Anxiety Misdiagnosed as Dementia? A Complex Case Successfully Treated Using a Multimodal Biofeedback Approach

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A sixty-three year old woman with a recent diagnosis of dementia sought biofeedback treatment for depression, anxiety, and cognitive impairment. Quantitative electroencephalography (QEEG) revealed brainwave patterns that could be associated with anxiety as well as possible dementia. The context of her forgetfulness and disorientation was explored and discovered to coincide with anxiety-related hyperarousal. Following a combination of heart rate variability (HRV) and neurofeedback (EEG biofeedback), she reported significant reductions in depression, anxiety, and cognitive impairment.

Mental health professionals may draw very different conclusions about presenting symptomatology of clients, possibly leading to misdiagnosis, unnecessary treatment, and potentially harmful outcomes. In this report, the authors present the case of J.C., a woman previously diagnosed with dementia, who sought biofeedback treatment to address issues of cognitive impairment, anxiety, and depression following her diagnosis. Over the course of treatment, it became increasingly evident that J.C.’s dementia-like symptoms were primarily a function of heightened states of arousal associated with anxiety. A treatment plan was developed to address her anxiety, depression and dementia-like symptoms with a combination of counseling, heart rate variability (HRV), and electroencephalographic (EEG) biofeedback.

Background
J.C., a 63-year-old, Caucasian female, was referred to a university-based neurotherapy clinic by a state vocational rehabilitation organization for evaluation and treatment. Presenting problems included depression, irritability, short-term memory issues, pain, disorientation, and impulsivity. J.C. reported having had a difficult childhood, marked by abuse, neglect, and health issues. In adulthood, J.C. struggled with depression for over 20 years. J.C. had been employed as an accountant for most of her career. From the time of her diagnosis, she was on medical disability and unable to work. Her dementia diagnosis followed an incident in which she had difficulty seeing and locating her new office building. In addition, she experienced disorientation and difficulty remembering things. She noted a worsening of depressive symptoms following the dementia diagnosis.

J.C.’s medication regimen at the time of intake included Aricept and Namenda for dementia; Wellbutrin, Pristiq, and Lamictal for depression; Crestor for cholesterol; Synthyroid for thyroid; Clonazepam for sleep; Tramadol for bone pain; and Prilosec for heartburn. Her demeanor was marked by frustration and impatience, and she seemed to have a generally negative outlook.

Pretreatment Assessment
J.C.’s mood and level of hopelessness were assessed using the Beck Anxiety Inventory (BAI), Beck Depression Inventory-II (BDI-II), and Beck Hopelessness Scale (BHS). In addition, her sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI) and her attention and impulsivity were measured using the Tests of Variables of Attention (TOVA), a computerized continuous performance assessment used to measure attention and impul-
sivity. A QEEG was performed to assess her current state of brainwave function.

On her pretreatment BAI, J.C. self-reported an overall mild level of anxiety-related symptoms, including hot flashes, dizziness, racing heart, nervousness, trembling, shakiness, sweating, feeling scared, and having fear related to losing control. She also indicated a severe inability to relax and a moderate fear of the worst happening. Her pretreatment BHS score was within the severe range; she endorsed items that indicated a lack of enthusiasm about her future, an inability to see pleasantness ahead, and feelings of being unlucky and unable to make things better. J.C. denied suicidal ideation. J.C.’s pretreatment results on the PSQI indicated that she was experiencing a clinically significant level of sleep difficulty, reporting a loss in sleep quality and difficulty sleeping due to having trouble falling asleep, breathing issues, nightmares, and worries that kept her awake. She also indicated that she took medication to sleep and that she would take over an hour to fall asleep without the medication.

The TOVA measures response time, response time variability, commission errors (impulsivity), and omission errors (inattention) within the auditory as well as visual modalities. On the pretreatment auditory TOVA, the overall results were deemed invalid because J.C. had difficulty distinguishing between the two tones and was significantly frustrated. She declined the option to continue the test on a different day. Her performance may have been negatively impacted by fatigue and/or frustration with the task. The results of the pretreatment visual TOVA were deemed valid. J.C. scored below average on response time variability and commission error (impulsivity) scores, suggesting that she struggled to impede impulsivity in both high-target and low-target frequency demand situations within the visual domain. Her response time and omission error scores improved from below average to average over the course of the test. Her performance suggested that she was able to improve her ability to attend after a five to ten minute adjustment period and when target frequency increased.

Pretreatment QEEG records evidenced a mixed fast and slow brainwave pattern, which can be associated with medication effects. This pattern might be expected given the number and types of medications J.C. was taking at the time she presented for treatment (Johnstone, Gunkelman, & Lunt, 2005). However, J.C.’s EEG record also showed a low-voltage-fast pattern. This is an EEG pattern often associated with anxiety and characterized by low EEG amplitudes across all the bandwidths of brain activity, with most of the activity in the higher, or faster, EEG frequency bands (e.g., beta activity > 29 cycles per second) (Johnstone et al. 2005). Additional activity observed in J.C.’s EEG that may be associated with anxiety (as well as medication effects) included sinusoidal and spindling beta from 20–26 Hz with an amplitude range of 5–40 uV centered along the vertex. Sinusoidal beta also is often correlated with obsessive-compulsive symptoms, anxiety or rumination, attention problems, and/or viral or toxic encephalopathy (Arns, Gunkelman, Olbrich, Sander, & Hegerl, 2011).

However, complicating the diagnostic picture with this 63-year-old client was the QEEG finding of a slower dominant frequency (posterior alpha occurred infrequently and occurred at 9 Hz rather than the expected 10 Hz), sometimes associated with dementia (Prichep, 2007). In addition, signs that could support the dementia diagnosis also included activity in the 1–2 Hz range with amplitude of 30–40 uV observed across the head, particularly in the posterior region. Her QEEG also showed slowing in the temporal lobe regions, which can be associated with auditory processing difficulties, memory issues, or difficulties accurately perceiving or interpreting social cues (Demos, 2005). However, dementia may lead to a decrease in overall mean frequency of the complete spectra and changes in coherence or synchronization patterns (Prichep, 2007), neither of which occurred in J.C.’s record.

Therefore, J.C.’s pretreatment QEEG, complicated by the number of medications she was taking, revealed mixed evidence for and against her previous diagnosis of dementia. It is important to consider that any given QEEG pattern can be associated with a variety of symptoms and/or conditions and that both QEEG data as well as subjective patient reports must be included in the overall evaluation and treatment planning process.

Interventions
J.C. began biofeedback treatment to address presenting concerns and develop better coping skills. Each session typically involved counseling, practice of diaphragmatic breathing skills, and HRV biofeedback followed by EEG biofeedback. Early discussion with J.C. revealed that her inability to see or find things seemed to coincide with moments of extreme stress. As she described such
instances, it became clear that she was quite frequently in a heightened state of arousal to the point of experiencing panic attacks. Consequently, the treatment program included a psycho-educational component to teach J.C. coping skills, such as diaphragmatic breathing. By learning and implementing diaphragmatic breathing, J.C. reported a dramatic decrease in her stress levels as well as a reduction in instances of getting lost or losing items. J.C. noted significant gains in her ability to manage life stressors, reduce anxiety levels, and in her practice of more positive self-talk.

HRV-biofeedback was implemented with J.C. as a way to improve her ability to self-regulate emotions, build body awareness, and promote deep breathing and a relaxed state prior to each neurofeedback treatment. Initially, J.C. was only able to take brief, shallow breaths, with limited use of her diaphragm. Although resistant at first, J.C. began to see improvement and to value regular practice once she established a greater comfort level and noticed positive results in her sessions. Over time, she was able to generalize her breathing practice to stressful situations, such as driving to new locations or finding a parking place in a crowded lot. She also used this practice to help her fall asleep at night.

J.C. attended EEG-biofeedback sessions either once or twice per week over the course of several months. The EEG-biofeedback treatment protocols included sessions initially targeting reduction of slow wave (2–8 Hz) activity and fast wave (20–30 Hz) activity at frontal (FZ) and central (CZ, C3) sites. The treatment protocol then focused on decreasing 2–10 Hz and 20–30 Hz while increasing 12–15 Hz in parietal sites (P3 and P4). Finally, 2–6 Hz and 6–11 Hz, along with 20–30 Hz, were reduced in the right fronto-temporal region between F8 and T4.

J.C. displayed and reported various changes throughout the course of treatment. Initially, she was not fully compliant with presession nutritional requirements and responded with irritation and frustration at any guidance during EEG biofeedback sessions. A notable change was apparent after several sessions of treatment at FZ. Unlike her irritable demeanor in prior sessions, she chatted happily with the therapist and her mood and affect appeared dynamically altered. In addition, she was dressed stylishly with particular attention given to hair and makeup. She made jokes and volunteered her thoughts easily, reflecting on how she had been able to overcome many struggles in her life. This was a contrast to prior sessions, where she expressed anger, regret, and a sense of loss about her life.

About halfway through treatment, J.C. reported that she had difficulty finding a parking space, she was calm, assessed the situation, and found a solution. She appeared to be open to suggestions, receptive to the therapist’s encouragement, and began to talk about feeling more alive, being able to “see the light,” and having real hope for her future.

Toward the end of her sessions, J.C. indicated hopefulness about the future and discussed “starting over” at 63 and how it was both frightening and exciting. Whereas, prior to treatment, she seemed to have trouble advocating for herself, she now appeared to become an active participant in her own physical and mental health. For example, she began to follow up with doctors and pay close attention to the effects of her medications.

Over the course of treatment, medically coordinated changes to medication included discontinuation of Pristiq, Lamictal, and Wellbutrin, for depression, and Namenda, for dementia. In addition, J.C. worked with her medical care providers and psychiatrist to find an amenable combination of medications to manage her pain and other physical conditions.

**Posttreatment Assessments**

The same measures were used to assess J.C. at the end of her treatment. J.C.’s posttreatment scores on the BAI, BDI-II, and BHS were all within the minimal range and indicated a significant decrease compared to her pretreatment scores. J.C.’s scores on the BAI decreased from the mild to the minimal range. Although her pretreatment scores on this measure were indicative of mild anxiety, J.C.’s reports of frequent, intense episodes of heightened arousal during times of stress indicated that she may have been underreporting anxiety symptoms on the pretreatment BAI. Over the course of treatment, J.C. reported an increased ability to moderate her anxiety during stressful situations through diaphragmatic breathing and positive thinking. As her anxiety decreased, so did her tendency to get lost, lose items, and become disoriented. J.C.’s improvement on BDI-II scores from the moderate to the minimal range suggested that her depression symptoms had decreased to subclinical levels following biofeedback and neurotherapy treatment. This measured improvement coincided with J.C.’s reports of being more hopeful and future-oriented, and advocating for her own health. J.C.’s improvement on BHS scores from the severe to the minimal range suggested that her sense of hopelessness had improved significantly after treatment. This apparent decrease in hopelessness aligned with J.C.’s decrease in depression symptoms as well as her reported hopefulness about the future, including excitement about “starting over” at age 63. The PSQI posttreatment score could not be
determined because J.C. skipped several items on the measure. Even though a score could not be obtained, she showed improvement, including falling asleep more quickly, no longer waking at night, no longer having difficulty breathing, no longer having nightmares, no longer having difficulty remaining alert during the day, having more enthusiasm to get things done, and no longer taking medication to help her sleep.

Following treatment, J.C.’s Auditory TOVA scores improved in the areas of response time, response time variability, commission errors, and omission errors, all of which shifted from invalid to average ranges. In addition, J.C.’s Visual TOVA scores improved in several areas. Response time variability improved from invalid to average, response time improved eight points within the average range, commission errors improved from invalid to above average, and omission errors improved from invalid to the higher end of average. After the posttreatment TOVA was completed, J.C. commented that she could focus and pay attention much better compared to the first time she completed the TOVA.

J.C.’s posttreatment QEEG record showed increased amplitudes in the occipital region at 9 and 10 Hz, and across the head from 6–20 Hz. There was decreased activity frontally from 2–5 Hz, and across the head from 22–30 Hz. These changes are consistent with what would generally be expected given the neurofeedback treatment protocols that were utilized to decrease 2–8 Hz and 20–30 Hz.

The increases in amplitude noted occipitally at 9 and 10 Hz were likely related to the client’s ability to meet situational demands. Dominant alpha activity seen after treatment in the eyes-closed record is an indication of improved ability to regulate brain arousal levels (Johnstone et al., 2005). J.C.’s record also indicated decreases in slow wave activity (from 2–5 Hz) in the frontal lobe region, a change associated with improved executive functioning, such as being able to maintain attention, impede impulsivity, and engage in problem solving tasks (Thompson & Thompson, 2003). J.C.’s record also showed decreases in the high beta range (20–30 Hz), which is typically associated with decreased anxiety, irritability, and ruminative thought patterns as well as improved frustration tolerance (Johnstone et al., 2005).

Conclusion
The diagnosis of dementia was reexamined after further exploration revealed that client J.C.’s dementia-like symptoms were possibly rooted in hyperarousal due to anxiety, as well as possible side effects of multiple medications. After undergoing multimodal biofeedback for physiological regulation and to normalize brainwave patterns associated with anxiety, rumination, attention problems, visual processing, and memory problems, J.C.’s depression, anxiety, and dementia-like symptoms and medication were dramatically reduced. This case demonstrates the complex nature of many mental health disorders and the importance of including psychophysiological assessment and intervention methods when treating these disorders.

References