the allodynia and hyperpathia characteristics.

Therefore, when a nerve is damaged and the cross-talk between pain pathways occurs, the result may be a reorganization of the inputs to the somatosensory cortex. Basing this theory upon the idea that sensations such as pain are representative of the cell population that is obtained during stimulation of the skin, Carter-Wilson (1991) theorized that this reorganization may result in a neural over-representation of the skin regions adjacent to the nerve injury. Therefore, instead of the nerve injury itself causing a small amount of pain, the entire appendage appears to send pain inputs to the cortex, thereby causing an increase in pain. If this is true then an electroencephalographic analysis may reveal how upper extremity chronic pain and especially RSD is affecting the cortical regions of the brain.

A technique that researchers are currently using to identify how the brain processes noxious information is the electroencephalogram (EEG). The EEG appears to be the result of synchronized variations in the membrane potentials of large numbers of neurons in the cerebral cortex (Duffy,

Iyer, & Surwillo, 1989). These potentials are defined as the summation of inhibitory postsynaptic potentials and excitatory postsynaptic potentials in the dendrites of the vertically oriented, cortical, pyrimidal neurons located in the top layer of the cerebral cortex ( Duffy, Iyer, & Surwillo, 1989). The summated electrical potentials are then amplified by electrodes placed on the scalp (Brown, 1972) and organized according to the International 10-20 System (See Appendix A). After being amplified, these signals are filtered and processed using a computer program that can then store the data for off-line processing. One observable factor detected by the EEG is brain wave frequency. These frequencies can be illustrated by different colors and represented by topographical maps. For example, quantitative electroencephalography (QEEG), which is represented by topographical mapping, can detect a particular high frequency rhythm which may be correlated to the performance of a particular task. The region producing this particular rhythm can then be identified as the approximate area responsible for processing the particular information (Wong, 1991).

Brain waves have been identified and categorized according to their frequency and amplitude. The frequency of a brain wave is determined in cycles per second and given

the term hertz (Hz). The amplitude, or strength, of a brain wave is determined by micro volts ( $\mu\text{V}$ ). Using these two criterion, four main types of brain waves have been determined. Starting from the lowest frequency wave, Andreassi (1989) mentions the delta rhythm as occurring at a rate of 0.5 to 3.5 Hz and having a magnitude of 20 to 200 micro volts. This wave is characteristic of deep sleep or activity observed in a region due to a tumor. The theta rhythm which is slightly higher in frequency (4.0 - 7.0 Hz) and fairly high in amplitude (20 to 100  $\mu\text{V}$ ) is representative of a very relaxed state. The rhythm that has been most linked to relaxation and most commonly observed in a participant who is sitting quietly in a relaxed manner with their eyes closed is alpha. This wave form occurs at the rate of 8 to 13 Hz and reveals a magnitude of 20 to 60  $\mu\text{V}$ . The highest frequency waves are those of the beta rhythm. These wave forms are very irregular in shape and exemplify a frequency between 14 to 30 Hz and an amplitude between 2 to 20  $\mu\text{V}$ . This frequency band has been characterized as an irregular rhythm which is evident during mental or physical activity (Andreassi, 1989). These four frequency bands are dependent upon the participant's state of arousal during the recording. For example, if a participant is very relaxed with their eyes closed they are

probably exhibiting an alpha rhythm. However, if they are asked to open their eyes the alpha rhythm will be blocked (Andreassi, 1989). Alpha blocking is defined as the change from low frequency, high amplitude waves to low amplitude, high frequency waves that are more characteristic of beta waves.

In studying EEG changes due to noxious stimuli, one must consider the arousal level of the individual during the procedure. Veerasarn and Stohler (1992) conducted a study in which each participant was administered one of two saline solutions. Some received a hypertonic saline which produced a painful sensation, while other participants received an isotonic saline which did not produce a painful sensation. It was found that the EEG recording did not significantly differentiate between those who were administered the painful and non-painful salines. Therefore, there was little difference between the EEG recordings of those with real pain and those with imagined pain. This implies that an EEG recording is sensitive to other stimuli such as the perception of pain and all efforts must be made to control the environment around the participant undergoing the EEG.

Many studies, using vast measures to control the experimental environment, such as Chen, Dworkin, Haug, and Gehrig's (1989b) study, have shown that the action of pain

can be viewed using topographical brain mapping. Evidence has also suggested that cortical and subcortical activity due to somatic inputs can be analyzed via vertex potentials recorded from electrodes placed on the scalp (Velasco, Velasco, & Olvera, 1985). Chen and Rappelsberger (1994) have documented that a pain state causes an increase in beta activity in the central region of the brain (e.g. parietal lobe). Specifically, the examination of the area containing the somatosensory cortex (C3, CZ, C4) may reveal an enhanced beta activity because of the noxious processing occurring in the somatosensory cortex (Chen & Rappelsberger, 1994). Studies using magnetoencephalography (MEG) recordings, which are exempt from the interference of the skull or the skin, also confirm the idea that pain inputs can be detected from the somatosensory cortex (Wong, 1991).

To examine the effects of pain on EEG recordings, the first study evaluated differences in brain wave activity between a control group, a chronic pain group, and an RSD group during two baseline measures. Based on the research by Chen and Rappelsberger (1994), which demonstrated that a pain state is characterized by enhanced beta activity in the central regions of the brain, it is assumed that EEG recordings will reveal an increase in beta and therefore a decrease in alpha in the chronic and RSD groups.

Specifically, during the baseline measure of eyes-closed condition, it is hypothesized that the no pain group (Group 1) will show significantly higher alpha amplitude than the chronic pain group (Group 2) and the RSD group (Group 3). In addition, during the eyes-closed condition, it is expected that a significantly higher beta amplitude will be evident in Group 2 and 3 than in Group 1. When all three groups open their eyes an alpha block would be expected because of the shift in arousal state. However, the magnitude of this alpha block may differ between the groups due to the painful stimuli experienced by Groups 2 and 3. It is hypothesized that by subtracting eyes-open alpha amplitude from the eyes-closed alpha amplitude, that Group 1 will show a significantly larger amplitude difference than Group's 2 and 3.

The purpose of the second study was to investigate the possible hemispheric differences between the three groups in regard to cognitive tasks. Previous research has shown that chronic pain patients often resemble normal individuals in regard to hemispheric activation. Specifically, chronic pain and no pain individuals tend to have a decrease in alpha activity in the left hemisphere during mathematical tasks; while visual-spatial tasks tend to elicit a decrease in alpha activity in the right hemisphere (De Benedittis & De



Gonda, 1985). It is proposed that Group 1 and Group 2 will reveal distinct contralateral decreases in alpha activity, compared to the eyes-open baseline, in the respective hemisphere depending on the task. However, due to the possible reorganization of inputs in the somatosensory cortex it is hypothesized that a bilateral decrease in alpha activity will be evident in Group 3 regardless of task.

Study 3 will attempt to identify EEG differences between participants undergoing a simplified Cold Water Pressor Test (CWPT). The CWPT has been a common technique used to identify the brain wave patterns of individuals under noxious stimulation. Backonja et al. (1991) showed that cold water stimulation caused a decrease in alpha in the central regions of the brain in healthy individuals. He relates this information to the possible activation of the somatosensory cortex. Similarly, researchers (Ferracuti, Seri, Mattia, & Crucco, 1994) also using healthy individuals free of pain, found a decrease in alpha activity in the somatosensory cortical area. Using immersion of the hand, Ferracuti et al. (1994) noted that attenuated alpha activity was evident in the contralateral hemisphere and seemed to be even more evident during right hand stimulation. It is hypothesized that Group 1 and Group 2 will show a contralateral decrease in alpha compared to the eyes-open

baseline measure in the parietal cortical areas, particularly the somatosensory region. However, Group 3, because of the possible reorganization and over representation of inputs to the somatosensory cortex, will reveal more of a bilateral decrease in alpha activity in the somatosensory regions.

## METHOD

### Participants

Twenty-six participants (F = 21 and M = 5) with a mean age of 39.3 years (SD = 9.2 years) were placed into three groups based upon specific criterion. The criteria for the control group was no history of chronic pain and no presence of pain during the procedure. Criteria for the chronic pain group was a diagnosis of chronic pain for at least one year and the RSD group was determined by a diagnosis received by their primary physician. Each participant took part in six experimental conditions. These included: eyes-open baseline, eyes-closed baseline, serial seven, a visual-spatial task using the Bender-Gestalt cards, and the CWPT used on both hands. All were naive to the experimental design; however, some patients from the chronic and RSD groups had experienced a limited amount of electromyography (EMG) biofeedback prior to participation in the present study.

The control group (F = 6 and M = 2) consisted of university students acquired from Cal State University San Bernardino (CSUSB) with a mean age of 35.6 years (SD = 10.8 years). This group consisted of only one right handed individual. All in this group had no history of chronic pain

and were not currently feeling pain at the time of the experiment.

Prior to the present study, upper extremity surgery had been performed on nine participants (F = 9 and M = 0) with a mean age of 42.1 years (SD = 7.4 years). After the surgery, these participants began to experience chronic pain in the afflicted areas. These participants were referred by orthopedic surgeons in Southern California who diagnosed them as having chronic pain for at least one year. Seventy-eight percent of this group was right handed and labeled their average pain level according to a 0 to 10 analog scale as 5.6 (SD = 2.2) and their duration of pain as 4.2 years (SD = 2.7 years). All characteristics represented by this group are shown in Table 1.

Nine additional participants, diagnosed with upper extremity RSD by their orthopedic surgeons in Southern California, were included in the study. The RSD group (F = 6 and M = 3) with a mean age of 39.9 years (SD = 9.2 years) consisted of all right handed individuals. RSD diagnosis was based upon criterion outlined by Buchta (1983) and inclusion of each participant according to these criteria are outlined in Table 2. This group labeled their average pain level according to a 0 to 10 analog scale as 6.4 (SD = 2.5) and the average duration of pain was 2.5 years (SD = 1.8 years).

Table 1  
Chronic Pain Group Medical History Summary

Pat#	Age	Sex	Hand	Surgery	Location	Duration	Severity
1	46	F	R	cervical disk herniation	neck & shoulders	1 yr, 9 mos	6
2	43	F	L	L5-S1 laminotomy & disectomy	lower back & shoulders	3 yrs, 5 mos	4
3	35	F	R	L5-S1 laminotomy & disectomy	lower back	6 yrs	8
4	46	F	L	left & right carpal tunnel release	right hand, elbow & forearm	10 yrs	6
5	55	F	R	right carpal tunnel release & alleviation of tenosynovitis in right thumb	right thumb	1 yr, 3 mos	3
6	44	F	R	ORIF of nonunion of a distal ulna fracture on left side	left hand & arm	1 yr, 8 mos	9
7	44	F	R	right & left first rib resections	both arms & hands	3 yrs	3
8	36	F	R	right & left carpal tunnel release	right & left hands	5 yrs	4

Table 2.  
RSD Group Medical History Summary

Pat#	Age	Sex	Hand	Surgery	Location	Duration	Severity	Pain Type	Physical Signs of RSD
1	37	M	R	none	left hand	6 mos	2	B	E, V, M, L, DM
2	35	F	R	none	left shoulder, arm & neck	1 yr	7	A	M, L, A
3	45	F	R	none	right hand & arm	4 yrs, 5 mos	3	A, T	E, V, L
4	48	M	R	anterior c-5/6+ c6-7 discectomy	left arm, shoulder & neck	2 yrs	7	A, T	E, D, L
5	30	M	R	a microvascular technique	left hand & arm	1 yr, 5 mos	9	B, A, T	E, V, M, L, A
6	58	F	R	closed reduction of right wrist	right wrist	7 mos	6	N/A	N/A
7	29	F	R	N/A	entire right arm	4 yrs, 6 mos	7	B, A, T	E, V, D, M, L, A
8	40	F	R	N/A	left arm & left facial side	5 yrs	9	N/A	N/A
9	37	F	R	N/A	left hand & arm	2 yrs, 6 mos	8	N/A	N/A

**Pain Types**

B= Burning A= Aching T= Throbbing

**Physical Signs Key**

E=Edema V=Vasomotor Instability D=Dystrophic Skin Changes  
 A=Allodynia L= Limitation of Movement DM=Dystonic Muscle Spasm  
 M=Mechanical Hyperalgesia N/A=Information Not Available

All participants gave informed consent (See Appendix B) of the procedure, approved by the Institutional Review Board at CSUSB, and were notified about their right to be excused from the study at any time. In the case of the RSD group, one individual did exercise the right to be excused from one of the tasks. All participants were treated in accordance with the "Ethical Principles of Psychologists and Code of Conduct" (American Psychological Association, 1992).

#### Apparatus

A personal history questionnaire was used for participant data collection and criterion fulfillment. It consisted of questions regarding the participants age, gender, handedness, location of pain currently being experienced, duration of this pain, and the severity of the pain according to a 0 to 10 Analog scale (See Appendix C).

Scalp recorded bioelectrical activity was obtained using a cap, consisting of 19 surface electrodes pre-positioned according to the International 10-20 System with reference to the linked ear lobes, which was placed upon the participant's head. Using a referential montage, even electrode numbers represented the right side of the head whereas odd numbered electrodes denoted the left side of the head. Those electrodes placed in the middle of the head were



indicated by a Z (See Appendix A). EEG was displayed upon a video screen and stored for off-line processing using a 24-channel Lexicor Neurosearch 24 electroencephalograph (model # PWR-B). After recording, all brain waves were analyzed using a fast Fourier transformation. This transform processed the amount of magnitude detected by each of the 19 electrode sites which was used as the dependent variable in all conditions. A sample rate of 256 Hz was used with the lowpass filter set at 64 Hz and the highpass filter set at .5 Hz. Therefore, all data was recorded between .5 and 64 Hz with a gain set at 32,000. A 60 Hz notch filter was also used in order to eliminate outside electrical interference within this frequency.

### Procedure

All twenty-six participants were selected from their group in random order so that a practice effect due to the setup would not be encountered. Each participant filled out the personal history questionnaire just before the EEG recordings began. The procedure began by introducing each participant to the setup process and equipment involved. All attempts were made to comfort and familiarize each participant with the procedure in which they would be involved. The cap was placed on the head and all electrodes

were carefully positioned and tested for conductivity. After setup was complete, the participant was able to view their brain wave activity for a brief time. This viewing demonstrated to the participant how very slight movements could be detected by the EEG and how decreased movement was essential for data collection. Once the participant was relaxed and movement was kept to a minimum, they were turned around to face a blank wall and a non-operating clock sitting on the desk in front of them.

Study 1, consisting of the baseline conditions of eyes-open and eyes-closed, was counterbalanced and performed first for comparison with the proceeding tasks. All participants were asked to focus on the clock while their eyes were open and to visualize a stationary object such as the sun while their eyes were closed. The magnitude spectra (amplitude of the wave found within a certain band width) was calculated using the following frequency bands: delta (0.0-4.0 Hz) theta (4.0-8.0 Hz) alpha (8.0-13.0 Hz) and beta (13.0-20.0 Hz). During these baseline conditions and for all of the following conditions, two min. were allotted for each condition and after careful observation artifacts were deleted yet never exceeded 20% of the total number of epochs. These artifacts were eliminated by a visual inspection performed by the experimenter. Criteria for

elimination consisted of noticeable eye rolls and blinks as well as noticeable signs of gross motor movement. The nonartifact data for all conditions were stored for off-line analysis.

Study 2 consisted of two conditions involving cognitive tasks. These tasks were counterbalanced and performed by all participants. One cognitive task involved counting by sevens. The participant was asked to begin counting from one by seven's while their eyes were open and they were focusing on the clock. All participants were instructed to keep a steady pace, and at any time if they lost count, to simply start over. To ensure that the task was being performed, after the 2 min. time period, participants were asked to say aloud the number they ended with. The second cognitive task consisted of using the Bender Gestalt cards. Each participant was requested to draw the geometric figure which was presented to them on each card. These cards were presented one at a time and turned by the experimenter so as to avoid movement. Also, the participants were instructed to move their hand from drawing to drawing very slowly.

The CWPT was used for the two conditions in study 3. This test required all participants to hold an ice cube in their hand for a maximum of 2 min. and both hands were

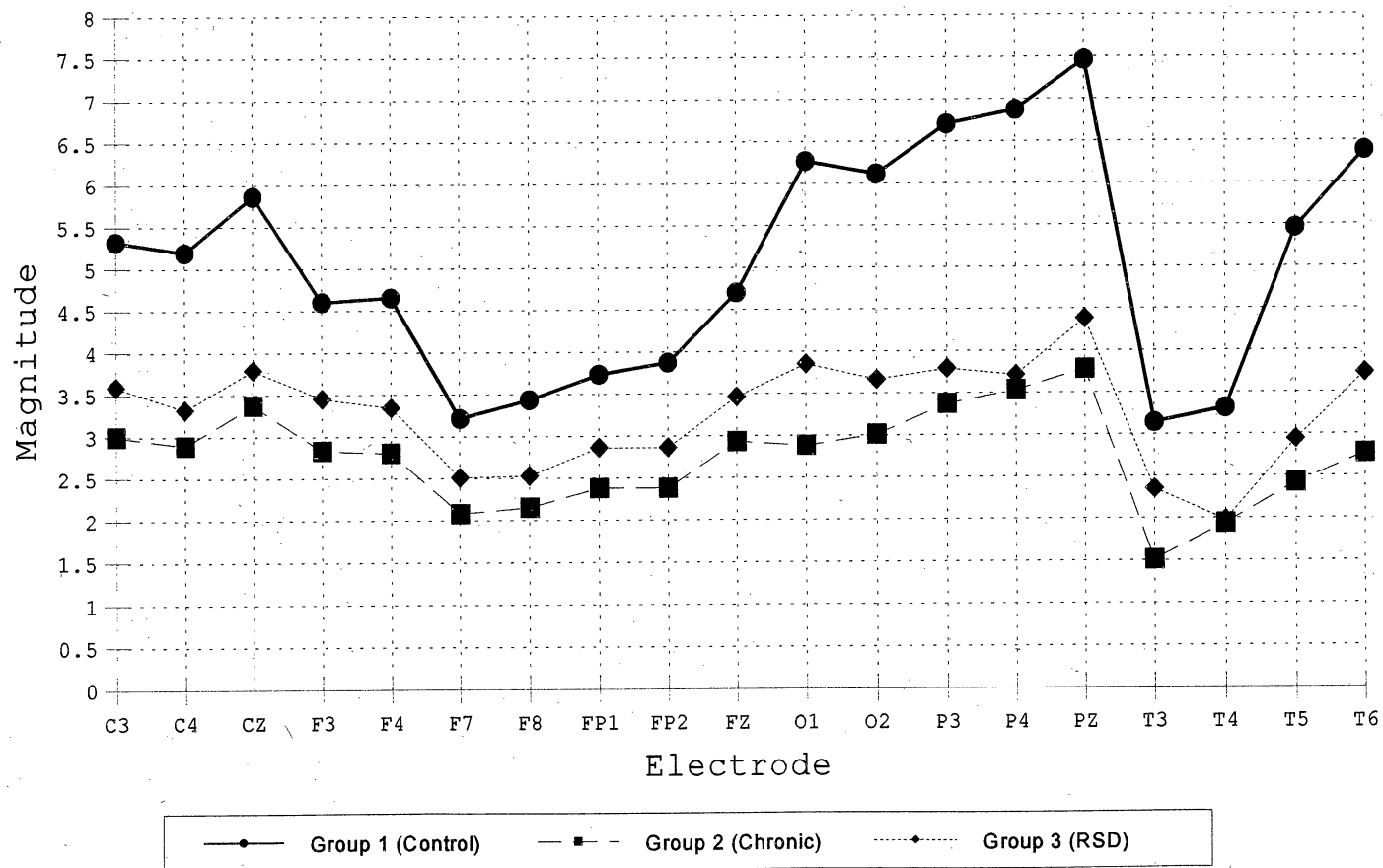
tested in a counterbalanced manner. This task was performed while the eyes were open and focusing on the clock. In order that there was no adverse effect due to the ice, a 3 min. interval was used between hand testing. After concluding the study all participants were debriefed (see Appendix D).

## RESULTS

Initial analyses were performed in order to observe any difference between the groups. The participant's severity of pain according to the 0 to 10 analog scale was analyzed for both pain groups using an independent  $t$  test. No significant difference between these groups was found  $t(16) = 1.10, p > .05$ . A second  $t$  test was performed in order to analyze the duration of pain for the two pain groups. Again, no significant result was found  $t(16) = 1.10, p > .05$ . Therefore, the two pain groups did not significantly differ in regard to pain level or duration of pain. Furthermore, in performing an Analysis of Variance (ANOVA) on the age factor for all three groups, no significant difference was found.

Figure 1 shows the mean alpha amplitudes per electrode site for all three groups during the eyes-closed condition. Another representation of this condition can be viewed via topographical maps (See Appendix E). These maps were solely used for visual purposes because they did not allow for statistical calculations. The overall mean amplitude during this baseline measure for Group 1 was 5.07, whereas for Groups 2 and 3, the mean amplitudes were 2.79 and 3.27 respectively. Therefore, it is quite evident that the alpha amplitude for Group 1 is higher than the two pain groups. It can also be seen that Groups 2 and 3 are very close in

Figure 1.  
Mean Alpha Amplitudes Per Electrode  
During the Eyes-Closed Condition



amplitude and follow a similar trend. Meaning, at all electrode sites even though Group 3 shows a higher amplitude than Group 2, the trends appear to parallel one another.

A 2x3x19 mixed design ANOVA was performed to test any group, eye condition, or electrode differences in alpha amplitude. A significant main effect in regard to the eyes-open and eyes-closed conditions was found  $F(1, 23) = 25.77$ ,  $p < .05$ . Regarding the eye condition main effect, the significant result confirms that an alpha block was shown to occur across all three groups. In addition, the group effect was found to be significant  $F(2, 23) = 3.78$ ,  $p < .05$  as well as the electrode main effect  $F(18, 414) = 24.71$   $p < .05$ . Additionally, a three way interaction was found to be significant  $F(18, 414) = 1.56$ ,  $p < .05$ . Confirming, Group 1's amplitudes were higher than the two pain group's amplitudes during the eyes-open and eyes-closed conditions at all electrode sites. And Group 3 showed a higher amplitude during the eyes-closed condition than Group 2 at all electrode sites. However, during the eyes-open condition, Group 2's amplitudes were higher than Group 3's amplitudes only at FP1 and FP2. Since the groups and electrode placements differed significantly, further analyses were performed using post hoc *t* tests. In order to create a more stringent analysis the *p* value for these tests

were set at .01 and only the electrode sites found to be significant at this level were mentioned. The first series of tests were designed to compare Groups 2 and 3. No significant results were found at any electrode site. This result confirmed no significant difference between these groups and in knowing this another set of tests were derived in order to compare Group 1 with the combined pain group of Group 2 and 3. These analyses resulted in a significant difference between the groups in regard to the electrode sites of O1, O2, P3, P4, T4, T5, and T6 (Table 3).

Mean beta amplitudes per electrode site for all three groups during the eyes-closed condition are presented in figure 2. Again, similar to Figure 1, the Group 1 overall mean amplitude level is higher (2.76) compared to Group 2 (1.86) and Group 3 (1.99). Likewise, Group 2 and Group 3's amplitudes are very similar. However, the figure now shows less of a parallel trend in that Group 2 and 3's amplitudes are greater than one another only at certain electrode sites.

Another 2x3x19 mixed design ANOVA was performed to assess any difference between eye conditions, groups, and electrode sites in regard to beta amplitude. A group effect was found to be significant  $F(2, 23) = 4.48, p < .05$  along with a significant main effect due to the electrode sites

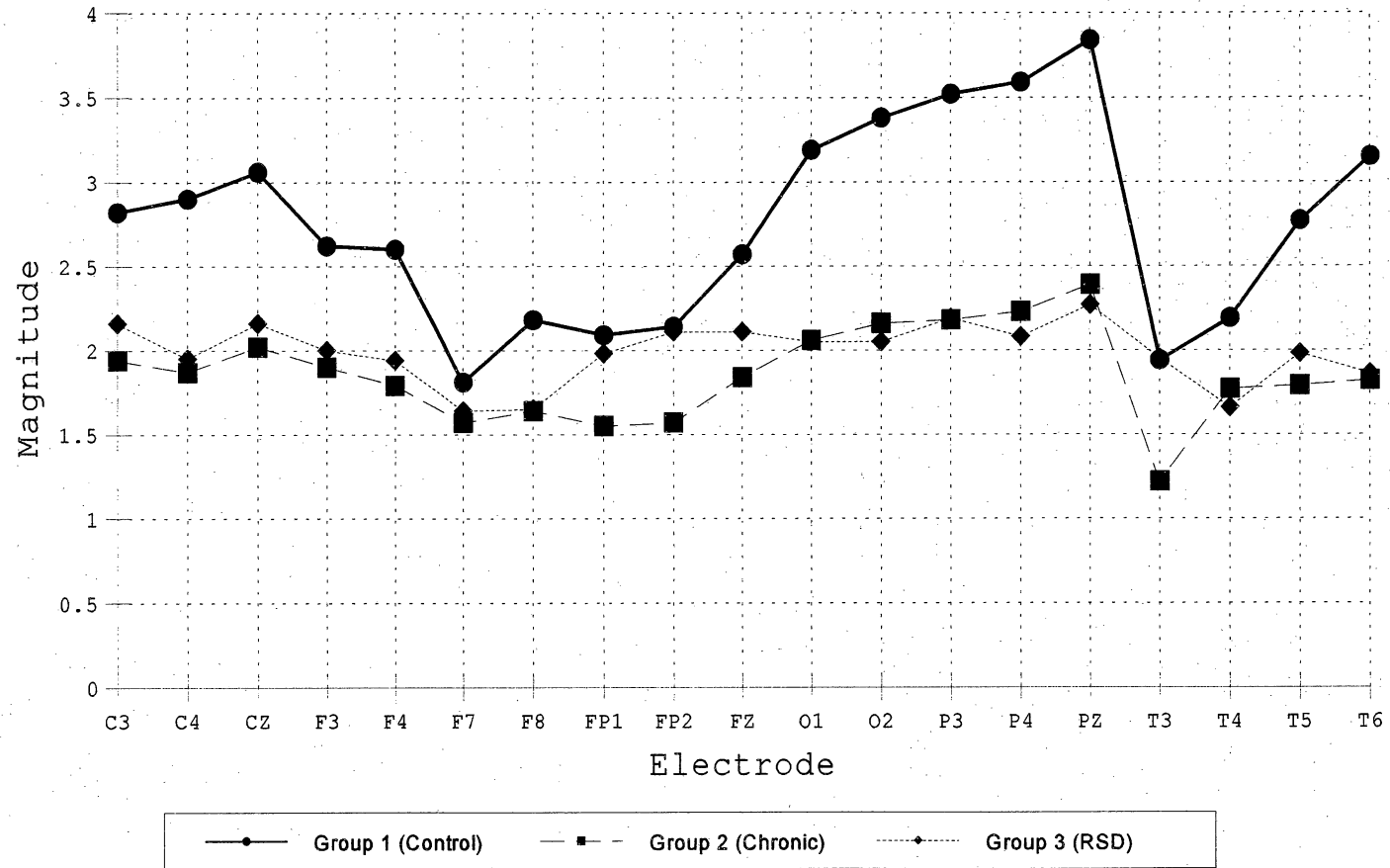


Table 3  
Planned Comparisons of Eyes Closed Alpha

Electrode	t Score
C3	-
C4	-
CZ	-
F3	-
F4	-
F7	-
F8	-
FP1	-
FP2	-
FZ	-
O1	3.099**
O2	3.099**
P3	2.657**
P4	2.692**
PZ	2.435*
T3	2.391*
T4	3.16**
T5	3.35**
T6	2.565**

<p>Key</p> <p>*P&lt;.05</p> <p>**P&lt;.01</p>
-----------------------------------------------

Figure 2.  
Mean Beta Amplitudes Per Electrode  
During the Eyes-Closed Condition



$F(18, 414) = 11.76, p < .05$ . Also, the main effect due to eye condition was found to be significant  $F(1, 23) = 10.30, p < .05$ . This significance again confirms the occurrence of an alpha block across all groups. Additionally, a three way interaction was found to be significant  $F(18, 414) = 1.74, p < .05$ . Meaning, Group 1's amplitudes were higher than the two pain group's amplitudes during the eyes-open condition at all electrode sites and higher than the two groups during the eyes-closed condition at all sites except T3 where Group 3 obtained a higher amplitude. Group 3 showed a higher amplitude during the eyes-closed condition than Group 2 at the electrode sites of C3, C4, CZ, F3, F4, F7, F8, FP1, FP2, FZ, P3, T3, T5, and T6. However, during the eyes-open condition, Group 3 showed a higher amplitude than Group 2 at electrode sites C3, C4, CZ, F3, F4, FP1, FP2, FZ, O1, P3, PZ, T3, T5, T6. Again, post hoc *t* tests set at a *p* level of .01 were used for further analysis. The first series of tests, designed to compare Groups 2 and 3, resulted in no significant findings. The second series of tests were used to compare Group 1 with the combined pain group consisting of Groups 2 and 3. These analyses resulted in a significant difference between the groups in regard to the electrode sites of C4, CZ, F4, F8, O1, O2, P3, P4, T5, and T6 (Table 4).

Table 4  
Planned Comparisons of Eyes Closed Beta

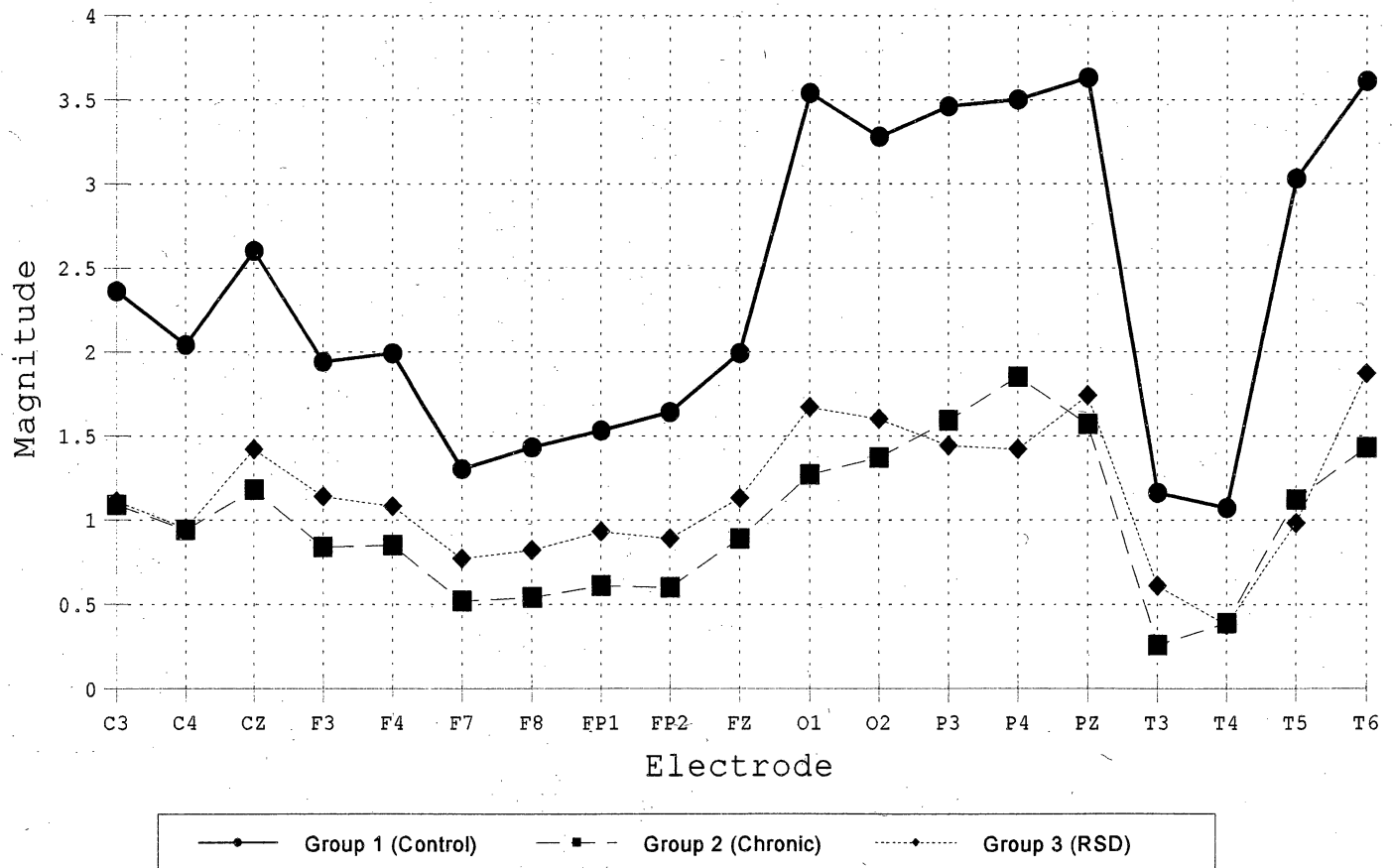
Electrode	t Score
C3	2.241*
C4	3.186**
CZ	2.689**
F3	2.48*
F4	2.732**
F7	1.161
F8	2.706**
FP1	1.344
FP2	1.187
FZ	1.779
O1	3.259**
O2	3.351**
P3	2.755**
P4	3.328**
PZ	2.016
T3	1.121
T4	1.85
T5	2.828**
T6	4.171**

<p>Key  * P&lt;.05  **P&lt;.01</p>
--------------------------------------------

In order to assess the difference in magnitude of the alpha block, the eyes-open alpha amplitudes were subtracted from the eyes-closed alpha amplitudes thereby producing one mean value per electrode site for each group. Figure 3 shows the amplitude levels for each group. Again, it is evident that Group 2 and Group 3 show lower mean amplitudes than Group 1. The difference between the eyes-open and eyes-closed conditions was greater for Group 1 showing an overall mean of 2.37 compared to Group 2 with a mean of .80 and Group 3 with a mean of 1.15. Again the interesting factor being the similarity of the chronic group and RSD group means especially at C3, C4, and T4. Similar to Figure 1, the amount of alpha amplitude is greater for Group 3 than for Group 2.

A 3x19 ANOVA was performed in order to test any group or electrode differences. This analysis resulted in a significant electrode main effect  $F(18, 414) = 11.88, p < .05$ . Two sets of  $t$  tests, set at a  $p$  level of .01 and designed in the same manner as the previous analysis, were performed. In regard to the comparison of Groups 2 and 3 there were no significant findings. However, when comparing these pain groups against

Figure 3.  
Mean Alpha Amplitudes Per Electrode  
Eyes-Open Subtracted From Eyes-Closed



Group 1 a significant difference was found at the electrode sites of O1 and T5 (Table 5).

Figure 4 shows the mean alpha amplitudes per electrode site for all three groups during the visual-spatial task (using the Bender-Gestalt cards). These amplitude levels were subtracted from the eyes-open baseline measure in order to readily observe the directionality of the amplitude readings. Most electrode sites did decrease in amplitude. However, by observing the overall mean amplitudes it was found that Group 2 deviated more from baseline as indicated by a mean of  $-.07$  than did Group 3, as indicated by a mean of  $-.21$ . However, Group 1 deviated to a greater extent shown by a mean amplitude of  $-.51$ . Of interest is the large decrease in the parietal region (p3, p4, pz) for Group 1 compared to Group 2 and Group 3.

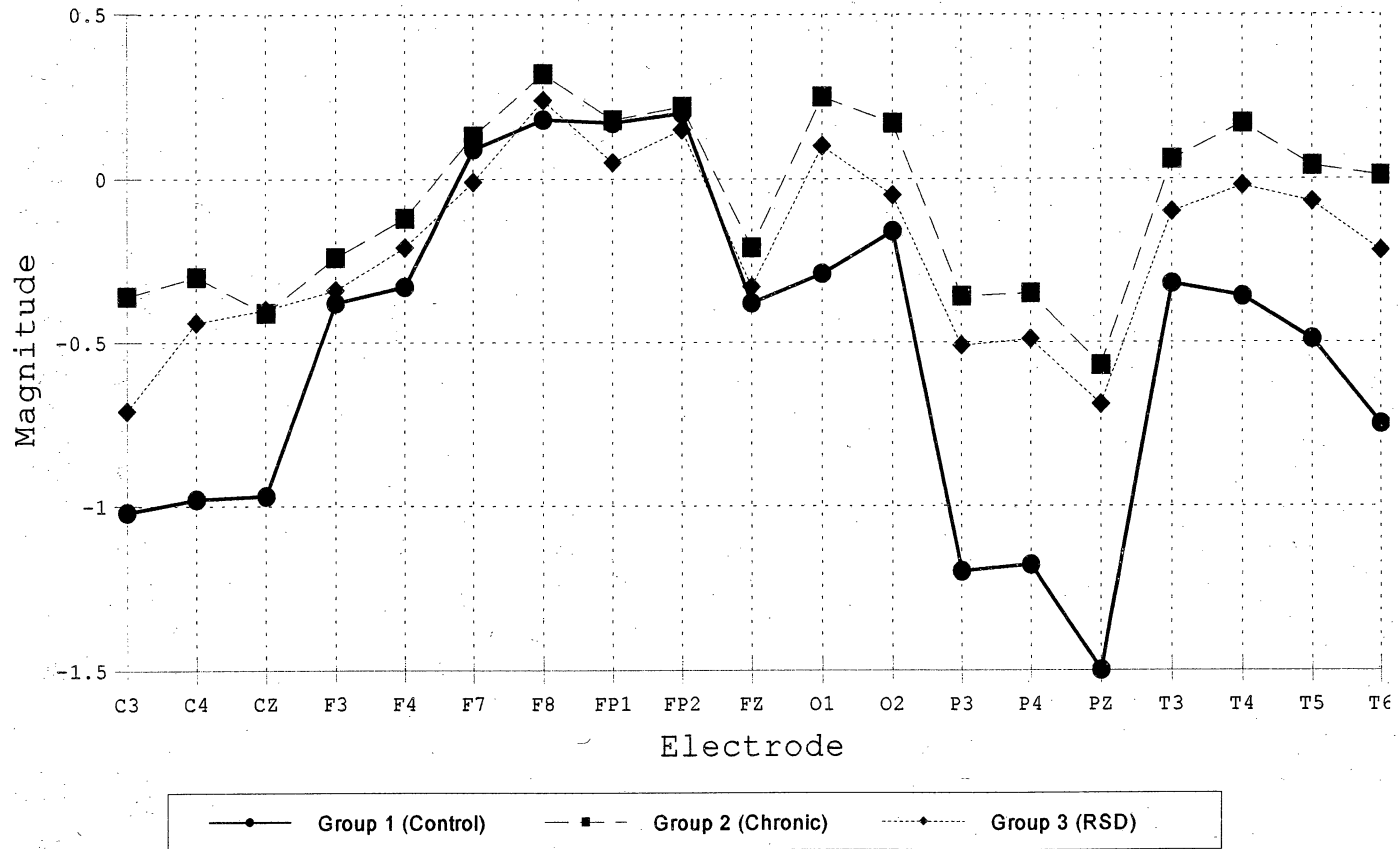
A series of 2x3 mixed design ANOVA's were performed to analyze any group or hemisphere effects due to the present task. The  $p$  value for this analysis was set at  $.05$ . There was no significant difference between groups. However, there was a significant hemisphere main effect when comparing the electrodes of F3/F4, F7/F8, FP1/FP2, and T5/T6. These results indicated that the right hemisphere electrode site had a higher alpha amplitude than the left hemisphere in

Table 5  
Planned Comparisons of Eyes Closed Minus  
Eyes Open Alpha

Electrode	t Score
C3	-
C4	-
CZ	-
F3	-
F4	-
F7	-
F8	-
FP1	-
FP2	-
FZ	-
O1	2.569**
O2	2.239*
P3	-
P4	-
PZ	-
T3	-
T4	2.113*
T5	2.732**
T6	-
Key: *P<.05 **P<.01	



Figure 4.  
Mean Alpha Amplitudes Per Electrode  
During the Visual-Spatial Task



most cases except for the placements of T5/T6 where the reverse was true (Table 6).

The mean amplitudes per electrode site for all three groups during the serial seven task are presented in Figure 5. These means were again subtracted from the eyes-open baseline. Figure 5 shows a positive direction in alpha for Group 1, signified by an overall mean of .21. However, Group 2 was only slightly positive at .05 and Group 3 resulted in a negative, alpha amplitude when compared to the eyes-open baseline marked by an overall mean of -.06. Also of interest is that out of the nineteen electrodes, thirteen are below the baseline alpha level for Group 3, whereas the other groups do not show this same result.

A series of 2X3 mixed design ANOVA's were performed with the intent to identify any group or hemisphere main effects due to the serial seven task. No significant results were found in regard to a group or hemisphere effect.

Figure 6 shows the mean amplitudes per electrode site for all three groups during the left hand CWPT. As with the cognitive tasks, the CWPT measures were also subtracted from eyes-open baseline so that the directionality of the magnitude readings would be more apparent. The overall amplitude means of .07 for Group 1, .21 for Group 2, and 0

Table 6.

Visual-Spatial Alpha by Hemisphere

Elect. pairs	Group		Hemisphere		Group x Hemisphere		Hemisphere Means	
	F score (2, 23)	P value	F score (1, 23)	P value	F score (2, 23)	P value	left	right
c3 x c4	1.81	.187	1.45	.241	.5	.613	-1.02	-.98
f3 x f4	.33	.725	7.14	.014	.37	.695	-.32	-.22
f7 x f8	0.21	.812	11.54	.002	.68	.518	.07	.2
fp1 x fp2	.15	.864	7.38	.012	1.25	.304	.13	.19
o1 x o2	.7	.508	.1	.750	.63	.543	-.29	-.16
p3 x p4	2.13	.142	.08	.784	.00	.996	-1.20	-1.18
t3 x t4	2.28	.125	.44	.515	.47	.629	-.32	-.36
t5 x t6	2.13	.141	5.48	.028	1.02	.375	-.16	-.30

Figure 5.  
Mean Alpha Amplitudes Per Electrode  
During the Serial Seven Task

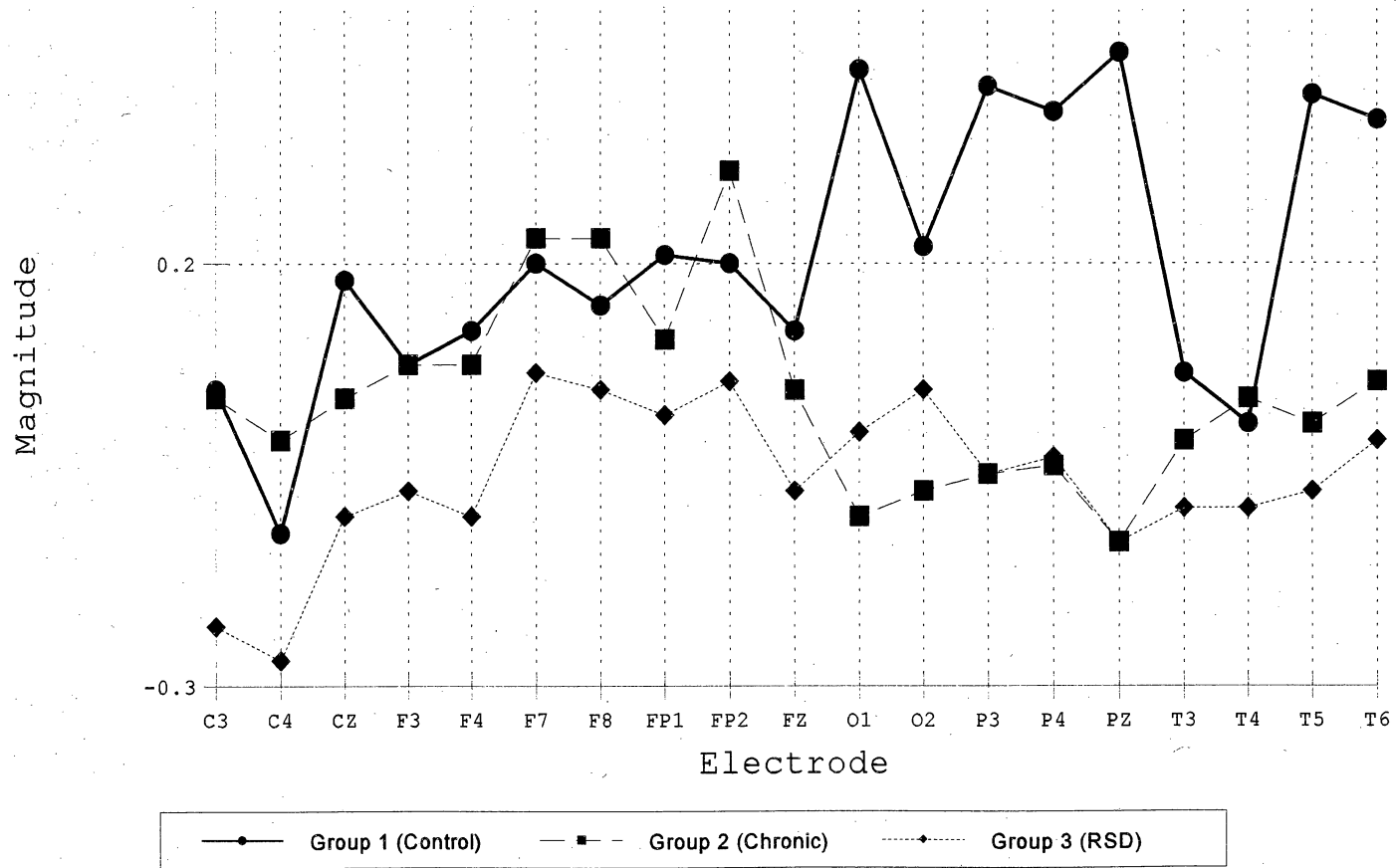
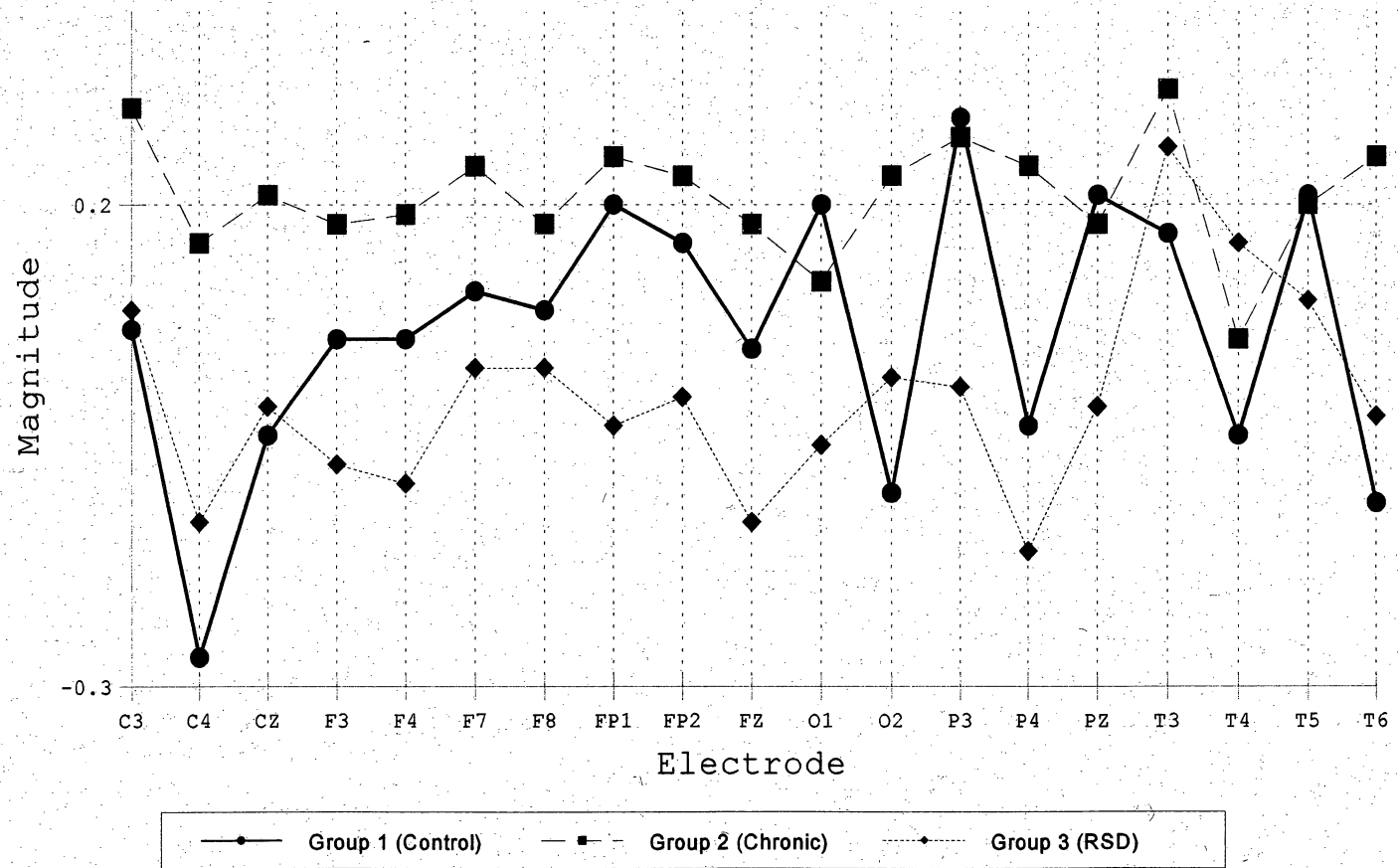


Figure 6.  
Mean Alpha Amplitudes Per Electrode  
During the Left Hand CWPT



for Group 3 are very close. Of interest is the hemispheric difference in regard to Group 1. Looking at the difference between electrodes C3/C4, O1/O2, P3/P4, T3/T4, and T5/T6, it is quite noticeable that the right hemisphere electrode is lower than the left hemisphere in all of these cases, whereas, this same trend is not followed by either of the pain groups.

A series of 2X3 mixed design ANOVA's were performed in order to identify any group or hemisphere differences. No significant group effect resulted. However, in regard to the hemisphere main effect when comparing C3/C4 a significance was found  $F(1, 23) = 9.31$   $p < .05$ . The same was true when comparing P3 and P4  $F(1, 23) = 6.92$   $p < .05$  (Table 7).

Table 7

Left Hand CWPT Alpha by Hemisphere

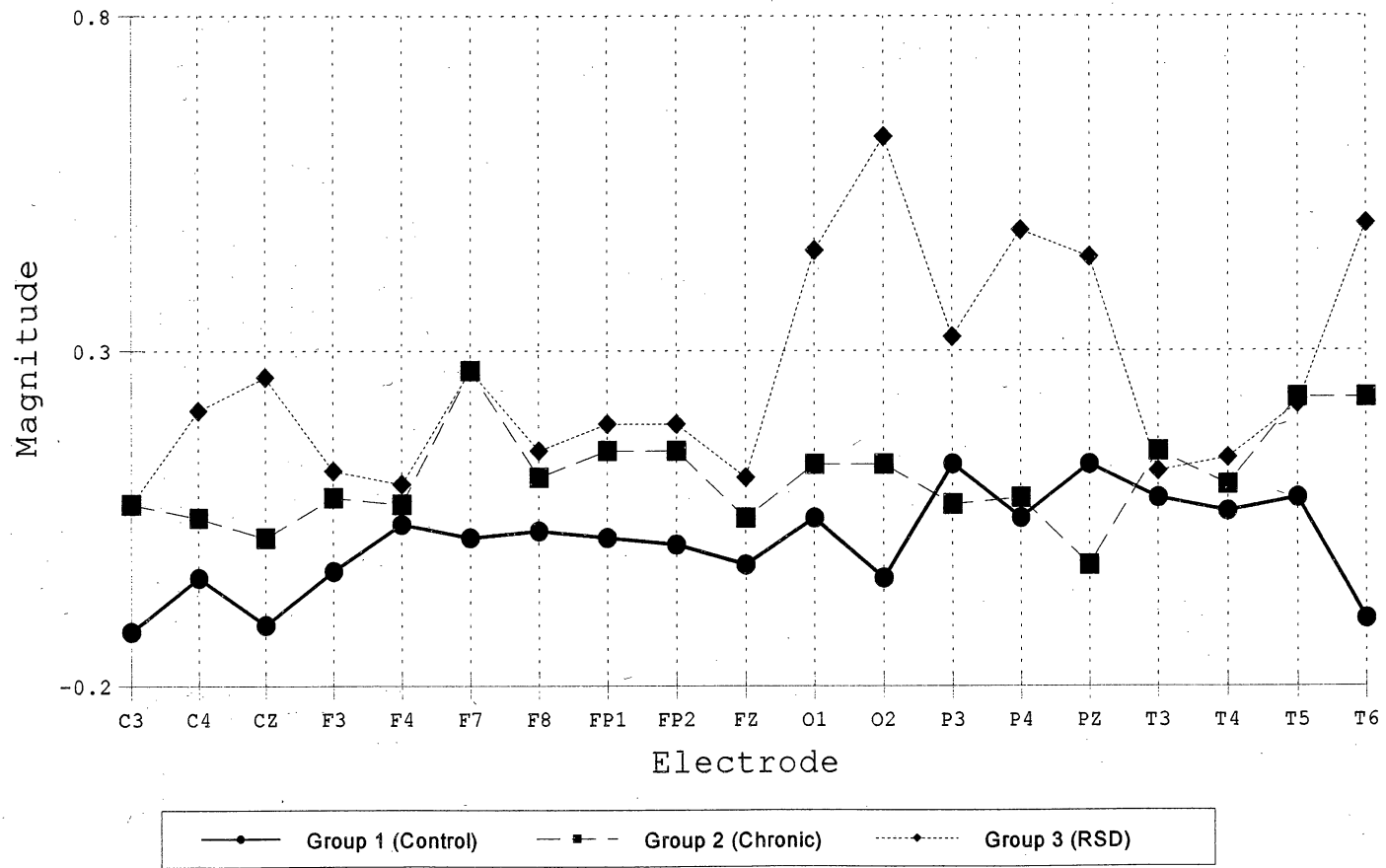
Site pairs	Group		Hemisphere		Group x Hemisphere		Hemisphere Means	
	F score	P value	F score	P value	F score	P value	left	right
c3 X c4	1.02 (2,23)	.377	9.31 (1,23)	.006	.62 (2,23)	.549	.07	-.27
p3 X p4	.94	.407	6.92	.015	1.68	.209	.29	-.03

Therefore, during the left hand CWPT there was a significant decrease in alpha in the right hemisphere (C4 and P4) compared to the left hemisphere.

The mean amplitude scores per electrode for all groups during the right hand CWPT are shown in Figure 7. In observing this graph, it can be seen that Group 3, with a mean amplitude of .26, increased their alpha from baseline to a greater extent than did Group 2 (.11) or Group 1 (.01). Of concern, is the trend which is seen in Group 3's amplitudes. For example, electrodes such as O1/O2, P3/P4, and T5/T6 show a decrease in alpha activity in the left hemisphere and vary greatly in amplitude between the two hemispheres. This trend is not evident in the other groups.

A series of 2X3 mixed design ANOVA's were performed on this data and no significant group or hemisphere difference was found.

Figure 7.  
Mean Alpha Amplitudes Per Electrode  
During the Right Hand CWPT





## DISCUSSION

In the present study, it was demonstrated that the control group obtained a significantly higher alpha amplitude at certain electrode sites than the pain groups in the eyes-closed condition. This confirmed the research by Chen and Rappelsberger (1994) that a pain state is identified by enhanced beta activity and therefore a decrease in alpha activity. This result may be due to the arousal differences between the pain and no pain groups. If the pain groups are initially aroused because of the pain they are experiencing, it would not seem uncharacteristic for them to obtain a lower alpha amplitude. The pain groups used in the current study were continuously aroused by painful stimuli possibly making them unable to relax and therefore decreasing the strength of the alpha amplitude comparatively to those without pain.

In order to further investigate the effects of a pain state that may exist in groups experiencing pain, as mentioned by Chen and Rappelsberger (1994), the beta amplitude was also analyzed during the eyes-closed condition. The present hypothesis, much like that of Chen and Rappelsberger (1994), proposed that the combined pain group of Group 2 and 3 would show an increase in beta

compared to Group 1. However, this was not confirmed. In fact, there was an opposite result which showed the control group obtaining a significantly higher amplitude for beta than the pain groups. An alternative explanation for this unexpected finding has been mentioned by Niedermeyer (1987). According to his research, individuals experiencing pain will often show a low amplitude level over all frequency band widths. The current study confirmed this result in that the chronic pain and RSD patients obtained low amplitude brain wave patterns much different from the higher amplitude control patients brain wave patterns. Regardless if they opened or closed their eyes, the overall amplitude was below those with no pain.

In the eyes-closed condition, the brain wave patterns obtained by the chronic and RSD groups are of considerable interest because of the parallelism which is seen. Not only are the RSD and chronic pain group's amplitudes much lower than the control group but the difference in magnitude between electrode sites is also vastly different. Furthermore, no matter how different the magnitude between electrode sites was for the chronic and RSD groups, these groups still mirrored one another and showed very little flexibility between brain regions.

Andreassi (1989) mentioned that when the eyes open alpha amplitude will decrease or block. In order to investigate the alpha amplitude difference during alpha block between groups, I examined the difference between the alpha amplitudes of eyes-open and eyes-closed conditions between the control group and the combined pain group consisting of the RSD and chronic pain patients. The data supported that of Andreassi (1989) in that all three groups did obtain a decrease in alpha while their eyes were open compared to when their eyes were closed. However, it was further hypothesized that due to noxious stimuli in the combined pain group, this difference between eyes-open and eyes-closed alpha would be significantly greater for the control group than for the pain groups. This hypothesis was also confirmed. It was found that the control group, at selective electrode sites, did significantly differ from the other groups. Again, it was demonstrated that the chronic pain and RSD groups had similar alpha amplitudes for all electrodes. In contrast, the control group showed large amplitude differences between electrode placements. This result further confirms the possible effect of the painful stimuli.

The first cognitive task consisted of the Bender-Gestalt cards. These cards were used as stimuli for the

purpose of the visual-spatial, drawing task. Past research has shown that this type of task tends to cause a decrease in alpha in the right hemisphere (De Benedittis & De Gonda, 1985). This decrease in alpha in the right hemisphere, compared to eyes-open baseline, was found only to be significant in the present study in regard to the electrode placements of T5/T6 and there was no significant group difference. Possibly, if a time line was used, one could observe many intervals during the task and show how the chronic pain or RSD individual handles differing situations, such as drawing figures, compared to the control group.

The overall theme of flexible and changing brain wave patterns as being indicative of normal functioning becomes very important when looking at the trend which occurred between the three groups during this task. The chronic pain and RSD groups are very similar in their trends to a point of almost parallelism. However, there is a noticeable difference between the pain groups and the control group. Also, the amplitude differences between the electrode sites for the control group demonstrate a changing and flexible trend very different from the static trend of the pain groups.

In order to further observe hemispheric differences between the three groups listed in the present study, a

serial seven task was used. The three groups did not show significant differences between themselves and contrary to De Benedittis and De Gonda (1985) there was no significant decrease in alpha activity in the left hemisphere during this task compared to the eyes-open baseline measure.

However, it was noticed in all three groups that for the most part there was a greater decrease in alpha in the right hemisphere rather than the left hemisphere. This was similar to the findings of De Benedittis and De Gonda (1985) when they mentioned that as a math task becomes more difficult the left hemisphere appears to become less involved. This is a very interesting finding and in order to further research this topic again a time line of seconds would be necessary during this task to look at how the hemispheric changes occur as the person progresses through higher and successively harder calculations.

Further investigating the effects of the serial seven task, the findings tend to support the view of Carter-Wilson (1991) in that because of the reorganization of pain inputs which are over represented in the somatosensory cortex, regardless of what task is implemented, the RSD individual will show a decrease in alpha in both hemispheres. This decrease in both hemispheres for the RSD group was shown in the current study and was not shared by the no pain and

chronic pain groups. In addition, it was noticeable that the RSD group had far more decreases in alpha than the other groups with the trend showing a much more static progression in comparison to the other groups.

The present study demonstrated a decrease in alpha in the contralateral hemispheres during the CWPT. This was shown by the group's significant hemispheric difference between C3 and C4 and between P3 and P4 during the CWPT using the left hand. These findings are consistent with the research of Backonja et al. (1991) in that there was a decrease in alpha from baseline in the central regions. Furthermore, these findings also support the results of Ferracuti, Seri, Mattia, and Crucco (1994) in that there was a decrease in alpha activity in the somatosensory cortex (C3/C4) and the decrease occurred in a contralateral manner. In regard to the right hand, the trend seemed to be similar with the left hand showing also a contralateral decrease in alpha. However, using the right hand, the results were not significant. It may prove beneficial to use a complete immersion CWPT for later study in order to evoke a greater response. This increase in response may also show a greater difference between the three groups. It is very apparent that the control group had a greater decrease in alpha on the contralateral side and the difference between the

amplitude of left and right electrode placements was varied much more than the pain groups. These differing trends may show the plasticity of the non noxiously stimulated brain.

Continued research in this area should analyze the brain wave activity upon a time frame of seconds. In doing so, one could analyze how the brain is affected by noxious stimuli initially, and then how the brain compensates for this stimuli. I believe that further research will demonstrate a difference, not only between how individuals with pain and those without pain initially perceive painful stimuli, but more importantly how these two groups compensate for the painful stimuli.

As Veerasarn and Stohler (1992) stressed, the arousal level factor in EEG studies is always important and must be of concern. The arousal level of the three groups was directly addressed in the present study. For example, when the eyes-open baseline alpha amplitude was subtracted from the eyes-closed alpha amplitude the arousal level due to pain was isolated. It would be assumed in making this calculation, that the results in regard to the control group would be due to the shift in arousal caused by the eye conditions only. However, it would also be assumed that along with the arousal of eyes opening, the pain groups

would additionally have the pain factor. Therefore, the difference we see between the control group's alpha amplitude and the pain group's alpha amplitude can be explained by the pain factor.

The current EEG findings were unable to support the view expressed by Carter-Wilson (1991) that due to the reorganization of pain inputs to the somatosensory cortex, an over-representation of the pain would result, causing painful sensations far surpassing the initial injury. However, this does not mean that a reorganization of pain inputs is not occurring. According to the results for the left hand CWPT, the RSD group's trend towards a bilateral decrease in alpha was found, which was contrary to the other groups. In addition, if the severity of the pain resulting from RSD was causing a reorganization of the somatosensory cortex, two things may result. The alpha level of RSD individuals would be far lower in amplitude compared to other pain groups or this reorganization is unlike anything previously studied and therefore uninterpretable at this time.

The cross-talk between the medial and lateral pain pathways discussed by Hooshmand (1993) appears to be a very plausible explanation for the severity of pain felt by RSD



patients. Again, if this cross-talk is in fact involved, more research is needed to analyze this effect on EEG. The brain wave rigidity noticed in the RSD group may in fact be the result of this cross-talk but more research is needed to answer this question.

Bennet and Xie (1988) discovered that they could manifest behaviors such as hyperalgesia and allodynia via a ligature placed around the tail of a rat. This research is vital because not only did they find these characteristics but they also discovered that the placement of the ligature caused neural responses to occur in the thalamus and cortical regions. This neural response sent to the thalamus is pertinent because it would seem that this response would therefore alter the thalamus in some way. This alteration then may change the pattern of brain waves since the thalamus appears to be the origination point for brain wave activity. More attention needs to be placed on animal models of RSD because under very controlled conditions and by way of canula placed electrodes the true neural response to this type of pain may be exposed.

In viewing Melzack's (1991) "neuromatrix" concept, it becomes evident that not only is the essence of pain multifaceted, but so are the pain mediated pathways to the mid and higher cortical regions. It must be noted that there

is probably little discrepancy on the effectiveness of the brain in its ability to compensate for dysfunctions. This may hold equally true for pain. Every individual seems to respond to pain differently. This may be partly due to psychological components but the fact is that however we try to track pain, be it by Melzack's pathways or the EEG, one must remember that pain appears to be the result of many complex, intertwined factors. In addition, Hooshmand (1993) stresses that an individual is diagnosed with RSD if they meet certain criteria. One such criterion is depression which is occurring because of the influence of the pain on the limbic system. Further research must focus on the effects of depression and how it may be viewed by EEG. As seen in the current study, the linkage may be between the suppressed amplitude in the parietal region and the limbic system involvement in depression. This linkage would be monumental in creating an additional noninvasive technique for diagnosing RSD.

Along the same lines of the complex factors affiliated with the sensation of pain, it does not seem uncharacteristic that studies such as the one represented by King and Nuss (1993), have shown that the use of electroconvulsive therapy (ECT) can reduce the pain associated with RSD. If electrical impulses are sent through

the thalamus, or the frontal-parietal cortex as mentioned by Canavero et al. (1993), the pain pathways will be disrupted in some manner. In addition, the memories associated with this pain are also affected and may lead to a lessening of the pain. One discovery that King and Nuss (1993) view as important is that the ECT may affect the limbic cortex, thalamus, and hypothalamus in such a way that the sympathetic nervous system is modulated. This modulation can suppress the sympathetic nervous system resulting in lessening of the response to pain and therefore an alleviation of pain. In this case it appears that the lessening of the pain factor is really the indicator of how much emotions play a role in the expression of pain.

Studies such as Verdugo, Campero, and Ochoa's (1994) have shown that administration of medications by way of sympathetic blocks are not always beneficial and therefore alternative methods must be looked at for the treatment of RSD. According to the current study, a difference in brain wave activity was evident in those with RSD compared to the control group. A pain state appears to be observed as a brain wave pattern of lower amplitude for alpha and beta. Therefore it would seem that more noninvasive research, such as EEG analysis and possible neurofeedback, would be beneficial for diagnosing and treating these individuals.

Since it has been discovered that the alpha rhythm is associated with a relaxed state, it would seem plausible that alpha training may be beneficial. Undergoing particular tasks while using neurofeedback may also help the individual learn how to deal with stressful events better and increase the plasticity of their brain wave patterns.

The ability to early diagnose and treat RSD was stressed by Hooshmand (1993) as being very critical for recovery. This may hold true for the result seen by way of the EEG as well. If the EEG changes seen in the RSD group are due to a reorganization of pain inputs to the somatosensory cortex, than being able to treat the symptoms of RSD before they increase to this stage may in fact be the most important goal. As can be noticed, the present study was performed on participants who had been diagnosed at least six months previous to the current study. This time frame should have been sufficient for the cortical changes to take place. Current study should focus on the duration of RSD and the progression of participants with this diagnosis. A study which took into effect these factors may discover the true cortical changes resulting from the pain and its effect on cortical structures over time.

Biofeedback may in fact be a very important procedure in lessening the effects of RSD such as vasomotor

instability. However, these effects appear to be secondary to the symptoms causing pain. It has been shown through Blanchard's (1979) study and by studies using hypnosis such as Gainer's (1992) that the sympathetic nervous system can be affected using these techniques. According to Gainer (1992), an overactive or unstable sympathetic nervous system can cause a reaction which causes severe vasoconstriction. This vasoconstriction in return can cause lowered temperature in the affected appendage and a decrease in blood flow both which are characteristics of RSD. This appears to be the clear link between RSD and noninvasive treatment using biofeedback. If biofeedback can be used to help in increasing blood flow and peripheral temperature then this is key for the RSD sufferer. More importantly, after techniques such as diaphragmatic breathing and other relaxation exercises have been taught, neurofeedback would also be very important in trying to get the chronic pain or RSD individual to raise their alpha amplitude. In raising their alpha amplitude a relaxed state would be achieved which appears, in viewing the present study, to be exactly what the pain groups would benefit from.

A psychological factor to RSD has also been viewed in the literature. Houdenhove and Vasquez (1993) have noted that helplessness may play a large role in the symptoms seen

in RSD. This view, much like Dorsel's (1989) theoretical model of a chronic pain behavior pattern, depict the RSD individual as someone who has lived a painful life prior to their injury and in return their physical pain becomes an acceptable release. These psychological attributes are not represented by the current study. However, further study using the combination of a psychological assessment and EEG analysis in RSD individuals may reveal a strong relation between the psychological and physiological characteristics of RSD.

As Melzack and Casey (1968) suggest, pain appears to be multifaceted in nature and that a traumatically painful syndrome such as RSD cannot be defined on a strict physiological level but certainly must be viewed on an emotional level as well. RSD, like all chronic pain syndromes, is very complicated in nature as Dorsel (1989) noted and because of this complicated nature cures seem to originate very slowly. Could the RSD sufferers emulate the soldiers of WWII as mentioned by Beecher (1956) simply by changing the situation in which they are in? No one denies the large psychological factor which is involved with pain.

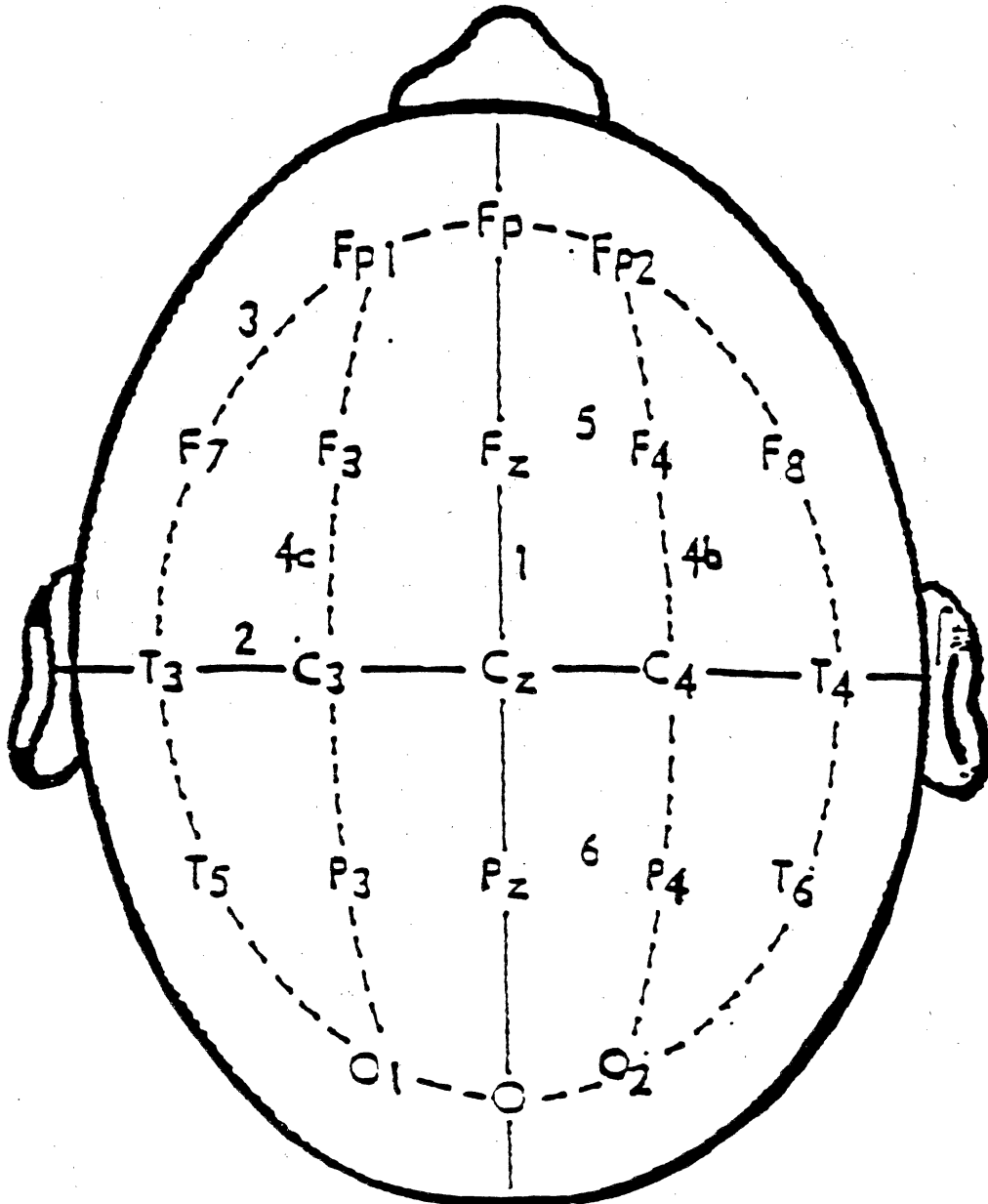
In conclusion, the contribution of this research has shown how noninvasive treatment such as biofeedback and neurofeedback may be very beneficial to those with chronic

pain and RSD. Possibly by the change in their relaxation state through these techniques may change their mental state and their focus on the pain much like the soldiers of WWII. This is not to say that medical treatment is not needed, but that this type of treatment be implemented within the overall treatment plan for chronic pain individuals.

APPENDIX A

International 10-20 System Montage

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## APPENDIX B

### Participant's Informed Consent

The procedure in which you are about to participate is designed to investigate brain wave activity in regard to individuals diagnosed with Reflex Sympathetic Dystrophy (RSD) and those without RSD diagnosis during various tasks. It is hoped that through this research, a diagnostic tool for RSD will be developed. This study is being conducted by Mark Clair, graduate student, California State University San Bernardino (CSUSB) under the supervision of Fred Newton Ph.D. , professor of psychology, CSUSB, and Kurt Bickford Ph.D. clinical psychologist. Experimental design for the current study has been approved by the Institutional Review Board of CSUSB .

For the purpose of data collection, a process known as electroencephalography (EEG) will be performed. This instrument is a noninvasive tool used to record various brain wave activity. All participants, during the recordings, will undergo a math and visual-spatial task as well as a simplified Cold Water Pressor Test (CWPT). The simplified CWPT consists of the placement of an ice cube in the palm of the hand. Research participation will consist of one session having a duration of approximately one to two hours.

All data collection will be coded in such a fashion that anonymity of all participants will be maintained at all times.

Please realize that your participation is strictly on a voluntary basis and you are free to withdraw from the study at any time without penalty. Any questions prior to the study, while undergoing the procedure, or after the study has been performed are encouraged. Questions about this study should be directed towards:

Mark Clair  
Department of Psychology  
(909) 880-7336

Frederick Newton, Ph.D.  
Department of Psychology  
(909) 880-5588

Participant's Signature \_\_\_\_\_ Date \_\_\_\_\_

APPENDIX C

Personal History Questionnaire

Patient # \_\_\_\_\_  
Handedness \_\_\_\_\_  
Age \_\_\_\_\_  
Sex \_\_\_\_\_  
Location of pain \_\_\_\_\_  
Duration of pain \_\_\_\_\_

Severity of pain presently being experienced  
0 1 2 3 4 5 6 7 8 9 10

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For physician use only:

edema \_\_\_\_\_  
limitation of movement \_\_\_\_\_  
vasomotor instability or symptoms \_\_\_\_\_  
allodynia \_\_\_\_\_  
dystrophic skin changes \_\_\_\_\_  
dystonic muscle spasms \_\_\_\_\_

Hypoesthesia :  
light touch \_\_\_\_\_ warm \_\_\_\_\_  
vibration \_\_\_\_\_ pinprick \_\_\_\_\_  
cold \_\_\_\_\_  
Mechanical Hyperalgesia \_\_\_\_\_

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Comments:

## **APPENDIX D**

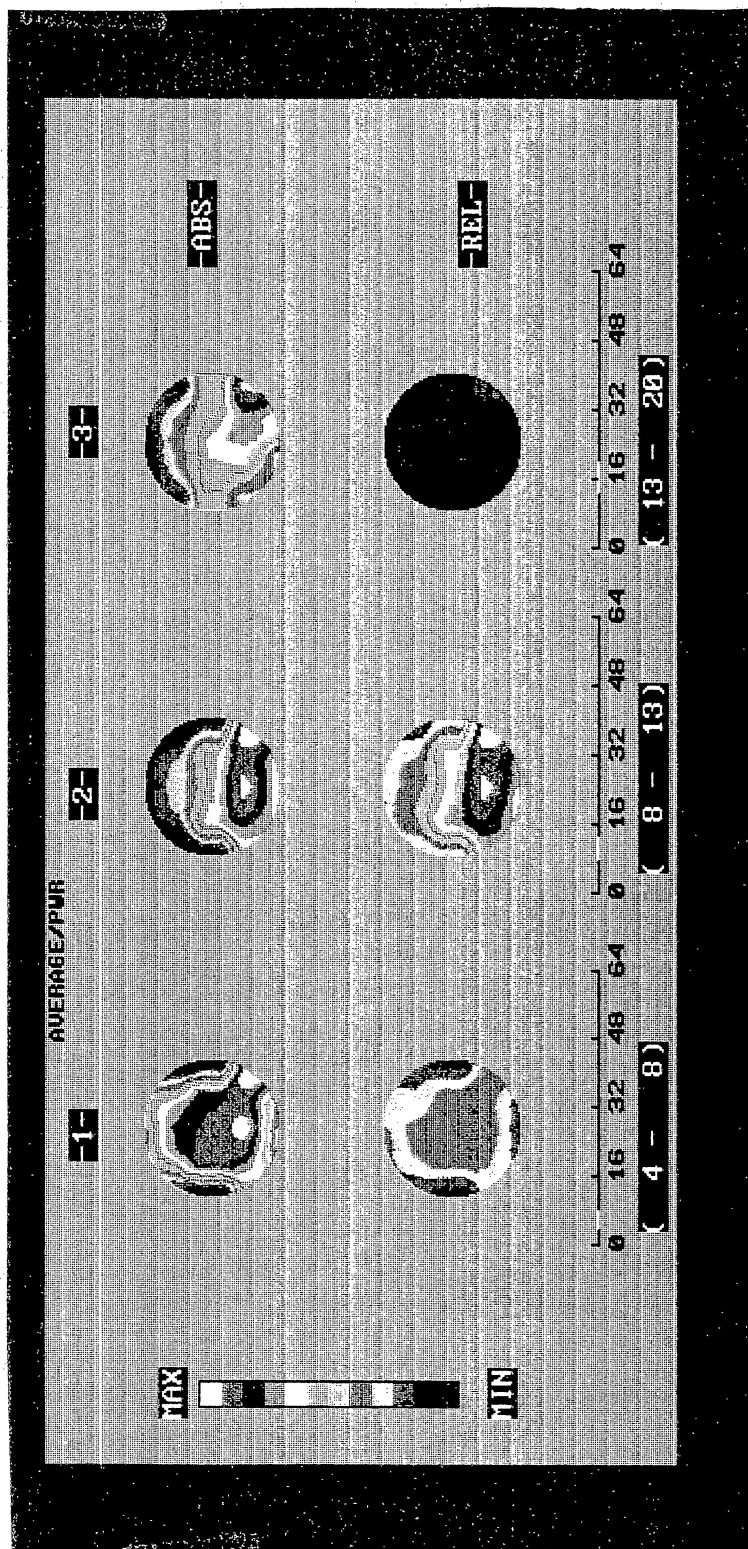
### **Debriefing Statement**

Thank you very much for your participation in this study. As was discussed on the consent form, the purpose of this study was to investigate brain-wave activity of individuals diagnosed with RSD and individuals without RSD. It is my desire that information obtained from this study will be helpful in developing a diagnostic tool for RSD.

If you have any questions about this study or the procedure in which you participated please contact Fred Newton Ph.D. at (909) 880-5588 or Mark Clair at (909) 880-7336. Results will be obtainable from Fred Newton c/o Department of Psychology, California State University San Bernardino upon the completion of the study.

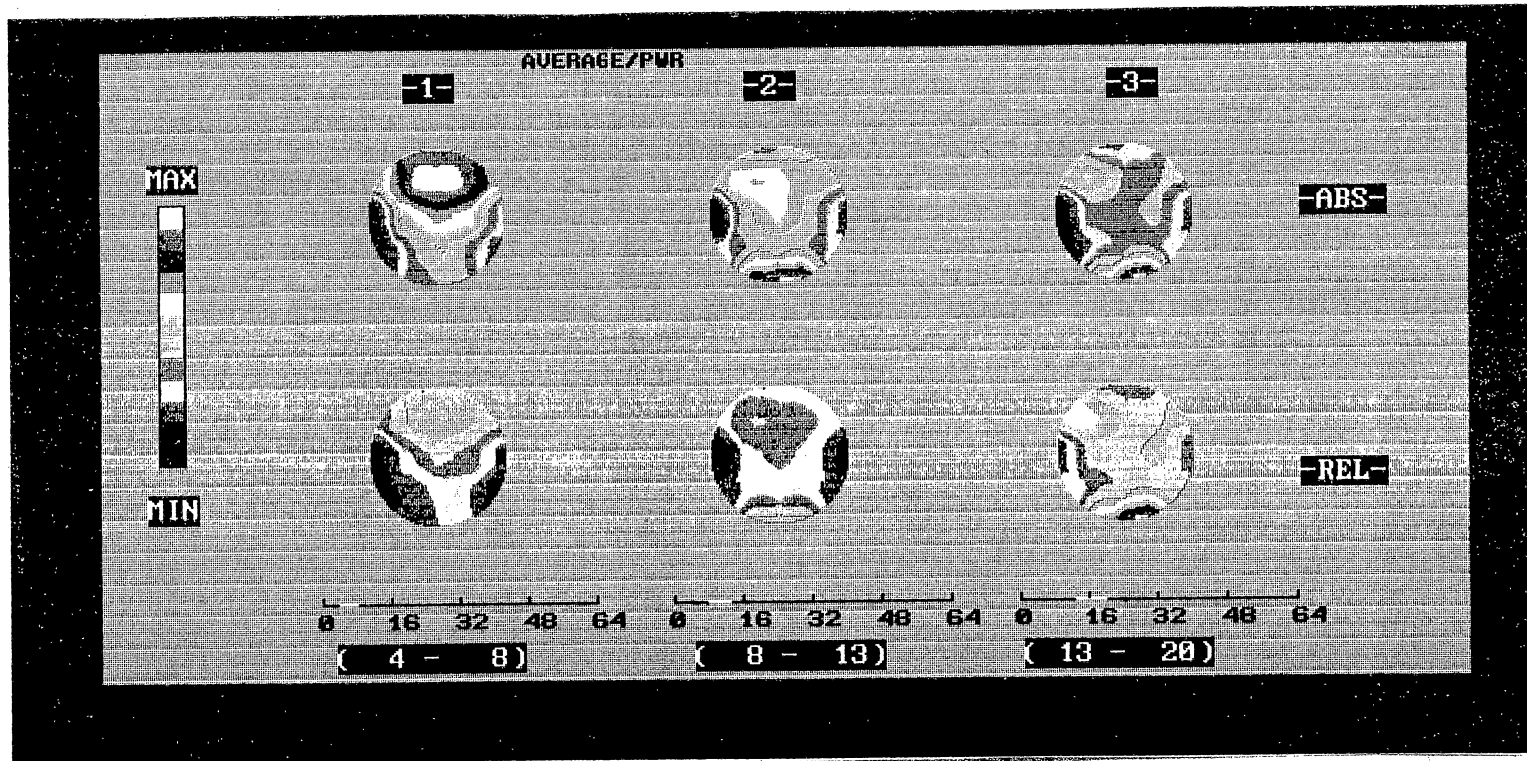
APPENDIX E

Topographical Map 1.  
Topographical Mapping of a Control Participant During the  
Eyes-Closed Baseline



Topographical Map 2.

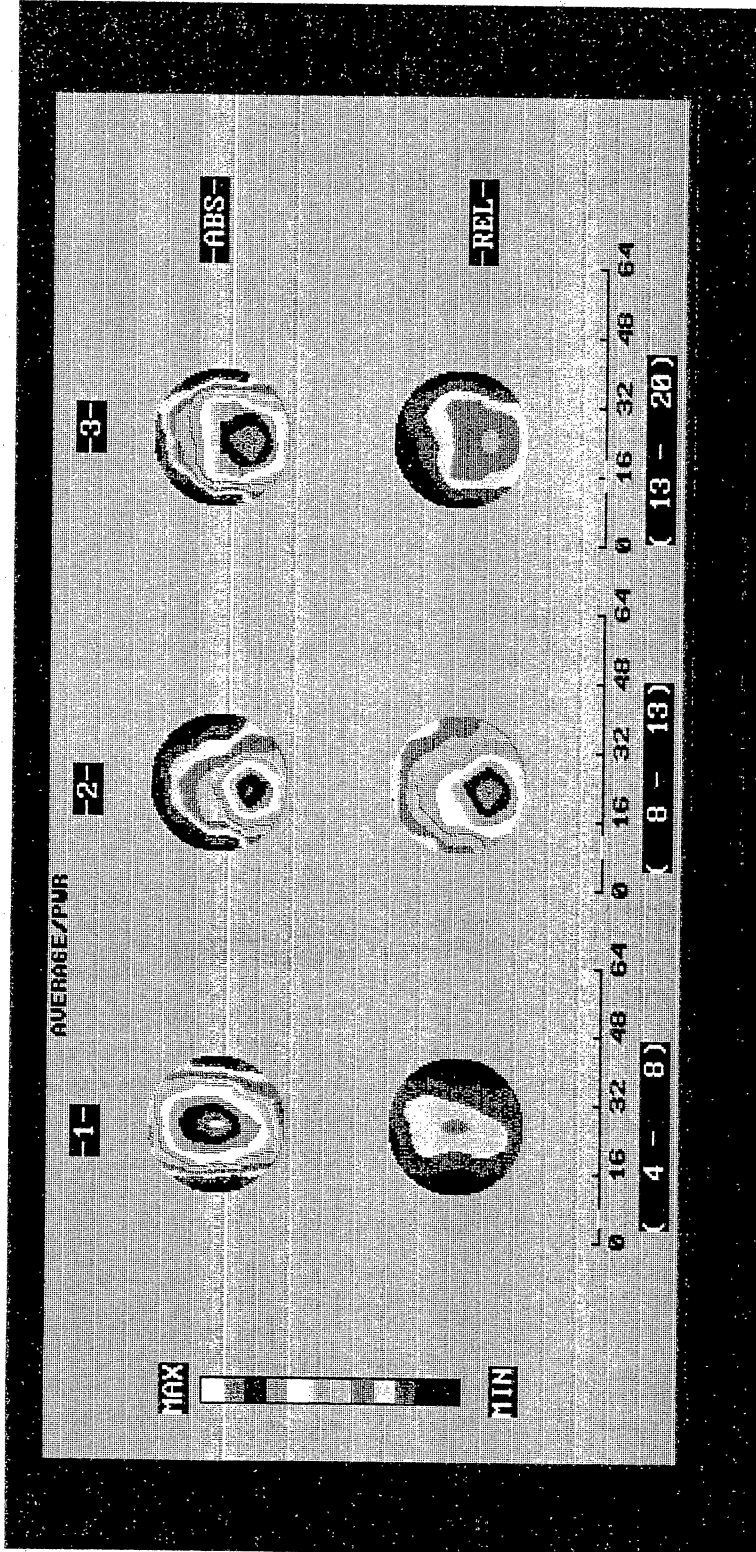
Topographical Mapping of a Chronic Pain Participant During the Eyes-Closed Baseline



APPENDIX E (continued)

APPENDIX E (continued)

Topographical Map 3.  
Topographical Mapping of an RSD Participant During the Eyes-  
Closed Baseline



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