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Describing Brain Activity of Persons With AD and Depressive Symptoms

Ezra C. Holston *

University of Tennessee-Knoxville College of Nursing, Knoxville, TN

A B S T R A C T

The purpose of this retrospective pilot study was to characterize depression of AD using electrophysiological changes in the brain activity of persons with AD and depressive symptoms. Participants had a mean age of 70.12 ± 12.68. Participants manifested an increase in absolute/relative theta activity ($p = .000$) over entire brain when compared to normative population-based database. Electrophysiological changes did not differ by age or gender except for increased absolute theta activity in the right lateral frontal areas (t-test = −2.31 to −2.39, $p = .04$) in females. An increased theta activity suggests that depressive symptoms may be part of AD symptomatology, not a co-morbid feature.

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Depression is a common co-morbid feature of Alzheimer's disease (AD) and has been identified in persons with AD since the 1980s (Green et al., 2003). Of the current 5.2 million persons with AD, approximately 20–40% have clinically manifested depressive symptoms, which will escalate to 3–6 million by 2050 (Alzheimer's Association [AA], 2014; Olin, Katz, Meyers, Schneider, & Lebowitz, 2002). These symptoms are clinically assessed as a depression disorder that persistently occurs with the disease process of AD as a co-morbidity of AD; however, the depressive symptoms may directly be associated with the symptomatology of AD and not a co-morbid feature (Holtzer et al., 2005).

In longitudinal studies using population-based samples, certain depressive symptoms occurred as early indicators of AD and markers of the progression of AD, constituting depression of AD, not a depression disorder (Copeland et al., 2003; Holtzer et al., 2005). The symptoms of depression of AD are depressed mood, loss of interest in social interaction and usual activities, fatigue, feelings of worthlessness, reoccurring thoughts of death, and changes in appetite, sleep, and psychomotor (Olin, Katz, et al., 2002; Zubenko et al., 2003). These depressive symptoms occur in 51–61% of persons with AD although they have a minimum prevalence of 15% in the general population (Copeland et al., 2003; Starkstein, Mizrabi, & Garau, 2005; Vida, Des Rosiers, Carrier, & Gauthier, 1994). The occurrence of these depressive symptoms can be sufficient to directly impact the functioning of persons with AD such as cognitive and behavioral functioning, although the depressive symptoms are not manifested in the expected daily occurrence during a 1-month time frame (Copeland et al., 2003; Olin, Katz, et al., 2002; Olin, Schneider, et al., 2002). Furthermore, depression of AD can be identified by structural changes from atrophy in certain cortical regions, altered neurotransmitters' production, and altered synaptic signaling in the brain activity.

Morphological changes have been reported in post mortem studies. Persons with depression and AD had more neuritic plaques and neurofibrillary tangles in the hippocampus and hippocampal atrophy than non-depressed persons with AD (Rapp et al., 2006). Morphological changes consisted of sulcal widening, ventricular dilatation, and white matter loss (Bowen, Najlerahim, Procter, Francis, & Murphy, 1989; Corsellis, 1962; Pantel et al., 1997; Tomlinson, Blessed, & Roth, 1968). These observed volumetric changes were similar to those observed in persons with AD (Pantel et al., 1997), suggesting a similarity in the morphological changes for depressive symptoms and AD as if they are the same symptomatology and not co-morbid features.

Depression, especially late-life, has been associated with decreased activation of serotonin (Bremner et al., 2003; Meltzer et al., 2004; Sheline et al., 2004), glutamate (Auer et al., 2000; Binesh, Kumar, Hwang, Mintz, & Thomas, 2004) and Gamma-aminobutyric acid (GABA) (Fatemi, Stary, Earle, Araghi-Niknam, & Eagan, 2005). In post mortem studies of AD, these abnormalities were reported as decreased serotonin activation in the frontal and temporal areas with decreased volume (Francis et al., 1987; Kepe et al., 2006; Lai et al., 2003; Palmer et al., 1987; Palmer, Stratmann, Procter, & Bowen, 1988), and decreased GABA in the frontal, temporal, and superior parietal areas (Lowe et al., 1988). Although these changes in GABA concentration have not been reported in ante mortem and biomarker studies (Armstrong et al., 2003; Lanctôt, Herrmann, Mazzotta, Khan, & Ingber, 2004), evidence suggests that the pathology of AD is associated with elevated concentration of extracellular glutamate from decreased gluatminergic activity in the cerebral cortex (Burbaeva et al., 2005; Greenamyre, Penney, D'Amato, & Young, 1987; Procter et al., 1988).

Down regulation of the serotonergic receptors has been related to the production of amyloid precursor protein (APP) via the pathway for the cleavage at the N and C termini of the beta amyloid (Aβ) domain (rapid secretion of Aβ peptides, β-secretase) (Nitsch, 1996; Nitsch, Deng, Growdon, & Wurtman, 1996). These observations could suggest that the same pathway for decreased serotonin activity contributes to

* Corresponding Author: Ezra C. Holston, PhD, RN, College of Nursing Building, Room 353, 1200 Volunteer BLVD, Knoxville, TN 37996.
E-mail address: eholston@utk.edu.

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both the manifestation of depression and the development of neuritic plaques and neurofibrillary tangles associated with AD. In addition, continued activation of this mechanism might lead to increased neuropathological abnormalities that are definitive signs of AD. Thus, the synergistic activation of neurochemical changes may contribute to the development and progression of depression of AD.

Numerous studies have characterized the electrophysiological changes in the brain activity of persons with AD (Bennys, Rondouin, Vergnes, & Touchon, 2001; Brassen et al., 2004; Brinkmeyer, Grass-Kapanke, & Ihl, 2004; Ikawa et al., 2000; Lindau et al., 2003) and depression (Davidson, Chapman, & Chapman, 1987; Flor-Henry, Lind, & Koles, 2004; Henriques & Davidson, 1990, 1991; Knott, Telner, Lapierre, Browne, & Horn, 1996; Lieber & Prichep, 1988; Prichep & John, 1992). Only four studies have described the electrophysiological changes in the brain activity of persons with AD and co-morbidity of depression. An increase in delta and theta activity was observed in persons with depression secondary to AD (Lieber, 1988). Relative theta activity increased with a decrease in relative alpha in depressed persons with AD (Pozzi, Golimstock, Petracchi, Garcia, & Starkstein, 1995; Pozzi et al., 1993). Relative theta activity increased in the central area of the brain with relative alpha decreasing in the posterior area (Deslandes et al., 2004). However, in these 4 studies, depressive symptoms are assessed as the disease process of a depression disorder (a co-morbid feature) and not a symptom of AD over the progression of AD. Therefore, the purpose of this retrospective, cross-sectional pilot study was to characterize depression of AD using electrophysiological changes in the brain activity of participants who had a diagnosis of dementia of the Alzheimer type (DAT) and manifested depressive symptoms. The findings parallel the abnormal features of persons with AD, supporting further examination of the electrophysiological changes in the brain activity of persons with AD and depression of AD.

METHODOLOGY

The objective of this pilot study was to describe how depression of AD is manifested in the brain activity of participants with a diagnosis of AD and manifested depressive symptoms. With a retrospective, descriptive design, 14 participants were selected from an electrophysiological database maintained by an electroencephalogram (EEG) clinical laboratory in a metropolitan, university-affiliated hospital. Institutional review boards for the university, school of medicine, and hospital approved the pilot study. The database from the EEG clinical laboratory consisted of electrophysiological data and clinical diagnosis for neurological conditions. Medical records of the participants were reviewed to validate the clinical diagnosis listed in the electrophysiological database and extract demographic and clinical data.

Participants' ages ranged from 50 to 89 (mean age 70.12 $\pm$ 12.68). They were English-speaking, community-dwellers diagnosed with dementia of Alzheimer's type (DAT). They had a history and currently manifested depressive symptoms but not a diagnosis for any mood/affective disorders. With their healthcare providers' permission, they discontinued any psychotropic or cognitive-acting medications 2 weeks prior to the evaluation period. A diagnosis of DAT was based on the DSM III-R (American Psychiatric Association, 1987) and probable AD according to the National Institute of Neurological and Communicative Disorders and Stroke (NINCDS) and the Alzheimer's Disease and Related Disorders Association (ADRDA) criteria (McKhann et al., 1984). Exclusion criteria included history of head trauma, substance abuse, any signs of abnormal structural brain pathology, diagnosis of delirium or vascular dementia, and continuation of any psychotropic or other cognitive-acting medication during the evaluation period.

Clinical Data Collection

Clinical data were extracted from the review of the participants' medical records in the university hospital and in the EEG clinical laboratory. Participants were assessed by healthcare providers in the departments of psychiatry, neurology, and radiology. These assessments included medical (physical examination, electrocardiogram, and blood analysis), neurological (assessment of speech, motor system, cranial nerves, sensory function, and extrapyramidal signs), and psychiatric evaluations (psychiatric interview, neuropsychological tests, and cognitive evaluations). Some participants received magnetic resonance imaging (MRI) or computed tomography (CT) imaging. Participants and/or family members provided medical history, current medications, family history of dementia and symptoms' onset, course, and duration. They also provided information about a history and/or current manifestation of depressive symptoms. In the psychiatric consults, depressive symptoms were assessed with the Beck Depression Inventory I and Hamilton Depression Scale.

Electrophysiological Data Collection

Electrophysiological data were extracted from the EEG clinical laboratory database. Participants were referred to the EEG clinical laboratory for a 24-hour electrophysiological evaluation to rule out any neurodegenerative conditions. The electrophysiological evaluation was performed by healthcare providers in the EEG clinical laboratory.

Participants were seated in a dark, soundproof room. Nineteen standard Ag/AgCl recording electrodes (Fp1, Fp2, F3, F4, C3, C4, P3, P4, O1, O2, F7, F8, T3, T4, T5, T6, Fz, Cz, and Pz) were attached to the scalp using water-soluble paste, according to the 10/20 International System (Prichep & John, 1992). Electro-oculogram electrodes (a differential eye channels) were diagonally placed above and below the eyes' orbit to detect any facial movement that would create artifacts in the EEG recording. An electrode was placed on the thorax to detect cardiac rhythm. One grounding electrode was placed on the dominant arm. Monopolar recordings were collected and referred to linked earlobes. The EEG amplifiers had a bandpass from 0.5 to 30 Hz (3 dB points) with a 60 Hz notch-filter. Impedances were maintained below 5000 ohms. The data were digitized with the A/D converter at 200 Hz with 12-bit resolution. Data acquisition was performed on a Nihon Kohden 2100-A EEG Acquisition System (Nihon Kohden Corp., Tokyo, Japan).

From the 24-hour electrophysiological evaluation, a minimum of 20 minutes of electrophysiological data was collected. During this 20-minute period, the participants remained conscious, at rest, and with their eyes closed. Trained EEG technicians also monitored the participants and the EEG recording for sleepiness, muscle movement, or eye movement. Trained EEG technicians used a FOCUS program to visually edited EEG data for distortions, sleepiness, or other artifacts that may affect electrophysiological analysis. This quality check procedure was supplemented with an automatic EEG artifact detection algorithm. Using NeuroGuide, only 1–2 minutes (24–48 artifact-free EEG epochs, 2.5 seconds long) were randomly selected and extracted from the 20 minutes of collected EEG data for electrophysiological analysis. These selected raw EEG epochs were again reviewed for artifact to ensure that the EEG epochs for statistical analysis were free of any artifacts. A split-half reliability and test–retest reliability of a minimum of .90 was maintained throughout the selection of the 24–48 artifact-free EEG epochs. The selected EEG epochs were quantified, normalized, and analyzed with the NeuroGuide.

EEG Data Analysis

For all 19 monopolar derivations, absolute power and relative power of the brain were computed using 4 frequencies or types of brain activity (delta = 1.5–3.5 Hz, theta = 3.5–7.5 Hz, alpha = 7.5–12.5 Hz, and beta = 12.5–25 Hz). NeuroGuide analysis methods have been previously described in detail (Thatcher, Biver, North, Curtin, & Walker, 2003). The essential features of NeuroGuide analysis are briefly provided. Power spectral analysis was performed on the artifact-free EEG data using Fast Fourier Transform (FFT). Identified electrophysiological

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