Neuropsychological Effects of Hostility and Pain on Emotion Perception

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ABSTRACT

Recent research on the neuropsychology of emotion and pain has indicated that emotion and pain are complex processes that may substantially influence each other. Disorders of negative emotion and pain are known to co-occur (Delgado, 2004); however, it is not clear whether negative emotional conditions lead to pain or whether increased pain experiences lead to negative emotion. Further, certain negative emotions, such as hostility or anger, may produce differential effects on the experience of pain, such that they may lead to an increase in pain or a decrease in pain. An increase or decrease in pain perception may lead to altered behavioral, cognitive, and neuropsychological effects in high hostility. In order to more clearly examine the aforementioned relationships, the current experiment examined auditory emotion perception before and after cold pressor pain in high and low hostile men. Additionally, quantitative electroencephalography (QEEG) was used to measure changes in cerebral activation as a result of auditory emotion perception and cold pressor pain. Results indicated that identification of emotion post-cold pressor differed as a function of hostility level and ear. The high hostile group increased identification of stimuli at the right ear after cold pressor exposure, while the low hostile group increased identification of stimuli at the left ear after cold pressor exposure. Primary QEEG findings indicated increased left temporal activation after cold pressor exposure and increased reactivity to cold pressor pain in the high hostile group. Low hostile men had a bilateral increase in high beta magnitude at the temporal lobes and a bilateral increase in delta magnitude at the frontal lobes after the cold pressor. Results suggest decreased cerebral laterality and left hemisphere activation for emotional and pain processing in high hostile men.

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Emotion and pain are complex phenomena that are universally experienced. Current views suggest that emotion contains valence, arousal, and motor activation components (Heilman & Gilmore, 1998). Chang, Arendt-Neilson, and Chen (2002) stated that pain is a complex experience that includes sensory, affective, cognitive, and motivational components. Further, pain may be linked to arousal and attention (Chen, 2001). Although emotion and pain are defined as separate constructs, their definitions contain many of the same elements and they may produce similar behavioral, physiological, and neuropsychological effects. Research indicates that emotion influences motor (Demaree et al., 2002), auditory (Gadea et al., 1995), somatosensory (Herridge, Harrison, & Demaree, 1997; Lee et al., 2002), visual (Klaassen et al, 2002; Coupland et al., 2004), and cardiovascular (Snyder, Harrison, & Shenal, 1998; Gendolla, Abele, & Krusken, 2001) systems. Similarly, pain produces relative changes in motor (Urban et al., 2004), auditory (Demaree & Harrison, 1997), somatosensory (Valeriani et al., 2004), visual (Herridge et al., 2004), and cardiovascular (Fillingim et al., 2002) systems. Further, imaging studies indicate that pain may produce cerebral activation that is similar to cerebral activation seen in negative emotion (Hseih, Hannerz, Ingvar, 1996; Coghill, Gilron, & Iadarola, 2001).

Previous research within emotion and pain has further noted that emotional traits may influence cerebral processing of emotion (Herridge et al. 2004) and pain (Janssen, 2002). Within this context, hostility may be a particularly important trait to examine. Hostility is associated with increased aggressive behavior, negative feelings towards others (Graves & Miller 2003), and increased physiological arousal (Keefe & Blumenthal, 1986). Due to its association with the development of cardiovascular disease (Matthews et al. 2004), hostility is one of the most

studied affective constructs. However, few studies have examined hostility's role in pain perception.

In order to more clearly elucidate the effects of emotion and pain on cerebral function in hostility, the current experiment used an emotional dichotic listening task and quantitative electroencephalogram (QEEG) to measure cerebral activation in high and low hostile men before and after exposure to a cold pressor[1]. Influential models in emotion include the right hemisphere model, the valence model, and the motivational model. Each of these models has provided a contribution to the understanding of functional cerebral processing in emotion. As will be presented, data also indicate that these models can provide insight into the functional cerebral processing of pain.

Right Hemisphere Hypothesis for Emotion and Pain

The right hemisphere hypothesis originally described the right hemisphere as important for the experience of all emotion (reviews see Borod, 1992; Demaree et al., 2005); however, modified versions now exist that describe differential roles for frontal and posterior cortex and include the left frontal lobe. It has been proposed that the left and right frontal lobes are important for the experience of positive and negative emotion, but that the right posterior cerebral regions are dominant for the perception of emotion (Borod, 1992). Borod et al. (2002) reviewed cases of unilateral brain damage and found support for the right hemisphere model in the perception and expression of emotion across facial and prosodic communication channels. Additional work has analyzed emotional deficits resulting from brain damage in relation to lesion location. Data indicate that right medial frontal lesions result in the inability to express emotional prosody (Heilman & Gilmore, 1998; Heilman, Leon, & Rosenbek, 2004), while

temporal-parietal lesions are associated with the inability to comprehend emotional prosody (Heilman, Scholes, & Watson, 1975; Ross et al., 1981).

Empirical research in non-brain damaged populations provides additional support for the right hemisphere model. In the auditory modality, Bryden and MacRae (1989) found a left ear advantage (right hemisphere processing) for the identification of emotion. Similarly, a left ear advantage was found for the emotional quality of tonal sequences (Bryden, Ley, & Sugarman, 1982). Harrison and Gorelczenko (1990) found support for the right hemisphere model in the visual modality. Participants identified facial affect faster when faces were presented to the left visual field (right hemisphere). Facial expression of emotion may also be more dependent on the right hemisphere. Dimberg and Petterson (2000) found that facial electromyography (EMG) activity in response to emotional stimuli was larger on the left side of the face, indicating a right hemisphere dominance for spontaneously evoked emotional expression. In a review of 49 studies of facial asymmetry in the expression of emotion, Borod, Haywood, and Koff (1997) concluded that the left hemiface (right hemisphere) was more involved than the right hemiface (left hemisphere) in the expression of emotion. The authors concluded that the results provided strong support for right cerebral dominance in emotional expression.

Borod (1992) suggests that the right hemisphere advantage for emotion arises from the fact that emotion has many characteristics (e.g. nonverbal, spatial, integrative, and patterned) that the right hemisphere is specialized to process. Several other authors have suggested that the right hemisphere's advantage for emotional processing may be due to a greater involvement of the right hemisphere in autonomic responses and arousal (Heller, 1993; Heilman,1997) that occur with emotion.

Right hemisphere activation in emotion may also be influenced by dynamic activation between the right-anterior cerebrum and the right posterior-cerebrum. Right frontal activation may relate to valence (Heilman, 1997), while right posterior activation may relate to arousal (Heller, 1993; Heilman, 1997). Additionally, relative activation of the anterior or posterior regions may influence the activation in the opposing region. Differential activation patterns may lead to dysfunctional emotional processing. For example, in a QEEG investigation of a patient with anger problems, Everhart and Harrison (1995) found that episodes of anger were accompanied by an increase in delta magnitude over the right frontal lobe concurrent with increased beta magnitude over the temporal lobe. Neuropsychological models of depression also focus on anterior-posterior cerebral activation patterns that lead to depression (e.g. Heller, 1993; Tucker, 1993; Crews & Harrison, 1995; Shenal, Harrison, & Demaree, 2003).

The noted right hemisphere involvement in emotion and emotional disorders may contribute to right hemisphere involvement in pain. Min and Lee (1997) examined somatic symptoms in patients with depressive disorders, anxiety disorders, and somatization disorders and found that comorbid somatic symptoms, especially pain, occurred more frequently at the left hemibody. The authors speculated that the emotional disorders are associated with a right hemisphere disturbance leading to left lateralization of pain and other somatic symptoms.

Right lateralization of pain (i.e. application of the right hemisphere hypothesis) has not traditionally been recognized as a critical component in neuropsychological models of pain. Anatomical pathways suggest that pain sensation should be similar at both the right and left side of the body (Lugo et al. 2002); however, several previous investigations have reported that lateralized processing of pain may occur (Hsieh, Hannerz, & Ingvar, 1996; Coghill, Gilron, & Iadarola, 2001). Lateralized pain occurs more frequently at the left hemi-body (Chandramouth,

Kanchan, & Ambadevi, 1993; Wittling, 1995) and higher ratings are given for painful stimuli applied to the left hemi-body (Pauli, Wiedemann, & Nickola, 1999; Lugo et al., 2002).

Recent imaging studies have also reported right lateralized cerebral activation in response to noxious stimuli. Coghill, Gilron, and Iadarola (2001) found right lateralized activation in the dorsolateral cortex, dorsal frontal cortex, and the inferior parietal lobe in response to a noxious thermal stimulus independent of the location of stimulation. However, no lateralization patterns were found when looking at pain intensity ratings. In a PET investigation of experimentally induced cluster headache attacks, Hsieh, Hannerz, and Ingvar (1996) concluded that the right hemisphere plays a preferential role in pain processing. EEG investigations suggest that cold pressor pain results in contralateral stimulation of the parietal cortex, however, the effect lasts longer over the right hemisphere (Ferracuti et al., 1994). Nevertheless, numerous other studies have indicated bilateral activation in response to pain (see Peyron, Laurent, & Garcia-Larrea, 2000 for a review). Bromm (2001) stated that bilateral activation of the secondary somatosensory cortex in response to pain is needed in order to differentiate the side of the body that is hurt from the side of the body that is unaffected. Bilateral activation of parietal cortex then may be the result of pain localization, rather than a result of pain intensity or perception.

In summary, data indicate a role for the right hemisphere in emotion and pain. This role may be further influenced by anterior or posterior function. The frontal lobe appears to be important in expression and valence perception, while the posterior brain may be important for perception and comprehension. However, the results are not unequivocal, especially with respect to pain. Given the fact that a large amount of literature exists for both emotion and pain, it is necessary to examine other neuropsychological models of emotion.

Valence Model of Emotion and Pain

Lesion studies, lateralization studies, and imaging studies have revealed functional differences between the left and right hemisphere in the processing of emotion. In an investigation of patients with unilateral cerebral damage, Adolphs, Jansari, and Tranel (2001) concluded that the perception of negative valences relies primarily on the right hemisphere, whereas positive valences are processed by both the right and the left hemispheres. Additionally, it is noted that damage to the right hemisphere produces a euphoric reaction versus a catastrophic reaction that occurs with damage to the left hemisphere (Heilman & Gilmore, 1998). Burton and Labar (1999) stated that lesions in the left hemisphere cause a disinhibition of negative affective valences of the right hemisphere thereby causing a release of negative emotion, while right hemisphere lesions result in the expression of positive emotion through disinhibition of the left hemisphere.

Additional support for the functional differences between the left and right hemispheres in emotional processing is provided by tachistoscopic presentation of emotional faces. Reuter-Lorenz, Givis, and Moscovitch (1983) presented happy, sad, and neutral faces to normal participants. Results indicated that reaction times to happy faces in the right visual field (RVF; left hemisphere) were faster, while reaction times to sad faces were faster in the LVF (right hemisphere). A similar design was used by Harrison and Gorelczenko (1990) who found an overall processing advantage for emotional faces when they were presented to the right hemisphere, however, the advantage was most prominent during the presentation of angry faces.

Further evidence of left hemispheric processing in positive emotion and right hemispheric processing in negative emotion is provided by imaging studies. Diego et al. (2004) found that moderate massage therapy lead to decreases in anxiety and stress and shifts to greater left frontal

EEG asymmetry, suggesting that induction of a positive affective state occurs with left frontal activation. Petruzzello, Hall, and Ekkekakis (2001) found that participants with greater left frontal EEG activation exhibited an increased positive reaction to exercise relative to participants with greater right frontal EEG activation. Blair et al. (1999) found increased right frontal glucose metabolism during the perception of angry faces. In a QEEG investigation of emotional memory and cerebral activation, Foster and Harrison (2002) found a significant positive correlation between the subjective intensity of angry memories and cerebral activation in the right frontal and right temporal cortices. Additional EEG data suggest that greater right frontal activation is associated with negative affect, while greater left frontal activation is associated with positive affect (Tomarken, Davidson, Henriques, 1990; Davidson, 1995). However, Bell and Fox (2003) failed to find baseline frontal asymmetries in groups of participants who scored high in either negative or positive affect. They hypothesized that this may be due to the broad range of emotions that can be classified as negative affect. Fox (1994) suggests that not all types of negative affect are associated with increased right frontal activation.

Despite some controversy, the cumulative data have led to the valence model of emotional processing. The model states that the right hemisphere is specialized for negative emotion, while the left hemisphere is specialized for positive emotion. The valence model is supported by characteristics of positive and negative emotion and functional processing asymmetries between the cerebral hemispheres. Positive emotions may be more communicative and linguistic requiring left hemispheric processing (Borod, Koff, & Buck, 1986; Borod et al., 2002). In contrast, negative emotions are related to danger or survival and require multimodal contributions, a quick scanning system, and Gesalt right hemispheric processing (Borod et al., 2002).

Neurochemical properties of the left and right hemispheres may also account for differences in emotional processing. Dopamine, a neurochemical associated with positive affect (see Ashby, Isen, & Turken, 1999 for a review) is found at higher levels in the left hemisphere (see Tucker & Williamson, 1984; Wittling, 1995 for reviews). Alternatively, the right hemisphere uses more norepinephrine and serotonin (see Tucker & Williamson, 1984; Wittling, 1995 for reviews). Altered levels of norepinephrine and serotonin are associated with negative affect such as, hostility, aggression (Cleare & Bond, 1997), and depression (Flory et al., 2004).

Understanding how the left and right hemisphere function in positive and negative affect can help describe cerebral activation in response to pain influences the affective component of pain. Schiff and Gagliese (1994) reported that reactions to cold pressor stimulation at either the left or right side resulted in emotional reactions that were consistent with activation of the contralateral hemisphere. Acute left sided pain (right hemisphere) resulted in higher group scores on measures of anxiety (increased right hemisphere activation, see Heller et al., 1997). Schiff and Gagliese (1994) also reported that right sided pain (left hemisphere) resulted in lower anxiety group scores than in the control group. The authors speculate that the right sided pain stimulation attenuated the emotional reaction to pain due to increased positive affect associated with left hemisphere activation. Lorenz, Minoshima, and Casey (2003) found that activation in the left and right dorsolateral prefrontal cortex (DLPFC) was correlated with activation in subcortical regions and was related to pain affect and intensity ratings. During periods of low activation in the left DLPFC pain unpleasantness ratings were significantly higher relative to periods of high activation in the left DLPFC. Additionally, periods of high activation in the left DLPFC were correlated with decreased activation in the midbrain and the anterior cingulate cortex (ACC). Periods of low right DLPFC were correlated with increased activation of the left and right insular

cortex and increases in pain unpleasantness and intensity ratings. Although the data are correlational, they suggest that the level of activation in the left and right frontal lobes may be associated with how positive or negative the pain is perceived.

Previous work suggests that positive affect can diminish pain, while negative affect may increase pain. Pain tolerance increases while viewing positive emotional pictures and decreases while viewing negative emotional pictures (Meagher, Arnau, & Rhudy, 2001). This effect may be described in terms of relative activation of the cerebral hemispheres. Positive emotion activates the left hemisphere (Lee et al., 2002), which may lead to inhibition of right hemisphere pain processing. Conversely, negative emotion activates the right hemisphere (Lee et al., 2002), which may lead to intensification of pain. However, negative emotion such as anxiety, fear, or anger can lead to pain inhibition. Bolles and Fanselow (1980) argued that threatening situations produce endogenous opioids in the brain that lead to pain inhibition. Wall (1979) proposed that pain associated with negative emotion occurs in phases. In an immediate phase, pain inhibition results from the need to recuperate or as a defensive reaction. Later, pain may be intensified in order to promote treatment and recovery of injury. Other authors have investigated the influences of physiological reactivity in anger its influence on pain inhibition. Janssen, Spinhoven, and Brosschot (2001) found that higher BP reactivity to experimentally induced anger (which may be related to increased levels of trait hostility or anger; see Davis, Matthews, & McGrath, 2000) prior to a cold pressor task increased pain tolerance. Burns, Bruehl, and Caceres (2004) further suggest that pain inhibition or intensification in anger may be related to an individuals preferred mode of anger expression. They suggest anger suppressors are capable of experiencing stress-induced analgesia, while anger expressors tend not to show the same effects in experimental research. Individuals who generally express their anger may evidence lower thresholds in the

laboratory due to the inability to express anger as they normally would which leads to non-compliance and avoidance of the pain stimulus.

The fact that negative emotion may be associated with increased or decreased pain suggests the importance of examining dynamic cerebral activation in response to noxious stimuli. The data presented here suggest that left hemisphere activation is important for positive emotion. Activation of the left hemisphere may be a way to decrease pain through increases in positive affect. Right hemisphere activation, in contrast, is important for negative emotion which is associated with an increase or decrease in pain. However, an alternative approach to the study of emotion exists that defines emotion in terms of the behavioral activation that is evoked by the emotional stimulus rather than in terms of positive or negative valence. Because pain requires a behavioral response this model may also apply to pain processing.

Motivational Models of Emotion and Pain

Motivational models of emotion focus on motor activation or the behavioral responses that are motivated by an emotion. Approach behaviors or states lead to greater left hemispheric activation, while withdrawal behaviors or states lead to greater right hemispheric activation in the frontal cortex (Davidson, 1993; 2000). According to Gray (2001), the distinction between approach and withdrawal emotional states is conceptually one of the clearest distinctions in emotion. In contrast to the valence model, left frontal activation is not associated with positive valence, but rather a behavioral approach state, whereas right frontal activation is associated with a behavioral withdrawal state and not negative valence (Harmon-Jones, 2004a). This distinction is important due to the fact that certain emotions, such as anger, have a negative valence but may produce behavioral approach rather than withdrawal.

Davidson's (1993) motivational model describes activation of the left frontal lobe as resulting in approach related behavior, while activation of the right frontal lobe is associated with withdrawal related behavior. Davidson (2003) states that left-sided prefrontal cortex (PFC) activation is required for the initiation of behavior related to appetitive goals and that hypoactivation of the left PFC may result in depression. Alternatively, right-sided PFC activation is related to behavioral inhibition and vigilance that is associated with negative or aversive emotional states and traits.

Gray (1990) details a Behavior Activation System (BAS) and a Behavior Inhibition System (BIS) for emotion. The BAS is related to emotions such as "hope" and "happiness," while the BIS is related to emotions such as "anxiety" and "fear." Personality measures relating to the BAS and BIS significantly correlate with anterior brain asymmetry indicative of approach or withdrawal states (Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997).

Heilman and Gilmore (1998) describe an approach/withdrawal system dependent on interactions between the anterior and posterior brain. The authors state that the right hemisphere has a special role in motor activation or for preparing an organism to respond to a stimulus. The frontal lobes are described as mediators of avoidance behaviors and the parietal lobes as mediators of approach behaviors. These ideas are supported by evidence from lesion studies. Lesions of the frontal lobes lead to the inability to inhibit responses, manual grasp responses, and inappropriate approach behaviors. Consequently, frontal lobe lesions produce approach behavior as a result of disinhibition of the parietal lobes. The parietal lobes (which mediate approach) are normally inhibited by the frontal lobes, when the frontal lobes are lesioned or deactivated the parietal lobes become disinhibited, producing excessive approach behavior. Lesions of the parietal lobes lead to neglect, deviations of eye, head, and arm movements, inability to respond,

and withdrawal behaviors (Heilman & Gilmore, 1998). This increase in withdrawal behavior may be a result of increased activation of the frontal lobe due to a decrease in parietal activation, leading to inhibition or suppression of approach behavior. Schutter et al. (2001) found support for parietal mediation of approach behavioral through measurement of asymmetrical activation of EEG activity.

Incorporating the valence and motivational models of emotion has traditionally been difficult because emotions such as anger or hostility have a negative valence, but can produce approach behaviors. High trait measures of anger, hostility, and aggression and anger induction have been found to correlate with increased baseline levels of left relative to right frontal activation (Harmon-Jones & Allen, 1998; Harmon-Jones, 2004b; Harmon-Jones & Sigelman, 2001). Harmon-Jones (2004a) suggests that anger generates approach behaviors that are aimed at resolving the anger, which may lead to acts of aggression.

However, work done by Harmon-Jones is at odds with prior research indicating right hemisphere function in negative emotion (i.e. Demaree et al., 2002; Foster & Harrison, 2004; Burton & Labar, 1999; Blair et al., 1999). To overcome the discrepancies it may be necessary to look at cerebral activation in brain areas other than the frontal lobes. In two case studies of patients with hostility and anger problems, it was found that hostility resulted from deactivation of the right frontal lobe and increased activation of the right temporal lobe (Everhart & Harrison, 1995; Demaree & Harrison, 1996). Demaree and Harrison (1997) found that high hostile participants activated the right hemisphere in response to a pain stressor as evidenced by changes in dichotic listening. These results suggest that the right posterior cortex is important for anger. Waldstein et al. (2000) found that negative emotion induction resulted in bilateral EEG activation of the frontal lobes and in the endorsement of anger. Waldstein et al. (2000) suggest

that anger may be related to either right or left frontal activation depending on how an individual handles emotion. Anger expressors are more likely to activate the left frontal lobe as a result of outwardly expressing anger through approach behaviors. Individuals who suppress anger are more likely to activate the right frontal lobe as a result of anger suppression and withdrawal from a situation (Waldstein et al., 2000).

An incorporation of Davidson's (1993) model and Heilman and Gilmore's (1998) model may provide the most parsimonious explanation for the cerebral activation concurrent with anger. Anger produces changes in both the anterior and posterior brain that are associated with behavioral approach or withdrawal. Additionally, it would lend support to Borod's (1992) addition to the right hemisphere model, indicating the importance of both the right and left frontal lobes, and the right posterior cortex in emotion.

Other emotions may be better served through this approach to emotion as well. In accordance with Davidson's model depression is most often associated with relative right frontal activation or left frontal hypoactivation (Baehr et al., 1998; Davidson, 1998) and produces social isolation and withdrawal behaviors. Additional evidence suggests that depression may also be concurrent with suppression of the right temporal-parietal cortex (see Heller, 1990). Incorporation of Heilman and Gilmore's (1998) model is necessary to account for noted changes in right posterior cortex during anger or hostility and depression. Further, this helps account for other behavioral correlates of depression such as decreased arousal and decreased performance on spatial tasks that require the use of the right parietal lobe (Henriques & Davidson, 1997).

The motivational approach may also be necessary to describe motor responses to pain. Behavioral responses to painful stimuli can be described in terms of approach and withdrawal states. Although pain is described as a sensation, it differs from other senses in that it is

immediately linked to behavioral withdrawal or approach. Sensory modalities, such as vision or hearing, are more immediately linked to environmental exploration (Janssen, 2002). It has even been suggested that pain be described as a homeostatic emotion because it is more akin to motivational systems such as hunger or thirst than it is to the other sensory modalities (Craig, 2003).

Motor responses to pain suggest that initial responses to pain are based on protective behavioral withdrawal reflexes that may be mediated by spinal pathways (Urban et al., 2004). However, when behavioral conflicts occur higher cortical functioning may be necessary to produce the appropriate behavioral response (Lorenz, Minoshima, & Casey, 2003). Hsieh et al. (1995) stated that the nature of intense acute pain requires that it is analyzed in terms of impending motor responses that are dependent on the perceived aversiveness of the stimuli.

Davidson's (1993) approach/withdrawal model of emotion specifically looks at hemispheric activation in the frontal lobes in relation to the behavioral response associated with positive and negative emotion. In neuroimaging studies of pain, bilateral activation (Lorenz et al., 2002; Lorenz, Minoshima, & Casey, 2003) and deactivation (Hsieh, Hannerz, & Ingvar, 1996; Tamura et al., 2004) of frontal cortex has been found. Investigations that have reported frontal activation with pain attribute the activation to the affective-motivational components of pain (Hsieh, Hannerz, & Ignvar, 1996; Fulbright et al., 2001). Positive or negative affective evaluation of a painful stimulus may influence resulting approach/withdrawal behaviors. Given the lack of research on pain and resulting approach/withdrawal behavior this is highly speculative; however, the fact that pain may result in left or right frontal hemisphere activation, as well as motivational behavior (Craig, 2003) supports the application of Davidson's (1993) approach/withdrawal model to pain processing.

In contrast to the previous investigations, Tamura et al. (2004) found that deactivation in the right frontal lobe was associated with a reduction in pain. Within the approach/withdrawal model, this may indicate a decrease in withdrawal related emotions leading to decreased pain as a result of a decrease in the amount of unpleasantness associated with the pain. Hsieh, Hannerz, and Ingvar (1996) suggested that bilateral deactivation in the prefrontal cortex found in response to pain was related to disengagement of attentional systems. They argue that pain is intrusive and causes inhibition of cognitive planning.

Evidence from migraine patients may lend additional support to the application of the approach/withdrawal model to pain. Avnon et al. (2004) proposed that evidence from participants with unilateral migraine suggests that the side of the migraine is positively associated with the level of cerebral activation in the corresponding hemisphere, such that right lateralized migraines occur with increased right hemispheric activation, while left lateralized migraines occur with increased left hemispheric activation. Fasmer and Oedegaard (2002) used migraine patients with unipolar and bipolar depression to describe this relationship. Fasmer and Oedegaard (2002) found that in bipolar patients, who may have more left hemisphere activation, migraine was more often located on the left side of the head. In contrast, in unipolar patients, with presumably more right hemisphere activation, the migraine was more often located on the right side of the head. Both unipolar and bipolar depression occur with differential behavioral patterns. Unipolar depression more often leads to social isolation and withdrawal. Bipolar depression, on the other hand, more often leads to differential periods of approach and withdrawal. The differential cerebral activation as a result of lateralized migraine may play a role in resulting affective state and motivational behavior. However, Brandt et al. (1990) investigated

the relationship between headache laterality, personality, and emotional traits and found no significant relationship.

Aversive and appetitive conditioning paradigms may be used to explain how emotions become associated with pain to produce approach or withdrawal responses. Results from Pavlov's (cited in Dickinson & Pearce, 1977) laboratory indicate that aversive shocks when paired with food elicit approach responses rather than withdrawal responses in dogs. The experiment indicates that the addition of desired reinforcement can alter the reflexive withdrawal response to escape the pain. In recent work with aversive and appetitive conditioning, Wunsch, Philippot, and Plaghki (2003) found an enhanced startle reflex in and higher intensity rating for pain associated with aversive stimuli and a diminished startle reflex and lower intensity rating for pain associated with appetitive stimuli. Previous work suggests that an enhanced startle reflex occurs during unpleasant emotional conditions, while a reduced startle response is associated with pleasant emotional conditions (Vrana, Spence, & Lang, 1988). The startle reflex is an index of defensive mobilization (Bradley et al., 2001) as such, increases in the startle reflex may be indicative of preparation for withdrawal. Accordingly, the study by Wunsch, Philippot, and Plaghki (2003) demonstrates that pain associated with aversive stimuli produces an increase in withdrawal behaviors, while pain associated with appetitive stimuli may produce a decrease in withdrawal behaviors.

The application of emotional models to pain is important due to the interactions that occur between the constructs. Emotion is noted to alter the experience of pain (Meagher, Arnau, & Rhudy, 2001) and pain may lead to increases in negative emotion (Janssen, Spinhoven, & Arntz, 2004). Altered pain experiences have been linked with anxiety (Grachev, Frederickson, & Apkarian, 2002), depression (Delgado, 2004), and anger or hostility disorders (Burns, Bruehl, &

Caceres, 2004). These negative emotional disorders typically produce increased pain; however, Janssen, Spinhoven, and Brosschot (2001) suggest that if anger is the result of pain it may have an inhibiting effect on pain. The differential effects that anger and hostility have on pain suggest a need to further empirically evaluate the neuropsychological effects of pain in individuals with increased levels of anger or hostility. In the past, investigations have focused on the relationship between pain and depression, while the role of anger and hostility in pain has been somewhat neglected (Janssen, Spinhoven, & Brosschot, 2001). The current proposal will focus on neuropsychological responses to pain which differ as a function of hostility level.

Hostility can be defined in terms of behavioral, cognitive, and physiological components. Behaviorally, hostility is described as aggressive behavior and social avoidance (Barefoot et al., 1989). Buss and Perry (1992) describe hostility as a cognitive component of aggression that includes negative feelings and injustice. Cognitively, hostility may also include hostile attributions of others and cynicism (Graves & Miller, 2003). Physiologically, hostility results in altered autonomic system functioning characterized by a higher resting heart rate (HR) and blood pressure (BP; Keefe, Castell, & Blumenthal, 1986) and increased HR and BP reactivity to stress (Davis, Matthews, & McGrath, 2000). Hostility's multifaceted definition, along with its link to cardiovascular disease (CVD; Matthews et al., 2004) have lead to increased study of the construct and several models of cerebral functioning that occur with heightened hostility.

Neuropsychology of Hostility

An extensive line of research exists detailing the neurobehavioral and neurophysiological effects of hostility. Altered facial affect recognition (Harrison & Gorelczenko, 1990, Larkin, Martin, & McClain, 2002), auditory (Demaree & Harrison, 1997; Mollet & Harrison, submitted for publication), somatosensory (Herridge, Harrison, & Demaree, 1997), visual (Harrison &

Gorelczenko, 1990; Herridge et al., 2004), motor (Demaree et al., 2002), and neuroendocrine systems (Saurez et al., 1998) have been found in individuals classified as high hostile by the Cook Medley Hostility Scale (Cook & Medley, 1954). Further, the altered systems functioning in hostility may be related to altered right cerebral functioning.

Hostility involves negative emotion, altered arousal level, and may lead to approach or withdrawal behavior depending on the environment or stress. Neuropsychological models of emotion suggest that the right hemisphere is important for negative emotion (Borod, 1992; Adolphs, Jansari, & Tranel, 2001). Right hemisphere lesions may result in reduced cortical and autonomic arousal (Morrow et al. 1981, Zoccolotti et al., 1982) and the right parietal cortex has been noted to play a crucial role in the maintenance of arousal (Heilman & Gilmore, 1998). Furthermore, approach/withdrawal models of emotion suggest that a functional relationship exists between the right frontal and the left frontal cortices whereby increased activation in the right frontal lobe leads to withdrawal related behaviors and decreased right frontal activation leads to approach behaviors (Davidson, 1993; 2000). A variation of this model also describes a relationship between right parietal activation and right frontal deactivation in producing approach/withdrawal related behaviors (Heilman & Gilmore, 1998; Schutter et al., 2001).

Given that the components of hostility involve right hemisphere function, increased levels of hostility may be concurrent with altered functioning in the right hemisphere. A proposed model of hostility suggests that, when confronted with an environmental stressor, high hostile individuals show increased lability within right hemispheric systems. This may be indicative of a decreased capacity to regulate behavioral, physiological, and neuropsychological effects of stress in high hostile individuals. Under confrontation, high hostiles may experience decreased activation in the right frontal lobe and increased activation in the temporal and parietal

cortices (Everhart & Harrison, 1995; Demaree & Harrison, 1996). Everhart and Harrison (1995) found support for this model in a patient with an anger disorder. Results from quantitative electroencephalogram (QEEG) indicated that during anger episodes the patient exhibited increased delta magnitudes at the right frontal lobe and increased beta magnitudes at the right temporal lobe.

Behavioral and physiological data from high hostiles also indicate altered right hemisphere functioning in hostility. Williamson and Harrison (2003) found that high hostiles produced more errors on a measure of design fluency (right frontal task) relative to low hostiles. Demaree and Harrison (1997) found that after a cold pressor high hostiles evidenced an increase in perception of speech sounds at the left ear (right hemisphere). High hostility has also been associated with decreased accuracy on facial affect perception tasks (Larkin, Martin, & McClain, 2002; Herridge et al., 2004). As noted previously, the right hemisphere may be specialized for facial affect perception (see Borod et al., 2002).

Increased physiological reactivity is generally found concurrent with increased levels of hostility (Demaree & Harrison, 1997; Shapiro et al. 2000). Data indicate that the right hemisphere plays a role in the regulation of HR and BP. Wittling et al. (1998) presented affective pictures to the right and left hemisphere and found that right hemisphere presentation lead to sympathetic nervous system changes. Yoon et al. (1997) measured changes in low-frequency (LF) and high frequency (HF) power before and after hemisphere inactivation. Results indicated lateralization in autonomic control and suggested right hemisphere control of sympathetic tone. Evidence from stroke populations further highlights the importance of the right hemisphere in mediating sympathetic arousal. Meyer et al. (2004) examined patients with right and left hemisphere stroke and found increased sympathetic tone was significantly associated with stroke

involving the right insular cortex. Lane and Schwartz (1987) suggested that lateralized cerebral responses to emotion cause lateralized imbalance in sympathetic input to the heart resulting in changes in cardiovascular reactivity. Foster and Harrison (2004) found that increased right cerebral activation is associated with increases in sympathetic tone and that relative differences in magnitude of cerebral asymmetries may determine overall changes in cardiovascular responses. In hostility, it is thought that increases in cardiovascular reactivity are resultant from a decreased capacity of the right frontal lobe to inhibit arousal that occurs as a result of activation of the right posterior cortex.

Empirical investigations within normal populations also provide support for altered right hemispheric activation in hostility and anger. Harmon-Jones and Allen (1998) found decreased resting right frontal activity in participants classified as high anger relative to participants classified as low anger. Waldstein et al. (2000) found that anger induction was consistent with bilateral frontal activation. This may be due to lack of data on participant trait hostility or anger level. The authors stated that anger can produce either approach or withdrawal behavior and that production of these behaviors may be related to an individuals preferred mode of anger expression. According to approach/withdrawal models of emotion anger expressers would activate the left frontal lobe, while anger suppressers would activate the right hemisphere. Thus, bilateral frontal activation may have resulted from a heterogeneous sample. However, in a similar sample of participants, Foster and Harrison (2002) found increased right temporal activation after anger induction. Although these results are from experiments involving anger, hostility and anger are defined in similar terms and scales that measure the constructs are often correlated. Mollet and Harrison (manuscript in preparation) found that scores from the Cook Medley Hostility Scale (Cook & Medley, 1954) were significantly correlated with scores on the

Buss and Perry Aggression Questionnaire (Buss & Perry, 1992). A significant correlation between the Cook Medley Hostility Scale (Cook & Medley, 1954) and the Spielberger Anger Scale was also found (Spicer & Chamberlain, 1996). Further, Spicer and Chamberlain found that high hostile participants reported experiencing anger more often.

Overall, data support the idea that hostility produces differential cerebral activation of the right hemisphere that may be dependent on trait or state. Evidence suggests that a dynamic system of activation and deactivation exists that includes both the anterior and posterior right hemisphere. Understanding cerebral activation may be important for predicting behavioral outcomes. In the current experiment, cerebral responses both at rest and after exposure to a painful stimulus are important because they may be altered in many of the same cerebral regions that are important for performing the emotional dichotic listening task that will be used.

Dichotic Listening

Dichotic listening is a task whereby two different stimuli are presented simultaneously to both ears. It was introduced as a laterality measure for speech processing by Kimura (1961a; 1961b). Since its introduction, researchers have designed novel manipulations that allow for the assessment of emotional prosody on stimulus detection (Bryden & MacRae, 1989, Wexler et al., 1986). The dichotic listening task in the current experiment is adapted from Voyer, Russell, and McKenna (2002). It consists of four words (bower, dower, power, and tower) spoken in four affective tones (happy, angry, neutral, and sad).

In investigations using emotional dichotic listening a consistent left ear advantage (right hemisphere) has been found for the perception of affective tone (Bryden, Ley, & Sugarman, 1982; Bryden & MacRae, 1989; Bulman-Flemming & Bryden, 1994; Snyder, Harrison, & Gorman, 1996; Jancke et al., 2001; Voyer, Russell, & McKenna, 2002). This is in contrast to a

right ear advantage (REA; left hemisphere) that is found for the identification of speech sounds (Kimura, 1961a). The left ear advantage for affective tone is thought to reflect the functional role of the right hemisphere in the perception of emotion (Bryden & MacRae, 1989), while the REA for speech is thought to reflect the left hemisphere's role in speech processing (Kimura, 1961a).

Imaging data provide further information about cerebral activation in emotional dichotic listening. Dichotic listening has been associated with temporal lobe functioning (Hugdahl, 1995) due to the auditory nature of the task. Imaging data indicate that emotional dichotic listening produces bilateral activation in the frontal, temporal, and parietal lobes (Jancke et al., 2001; Jancke & Shah, 2002; Jancke et al., 2003). Frontal lobe activation may result from vigilance to the stimuli (Jancke & Shah, 2002), while temporal lobe activation is a result of the auditory stimuli (Hugdahl, 1995). Bilateral activation may be a result of callosal transfer of verbal information to the left hemisphere and emotional information to the right hemisphere (Jancke et al. 2001).

Alternatively, bilateral activation may be a result of presentation of both positive and negative emotional tone. According to the valence hypothesis, the left posterior should be important for positive emotion, while the right posterior should be important negative emotion. Erhan et al. (1998) found partial support for the valence hypothesis using an emotional dichotic listening task. The authors found that participants who displayed a strong left ear advantage for the identification of emotional prosody also tended to show a greater left ear advantage in the identification of negative prosody relative to positive prosody.

Further, emotional status of participants may also influence which dichotic stimuli are attended to. As previously described, negative emotion and hostility may impact the right hemisphere and alter functioning in the frontal and temporal lobes. Activation of the frontal and

temporal lobes is important due to their role in attention to stimuli and auditory perception of stimuli (see above). In previous research negative emotional states have been shown to decrease the right ear advantage in the identification of speech sounds. Demaree and Harrison (1997) found an increase in the number of correct answers at the left ear when high hostile participants were given a cold pressor stressor, whereas the cold pressor increased the number of correct answers at the right ear for low hostile participants. A relatively low right ear advantage for processing speech sounds was found for repressors and high anxious participants relative to low anxious participants. This is consistent with the idea that repressors and high anxious participants have greater relative activation of the right hemisphere, producing avoidance for the laboratory task and inhibiting speech processing in the left hemisphere (Wexler et al., 1986). Gadea et al. (2005) found that negative emotional induction produced an increase in identification of dichotic stimuli at the left ear and a decrease in identification of dichotic stimuli at the right ear.

However, opposite effects of negative emotion on dichotic listening are also evident. In an experiment (Al'Absi, Hugdahl, & Lovallo, 2002) measuring cortisol secretion, which has been found to increase during negative emotions, men who secreted high amounts of cortisol in response to stress demonstrated a higher number of correct responses during dichotic listening than low cortisol responders. The authors attributed the increased performance of high cortisol responders on the dichotic listening task to a heightened arousal level and enhanced sensory intake leading to better selective attention (Al'Absi, Hugdahl, & Lovallo, 2002). Increases in arousal are related to right parietal activation (Heilman & Gilmore, 1998) which may influence attention to stimuli at the left hemibody. During manipulation of arousal level, a high negative arousal level was associated with more correct answers at the left ear and a decrease in correct answers at the right ear (Asbjornsen, Hugdahl, & Bryden, 1992). However, for high hostiles

increases in arousal may lead to decreased performance. Bell and Fox (2003) suggest that there may be an inverted U-shaped function that describes how cerebral activation influences cognitive performance. A "normal" level of hemispheric arousal may be advantageous to cognitive performance, while "extreme" levels of hemispheric arousal may be disadvantageous (Bell & Fox, 2003). High hostile individuals are noted to have increased physiological arousal at rest (Spicer & Chamberlain, 1996) and demonstrate heightened cardiovascular lability (Davis, Matthews, & McGrath, 2000). Heightened reactivity to an arousal manipulation in a system that is already physiologically aroused may lead to an extreme level of arousal that produces performance deficits.

Cold Pressor

The cold pressor was chosen as a painful stimulus because it has been noted to change dichotic laterality effects (Demaree & Harrison, 1997), because it is relatively easy to administer, and because it is a popular method of painful stimulation that does not produce long-term changes. Further, the cold pressor induces cerebral changes that may affect performance on the dichotic listening task. Chang, Arendt-Nielson, and Chen (2002) found that during a cold pressor test, participants had increased low-frequency activity (delta and theta bandwidths) in the bilateral frontal region and increased high-frequency activity (beta-1 and beta-2 bandwidths) in the bilateral temporal region. Di Piero et al. (1994) reported that the cold pressor not only produced severe pain in participants but also activated contralateral frontal and bilateral temporal regions as measured by single-photon emission tomography (SPET). The pattern of activation suggests that painful stimuli are able to activate cortex via somatosensory pathways (Di Piero et al., 1994). Submerging the participants' left arm in the cold pressor should activate the right brain regions that are associated with emotion perception, negative emotion, hostility and

arousal. Bilateral frontal and temporal activation are evident in emotional dichotic listening (see Jancke et al. 2001; Jancke & Shah, 2002) and the activation associated with the cold pressor is expected to increase negative affect perception on the emotional dichotic listening task for high hostiles. Additionally, activation of the parietal lobe has also been found subsequent to a cold pressor. QEEG studies examining the effects of the cold pressor indicate that cold pressor stimulation produces alpha-2 desynchronization over the contralateral parietal electrodes of the stimulated hand and that this effect lasts longer over the right hemisphere (Ferracuti, 1994). Increased activation of the right parietal lobe may lead to increased arousal and significantly alter performance on the dichotic listening task post-cold pressor pain.

Rationale

The current experiment was designed to examine the influence of hostility and pain on emotion perception using dichotic listening. The experiment measured high and low hostile participants' cerebral activation in response to pain. Specifically, high and low hostiles were asked to complete an emotional dichotic listening task before and after pain stress. It was thought that in high hostile individuals cold pressor pain would increase right temporal and parietal activation and increase perception of negative emotion. In contrast, it was expected that low hostile individuals would experience stable or relative increased left posterior brain activation in response to pain.

In order to increase the homogeneity of variance attributable to cerebral laterality, only men were recruited for participation. Considerable evidence suggests that differences in emotional processing and laterality exist among men and women (e.g., Ley & Bryden, 1979; Harrison, Gorelczenko, & Cook, 1990; Crews & Harrison, 1994; Hiscock et al, 2001). To avoid confounding these laterality effects the exclusion of women was necessary.

Variables

Independent Variables

Group. The independent variable group was based on scores from the Cook Medley Hostility Scale (Cook & Medley, 1954). Participants were classified as either high or low hostile.

Pain. For the dichotic listening analysis the independent variable pain consisted of two levels: pre-cold pressor and post-cold pressor. The pain variable was also used in the QEEG analysis for QEEG data collected immediately before and immediately after the cold pressor.

Affect. The variable affect consisted of three levels including neutral, happy, and angry affective tone.

Condition. The condition variable was included in the QEEG analysis and always consisted to of two levels. Since data were recorded around two separate task conditions, condition was comprised of two pre- and post-conditions. The first set of analysis included pre- and post-dichotic listening 1. The second analysis included pre- and post-dichotic listening 2.

Hemisphere. Hemisphere was used in the QEEG analysis and included data recorded from the locations located in the right and left hemispheres.

Location. Location was used in the QEEG analysis and included the following five levels: frontal-1 (F3/F4), frontal-2 (F7/F8), temporal-1 (T3/T4), temporal-2 (T5/T6) and parietal (P3/P4).

Dependent Variables

Score. The number of correctly identified stimuli was totaled for each affect at the left and right ears.

Laterality Index. The following formula was used to compute a laterality index:

$$\frac{\text{Number correct at the right ear - Number correct at the left ear}}{\text{Number correct at the right ear + Number correct at the left ear}}$$

Thus, scores for the laterality range from -1 to 1, with a negative number indicating a left ear advantage and a positive number indicating a right ear advantage.

QEEG. QEEG was used to measure delta (2-4 Hz), low beta (13 to 21 Hz) and high beta (21 to 32 Hz) magnitudes (μV) at the F3, F4, F7, F8, T3, T4, T5, T6, P3 and P4 electrode sites. Electrodes were arranged according to the 10/20 international system. During analysis, data was grouped into five locations: frontal-1 (F3/F4), frontal-2 (F7/F8), temporal-1 (T3/T4), temporal-2 (T5/T6), and parietal (P3/P4).

Hypotheses

Dichotic Listening

1. It was expected that participants would show increased identification of affect at the left ear relative to the right ear.
2. A main effect of affect was expected for the laterality index. Increased left lateralization for the identification of angry affect was expected.
3. A group x affect interaction was predicted for scores from the right and left ears. It was expected that the high hostile men would show increased identification of angry affect relative to the low hostile men.
4. A main effect of pain was expected for the scores from the left ear and the laterality index. It was predicted that after the cold pressor participants would show an increase in accurate identification of stimuli at the left ear.

5. Pain x affect interaction at the left ear was expected to indicate increased identification of angry affect at the left ear after cold pressor administration.
6. A group x pain interaction was expected for scores from the right and left ears. For the left ear, it was expected that after the cold pressor high hostiles would increase identification of stimuli at the left ear. At the right ear, it was predicted that low hostile participants would show a relative increase in the identification of stimuli after the cold pressor.
7. A group x pain x affect interaction at the left ear was expected to indicate increased identification of angry stimuli within the high hostile group after the cold- pressor.

Self-Report Data

1. Participants in the high hostile group were expected to report experiencing lower levels of pain and stress from the cold pressor relative to the low hostile group.

QEEG

1. The emotional dichotic listening task was expected to produce bilateral activation at the temporal region as a result of the verbal and emotional content of the stimuli. It was expected that a condition x location interaction would indicate increases in low (13-21 Hz) and high beta (22-32 Hz) magnitudes at the temporal-1 and temporal-2 locations for both trials of the dichotic listening task.
2. The cold pressor was expected to increase right temporal and parietal activation in participants as evidenced by increases in low (13-21 Hz) and high beta (22-32 Hz) magnitude at the right temporal-1, temporal-2, and parietal locations. Thus, a pain x hemisphere x location interaction was predicted.

3. High hostiles were expected to show increased reactivity to the cold pressor at the right hemisphere relative to the low hostile group. It was thought that a group x pain x hemisphere interaction would indicate increased low (13-21 Hz) and high (22-32 Hz) beta magnitudes in the right hemisphere for the high hostile group.
4. A group x pain x location interaction was expected for the delta bandwidth. Specifically, it was expected high hostile participants would show a decrease in frontal activation evidenced by an increase in low frequency high delta activity (2-4 Hz) at the frontal-1 and frontal-2 locations after cold pressor administration.
5. The cold pressor was expected produce relative increases in left hemispheric activation in low hostile participants as evidenced by increases in low (13-21 Hz) and high beta (22-32 Hz). A group x pain x hemisphere interaction was predicted to indicate relative activation in the left hemisphere in the low hostile group as a function of the cold pressor.

Method

Participants

Participants were recruited from the undergraduate psychology population. They completed an online pre-screening that included an Informed Consent Form (Appendix A), a Medical History Questionnaire (Appendix B), the Coren, Porac, and Duncan Laterality Questionnaire (Coren, Porac, & Duncan, 1979; Appendix C), and the Cook Medley Hostility Scale (Cook & Medley, 1954). Participants were excluded if they were left handed, had any uncorrected visual impairments, a history of head injury, any hearing impairments, a major medical disorder (i.e. thyroid condition, diabetes, etc.), a neurological disorder (i.e. Parkinson's Disease, Huntington's Disease, etc.), a history of neurological problems, a history of mental illness, were being treated for a current mental illness (i.e. depression, anxiety), or did not meet

scoring criteria on the Cook Medley Hostility Scale (Cook & Medley, 1954). Participants were asked to refrain from smoking for 2 hours and caffeine and alcohol for 12 hours prior to participation in the experiment.

Forty-six men completed the experiment. Fourteen participants were excluded for not meeting scoring criteria on the Cook Medley Hostility Scale (Cook & Medley, 1954) on the testing day. Two participants were excluded for removing their hands from the cold pressor early. Four participants were excluded for having beta magnitudes at one of the temporal lobe locations (either T3, T4, T5, or T6) 2 standard deviations above the mean (two from the high hostile group and two from the low hostile group). The final analysis included 13 high and 13 low hostile men between the ages of 18-24 (M=19.50, SD=1.50). Of the twenty-two participants that reported an ethnic/racial background, 14 were Caucasian, 3 were African American, 4 were Asian, and 1 was African.

Self-Report

Medical History Questionnaire: The Medical History Questionnaire is a 33 item self-report inventory that assesses the participant's current and previous health. Participants were asked to report and to explain medical conditions and history that may interfere with the experiment.

Coren, Porac, and Duncan Laterality Questionnaire: The Coren, Porac, and Duncan Laterality Test (Coren, Porac, & Duncan, 1979) is a 13 item self-report questionnaire to determine handedness. Scores can range from -13 to +13 indicating extreme left or right handedness. A score of +7 was required for participation in the experiment.

Cook Medley Hostility Scale: The Cook Medley Hostility Scale (Cook & Medley, 1954) is a self-report questionnaire purported to tap cynicism, anger, suspiciousness, and resentment in the hostility construct (Smith & Frohm, 1985). The scale consists of 50 true false items.

Participants who scored 29 or above were placed in the high hostile group, while those who scored 19 or below were classified as low hostile. This grouping criterion has been used previously in our lab (Harrison & Gorelczenko, 1990; Demaree & Harrison, 1997; Demaree et al., 2002; Williams & Harrison, 2003) and has been found to be successful. The general nature of the questions make it a trait, rather than state indicator of hostility (Demaree & Harrison, 1997). Its validity as a predictor of medical and psychological outcomes has made it one of the more commonly used measurements of hostility (Contrada & Jussim, 1992). Although the scale has been criticized as lacking the internal structure to predict psychological traits (Contrada & Jussim, 1992) its success in measuring hostility in previous research (Demaree & Harrison, 1997; Herridge, Harrison, & Demaree, 1997; Harrison & Gorelczenko, 1990; Davis, Matthews, & McGrath, 2000, Larkin, Martin, & McClain, 2002; Shapiro et al., 2000) demonstrates that it is a valid measure. Some example questions from the scale include "I have sometimes stayed away from another person because I feared saying or doing something that I might regret afterwards." "I feel that I have often been punished without cause." "I have often had to take orders from someone who did not know as much as I did."

Physiological

QEEG: QEEG was recorded and analyzed using the Lexicor Neurosearch – 24 system (Lexicor Medical Technology, 1992). The data were quantified online to digital values with a Gateway 486 DX computer for display, storage, and analysis. A high pass filter was used to eliminate low frequency artifact (below 2 Hz). The amplification factor was set to 32,000 with a sampling rate of 256 samples/second. Participants were fitted with a lycra electrode cap (Electro-Cap International, Inc.) by measuring the distance from the inion to the nasion. The participant's forehead was marked with a grease pencil 10% of the measured distance above the nasion. Head

circumference was measured by passing the measuring tape through the mark on the forehead. This measurement determined the cap size. The cap contained 19 electrodes arranged in the 10/20 International System. The participant's forehead and earlobes were wiped with an alcohol swab. The earlobes were then lightly abraded using a cotton swab and NuPrep (D.O. Weaver and Co.). The reference electrodes were then placed firmly on the participants earlobes. Two sponge disks were placed over the FP1 and FP2 electrodes on the electrocap. The disks were placed on the forehead on either side of the grease mark and the cap was pulled over the back of the participants scalp. The reference leads on the cap were attached to the leads on the earlobes and the cap was plugged into the electroboard. A blunt needle was attached to a syringe filled with NuPrep and used to prepare the electrode sites. Next, a blunt needle was attached to a syringe needle filled with electrode gel (Electro-Cap International, Inc.). The syringe was used to fill all the electrodes with electrode gel. An electrode impedance meter (Lexicor Medical Technology, Model 1089 MKII) was used to measure the impedance at each electrode. Impedance was adjusted to 5 k Ω or less at each site.

EOG. Auxiliary channels of the NeuroSearch-24 and silver-silver chloride electrodes filled with electrode gel (Electro-Cap International, Inc.) were used to measure EOG activity over the participant's left and right eyes. A bipolar electrode arrangement was used for each eye. One electrode was placed about 2 cm above the supraorbital margin and the other electrode was placed over the cheekbone. An alcohol pad was used to prep the EOG electrode sites.

Apparati

Dichotic Listening: Stimuli for the dichotic listening test consisted of four words (power, tower, dower, and bower) spoken by a male voice in three affective conditions (neutral, happy, and angry). The stimuli were recorded following the procedure used by Bryden and MacRae

(1989). They were adapted from Voyer, Russell, and McKenna (2002) and were administered using a similar procedure. Stimuli were recorded on a computer with a 16-bit sound card at a sampling rate of 22 kHz and 8-bit quality. Words were adjusted to a duration of 550 ms and an intensity of 70 decibels (dB). Each word and affective combination was presented to each ear, resulting in 72 trials. Before each testing condition, participants completed four practice trials in order to prepare them for and to familiarize them with, the task. The inter-trial interval was 5 s. Stimuli were presented to participants at 75 dB via Sony earphones. The dB level was re-checked after every 7 participants. Position of the earphones was counterbalanced between participants to control for any intensity differences. Participants were given a data sheet (see Appendix D) and asked to circle the emotion they heard most clearly on each trial.

Cold Pressor: Ice water for the cold pressor was maintained at 0-3 degrees Celsius using a small ice cooler. Water temperature was measured using a standard mercury thermometer. The cooler was located next to the participant's left arm.

Procedure

All procedures were approved by the Department of Psychology Human Subjects Committee and the Virginia Tech Institutional Review Board. After reviewing the results from the online pre-screening questionnaires, eligible participants were invited to the lab for participation. Upon arrival at the lab, participants were asked to complete the Informed Consent Form (see Appendix E). After completion of the Informed Consent Form, participants were fitted with the QEEG cap and EOG electrodes using the procedures outlined above. The experimenter then left the room. Participants heard the instructions for the remainder of the experiment through earphones. The experimenter watched the participants through a two-way mirror and

communicated with them via an intercom. To begin the experiment, participants were given the following instructions:

> Please sit relaxed in the chair with your eyes close. Please try to not to move your eyes, neck, or head and stay as relaxed as possible.

A 2 minute baseline QEEG and EOG sample was then recorded. Participants heard these instructions prior to collection of each QEEG sample. Following the baseline measurement, participants listened to the dichotic listening stimuli binaurally. Each word, spoken in each affective tone was played so that participants were familiar with the verbal and affective qualities of the stimuli. Next, they were read the instructions at the top of the dichotic listening data sheet and given an opportunity to ask questions. At completion of the dichotic listening task, a two minute QEEG and EOG sample was collected. Next, participants were asked to place their hand in the ice water. They heard the following instructions:

> When instructed, please place your left hand in the water to a point about one inch above your wrist. Please keep you hand in the water until instructed to remove it. This may be uncomfortable or painful, but please try and keep your hand in the water for the entire time. Do you have any questions? Begin.

After 45 seconds, participants were told to remove their hand and a two minute QEEG and EOG sample was collected. The dichotic listening task was then administered again, following the same procedure as before; however, a familiarization phase was not included. At completion of the second trial of the dichotic listening task, a final two minute QEEG and EOG measurement was collected. Upon completion of the experiment the QEEG cap and EOG electrodes were removed. Participants were asked to complete a questionnaire assessing the cold pressor (see Appendix F). They also re-took the Cook Medley Hostility Scale (Cook & Medley,

1954). Participants were then allowed to ask any questions they had regarding the experiment and then excused (see Appendix G for a flow chart of the procedures).

Results

Self Report Questionnaire Analysis

Separate t-tests were used to compare group means from the Coren, Porac, and Duncan Laterality Questionnaire (Coren, Porac, & Duncan, 1979) and the Cook Medley Hostility Scale (Cook & Medley, 1954). Results indicated that groups were statistically equivalent on the laterality questionnaire ($t(24) = .46$, $p = .64$). The mean score for the hostile group on the laterality questionnaire was 11.46 ($SD = 1.85$). The mean score for the low hostile group on the laterality questionnaire was 11.08 ($SD = 2.36$). Scores for the high and low hostile groups on the Cook Medley Hostility Scale (Cook & Medley, 1954) were significantly different ($t(24) = 13.51$, $p < .0001$). The high hostile group ($M = 33.85$, $SD = 3.99$) scored significantly higher on the Cook Medley Hostility Scale (Cook & Medley, 1954) relative to the low hostile group ($M = 14.14$, $SD = 2.36$).

A one-way ANOVA was used to examine group differences in level of stress and pain experienced during the cold pressor. A separate ANOVA was computed for the stress scores and the pain scores. Groups did not differ on their self-reported level of stress ($F(1, 24) = .42$, $p = .52$). However, a main effect of group ($F(1, 24) = 5.25$, $p < .03$) on the level of self-reported pain experienced during the cold pressor was found. Post-hoc comparisons indicated that high hostile men reported experiencing significantly more pain ($M = 5.54$, $SD = 1.05$) than low hostile men ($M = 4.46$, $SD = 1.33$).

Dichotic Listening Analysis

The following procedures were used to analyze data from the dichotic listening task. First, a three factor mixed design ANOVA was used. The ANOVA contained the following factors: the fixed effect of group (high or low hostile) and the repeated measures of affect (happy, angry, and neutral) x pain (pre- or post-cold pressor). Separate ANOVAs were computed for the total number of correctly identified affective tones at the right ear and the total number of correctly identified affective tones at the left ear. Additionally, a laterality index was calculated in order to determine ear advantage. A separate ANOVA was computed with laterality index as the dependent variable. The following formula was used to compute the LI:

$$\frac{\text{Number correct at the RE - Number correct at the LE}}{\text{Number correct at the RE + Number correct at the LE}}$$

Thus, scores for the LI range from -1 to 1, with a negative number indicating a left ear advantage and a positive number indicating a right ear advantage.

Right Ear

A main effect of pain ($F(1, 24) = 4.23$, $p = .05$) indicated that the cold pressor significantly increased the number of correctly identified stimuli at the right ear. In the pre-cold pressor condition the mean number of correctly identified stimuli at the right ear was 9.15 (SD = 4.27), while the mean number of correctly identified stimuli at the right ear in the post cold pressor condition was 9.86 (SD = 5.07). A group x pain interaction ($F(1, 24) = 5.91$, $p < .02$) indicated that this effect was primarily due to the high hostile group. Post-hoc analyses using Tukey's HSD indicated that the high hostile group significantly increased the number of stimuli identified at the right ear in the post-cold pressor condition, while the means for the low hostile group were not significantly different from pre- to post-cold pressor condition (see Figure 1).

A pain x affect interaction ($F (2, 48) = 4.32$, $p < .02$) further indicated that the increase in number of correctly identified stimuli at the right ear after the cold pressor was primarily due to an increase in identification of angry affect in the post-cold pressor condition. Post-hoc comparisons revealed that participants identified significantly more angry affective tones in the post-cold pressor condition. However, identification of neutral and happy stimuli was not significantly different as a function of the cold pressor (see Figure 2).

No other effects at the right ear were significant. Table 1 presents the ANOVA source table for the number of correctly identified stimuli at the right ear.

Left Ear

A group x pain interaction ($F (1, 24) = 4.92$, $p < .04$) was found for number of correctly identified stimuli at the left ear. The high hostile group evidenced decreased identification of stimuli at the left ear in the post cold pressor condition, while the low hostile group evidenced increased identification of stimuli at the left ear in the post cold pressor condition (see Figure 3). However, post-hoc analyses using Tukey's HSD revealed that these effects were not significant. Post-hoc comparisons further indicated that high and low hostiles did not significantly differ on identification of affect at the left ear in the pre- condition; however, in the post- condition low hostiles performed significantly better than high hostiles.

No other effects were significant at the left ear. See Table 2 for the ANOVA source table.

Laterality Index

A group x pain interaction ($F (1, 24) = 5.03$, $p < .04$) was found. The laterality index for the low hostile group indicated a greater left ear advantage in both the pre- and post-cold pressor conditions. The laterality index for the high hostile group shifted from a left ear advantage in the pre-cold pressor condition to no ear advantage in the post-cold pressor condition (see Figure 4).

The low hostile group exhibited an increased left ear advantage in the post cold pressor condition. Post-hoc comparisons using Tukey's HSD indicated that the low hostile group demonstrated a significantly increased left ear advantage in both the pre- and post-cold pressor conditions. However, within groups comparisons were not significantly different as a function of the cold pressor.

No other effects were significant for the laterality index. See Table 3 for the ANOVA source table.

Due to the length of the dichotic listening task (72 trials, 8 minutes and 03 seconds) a second set of analysis was conducted that included the additional effect of block (block 1 (trials 1-36) and block 2 (trials 37-72)). Three separate four factor mixed design ANOVAs with the fixed effect of group (high or low hostile) and with the repeated measures of pain (pre- or post-cold pressor), block (block 1 or block 2), and affect (neutral, happy or angry) were run on the number of stimuli correctly identified at the right and left ears and on the laterality index. The following effects of block were found.

Right Ear

A block x affect interaction ($F(2, 48) = 6.41$, $p < .003$) indicated that the accuracy in the identification of angry stimuli increased at the end of the task, while accurate identification neutral affect significantly decreased at the end of the task. Identification of happy affect did not differ as a function of block (see Figure 5). However, post-hoc comparisons indicated that this was not a reliable effect. Means were not significantly different between blocks when compared using Tukey's HSD.

Other results corresponded with the previous ANOVA. A main effect of pain ($F(1, 24) = 4.23$, $p = .05$) was found. Participants identified significantly more stimuli at the right ear in the

post-cold pressor condition (pre-cold pressor: M = 4.58, SD = 2.43, post-cold pressor: M = 4.93, SD = 2.69).

A group x pain interaction ($F(1, 24) = 5.91$, $p < .02$) indicated that the main effect of pain was primarily due to the high hostile group. Post-hoc comparisons indicated that high hostiles identified significantly more stimuli at the right ear in the post-cold pressor condition (M = 5.58, SD = 2.93) relative to the pre-cold pressor condition (M = 4.81, SD = 2.64). Means from pre- (M = 4.35, SD = 2.19) to post-cold pressor (M = 4.28, SD = 2.27) condition were not significantly different for the low hostile group.

A pain x affect interaction ($F(2, 48) = 4.32$, $p < .02$) indicated that the cold pressor increased identification of angry affect. Post-hoc comparisons indicated that accurate identification of angry affect was significantly increased after administration of the cold pressor (pre-cold pressor: M = 4.23, SD = 2.81; post-cold pressor: M = 5.21, SD = 2.57). Changes in identification of happy (pre-cold pressor: M = 4.69, SD = 2.81, post-cold pressor: M = 4.81, SD = 3.13) and neutral affect (pre-cold pressor: M = 4.81, SD = 2.41, post-cold pressor: M = 4.77, SD = 2.35) as a function of the cold pressor were not significantly different as indicated by post-hoc comparisons.

No other effects were significant at the right ear.

Left Ear

A block x affect interaction ($F(2, 48) = 5.91$, $p < .005$) indicated that identification of angry and neutral affect at the left ear underwent a pattern similar to that of identification of angry and neutral affect at the right ear. However, post-hoc comparisons indicated that changes in accurate identification of angry affect at the left ear were not significant. The only change that was significant during post-hoc comparison was a decrease in identification of neutral stimuli in

block 2 (see Figure 6). Additionally in block 1, identification of the happy and neutral tones was significantly greater than identification of the angry tone. In block 2, there was no significant difference in identification of happy, angry, or neutral tone.

A pain x block x affect interaction ($F (2, 48) = 3.62$ $p < .03$) indicated that this effect was modified by cold pressor administration. Post-hoc comparisons revealed that in block 1 of the pre-cold pressor condition, happy and neutral affect were identified significantly more than angry affect; however, there were no significant differences in identification of the affective tones in block 2 of the pre-cold pressor condition. In the post-cold pressor condition identification of the stimuli was not significantly different between blocks or affective tones (see Figure 7).

A group x pain interaction ($F (1, 24) = 4.92$, $p < .04$) was again significant. High hostiles had a significant reduction in the accuracy of affect identification at the left ear after cold pressor administration (pre-cold pressor: $M = 5.94$, $SD = 2.90$, post-cold: $M = 5.51$, $SD = 2.86$), while low hostiles identified more affect at the left ear as a function of the cold pressor (pre-cold pressor: $M = 6.40$, $SD = 2.34$, post-cold: $M = 6.81$, $SD = 2.34$). These differences were not significant when compared with Tukey's HSD. However, low hostiles in the post-cold pressor condition were significantly better at identification of affect when compared with the high hostile group in the pre- and post-cold pressor conditions.

No other effects were significant for scores at the left ear. The ANOVA source table is presented in Table 5.

Laterality Index

No significant effects of block were found for the laterality index. However, the group x pain interaction ($F (1, 24) = 6.92$, $p < .01$) was reliable. Consistent with the previous results, high

hostiles demonstrated less left lateralization for the identification of affective tones relative to the low hostile group. High hostiles had a reduced left ear advantage in the pre-cold pressor condition (M = -.11, SD = .37) that shifted to a no ear advantage in the post-cold pressor condition (M = .02, SD = .41). Low hostiles had a larger left ear advantage in both pre- (M = -.21, SD = .31) and post-cold pressor (M = -.26, SD = .29). Post-hoc comparisons indicated that the laterality index for the high and low hostiles groups was not significantly different in the pre-cold pressor condition; however, in the post-cold pressor condition low hostiles were significantly more left lateralized relative to the high hostile group.

The ANOVA source table is presented in Table 6.

Because no group differences were found for the accurate identification of the different affect an additional ANOVA was performed on the number of errors committed. An error score was computed by subtracting the number of correctly identified stimuli for each affect from the actual number of stimuli identified for each affect. A group (high or low hostile) x pain (pre- or post-cold pressor) x affect (neutral, happy or angry) ANOVA was then computed.

Error Score

A main effect of pain ($F(1, 24) = 11.60$, $p < .002$) indicated that the cold pressor significantly affected the number of errors committed. In the pre-cold pressor condition participants committed significantly more errors (M = 2.51, SD = 3.16) than they did in the post-cold pressor condition (M = 1.81, SD = 2.40).

A main effect of affect ($F(2, 48) = 11.16$, $p < .0001$) indicated that participants made significantly more errors during the identification of neutral stimuli (M = 3.92, SD = 3.53) relative to the identification of happy stimuli (M = 1.65, SD = 1.86) and angry stimuli (M = .90, SD = 1.81).

A pain x affect interaction ($F(2, 48) = 5.03$, $p < .01$) indicated that the number of errors committed was significantly different as a function of the cold pressor. Post-hoc comparisons indicated that the cold pressor significantly decreased the number of errors committed for the neutral stimuli, but did not significantly affect the number of errors committed during identification of happy or angry stimuli (see Figure 8).

No other effects were significant for the error score. See Table 7 for the ANOVA source table.

QEEG Analysis

Individual QEEG traces were visually inspected and artifacted offline. Raw data underwent Fast Fourier Transform and magnitudes were computed. Magnitude is defined as the voltage of a wave measured peak-to-peak and is reported in microvolts (μV; Stewart et al., 2001). Only artifact-free samples were retained for analysis. Artifacting the epochs involved deleting any one-second epoch noted to contain QEEG activity in which the magnitude exceeded ±50 μV as well as epochs containing eye movement artifacts as identified by EOG activity. QEEG samples had to contain at least 48 one-second epochs (e.g. at least 80% of each one-minute sample had to remain) to be included in the analysis.

To examine baseline effects of hostility and the effects of the dichotic listening task on the QEEG data a four factor mixed design ANOVA was used. The ANOVA included the fixed effects of group (high or low hostile) and the repeated measures of condition (pre- or post-dichotic listening trial 1), hemisphere (right or left), and location (frontal-1, frontal-2, temporal-1, temporal-2, or parietal). Separate ANOVAs were computed for the delta, low beta, and high beta bandwidths.

Delta (2-4 Hz)

A significant main effect of group ($F(1, 24) = 7.49$, $p < .01$) indicated that the low hostile group evidenced significantly higher delta magnitudes ($M = 5.22$, $SD = 1.49$) relative to the high hostile group ($M = 4.25$, $SD = 1.22$).

A main effect of location ($F(4, 96) = 42.90$, $p < .0001$) indicated that delta magnitudes were significantly higher at the frontal-1 and parietal locations relative to all other locations. Delta magnitudes at the temporal-1 location were significantly lower relative to all other sites (see Table 8).

No other effects were significant within the delta bandwidth. See Table 9 for the ANOVA source table.

Low Beta (13-21 Hz)

For low beta magnitude, a significant main effect of location was found ($F(4, 96) = 33.62$, $p < .0001$). Low beta values were significantly higher at the parietal location relative to the other four locations. Low beta was significantly higher at the temporal-2 location and the frontal-1 location relative to the temporal-1 and the frontal-2 locations (see Table 8).

A group x location interaction ($F(4, 96) = 2.43$, $p = .05$) indicated differences in low beta magnitude as a function of hostility level. However, post-hoc comparisons revealed that there were no significant between group differences in low beta magnitudes at the respective locations Both high and low hostile had significantly higher low beta magnitude at the parietal location relative to both frontal locations (see Figure 9).

Condition x hemisphere was significant ($F(1, 24) = 11.00$, $p < .003$). Low beta magnitude at the left hemisphere decreased as a function of the dichotic listening, while low beta magnitude at the right hemisphere increased as a function of the dichotic listening task; however, post-hoc

comparisons using Tukey's HSD indicated that these differences were not significant (see Figure 10).

A condition x location interaction ($F (4, 96) = 8.13$, $p < .0001$) was found for low beta magnitude. Post-hoc comparisons revealed that there were no significant differences in low beta magnitude from pre-dichotic listening 1 to post-dichotic listening 1 (see Figure 11). However, low beta magnitudes at the parietal location were again significantly greater relative to low beta magnitudes at the frontal-1, frontal-2, temporal-1, and temporal-2 locations.

A group x condition x location interaction ($F (4, 96) = 5.07$, $p < .0009$) was significant. Post-hoc comparisons again revealed that no differences in low beta magnitude existed at each location from pre- to post conditions. Further, no significant effects of group were found. The only significant difference during post-hoc comparisons was increased low beta magnitudes at the parietal location for both the high and low hostile groups across pre- and post-task conditions (see Figure 12).

No other effects were significant for low beta. The ANOVA source table is presented in Table 10.

High Beta (22-32 Hz)

For high beta a significant main effect of location ($F (4, 96) = 7.46$, $p < .0001$) was found. High beta magnitudes were significantly higher at the temporal-1, temporal-2, and parietal locations relative to the frontal-1 and frontal-2 locations (see Table 8).

A condition x hemisphere interaction ($F (1, 24) = 5.12$, $p < .03$) indicated that high beta magnitudes in the right and left hemispheres differed as a function of the task condition. Post-hoc comparisons indicated that the left hemisphere evidenced significantly higher beta magnitudes in the pre-dichotic listening 1 condition (baseline). After completion of the dichotic

listening task, participants evidenced a significant reduction in high beta magnitude over the left hemisphere (see Figure 13).

A condition x location interaction ($F (4, 96) = 9.45$, $p < .0001$) indicated that high beta magnitudes differed across locations as a function of the dichotic listening task. Post-hoc comparisons using Tukey's HSD indicated that there was a significant reduction in high beta magnitude at the temporal-1 location after completion of the dichotic listening task (see Figure 14); however, high beta magnitude at the other locations did not differ as a function of the dichotic listening task. A group x condition x location interaction ($F (4, 96) = 5.16$, $p < .0008$) further indicated that this effect differed as a function of group. Post-hoc comparisons using Tukey's HSD revealed that the high hostile group underwent a significant reduction in high beta magnitude at the temporal-1 location (see Figure 15) after completion of the dichotic listening task.

A condition x hemisphere x location interaction ($F (4, 96) = 3.13$, $p < .02$) was found. Post-hoc analysis indicated that high beta at the left temporal-1 location (T3) was significantly higher than high beta at the right temporal-1 location (T4) in the pre-dichotic listening 1 condition. However, after completion of the dichotic listening task there were no significant differences between right and left temporal-1 locations. Further, high beta magnitudes at the temporal-1 location were significantly higher than high beta magnitudes at the frontal-2 location at baseline. This effect was not present after completion of the dichotic listening task (see Figure 16).

No other effects were significant for the high beta bandwidth.

To examine the effects of the cold pressor on QEEG activity a group (high or low hostile) pain (pre- or post-cold pressor) x hemisphere (left or right) x location (frontal-1, frontal-2, temporal-1, temporal-2, or parietal) mixed design ANOVA was be used. A separate ANOVA

was computed for data delta, low beta, and high beta bandwidths. See Table 11 for the ANOVA source table.

Delta (2-4 Hz)

A significant main effect of group was found for delta magnitude ($F(1, 24) = 6.03$, $p < .02$). Similar to the previous ANOVA, low hostiles evidenced significantly higher delta magnitudes ($M = 5.12$, $SD = 1.42$) relative to the high hostile group ($M = 4.31$, $SD = 1.22$).

A significant main effect of location was found ($F(4, 96) = 54.68$, $p < .0001$). Participants evidenced significantly higher delta at the frontal-1 location and the parietal location relative the temporal locations. Delta magnitude at temporal-2 location was also significantly higher than delta magnitude at the temporal-1 location (see Table 8).

A significant pain x location interaction ($F(4, 96) = 5.25$, $p < .0007$) was present. Post-hoc comparisons indicated that delta magnitudes at the frontal-2 location significantly increased as a function of cold pressor administration (see Figure 17).

No other effects were significant for the delta bandwidth. See Table 12 for the ANOVA source table.

Low Beta (13-21 Hz)

A main effect of pain ($F(1, 24) = 8.61$, $p < .007$) indicated that low beta magnitude significantly increased from the pre-cold pressor ($M = 8.09$, $SD = 2.94$) to the post cold pressor ($M = 8.57$, $SD = 3.22$) condition. A group x pain interaction ($F(1, 24) = 5.83$, $p < .02$) indicated that this effect was primarily due to the high hostile group. Post-hoc comparisons indicated that high hostile men showed a significant increase in low beta magnitude in the post cold pressor condition; however, low beta magnitudes for low hostile men did not significantly change after cold pressor administration (see Figure 18).

Low beta magnitudes were also significantly different as a function of location ($F(4, 96) = 35.41$, $p < .0001$). Low beta was significantly higher at the posterior locations relative to the frontal locations (see Table 8). A group x location interaction ($F(4, 96) = 2.54$, $p < .04$) indicated that this was primarily due to the low hostile men. Post-hoc comparisons indicated that low hostiles evidenced significantly higher low beta magnitudes at the parietal location relative to the high hostile group. Low beta was not significantly different between groups at any other location (see Figure 19).

A significant pain x location interaction ($F(4, 96) = 11.16$, $p < .0001$) further indicated that low beta magnitude differed across sites as a function of cold pressor administration. Post-hoc comparisons indicated significantly increased low beta magnitudes at the frontal-1, temporal-2, and the parietal locations in the pre-cold pressor condition. However, in the post-cold pressor condition low beta magnitude was significantly higher at the temporal-1, temporal-2, and parietal locations relative to the frontal-1 and frontal-2 locations. These comparisons indicate that the cold pressor significantly increased low beta magnitudes at the temporal-1 location (see Figure 20). A group x pain x location interaction ($F(4, 96) = 6.98$, $p < .0001$) indicated that the increase in low beta at the temporal-1 location was primarily due to the high hostile group. Tukey's HSD post-hoc comparisons indicated that high hostiles evidenced significantly higher low beta magnitude at the temporal-1 location in the post-cold pressor condition. Low hostiles evidenced significantly increased low beta across all locations except for the temporal-1 location in the pre-cold pressor condition. In the post-cold pressor condition low hostiles had significantly increased low beta magnitude at the temporal-2 and parietal locations (see Figure 21).

A hemisphere x location interaction ($F(4, 96) = 2.62$, $p < .04$) was significant. Post-hoc comparisons revealed that no significant differences existed between low beta magnitudes at the

right and left hemispheres. In both the right and left hemispheres low beta magnitude was significantly higher at the frontal-1, temporal-1, temporal-2, and parietal locations relative to the frontal-2 location. Low beta magnitudes at the left temporal-2 location (T5) and the left parietal location (P3) were significantly higher than the left frontal-2 location (F3). In the right hemisphere, right parietal (P4) low beta magnitude was significantly higher than right frontal-1 (F4) low beta magnitude. Further, in both the right and left hemispheres low beta was significantly higher at the frontal-1 location relative to the frontal-2 location (see Figure 22).

A pain x hemisphere x location interaction ($F (4, 96) = 3.47$, $p < .01$) indicated that the cold pressor differentially influenced low beta magnitudes as a function of hemisphere and location. Post-hoc comparisons revealed that low beta magnitudes were not significantly different between the right and left hemispheres at the respective locations in the pre-cold pressor condition. However, in the post cold pressor condition post-hoc comparisons indicated a significant increase in low beta magnitude at the left temporal-1 location (T3). This effect was not found for the homologous location at the right hemisphere (i.e. T4; see Figure 23).

No other effects were significant for low beta magnitudes. See Table 13 for the ANOVA source table.

High Beta (22-32 Hz)

Changes in high beta magnitudes were similar to those found for low beta magnitudes. First, a significant main effect of pain ($F (1, 24) = 32.09$, $p < .0001$) indicated that high beta significantly increased from the pre-cold pressor condition ($M = 6.16$, $SD = 2.12$) to the post-cold pressor condition ($M = 7.31$, $SD = 3.12$). A significant group x pain interaction ($F (1, 24) = 9.28$, $p < .006$) indicated that this effect was primarily due to the high hostile group. Post-hoc comparisons indicated that high hostile men had significantly higher high beta magnitudes in the

post-cold pressor condition relative to the pre-cold pressor condition. High hostile men in the post-cold pressor condition also evidenced significantly higher high beta magnitudes relative to low hostile men in the post-cold pressor condition (see Figure 24).

High beta magnitude was significantly different as a function of location ($F(4, 96) = 8.35$, $p < .0001$). High beta was significantly higher at the temporal-1 and the parietal locations relative to high beta at the frontal-1 and frontal-2 locations (see Table 8).

A pain x location interaction ($F(4, 96) = 12.92$, $p < .0001$) indicated that the cold pressor increased high beta magnitude across locations. Post-hoc comparisons revealed that increases in high beta magnitude as a function of the cold pressor reached significance at all posterior locations (i.e. the temporal-1, temporal-2, and parietal locations); however, the increase was not significant for the frontal-1 and the frontal-2 locations (see Figure 25).

A significant group x pain x location interaction ($F(4, 96) = 6.00$, $p < .0002$) was found. Post-hoc comparisons revealed that the cold pressor significantly increased high beta magnitude at the temporal-1 and temporal-2 locations in the high hostile group; however, high beta magnitudes at the respective locations were not significantly different for the low hostile group (see Figure 26). Additionally, in the post-cold pressor condition high hostiles had significantly higher high beta magnitudes at the temporal-1 location and the parietal location relative to the low hostile group.

A significant pain x hemisphere x location ($F(4, 96) = 3.45$, $p < .01$) indicated that the cold pressor differentially affected cerebral activation in the right and left hemispheres (see Figure 27). Post-hoc comparisons indicated increased activation at the left temporal-1 location (T3) relative to the right temporal-1 location (T4) in the post-cold pressor condition. Further, in the right hemisphere high beta magnitude significantly increased at the temporal-1 (T4),

temporal-2 (T6), and the parietal (P4) locations from pre- to post-cold pressor conditions. In the left hemisphere, high beta significantly increased at the temporal-1 (T3) and temporal-2 (T5) locations only.

No other significant effects were found for high beta. See Table 14 for the ANOVA source table.

Finally, to examine the effects of the second dichotic listening task on QEEG activity a group (high or low hostile) x condition (pre- or post-dichotic listening 2) x hemisphere (left or right) x location (frontal-1, frontal-2, temporal-1, temporal-2, or parietal) mixed design ANOVA was used. A separate ANOVA was computed for data delta, low beta, and high beta bandwidths.

Delta (2-4 Hz)

Consistent with the previous results, a significant main effect of group ($F(1, 24) = 4.52$, $p < .04$) was found. Low hostiles had significantly higher delta values ($M = 5.09$, $SD = 1.42$) relative to the high hostile group ($M = 4.36$, $SD = 1.22$).

Additionally, a main effect of location ($F(4, 96) = 48.54$, $p < .0001$) indicated increased delta values at the frontal and parietal locations relative to the temporal locations (see Table 8).

No other effects were significant for the delta bandwidth. See Table 15 for the ANOVA source table.

Low Beta (13-21 Hz)

For low beta a significant effect of location ($F(4, 96) = 36.99$, $p < .0001$) was noted again. Low beta values were significantly higher at the parietal location relative to all other locations Low beta magnitudes at the frontal-2 location were significantly lower relative to all other sites (see Table 8).

A group x location interaction ($F(4, 96) = 2.73$, $p < .03$) indicated that low beta magnitude differed across locations as a function of group. Post-hoc comparisons revealed that low hostile men had higher low beta magnitude at the parietal location relative to high hostile men. Low beta magnitude at the parietal location in the low hostile group was also significantly higher than all other locations within the low hostile group. Low hostiles also evidenced increased low beta at the frontal-1 and temporal-2 locations relative to the frontal-2 and the temporal-1 locations. High hostiles evidenced increased low beta magnitudes at the frontal-1, temporal-1, temporal-2 and parietal locations relative to the frontal-2 location (see Figure 28).

A condition x location interaction ($F(4, 96) = 8.60$, $p < .0001$) indicated that participants increased low beta magnitudes after completion of the dichotic listening task across all locations except the temporal-1 location. Post-hoc comparisons revealed that these increases were not significant. However, the post-hoc comparisons did reveal that low beta magnitudes at the temporal-1 location underwent a significant reduction after completion of the dichotic listening task (see Figure 29). This effect was further influenced by group. A significant group x condition x location interaction ($F(4, 96) = 6.42$, $p < .0001$) indicated that the decrease in low beta at the temporal-1 location after completion of the dichotic listening task was primarily due to the high hostile group. Post-hoc comparisons revealed that there was a significant reduction in low beta magnitude at the temporal-1 location for the high hostile men. Low beta magnitudes at all other locations did not significantly differ from pre- to post-conditions. Low hostiles did not evidence significant changes in low beta magnitudes as a function of the dichotic listening task. Between groups comparisons indicated that in the pre-condition low hostile men had significantly increased low beta magnitude at the frontal-1 and the parietal locations. In the post-condition, the

only significant difference between groups was at the parietal location, where low hostiles evidenced increased low beta magnitude relative to the high hostile group (see Figure 30).

No other effects for low beta were significant. See Table 16 for the ANOVA source table.

High Beta (22-32 Hz)

A significant main effect of condition ($F (1, 24) = 11.78$, $p < .002$) was noted for high beta magnitudes. High beta magnitude significantly decreased from pre-dichotic listening 2 condition ($M = 6.83$, $SD = 2.90$) to post-dichotic listening 2 condition ($M = 6.18$, $SD = 2.20$). A group x condition interaction ($F (1, 24) = 4.99$, $p < .04$) indicated that this effect was primarily due to the high hostile group. Post-hoc comparisons using Tukey's HSD indicated that completion of the dichotic listening 2 task led to a significant reduction in cerebral activation in high hostile men, while no significant changes were noted for the low hostile group (see Figure 31)

A significant main effect of location ($F (4, 96) = 7.72$, $p < .0001$) indicated that high beta was significantly higher at the temporal-1, temporal-2, and parietal locations relative to the frontal-2 location. There was no significant difference between high beta magnitude between the temporal locations and the frontal-1 location. Additionally, the frontal-1 location was not significantly different from the frontal-2 location (see Table 8).

A condition x location interaction ($F (4, 96) = 7.50$, $p < .0001$) was significant. Post-hoc comparisons indicated that the only significant difference in high beta magnitudes from pre- to post-task conditions was at the temporal-1 location. High beta magnitude at the temporal-1 location significantly decreased after completion of the second dichotic listening task. Further, in the pre-task condition high beta magnitude was significantly higher at the temporal-1, temporal-2, and the parietal locations relative to the frontal-2 location. High beta at the temporal-1 and parietal locations was also significantly higher than high beta the frontal-1 location. However,

after completion of the dichotic listening task only high beta magnitude at the parietal location remained significantly higher relative to high beta magnitude at the frontal-2 location. High beta at the parietal location was also significantly higher than high beta at the temporal-1 location (see Figure 32).

Further, a significant group x condition x location interaction ($F (4, 96) = 7.17$, $p < .0001$) indicated the high beta magnitudes differed between groups. Post-hoc comparisons indicated that for the high hostile group, high beta magnitudes at the temporal-1 location were higher in the pre-task condition relative to the post-task condition. For the low hostile group, post-hoc comparisons revealed that high beta magnitudes at each location were not significantly different from pre- to post-task conditions. Further groups demonstrated relatively similar high beta magnitudes at each site from pre- to post-task conditions, except at the temporal-1 location. At this location, high hostiles had significantly higher high beta magnitudes in the pre-task condition relative to the low hostile group (see Figure 33).

No other effects were significant for high beta magnitudes. See Table 17 for the ANOVA source table.

Because cold pressor exposure led to group differences in performance on the dichotic listening task, several more refined ANOVAs were conducted on QEEG data collected before and after the cold pressor. The ANOVAs included only the frontal-1 and temporal-1 locations because these were the primary areas of interest. Additionally, the preceding analysis indicated significant changes at these locations as a function of the cold pressor. It was thought that the additional ANOVAs would help establish a more concrete relationship between cerebral activation to pain and hostility. First, a group (high or low hostile) x pain (pre- or post-cold pressor) x hemisphere (left or right) x location (frontal-1 or temporal-1) mixed design ANOVA

was used. A separate ANOVA was computed for data from the delta, low beta, and high beta bandwidths. After completion of this analysis, high and low hostile groups were analyzed individually using QEEG data collected before and after the cold pressor. A pain (pre- or post-cold pressor) x hemisphere (left or right) x location (frontal-1 or temporal-1) mixed design ANOVA was computed for the delta, low beta, and high beta bandwidths. The results are presented below.

Delta (2-4 Hz)

A significant main effect of group ($F(1, 24) = 6.06$, $p < .02$) was found. The low hostile group evidenced significantly higher delta magnitudes ($M = 4.90$, $SD = 1.49$) relative to the high hostile group ($M = 4.20$, $SD = 1.28$). A significant main effect of location ($F(1, 24) = 277.78$, $p < .0001$) indicated that delta magnitude was significantly higher at the frontal-1 location ($M = 5.55$, $SD = 1.13$) relative to the temporal-1 location ($M = 3.55$, $SD = .92$).

A pain x location interaction ($F(1, 24) = 5.31$, $p < .030$) was significant. However, post-hoc comparisons indicated that delta magnitude at the frontal-1 and the temporal-1 locations did not significantly change as a function of the cold pressor. Delta magnitude at the frontal-1 location was significantly higher than delta magnitude at the temporal-1 location in both the pre- and post-cold pressor conditions (see Figure 34).

A reliable group x pain x location interaction ($F(1, 24) = 9.10$, $p < .006$) indicated that the high and low hostiles responded differently to the cold pressor. Post-hoc comparisons indicated that the low hostiles evidenced significantly increased delta magnitudes at the frontal-1 location after cold pressor pain. Delta magnitudes at the frontal-1 location were not significantly different as a function of the cold pressor for the high hostile group. Delta magnitude at the temporal-1

location was not significantly different from pre- to post-cold pressor condition for either group (see Figure 35).

No other effects for the delta bandwidth were significant. Table 18 presents the ANOVA source table.

Low Beta (13-21 Hz)

A significant main effect of pain ($F(1, 24) = 10.79$, $p < .003$) indicated that the cold pressor significantly increased low beta magnitude (pre-cold pressor: $M = 7.38$, $SD = 2.29$; post-cold pressor: $M = 8.21$, $SD = 3.14$). A group x pain interaction ($F(1, 24) = 10.15$, $p < .004$) indicated that changes in low beta magnitude were primarily due to the high hostile group. Post-hoc comparisons revealed that in the pre-cold pressor condition low beta magnitude for the low hostile group was significantly higher for the low hostile group. However, high hostiles evidenced significantly increased low beta magnitudes as a function of the cold pressor, while the low hostile did not undergo a significant change in low beta magnitude after the cold pressor. (see Figure 36).

A pain x location interaction ($F(1, 24) = 15.76$, $p < .0006$) indicated changes in low beta magnitude after cold pressor exposure. Post-hoc comparisons revealed that there was a significant increase in low beta magnitude at the temporal-1 location from pre- to post-cold pressor conditions. In the pre-cold pressor condition low beta at the frontal-1 location was significantly higher than low beta magnitude at the temporal-1 location. However, in the post-cold pressor condition low beta magnitude at the frontal-1 location was not significantly different from low beta magnitude at the temporal-1 location (see Figure 37). This effect was further influenced by group. A significant group x pain x location interaction ($F(1, 24) = 6.83$, $p < .015$) indicated that the increase in low beta at the temporal-1 location after the cold pressor was

primarily due to the high hostile group. Post-hoc comparisons revealed that there was a significant increase in low beta magnitude at the temporal-1 location for the high hostile group after cold pressor exposure. This increase was not noted in the low hostile group. Low beta magnitudes at the frontal-1 location were not significantly affected by the cold pressor for either group. However, the in the pre-cold pressor condition low hostiles had significantly higher low beta magnitudes at the frontal-1 location relative to the high hostile group. In the post-cold pressor condition there was no significant difference between groups at the frontal-1 location (see Figure 38).

No other effects were significant for low beta magnitude. The ANOVA source table is presented in Table 19.

High Beta (22-32 Hz)

Results for high beta magnitude were similar to those for low beta. A significant main effect of location ($F(1, 24) = 4.15$, $p = 0.05$) was present. High beta values were significantly higher at the temporal-1 location ($M = 7.40$, $SD = 4.18$) relative to the frontal-1 location ($M = 6.23$, $SD = 1.72$).

A significant main effect of pain ($F(1, 24) = 23.76$, $p < .0001$) indicated that the cold pressor significantly increased high beta magnitude (pre-cold pressor: $M = 6.02$, $SD = 2.13$; post-cold pressor: $M = 7.61$, $SD = 3.91$).

A group x pain interaction ($F(1, 24) = 9.92$, $p < .004$) again indicated that the increase in high beta magnitude as a function of the cold pressor was primarily due to the high hostile group. Post-hoc comparisons revealed that high hostile men evidenced significantly increased high beta magnitudes as a function of the cold pressor, while the low hostile men did not undergo a significant change in high beta magnitude after cold pressor exposure (see Figure 39).

A pain x location interaction ($F (1, 24) = 19.87$, $p < .0002$) indicated that changes in high beta magnitude after cold pressor exposure were a function of location. Post-hoc comparisons revealed that there was a significant increase in high beta magnitude at the temporal-1 location from pre- to post-cold pressor conditions. There was no significant change in high beta magnitude at the frontal-1 location after cold pressor administration. In the pre-cold pressor condition there was no difference in high beta magnitudes between locations; however, in the post-cold pressor condition high beta magnitudes at the temporal-1 location were significantly higher than high beta magnitudes at the frontal-1 location (see Figure 40). This effect was further influenced by group. A significant group x pain x location interaction ($F (1, 24) = 7.59$, $p < .011$) indicated that the increase in high beta at the temporal-1 location after the cold pressor was primarily due to the high hostile group. Post-hoc comparisons revealed that there was a significant increase in high beta magnitude at the temporal-1 location for the high hostile men after cold pressor exposure. This effect was not significant in the low hostile group. Additionally, high beta magnitude at the temporal-1 location for the high hostile group in the post-cold pressor condition was significantly higher relative to the low hostile group. High beta magnitude at the frontal-1 location was not significantly affected by the cold pressor for either group. Further, high beta at the frontal-1 location was not significantly different between groups in either condition (see Figure 41).

A pain x hemisphere x location interaction ($F (1, 24) = 5.17$, $p < .03$) was significant. Post-hoc comparisons revealed that there were no significant differences between locations or hemispheres in the pre-cold pressor condition. After exposure to the cold pressor there was a significant increase in high beta magnitude at the temporal-1 location, but not at the frontal-1 location. Further, high beta magnitude in the frontal-1 location was not significantly different

between the right and left hemispheres. High beta magnitude at the left temporal-1 location (T3) was significantly higher than high beta magnitude at the right temporal-1 location (T4) in the post-cold pressor condition (see Figure 42).

No other effects were significant for high beta magnitude were significant. The ANOVA source table is presented in Table 20.

High Hostiles - Delta (2-4 Hz)

The only significant effect for the delta bandwidth within the high hostile group was a main effect of location ($F (1, 12) = 84.44$, $p < .0001$). Significantly higher delta magnitude was found at the frontal-1 location ($M = 5.09$, $SD = 1.09$) relative to the temporal-1 location ($M = 3.29$, $SD = 0.69$).

See Table 21 for the ANOVA source table.

High Hostiles - Low Beta (13-21 Hz)

A significant main effect of pain ($F (1, 12) = 13.73$, $p < .003$) was found for low beta magnitude. Post-hoc analysis indicated that the high hostile men had significantly higher low beta magnitudes in the post-cold pressor condition (pre-cold pressor: $M = 6.76$, $SD = 1.96$; post-cold pressor: $M = 8.39$, $SD = 3.58$). A pain x location interaction ($F (1, 12) = 12.66$, $p < .004$) indicated that this was primarily due to an increase in low beta magnitude at the temporal-1 location (see Figure 43). Post-hoc comparisons revealed that low beta magnitude significantly increased at the temporal-1 location in the post-cold pressor condition, but low beta magnitude at the frontal-1 location was not significantly different as a function of the cold pressor.

No other effects were significant for low beta magnitude. See Table 22 for the ANOVA source table.

High Hostiles - High Beta (22-32 Hz)

A significant main effect of pain ($F(1, 12) = 19.88$, $p < .0008$) was found for high beta magnitude. High beta magnitudes significantly increased in the post-cold pressor condition (pre-cold pressor: $M = 5.71$, $SD = 1.82$; post-cold pressor: $M = 8.32$, $SD = 4.63$). A pain x location interaction ($F(1, 12) = 14.36$, $p < .003$) indicated that this was primarily due to an increase in high beta magnitude at the temporal-1 location (see Figure 44). Post-hoc comparisons revealed that high beta magnitude significantly increased at the temporal-1 location in the post-cold pressor condition, but not at the frontal-1 location. Further support for this interaction was provided by a main effect of location ($F(1, 12) = 4.96$, $p < .05$). Significantly increased high beta magnitudes were found at the temporal-1 location ($M = 8.06$, $SD = 4.95$) relative to the frontal-1 location ($M = 5.98$, $SD = 1.22$).

A pain x hemisphere interaction was also present ($F(1, 12) = 4.75$, $p < .05$). Post-hoc comparisons using Tukey's HSD indicated that high beta magnitudes at the right and left hemispheres were not significantly different in the pre-cold pressor condition. In the post-cold pressor condition, significantly increased high beta magnitude at the both the right and left hemispheres was found. Additionally, high beta magnitude at the left hemisphere was significantly increased relative to high beta magnitude at the right hemisphere in the post-cold pressor condition (see Figure 45).

A pain x hemisphere x location interaction ($F(1, 12) = 5.86$, $p < .03$) was significant. Post-hoc comparisons indicated that there was no significant change in high beta magnitude at the frontal-1 location as a function of the cold pressor. High beta magnitude at the temporal-1 location was significantly increased after cold pressor exposure. Additionally, high beta

magnitude at the left temporal-1 location (T3) was significantly higher than high beta magnitude at the right temporal-1 location (T4) in the post-cold pressor condition (see Figure 46).

No other effects were significant for high beta magnitude. The ANOVA source table is presented in Table 23.

Low Hostiles - Delta (2-4 Hz)

For the low hostile group, a main effect of location ($F(1, 12) = 253.62$, $p < .0001$) was found in the delta bandwidth. Low hostile men evidenced significantly higher delta magnitudes at the frontal-1 location ($M = 6.00$, $SD = .98$) relative to the temporal-1 location ($M = 3.81$, $SD = 1.04$).

A pain x location interaction ($F(1, 12) = 7.79$, $p < .02$) indicated that the cold pressor increased delta magnitudes at the frontal-1 location (pre-cold pressor: $M = 5.78$, $SD = .89$; post-cold pressor: $M = 6.22$, $SD = 1.03$) and at the temporal-1 location (pre-cold pressor: $M = 3.95$, $SD = 1.21$; post-cold pressor: $M = 3.67$, $SD = .84$) from pre- to post cold pressor conditions. However, post-hoc comparisons indicated that these increases were not significant.

No other effects were significant for the delta bandwidth within the low hostile group. The ANOVA source table is presented in Table 24.

Low Hostiles - Low Beta (13-21 Hz)

For low beta magnitudes within the low hostile group, a main effect of location ($F(1, 12) = 6.59$, $p < .025$) was significant. Low hostile men had significantly higher low beta magnitude at the frontal-1 location ($M = 8.61$, $SD = 2.06$) relative to the temporal-1 location ($M = 7.40$, $SD = 2.82$).

No other effects were significant for the low hostile group within the low beta bandwidth. The ANOVA source table is presented in Table 25.

Low Hostiles - High Beta (22-32 Hz)

For the high beta bandwidth, a pain x location interaction was present ($F(1, 12) = 7.71$, $p < .02$). Similar to the high hostile group, post-hoc comparisons revealed a significant increase in high beta magnitude at the temporal-1 location as a function of the cold pressor (see Figure 47).

No other effects were significant for the low hostile group within the high beta bandwidth. The ANOVA source table is presented in Table 26.

Discussion

The current experiment examined cerebral activation in high and low hostile men during emotion perception and before and after exposure to cold pressor pain. Cerebral activation was measured functionally through dichotic listening and neurophysiologically using QEEG. The primary findings of the experiment indicate a reduction in lateralization of emotion perception and activation of the left hemisphere in response to cold pressor pain in high hostile men. Although, reduced laterality for emotion perception replicates previous research within high hostile men (Herridge et al. 2004), this effect was not predicted, nor was left hemisphere activation to the cold pressor. Originally, it was predicted that high hostile men would show increased right lateralization of emotion perception and activation of the right hemisphere in response to cold pressor pain. While the primary findings of the current experiment were unexpected, they provide additional data regarding hostility and may lead to a better understanding and conceptualization of the neuropsychological effects of hostility on emotion perception and reactivity to cold pressor pain in high and low hostile men.

Results from the dichotic listening task indicate that cerebral lateralization of emotion perception differed among high and low hostile men. Further, lateralization of emotion perception was noted to change as a function of cold pressor administration. However, the effects

of the cold pressor were diametrically opposite to the hypothesized relationship. It was thought that the cold pressor would increase identification of emotion at the left ear for the high hostile group, while the low hostile group was expected to show a relative increase in identification of emotion at the right ear in response to the cold pressor. Instead, results indicated that the high hostile group increased identification of stimuli at the right ear after exposure to the cold pressor. This is suggestive of an increase in left temporal activation as a result of the cold pressor in the high hostile group. In contrast, the low hostile group had an increase in the identification of stimuli at the left ear as a function of the cold pressor. Indeed, QEEG data provide additional support for these effects. Primary QEEG results indicated significant increases in low and high beta activation at the left anterior temporal location (T3) for the high hostile group after administration of the cold pressor. For the low hostile group, QEEG results indicated a bilateral increase in beta activity at the anterior temporal and parietal lobes and a bilateral increase in delta magnitude at the frontal lobes after the cold pressor. Increased bilateral beta activity at the temporal and parietal lobes may be indicative of an increase in general arousal level in the low hostile group after the cold pressor.

Several major hypotheses for the present experiment were based on a similar experiment in our laboratory. Demaree and Harrison (1997) found that high hostile participants respond to the cold pressor task with increased activation of the right hemisphere as measured by dichotic listening. However, the current dichotic listening results are suggestive of activation of the left hemisphere in response to the cold pressor in high hostiles. While the current results appear to be contradictory to Demaree and Harrison (1997), further inspection of the experimental task demands suggests the results are quite similar. Demaree and Harrison (1997) measured dichotic listening in high and low hostile men using linguistic (consonant-vowel sounds) stimuli. The

current experiment used an emotional dichotic listening task. In both experiments high hostile men showed increased identification of stimuli at the non-dominant ear for the task after exposure to the cold pressor. The experiments suggest that there was increased activation of the non-dominant hemisphere for the task in high hostile men following the cold pressor. In the present study, results indicated increased identification of stimuli at the right ear after the cold pressor rather than the left ear.

Previous research using emotional dichotic listening indicates a left ear advantage for the identification of emotion (Bryden, Ley, & Sugarman, 1982; Bryden & MacRae, 1989; Bulman-Flemming & Bryden, 1994; Snyder, Harrison, & Gorman, 1996; Jancke et al., 2001; Voyer, Russell, & McKenna, 2002). Laterality indices for both the high and low hostile groups indicated a left ear advantage in the pre-cold pressor condition. Moreover, the laterality index for the low hostile group indicated that low hostile men identified more stimuli at the left ear relative to the high hostile group. In the post-cold pressor condition the laterality index indicated that high hostile men underwent a reduction in laterality as a function of the cold pressor, while the low hostile men showed increased laterality as a function of the cold pressor. Further, in the post-cold pressor condition low hostile men identified significantly more affect tones at the left ear relative to the high hostile men. These results indicate reduced laterality for emotion in high hostile men and suggest that cerebral lateralization for emotion changes as a function of the cold pressor in high and low hostile men.

Reduced cerebral laterality in high hostile men during emotion perception has been noted in the visual modality as well. Herridge et al. (2004) found that overall accuracy in facial affect identification was similar in the right and left visual fields for high hostile men; however, low hostile men showed a marked left visual field advantage for facial affect identification. Further,

high hostile men were more accurate in the identification of angry and happy faces in the right visual field relative to low hostile men. The results were interpreted as evidence for right cerebral dysfunction in hostility. Thus, the current results provide additional evidence that high hostile men have reduced right lateralization for emotional processing across sensory perceptual systems.

Reduced cerebral laterality for linguistic speech processing as measured by dichotic listening (i.e. reduced right ear advantage for identification of consonant-vowel sounds) has been found in schizophrenia (Wexler, Giller, & Southwick, 1991; Ragland et al., 1992; Bruder et al., 1995), depression (Bruder et al.,1992; Wale & Carr, 1990), and social phobia (Bruder et al., 2004). Reductions in lateralized linguistic speech processing among groups with psychopathologies have been attributed to dysfunction within the left hemisphere (Bruder et al., 2004). Accordingly, reduced cerebral laterality for emotion found within high hostile men in the present experiment may be related to right hemisphere dysfunction in hostility.

However, it should be noted that reduced cerebral laterality is not always interpreted as indicative of hemispheric dysfunction. Sex difference literature has repeatedly reported reduced cerebral laterality in women (Baxter et al. 2003; Hiscock et al., 1994; Hiscock et al., 1995). Reduced laterality in women is usually used to describe why women may excel at one task and have deficits in another. For example, Levy (1969) proposed that women's increased performance on verbal tasks arises from using both the right and left hemispheres for language tasks. However, spatial performance, which is typically a right hemisphere task, decreases due to bilateral language representation. Bilateral representation of language is thought to interfere with the development of neural areas that are used for spatial processing (Levy & Reid, 1978; McGlone, 1980). A similar hypothesis may be made for high and low hostile men when the

current data are considered with respect to previous research. Hostility has been conceptualized as primarily affecting the right hemisphere.

The results from the present experiment indicate that heightened hostility also affects the left hemisphere. A reduction in right lateralization for emotional processing in the post-cold pressor condition suggests that high hostiles differentially relied on emotional processing by the left hemisphere after the cold pressor. Use of the left hemisphere for emotional processing in high hostile men may be an attempt to compensate for a reduced functional capacity of the right hemisphere after cold pressor pain. Case studies of split-brain patients indicate that language functioning may develop in the right hemisphere when it is disconnected from the left hemisphere (Gazzaniga, Ivry, & Mangun, 2002). Bates et al. (2001) reported that when unilateral brain damage occurs early in life, there is reorganization of language functioning, such that the right hemisphere is able to acquire language. These examples indicate that changes in cerebral lateralization or brain organization are possible after injury. It is plausible then, that if emotional processing centers in the right hemisphere are compromised due to heightened levels of hostility, emotion processing may shift to the left hemisphere. Additionally, current results indicate that the cold pressor primarily activated the left hemisphere in high hostile men. This effect has not been noted in previous research and may be related to the emotion associated with cold pressor pain. Pain is defined as having both sensory and affective components. Thus, left hemisphere activation in high hostile men may reflect cerebral processing of the emotional component of the pain.

Although shifting emotional processing to the left hemisphere when the right hemisphere is compromised may be seen as an adaptive response, it does not necessarily suggest that accurate processing of emotional stimuli will occur. Gazzaniga, Ivy, and Magnun (2002) point

out that when speech is produced from the right hemisphere, it is often quite different from speech that is produced in the left hemisphere. Right hemisphere speech generally includes only one word utterances (Gazzaniga, Ivy, & Magnun, 2002). Processing emotion from the left hemisphere may also undergo similar changes. The left hemisphere is described as a sequential processor. Processing emotion as a series of sequential events may lead to misinterpretation of emotional events or singling out one aspect of an emotional stimulus. Misinterpretation of emotional events may be related to increased feelings of negativity and feeling that others are in opposition to you that is noted in the hostile construct. Perseveration of negative information may also occur. If negative emotional stimuli are more salient, as has been suggested (Dahl, 2001), high hostile men might be more likely to pick a negative emotional stimulus or event out of a series and perseverate on it.

Moreover, using the left hemisphere for emotional processing may lead to negative outcomes for other left hemisphere functions. Traditionally, the left hemisphere is associated with expression and comprehension of speech processing. Recruiting those language areas for the processing of emotion may lead to a reduction in verbal fluency and a reduction in verbal learning or speech comprehension. Indeed, in a verbal learning investigation, high hostile men were noted to acquire lists of words slower than low hostile men (Mollet & Harrison, submitted for publication). Altered speech expression and comprehension may have implications for social interactions. High hostile men may have difficulty expressing themselves during confrontation. Failure to successfully communicate may lead to increased stress and aggression in high hostile men. Further, failure to understand or appreciate the speech of others may lead to a lack of situational awareness and a tendency to attribute the stressor or problem to others. Increased

stress, aggression, and a tendency to assume others are the source of problems are all noted within the hostile construct.

Thus, a working hypothesis regarding reduced cerebral lateralization in hostility that is analogous to hypotheses regarding reduced cerebral lateralization in women may be that bilateral representation of emotion perception in high hostiles could give them an advantage for emotional processing. However, bilateral representation of emotion perception may also produce deficits on language tasks. Further, stronger support for this hypothesis may be found if research is conducted in populations other than undergraduate students. Undergraduates typically come from affluent backgrounds and are required to maintain high levels of verbal proficiency.

While the dichotic listening results of the present experiment are supportive of reduced laterality for emotion perception within high hostile men (a replication of previous research), several other hypotheses based on empirical data went unsupported. In previous research, high hostile men have displayed a negative affective bias in the visual modality (Harrison & Gorelczenkco, 1990) and in affective learning (Mollet & Harrison, submitted for publication). Further, there is a significant amount of literature that indicates mood congruency for affective memory and perception (see Rusting, 1998 for a review). Based on this it was expected that high hostiles would show increased accuracy in the identification of angry stimuli. No support was found for this hypothesis. The lack of a negative affective bias may be related to a reluctance to endorse anger in the presence of the experimenter.

The experiment also failed to find evidence of differential functioning of the right and left frontal lobes in high hostiles after exposure to the cold pressor. Decreased right frontal functioning in high hostile men as measured by performance on a design fluency task has been noted (Williamson & Harrison, 2003). Moreover, increases in right frontal delta magnitudes

were noted during anger episodes in a patient with an anger disorder (Everhart & Harrison, 1995). It was hypothesized that high hostiles would evidence increased delta magnitude at F4 and F8 after the cold pressor. This effect was not present. Instead, QEEG data suggest bilateral frontal deactivation for the low hostile group as a result of the cold pressor. Low hostile men had a significant increase in delta activity at F3 and F4 in the post-cold pressor condition. This effect was not present for the high hostile group. However, a bilateral increase in delta magnitude at F7 and F8 after the cold pressor was found in both high and low hostile men.

It was hypothesized that high hostile men would report experiencing less pain as a result of the cold pressor. Previous investigations have indicated that anger may be related to an increase in pain tolerance (Janssen, Spinhoven, & Brosschot, 2001; Burns, Bruehl, & Caceres, 2004) and that this relationship may be mediated through an increased BP reactivity to pain during anger. Instead, results indicated that high hostile participants reported experiencing a significantly higher level of pain as a result of the cold pressor relative to the low hostile participants. However, this finding is not unsupported in the literature. Participants who have scored high on measures of anger suppression or anger-in have been noted to report increased pain (Janssen, Spinhoven, & Brosschot, 2001). Moreover, QEEG data in the present experiment provide support for increased pain in high hostile men. The high hostile group showed increased cerebral activation to the cold pressor, especially at the anterior left temporal lobe. As discussed above, this may be related to cerebral processing of the emotional component of pain. Differential processing of pain in high and low hostile men may be responsible for the increased level of pain reported within the high hostile group.

Other results from the experiment provide information about cerebral activation during emotional processing. Dichotic listening results provided support for the importance of the right

hemisphere in emotional processing. Both groups showed increased accuracy in the identification of emotion at the left ear. Results also indicated that identification of affect changed as the task progressed. During the first 36 trials of the task, participants identified more neutral stimuli at both the right and left ears. In the last 36 trials there was increased identification of angry stimuli at both the right and left ears. QEEG data may provide insight to this effect. Increased left hemisphere activation at baseline and after exposure to the cold pressor, were noted. Thus, increased identification of neutral stimuli during the first 36 trials may be a function of left hemisphere activation. Further, after completion of the first dichotic listening task there was a significant decrease in left hemisphere high beta magnitude. Decreased left hemisphere activation may have led to the shift from identification of neutral to angry stimuli as the task progressed.

Influences of the cold pressor on the dichotic listening task were also noted. However, the results were diametrically opposite of the predicted relationship. It was expected that the cold pressor would increase identification of affect at the left ear and that this effect would be most noted in the identification of angry affect within the high hostile group. Instead, results indicated an increase in identification of angry stimuli at the right ear as a function of the cold pressor regardless of hostility level. However, a group x pain interaction indicated that the increase in correctly identified stimuli at the right ear was primarily due to the high hostile group. The lack of increased identification of stimuli at the left ear may be partially due to a ceiling effect. It may be that the cold pressor did not increase identification of affect at the left ear because this value was already elevated in the pre-cold pressor condition. Regardless, these results seem to provide evidence against the valence model. No support was found for lateralization of positive and negative affect to the left and right hemispheres before or after the cold pressor.

Results from the QEEG data collected before and after the dichotic listening trials correspond with previous research examining cerebral functioning during completion of this task. Increased activation was noted at the frontal-1 and parietal locations during both trials of the dichotic listening task as evidenced by increased high and low beta magnitudes. Other neuroimaging investigations using similar tasks have reported bilateral frontal, temporal, and parietal activation as a function of emotional dichotic listening task (Jancke et al., 2001; Jancke & Shah, 2002; Jancke et al., 2003). The lack of temporal activation as a function of the dichotic listening task in the current experiment may be related to the fact that the high and low beta magnitudes at the temporal-1 location were significantly elevated at baseline and after exposure to the cold pressor.

QEEG data collected before and after the cold pressor provides additional information about regional brain activity after cold pressor exposure. Primary findings indicated increased delta at the frontal-2 location and increased low and high beta at the temporal-1, temporal-2 and parietal locations. For the low and high beta bandwidths, right hemisphere activation was greater at the parietal location, while left hemisphere activation was greater for the temporal locations. Increased delta at the frontal-2 location and increased beta at the right parietal location coincides with previous research examining cerebral activation to the cold pressor (see Di Piero et al., 1994; Ferracuti, 1994; Chang, Arendt-Nelson, & Chen, 2002).

Left temporal activation in response to the cold pressor has not been reported; however, there is one report of left hemisphere activation after painful heat. Schlereth et al. (2003) reported increased activation in the left insular region after exposure to painful heat stimuli regardless of which side of the body was stimulated. The authors suggest that the left hemisphere may play an important role in the early discriminative components of pain processing, whereas

the right hemisphere may be more important in processing the late components of pain. This interpretation may correspond with application of motivational models of emotion to pain processing. The early components of pain may be associated with approach behavior as a result of trying to alleviate the pain or remove the painful stimuli and thus require left hemisphere activation. Later components of pain may be more related to withdrawal and right hemisphere activation as a result of an affective response associated with the pain. Preliminary support for this interpretation is provided by results the dichotic listening task. For dichotic listening, a pain x block x affect interaction for the number of correctly identified stimuli at the left ear indicated that identification of neutral stimuli decreased, while identification of affect increased in the second block of the post-cold pressor condition. This may indicate changes in right hemispheric functioning during completion of the dichotic listening task. Specifically, it is possible that cerebral activation in the right hemisphere increased as the task progressed. Moreover, this interaction was not significant at the right ear. Perhaps a longer task may be better at detecting shifts in left and right hemispheric activation as a result of pain processing. QEEG data collected before and after the dichotic listening trials may provide additional support for a left hemisphere role in processing the early components of pain. After completion of the second trial of dichotic listening, the increased left hemisphere activation that was noted after the cold pressor was no longer present. Moreover, a condition x hemisphere x location interaction indicated that high beta magnitudes at the left temporal-1 location (T3) were significantly increased relative to the right temporal-1 location (T4) at baseline. Heightened left anterior temporal high beta magnitude at baseline may reflect cerebral processing of pain experienced while participants were fitted with the EEG cap. Although the cap fitting was not intended to be a painful experience and relatively few participants complained of pain during the fitting, preparation of the electrodes

may be an uncomfortable experience. While this may be speculative, it deserves future consideration as it may reflect a methodological problem in collecting baseline EEG data. Inclusion of a self-report questionnaire assessing pain during cap fitting may serve as a possible remedy for this problem.

An alternative explanation to left hemisphere activation to the cold pressor is provided by Chang, Ardent-Nielson, and Chen (2002). They suggest that bilateral increases in beta activity at frontal and temporal sites may be related to increased muscle tension in response to cold pressor pain. Indeed, frontal and temporal electrodes overlap facial muscles. A recent investigation in our laboratory noted increased facial motor tone (as measured by EMG) at the left and right masseter in high and low hostile men in response to the cold pressor. Further, EMG activity was greater in high hostile men, especially at the left masseter (Rhodes & Harrison, unpublished). However, others (Reinert, Treede, & Bromm, 2000) have concluded that increased beta activation to pain is reflective of hyperarousal. In the present study, it is likely that increases in beta activation reflect changes in cerebral activation and arousal level due to cold pressor pain. There was a consistent group x pain interaction for the high and low beta bandwidths that indicated increased reactivity to the cold pressor pain in the high hostile group. In contrast, low hostiles seemed to have exhibited hypoarousal. A consistent main effect of group in the delta bandwidth indicated that low hostiles had increased slow-wave activity.

Evidence for the validity of the QEEG data is provided by the performance of the two groups on the dichotic listening task. First, in the post-cold pressor condition, high hostiles identified significantly more stimuli at the right ear, which is suggestive of increased left hemisphere activation. Second, overall performance on the dichotic listening task in the pre- and post-cold pressor conditions suggests that the groups were at different levels of arousal

throughout the experiment. Heightened levels of hostility have traditionally been associated with increased reactivity to the cold pressor. Arousal theory states that performance varies on an inverted U function, with over- and under-arousal leading to decrements in performance. Since the cold pressor increased performance (increased the left ear advantage for emotional processing) in the low hostile group, it may be that the cold pressor increased arousal level and subsequent performance. In contrast, the high hostile group may have experienced opposite effects. The cold pressor led to a decrease in performance in the high hostile group (decreased the left ear advantage for emotional processing), suggesting that high hostiles were in a state of hyperarousal after cold pressor administration.

Conclusions

There are several theoretical implications from the present experiment. In general, the results provide evidence against the valence model of emotional processing. As previously discussed, left hemisphere activation in the valence model of emotion is associated with increases in positive affect and a reduction in the experience of pain. Here, increased left hemisphere activation was found within high hostile men after exposure to cold pressor pain. Further, the increase in left hemisphere activation in the high hostile men occurred with an increased level of self-reported pain. The results are interpreted as providing evidence of left hemisphere processing of emotion and left hemisphere activation to a painful stimulus in high hostile men.

These results seem to support the motivational model of emotion, which suggests that left hemisphere activation is related to approach behaviors, rather than positive affect. Left pre-frontal activation has been found concurrent with anger in a number of other projects (Harmon-Jones & Allen, 1998; Harmon-Jones & Siegalman, 2001; Harmon-Jones et al., 2004). While, no

frontal asymmetries were noted here, left-right asymmetry was noted in high hostile men at the left anterior temporal lobe. Within the motivational model of emotional processing, relatively few experiments have demonstrated left temporal lobe activation in hostility. Aftanas, Reva, Savotina, and Makhnev (2006) reported increases in activation within the left anterior temporal cortex during the processing of negative emotion in normal participants. The authors suggest activation in the left anterior temporal cortex in response to anger may be related to verbalization that often accompanies anger. Indeed, this may be an area for future research to investigate. Measures of verbal fluency before and after exposure to pain may provide an additional approach to studying left cerebral involvement in pain processing. Further, examination of the emotional content of the words produced might provide an indication of the emotional state after pain exposure. Additional research examining anterior and posterior asymmetries during pain and emotional processing in hostility may contribute to theoretical models of hostility. Examining regional patterns of brain asymmetry in the anterior and the posterior cortex in anxiety has helped define and describe the construct of anxiety (see Heller et al. 1997). Perhaps application of this approach to hostility will lead to new discoveries about cerebral activation during anger.

The current findings have implications for cardiovascular regulation in hostility as well. Lane and Jennings (1995) purpose a brain-heart laterality hypothesis that examines lateralized control of sympathetic input to the heart. The authors suggest that asymmetric hemispheric arousal is transmitted ipsilaterally to the heart. Individuals who experience increased left hemisphere activation during emotion are at greater risk for sudden cardiac death due to lateralized sympathetic input to the heart (Lane & Jennings, 1995). A lateralized imbalance in sympathetic input to the heart increases the likelihood of ventricular fibrillation and sudden death (Lane & Schwartz, 1987). Since no cardiovascular data was recorded in the current experiment,

no conclusions can be made concerning sympathetic regulation in the current sample. However, hostility has emerged from cardiovascular risk studies as a factor that may predict development and progression of cardiovascular disease. Cardiovascular problems in individuals who are high hostile may be in part mediated by representation of emotion in the left hemisphere.

Results of the current experiment also provide support for other theoretical models of brain functioning. The initial analysis of the QEEG data within both groups provides additional support for a functional cerebral systems model whereby anterior cerebral regions inhibit posterior regions. As previously discussed, it is thought that the frontal lobes exert inhibitory control over the temporal and parietal lobes. During stress or pain, increases in cerebral arousal are thought to result from a lack of frontal regulation. In the current experiment, the initial analysis indicated that the cold pressor produced an increase in posterior brain activation that was concurrent with deactivation of the frontal lobes. This was evidenced by increased beta at the temporal-1 and parietal locations and increased delta at the frontal-2 location as a function of the cold pressor. However, when a more refined ANOVA was computed using only values from the frontal-1 and temporal-1 locations this relationship was not found for the high hostile group. High hostile men did not show a significant reduction in delta magnitude at the frontal-1 location as a function of the cold pressor; however, increased temporal activation was present. The lack of a relationship between deactivation of the frontal lobes and activation of the temporal lobes may be indicative of differential cerebral organization in the high hostile group.

The current experiment presents results that challenge the hypothesized neuropsychological model of hostility. Primary findings of the experiment suggest reduced cerebral laterality for emotional processing with left hemisphere activation to emotion and cold pressor pain in high hostile men. While, these results may seem controversial they may lead to a

better conceptualization of the neuropsychological effects of hostility. The present experiment may help identify underlying cerebral activity that contributes to hostile behavior and physiological responses that are associated with heightened hostility. Future research should continue to investigate how a heightened level of hostility may influence left hemisphere functioning.

References

Adolphs, R., Jansari, A., & Tranel, D. (2001). Hemispheric perception of emotional valence from facial expressions. *Neuropsychology, 15(4),* 516-524.

Aftanas, L.I., Reva, N.V., Savotina, L.N., & Makhnev, V.P. (2006). Neurophysiological correlates of induced discrete emotions in humans: an individually oriented analysis. *Neuroscience and Behavioral Physiology, 36(2),* 119-30.

Al'Absi, M., Hugdahl, K., Lovallo, W.R. (2002). Adrenocortical stress responses and altered working memory performance. *Psychophysiology, 39,* 95-99.

Asbjornsen, A., Hugdahl, K., & Bryden, M.P. (1992). Manipulations of subjects' level of arousal in dichotic listening. *Brain and Cognition, 19(2),* 183-194.

Ashby, G.F., Isen, A.M., & Turken, A.U. (1999). A neuropsychological model of positive affect and its influence on cognition. *Psychological Review, 106(3),* 529-550.

Avnon, Y., Nitzan, M., Sprecher, E., Rogowski, Z., & Yarnitsky, D. (2004). Autonomic asymmetry in migraine: augmented parasympathetic activation in left unilateral migraineurs. *Brain, 127,* 2099-2108.

Baehr, E.I., Rosenfield, J.P., Baehr, R., & Earnest, C. (1998). Comparison of two EEG asymmetry indices in depressed patients vs. normal controls. *International Journal of Psychophysiology, 31,* 89-92.

Barefoot, J.C., Dodge, K.A., Peterson, B.L., Dahlstrom, W.G., & Williams, R.B. (1989). The Cook-Medley hostility scale: item content and ability to predict survival. *Psychosomatic Medicine, 51(1),* 46-57.

Bates, E., Reilly, J., Wulfeck, B., Dronkers, N., Opie, M., Fenson, J., Kriz, S., Jeffries, R., Miller, L., & Herbst, K. (2001). Differential effects of unilateral lesions on language production in children and adults. *Brain and Language, 79,* 223-265.

Baxter, L.C., Saykin, A.J., Flashman, L.A., Johnson, S.C., Guerin, S.J., Babcock, D.R. & Wishart, H.A. (2003). Sex differences in semantic language processing: a functional MRI study. *Brain and Language, 84,* 264–272.

Bell, M.A., & Fox, N.A (2003). Cognition and affective style: Individual differences in brain electrical activity during spatial and verbal tasks. *Brain and Cognition, 53,* 441-451.

Biaggio, M. K., & Godwin, W. H. (1987). Relation of depression to anger and hostility constructs. *Psychological Reports, 61,* 87–90.

Blair, R.J.R., Morris, J.S., Frith, C.D., Perrett, D.I., & Dolan, R.J. (1999). Dissociable neural responses to facial expressions of sadness and anger. *Brain, 122,* 883-893.

Bolles, R.C., & Fanselow, M.S. (1980). A perceptual-defensive-recuperative model of fear and pain. *Behavioral and Brain Sciences, 3,* 291-323.

Borod, J.C. (1992). Interhemispheric and intrahemishperic control of emotion: a focus on unilateral brain damage. *Journal of Consulting and Clinical Psychology, 60(3),* 339-348.

Borod, J.C., Bloom, R.L., Brickman, A.M., Nakhutina, L., & Curko, E.A. (2002). Emotional processing deficits in individuals with unilateral brain damage. *Applied Neuropsychology, 9 (1),* 23-36

Borod, J.C., Haywood, C.S., & Koff, E. (1997). Neuropsychological aspects of facial asymmetry during emotional expression: A review of the normal adult literature. *Neuropsychology Review, 7,* 41-60.

Borod, J.C., Koff, E., & Buck, R. (1986). The neuropsychology of facial expression in normal and brain-damaged subjects. In P.Blanck, R. Buck, & R. Rosenthal (Eds.), *Nonverbal communication in the clinical context (196-222).* University Park: Pennsylvania State University Press.

Bradley, M.M., Codispoti, M., Sabatinelli, D., & Lang, P. (2001). Emotion and motivation II: sex differences in picture processing. *Emotion, 1(3),* 300-319.

Brandt, J.B., Celentano, D., Stewart, W., Linet, M., & Folstein, M.F. (1990). Personality and emotional disorder in a community sample of migraine headache sufferers. *American Journal of Psychiatry, 147,* 303-308.

Bromm, B. (2001). Brain images of pain. *News in Physiological Sciences, 16,* 244-249.

Bruder, G.E., Rabinowicz, E., Towey, J., Brown, A., Kaufmann, C.A., Amador, X., Malaspina, D., & Gorman, J.M. (1995). Smaller right ear (left hemisphere) advantage for dichotic fused words in patients with schizophrenia. *American Journal of Psychiatry, 152(6),* 932-935.

Bruder, G.E., Schneier, F.R., Stewart, J.W., McGrath, P.J., & Quitkin, F. (2004). Left hemisphere dysfunction during verbal dichotic listening tests in patients who have social phobia with or without comorbid depressive disorder. *American Journal of Psychiatry, 161,* 72-78.

Bruder, G.E., Stewart, J.W., Towey, J.P., Friedman, D., Tenke, C.E., Voglmaier, M.M., Leite, P., Cohen, P., Quitkin, F.M. (1992). Abnormal cerebral laterality in bipolar depression: convergence of behavioral and brain event-related potential findings. *Biological Psychiatry, 32(1),* 33-47.

Bruehl, S., Burns, J.W., Chung, O.Y., Ward, P., & Johnson, B. (2002). Anger and pain sensitivity in chronic low back pain patients and pain-free controls: the role of endogenous opioids. *Pain, 99(1-2),* 223-33.

Bryden, M.P., Ley, R.G., & Sugarman, J.H. (1982). A left-ear advantage for identifying the emotional quality of tonal sequences. *Neuropsychologia, 20(1),* 83-87.

Bryden, M.P. & MacRae, L. (1989). Dichotic Laterality Effects Obtained with Emotional Words. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology, 1(3),* 171-176.

Bulman-Fleming, M.B., & Bryden, M.P. (1994). Simultaneous verbal and affective laterality effects. *Neuropsychologia, 32,* 787-797.

Burns, J.W., Bruehl, S., & Caceres, C. (2004). Anger management style, blood pressure reactivity, and acute pain sensitivity: Evidence for "Trait x Situation" models. *Annals of Behavioral Medicine, 27(3),* 195-204.

Burton, L.A., & Labar, D. (1999). Emotional status after right vs. left temporal lobectomy. *Seizure, 8(2),* 116-119.

Buss, A., & Perry, M. (1992). The aggression questionnaire. *Journal of Personality and Social Psychology, 63(3)*, 452-459.

Carmody, T. P., Crossen, J. R., & Wiens, A. N. (1989). Hostility as a health risk factor: Relationships with neuroticism, Type A behavior, attentional focus, and interpersonal style. *Journal of Clinical Psychology, 45,* 754–762.

Chandramouth, R., Kanchan, B., & Ambadevi, B. (1993). Right-left asymmetry in tonic pain perception and its modification by simultaneous contralateral noxious stimulation. *Neuropsychologia, 31,* 687-694.

Chang, P.F., Arendt-Nielson, L., & Chen, A.C.N. (2002). Differential cerebral responses to aversive auditory arousal versus muscle pain: Specific EEG patterns are associated with human pain processing. *Experimental Brain Research, 147,* 387-393.

Chen, A.C.N., Dworkin, S.F., Haug, J. & Gehrig, J. (1989). Topograpic brain measures of human pain and pain responsivity. *Pain*, 37**,** 129–141.

Chen, A.C.N. (2001). New perspectives in EEG/MEG brain mapping and PET/fMRI neuroimaging of human pain. *International Journal of Psychophysiology, 42,* 147-159.

Cleare, A.J. & Bond, A.J. (1997). Does central serotonergic function correlate with aggression? A study using D-fenfluramine in healthy subjects. *Psychiatry Research, 69(2-3),* 89-95.

Coghill, R.C., Gilron, I., & Iadarola, J. (2001). Hemispheric lateralization of somatosensory processing. *Journal of Neurophysiology, 85(6),* 2602-2612.

Coupland, N.J., Sustrik, R.A., Ting, P., Li, D., Hartfeil, M., Singh, A.J., & Blair, R.J. (2004). Positive and negative affect differentially influence identification of facial emotions. *Depression and Anxiety, 19(1),* 31-34.

Contrada, R.J., & Jussim, L. (1992). What does the Cook-Medley hostility scale measure? In search of an adequate measurement model. *Journal of Applied Social Psychology, 22,* 615-627.

Cook, W., & Medley, D. (1954). Propose hostility and pharisaic-virtue scales for the MMPI. *Journal of Applied Psychology, 38,* 414-418.

Coren, S.P., Porac, C., & Duncan, P. (1979). A behaviorally validated self-report inventory to assess 4 types of lateral preferences. *Journal of Clinical Neuropsychology, 1,* 55-64.

Craig, A.D. (2003). A new view of pain as a homeostatic emotion. *Trends in Neurosciences, 26(6),* 303-307.

Crews, W. D., & Harrison, D. W. (1994). Sex differences and cerebral asymmetry in facial affect perception as a function of depressed mood. *Psychobiology, 22,* 112-116.

Crews, W.D., Jr., & Harrison, D.W. (1995). Neuropsychological test performances of young depressed outpatient women. An examination of executive functions. *Archives of Clinical Neuropsychology, 14(6),* 517-529.

Dahl, M. (2001). Asymmetries in the processing of emotionally valenced words. *Scandinavian Journal of Psychology, 42,* 97-104.

Davidson, R.J. (1993). Cerebral asymmetry and emotion: Conceptual and methodological conundrums. *Cognition and Emotion, 7,* 115-138.

Davidson, R.J. (1995). Cerebral asymmetry, emotion, and affective style. In R.J. Davidson & K. Hughdahl (Ed.), *Brain Asymmetry (361-387).* Cambridge: MIT Press.

Davidson, R.J. (1998). Anterior electrophysiological asymmetries, emotion, and depression: Conceptual and methodological conundrums. *Psychophysiology, 35,* 607-614.

Davidson, R.J. (2000). What does the prefrontal cortex "do" in affect: Perspectives on frontal EEG asymmetry research. *Biological Psychology, 67(1-2),* 219-234.

Davidson, R.J. (2003). Affective neuroscience and psychophysiology: Toward a synthesis. *Psychophysiology, 40,* 655-665.

Davis, M.C., Matthews, K.A., McGrath, M.A. (2000). Hostile attitudes predict elevated vascular resistance during interpersonal stress in men and women. *Psychosomatic Medicine, 62,* 17-25.

Delgado, P.L. (2004). Common pathways of depression and pain. *Journal of Clinical Psychiatry, 65(12),* 16-19.

Demaree, H.A., Everhart, D.E., Youngstrom, E.A., & Harrison, D.W. (2005). Brain lateralization of emotional processing: Historical roots and a future incorporating "dominance." *Behavioral and Cognitive Neuroscience Reviews, 4(1),* 3-20.

Demaree, H. A., & Harrison, D. W. (1996). Case Study: Topographical brain mapping in hostility following mild closed-head injury. *The International Journal of Neuroscience, 87,* 97-101.

Demaree, H., & Harrison, D. (1997). Physiological and neuropsychological correlates of hostility. *Neuropsychologia, 35,* 1405-1411.

Demaree, H. A., Higgins, D. A., Williamson, J. B., & Harrison, D.W. (2002). Asymmetry in hand grip strength and fatigue in low- and high-hostile men. *International Journal of Neuroscience, 112(4),* 415-428.

Diego, M.A., Field, T., Sanders, C., & Hernandez-Reif, M. (2004). Massage therapy of moderate and light pressure and vibrator effects on EEG and heart rate. *International Journal of Neuroscience, 114(31),* 31-45.

Dickinson, A., & Pearce, J.M. (1977). Inhibitory interaction between appetive and aversive stimuli. *Psychological Bulletin, 84,* 690-711.

Dimberg, U., & Petterson, M. (2000). Facial reactions to happy and angry facial expressions: Evidence for right hemisphere dominance. *Psychophysiology, 37,* 693-696.

DiPiero, V., Ferracuti, S., Sabatini, U., Pantano, P., Cruccu, G., & Lenzi, G.L. (1994). A cerebral blood flow study on tonic pain activation in man. *Pain, 56(2),* 167-173.

Erhan, H., Borod, J.C., Tenke, C.E., & Bruder, G.E. (1998). Identification of emotion in a dichotic listening task: Event-related brain potential and behavioral findings. *Brain and Cognition, 37,* 286-307.

Everhart, D.E., & Harrison, D.W. (1995). Hostility following right CVA: Support for right orbital frontal deactivation and right temporal activation. *Journal of Neurotherapy, 1(2),* 55-59.

Fasmer, O.B., & Oedegaard, K.J. (2002). Laterality of pain in migraine with comorbid unipolar depressive and bipolar II disorders. *Bipolar Disorders, 4,* 290-295.

Fava, M., Rosenbaum, J. F., Pava, J. A., McCarthy, M. K., Steingard, R. J., & Boufiddes, E. (1993). Anger attacks in unipolar depression: I. Clinical correlates and response to fluoxetine treatment. *American Journal of Psychiatry, 150,* 1158–1163.

Felsten, G. (1995). Cynical hostility influences anger, but not cardiovascular reactivity during competition with harassment. *International Journal of Psychophysiology, 19(3),* 223-31.

Felsten, G. (1996). Hostility, stress, and symptoms of depression. *Personality & Individual Differences, 21,* 461–467.

Ferracuti, S., Seri, S., Mattia, D., & Cruccu, G. (1994). Quantitative EEG modifications during the cold water pressor test: Hemispheric and hand differences. *International Journal of Psychophysiology, 17(3),* 261-268.

Flory, J.D., Manuck, S.B., Matthews, K.A., & Muldoon, M.F. (2004). Seretonergic function in the central nervous system is associated with daily ratings of positive mood. *Psychiatry Research, 129,* 11-19.

Fillingim, R.B., Browning, A.D., Powell, T., & Wright, R.A. (2002). Sex differences in perceptual and cardiovascular responses to pain: The influence of a perceived ability manipulation. *Journal of Pain, 3(6),* 439-445.

Foster, P.S., & Harrison, D.W. (2002). The relationship between magnitude of cerebral activation and intensity of emotional arousal. *International Journal of Neuroscience, 112,* 1463-1477.

Foster, P.S., & Harrison, D.W. (2004). The covariation of cortical electrical activity and cardiovascular responding. *International Journal of Psychophysiology, 52,* 239-255.

Fox, N.A. (1994). Dynamic cerebral processes underlying emotion regulation. *Monographs of the Society for Research in Child Development, 59(2-3),* 152-166.

Fulbright, R.K., Troche, C., Skudlarski, P., Gore, J, & Wexler, B.E. (2001). Functional MR imaging of regional brain activation associated with the affective experience of pain. *American Journal of Roentgenology, 177,* 1205-1210.

Gadea, M., Gomez, C., Gonzalez-Bono, E., Espert, R., & Salvador, A. (1995). Increased cortisol and decreased right ear advantage (REA) in dichotic listening following a negative mood induction. *Psychoneuroendocrinology, 30(2),* 129-138.

Gazzaniga, M.S., Ivry, R., & Mangun, G.R. (2002). *Cognitive Neuroscience: The Biology of the Mind.* New York: W.W. Norton & Company, Inc.

Grachev, I.D., Fredrickson, B.E., & Apkarian, A.V. (2002). Brain chemistry reflects dual states of pain and anxiety in chronic low back pain. *Journal of Neural Transmission, 109(10),* 1309-1334.

Graves, K.D., & Miller, P.M. (2003). Behavioral Medicine in the prevention and treatment of cardiovascular disease. *Behavior Modification, 27(1),* 3-25.

Gray, J.A. (1990). Brain systems that mediate both emotion and cognition. *Cognition and Emotion, 4,* 269-288.

Gray, J.A. (2001). Emotional modulation of cognitive control: Approach-withdrawal states double-dissociate spatial from verbal two-back task performance. *Journal of Experimental Psychology, 130(3),* 436-452.

Harmon-Jones, (2004a). Contributions from research on anger and cognitive dissonance to understanding the motivational functions of asymmetrical frontal brain activity. *Biological Psychology, 67(1-2),* 51-76.

Harmon-Jones, E. (2004b). On the relationship of anterior brain activity and anger: examining the role of attitude toward anger. *Cognition and Emotion, 18,* 337–361.

Harmon-Jones, E., & Allen, J.J. (1997). Behavioral activation sensitivity and resting frontal EEG asymmetry: Covariation of putative indicators related to risk for mood disorders. *Journal of Abnormal Psychology, 106,* 159-163.

Harmon-Jones, E., & Allen, J.J. (1998). Anger and frontal brain activity: EEG asymmetry consistent with approach motivation despite negative affective valence. *Journal of Personality and Social Psychology, 74,* 1310–1316.

Harmon-Jones, E., & Sigelman, J. (2001). State anger and frontal brain activity: evidence that insult-related relative left prefrontal activation is associated with experienced anger and aggression. *Journal of Personality and Social Psychology, 80,* 797–803.

Harmon-Jones, E., Vaughn-Scott, K., Mohr, S., Sigelman, J., & Harmon-Jones, C. (2004).The effect of manipulated sympathy and anger on left and right frontal cortical activity. *Emotion, 4(1),* 95-101.

Harrison, D.W., & Gorelczenko, P.M. (1990). Functional asymmetry for facial affect perception in high and low hostile men and women. *International Journal of Neuroscience, 55(2-4),* 89-97.

Harrison, D.W., Gorelczenkco, P.M., & Cook, J. (1990). Sex differences in the functional asymmetry for facial affect perception. *International Journal of Neuroscience, 52(1-2),* 11-6.

Heilman, K.M. (1997). The neurobiology of emotional experience. *Journal of Neuropsychiatry and Clinical Neuroscience, 9(3),* 439-48.

Heilman, K.M., & Gilmore, R.L. (1998). Cortical influences in emotion. *Journal of Clinical Neurophysiology, 15(5),* 409-423.

Heilman, K.M, Leon, S.A., & Rosenbek, J.C. (2004). Affective aprosodia from a medial frontal stroke. *Brain and Language, 89(3),* 411-416.

Heilman, K.M, Scholes, R., & Watson, R.T. (1975). Auditory affective agnosia. *Journal of Neurology, Neurosurgery, and Psychiatry, 38,* 1018-1020.

Heller, W. (1990). The neuropsychology of emotion: Developmental patterns and implications for psychopathology. In N. Stein, B. L. Leventhal. & T. Trabasso (Ed.), *Psychological and biological approaches to emotion (167–211).* Hillsdale: Erlbaum.

Heller, W. (1993). Neuropsychological mechanisms of individual differences in emotion, personality, and arousal. *Neuropsychology, 7,* 476-489.

Heller, W., Nitschke, J.B., Etienne, M.A., & Miller, G.A.(1997). Patterns of regional brain activity differentiate types of anxiety. *Journal of Abnormal Psychology, 106(3),* 376–385.

Henriques, J., & Davidson, R..J. (1997). Brain electrical asymmetries during cognitive task performance in depressed and nondepressed subjects. *Biological Psychiatry, 42,* 1039-1050.

Herridge, M.L., Harrison, D.W., & Demaree, H. (1997). Hostility, facial configuration, and bilateral asymmetry on galvanic skin response. *Psychobiology, 25(1),* 71-76.

Herridge, M.L., Harrison, D.W., Mollet, G.A., & Shenal, B.V. (2004). Hostility and facial affect recognition: effects of a cold pressor stressor on accuracy and cardiovascular reactivity. *Brain and Cognition, 55(3),* 564-571.

Hiscock, M., Inch, R., Jacek, C., Hiscock-Kalil, C. & Kalil, K.M. (1994). Is there a sex difference in human laterality? I. An exhaustive survey of auditory laterality studies from six neuropsychological journals. J*ournal of Clinical and Experimental Neuropsychology. 16,* 423–435.

Hiscock, M., Israelian, M., Inch, R., Jacek, C. & Hiscock-Kalil, C. (1995). Is there a sex difference in human laterality? II. An exhaustive survey of visual laterality studies from six neuropsychological journals. *Journal of Clinical and Experimental Neuropsychology, 17,* 590–610.

Hiscock, M., Perachio, N., & Inch, R. (2001). Is there a sex difference in human laterality? IV. An exhaustive survey of dual-task interference studies from six neuropsychology journals. *Journal of Clinical and Experimental Neuropsychology, 23(2),* 137-148.

Hsieh, J.C., Belfrage, M., Stone-Elander, S., Hansson, P., & Ingvar, M. (1995). Central representation of chronic ongoing neuropathic pain studied by positron emission tomography. *Pain, 65,* 225-236.

Hsieh, J.C., Hannerz, J., & Ingvar, M. (1996). Right-lateralised central processing for pain of nitroglycerin-induced cluster headache. *Pain, 67(1),* 59-68.

Hugdahl, K. (1995). Dichotic listening: probing temporal lobe functional integrity. In Davidson, R.J. and Hugdahl, K. ed. *Brain Asymmetry.* Cambridge, MA: MIT Press, 123-156.

Jancke, L., Buchanan, T.W., Lutz, K., & Shah, N.J. (2001). Focused and nonfocused attention in verbal and emotional dichotic listening: An FMRI study. *Brain and Language, 78,* 349-363.

Jancke, L., & Shah, N.J. (2002). Does dichotic listening probe temporal lobe functions? *Neurology, 58,* 736-743.

Jancke, L., Specht, K., Shah, J.N., & Hugdahl, K. (2003). Focused attention in a simple dichotic listening task: an fMRI experiment. *Cognitive Brain Research, 16,* 257-266.

Janssen, S.A. (2002). Negative affect and sensitization to pain. *Scandinavian Journal of Psychology, 43,* 131-137.

Janssen, S.A., Spinhoven, P., & Arntz, A. (2004). The effects of failing to control pain: an experimental investigation. *Pain, 107(3),* 227-33.

Janssen, S.A., Spinhoven, P., & Brosschot, J.F. (2001). Experimentally induced anger, cardiovascular reactivity, and pain sensitivity. *Journal of Psychosomatic Research, 51,* 479-485.

Kimura, D. (1961a). Cerebral dominance and the perception of verbal stimuli. *Canadian Journal of Psychology, 15,* 166-171.

Kimura, D. (1961b). Cerebral dominance and the perception of verbal stimuli. *Canadian Journal of Psychology, 15,* 156-165.

Keefe, F.J., Castell, P.J., & Blumenthal, J.A. (1986) Angina pectoris in type A and type B cardiac patients. *Pain, 27(2),* 211-8.

Klaasen, T., Riedel, W.J., Deutz, N.E.P., & Van Praag, H.M. (2002). Mood congruent memory bias induced by tryptophan depletion. *Psychological Medicine, 32,* 267-272.

Lane, R.D., & Jennings, J.R. (1995). Hemispheric asymmetry, autonomic asymmetry, and the problem of sudden cardiac death. In R.J. Davidson & K. Hugdahl (Eds.), *Brain Asymmetry* (271-304). Cambridge, MA: MIT Press.

Lane, R.D., & Schwartz, G.E. (1987). Induction of lateralized sympathetic input to the heart by the CNS during emotional arousal: A possible neurophysiologic trigger of sudden cardiac death. *Psychosomatic Medicine, 49(3),* 275-284.

Larkin, K.T., Martin, R.R., & McClain, S.E. (2002). Cynical hostility and the accuracy of decoding facial expressions of emotions. *Journal of Behavioral Medicine, 25(3),* 285-292.

Lee, G.P., Meador, K.J., Loring, D.W., & Bradley, K.P. (2002). Lateralized changes in autonomic arousal during emotional processing in patients with unilateral temporal lobe seizure onset. *International Journal of Neuroscience, 112(6),* 743-57.

Levy, J. (1969). Possible basis for the evolution of lateral specialization of the human brain. *Nature, 224,* 614–5.

Levy, J., & Reid, M. (1978). Variations in cerebral organization as a function of handedness, hand posture in writing, and sex. *Journal of Experimental Psychology: General, 107,* 119–144.

Ley, R., & Bryden, M. (1979). Hemispheric differences in processing emotions and faces. *Brain and Language, 7,* 127-138.

Lorenz, J., Cross, D., Minoshima, S., Morrow, T., Paulson, P., & Casey K. (2002). A unique representation of heat allodynia in the human brain. *Neuron, 35,* 383-93.

Lorenz, J., Minoshima, S., & Casey, K.L. (2003). Keeping pain out of the mind: The role of the dorsolateral prefrontal cortex. *Brain, 126,* 1079-1091.

Lugo, M., Isturiz, G., Lara, C., Garcia, N., & Eblen-Zaijur, A. (2002). Sensory lateralization in pain subjective perception for noxious heat stimulus. *Somatosensory and Motor Research, 19(3),* 207-212.

Matthews, K.A., Gump, B.B., Harris, K.F., Haney, T.L., & Barefoot, J.C. (2004). Hostile behaviors predict cardiovascular mortality among men enrolled in the Multiple Risk Factor Intervention Trial. *Circulation, 109(1),* 66-70.

McGlone, J. (1980). Sex differences in human brain asymmetry: a critical survey. *Behavioral Brain Science, 3,* 215–263.

Meagher, M.W., Arnau, R.C., & Rhudy, J.L. (2001). Pain and emotion: Effects of affective picture modulation. *Psychosomatic Medicine, 63,* 79-90.

Meyer, S. Strittmatter, M., Fischer, C., Georg, T., & Schmitz, B. (2004). Lateralization in autonomic dysfunction in ischemic stroke involving the insular cortex. *Neuroreport, 15(2),* 357-361.

Min, S., & Lee, B. (1997). Laterality in somatization. *Psychosomatic Medicine, 59(3),* 236-240.

Mollet, G.A., & Harrison, D.W. (submitted for publication). Affective verbal learning in hostility: An increased primacy effect and bias for negative emotional material. Manuscript submitted for publication.

Mollet, G.A. & Harrison, D.W. (in preparation). Sex differences in endorsement of hostility and aggression. Manuscript in preparation for publication.

Morrow, L., Vrtunski, P.B., Kim, Y., & Boller, F. (1981). Arousal responses to emotional stimuli and laterality of lesion. *Neuropsychologia, 19(1),* 65-71.

Pauli, P., Wiedemann, G., & Nickola, M. (1999). Pain sensitivity, cerebral laterality, and negative affect. *Pain, 80,* 359-364.

Petruzzello, S.J., Hall, E.E., & Ekkekakis, P. (2001).Regional brain activation as a biological marker of affective responsivity to acute exercise: Influence of fitness. *Psychophysiology, 38,* 99-106.

Peyron, R., Laurent, B., & Garcia-Larrea, L. (2000). Functional imaging of the brain responses to pain. A review and meta-analysis. *Clinical Neurophysiology, 30(5),* 263-288.

Ragland, J.D., Goldberg, T.E., Wexler, B.E., Gold, J.M., Torrey, E.F., & Weinberger, D.R. (1992). Dichotic listening in monozygotic twins discordant and concordant for schizophrenia. *Schizophrenia Research, 7(2),* 177-83.

Reuter-Lorenz, P.A., Givis, R.P., & Moscovitch, M. (1983). Hemispheric specialization and the perception of emotion: evidence from right-handers and from inverted and non-inverted left-handers. *Neuropsychologia, 21(6),* 687-692.

Reinert, A., Treede R. & Bromm, B. (2000). The pain inhibiting pain effect: An electrophysiological study in humans. *Brain Research, 862,* 103–110.

Ross, E.D., Harney, J.H., deLacoste-Utamsing, C., & Purdy, P.D. (1981). How the brain integrates affective and propostitional language into a unified behavioral function. Hypothesis based on clinicoanatomic evidence. *Archives of Neurology, 38,* 745-748.

Rusting, C.L. (1998). Personality, mood, and cognitive processing of emotional information: three conceptual frameworks. *Psychological Bulletin, 124(2),* 165-196.

Schiff, B.B., & Gagliese, L. (1994). The consequences of experimentally induced and chronic unilateral pain: reflections of hemispheric lateralization of emotion. *Cortex, 30(2),* 255-267.

Schlereth, T., Baumgartner, U., Magerl, W., Stoeter, P., Treede, R.D. (2003). Left-hemisphere dominance in early nociceptive processing in the human parasylvian cortex. *Neuroimage, 20(1),* 441-54.

Schutter, D., Putman, P., Hermans, E., & van Honk, J. (2001). Parietal electroencephalogram beta asymmetry and selective attention to angry facial expressions in healthy human subjects. *Neuroscience Letters, 314(1-2),* 13-16.

Sendowski, I., Savourey, G., Launay, J.C., Besnard, Y., Cottet-Emard, J.M., Pequignot, J.M. & Bittel, J. Sympathetic stimulation induced by hand cooling alters cold-induced vasodilatation in humans. *European Journal of Applied Physiology, 81(4),* 303-309.

Shapiro, P.A., Sloan, R.P., Bagiella, E., Kuhl, J.P., Anjilvel, S., & Mann, J.J. (2000). Cerebral activation, hostility, and cardiovascular control during mental stress. *Journal of Psychosomatic Research, 48,* 485-491.

Shenal, B.V., Harrison, D.W., & Demaree, H.A. (2003). The neuropsychology of depression: A literature review and preliminary model. *Neuropsychology Review, 13,* 33-42.

Smith, T.W., & Frohm, K.D. (1985). What's so unhealthy about hostility? Construct validity and psychosocial correlates of the Cook and Medley Ho scale. *Health Psychology, 4(6),* 503-520.

Snyder, K.A., Harrison, D.W., & Gorman, W.J. (1996). Auditory affect perception in a dichotic listening paradigm as a function of verbal fluency classification. *International Journal of Neuroscience, 84(1-4),* 65-74.

Snyder, K. A., Harrison, D. W., & Shenal, B. V. (1998). The affective auditory verbal learning test: Peripheral arousal correlated. *Archives of Clinical Neuropsychology, 13,* 251-258.

Spielberger, C.D., Johnson, E.H., Russell, S.F., Crane, R., Jacobs, G.A., & Worden, T.J. (1985). The experience and expression of anger: Construction and validation of and anger expression scale. In M.A. Chesney and R.H. Rosenman (eds.), *Anger and Hostility in Cardiovascular and Behavioral Disorders (5-30).* Hemisphere, Washington, DC.

Spicer, J., & Chamberlain, K. (1996). Cynical hostility, anger, and resting blood pressure. *Journal of Psychosomatic Research, 40(4),* 359-368.

Stewart, G., Steffler, D.J., Lemoine, D.E., & Leps, J.D. (2001). Do quantitative EEG measures differentiate hyperactivity in attention deficit/hyperactivity disorder? *Child Study Journal, 31(2),* 103-119.

Sutton, S.K., & Davidson, R.J. (1997). Prefrontal brain asymmetry: A biological substrate of the behavioral approach and inhibition systems. *Psychological Science, 8,* 204-210.

Suarez, E.C., Kuhn, C.M., Schanberg, S.M., Williams, R.B., Jr., Zimmermann, E.A. (1998). Neuroendocrine, cardiovascular, and emotional responses of hostile men: The role of the interpersonal challenge. *Psychosomatic Medicine, 60(1),* 78-88.

Tamura, Y., Okabeb, S., Ohnishic, T., Saitod, D.N., Araib, N., Mochioa, S., Inouea, K., & Ugawa, Y. (2004). Effects of 1-Hz repetitive transcranial magnetic stimulation on acute pain induced by capsaicin. *Pain, 107(1-2),* 107-115.

Tomarken, A. J., Davidson, R. J., & Henriques, J. B. (1990). Resting frontal brain asymmetry predicts affective responses to films. *Journal of Personality and Social Psychology, 59,* 791-801.

Tucker, D.M. (1993). Emotional experience and the problem of vertical integration: Discussion of the special section on emotion. *Neuropsychology, 7,* 500-509.

Tucker, D. M., & Williamson, P. A. (1984). Asymmetric neural control systems in human self-regulation. *Psychological Review, 91(2),* 185-215.

Urban, P.P, Solinski, M., Best., C., Rolke, R., Hopf, H.C., & Dieterich, M. (2004). Different short-term modulation of cortical motor output to distal and proximal upper-limb muscles during painful sensory nerve stimulation. *Muscle and Nerve, 29,* 663-669.

Valeriani, M., Tinazzi, M., Le Pera, D., Restuccia, D., De Armas, L., Maiese, T., Tonali, P., Arendt-Nielsen, L. (2004). Inhibitory effect of capsaicin evoked trigeminal pain on warmth sensation and warmth evoked potentials. *Experimental Brain Research, 160,* 29-37.

Voyer, D., Russell, A., & McKenna, J. (2002). On the reliability of laterality effects in a dichotic emotion recognition task. *Journal of Clinical and Experimental Neuropsychology, 24(5),* 605-614.

Vrana, S.R., Spence, E.L., & Lang, P.J. (1988). The startle probe response: A new measure of emotion? *Journal of Abnormal Psychology, 97,* 487-491.

Waldstein, S.R., Kop, W.J., Schmidt, L.A., Haufler, A.J., Krantz, D.S., & Fox, N.A. (2000). Frontal electrocortical and cardiovascular reactivity during happiness and anger. *Biological Psychology, 55,* 2-23.

Wale, J., & Carr, V. (1990). Differences in dichotic listening asymmetries in depression according to symptomatology. *Journal of Affective Disorders, 18(1),* 1-9.

Wall, P.D. (1979). On the relation of injury to pain. *Pain, 6,* 253-264.

Wexler, B.E., Giller, E.L., Southwick, S. (1991). Cerebral laterality, symptoms, and diagnosis in psychotic patients. *Biological Psychiatry, 29(2),* 103-116.

Wexler, B.E., Schwartz, G., Warrenburg, S., Servis, M., & Tarlatzis, I. (1986). Effects of emotion on perceptual asymmetry interactions with personality. *Neuropsychologia, 24(5),* 699-710.

Williamson, J.B., & Harrison, D.W. (2003). Functional Cerebral Asymmetry in hostility: A dual task approach with fluency and cardiovascular regulation. *Brain and Cognition, 52(2),* 167-174.

Winer, B.J. (1971). Statistical principles in experimental design. New York: McGraw Hill.

Wittling, W. (1995). Brain asymmetry in the control of autonomic-physiologic activity. In R. J. Davidson & K. Hugdahl (Ed), *Brain asymmetry (305-358).* Cambridge: MIT Press.

Wittling, W., Block, A., Schweiger, E., & Genzel, S. (1998). Hemisphere asymmetry in sympathetic control of the human myocardium. *Brain and Cognition, 38,* 17-35.

Wunsch, A., Philippot, P., & Plaghki, L. (2003). Affective associative learning modifies the sensory perception of nociceptive stimuli without participant's awareness. *Pain, 102,* 27-38.

Yoon, B. W., Morillo, C. A., Cechetto, D. F., & Hachinski, V. (1997). Cerebral hemispheric lateralization in cardiac autonomic control. *Archives of Neurology, 54,* 741-744.

Zoccolotti, P., Scabini, D., & Violani, C. (1982). Electrodermal responses in patients with unilateral brain damage. *Journal of Clinical Neuropsychology, 4(2),* 143-150.

Footnotes

1. Although the current experiment conceptualizes the cold pressor as a painful stimulus, it should be noted that the cold pressor is also a thermodynamic challenge that produces additional autonomic activity. Exposure to the cold pressor produces general sympathetic activation and a change in skin temperature that is accompanied by vasoconstriction (Sendowski et al., 2000).

Table 1

ANOVA source table for Number Correct at the Right Ear

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 24	120.31	120.31	2.42	0.13
Pain	1, 24	19.39	19.39	4.23	0.05
Affect	2, 48	0.47	0.23	0.01	0.99
Group x Pain	1, 24	27.08	27.08	5.91	0.02
Group x Affect	2, 48	27.39	13.70	0.39	0.68
Pain x Affect	2, 48	31.40	15.70	4.32	0.02
Group x Pain x Affect	2, 48	2.01	1.00	0.28	0.75

Table 2

ANOVA source table for Number Correct at the Left Ear

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 24	120.31	120.31	2.32	0.14
Pain	1, 24	0.006	0.006	0.00	0.97
Affect	2, 48	76.27	38.13	1.07	0.35
Group x Pain	1, 24	27.08	27.08	4.92	0.04
Group x Affect	2, 48	0.47	0.24	0.01	0.99
Pain x Affect	2, 48	15.17	7.58	1.51	0.23
Group x Pain x Affect	2, 48	2.86	1.43	0.28	0.74

Table 3

ANOVA source table for the Laterality Index

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 24	1.46	1.46	3.53	0.07
Pain	1, 24	0.08	0.08	1.15	0.29
Affect	2, 48	0.15	0.08	1.81	0.18
Group x Pain	1, 24	0.35	0.35	5.03	0.03
Group x Affect	2, 48	0.22	0.11	1.81	0.09
Pain x Affect	2, 48	0.14	0.07	1.83	0.17
Group x Pain x Affect	2, 48	0.09	0.05	1.19	0.31

Table 4

ANOVA source table for Number Correct at the Right Ear including the Block variable

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 24	60.16	60.16	2.42	0.13
Pain	1, 24	9.70	9.70	4.23	0.05
Affect	2, 48	0.24	0.12	0.01	0.99
Block	1, 24	0.72	0.72	0.38	0.54
Group x Pain	1, 24	13.54	13.54	5.91	0.02
Group x Affect	2, 48	13.70	6.85	0.39	0.68
Group x Block	1, 24	1.70	1.70	0.90	0.35
Pain x Affect	2, 48	15.70	7.85	4.32	0.02
Pain x Block	1, 24	0.26	0.26	0.13	0.72
Block x Affect	2, 48	35.90	17.95	6.41	0.003
Group x Pain x Affect	2, 48	1.01	0.50	0.28	0.76
Group x Block x Affect	2, 48	7.24	3.62	1.29	0.28
Pain x Block x Affect	2, 48	4.67	2.33	1.61	0.21
Group x Pain x Block x Affect	2, 48	2.16	1.08	0.75	0.48

Table 5

ANOVA source table for Number Correct at the Left Ear including the Block Variable

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 24	60.16	60.16	2.32	0.14
Pain	1, 24	0.003	0.003	0.00	0.97
Affect	2, 48	38.13	19.07	1.07	0.35
Block	1, 24	4.88	4.88	2.13	0.16
Group x Pain	1, 24	13.54	13.54	4.92	0.04
Group x Affect	2, 48	0.24	0.12	0.01	0.99
Group x Block	1, 24	2.00	2.00	0.87	0.36
Pain x Affect	2, 48	7.58	3.79	1.51	0.23
Pain x Block	1, 24	0.003	0.003	0.00	0.97
Block x Affect	2, 48	37.10	18.55	5.91	0.005
Group x Pain x Affect	2, 48	1.43	0.71	0.28	0.75
Group x Block x Affect	2, 48	0.66	0.33	0.11	0.90
Pain x Block x Affect	2, 48	10.85	5.43	3.62	0.03
Group x Pain x Block x Affect	2, 48	2.16	1.08	0.72	0.49

Table 6

ANOVA source table for the Laterality Index including the Block Variable

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 24	2.76	2.76	3.25	0.08
Pain	1, 24	0.12	0.12	1.25	0.27
Affect	2, 48	0.41	0.21	2.29	0.11
Block	1, 24	0.08	0.08	1.01	0.32
Group x Pain	1, 24	0.69	0.69	6.92	0.01
Group x Affect	2, 48	0.45	0.22	2.50	0.09
Group x Block	1, 24	0.008	0.008	0.11	0.74
Pain x Affect	2, 48	0.15	0.08	1.93	0.16
Pain x Block	1, 24	0.03	0.03	0.35	0.56
Block x Affect	2, 48	0.03	0.01	0.35	0.70
Group x Pain x Affect	2, 48	0.05	0.02	0.58	0.56
Group x Block x Affect	2, 48	0.06	0.03	0.76	0.47
Pain x Block x Affect	2, 48	0.02	0.009	0.35	0.71
Group x Pain x Block x Affect	2, 48	0.07	0.04	1.32	0.28

Table 7

ANOVA source table for the Error Score

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 24	0.006	0.006	0.00	0.98
Pain	1, 24	19.39	19.39	11.60	0.002
Affect	2, 48	257.01	128.51	11.16	0.0001
Group x Pain	1, 24	0.006	0.006	0.00	0.95
Group x Affect	2, 48	28.71	14.35	1.25	0.30
Pain x Affect	2, 48	16.24	8.12	5.03	0.01
Group x Pain x Affect	2, 48	0.24	0.12	0.08	0.93

Table 8

Means and standard deviations for the main effect of Location across task conditions and bandwidths

	Dichotic Listening 1		*Cold Pressor*		*Dichotic Listening 2*	
Delta	*Mean*	*SD*	*Mean*	*SD*	*Mean*	*SD*
Frontal-1	5.49, a	1.20	5.55, a	1.12	5.50, a	1.12
Frontal-2	4.77, b	1.41	5.01, b	1.51	4.93, b	1.41
Temporal-1	3.55, c	0.88	3.55, d	0.92	3.52, d	0.83
Temporal-2	4.43, b	1.35	4.24, c	1.18	4.37, c	1.38
Parietal	5.43, a	1.38	5.22, a, b	1.13	5.31, a, b	1.29
Low Beta						
Frontal-1	8.01, b, c	2.04	7.98, c	2.03	8.06, b, c	2.00
Frontal-2	6.33, c	1.56	6.38, d	1.73	6.34, d	1.58
Temporal-1	7.56, b, c	3.00	7.60, c	3.36	7.44, c	3.13
Temporal-2	8.97, b	2.72	9.13, b	2.79	8.98, b	2.84
Parietal	10.50, a	3.21	10.55, a	3.42	10.40, a	3.21
High Beta						
Frontal-1	6.12, c	1.62	6.23, b, c	1.72	6.12, b, c	1.48
Frontal-2	5.45, c, b	1.38	5.56, c	1.74	5.47, c	1.52
Temporal-1	7.27, a	3.77	7.40, a, b	4.18	7.05, a, b	4.13
Temporal-2	6.79, a, b	2.34	7.05, a	2.17	6.72, a, b	2.15
Parietal	7.17, a, b	2.30	7.42, a	2.58	7.18, a	2.38

**Note:* Means with the same letter are not significantly different.

Table 9

ANOVA source table for the delta bandwidth

Condition: Dichotic Listening 1

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 24	122.80	122.80	7.49	0.01
Condition	1, 24	3.06	3.06	2.34	0.14
Hemisphere	1, 24	0.10	0.10	0.05	0.83
Location	4, 96	264.55	66.14	42.90	0.0001
Group x Condition	1, 24	2.18	2.18	1.67	0.21
Group x Hemisphere	1, 24	0.22	0.22	0.11	0.75
Group x Location	4, 96	7.15	1.79	1.16	0.33
Condition x Hemisphere	1, 24	0.33	0.33	1.45	0.24
Condition x Location	1, 24	1.22	0.30	2.24	0.07
Hemisphere x Location	4, 96	3.03	0.76	2.36	0.06
Group x Condition x Hemisphere	1, 24	0.70	0.70	3.08	0.09
Group x Condition x Location	4, 96	0.64	0.16	1.18	0.33
Group x Hemisphere x Location	4, 96	1.74	0.43	1.36	0.25
Condition x Hemisphere x Location	4, 96	0.14	0.04	0.65	0.63
Group x Condition x Hemisphere x Location	4, 96	0.25	0.06	1.17	0.33

Table 10

ANOVA source table for the low beta bandwidth

Condition: Dichotic Listening 1

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 24	149.69	149.69	2.09	0.16
Condition	1, 24	3.52	3.52	1.29	0.27
Hemisphere	1, 24	0.31	0.31	0.03	0.87
Location	4, 96	1017.95	254.49	33.62	0.0001
Group x Condition	1, 24	5.00	5.00	1.83	0.19
Group x Hemisphere	1, 24	2.28	2.28	0.22	0.65
Group x Location	4, 96	73.72	18.43	2.43	0.05
Condition x Hemisphere	1, 24	7.06	7.06	11.00	0.003
Condition x Location	4, 96	29.94	7.49	8.13	0.0001
Hemisphere x Location	4, 96	12.40	3.10	1.39	0.24
Group x Condition x Hemisphere	1, 24	0.86	0.86	1.35	0.26
Group x Condition x Location	4, 96	18.66	4.67	5.07	0.0009
Group x Hemisphere x Location	4, 96	3.06	0.77	0.34	0.85
Condition x Hemisphere x Location	4, 96	4.12	1.03	2.13	0.08
Group x Condition x Hemisphere x Location	4, 96	1.34	0.34	0.69	0.60

Table 11

ANOVA source table for the high beta bandwidth

Condition: Dichotic Listening 1

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 24	9.32	9.32	0.20	0.66
Condition	1, 24	4.36	4.36	2.39	0.13
Hemisphere	1, 24	9.10	9.10	0.85	0.37
Location	4, 96	245.10	61.27	7.46	0.0001
Group x Condition	1, 24	12.43	12.43	2.39	0.13
Group x Hemisphere	1, 24	0.96	0.96	0.09	0.77
Group x Location	4, 96	17.04	4.26	0.52	0.72
Condition x Hemisphere	1, 24	7.78	7.78	5.12	0.03
Condition x Location	1, 24	47.90	11.97	9.45	0.0001
Hemisphere x Location	4, 96	20.41	5.10	1.48	0.21
Group x Condition x Hemisphere	1, 24	0.90	0.90	0.59	0.45
Group x Condition x Location	4, 96	26.14	6.54	5.16	0.0008
Group x Hemisphere x Location	4, 96	14.09	3.52	1.02	0.40
Condition x Hemisphere x Location	4, 96	11.82	2.96	3.13	0.02
Group x Condition x Hemisphere x Location	4, 96	8.91	2.23	2.36	0.06

Table 12

ANOVA source table for the delta bandwidth

Condition: Cold Pressor

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 24	85.05	85.05	6.03	0.02
Pain	1, 24	2.37	2.37	2.44	0.13
Hemisphere	1, 24	0.44	0.44	0.17	0.69
Location	4, 96	272.33	68.08	54.68	0.0001
Group x Pain	1, 24	0.26	0.26	0.27	0.61
Group x Hemisphere	1, 24	0.13	0.13	0.05	0.82
Group x Location	4, 96	4.13	1.03	0.83	0.51
Pain x Hemisphere	1, 24	0.03	0.30	0.12	0.73
Pain x Location	1, 24	5.12	1.28	5.25	0.0007
Hemisphere x Location	4, 96	0.24	0.06	0.17	0.95
Group x Pain x Hemisphere	1, 24	0.0009	0.0009	0.00	0.95
Group x Pain x Location	4, 96	2.34	0.59	2.40	0.06
Group x Hemisphere x Location	4, 96	1.63	0.41	1.16	0.33
Pain x Hemisphere x Location	4, 96	0.18	0.04	0.25	0.91
Group x Pain x Hemisphere x Location	4, 96	0.66	0.16	0.92	0.46

Table 13

ANOVA source table for the low beta bandwidth

Condition: Cold Pressor

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 24	129.10	129.19	1.52	0.23
Pain	1, 24	30.19	30.19	8.61	0.007
Hemisphere	1, 24	6.23	6.23	0.64	0.43
Location	4, 96	1043.07	260.77	35.41	0.0001
Group x Pain	1, 24	20.44	20.44	5.83	0.024
Group x Hemisphere	1, 24	0.27	0.27	0.64	0.43
Group x Location	4, 96	74.92	18.73	2.54	0.04
Pain x Hemisphere	1, 24	1.91	1.91	0.92	0.35
Pain x Location	1, 24	52.68	13.17	11.16	0.0001
Hemisphere x Location	4, 96	22.14	5.53	2.62	0.04
Group x Pain x Hemisphere	1, 24	0.00002	0.00002	0.00	1.00
Group x Pain x Location	4, 96	32.96	8.24	6.98	0.0001
Group x Hemisphere x Location	4, 96	1.05	0.26	0.12	0.97
Pain x Hemisphere x Location	4, 96	12.18	3.04	3.47	0.01
Group x Pain x Hemisphere x Location	4, 96	5.30	1.32	1.51	0.21

Table 14

ANOVA source table for the high beta bandwidth

Condition: Cold Pressor

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 24	5.18	5.18	0.09	0.77
Pain	1, 24	171.35	171.35	32.09	0.0001
Hemisphere	1, 24	2.94	2.94	0.39	0.54
Location	4, 96	274.57	68.64	8.35	0.0001
Group x Pain	1, 24	49.54	49.54	9.28	0.006
Group x Hemisphere	1, 24	4.12	4.12	0.54	0.47
Group x Location	4, 96	48.17	12.04	1.46	0.22
Pain x Hemisphere	1, 24	3.15	3.15	1.39	0.25
Pain x Location	1, 24	94.28	23.57	12.92	0.0001
Hemisphere x Location	4, 96	19.73	4.93	1.75	0.15
Group x Pain x Hemisphere	1, 24	0.72	0.72	0.31	0.58
Group x Pain x Location	4, 96	43.81	10.95	6.00	0.0002
Group x Hemisphere x Location	4, 96	8.30	2.08	0.74	0.57
Pain x Hemisphere x Location	4, 96	12.11	3.03	3.45	0.01
Group x Pain x Hemisphere x Location	4, 96	7.00	1.75	1.99	0.10

Table 15

ANOVA source table for the delta bandwidth

Condition: Dichotic Listening 2

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 24	70.60	70.60	4.52	0.04
Condition	1, 24	0.72	0.72	0.53	0.47
Hemisphere	1, 24	0.06	0.06	0.04	0.84
Location	4, 96	266.23	66.56	48.54	0.0001
Group x Condition	1, 24	0.06	0.06	0.04	0.84
Group x Hemisphere	1, 24	0.02	0.02	0.01	0.92
Group x Location	4, 96	9.02	2.25	1.64	0.17
Condition x Hemisphere	1, 24	0.27	0.27	1.19	0.29
Condition x Location	1, 24	0.73	0.18	0.69	0.60
Hemisphere x Location	4, 96	1.06	0.26	0.64	0.64
Group x Condition x Hemisphere	1, 24	0.04	0.04	0.18	0.67
Group x Condition x Location	4, 96	0.47	0.12	0.45	0.77
Group x Hemisphere x Location	4, 96	1.39	0.35	0.84	0.50
Condition x Hemisphere x Location	4, 96	0.09	0.02	0.15	0.96
Group x Condition x Hemisphere x Location	4, 96	0.92	0.22	1.54	0.20

Table 16

ANOVA source table for the low beta bandwidth

Condition: Dichotic Listening 2

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 24	128.70	128.70	1.74	0.20
Condition	1, 24	0.11	0.11	0.03	0.86
Hemisphere	1, 24	0.11	0.11	0.01	0.92
Location	4, 96	984.41	246.10	36.99	0.0001
Group x Condition	1, 24	0.69	0.69	0.19	0.66
Group x Hemisphere	1, 24	5.67	5.67	0.50	0.49
Group x Location	4, 96	72.68	18.17	2.73	0.03
Condition x Hemisphere	1, 24	2.51	2.51	2.96	0.10
Condition x Location	1, 24	62.65	15.66	8.60	0.0001
Hemisphere x Location	4, 96	12.37	0.95	0.43	0.76
Group x Condition x Hemisphere	1, 24	0.02	0.02	0.02	0.88
Group x Condition x Location	4, 96	46.77	11.69	6.42	0.0001
Group x Hemisphere x Location	4, 96	3.82	0.95	0.43	0.79
Condition x Hemisphere x Location	4, 96	3.68	0.92	1.70	0.16
Group x Condition x Hemisphere x Location	4, 96	2.70	0.67	1.25	0.30

Table 17

ANOVA source table for the high beta bandwidth

Condition: Dichotic Listening 2

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 24	0.0002	0.0002	0.00	1.00
Condition	1, 24	55.38	55.38	11.78	0.002
Hemisphere	1, 24	2.82	2.82	0.26	0.61
Location	4, 96	210.33	52.58	7.72	0.0001
Group x Condition	1, 24	23.48	23.48	4.99	0.04
Group x Hemisphere	1, 24	0.87	0.87	0.08	0.78
Group x Location	4, 96	27.21	6.80	1.00	0.41
Condition x Hemisphere	1, 24	5.10	5.10	2.22	0.15
Condition x Location	1, 24	86.05	21.51	7.50	0.0001
Hemisphere x Location	4, 96	14.74	3.69	1.01	0.41
Group x Condition x Hemisphere	1, 24	0.01	0.01	0.01	0.94
Group x Condition x Location	4, 96	82.29	20.57	7.17	0.0001
Group x Hemisphere x Location	4, 96	3.03	0.76	0.21	0.93
Condition x Hemisphere x Location	4, 96	7.99	1.99	1.66	0.16
Group x Condition x Hemisphere x Location	4, 96	1.75	0.44	0.36	0.83

Table 18

ANOVA source table for the delta bandwidth including only the Frontal-1 and Temporal-1 locations

Condition: Cold Pressor

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 24	26.47	26.47	6.06	0.02
Pain	1, 24	0.02	0.02	0.04	0.84
Hemisphere	1, 24	0.05	0.05	0.04	0.85
Location	1, 24	208.00	208.00	277.78	0.0001
Group x Pain	1, 24	0.17	0.17	0.48	0.50
Group x Hemisphere	1, 24	0.03	0.03	0.02	0.88
Group x Location	1, 24	1.96	1.96	2.62	0.12
Pain x Hemisphere	1, 24	0.005	0.005	0.03	0.86
Pain x Location	1, 24	1.26	1.26	5.31	0.03
Hemisphere x Location	1, 24	0.06	0.06	0.14	0.71
Group x Pain x Hemisphere	1, 24	0.13	0.13	0.88	0.36
Group x Pain x Location	1, 24	2.16	2.16	9.10	0.006
Group x Hemisphere x Location	1, 24	0.04	0.04	0.10	0.75
Pain x Hemisphere x Location	1, 24	0.02	0.02	0.23	0.64
Group x Pain x Hemisphere x Location	1, 24	0.34	0.34	3.34	0.08

Table 19

ANOVA source table for the low beta bandwidth including only Frontal-1 and Temporal-1 locations

Condition: Cold Pressor

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 24	9.52	9.52	0.38	0.55
Pain	1, 24	35.81	35.81	10.79	0.003
Hemisphere	1, 24	6.27	6.27	1.04	0.32
Location	1, 24	7.35	7.35	0.60	0.45
Group x Pain	1, 24	33.68	33.68	10.15	0.004
Group x Hemisphere	1, 24	0.19	0.19	0.03	0.86
Group x Location	1, 24	36.47	36.47	2.97	0.10
Pain x Hemisphere	1, 24	6.91	6.91	3.01	0.10
Pain x Location	1, 24	40.60	40.60	15.76	0.0006
Hemisphere x Location	1, 24	11.88	11.88	3.12	0.09
Group x Pain x Hemisphere	1, 24	1.65	1.65	0.72	0.41
Group x Pain x Location	1, 24	17.60	17.60	6.83	0.02
Group x Hemisphere x Location	1, 24	0.11	0.11	0.03	0.87
Pain x Hemisphere x Location	1, 24	6.20	6.20	3.67	0.07
Group x Pain x Hemisphere x Location	1, 24	1.87	1.87	1.10	0.30

Table 20

ANOVA source table for the high beta bandwidth including only Frontal-1 and Temporal-1 locations

Condition: Cold Pressor

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 24	8.32	8.32	0.27	0.61
Pain	1, 24	130.89	130.89	23.76	0.0001
Hemisphere	1, 24	3.05	3.05	0.52	0.48
Location	1, 24	71.56	71.56	4.15	0.05
Group x Pain	1, 24	54.63	54.63	9.92	0.004
Group x Hemisphere	1, 24	8.00	8.00	1.36	0.25
Group x Location	1, 24	42.48	42.48	2.46	0.13
Pain x Hemisphere	1, 24	7.31	7.31	1.98	0.17
Pain x Location	1, 24	74.64	74.64	19.87	0.0002
Hemisphere x Location	1, 24	12.41	12.41	2.41	0.13
Group x Pain x Hemisphere	1, 24	3.93	3.93	1.98	0.17
Group x Pain x Location	1, 24	28.50	28.50	7.59	0.01
Group x Hemisphere x Location	1, 24	2.50	2.50	0.49	0.49
Pain x Hemisphere x Location	1, 24	7.16	7.16	5.17	0.03
Group x Pain x Hemisphere x Location	1, 24	2.91	2.91	2.10	0.16

Table 21

ANOVA source table for the delta bandwidth including only the High Hostile Group

Condition: Cold Pressor

Source	*df*	*SS*	*MS*	*F*	*p*
Pain	1, 12	0.04	0.04	0.09	0.76
Hemisphere	1, 12	0.0009	0.0009	0.00	0.97
Location	1, 12	84.78	84.78	84.44	0.0001
Pain x Hemisphere	1, 12	0.04	0.04	0.36	0.56
Pain x Location	1, 12	0.06	0.06	1.38	0.26
Hemisphere x Location	1, 12	0.10	0.10	0.62	0.45
Pain x Hemisphere x Location	1, 12	0.09	0.09	1.38	0.26

Table 22

ANOVA source table for the low beta bandwidth including only the High Hostile Group

Condition: Cold Pressor

Source	*df*	*SS*	*MS*	*F*	*p*
Pain	1, 12	69.47	69.47	13.71	0.003
Hemisphere	1, 12	4.32	4.32	0.96	0.35
Location	1, 12	5.54	5.54	0.30	0.60
Pain x Hemisphere	1, 12	7.65	7.65	2.57	0.14
Pain x Location	1, 12	55.83	55.83	12.66	0.004
Hemisphere x Location	1, 12	7.11	7.11	2.52	0.14
Pain x Hemisphere x Location	1, 12	7.43	7.43	3.34	0.09

Table 23

ANOVA source table for the high beta bandwidth including only the High Hostile Group

Condition: Cold Pressor

Source	*df*	*SS*	*MS*	*F*	*p*
Pain	1, 12	177.32	177.32	19.88	0.0008
Hemisphere	1, 12	10.47	10.47	2.04	0.18
Location	1, 12	112.15	112.15	4.96	0.05
Pain x Hemisphere	1, 12	10.99	10.99	4.75	0.05
Pain x Location	1, 12	97.70	97.70	14.36	0.003
Hemisphere x Location	1, 12	13.02	13.02	3.44	0.09
Pain x Hemisphere x Location	1, 12	9.60	9.60	5.86	0.03

Table 24

ANOVA source table for the delta bandwidth including only the Low Hostile Group

Condition: Cold Pressor

Source	*df*	*SS*	*MS*	*F*	*p*
Pain	1, 12	0.15	0.15	0.53	0.48
Hemisphere	1, 12	0.08	0.08	0.04	0.84
Location	1, 12	125.18	125.18	253.62	0.0001
Pain x Hemisphere	1, 12	0.09	0.09	0.52	0.49
Pain x Location	1, 12	3.36	3.36	7.79	0.02
Hemisphere x Location	1, 12	0.009	0.009	0.00	0.97
Pain x Hemisphere x Location	1, 12	0.27	0.27	1.98	0.19

Table 25

ANOVA source table for the low beta bandwidth including only the Low Hostile Group

Condition: Cold Pressor

Source	*df*	*SS*	*MS*	*F*	*p*
Pain	1, 12	0.02	0.02	0.01	0.92
Hemisphere	1, 12	2.13	2.13	0.28	0.60
Location	1, 12	38.28	38.28	6.59	0.03
Pain x Hemisphere	1, 12	0.90	0.90	0.56	0.47
Pain x Location	1, 12	2.37	2.37	3.20	0.10
Hemisphere x Location	1, 12	4.87	4.87	1.02	0.33
Pain x Hemisphere x Location	1, 12	0.63	0.63	0.55	0.47

Table 26

ANOVA source table for the high beta bandwidth including only the Low Hostile Group

Condition: Cold Pressor

Source	*df*	*SS*	*MS*	*F*	*p*
Pain	1, 12	8.20	8.20	3.91	0.07
Hemisphere	1, 12	0.59	0.59	0.09	0.77
Location	1, 12	1.88	1.88	0.16	0.70
Pain x Hemisphere	1, 12	0.26	0.26	0.16	0.70
Pain x Location	1, 12	5.44	5.44	7.71	0.02
Hemisphere x Location	1, 12	1.88	1.88	0.29	0.60
Pain x Hemisphere x Location	1, 12	0.47	0.47	0.41	0.53

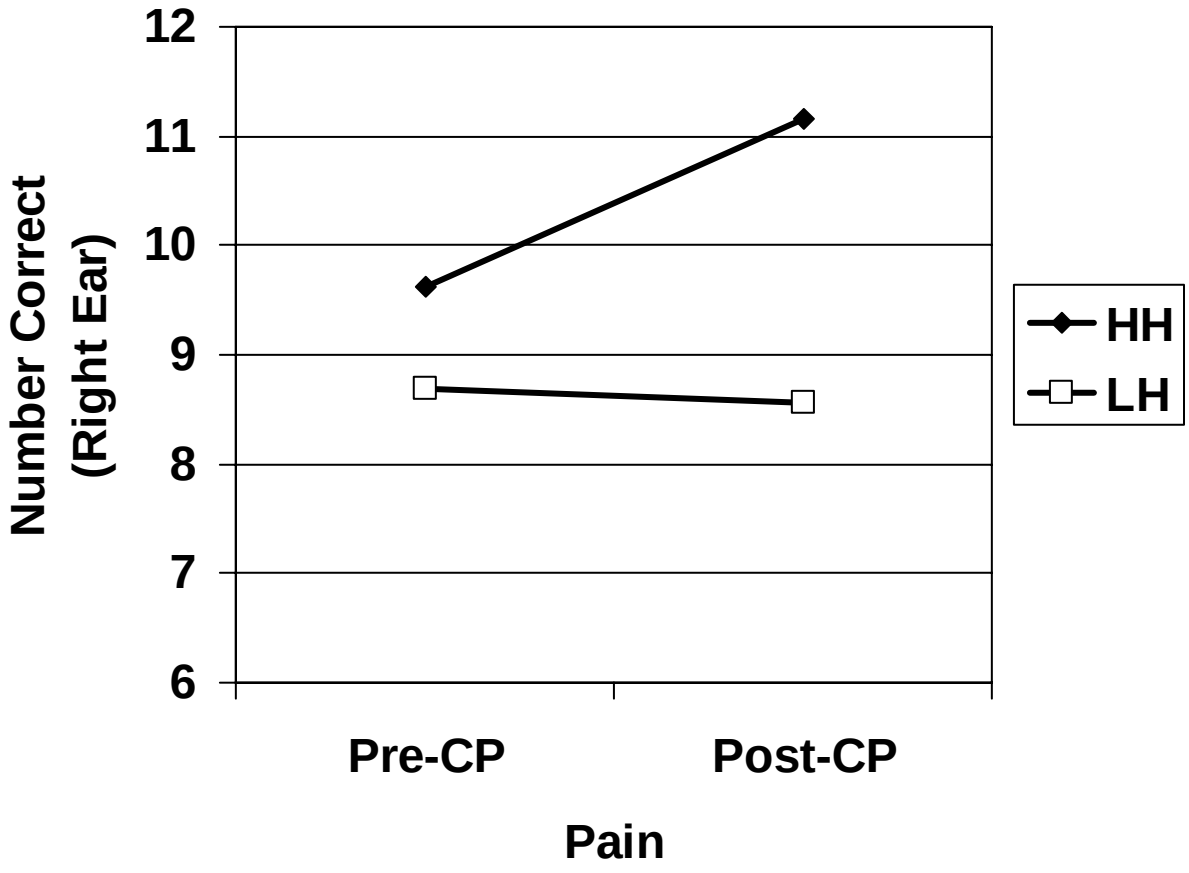


Figure 1. Group differences in identification of stimuli at the right ear as a function of the cold pressor.

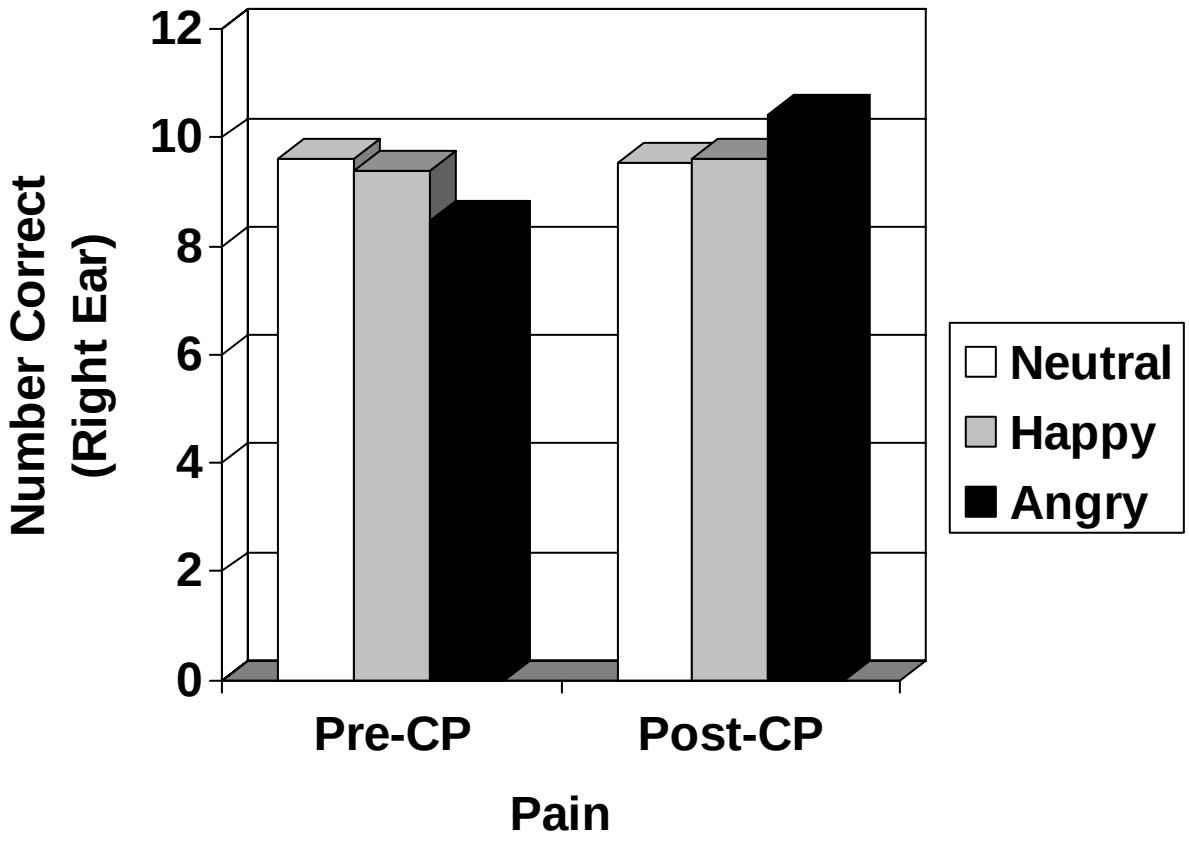


Figure 2. Changes in affect identification at the right ear as a function of the cold pressor.

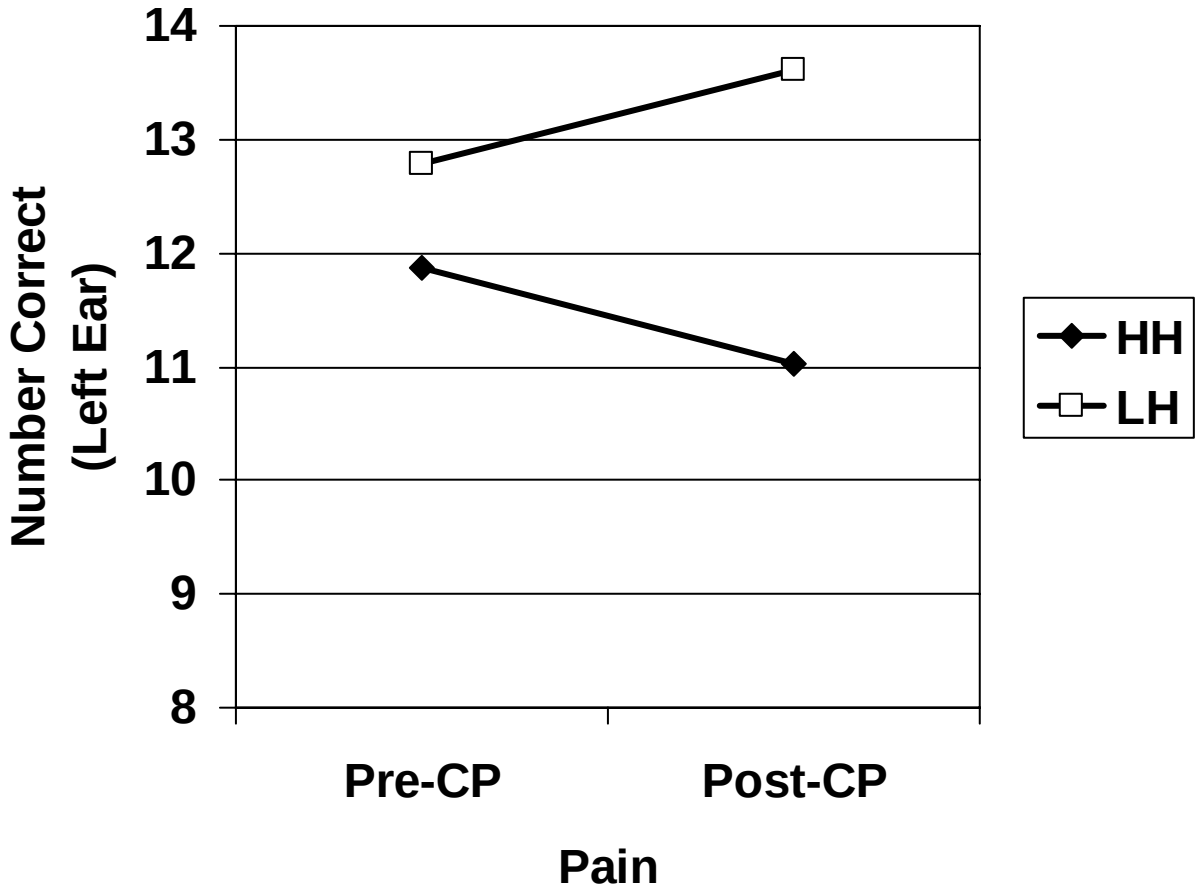


Figure 3. Group differences in number correct at the left ear as a function of the cold pressor.

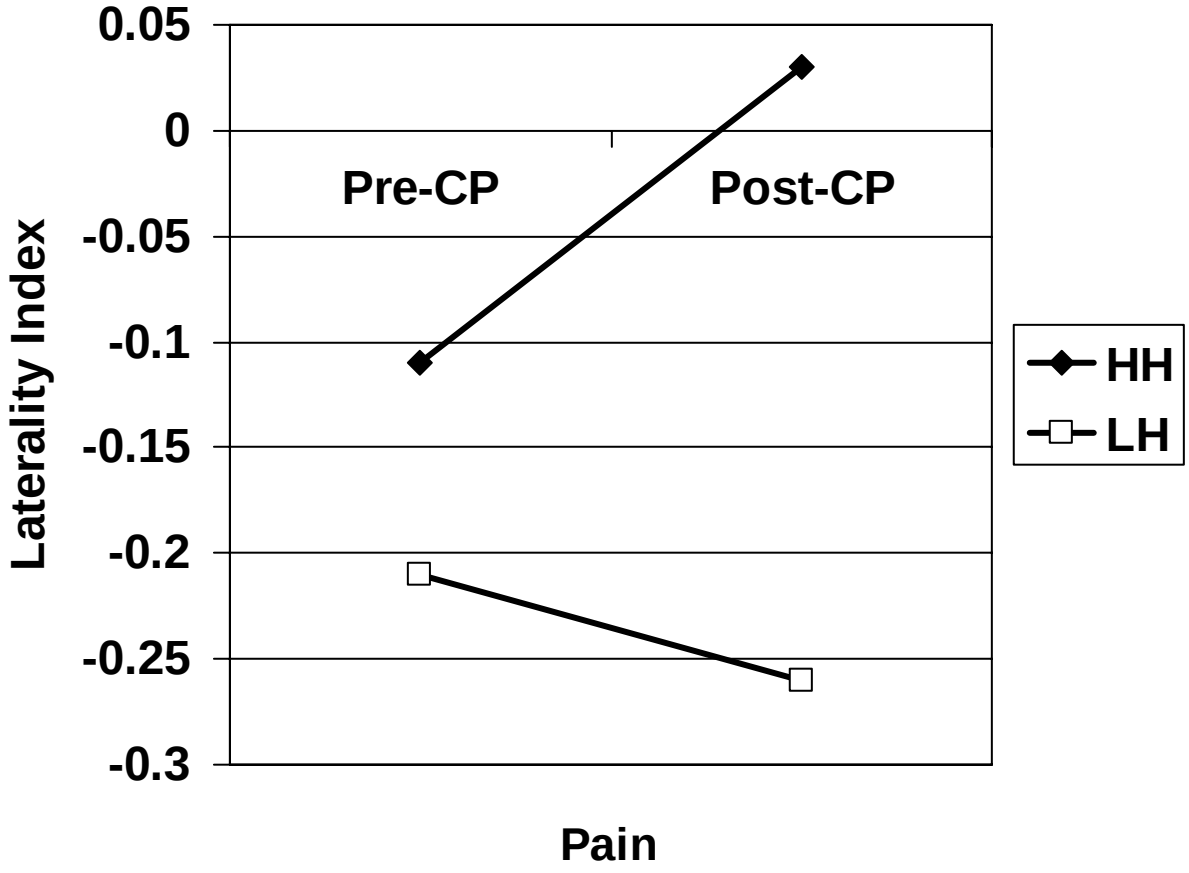


Figure 4. Group differences in laterality indices as a function of the cold pressor.

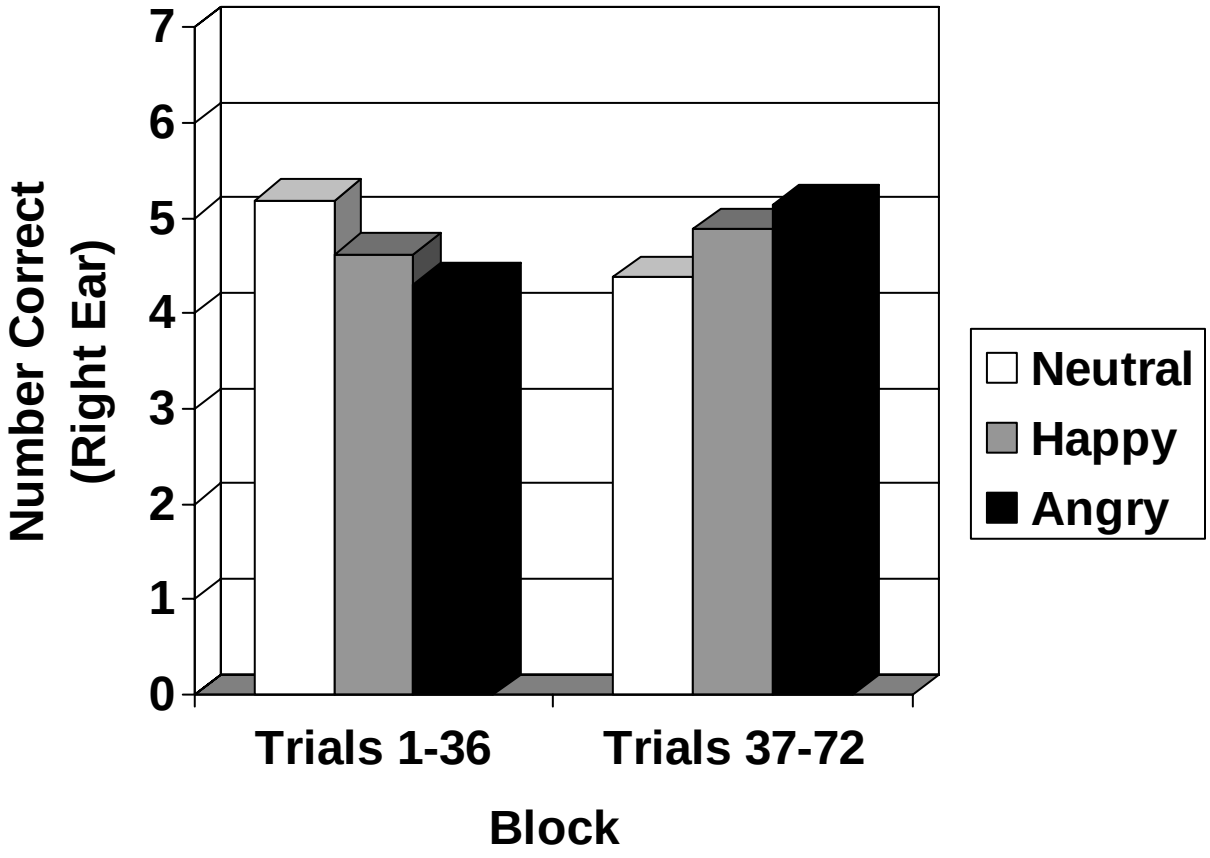


Figure 5. Number correct at the right ear as a function of affect and block.

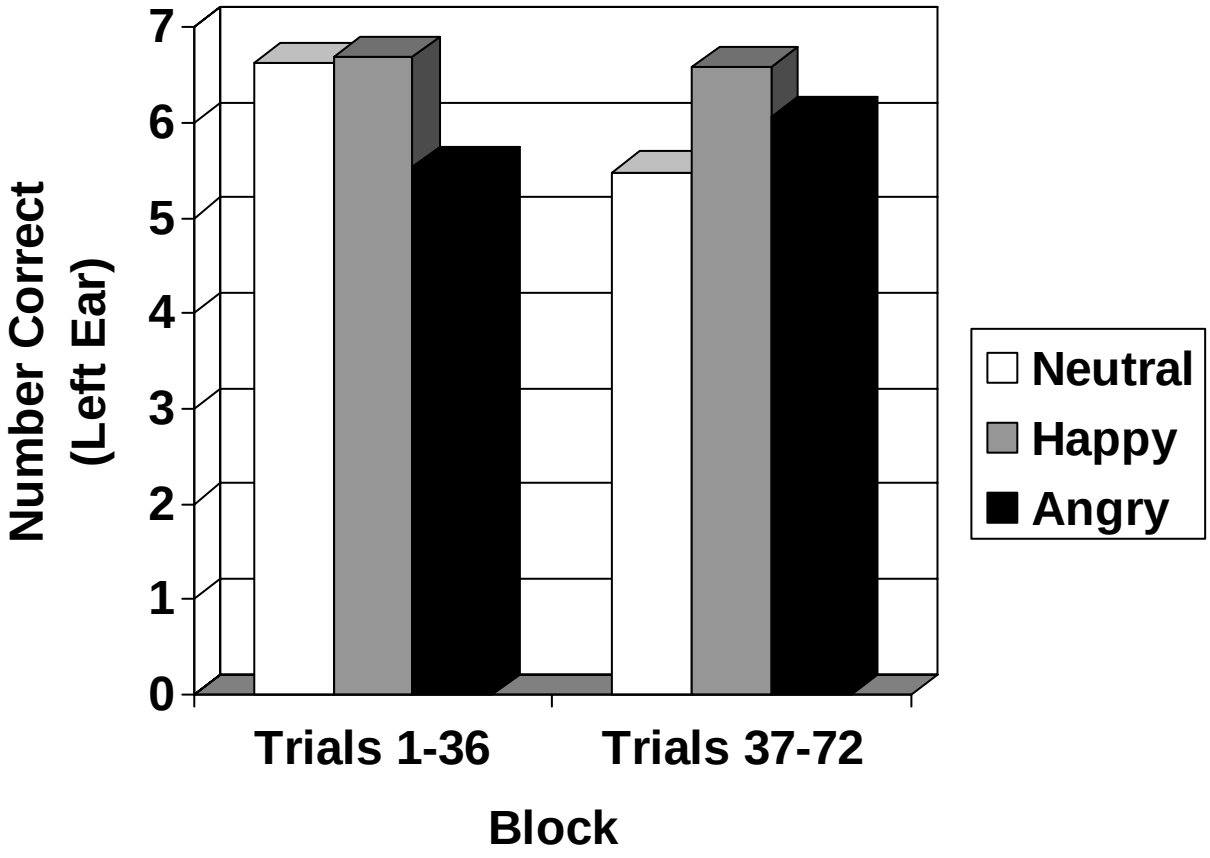


Figure 6. Changes in the number correct at the left ear as a function of affect and block.

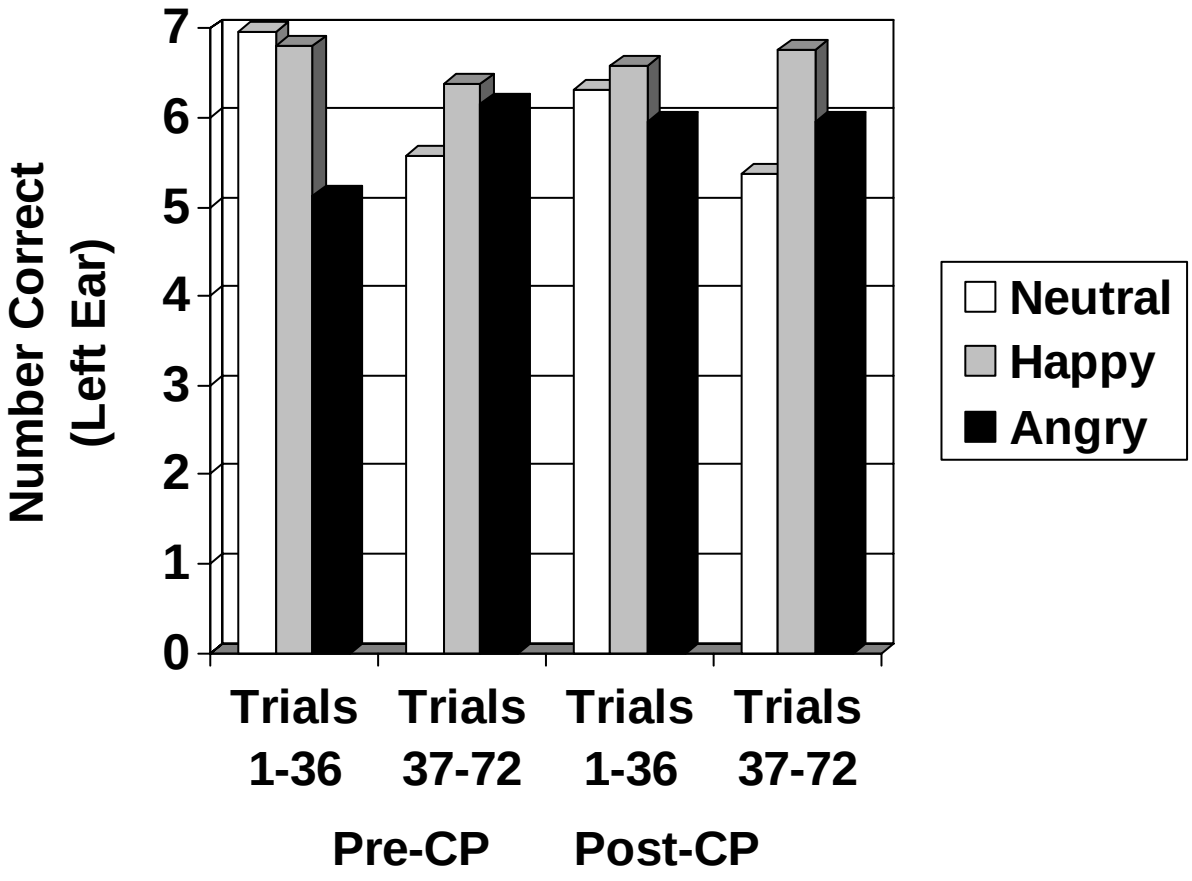


Figure 7. Number of correctly identified stimuli at the left ear as a function of affect, block, and pain condition.

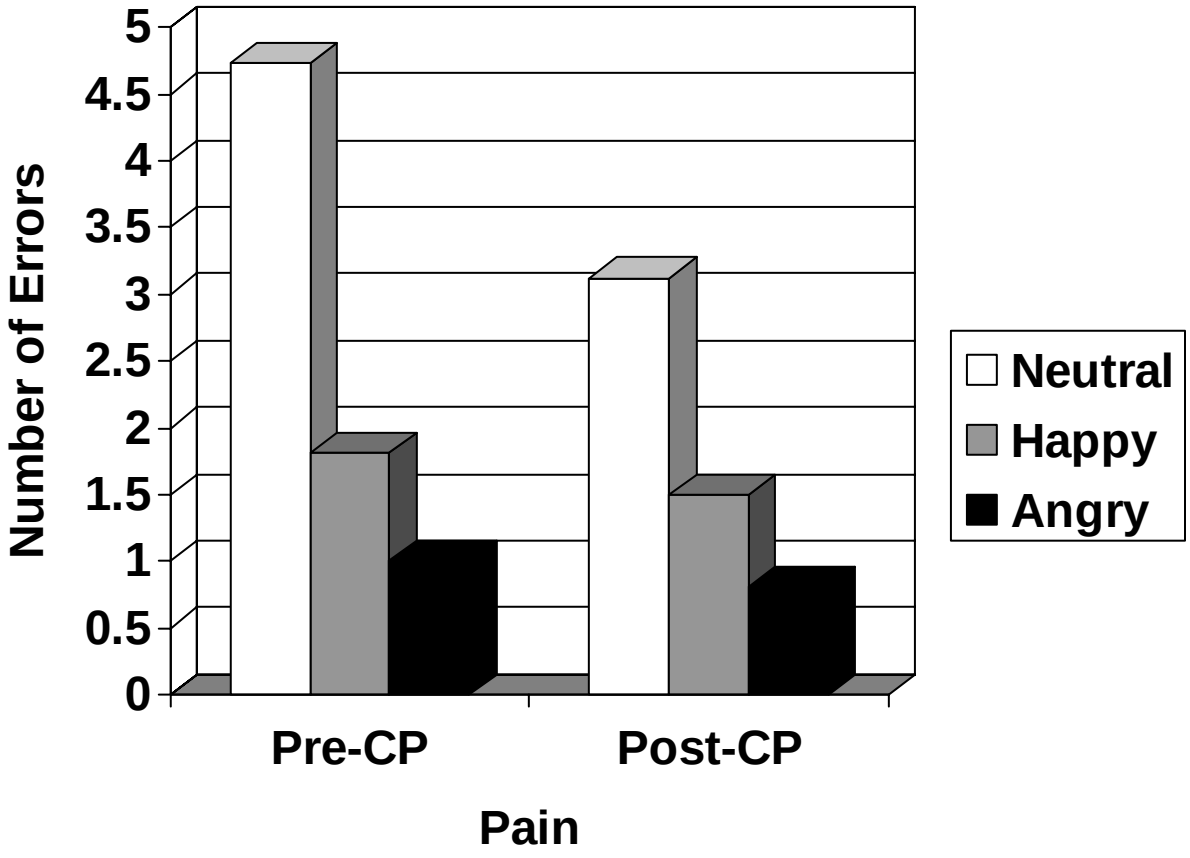


Figure 8. Number of errors as a function of affect and pain condition.

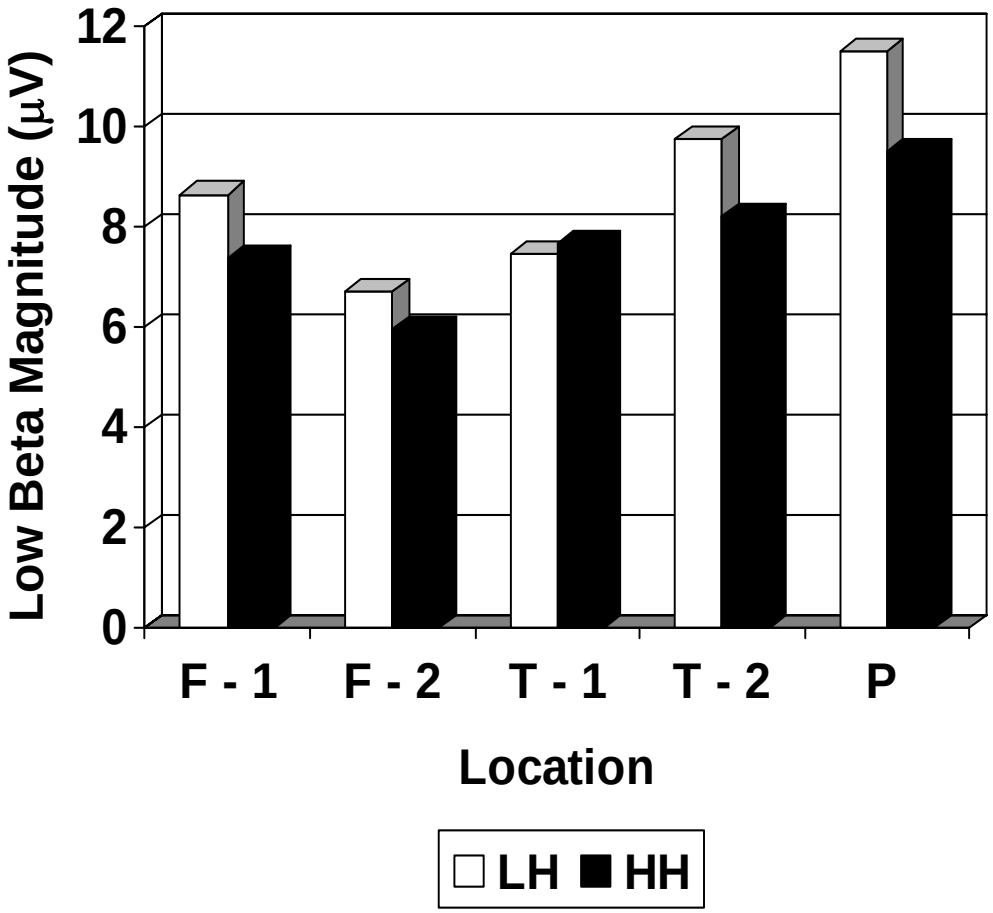


Figure 9. Group differences in low beta magnitude at each location.

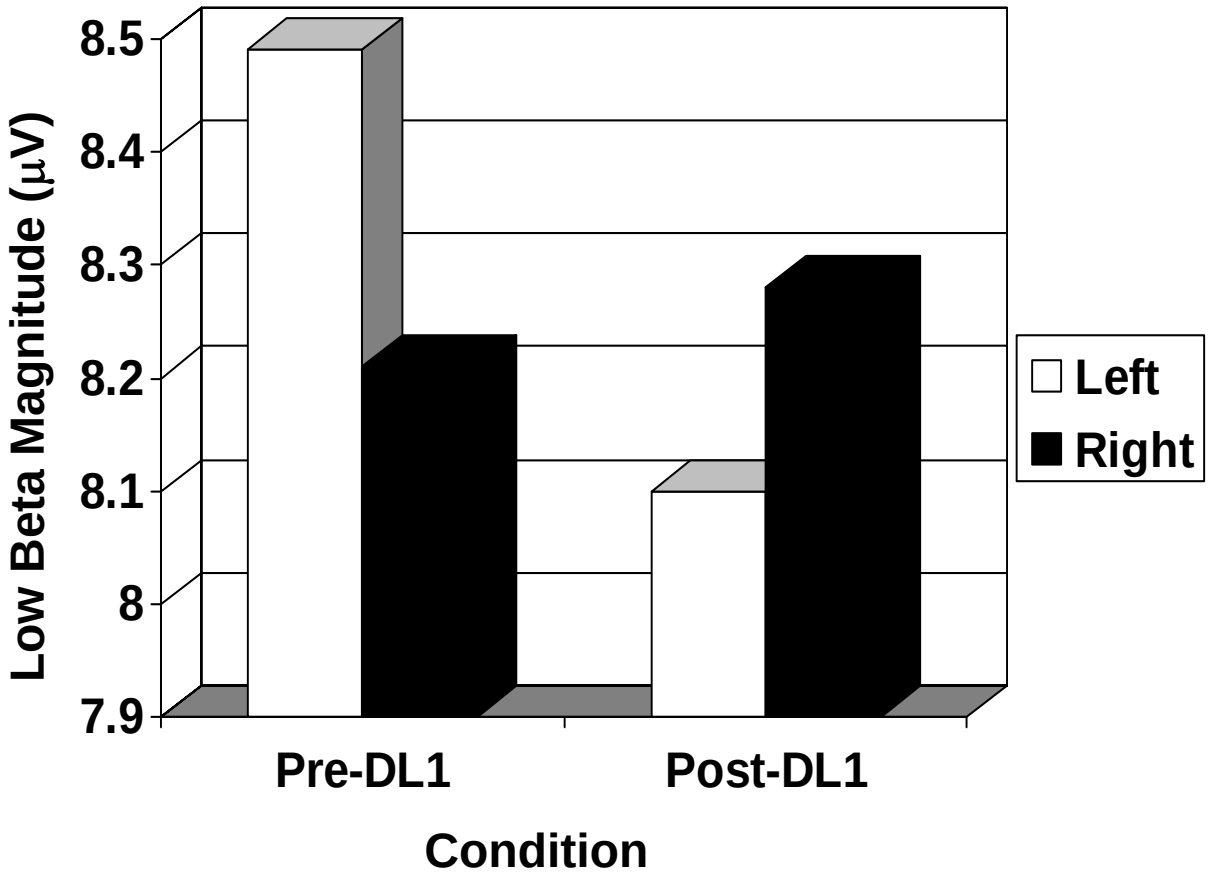


Figure 10. Low beta magnitude at each hemisphere from pre- to post-dichotic listening 1 task conditions.

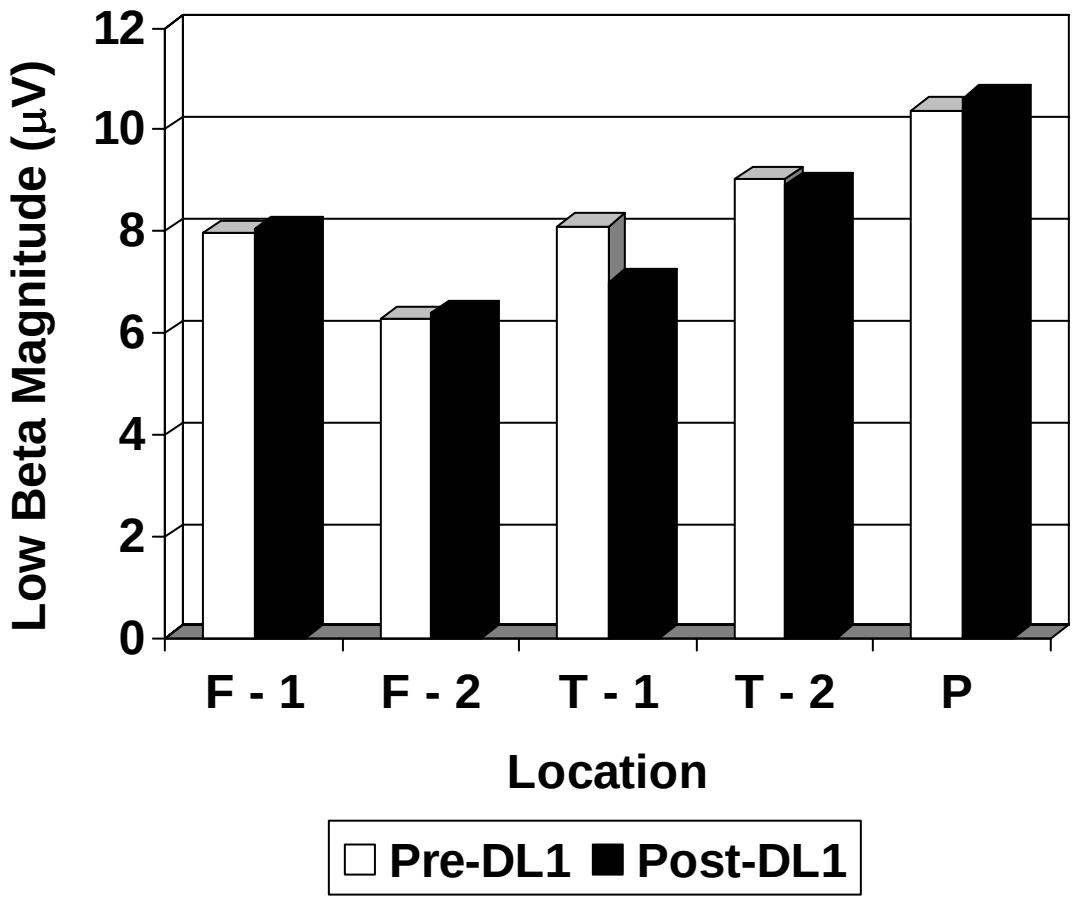


Figure 11. Low beta magnitude at each location from pre- to post-dichotic listening 1 task conditions.

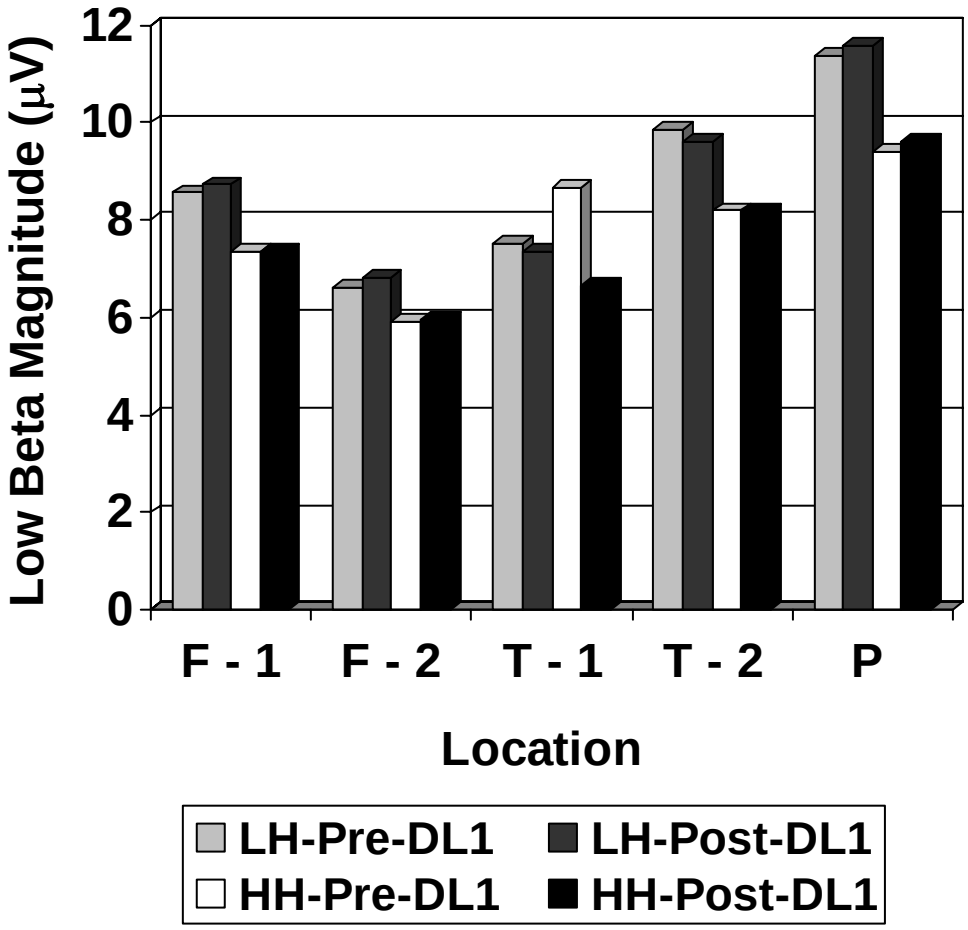


Figure 12. Low beta magnitude at each location as a function of group and dichotic listening 1 task conditions.

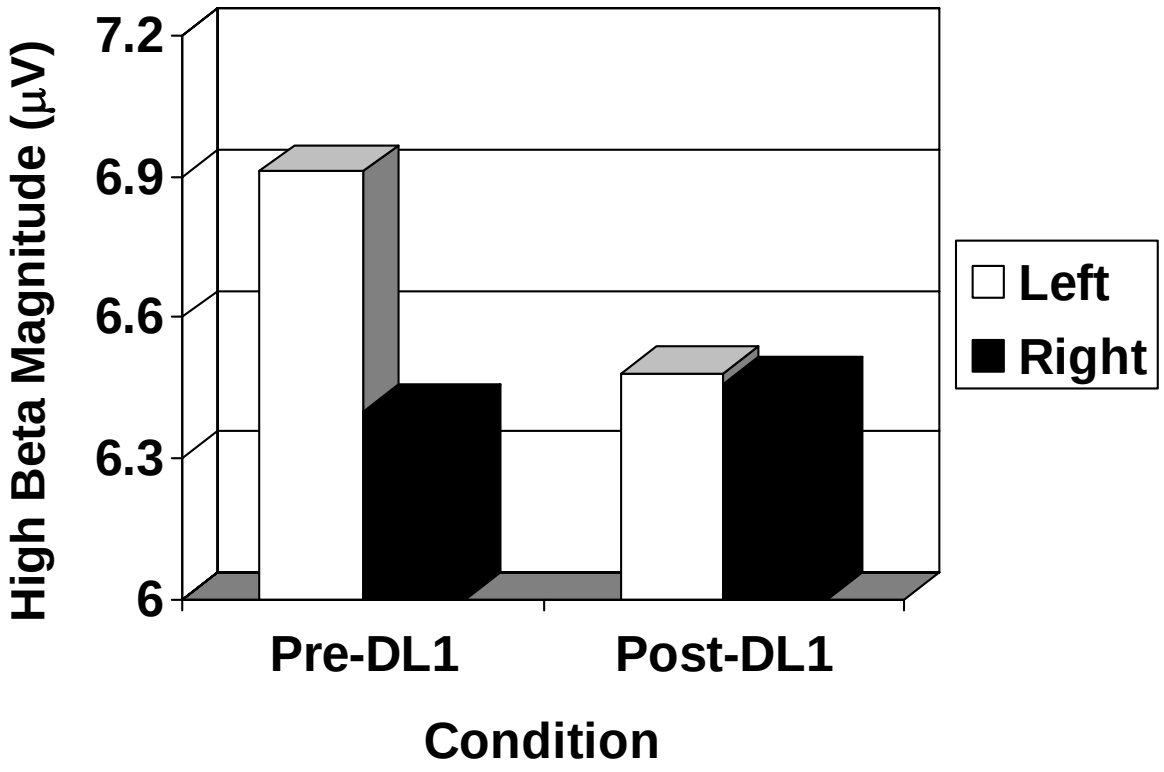


Figure 13. High beta magnitude at the left and right hemispheres from pre- to post-dichotic listening 1 conditions.

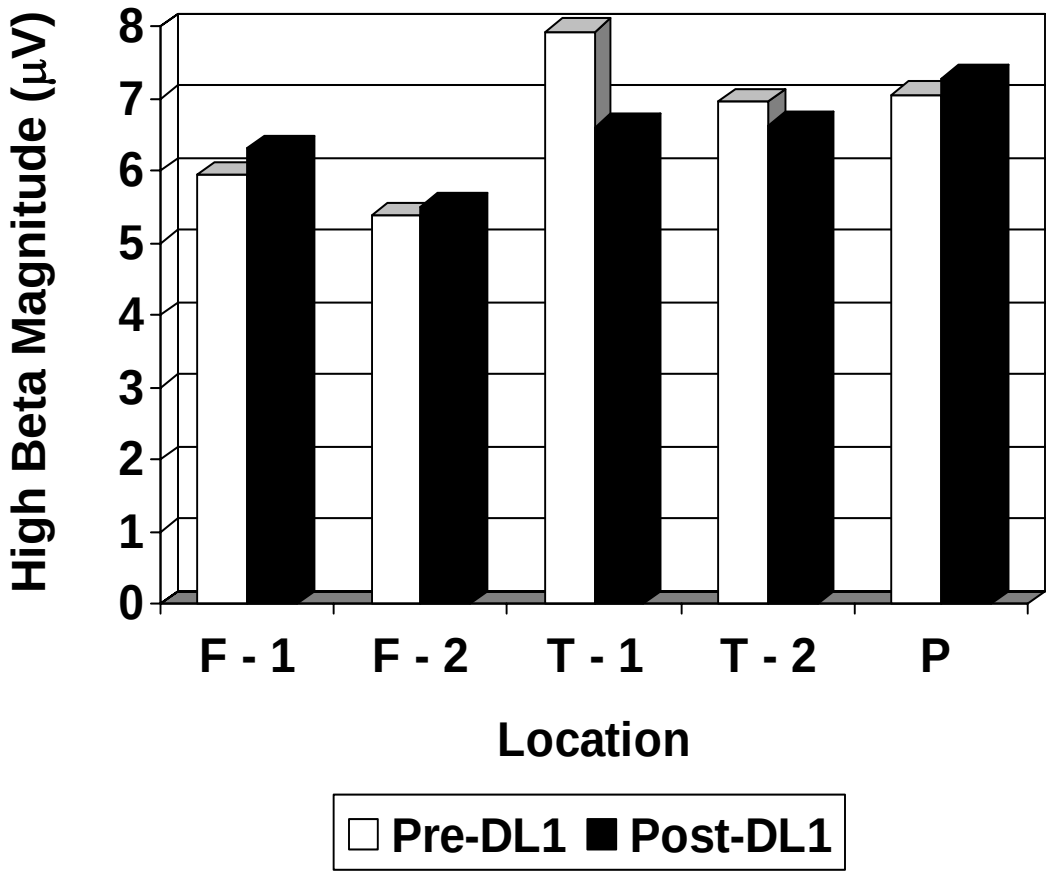


Figure 14. High beta magnitude at each location as a function of the dichotic listening 1 task.

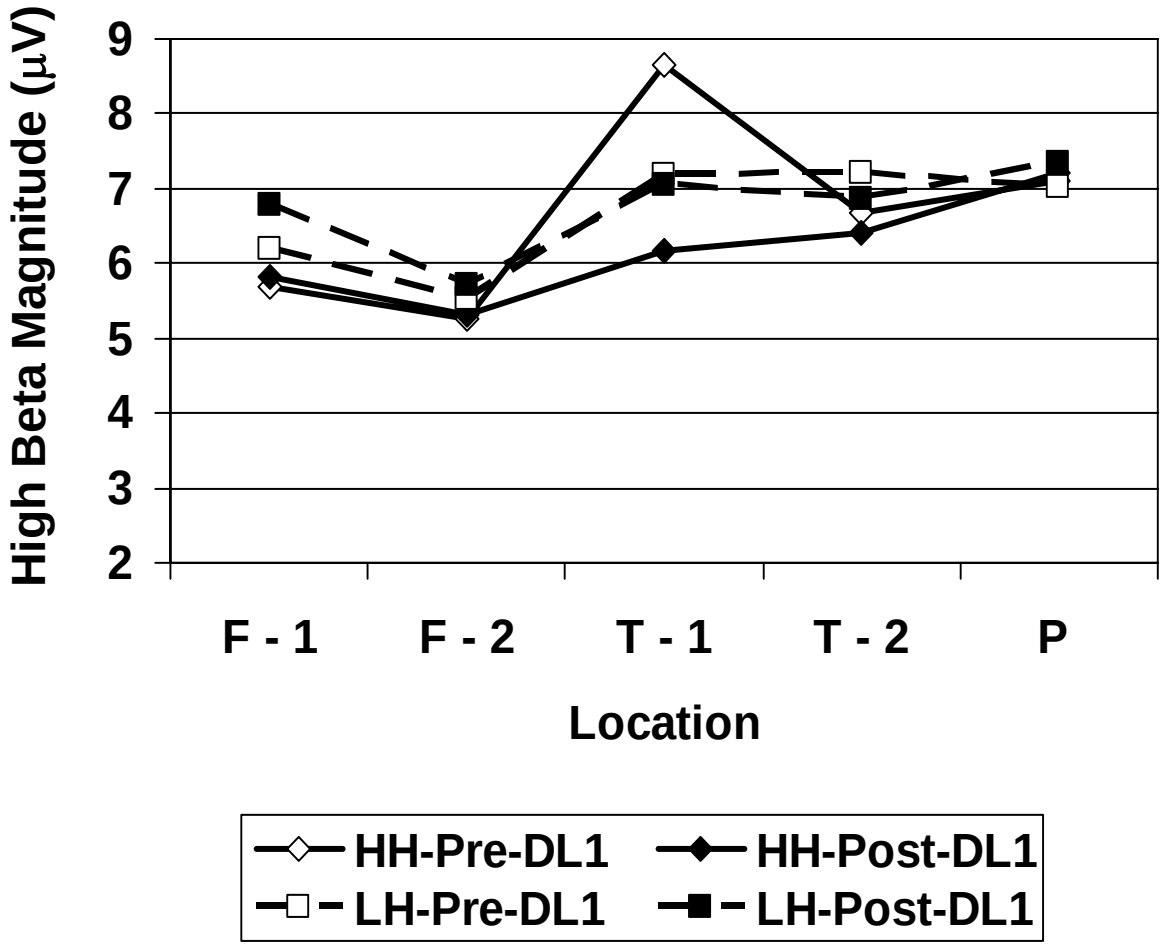


Figure 15. Group differences in high beta magnitude at each location as a function of the dichotic listening 1 task.

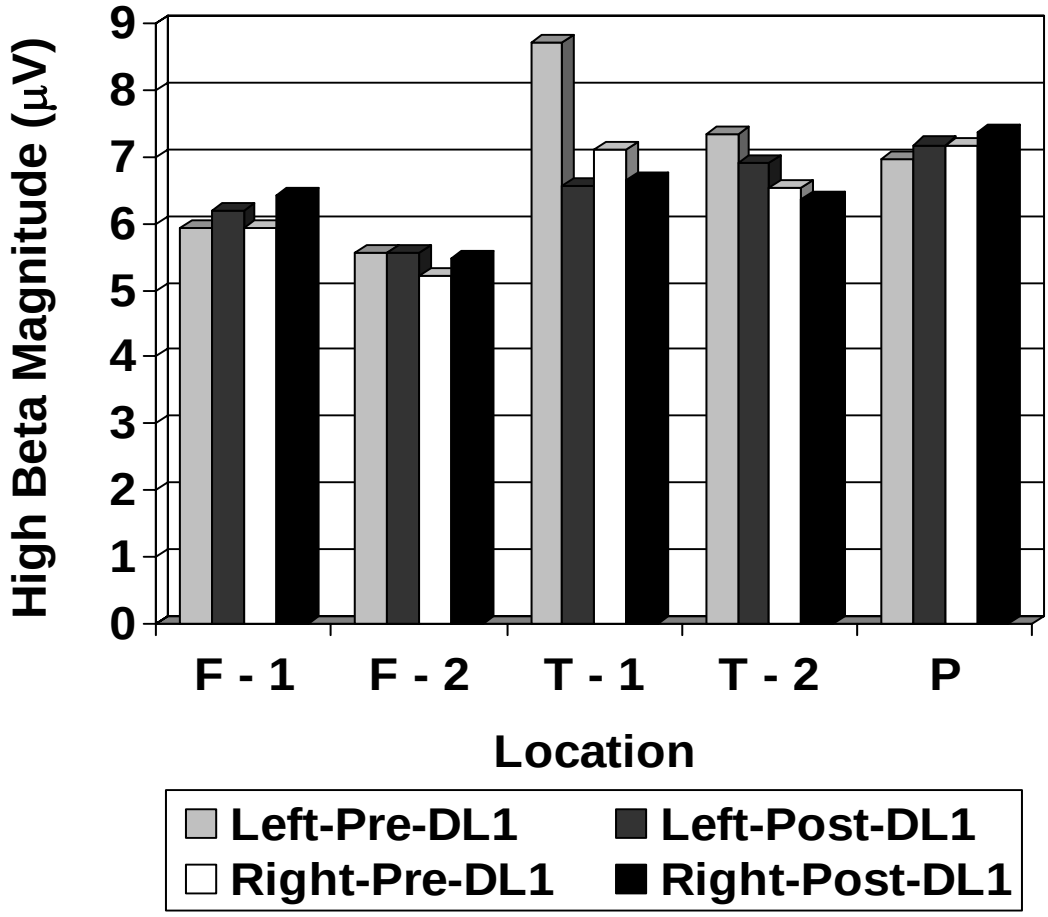


Figure 16. High beta magnitude at each location within the right and left hemispheres as a function of the dichotic listening 1 task.

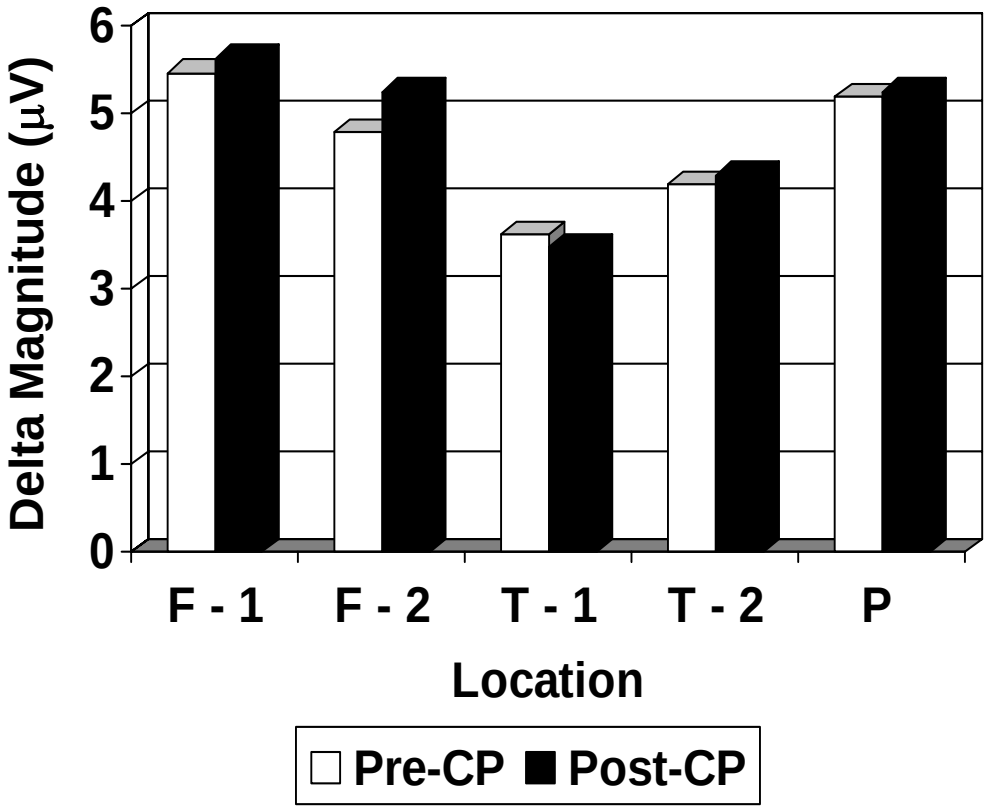


Figure 17. Delta magnitude at each location as a function of the cold pressor.

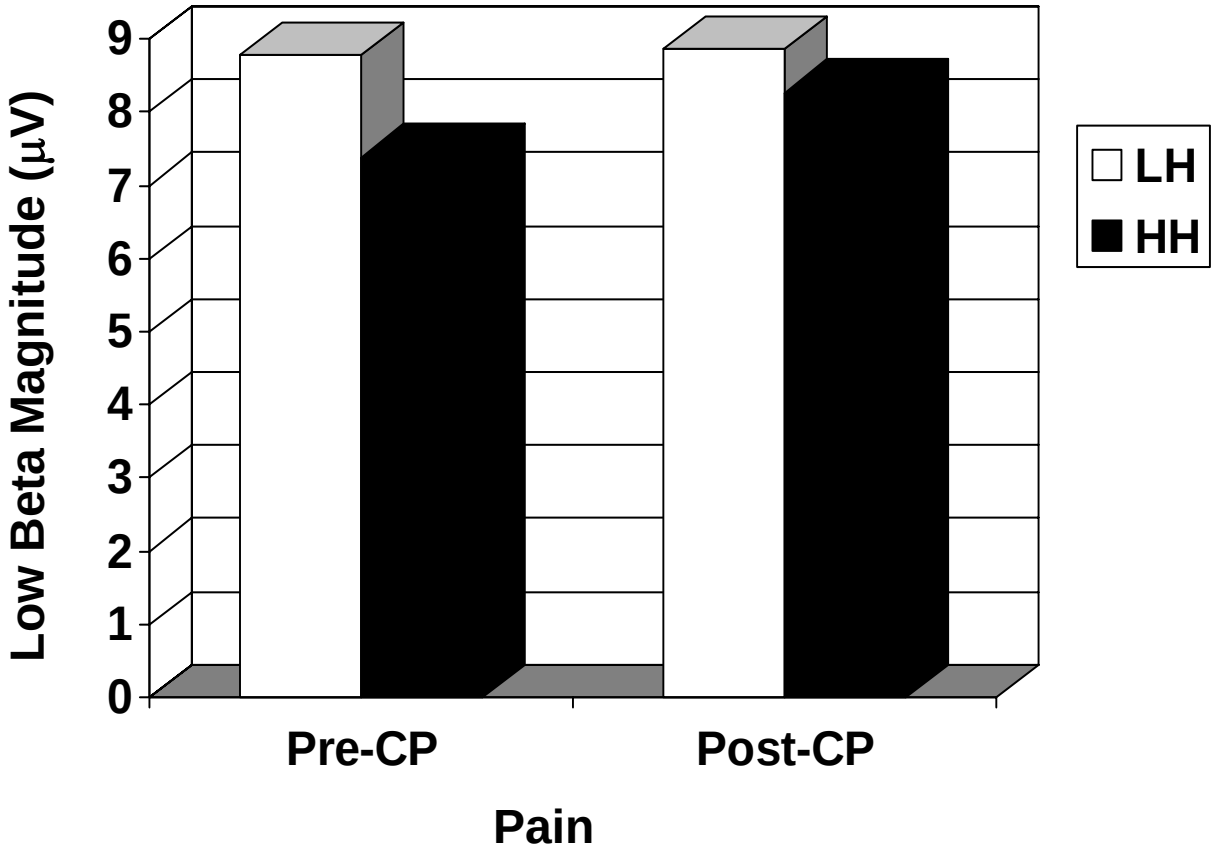


Figure 18. Group differences in low beta magnitude as a function of the cold pressor.

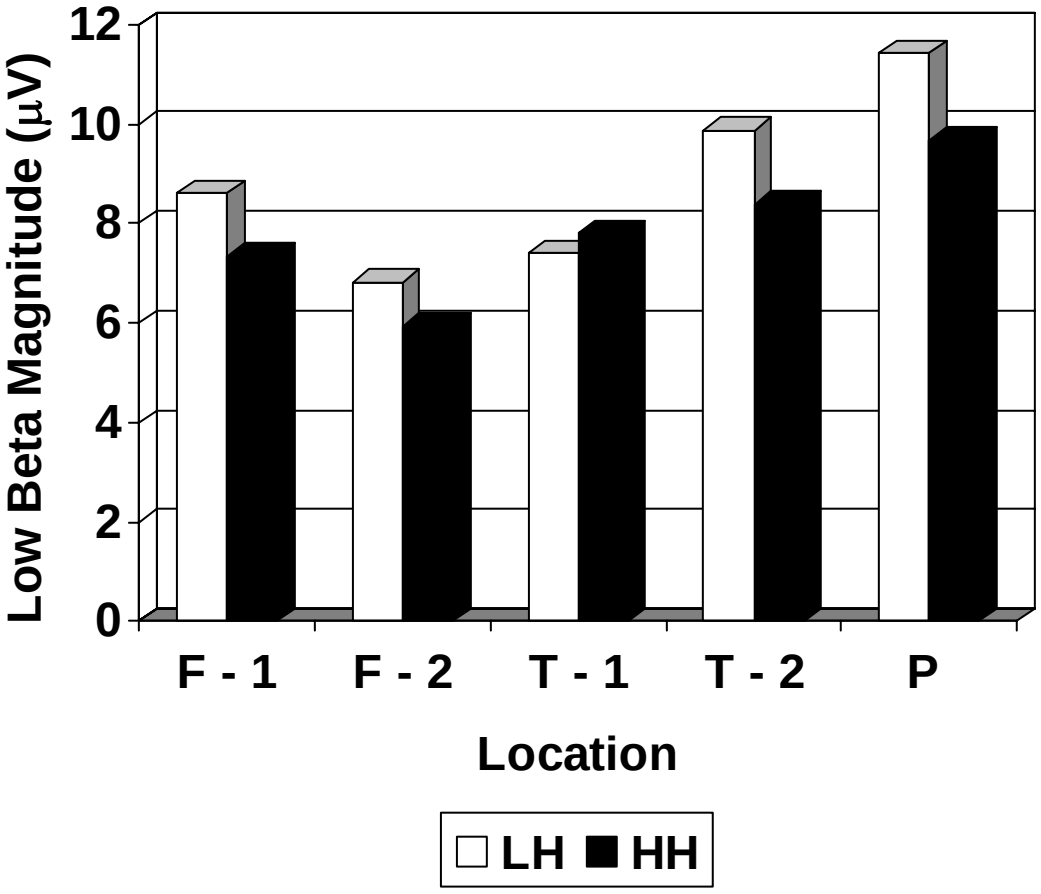


Figure 19. Low beta magnitude at each location as a function of group.

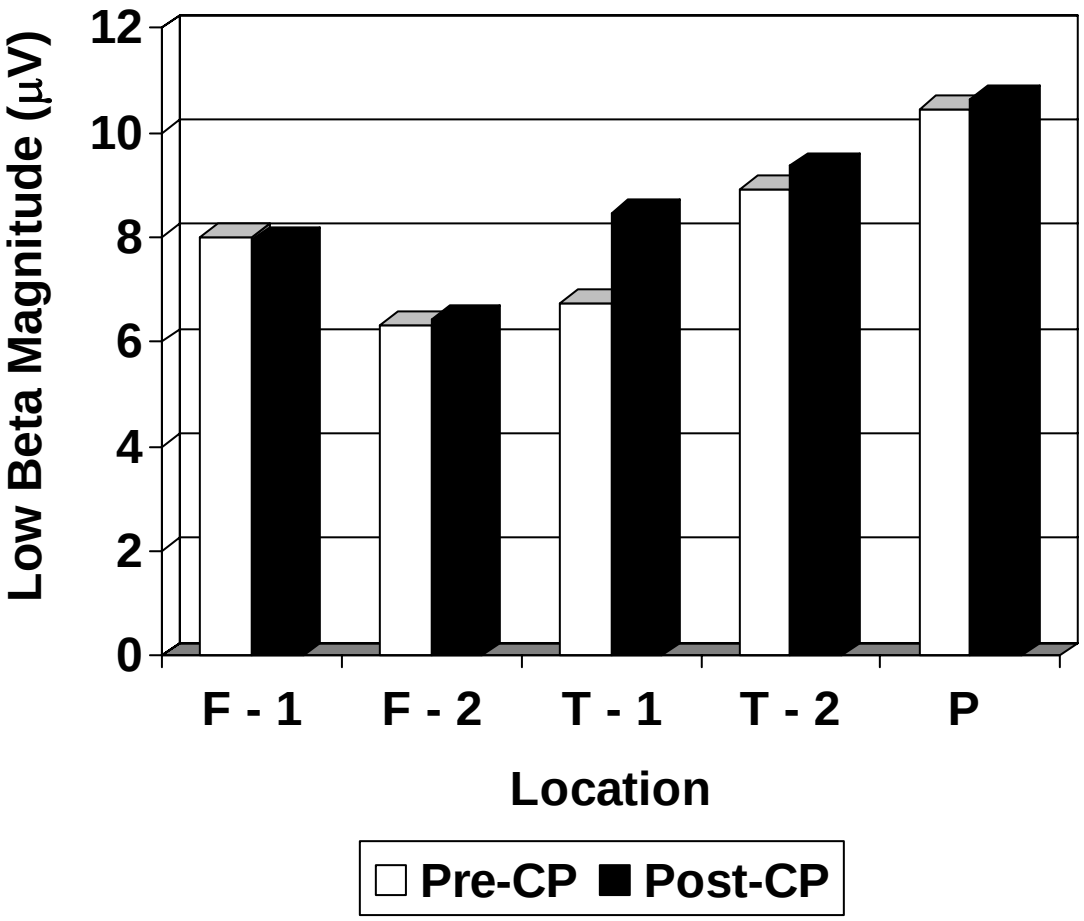


Figure 20. Low beta changes at each location from pre- to post-cold pressor conditions.

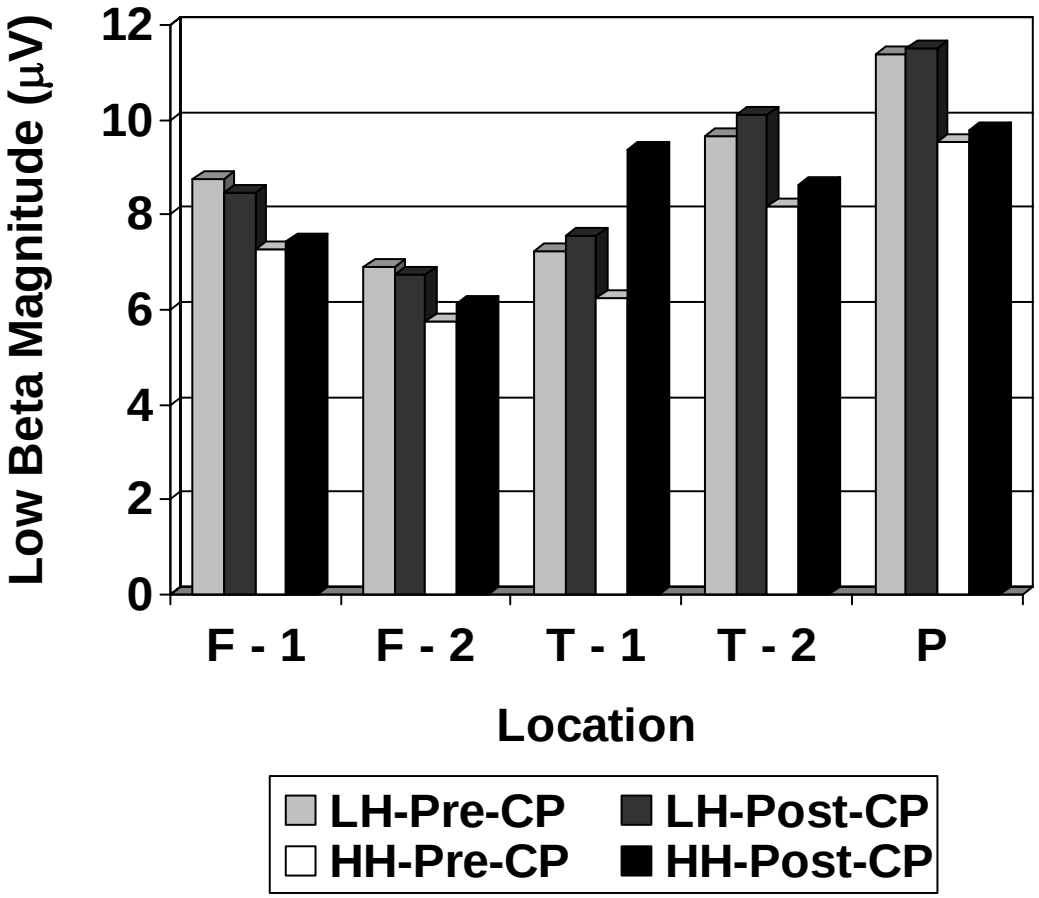


Figure 21. Group differences in low beta at each location as a function of the cold pressor.

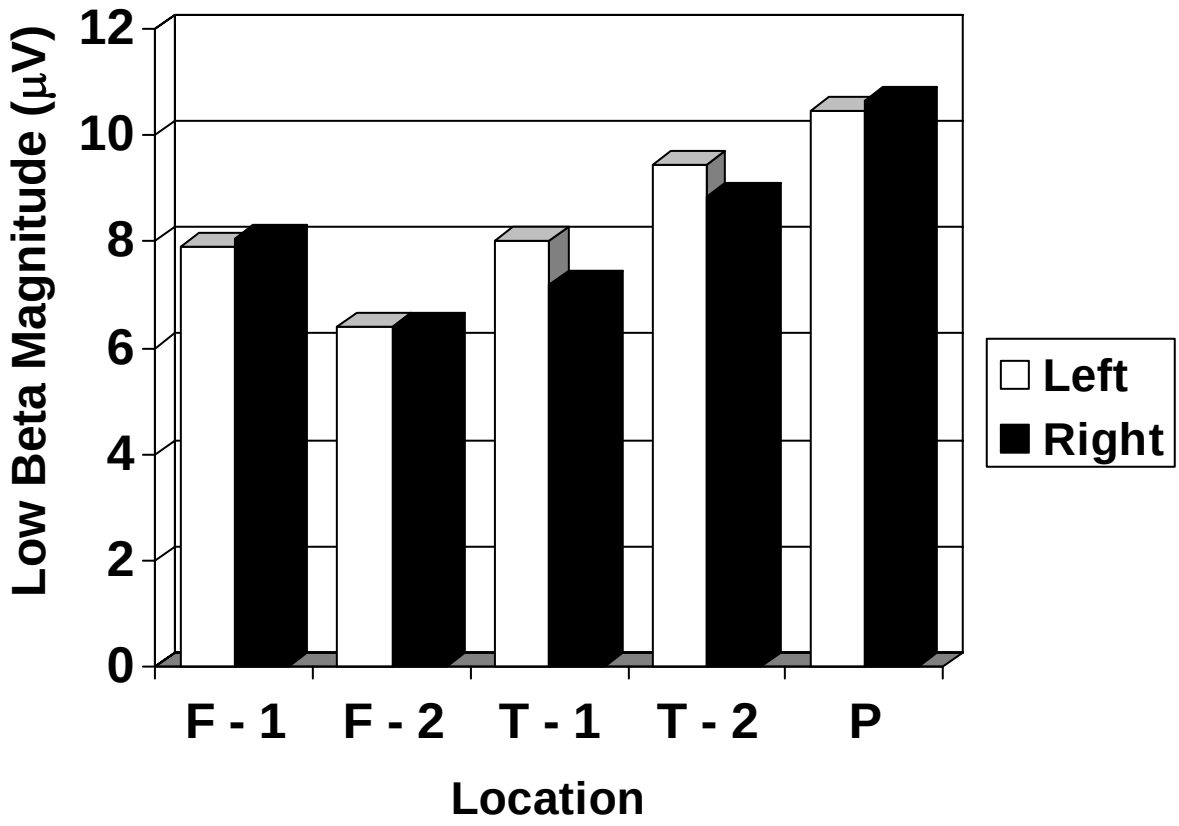


Figure 22. Low beta as a function of location and hemisphere in the cold pressor condition.

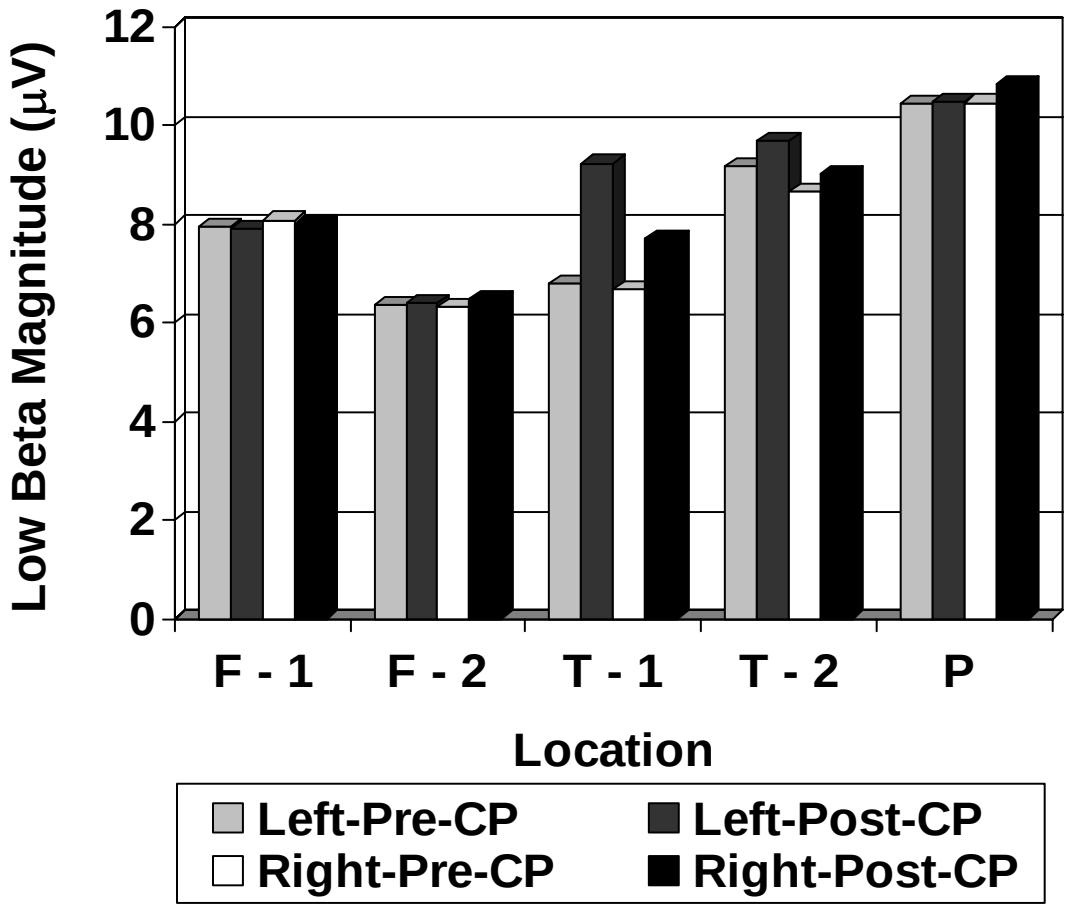


Figure 23. Low beta magnitude at each location within the left and right hemispheres as a function of the cold pressor.

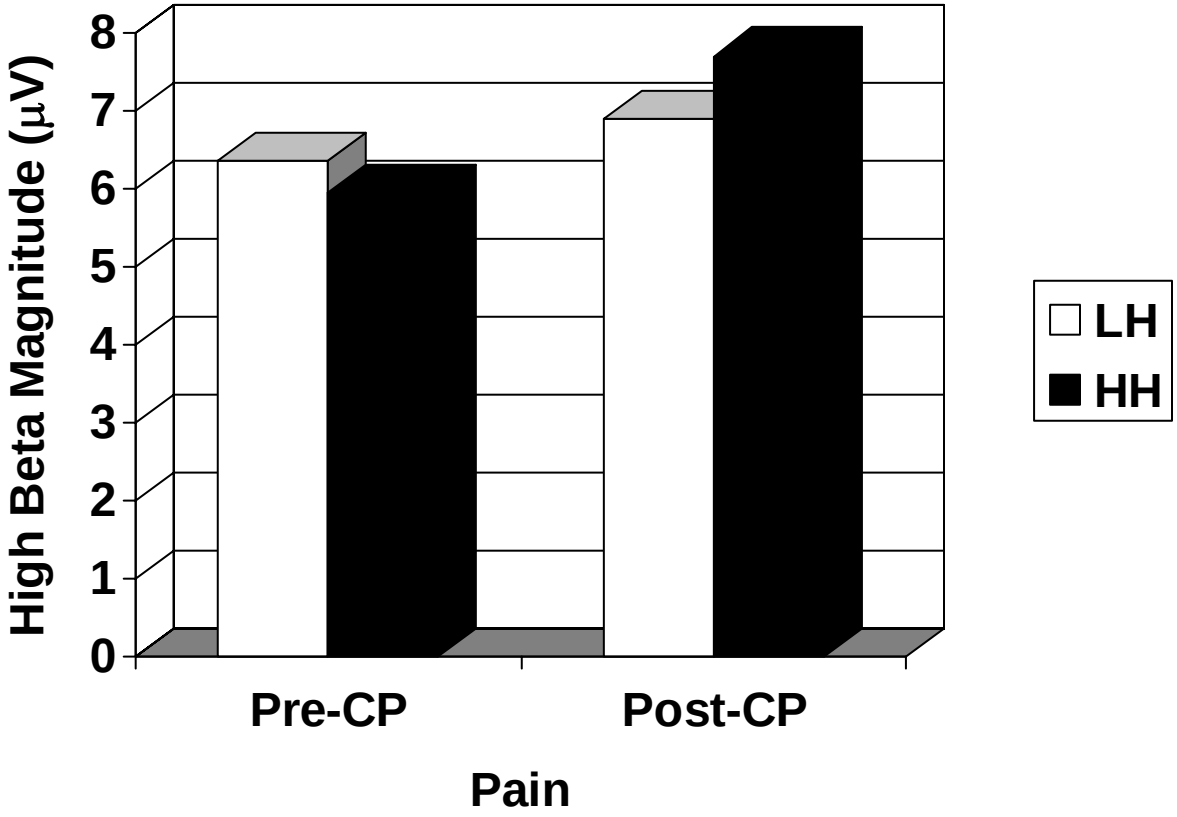


Figure 24. Group differences in high beta magnitude as a function of the cold pressor.

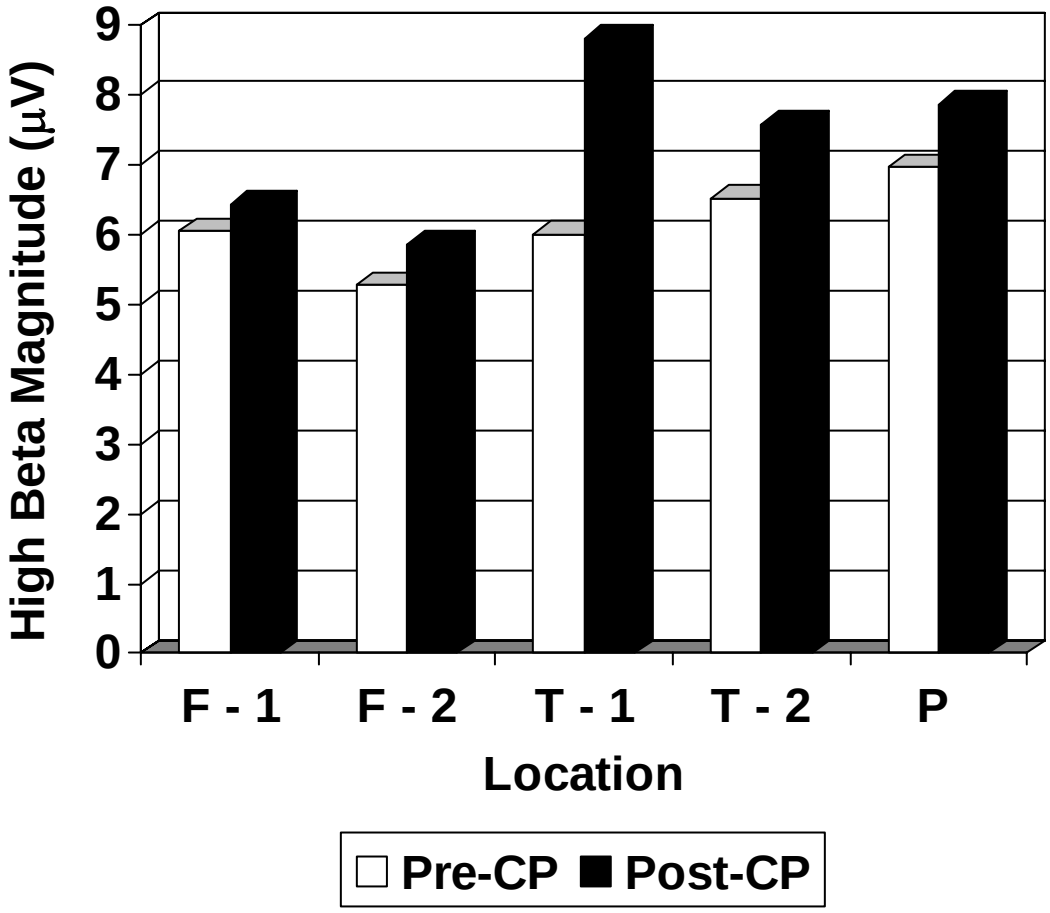


Figure 25. High beta magnitude at each location as a function of the cold pressor.

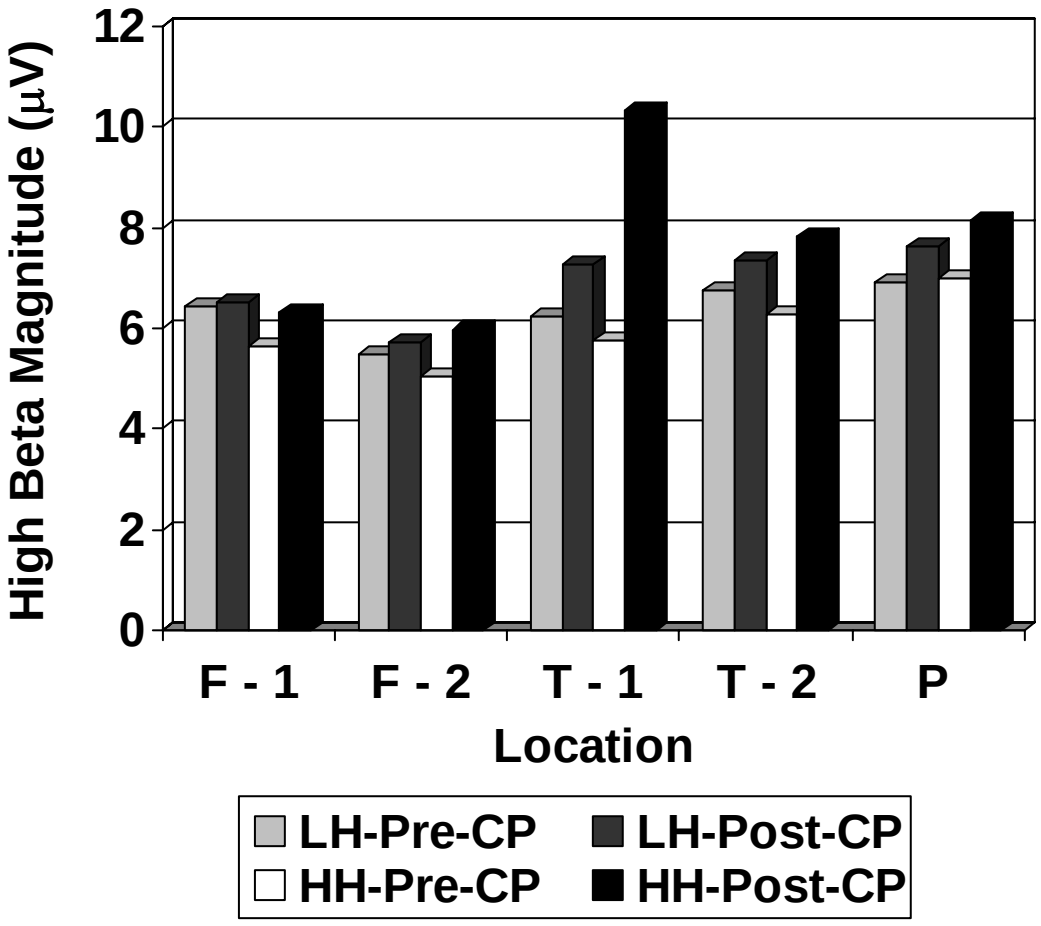


Figure 26. Group differences in high beta magnitude at each location as a function of the cold pressor.

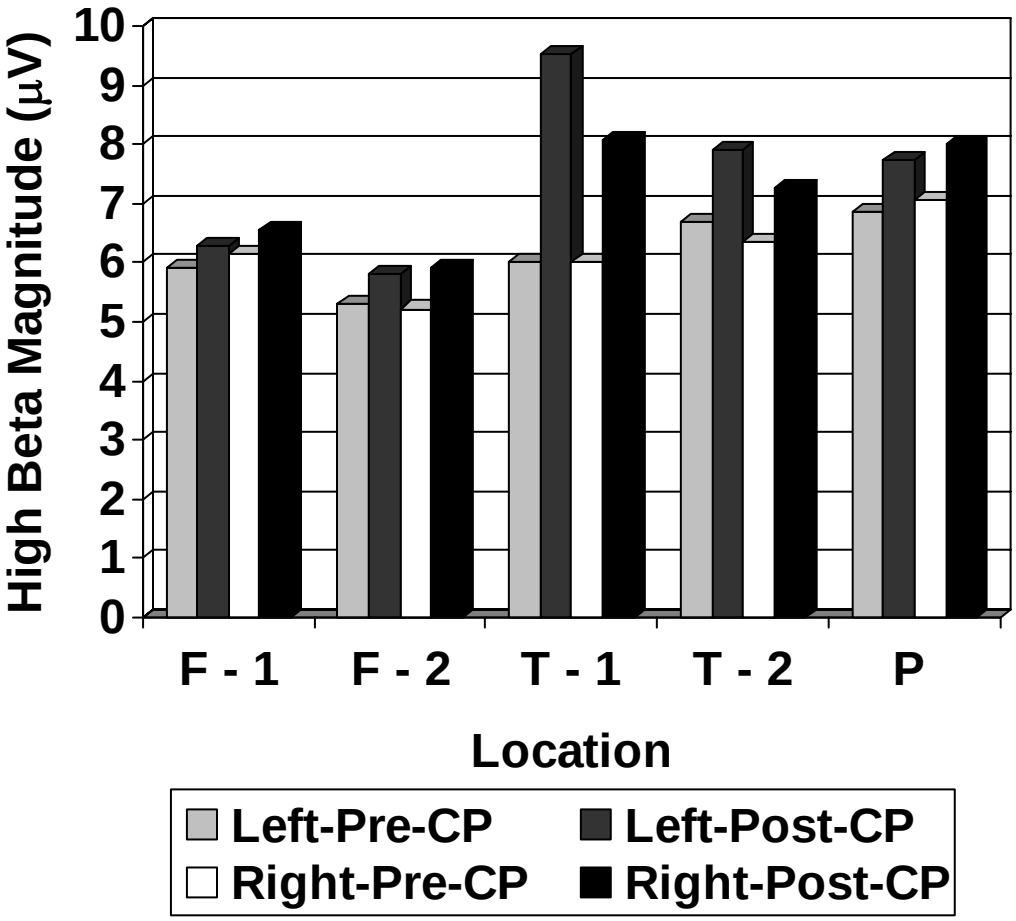


Figure 27. High beta magnitude at each location within the right and left hemispheres as a function of the cold pressor.

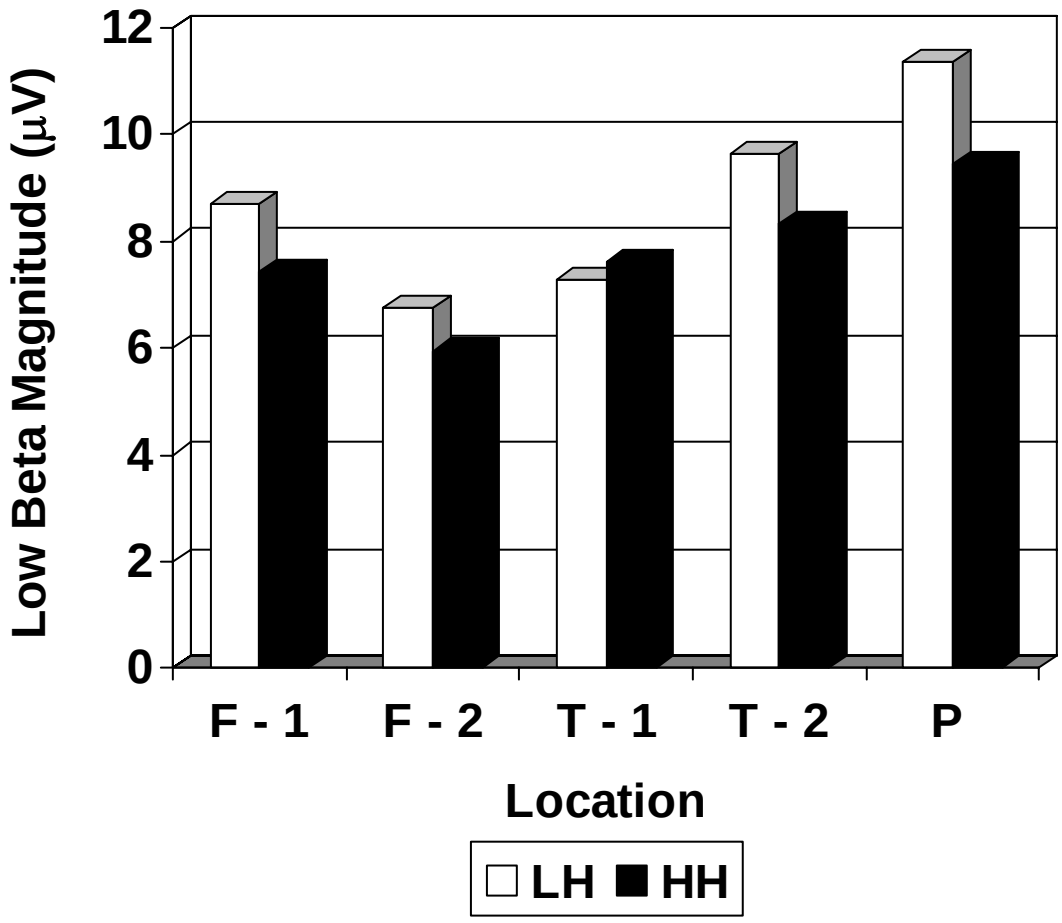


Figure 28. Group differences in low beta magnitude at each location.

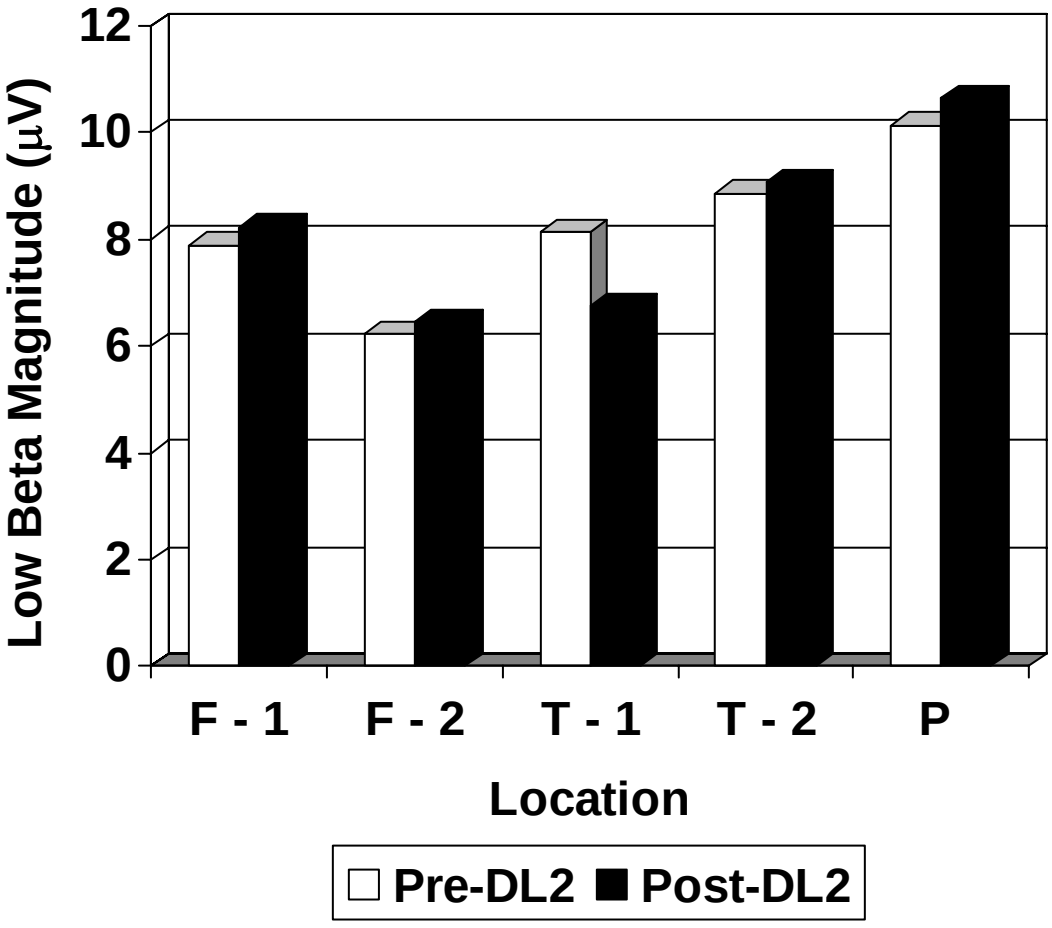


Figure 29. Changes in low beta magnitude at each location as a function of the dichotic listening 2 condition.

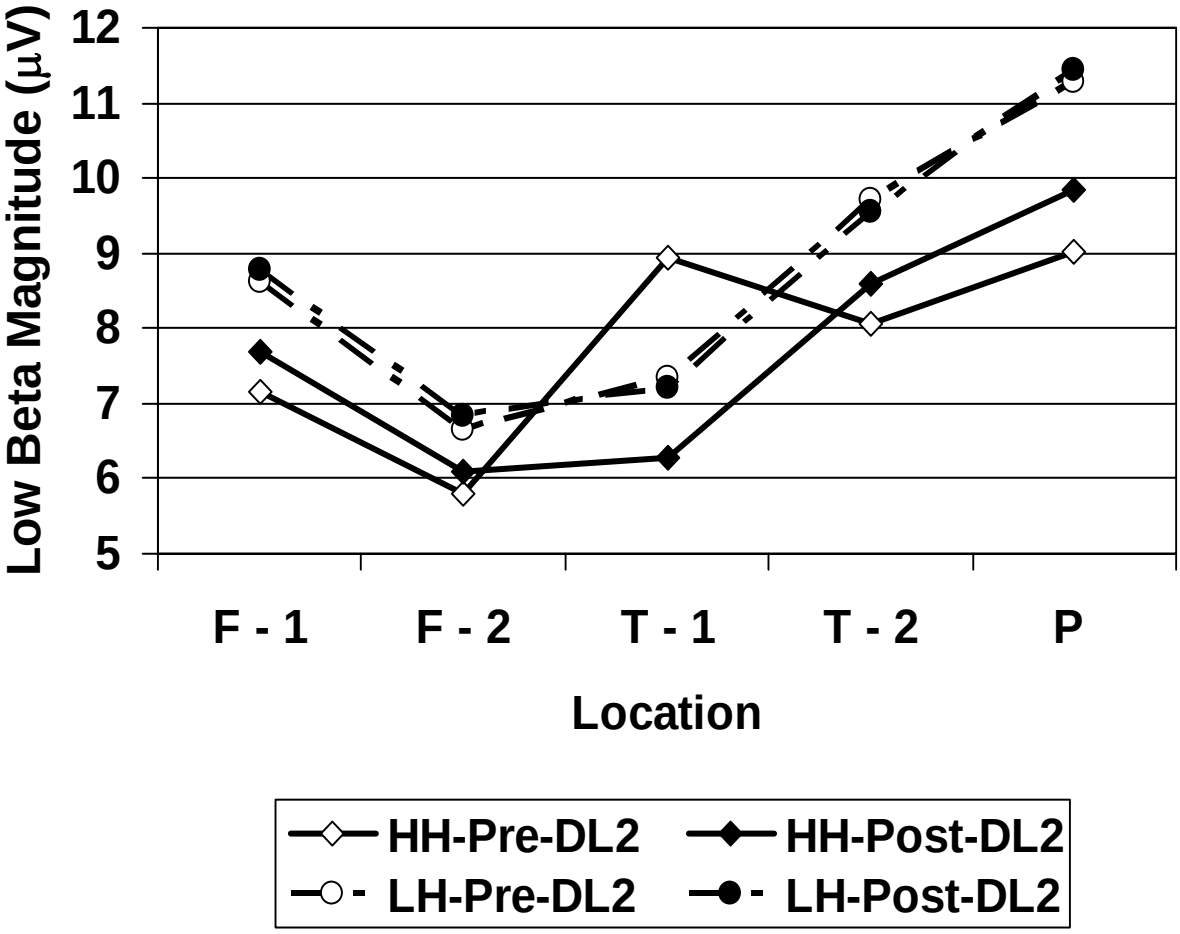


Figure 30. Group differences in low beta magnitude at each location in the pre- and post-dichotic listening 2 conditions.

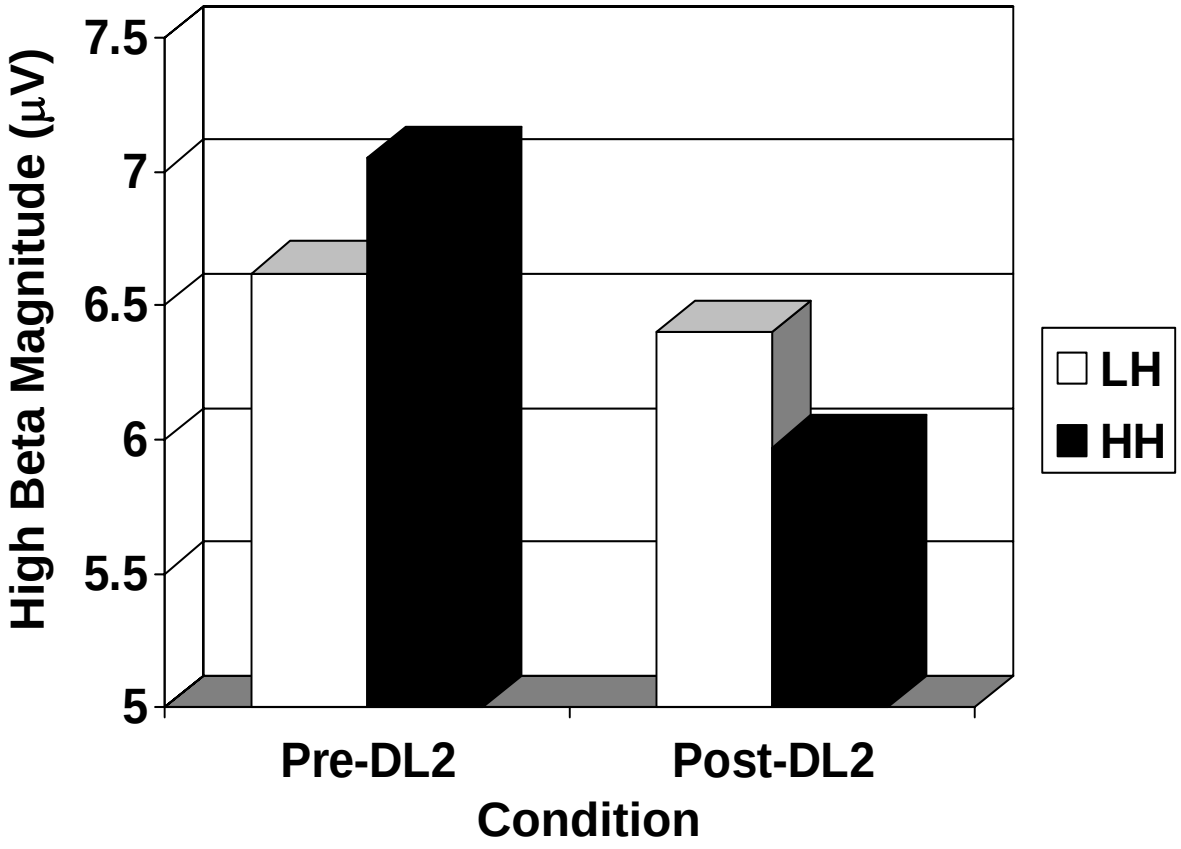


Figure 31. High beta magnitude changes from pre- to post-dichotic listening 2 conditions as a function of group.

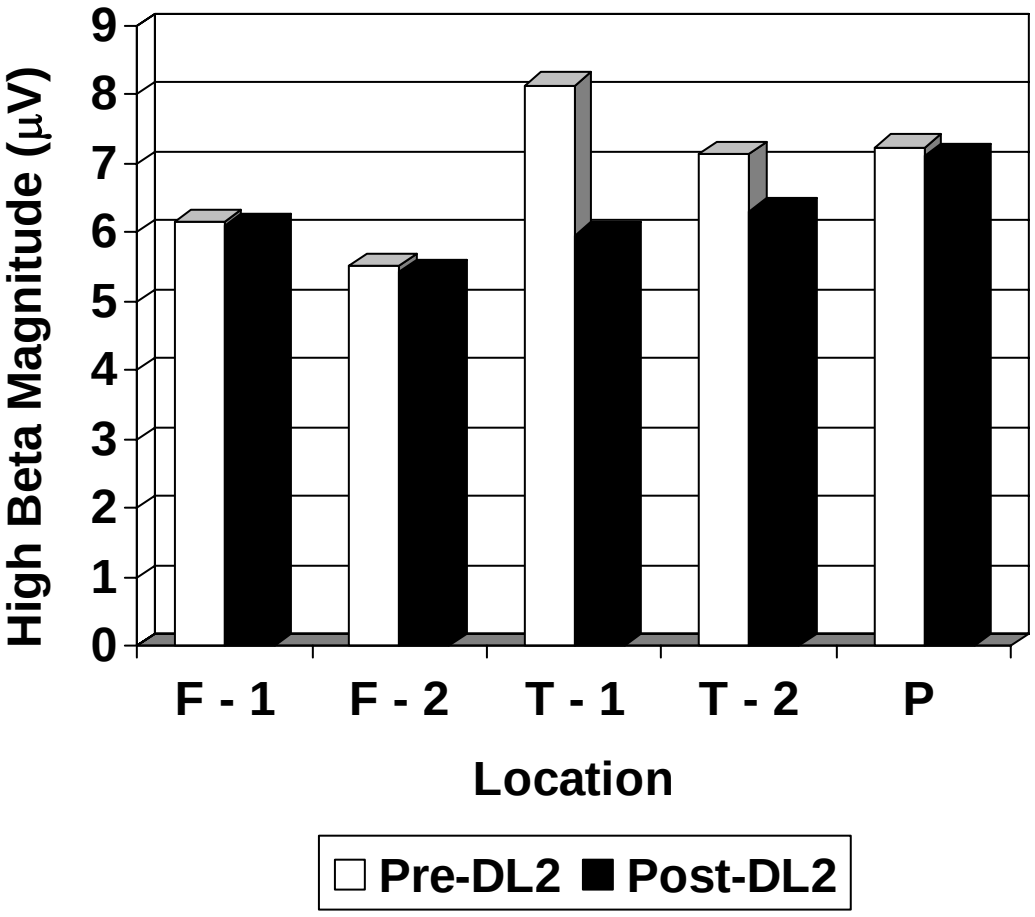


Figure 32. High beta magnitudes at each location in the pre- and post-dichotic listening 2 conditions.

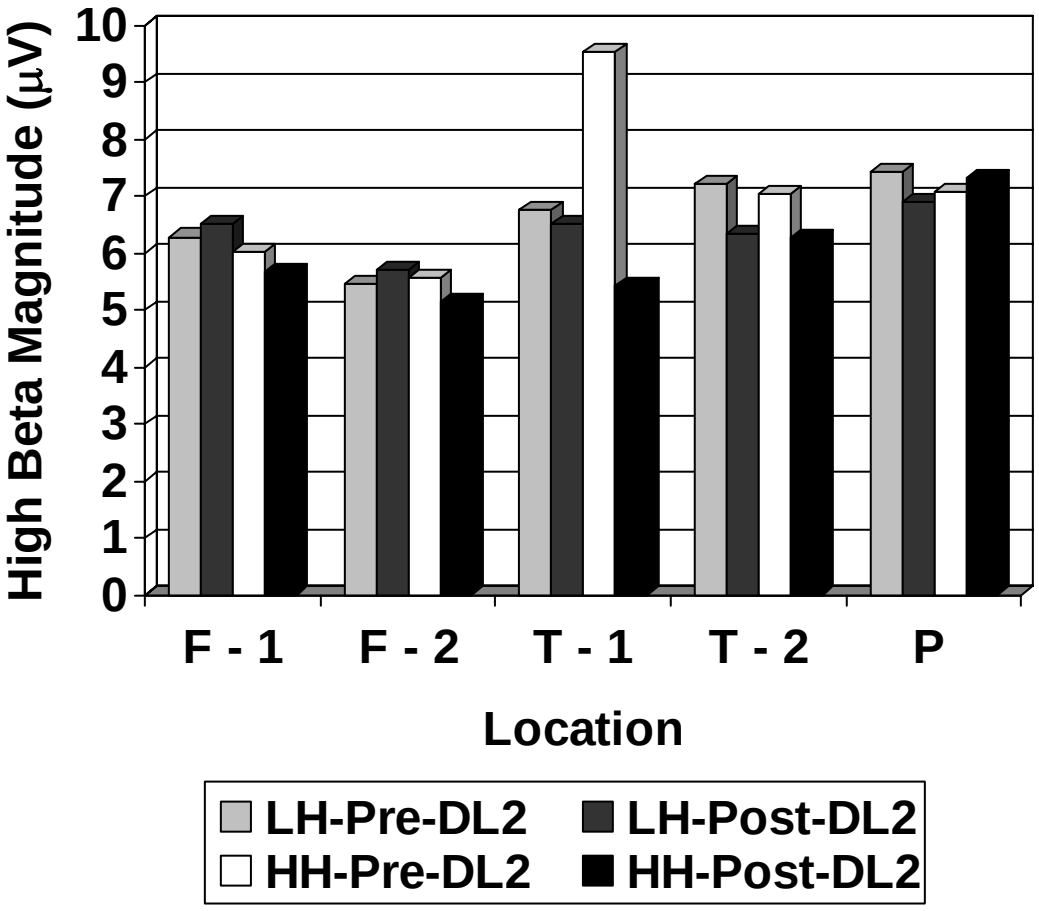


Figure 33. High beta magnitude changes from pre- to post-dichotic listening 2 at each location as a function of group.

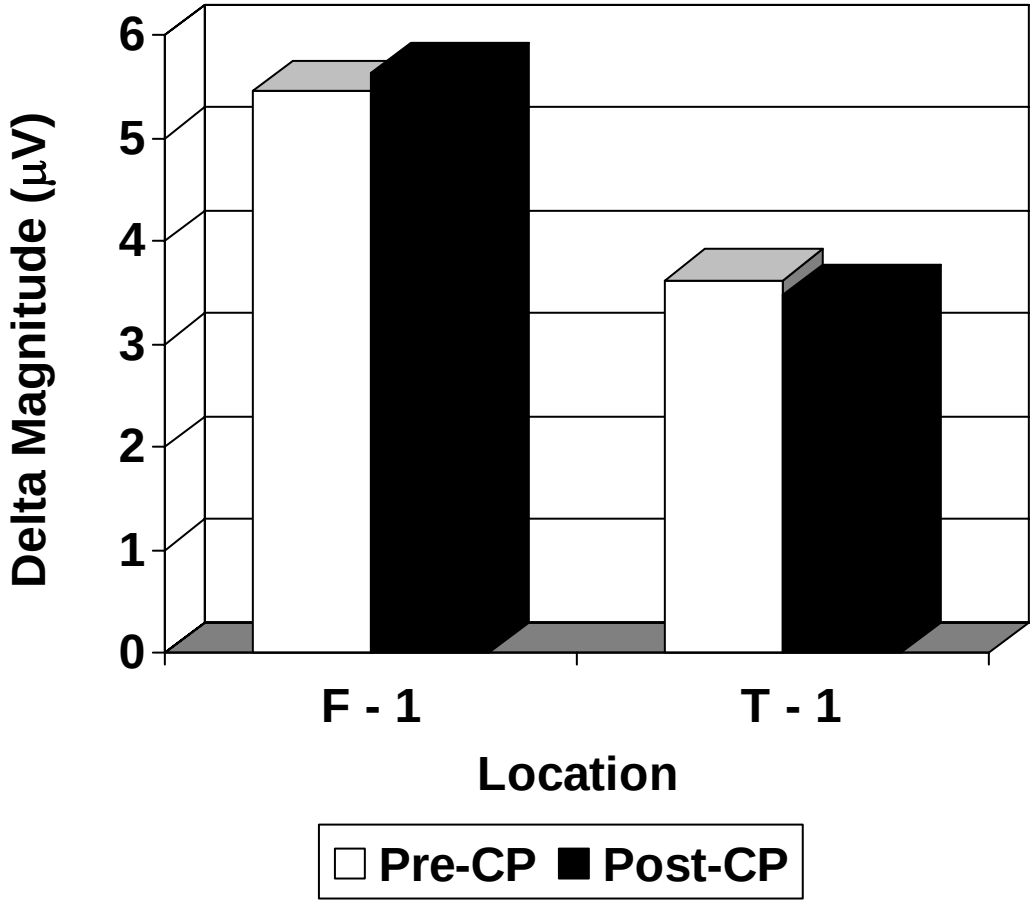


Figure 34. Delta magnitude at the frontal-1 and temporal-1 locations as a function of the cold pressor.

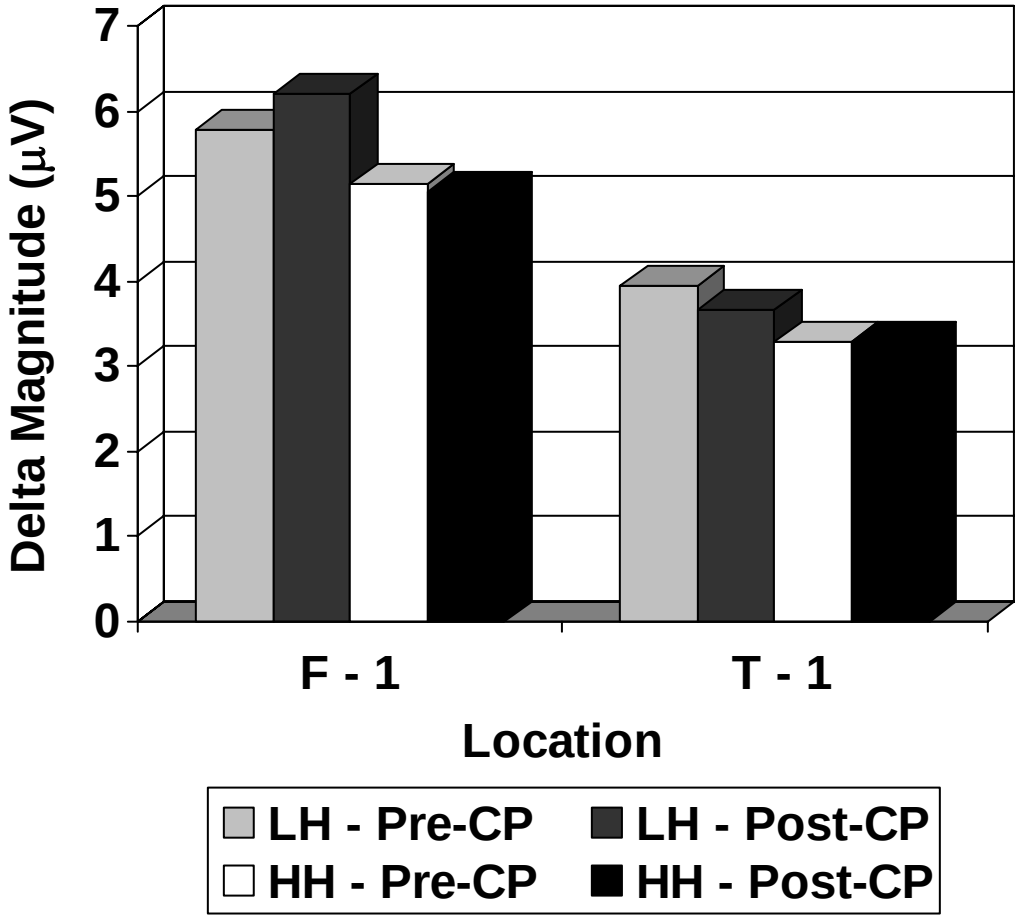


Figure 35. Delta magnitude at the frontal-1 and temporal-1 locations as a function of group and the cold pressor.

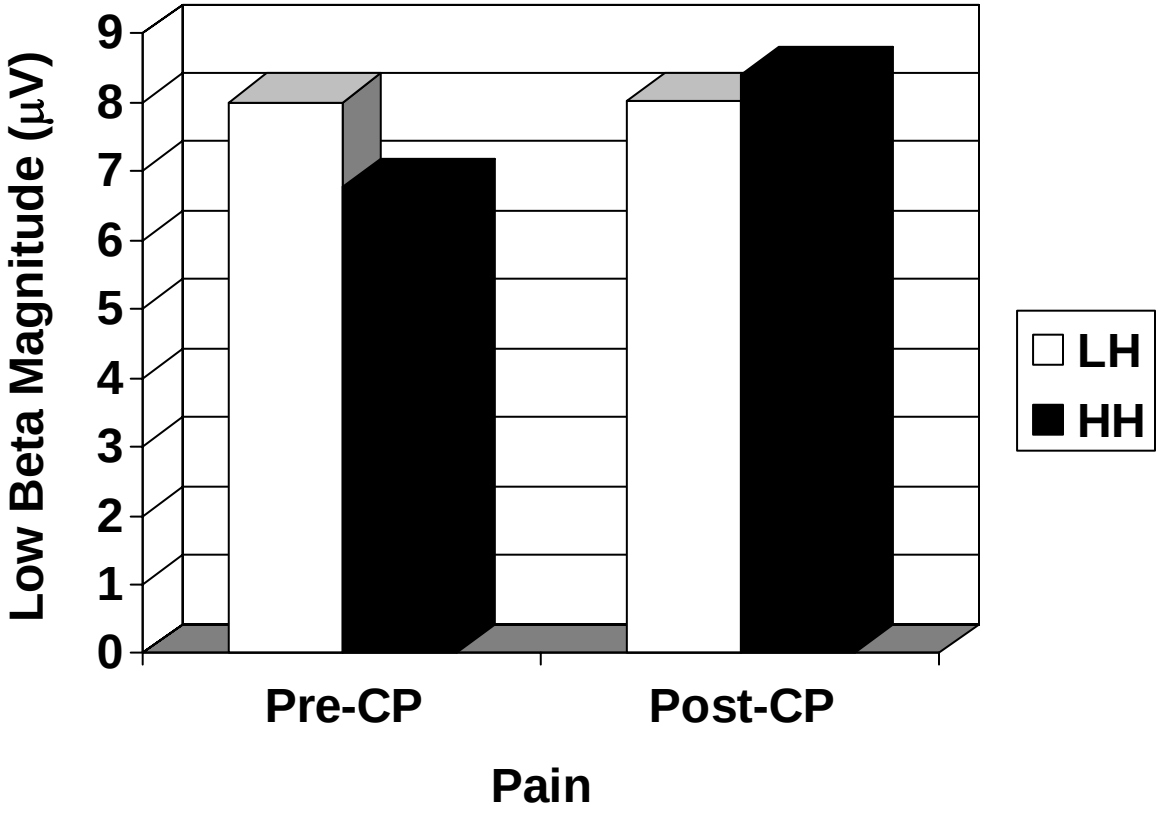


Figure 36. Group differences in low beta magnitude as a function of the cold pressor.

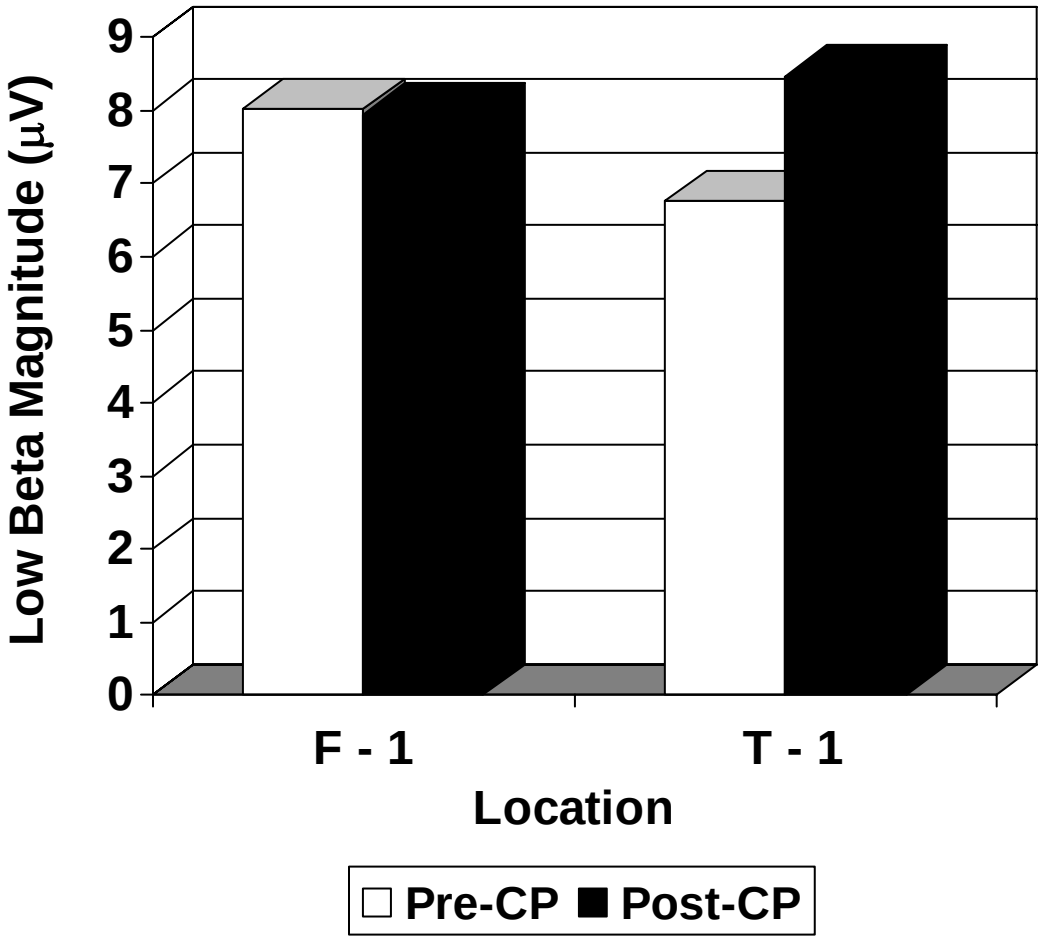


Figure 37. Changes in frontal-1 and temporal-1 low beta magnitude as a function of the cold pressor.

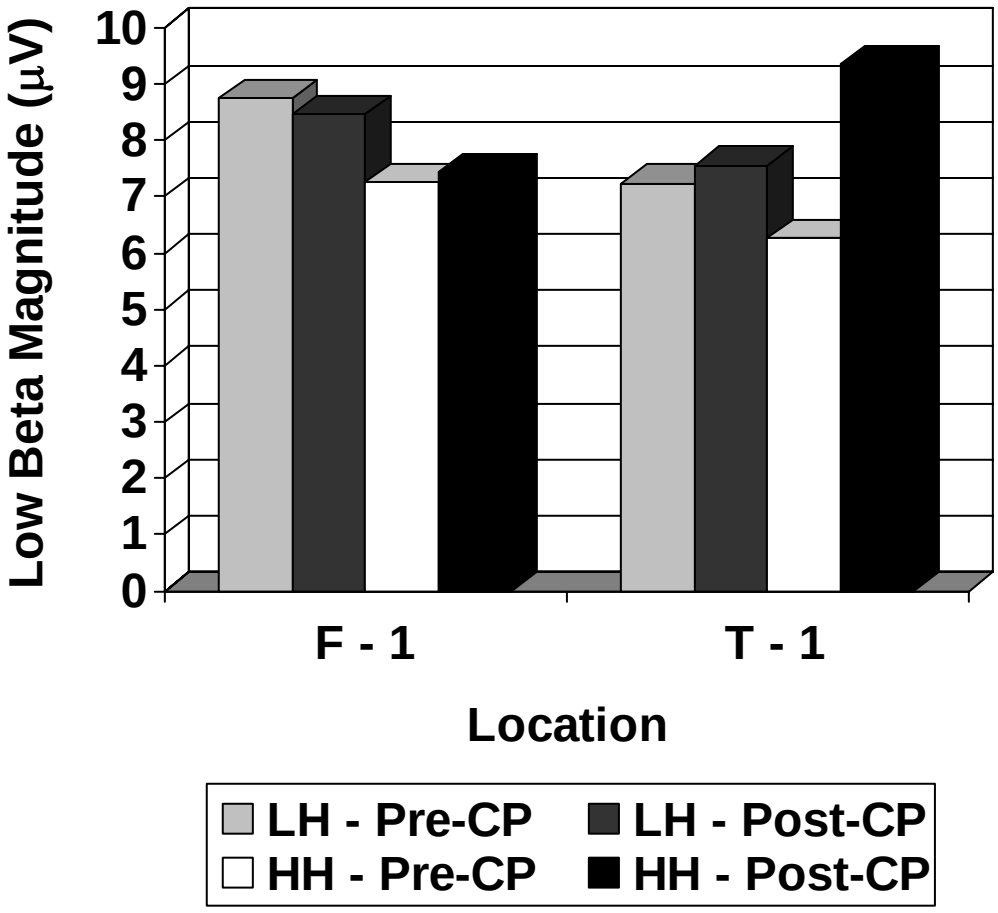


Figure 38. Low beta changes at the frontal-1 and temporal-1 locations as a function of hostility level and the cold pressor.

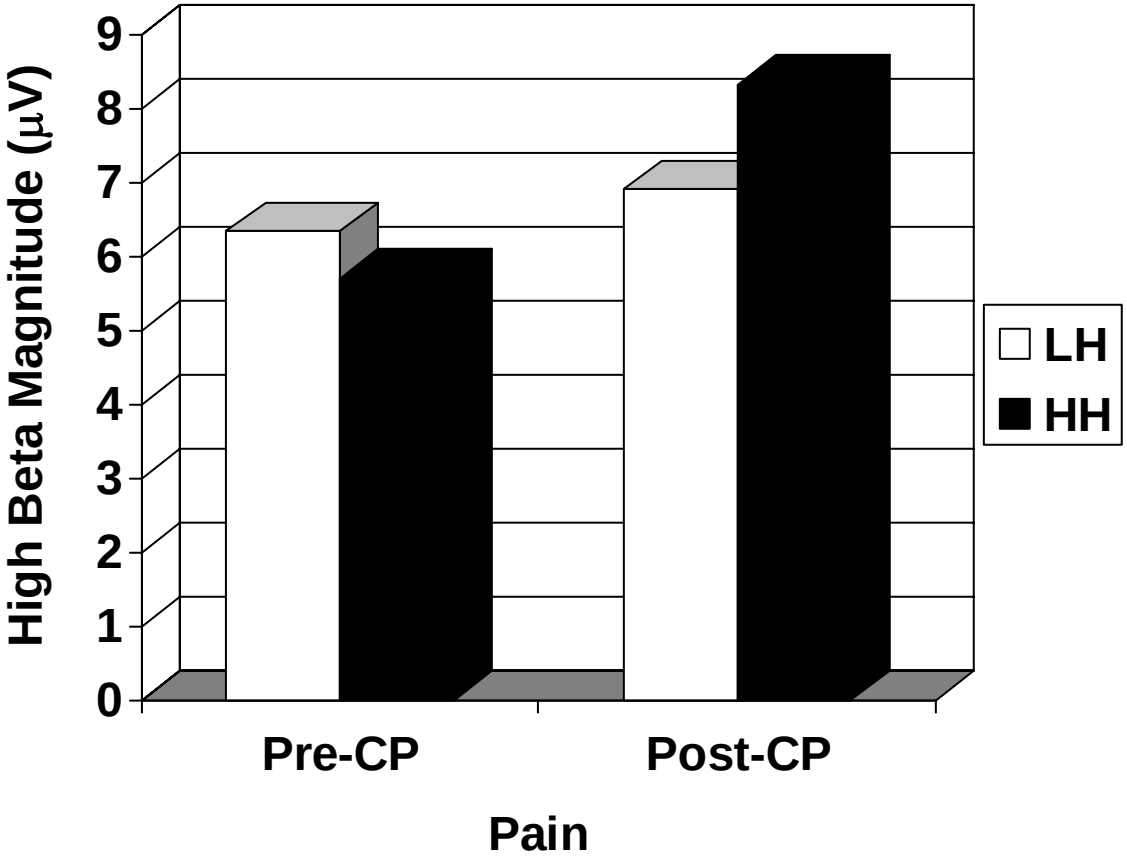


Figure 39. Group differences in high beta magnitude as a function of the cold pressor.

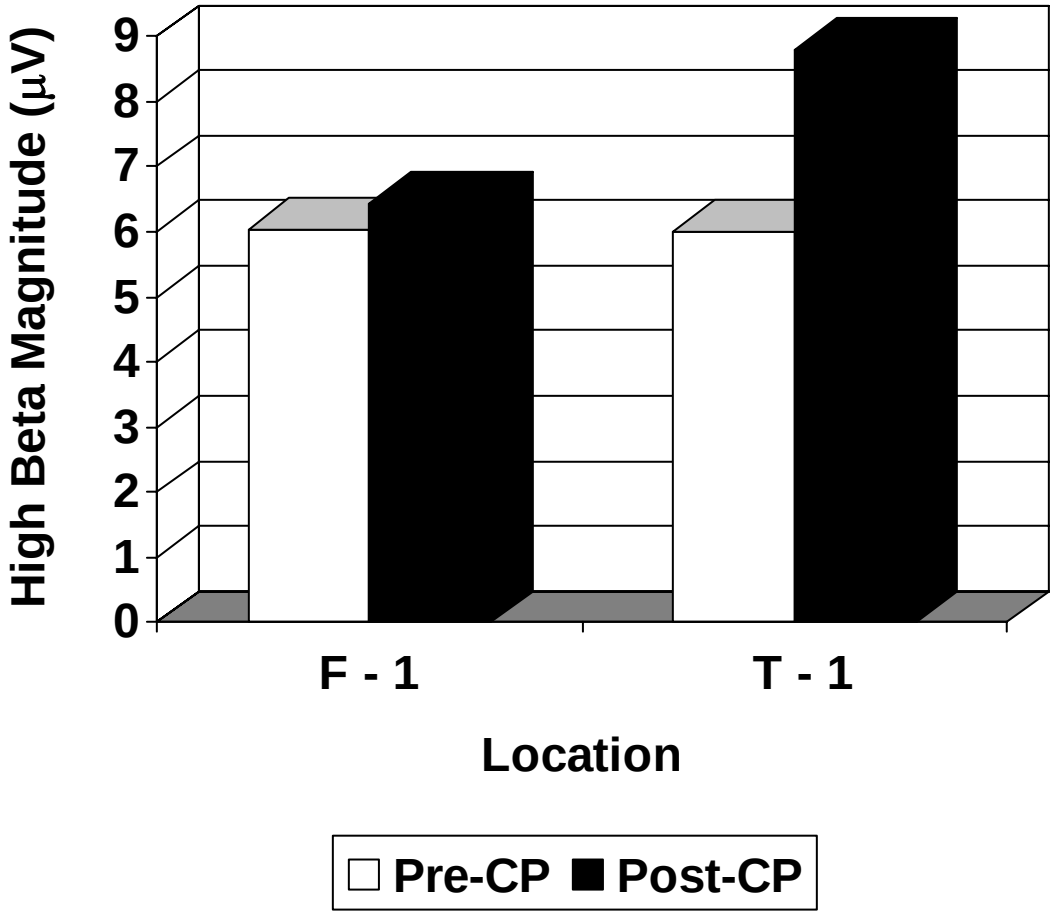


Figure 40. Changes in high beta magnitude at the frontal-1 and temporal-1 locations as a function of the cold pressor.

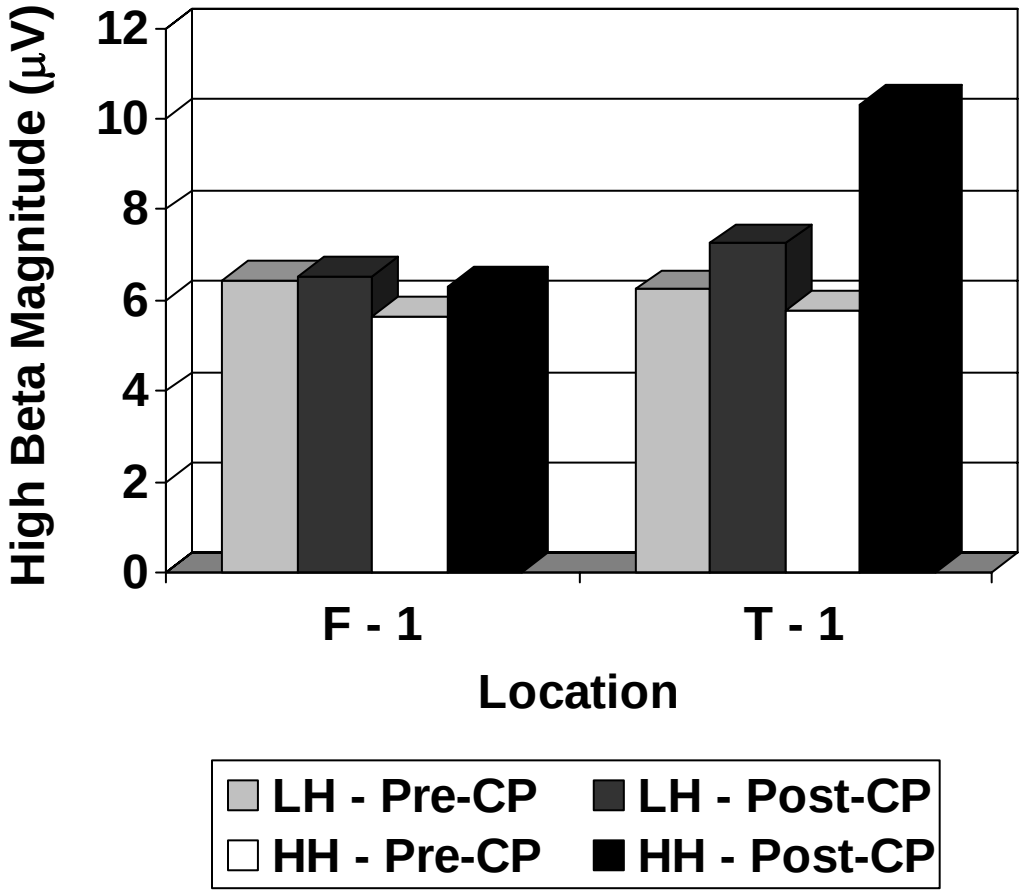


Figure 41. Group differences in high beta magnitude at the frontal-1 and temporal-1 locations as a function of the cold pressor.

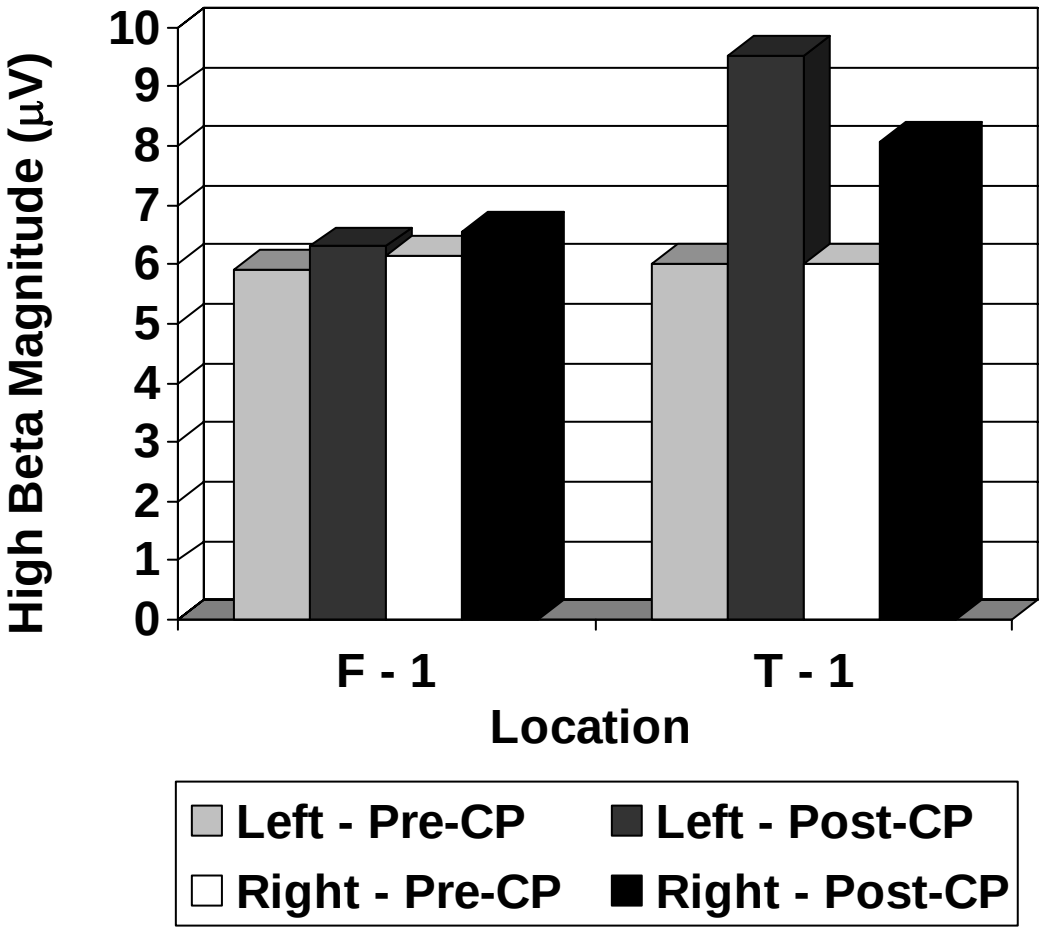


Figure 42. Changes in high beta at the frontal-l and temporal-1 locations within each hemisphere as a function of the cold pressor.

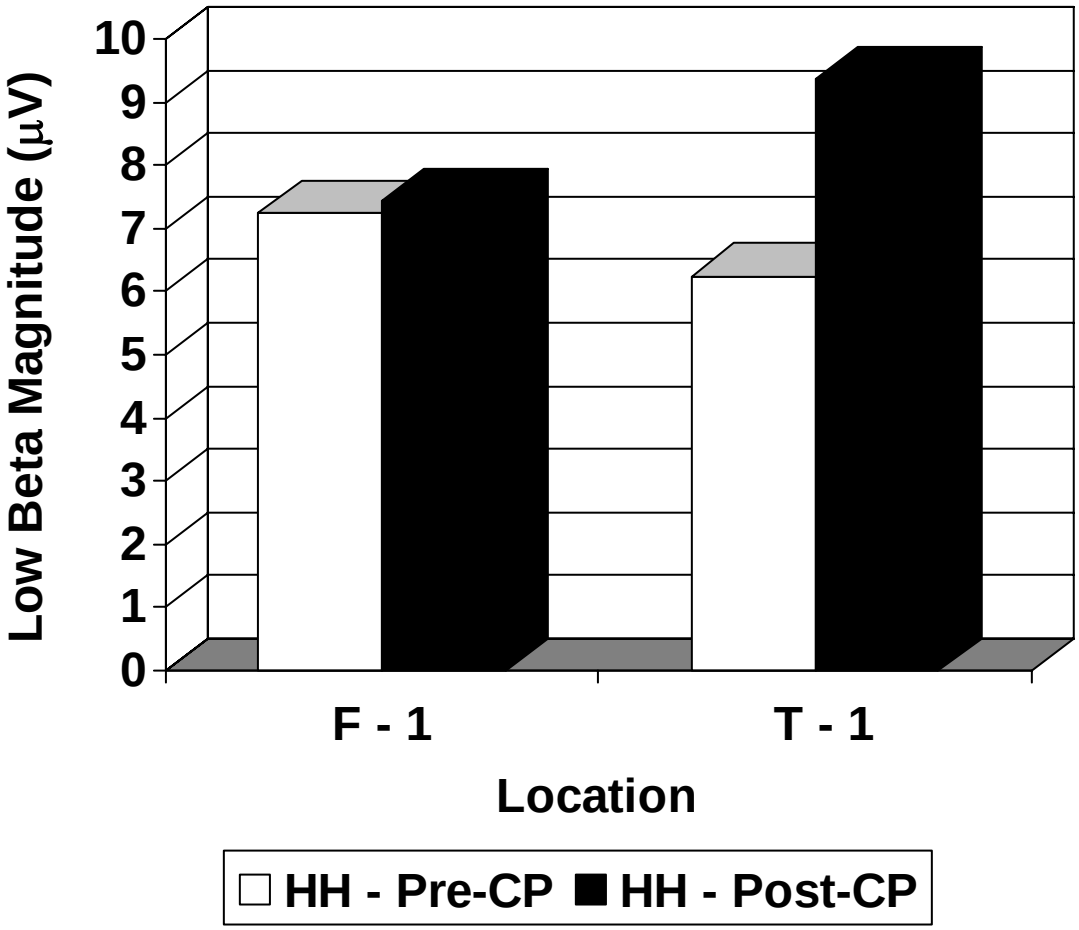


Figure 43. Low beta magnitude changes as a function of location and the cold pressor.

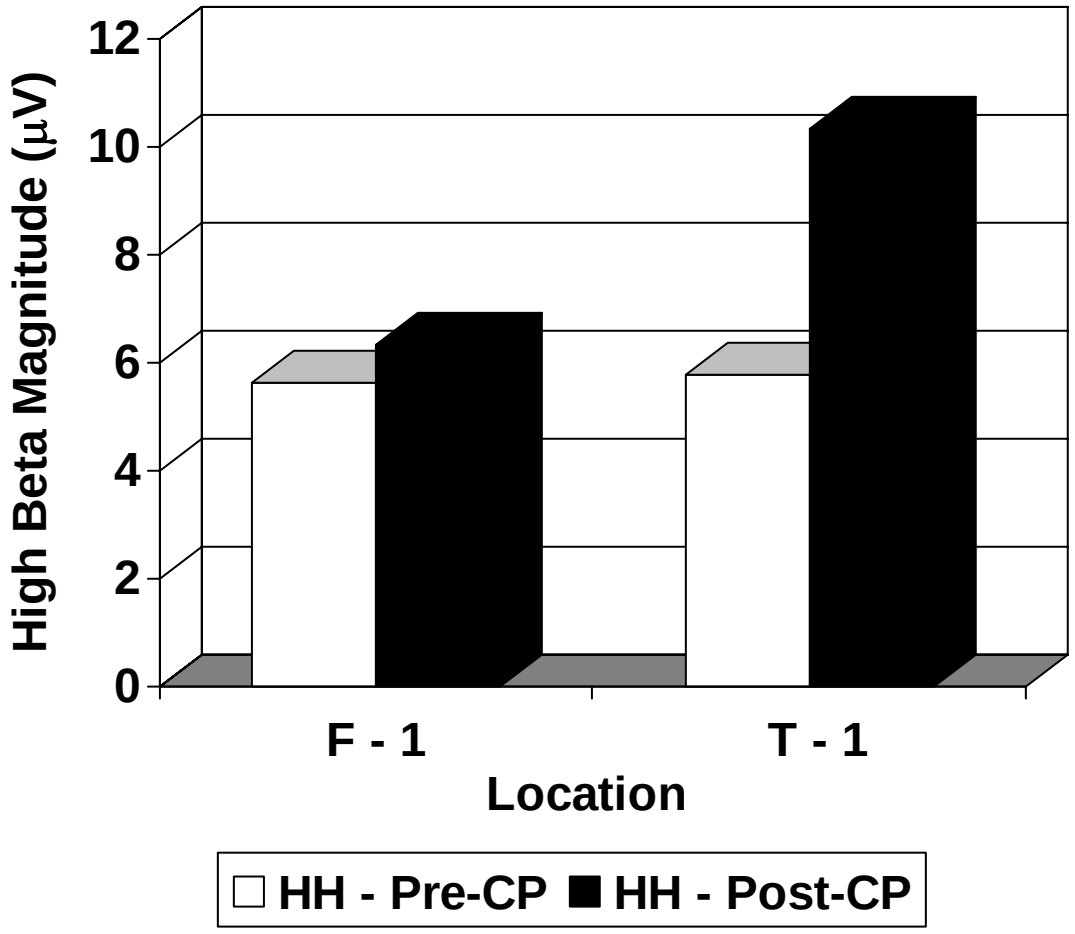


Figure 44. Changes in high beta magnitude as a function of location and pain condition in the high hostile group.

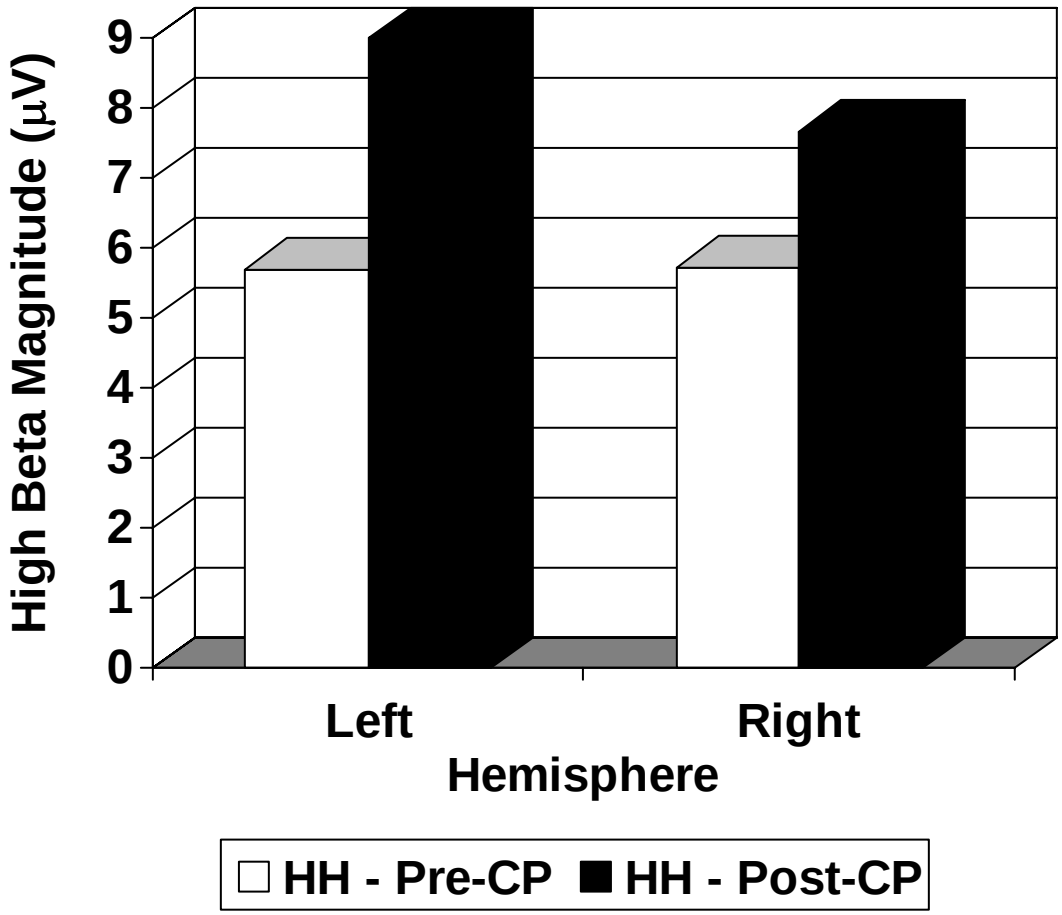


Figure 45. High beta magnitude at the left and right hemispheres in the pre- and post-cold pressor conditions for the high hostile group.

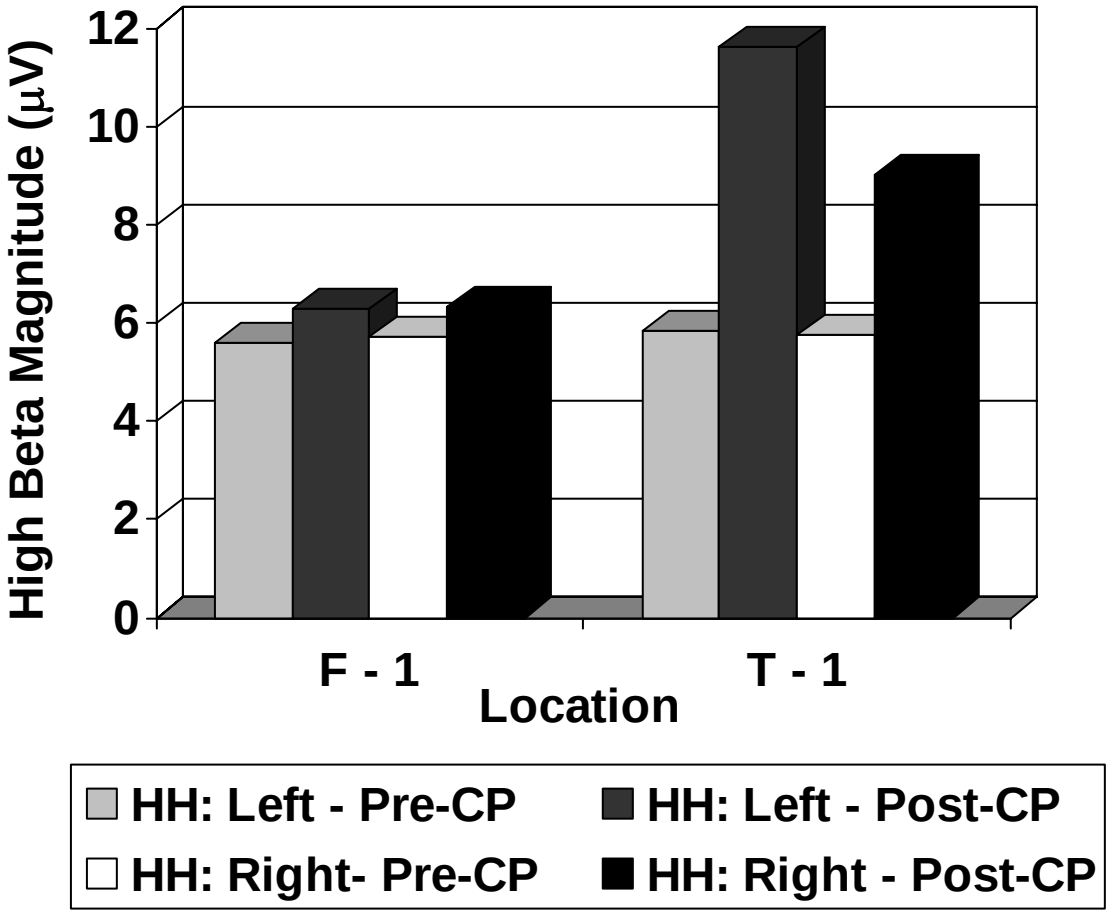


Figure 46. Changes in high beta at each location within the right and left hemispheres a function of the cold pressor for the high hostile group.

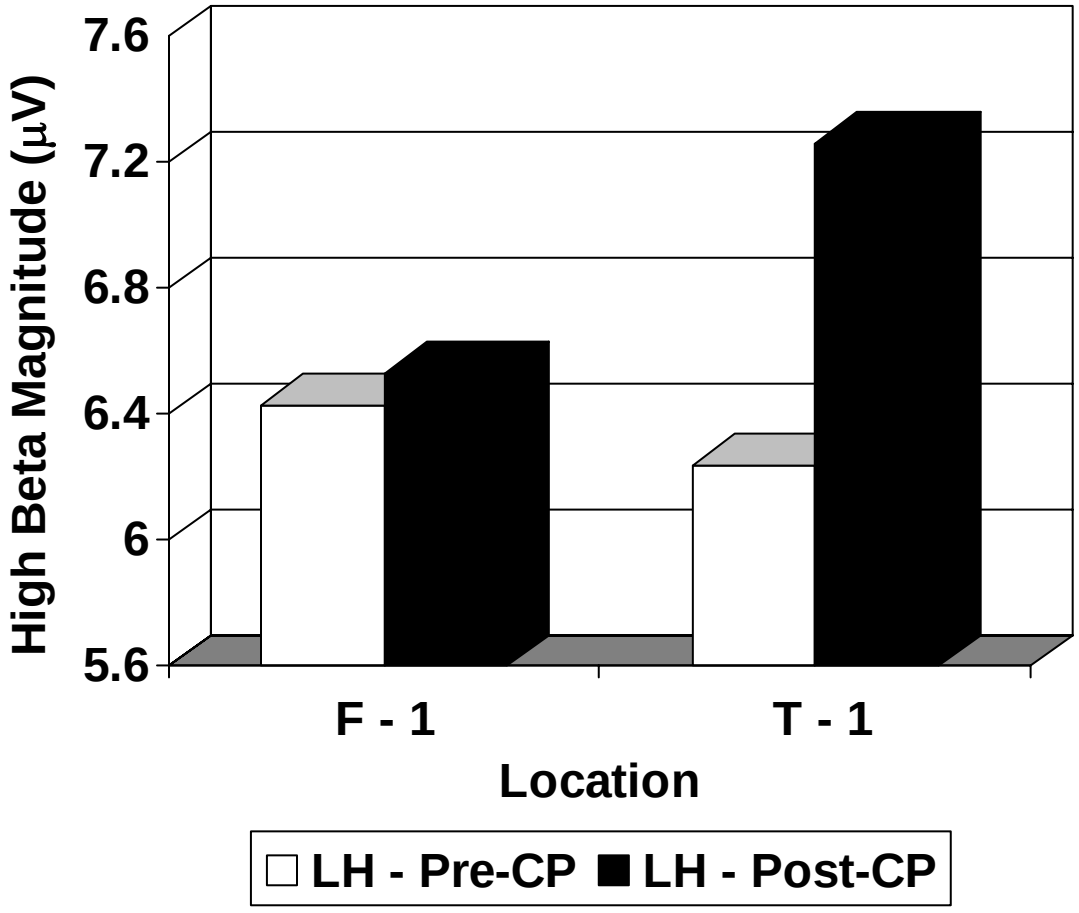


Figure 47. Changes in high beta magnitude for the low hostile group at the frontal-1 and temporal-1 locations as a function of the cold pressor.

Appendix A

Informed Consent (Pre-Screening)

Title: Neuropsychological Effects of Hostility and Pain on Emotion Perception

IRB Approval #: 05-346

1. **PURPOSE OF THE EXPERIMENT:** To learn more about the effects of pain on emotion perception and neuropsychological functioning.

2. **PROCEDURE TO BE FOLLOWED IN THE STUDY:** Participants for the study will be identified after completion questionnaires that will be administered via a secured website. Qualified participants will be contacted and requested to continue based on their questionnaire results.

3. **ANONYMITY OF SUBJECTS AND CONFIDENTIALITY OF THE RESULTS:** Identifying information will be kept confidential and will not be released to anyone other than the individuals directly working on the project without your written consent. The information you provide will have your name removed and is given a number. Information will only be associated with a subject number that will be used during analysis and written reports of the research.

4. **DISCOMFORT AND RISKS FROM PARTICIPATING IN THE STUDY:** Some of the questionnaires may contain material that you find embarrassing or uncomfortable to answer. You may omit any questions that you find embarrassing or uncomfortable. If you have any questions after completing the questionnaires or have any problems associated with the study, you may contact Gina Mollet (231-3235) or Dr. David W. Harrison (231-4422) and they will assist you directly or direct you to appropriate services.

5. **EXPECTED BENEFITS:** Your participation in this project will aid in the understanding of emotion and brain activation. No guarantee of benefits has been made to encourage your participation.

6. **FREEDOM TO WITHDRAW:** You are free to withdraw from the study at any time without penalty and your decision to withdraw will not affect your psychology course grade or application of points.

7. **EXTRA CREDIT COMPENSATION:** You will receive 1 extra credit point for your psychology course by participation in this portion of the study.

8. **USE OF RESEARCH DATA:** The information gathered from this study will be used for scientific and/or educational purposes. The findings may be presented at scientific meetings and/or published and reproduced in professional journals or books. The findings may also be used for other purposes that Virginia Tech's Department of Psychology deems proper in the interest of education, knowledge, and research.

9. **APPROVAL OF RESEARCH:** This project has been approved by the Human Subjects Committee of the Department of Psychology and the Institutional Review Board of Virginia Tech.

10. **PARTICIPANTS PERMISSION:** I have read and understand the above description of the study. I have had the opportunity to ask questions and all have been answered in an appropriate manner. I hereby acknowledge the above and voluntarily give my consent to participate in this study. I realize that I may withdraw at any time without penalty and that I may contact one of the people listed below at any time if I have questions regarding the study.

Gina A. Mollet Primary Researcher	231-3235
David W. Harrison, Ph.D. Faculty Advisor	231-4422
Jack Finney, Ph.D. Department Chair, Department of Psychology	231-6670
David Moore, Ph.D. Institutional Review Board Chair, Research Division	231-4991

Participant's Signature: _____ Date: _____

Participant's ID: _____ Participant's Telephone #:_____

Appendix B

Medical History Questionnaire

Name: _____ Age: _____

Race/Ethnic Origin: _____ Average Grades: _____

Please list all of your past and present surgeries, medical procedures, major diseases/illnesses, and medical conditions (include dates when possible):

_____

_____

_____

Please circle the appropriate response and explain any "Yes" responses below.

1. Have you ever been knocked unconscious for more than 5 minutes?	Yes	No
2. Have you ever had a head injury?	Yes	No
3. Have you ever had a stroke or aneurysm?	Yes	No
4. Have you ever or are you currently experiencing any fainting spells or black outs?	Yes	No
5. Are you able to read, write, and spell without difficulty?	Yes	No
6. Have you ever been diagnosed with any learning disabilities?	Yes	No
7. Have you ever been diagnosed with memory loss, cognitive impairment, or thinking problems, or are you experiencing such problems at present?	Yes	No
8. Do you have a past or present history of epilepsy or seizures?	Yes	No
9. Do you have any paralysis?	Yes	No
10. Have you ever had neurological surgery?	Yes	No
11. Do you have a history of cardiac or respiratory arrest or lack of oxygen (e.g., hypoxia)?	Yes	No
12. Do you have a past or present history of neurological disorders (e.g., Parkinson's Disease, Multiple Sclerosis, or Huntington's Disease)?	Yes	No
13. Do you have a past or present history of alcohol or drug problems?	Yes	No

14. Are you currently consuming three or more alcoholic beverages per day?	Yes	No
15. Past or present diagnosed psychological/psychiatric problems?	Yes	No
16. Have you ever received psychological/psychiatric counseling?	Yes	No
17. Have you ever been hospitalized in a psychiatric facility/hospital?	Yes	No
18. Are you currently taking any prescription medications?	Yes	No
19. Do you currently have any diagnosed medical conditions or illnesses?	Yes	No
20. Do you have any uncorrected vision problems?	Yes	No
21. Do you have an uncorrected hearing impairment?	Yes	No
22. Do you have any problems or pain with movement (e.g., severe hand, arm, or shoulder pain with movement)?	Yes	No
23. At present, do you have any uncontrolled metabolic disorders such thyroid conditions or diabetes?	Yes	No
25. Have you ever been diagnosed with arthritis?	Yes	No
26. Do you have Raynaud's disease?	Yes	No
27. Are you currently experiencing any cardiovascular disorders or have you previously been diagnosed with any cardiovascular disorders?	Yes	No
28. Do you now or have you ever had a heart murmur?	Yes	No
29. Do you now or have you ever had pressure, pain or tightness in the chest?	Yes	No
30. Do you now or have you ever had pressure, pain or tightness in the chest brought on by exertion?	Yes	No
31. Have you ever been diagnosed with asthma?	Yes	No
32. Do you have a history of child abuse?	Yes	No
33. Do you have a history of broken bones?	Yes	No
34. Do you smoke?	Yes	No
35. Do you consume caffeine (soda, coffee, tea, etc.)?	Yes	No

36. Do you consume alcohol? Yes No

Please explain any "Yes" responses:

_____
_____
_____

Appendix C

Coren, Porac, and Duncan Laterality Questionnaire

Circle the appropriate number after each item:	Right	Left	Both
With which hand would you throw a ball to hit a target?	1	-1	0
With which hand do you draw?	1	-1	0
With which hand do you use an eraser on paper?	1	-1	0
With which hand do you remove the top card when dealing?	1	-1	0
With which foot do you kick a ball?	1	-1	0
If you wanted to pick up a pebble with your toes, which foot would you use?	1	-1	0
If you had to step up onto a chair, which foot would you place on the chair first?	1	-1	0
Which eye would you use to peep through a keyhole?	1	-1	0
If you had to look into a dark bottle to see how full it was, which eye would you use?	1	-1	0
Which eye would you use to sight down a rifle?	1	-1	0
If you wanted to listen to a conversation going on behind a closed door, which ear would you place against the door?	1	-1	0
If you wanted to listen to someone's heartbeat, which ear would you place against their chest?	1	-1	0
Into which ear would you place the earphone of a transistor radio?	1	-1	0

# of Right + # of Left = Total Score
_____ + _____ = _____

Is your mother left or right hand dominant? _____

Is your father left or right hand dominant? _____

Appendix D

Data Sheet

Participant #: _____ Date: _____ Trial #:_____

Instructions

This task assesses your ability to hear emotions. On each trial you will hear two words pronounced in two different emotional tones (one at each ear). Your task is to identify which emotions you hear. Respond by circling the appropriate emotions for each trial. For example, if you hear the emotion of anger in one ear and the emotion of happiness in the other ear, you would circle "angry" and "happy." There will be 6 practice trials followed by 72 experimental trials. You will get a 40 second break after the first 36 trials. Ask the experimenter if you have any questions.

Practice Trials

1.	angry	happy	neutral
2.	angry	happy	neutral
3.	angry	happy	neutral
4.	angry	happy	neutral
5.	angry	happy	neutral
6.	angry	happy	neutral

Go to the next page for the experimental trials

Experimental Trials

1.	angry	happy	neutral
2.	angry	happy	neutral
3.	angry	happy	neutral
4.	angry	happy	neutral
5.	angry	happy	neutral
6.	angry	happy	neutral
7.	angry	happy	neutral
8.	angry	happy	neutral
9.	angry	happy	neutral
10.	angry	happy	neutral
11.	angry	happy	neutral
12.	angry	happy	neutral
13.	angry	happy	neutral
14.	angry	happy	neutral
15.	angry	happy	neutral
16.	angry	happy	neutral
17.	angry	happy	neutral
18.	angry	happy	neutral
19.	angry	happy	neutral
20.	angry	happy	neutral
21.	angry	happy	neutral
22.	angry	happy	neutral

23.	angry	happy	neutral
24.	angry	happy	neutral
25.	angry	happy	neutral
26.	angry	happy	neutral
27.	angry	happy	neutral
28.	angry	happy	neutral
29.	angry	happy	neutral
30.	angry	happy	neutral
31.	angry	happy	neutral
32.	angry	happy	neutral
33.	angry	happy	neutral
34.	angry	happy	neutral
35.	angry	happy	neutral
36.	angry	happy	neutral
37.	angry	happy	neutral
38.	angry	happy	neutral
39.	angry	happy	neutral
40.	angry	happy	neutral
41.	angry	happy	neutral
42.	angry	happy	neutral
43.	angry	happy	neutral
44.	angry	happy	neutral
45.	angry	happy	neutral

46.	angry	happy	neutral
47.	angry	happy	neutral
48.	angry	happy	neutral
49.	angry	happy	neutral
50.	angry	happy	neutral
51.	angry	happy	neutral
52.	angry	happy	neutral
53.	angry	happy	neutral
54.	angry	happy	neutral
55.	angry	happy	neutral
56.	angry	happy	neutral
57.	angry	happy	neutral
58.	angry	happy	neutral
59.	angry	happy	neutral
60.	angry	happy	neutral
61.	angry	happy	neutral
62.	angry	happy	neutral
63.	angry	happy	neutral
64.	angry	happy	neutral
65.	angry	happy	neutral
66.	angry	happy	neutral
67.	angry	happy	neutral
68.	angry	happy	neutral

69.	angry	happy	neutral
70.	angry	happy	neutral
71.	angry	happy	neutral
72.	angry	happy	neutral

Appendix E

Informed Consent (Testing Day)

Title: Neuropsychological Effects of Hostility and Pain on Emotion Perception

IRB Approval #: 05-346

1. **PURPOSE OF THE EXPERIMENT:** The purpose of this experiment is to measure brain and behavior changes in response to pain. The experiment will use electroencephalography, heart rate, and blood pressure to examine the neuropsychophysiological changes that occur in response to pain. A dichotic listening task will be used to examine emotion perception.

2. **PROCEDURE TO BE FOLLOWED IN THE STUDY:** You will be asked to complete this consent. You will then be connected to the electroencephalogram (EEG). You will then be asked to relax for three to four minutes and baseline readings will be taken. Following this, you will be asked to complete a dichotic listening task. After completion, you will be asked to place your hand in ice water. Next, you will be asked to complete the dichotic listening task a second time. EEG, will be monitored throughout the experiment. You are required to appear only once for the experiment, which should last no longer than 90 minutes. At the completion of the experiment you will be disconnected from the EEG and allowed to ask any questions you should have relevant to the experiment.

3. **ANONYMITY OF SUBJECTS AND CONFIDENTIALITY OF THE RESULTS:** Identifying information will be kept confidential and will not be released to anyone other than the individuals directly working on the project without your written consent. The information you provide will have your name removed and is given a number. Information will only be associated with a subject number that will be used during analysis and written reports of the research.

4. **DISCOMFORT AND RISKS FROM PARTICIPATING IN THE STUDY:** Participation in EEG data collection may produce discomfort. The placement of ear electrodes may pinch your ears and be potentially painful. Wearing the electro-cap may produce pressure around your head that is uncomfortable and may produce possible abrasions on your forehead. Other risks include slight discomfort from the abrasive cleaning and remote risk of infection. To ensure your safety from infection, all electrodes, electrode caps, and other equipment contacting the body will be thoroughly sanitized before each experimental session. The experimenters will also wear latex gloves while applying electrode caps and individual electrodes. These gloves will be thrown away immediately afterwards. Electrode gel injectors and blunt needles for attaching to the injector come in enclosed packages and are sanitized by the distributor. They will also be discarded after use. All containers and materials will be cleaned and/or disposed of following procedures approved for BioHazard disposition. Container(s) for the injectors and needles will be clearly labeled “BioHazard” and will be separate from other laboratory waste.

Cotton swabs are used to apply the cleanser to the skin (other than scalp) and disposed of immediately afterwards. Electrodes are lightly scrubbed with Ivory soap to remove any gel and then sterilized, according to standardized procedures with a solution purchased from the manufacturer. Given these procedures, the likelihood of transmission of human diseases in quite minimal if not nonexistant. Using standardized procedures, your skin is cleansed to reduce impedance from natural oils at each electrode site. These cleansings are accomplished with the use of NuPrep, a commonly used antiseptic cleanser accepted in EEG research laboratories in the US and Europe. Such procedures are recommended by the Electrocap company and follow US government hospital regulations. Since those ho have skin allergies have been shown to rarely experience a skin rash from NuPrep, you will be asked if you have ever had any skin allergies. If so, only rubbing alcohol will be used to clean the skin. The researchers will monitor any recommended changes and update procedures when necessary.

Minor discomfort may also be experienced when you submerge your hand in the ice water; however, this should not last more than a few minutes.

Safeguards that will be used to minimize your discomfort include the continuous opportunity to terminate the experiments without penalty to yourself (as in losing extra credit points) should you ever feel uncomfortable. A thorough debriefing discussing any issues that may be of concern to you will also be provided at the end of the experiment. At that time you will be given ample opportunity to ask any additional questions about the research that you feel were inadequately addressed by the debriefing. Some of the questionnaires may contain material that you find embarrassing or uncomfortable to answer. You may omit any questions that you find embarrassing or uncomfortable. Additionally, you may experience some mild to moderate discomfort during the course of this experiment. This should be relatively minor discomfort lasting no longer than a few minutes. If you have any questions after leaving the experiment or have any problems associated with the study, you may contact Gina Mollet (231-3235) or Dr. David W. Harrison (231-4422) and they will assist you directly or direct you to appropriate services.

5. **EXPECTED BENEFITS:** Your participation in this project will aid in the understanding of emotion and brain activation. You may also benefit from learning how professional neuropsychological research is conducted. No guarantee of benefits has been made to encourage your participation.

6. **FREEDOM TO WITHDRAW:** You are free to withdraw from the study at any time without penalty and your decision to withdraw will not affect your psychology course grade or application of points.

7. **EXTRA CREDIT COMPENSATION:** You will receive 2 extra credit points for your psychology course by participation in this portion of the study.

8. **USE OF RESEARCH DATA:** The information gathered from this study will be used for scientific and/or educational purposes. The findings may be presented at scientific meetings and/or published and reproduced in professional journals or books. The findings may also be used for other purposes that Virginia Tech's Department of Psychology deems proper in the interest of education, knowledge, and research.

9. **APPROVAL OF RESEARCH:** This project has been approved by the Human Subjects Committee of the Department of Psychology and the Institutional Review Board of Virginia Tech.

10. **PARTICIPANTS PERMISSION:** I have read and understand the above description of the study. I have had the opportunity to ask questions and all have been answered in an appropriate manner. I hereby acknowledge the above and voluntarily give my consent to participate in this study. I realize that I may withdraw at any time without penalty and that I may contact one of the people listed below at any time if I have questions regarding the study.

Gina A. Mollet Primary Researcher	231-3235
David W. Harrison, Ph.D. Faculty Advisor	231-4422
Jack Finney, Ph.D. Department Chair, Department of Psychology	231-6670
David Moore, Ph.D. Institutional Review Board Chair, Research Division	231-4991

Participant's Signature: _____ Date: _____

Participant's ID: _____ Participant's Telephone #:_____

Appendix F

Cold Pressor Assessment

Please circle the most appropriate answer to each of the following questions:

1. How stressful was putting your hand in the ice water?

(1 = not stressful at all, 4 = moderately stressful, 7 = extremely stressful)

1 2 3 4 5 6 7

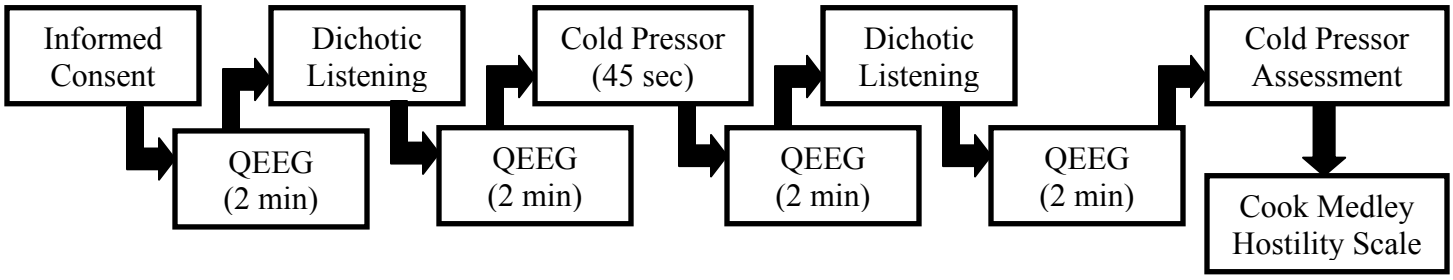
2. What level of pain did you experience when you put your hand in the ice water?

(1 = not painful at all, 4 = moderately painful, 7 = extremely painful)

1 2 3 4 5 6 7

Appendix G

Flow Chart of Procedures



Virginia Tech
VIRGINIA POLYTECHNIC INSTITUTE
AND STATE UNIVERSITY

Institutional Review Board

Dr. David M. Moore
IRB (Human Subjects)Chair
Assistant Vice President for Research Compliance
CVM Phase II- Duckpond Dr., Blacksburg, VA 24061-0442
Office: 540/231-4991; FAX: 540/231-6033
email: moored@vt.edu

DATE: May 16, 2005

MEMORANDUM

TO: David W. Harrison Psychology 0436
Gina Mollet Psychology 0436

FROM: David Moore

SUBJECT: **IRB Expedited Approval:** "Neuropsychological Effects of Hostility and Pain on Emotion Perception" IRB # 05-346

This memo is regarding the above-mentioned protocol. The proposed research is eligible for expedited review according to the specifications authorized by 45 CFR 46.110 and 21 CFR 56.110. As Chair of the Virginia Tech Institutional Review Board, I have granted approval to the study for a period of 12 months, effective May 16, 2005.

Virginia Tech has an approved Federal Wide Assurance (FWA00000572, exp. 7/20/07) on file with OHRP, and its IRB Registration Number is IRB00000667.

cc: File
Department Reviewer: Jack W. Finney

VITA
GINA A. MOLLET

PERSONAL INFORMATION

Academic Address:	Department of Psychology
	109 Williams Hall
	Virginia Polytechnic Institute and State University
	Blacksburg, VA 24061-0436
Phone:	540-231-9174
Email Address:	gmollet@vt.edu

EDUCATION

B.A. 1998-2000	University of Minnesota, Morris
	Major Field of Study: Psychology and Spanish
B.A. 2000-2002	University of South Dakota
	Major Field of Study: Psychology and Spanish
M.S. 2002-2004	Virginia Polytechnic Institute and State University
	Major Field of Study: Neuropsychology
	Thesis Title: Hostility and Negative Emotion Induction: Implications for Verbal Learning and Cardiovascular Regulation.
	Major Advisor: David W. Harrison, Ph.D.
Ph.D. 2004-present	Virginia Polytechnic Institute and State University
(expected 2006)	Blacksburg, VA
	Major Field of Study: Neuropsychology
	Dissertation Title: Neuropsychological Effects of Hostility and Pain on Emotion Perception
	Major Advisor: David W. Harrison, Ph.D.

POSITIONS AND APPOINTMENTS

2005 *Graduate Research Assistant*
Grado Department of Industrial and Systems Engineering
Assessment and Cognitive Ergonomics (ACE) Laboratory
Project Title: Sensemaking in Military Combat
Virginia Polytechnic Institute and State University, Blacksburg, Virginia

2005-present *Tutor/Mentor*
Student Athletic Academic Services, Virginia Polytechnic Institute and State University, Blacksburg, Virginia

2002-present *Graduate Teaching Assistant*
Virginia Polytechnic Institute and State University, Blacksburg, Virginia
Courses Taught: Introductory Psychology Recitation, Cognitive Psychology Lab, Advanced Lab in Learning, Psychology of Learning, Introductory Psychology, Nervous Systems and Behavior

2003 *Lab Assistant*
Neuroscience Primate Vision Lab
University of South Dakota, Vermillion, South Dakota

1999 *Teaching Assistant*
Orouba Language School, Cairo Egypt

CLINCIAL TRAINING

2002-2004 *Clinical Neuropsychology Laboratory*
Psychological Services Center
Virginia Polytechnic Institute and State University, Blacksburg, Virginia
Trained in Quantitative Electroencephalogram, standardized test administration, and syndrome analysis

HONORS AND AWARDS

1998 Lisa Bernard Memorial Scholarship

1998 William T. Stout Scholarship

1998 Academic Scholarship

1998-2002	Dean's List
2002	Study Abroad Scholarship
2004	Graduate School Scholarship

PROFESSIONAL ORGANIZATIONS AND ACTIVITIES

Psi Chi Honor Society for Psychology

Phi Beta Kappa Honor Society

Sigma Delta Pi Honor Society for Spanish

Golden Key International Honor Society

American Psychological Association – Student Affiliate

American Psychological Association of Graduate Students

Graduate Student Association of Virginia Tech – Psychology Department Delegate (2002-2003)

American Psychological Society

Peer Reviewer for American Psychological Society

Peer Reviewer for *Neuropsychology Review*

CURRENT RESEARCH AND SCHOLARLY INTERESTS

Neuropsychology of emotion, hostility, neuropsychology of pain, cardiovascular psychophysiology, emotional correlates of head trauma, sex differences

GRANTS SUBMITTED

2005	NIH PA 03-169 Basic and Transitional Research in Emotion Project Title: Neuropsychological Effects of Hostility and Pain on Emotion Perception. Unfunded.

GRANTS RECEIVED

2004	Graduate Student Association Travel Fund
2004	Graduate Research Development Program
2004	Graduate Student Association Travel Fund
2005	Galper Fund Award
2005	Wisconsin Symposium on Emotion Travel Grant

2005 Graduate Research Development Project

PEER REVIEWED PUBLICATIONS

Mollet, G.A. (2003). Sensation and perception. In Lehman, P. K., Dula, C. S., & Finney, J. W. (Eds.), *Introductory Psychology: Recitation reader,* pp. 57-58**.** Boston: McGraw-Hill.

Herridge, M., Harrison, D.W., Mollet, G.A., & Shenal, B. (2004). Hostility and emotional facial recognition: effects of arousal on perceptual accuracy and cardiovascular reactivity. *Brain and Cognition*, *55,* 564-571.

Emerson, C.S., Mollet, G.A, & Harrison, D.W. (2005). Anxious-depression in boys: an evaluation of executive functioning. *Archives of Clinical Neuropsychology, 20(4),* 539-546.

PUBLISHED ABSTRACTS

Foster, P. S., Beck, A. L., Mollet, G. A., & Harrison, D. W. (2003). Homologous hemispheric comparisons using quantitative electroencephalography: Is symmetry to be expected? *Archives of Clinical Neuropsychology, 18,* 796-797.

Foster, P.S., Mollet, G.A., Walters, R.P., & Harrison, D.W. (2004). Quantitative electroencephalographic evidence for the double dissociation of the trial making test. *Archives of Clinical Neuropsychology, 19,* 903.

Foster, P.S., Walters, R.P., Mollet, G.A., & Harrison, D.W. (2004). Performance on the figure trail making test as a function of right frontal lobe delta activity. *Archives of Clinical Neuropsychology, 19,* 903-904.

Foster, P.S., Walters, R.P., Mollet, G.A., & Harrison, D.W. (2004). Hostility as a function of right frontal lobe slow wave activity. *Archives of Clinical Neuropsychology, 19,* 941-942.

Mollet, G.A, Emerson, C.S., & Harrison, D.W. (2004). Anxious-depression in childhood: An evaluation of executive functions. *Journal of the International Neuropsychological Society, 10 (S1),* 142.

Mollet, G.A., Harrison, D.W., Walters, R.P., & Foster, P.S. (2004). Thalamic syndrome: Lateralized emotional multimodal hallucinations. *Archives of Clinical Neuropsychology, 19,* 937.

Mollet, G.A., & Harrison, D.W. (2005). Affective auditory verbal learning: Evidence for a negative emotional bias in hostility. *Journal of the International Neuropsychological Society, 11(S1),* 35.

Mollet, G.A., Walters, R.P., Harrison, D.W., & Foster, P.S. (2005). Alexia without agraphia: A traditional vs. a non-traditional case. *Journal of the International Neuropsychological Society, 11(S1),* 123-124.

Carmona, J., Harrison, D.W., Mollet, G.A., & Harrison, K. Support for frontal deactivation and posterior activation to rotary stress. *Archives of Clinical Neuropsychology, 20 (7),* 920.

Mollet, G.A., Walters, R.P., Harrison, D.W., & Holland, A.K. (2005). EOG Smooth Pursuit Errors as a Function of Hostility. *Archives of Clinical Neuropsychology, 20 (7),* 846.

Mollet, G.A., Holland, A.K., & Harrison, D.W. (2005). Effects of Bright Light on Unilateral Stroke: Modified Receptive Speech Deficits in Left Hemisphere Stroke Patients. *Archives of Clinical Neuropsychology, 20 (7),* 859.

Holland, A.K., Mollet, G.A, & Harrison, D.W. (in press). Sex Differences in Motor Precision as a Function of Cerebral Asymmetry. *Journal of the International Neuropsychological Society, abstract in press.*

Mollet, G.A., Holland, A.K., Kofalt, J.D., & Harrison, D.W. (in press). Differences in Hand Grip Strength as a Function of Hostility, Sex, and Stress. *Journal of the International Neuropsychological Society, abstract in press.*

Walters, R.P., Mollet, G.A., & Harrison, D.W. (in press). High Hostile Men: Increased Error Rate for Nonverbal Fluency. *Journal of the International Neuropsychological Society, abstract in press.*

MANUSCRIPTS ACCEPTED FOR PUBLICATION

Mollet, G.A. & Harrison, D.W. (Manuscript accepted for publication). Emotion and Pain: A Functional Cerebral Systems Integration. Manuscript accepted for publication.

MANUSCRIPTS SUBMITTED FOR PUBLICATION

Mollet, G.A., Foster, P.S., Walters, R.P., & Harrison, D.W. (2005).Thalamic syndrome: Lateralized multimodal hallucinations. Manuscript submitted for publication.

Mollet, G.A., & Harrison, D.W. (2005). Affective verbal learning in hostility: An increased primacy effect and bias for negative emotional material. Manuscript submitted for publication.

Mollet, G.A., & Harrison, D.W. (2005). Effects of hostility and pain on affective verbal learning in women. Manuscript submitted for publication.

Higgins, D.A., Mollet, G.A., & Harrison, D.W. (2005). Cardiovascular reactivity to speech processing and cold pressor stress: Evidence for sex differences in dynamic functional cerebral laterality. Manuscript submitted for publication.

MANUSCRIPTS IN PREPARATION

Mollet, G.A., Walters, R.P., & Harrison, D.W. (in preparation). Alexia without agraphia: A typical vs. an atypical lesion. Manuscript in preparation for publication.

Rhodes, R.D., Harrison, D.W., Demaree, H.A. (in preparation). Cerebral asymmetry in emotion and cardiovascular regulation: A functional systems analysis. Manuscript in preparation for publication.

POSTERS PRESENTED

Beck, A.L., Mollet, G.A., Foster, P.S., Walters, R.P., & Harrison, D.W. (2003). Thalamic syndrome: Lateralized multimodal hallucinations. Poster presented at the 19th Annual Virginia Tech Graduate Research Conference.

Mollet, G.A., Emerson, C.S., Beck, A.L., & Harrison, D.W. (2003). Frontal lobe dysfunction in anxious-depressed boys. Poster presented at the 19th Annual Virginia Tech Graduate Research Conference.

Foster, P. S., Beck, A. L., Mollet, G. A., & Harrison, D. W. (2003). Homologous hemispheric comparisons using quantitative electroencephalography: Is symmetry to be expected? Poster presented at the annual meeting of the National Academy of Neuropsychology, Dallas, TX.

Walters, R.P., Beck, A.L., Harrison, D.W., & Mollet, G.A. (2003). A lack of relationship between auditory and visual hallucinations in a rehabilitative population. Poster presented at the 19th Annual Virginia Tech Graduate Research Conference.

Foster, P.S., Mollet, G.A., Walters, R.P., & Harrison, D.W. (2004). Quantitative electroencephalographic evidence for the double dissociation of the trial making test. Poster presented at the 24th Annual Conference of the National Academy of Neuropsychology, Seattle, WA. November, 2004.

Foster, P.S., Walters, R.P., Mollet, G.A., & Harrison, D.W. (2004). Performance on the figure trail making test as a function of right frontal lobe delta activity. Poster presented at the 24th Annual Conference of the National Academy of Neuropsychology, Seattle, WA. November, 2004.

Foster, P.S., Walters, R.P., Mollet, G.A., & Harrison, D.W. (2004). Hostility as a function of right frontal lobe slow wave activity. Poster presented at the 24th Annual Conference of the National Academy of Neuropsychology, Seattle, WA. November, 2004.

Mollet, G.A, Emerson, C.S., & Harrison, D.W. (2004). Anxious-depression in childhood: An evaluation of executive functions. Poster presented at the 32nd annual meeting of the International Neuropsychological Society, Baltimore, MD, February 2004.

Mollet, G.A., Walters, R.P., & Harrison, D.W. (2004). Alexia without agraphia: Connecting the disconnection. Poster presented at the 20th Annual Virginia Tech Graduate Research Conference.

Mollet, G.A., Harrison, D.W., Walters, R.P., & Foster, P.S. (2004). Thalamic syndrome: Lateralized emotional multimodal hallucinations. Poster presented at the 24th Annual Conference of the National Academy of Neuropsychology, Seattle, WA. November, 2004.

Mollet, G.A., & Harrison, D.W. (2005). Affective auditory verbal learning: Evidence for a negative emotional bias in hostility. Poster presented at the 33rd Annual Meeting of the International Neuropsychological Society, St. Louis, MO. February, 2005.

Mollet, G.A., Walters, R.P., Harrison, D.W., & Foster, P.S. (2005). Alexia without agraphia: A traditional vs. a non-traditional case. Poster presented at the 33rd Annual Meeting of the International Neuropsychological Society, St. Louis, MO. February, 2005.

Mollet, G.A., & Harrison, D.W. (2005). Effects of Sex and Pain on Affective Verbal Learning. Poster presented at the Poster presented at the 21st Annual Virginia Tech Graduate Research Conference.

Mollet, G.A., & Harrison, D.W. (2005). A Bias for Negative Emotion in Verbal Learning. Poster presented at the Poster presented at the 21st Annual Virginia Tech Graduate Research Conference.

Harrison, D.W., & Mollet, G.A. (2005). Affective verbal learning: Evidence for a negative emotional bias. Poster presented at the 2005 American Psychological Association Annual Convention, Washington, DC, August, 2005.

Harrison, D.W., & Mollet, G.A. (2005). Effects of a pain stressor on affective verbal learning. Poster presented at the 2005 American Psychological Association Annual Convention, Washington, DC, August, 2005.

Mollet, G.A., & Harrison, D.W. (2005). Influence of pain stress and sex on affective verbal learning. Poster presented at the 2005 American Psychological Association Annual Convention, Washington, DC, August, 2005.

Mollet, G.A., & Harrison, D.W. (2005). Effects of hostility on affective verbal learning in women. Poster presented at the 2005 American Psychological Association Annual Convention, Washington, DC, August, 2005.

Carmona, J., Harrison, D.W., Mollet, G.A., & Harrison, K. Support for frontal deactivation and posterior activation to rotary stress. Poster presented at the 2005 National Academy of Neuropsychology Annual Meeting, Tampa, FL, October, 2005.

Mollet, G.A., Walters, R.P., Harrison, D.W., & Holland, A.K. (2005). EOG Smooth Pursuit Errors as a Function of Hostility. Poster presented at the 2005 National Academy of Neuropsychology Annual Meeting, Tampa, FL, October, 2005.

Mollet, G.A., Holland, A.K., & Harrison, D.W. (2005). Effects of Bright Light on Unilateral Stroke: Modified Receptive Speech Deficits in Left Hemisphere Stroke Patients. Poster presented at the 2005 National Academy of Neuropsychology Annual Meeting, Tampa, FL, October, 2005.

Holland, A.K., Mollet, G.A, & Harrison, D.W. (2006). Sex Differences in Motor Precision as a Function of Cerebral Asymmetry. Poster presented at the 34th Annual Meeting of the International Neuropsychological Society, Boston, MA. February, 2006.

Mollet, G.A., Holland, A.K., Kofalt, J.D., & Harrison, D.W. (2006). Differences in Hand Grip Strength as a Function of Hostility, Sex, and Stress. Poster presented at the 34th Annual Meeting of the International Neuropsychological Society, Boston, MA. February, 2006.

Walters, R.P., Mollet, G.A., & Harrison, D.W. (2006). High Hostile Men: Increased Error Rate for Nonverbal Fluency. Poster presented at the 34th Annual Meeting of the International Neuropsychological Society, Boston, MA. February, 2006.