BIO ACCENT

BAOJ Neurology

Harrison DW, et al., BAOJ Neuro 2015 1:1
1: 001

Research Article

Quantitative Electroencephalogram Assessment of Expressive Aphasia

Foster PS[1], Harrison PK[2], Williamson JB[3], Campbell RW[2], Harrison DW[2*]

[1]*Department of Psychology, Middle Tennessee State University, USA*

[2]*Department of Psychology, Virginia Polytechnic Institute and State University, USA*

[3]*Department of Neurology, University of Florida, USA*

Abstract

Research has indicated that expressive aphasia results from left frontal lobe dysfunction, whereas expressive aprosodia has resulted from right frontal lobe dysfunction. The neuroanatomical localization of nonfluent aphasia has been investigated using numerous different neuroimaging procedures, such as computerized tomography, magnetic resonance imaging, positron emission tomography, and measures of regional cerebral blood flow. However, investigations using quantitative electroencephalography (QEEG) have only rarely been reported. The present investigation reports the case of an individual dually diagnosed with nonfluent aphasia and dysprosodia for whom standardized testing and syndrome analysis had been conducted. The results from this neuropsychological evaluation were then used to generate an a priori hypothesis of diffuse bilateral frontal lobe dysfunction. This hypothesis was then testing using QEEG. The results supported the impression of bilateral frontal lobe dysfunction. It was also found that increasing symptom severity correlated with increasing levels of brain dysfunction.

Keywords: Aphasia; Aprosodia; Quantitative Electroencephalography; QEEG; Expressive Speech; Frontal Lobe; Brain Asymmetry; Laterality; Speech; Language.

Quantitative Electroencephalogram Assessment of Expressive Aphasia

Aphasia refers to a class of neuropsychological disorders that involve the loss or impairment of language as a result of brain damage [2]. Aphasia may result from many different types of brain insults that produce lesions, such as vascular disorders, trauma, neoplasms, and infections [5]. However, the neuroanatomical location of the insult, rather than the etiology of the insult, is the crucial factor in determining aphasic symptomatology [5]. Indeed, the neuroanatomical site of the brain pathology and the associated syndrome is the distinguishing factor for the many different classifications of aphasia that exist. Broadly speaking, perisylvian aphasias refer to those classifications of aphasia that result from brain pathology located within the immediate vicinity of the Sylvian fissure. Conversely, aphasias that result from brain pathology that is not within the immediate vicinity of the sylvian fissure are referred to as extrasylvian or transcortical aphasias [4, 5]. The perisylvian aphasias may be further classified as to whether they are fluent (Wernicke's) or nonfluent (Broca's). Fluent aphasia involves impairment in auditory verbal comprehension and the repetition of spoken language. Additionally, the fluent speech may present as odd in nature or completely meaningless [5, 9, 16].

As an expressive speech deficit, nonfluent aphasia is typically associated with sparse verbal output, the use of short sentences, and many intervening pauses. Speech, in addition to being nonfluent, may be poorly articulated and produced with considerable effort. Unlike fluent aphasia, however, the comprehension of speech is typically intact [4, 5, 9, 6, 24]. Nonfluent aphasia also has resulted in the appearance of stuttering [5, 30]. Furthermore, individuals with nonfluent aphasia may exhibit mild aprosodia, or alterations in the rhythm, inflection, melody, and pitch of their speech [5, 7, 33].

Although discrepant findings concerning the neuroanatomical location of nonfluent aphasia may be found [3, 10], the vast majority of research implicates frontal lobe dysfunction in producing nonfluent aphasia [1, 5, 6, 8, 10, 12, 14, 17, 19, 27, 29, 13]. More specifically, nonfluent aphasia is associated with dysfunction within the opercular portion of the third transverse frontal convolution, or Brodman's area 44 [2, 8, 24]. Lesions producing nonfluent aphasia may also involve the lower portion of the motor strip or other cortical areas either anterior or superior to Brodman's area 44 [8]. Consistent with the neuroanatomical locations involved with nonfluent aphasia, stuttering has also been found to result from lesions within the left frontal lobe [23, 31, 32]. Right hemisphere dysfunction, in contrast, has been implicated in other speech disorders [22], particularly with the dysprosodic aspect of speech [33].

The finding that nonfluent aphasia and aprosodia result from left

***Corresponding author:** Harrison DW, Department of Psychology, Behavioral Neuroscience Laboratory, Virginia Polytechnic Institute, USA, E-mail: dwh@vt.edu

Sub Date: March 10, 2015, **Acc Date:** March 24, 2015, **Pub Date**: March 26, 2015

Citation: Foster PS, Harrison PK, Williamson JB, Campbell RW, Harrison DW (2015) Quantitative Electroencephalogram Assessment of Expressive Aphasia. BAOJ Neuro 1: 001.

and right frontal lobe lesions, respectively, has received support from various neuroimaging procedures [5]. Specifically, the neuroimaging procedures that have implicated the frontal lobes in aphasia and aprosodia have included computerized tomography [3, 10, 18, 19, 27], positron emission tomography [12], magnetic resonance imaging [1, 17, 29] and measures of regional cerebral blood flow [21,26]. However, a review of the relevant literature indicated that quantitative electroencephalography (QEEG) has not been widely utilized to investigate the neuroanatomical basis of nonfluent aphasia [11].

The use of QEEG as an investigative tool is supported given that this measure has been validated [11] and is certainly more convenient and more efficient that most other neuroimaging methods. Quantitative electroencephalography provides researchers and clinicians with an effective and flexible neuropsychological assessment tool that enables interhemispheric comparisons in patients with suspected cerebral dysfunction [25]. Additionally, QEEG permits statistical analyses of comparisons among electrode sites within a single individual. Using a within subjects design affords the QEEG a high level of sensitivity to the patient's unique premorbid level of functioning. Furthermore, QEEG permits the investigator to test the a priori hypotheses of cerebral dysfunction that result from neuropsychological assessment [25]. Hence, QEEG may be used as a means of confirming the suspected neuroanatomical locations of cerebral dysfunction. Moreover, QEEG may be useful in the confirmation of "functional lesions" where distinct anatomical anomalies are not apparent from gross imaging techniques (e.g. CT-Scan), but where reliable metabolic and electrical changes are present.

The present investigation concerns the neuropsychological evaluation of an individual dually diagnosed with nonfluent dysphasia and dysprosodia in which QEEG was utilized as a neuropsychological procedure to provide for convergent validation of suspected cerebral dysfunction.

Method

Case History

The patient was a right handed 29 year old Caucasian male who graduated from high school and who also attended college for half of one semester. The patient reported that he had never been hospitalized for either emotional or medical problems. He had no current medical problems and denied any history of head trauma. The patient's mother reported that he experienced some type of seizure at approximately the age of one and a half years. She was unable to provide details concerning the seizure. The seizure had ceased upon arrival at the hospital and was an isolated incident. Family medical history was remarkable for the mother stuttering mildly on an occasional basis. Additionally, the patient's mother indicated that his great aunt suffered from some type of palsy and also may have had Huntington's chorea. However, she was unable to provide more specific details. The patient denied illicit drug use, but reported drinking alcohol occasionally on a social basis. The patient was referred for a neuropsychological evaluation due to problems with stuttering. Consultation with the parents indicated that the patient had stuttered for his entire life. An ebb and flow pattern to stuttering was reported by both the patient and his father. Essentially, it was reported that the patient's stuttering became more severe whenever he experienced anxiety or stress. He had previously received treatment for his stuttering, but he indicated that this treatment was unsuccessful.

Procedure

Standardized Testing: To obtain an indication of the patient's memory functioning, the Denman Neuropsychology Memory Scale (Sidney Denman, 1984) was administered, which consists of a collection of well established memory subtests. Specifically, the administration of this scale includes the Verbal Memory subtests of Immediate Recall of a Story, Paired Associate Learning, Memory for Digits, Remote Verbal Information, Delayed Paired Associates, and Delayed Recall of a Story. Additionally, the scale also includes the Nonverbal Memory subtests of Immediate Recall of a Figure, Memory for Human Faces, Remote Verbal Information, and Delayed Recall of a Figure. However, it should be noted that the Musical Tones and Melodies subtest was not administered due to equipment malfunction. Thus, the Nonverbal and the Full Scale Memory Quotients were calculated using the short form version of this testing instrument. The results of this test yielded Scaled Scores of 6 and 8 on the Remote Verbal Information and Remote Nonverbal Information subtests, respectively. Further, the patient obtained a Verbal Memory Quotient of 48, a Nonverbal Memory Quotient of 68, and a Full Scale Memory Quotient of 43. Review of the results from the Denman Neuropsychology Memory Scale indicated that the Verbal Memory Quotient was significantly ($p < .001$) lower than the Nonverbal Memory Quotient. Profile analysis also indicated that the patient possessed significant relative strengths with the Remote Verbal and Nonverbal Information as well as the Memory for Digits subtests.

Syndrome Analysis: The visual screening revealed impairment in visual smooth pursuit with pursuit within the right hemispace. Tactile startle to confrontation was heightened on confrontation at the right hemibody. Bilateral Rapid Alternating Movements were effortful and poorly coordinated. Grip strength was measured at 37kg at the left and 31kg at the right hand. The Dynamometer Perseveration Test indicated perseveration in grip strength at both hands. Specifically, when asked to grip the hand dynamometer "half as hard," his grip strength measured 33kg at the left and 25kg at the right hand. Passive Range of Motion yielded increased flexor and extensor tone at both arms concurrent with speech. Lingual praxis was remarkable for the tongue extending to the left. Facial affect expression to confrontation revealed decreased facial expression over the frontalis muscles. Ambulatory gait revealed slightly bilateral adduction of the feet and forward postural tilt. Rhomberg's test

revealed forward drift with posture also shifted about the neuraxis to the right. He exhibited constructional and organizational deficits during the Draw a House and Draw a Clock tests. Behavioral speed and sequencing was investigated using the Trail Making Test Parts A and B. Performance on this test was impaired, with completion of Part B requiring 126 seconds. His performance on this test was also quite perseverative in that several lines were drawn between each of the numbers and letters. He was also noted to be nonfluent during the Controlled Oral Word Association Test, generating 11 words beginning with the letter F, 6 words beginning with A, and 9 words beginning with the letter S, with the examiner recording responses. However, there were 7 perseverative errors with words beginning with the letter S. Performance on the Ruff Figural Fluency test was also in the impaired range. Specifically, he obtained an error ratio of .0476, generating 21 unique designs and 1 perseverative error, placing him in the 1.1 percentile. Evaluation of propositional speech was notable for diminished fluency to confrontation and in conversation as well as articulation errors, perseverative phonemes, and phonemic paraphasic errors. The patient was also noted to stutter severely throughout the evaluation. Additionally, his speech was ballistic with rapid, poorly regulated initiation.

Based on the findings from the standardized testing and syndrome analysis it was hypothesized that the patient suffered from diffuse bilateral frontal lobe dysfunction.

Quantitative Electroencephalogram: The QEEG was conducted following the completion of standardized testing and the syndrome analysis to provide a third assessment protocol to contribute to convergent validation of cerebral dysfunction. The QEEG served as a confirmatory tool to test the a priori hypothesis of diffuse bilateral frontal lobe dysfunction. Specifically, it was hypothesized that high delta (2.0 – 4.0Hz) and theta (5.0 – 7.0Hz) magnitude (μV) values would be significantly increased at the frontal electrode sites as compared to the more posterior sites of the brain. Also, since aphasia is typically associated with left frontal dysfunction and dysprosodia with right frontal lobe dysfunction, it was hypothesized that heightened delta and theta values would exist across the left and the right frontal lobes. Further, the existence of a positive relationship was expected between the severity of dysfunction in the frontal lobes and the severity of the patient's stuttering. More specifically, it was hypothesized that significant positive correlations would be found between the severity of the patient's stuttering and changes in both high delta and theta magnitude values at the frontal electrode sites.

Quantitative electroencephalography was measured using a Neurosearch-24 (Lexicor Medical Technology). The patient was fitted for a lycra electrode cap containing 19 electrodes arranged according to the International 10/20 System. QEEG was measured using linked ear references. The impedances for all electrodes were less than 10kΩ, and in most instances less than 7kΩ. A sampling rate of 256Hz was used and frequencies below 2Hz were eliminated by a high pass filter. The EEG bandwidths analyzed included high delta (2.0 – 4.0Hz) and theta (5.0 – 7.0Hz).

Subsequent to placement of the lycra electrode cap and testing the impedence levels of the electrodes the patient was instructed to close his eyes and relax while seated in a comfortably padded reclining chair in a near supine position. The patient was allowed to relax for 5 minutes, after which time a baseline measurement of QEEG was obtained. The patient was then exposed to six different conditions requiring him to speak. Briefly, the patient was instructed to solve simple math problems, to describe his job, to recite the alphabet, to count forward, to describe his leisure activities, and to count backwards from 100 by 3. The patient was allowed to relax for at least one minute between each of these conditions. The level of severity of his stuttering during each of these conditions was assessed by the principle investigator. Essentially, the severity of stuttering was rated using a 10 point Likert type scale with 0 representing the absence of stuttering and 10 representing severe stuttering. A total of 60 one second epochs were recorded for each of these conditions, including the baseline condition. Each epoch was individually artifacted to remove eye movement and muscle artifacts.

Results

For purposes of conducting all analyses, magnitude values are represented in μV. High delta and theta magnitude values obtained during the eyes closed baseline condition were noted to be higher at the frontal poles (FP1 and FP2) as compared to the more posterior regions of the same hemisphere (F7, F3, T3, C3, T5, P3, O1 and F8, F4, T4, C4, T6, P4, O2). High delta magnitude values at the F7 and F3 electrode sites were higher than the T3 and T5 electrode sites and were also higher at the F4 electrode site as compared to the T4 and T6 electrode sites. Similarly, theta magnitude values at the F3 electrode site were higher than the T3 and T5 electrode sites and were also higher at the F4 electrode site as compared to the T4 and T6 electrode sites. The magnitude values for high delta across the left and right cerebral hemispheres are displayed in Figures 1 and 2.

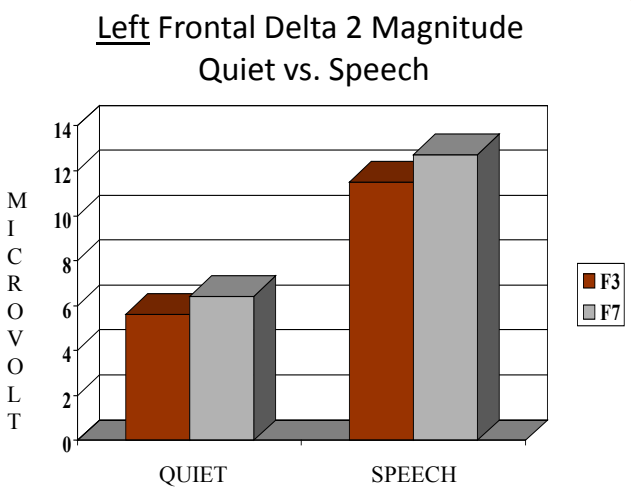

Figure 1. High Delta Magnitude Values Across the Left Frontal Region During Quiet and Expressed Speech Conditions.

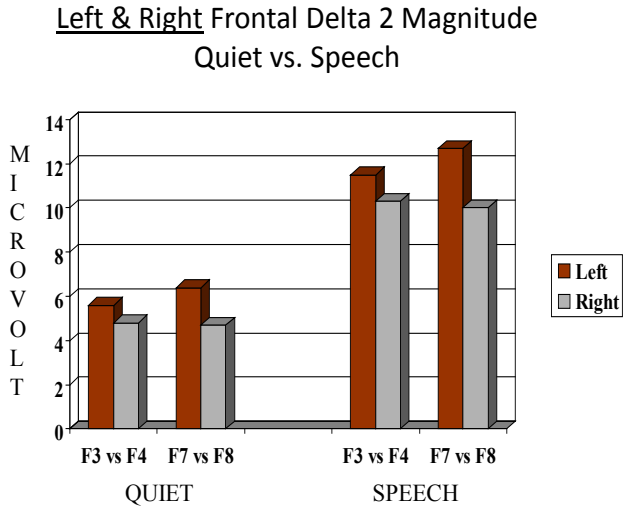

Figure 2. Theta Magnitude Values Across the Left and Right Cerebral Hemispheres for the Eyes Closed Baseline Condition.

Statistical analyses were conducted for purposes of comparing the average high delta and theta magnitude values for the anterior regions (FP1, FP2, F7, F8, F3, F4) versus the posterior regions of the brain (T3, T5, P3, T4, T6, P4). The central (C3, C4) and occipital (O1, O2) sites were excluded from these analyses to keep the group sizes equal. To conduct statistical analyses each electrode site was considered as a separate case. The results of a oneway ANOVA indicated that high delta magnitude values were significantly greater at the anterior regions as compared to the posterior regions of the brain, $F(1,10) = 6.57$, $p < .05$. However, no significant differences in theta magnitude values were found between the anterior and posterior regions of the brain, $F(1, 10) = 3.29$, $p = ns$. Consult Table 1 for the means and standard deviations of the high delta and theta magnitude values for the anterior and posterior regions for the baseline condition.

The hypothesis of diffuse bilateral frontal lobe dysfunction was tested by analyzing the high delta and theta magnitude values obtained from the condition in which the patient exhibited the most severe level of stuttering, namely, the condition in which he was instructed to describe his current job. The findings were consistent with those obtained from the baseline condition. Specifically, the high delta and theta magnitude values obtained for the speaking condition were higher at the frontal poles (FP1 and FP2) than at all other more posterior electrode sites (F7, F3, T3, T5, C3, P3, O1 and F8, F4, T4, T6, C4, P4, O2). Furthermore, high delta and theta magnitude values were higher at the F7 and F3 electrode sites than at all the more posterior electrode sites and was also higher at the F8 and F4 electrode sites as compared to all more posterior sites.

Statistical analyses were performed with the high delta and theta magnitude values to determine whether significant differences existed in magnitude between the anterior (FP1, FP2, F7, F8, F3, F4) and the posterior (T3, T5, P3, T4, T6, P4) regions of the brain. These analyses were conducted in the same manner as described previously. The results of a oneway ANOVA indicated that both high delta and theta magnitude values were significant greater at the anterior regions of the brain than at the posterior regions, $F(1, 10) = 8.92$, $p = .05$ and $F(1, 10) = 11.60$, $p < .01$, respectively. Consult Table 1 for the means and standard deviations of the high delta and theta magnitude values for the anterior and posterior regions for the speaking condition.

Comparisons among the left and right frontal lobes were made by examining the theta and high delta magnitude values for the left frontal lobe (FP1, F3, F7) and the right frontal lobe (FP2, F4, and F8) during the baseline and the speaking conditions. As can be seen in Figure 2, the high delta magnitude values between homologous sites in the left and right frontal lobes differ by no more than .2 μV in the baseline condition. Similarly, the theta magnitude values differ by no more than .7 μV in the baseline condition. However, differences between homologous sites within the left and right frontal lobes do emerge in the speaking condition. Specifically, the high delta magnitude value of the FP2 (25.6 μV) electrode site was higher than that of the FP1 (22.3 μV) electrode site. This same pattern was also found in comparing the theta magnitude values for these same electrode sites (FP2 being 21.7 μV and FP1 being 19.8 μV).

Correlational analyses were conducted to determine the existence of a positive relationship between severity of frontal lobe dysfunction and severity of stuttering. For purposes of examining this relationship, comparisons were made at the FP1 and FP2 electrode sites as these sites were associated with the greatest level of dysfunction for both the speaking and baseline conditions (see above).

Condition	Region	Hz	M	SD
		High Delta	7.05	1.08
Anterior				
Baseline	Theta	9.38	1.47	
		High Delta	5.52	.78
Posterior				
		Theta	7.80	1.29
		High Delta	16.5	5.36
	Anterior			
Speaking		Theta	15.73	3.64
		High Delta	9.27	.75
	Posterior			
		Theta	9.85	1.30

Table 1: Means and Standard Deviations for High Delta and Theta Magnitude Values for the Anterior and Posterior Regions During the Baseline and Speaking Conditions
Note. Values are represented in μV.

Condition		Rating
1	Solving simple math problems.	2
2	Job description.	10
3	Recite the alphabet.	0
4	Counting forward.	0
5	Describe leisure activities.	8
6	Counting backward.	5

Table 2: Stuttering Severity Ratings for Each of the Speaking Conditions

To conduct correlational analyses changes in high delta magnitude for each speaking condition were recorded during the eyes closed baseline condition. Correlational analyses were then conducted between the stuttering severity rating scores and the changes in high delta magnitude. The mean severity rating across all speaking conditions was 4.17 (SD = 4.22). Table 2 presents the stuttering severity rating for each speaking condition.

Correlational analyses indicated significant positive correlations between changes in high delta magnitude values and stuttering severity rating scores for both the FP1 (r = .95, p < .01) and the FP2 (r = .95, p < .01) electrode sites.

Discussion

The a priori hypothesis of diffuse bilateral frontal lobe dysfunction was supported by the results of the present investigation.

Visual inspection of the high delta and theta magnitude values for both the baseline and speaking conditions indicated that the frontal poles were associated with higher magnitude values for these frequencies than the more posterior electrode sites of the same hemisphere. This conclusion was substantiated by conducting statistical analyses, which yielded increased high delta magnitude values over the anterior regions as compared to the posterior regions of the brain. Given that the presence of marked delta activity in the waking state is associated with the presence of brain dysfunction [15, 20], the hypothesis of diffuse bilateral frontal lobe dysfunction in the present case is substantiated. Furthermore, although theta magnitude values were not significantly different between the anterior and posterior regions of the brain for the baseline condition, significant differences between the anterior and posterior regions were found for the speaking condition. Equal levels of dysfunction were also found to exist between almost all homologous sites within the left and right frontal lobes in both

the baseline and speaking conditions, as hypothesized. However, increased slowing was noted to exist over the right frontal pole, as compared to the left frontal pole, in the speaking condition. Thus, the results of the present investigation provide further support for the association of frontal lobe dysfunction with nonfluent aphasia and dysprosodia.

Whereas previous investigations have found left frontal lobe dysfunction using neuroimaging procedures such as computerized tomography [3, 10, 18, 19, 27], magnetic resonance imaging [1, 17, 29], positron emission tomography [12] and measures of regional cerebral blood flow [21, 26], the present investigation utilized quantitative electroencephalography. Hence, the present findings not only support previous findings concerning the neuroanatomical correlates of nonfluent aphasia, but also support the use of QEEG as a diagnostic tool. The combination of standardized testing, syndrome analysis, and QEEG provides clinicians and researchers with a powerful means of investigating the relationship between the brain and behavior. This procedure also conforms closely to the scientific method, since the QEEG is used to test the a priori hypotheses resulting from analysis of the data from standardized testing and syndrome analysis. Therefore, converging validation of localized cerebral dysfunction may result from this tripartite assessment approach. Indeed, predicting the neuroanatomical location of brain dysfunction and testing the prediction using QEEG affords researchers and clinicians great confidence in their findings. Additionally, QEEG is a noninvasive procedure and the equipment needed is relatively inexpensive and requires little physical space. Thus, the use of QEEG as a tool for confirming a priori hypotheses regarding brain dysfunction should be particularly appealing to researchers and clinicians.

The present investigation found a positive relationship between increasing symptom severity and increasing levels of brain dysfunction. Based on these findings it may be inferred that larger lesions may yield more severe symptomatology. Although some researchers have discussed the existence of a relationship between symptom severity and brain activity [28], more research appears warranted. Additionally, the present findings carry implications for rehabilitation with patients suffering from brain injuries or cerebral lesions resulting from other insults. The existence of a positive relationship between symptom severity and brain dysfunction may indicate that decreasing severity of symptoms may result in decreasing brain dysfunction. Thus, QEEG may be used as a clinical outcome measure for clinicians conducting therapy for patients. It is the hope of the present authors that this investigation will stimulate further research concerning the relationship between symptom severity and severity of brain dysfunction and how this relationship may be used as a measure of treatment outcome. Also, it is hoped that this investigation stimulates further research using QEEG as a diagnostic tool.

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