NeuroRegulation

The Official Journal of

ISNR

INTERNATIONAL SOCIETY FOR NEUROREGULATION & RESEARCH

Volume 10, Number 1, 2023

NeuroRegulation

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NeuroRegulation (ISSN: 2373-0587) is published quarterly by the International Society for Neuroregulation and Research (ISNR), 13876 SW 56th Street, PMB 311, Miami, FL 33175-6021, USA.

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NeuroRegulation

ISNR
INTERNATIONAL SOCIETY FOR
NEUROREGULATION & RESEARCH

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Psychophysiology and Cognitive Functioning in Elderly: The Skin Conductance as a Reliable Marker of Memorization and Rememorization Capability

*Carlo Pruneti[1], Chiara Cosentino[2], and Sara Guidotti[1]**

[1]Clinical Psychology, Clinical Psychophysiology and Clinical Neuropsychology Labs, University of Parma, Parma, Italy
[2]Department of Medicine and Surgery, University of Parma, Parma, Italy

Abstract

Objectives. The aim of the study is to assess the role of physiological activation in favoring the benefits of a series of sessions of reminiscence therapy (RT). **Methods.** Seven healthy elders (age: 87.7 ± 4.6) were recruited. A Psychophysiological Stress Profile (in three phases: baseline, stress, recovery) has been recorded in order to register the skin conductance (level and response, SCL-SCR). During the stress condition the Mini-Mental State Examination and Semantic Fluency Test were administered. The cognitive functioning was reassessed after seven sessions of RT. **Results.** On the basis of the SCR value (during stress condition), two groups have been made: high responders (HRs) and low responders (LRs). At baseline, HRs significantly differ in SCR (stress phase) and MMSE total score. After the RT, the same group reported higher scores in memory recall and lexical access. **Discussion.** A relation between physiological arousal and cognitive performance has been confirmed.

Keywords: autonomic nervous system; skin conductance; cognition; arousal; older people

Citation: Pruneti, C., Cosentino, C., & Guidotti, S. (2023). Psychophysiology and cognitive functioning in elderly: The skin conductance as a reliable marker of memorization and rememorization capability. *NeuroRegulation, 10*(1), 2–10. https://doi.org/10.15540/nr.10.1.2

***Address correspondence to**: Sara Guidotti, PhD Student, Clinical Psychology, Clinical Psychophysiology and Clinical Neuropsychology Labs., Dept. of Medicine and Surgery, Via Volturno, 39 43126 Parma, Italy. Email: sara.guidotti@unipr.it

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Edited by:
Rex L. Cannon, PhD, Currents, Knoxville, Tennessee, USA

Reviewed by:
Rex L. Cannon, PhD, Currents, Knoxville, Tennessee, USA
Randall Lyle, PhD, Mount Mercy University, Cedar Rapids, Iowa, USA

Introduction

The number of people aged above 65 years worldwide is increasing more and more, and it is estimated that it will reach 1.5 billion in 2050. Considering this assumption, healthy aging is becoming an important issue globally because of the increasing cost of healthcare (World Health Organization, 2011). Moreover, older adults' health status and health promotion issues are of critical concern because about one-fifth of older adults experience an age-related disorder with associated mental or physical disability. As a matter of fact, the most common neurological and psychopathological disorders in older adults are dementia, depression, anxiety, and substance abuse problems. In this scenario, health and social services, as well as long-term care for older adults, play important roles in promoting their mental health (World Health Organization, 2015; Yen & Lin, 2018).

According to recent estimates, older adults (> 65 years old) in long-term care are 5.3% in Australia (Australian Institute of Health and Welfare, 2012); 0.08% in Malaysia (Department of Statistics Malaysia, 2012); 3.9% in the United States of America (Jones et al., 2009); 4.1% in United Kingdom (Laing & Buisson, 2012); 3.2% in Germany (Molinuevo, 2008; Syed Elias et al., 2015), and 0.08% in Italy (Ministero della Salute, 2021). Although these percentages indicate only a small proportion of the population, the level and the type of care required is significantly demanding and challenging. Furthermore, this public issue will become even larger considering that the world population of older adults is increasing disproportionally (World Health Organization, 2012).

Nevertheless, even considering the healthy aging, during this stage older adults find themselves having to cope with their feelings about life events (Erikson, 1959). For instance, adapting to the social environment, enjoying support, and being empowered to live and die with dignity are common topics (World Health Organization, 2015). Regarding this matter, reminiscence therapy (RT) generally helps older adults review their memories and past experiences, promoting a successful aging and the acceptance of death (Yen & Lin, 2018). In addition, RT demonstrated to benefit older patients with and without mental health problems (Syed Elias et al., 2015; Woods et al., 2005). Reminiscence, by definition, recalls past memories (Westerhof et al., 2010). More specifically, RT uses the recall of past events, feelings, and thoughts to facilitate pleasure, better quality of life, and better adjustment to present circumstances (Bulechek et al., 2008). Participants are free to discuss their life stories and they can focus on both pleasant and sad memories. Eight functions of RT were identified (Webster, 1993). Briefly, these were (1) identity and appreciating oneself; (2) problem solving and recognizing one's own strengths in dealing with problems; (3) death preparation and facilitating acceptance of death; (4) teach/inform and sharing life stories with the intent to teach; (5) conversation and developing ways of communication with other people; (6) bitterness revival and revisiting memories of difficult life events; (7) boredom reduction and reminiscing to relieve feelings of boredom; as well as (8) intimacy maintenance and remembering significant people. It was found that the eight functions of RT could be grouped according to three higher order dimensions linked to well-being: positive self-functions, negative self-functions, and prosocial functions. In summary, RT has (1) positive self-functions that are referred to preserving or developing self-awareness and included reminiscence for identity, problem solving, and death preparation; (2) negative self-functions that are related to regrets about the past and rumination and included bitterness revival, boredom reduction, and intimacy maintenance; and, (3) lastly, prosocial functions of reminiscence fostered relatedness with others such as conversation and teach/inform (Cappeliez et al., 2007; Cappeliez & Robitaille, 2010). For instance, describing negative feelings that were induced by a tragedy and then releasing the related grief with the support from the others may provide psychological relief for some individuals. In addition, although cognitive capacity may decrease also in healthy aging, cognitive performance can be stimulated by exercising memory systems and improving plasticity (Yen & Lin, 2018). Although some therapists prefer to use individual RT (Chong, 2000), when comparing it with group RT in long-term care, at least three authors preferred group RT since it encouraged social contact between the residents, enhanced communication skills, and established new relationships (Burnside & Haight, 1994; Roos & Malan, 2012; Zhou et al., 2012). Furthermore, a systematic review of RT for the treatment of depression established that the social role function of the group was the defining factor that made it more effective RT (Housden, 2009). Indeed, group RT usually comprises 6 to 10 participants in each therapy session to enhance group dynamics, whereas individual RT is conducted on a one-to-one basis (Chong, 2000). Because of its nonpharmacological nature, RT is usually used as an intervention for older adults in dementia care, long-term care, and hospice care (Cotelli et al., 2012). Moreover, given today's challenges in long-term care, this approach is valuable because it can be conducted during normal activities of daily life (i.e., during mealtime, walking around the facility; Klever, 2013).

Considering that the elderly population is increasing over time, it may be useful to identify the individual characteristics that make a person suitable for a specific nonpharmacological intervention. For instance, in the case of RT, the cognitive stimulation and the training of memory retention skills are supported by the emotional processing provoked by life events and related memories. Therefore, an emotional-psychophysiological activation is elicited in order to favor the re-elaboration of mnesic content. There are various studies that provide important information through the recording of psychophysiological parameters. More specifically, the literature is rich in studies that assessed the connection between emotional-psychophysiological arousal and cognition, but, to our knowledge, there is no research that investigated the role of the skin conductance (SC) (or galvanic skin response [GSR] or electrodermal activity [EDA]) as a reliable marker of cognitive efficiency and a predictor of the benefit obtained by a cognitive training in elderly subjects.

The hypothesis that drove the present study is based on the fact that the rapid phasic components of SC (the skin conductance response [SCR]) is the parameter that best reflects the phasic components of autonomic arousal (Fowles et al., 1981; Tranel & Damasio, 1984). More specifically, SCR has long been known as a sensitive index of psychosomatic arousal that is strictly connected to emotional activations, mental processing, and focused attention, especially considering stimuli with emotional or social valence (Bechara et al., 1996; Cacioppo et al., 2007; Gatti et al., 2018; Palomba et al., 2000; Pruneti et al., 2021). For instance, previous studies demonstrated that SC, both during the rest phase (skin conductance level [SCL]) and during the stress presentation (SCR), is closely connected with the activation of specific brain areas responsible for processing the emotional value of a stimulus, such as the hypothalamus and amygdala (Gatti et al., 2018; Pruneti et al., 2021). Furthermore, other brain regions responsible for sustained and focal attention (i.e., prefrontal and orbito-frontal cortexes) emerged to be associated with autonomic activation (Pruneti et al., 2021). In summary, it is precisely for this reason that SCR is commonly considered by researchers as a good index of emotional and motivational involvement (Pennisi & Sarlo, 1998; Pribram & McGuinness, 1975). However, although these data are promising, only a few studies highlight the need for direct sympathetic autonomic activation to support the efficiency of these cognitive functions. The aim of this work was to confirm the association between neuropsychological and psychophysiological variables in a group of elderly subjects, selected according to the absence of serious neurodegenerative disorders. Considering the SCR as an index of emotional reactivity to create a group of high responders (HRs) to be compared with low responders (LRs), it has been hypothesized that HRs might benefit more from a series of RT sessions aimed at improving memory retrieval.

Materials and Methods

Study Design and Participants

In this exploratory and cross-sectional study, seven subjects aged between 82 and 94 years old (87.7 ± 4.6) were consecutively examined. The criteria for inclusion in the study were absence of neurodegenerative disorders or physical diseases; low-mild cognitive deficit assessed by the Short Portable Mental Status Questionnaire (SPMSQ; Pfeiffer, 1975); score less than 6 at the Geriatric Depression Scale (GDS; Yesavage et al., 1982); no assumption of psychoactive drugs.

Institutional Review Board Statement

This study was conducted in accordance with the recommendations of the local ethic committee at the University of Parma. In Italy, until 2018, no ethical approval was required for observational nature studies, since they were not defined as medical or clinical research, according to the Italian law No. 211/2003. The study was conducted before 2018 and included nonclinical surveys which used noninvasive measures. Furthermore, this study complies with the Declaration of Helsinki and with Italian privacy law (Legislative decree No. 196/2003). No treatments or false feedback were given, and no potentially harmful evaluation methods were used. Participation was voluntary, and participants could drop out at any time without any negative consequences. All data were stored only by using an anonymous ID for each participant and the data obtained were used solely for scientific purposes.

Procedures

The present research was developed in collaboration with a nursing home settled in Parma district (Azienda Pubblica di Servizi alla Persona, Parma), in which about 90 people with different characteristics live. Recruitment was done by two psychologists. In particular, a neuropsychological and psychophysiological assessment was made with devices and personnel from the Clinical Psychology, Clinical Psychophysiology, and Clinical Neuropsychology Labs at the University of Parma (Dept. of Medicine and Surgery), where all data were processed and analyzed.

For the first data collection, patients were taken to a quiet room and were informed by a research assistant about the study procedures. After providing informed consent, patients were administered the neuropsychological tests and were familiarized with the equipment (e.g., cables) and the procedure of the psychophysiological evaluation (see below). For the next 2 months, the selected patients received seven RT sessions. Lastly, for the second data collection, patients were readministered the neuropsychological tests.

Measures

The *Mini-Mental State Examination* (MMSE; Folstein et al., 1975) was used to assess overall general cognitive ability. The MMSE is a set of 11 questions that investigate five areas of cognitive functioning (orientation, immediate memory/recording, attention/concentration, delayed recall, and language). This instrument is currently the most widely used test for cognitive screening in clinical practice and is mentioned by several guidelines for the assessment of dementia and cognitive disorders. Indeed, it shows good sensitivity and reliability with Cronbach's α = 0.91 (Marioni et al., 2011).

The *Semantic Fluency Test* (SFT; Costa et al., 2014) was used to quantitatively assess the vocabulary. The subtest used by Costa and colleagues is a revised version of the test used by Novelli et al. (1986). In this version, the subject is asked to say as many words as possible belonging to the colors, animals, and fruits categories in three different trials, which also lasted 60 s each.

The *Psychophysiological Stress Profile* (PSP; Cosentino et al., 2018; De Vincenzo et al., 2022; Fuller, 1979; Pruneti, Fontana, et al., 2011; Pruneti & Guidotti, 2022; Pruneti, Guidotti, et al., 2022; Pruneti, Lento, et al., 2010) structured in three phases was implemented. In the baseline phase (6 min), each participant was instructed to close their eyes and remain still and relaxed. During the stress phase (4 min), the participant was administered the neuropsychological tests (MMSE and SFT). Lastly, in the recovery phase (6 min) the participant was instructed to relax again. The SCL and SCR parameters were recorded giving a very low-intensity electrical direct current by means of two electrodes placed on the first and second fingers of the nondominant hand. More specifically, two gold plated electrodes were used. The employed technology device was the “psycholab VD 13” by SATEM (Rome, Italy). The Modulab was connected by means of an infrared cable with a PC and all the data was detected and processed by PC soft VD 13SV VERSION 5.0 Works program software by SATEM (Rome, Italy).

Statistical Analysis

Statistical analysis was performed using Microsoft Excel and IBM SPSS Statistics (Version 28.0.1.0). Differences between HR and LR participants with respect to neuropsychological and psychophysiological variables at baseline were assessed using the Mann-Whitney U Test. Considering the small sample size, a Spearman’s Correlation was implemented to examine the association between the variables investigated. To test our hypothesis, another Mann Whitney U test was conducted in order to calculate statistical differences between the two groups on the neuropsychological test scores after the seven RT sessions. All statistics were considered significant if $p < 0.05$.

Results

All of the participants were males, married, and retired. The sample was divided in half considering the SCR value obtained during the stress condition of the PSP. The neuropsychological and psychophysiological features of the two groups of the sample are shown in Table 1 where differences at baseline between HR and LR are shown. Three participants were considered HRs while four participants were considered LRs. Considering the psychophysiological evaluation, HRs and LRs significantly differed only in the SCR value of the stress phase ($p < 0.03$). Moreover, HR participants reported higher scores on the MMSE ($p < 0.05$).

The associations between variables are reported in Table 2. The SCL recorded during the stress phase was negatively and moderately associated with both SCR stess ($p < 0.05$) and SCR recovery ($p < 0.05$) levels. Furthermore, SCR stress level was positively and moderately correlated with the total score of the MMSE ($p < 0.05$).

Lastly, the differences between HRs and LRs after the RT are shown in Table 3 where the comparison of the neuropsychological Lastly, the differences between HRs and LRs after the RT are shown in Table 3 where the comparison of the neuropsychological tests’ scores between groups highlights significantly higher scores of recall ($p < 0.03$) and verbal fluency ($p < 0.03$) in HRs.

Discussion

The basic assumption underlying the present research was that psychophysiological arousal is strictly associated with cognitive efficiency. The aim of the present pilot study was to evaluate the possibility of dividing a group of healthy elderly people according to the parameter of the SCR in the mental stress condition and, therefore, to create a group of HR and a group of LR. More specifically, the difference between the two groups after cognitive training aimed at implementing memory recovery skills has been investigated.

Once the two groups, HR and LR, were created, significant differences in psychophysiological parameters and neuropsychological scores emerged. In particular, in the HR group, there are significantly higher values than the LR group both in the SCR (stress condition) and in the overall MMSE score. Moreover, it has emerged that the two variables are significantly associated, according to the Spearman’s Correlation calculation. These data are in line with several previous studies that have shown that skin conductance reactivity correlates with mental performance (Cacioppo et al., 2007; Kim et al., 2019; Lim et al., 1996; Pruneti et al., 2021).

The second data collection served to investigate the hypothesis that guided the present research because the aim was to investigate the significant difference between the two groups after seven sessions of RT. To our knowledge, this aspect has never been investigated so far. However, it was hypothesized that subjects placed in the HR group

Table 1

Comparisons Of Neuropsychological and Psychophysiological Features Between High Responders and Low Responders During the First Data Collection.

	High Responders (*n* = 3)		Low Responders (*n* = 4)		*U*	*p*
	M	*SD*	*M*	*SD*		
Neuropsychological Assessment						
Mini-Mental State Examination						
Registration	1.67	1.5	1.75	1.3	3.0	0.18
Recall	3.00	0.0	2.50	0.6	6.0	1.00
Calculation	2.30	2.3	0.50	0.6	2.0	0.12
Total score	26.90	2.8	21.68	2.2	0.5	0.05
Semantic Fluency Test	22.00	5.6	19.00	5.0	3.5	0.37
Psychophysiological Assessment						
Skin Conductance Level						
Baseline	2.79	0.51	2.79	0.25	0.06	1.00
Stress	2.71	0.01	2.78	0.02	0.88	1.00
Recovery	2.80	0.05	2.79	0.01	1.56	0.43
Skin Conductance Response						
Baseline	2.75	0.66	2.80	1.00	1.22	0.49
Stress	6.87	0.27	2.47	1.18	7.00	0.03
Recovery	4.59	1.98	1.99	0.98	1.22	0.49

Table 2
Relationships Between Variables in the Whole Sample.

	1	2	3	4	5	6	7	8	9	10
1 SCL Baseline	1.00									
2 SCL Stress	0.38									
3 SCL Recovery	0.00	−0.12								
4 SCR Baseline	0.07	−0.09	0.58							
5 SCR Stress	−0.04	−0.85*	0.04	0.22						
6 SCR Recovery	−0.15	−0.78*	0.11	0.00	0.89					
7 MMSE Registration	0.10	0.10	−0.10	−0.10	0.28	0.38				
8 MMSE Recall	0.00	−0.33	0.00	−0.32	0.16	0.00	0.00			
9 MMSE Calculation	−0.25	−0.7	−0.3	−0.6	0.54	0.66	−0.05	0.35		
10 MMSE Total score	0.09	−0.71	−0.34	0.09	0.79*	0.49	0.00	0.40	0.48	
11 Semantic Fluency Test	−0.73	−0.67	−0.07	0.35	0.54	0.41	−0.05	−0.08	0.22	0.47

*$p < 0.05$. SCL = skin conductance level; SCR = skin conductance response; MMSE = Mini-Mental State Examination.

Table 3
Comparison of the Neuropsychological Tests Scores Between High Responders and Low Responders During the Second Data Collection.

	High Responders (*n* = 3)		Low Responders (*n* = 4)		*U*	*p*
	M	*SD*	*M*	*SD*		
Neuropsychological Assessment						
Mini-Mental State Examination						
Registration	3	0.0	1.00	0.82	6.0	0.03
Recall	3	0.0	3.00	0.00	0.0	1.00
Calculation	3	2.0	1.50	1.00	3.0	0.23
Semantic Fluency Test	31	5.2	21.25	4.60	0.0	0.03

could benefit most from cognitive training. This suggestion was confirmed because the subjects considered more reactive from a psychophysiological point of view reported higher scores in the MMSE subscale that investigates memory retrieval. Furthermore, the same subjects also appear to have improved their lexical access.

These data, although preliminary, allow to confirm what was repeatedly demonstrated about the relationship between cognition and arousal. The association of the measurement of skin conductance to cognitive performance has provided objective and quantitative features of emotional and motivational aspects that drive learning processes. Our results are in line with previous research that suggested that this parameter can be a sensitive and reliable measure of learning (Eisenstein, Bonheim, et al., 1995; Eisenstein, Eisenstein, et al., 1990). In addition, the present study shows that the SCR can be a good indicator of the level of cognitive efficiency even in a group of elderly people.

The close connection between arousal and mental efficiency confirms what was reported more than 100 years ago with the Yerkes-Dodson curve (Yerkes &

Dodson, 1908). According to this law, an optimal level of psychophysiological activation can facilitate the achievement of good results in terms of mental and physical performance. In fact, a low level of performance would be observable in both emotional hyperactivation and hypoactivation (Calabrese, 2008). Identifying the right level of physiological activation, which also involves stress management skills, is a useful aspect for many health professionals such as psychologists, neurologists, speech therapists, and physiotherapists, as well as teachers and educators. In fact, in clinical practice everyone knows that the level of activation, and, therefore, of motivation and collaboration, greatly influences the commitment, both mental and physical, and, thus, the performance. For instance, from the diagnostic to the rehabilitative fields, the optimal level of mental effort is fundamental in order to understand the real difficulties of the patients. Additional data, such as a measure of the emotional effort, can be derived within a psychophysiological evaluation which might be useful to test how difficult and complex is perceived the task administered to the subject examined. In fact, it is possible that low performance corresponds to a minimum level of effort (typical of manipulative and simulative attitudes but also indicative of serious impairment given by depression, for example) or a high effort (found in conditions of anxious hyperactivation or by real cognitive disorders present that cause distress). Furthermore, there are other practical implications of the present study considering the cognitive dimension.

Moreover, dysfunctional learning processes and memory retrieval are usually assessed through verbal tests that are cortically mediated and difficult to quantify. Nonverbal autonomic responses, like the SC, are mediated at a lower brain level, most probably the brain stem. Unlike verbal tests, the SC should be independent of language ability, education level, cultural background, intelligence quotient, and should be less subject to influence by experimenter-subject interaction. Furthermore, it can be quantified, and the subject is unaware of its occurrence or change over time. If cortical defects in learning and memory have autonomic correlates, then measures such as the SC may prove useful diagnostically in detecting a loss in learning and memory as a function of age and/or pathology at a very early stage when the loss may be more amenable to treatment. Its ability to be quantified at such a high level of measurement relative to verbal measures also may prove useful in the following progression of the loss as well as in assessing treatment efficacy. These aspects are often fundamental for a correct differential diagnosis.

However, conditions characterized by neurological and psychiatric disorders were excluded in our study. Thus, the different levels of stress response in terms of SCR can be read in terms of interindividual variability. Considering this, people who are more physiologically responsive are also easier to be emotionally engaged. This aspect could have effects on the effectiveness of an RT intervention which aims to stimulate cognitive functions by leveraging the ability to get excited by talking about personal life events.

Conclusion

These results, although preliminary, confirm the strong connection between cognition and psychophysiological activation, supporting the results of several previous studies (Gray et al., 2009; Logothetis et al., 2001; Pruneti & Boem, 1995; Pruneti et al., 2021).

To conclude, further studies with larger samples are certainly needed to confirm the preliminary data that emerged in this study. However, the results appear promising and future confirmations may also bring benefits in the clinical setting. In fact, carrying out a multidimensional assessment, which involves a psychophysiological assessment, could offer important suggestions to better interpret the dynamic balance of the person and their information processing systems, both tacitly and explicitly (Reda, 1988, 2016). These assumptions could therefore have repercussions both for the diagnostic and the intervention phases and even help decide what is the best treatment for that phase of that person's life.

Author Declarations

The authors declare no conflict of interest. This research received no external funding.

The data presented in this study are available upon reasonable request from the corresponding author.

References

Australian Institute of Health and Welfare. (2012). *Residential aged care in Australia 2010-11: A statistical overview*. Aged care statistics series no. 36. Cat. No. AGE 68. Canberra: AIHW. https://www.aihw.gov.au/reports/aged-care/residential-aged-care-in-australia-2010-11/contents/table-of-contents

Bechara, A., Tranel, D., Damasio, H., & Damasio, A. R. (1996). Failure to respond autonomically to anticipated future outcomes following damage to prefrontal cortex. *Cerebral Cortex*, *6*(2), 215–225. https://doi.org /10.1093/cercor/6.2.215

Bulechek, G. M., Butcher, H. K., & Dochterman, J. M. (Eds.). (2008). *Nursing Intervention Classification (NIC)* (5th ed.). St. Louis, MO: Mosby.

Burnside, I., & Haight, B. (1994). Reminiscence and life review: Therapeutic interventions for older people. *The Nurse practitioner*, *19*(4), 55–61. https://doi.org/10.1097/00006205-199404000-00011

Cacioppo, J. T., Tassinary, L. G., & Berntson, G. G. (Eds.). (2007). *Handbook of psychophysiology* (3rd ed.). Cambridge University Press. https://doi.org/10.1017/CBO9780511546396

Calabrese, E. J. (2008). Stress biology and hormesis: The Yerkes-Dodson law in psychology—A special case of the hormesis dose response. *Critical Reviews in Toxicology*, *38*(5), 453–462. https://doi.org/10.1080/10408440802004007

Cappeliez, P., Rivard, V., & Guindon, S. P. (2007). Functions of reminiscence in later life: Proposition of a model and applications. *European Review of Applied Psychology, 57*(3), 151–156. https://doi.org/10.1016/j.erap.2005.02.014

Cappeliez, P., & Robitaille, A. (2010). Coping mediates the relationships between reminiscence and psychological well-being among older adults. *Aging & Mental Health*, *14*(7), 807–818. https://doi.org/10.1080/13607861003713307

Chong, A. (2000). Reminiscence group for Chinese older people—A cultural consideration. *Journal of Gerontological Social Work, 34*(2), 7–22. https://doi.org/10.1300 /J083v34n02_03

Cosentino, C., Sgromo, D., Merisio, C., Berretta, R., & Pruneti, C. (2018). Psychophysiological Adjustment to ovarian cancer: Preliminary study on Italian women condition. *Applied Psychophysiology and Biofeedback*, *43*(2), 161–168. https://doi.org/10.1007/s10484-018-9395-3

Costa, A., Bagoj, E., Monaco, M., Zabberoni, S., De Rosa, S., Papantonio, A. M., Mundi, C., Caltagirone, C., & Carlesimo, G. A. (2014). Standardization and normative data obtained in the Italian population for a new verbal fluency instrument, the phonemic/semantic alternate fluency test. *Neurological Sciences, 35*(3), 365–372. https://doi.org/10.1007/s10072-013-1520-8

Cotelli, M., Manenti, R., & Zanetti, O. (2012). Reminiscence therapy in dementia: A review. *Maturitas*, *72*(3), 203–205. https://doi.org/10.1016/j.maturitas.2012.04.008

De Vincenzo, F., Cosentino, C., Quinto, R. M., Di Leo, S., Contardi, A., Guidotti, S., Iani, L., & Pruneti, C. (2022). Psychological adjustment and heart rate variability in ovarian cancer survivors. *Mediterranean Journal of Clinical Psychology, 10*(1). https://doi.org/10.13129/2282-1619/mjcp-3318

Department of Statistics Malaysia. (2012). *Social statistics bulletin Malaysia 2012*. Malaysia: Department of Statistics Malaysia. https://www.statistics.gov.my/portal/download_Labour/files/BPS/Buletin_Perangkaan_Sosial_ 2014.pdf

Eisenstein, E. M., Bonheim, P., & Eisenstein, D. (1995). Habituation of the galvanic skin response to tone as a function of age. *Brain Research Bulletin*, *37*(4), 343–350. https://doi.org/10.1016/0361-9230(95)00004-6

Eisenstein, E. M., Eisenstein, D., Bonheim, P., & Welch, E. A. (1990). Habituation of the galvanic skin response in adult males as a function of age. *Physiology & Behavior*, *48*(1), 169–173. https://doi.org/10.1016/0031-9384(90)90279-d

Erikson, E. H. (1959). *Identity and the life cycle*. New York, NY: International Universities Press.

Folstein, M. F., Folstein, S. E., & McHugh, P. R. (1975). "Mini-mental state": A practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research*, *12*(3), 189–198. https://doi.org/10.1016/0022-3956(75)90026-6

Fowles, D. C., Christie, M. J., Edelberg, R., Grings, W. W., Lykken, D. T., & Venables, P. H. (1981). Publication recommendations for electrodermal measurements. *Psychophysiology*, *18*(3), 232–239. https://doi.org/10.1111 /j.1469-8986.1981.tb03024.x

Fuller, G. D. (1979). *Biofeedback methods and procedures in clinical practice*. San Francisco, CA: Biofeedback Press.

Gatti, E., Calzolari, E., Maggioni, E., & Obrist, M. (2018). Emotional ratings and skin conductance response to visual, auditory and haptic stimuli. *Scientific Data*, *5*, Article 180120. https://doi.org/10.1038/sdata.2018.120

Gray, M. A., Minati, L., Harrison, N. A., Gianaros, P. J., Napadow, V., & Critchley, H. D. (2009). Physiological recordings: Basic concepts and implementation during functional magnetic resonance imaging. *NeuroImage*, *47*(3), 1105–1115. https://doi.org/10.1016/j.neuroimage.2009.05.033

Housden, S. (2009). The use of reminiscence in the prevention and treatment of depression in older people living in care homes: a literature review. *Groupwork, 19*(2), 28–45. https://doi.org/10.1921/gpwk.v19i2.674

Jones, A. L., Dwyer, L. L., Bercovitz, A. R., & Strahan, G.W. (2009). *The National Nursing Home Survey: 2004 overview*. National Center for Health Statistics. Vital and Health Statistics, *13*(167). Maryland: DHHS Publication. https://www.cdc.gov/nchs/data/series/sr_13/sr13_167.pdf

Kim, A. Y., Jang, E. H., Choi, K. W., Jeon, H. J., Byun, S., Sim, J. Y., Choi, J. H., & Yu, H. Y. (2019). Skin conductance responses in Major Depressive Disorder (MDD) under mental arithmetic stress. *PLoS ONE*, *14*(4), Article e0213140. https://doi.org/10.1371/journal.pone.0213140

Klever, S. (2013). Reminiscence therapy: Finding meaning in memories. *Nursing*, *43*(4), 36–37. https://doi.org/10.1097 /01.NURSE.0000427988.23941.51

Laing & Buisson. (2012). Care of elderly UK market survey 2012/13. London: Laing and Buisson. https://www.laingbuisson.co.uk/MediaCentre/PressReleases /CareofElderly2012 13PressRelease.aspx

Lim, C. L., Barry, R. J., Gordon, E., Sawant, A., Rennie, C., & Yiannikas, C. (1996). The relationship between quantified EEG and skin conductance level. *International Journal of Psychophysiology*, *21*(2–3), 151–162. https://doi.org/10.1016 /0167-8760(95)00049-6

Logothetis, N. K., Pauls, J., Augath, M., Trinath, T., & Oeltermann, A. (2001). Neurophysiological investigation of the basis of the fMRI signal. *Nature*, *412*(6843), 150–157. https://doi.org/10.1038/35084005

Marioni, R. E., Chatfield, M., Brayne, C., Matthews, F. E., & Medical Research Council Cognitive Function and Ageing Study Group (2011). The reliability of assigning individuals to cognitive states using the Mini Mental-State Examination: A population-based prospective cohort study. *BMC Medical Research Methodology*, *11*, Article 127. https://doi.org /10.1186/1471-2288-11-127

Ministero della Salute. (2021). *Rapporto long term care 2021*. Ministero del lavoro e delle politiche sociali.

Molinuevo, D. (2008). *Services for older people in Europe: Facts and figures about long term care services in Europe*. European Social Network. https://health.ec.europa.eu /system/files/2016-11 /services_older_0.pdf

Novelli, G., Papagno, C., Capitani, E., Laiacona, M., Cappa, S. F., & Vallar, G. (1986). Tre test clinici di memoria verbale a lungo termine: Taratura su soggetti normali [Three clinical tests for the assessment of verbal long-term memory function: Norms from 320 normal subjects]. *Archivio di Psicologia, Neurologia e Psichiatria, 47*(2), 278–296.

Palomba, D., Sarlo, M., Angrilli, A., Mini, A., & Stegagno, L. (2000). Cardiac responses associated with affective processing of unpleasant film stimuli. *International Journal of Psychophysiology*, *36*(1), 45–57. https://doi.org/10.1016/s0167-8760(99)00099-9

Pennisi, P., & Sarlo, M. (1998). *Indici elettrofisiologici in psicologia*. CLEUP.

Pfeiffer, E. (1975). A short portable mental status questionnaire for the assessment of organic brain deficit in elderly patients. *Journal of the American Geriatrics Society*, *23*(10), 433–441. https://doi.org/10.1111/j.1532-5415.1975.tb00927.x

Pribram, K. H., & McGuinness, D. (1975). Arousal, activation, and effort in the control of attention. *Psychological Review*, *82*(2), 116–149. https://doi.org/10.1037/h0076780

Pruneti, C. A., & Boem, A. (1995). Physiological response in healthy subjects and in patients after myocardial infarction, elicited by a new computerised version of the Raven Coloured PM 47 as a mental stress test. *Functional Neurology*, *10*(4–5), 195–201.

Pruneti, C., Fontana, F., Carrozzo, E., & Fante, C. (2011). Autonomic reactivity, emotions and stress response in psychopathology. *Applied Psychophysiolology and Biofeedback*, *36*, 218.

Pruneti, C., & Guidotti, S. (2022). Cognition, behavior, sexuality, and autonomic responses of women with Hypothalamic Amenorrhea. *Brain Sciences, 12*(11), 1448. https://doi.org/10.3390/brainsci12111448

Pruneti, C., Guidotti, S., Lento, R. M., & Renda, N. (2022). Dissociation between cognitive-behavioral and emotional-psycho- physiological aspects in eating disorders and its pre-post treatment stability. *Journal of Psychopathology, 28*(1), 30–38. https://doi.org/10.36148/2284-0249-451

Pruneti, C. A., Lento, R. M., Fante, C., Carrozzo, E., & Fontana, F. (2010). Autonomic arousal and differential diagnosis in clinical psychology and psychopathology. *Giornale Italiano di Psicopatologia/Italian Journal of Psychopathology, 16*(1), 43–52.

Pruneti, C., Vanello, N., Paterni, M., Landini, L., Guidotti, S., & Ferdeghini, E. M. (2021). Combined functional magnetic resonance imaging and skin conductance to detect localized neural response to psychological stress: A pilot study. *Archives Italiennes de Biologie*, *159*(1), 21–27. https://doi.org/10.12871/00039829202112

Reda, M. A. (1988). L'atteggiamento diagnostico in psicoterapia cognitiva. *Psicobiettivo, 3*, 31–40.

Reda, M. A. (2016). *Sistemi cognitivi complessi e psicoterapia*. Roma: Carocci Editore.

Roos, V., & Malan, L. (2012). The role of context and the interpersonal experience of loneliness among older people in a residential care facility. *Global Health Action*, *5*(1), Article 18861. https://doi.org/10.3402/gha.v5i0.18861

Syed Elias, S. M., Neville, C., & Scott, T. (2015). The effectiveness of group reminiscence therapy for loneliness, anxiety and depression in older adults in long-term care: A systematic review. *Geriatric Nursing*, *36*(5), 372–380. https://doi.org/10.1016/j.gerinurse.2015.05.004

Tranel, D., & Damasio, H. (1994). Neuroanatomical correlates of electrodermal skin conductance responses. *Psychophysiology*, *31*(5), 427–438. https://doi.org/10.1111/j.1469-8986.1994.tb01046.x

Webster, J. D. (1993). Construction and validation of the Reminiscence Functions Scale. *Journal of Gerontology*, *48*(5), P256–P262. https://doi.org/10.1093/geronj/48.5.p256

Westerhof, G. J., Bohlmeijer, E., & Webster, J. D. (2010). Reminiscence and mental health: A review of recent progress in theory, research and interventions. *Ageing & Society, 30*(4), 697–721. https://doi.org/10.1017/S0144686X09990328

Woods, B., Spector, A., Jones, C., Orrell, M., & Davies, S. (2005). Reminiscence therapy for dementia. *Cochrane Database of Systematic Reviews*, (2), CD001120. https://doi.org/10.1002/14651858.CD001120.pub2

World Health Organization. (2011). *Global health and aging*. National Institute on Aging, National Institutes of Health. https://www.nia.nih.gov/sites/default/files/2017-06/global_health_aging.pdf

World Health Organization. (2012). *Ageing and life course: Interesting facts about ageing*. Geneva: WHO. https://www.who.int/health-topics/ageing

World Health Organization. (2015). *Mental health and older adults*. https://www.who.int/mediacentre/factsheets/fs381/en/

Yen, H.-Y., & Lin, L.-J. (2018). A systematic review of reminiscence therapy for older adults in Taiwan. *Journal of Nursing Research*, *26*(2), 138–150. https://doi.org/10.1097/jnr.0000000000000233

Yesavage, J. A., Brink, T. L., Rose, T. L., Lum, O., Huang, V., Adey, M., & Leirer, V. O. (1982). Development and validation of a geriatric depression screening scale: A preliminary report. *Journal of Psychiatric Research*, *17*(1), 37–49. https://doi.org/10.1016/0022-3956(82)90033-4

Yerkes, R. M., & Dodson, J. D. (1908). The relation of strength of stimulus to rapidity of habit-formation. *Journal of Comparative Neurology and Psychology, 18*(5), 459–482. https://doi.org/10.1002/cne.920180503

Zhou, W., He, G., Gao, J., Yuan, Q., Feng, H., & Zhang, C. K. (2012). The effects of group reminiscence therapy on depression, self-esteem, and affect balance of Chinese community-dwelling elderly. *Archives of Gerontology and Geriatrics*, *54*(3), e440–e447. https://doi.org/10.1016/j.archger.2011.12.003

Received: November 23, 2022
Accepted: December 22, 2022
Published: March 30, 2023

NeuroRegulation

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Psycho-neuro-biological Correlates of Beta Activity

Caroline Leaf[1], Robert P. Turner[2], Charles S. Wasserman[3], René M. Paulson[4], Nicholas Kopooshian[5], Gabrielle Z. Lynch[4], and Alexandria M. G. Leaf[1]*

[1]Switch on Your Brain, LLC, Southlake, Texas, USA
[2]Department of Pediatrics, Medical University of South Carolina, South Carolina, USA
[3]University of Connecticut, Storrs, Connecticut, USA
[4]Elite Research, LLC, Irving, Texas, USA
[5]Drexel University College of Medicine, Philadelphia, Pennsylvania, USA

Abstract

Chronic stress and anxiety in everyday life can lead to sympathetic hyperactivity. This can be observed as behavioral, chemical, and neurological changes, including increased rumination, anxiety, and depression, and chemical changes in biological markers like homocysteine. In the EEG, increased beta (13–30 Hz) wave activity, especially high beta (> 20 Hz) has long been noted in anxiety states. However, recent research indicates that low beta waves (13–20 Hz) may play a role as well. The current paper presents a pilot study that assessed the Neurocycle's efficacy as a nonpharmacological mind-management therapy for people who struggle with anxiety and depression. We assessed psychometrics, blood-serum homocysteine levels, and quantitative electroencephalography (qEEG). Efficacy of the Neurocycle was demonstrated by improved psychometric self-assessment over the study. We observed a positive correlation between subject's low beta relative power and homocysteine levels. The findings validate the Neurocycle's efficacy for improving mental health as measured by behavioral, chemical, and neurological measures. Altogether, these findings support low beta's role in stress/anxiety manifestation given that its modulation significantly correlated with stress biomarkers in patients' blood samples and stress and anxiety self-assessments. Future work should expand these findings with larger datasets to confirm the ranges of healthy and maladaptive low beta.

Keywords: qEEG; beta; stress; anxiety; homocysteine

Citation: Leaf, C., Turner, R. P., Wasserman, C. S., Paulson, R. M., Kopooshian, N., Lynch, G. Z., & Leaf, A. M. G. (2023). Psycho-neuro-biological correlates of beta activity. *NeuroRegulation, 10*(1), 11–20. https://doi.org/10.15540/nr.10.1.11

***Address correspondence to**: Charles S. Wasserman, M.S. 164 Green Manor Rd., Manchester, CT 06042, USA. Email: Charles.Wasserman@uconn.edu

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Edited by:
Rex L. Cannon, PhD, Currents, Knoxville, Tennessee, USA

Reviewed by:
Rex L. Cannon, PhD, Currents, Knoxville, Tennessee, USA
Randall Lyle, PhD, Mount Mercy University, Cedar Rapids, Iowa, USA

Introduction

Increasing evidence suggests a correlation between resting-state electroencephalography (EEG) activity and anxiety symptoms in patients. Specifically, an increase of beta (13–30 Hz) and a decrease in alpha (8–12 Hz) waves have been associated with higher states of anxiety (Hammond, 2005; Ribas et al., 2018; Tharawadeepimuk & Wongsawat, 2014; Thompson & Thompson, 2007). Furthermore, studies evaluating methods of reducing anxiety have found that a decrease in beta activity is directly correlated with lower anxiety levels (Sherlin et al., 2010; Walker, 2010). These results have been consistently verified across multiple clinical conditions (i.e., PTSD, anxiety spectrum disorders), as well as across diverse anxiety treatment methods—from neurofeedback therapy to SSRI treatments to mindfulness and meditation—overall, confirming the relationship between beta wave activity and anxiety factors. However, which ranges of beta specifically play a role in this interrelation have still not been confirmed or normed in the literature. While many studies, including Díaz et al. (2019), have correlated high beta (which they defined as 22–30 Hz) with anxiety factors (Díaz et al., 2019; Tarrant et al., 2018; Tas et al., 2015; Walker, 2010), increased low beta (13–20 Hz) and overall beta activity (13–30 Hz) have also been correlated with anxiety, stress, and fear factors (Ribas et al., 2018). Thus, the current pilot study seeks to contribute to the field's developing knowledge of the relationship between beta wave activity and anxiety to improve understanding of how beta modulation can be integrated into therapy modalities in the treatment of anxiety and depression-related mental health struggles.

Interrelation Between Beta Activity and Anxiety

The past decade of EEG studies has confirmed early seminal research of a relationship between beta wave activity—overall, low, and high—and an umbrella of anxiety factors. Recently, Ribas et al. (2018) provided percentage ranges at which beta activity and anxiety risk factors correlate; measuring at T3 and T4, their qEEG assessments identified levels of overall beta wave activity greater than 17% and high beta wave activity greater than 10% with subjects' fear, panic, insecurity, phobia, and anxiety. Though high beta wave activity is typically associated with anxiety and stress issues (Díaz et al., 2019; Tarrant et al., 2018; Tas et al., 2015; Walker, 2010), findings such as those from Ribas et al. (2018) help to clarify how both low beta and high beta are related to anxiety factors and an increased percentage of either can be correlated to increased anxiety factors. Direct modulation of beta wave amplitude via EEG-based biofeedback (neurofeedback [NFB]) therapy (21–30 Hz) for decreasing anxiety levels has also confirmed the interrelationship of beta wave activity and anxiety factors. Walker (2010) demonstrated how reductions in beta wave amplitude yielded statistically significant reductions in self-reported anxiety, indicating that decreased beta wave activity decreased anxiety symptoms. Moreover, heightened beta amplitudes have been correlated with anxiety in its manifestations in other mental health disorders, including posttraumatic stress disorder (PTSD; Roohi-Azizi et al., 2017) as well as bipolar disorder, schizophrenia, and addiction (Kesebir & Yosmaoğlu, 2020).

Therapy modalities for anxiety have also yielded important findings as to how beta activity and anxiety are related. Clinical studies of how pharmacological anxiety spectrum disorder treatments impact beta wave activity reveal that decreases in anxiety via methods such as SSRI treatment significantly correlate with reductions in prefrontal and frontal beta as well as high beta 6 months post-SSRI treatment (Tas et al., 2015). Another pilot study evaluating the effects of virtual reality on reducing anxiety found that relative power high beta activity decreased while low beta increased after sessions of treatment to decrease anxiety (Tarrant et al., 2018). Traditional holistic therapy modalities for anxiety such as yoga, meditation, and breathing techniques have also demonstrated reduced overall beta wave amplitude alongside improved mental state (Kaushik et al., 2020). As each study's reduction in anxiety via said therapeutic methods resulted in a change of beta wave activity, the multiple modalities of therapies used in these studies all validate the interrelationship between beta wave activity and anxiety factors.

The Search for Consistency in Defining the Beta-Anxiety Relationship

While numerous studies have focused on overall beta and high beta activity, there is a lack of consistency across the definitions of low versus high beta amongst different researchers using varying cutoff frequencies and a lack of consistency regarding which beta wave range is associated with anxiety factors. Between low and high beta, Díaz et al. (2019) suggests that low beta is associated with quiet and introspective thinking, which they termed the "healthy range" of beta. The researchers found that low beta (13–20 Hz) reduced in global coherence (a measurement of interhemispheric comodulation) from 55% to 15–20% when transitioning from a resting state to a demanding task, indicating that coherence within the lower beta frequencies was more closely associated with rest and could be differentiated from higher beta, which can be implicated in anxiety symptoms (Díaz et al., 2019). However, Milner et al. (2020) reported that amongst patients with high tinnitus-related distress, higher-amplitude low beta (13–20 Hz) activity was observed, indicating an association between increased low beta and ruminating cognitive-emotional processing. This type of internally focused thinking is associated with increased low beta and can result in more negative thinking types like rumination (Apazoglou et al., 2019) and anxiety in excess. Some of the most recent research has identified that high-amplitude low beta waves are related to a persistent sympathetic hyperactivity state that influences mental stress (Kopańska et al., 2022). These associations of differing aspects of anxiety with different ranges of Beta frequencies show how the neighboring frequency bands can interact with or be impacted by anxiety levels in distinct manners, and relationships must be assessed across the spectra to understand how anxiety manifests in the qEEG and can therefore be addressed therapeutically.

The Need for a Psycho-Neuro-Biological Approach

The World Health Organization (WHO; 2022) has reported that there has been a 13% increase in mental health conditions and substance abuse disorders since 2019. However, despite this increase, current pharmacological treatments do not offer lasting treatment or resolution for these disorders (Ivanov & Schwartz, 2021), and the aforementioned lack of standardization across the field regarding the relationship between neural frequencies and mental health conditions has necessitated more research. Specifically, Newson and Thiagarajan (2019) called for researchers to contribute to the creation of a large qEEG database that could be assessed to inform and standardize norms of neurological function and related mental health outcomes. The need for more direct application of such neuroscientific research to the development of clinical practices and the treatment of mental illness has also been established (Ivanov & Schwartz, 2021). Given these identified gaps in the research and treatment of mental health conditions and their associated brain wave manifestations, a melding of neurophysiological, psychosocial, and biomedical streams of research are necessary to produce methods of jointly assessing biological and psychosocial measures and tailoring interventions in a patient-centered approach.

As such, the current pilot study takes a novel psycho-neuro-biological approach to the study of beta activity, their association with high/low stress and anxiety, and the effective management and resolution of anxiety symptomatology. To circumvent the lack of established cutoff low and high beta frequency definitions, a unique approach was taken that combines psychological, neurological, and biological measurements of participants' stress and anxiety levels to confirm the relationship between a reduction in anxiety and stress and its corresponding reduction in low beta wave activity for participants. Implementing the Neurocycle—a nonpharmacological, mind-management, and mind-directed neuroplasticity therapy modality for mental health improvement and anxiety and depression-related symptoms reduction—our study aimed to assess whether the Neurocycle intervention has a substantively positive impact on psychological and neurophysiological measures in a population of subjects with mental health and neurological symptoms. The following hypotheses were generated:

> H1: There will be change in the subjects' neurophysiological functioning, as measured by qEEG analysis of low beta relative power throughout the Neurocycle program.
>
> H2: There will be change in the subjects' biophysical anxiety symptoms throughout the completion of the Neurocycle program, as measured by blood serum homocysteine levels.
>
> H3: There will be positive change in the subjects' psychological well-being after the completion of the Neurocycle program, as measured by psychometric assessments of stress and anxiety.

Altogether, this psycho-neuro-biological approach will provide the more detailed neurophysiological data called for by Newson and Thiagarajan (2019) through a mapping of the psychological, neurological, and biological identifiers of anxiety, helping to describe low beta neural activity and its relationship with mental health conditions within the nexus of their neurophysiological, biological, and psychosocial tripartite nature.

Materials and Methods

Study Design

A double-blind randomized clinical trial (RCT) pilot study was selected for its suitability in determining if an intervention has a meaningful effect on key outcome measures of interest and its ability to establish high confidence in causal claims (Spieth et al., 2016). The study design, instruments, and protocol were approved by the Sterling Institutional Review Board (approval ID no. 7281-RPTurner). A total of 14 participants were recruited based on power analysis of convenience sampling; *a priori* power analysis was conducted using G*Power 3.1.9.2, and, assuming a moderate to high effect size ($f = 0.30$, power $[1 - \beta] = 0.80$) and alpha (α) of 0.05 for a between-within subjects analysis of variance (ANOVA) with two groups and six repeated measures, the necessary sample size was verified as 12 to detect a significant effect in the population (Cohen, 1988; Erdfelder et al., 1996; Faul et al., 2007) and an additional two participants for potential attrition during the study period. Participants for this study were recruited from patients and employees of Network Neurology and from additional flyers for this clinical trial posted around Network Neurology and at local colleges within a 15-mile radius of the Network Neurology office. To ensure participants met the recruitment criteria of preexisting anxiety and/or depression, the research team recruited a total initial pool of 30 recruits in a prescreening phase to reach the desired sample size of 14 participants for the pilot study given the current prevalence of depression and generalized anxiety disorders in clinical settings (70–80% [> 14 on the HAM-D; Trivedi et al., 2006] and 50% [> 18 on the HAM-A; Ruiz et al., 2011], respectively).

To select the 14 participants from the initial 30 recruits, inclusion and exclusion criteria were applied. The inclusion criteria for this study consisted of: (a) consent to participate in the study; (b) 18 years of age or older; (c) a score of 14 or above on the HAM-D depression scale; (d) a score of 18 or above on the HAM-A anxiety scale; and (e) completion of the pilot study. The exclusion criteria for this study consisted of: (a) prior experience or familiarity with Dr. Leaf's books, applications, or teachings (due to possible study bias); (b) concurrent diagnosis of epilepsy or refractory depression (due to complexity of comorbid diagnoses); (c) current prescription of more than 3 psychotropic medications (due to confounding factors in brain analysis and masking of symptoms); (d) a score of less than 14 on the HAM-D depression scale; (e) a score of less than 18 on the HAM-A anxiety scale; and/or (f) incomplete study participation.

After the final 14 participants were selected, they were provided with an Informed Consent explaining the purpose and background of the study, its procedures, its duration (including their right to cease participation at any point during the study), the risks and discomfort associated with the assessments (e.g., potential discomfort from blood draw and qEEG procedures), potential benefits to the participants, costs (none) and compensation for the study (access to the Neurocycle app), protection of their privacy, and contact information for the study personnel. The subjects were randomly assigned to the "treatment" group (*n* = 7), the Neurocycle, or the "control" group (*n* = 7), which received no special attention beyond the standard of care of their physician. During the study, attrition occurred following baseline measurements in both groups (control: attrition of *n* = 1, for a final total of *n* = 6; treatment: attrition of *n* = 2, for a final total of *n* = 5). Replacement of missing data was not a possible strategy for addressing attrition given that individualized brain mapping could not be replaced by random values. However, attrition bias was avoided by removing any partial data from participants who dropped out from the final dataset as these participants violated the inclusion criteria of completing the pilot study. Therefore, their entire profiles were removed from the final samples, and data integrity was maintained.

Materials

The intervention utilized the Neurocycle program hosted on the Neurocycle app. The Neurocycle (Leaf, 1997, 2021) is a 63-day mind-directed self-help mental health program created by Dr. Caroline Leaf that is implemented in three phases of 21 days for a total of 63 consecutive days. These three phases are administered through the Neurocycle app, in which participants are directed via daily audio and video recordings through the five-step Neurocycle process of Gather Awareness, Reflect, Write, Recheck, and Active Reach, which provide a scientifically validated framework for participants to identify, face, process, and manage intrusive toxic thoughts that cause distress, including symptoms of anxiety and depression (Idris, 2020; Leaf, 1997, 2021). This approach acknowledges that individuals can reconceptualize and take control of their mental health through mind-management and provides development in the required skills to actualize the benefits of mindfulness: self-regulation, resilience, reconceptualization, and exposure (Shapiro et al., 2006).

Measurements, Instruments, and Data Collection

The psycho-neuro-biological effects of the program were assessed using a novel three-phase structure in a pilot study to test the effectiveness of the Neurocycle. The psychological effects of the Neurocycle were measured by the Leaf Mind Management (LMM) scale and triangulated with the Hospital Anxiety and Depression Scale Anxiety and Depression subscale (HADS-A & HADS-D; Bjelland et al., 2002) and the BBC Subjective Well-Being Scale (BSC; Pontin et al., 2013). The neurophysiological effects of the Neurocycle were assessed using surface qEEG functional analysis. The psychological and neurophysiological effects were then confirmed in bloodwork analysis to measure participants' homocysteine levels, which are known to increase alongside stress, anxiety, and depression (Kevere et al., 2014). This combined approach was designed to address criticisms in the field of psychology that self-assessments are inherently flawed measurement tools on their own due to biases that can be beneath our consciousness or socially motivated (Chen et al., 2013; Karpen, 2018). Additionally, the tripartite approach addresses the lack of consensus in the field of electroencephalography regarding what constitutes high and low beta frequencies and their exact relationship with stress and anxiety in brain function by providing a third measurement to confirm a change in anxiety and stress. The assessments were administered in a staged format that captured key insight into the changes in participants' stress and anxiety across six distinct time periods: preintervention (Day 0), on Days 7, 14, 21, and 42, and postintervention on Day 63. The schedule of assessment administration is provided in Table 1 below, and descriptions of each assessment phase follow.

Table 1
Mean and Standard Errors of Confirmation Measures and Correlations for Treatment Group

Measure	Pre-Screen	Day 0	Day 7	Day 14	Day 21	Day 42	Day 63
Clinical Anxiety (HAM-A)	X						
Clinical Depression (HAM-D)	X						
Psychological Effects (BBC-SWB)		X	X	X	X	X	X
Self-Report Anxiety (HADS-A)		X	X	X	X	X	X
Self-Awareness and Mind Management of Stress and Anxiety (LMM)		X	X	X	X	X	X
Neurophysiological Effects (qEEG)		X			X		X
Bloodwork (Homocysteine)		X			X		X

Neurophysiological Assessment

Participants underwent three qEEG sessions for neuroimaging analysis on Days 0, 21, and 63 to assess neural activity changes from baseline to the completion of the first phase of the intervention (Day 21) and then from this phase to the completion of the entire program (Day 63). For each recording, subjects were seated in a quiet, comfortable room and allowed to relax in a comfortable armchair. Nineteen electrode sites were located according to the international 10-20 system, cleaned using a mild abrasive gel (Nu-Prep), and electrodes tested to obtain impedances below 5 kΩ. Subjects were instructed to sit quietly without movement while EEG was recorded at a 250 Hz sampling rate (Mitsar EEG-201). Subjects were prompted to relax to reduce muscle artifact if noted by the researcher at time of recording. Participants' qEEG was recorded for 10 minutes with their eyes open and another 10 minutes with their eyes closed. Only eyes-open data are reported on in this paper.

Psychological Assessment

Self-assessment of psychometric indicators was provided by participants during all six key stages of the intervention's administration: Days 0, 7, 14, 21, 42, and 63. The primary assessment tool implemented was the LMM scale, which was designed by the principle investigator (PI) to assess autonomy, awareness, toxic thoughts and isolation, toxic stress and anxiety, barriers and challenges, and empowerment and life satisfaction. The LMM has shown strong structural validity and reliability in testing (publication pending); Cronbach's alphas for subfactors ranged from .62 to .90 with an overall factor that ranged from .77 to .80. The LMM measures subjects' changes in awareness, processing, reconceptualization, and control of reactions and responses to the circumstances of life that cause feelings of anxiety and depression. As such, it is a tool for assessing participants' mindfulness of their mental health and the necessary mediators—self-regulation, resilience, reconceptualization, and relived experience—to respond healthily to stress and anxiety. Improvements in stress and anxiety can be measured by increases in the autonomy, awareness, and empowerment subscales alongside decreases in the toxic thoughts, toxic stress, and barriers subscales. To validate the LMM assessment in this study, traditional measures of anxiety, stress, and depression were also administered, including the HADS-A, HADS-D (Bjelland et al., 2002), and BBC-SWB (Pontin et al., 2013) instruments. The HADS-A and HADS-D are 4-point Likert scale each with seven items possessing strong validity and reliability with Cronbach alphas that range from .68 to .89 (Bjelland et al., 2002). Likewise, the BBC-SWB is a 5-point Likert scale with 24 items and has been found both a reliable and valid instrument that also possesses strong Cronbach alphas that range from .74 to .95, indicating very strong reliability (Pontin et al., 2013). By administering these instruments across six time periods, the evolution of change in the participants' well-being, depression, and stress and anxiety levels could be tracked alongside and between qEEG and blood measurements, filling in the qualitative explanation of the participants' mental health changes.

Biological Assessment

Participants were sampled for blood-measured homocysteine levels, elevated levels of which are known to be associated with an individual's elevated stress and anxiety levels and direct neurotoxic effects (Aghayan et al., 2020; Chung et al., 2017) given that this sulphurated amino acid is responsible for mediating methylation, which is critical for nervous system balance and health (Kennedy, 2016). This assessment was performed in three parts. Blood samples were drawn by a contracted phlebotomist in 10 mL vials preintervention on Day 0, after the initial phase of the intervention on Day 21, and postintervention on Day 63. Blood amino acid analysis for homocysteine levels was then performed by a contracted lab and reported to the researchers as follows: normal range: 5–15 mcmol/L; moderately elevated range: 15–30 mcmol/L; intermediately elevated range: 30–100 mcmol/L; and severely elevated range: < 100 mcmol/L (Haldeman-Englert et al., 2022).

The qEEG data for each subject was preprocessed using the Harvard Automated Preprocessing Pipeline for Electroencephalography (HAPPE; Gabard-Durnam et al., 2018) to remove artifactual contributions to the data such as eye, muscle, electrical, and movement-related artifacts. The resulting data was analyzed using a sliding window FFT to obtain power spectral density estimates for each electrode site. Then, relative power was calculated for each frequency band relative to the total power in the 1–80 Hz range. Relative power was used for analyses to allow direct comparison from one subject to another, controlling for interpersonal differences in overall EEG amplitude. In this study, all-electrode-averaged low beta relative power (13–20 Hz) was analyzed.

The data gathered from the qEEG, bloodwork, and psychometric assessments were analyzed altogether using IBM SPSS v27. Overall study analysis was examined with the original planned mixed (between-within subjects) ANOVA with the two groups (treatment and control) over six repeated measures (the pretest and five follow-up measures) was performed. The overall main effects of group, time, and the interaction of group and time were assessed to determine if the effects of the intervention had an impact on the study outcomes. Pairwise group comparisons over time were calculated using the Bonferroni method to adjust for multiple comparisons. To examine the specific hypotheses outlined in this paper, linear multiple regression models and simple regressions were conducted to examine the relationships among the specific variables of interest, as well as nonparametric correlations to assess potential triangulating relationships. The alpha (α) level for this pilot study was set at .10.

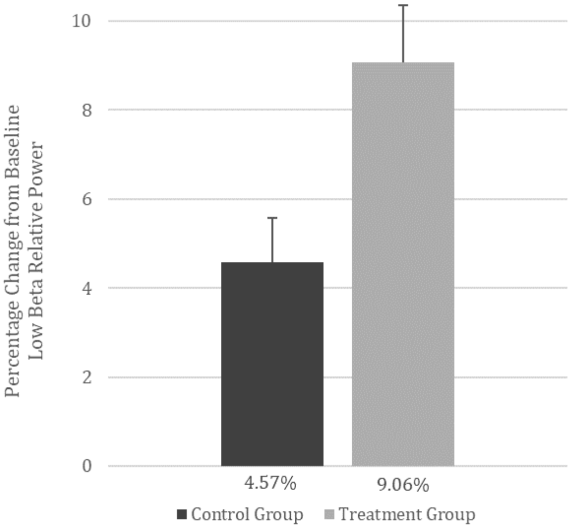
Results

Our multivariate linear regression model showed that the LMM toxic-stress subscale and homocysteine levels were significant predictors and accounted for 41.4% of the variance of global average low beta relative power changes from Day 1 to Day 63, $F = 4.49$, $p < .05$, $R^2 = 41.4\%$. Looking at the individual predictors of the model, we can see that the strongest indicator was the change in homocysteine with a beta coefficient (standardized) of .613 ($p = .036$). Additionally, the LMM toxic stress subscale change was meaningful (moderate) at .395 ($p = .142$). These results indicate that the greater change in homocysteine was a prime predictor of change in average low beta relative power. Furthermore, within participants' change in the LMM toxic stress over the course of the study, greater change in toxic stress was related to greater change in average low beta relative power regardless of homocysteine levels.

These results confirmed H1, H2, and H3. Overall, participants' average low beta relative power changes correlate with the trajectory of change in neurophysiological functioning during the Neurocycle. At baseline there was no statistically significant difference in low beta relative power between the treatment and control group, $t(5.89) = 1.60$, $p = .118$, but by Day 21 we observe a statistically significant difference between the groups, $t(9) = 1.71$, $p = .089$, see Figure 1.

The neurophysiological improvement is confirmed in the correlations of decreased LMM Toxic Stress subscale scores with decreased HADS-A Anxiety ($\rho = .894$, $p < .001$) and HADS-D Depression ($\rho = .592$, $p = .046$) subscale scores for intervention participants. Together, these correlations validated H1.

Figure 1. *Low Beta (13–20Hz) Relative Power Percentage Change From Baseline to Day 21 for the Treatment and Control Groups.*



H2 was confirmed through the corresponding correlation between average low beta relative power and blood serum homocysteine levels (ρ = .755, p = .007). Given that homocysteine and average low beta relative power wave activity decreased from Day 21 to 63, as self-reported anxiety and depression improved as evidenced by the lowered HADS-A and LMM Toxic Stress scores (ρ = .894, p-value < .001), biophysical anxiety symptoms were clearly lessened. Thus, positive change occurred, confirming H2.

Additionally, the same psychometric assessments confirmed that intervention participants experienced a reduction in their anxiety from Day 21 to Day 63 of the program. Analyses confirmed a statistically significant change in participants' low beta relative power (M_{diff} = .0052, SE = .003, t = 1.75, p = .078). Due to low sample sizes in the pilot study, multivariate correlational analyses by group were not possible; however, there are corresponding relationships of percent change low beta relative power with change in homocysteine levels (ρ = .852, p = .033), and the psychometric tests of depression and anxiety via lowered HADS-A (ρ = .866, p = .067) and LMM Toxic Stress scores (ρ = .689, p = .099), see Table 2. Thus, H3 was confirmed.

Table 2
Mean and Standard Errors of Confirmation Measures and Correlations for Treatment Group

Measure	Day 21 Mean	Day 21 SE	Day 63 Mean	Day 63 SE	% Change Low Beta Correlation
Low Beta Relative Power	0.118	0.005	0.112	0.007	-
Bloodwork (Homocysteine)	187.80	23.64	173.69	17.06	.852*
Self-Report Anxiety (HADS-A)	7.25	3.25	7.00	3.03	.866*
Self-Awareness and Mind Management of Stress and Anxiety (LMM)	6.00	.32	5.25	.37	.689*

Note. *Significant correlation (ρ) with percent change from baseline low beta relative power, p < .10.

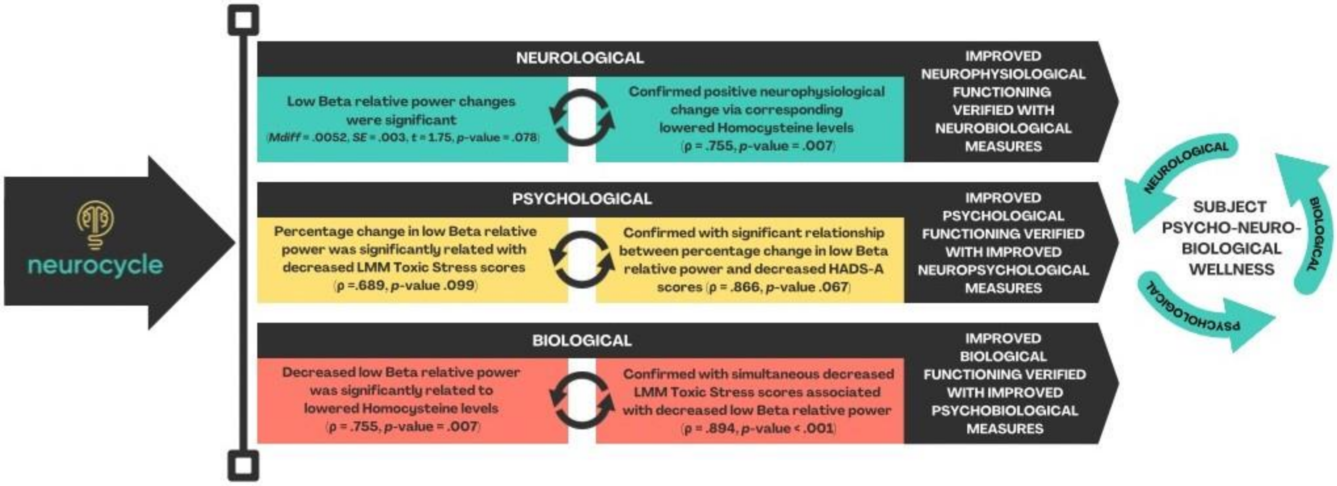
Discussion

Though low beta has historically been associated with positive mental state aspects, such as focused energy (Abhang et al., 2016; Díaz et al., 2019; Tarrant et al., 2018), the musing thought capability of this wavelength can become detrimental if too high a relative power is reached (Apazoglou et al., 2019). For instance, abundant high-amplitude low beta wave activity is related to persistent sympathetic hyperactivity that influences mental stress (Kopańska, 2022). Thus, the relative power of low beta appears to be a factor in the modulation between the self-monitoring and internal focus capabilities of beta and more toxic applications of reflective thought, such as rumination (Apazoglou et al., 2019). Figure 2 displays how the psycho-neuro-biological results of the current study support this understanding of low beta wave relative power modulated in relation to overall subject wellness.

The current study's results suggest that changes in global average low beta relative power and blood serum homocysteine levels are associated with participants' anxiety and stress, as indicated by the HADS-A and LMM Toxic Stress scale. The results from the three different manners of measurement—neurological measurement with the qEEG, psychological measurement with the LMM and HADS-A, and biological measurement with homocysteine—show interactive, statistically significant relationships that validate the data through each field. While all subject's low beta increased from the beginning of the study to Day 21, that increase was significantly stronger in the treatment group as they engaged in the treatment process, as shown in Figure 1. It is important to acknowledge that improving mental health is not a linear process from start to end and experiencing an increase in symptoms before they get better is common across many therapeutic modalities. Throughout the rest of the study from Day 21 to 63, each independent measurement modality was verified by two other modalities, as described in Figure 2. The positive neurophysiological change resulting from the decrease in global average low beta relative power from Day 21 to Day 63 of the study was supported by the biological measurement of the participants' decreased homocysteine levels. Following, the improved biological functioning resulting from the decreased blood homocysteine levels was verified with psychosocial assessments of participants' decreased stress and anxiety. Coming full circle, this improved psychological functioning was then verified by correlating both sets of significant results from the two psychosocial assessments—the HADS-A and LMM Toxic Stress scale—with their significant association with global average low beta relative power. While the finding of association between global low beta, homocysteine, and psychometric measure of stress was found over the entire set of participants, only the treatment group showed a significant reduction in symptoms as shown by the decrease in the HADS-A and LMM Toxic Stress scale. It is important to note that the qEEG recordings were made during an at-rest condition that was not designed to elicit any specific emotional response, which may account for some of the differences in beta frequencies engaged between this study and other findings in the qEEG literature featuring studies that utilized varied levels of stressors (Díaz et al., 2019; Ribas et al., 2018; Tharawadeepimuk & Wongsawat, 2014).

Answering Newson and Thiagarajan's (2019) call for more qEEG contributions toward the understanding of neurological function and related mental health outcomes, these tripartite statistical relationships have therefore shown that low beta is involved in the management of anxiety. Furthermore, the current data indicates that lower low beta relative power may be associated with improved perspectives of subjects' stress and anxiety. This finding emphasizes the importance of addressing low beta when dealing with anxiety and mental well-being, thereby emphasizing the significance of the Neurocycle as a mindfulness tool that directly interfaces with low beta wave activity. As this was a

Figure 2. *Summary of the Psycho-Neuro-Biological Impact of the Neurocycle Program (Days 21–63): Global Average Low Beta Relative Power, Homocysteine, and Psychosocial Measurements.*



Note. LMM = Leaf Mind Management Scale; HADS-A = Hospital Anxiety and Depression Scale: Anxiety Subscale.

pilot study, future research should confirm these relationships with larger data sets and longitudinal studies to provide normative ranges for understanding low beta's involvement in anxiety magnification and mitigation. Such ranges could inform therapy modalities and improve patient care with treatments that directly address the manifestation of anxiety at its psycho-neuro-biological roots (Ivanov & Schwartz, 2021).

Conclusion

The present pilot study was conducted to assess the efficacy of the Neurocycle for improving the psycho-neuro-biological wellness of participants as measured by global average low beta relative power, homocysteine blood levels, LMM Toxic Stress subscale scores, and HADS-A scores. Neurophysiological changes were observed as an indicator of improved mental wellness through improved psychosocial state as indicated by decreased LMM Toxic Stress subscale scores and decreased HADS-A anxiety scores. Neurological and mental improvement was validated with measurement of decreased homocysteine and low beta levels, from Day 21 to Day 63 of the study, coinciding with decreased self-report of symptoms of stress and anxiety. The correlation of these results provides novel support for the connection between low beta and poor mental health indicators such as rumination or active anxious focus.

Though high beta is typically associated with stress and anxiety, the reduction of low beta wave amplitude in the current results was significantly associated with lowered participant stress and anxiety, revealing that both low and high beta are involved in the mind management of stress and anxiety. Altogether, this study's psycho-neuro-biological approach provides evidence for the efficacy of the Neurocycle for mind management and stress and anxiety reduction. Continued work should expand the data from this pilot with larger-scale and longitudinal research to establish the exact ranges of beneficial versus maladaptive low beta.

Author Declarations

Dr. Caroline Leaf has ownership in Switch on Your Brain and the Neurocycle and financially benefits from royalties for the intellectual property that is subject to evaluation or improvement through the research presented here. Financial interest concerns were addressed through the adoption of a double-blind research design and involvement of a third-party research consultation firm. There are no conflicts of interest or grant support to disclose.

References

Abhang, P. A., Gawali, B., & Mehrotra, S. C. (2016). *Introduction to EEG-and speech-based emotion recognition*. Academic Press.

Aghayan, S. S, Farajzadeh, A., Bagheri-Hosseinabadi, Z., Fadaei, H., Yarmohammadi, M., & Jafarisani, M. (2020). Elevated homocysteine, as a biomarker of cardiac injury, in panic disorder patients due to oxidative stress. *Brain and Behavior*, *10*(12), Article e01851. https://doi.org/10.1002/brb3.1851

Apazoglou, K., Küng, A.-L., Cordera, P., Aubry, J.-M., Dayer, A., Vuilleumier, P., & Piguet, C. (2019). Rumination related activity in brain networks mediating attentional switching in euthymic bipolar patients. *International Journal of Bipolar Disorders*, *7*(1), Article 3. https://doi.org/10.1186/s40345-018-0137-5

Bjelland, I., Dahl, A. A., Haug, T. T., & Neckelmann, D. (2002). The validity of the Hospital Anxiety and Depression Scale: an updated literature review. *Journal of Psychosomatic Research*, *52*(2), 69–77. https://doi.org/10.1016/S0022-3999(01)00296-3

Chen, Y., Shi, W., & Ying, H. (2013). The self-evaluation bias in rating one's ability: The Dunning-Kruger effect. *Advances in Psychological Science*, *21*(12), 2204–2213. https://doi.org/10.3724/SP.J.1042.2013.02204

Chung, K.-H., Chiou, H.-Y., & Chen, Y.-H. (2017). Associations between serum homocysteine levels and anxiety and depression among children and adolescents in Taiwan. *Scientific Reports*, *7*(1), Article 8330. https://doi.org/10.1038/s41598-017-08568-9

Cohen, J. (1988). Set correlation and contingency tables. *Applied Psychological Measurement*, *12*(4), 425–434. https://doi.org/10.1177/014662168801200410

Díaz, H., Cid, F. M., Otárola, J., Rojas, R., Alarcón, O., & Cañete, L. (2019). EEG Beta band frequency domain evaluation for assessing stress and anxiety in resting, eyes closed, basal conditions. *Procedia Computer Science*, *162*, 974–981. https://doi.org/10.1016/j.procs.2019.12.075

Erdfelder, E., Faul, F., & Buchner, A. (1996). GPOWER: A general power analysis program. *Behavior Research Methods, Instruments, & Computers*, *28*, 1–11. https://doi.org/10.3758/BF03203630

Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). G* Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods*, *39*(2), 175–191. https://doi.org/10.3758/BF03193146

Gabard-Durnam, L. J., Mendez Leal, A. S., Wilkinson, C. L., & Levin, A. R. (2018). The Harvard Automated Processing Pipeline for Electroencephalography (HAPPE): Standardized processing software for developmental and high-artifact data. *Frontiers in Neuroscience*, *12*, 97. https://doi.org/10.3389/fnins.2018.00097

Haldeman-Englert, C., Turley, R., & Novick, T. (2022). Homocysteine. In *Health Encyclopedia*. University of Rochester Medical Center.

Hammond, D. C. (2005). Neurofeedback treatment of depression and anxiety. *Journal of Adult Development*, *12*, 131–137. https://doi.org/10.1007/s10804-005-7029-5

Idris, Z. (2020). Quantum physics perspective on electromagnetic and quantum fields inside the brain. *The Malaysian Journal of Medical Sciences*, *27*(1), 1–5. https://doi.org/10.21315/mjms2020.27.1.1

Ivanov, I., & Schwartz, J. M. (2021). Why psychotropic drugs don't cure mental illness—But should they? *Frontiers in Psychiatry*, *12,* Article 579566. https://doi.org/10.3389/fpsyt.2021.579566

Karpen, S. C. (2018). The social psychology of biased self-assessment. *American Journal of Pharmaceutical Education*, *82*(5), 6299. https://doi.org/10.5688/ajpe6299

Kaushik, M., Jain, A., Agarwal, P., Joshi, S. D., & Parvez, S. (2020). Role of yoga and meditation as complimentary therapeutic regime for stress-related neuropsychiatric disorders: Utilization of brain waves activity as novel tool. *Journal of Evidence-Based Integrative Medicine*, *25*. https://doi.org/10.1177/2515690x20949451

Kennedy, D. O. (2016). B vitamins and the brain: Mechanisms, dose and efficacy—A review. *Nutrients*, *8*(2), 68. https://doi.org/10.3390/nu8020068

Kesebir, S., & Yosmaoğlu, A. (2020). QEEG-spectral power density of brain regions in predicting risk, resistance and resilience for bipolar disorder: A comparison of first degree relatives and unrelated healthy subjects. *Heliyon*, *6*(6), Article e04100. https://doi.org/10.1016/j.heliyon.2020.e04100

Kevere, L., Purvina, S., Bauze, D., Zeibarts, M., Andrezina, R., Piekuse, L., Brekis, E., & Purvins, I. (2014). Homocysteine and MTHFR C677T polymorphism in children and adolescents with psychotic and mood disorders. *Nordic Journal of Psychiatry*, *68*(2), 129–136. https://doi.org/10.3109/08039488.2013.782066

Kopańska, M., Dejnowicz-Velitchkov, A., Bartman, P., & Szczygielski, J. (2022). MiniQEEG and neurofeedback in diagnosis and treatment of COVID-19-related panic attacks: A case report. *Brain Sciences*, *12*(11), 1541. https://doi.org/10.3390/brainsci12111541

Leaf, C. M. (1997). *The Mind-mapping Approach: A model and framework for Geodesic Learning* (Doctoral dissertation, University of Pretoria). https://hdl.handle.net/2263/71220

Leaf, C. (2021). *Cleaning up your mental mess: 5 simple, scientifically proven steps to reduce anxiety, stress, and toxic thinking*. Baker Books.

Milner, R., Lewandowska, M., Ganc, M., Nikadon, J., Niedziałek, I., Jędrzejczak, W. W., & Skarżyński, H. (2020). Electrophysiological correlates of focused attention on low- and high-distressed tinnitus. *PLoS ONE*, *15*(8), Article e0236521. https://doi.org/10.1371/journal.pone.0236521

Newson, J. J., & Thiagarajan, T. C. (2019). EEG frequency bands in psychiatric disorders: A review of resting state studies. *Frontiers in Human Neuroscience*, *12*, 521. https://doi.org/10.3389/fnhum.2018.00521

Pontin, E., Schwannauer, M., Tai, S., & Kinderman, P. A. (2013). A UK validation of a general measure of subjective well-being: The modified BBC subjective well-being scale (BBC-SWB). *Health and Quality of Life Outcomes*, *11*(1), Article 150. https://doi.org/10.1186/1477-7525-11-150

Ribas, V. R., Ribas, R. G., Nóbrega, J. D., Nóbrega, M. V., Espécie, J. A., Calafange, M. T., Calafange, C. D., & Martins, H. A. (2018). Pattern of anxiety, insecurity, fear, panic and/or phobia observed by quantitative electroencephalography (QEEG). *Dementia & Neuropsychologia*, *12*(3), 264–271. https://doi.org/10.1590/1980-57642018dn12-030007

Roohi-Azizi, M., Azimi, L., Heysieattalab, S., & Aamidfar, M. (2017). Changes of the brain's bioelectrical activity in cognition, consciousness, and some mental disorders. *Medical Journal of the Islamic Republic of Iran*, *31*(1), 53. https://doi.org/10.14196/mjiri.31.53

Ruiz, M. A., Zamorano, E., García-Campayo, J., Pardo, A., Freire, O., & Rejas, J. (2011). Validity of the GAD-7 scale as an outcome measure of disability in patients with generalized anxiety disorders in primary care. *Journal of Affective Disorders*, *128*(3), 277–286. https://doi.org/10.1016/j.jad.2010.07.010

Shapiro, S. L., Carlson, L. E., Astin, J. A., & Freedman, B. (2006). Mechanisms of mindfulness. *Journal of Clinical Psychology*, *62*(3), 373–386. https://doi.org/10.1002/jclp.20237

Sherlin, L., Arns, M., Lubar, J., & Sokhadze, E. (2010). A position paper on neurofeedback for the treatment of ADHD. *Journal of Neurotherapy*, *14*(2), 66–78. https://doi.org/10.1080/10874201003773880

Spieth, P. M., Kubasch, A. S., Penzlin, A. I., Illigens, B. M. W., Barlinn, K., & Siepmann, T. (2016). Randomized controlled trials–A matter of design. *Neuropsychiatric Disease and Treatment*, *12*, 1341–1349. https://doi.org/10.2147/NDT.S101938

Tarrant, J., Viczko, J., & Cope, H. (2018). Virtual reality for anxiety reduction demonstrated by quantitative EEG: A pilot study. *Frontiers in Psychology*, *9*, 1280. https://doi.org/10.3389/fpsyg.2018.01280

Tas, C., Cebi, M., Tan, O., Hizli-Sayar, G., Tarhan, N., & Brown, E. C. (2015). EEG power, cordance and coherence differences between unipolar and bipolar depression. *Journal of Affective Disorders*, *172*, 184–190. https://doi.org/10.1016/j.jad.2014.10.001

Tharawadeepimuk, K., & Wongsawat, Y. (2014). QEEG evaluation for anxiety level analysis in athletes. In *The 7th 2014 Biomedical Engineering International Conference* (pp. 1–4). Fukuoka, Japan: IEEE. https://doi.org/10.1109/BMEiCON.2014.7017400

Thompson, L., & Thompson, M. (2007). Autistic spectrum disorders including Asperger's syndrome EEG & QEEG findings, results, & neurophysiological rationale for success using neurofeedback training. In *Applied Psychophysiology and Biofeedback* (vol. 32, no. 3–4, pp. 213–213). Springer.

Trivedi, M. H., Rush, A. J., Wisniewski, S. R., Nierenberg, A. A., Warden, D., Ritz, L., Norquist, G., Howland, R. H., Lebowitz, B., McGrath, P. J., Shores-Wilson, K., Biggs, M. M., Balasubramani, G. K., Fava, M., & STAR*D Study Team (2006). Evaluation of outcomes with citalopram for depression using measurement-based care in STAR*D: implications for clinical practice. *The American Journal of Psychiatry*, *163*(1), 28–40. https://doi.org/10.1176/appi.ajp.163.1.28

Walker, J. E. (2010). Using QEEG-guided neurofeedback for epilepsy versus standardized protocols: Enhanced effectiveness? *Applied Psychophysiology and Biofeedback*, *35*, 29–30. https://doi.org/10.1007/s10484-009-9123-0

World Health Organization [WHO]. (2022). World mental health report: Transforming mental health for all. Mental Health and Substance Use. https://www.who.int/teams/mental-health-and-substance-use/world-mental-health-report

Received: February 8, 2023
Accepted: February 26, 2023
Published: March 30, 2023

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Intractable Epilepsy Controlled by Neurofeedback and Adjunctive Treatments: A Case Report

Robert P. Turner[1], Vietta E. Wilson[2], Jay D. Gunkelman[3], Alexandra A. Harvison[4], and Linda A. Walker[5]*

[1]Network Neurology Health, Charleston, South Carolina, USA
[2]York University, Toronto, Ontario, Canada
[3]Brain Science International, Pinole, California, USA
[4]Behavioral Psychology, Brno, Czech Republic
[5]Inland Seas Neurotherapy and Counseling, Traverse City, Michigan, USA

Abstract

This case report documents the treatment of a female patient with intractable temporal lobe epilepsy with secondary generalization. At the age of 13, the patient was hospitalized with ~120 seizures in a day, some of which were life-threatening. After hospital discharge, despite a regimen of multiple antiseizure medications, the patient still experienced ~90 seizures per day. After the interventions described in this work, over 500 neurofeedback sessions guided by EEG or qEEG data and adjunctive treatments including mental skills coaching, the patient became seizure- and medication-free, progressing from poor academic performance and inability to carry out normal daily life to attending university as a student athlete playing an NCAA Division I sport. This case emphasizes that, with professional guidance and supervision, it is possible for people with epilepsy or their caregivers to provide the extensive, long-term neurofeedback and adjunctive training necessary for reduction and control of intractable seizures.

Keywords: epilepsy; qEEG; EEG; neurofeedback; mental training; case report

Citation: Turner, R. P., Wilson, V. E., Gunkelman, J. D., Harvison, A. A., & Walker, L. A. (2023). Intractable epilepsy controlled by neurofeedback and adjunctive treatments: A case report. *NeuroRegulation, 10*(1), 21–30. https://doi.org/10.15540/nr.10.1.21

***Address correspondence to**: Robert Turner, MD, 2245-C Ashley Crossing Drive, PMB 163, Charleston, SC, USA. Email: robertturner@networkneurology.com

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Edited by:
Rex L. Cannon, PhD, Currents, Knoxville, Tennessee, USA

Reviewed by:
Rex L. Cannon, PhD, Currents, Knoxville, Tennessee, USA
Randall Lyle, PhD, Mount Mercy University, Cedar Rapids, Iowa, USA

Introduction

Epilepsy is a common disorder affecting approximately 85 million people worldwide (Singh & Trevick, 2016), including an estimated 470,000 children in the United States. Epilepsy requires self-management to optimize seizure control and minimize impact (Centers for Disease Control and Prevention, 2020). Current allopathic treatments for epilepsy include antiseizure medications, but at least one-third of people with epilepsy (PWE) still have persistent, uncontrolled seizures, currently defined as "intractable epilepsy" (Asadi-Pooya et al., 2016). Children on antiseizure medications demonstrate worse educational and health outcomes, more hospitalizations, and increased morbidity and mortality when compared with age-matched peers (Fleming et al., 2019).

PWE are often referred for possible epilepsy surgery, though reported success rates vary by center. At one center, more than 75% of patients who underwent appropriate epilepsy surgery experienced a meaningful improvement in quality of life (QOL; Benevides et al., 2021), and another center has reported that 47% of those who underwent epilepsy surgery were seizure free at 5-year follow-up (Mohan et al., 2018). Risks and complications of epilepsy surgery may include visual field defects, motor impairments, intracranial complications, and neurocognitive dysfunction (Kohlhase et al., 2021). Given that epilepsy is a disease of neural networks, it is to be expected that focal resective epilepsy surgical interventions are often unable to render freedom from seizures (Engel, 2013; Engel et al., 2013).

Adjunctive or complementary treatments for epilepsy approaches often address the broader health aspects affecting seizures in PWE. These interventions include nutritional therapies, lifestyle changes, cognitive training, and behavioral treatments that are likely underutilized in the treatment of epilepsy (Haut et al., 2019; Yardi et al., 2020). In a 2021 physician survey, approximately 70% of 1,000 physicians from 25 countries endorsed the use of complementary or alternative modalities (Mesraoua et al., 2021). It is likely that patients and caregivers will continue to seek out these alternative strategies in situations where antiseizure medications fail to control seizures or produce unacceptable side effects (Nagai et al., 2019).

Biofeedback (BFB) and Neurofeedback (NFB) as an Alternative Treatment

Electroencephalogram (EEG) BFB, also called NFB, was first reported by M. B. Sterman, who identified and operantly trained the sensorimotor rhythm (SMR) in cats (Sterman et al., 1969; Wyrwicka & Sterman, 1968). This led to successfully applying SMR NFB training to PWE (Sterman et al., 1974). Examples of case studies (Selaa & Shaked–Toledanob, 2014) and small group nonrandomized studies (Frey, 2016; Kohlhase et al., 2021) have reported favorable results. A meta-analysis reported SMR training significantly decreased seizure rate in more than 70% of the cases reviewed (Tan et al., 2009). A 2019 review concluded that NFB is possibly efficacious in the treatment of pediatric epilepsy, though lacking sufficient research (Nigro, 2019). A recent double-blind, sham-controlled study of children and adolescents with epilepsy noted significant improvements in cognitive functioning and quality of life measures following NFB training (Morales-Quezada et al., 2019).

Case Presentation

Ethical Approval

IRB approval is not required for case reports. The patient, now an adult, read and approved this document prior to publication and provided informed consent for use of her medical history.

Early Evidence of Epilepsy

Table 1 lists a brief chronology of the patient's symptoms, attempted interventions, as well as any symptom changes that correlated in time with interventions. The information presented was retrieved from the patient's medical records and contemporaneous notes maintained by her mother.

Table 1

Chronology of Symptoms, Signs, and Interventions

Age (Years)	Symptom Summary	Intervention	Any Apparent Symptom Changes After Intervention
8–10	Forgetful, anxious, epigastric pain, ADHD diagnosis. EEG: single paroxysm consistent with seizure.	NFB: begins training series for ADHD symptoms.	Slight improvement in emotions, but attention problems persisted.
11–12	Difficulty with peers, emotional, can't recall instructions; blank staring. EEG: epileptiform discharges	NFB: same; Other: social skills training.	Better outward management of emotions. Sport: won regional titles.
13 (Jan–Apr)	Sharp school decline, eye flutters, TLE diagnosis, absence seizures every 5–10 min, fatigue. Sport: impaired but some wins; EEG: seizure activity; MRI: No struct defect.	NFB: same; Sleep: naps for fatigue.	
13 (Late Apr)	Admitted to ER; episode of status epilepticus; first tonic-clonic seizures, peak of 250 seizures/day.	Med: (in ER) DZP, MDZ; (later in hospital) LEV, LAC; NFB: now treating epilepsy.	Sedated by meds. Seizures reduced to ~90 per day. EEG: seizures reduced during NFB recording sessions.
13 (May–Jun)	Hospital discharge, multiple tonic-clonic seizures/day, poor memory/coherence, weak, seizures more severe during menses.	Med: LEV, LAC, CBZ; NFB: same; Sleep: extended daily naps.	Seizures reduced to ~80 per day with continued aura, epigastric pain, sleep seizures, heart and lung stoppage.

Table 1

Chronology of Symptoms, Signs, and Interventions

Age (Years)	Symptom Summary	Intervention	Any Apparent Symptom Changes After Intervention
14 (Jul)	~80–90 seizures/day, with LOC 70% of time, drop seizures with unbearable epigastric pain.	Sport: mental skills train; Diet: low glycemic, supplements, probiotics, CBD, homeopathy, acupuncture; Med: reduced; NFB: 5–8 1-min sessions/day; BFB: diaphragmatic breathing/HRV; Sleep: same.	Able to do more activities; fitness level improved, able to use visualization of sports skills. Doctors pleased with seizure reduction progress.
14 (Aug–Oct)	Drop seizures while playing sports during shifts in emotion, sharp, unbearable epigastric pain; most intense when striking the ball; ~60 episodes/day.	Sport: mental skills train, daily practice between seizures but none during menses; Diet: low glycemic, supplements, probiotics; homeopathy; Med: none; NFB/BFB: increased; Sleep: same.	Won sports tournament in spite of multiple seizures during matches. Still weak.
14 (Nov–Feb)	Epigastric pain, fatigue despite long sleep, fewer seizures overall, but intense 2- to 5-min lung-stopping tonic-clonics during week of menses (~15/day). Anxious and tearful.	Sport: same; Diet: low glycemic, probiotics, zinc, selenium, magnesium, B-complex; NFB: target seizure foci, increased session time; BFB: increased; Sleep: same.	Seizures reduce to 30–40/day with fewer lung stoppages; reduced fatigue/epigastric pain; fewer tonic-clonics; Sport: played well, 2–3 tonic-clonics during games; EEG: elevated beta but no waking seizures; Sleep EEG: no night seizures.
14–15 (Apr–Jul)	Improved sleep and strength, able to play many sports matches except during menses. Seizures: fewer with LOC; able to remain coherent, hand stiffening common.	Diet: low glycemic, supplements; NFB: same; BFB: learned to use HRV and diaphragm breathing to reduce length of seizures and avoid onset of lower intensity seizures; Sleep: same.	Epigastric pain ceases, reduced LOC during seizures with more ability to communicate/function. Sport: attained international ranking; EEG: reduced beta amp from 16 SD to 8 SD; BFB: felt empowered to prevent or blunt seizures.
15 (Aug)	Shorter, milder seizures, ~8/day, LOC uncommon (< 2%). Hand stiffening only.	Diet, NFB, BFB, Sleep: same.	Felt increased control. EEG: beta amp dec further; Sport: professional tournaments, played during menses.
15 (Oct–Dec)	~5 episodes/day; last drop seizure noted; dizzy but no LOC; EEG: slowing but no discharges, beta amp improved.	Diet: low glycemic; NFB, BFB: same; Sleep: naps discontinued.	Reduced to ~3 episodes/day; Reduced anxiety, went on walks alone, straight A's, built peer relationships, less anxiety; Sport: games now possible during menses.
16–17	Occasional dizziness, no LOC, no drop seizures; hands can stiffen for 10–15 s if fatigued. Improved focus under stressful conditions.	Diet: dairy-free and reduced gluten; NFB, BFB: focused on motor quieting for performance, rather than seizures, 1–2/week.	Academic improvement, able to travel on flights; Sport: increased stamina for multiple matches, awarded full NCAA Division I university scholarship.
17 (Mar–Jun)	Two brief, mild episodes of epileptic activity, both associated with lack of sleep. No LOC.	Diet: gluten- and dairy-free; NFB: intense 30 sessions over 6 weeks; BFB: same.	EEG: beta amplitudes normalized. Sport: semifinalist in two professional tournaments.
18	Seizure-free, difficulty with sleep.	NFB: 1/week; BFB: 2–3/week.	Attended university away from home as an athlete.

BFB: biofeedback; CBD: cannabidiol; CBZ: clobazam; DZP: diazepam; HRV: heart rate variability training; LAC: lacosamide; LEV: levetiracetam; LOC: loss of consciousness; MDZ: midazolam; MSC: mental skills coaching; NFB: neurofeedback; TLE: temporal lobe epilepsy.

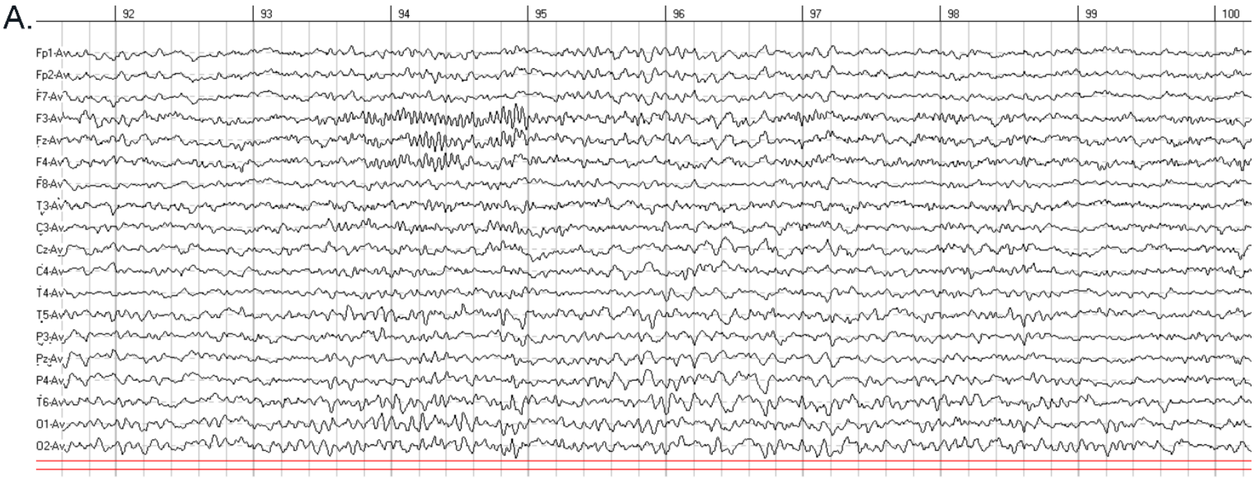
At 8 years of age (2010), the patient's teachers reported attention issues such as forgetting instructions and assignments, and over the following year she developed anxious behaviors and difficulty relating to her peers.

At 10.5 years old, an EEG recorded the first report of possible epileptiform activity, although there were no apparent outward or behavioral manifestations of epilepsy. On a follow-up EEG at age 11 (2013), the neurologist highlighted that the EEG was "consistent with epileptic seizure activity" (Figure 1). In this recording, 20–24 Hz beta spindles are present frontally and fast 12–13 Hz alpha frequencies are present posteriorly, both indicating CNS overarousal. Subtle spikes and slower activity are also noted left temporally (T5). All EEGs for this case review were collected in the eyes-open (EO) and eyes-closed (EC) conditions according to the American Clinical Neurophysiology Society guidelines using the International 10–20 electrode placement system, and all were reviewed and interpreted by neurologists before an NFB specialist decided on training locations and frequencies. The EEGs were processed using a 4-s epoch Hanning window, with a 50% overlapping "sliding window" to process deartifacted epochs.

Behavioral Signs and Clinical Diagnosis of Epilepsy

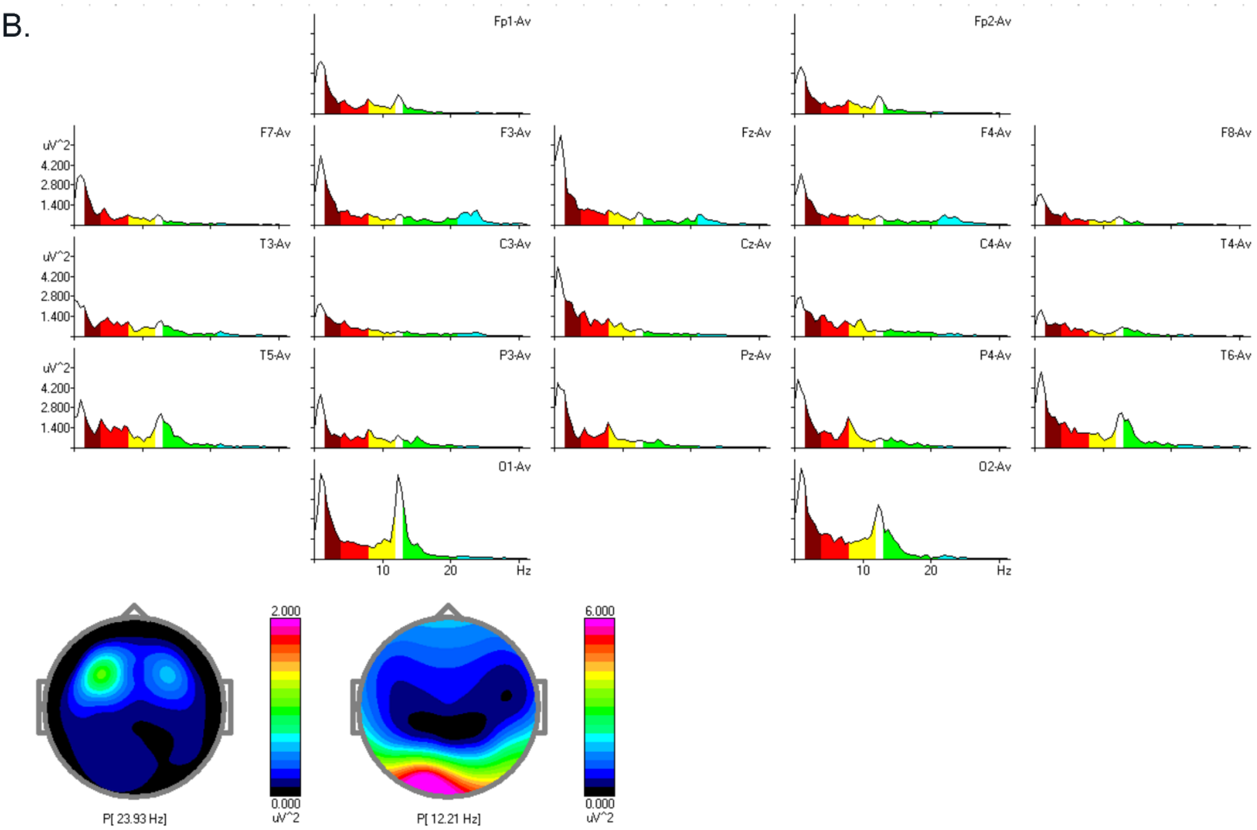
At age 13 (2015), the first outward signs of seizures were recognized, with rapid eye-blinking, difficulty with breathing, and choking sounds. Neurologists diagnosed temporal lobe epilepsy (TLE) and recommended medications should be considered, although no antiseizure medication was prescribed at that time. The patient's seizures became progressively worse, with more frequent and intense episodes over the next 2 months. In one day, she experienced over 120 seizures, including full body and lung stoppages, and was admitted to the emergency room, followed by an episode of status epilepticus. While hospitalized over the next week, she experienced a peak of ~250 visible seizures per day, including tonic rigidity followed by clonic convulsive activity, choking, cessation of breathing, loss of consciousness and eye. Auras of intense abdominal pain were reported during each seizure.

Figure 1. *Early EEG and qEEG Recordings.*



Note. These recordings were collected in 2013 when the patient was 11 years old, 2 years before she was diagnosed with TLE. (A) EEG and qEEG from EC baseline were collected during the evaluation for NFB for ADHD. Note the 20–24 Hz beta spindles seen frontally, with very fast 12–13 Hz alpha frequencies posteriorly. Subtle spikes and slower activity are also noted left temporally (T5).

Figure 1. *Early EEG and qEEG Recordings.*



Note. (B) In the power spectrum graphical display and topographic mapping, these faster alpha and beta spindles can be seen, indicating CNS overarousal. The temporal epileptiform transients are not seen in the qEEG's averaged spectral displays.

Medications and Adjustments

When hospitalized, the patient began antiseizure medications, up to 400 mg of lacosamide (LAC) and 2500 mg of levetiracetam (LEV) daily. These treatments reduced observed seizures from 250/day to about 90/day. One month after her initial hospitalization, clobazam (CBZ) was added and titrated up to 5 mg daily. During this time, she had very forceful, seizure-related automatisms of uncontrolled hand/fist pounding documented with EEG monitoring.

There was significant sedated demeanor, so medications were reduced under medical supervision. LEV was completely tapered over the following 7 weeks, and with its reduction the peri-ictal automatisms completely ceased. Even with LEV discontinued, she continued to have clouding of her sensorium for the next 5 weeks, so LAC was phased out slowly over the following 8 weeks. CBZ was then also tapered over a 10-week period. Although she was still exhibiting clinical seizures, frequency of events decreased, and she exhibited much-improved cognitive functioning and awareness.

Neurofeedback and Adjunctive Treatments

Encouraged by reported cases of success with NFB for control of seizures (Tan et al., 2009), the patient's parents proposed to the hospital's neurologists that medications should be withdrawn due to the side effects and NFB begun for seizures and epilepsy. The hospital neurologists involved in her care were not supportive of NFB. Her parents nonetheless decided to pursue medication taper and EEG/qEEG-guided NFB, along with adjunctive lifestyle changes and mental skills coaching (MSC). Behavioral side effects of the medications were carefully monitored and successfully eliminated during medication taper.

The patient's mother, who had obtained training and supervision in providing NFB for ADHD, sought out a treatment team of neurologists, epileptologists, and other clinicians experienced with NFB in PWE. She arranged for EEGs to be acquired and EEG monitoring, and the patient began MSC. An NFB-experienced neurologist recommended six NFB sessions to decrease slow 1–5 Hz activity primarily over F3/F4, followed by more sessions of F7/F8 and then over Fz, as well as decreasing fast beta activities of 21–30 Hz at all sites except T3/T4. This did not resolve any behavioral symptoms, and the patient was referred to a pediatric epileptologist /neurologist who was trained and board-certified in pediatric epilepsy as well as EEG, qEEG, and NFB.

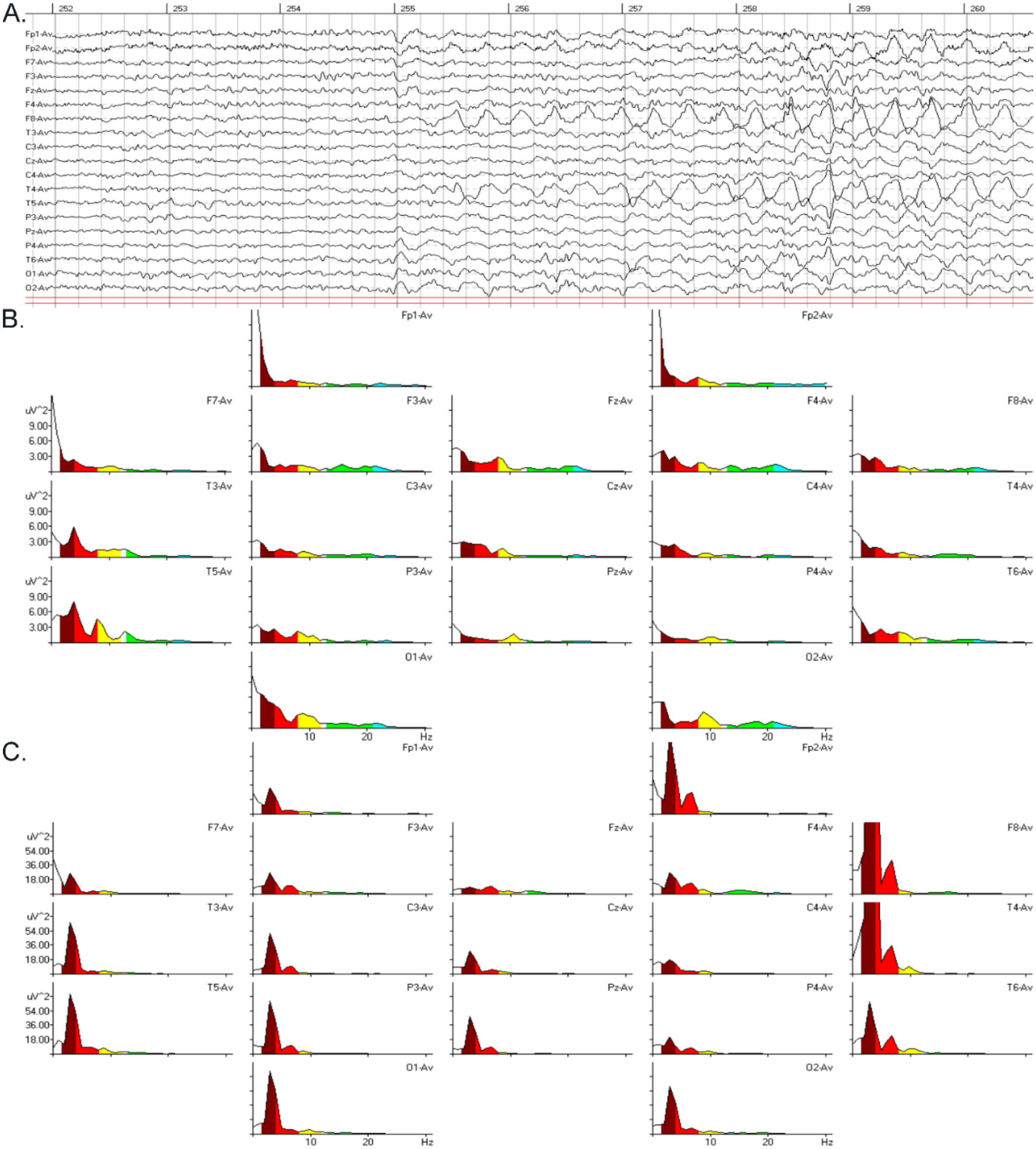
The pediatric neurologist emphasized enhancing SMR (Cz, C4, Fz) and suppressing delta and theta at those sites. Despite over 80 NFB sessions, there was little improvement noted. An NFB trainer recommended fine tuning the sites to between T3/C3 and T4/Cz and more sessions of C3/C4. By early December 2015 (age 14), with little change evident after the NFB approaches noted above, a comprehensive consultation was obtained from a qEEG and NFB expert with decades of EEG experience and training in applied clinical psychophysiology, who recommended use of combined EEG and qEEG to provide a complete assessment to guide training. EEGs continued to show right temporal discharges and more frequent left posterior temporal discharges (Figure 2).

At his direction, the training parameters were altered to enhance SMR at T5-Cz while suppressing both slow (2–7 Hz) and fast (22–30 Hz) activity over 35 sessions. Fifty-one more training sessions followed at T5/T6 and 28 sessions of T5 and O2 training. Following this, neurobehavioral improvements were noted, with fewer akinetic (“drop”) seizures, which generally lasted about 20 seconds, with frequency decreasing to less than 1/day. The patient continued to have drop seizures when participating in sports and would resume play after a seizure. During menstrual periods, seizures were significantly more frequent and severe. Based upon research at the time (Strehl et al., 2005), NFB was changed to 40 sessions of slow cortical potential (SCP) training, but this had little apparent effect. Then, 30 more sessions were conducted using O1 and FCz, followed by 15 sessions of T3-FCz. Following this, “drop” seizures ceased, but clonic/motor seizures (seen as stiffening and clouding, but not total loss, of consciousness) remained. An aura of abdominal pain prior to seizures continued to occur. Updated EEG/qEEG data suggested NFB training back to SMR at T5-CZ. To address the presumed deep-brain sources of the preictal abdominal pain (Morales-Quezada et al., 2019), training was moved to T3-Cz, with some intermittent training at T5-Fz or T6-Cz. For rationale for these training protocols, the aura of abdominal pain was suggestive of a deep temporal lobe source at or near the Sylvian fissure or insular cortex, where epileptiform discharges may elicit visceral effects, including abdominal pain (Balabhadra et al., 2020; Cerminara et al., 2013). This early aura was one of the most persistent and one of the last symptoms to disappear during treatment. Early treatments were limited by seizure frequency interruptions, and training took place 30, 45, or 60 seconds at a time. Over time, they were extended to six 5-min sessions. The patient had NFB training twice daily for several months (when possible).

Concurrent with NFB, adjunctive treatments were attempted (Table 1). A low glycemic index diet appeared to correlate with improvement in seizures. Other dietary supplements were discontinued due to lack of perceived benefit, including cannabidiol, Omega 3 fish oil, and probiotics (L. rhamnosus and B. longum). Toxicology testing for heavy metals indicated that the patient had elevated copper and low zinc levels. Additional testing including organic acids and infectious/fungi testing, mitochondrial function assessment, levels of oxalates, and other key elemental substances, but these tests did not yield diagnostic clarity. After a monitored chelation and vitamin supplement combination, vitamin and essential metal absorption improved, but had only small impact, if any, on seizure reduction (although there may have been a positive impact on her sleep quality). Homeopathic interventions for one year had no evident impact on seizures.

Because menstruation was associated with increased seizures, transdermal progesterone was attempted, which may have correlated with decreased seizure frequency but not intensity. Electrodermal response BFB training was attempted but not perceived as effective for seizure reduction. Other BFB modalities, including heart rate variability (HRV), abdominal breathing, and muscle relaxation had a positive impact on managing anxiety, reducing triggers for seizures, and reducing or shortening seizures.

Figure 2. *EEG/qEEG During Symptomatic TLE, Age 14*.



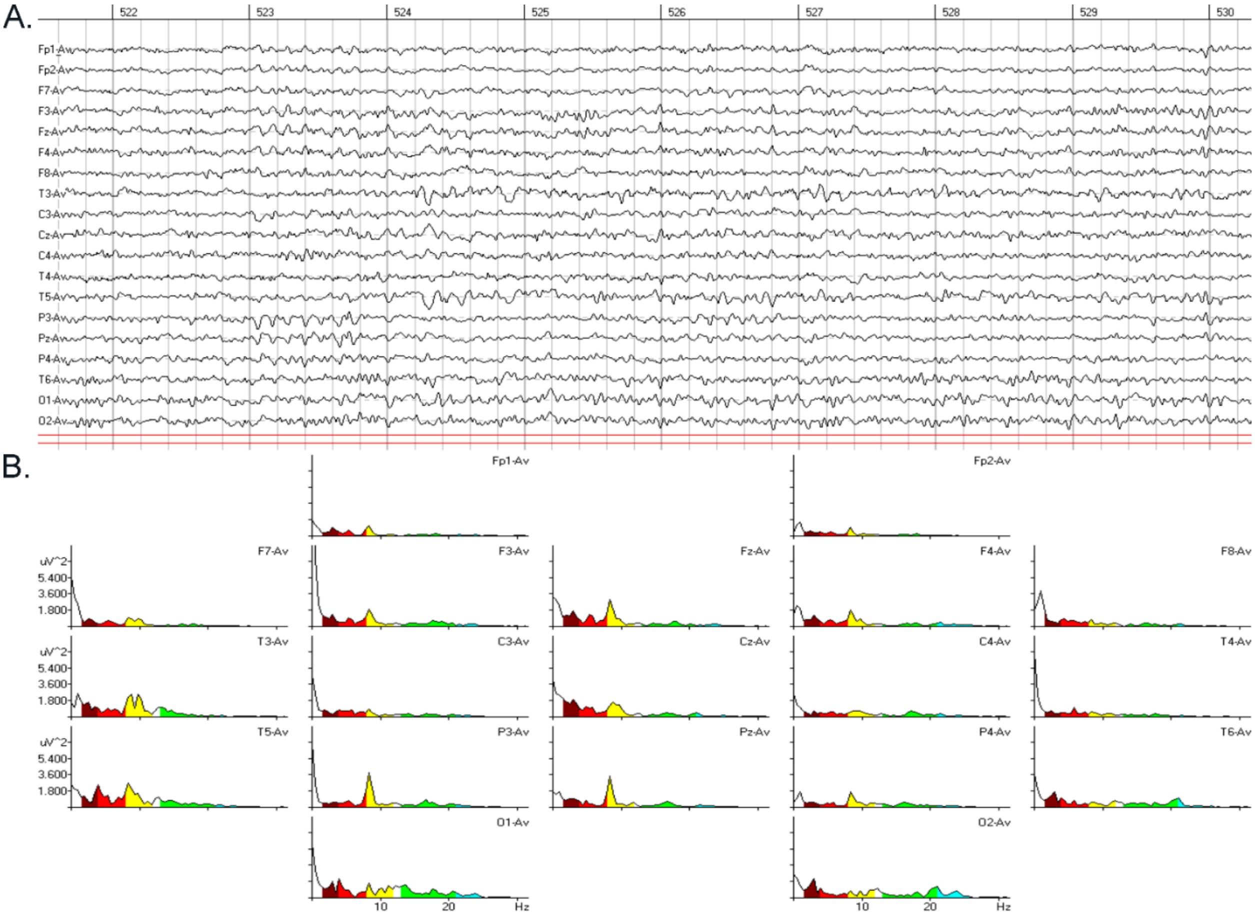
Note. (A) EEG waveforms include a paroxysmal right frontal-temporal discharge. The baseline period showed altered left temporal EEG spectral power, which seemed to trigger the more prominent right hemispheric event. (B) Represents the EEG spectra for the baseline period. (C) Represents the EEG spectra during the right frontal-temporal paroxysmal discharge.

Her overall treatment plan also included mental skills coaching. The skills included progressive relaxation, imagery, HRV, breathing, attentional focusing, and cognitive restructuring to enhance awareness of the feelings of stress or any sensations prior to onset of a seizure, followed by immediate practice of self-regulation skills to quickly alter the mind and body responses. Her ability to produce these changes was documented with BFB training instruments (HRV and EEG). This provided a link between her behavior and body responses and enhanced her self-control and self-confidence. Consistent coaching improved compliance and seeing changes in her daily life resulted in additional confidence and motivation.

Full Abatement of Epilepsy Symptoms

By age 16 (2017/2018), following two years of daily NFB SMR training sessions, the patient did not experience any witnessed akinetic/“drop” seizures, aura/epigastric pain or motor tremulousness, or tonic “stiffening” seizures. She reached two professional semifinals in her sport, illustrating her ability to perform at elite levels without seizures, despite suboptimal sleep, intense sports-cardiovascular challenges, and the mental stressors of university recruitment interviews. An EEG recording at this time, taken after a 12-hour international flight, demonstrated residual elevated spectral power and slowing over the left temporal regions (Figure 3). However, no spikes or abnormal paroxysmal discharges were noted.

Figure 3. *EEG/qEEG After Epilepsy Symptoms Abated, Age 16.*



Note. (A) EEG waveform and (B) Power spectrum graphical display and topographic mapping. Note subtle residual low voltage slowing left temporally. EEG also demonstrated lack of epileptiform discharges and no abnormal paroxysms, with diminution of frontal beta spindling noted in earlier recordings.

At age 17 (2019), the patient experienced the final two epileptic episodes through the writing of this report. In the first, she experienced a 5- to 8-second period of dizziness that occurred after period of illness and physical exhaustion. In the second, she experienced an 8- to 10-second period of hand stiffening on a 17-hour international flight with a 5-hour layover and no sleep. In both cases, she did not lose consciousness.

Current Status

As of the time of writing, the patient remains seizure- and medication-free and carries a full academic load and daily sports workouts at university. Her NFB training is ongoing, and she reports improved attention/focus in her sport and feels at optimal health when she maintains two NFB training sessions per week. While she has shown some continued epileptiform activity in her EEG up to 20 seconds, she has had no outward signs, with the exception of occasional light headedness. She is aware that, in order to remain seizure- and medication-free, intermittent follow up NFB sessions may be required long-term, perhaps for the rest of her life.

Discussion

This successful approach to controlling severe epilepsy was a multi-disciplinary effort, working with a highly motivated patient and parents or caregivers. Although a case report of only one patient, the authors feel the foundation of her successful achievement of controlling, and ultimately eliminating, epilepsy was NFB training over a long period. The monitoring and tracking of NFB training sessions and their outcomes, with ongoing monitoring of the underlying EEG and qEEG changes, allowed the neurologist and EEG specialists to give personalized recommendations and guidance for fine-tuning of training interventions to maximize the beneficial outcomes. When there was little improvement following NFB sessions targeting the prominent right temporal lobe IEDs, NFB training was altered to target the less-prominent left temporal lobe IEDs, and this was followed by behavioral improvements and, eventually, complete seizure cessation.

Concurrent with NFB, adjunctive modalities and coaching helped to improve motivation and health and facilitated the transfer of self-control from training sessions to real-life situations. Importantly, the patient was able to achieve a sense of competence, motivation, and self-confidence from participating in her own treatment. Her physicians and other healthcare providers were knowledgeable and experienced in EEG, qEEG, epilepsy, and NFB training. All these factors were needed to develop, execute, and sustain a relevant treatment plan that was administered at home. This, supplemented with parental or caregiver support and ongoing coaching, allowed for the patient's success in school, sports, and independence in daily life.

While many PWE cannot attend specialized clinics, this case study documents that, with appropriate EEG equipment, training, and guidance and internet access, it is possible to receive extensive help for epilepsy remediation in a cost-effective manner. Technology allows such training to be largely home-based, professionally supervised, and implemented by a trained caregiver, with online professional supervision as required. The development of more sophisticated mobile apps and equipment should aid in the monitoring and training of an individual's brain/body physiologic states during daily life (Dozières-Puyravel et al., 2020).

Conclusions

This case report documents the use of NFB, along with adjunctive interventions, for a young female who progressed from severe, medically intractable temporal lobe epilepsy to a current performance as a university student and athlete in a NCAA Division I sport. Those of us involved in her clinical care and management believe that her story can bring hope and inspiration to others experiencing intractable epilepsy and that it will encourage research in alternative therapies.

Author Acknowledgement

The authors thank Rachel Tittle for editing assistance.

Author Declaration

There are no financial interests or conflicts to report.

References

Asadi-Pooya, A. A., Nei, M., Sharan, A., & Sperling, M. R. (2016). Patient historical risk factors associated with seizure outcome after surgery for drug-resistant nonlesional temporal lobe epilepsy. *World Neurosurgery*, *91*, 205–209. https://doi.org/10.1016/j.wneu.2016.04.022

Balabhadra, A., Malipeddi, A., Ali, N., & Balabhadra, R. (2020). Abdominal epilepsy: A rare cause of unexplained abdominal pain. *Cureus, 12*(8), Article e10120. https://doi.org/10.7759/cureus.10120

Benevides, M. L., Costa Nunes, J., Guarnieri, R., Pauli, C., Wolf, P., Lunardi, M., Kondageski, C., Neves Linhares, M., Lin, K., & Walz, R. (2021). Quality of life long after temporal lobe epilepsy surgery. *Acta Neurologica Scandinavica*, *143*(6), 629–636. https://doi.org/10.1111/ane.13406

Centers for Disease Control and Prevention. (2020). *Epilepsy data and statistics: Centers for Disease Control and Prevention*. https://www.cdc.gov/epilepsy/data/index.html

Cerminara, C., El Malhany N., Roberto, D., & Curatolo, P. (2013). Focal epilepsy with ictal abdominal pain: A case report. *Italian Journal of Pediatrics, 39*, Article 76. https://doi.org/10.1186/1824-7288-39-76

Dozières-Puyravel, B., Danse, M., Goujon, E., Höhn, S., & Auvin, S. (2020). Views of adolescents and their parents on mobile apps for epilepsy self-management. *Epilepsy & Behavior, 106*, Article 107039. https://doi.org/10.1016/j.yebeh.2020.107039

Engel, J., Jr. (2013). Why is there still doubt to cut it out? *Epilepsy Currents, 13*(5), 198–204. https://doi.org/10.5698/1535-7597-13.5.198

Engel, J., Jr., Pitkänen, A., Loeb, J. A., Dudek, F. E., Bertram, E. H., 3rd, Cole, A. J., Moshé, S. L., Wiebe, S., Jensen, F. E., Mody, I., Nehlig, A., & Vezzani, A. (2013). Epilepsy biomarkers. *Epilepsia,* 54(Suppl. 4), 61–69. https://doi.org/10.1111/epi.12299

Fleming, M., Fitton, C. A., Steiner, M. F. C., McLay, J. S., Clark, D., King, A., Mackay, D. F., & Pell, J. P. (2019). Educational and health outcomes of children and adolescents receiving antiepileptic medication: Scotland-wide record linkage study of 766 244 schoolchildren. *BMC Public Health*, *9*(1), Article 595. https://doi.org/10.1186/s12889-019-6888-9.

Frey, L. (2016). Impact of sensorimotor rhythm neurofeedback on quality of life in patients with medically-refractory seizures. *NeuroRegulation*, *3*(1), 3–6. https://doi.org/10.15540/nr.3.1.3

Haut, S. R., Gursky, J. M., & Privitera, M. (2019). Behavioral interventions in epilepsy. *Current Opinion in Neurology*, *32*(2), 227–236. https://doi.org/10.1097/WCO.0000000000000661

Kohlhase, K., Zöllner, J. P., Tandon, N., Strzelczyk, A., & Rosenow F. (2021). Comparison of minimally invasive and traditional surgical approaches for refractory mesial temporal lobe epilepsy: A systematic review and meta-analysis of outcomes. *Epilepsia*, *62*(4), 831–845. https://doi.org/10.1111/epi.16846

Mesraoua, B., Kissani, N., Deleu, D., Elsheikh, L., Ali, M., Melikyan, G., Hail, H. A., Wiebe, S., & Asadi-Pooya, A. A. (2021). Complementary and alternative medicine (CAM) for epilepsy treatment in the Middle East and North Africa (MENA) region. *Epilepsy Research, 170*, 106538. https://doi.org/10.1016/j.eplepsyres.2020.106538

Mohan, M., Keller, S., Nicolson, A., Biswas, S., Smith, D., Osman Farah, J., Eldridge, P., & Wieshmann, U. (2018). The long-term outcomes of epilepsy surgery. *PLoS ONE*, *13*(5), e0196274. https://doi.org/10.1371/journal.pone.0196274

Morales-Quezada, L., Martinez, D., El-Hagrassy, M. M., Kaptchuk, T. J., Sterman, M. B., & Yeh, G. Y. (2019). Neurofeedback impacts cognition and quality of life in pediatric focal epilepsy: An exploratory randomized double-blinded sham-controlled trial. *Epilepsy & Behavior, 101*(Pt. A), 106570. https://doi.org/10.1016/j.yebeh.2019.106570

Nagai, Y., Jones, C. I., & Sen, A. (2019). Galvanic skin response (GSR)/electrodermal/skin conductance biofeedback on epilepsy: A systematic review and meta-analysis. *Frontiers in Neurology, 10*, 377. https://doi.org/10.3389/fneur.2019.00377

Nigro, S. E. (2019). The Efficacy of Neurofeedback for Pediatric Epilepsy. *Applied Psychophysioly and Biofeedback, 44*(4), 285–290. https://doi.org/10.1007/s10484-019-09446-y

Selaa, R., & Shaked–Toledanob, M. (2014, June). Neurofeedback treatments enable the EEG-normalization and total seizure control of epilepsy–A case study. *Applied Psychophysiology and Biofeedback, 39*(2), 142–143.

Singh, A., & Trevick, S. (2016). The epidemiology of global epilepsy. *Neurologic Clinics*, *34*(4), 837–847. https://doi.org/10.1016/j.ncl.2016.06.015

Sterman, M. B., Macdonald, L. R., & Stone, R. K. (1974). Biofeedback training of the sensorimotor electroencephalogram rhythm in man: Effects on epilepsy. *Epilepsia*, *15*(3), 395–416. https://doi.org/10.1111/j.1528-1157.1974.tb04016.x

Sterman, M. B., Wyrwicka, W., & Roth, S. (1969). Electrophysiological correlates and neural substrates of alimentary behavior in the cat. *Annals of the New York Academy of Sciences*, *157*(2), 723–739. https://doi.org/10.1111/j.1749-6632.1969.tb12916.x

Strehl, U., Kotchoubey, B., Trevorrow, T., & Birbaumer, N. (2005). Predictors of seizure reduction after self-regulation of slow cortical potentials as a treatment of drug-resistant epilepsy. *Epilepsy & Behavior*, *6*(2), 156–166. https://doi.org/10.1016/j.yebeh.2004.11.004

Tan, G., Thornby, J., Hammond, D. C., Strehl, U., Canady, B., Arnemann, K., & Kaiser, D. A. (2009). Meta-analysis of EEG biofeedback in treating epilepsy. *Clinical EEG and Neuroscience*, *40*(3), 173–179. https://doi.org/10.1177/155005940904000310

Wyrwicka, W., & Sterman, M. B. (1968). Instrumental conditioning of sensorimotor cortex EEG spindles in the waking cat. *Physiology & Behavior*, *3*(5), 703–707. https://doi.org/10.1016/0031-9384(68)90139-x

Yardi, R., Morita-Sherman, M. E., Fitzgerald, Z., Punia, V., Bena, J., Morrison, S., Najm, I., Bingaman, W., & Jehi, L. (2020). Long-term outcomes of reoperations in epilepsy surgery. *Epilepsia*, *61*(3), 465–478. https://doi.org/10.1111/epi.16452

Received: January 21, 2023
Accepted: February 12, 2023
Published: March 30, 2023

NeuroRegulation

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In Neurofeedback Training, Harder is Not Necessarily Better: The Power of Positive Feedback in Facilitating Brainwave Self-Regulation

Revital Yonah[1*]

[1]Private practice, Jerusalem, Israel

Abstract

Neurofeedback is gaining recognition as an efficient, effective treatment for a variety of different psychological and neuropsychiatric disorders. Its value has been shown in robust clinical studies. However, a certain percentage of clients do not respond to this treatment modality. We suggest performing easier sessions so that clients receive an increased rate of positive feedback. This may encourage positive response to neurofeedback. Research has shown that implicit learning, the type of learning involved in neurofeedback, is better achieved with high levels of positive feedback. In addition, psychological factors related to attention, motivation, cooperation, and positive affect may also be contributing to this facilitatory effect. The relevant theoretical background and supporting evidence are provided.

Keywords: neurofeedback; EEG-Biofeedback; implicit learning; basal-ganglia; threshold; thresholding; reward; positive feedback

Citation: Yonah, R. (2023). In neurofeedback training, harder is not necessarily better: The power of positive feedback in facilitating brainwave self-regulation. *NeuroRegulation, 10*(1), 31–41. https://doi.org/10.15540/nr.10.1.31

***Address correspondence to**: Revital Yonah, POB 18144, Jerusalem, 9118101, Israel. Email: RevitalYonah@gmail.com

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Edited by:
Rex L. Cannon, PhD, Currents, Knoxville, Tennessee, USA

Reviewed by:
Rex L. Cannon, PhD, Currents, Knoxville, Tennessee, USA
Tanya Morosoli, MSc: 1) Clínica de Neuropsicología Diagnóstica y Terapéutica, Mexico City, Mexico; 2) ECPE, Harvard T. H. Chan School of Public Health, Boston, Massachussetts, USA

Background

EEG neurofeedback (also known as EEG-biofeedback or brainwave self-regulation) has been used to treat a variety of neuropsychiatric disorders (Niv, 2013). Success rates vary and have often been reported to be high, but invariably some trainees are slow to respond or do not show any response to this treatment modality (Rogala et al., 2016; Zuberer et al., 2015). Indeed, nonresponse rates were reported to vary between 16% and up to 57% in some cases (Alkoby et al., 2018). The reasons for this nonresponse are not yet well understood (Oblak et al., 2019). Part of it may be due to the use of standardized protocols that do not target the individual dysregulation in these studies. Other methodological factors may be responsible for these rates of nonresponse, such as number and length of sessions, intersession intervals, type of threshold used (automatically vs. manually adjusted), trainer-participant interface (Gruzelier, 2014), as well as schedules of reinforcement (Sherlin et al., 2011) and types, and modalities of feedback (Strehl, 2014). The ability of the clinician to instill a motivation to succeed in the client has also been cited as crucial (Sterman & Egner, 2006). As neurofeedback is gaining increasing acceptance and recognition by the mainstream medical establishment, it is important to elucidate the factors and parameters that can facilitate learning and enhance treatment results.

Neurofeedback is based on the principles of operant conditioning of brainwave activity (Birbaumer et al., 2013; Collura, 2014; Sitaram et al., 2017). Clients are fed back information about their electrophysiological activity and are taught to modify this activity by means of positive and negative feedback received through the sensory modalities (i.e., visual, auditory, or tactile feedback). When a client’s brainwave activity comes closer to the desired target activity (usually, age-group norms), alleviation of neuropsychiatric symptoms often ensues. The very act of learning to regulate one's brainwave activity may rid clients of mental barriers that have plagued them for years, and, albeit rarely, this may even happen in the course of just two sessions (van der Kolk, 2014).

An average neurofeedback training series may take anywhere between 40 and 80 sessions (Barabasz & Barabasz, 1999), with some individuals requiring more sessions to obtain satisfactory results. In some cases, part of the reason the process takes a long time is that it is not always obvious which training protocols would be most effective for a given client, even when the protocol selection is based on a neurometric assessment (i.e., qEEG test), intake, and a thorough anamnesis. Practitioners normally start with one or two training protocols for a few sessions based on these assessments. They monitor the client's response, and either continue with the initial protocols, if response is satisfactory, or change to other protocols, if there is no response or if the response is less than optimal (Fisher, 2014; Johnson & Bodenhamer-Davis, 2009). When clients are fast responders, fine-tuning of the protocol selection process can be accomplished more rapidly. However, when clients take a long time to respond—with some, it may require 20 or more sessions before we can notice and start appreciating the effects of the training (Pallanti, as cited in Gastaldi, 2023)—then the practitioner's job of fine-tuning the protocol selection process is more difficult and requires more time. Practitioners may also wonder in such cases whether the client is a nonresponder to neurofeedback, or whether the problem is with the protocol they selected. Speeding up clients' response in such cases may aid the process.

Thresholding plays a crucial role in this respect. It has already been suggested that setting thresholds optimally may save up to 35% of the overall training time, which may be translated into significant reductions in training costs for clients (Davelaar, 2017). Here we suggest that performing training sessions with thresholds yielding relatively high success rates (and therefore a high incidence of positive feedback) may accelerate clients' neurofeedback learning and response. In other words, when performing neurofeedback sessions, clients should receive more positive than negative feedback to achieve success in training. To explain this, we should first refer to some basic theoretical principles of learning, and more specifically, of reinforcement learning or operant conditioning.

Basic Behaviorist Principles of Learning

Thorndike (1911, as cited in Sherlin et al., 2011) first formulated the Law of Effect, which states that reward raises the likelihood that the target behavior will reoccur while punishment decreases that likelihood. Skinner further developed the idea of operant conditioning based on this law (Skinner, 1945).

The neural correlates of reinforcement learning, or operant conditioning, are varied. Learning from reward seems to involve partially different networks and structures than learning to avoid punishment (Elliott et al., 2010). Dopaminergic neurons in the striatum and frontal cortices (Bromberg-Martin et al., 2010) as well as in the substantia nigra and ventral tegmental area (Sulzer et al., 2013) seem to play a key role in reward learning. Learning to avoid punishment involves the insula and lateral orbitofrontal cortex, among other regions (Elliott et al., 2010; O'Doherty et al., 2001; Wächter et al., 2009).

There are positive and negative rewards, and positive and negative punishments. Positive rewards are when we provide something desirable to an organism to increase the targeted behavior. Negative rewards are when we remove something undesirable from the organism with the intention of increasing the targeted behavior. Positive punishments are when we give or do something unpleasant as punishment to decrease the likelihood of a certain behavior. Negative punishments are when we remove something desirable from the organism with the intention of decreasing the likelihood that a certain behavior will reoccur (Sherlin et al., 2011).

For simplicity's sake, in this paper we employ the broader terms of positive feedback and negative feedback to refer to rewards and punishments, respectively, without resorting to the more refined categories based on the types of reinforcers employed. That is, here positive feedback refers to either positive or negative reward, and negative feedback refers to either positive or negative punishment.

There are different neurofeedback technologies, with various methods of providing feedback to clients, employing positive feedback, negative feedback or a combination of both. Since clients often know that the absence of positive feedback is really negative (i.e., it means that their brainwaves are not reaching the target activity), the absence of such positive feedback may be experienced by them as negative feedback, an indication of failure. In such cases, this would serve as an internal, or secondary, punishment. The opposite is true as well: the absence of negative feedback may be perceived by clients as rewarding, an indication of success, even if no reward is actually obtained. In this case, this would serve as an internal, or secondary, reward.

Thresholds and Success Rates in Neurofeedback Training

In neurofeedback, different aspects of brainwave activity can be trained up or down. Amplitude, coherence, percent time, and symmetry indices are just few examples of neural activity that can be trained and modified through neural feedback. Here we refer to amplitude training, but the same principles may hold true for other aspects of neural activity as well.

Brainwaves are referred to in terms of their frequency and amplitude. Frequency is the number of cycles per second, measured in units of hertz (e.g., theta: 4–8 Hz, alpha: 8–12 Hz, etc.). Brainwave amplitude refers to magnitude, measured in units of microvolts. Amplitude in any given brainwave frequency is determined by the degree of synchronization of neurons at that specific frequency under a certain electrode site (Daffertshofer & van Wijk, 2011). When we attempt to enhance a frequency and train its amplitude up, for example, we set a certain value as the “threshold”: every time the brain produces this frequency at amplitudes that are at or higher than the threshold, the client receives positive feedback, and every time the brain produces amplitudes that are lower than this threshold, the brain receives negative feedback. The opposite is true for frequencies we attempt to suppress. In such inhibit frequencies, the client receives positive feedback for amplitudes at or below the threshold. The threshold determines the difficulty level of the training. If placed high in reward protocols, or low in inhibit protocols, it may yield relatively low success rates, which translates to a lower incidence of positive feedback provided to clients.

There are different ways of setting training thresholds (Vernon et al., 2009). A threshold can be a fixed value. This fixed value can be preset, based on previous experience, previous results of the client, or professional literature, or it can be equal to the average amplitudes at rest or a proportion of this average; alternatively, it can be a changing value designed to yield a fixed success rate (i.e., automatic threshold). A common perception among clients and clinicians, especially those new to neurofeedback, is the harder the training, the more efficient it is. The tacit assumption here is the brain is like a muscle, and the more “weights” we load onto it, the better the results. Sessions conducted under this assumption may therefore yield success rates of around 30–40% or lower. That is, clients would meet the target brainwave activity or go beyond it in the desired direction only around 30% or 40% of the time or less, and the rate of compensation would be accordingly low.

In this paper we would like to suggest setting the threshold so it yields higher success rates. This will yield a higher incidence of positive feedback during a session, which is preferable, as it may yield more robust clinical results, faster.

The Power of Positive Feedback

As mentioned above, one common way of setting a threshold is to place it at exactly the average amplitude at baseline. Thus, the client’s brainwave activity at rest would go above this value roughly 50% of the time. Here we suggest setting a threshold that is easier to pass (i.e., a lower threshold in reward frequencies or a higher threshold in inhibit frequencies). This would be one yielding significantly more than 50% success rates. We believe this is preferable, as it may contribute towards a more effective and efficient training. The reasons for this are physiological and psychological in nature, as detailed below. To explain this, we would use a simple protocol as an example, sensorimotor rhythm (SMR) up at CZ, but the same rationale may hold true for other, more complex protocols as well.

In an SMR up protocol, if we use the average amplitudes at baseline as the threshold, we provide negative feedback to clients every time the amplitude is below, or even just below, what it was at baseline. However, when we do this, we basically provide the brain with negative feedback for producing SMR activity that is very close to the desired level, even if it does not meet it. This may make it harder for the brain to learn the desired pattern of activity and in some cases may even teach the brain to inhibit it. It was noted in a different context that if positive feedback is withheld for an activity falling just short of the threshold, this may discourage the increase of the desired brainwave activity (Hardt & Kamiya, 1976).

According to the principles of shaping, we reward the brain not only when it meets the criterion (i.e., threshold), but also when it comes close to it. This way, we indicate to the brain the direction it has to shift its activity in order to receive positive feedback. Activity very significantly distant from the target should not be rewarded, so the brain does not unlearn the desired pattern of activity (Davelaar, 2018). One of the problems with negative feedback is it carries little specificity, which makes it harder for clients to know how to improve (Reinschluessel & Mandryk, 2016). Positive feedback, on the other hand, contains such information. While this practice is accepted by some for the first stages of training, here we suggest that not only at the beginning of neurofeedback training but throughout the training series, clients should preferably receive more positive than negative feedback when training.

Ideally, there should be a gradation of feedback, so activity that is very far from the threshold receives more negative feedback than activity somewhat closer to the threshold. In many neurofeedback systems such gradation exists. Ideally, as the brain learns the desired pattern of activity and produces higher and higher amplitudes on average, the new thresholds should be updated accordingly, but still allow for higher percentage of positive, compared to negative, feedback.

How do we set the thresholds? The optimal threshold setting is unknown (Davelaar, 2017) and this question remains to be determined in controlled experiments. Experience shows setting the threshold to around 60–80% of the average amplitudes at baseline in reward frequencies, and between 120% and 140% of the average amplitudes at baseline in inhibit frequencies, may be safe and effective in encouraging the brain to change its electrophysiological activity in the desired direction (Egner et al., 2004; Ros et al., 2009; Vernon et al., 2009). Success rates at such sessions may be 60–80%, which is more informative to the brain than the 50% or so normally achieved when the threshold is set to be equal to the average amplitudes at baseline (Nam & Choi, 2020). More research must be conducted to determine the optimal level of thresholds (Vernon et al., 2009). This observation finds support also when considering the nature of the learning process in neurofeedback, as we explain next.

Implicit Learning is Better Achieved With Positive Feedback

Neurofeedback is a form of implicit, procedural learning, a type of skill learning that can be acquired even without conscious awareness (Birbaumer et al., 2013; Ramot et al., 2016; Siniatchkin et al., 2000; Sitaram et al., 2017). The neural network engaged in neurofeedback is wide and involves both cortical and subcortical structures. Among these, the basal ganglia seem to play a major role as a part of the corticostriatal loop (Birbaumer et al., 2013; Emmert et al., 2016; Koralek et al., 2012; Lam et al., 2020; Skottnik et al., 2019), with dopaminergic and glutamatergic synapses (Sitaram et al., 2017). These nuclei are involved in other types of implicit learning as well (Heindel et al., 1989; Poldrack et al., 2001). Their involvement in neurofeedback was demonstrated in both human functional magnetic resonance imaging (fMRI) studies (Emmert et al., 2016; Sitaram et al., 2017) and animal studies (Koralek et al., 2012; Schafer & Moore, 2011).

Research has shown implicit learning is better achieved with correct feedback than with error feedback—that is, with positive, rather than negative feedback (Loonis et al., 2017). The reason for better implicit learning with less error feedback (or "errorless learning") may be that errors cause people to use explicit cognitive processes in trying to form better strategies for success. This may overload the system and, paradoxically, impair implicit learning (Chafee & Crowe, 2017; Maxwell et al., 2001; Poolton et al., 2005). Loonis and colleagues found category-saccade learning, a type of implicit learning, improved more after correct choices and positive feedback than after incorrect choices and negative feedback. They found negative feedback in this type of task appears to interfere with the learning process: performance worsened after an incorrect trial and subsequent reaction times increased. In equivalent explicit learning tasks, performance was almost the same after positive and negative feedback (Loonis et al., 2017). Interestingly, Sasaki et al. (2010) suggested successful performance of a visual perceptual learning task, a form of procedural learning, yields a sense of achievement. This serves as an internal reward, as opposed to an externally provided physical reward. This internal reward, in turn, reinforces the implicit learning of task-irrelevant features, which are presented simultaneously as the task-relevant features (i.e., implicitly). Similarly, Shibata and coauthors found fake, larger-gradient positive feedback enhanced performance on visual perceptual learning more than genuine feedback.

They suggested the same reason: positive feedback was perceived by subjects as a form of praise, and this has implicitly facilitated learning (Shibata et al., 2009). Task-irrelevant learning may occur only if the irrelevant stimuli or features are presented subliminally, so the conscious attention system does not detect them (Tsushima et al., 2008). The evaluation of one's performance by the feedback provided seems to be performed by the frontal cortex, and this evaluation directs the basal ganglia and part of the forebrain to control the rate of implicit, perceptual learning (Shibata et al., 2009).

This phenomenon is also demonstrated with amnesiacs, in whom the hippocampus is damaged. The ability of such patients to acquire explicit learning is compromised, whereas their ability to acquire implicit learning is relatively intact. Amnesia patients perform skill learning, a type of implicit learning, better when correct (positive) rather than error (negative) feedback is emphasized (Evans et al., 2000). In this case, however, an alternative explanation may be that amnesiacs have difficulty remembering and employing explicit cognitive strategies. They therefore perform better with "errorless learning" than with "errorful learning." Also, off-medication Parkinson's disease patients, who have basal-ganglia damage, learned procedural tasks better when punishment was employed as feedback rather than reward (Argyelan et al., 2018). Once back on medication, dopamine medications changed this pattern, so the patients acquired procedural learning better from reward than from punishment. This may stress the importance of the basal ganglia, a key component in neurofeedback learning as well, in acquiring procedural learning from positive feedback or reward.

Neurofeedback is considered by most an implicit form of learning (Lam et al., 2020; Ramot et al., 2016; Siniatchkin et al., 2000). Since implicit learning is better achieved with positive feedback, this may yield further support to the observation that neurofeedback sessions should be conducted with a relatively high incidence of positive feedback. Sessions conducted this way may be more efficient and effective than sessions conducted with equal or higher incidence of negative feedback.

Maxwell and colleagues suggested errorful learning relies more on explicit processes and involves hypothesis testing about different strategies (Maxwell et al., 2001). Kober and coauthors proposed testing of strategies for success in neurofeedback imposes a cognitive load on trainees, which may harm their performance. They advise that neurofeedback training is better performed without employing such conscious, explicit strategies (Kober et al., 2013). Lam and colleagues found error monitoring networks are of lesser relevance to neurofeedback learning (Lam et al., 2020), which again stresses the fact that neurofeedback may be based more on learning from positive feedback than error feedback. Naturally, a certain percentage of negative feedback is necessary, but more positive than negative feedback is preferable.

Some Additional Considerations in Favor of Employing High Rates of Positive Feedback

To be effective, positive feedback should preferably be provided more often than negative feedback. This would be the case when we set the threshold lower than the average baseline amplitudes for reward frequencies, and higher than average baseline amplitudes for inhibit frequencies. In addition to the physiological and learning-related aspects discussed above, there are also psychological considerations in favor of employing more positive than negative feedback in neurofeedback training sessions.

When the session is too difficult, with relatively low levels of positive feedback, clients tend to try to artificially control the feedback. They do so unconsciously by shifting in their chairs, touching the sensors, moving their arms, legs, or facial muscles, or otherwise trying to control the feedback with their muscles rather than with their brainwave activity. This interferes with the session and training process and decreases the chances learning occurs.

It was found that negative feedback may demotivate participants (Reinschluessel & Mandryk, 2016) and make them avoid participating in a task, even when the task is an otherwise enjoyable game (Lin et al., 2006). When clients are children, they may refuse to continue a neurofeedback session, without being able to verbalize the reason for their refusal. This may affect their motivation to complete the remainder of the training series. Some adult clients, especially people who are anxious or depressed, tend to judge themselves harshly for their performance. If such clients believe they are not receiving enough positive feedback, they tend to interpret it as if they are failing to perform the training satisfactorily. This may cause them to be stressed, tense, and anxious and, as a result, they may try to control the feedback by exerting excessive mental effort. As mentioned earlier, such an effort may be counterproductive. Kober et al. (2013) have shown exerting mental effort and trying to consciously control the feedback causes cognitive overload that may hamper learning. This could also cause fatigue relatively early in the session, which is counterproductive for successful training (Shourie et al., 2018). The optimal way of training appears to be by releasing conscious control, keeping an open focus, and letting the brain naturally process the feedback and respond to it (Fehmi & Robbins, 2008). To ensure this, clients must be relaxed, and this state cannot be achieved when clients receive a high rate of negative feedback.

Clients may be more comfortable and motivated to cooperate when thresholds are easier to pass. A high incidence of positive feedback boosts their confidence, and this may have a beneficial effect on their motivation, cooperation, and consequently, on their overall success in the training (Van Doren et al., 2017). Positive feedback produces signals of internal reward, and this in turn may enhance implicit learning (Sasaki et al., 2010). Even when the positive feedback is false, it may still boost learning, for the same psychological reasons (Shibata et al., 2009). In support, motivational factors were positively correlated with Brain-Computer Interface (BCI) performance (Barbero & Grosse-Wentrup, 2010; Nijboer et al., 2010). Motivation and mood were found to be at least moderate predictors of success in neurofeedback and BCI training (Cohen Kadosh & Staunton, 2019).

Attention is another factor crucial for neurofeedback learning. Setting the threshold too high in reward protocols, or too low in inhibit protocols, so the difficulty level is high and the incidence rate of positive feedback is low, may harm the client's ability to be attentive for the duration of the session and interfere with the learning process (Cohen Kadosh & Staunton, 2019).

In summary, if the threshold is set so the training difficulty level is high, then too little feedback information is provided for the brain to learn from. This may frustrate clients, demotivate them, hamper their mood, and may be too taxing for them in terms of their attention resources. Clients may try to control the feedback with their muscle activity and even refuse to continue training, if too little positive feedback is provided. This is especially true for the first few sessions a naïve neurofeedback client performs but is also true for more experienced trainees as well. Working with thresholds yielding higher success rates and higher incidences of positive feedback may be preferable. This is particularly the case with young children or anxious adults. This may allow for better learning and better clinical results. However, if the threshold is set so the session is too easy, this may be counterproductive. Both too little and too much positive feedback may inhibit clients' ability to learn to self-regulate (Vernon et al., 2009).

Supporting Research and Evidence

Support for the observation that thresholds yielding a higher incidence of positive feedback are preferable comes from clinicians and researchers, who have employed such thresholds. For example, Thompson and Thompson (1998) stated that for reward frequencies, the threshold is set 0.2 to 0.6 microvolts lower than the client's average, whereas for inhibit frequencies, the threshold is set 1 to 2 microvolts higher than the client's average. Others have placed the threshold at 80% of the baseline average of the reward frequency, and at 120% and even 160% of the baseline average of the inhibit frequency (Egner et al., 2004; Ros et al., 2009). Ros et al. (2017) used thresholds that yielded 60% positive feedback and 40% negative feedback. Lubar suggested when clients get stuck on a plateau in their learning curve and show no progress in neurofeedback training, to set the threshold lower, so that they receive more positive feedback (Ayers et al., 2000). Van Doren and coauthors showed that when ensuring clients receive at least 50% positive feedback during neurofeedback, their performance improves compared to thresholds yielding lower reward incidence in an alpha-theta protocol (Van Doren et al., 2017). Others reported using at least 70% positive feedback (White & Richards, 2009). In addition, Davelaar (2017) found in a computational analysis of a neurofeedback protocol that lower (that is, "easier") thresholds were associated with faster learning and higher (that is, "tougher") thresholds were associated with unlearning the target pattern of activation. Vernon et al. (2009) noted that, in studies employing alpha enhancement protocols, thresholds have been placed anywhere between 50% and 85% the amount of alpha seen at rest. This makes training easier than with a threshold set at 100% the average amplitude at baseline (Vernon et al., 2009). In reference to Knox (1980), who suggested a range of possible thresholds, Vernon and colleagues noted that thresholds exceeded by 75% during resting baseline period would probably be both easier and more effective for training than thresholds exceeded by lower percentages, because with higher percentages clients receive more feedback information (Vernon et al., 2009).

A recent pioneering study by Nam and Choi (2020) has yielded empirical results lending support to this thesis. The researchers found in an SMR enhancement session, setting the training threshold so subjects receive more reward (80% reinforcement rate) was more effective than setting it so subjects receive less reward (50% and 30% reinforcement rates).

An fMRI study revealed that during the first training session, neurofeedback signals of failure (i.e., negative feedback) were correlated with deactivations in the precuneus/posterior cingulate. Neurofeedback signals of success were correlated later in the process with deactivations in the medial prefrontal/anterior cingulate cortex (Radua et al., 2018). The level of deactivation in the anterior node predicted the efficacy of the training in reducing anxiety. These results indicate a higher sensitivity to signals of failure at the beginning of neurofeedback learning and to signals of success later in the learning. In the earlier stages of neurofeedback learning, clients may be apprehensive about their ability to learn from feedback and may consequently try to control the feedback consciously. Later, but still at an early stage of learning, this kind of learning diminishes, and learning from positive feedback takes the leading role in the training process. The only predictor of neurofeedback success in Radua et al.'s (2018) study was the level of deactivations in the anterior node. For most participants, this shift occurred as early as the middle of the first session. This study yields further support to the observation that learning from positive feedback has a central role in neurofeedback.

During operant conditioning, following the delivery of reward, an alpha-like activity called postreinforcement synchronization (PRS) occurs (Collura, 2014), the amount of which is related to the speed of learning (Marczynski et al., 1981). It was previously suggested that meaningful information is too difficult to extract from complex neurofeedback games, and such games do not allow for PRS to occur (Sherlin et al., 2011). Employing the same reasoning, it may be the case that neurofeedback training that is too difficult (i.e., does not provide enough positive feedback), may not allow for the PRS complex to occur and therefore may hamper learning. Indeed, it was demonstrated that cognitive tasks of high-load (Sterman et al., 1993) or low-desirability of the reward (Clemente et al., 1964) interfere with PRS.

There have been researchers who placed the threshold above the average amplitudes at baseline in reward frequencies and below the average amplitude at baseline in inhibit frequencies so success rates and reward incidence were lower (Sterman & Egner, 2006). It has been claimed that placing the threshold this way may make the training tedious for clients (Othmer, 2009). In addition, in terms of information-theory, the brain may not receive enough feedback information to work with. Increasing the reward incidence makes the session more rewarding and engaging, and the training more efficient (Othmer, 2009).

Some have objected, on theoretical grounds, to employing very high reward rates, but admit training this way yields good clinical results (Othmer, 2009). Still, it is important not to “choke” the system. The threshold should not be set so low that we would be rewarding too little of the desired activity, because we would then be training the brain to inhibit the desired activity. It has been shown in other contexts of learning that when clients can earn very large rewards, this harms their performance level (i.e., the “choking” effect of very large rewards; Mobbs et al., 2009). The striatum may be involved in this phenomenon (Chib et al., 2012), which seems to have a dopaminergic basis (Mobbs et al., 2009).

Summary and Discussion

The importance of feedback parameters to the success of neurofeedback training cannot be overrated. The way the threshold is set has a crucial effect on learning in neurofeedback (Vernon et al., 2009), and placing the threshold too high or too low may yield either no response or a response opposite to the desired target behavior (Davelaar, 2017). Debates about thresholding have been continuing for quite some time. Despite the importance of thresholding, there is not enough research on the topic (Nam & Choi, 2020; Van Doren et al., 2017; Vernon et al., 2009).

Different types and modalities of feedback yield different levels of success in training. Feedback can be visual, auditory, or tactile; it can be proportional or binary, immediate or delayed, simple or complex. Visual feedback that is proportional, immediate, and simple seems to better support learning (Strehl, 2014). There are also different schedules of reinforcement (e.g., continuous or intermittent; Sherlin et al., 2011) and research is continuing to determine which may be more effective. Feedback can be provided in different ways and affect clinical outcomes. Regardless of the feedback method selected, a higher rate of positive feedback (i.e., more positive than negative feedback) may be preferable.

Neurofeedback is considered an implicit form of learning (Birbaumer et al., 2013; Emmert et al., 2016; Sitaram et al., 2017). Research has shown implicit learning is better acquired when more positive than negative feedback is given to participants (Loonis et al., 2017). In fact, the power of positive feedback is so strong, that even false positive feedback may enhance learning (Sasaki et al., 2010; Shibata et al., 2009). In addition, psychological factors related to motivation, positive affect, mood, self-confidence, and attention contribute to this phenomenon as well (Barbero & Grosse-Wentrup, 2010; Cohen Kadosh & Staunton, 2019; Curran & Stokes, 2003).

Higher levels of positive feedback in the initial stages of training are in accordance with the principles of shaping (i.e., even behaviors that do not meet the target are initially rewarded). But it seems that in neurofeedback, like in other forms of implicit learning, clients should receive more positive than negative feedback not only at the beginning of training but also in later stages of the training series. If the threshold is set so that it is relatively easy to pass and produces larger rates of positive feedback, implicit learning is more easily acquired, clients are more motivated and cooperative, and training becomes more effective and efficient. This may facilitate and shorten the process of fine-tuning the protocol selection process and help clients acquire brainwave self-regulation faster. It may therefore decrease the amount of time required to achieve the training goals. It may also prevent clients from dropping out due to lack of initial response. Utilizing a 10-minute-long session design, Nam and Choi (2020) have provided some preliminary empirical evidence that higher success rates in neurofeedback yielded better results. Research should be conducted to empirically validate the observation that in longer sessions and in later stages of the training as well, higher levels of positive feedback should be employed to achieve more efficient learning.

Reward alone may be less effective than a combination of reward and punishment (Klöbl et al., 2020). Having punishment is motivating too—the motivation to avoid it—and is important for learning (Mohammadi et al., 2018).

There are some accounts that different personality types respond differently to negative and positive feedback (Frank et al., 2005). For example, extroverts learn better from positive feedback and introverts learn better from negative feedback (Boddy et al., 1986). This distinction has not yet been given sufficient attention in neurofeedback research. Experience shows both personality types seem to benefit from neurofeedback training with relatively high rates of positive feedback, probably due to the implicit nature of neurofeedback learning.

Given the importance of positive feedback in neurofeedback training, it is possible some studies that did not find a robust effect for neurofeedback were employing thresholds yielding lower levels of positive feedback. Thus, information about the way thresholds were set and consequent success rates should be provided in neurofeedback research studies (Van Doren et al., 2017).

Some studies that used automatic thresholding to keep a high reward rate constant (80%) failed to find any specific effects for neurofeedback (Lansbergen et al., 2011; Logemann et al., 2010). The problem in such studies may not lay with the high reward rate, but with the fact that the threshold was automatically adjusted every 30 seconds to keep this rate constant. With such settings, no matter what the clients were doing, they were rewarded at the same rate, which may have, in fact, trained them in opposite directions at times (Ayers et al., 2000; Sherlin et al., 2011).

Using positive reinforcement in neurofeedback games is more efficient than using negative reinforcement (Reinschluessel & Mandryk, 2016). A plausible strategy for training in neurofeedback systems that use negative reinforcement (e.g., systems in which the game freezes when brainwaves do not meet the target) may be to reframe the feedback by asking clients to view the negative state (i.e., the frozen game) as the default state, and the removal of this state as a reward for their achievements.

Lastly, research should be conducted to determine whether a higher rate of positive than negative feedback is effective in all types of protocols and frequency bands or only in some of them; in all kinds of neural indices or just in part of them; with all forms of neurofeedback or only with specific feedback modalities; for all clients or for only specific clinical populations or personality types. Once these questions are empirically answered, the field of neurofeedback may take a substantial leap forward.

Author Disclosure

The author does not have any grants, financial interests or conflicts to disclose.

References

Alkoby, O., Abu-Rmileh, A., Shriki, O., & Todder, D. (2018). Can we predict who will respond to neurofeedback? A review of the inefficacy problem and existing predictors for successful EEG neurofeedback learning. *Neuroscience*, *378*, 155–164. https://doi.org/10.1016/j.neuroscience.2016.12.050

Argyelan, M., Herzallah, M., Sako, W., DeLucia, I., Sarpal, D., Vo, A, Fitzpatrick, T., Moustafa, A. A., Eidelberg, D., & Gluck, M. (2018). Dopamine modulates striatal response to reward and punishment in patients with Parkinson's disease: A pharmacological challenge fMRI study. *NeuroReport, 29*(7), 532–540. https://doi.org/10.1097/wnr.0000000000000970

Ayers, M. E., Sams, M. W., & Sterman, M. B. (2000). When to inhibit EEG activity instead of reinforcing and inhibiting simultaneously. *Journal of Neurotherapy, 4*(1), 83–93. https://doi.org/10.1300/J184v04n01_10

Barabasz, A. F., & Barabasz, M. (1999). Treating ADHD with hypnosis and neurotherapy. *Paper presented at the 1999 Annual Convention of the American Psychological Association*. Boston, MA. https://files.eric.ed.gov/fulltext/ED435076.pdf

Barbero, Á., & Grosse-Wentrup, M. (2010). Biased feedback in brain-computer interfaces. *Journal of NeuroEngineering and Rehabilitation, 7*, Article 34. https://doi.org/10.1186/1743-0003-7-34

Birbaumer, N., Ruiz, S., & Sitaram, R. (2013). Learned regulation of brain metabolism. *Trends in Cognitive Sciences, 17*(6), 295–302. https://doi.org/10.1016/j.tics.2013.04.009

Boddy, J., Carver, A., & Rowley, K. (1986). Effects of positive and negative verbal reinforcement on performance as a function of extraversion-introversion: Some tests of Gray's theory. *Personality and Individual Differences, 7*(1), 81–88. https://doi.org/10.1016/0191-8869(86)90111-X

Bromberg-Martin, E. S., Matsumoto, M., & Hikosaka, O. (2010). Dopamine in motivational control: Rewarding, aversive, and alerting. *Neuron, 68*(5), 815–834. https://doi.org/10.1016/j.neuron.2010.11.022

Chafee, M. V., & Crowe, D. A. (2017). Implicit and explicit learning mechanisms meet in monkey prefrontal cortex. *Neuron, 96*(2), 256–258. https://doi.org/10.1016/j.neuron.2017.09.049

Chib, V. S., De Martino, B., Shimojo, S., & O'Doherty, J. P. (2012). Neural mechanisms underlying paradoxical performance for monetary incentives are driven by loss aversion. *Neuron, 74*(3), 582–594. https://doi.org/10.1016/j.neuron.2012.02.038

Clemente, C. D., Sterman, M. B., & Wyrwicka, W. (1964). Post-reinforcement EEG synchronization during alimentary behavior. *Electroencephalography and Clinical Neurophysiology, 16*(4), 355–365. https://doi.org/10.1016/0013-4694(64)90069-0

Cohen Kadosh, K., & Staunton, G. (2019). A systematic review of the psychological factors that influence neurofeedback learning outcomes. *NeuroImage, 185,* 545–555, https://doi.org/10.1016/j.neuroimage.2018.10.021

Collura, T. F. (2014). *Technical foundations of neurofeedback* (pp. 16–17). Routledge.

Curran, E. A., & Stokes, M. J. (2003). Learning to control brain activity: A review of the production and control of EEG components for driving brain–computer interface (BCI) systems. *Brain and Cognition, 51*(3), 326–336. https://doi.org/10.1016/S0278-2626(03)00036-8

Daffertshofer, A., & van Wijk, B. C. M. (2011). On the influence of amplitude on the connectivity between phases. *Frontiers in Neuroinformatics, 5*, 6. https://doi.org/10.3389/fninf.2011.00006

Davelaar, E. J. (2017). A computational approach to developing cost-efficient adaptive-threshold algorithms for EEG neuro feedback. *International Journal of Structural and Computational Biology, 1*(2), 1–4. https://eprints.bbk.ac.uk/id/eprint/21537/1/21537.pdf

Davelaar, E. J. (2018). Mechanisms of neurofeedback: A computation-theoretic approach. *Neuroscience, 378*, 175–188. https://doi.org/10.1016/j.neuroscience.2017.05.052

Egner, T., Zech, T. F., & Gruzelier, J. H. (2004). The effects of neurofeedback training on the spectral topography of the electroencephalogram. *Clinical Neurophysiology, 115*(11), 2452–2460. https://doi.org/10.1016/j.clinph.2004.05.033

Elliott, R., Agnew, Z., & Deakin, J. F. W. (2010). Hedonic and informational functions of the human orbitofrontal cortex. *Cerebral Cortex, 20*(1), 198–204. https://doi.org/10.1093/cercor/bhp092

Emmert, K., Kopel, R., Sulzer, J., Brühl, A. B., Berman, B. D., Linden, D. E. J., Horovitz, S. G., Breimhorst, M., Caria, A., Frank, S., Johnston, S., Long, Z., Paret., C., Robineau, F., Veit, R., Bartsch, A., Beckmann, C. F., Van De Ville, D., & Haller, S. (2016). Meta-analysis of real-time fMRI neurofeedback studies using individual participant data: How is brain regulation mediated? *NeuroImage, 124*(Part A), 806–812. https://doi.org/10.1016/j.neuroimage.2015.09.042

Evans, J. J., Wilson, B. A., Schuri, U., Andrade, J., Baddeley, A., Bruna, O., Canavan, T., Del Sala, S., Green, R., Laaksonen, R., Lorenzi, L., & Taussik, I. (2000). A comparison of "errorless" and "trial-and-error" learning methods for teaching individuals with acquired memory deficits. *Neuropsychological Rehabilitation, 10*(1), 67–101. https://doi.org/10.1080/096020100389309

Fehmi, L. G., & Robbins, J. (2008). *The open-focus brain: harnessing the power of attention to heal mind and body* (pp. 29–40). Boulder, CO: Shambhala Publications.

Fisher, S. F. (2014). *Neurofeedback in the treatment of developmental trauma: Calming the fear-driven brain* (pp. 277–324). W.W. Norton & Company.

Frank, M., Woroch, B. S., & Curran, T. (2005). Error-related negativity predicts reinforcement learning and conflict biases. *Neuron, 47*(4), 495–501. https://doi.org/10.1016/j.neuron.2005.06.020

Gastaldi, F. (2023, February). *Neurofeedback, tutto sulla tecnica scelta da Marco Mengoni per liberarsi dallo stress*. Vanity Fair. https://www.vanityfair.it/article/neurofeedback-come-funziona-tecnica-marco-mengoni-anti-stress

Gruzelier, J. H. (2014). EEG-neurofeedback for optimising performance. III: A review of methodological and theoretical considerations. *Neuroscience & Biobehavioral Reviews*, *44*, 159–182. https://doi.org/10.1016/j.neubiorev.2014.03.015

Hardt, J. V., & Kamiya, J. (1976). Conflicting results in EEG alpha feedback studies. *Biofeedback and Self-Regulation, 1*(1), 63–75. https://doi.org/10.1007/BF00998691

Heindel, W. C., Salmon, D. P., Shults, C. W., Walicke, P. A., & Butters, N. (1989). Neuropsychological evidence for multiple implicit memory systems: A comparison of Alzheimer's, Huntington's, and Parkinson's disease patients. *The Journal of Neuroscience, 9*(2), 582–587. https://doi.org/10.1523/JNEUROSCI.09-02-00582.1989

Johnson, M. L., & Bodenhamer-Davis, E. (2009). QEEG-based protocol selection: A study of level of agreement on sites, sequences, and rationales among a group of experienced QEEG-based neurofeedback practitioners. *Journal of Neurotherapy*, *13*(1), 41–66. https://doi.org/10.1080/10874200802668416

Klöbl, M., Michenthaler, P., Godbersen, G. M., Robinson, S., Hahn, A., & Lanzenberger, R. (2020). Reinforcement and punishment shape the learning dynamics in fMRI neurofeedback. *Frontiers in Human Neuroscience, 14*, 304. https://doi.org/10.3389/fnhum.2020.00304

Knox, S. S. (1980). Distribution of 'criterion' alpha in the resting EEG: Further argument against the use of an amplitude threshold in alpha biofeedback training. *Biological*

Psychology, 11(1), 1–6. https://doi.org/10.1016/0301-0511(80)90021-6

Kober, S. E., Witte, M., Ninaus, M., Neuper, C., & Wood, G. (2013). Learning to modulate one's own brain activity: The effect of spontaneous mental strategies. *Frontiers in Human Neuroscience, 7*, 695. https://doi.org/10.3389/fnhum.2013.00695

Koralek, A. C., Jin, X., Long II, J. D., Costa, R. M., & Carmena, J. M. (2012). Corticostriatal plasticity is necessary for learning intentional neuroprosthetic skills. *Nature, 483*(7389), 331–335. https://doi.org/10.1038/nature10845

Lam, S.-L., Criaud, M., Alegria, A., Barker, G. J., Giampietro, V., & Rubia, K. (2020). Neurofunctional and behavioural measures associated with fMRI-neurofeedback learning in adolescents with Attention-Deficit/Hyperactivity Disorder. *NeuroImage Clinical, 27*, 102291. https://doi.org/10.1016/j.nicl.2020.102291

Lansbergen, M. M., van Dongen-Boomsma, M., Buitelaar, J. K., & Slaats-Willemse, D. (2011). ADHD and EEG-neurofeedback: A double-blind randomized placebo-controlled feasibility study. *Journal of Neural Transmission, 118,* 275–284. https://doi.org/10.1007/s00702-010-0524-2

Lin, J. J., Mamykina, L., Lindtner, S., Delajoux, G., & Strub, H. B. (2006). Fish'n'Steps: Encouraging physical activity with an interactive computer game. In P. Dourish, & A. Friday (Eds.)*,* *UbiComp 2006: Ubiquitous computing*. Lecture Notes in Computer Science, vol. 4206. Springer, Berlin, Heidelberg. https://doi.org/10.1007/11853565_16

Logemann, H. N. A., Lansbergen, M. M., Van Os, T. W. D. P., Böcker, K. B. E., & Kenemans, J. L. (2010). The effectiveness of EEG-feedback on attention, impulsivity and EEG: A sham feedback controlled study. *Neuroscience Letters, 479*(1), 49–53. https://doi.org/10.1016/j.neulet.2010.05.026

Loonis, R. F., Brincat, S. L., Antzoulatos, E. G., & Miller, E. K. (2017). A meta-analysis suggests different neural correlates for implicit and explicit learning. *Neuron, 96*(2), 521–534.e7. https://doi.org/10.1016/j.neuron.2017.09.032

Marczynski, T. J., Harris, C. M., & Livezey, G. T. (1981). The magnitude of post-reinforcement EEG synchronization (PRS) in cats reflects learning ability. *Brain Research, 204*(1), 214–219. https://doi.org/10.1016/0006-8993(81)90667-3

Maxwell, J. P., Masters, R. S. W., Kerr, E., & Weedon, E. (2001). The implicit benefit of learning without errors. *The Quarterly Journal of Experimental Psychology: A Human Experimental Psychology, 54A*(4), 1049–1068. https://doi.org/10.1080%2F713756014

Mobbs, D., Hassabis, D., Seymour, B., Marchant, J. L., Weiskopf, N., Dolan, R. J., & Frith, C. D. (2009). Choking on the money: Reward-based performance decrements are associated with midbrain activity. *Psychology Science, 20*(8), 955–962. https://doi.org/10.1111/j.1467-9280.2009.02399.x

Mohammadi, H. S., Pirbabaei, E., Sisi, M. J., & Sekhavat, Y. A. (2018). ExerBrain: A comparison of positive and negative reinforcement in attention training using BCI based computer games. In *2018 2nd National and 1st International Digital Games Research Conference: Trends, Technologies, and Applications (DGRC)*, *11,* 167–171. Tehran, Iran. https://doi.org/10.1109/DGRC.2018.8712048.

Nam, S., & Choi, S. (2020). Effect of threshold setting on neurofeedback training. *NeuroRegulation, 7*(3), 107–117. https://doi.org/10.15540/nr.7.3.107

Nijboer, F., Birbaumer, N., & Kübler, A. (2010). The influence of psychological state and motivation on brain–computer interface performance in patients with amyotrophic lateral sclerosis–a longitudinal study. *Frontiers in Neuropharmacology, 4,* 55. https://doi.org/10.3389/fnins.2010.00055

Niv, S. (2013). Clinical efficacy and potential mechanisms of neurofeedback. *Personality and Individual Differences, 54*(6), 676–686. https://doi.org/10.1016/j.paid.2012.11.037

Oblak, E. F., Sulzer, J. S., & Lewis-Peacock, J. A. (2019). A simulation-based approach to improve decoded neurofeedback performance. *NeuroImage*, *195*, 300–310. https://doi.org/10.1016/j.neuroimage.2019.03.062

O'Doherty, J., Kringelbach, M. L., Rolls, E. T., Hornak, J., & Andrews, C. (2001). Abstract reward and punishment representations in the human orbitofrontal cortex. *Nature Neuroscience*, *4*(1), 95–102. https://doi.org/10.1038/82959

Othmer, S. (2009). In J. R. Evans, T. H. Budzynski, H. K. Budzynski, & A. Abarbanel (Eds.), *Introduction to quantitative EEG and neurofeedback: Advanced theory and applications* (pp. 3–26). Academic Press.

Poldrack, R. A., Clark, J., Paré-Blagoev, E. J., Shohamy, D., Moyano, J. C., Myers, C., & Gluck, M. A. (2001). Interactive memory systems in the human brain. *Nature, 414,* 546–550. https://doi.org/10.1038/35107080

Poolton, J. M., Masters, R. S. W., & Maxwell, J. P. (2005). The relationship between initial errorless learning conditions and subsequent performance. *Human Movement Science, 24*(3), 362–378. https://doi.org/10.1016/j.humov.2005.06.006

Radua, J., Stoica, T., Scheinost, D., Pittenger, C., & Hampson, M. (2018). Neural correlates of success and failure signals during neurofeedback learning. *Neuroscience*, *378*, 11–21. https://doi.org/10.1016/j.neuroscience.2016.04.003

Ramot, M., Grossman, S., Friedman, D., & Malach, R. (2016). Covert neurofeedback without awareness shapes cortical network spontaneous connectivity. *PNAS Proceedings of the National Academy of Sciences of the United States of America, 113*(17), E2413–E2420. https://doi.org/10.1073/pnas.1516857113

Reinschluessel, A. V., & Mandryk, R. L. (2016). Using positive or negative reinforcement in neurofeedback games for training self-regulation. In *Proceedings of the 2016 Annual Symposium on Computer-Human Interaction in Play* (pp. 186–198). https://doi.org/10.1145/2967934.2968085

Rogala, J., Jurewicz, K., Paluch, K., Kublik, E., Cetnarski, R., & Wróbel, A. (2016). The do's and don'ts of neurofeedback training: A review of the controlled studies using healthy adults. *Frontiers in Human Neuroscience, 10,* 301. https://doi.org/10.3389/fnhum.2016.00301

Ros, T., Moseley, M. J., Bloom, P. A., Benjamin, L., Parkinson, L. A., & Gruzelier, J. H. (2009). Optimizing microsurgical skills with EEG neurofeedback. *BMC Neuroscience, 10*(1), Article 87. https://doi.org/10.1186/1471-2202-10-87

Ros, T., Frewen, P., Théberge, J., Michela, A., Kluetsch, R., Mueller, A., Candrian, G., Jetly, R., Vuilleumier, P., & Lanius, R. A. (2017). Neurofeedback tunes scale-free dynamics in spontaneous brain activity. *Cerebral Cortex, 27*(10), 4911–4922. https://doi.org/10.1093/cercor/bhw285

Sasaki, Y., Nanez, J. & Watanabe, T. (2010). Advances in visual perceptual learning and plasticity. *Nature Reviews Neuroscience, 11,* 53–60. https://doi.org/10.1038/nrn2737

Schafer, R. J., & Moore, T. (2011). Selective attention from voluntary control of neurons in prefrontal cortex. *Science, 332*(6037), 1568–1571. https://doi.org/10.1126/science.1199892

Sherlin, L. H., Arns, M., Lubar, J., Heinrich, H., Kerson, C., Strehl, U., & Sterman, M. B. (2011). Neurofeedback and basic learning theory: Implications for research and practice. *Journal of Neurotherapy, 15*(4), 292–304. https://doi.org/10.1080/10874208.2011.623089

Shibata, K., Yamagishi, N., Ishii, S., & Kawato, M. (2009). Boosting perceptual learning by fake feedback. *Vision Research, 49*(21), 2574–2585. https://doi.org/10.1016/j.visres.2009.06.009

Shourie, N., Firoozabadi, M., & Badie, K. (2018). Fuzzy adaptive neurofeedback training: An efficient neurofeedback training procedure providing a more accurate progress rate for trainee. *Biomedical Signal Processing and Control, 44,* 75–81. https://doi.org/10.1016/j.bspc.2018.02.009

Siniatchkin, M., Kropp, P., & Gerber, W.-D. (2000). Neurofeedback–The significance of reinforcement and the search for an appropriate strategy for the success of self-regulation. *Applied Psychophysiology and Biofeedback 25*(3), 167–175. https://doi.org/10.1023/A:1009502808906

Sitaram, R., Ros, T., Stoeckel, L., Haller, S., Scharnowski, F., Lewis-Peacock, J., Weiskopf, N., Blefari, M. L., Rana, M., Oblak, E., Birbaumer, N., & Sulzer, J. (2017). Closed-loop brain training: The science of neurofeedback. *Nature Reviews Neuroscience, 18*(2), 86–100. https://doi.org/10.1038/nrn.2016.164

Skinner, B. F. (1945). The operational analysis of psychological terms. *Psychological Review, 52*(5), 270–277. https://doi.org/10.1037/h0062535

Skottnik, L., Sorger, B., Kamp, T., Linden, D., & Goebel, R. (2019). Success and failure of controlling the real-time functional magnetic resonance imaging neurofeedback signal are reflected in the striatum. *Brain and Behavior, 9*(3), Article e01240. https://doi.org/10.1002/brb3.1240

Sterman, M. B., & Egner, T. (2006). Foundation and practice of neurofeedback for the treatment of epilepsy. *Applied Psychophysiology and Biofeedback, 31*(1), 21–35. https://doi.org/10.1007/s10484-006-9002-x

Sterman, M. B., Mann, C. A., & Kaiser, D. A. (1993, February). Quantitative EEG patterns of differential in-flight workload. In NASA. Johnson Space Center, Sixth Annual Workshop on Space Operations Applications and Research (SOAR 1992), Volume 2.

Strehl, U. (2014). What learning theories can teach us in designing neurofeedback treatments. *Frontiers in Human Neuroscience*, *8*, 894. https://doi.org/10.3389/fnhum.2014.00894

Sulzer, J., Sitaram, R., Blefari, M. L., Kollias, S., Birbaumer, N., Stephan, K. E., Luft, A., & Gassert, R. (2013). Neurofeedbackmediated self-regulation of the dopaminergic midbrain. *NeuroImage, 83,* 817–825. https://doi.org/10.1016/j.neuroimage.2013.05.115

Thompson, L., & Thompson, M. (1998). Neurofeedback combined with training in metacognitive strategies: Effectiveness in students with ADD. *Applied Psychophysiology and Biofeedback, 23*(4), 243–263. https://doi.org/10.1023/A:1022213731956

Thorndike, E. L. (1999). Animal intelligence (p. v). Bristol, UK: Thoemmes. (Original work published 1911).

Tsushima, Y., Seitz, A. R., & Watanabe, T. (2008). Task-irrelevant learning occurs only when the irrelevant feature is weak. *Current Biology, 18*(12)*,* R516–R517. https://doi.org/10.1016/j.cub.2008.04.029

Van der Kolk, B. (2014). *The body keeps the score: Brain, mind, and body in the healing of trauma* (pp. 309–329). Penguin Publishing Group.

Van Doren, J., Heinrich, H., Bezold, M., Reuter, N., Kratz, O., Horndasch, S., Berking, M., Ros, T., Gevensleben, H., Moll, G. H., & Studer, P. (2017). Theta/beta neurofeedback in children with ADHD: Feasibility of a short-term setting and plasticity effects. *International Journal of Psychophysiology, 112,* 80–88. https://doi.org/10.1016/j.ijpsycho.2016.11.004

Vernon, D., Dempster, T., Bazanova, O. M., Rutterford, N., Pasqualini, M., & Andersen, S. (2009). Alpha neurofeedback training for performance enhancement: Reviewing the methodology. *Journal of Neurotherapy, 13*(4), 214–227. https://doi.org/10.1080/10874200903334397

Wächter, T., Lungu, O. V., Liu, T., Willingham, D. T., & Ashe, J. (2009). Differential effect of reward and punishment on procedural learning. *Journal of Neuroscience, 29*(2), 436–443. https://doi.org/10.1523/JNEUROSCI.4132-08.2009

White, N. E., & Richards, L. M. (2009). Alpha–theta neurotherapy and the neurobehavioral treatment of addictions, mood disorders and trauma. In J. R. Evans, T. H. Budzynski, H. K. Budzynski, & A. Abarbanel (Eds.). *Introduction to quantitative EEG and neurofeedback: Advanced theory and applications* (pp. 143–164). Academic Press.

Zuberer, A., Brandeis, D., & Drechsler, R. (2015). Are treatment effects of neurofeedback training in children with ADHD related to the successful regulation of brain activity? A review on the learning of regulation of brain activity and a contribution to the discussion on specificity. *Frontiers in Human Neuroscience, 9,* 135. https://doi.org/10.3389/fnhum.2015.00135

Received: November 26, 2022
Accepted: March 19, 2023
Published: March 30, 2023

NeuroRegulation

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NEUROREGULATION & RESEARCH

The Suisun Summit 2022: Emerging Themes and Open Questions in qEEG and Neurotherapies

Rogene M. Eichler West[1,2], Mary Tracy[3], Ronald J. Swatzyna[4], Robert P. Turner[5], Michael Pierce[6], Mark Jones[7], Nicky Whittredge[8], Jessica Eure[9], Giancarlo Licata[10], Taylor Capozziello[11], Rachman Chung[12], Clementine Clerc[13], Shari Johansson[6], Myler Leachman[14], Christy Lewis[15], Yvonne Tate[16], Gay L. Teurman[17], Crystal Turman[18], Linda Weber[19], Mica Stumpf[20], and Jay Gunkelman[21]*

[1]Northwest Neuro Professionals, LLC, Seattle, Washington, USA
[2]Pacific Northwest National Laboratory, Seattle, Washington, USA
[3]NeuroTraining Strategies, Davis, California, USA
[4]Houston Neuroscience Brain Center, Houston, Texas, USA
[5]Network Neurology Health, Charleston, South Carolina, USA
[6]Grey Matters Neurofeedback & Counseling, Lakewood, Colorado, USA
[7]University of Texas at San Antonio, Well Mind Center, San Antonio, Texas, USA
[8]Saybrook University, Pasadena, California, USA
[9]Virginia Center for Neurofeedback, Attachment & Trauma, Charlottesville, Virginia, USA
[10]Vital Brain Health, Pasadena, California, USA
[11]Elite Physiology, Inc, San Francisco, California, USA
[12]NeuroAxis Health, San Francisco, California, USA
[13]OPTIMA neurofeedback, Marseille, France
[14]Convergent Brain Diagnostics, PC, El Dorado Hills, California, USA
[15]The Biofeedback, Education, & Training Center, PLLC, Plano, Texas, USA
[16]TRS Neurofeedback, Lakewood, Colorado, USA
[17]Brain Health Clinic, Sacramento, California, USA
[18]Wellspring Counseling Services, LLC, Satellite Beach, Florida, USA
[19]Mindful Feedback, London, Ontario, Canada
[20]East Bay Agency for Children, Oakland, California, USA
[21]Private Consulting, Suisun, California, USA

Abstract

The Suisun Summit 2022 was a gathering of 70 clinicians, educators, and researchers in quantitative electroencephalography and neurofeedback. During this 5-day event, several themes emerged in talks or discussion groups: EEG/qEEG Reading Skills; Medication Effects and Pharmaco-EEG; Technological Advancements; Emerging Concerns; and Growing Community Prestige through Research. Participants were asked to summarize what they believed to be the most important messages from the event to share with colleagues who were not in attendance, resulting in this review. A unifying concept for all the themes was a desire for higher quality, standardized EEG/qEEG education that provides depth as well as breadth. Models of clinical care that encourage open communication with prescribers and functional medicine specialists were strongly emphasized. Abstracts from all presentations are attached in Addendum B.

Keywords: EEG; qEEG; neurofeedback; education; conference review; best practices

Citation: Eichler West, R. M., Tracy, M., Swatzyna, R. J., Turner, R. P., Pierce, M., Jones, M., Whittredge, N., Eure, J., Licata, G., Capozziello, T., Chung, R., Clerc, C., Johansson, S., Leachman, M., Lewis, C., Tate, Y., Teurman, G. L., Turman, C., Weber, L., Stumpf, M., & Gunkelman, J. (2023). The Suisun Summit 2022: Emerging themes and open questions in qEEG and neurotherapies. *NeuroRegulation, 10*(1), 42–60. https://doi.org/10.15540/nr.10.1.42

***Address correspondence to**: Rogene M. Eichler West, PhD, Northwest Neuro Professionals, LLC, 11054 19th Ave NE, Seattle, WA 98125, USA. Email: dr.west@nwneuro.pro

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Edited by:
Rex L. Cannon, PhD, Currents, Knoxville, Tennessee, USA

Reviewed by:
Rex L. Cannon, PhD, Currents, Knoxville, Tennessee, USA
Randall Lyle, PhD, Mount Mercy University, Cedar Rapids, Iowa, USA

Introduction

The Suisun Summit was a gathering of nearly 70 clinicians, educators, and researchers in quantitative electroencephalography (qEEG) and neurofeedback on October 12–16, 2022, in Suisun City, California. For most, this was the first in-person professional conference they attended since before the COVID pandemic. Twelve presentations and group discussions were organized as a 5-day, single-track event, that was flawlessly executed by Mary Tracy. Many participants commented that it was the best conference they have ever attended, both for the quality of the presentations and the inclusive sense of comradery they experienced.

International Society for Neuroregulation and Research (ISNR) President Mark Jones is on a mission that his term at the helm be known for reinvigorating the "R stands for research" into the identity of ISNR. He and Ron Swatzyna set a tone for the event on the first day with an encouraging discussion on developing support for clinicians to design research projects and gather data in their private practices. The final discussion, led by Rogene Eichler West, organized attendees to co-author this conference review so that they might gain some experience with the process of publishing in a peer-reviewed journal.

The group first identified common cross-cutting themes and then were asked to remark on: (a) the current state of knowledge; (b) the new material that was introduced during the Summit that other professionals in the field should know about; and (c) the open questions that were driving the future of the field. Multiple themes emerged: qEEG Reading Skills; Medication Effects and Pharmaco-EEG; Technological Advancement; Growing Community Prestige through Research; and Emerging Concerns. From here, smaller groups were formed with each responsible for writing a summary of each theme. Each group had the opportunity to offer feedback on the sections submitted by the other groups.

The reminder of this paper explores those themes in greater detail. The paper closes with some comments on the history of the event, born out of two workshops in 2019, as well as the origin of the Onto Innovations Study Group, many of whose members were in attendance.

QEEG Reading Skills

An emphasis on improving qEEG reading skills using visual inspection as the first step in the interpretation of neuropsychiatric symptoms in the brain is a challenge of continuous refinement. There are various pitfalls to avoid and many subtleties that require exposure to many EEGs for reliable interpretation. This summit was not intended to be a didactic for a qEEG exam, nor was it a tutorial on abnormal waveforms. Instead, EEG reading skills were emphasized in the context of case reviews. Jay Gunkelman presented talks on "Unexpected Epileptiform Content in the EEG" and "PTSD Biomarkers with ERP and EEG." Tiff Thompson delivered the presentation on "Pediatrics and Developmental Trauma in the Brain." Angelika Sadar discussed important reading skills during her moderated discussion of "Medications and EEG Correlates," as did Ron Swatzyna and Jay Gunkelman, who presented on "Pharmaco-EEG and Phenotypes: The Interface between Neurology, Psychiatry, and Neuromodulation."

The Challenges to Becoming Competent in Reading Raw EEG

Philosophically, and from a practical standpoint, what unites most participants at this event is the fundamental assumption that interpreting a qEEG requires the ability to read and interpret the raw EEG first before processing it with higher order statistics (i.e., Fast Fourier Transforms, comparison with normative databases, and displays of head maps). Processed algorithms are averaged over all epochs in a recording, which means that important but less frequently occurring features, such as isolated epileptiform discharges, may not show up in the final analysis. Understanding how filters can alter waveform morphology could explain the appearance of multiple peaks in the power spectrum. Furthermore, it is not always possible to remove all sources of artifacts; therefore, one needs training to develop confidence in determining that the higher order statistics and brain maps are a true reflection of the raw EEG features and have not been influenced by artifacts or skewed by frequencies outside of their range, such as alpha loading into theta or beta bands, since the brain maps have fixed frequencies. For instance, slow alpha loading into the theta band results in a high theta beta ratio, and leptokurtic alpha appears as hypercoherence in that single frequency.

Our competence as clinicians and outcomes for our clients improve significantly when we commit ourselves to acquiring these skills. For example, the pattern of waxing, waning, and intermittent coherence of frontal alpha suggests issues with vigilance and perhaps chronic poor sleep quality. A qEEG clinician trained in reading raw EEG data will have a better sense of whether a complaint of attention issues might first be addressed through improved sleep hygiene. Dropping into stage 2 sleep before 300 seconds (technically known as "mean sleep latency time") necessitates a referral for sleep monitoring as untreated sleep apnea can be life-threatening and can explain many presenting symptoms that could improve with better quality sleep. And certainly, a neurofeedback session on someone who is unable to maintain vigilance is of dubious value.

Opportunities to become competent in seeing these patterns, developing skills in interpreting EEG data using different montages that verify the focal localization and identification of abnormal EEG features, and awareness of the multitude of clinical interpretations, especially in the realm of mental health, are difficult to find in this burgeoning field. This is why we emphasize the attainment of a higher standard of EEG skills by encouraging students to become Board Certified in qEEG.

Formal Training of Neurologists and Psychiatrists in Reading Raw EEG

Formal training in reading EEG is typically restricted to neurologists and some psychiatrists. However, even in this population, 43.2% of neurology residencies do not include dedicated EEG rotations. A residency that does contain an EEG rotation only provides 6 weeks of EEG training, and 74% of neurology trainees will receive no further EEG training beyond this residency (Weber et al., 2016). Beyond this, more highly specialized training requires an additional 1 or 2 years in an epilepsy or sleep medicine fellowship and a board exam after completing a 4-year neurology residency after medical school. These specialists may further specialize in pediatrics and neonatal populations.

Even though medical professionals and qEEG analysts like us are looking at the same data, medical specialists do not learn clinical correlates associated with mental health, and we as qEEG analysts do not have the specialty training to make definitive calls for medical referrals. How can we be certain that appropriate help for our clients is not lost in the space between our scopes of practice? There are two steps we can take to move forward: (a) we can continue to develop learning opportunities for standardized and structured training in EEG reading skills and interpretation for mental health practitioners; (b) we can seek to build bridges between the communities so that appropriate communication can be facilitated, and cross-referrals can be made.

Nonmedical qEEG Certification and the Importance of Ongoing Education

Currently the standard in nonmedical EEG is a qEEG certification as a Diplomate or Technologist through the International qEEG Certification Board (IQEEGCB). Passing the qEEG Board exam is intended to demonstrate one's general knowledge of electrophysiological processes, instrumentation features, and clinical correlates of EEG and qEEG findings. In addition, the ability to confidently record, artifact, and process the EEG using various technologies for clinical interpretation is one gold standard for attaining certification. However, even with these credentials, it may take years of practice and the examination of thousands of EEGs with mentoring from an expert to become a competent qEEG Analyst. The IQEEGCB requirement for writing five qEEG reports, and 10 hours of mentoring by a qEEG Diplomate is not enough. It is for this reason that opportunities for ongoing education such as weekly grand rounds or presentations that emphasize EEG reading skills are held in such high regard by the attendees of this event.

The Power of Case Studies

Jay Gunkelman, who was the featured expert in EEG and qEEG at the Summit, is technically an EEG technician, not a clinician. His profound influence and deep commitment to the education of this community emphasizes the importance of intensive training and experience over titles. Jay's method of instruction is centered on case studies. He presented an example where a 55-year-old female, in otherwise good health, developed trouble with word finding and lost the ability to sing. While reviewing her EEG with a clinical colleague, he pointed out sharp slow transients emanating from the temporal area. Jay understood that this waveform, particularly at this location, suggested deleterious vascular changes, perhaps in the aortic arch, left brachial or left carotid artery. The clinician then pushed for a referral for further testing in the form of a magnetic resonance angiography (MRA), which specifically evaluates the condition of blood vessels. The MRA revealed an aneurysm, which resulted in proper medical intervention, likely saving this person's life.

That story has an amazingly happy ending. It illustrates the power of raw EEG interpretation in the hands of those properly trained, and in partnership with a clinician whose scope of practice allows further medically based investigation.

In another case study, we learned of a client whose family intervened in a scheduled neurosurgery procedure of a family member in favor of trying neurofeedback training. This young woman was diagnosed with a seizure disorder and was given the option of surgery as a last resort. Fortunately, the family became aware of the potential of neurofeedback to stabilize the dysregulated pattern. Gunkelman and his team worked with the neurologist to offer an alternative treatment, and this young woman is now living a seizure-free life without having to undergo neurosurgery.

Examples of Clinical Correlates That Are the Product of Raw EEG Interpretation

Throughout the various talks, several other correlates were discussed, along with how their presence indicated the most efficacious treatment selection:

- Beta spindles can indicate several issues from anxiety to insomnia to inflammatory processes to benzodiazepine or amphetamine intoxication, with distribution, amplitude, and frequency contributing to the interpretation (Swatzyna et al., 2015).
- Paroxysmal activity is present in between 20% and 60% of clients with attention-deficit/hyperactivity disorder (ADHD) and autism spectrum disorder (ASD), which need to be stabilized before other interventions are pursued (Swatzyna et al., 2015).
- Phenotype classification may reveal physiological issues that need to be addressed before engaging in other neurotherapies. For example, low power EEGs (less than ± 25 mV) should first be assessed for metabolic insufficiencies, infections or toxicities, or traumatic brain injuries.

Tiff Thompson, a specialist in neonatal and pediatric EEG and trained as a registered EEG tech as well as a licensed clinical psychologist, presented on “Pediatrics and Developmental Trauma in the Brain.” Her talk stood out because of the use of a timeline to introduce the developmental evolution of EEG markers, neurobiology, and behaviors from birth to adolescence. This approach was both complementary to clinical case studies and integrative of the emergence of expected waveforms. It also took the form of a longitudinal case study, featuring her own children!

Mastering EEG Tools

There are a great many analytic tools available, each with its strengths and weaknesses. A practitioner must know how to choose the correct tool for each kind of analysis. “You are only as good as your data and your ability to quantitatively process that data” (Gunkelman, 2014). For example, the “correct” montage is the one that allows you to verify the signal source in a particular way. The Linked Ears montage is the derivation used to create databases, so this montage needs to be used when referencing a normative database. This montage can be contaminated with ear lead and temporal artifacts, however, which creates the appearance of elevated coherence. Neurologists use bipolar montages to best localize the origin of a particular waveform (Nunez & Srinivasan, 2006).

EEG Reading Skills and Neurofeedback

EEG reading skills directly impact one’s ability to assess appropriate neurofeedback software to accurately target the marker of greatest concern. A “hot-spot-ologist” might try to train down a beta peak at 20 Hz, only to be training down a 10 Hz alpha with a notched appearance, because of filter settings. Focal slowing, for example, might not be adequately trained using standard band and reward settings if the occurrence is relatively infrequent. Excess frontal beta or peak alpha frequencies above 12 Hz can indicate dysregulation but may also indicate markers of the drive and motivation of peak performers. Training the clients to turn this ability on and off at will is far more powerful than squashing excess frontal beta because their *z*-score is an outlier.

Medication Effects and Pharmaco-EEG

Two of the presentations were explicitly about the impact of medications on EEG or, conversely, the use of EEG to better select medications. Angelika Sadar moderated a group discussion on “Medications and EEG Correlates”; Ron Swatzyna and Jay Gunkelman presented a talk on “Pharmaco-EEG and Phenotypes: The Interface between Neurology, Psychiatry, and Neuromodulation.”

Influence of and Selection of Drugs From Raw Data

The discussion with Angelika Sadar reinforced the importance of learning to disambiguate medication-related features in the raw data on the EEG from those generated by the brain for more accurate interpretation, as it is unethical for qEEG analysts to instruct a client to taper off medications before collecting a qEEG unless they are also the prescribing MD. While some medications can be expected to have the same effect on all brains, such as diffuse presentation of beta associated with benzodiazepines or amphetamines, other drugs will exert their effect based on a baseline of overarousal or underarousal. This knowledge is useful when selecting an applied psychophysiological protocol appropriate to the underlying brain patterns rather than their chemically shifted state.

Jay Gunkelman cited two studies (Arns et al., 2008; Loo et al., 2016) where the EEG phenotype model was instrumental in an improved success rate when selecting ADHD medications. He gave examples where the choice of the medication for psychotic illness, bipolar disorder, epileptiform activity, or posttraumatic stress disorder (PTSD) was best selected by the presence of beta spindles, global/focal slowings, and/or paroxysms, rather than the primary symptoms or Diagnostic and Statistical Manual (DSM) diagnostic labels. These recommendations fall under the rubric of "Personalized Medicine" based on phenotypic biomarkers and must be facilitated by qEEG analysts in conjunction with the prescribing physician or other professionals. It is noteworthy to mention that the general medical use of genotypic and phenotypic biomarkers to better select treatment for an individual is predicted to become a $500 billion market by 2027 (Mehra, 2022). Success in oncology, cardiology, and neurology will likely influence the pursuit in other fields, such as mental health. Journals such as *Clinical EEG and Neuroscience* have taken notice, devoting an upcoming special issue based on work in this area presented at this year's International Pharmaco-EEG Society Annual Meeting.

Ron Swatzyna strongly agreed with these observations. In reviewing thousands of EEGs from his own clients with mental health diagnoses, he identified four main neurobiomarkers that account for most medication failure: encephalopathy, focal slowing, beta spindles, and transient discharges. One or more of these neurobiomarkers were identified in all refractory cases. Swatzyna et al. (2014) found that multiple neurobiomarkers were associated with more psychopathology, diagnoses, and medication prescribed.

There is a growing enthusiasm in the qEEG community that replacing the traditional DSM and International Classification of Diseases (ICD) diagnostic criteria that psychiatrists rely on for medication prescriptions with EEG biomarkers would result in improved outcomes. In February, World Psychiatry (Leichsenring et al., 2022) published a review and meta-analytic evaluation of recent meta-analyses highlighting the limited gain for both psychotherapies and pharmacotherapies over placebo or treatment as usual (TAU). The highest impact psychiatric journal is suggesting that a paradigm shift is required to achieve further progress. Now is our time to provide research supporting the use of EEG and qEEG for medication selection especially in refractory cases.

Partnering with Prescribers

One of the main frustrations for mental health practitioners trained in qEEG is clearing the way for open and respectful communication with prescribers, enabling information to be shared within the boundaries of an individual's licensure and training.

As qEEG clinicians, unless we are the prescribing professional, we would be creating legal liability for ourselves and may do harm to our patients or clients if we encouraged or even agreed with a patient to taper off medications before a qEEG or because of the findings in a qEEG. We must therefore refer the patient or client to their prescribing professional or have a conversation with the said person before any medication changes are made under the authorization of the prescribing professional.

The question arose whether members of this community had an *ethical* responsibility to bring this information to a prescriber if a client's EEG appeared to be subject to increasing instability due to a choice of medication. For example, some medications exacerbate seizure discharges or even trigger outright seizures in some patients who have isolated epileptiform discharges (IEDs) in their brains, which can only be detected with an EEG. Psychiatrists have no way of knowing about the presence of silent discharges in the brain and therefore might appreciate that information to make an informed decision regarding medication choice. This situation becomes more complicated as several neurobiomarkers, such as positive identification of IEDs, can only legally be reported by a neurologist, who will rarely get involved unless there is clear evidence of epileptiform morphology in the EEG or a report of an outright seizure.

Participants developed a list of ideas to help improve the communication process between providers:

1) Providing the prescriber with a list of peer-reviewed research articles to back up the EEG findings. How can our professional organizations help provide an up-to-date portal of this kind of information, so that each individual clinician

need not have to continuously update their own corpus of new research in this area?

2) Networking with client's prescribing practitioners by offering a professional courtesy assessment to the practitioner or their clinical team. Offering to tell them about their own brain will engender some personal interest and help them to develop confidence in your clinical skills.
3) Creating a one-page informational sheet to be used as a handout to introduce prescribers to EEG-guided medication selection. Can one of our professional organizations produce an authoritative version that can be used by the entire community?

Successfully collaborating with prescribers to build an effective and knowledgeable team is essential to the continuity of care for clients. For qEEG practitioners who are not licensed as psychiatrists, it is important to work with a knowledgeable, licensed prescriber. One way to network with local psychiatrists is to join any regional listserv of mental health professionals. There are often group meetups for providers soliciting referrals. One suggestion was to consider hosting a meetup and to post it on the listserv.

Psychiatrists have a basic understanding of the EEG, though they don't often get as much EEG training in school as many would like. Those who have received training most certainly would not have had exposure to the application of EEG to medication selection, as this is a relatively new specialty. It is recommended that qEEG analysts develop a relationship with a prescribing professional. As the relationship grows, it is important to make this relationship a reciprocal one: the psychiatrist will have his or her own informed reasoning for choosing a medication. The group recommended sharing what we know and learning how the prescribing professional formulates a decision. Collaboration is encouraged.

Technological Advancements

Three talks introduced new technologies in the form of algorithms or new hardware. Peter Gast and Michael Villanueva gave a joint presentation entitled "Art of Dimension" where they demonstrated motivations for alternative representations of data for improved insight and analysis. Rogene Eichler West presented an introduction to a kind of analysis that has become pervasive in many fields, machine learning, in her talk, "Machine Learning Algorithms for the Identification of Signals in the Noise." Finally, attendees received a demonstration of the latest innovations in wearable EEG during Seung Wan Kan and Daekeun Kim's talk, "Vision of New Decade: Building a digital mental care platform with qEEG brain mapping and neuromodulation." For those new to the field, Dave Siever reviewed the power of audio-visual entrainment during his talk, "Physiology and Clinical Applications of Audio-Visual Entrainment Technology" (Siever & Collura, 2017).

The Art of Dimension

Gast (also known by the name Makoto Miyakoshi) is a researcher and programmer who plays an active advisory role in the EEGLAB community. At the start of his presentation, Gast set the tone for his talk using a video of the "Ambiguous Cylinder Illusion," a popular YouTube video and winner of the 2016 Best Illusion of The Year Contest. The message was that our perception of data is dependent on how it is displayed. For example, a common practice in clinical EEG is to collapse the time dimension and represent the data as power spectral density averages. Although this method of reducing dimensionality makes the ensuing information simpler and quicker to view, it also can create false impressions about the nature of the EEG and the salient information contained therein.

The importance of the transient and intermittent features of EEG was illustrated by a second analogy, first by playing an audio clip of a popular Beatles song normally and then by compressing the entire song into a couple of seconds; averaging EEG power from a multiminute recording into a single image is similarly reductive. Gast demonstrated an alternative way of viewing the data known as a peak-power frequency ribbon analysis, a graphical representation that reveals a low-dimensional representation of stationary power distribution in the time domain. Visually, this appeared as a shorthand for viewing the whole length of a multiminute EEG recording while simultaneously retaining important temporal features.

Michael Villanueva, a clinical psychologist and neurofeedback practitioner brought the concepts introduced by Gast into a practical clinical framework. Most EEG analysis software relies on spectral averages to create comparisons with normative databases. But given the transient nature of EEG, as demonstrated in the audio analogy, we may grow dissatisfied with the lack of any temporal resolution in such comparisons. Furthermore, although *p*-values associated with differences in spectral power density between an individual and the database may help identify areas of interest, significance (*p*-values) may not be relevant given the intended application of neurofeedback.

As a more appropriate method for comparison of either pre–post or individual-database EEG time series data, Villanueva proposed the application of Cohen's D, or the standardized mean difference. Villanueva announced the future release of a "d-Matrix" toolbox within EEGLAB, created by Gast, that can apply the Cohen's *d* effect size calculation to a set of EEG time series. This comparison allows for the retention of the time dimension and "provides a beautiful spatial overview of cortical power levels," giving a unique window into the hitherto unseen effects engendered by neurofeedback.

An Introduction to Machine Learning

Rogene Eichler West is a research scientist, former clinician, and qEEG-diplomate, who provided a broad-reaching introduction to machine learning and its potential applications for EEG. She pointed out that machine learning approaches have begun to surpass the use of standard statistics in discovering and classifying high-dimensional relationships in data features. Because of this ubiquity, it will become important for clinicians to learn to skeptically read the machine learning literature rather than to accept the conclusions at face value.

Machine learning comprises a variety of computational approaches to data that vary in their complexity, data requirements, computing power consumption, and use-case considerations. Machine learning algorithms are divided into supervised learning, for which examples must be provided to learn from, and unsupervised learning, where the data is clustered based on some distance measure. The simplest of these methods, such as simple linear regressions and decision trees are already intuitively used by most people.

The use of more advanced methods hold promise for identifying yet-unknown features or correlates of EEG signals, but also present unique challenges. One such challenge is the time-consuming step of generating labeled data for supervised algorithms by humans with expertise in the domain of relevant knowledge. A second challenge is to develop strategies for handling imbalanced datasets, which frequently occur in clinical data where the number of normal examples to learn from outnumber the clinically significant ones. A third challenge is to develop a feature space to differentiate salient classes.

Early machine learning applications, such as neural networks, were motivated by an idealized model of how collections of neurons worked together to solve problems. Consequently, language developed such that terms like neurons, synaptic weights, channels, and epochs hold a different meaning for data scientists than EEG clinicians and researchers—and this can cause confusion.

Examples were given for how to critically read a study. For example, a valid result should have a separation between training data, cross-validation data, and testing data, lest performance measures such as accuracy are reported as inappropriately high.

Machine learning algorithms are available for development in major programming languages and on platforms such as EEGLAB, as are a growing number of open-access EEG data collections, such as TDBRAIN released by Martijn Arns' BrainClinic's Foundation.

The Next Generation of iMediSync's Digital Mental Care Platform

Seung Wan Kan and Daekeun Kim provided their vision for the next 10 years of iMediSync and a summary of the company's history since its founding in 2012. Their goal was to create an integrated and user-friendly EEG system so that more health professionals could make use of EEG data in their clinical practice. iMediSync has since developed an age-and-sex-matched qEEG normative database, iSyncBrain, which is available as a paid service for processing EEG data from many standard EEG devices. The iSyncBrain processing software can rapidly and automatically artifact the data using an AI-based algorithm and then compile the results into a downloadable report. iMediSync has also developed a machine learning algorithm for the detection of cognitive impairment, along the spectrum of Alzheimer's disease disorders.

Participants had the opportunity to try their new integrated EEG helmet called iSyncWave. This helmet integrates high-impedance dry sensors, an EEG amplifier, NIR-LEDs for photobiomodulation, and a wireless communication module so that data can be recorded directly to a Samsung tablet and then uploaded to the iSyncBrain server for processing. The collection software automates the recording of 2 min of eyes open and then 2 min of eyes-closed data. While several devices are currently available on the market to deliver transcranial photobiomodulation (tPBM), the iMediSync team proposes to turn the integration of

EEG and tPBM into a viable telemedicine diagnostic and personalized therapeutic device for mild cognitive decline (MCI) and dementia.

iMediSync hopes to expand the scope of their AI-based EEG analysis tools and to bring their vertically integrated EEG solutions to diverse health professionals. They reported working with medical boards to document their processes to be considered for insurance billing.

Emerging Concerns

The impact of environmental factors on brain health, such as electromagnetic radiation (EMF) and toxins in the air, water, and food, is of growing concern to a great number of attendees. There is a realization that more attention must be paid to helping clients identify and avoid exposure to these factors. However, most clinicians felt that they needed to be better educated on this topic themselves. Two talks highlighted issues to consider. Neurologist and Pediatric Epileptologist Rusty Turner spoke to the harm that our radio-enabled world is silently causing in his talk, “EMR: The Most Unrecognized Influence on EEG.” Chiropractic neurologist and former professor of neurochemistry Michael Pierce then presented on the biochemistry underlying several neurodegenerative processes in his talk, “Toxic, Metabolic and EMF Effects on EEG and the Brain.”

Toxins All Around

Dr. Turner illustrated the longstanding, yet unrecognized, adverse effects of artificial environmental electromagnetic radiation (EMR) on all life, specifically human life, and the value that careful EEG review has in demonstrating external effects of environmental EMR. Exponentially increasing EMR exposure of humans worldwide is essentially unrecognized despite its pervasive, albeit invisible, presence throughout the world. Clues arise in careful review of the EEG data being collected every day by clinicians in our field. This EMR exposure consists of increasing use of wireless networks, cell phones and towers, etc. and is demonstrated from a multitude of publications over the past 60 years. Adverse effects are also seen on genetic and reproductive health and especially on the developing nervous system. The developing nervous system is more susceptible to EMR, and neurodevelopmental anomalies and seizures or epilepsy are increasingly associated with such exposures—both pre- and postnatally. They are also manifested throughout the lifespan as is being increasingly seen in mental ill-health disorders worldwide. Turner recommended all providers of healthcare, including those of us in the neuroregulation or neuromodulation field follow the Precautionary Principle (*taking preventative action in the face of uncertain and/or conflicting scientific evidence*) given the substantial literature involving human or animal studies and worldwide deployment of wireless technology. Evidence exists that worldwide, pervasive, increasing EMR exposure results in progressive manifestation of seizures or epilepsy, disorders of the developing nervous system, sleep, and mental health disorders, as well as systemic disorders involving the cardiovascular and GI systems.

Dr. Pierce voiced similar concerns about the toxic environment in which we find ourselves immersed. He was joined by Jay Gunkelman in sharing anecdotes concerning the lack of protections offered by the regulatory process, and he further raised concerns with regulatory peer review citing a body of work by John Ioannidis. While encouraging referrals when alarming features in an EEG are observed, he emphasized that most indications of toxins or metabolic disorders in EEG are more subtle and not in a one-to-one correlation with specific insults due to biochemical individuality and underlying endophenotypes.

Pierce enumerated a plethora of environmental stressors such as chronic exposure to heavy metals, molds, industrial solvents, and agrichemicals. He pointed out unexpected sources of exposure, as well as the deleterious synergistic impact at the blood-brain barrier when chemical and EMR exposures are combined. The clinical implications are often observed as forms of insulin resistance, lectin intolerance, thyroid conditions, adrenal or sex hormone imbalances, inflammation and oxidative stress, alterations in methylation pathways, and anemias. He emphasized the utility of comprehensive history collection and lab testing as well as the efficacy of detoxification through supplementation and therapeutic diets.

Questions Directing a Course of Action

In the follow-up discussions, many echoed the need to form the same kinds of partnerships with functional medicine specialists as with medication prescribers. However, what differs with emerging concerns is that by their very definition, the clinical path forward for diagnosis and treatment is not well defined.

- How do we handle potential diagnostic findings in our qEEG recordings that are affecting the

brain and may be due to causative factors that are clearly not well defined (e.g., EMF exposure)? All too often, these findings and symptoms are medically dismissed by a client's general practitioner or specialist.
- How and to whom do we refer in such cases? What is the process of proper referral and tracking? Who coordinates overall care? When is it time for second opinions? How do we improve patient education beyond simple referrals?
- How do we resolve difference between patient goals and clinician priorities in EMF and toxic or environmental cases, where the problems may not be recognized as orthodox diagnoses or treatments, despite measurable contribution to EEG changes?
- How do we prioritize issues, symptoms, and mechanisms of injury when so much is unknown regarding continuing environmental threats to the patient?
- There is often no standard of care for environmental exposures despite recognizing their long-term impact on brain function. How do we approach the relative quality of evidence in our cases? How do we report these outcome measures in case study data?
- How do we relate EEG findings to symptoms or lab findings when they are stand-alone indicators of dysfunction? Can EEG findings be integrated into a comprehensive brain diagnosis that has room to include emerging environmental factors?

A New Normal for How the Body Keeps the Score

As environmental toxins exponentially accumulate, changes are happening in the landscape of disease that we appear to be accepting as "normal." Insidiously, this change is happening sufficiently slowly and invisibly that it is perceived to be unremarkable. Despite the concern for an impact on brain health being called out at this year's meeting of the American Neurological Association (Lakhani, 2022), traditional assessments and interventions remain insufficient to help our patients and clients identify causes and implement solutions to regain their health.

EEG practitioners are often on the front lines of working with persons suffering from an environmental exposure because the symptoms often have a mental health component such as depression, anxiety, or brain fog. While we come from a variety of disciplines to study brain dysregulation and promote healing, there is an overwhelming convergence in the stories we share about the health challenges of our clients and the studies we share with each other validating our observations (Ventriglio et al., 2021).

When van der Kolk (1994) first reminded us that the body keeps the score, his intention was to bring our attention to the ways that dysregulation lives on invisibly in the body after a trauma. We are waking up to the observation that that scoreboard also reflects a game played against the very air we breathe and water we drink. While knowledge of a trauma provided us with a therapeutic direction to try to even the score, our community is somewhat lost in the ability to offer recommendations to these new environmental threats. This is especially true in that most mental health clinicians have no training in the ability to recognize, or even screen for, underlying metabolic and toxic impacts on mental health. Even if we suspect an underlying issue, we find ourselves isolated from a network of providers with whom we might work to find the root cause. As a first step, we propose working more closely with the functional medicine community to develop standard screenings from which we might refer and coordinate care.

Standardized Intakes and a Model of Client Care

It is important for the neurofeedback community to create a standard system for screening the most common physical issues that need to be addressed as a referral, prior to neurofeedback treatment. For example, most experienced practitioners understand to refer out a client with untreated thyroid issues, sleep apnea, mold poisoning, toxic exposure, or inflammation due to undiagnosed causes prior to beginning neurofeedback. Yet, this knowledge is not part of our standardized training, and what constitutes a thorough clinical and medical history widely varies. It is also useful for greater training on physical symptoms and health issues to probe when a particular EEG pattern is observed, such as low power EEG indicating toxic or metabolic issues and beta spindling possibly indicating inflammation.

This system of intake and screening should be supported by models of client care that include what types of medical professionals to refer to for various physical issues and the proper process for initiating these referrals. This intake should include chronological health histories, developmental milestones, review of organ systems, metabolic symptom questionnaires, and questions related to infections and potential toxic exposures. Such a model should address when and how to obtain second opinions or consultation for routine laboratory assessments and who is responsible for overseeing this process to facilitate the highest quality of care.

The resulting system needs to be easy and supportive for clients. Already, new clients are subjected to an increasing number of questions and assessment forms to become established at a practice. Complicated histories place a burden on the client's capacity to express the most urgent priorities in an uncomplicated form. Since the outbreak of the COVID-19 pandemic, and with the growing impact of environmental toxins, patients are presenting with more complex symptoms and histories. Many patients are already feeling lost and unsupported by healthcare practitioners.

Clients who are already overwhelmed may also find it difficult to prioritize the order of potential environmental threats to eliminate or the order in which to pursue health interventions. Expectations should be set so that they understand that attempts to address their symptoms may require a degree of trial and error. An effective model of care might offer a decision tree with suggested priorities so that the interventions most likely to yield success are tried first.

Finally, there is a pressing need to develop and institute pertinent education for clients to help them understand the symptoms associated with various medical issues and the appropriate process to follow in navigating their care. While education already exists regarding conventional health practices, such as diet and exercise and the effects of recreational drug use that are foundational to good neurofeedback treatment outcomes, there is also a huge amount of conflicting information that causes confusion and limits compliance. Unambiguous, authoritative, age-appropriate, and readily available education modules might be developed by experts in relevant disciplines within our community and made available through professional organizations.

The Standard of Care: Partnerships With Complementary and Alternative Medicine

Standards of care in both physical medicine and in mental health practice lie at the nexus between state regulatory boards and professional association consensus statements. A growing demand for science-based and brain-based measures of mental health and brain tissue physiology is placing pressure on private therapists to integrate complementary and alternative medicine (CAM) reports and methods into their care. Some of these methods are within their scope of practice and some of them are outside their scope, requiring interdisciplinary communication and integration with multiple disciplines. A basic science understanding of these mechanisms is demanded by patient populations and families, and communication skills across disciplines are required more and more. Political forces and captured regulatory agencies add more complexity to the pressures on mental health clinicians as they serve patients and seek root causes. Many of the modes of physiologic and metabolic brain investigation are science-based but have not yet developed validation, yet clients seek them with great interest, and some doctors discharge patients for the infraction of using CAM services. As clinical science marches forward, there is a structural gap that will always exist between early development and validation of methods, within which lie both risky and unproven methods and at the same time contain what will become known as some of the safest, most sustainable, and effective methods ever discovered. A balanced approach to this unknown gap is required without either discarding promising methods or accepting methods without critical thinking. New clinical ideas must be given a clinical chance to blossom and financial perverse incentives must be transparent while these studies are produced.

Since normal brain function depends on normal neuronal metabolism, which is related to systemic homeostasis of metabolites, such as glucose, electrolytes, amino acids, and ammonia (Lin, 2005) we recommend a more comprehensive laboratory panel which may include CBC, comprehensive metabolic panel, fasting glucose, HbA1C, iron, ferritin, TIBC, B12, MMA, folate, homocysteine, CRP, fibrinogen, magnesium, thyroid panel, TGF-1, heavy metal panels (serum and urine), and other infectious and/or autoimmune markers that are relevant for each patient. If a healthcare provider does not have the scope of practice to order laboratory tests, it is recommended that they should collaborate with a health professional who can provide these services.

Growing Community Prestige Through Research

Ron Swatzyna and Mark Jones discussed ways to increase research on neurofeedback by gathering data from clinician's private practices and collaborating in sharing data in ways that create substantial multisite analyses. Swatzyna described ways he has amassed a large database of patients through his own practice, including EEG analysis and biomarkers, medications, and diagnoses. He has published on Pharmaco-EEG topics and continues to promote research through an institutional review board (IRB) he helped create (through the local Sigma Xi chapter at Rice University and the Texas Medical Center). Jones shared his vision as the current president of the International Society for NeuroRegulation and Research (ISNR) for “the R in ISNR,” a collaborative citizen-science approach to sharing data from across multiple practices and agencies to create an open-source database in which participants can upload their respective data sets and do analysis via queries of the online database, possibly housed at the university where he teaches. Swatzyna and Jones facilitated discussion on the various nuances of research designs, IRB requirements, and eventually establishing standardized assessments and protocols related to specific syndromes to facilitate robust statistical analysis.

Generating further research will significantly contribute to our understanding of mental health disorders, and the unfolding understanding of individualized neurobiomarkers is key to improving pharmacotherapy outcomes. Ron Swatzyna stressed the need and benefits of partnering with editors that are versed in qEEG and neurofeedback and who are open to cross-publishing in journals such as the *World Psychiatry Journal* such that both communities are being exposed to cross-cutting discoveries.

While many participants expressed a desire to become involved with research, many expressed a sense of being overwhelmed by how to get started. Ours is a heterogeneous community, and just as our backgrounds and licenses differ, so do our experiences participating in research. Many participants have never designed an experiment, performed (or evaluated) a statistical analysis, or published research. As with many of the other themes above, many expressed a desire for additional educational opportunities.

History of the Suisun Summit

The Suisun Summit was born out of two events that took place in 2019. The first was the "Back to Basics" workshop that was held in Suisun at the Hampton Inn from Feb 6–10, 2019, featuring Jay Gunkelman’s teachings on reading and interpreting the raw EEG. In the summer of 2018, it had become apparent that Jay’s health would no longer allow him to travel to professional meetings. Mary Tracy and Brian Judd suggested to Jay that a conference be organized near his home, and he was thrilled to accept the offer. They invited 20 people who were interested in the visual interpretation of the EEG with higher order processing using WinEEG. Tracy sincerely wanted people to begin hearing Gunkelman teach in person at the most fundamental level. She believed that the international EEG community would benefit from refocusing on the interpretation of the raw EEG instead of relying on brain mapping alone to tell the story. Linda Walker and Gunkelman co-taught this 5-day event. This very successful workshop spawned another workshop, Onto Innovations, at the same location in October of 2019, which Cindy Reynolds and Candia Smith facilitated. Other presenters at this meeting included our dear, late friend Harry Kerasidis, as well as Michael Villanueva and his colleague Clement Lee.

Another colleague who we lost in July of 2021, Joe Castellano, attended this second event and took the name to begin the online Onto Innovations Study Group. The group received Gunkelman's BSI (Brain Science International) collection of papers and talks, which, in addition to the classic textbook, *Niedermeyer’s Encephalography*, became the basis for biweekly meetings (Schomer & da Silva, 2011). Topics evolved to include content beyond these resources, but with a continued focus on the intersection between neuroscience and mental health. Membership required making a good faith effort to attend as many sessions as possible to support presentations by one’s colleagues, but also that members commit to making their own presentation to the group annually. Group members felt the loss of Castellano deeply and are grateful to Dave Siever and Mary Tracy for picking up the reins to keep the group moving forward.

The Suisun Summit was originally intended to be an opportunity for members of the Onto Innovations Study Group, who had grown close through their biweekly online meetings during the pandemic, to finally meet without a computer screen between them. Word of mouth grew such that the number of participants soon hit the limit of the seating capacity of the largest event venue in Suisun City.

A delightful and comradery-building aspect of this meeting was that there were opportunities to get to know colleagues through their artistic gifts. Participants enjoys musical offerings by Dave Siever, Rusty Turner, Tony Jackson, and Rebekah Walker.

The Suisun Summit will take place again in 2023. While the number of in-person attendees must remained capped at 70, the new planning committee will arrange to livestream the event.

Conclusion

Several themes emerged in talks directly or in discussion groups at the Suisun Summit 2022: qEEG Reading Skills; Medication Effects and Pharmaco-EEG; Technological Advancement; Emerging Concerns; and Growing Community Prestige Through Research. Participants were asked to summarize what they believed to be the most important messages from the event to share with colleagues who were not in attendance, resulting in this review. Cross-cutting all the themes was a desire for higher quality, standardized education that provides depth as well as breadth, and models for clinical care that provided closer relationships between qEEG analysts, medication prescribers, and functional medicine specialists.

We are uniquely poised as a profession of qEEG analysts to offer services to the public that are not available with any other medical or nonmedical practitioner. For example, our ability to identify and locate isolated interictal discharges in the brain during routine EEG screening for seemingly unrelated presenting issues allows us to make referrals to neurologists or epileptologists for proper medical diagnosis and perhaps medication management. Indeed, the biomarkers and EEG features that are correlated with numerous physical (e.g., cerebral ischemia, traumatic brain injury [TBI], stroke, etc.) and neuropsychiatric (e.g., migraine headaches, ADHD, affective dysregulation, ASD, etc.) problems are routinely identified in our processing of the EEG and we can develop training protocols or neuromodulatory treatment to address these symptoms.

In many ways, our dependence on maps and their automated interpretation have already taken away much of our power as clinicians, leaving us in the role of technicians when it comes to the potential power of qEEG in our practices (see addendum “Neidermeyer’s Lament.”). Future developments in the application of AI algorithms which can correlate EEG and qEEG patterns, symptom questionnaires and laboratory biomarkers have the potential of becoming a more sensitive and specific method for identifying these disorders. Yet as clinicians, we need a greater degree of standardized training to possess knowledge of these indicators for ourselves, to verify and corroborate the findings.

United in our respect across the professions and our sense of comradery, achieving our common goal of restoring and promoting brain health, we call upon each other and our professional organizations to inspire the development of higher quality standardized education in qEEG reading skills, the development of an authoritative compendium of clinical correlates for mental health and emerging concerns, and models for clinical care that provided closer relationships to prescribers and functional medicine specialists.

Acknowledgements

The authors would like to thank the following people for their participation in discussions, for providing edits to drafts, or for their endorsement of this summary of the Suisun Summit: Rebekah Walker, Nathalie Clerc, Dave Siever, David C. Johansson

Author Declarations

None of the authors received any financial support, hold a financial interest, or have any conflicts of interest to disclose.

References

Arns, M., Gunkelman, J., Breteler, M., & Spronk, D. (2008). EEG phenotypes predict treatment outcome to stimulants in children with ADHD. *Journal of Integrative Neuroscience*, *7*(03), 421–438. https://doi.org/10.1142/s0219635208001897

Byrom, B., McCarthy, M., Schueler, P. & Muehlhauser, W. (2018). Brain monitoring devices in neuroscience clinical research: The potential of remote monitoring using sensors, wearables, and mobile devices. *Clinical Pharmacology & Therapeutics*, *104*(1), 59–71. https://doi.org/10.1002/cpt.1077

Gunkelman, J. (2014). Medication prediction with electroencephalography phenotypes and biomarkers. *Biofeedback*, *42*(2), 68–73. https://doi.org/10.5298/1081-5937-42.2.03

Lakhani, N. (2022, October 23). *Exposure to environmental toxins may be root of rise in neurological disorders*. The Guardian. https://www.theguardian.com/us-news/2022/oct/23/environmental-toxins-neurological-disorders-parkinsons-alzheimers

Leichsenring, F, Steinert, C, Rabung, S, & Ioannidis, J. P. A. (2022). The efficacy of psychotherapies for mental disorders in adults: An umbrella review and meta-analytic evaluation of recent meta-analyses. *World Psychiatry*, *21*(1), 133–145. https://doi.org/10.1002/wps.20941

Lin, C.-C. K. (2005). EEG manifestations in metabolic encephalopathy. *Acta Neurologica Taiwanica*, *14*(3), 151–161.

Loo, S. K., Bilder, R. M., Cho, A. L., Sturm, A., Cowen, J., Walshaw, P., Levitt, J., Del’Homme, M., Piacentini, J., McGough, J. J., & McCracken, J. T. (2016). Effects of d-methylphenidate, guanfacine, and their combination on electroencephalogram resting state spectral power in attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, *55*(8), 674–682. https://doi.org/10.1016/j.jaac.2016.04.020

Mehra, A. (2022, November 11). *Personalized medicine industry sizeworth $~500 billion by 2027*. Market Research Firm. https://www.marketsandmarkets.com/PressReleases/personalizedmedicine

Nunez, P. L., & Srinivasan R. (2006). *Electric fields of the Brain: The Neurophysics of EEG*. Oxford University Press.

Schomer, D. L., & da Silva, F. H. L. (Eds.) (2011). *Niedermeyer's electroencephalography: Basic principles, clinical applications, and related fields* (6th ed.). Lippincott Williams & Wilkins.

Siever, D., & Collura, T. (2017). Chapter 3 - Audio–visual entrainment: Physiological mechanisms and clinical outcomes. In J. R. Evans, & R. P. Turner, *Rhythmic stimulation procedures in neuromodulation* (pp. 51–95). Academic Press. https://doi.org/10.1016/b978-0-12-803726-3.00003-1

Swatzyna, R. J., Kozlowski, G. P., & Tarnow, J. D. (2015). Pharmaco-EEG: A study of individualized medicine in clinical practice. *Clinical EEG and Neuroscience*, *46*(3), 192–196. https://doi.org/10.1177/1550059414556120

Swatzyna, R. J., Tarnow, J. D., Tannous, J. D., Pillai, V., Schieszler, C., & Kozlowski, G. P. (2014). EEG/QEEG technology identifies neurobiomarkers critical to medication selection and treatment in refractory cases. *Journal of Psychology & Clinical Psychiatry, 1*(7), 00046. https://doi.org/10.15406/jpcpy.2014.01.00046

Van der Kolk, B. A. (1994). The body keeps the score: Memory and the evolving psychobiology of posttraumatic stress. *Harvard Review of Psychiatry*, *1*(5), 253–265. https://doi.org/10.3109/10673229409017088

Ventriglio, A., Bellomo, A., di Gioia, I., Di Sabatino, D., Favale, D., De Berardis, D., & Cianconi, P. (2021). Environmental pollution and mental health: A narrative review of literature. *CNS Spectrums*, *26*(1), 51–61. https://doi.org/10.1017/s1092852920001303

Weber, D., McCarthy, D., & Pathmanathan, J. (2016). An effective automated method for teaching EEG interpretation to neurology residents. *Seizure*, *40*, 10–12. https://doi.org/10.1016/j.seizure.2016.05.009

Received: December 31, 2022
Accepted: January 23, 2023
Published: March 30, 2023

Addendum A

Niedermeyer’s Lament: Toward a Fundamental Understanding of the EEG

A Note on the Examination of Contemporary Practices of EEG Acquisition, Processing, and the Neuropsychiatric Interpretation of Brain Activity

Mary L. Tracy, MA PhD MFT BCN QEEG-D
Davis, CA 95616

“*A certain malaise has inched its way into the hearts of thousands of electroencephalographers who have started to feel the grip of stagnation … the feeling of doing pragmatically useful work with an ill-understood method has been depressing to many workers in the field ... A more real danger, perhaps, is presented by the poorly trained colleagues who are tarnishing the image of EEG ... In these times of challenge, a review of the state-of-affairs in EEG seems to be appropriate.”* Ernst Niedermeyer, Spring, 1981.

Ernst Niedermeyer wrote these words in a preface to his tome on the basic principles, clinical applications, and related fields of electroencephalography. Although written in 1981, these excerpts from *Niedermeyer’s Electroencephalography* are as fresh and salient to our current state of affairs in EEG as they were in the era that he composed them. Today, rapidly expanding technological advances and automated algorithms for higher order statistical processing of EEG, along with the easily produced, spectacular neuroimaging displays are replacing the considerations of the raw EEG that are at the basis of these transformations. Well-intentioned students and professionals who are excited about the prospects of a career in EEG assessment and neurofeedback are becoming fully armed with EEG software platforms that promise an easy solution to the detection of dysregulation in the brain, replacing the otherwise difficult and tedious undertaking of a basic education in electroencephalographic principles. There is a clear danger in putting this technology into the hands of licensed clinicians and researchers who are undereducated in the identification of meaningful electroencephalographic features and the functional association of raw EEG findings to psychiatric and behavioral symptoms. Intelligent and curious students who expect that higher-order software programs for EEG processing alone will rescue them from the arduous task of truly understanding how to both detect and interpret basic EEG morphology and features will become quickly disillusioned when they realize that they do not know how to interpret the results, no matter how spectacular the displays.

To further complicate this picture, the market is being flooded with low-cost gadgetry that claims to reliably record EEG and facilitate neurofeedback training without any user access to the specifications, quality, or reliability of these devices. The global connected health and wellness devices market was estimated to be $123.2 billion in 2015 and is expected to reach $612.0 billion by 2024 (Byrom et al., 2018). Especially in this time of global pandemic, neurofeedback practitioners are desperate to employ remote training using inexpensive EEG technology that has not been rigorously tested or undergone clinical trials. There exists a large gap, some would say a veritable chasm, between classically trained electroencephalographers and contemporary consumers of novel EEG systems who have little understanding or appreciation of the origin, meaning, and functional significance of the EEG. Even medically trained neurologists, who typically have about 6 months of training in EEG interpretation, generally use visual examination of the EEG solely for the identification of epileptic features. Epileptologists and electroencephalographers have at least 1.5 years of specialized training in the identification of abnormal features in the EEG as they pertain to seizure disorders, but, like neurologists, they are unlikely to refer patients to neurofeedback specialists as there is little understanding in the medical community of the benefit of neurofeedback for training cortical networks in epileptic disorders. In fact, there is a vast literature on both the analysis of raw EEG and the neuropsychiatric correlates of EEG phenomena, as well as the clinical application to neurofeedback and neuromodulatory training that is still virtually unknown in the medical establishment.

A part of Niedermeyer’s lament about the underutilization of EEG addressed the phenomenal “progress of the new methods of structural diagnosis.” EEG has been relegated to an antiquated status as more expensive imaging methods like MRI, CT, PET, and SPECT scans took precedence in the medical analysis of brain health beginning in 1977. The problem with this is that imaging methods reveal structural detail in the brain *but have no bearing on functional relationships of brain networks*, which can be beautifully elucidated with EEG analysis with good temporal resolution. This leaves the EEG, which is a superior tool in describing the functional significance of psychiatric symptoms, to an underutilized realm that is poorly understood by the practitioners who could benefit from it the most.

Indeed, there is no real occupational classification for the individual who has mastered traditional electroencephalographic assessment as it applies to the interpretation of functional localization of patient symptoms and behaviors in the brain. They are not "neurophysiologists," nor "psychophysiologists." Neither are they purely electroencephalographers. A more precise terminology for the work and skill base of the EEG technician who interprets psychiatric symptoms with a fundamental understanding of the EEG would be "neuropsychiatric or neuropsychological electroencephalographer" (personal communication, Jay Gunkelman August 2020). This occupational classification, used in a professional context, has yet to be introduced to those who practice EEG interpretation or who use EEG as a foundation for developing neurofeedback and neuromodulatory protocols for optimizing brain function.

The purpose of this note is to bring attention to the "pragmatically useful work" of raw EEG interpretation as a foundational and practical platform for examining the validity and application of modern EEG technology to the clinical interpretation of neurological and psychiatric phenomena. It is time to explore the possibility of introducing and training individuals in the extended neuroscience community in the occupation of "neuropsychiatric electroencephalography," beginning with a rigorous educational foundation in the instrumentation and electronics that support EEG recordings, as well as raw EEG interpretation. Without this foundation, the necessary tools for the interpretation of the research and clinical literature in the field are questionable, and the meaning derived from such explorations will only further muddy the waters of this "pragmatically useful work."

Addendum B

Abstracts - Suisun Summit 2022

Suisun Summit 2022, October 12–16, Suisun, California

Expect the Unexpected: Epilepsy and the Foundation of Neurofeedback

Jay Gunkelman

In the beginning of the field of neurofeedback (NF) there were those doing “state-based” EEG training like Joe Kamiya and Elmer Green, and those doing “clinical” work with epilepsy associated largely with Barry Sterman or Nils Birbaumer. ADHD and other applications came later, but the scientific proof level work in epilepsy was quite impressive even in the mid-1970s.

I would dare to suggest that despite the efficacy proofs, the bulk of the NF practitioners today do not work with epilepsy as a primary indication for clinical work—at least not knowingly. However, approximately 25% of those with ADHD and from 60% of those with ASD have “unexpected” epileptiform discharges or paroxysms, meaning there are clients with these patterns who need our help. There are many thousands of clients with intractable epilepsy and thousands more with unexpected epileptiform discharges.

A recent series of severe intractable epileptic cases will be used to illustrate the life-changing nature of applying NF to these cases. Very current publications on the efficacy of treating psychiatric clients who have epileptiform activity, but no seizure history will be shared, as will publications challenging the standard of practice in psychiatry in treating these clients without reviewing the EEG.

A plea from a mother whose daughter was successfully treated for intractable epilepsy asking the field to provide access to far more practitioners who are willing to accept these cases will be shared.

Support for Clinicians to Design Research Projects and Gather Data in Their Private Practices

Moderators: *Ron Swatzyna and Mark Jones*

Clinical research is valuable and necessary. Innovative therapeutic approaches need proof of concept studies which are vital for our field to grow. However, there is a dearth of case studies and clinical research in general, despite noteworthy findings. This presentation lays the groundwork for making your center a clinical research facility. We start with explaining how to collect and categorize data, writing and publishing case studies, building a research team, securing an institution review board (IRB) approval, data mining, and publishing clinical research. The “old guard” is graying so now is the time to highlight your work, publish your findings, and help our field grow.

Medications and EEG Correlates

Moderator: *Angelika Sadar*
Panel: *Ron Swatzyna, Jay Gunkelman, and Robert ‘Rusty’ Turner*

It is not uncommon that a person seeking an EEG is taking one or more prescribed medications, and maybe taking various nonprescribed substances (including vitamins, supplements, and self-prescribed) as well. For the purposes of this discussion, we will refer to all natural and unnatural products that affect the EEG as substances. As practitioners, we are faced with discussing how substances may affect the EEG, but we may not be licensed to make recommendations. Following the data acquisition, we are faced with discussing the impact of the substances on the readings obtained. Then, we are faced with considering how the ensuing neurofeedback (and other interventions) may impact the effect of the substances. With the knowledge of our panel of experts who can address the impact of substances on the EEG, we will discuss the broader topics related to best practices for working with patients who are utilizing substances of any kind.

Art of Dimension

Michael Villanueva and Peter Gast

In this joint presentation, Michael (Clinical Psychologist) and Peter (EEG researcher/programmer), distill a fundamental "takeaway" from their 7-year-long corroboration while sharing the resulting new analytic tools. Peter will discuss the relationship between dimension reduction and data analysis. Criticizing the widespread overreliance on power spectral density analysis, he will propose two alternative analyses designed to overcome its limitations. The first method, called the peak-power frequency ribbon analysis, is a spectrogram sorted by instantaneous frequencies. The ribbon analysis reveals a low-dimensional representation of stationary power distribution in the time domain. The second method designed to test pre–post EEG data is called the d-Matrix. The d-Matrix implements Cohen's *d* obtained from before–after comparisons visualized in a channel-by-frequency matrix. Starting with the ribbon analysis, Michael will demonstrate how sorting by frequencies gives the clinician a radically different temporal visualization into fluctuations of the alpha band and their relationship to clients' symptomatology. Next, he will compare pre–post EEG data using WinEEG software and then use the same data to demonstrate the d-Matrix (within an EEGLAB environment). Neurofeedback practitioners seek to alter specific frequency ranges by channel location; thus, significance (*p* value) is the least relevant, while effect size is the most pertinent to the client. The d-Matrix provides a beautiful spatial overview of cortical power levels, giving a unique window into the hitherto unseen effects engendered by neurofeedback. In sum, our proposed methods can facilitate an efficient, statistically rigorous neurofeedback clinical practice.

EMR: The Most Unrecognized Influence on EEG

Rusty Turner

The premise for this presentation will be mechanistic, focusing on documented problems related to wireless communications radiation and HEV-blue light from screens, with EEG and qEEG data presented:

(1) cause morphologic changes in erythrocytes including echinocyte and rouleaux formation that can contribute to hypercoagulation
(2) impair microcirculation and reduce erythrocyte and hemoglobin levels exacerbating hypoxia
(3) amplify immune system dysfunction, including immunosuppression, autoimmunity, and hyperinflammation
(4) increase cellular oxidative stress and the production of free radicals resulting in vascular injury and organ damage
(5) increase intracellular Ca2+ essential for viral entry, replication, and release, in addition to promoting proinflammatory pathways; and
(6) worsen neurologic and developmental disorders, heart arrhythmias and cardiac disorders.

Pharmaco-EEG and Phenotypes: The Interface Between Neurology, Psychiatry and Neuromodulation

Ron Swatzyna and Jay Gunkelman

No Abstract Available

Physiology and Clinical Application of Audio-Visual Entrainment Technology

Dave Siever

Since the discovery of photic driving by Adrian and Matthews in 1934, much has been discovered about the benefits of brainwave entrainment (BWE) or audio-visual entrainment (AVE), as it is commonly known today. The brain responds extremely well to stimulation. However, the concept of AVE implies a frequency-following response, where the frequency of the brain synchronizes to the frequency of stimulation. While this is true, frequency-following is the least of what AVE is about.

AVE increases cerebral blood flow, stimulates beneficial neurotransmitters, has profound calming effects on the mind and body, induces a calming meditative mind state, is very effective for teaching heart rate variability (HRV) in highly anxious persons who fail HRV biofeedback training, increases brain lactate and ATP, triggers protective heat-shock protein, excites microglia, improves neuronal efficiency, and excites noninflammatory cytokines, which, in turn, promote microglial phagocytic states, such as IL-6 and IL-4, and increased expression of microglial chemokines, such as M-CSF and MIG.

There have been dozens of AVE devices marketed over the decades, but most do not consider scientifically derived stimulation methods and frequencies used. As a result, AVE has, to some degree, fallen into a New Age type of category. The truth of it is that AVE has strong empirical evidence as to its efficacy in physiological effects and clinical applications.

Research on the effectiveness of AVE in promoting relaxation, cognition, and hypnotic induction, treating ADD, PMS, SAD, PTSD, migraine headache, chronic pain, anxiety, depression, episodic memory, cognitive decline in seniors, and potential for treating early-onset Alzheimer's disease is now available. Recent discoveries have shown AVE to be a powerful means of recovery from traumatic brain injuries (TBI) of a newly identified type, termed thalamocortical disconnect (TCD). The TCD type of TBI is quite common and characterized by general anxiety, obsessive-compulsiveness, and severe insomnia.

Machine Learning Algorithms for the Identification of Signals in the Noise

Rogene Eichler West

Classifying collections of EEG subtypes, such as phenotypes, or identifying particular waveforms, such as a spike and wave, are typically performed visually by an expert. In some research endeavors, it is not clear what characteristics are the most important to look for when labeling each dataset as belonging to a particular clinical group. In situations such as long-term monitoring, it is tedious for a neurologist to observe the data in real time to catch an elusive marker, and therefore this manual process is prone to error. In both of these scenarios, it would be useful to have a software tool perform with similar levels of sensitivity and specificity as an expert. Machine learning approaches on EEG data are beginning to meet this performance criteria. In this talk, the underlying principles behind machine learning algorithms will be presented. Concepts to be explored include supervised versus unsupervised learning; gradient descent and clustering; underfitting and overfitting; evaluating performance; and dealing with small datasets. The goal of this talk is to familiarize clinical professionals with machine learning concepts that they may encounter in the literature, so that they might have confidence when interpreting the conclusions of such studies.

Toxic, Metabolic and EMF Effects on EEG and the Brain

Michael Pierce

This 45-min discussion is a brief survey of the known brainwave changes seen in toxicity, metabolic disorders, and EMF studies with correlation to common exposures. While there is not generally a one-to-one correlation from specific brainwaves or paroxysms to discrete exposures, there are common or global patterns that emerge across a broad range of chemical and electrical exposures, and EMF exposures facilitate barrier disruptions and transfer of metals. Regional brain tissue metabolic vulnerabilities to substances and hypoxia are also surveyed. Significant brainwave findings indicate clinical and lab correlations for specific diagnostic follow up. Discussion of typical cases and citations are included.

Pediatrics and Developmental Trauma in the Brain

Tiff Thompson

No Abstract Available

Vision of New Decade: Building a Digital Mental Health Care Platform With qEEG Brain Mapping and Neuromodulation

Seung Wan Kan and Daekeun Kim

In the last three decades, neurofeedback has evolved from experience-based to quantitative EEG (qEEG)-guided practices that have been applied to various neuropsychiatric disorders. Despite their potential values, neither neurofeedback nor qEEG brain mapping are recognized by conventional medical specialists as legitimate methods for functionally analyzing the brain. During the recent COVID pandemic period, digital healthcare and digital therapeutics attracted attention, especially for mental health purposes. iMediSync Inc. has the vision of contributing to global mental health care and wellness and the evolution of human consciousness using integrative AI mental health care platform services. We have developed four innovative solutions in the qEEG and neuromodulation areas.

First, iMediSync has developed the only sex- and age-matched qEEG normative DB which was initiated at Seoul National University in 2012. Sex differences can affect EEG variance according to development and aging, but there has been no qEEG normative DB differentiating biological sex before iSyncBrain.

Second, there has been a barrier for clinical practitioners to apply qEEG to ordinary practice, as most conventional qEEG analysis software requires manual processing, which takes time and requires experience and knowledge. So, we developed iSyncBrain, a user-friendly, AI-powered, automated EEG signal processing system using SaaS (software as a service).

Third, to expand the application of qEEG to more than functional brain mapping and endophenotyping, and to identify qEEG features as digital biomarkers for the diagnosis of specific mental disorders, we developed qEEG-specialized AI/ML technology and created a qEEG-based algorithm for differential detection of either Alzheimer-induced or non-Alzheimer-induced amnestic mild cognitive impairment (aMCI) from normal aging.

Fourth, brainwave measurement with conventional wet-type EEG sensor systems is not user-friendly and EEG acquisitions cannot easily be accomplished in real-world situations such as school, sports, or military camps. We developed iSyncWave, a wireless, wearable EEG helmet with 19 dry sensors and NIR-LED stimulators, which can both measure EEG and provide neuromodulation. It demonstrates high signal quality and interconnects with the iSyncBrain software platform.

By integrating these four core technologies, iMediSync is opening a new era of qEEG-centered digital mental health care platforms which will support data-driven mental care services either online or offline.

PTSD Biomarkers with ERP and EEG

Jay Gunkelman

Trauma leaves a lasting “mark” in the brain’s function, whether from developmental issues with attachment or changes later in life. This talk will show the relationship between the ERP and EEG findings seen in trauma, with a special focus on the eyes-open and closed EEG features involved. A discussion of the underlying mechanisms for these findings will be presented.

The Suisun Summit 2022: Emerging Themes and Open Questions in qEEG and Neurotherapies

Moderator: *Rogene Eichler West*

The purpose of this session is to organize interested participants towards the co-authorship of a journal article summarizing the themes and questions covered during the summit. We will begin by whiteboarding the most important topics to include. We will then break into small groups to add details to particular sections. We will then come together as a group again to determine homework assignments and deadlines for submitting a final production. We will aim for submission to the journal *NeuroRegulation*. Authorship requires submitting two to three paragraphs towards the completed document. If you have never published before, get your first publication credit just for writing up what you learned this week with your friends.