Healthy high-hostiles evidence low-alpha power (7.5–9.5 Hz) changes during negative affective learning

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Abstract

The present experiment was designed to better understand the impact of positive and negative emotional processing among low- and high-hostile individuals. Based on previous research which found increased sympathovagal balance among low-hostiles to the negative version of the Affective Auditory Verbal Learning Test (AAVL), it was hypothesized that low-hostiles would experience increased cortical arousal to this stimulus whereas their high-hostile counterparts would not. As expected, low-hostiles experienced significantly reduced low-alpha power (7.5–9.5 Hz) relative to high-hostiles during the presentation of the negative AAVL. In a replication of prior research, significant primacy and recency effects were noted for the negative and positive word lists, respectively. Results are discussed in terms of cerebral activation theory and the potential impact of emotional processing among high-hostile individuals and their likelihood to develop coronary heart disease.

Keywords: Hostility; Alpha power; Verbal learning; Emotional memory

1. Introduction

Personality differences have long been related to autonomic arousability. For example, Eysenck, in his Psychoticism-Extraversion-Neuroticism (PEN) model, postulated that personality factors are associated with arousal within both the reticular activation system (RAS) and visceral brain areas (VBA) (e.g., Eysenck, 1967, 1970). Based on his research, Eysenck suggested that “introverts” evidence increased chronic brain arousal from RAS activation whereas “neurotics” experience autonomic hyper-reactivity due to a low threshold for VBA arousal. Jeffrey Gray (1981), although he agreed with many of Eysencks suppositions, focused on impulsivity (high neuroticism and extraversion) and anxiety (high neuroticism but low extraversion) as important traits related to individual difference research. Gray proposed two motivational brain pathways called the Behavioral Inhibition (BIS) and Behavioral Activation Systems (BAS). The BAS is the appetitive motivation system that typically induces “approach” behavior and is generally associated with positive affect such as “hope” and “happiness” (e.g., Corr, 2001; Depue & Iacono, 1989; Fowles, 1994; Gray, 1990, 1994; Matthews & Gilliland, 1999, 2001) and may be associated with increased sympathetic arousal as measured by pre-ejection period (Beauchaine, 2001). The BIS, conversely, inhibits behavior in response to threatening stimuli and is most closely associated with negative affect such as fear and anxiety (e.g., Fowles, 1988, 1994; Gray, 1990, 1994; Matthews & Gilliland, 1999, 2001; Meyer, Johnson, & Winters, 2001) and may be associated with increased tonic skin conductance (Beauchaine, 2001). Clearly, BIS and BAS arousal are important to emotional experience and autonomic arousal.

Because it has been found to predict coronary artery disease (CAD) (Dembroski, MacDougall, Costa, &
Grandits, 1989; Hecker, Chesney, Black, & Frautschi, 1988; Littman, 1993; Miller, Smith, Turner, Guijarro, & Hallet, 1996; Siegman, Townsend, Civelek, & Blumenthal, 2000), hostility is an important, relatively discrete, personality trait. High-hostile individuals have been found to be particularly prone to CAD. Siegman and colleagues (2000), for example, found that hostility was a significant predictor of CAD with a relative risk ratio of 1.22. Results from two meta-analyses have independently supported the notion that hostility level increases risk for CAD (Booth-Kewley & Friedman, 1987; Miller et al., 1996). Furthermore, among persons diagnosed with CAD, hostility level is positively associated with disease severity and negatively associated with rate of recovery (Dembroski, 1985; Koshenuvo et al., 1988; MacDougall, Dembroski,Dimsdale, & Hackett, 1985; Powell & Thoresen, 1985; Williams Jr. et al., 1980). Given that cardiovascular disease accounts for about 40% of mortality within most industrialized nations (and is associated with enormous personal, emotional, and financial costs), a better understanding of how hostility increases the risk for CAD is clearly warranted (American Heart Association, 2001).

High levels of trait hostility have been found to predict increased physiological reactivity to mental and emotional stressors (Demaree & Harrison, 1997; Demaree, Harrison, & Rhodes, 2000; Everson, McKey, & Lovallo, 1995; Fredrickson et al., 2000; Smith & Gallo, 1999; Vogeley, 1998). For example, Demaree and Harrison (1997) determined that, relative to low-hostiles, high-hostile individuals evidenced significantly greater heart rate (HR) increases to the administration of a painful cold-pressor stimulus. Fredrickson and her colleagues (2000) further determined that high-hostile individuals had significantly increased blood pressure (BP) reactivity and decreased recovery to “relived anger” experiences. Indeed, although not without some debate (see Myrtek, 1995; Suls & Wan, 1993), a review of relevant literature supports the hostility–reactivity hypothesis (for a review, see Brummett & Williams Jr., 1998). It is important to note, however, that these autonomic data may be somewhat misleading because they are “end-organ” measures (e.g., HR, BP).

Because “end organ” measures are affected by both the sympathetic (“speeding”) and parasympathetic (“slowing”) nervous systems and are therefore relatively non-specific, the present researchers recently conducted a study designed to assess the impact of hostility on more discrete measures of autonomic arousal during positive and negative emotional processing (Demaree & Everhart, in press). Specifically, electrocardiogram (ECG) data were collected before, during, and after participants learned a positive or negative word list (known as the Affective Auditory Verbal Learning Test, or AAVL, Snyder & Harrison, 1997). ECG data were submitted for spectral analysis, yielding low frequency (LF, an indicator of sympathetic arousal; .05–.15Hz) and high frequency (HF, an indicator of parasympathetic arousal; .15–.40Hz) power. The ratio of these two variables (LF/HF) may represent sympathovagal balance, or the relative contribution of sympathetic to parasympathetic input at the sinoatrial node of the myocardium (Task Force, 1996). The Demaree and Everhart (in press) study yielded two important findings. First, at baseline, high-hostiles experience significantly reduced parasympathetic arousal relative to low-hostiles. Second, to the negative word list, low-hostiles surprisingly evidenced significant LF/HF increases during both reactivity and recovery phases whereas high-hostiles showed no significant changes. This latter finding, using more sensitive measures of autonomic functioning than have traditionally been used, is notable because it conflicts with previous conclusions drawn from “end-organ” data. It is important to note, however, that these disparate results may reflect the unusual nature of the task (i.e., the AAVL), which is significantly different from most stressors used in this line of research (e.g., cold-pressor, receiving verbal harassment while attempting unsolvable anagrams, etc.). Given Bernston, Cacioppo, and Quigley (1991, 1993) Theory of Autonomic Space—which stipulates that sympathetic and parasympathetic influences may act independently, reciprocally, or even coactively—we may not infer from the LF/HF data alone whether sympathetic or parasympathetic influences increased or decreased among any of our participants. However, since it was also found that the two Hostility groups evidenced no significant difference with regard to respiratory sinus arrhythmia (RSA, a sensitive indicator of parasympathetic arousal) during the negative AAVL, we may cautiously suggest that low-hostiles evidenced increased sympathetic arousal whereas high-hostiles did not.

To understand the hostility–CAD relationship, it is important to acknowledge that both decreased vagal tone and reduced autonomic flexibility may predict CAD (Brosschot & Thayer, 1998; Friedman & Thayer, 1998a, 1998b; Stein & Kleiger, 1999), both of which were found among high-hostile individuals (Demaree & Everhart, in press) to the negative AAVL. The present research was designed to better understand the neural correlates of emotional processing in hostility by determining the impact of the AAVL on cortical arousal, as measured by electroencephalography (EEG), among low- and high-hostile individuals. Because relative activation of the sympathetic nervous system is associated with increased glucose and oxygen utilization and an aroused and vigilant animal, it was hypothesized that low-hostiles would evidence increased cortical arousal to the negative AAVL (as indicated by decreased alpha power which is inversely associated with neural firing, see Davidson & Henriques, 2000; Glass, 1966; Lindsley & Wicke, 1974; Shagass, 1972) relative to high-hostiles.
Also, replicating previous research (Snyder & Harrison, 1997; Snyder, Harrison, & Shenal, 1998), it was hypothesized that a significant primacy effect would be found on the negative version of the AAVL whereas a significant recency effect would be found on positive AAVL.

2. Method

2.1. Participants

Right-handed male (N = 12) and female (N = 36) participants (N = 48) between the ages of 18 and 23 years were recruited from the undergraduate subject pool within the Department of Psychology at East Carolina University. All participants obtained extra credit toward their psychology course for participation. The research was approved by the University and Medical Center Review Board at East Carolina University.

2.2. Word lists

Negative and positive affective word lists of the AAVL were developed using the Toglia and Battig (1978) index of word norms, which replicated and expanded the work of previous researchers (Locascio & Ley, 1972). A total of 2854 words were evaluated by 2500 undergraduates at the University of Colorado using a 7-point Likert scale for concreteness, imagery, categorizability, meaningfulness, familiarity (FAM), number of attributes, and pleasantness (PLS). Construction of the affective lists of the AAVL test consisted of selecting 15 words having the highest mean PLS rating and 15 words having the lowest mean PLS ratings from the subset of all words with a FAM rating of at least 5.0 or greater (see Snyder & Harrison, 1997; Snyder et al., 1998). Positive and negative word lists were tape recorded onto TDK IECI/TYPE I audio tapes at a rate of approximately 2s/word and played at about 50dB on a Sony CFD-G30 CD Radio Cassette Player. A female actor was used for all voice recordings.

2.3. Quantitative EEG recording procedures

Recording procedures are identical to those previously described by Everhart and Demaree (2003). Participants were tested in the Cognitive Neuroscience Laboratory located within the Department of Psychology at East Carolina University. The participants were seated in a reclining chair and were fitted with a lycra electrode cap (Electro-Cap International). Within the electrode cap, electrodes are arranged according to the 10-20 international system (Jasper, 1958). Thus, EEG was recorded from 19 active electrode sites using linked ears (A1 and A2) as a reference (monopolar montage). The use of linked ears has been recommended for asymmetry research when using a specific, rather than computed, reference location (Davidson, 1998; but see Hagemann, Naumann, & Thayer, 2001). In order to record eye movements, one electrode was placed at the outer canthus of the left eye (horizontal eye movements), and one electrode was placed supraorbitally above the left eye (vertical eye movements). Thus, eye movements were recorded on two separate auxiliary channels. The EEG signal was amplified and converted on line to digital using a Lexicor Neurosearch—24 Brain Mapping System (Lexicor Medical Technology, 1992). The amplification factor was 32,000 with a sampling rate of 256 samples/s. A high-pass filter was used to eliminate slow wave frequencies that were less than 2 Hz, and a low-pass filter was used to eliminate fast wave frequencies that were more than 128 Hz. A 60 Hz notch filter was used to eliminate 60 Hz line noise. Data were stored and analyzed on a PC Pentium Computer. The EEG data were converted on line for display, storage, and analysis. Participants were instructed to close their eyes at all recordings. In total, four 1-min EEG samples were recorded for each participant (Baseline, immediately following learning trial 3 of the AAVL, immediately following trial 5 of the AAVL, and 5 min following completion of the AAVL). Artifacting was completed using a two-step process. First, epochs that contained signals that were greater than ±50 µV were automatically eliminated via computer software. Second, based on visual inspection of auxiliary channels, epochs that contained significant eye movements were eliminated. In all cases, at least 85% (i.e., 51 one-second epochs) of each of the 1-min recordings remained for Fast Fourier Transformation (FFT), and low- and high-hostile groups did not differ significantly in the amount of artifact-edited data. Power spectral analysis (using FFT) was subsequently performed on 1-s epochs; the data for each 1-s epoch were averaged together for each of the four 1-min recording periods.

2.4. Procedures

Upon arrival, participants were asked to read and sign an informed consent form. A brief medical history survey was used to eliminate participants with a history of head injury, drug use (e.g., alcohol, prescription drugs, etc.), psychological problems (e.g., depression, attention deficit disorder, etc.), and diagnosis of learning disabilities. Participants were asked to complete the behavioral laterality inventory (Coran, Porac, & Duncan, 1979) in order to ensure right-hand dominance. A score of +6 on the inventory was required for further participation. Participants were also administered the CMHO (Cook & Medley, 1954), a 50-item true/false questionnaire that is a valid predictor or medical, psy-
chological, and interpersonal outcomes (Han, Weed, Calhoun, & Butcher, 1995). Although several scales measuring “hostility” exist, the CMHO was selected primarily because of its inclusion in the Minnesota Multiphasic Personality Inventory. Previous studies that have used this measure have found good convergent and discriminant validity (Barefoot, Dodge, Peterson, Dahlstrom, & Williams, 1989). For instance, high correlations with other MMPI-2 scales suggest that the CMHO is related to general psychopathology and negative affectivity (Han et al., 1995). However, Smith and Frohm (1985) found that the CMHO correlates significantly higher with trait anger than self-reported trait anxiety or depression. Moreover, factor analysis of the CMHO reveal a general factor involving both “cynicism” and “distrust” (Greenglass & Julkunen, 1989).

The CMHO cut-off for hostility level for both men and women was 20 (“low-hostile” <20 and “high-hostile” >20). Participants in the low- and high-hostile groups were randomly placed into the positive (Low Hostility, N = 12 and High Hostility, N = 13) and negative (Low Hostility, N = 10 and High Hostility, N = 12) AAVL conditions, resulting in roughly equivalent cell sizes. The experimenter was blind to participant hostility level. Upon completion of questionnaires, the electrode application procedures described above were initiated. Following preparation and after a 10-min adaptation period, participants were asked to provide a numerical rating of overall mood state using a Likert scale (presented visually) where +10 was considered an “extremely positive” mood state and −10 was considered an “extremely negative” mood state. Immediately after response, the experimenter left the room, and baseline EEG recording was initiated for the duration of 1 min. Depending on arrival, a positive or negative words list was read to the participants using a previously recorded audiotape of a female voice reading the lists at about 1 words/s. Instructions were also read by the female voice on the audiotape, which was played through the intercom system at about 50 dB. Participants received the following instructions from the original RAVL on Trial 1: “I am going to read you a list of words. Please listen carefully. When I stop, you are to say back as many words as you can remember. Say the words in any order you remember. Just try to remember as many as you can.” Instructions for Trial 2 through Trial 5 were also as follows: “Now I’m going to read the same list again. When I stop again, I want you to tell me as many words as you can remember, including words you said the first time. It doesn’t matter what order you say them. Just say as many words as you can remember whether or not you said them before.” When the participant could no longer recall any more words or a maximum of 3 min had past, the next trial began. Participants’ responses were recorded on the data sheet. Following completion of the fifth learning trial, participants were again asked to provide a Likert scale rating of mood state using the same visual scale described above.

The experimental procedures are identical to those described by Snyder and Harrison (1997) with the exception of EEG recording. In total, four 1-min samples were recorded for each participant. A 1-min baseline recording (Baseline) was initiated following a 10-min adaptation period and prior to initiation of the AAVL. One-min samples were also recorded immediately after the third trial of the AAVL (T3), immediately after the final trial of the AAVL (T5), and 5 min following completion of the AAVL (Recovery). After the final EEG recording, participants were debriefed and thanked for their participation.

3. Results

3.1. Behavioral data: Learning/recall patterns

Independent analyses of variance (ANOVAs) were performed on three dependent memory variables—primacy (words 1–5), recency (words 11–15), and overall memory data. Each ANOVA was performed with independent factors of Hostility (low and high), Valence (positive and negative word lists from the AAVL), and a repeated measure of Trial (1–5). All pairwise comparisons were made using Tukey’s Honestly Significant Difference test to control for experimentwise error ratio (Winer, 1971).

For Primacy data, analysis revealed a significant main effect of Valence [F(1, 44) = 32.43, p < .001, d = 1.65]. Fewer primacy words were recalled from the positive version of the AAVL (M = 3.59; SD = .70) than from the negative (M = 4.53; SD = .37). A significant main effect of Trial was also found [F(4, 176) = 22.81, p < .001]. Fewer primacy words were recalled during Trial 1 (M = 3.29; SD = 1.16, dtrials 1-2 = 0.65) than during Trial 2 (M = 3.94; SD = 1.26, dtrials 1-2 = 0.65), Trial 3 (M = 4.18; SD = .96, dtrials 1-3 = .90), Trial 4 (M = 4.32; SD = .78, dtrials 1-4 = 1.04), and Trial 5 (M = 4.57; SD = .68, dtrials 1-5 = 1.29). Moreover, fewer words were recalled during Trials 2 (dtrials 2-3 = 0.63) and 3 (dtrials 3-5 = 0.39) and 4 (dtrials 4-5 = 0.25) relative to Trial 5. A Valence by Trial interaction effect was also found for Primacy data [F(4, 176) = 3.39, p < .01]. Relative to negative words, fewer positive AAVL words were recalled during Trials 1, 4, and 5, but not Trials 2 and 3.

For recency data, analysis revealed a significant main effect of Valence [F(1, 44) = 20.51, p < .001, d = 1.17]. Specifically, fewer recency words were recalled from the negative version of the AAVL (M = 3.15; SD = .68) than from the positive (M = 3.85; SD = .52). A significant main effect of Trial was also found [F(4, 176) = 36.45, p < .001, d]. Specifically, more recency words were...
were recalled during Trial 3 ($M = 3.86; SD = 1.05$, $d_{\text{trials 1-3}} = 1.38$, $d_{\text{trials 2-3}} = .72$), Trial 4 ($M = 3.99; SD = .94$, $d_{\text{trials 1-4}} = 1.51$, $d_{\text{trials 2-4}} = .84$), and Trial 5 ($M = 4.15; SD = .94$, $d_{\text{trials 1-5}} = 1.66$, $d_{\text{trials 2-5}} = .99$) than during Trial 1 ($M = 2.40; SD = 1.30$), and Trial 2 ($M = 3.10; SD = 1.01$).

For total recall data, analysis revealed a significant main effect of Trial [$F(4,176) = 144.99$, $p < .001$]. The number of words recalled increased significantly from Trial 1 ($M = 7.37; SD = 1.69$) to Trial 2 ($M = 9.93; SD = 1.95$, $d_{\text{trials 1-2}} = 1.37$) and from Trial 2 to Trial 3 ($M = 11.70; SD = 1.97$, $d_{\text{trials 2-3}} = .95$). The number of words recalled also increased significantly from Trial 4 ($M = 12.04; SD = 1.95$) to Trial 5 ($M = 12.72; SD = 1.76$, $d_{\text{trials 4-5}} = 0.36$).

3.2. Behavioral data: Self-report of mood state

Pretest and posttest ratings on mood state using the Likert scale previously described were recorded. The ratings were converted into one score (Pre–Post) for comparison purposes. One-way ANOVA yielded a significant difference between the positive and negative word lists [$F(1,44) = 6.09$, $p < .01$]. Participants who received the negative word list ($M = 1.28; SD = 1.88$) reported a greater decline in mood state (i.e., toward the negative direction) than did participants who received the positive word list ($M = .29; SD = 1.21$, $d = 0.64$). Thus, mood state is affected differently by the two word lists.

3.3. Data reduction and QEEG analyses

Analyses were restricted to frequencies within the alpha band (8–13 Hz) and to the following electrode sites: F3, F4; F7, F8, T3, T4; T5, T6; C3, C4; P3, P4; and O1, O2. Initially, the alpha band was separated into three components including low alpha (7.5–9.5 Hz), mid alpha (9.5–11.5 Hz), and high alpha (11.5–13.0 Hz), as recommended by (Crawford, Clarke, & Kitner-Triolo, 1996). The rationale for separating the alpha band is supported in that there are functionally different rhythmic alpha components (Barlow, 1993), and factor analyses have indicated that there are three discrete alpha band factors (Andresen, Stemmler, Thom, & Irrgang, 1984). Moreover, Crawford et al. (1996), found that only the low-alpha band successfully differentiated between positive and negative mood states, and a previous study in our laboratory that utilized positive and negative affective word lists found differences only within the low-alpha band (Everhart & Demaree, 2003). In order to improve homogeneity of variance (Davidson, 1998; Davidson & Henriques, 2000; Winer, 1971) band values were transformed using the log(x). Three independent ANOVAs were performed (one for each component) with the between factors of Hostility (low and high) and Valence (positive or negative) and the repeated factors of Hemisphere (left and right), Site (F3,F4; F7,F8; T3,T4; C3,C4; P3,P4; and O1,O2), and Trial (Baseline, Trial 3, Trial 5, Recovery). The relative contribution of each significant effect was assessed by computing the percentage of the total treatment variance (abbreviated as “ptv”) that was due to that effect. Of note, any effect that accounted for less than one half of one percent of the total treatment variance was treated as trivial and dismissed. Initial analyses found no interactions or main effects involving hostility or valance within the mid- (9.5–11.5 Hz) and high-alpha (11.5–13.0) bands. These frequency bands were no longer considered for further analyses.

For the low-alpha band, there were significant main effects for site [$F(6,38) = 245.64$, $p < .001$, $ptv = 75.4$] and hemisphere [$F(1,43) = 27.88$, $p < .001$, $ptv = 0.8$]. Alpha power was significantly greater over the right hemisphere ($M = 573, SD = .440$) than over the left hemisphere ($M = .524, SD = .424, d = .11$). The only significant and non-trivial interaction was Hostility × Site × Valence [$F(6,38) = 2.80$, $p = .024$, $ptv = 1.0$]. This interaction was further investigated by examining the Hostility × Site interaction at each level of valence. The Hostility × Site interaction was significant with the negative words [$F(6,15) = 3.10, p = .035, ptv = 4.9$], but not with the positive words [$F(6,18) = .71, p = .64$]. The significant Hostility × Site interaction with the negative words was further investigated by evaluating the effect of hostility at each site. Although mean low-alpha power was greater for high-hostiles than for low-hostiles at every site (see Fig. 1), the difference reached statistical significance only for the occipital sites [$F(1,20) = 5.67, p = .027, \eta^2 = .22$]. At the

![Fig. 1. Mean low-alpha (7.5–9.5 Hz) power for low- and high-hostiles during the negative word list of the AAVL. F3–F4, mid frontal; F7–F8, mid temporal; T3–T4, anterior temporal; T5–T6, posterior temporal; C3–C4, central; P3–P4, parietal; O1–O2, occipital. Note. The site pairs listed above reflect the average of left and right hemisphere scalp sites. Decline in alpha power is inversely correlated with brain activation.](image-url)
occipital site, the mean for high-hostiles was 1.10 (SD = .59), while the mean for low-hostiles was .59 (SD = .36), d = 1.02.

### 3.4. QEEG analyses using hostility as a continuous variable

Data were also analyzed using the independent variable of hostility as a continuous measure rather than dichotomous (i.e., low and high hostility). A preliminary polynomial regression analysis indicated that the effect of hostility on alpha power was linear. The factorial analysis produced a pattern of significant effects that were exactly the same as that observed when the analysis was completed with dichotomized hostility. The significant Hostility x Site x Valence interaction from this analysis is illustrated in Fig. 2. The correlation between hostility and low alpha was greater for negative words than for positive words at every site, but that correlation was significant only for the occipital sites (r = .45, p < .04). Regarding positive words, all correlations were trivial (r < .21 for each site), and none reached significance. In summary, the pattern of results found during exploratory analyses using hostility as a continuous variable was essentially identical to the findings reported for low- and high-hostiles (i.e., dichotomous). Thus, reported hostility level was positively associated with alpha power during negative affective learning.

## 4. Discussion

The purpose of the present experiment was to examine changes in brain arousal among low- and high-hostiles during positive and negative emotional learning. As has previously been discussed, high-hostiles evidence reduced sympathovagal reactivity to the negative AAVL whereas low-hostiles evidence significant increases of LF/HF to these stimuli (Demaree & Everhart, in press). While it remains unclear why high-hostiles experienced reduced reactivity relative to low-hostiles (although we speculate that it is due to the nature of the stressor, i.e. the AAVL, or a sign of autonomic “inflexibility”), understanding the cerebral mechanisms which underlie these disparate autonomic reactions is important because reduced autonomic flexibility and decreased parasympathetic arousal may be an important predictor of CAD (Brosschot & Thayer, 1998; Stein & Kleiger, 1999). Because the central nervous system directly affects autonomic processing (e.g., Lane & Jennings, 1998), the present experiment was designed to determine the impact of negative list learning among low- and high-hostiles on low-alpha power (which is reciprocally related to brain arousal). The primary finding of the present experiment is that, during processing of the negative AAVL, low-hostiles experienced significantly decreased low-alpha power within occipital brain regions relative to their high-hostile counterparts. This discrepancy of low-alpha power was expected as low-hostiles also experience increased sympathovagal balance in response to the same stimuli (Demaree & Everhart, in press). This finding is consistent with a previous experiment that used a similar age group (although hostility level was not examined) in which decreased alpha power was observed using the same negative word list of the AAVL (Everhart & Demaree, 2003). Similarly, no significant findings were observed for the positive word list.

Brain arousal differences in response to the negative AAVL may reflect trait differences to negative affect. While relatively little research has examined the interaction of hostility and affective valence on brain arousal, prior research may support this conclusion. For example, Raine, Lencz, Bihrl, LaCasse, and Colletti (2000) found that community-dwelling persons with Antisocial Personality Disorder have an 11% reduction of prefrontal gray matter (decreased cortical arousal) and experience significantly less autonomic arousal in response to a stressor as compared to psychiatric controls. Also, Blackburn (1975) found that hostility level of 80 men housed in a secure psychiatric facility was significantly related to decreased alpha power in response to the aversive cold-pressor test.

It is interesting to note that EEG alpha power biofeedback has been used to modify emotional responses to affective stimuli. For example, Allen, Harmon-Jones, and Cavender (2001) found that biofeedback promoting greater right-relative to left-frontal alpha power led to a more positive affective response (verified by facial electromyography) to evocative film clips. The present research indicates that, among high-hostile individuals, biofeedback designed to decrease posterior alpha power during negative emotional processing may reduce...
cardiovascular risk by allowing more variable autonomic response.

It should be noted that group cerebral arousal differences in response to the negative AAVL may be explained, at least in part, by memory load. It has been well established that increasing memory load causes decreased RSA within the encoder (Aasman, Mulder, & Mulder, 1987; Redondo & Del Valle-Inclán, 1992). Because low-hostiles may have a reduced number of associations with negative words (e.g., murder, kill, etc.), the negative AAVL may represent a more difficult memory task for them relative to their high-hostile counterparts. Consequently, low-hostiles may evidence increased posterior arousal when modulating sympathovagal balance whereas high-hostiles do not.

It is interesting to note that significant findings were isolated to the low-alpha power, as no group differences in affective learning were observed for mid- and high-alpha bands. This finding is also consistent with our previous study (Everhart & Demaree, 2003) that utilized the same positive and negative word lists. The previous study reported no differences for other commonly used band-widths (i.e., delta, theta, and beta). The reason for the isolation of significant differences to the low-alpha band is uncertain. It is possible that this particular band-width is more sensitive to the demands of the negative word list of the AAVL. Similarly, other laboratories have reported that the low-alpha band may be optimal for the examination of differences in emotion processing or differentiation of mood states (see for example, Crawford et al., 1996) or possibly for sustained attentional abilities during the performance of tracking or decision making tasks (Crawford, Knebel, Vendemia, Kaplan, & Ratcliff, 1995; Crawford & Vasilescu, 1995). Future studies will attempt to disentangle the relationship between various EEG frequency bands, cognitive function, and emotion processing.

The cerebral mechanisms involved in the regulation of hostility, autonomic function (i.e., sympathovagal balance), and emotional learning are poorly understood. It is well known that the cerebral cortex is involved in the regulation of autonomic function. For instance, direct projections exist between various cortical structures (prefrontal cortex, the cingulate cortex, and the temporal lobe) and the subcortical structures (amygdala, hypothalamus, brainstem, and spinal cord) that are thought to regulate sympathovagal balance (Lane & Jennings, 1998). Of note, however, the role of cortical structures in autonomic regulation may be modulatory rather than primary. Perhaps the structure that is most relevant to the present study is the amygdala. The amygdala is thought to act as a transmodal gateway for linking sensory representations with mental and autonomic correlates of emotional and motivational valence (Mesulam, 2000). Thus, the amygdala modulates the hippocampus and plays a large role in memory for emotional events (Cahill & McGaugh, 1998). Indeed, amygdala activity during viewing of emotional stimuli correlates with long-term explicit memory for the stimuli (Canli, Zhao, Brewer, Gabrielli, & Cahill, 2000). Amygdaloid activity is also associated with aggression (Dicks, Myers, & Kling, 1969) and with the actions of various stress hormones (McGaugh, 2000).

Others have speculated that disruptions within the circuits involving the amygdala and its connections may in part be associated with a wide-range of human psychopathology (Mesulam, 2000; Packard & Cahill, 2001). With regard to the present experiment, it is plausible that baseline differences in resting amygdala activity among high- and low-hostiles may account for the differences noted in sympathovagal balance (Demaree & Everhart, in press) and the cortical arousal observed during emotional learning in the current experiment. The authors speculate that differences in emotion and motivation between high- and low-hostiles may produce differences in encoding strategy and thus, less cortical arousal is seen among high-hostiles during negative word learning.

One shortcoming of the present study is that no attempts were made to control for the potential use of differing encoding strategies between low- and high-hostiles. In future studies, it will be important to investigate the possibility of such differences. Moreover, the experimental design does not permit examination of the function of subcortical structures, notably the amygdala, as EEG is at best a crude measure of brain electrical activity. Given this, our inferences are only speculative (although consistent with current theories regarding the function of the amygdala). Another limitation of the study lies in the inability to examine for potentially meaningful gender differences, and it is well known that men and women process emotional stimuli differently (see for example Everhart, Shucard, Quatrin, & Shucard, 2001). More specifically, the total sample in this study is comprised primarily of women (75%) and thus only three men were included in each of the four cells. Given this, reliable examination of possible gender-related differences cannot be performed in this study.

References


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