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Neuropsychological effects of hostility and pain on emotion perception

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In order to examine the neuropsychological effects of hostility on emotional and pain processing, auditory emotion perception before and after cold pressor pain in high and low hostile men was examined. Additionally, quantitative electroencephalography (QEEG) was recorded between each experimental manipulation. Results indicated that identification of emotion post cold pressor differed as a function of hostility level and ear. 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. Taken together, results from the dichotic listening task and the QEEG suggest decreased cerebral laterality and left hemisphere activation for emotional and pain processing in high hostile men.

Keywords: Hostility; Electroencephalography; Emotion; Cold pressor; Dichotic listening; Pain.

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, Higgins, Williamson, & Harrison, 2002), auditory (Gadea, Gomez, Gonzalez-Bono, Espert, & Salvador, 1995), somatosensory (Herridge, Harrison, & Demaree, 1997; Lee, Meador, Loring, & Bradley, 2002), visual (Coupland et al., 2004; Klaassen, Riedel, Deutz, & Van Praag, 2002), and cardiovascular (Gendolla, Abele, & Krusken, 2001; Snyder, Harrison, & Shenal, 1998) systems. Similarly, pain produces relative changes in motor (Urban et al., 2004), auditory (Demaree & Harrison, 1997), somatosensory (Valeriani et al., 2004), visual (Herridge, Harrison, Mollet, & Shenal, 2004), and cardiovascular (Fillingim, Browning, Powell, & Wright, 2002) systems. Further, imaging studies indicate that pain may produce cerebral activation that is similar to cerebral activation seen in negative emotion (Coghill, Gilron, & Iadarola, 2001; Hseih, Hannerz, & Ingvar, 1996).

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. Behaviorally, hostility is described as aggressive behavior and social avoidance (Barefoot, Dodge, Peterson, Dahlstrom, & Williams, 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

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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 and its link to cardiovascular disease (CVD; Matthews, Gump, Harris, Haney, & Barefoot, 2004) have led to increased study of the construct.

Empirical investigations within normal populations indicate 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 individual's preferred mode of anger expression. According to approach/withdrawal models of emotion anger expressers would activate the left frontal lobe, while anger suppressors 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.

Dichotic listening

Dichotic listening is a task primarily used to by experimental neuropsychologists to examine auditory lateralization (Jancke, Specht, Shah, & Hugdahl, 2003). Dichotic listening can also be used to assess emotion lateralization. When looking at emotion, it is noted that changes in vocal tone indicate changes in emotion (Shipley-Brown, Dingwall, Berlin, Yeni-Komshian, & Gordon Salant, 1988). Thus, varying the tone of voice of the dichotic messages can provide an alternative measure for emotion lateralization (Bryden, 1988). Previous research using dichotic listening to assess emotion lateralization has primarily used neutral words with varying emotional prosody (Bryden & MacCrae, 1989; Snyder, Harrison, & Gorman, 1996). Participants are asked to identify the emotional tone or the word that was spoken. Other tasks exist whereby affective sentences are presented with neutral sentences. In this case participants are asked to identify the emotional tone and content of the sentences (Ley & Bryden, 1982). Emotional dichotic listening tasks may also employ the use of nonlinguistic affective sounds presented in positive or negative tones (i.e., giggles, groans, sighs; Pollak, Holt, & Wismer Fries, 2004). Previous research using emotional dichotic listening tasks indicate a left ear advantage (right hemisphere) for the perception of affective tone (Bryden, Ley, & Sugarman, 1982; Bryden & MacRae, 1989; Bulman-Fleming & Bryden, 1994; Jancke, Buchanan, Lutz, & Shah, 2001; Snyder et al., 1996; Voyer, Russell, & McKenna, 2002). The left ear advantage for affective tone is thought to reflect the functional role of the right hemisphere in emotion perception (Bryden & MacRae, 1989).

Moreover, imaging data indicate that emotional dichotic listening tasks produce 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 for emotional processing, the left posterior should be important for positive emotion, while the right posterior should be important negative emotion. Erhan, Borod, Tenke, and Bruder (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. 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. Wexler, Schwartz, Warrenburg, Servis, and Tarlatzis (1986) found a relatively low right ear advantage for processing speech sounds in 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 also exist. In an experiment 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 (Al'Absi, Hugdahl, & Lovallo, 2002). 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 et al., 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 et al., 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. In order to examine this aspect of hostility, participants completed the dichotic listening task twice and were exposed to a cold pressor in between trials.

Cold pressor

The cold pressor was chosen as a painful stimulus for the experiment 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 et al. (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 participant's 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. Quantitative electroencephalography (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, Seri, Mattia, & Cruccu, 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 expected that participants would show increased identification of affect at the left ear relative to the right ear. The pain stress was expected to increase affect identification, particularly angry affect identification, at the left ear through activation of the right hemisphere. Moreover, the effect was expected to be significantly higher within the high hostile group. Several hypotheses also existed for the QEEG data. The

emotional dichotic listening task was expected to produce bilateral activation at the temporal region due to the fact that the stimuli were both verbal and emotional. It was expected that increases in beta (22–32 Hz) magnitudes at the temporal location for both trials of the dichotic listening task would be noted. The cold pressor was expected to increase right temporal and right parietal activation in participants as evidenced by increases in beta (22–32 Hz) magnitude at the right temporal and parietal locations. Further, high hostiles were expected to show increased reactivity to the cold pressor at the right hemisphere relative to the low hostile group. Within the delta bandwidth it was expected that 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 location after cold pressor administration. A self-report measure was also included at the end of the experiment in order to assess the level of pain and stress that participants experienced as a result of the cold pressor. Participants in the high hostile group were expected to report experiencing lower levels of pain and stress from the cold pressor than the low hostile group.

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., Crews & Harrison, 1994; Harrison, Gorelczenko, & Cook, 1990; Hiscock, Perachio, & Inch, 2001). To avoid confounding these laterality effects the exclusion of women was necessary.

METHOD

Participants

Participants were recruited from the undergraduate psychology population. They completed an online prescreening that included an informed consent form, a medical history questionnaire, the Coren, Porac, and Duncan Laterality Questionnaire (Coren, Porac, & Duncan, 1979), and the Cook–Medley Hostility Scale (Cook & Medley, 1954).[1] The medical history questionnaire was a 36-item self-report questionnaire that assessed history of previous head injuries, neurological conditions, mental illness history, and use of drugs and alcohol. Participants were excluded if they were left-handed, if they 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, or were being treated for a current mental illness (i.e., depression, anxiety). Moreover, to be placed in the high hostile group participants had to score 29 or above on the Cook–Medley Hostility Scale, while those in the low hostile group had to score 19 or below. Cutoff scores for the groups were based on previous research in our laboratory (Demaree & Harrison, 1997; Harrison & Gorelczenko, 1990; Williamson & Harrison, 2003) and found to be successful. Participants were asked to refrain from smoking for 2 hours and from caffeine and alcohol for 12 hours prior to participation in the experiment.

A total of 46 men completed the experiment. A total of 14 participants were excluded for not meeting scoring criteria on the Cook–Medley Hostility Scale on the testing day. These participants completed the entire questionnaire and scored in the middle range, between 20 and 28. A total of 2 participants were excluded for removing their hands from the cold pressor early. A total of 4 participants (2 from the high hostile group and 2 from the low hostile group) were excluded for having beta magnitudes at one of the temporal lobe locations (T3, T4, T5, or T6) 2 standard deviations above the mean. The large beta magnitudes were noted after the participants had placed their hands in the cold pressor. Upon inspection of the QEEG data, there appeared to be electromyogram (EMG) interference at the temporal lobe sites for those participants. Perhaps the participants clenched their jaws while placing their hands in the cold pressor. The final analysis included 13 high and 13 low hostile men between the ages of 18–24 years (M = 19.50, SD = 1.50). The mean age of the high hostile group was 20.27 (SD = 1.55), and the mean age of the

[1]The Cook Medley Hostility Scale 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. The current grouping criterion has been used previously in our lab (Demaree & Harrison, 1997; Demaree et al., 2002; Harrison & Gorelczenko, 1990; Williamson & 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). 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."

low hostile group was 18.91 (SD = 0.83). Of the 22 participants that reported an ethnic/racial background, 14 were Caucasian (5 in the high hostile group, 9 in the low hostile group), 3 were African American (2 in the high hostile group, 1 in the low hostile group), 4 were Asian (3 in the high hostile group, 1 in the low hostile group), and 1 reported being African (in the high hostile group).

Physiological measures

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 participant's 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 participant's 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 was 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 kΩ or less at each site.

Electrooculogram (EOG)

Auxiliary channels of the NeuroSearch-24 and silver/silver chloride electrodes filled with electrode gel (Electro-Cap International, Inc.) were used to measure electrooculogram (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.

Apparatus

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 adapted from Voyer et al. (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 4 practice trials in order to prepare them for and to familiarize them with the task. The intertrial interval was 5 s. Stimuli were presented to participants at 75 dB via Sony earphones. The dB level was rechecked after every 7 participants. Position of the earphones was counterbalanced between participants to control for any intensity differences.

Cold pressor

Ice water for the cold pressor was maintained at 0–3 degrees Celsius using a small ice cooler located next to the participant's left arm. Water temperature was measured continuously throughout the experiment using a standard mercury thermometer. Participants submerged their hands in the ice water, and their hands made contact with the ice. The water was not circulated during the experiment.

Self-report

Cold pressor assessment

Upon completion of the study participants completed a cold pressor assessment. They were asked to rate their level of pain and stress in response to

the cold pressor on a 7-point Likert scale (1 being not painful or stressful and 7 being extremely painful or stressful).

Procedure

All procedures were approved by the Department of Psychology Human Subjects Committee and the Virginia Tech Institutional Review Board. After completion of the online prescreening, eligible participants were invited to the lab. Upon arrival at the lab, participants were asked to complete the informed consent form. After completion of the informed consent form, participants were fitted with the QEEG cap and EOG electrodes. 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. The experiment began with the following instructions:

> Please sit relaxed in the chair with your eyes closed. 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. Next, participants were given a data sheet and were asked to circle the emotion that they heard most clearly on each trial of the dichotic listening task. After completion of the dichotic listening task, a 2-minute QEEG and EOG sample was collected. Next, participants 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 2-minute QEEG and EOG sample was collected. The dichotic listening task was then administered again. At completion of the second trial of the dichotic listening task, a final 2-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. They also retook the Cook–Medley Hostility Scale. Participants were then debriefed and excused.

RESULTS

Self-report questionnaire analysis

Separate t tests were used to compare group means from the Coren, Porac, and Duncan Laterality Questionnaire and the Cook–Medley Hostility Scale. Results indicated that groups were statistically equivalent on the laterality questionnaire, $t(24) = 0.46$, $p = .64$. 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 ($SD = 2.36$). Hostility scores collected on the testing day were used to place participants in high and low hostile groups. If participants did not meet scoring criteria on the testing day they were eliminated from the analysis. Scores for the high and low hostile groups on the Cook–Medley Hostility Scale 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 than on the low hostile group ($M = 14.14$, $SD = 2.36$).

A one-way analysis of variance (ANOVA) was used to examine group differences on 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, $F(1, 24) = 0.42$, $p = .52$. However, a main effect of group, $F(1, 24) = 5.25$, $p < .03$, 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

In order to assess the effects of the hostility level and the cold pressor on affect identification at the left and right ears 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) × Pain (pre or post cold pressor). Additionally, a laterality index (LI) was calculated in order to determine ear advantage. The following formula was used to compute the LI:

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

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. Separate ANOVAs were computed for the total number of correctly identified affective tones at the right ear, the total number of correctly identified affective tones at the left ear, and the LI as dependent variables. All post hoc comparisons were made using Tukey's honestly significant difference (HSD).

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 × Pain interaction, $F(1, 24) = 5.91$, $p < .02$, indicated that this effect was primarily due to the high hostile group. Post hoc analyses 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 × 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 ($M = 10.42$, $SD = 4.90$) than in the pre cold pressor condition ($M = 8.46$, $SD = 3.33$). However, identification of neutral (pre cold pressor: $M = 9.62$, $SD = 4.23$; post cold pressor: $M = 9.54$, $SD = 4.33$) and happy (pre cold pressor: $M = 9.38$, $SD = 5.14$; post cold pressor: $M = 9.62$, $SD = 5.97$) stimuli was not significantly different as a function of the cold pressor.

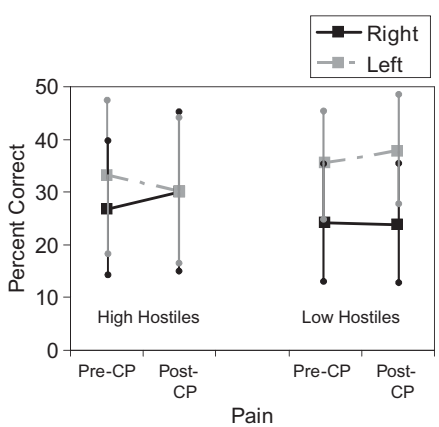

Figure 1. Number of correctly identified stimuli at the right and left ears as a function of group and pain. CP = cold pressor.

Left ear

A Group × 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. However, post hoc analyses 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. The results are presented in Figure 1 with the Group × Pain interaction from the right ear and from the left ear for comparison purposes.

Laterality index

A Group × 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 ($M = –0.21$, $SD = 0.26$) and post cold pressor ($M = –0.26$, $SD = 0.25$) conditions. The laterality index for the high hostile group shifted from a left ear advantage in the pre cold pressor condition ($M = –0.11$, $SD = 0.36$) to a no ear advantage in the post cold pressor condition ($M = 0.03$, $SD = 0.40$). The low hostile group exhibited an increased left ear advantage in the post cold pressor condition. Post hoc comparisons 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.

QEEG analysis

Individual QEEG traces were visually inspected and artifacted offline. 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. A preliminary analysis was computed that included data from all frontal, temporal, and parietal sites. Based on the findings of this analysis and our interest in the delta and beta bandwidths and the effects of hostility at the frontal, temporal, and parietal sites a more refined analysis was computed. In the final analysis only data from the delta (2–4 Hz) and beta (22–32 Hz) bandwidths within the primary regions of interest (F3, F4, T3, T4, P3, and P4) were included in the analysis. These electrode sites were chosen because they produced the strongest results. Additionally, previous research examining cerebral response to the dichotic listening (see Jancke et al., 2001; Jancke & Shah, 2002) and the cold pressor (Chang et al., 2002; Di Piero et al., 1994) tasks has illustrated a relationship between the frontal, temporal, and parietal areas and these tasks.

Since QEEG data were collected before and after three separate manipulations, data were analyzed in three stages. First, to examine baseline effects of hostility and the effects of the dichotic listening task on the QEEG activity 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, temporal, and parietal). Separate ANOVAs were computed for the delta and beta bandwidths. All post hoc comparisons were made using Tukey's HSD.

Delta (2–4 Hz)—pre and post dichotic listening, Trial 1

A main effect of group, $F(1, 24) = 8.73$, $p < .007$, indicated that the high hostile group ($M = 4.33$, $SD = 1.21$) had significantly lower delta magnitudes than the low hostile group ($M = 5.31$, $SD = 1.56$).

Beta (22–32 Hz)—pre and post dichotic listening, Trial 1

A Condition × Location, $F(2, 48) = 13.22$, $p < .0001$, interaction was significant. Post hoc comparisons indication that the only significant change from pretask ($M = 7.92$, $SD = 4.32$) to posttask ($M = 6.61$, $SD = 3.04$) conditions was at the temporal location. Means at the frontal (pre dichotic listening, Trial 1: $M = 5.94$, $SD = 1.56$; post dichotic listening, Trial 1: $M = 6.31$, $SD = 1.97$) and parietal (pre dichotic listening, Trial 1: $M = 7.06$, $SD = 2.20$; post dichotic listening, Trial 1: $M = 7.27$, $SD = 2.41$) locations were not significantly different as a function of the task. In the pretask condition means were significantly higher at the temporal and parietal locations than at the frontal location. However, in the posttask condition only means at the parietal location were significantly higher than those at the frontal location.

A significant Group × Condition, $F(1, 24) = 5.41$, $p < .03$, interaction indicated that beta magnitudes for the high hostile group were higher in the pre dichotic listening Trial 1 condition ($M = 7.15$, $SD = 3.60$) than in the posttask condition ($M = 6.39$, $SD = 2.22$). However, the low hostile group underwent an opposite pattern. Beta magnitudes for the low hostiles were higher in the posttask condition ($M = 7.10$, $SD = 2.78$) than in the pretask condition ($M = 6.80$, $SD = 2.19$). Post hoc comparisons indicated that the changes as a function of the task were not significant.

A Condition × Hemisphere interaction, $F(1, 24) = 5.21$, $p < .032$, was significant. Post hoc comparisons indicated that there was a significant decrease in beta magnitude at the left hemisphere from pretask ($M = 7.21$, $SD = 3.38$) to posttask ($M = 6.64$, $SD = 2.29$) condition. Means at the right hemisphere did not significantly change from pre- ($M = 6.74$, $SD = 2.50$) to posttask ($M = 6.82$, $SD = 2.76$) conditions. Additionally, means at the right and left hemispheres were not significantly different in the pre or posttask condition.

A Condition × Hemisphere × Location interaction, $F(2, 48) = 4.87$, $p < .012$, was significant. Post hoc comparisons indicated that in the pretask condition means at the right temporal location were significantly higher than means at the left temporal location. Further, means at the left and right temporal and parietal locations were significantly higher than means at the left and right frontal location in the pretask condition. In the posttask condition means were not significantly different between the left and right hemispheres at each location. However, means at the parietal location were still significantly higher than means at the frontal location (see Table 1 for means and standard deviations).

The second stage of analysis examined data collected before and after the cold pressor. This analysis was conducted in order to examine cerebral activation in high and low hostile participants before and after the cold pressor. A four-factor mixed-design ANOVA was used. The ANOVA included the fixed effect of group (high or low hostile) and the repeated measures of pain (pre or post cold pressor), hemisphere (right or left), and location (frontal, temporal, and parietal). Separate ANOVAs were computed for the delta and beta bandwidths.

TABLE 1
Means and standard deviations for the Condition × Hemisphere × Location interaction for the beta bandwidth before and after the dichotic listening Trial 1 task

	Condition			
	Pre dichotic listening Trial 1		*Post dichotic listening Trial 1*	
Location	*Mean*	*SD*	*Mean*	*SD*
Right frontal	5.94	1.17	6.43	2.24
Left frontal	5.93	1.16	6.17	1.68
Right temporal	7.12	3.27	6.65	3.19
Left temporal	8.72	5.10	6.57	2.95
Right parietal	7.16	2.49	7.37	2.78
Left parietal	6.97	1.90	7.18	2.04

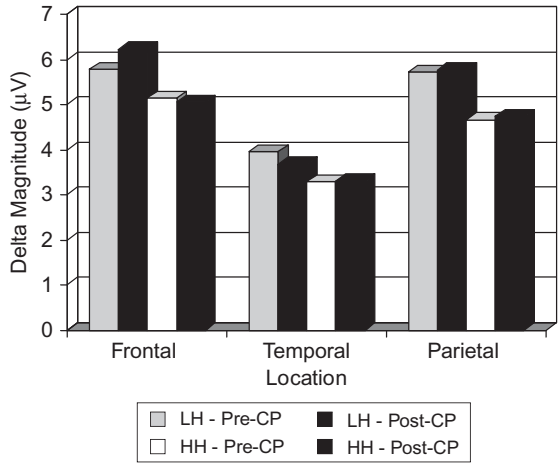

Figure 2. Delta magnitude at each location as a function of group and the cold pressor. CP = cold pressor. LH = low hostile. HH = high hostile.

Delta (2–4 Hz)—pre and post cold pressor

A main effect of group, $F(1, 24) = 7.74$, $p < .010$, indicated that the low hostile group ($M = 5.18$, $SD = 1.43$) had significantly higher delta magnitudes than did the high hostile group ($M = 4.36$, $SD = 1.18$).

A Pain × Location interaction, $F(2, 48) = 3.90$, $p < .03$, was significant. Delta magnitudes were significantly higher at the frontal location ($M = 5.46$, $SD = 1.09$) than at the temporal ($M = 3.62$, $SD = 1.06$) and the parietal ($M = 5.19$, $SD = 1.17$) locations pre cold pressor. Additionally, delta magnitudes at the parietal location were significantly higher than delta magnitudes at the temporal location pre cold pressor. The same pattern was present post cold pressor. Delta magnitudes at the frontal location ($M = 5.63$, $SD = 1.17$) were significantly higher than magnitudes at the temporal ($M = 3.48$, $SD = 0.75$) and the parietal ($M = 5.25$, $SD = 1.10$) locations. Again, parietal delta magnitudes were significantly higher than temporal delta magnitudes. However, there were no significant changes within each location as a function of the cold pressor.

A Group × Pain × Location, $F(2, 48) = 6.81$, $p < .0025$, interaction was significant. Post hoc comparisons indicated that there were no significant changes within the high hostile group from pre to post cold pressor conditions across locations. For low hostiles, there was a significant increase in delta magnitude at the frontal location from pre to post cold pressor conditions. Low hostiles had significantly higher delta magnitudes at the frontal and parietal locations in both pre and post cold pressor conditions than did the high hostile group (see Figure 2).

Beta (22–32 Hz)—pre and post cold pressor

A significant main effect of pain, $F(1, 24) = 28.86$, $p < .0001$, indicated that the cold pressor significantly increased beta magnitudes (pre cold pressor: $M = 6.34$, $SD = 2.27$; post cold pressor: $M = 7.70$, $SD = 3.54$).

A Group × Pain interaction, $F(1, 24) = 8.86$, $p < .007$, indicated that the increase in 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 beta magnitudes as a function of the cold pressor (pre cold pressor: $M = 6.14$, $SD = 8.26$; post cold pressor: $M = 8.26$, $SD = 4.17$), while the low hostile men did not undergo a significant change in beta magnitude after cold pressor exposure (pre cold pressor: $M = 6.53$, $SD = 2.39$; post cold pressor: $M = 7.14$, $SD = 2.68$).

A Pain × Location interaction, $F(2, 48) = 15.53$, $p < .0001$, 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 location from pre ($M = 6.01$, $SD = 2.51$) to post cold pressor ($M = 8.80$, $SD = 4.99$) conditions. There was no significant change in high beta magnitude at the frontal (pre cold pressor: $M = 6.04$, $SD = 1.70$; post cold pressor: $M = 6.42$, $SD = 1.74$) or at the parietal (pre cold pressor: $M = 6.96$, $SD = 2.41$; post cold pressor: $M = 7.87$, $SD = 2.68$) location as a function of the cold pressor. In the pre cold pressor condition means at the parietal location were significantly higher then means at the temporal location; however, in the post cold pressor condition there was

no difference in beta magnitudes between locations. Further, in the post cold pressor condition beta magnitudes at the temporal and parietal locations were significantly higher than beta magnitudes at the frontal location. This effect was further influenced by group. A significant Group × Pain × Location interaction, $F(2, 48) = 7.51$, $p < .002$, indicated that the increase in beta at the temporal location after the cold pressor was primarily due to the high hostile group. Post hoc comparisons indicated that there was a significant increase in beta magnitude at the temporal location for high hostile men after cold pressor exposure. This effect was not significant in the low hostile group. Additionally, beta magnitude at the temporal location for the high hostile group in the post cold pressor condition was significantly higher then beta magnitudes for the low hostile group. Beta magnitude at the frontal and parietal locations was not significantly affected by the cold pressor for either group. Further, beta at the frontal and the parietal locations was not significantly different between groups in either condition (see Figure 3).

A Pain × Hemisphere × Location interaction, $F(2, 48) = 4.63$, $p < .01$, was significant. Post hoc comparisons indicated 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 beta magnitude at the left and right temporal locations. Further, in the post cold pressor condition beta magnitude at the left temporal location (T3) was significantly higher than beta magnitude at the right temporal location (T4). There was no change in beta magnitude at the frontal or parietal locations from pre to post cold pressor conditions. Further, beta magnitude in the frontal and the parietal locations was not significantly different between the right and left hemispheres in either condition (see Table 2 for means and standard deviations).

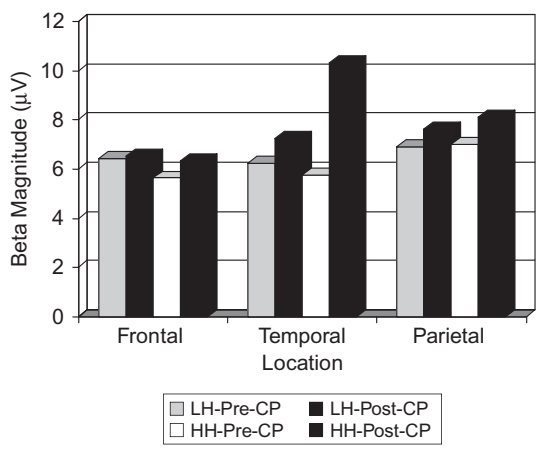

Figure 3. Beta magnitude at each location as a function of group and the cold pressor. CP = cold pressor. LH = low hostile. HH = high hostile.

TABLE 2
Means and standard deviations for the Pain × Hemisphere × Location interaction for the beta bandwidth before and after the cold pressor

	Condition			
	Pre cold pressor		*Post cold pressor*	
Location	*Mean*	*SD*	*Mean*	*SD*
Right frontal	6.16	1.91	6.55	2.00
Left frontal	5.91	1.48	6.30	1.44
Right temporal	6.02	2.79	8.06	4.23
Left temporal	6.00	2.25	9.53	5.64
Right parietal	7.05	2.75	8.00	3.09
Left parietal	6.87	2.06	7.75	2.26

Finally, to examine the QEEG collected before and after the second trial of dichotic listening, a four-factor mixed-design ANOVA was used. The analysis was done to examine cerebral activation before and after the dichotic listening task. The ANOVA included the fixed effects of group (high or low hostile) and the repeated measures of condition (pre or post dichotic listening, Trial 2), hemisphere (right or left), and location (frontal, temporal, and parietal). Separate ANOVAs were computed for the delta and beta bandwidths.

Delta (2–4 Hz)—pre and post dichotic listening, Trial 2

A main effect of group, $F(1, 24) = 6.90$, $p < .014$, again indicated that delta magnitudes were significantly higher for the low hostile group ($M = 5.18$, $SD = 1.51$) than for the high hostile group ($M = 4.36$, $SD = 1.17$).

A main effect of location, $F(2, 48) = 119.35$, $p < .0001$, indicated that delta magnitudes at the frontal ($M = 5.49$, $SD = 1.11$) and the parietal locations ($M = 5.31$, $SD = 1.29$) were significantly higher than delta magnitudes at the temporal location ($M = 3.52$, $SD = 0.83$).

Beta (22–32 Hz)—pre and post dichotic listening, Trial 2

A Group × Condition, $F(1, 24) = 7.19$, $p < .01$, interaction was significant. Post hoc comparisons indicated that the high hostile group underwent a significant reduction in beta magnitudes from pretask ($M = 7.53$, $SD = 3.90$) to posttask ($M = 6.14$, $SD = 2.26$) condition. Beta magnitude for the low hostile group was not significantly different from

pre task (M = 6.82, SD = 2.55) to post task (M = 6.64, SD = 2.52). Further, high hostiles had significantly higher beta magnitudes in the pretask condition than did the low hostile group.

A Condition × Location, $F(2, 48) = 7.47$, $p < .002$, interaction was significant. Post hoc comparisons indicated that in the pretask condition beta magnitudes at the temporal location (M = 8.14, SD = 5.0) were significantly higher than beta magnitudes at the frontal location (M = 6.15, SD = 1.35). Beta magnitudes at the parietal location (M = 7.24, SD = 2.14) in the pretask condition were not significantly different from those at either the frontal or the temporal location. In the posttask condition there was no significant difference between locations (frontal: M = 6.09, SD = 1.61; temporal: M = 5.97, SD = 2.69; parietal: M = 7.11, SD = 2.62). The only location to significantly change from pre- to posttask conditions was the temporal location. There was a significant reduction in beta magnitude at the temporal lobes in the posttask condition.

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. High and low hostile groups were analyzed individually using QEEG data collected before and after the cold pressor. Further, since the primary findings from the QEEG data were at the frontal and temporal locations, the parietal location was eliminated from the refined ANOVA. A Pain (pre or post cold pressor) × Hemisphere (left or right) × Location (frontal and temporal) mixed-design ANOVA was computed for the delta and beta bandwidths.

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) than at the temporal-1 location (M = 3.29, SD = 0.69).

High hostiles—beta (22–32 Hz)

A significant main effect of pain, $F(1, 12) = 19.88$, $p < .0008$, was found for beta magnitude. 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 × Location interaction, $F(1, 12) = 14.36$, $p < .003$, indicated that this was primarily due to an increase in beta magnitude at the temporal location. Post hoc comparisons revealed that beta magnitude significantly increased at the temporal location in the post cold pressor condition (pre cold pressor: M = 5.78, SD = 2.41; post cold pressor: M = 10.33, SD = 5.78) but not at the frontal location (pre cold pressor: M = 5.64, SD = 0.99; post cold pressor: M = 6.32, SD = 1.35). Further support for this interaction was provided by a main effect of location, $F(1, 12) = 4.96$, $p < .05$. Beta magnitudes at the temporal location (M = 8.06, SD = 4.95) were significantly higher than beta magnitudes at the frontal location (M = 5.98, SD = 1.22).

A Pain × Hemisphere interaction was also present, $F(1, 12) = 4.75$, $p < .05$. Post hoc comparisons indicated that beta magnitudes at the right and left hemispheres were not significantly different in the pre cold pressor condition (right: M = 5.72, SD = 2.00; left: M = 5.70, SD = 1.67). In the post cold pressor condition, significantly increased beta magnitude at the both the right and the left hemispheres was found (right: M = 7.68, SD = 3.51; left: M = 8.97, SD = 5.52). Additionally, beta magnitude at the left hemisphere was significantly increased relative to high beta magnitude at the right hemisphere in the post cold pressor condition.

A Pain × Hemisphere × Location interaction, $F(1, 12) = 5.86$, $p < .03$, was significant. Post hoc comparisons indicated that there was no significant change in beta magnitude at the frontal location as a function of the cold pressor. Beta magnitude at the temporal location was significantly increased after cold pressor exposure. Additionally, high beta magnitude at the left temporal location (T3) was significantly higher than high beta magnitude at the right temporal location (T4) in the post cold pressor condition (see Figure 4).

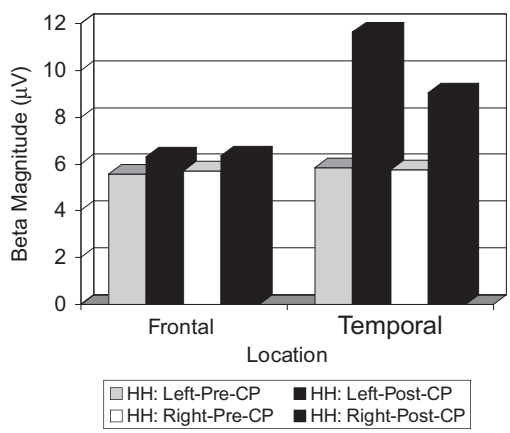

Figure 4. Beta magnitudes for the high hostile group (HH) at the frontal and temporal locations as a function of hemisphere and the cold pressor (CP).

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 location ($M = 6.00$, $SD = 0.98$) than at the temporal location ($M = 3.81$, $SD = 1.04$).

A Pain × Location interaction, $F(1, 12) = 7.79$, $p < .02$, indicated that the cold pressor increased delta magnitudes at the frontal location (pre cold pressor: $M = 5.78$, $SD = 0.89$; post cold pressor: $M = 6.22$, $SD = 1.03$) and at the temporal location (pre cold pressor: $M = 3.95$, $SD = 1.21$; post cold pressor: $M = 3.67$, $SD = 0.84$) from pre to post cold pressor conditions. However, post hoc comparisons indicated that these increases were not significant.

Low hostiles—beta (22–32 Hz)

For the beta bandwidth, a Pain × 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 beta magnitude at the temporal location (pre cold pressor: $M = 6.24$, $SD = 2.64$; post cold pressor: $M = 7.26$, $SD = 3.54$) as a function of the cold pressor. Means at the frontal location were not significantly different as a function of the cold pressor (pre cold pressor: $M = 6.43$, $SD = 2.14$; post cold pressor: $M = 6.53$, $SD = 2.07$).

DISCUSSION

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 may lead to a better conceptualization of the neuropsychological effects of hostility on emotion perception and reactivity to pain.

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. QEEG results within the high hostile group indicated significant increases in beta activation at the left temporal location (T3) after exposure to the cold pressor.

The pattern of dichotic listening stimuli identification before and after a cold pressor stressor in the high hostile group may be indicative of decreased laterality for emotional processing within high hostile men. 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 than did 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 than did 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. It is important to note that the reduction in cerebral lateralization for emotional processing is not indicative of a deficit, but rather illustrates differential cerebral processing in the groups compared here.

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 than

were low hostile men. Thus, the current results provide additional evidence that high hostile men have reduced right lateralization for emotional processing across sensory perceptual systems when compared to low hostile men.

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 (Bruder et al., 1995; Ragland et al., 1992; Wexler, Giller, & Southwick, 1991), depression (Bruder et al., 1992; Wale & Carr, 1990), and social phobia (Bruder, Schneier, Stewart, McGrath, & Quitkin, 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 changes in right hemisphere organization that occurs with heightened hostility.

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 function differently in high hostile individuals, emotion processing may shift to the left hemisphere. Additionally, QEEG 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 et al. (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 et al., 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, which 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, 2007). Altered speech expression and comprehension may have implications for social interactions. High hostile men may have difficulty expressing themselves during confrontation. 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.

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 (Burns, Bruehl, & Caceres, 2004; Janssen, Spinhoven, & Brosschot, 2001) 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 than did 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 et al., 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. 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.

Influences of the cold pressor on the dichotic listening task were also noted. However, the results were diametrically opposite to 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 × 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 of emotional processing. 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 and parietal locations during both trials of the dichotic listening task as evidenced by increased 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 beta magnitudes at the temporal location were significantly elevated at baseline and after exposure to the cold pressor.

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

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, Baumgartner, Magerl, Stoeter, and Treede (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.

An alternative explanation to left hemisphere activation to the cold pressor is provided by Chang et al. (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, 2004). 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 × 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 underarousal 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

It is important to consider several limitations of the current study that may influence the application of the results. First, the participants were recruited from a college population. This led to a small population of potential participants and also to a restricted age range. Future studies of this nature should seek to include older and younger populations to examine the influence of age on hostility. Additionally, the current sample size is relatively low. It would be beneficial to examine larger groups of individuals and to include female participants. An additional problem of the experiment may have been the way the cold pressor was administered. The water in the cold pressor was not continuously circulated. It is possible that a lining of warm water might have developed around the participant's hand. This might explain why some participants did not report a great deal of pain or stress during the cold pressor. Moreover, due to our interest in the response of the right hemisphere all participants submerged their left hand in the cold pressor. Perhaps using both hands in the cold pressor might have produced differential results. It should also be noted that the order of data collection might have been problematic for addressing our specific hypotheses. QEEG data were collected after each experimental manipulation. However, the current design was used to reduce the amount of artifact due to movement in the data.

There were also a large number of participants who were excluded from the analysis due to their scores on the Cook–Medley Hostility Scale on the day of the experiment. The large number of participants ($n = 14$) may be indicative of several problems and limit the sample variance of the current experiment. First, participants were prescreened in an online questionnaire. The lack of retest reliability may indicate that the online screening method does not produce consistent results. Alternatively, participants may have chosen answers that were more socially desirable in the lab setting. In either case, we excluded participants who did not meet scoring criteria due to our interest in group differences in high and low hostile individuals and due to criteria set out before the experiment was conducted. While these criteria may reduce sample variance and question the reliability of the results, they were based on previous research in our lab (Demaree & Harrison, 1997; Harrison & Gorelczenko, 1990; Mollet & Harrison, 2007; Williamson & Harrison, 2003) that has examined group differences in high and low hostile individuals across sensory and motor modalities. By continuing to use the same criteria, we are able to integrate research findings and develop a theoretical framework for brain function and behavior in a specific group of the population (i.e., high and low hostile individuals).

Despite the limitations of the experiment, there are several theoretical implications for prominent models of emotion processing as a result of the current experiment. In general, the results provide evidence against the valence model of emotional processing. According to the valence model, left hemisphere activation 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 and 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 prefrontal activation has been found concurrent with anger in a number of other projects (Harmon-Jones & Allen, 1998; Harmon-Jones & Sigelman, 2001; Harmon-Jones, Vaughn-Scott, Mohr, Sigelman, & Harmon-Jones,

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 that 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, Nitschke, Etienne, & Miller, 1997). Perhaps application of this approach to hostility will lead to new discoveries about cerebral activation during anger.

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. 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 and parietal locations and increased delta at the frontal location as a function of the cold pressor. However, when a more refined ANOVA was computed using only values from the frontal and temporal 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 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.

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. 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.

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