

Reprint (1-2)5

# Hostility Following Right CVA: Support for Right Orbital Frontal Deactivation and Right Temporal Activation

**D. Erik Everhart, M.S., and David W. Harrison, Ph.D.**

*There is consensus among researchers that the right versus left cerebral hemisphere is specialized for the experience of negative emotion. There is currently a need for research which specifically investigates cortical theories of hostility. Topographical brain mapping and QEEG techniques were used to test hypotheses of concurrent decreased activation of the right orbital-frontal region and increased activation of the right temporal region during the experience of hostility. The patient was a 44-year old, right-handed female with status-post right CVA, left hemiparesis, and feelings of hostility and suspiciousness toward others. Results support a priori hypotheses, in that a relative decrease in beta activity was displayed over the right orbital frontal region concurrent with a relative increase in beta activity over the right temporal region. Implications for the utility of topographical brain mapping and QEEG in the neuropsychological evaluation are discussed.*

## Introduction

There is consensus among researchers that the right cerebral hemisphere is specialized relative to the left cerebral hemisphere for the experience of negative emotions (Heilman, Bowers, & Valenstein, 1993; Davidson & Fox, 1984; Tucker, 1981). Research using the electroencephalogram supports the notion that the right hemisphere is specialized for the experience of negative emotions. For example, in an investigation of EEG changes during induced emotional states, the right frontal cortex displayed an increase in activation during negative affect while a similar activation was found in the left frontal cortex during positive affective states (Davidson, Schwartz, Saron, Bennett, & Goleman, 1979). Similar results were found when Ahern and Schwartz (1985) examined the EEG records of subjects who were asked emotion-laden questions. Results indicate left-hemispheric activation in the frontal zones for positive emotions and right-hemispheric frontal activation for negative emotions. Support for right-hemispheric activation during experienced negative emotion has been further bolstered by the findings of Davidson, Ekman, Saron, Senulis, and Frieman (1990). During this study, subjects watched films to promote happiness or disgust while EEG and facial expression were concurrently recorded. Disgust was found to be associated with right-sided activation in the frontal and anterior temporal regions, whereas happiness was accompanied by left-sided anterior temporal activation.

There is currently a need for more specific research on hostility. The research thus far suggests temporal lobe involvement in the experience of hostility. For example, investigation of a sample of 141 patients with brain tumors with cortical lesions caused by microsurgical tumor resection

indicated that patients with lesions of the ventral frontal cortex or lesions of the temporoparietal cortex reported worse mood states (irritability/anger) than did patients with other lesions (Irle, Peper, Wowra, & Kunze, 1994). Edlund, Swann, and Clothier (1987) also described temporal lobe involvement with the experience of hostility. Six patients with atypical panic disorder involving hostility, irritability, severe derealization, and social withdrawal all evidenced temporal lobe EEG abnormalities.

A neuropsychological explanation of hostility would suggest that the anterior right cerebrum is responsible for the inhibition or regulation of autonomic functioning as well as the expression of heightened hostility. The orbital-frontal cortex has extensive interconnections with the amygdaloid bodies of the anterior temporal region. The latter region has been frequently described as responsible for increasing hostility. Heilman et al. (1993) hypothesize that these two extensively interconnected regions interact with each other to yield a relatively conservative and stable aggression level among normals. Thus, ablation of right orbitofrontal regions or stimulation of right anteriomedial temporal regions may yield anger/aggression. Similarly, passivity or flattened affective expression may result from stimulation of right orbito-frontal regions or ablation of right anteromedial temporal regions.

Recently, Everhart, Demaree, and Harrison (1995) found support for the right orbito-frontal deactivation/anteromedial temporal activation hypothesis in an individual with extreme hostility toward others. In this case study using topographical brain mapping and QEEG, marked bilateral delta activity as well as heightened right parietaltemporal beta activity was noted following a closed head injury. Reportedly, there were no indications of heightened hostility prior to the injury.

The present hypothesis was also tested in the following case study of a female patient with status-post right CVA using topographical brain mapping procedures and QEEG, a relatively new technology for examining specific regions of cortical activity. In phase one of the present experiment, topographical brain mapping and QEEG are used to test the hypothesis of heightened activity over the right temporal lobe relative to the left temporal lobe. In phase two of the experiment, oscillation of the left upper extremity is used to augment the patient's symptoms and to elicit verbal discourse in which the patient can communicate her feelings or associated cognitions. It was hypothesized that right temporal lobe activation would be induced by this procedure, as well.

### **Case Study**

A 44-year old, right-handed female presented with one-year status-post right CVA, left hemiparesis, and complaints of angry feelings towards others, perceived movement in a vertical plane with floating sensations, and "black, spidery" visual phenomena at her left visual field towards the midline. Neuropsychological evaluation, which utilized a number of neuropsychological tests and syndrome analysis, suggested that dysfunction was within the right parietotemporal region.

### **Method**

Topographical brain mapping and QEEG were used in two phases in order to evaluate the patient. During phase one, QEEG was recorded from 21 scalp locations while the patient was asked to lie motionless with eyes open in a supine position on a reclining chair. The chair was located in a sound attenuated chamber. A lycra electrode cap was fitted according to the appropriate anatomical marks on the subject's head. The cap was attached via elastic straps to a body harness around the subject's chest to ensure that the cap was securely positioned. QEEGs were recorded from 21 scalp locations and referenced to the earlobes. Impedance levels at each electrode site were measured to be below 10 kohm. A total of 120 one-second epochs of data were recorded for phases. A sampling rate of 128 was used and a high pass filter eliminated frequencies below 2 Hz. Epochs contaminated by eye-blinks and/or movement artifact were eliminated from the analysis. The magnitude (m V) of standard beta (13-21 Hz) and delta (2-4 Hz) across remaining epochs was assessed using the Neurosearch-24 (Lexicor Medical Technology, 1992).

During phase two the procedure was identical to that used in phase one, with the exception of the utilization of a left, lateral arm oscillation procedure. Specifically, the patient's left arm was oscillated by the examiner. It should be noted that during phase two, the patient reported increased feelings of hostility. The oscillation procedure had been reliably used during the previous neuropsychological examination to induce hostility and complaints of dissociation (i.e., floating sensations). Epochs containing artifact were eliminated from analysis. The magnitude (LV) of standard beta (13-21 Hz) and delta (2-4 Hz) were also assessed for phase two.

## Results

As indicated above, the magnitude (V) of beta (13-21 Hz) was assessed during two conditions. During the eyes open (phase one) condition, beta asymmetry between left and right temporal lobes was indicated (see Table 1).

Specifically, the right anterior temporal region (T4) displayed greater activation or arousal than the left anterior temporal region (T3). In contrast, the left posterior temporal region (T5) demonstrated greater activation or arousal than the right posterior temporal region (T6). Additionally, beta activity over left and right parietal regions (P3 and P4, respectively) was symmetrical, while the left orbital frontal region (F7) demonstrated greater activity than the right orbital-frontal region (F8). No marked asymmetries (See Table 1) were noted in Delta (2-4 Hz) activity within the cortical regions discussed above (F7, F8, T3, T4, T5, T6, P3, P4,)

During the second phase (lateral arm oscillation procedure), the patient expressed feelings of floating and falling to the left and backwards with oscillation of the limb, as well as an increase in hostility.

Analyses of data revealed a slight decrease in beta activity over the right orbital frontal region (F8) and a marked increase in beta activity in right temporal regions (T4 and T6). Beta activity over the right parietal region appeared to remain relatively consistent through both phases. The lateral arm oscillation procedure might have been expected to increase activity over C4. However, beta activity over this region remained constant at 9.1 kV during both phases. A topographical -display of beta activity is seen in Figure 1. Note the demonstrated deactivation over the right orbitalfrontal region and activation over the right temporal region for beta (13-21

Hz) activity.

**Table 1 - Magnitude (mV) of Beta (13-21 Hz) and Delta (2-4 Hz) activity during eyes-open condition (phase one.)**

	Left Hemisphere		Right Hemisphere	
<b>Beta</b>	F7	6.4	F8	5.7
	T3	6.3	T4	9.0
	T5	13.8	T6	11.9
	P3	11.4	P4	11.4
<b>Delta</b>	F7	4.3	F8	3.4
	T3	2.9	T4	2.4
	T5	4.2	T6	3.9
	P3	5.1	P4	4.8

**Table 1 - Comparison of Magnitude (mV) of Beta (13-21 Hz) and Delta (2-4 Hz) activity during eyes-open condition (phase one) and lateral am condition (phase two).**

	Left Hemisphere		Right Hemisphere	
<b>Beta</b>	F7	5.7	F8	5.4
	T3	9.0	T4	17.8
	T5	11.9	T6	20.4
	P3	11.4	P4	11.0
<b>Delta</b>	F7	3.4	F8	6.6
	T3	2.4	T4	6.7
	T5	3.9	T6	8.3
	P3	4.8	P4	7.9

Delta activity (2-4 Hz) during phase two of the experiment increased over all cortical regions discussed above. Table 2 demonstrates increased delta activity within the right cortical regions.

### Discussion

Taken together, these results corroborate with findings from the neuropsychological evaluation, suggesting dysfunction within the right parieto-temporal region. These findings support previous experimental findings, where relative right orbital frontal lobe deactivation was found concurrent with relative right temporal lobe activation during increased hostility following right CVA. The

findings from this case study were also consistent with Everhart et al. (1995) where heightened delta activity was found in the right orbital frontal region and an increase in beta activity was **found** in the right versus the left temporal region. In the present investigation induction occurred during the lateral arm oscillation procedure, suggesting right anterior deactivation and posterior activation concurrent with the patient's reported experience of hostility. Reported hostility was consistent with the behavioral display of anger, facial configuration, and affective prosody.

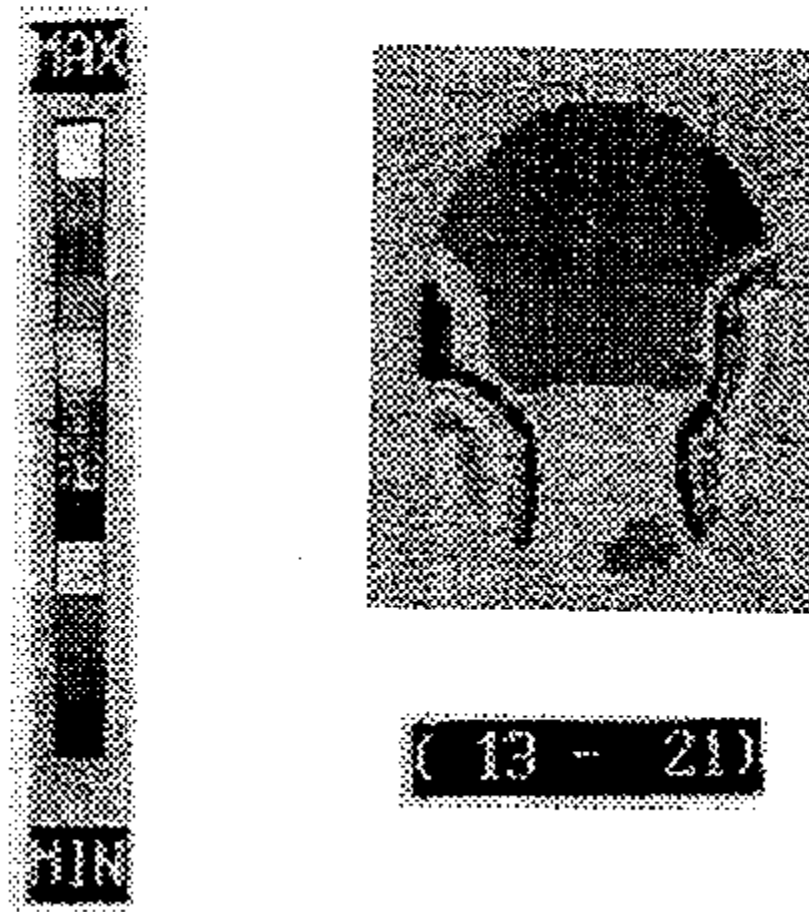
At present, there are few, if any, group studies which have investigated neurophysiological correlates of hostility in individuals with right CVAs via topographical brain mapping and QEEG. The lack of research in this area may be due to the difficulty in obtaining a group of compliant and hostile individuals, or due to the relatively underdeveloped utility of topographical brain mapping and QEEG in neuropsychological evaluation and theory-driven hypothesis testing.

It should be noted that there are certain limitations when using standard EEG scalp, locations (i.e., F8, F1) to describe precise neuroanatomical regions (such as orbital frontal). In general, only 21 scalp locations are used to assess the activity of numerous cortical regions. Thus, some degree of speculation is required when assessing cortical activity through use of EEG data.

The concept of activation with respect to topographical brain mapping and QEEG is also an area that at present has limitations. More specifically, there is not clear consensus within the literature as to which bandwidths should be used to describe activation and deactivation. Future studies with hostile individuals may advance the knowledge base regarding the concept of cortical activation and bandwidth use.

The next logical step with research of neuropsychological theories of hostility appears to entail a large group study of hostile individuals. Continued use of topographical brain mapping and QEEG will assist in the refinement of current techniques, as well as advances in the understanding of neuropsychological theories of hostility.

**Figure 1**  
**Beta Activity During Phase 2-Lateral Arm**  
**Oscillation Procedure**



Use of topographical brain mapping and QEEG appear increasingly promising. Duffy (1994) has suggested that QEEG might be increasingly used for independent confirmation of psychological hypotheses, and assistance in determining functional correlates of activation of specific cortical regions. QEEG also potentially promises to provide valuable assistance to the clinical neuropsychological evaluation, as well as support for neuropsychological theories of emotion such as hostility.

#### **References**

**Ahern, G. L., & Schwartz, G. E. (1985). *Differential lateralization for positive and negative emotion in the human brain: EEG spectral analysis. Neuropsychologia, 23(6), 745-755.***

**Davidson, R. J., Ekman, P., Saron, C. D., Senulis, J. A., & Friesen, W V (1990). *Approach-withdrawal and cerebral asymmetry: Emotional expression and brain physiology I. Journal of Personality and Social Psychology, 58(2), 330-341.***

**Davidson, R. J., Schwartz, G. E., Saron, C., Bennett, J., & Goleman, D. J. (1979). *Frontal versus parietal asymmetry during positive and negative affect. Psychophysiology, 16, 202-203.***

**Duffy, F. H. (1994). *The role of quantified electroencephalography in psychological research. In G. Dawson & K. W Fischer (Eds.), Human behavior and the developing brain (pp. 93-133). New York: The Guilford Press.***

**Edlund, M. J., Swann, A. C., & Clothier, J. (1987). *Patients with panic attacks and abnormal EEG results. American Journal of Psychiatry, 144(4), 508-509.***

**Everhart, D. E., Demaree, H. A., & Harrison, D. W. (1995). *'Ibtopographical brain mapping: Hostility following closed head injury. The Clinical Neuropsychologist, 9(3), 280.***

**Fox, N. A., & Davidson, R. J. (1984). *Hemispheric substrates for affect: a developmental model. In N. A. Fox & R. J. Davidson (Eds.), The psychobiology of affect development. Hillsdale, NJ: Erlbaum.***

**Heilman, K. M., Bowers, D., & Valenstein, E. (1993). *Emotional disorders associated with neurological disease. In K. M. Heilman & E. Valenstein (Eds.), Clinical Neuropsychology (3rd ed., pp. 461-497). New York: Oxford University Press.***

**Irle, E., Peper, M., Wowra, B., & Kunze, S. (1994). *Mood changes after surgery for tumors of the cerebral cortex. Archives of Neurology, 5(2), 164-174.***

**NeuroSearch-24. (1992). Lexicor Medical Technology, Inc. Boulder, CO.**

**Tucker, D. M. (1981). *Lateral brain function, emotion, and conceptualization. Psychological Bulletin, 89, 19-46.***

**About the Authors:**

*D. Erik Everhart, M.S., received his M.S. degree in 1995 from Virginia Polytechnic Institute and State University. He is interested in cortical features of anxiety and hostility, and how increased cortical arousal associated with anxiety and hostility may influence perception and expression of emotion. At present he is pursuing his Ph.D. in Clinical Psychology.*

*David W. Harrison, Ph.D., received his Ph.D. in 1983 and completed his postdoctoral fellowship in 1985 at the*

## ISNR Copyrighted Material

*University of Georgia at Athens. He currently is associate professor at Virginia Polytechnic Institute and State University in Blacksburg, VA, and is interested in the neuropsychology of emotion.*

*Address correspondence to: David W Harrison, Ph.D., Department of Psychology, Virginia Polytechnic Institute and State University, Blacksburg, VA 24061.*