Brain Waves Reflect Cognition-Emotion State as a Diagnostic Tool for Intervention in Dysfunctional States: A Real-World Evidence

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Abstract: *Objective*: This study aims to characterize electrical signals to establish a diagnosis of cognitive-emotional dysfunction and guide a successful therapeutic intervention. Therefore, the present study aimed to observe these frequency bands in a sample of dysfunctional neurological behaviors to establish a neural marker of neural dysfunction that helps diagnose and monitor treatment.

Methods: A descriptive retrospective (extracted from the database) observational study design based on real-world historical data from routine clinical practice**.** According to DSM-5, low academic achievement (n =70), disruptive behavior (externalizing behavior problems) (n=70), and somatic syndrome disorder (n=70) were the subjects. The mean age of the sample was 14.13 (SD = 1.46; range 12-18), 31.5% women. The measuring instrument was the NeXus-10, which is suitable for acquiring a wide range of physiological signals. Brain electrical activity was recorded by using the quantitative electroencephalograph (qEEG) in accordance with the 10-20 International Electrode Placement System. In particular, the specific form of miniQ (mini-qEEG) was used.

Results: A pattern record present in all cases were identified. The record refers to (a) activity along the midline, namely, Fz-Cz-Pz, (b) activity from the center (Cz) to back, namely, Pz-O1 and O2, (c) activity from the center (Cz) forward (Fz), and (d) comparison between hemispheres. The characteristics of theta, alpha, and beta waves define the characteristic pattern of neurological dysfunction. The reversal of the dysfunctional pattern coincided with the remission of the clinical symptoms after treatment, which occurred in 87,6% of the subjects. We define remission as not meeting DSM-5 criteria.

Conclusion: This study suggests that miniQ register could be considered a simple and objective tool for studying neurological dysfunction. This dysfunction is explained according to current neurological knowledge of interactive cognition-emotion processing. MiniQ may be a cheap and reliable method and a promising tool for the investigation in the field.

Keywords: Electrophysiology, brain waves, behavior, learning, somatic disorder.

INTRODUCTION

Brain electrical activity can be recorded in the form of waves, namely alpha, beta, gamma, delta, and theta, being a relatively simple practical procedure within the scope of the practice of any professional in the field. The functional significance of these waves is not fully understood. Their functional role remains debated. Concerning the functional significance of brain waves, some well-established characteristics should be kept in mind. Slow frequencies (delta 0-4 Hz, theta 4-8 Hz, slow alpha 8-10 Hz) and fast frequencies (high alpha 11-12 Hz and beta 12-20 Hz) can be differentiated in the sense of slow versus fast. The faster activity ranges are thought to be involved in cortico-cortical interactions during information processing. The faster the activity, the more neural areas work asynchronously. The slower it is, the fewer areas do it. The faster the pace, the more asynchrony; the slower, the more synchrony. Asynchrony is characteristic of cortical-cognitive processing, while synchrony is characteristic of subcortical processing.

It is assumed that the higher frequencies translate cortical–cognitive processing; on the other hand, the lower frequencies convey subcortical processing. Subcortical processing happens mostly according to massed synchronized neural firing, which is detrimental to information processing [1]. Experimental evidence has demonstrated the link between the wave and brain territory. The entorhinal neurons oscillate at 5-7 Hz (near the theta rhythm), the parabrachial neurons (pons) at close to 10 Hz (near the alpha rhythm), and the cortical neurons at frequencies ranging from 10 to 50 Hz (near that reported for the activated cortical rhythm) [2-8]. Transitions between gamma-band oscillations (cognitive processing) and low-frequency (subcortical processing) bursts can occur rapidly [9]. Sleep-like slow waves have been positively associated with automatization (subconscious processing), and beta has been inversely related to it. The delta is active within brain networks that connect the cortex with the

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insula, amygdala, and brainstem [10]. Gamma is currently of limited clinical value.

The difference between cortical and subcortical processing seems well established. We can differentiate external (more modern) informative data processing neuro-cognitive network (frontal, parietal, occipital, and outer temporal cortex) from internal (oldest) neuro-sensitive network (amygdala, cingulate, mvPFC) [11]. This is not different from what happens with somatic processing, where sensitivity processing appears differentiated from motor processing. Homunculus has been well known for a long time. Since we are born, both networks have continuously interacted in parallel. Every experience involves informational data that leads to feeling right or wrong to a varying degree. Even the conceptual knowledge is stored as patterns of neural activity that encode affective information about each concept, contrary to the long-held idea that concept representations are independent [12]. Although the cognitive processing of informational data is easily understood, the same is not the case with emotional processing.

Emotions like sadness, fear, disgust, anger, disappointment, frustration, etc., share bad feelings. The terms have been coined based on describing behaviors. Each and every one of the emotions shares a good or bad feeling, and the sensitive brain processes and memorizes good or bad feelings. This is so always and in any case. Repeated and accumulated bad feeling experiences make the sensitive, emotional network more sensitive and, without being able to discriminate, interprets bad feelings as danger, fear, and insecurity (poor self-confidence). At each instant, the sum accumulated throughout the life of good or bad feelings makes the neurons that process feeling more or less sensitive. More sensitive means are more reactive. Sensory neurons (amygdala as a prototype) are unable to process informative data; they process sensitivity. The sensitive brain is a sensitive tissue that does not think.

The crucial issue is that cognitive processing (thoughts) and behaviors, in general, are biased when accumulated painful (dangerous) sensitivity is higher than pleasant. Certainly, the research by LeDoux *et al*. [13, 14] on processing fear is the most relevant. In a situation of fear (danger), LeDoux brilliantly demonstrated that such a sensitivity first unconsciously reaches the amygdala, and later the cerebral cortex receives conscious informative data. The amygdala reacts by sending signals indiscriminately everywhere, including the cortex, to trigger defensive behavior. The information about what is happening also reaches the cortex without going through the amygdala, but it does so later. What is relevant is that when the cortex receives direct information without going through the amygdala, it has already reacted to the signal from the amygdala and does not change its activity. Activity is thought (language) activity that is put into action as cognitive bias. It is not unreasonable that the cortex uses what is memorized and applies reasonable explanations for the situation in each case. It is a perfect unconscious self-deception.

The reality of unconscious cognitive and sensory-emotional processing is unquestionable [15-20]. For instance, the consciously verbally reported strategy to perform a task may not be the one the child used, according to the observable eye movements of the child [21]. It takes About 40 mins to form a conscious opinion of a stranger, and it is enough time to observe the subject's face and body language. The brain-mind can interpret it unconsciously if its facial features and body language inspire confidence or danger [22]. The always active processing of fear (danger) turns out to be crucial because it unconsciously conditions (biases) cognitive processing and behavior [13,14,23-25], promoting masked cognitive and behavioral bias (defensive behavior). One study [26] concludes that the brain might cheat when learning or behaving, building memorized answers to respond to similar questions (cognitive-behavioral bias).

Anticipatory unconscious processing before conscious processing in decision-making has been reported [27, 28]. Particularly, neuroimaging study demonstrated a decision could be encoded in the brain activity of the prefrontal and parietal cortex up to 10 s before it enters awareness [29]. It is not unreasonable to conclude that anticipatory unconscious processing has to do with subsequent decision-making. The corollary is that the most surprising thing is that humans reason rather poorly, irrational conscious biases in decision-making being common [30, 31]. When anyone makes a decision, they take it based on pure conscious rational arguments of pros and cons. These pros and cons may be unconsciously biased.

The key question is how reliable our thoughts are and the language that conveys them. Irrationality is very common, and verbal self-reporting is questionable. It can be a bias. This contrasts sharply with the classical view that pro and con reasoning is the most reliable way to arrive at sound decisions [32].

Fortunately, a good part of the biases can be diagnosed with the appropriate knowledge [33-35]. In neuroscientific terms, the individual's personality is determined by self-confidence, which is a sensitivity (feeling). We can assume that the placebo effect is unquestionable and linked to the power of beliefs. Positive beliefs are neurologically processed as self-confidence, and self-confidence is well-being (less pain or no pain). It has been found that activation of neural reward systems via non-pharmacologic means can reduce the experience of pain [36]. We work in such a way that the more unconscious self-confidence, the less accumulated negative sensitivity (pain-danger), which decreases the unconsciously generated defensive behaviors, including cognitive biases. The crucial point is to distinguish a compensatory (defensive) conscious state of well-being from (due to) the subconscious discomfort due to insecurity (non-confidence) as part of a biased activity (thought or behavior). The reality is that we decide what makes us feel better consciously or unconsciously (intuitively).

In this theoretical neural framework, neural dysfunction means the balance between cortical cognitive and subcortical sensory processing dysfunction. There are associations between personal states and wavelengths, which can provide targets for diagnosis and treatment. It would still be of value if one could recognize the contribution of emotion to dysfunctional behavior. Emotion significantly affects cognitive processes, as proved by research in the past decades. The emotional state is seen as a dimension instead of a state of a specific emotion, such as fear, sadness, joy, or greed, for example. Using emotion as a dimension simplifies the assessment and the quantification process of emotion. Lower frequencies give an indicator of emotional processing. Different brain wave patterns have been linked to different mental states, namely, learning disabilities, cognitive abnormalities, attention deficit hyperactivity disorder, antisocial behavior, autism spectrum disorder, traumatic brain injury, schizophrenia, Parkinson's disease, Alzheimer's disease, mild cognitive impairment, dementia, depression, anxiety, and so on.

EEG is a widely used tool for studying the brain. A common approach to measuring cortical activity across the brain is the electroencephalogram (EEG), which can reflect local neuronal activity as well as connectivity among brain regions. This study aims to characterize electrical signals to establish a diagnosis of cognitive-emotional dysfunction and guide a successful therapeutic intervention. Therefore, the present study aimed to observe these frequency bands in a sample of dysfunctional behaviors to establish a neural marker of neural dysfunction that helps diagnose and monitor treatment. We consider behavior in a neurological sense as an external manifestation of the person, that is, the output of central brain processing. In this sense, EEG analysis expresses cognitive and emotional intelligence [37].

METHODS

This descriptive retrospective observational study design is based on real-world historical data from routine clinical practice. Real-world data (RWD) are data relating to patient health status and/or the delivery of health care routinely collected from various sources, including observational studies (prospective or retrospective). Observational studies may provide credible evidence. Patient registries are a source of RWD. A patient registry is an organized system that uses observational study methods to collect uniform data (clinical and other) to evaluate specified outcomes for a population. Each one of the patients treated at the Fundació Carme Vidal has a clinical history that is incorporated into the registry.

Participants

The urban middle-class participants, predominantly from the urban area of a large city, were recruited from among those who come to the Foundation Carme Vidal de NeuroPsicoPedagogía due to low academic achievement (n=70), disruptive behavior (externalizing behavior problems) (n=70), and somatic syndrome disorder (n=70). The mean age of the sample was 14.13 (SD = 1.46; range 12-18), with 31.5% women. According to school information, low achievement was defined as achievement lower than the 25th percentile of the norm group across one academic year. This criterion was considered the most reliable based on real-world evidence. Disruptive behavior (externalizing behavior problems) and somatic syndrome disorder was defined according to DSM-5. The vast majority of disruptive behavior is especially aggression and poor interpersonal relationships with peers and teachers.

Trained psychotherapists qualified in educational psychology performed the procedure of recruitment, getting information directly from the school and health registration. Unstructured informal and open-ended interview was carried out with participants and parents of cases. A pediatric neurologist assessed each case by practicing a discerning clinical history and checking

registered personal medical history to confirm diagnoses of disruptive behavior and somatic disorders according to DSM-5 and rule out any other comorbidity or condition. Dyslexia, dysphasia, dyscalculia, ADHD, and psychiatric comorbidity were ruled out. As needed, the following studies were performed: auditory and visual event-related potential, otorhinolaryngology, ophthalmological exploration, cardiological examination, thyroid study, sonography, and video-EEG. Exclusion criteria were any child psychiatric disorders, comorbidity, previous medication, or other therapy in progress. Samples weren't grouped via socioeconomic background, sensory deprivation, ethnic pattern, or cultural or instructional factors.

All participants were administered the Cognitive Assessment System [37] to obtain their cognitive patterns in terms of planning, attention, and successive and simultaneous processing. The informed consent form was signed by the parents or guardians of each participant. The study follows the guidelines of the Fundació Carme Vidal human research ethics committee.

Assessment

The measuring instrument was the NeXus-10 (Mind Media®) which is suitable for acquiring a wide range of physiological signals. Up to four channels of EEG, EMG, ECG, and EOG signals are available. Heart rate, skin conductance, respiration, and temperature can be recorded. The NeXus-10 also has an extra input for oximetry or triggers and one input for digital sensors.

The NeXus-10 communicates wirelessly by Bluetooth or uses the USB extender cable to record data at higher sample rates. A high-grade lithium-ion battery pack and an SD flash memory card slot enable full ambulatory use of this portable device. The NeXus-10 is equipped with a display conveniently showing the device's status. We used five bands, with ranges set at <4 Hz (delta), 4–8 Hz (theta), 8–13 Hz (alpha), 13–30 Hz (beta), and >30 Hz (gamma) by the literature.

Procedure

Electrode gel provided appropriate conductance between the electrode and the skin. Before EEG data measurement, electrode impedance was reduced to less than 5kΩ to ensure good electrode connection to the scalp. The input signals were referenced to the ears and bandpass filtered with 0.5 and 50 Hz cutoff frequencies. Brain electrical activity was recorded using the quantitative electroencephalograph (qEEG) by the 10-20 International Electrode Placement System. In particular, the specific form of miniQ (mini-qEEG) was used. Recording conditions were awake and alert, eyes open and eyes closed. The electrodes, one for each point, were successively placed on the participant's head as follows: (a) Fz, Cz, Pz (midline) for 2 minutes with open eyes and 2 minutes with closed eyes; (b) C3, C4, P3, P4 for 2 minutes with open eyes; (c) O1, O2, P5. P6 for 2 minutes with open eyes and 2 minutes with closed eyes; (d) F3, F4 for 2 minutes with open eyes, doing the silent reading, and 2 minutes while the subject was performing the mathematical calculation. The device allows other variables to be recorded, such as skin conduction, corporal temperature, heart rate variability, breathing frequency, and EMG. The raw recording was digitized for data storage, manually edited to reduce artifacts (eye movement, EMG, body movement, etc.), and subjected to quantitative analysis (Fourier analysis).

The therapeutic intervention procedure has been previously described and reported. This study was carried out in accordance with the recommendations of the Institutional Review Board of the Fundació Carme Vidal de NeuroPsicoPedagogia, and with their approval of the study protocol. All subjects gave written informed consent to the Declaration of Helsinki.

Statistical Analysis

The principal components factor analysis (Varimax rotation) was applied to identify a correlation matrix that identifies the association level between brain waves and cognitive processes. A process of extraction groups individual items according to their level of inter-correlation into a smaller number of categories or factors. A rotation process maximizes the separation of these factors to give an interpretable solution.

RESULTS

Factorial analysis was applied to the recordings of brain waves in F3, F4, C3, C4, T5, and T6, and the results of the application of the Cognitive Assessment System. As Tables **1-6** show, the brain waves appear differentiated into slow and fast as independent entities when he/she is not engaged in a specific task. This is so at all recording points. For their part, cognitive processes become independent of brain waves as a different entity. The different entities are referred to as groups or grouped values in the tables. In this case, the group is equivalent to a factor.

Table 1: Brain Waves for F3 doing any Mental Activity. Factor Analysis (N=210)

	Waves group 1	Waves group 2	Cognitive processes
COGNITIVE PROCESSES			
Planning	-0.036	-0.004	0.820
Simultaneous	0.077	-0.101	0.780
Attention	0.066	0.083	0.797
Successive	0.047	0.067	0.737
BRAIN WAVES			
F3 delta	0.706	0.073	0.022
F3 theta	0.899	0.122	0.011
F3 alpha	0.952	0.138	0.053
F3 alpha1	0.914	0.111	0.088
F3 alpha2	0.914	0.184	0.041
F3 smr	0.782	0.509	0.042
F3 beta	0.752	0.590	0.025
F3 beta1	0.763	0.548	0.044
F3 beta2	0.538	0.801	0.075
F3 beta3	0.324	0.913	0.056
F3 beta4	0.105	0.960	-0.023
F3 high_beta	0.217	0.950	-0.011
F3 gamma	0.057	0.942	0.006
Eigenvalue	8.64	2.77	2.39
Variance explained	50.83%	16.29%	14.06%
Principal Components			
Varimax rotation			

Table 2: Brain Waves for F4 doing any Mental Activity. Factor Analysis (N=210)

	Waves group 1	Waves group 2	Cognitive processes
COGNITIVE PROCESSES			
Planning	0.065	-0.053	0.798
Simultaneous	-0.018	0.033	0.768
Attention	0.100	0.062	0.786
Successive	0.050	0.035	0.733
BRAIN WAVES			
F4 delta	0.055	0.645	-0.151
F4 theta	0.234	0.874	-0.011
F4 alpha	0.265	0.911	0.110
F4 alpha1	0.246	0.878	0.121
F4 alpha2	0.343	0.851	0.117
F4 smr	0.640	0.663	0.064
F4 beta	0.672	0.630	0.027
F4 beta1	0.929	0.289	0.079
F4 beta2	0.862	0.434	0.032
F4 beta3	0.932	0.273	0.073
F4 beta4	0.947	0.154	0.045
F4 high_beta	0.916	0.289	0.031
F4 gamma	0.921	0.056	0.087
Eigenvalue	8.88	2.42	2.21
Variance explained	52.28%	14.26%	13%
Principal Components			
Varimax rotation			

Table 3: Brain Waves C3 doing any Mental Activity. Factor Analysis (N=210)

	Waves group 1	Waves group 2	Cognitive processes	Waves group 3
COGNITIVE PROCESSES				
Planning	-0.094	-0.069	0.821	-0.023
Simultaneous	0.099	0.128	0.757	0.098
Attention	0.097	0.070	0.789	-0.130
Successive	-0.007	0.020	0.743	-0.036
BRAIN WAVES				
C3 delta	0.100	0.079	-0.102	0.931
C3 theta	0.105	0.642	0.029	0.590
C3 alpha	0.035	0.977	0.046	0.027
C3 alpha1	0.028	0.920	0.057	0.062
C3 alpha2	0.109	0.904	0.034	0.006
C3 smr	0.593	0.652	0.049	0.258
C3 beta	0.686	0.591	0.036	0.205
C3 beta1	0.766	0.419	0.067	0.225
C3 beta2	0.893	0.354	0.073	0.066
C3 beta3	0.965	0.044	0.042	0.049
C3 beta4	0.963	-0.037	-0.009	0.021
C3 high_beta	0.954	0.037	-0.021	0.011
C3 gamma	0.927	-0.079	0.015	-0.031
Eigenvalue	7.15	3.26	2.44	1.069
Variance explained	42.06%	19.15%	14.35%	6.29%
Principal Components				
Varimax rotation				

Table 4: Brain Waves for C4 doing any Mental Activity. Factor Analysis (N=210)

	Waves group 1	Waves group 2	Cognitive processes	Waves group 3
COGNITIVE PROCESSES				
Planning	-0.009	-0.036	0.802	-0.164
Simultaneous	0.194	0.121	0.754	0.141
Attention	0.083	0.067	0.776	-0.226
Successive	-0.011	-0.021	0.771	0.108
BRAIN WAVES				
C4 delta	0.142	0.102	-0.102	0.914
C4 theta	0.099	0.568	0.002	0.727
C4 alpha	0.016	0.974	0.053	0.101
C4 alpha1	-0.007	0.902	0.064	0.137
C4 alpha2	0.129	0.906	0.015	0.034
C4 smr	0.621	0.621	-0.013	0.197
C4 beta	0.657	0.590	0.014	0.166
C4 beta1	0.974	0.078	0.076	0.031
C4 beta2	0.894	0.357	0.056	0.046
C4 beta3	0.973	0.068	0.069	0.027
C4 beta4	0.962	-0.019	0.046	0.083
C4 high_beta	0.934	0.084	0.043	0.111
C4 gamma	0.910	-0.093	0.091	0.014
Eigenvalue	7.18	3.34	2.43	1.14
Variance explained	42.27%	19.67%	14.30%	6.68%
Principal Components				
Varimax rotation				

Table 5: Brain Waves for T5 doing any Mental. Activity Factor Analysis (N=210)

	Waves group 1	Waves group 2	Cognitive processes
COGNITIVE PROCESSES			
Planning	-0.039	0.004	0.801
Simultaneous	0.010	0.013	0.796
Attention	-0.046	-0.030	0.803
Successive	0.112	-0.140	0.746
BRAIN WAVES			
T5 delta	0.242	0.547	-0.102
T5 theta	0.251	0.827	0.002
T5 alpha	0.093	0.947	0.053
T5 alpha1	0.113	0.929	0.064
T5 alpha2	0.202	0.872	0.015
T5 smr	0.660	0.677	-0.013
T5 beta	0.796	0.512	0.014
T5 beta1	0.809	0.492	0.076
T5 beta2	0.906	0.372	0.056
T5 beta3	0.958	0.209	0.069
T5 beta4	0.979	0.078	0.046
T5 high_beta	0.976	0.103	0.043
T5 gamma	0.965	0.014	0.091
Eigenvalue	8.44	2.96	2.41
Variance explained	49.64%	17.40%	14.19%
Principal Components			
Varimax rotation			

Table 6: Brain Waves for T6 doing any Mental Activity. Factor Analysis (N=210)

	Waves group 1	Waves group 2	Cognitive processes
COGNITIVE PROCESSES			
Planning	-0.014	-0.012	0.816
Simultaneous	-0.046	0.041	0.796
Attention	-0.070	-0.037	0.806
Successive	0.025	-0.128	0.746
BRAIN WAVES			
T6 delta	0.392	0.493	-0.067
T6 theta	0.247	0.827	-0.118
T6 alpha	0.061	0.955	-0.008
T6 alpha1	0.089	0.908	-0.002
T6 alpha2	0.128	0.899	0.019
T6 smr	0.491	0.770	-0.058
T6 beta	0.602	0.690	-0.096
T6 beta1	0.955	0.260	-0.026
T6 beta2	0.847	0.479	-0.027
T6 beta3	0.948	0.264	-0.002
T6 beta4	0.978	0.126	-0.030
T6 high_beta	0.949	0.210	-0.066
T6 gamma	0.952	0.009	-0.006
Eigenvalue	8.35	2.84	2.49
Variance explained	49.13%	16.68%	14.64%
Principal Components			
Varimax rotation			

On the other hand, the following pattern was identified according to specific analyzes:

1. Activity along the midline, namely, Fz-Cz-Pz:

    1.1. At Cz, the central location of the medial line, higher theta, and low Alpha waves along with slower sensorimotor rhythm (SMR). It is about slow wave hyperactivity.

    1.2. At Cz with eyes closed, the Alpha wave is dominant (higher in amplitude) as expected, but Beta and Theta are too high. It is also found that the Beta wave remains practically at the same value in amplitude with closed eyes (it does not decrease in amplitude when closing eyes as expected). It is about hyperactivity of Theta and Beta.

    1.3. At Cz and maybe Pz, the scale of progressive decrease in amplitude of the different Beta waves is not met, that is, B1>B2>B3>B4. It is about beta hyperactivity.

    1.4. High Theta/Beta ratio. Predominant activity of theta over beta.

2. Activity from the center (Cz) to back, namely, Pz-O1 and O2:

    2.1. The slow-wave Theta does not rise in amplitude from the center backward as expected. It is about dysfunctional-displaced Theta activity.

    2.2. The Beta wave is expected to be more dominant (higher in amplitude) in the front (Fz) than in the back (Pz). On the contrary, it is higher (dominant) in the central (Cz) and parietal (Pz) areas than in the frontal area (Fz). It is about dysfunctional-displaced Beta activity.

    2.3. Alpha blockade (eyes-closed response) must increase Alpha amplitude, but the increase is very small in the central (Cz) area (30% would be expected). In occipital (01-O2), alpha decreases with eyes closed instead of increasing (an increase of around 50% would be expected). It is about hypoactive Alpha.

    2.4. Peak Alpha higher or slower than expected. It is about dysfunctional Alpha.

3. Activity from the center (Cz) forward (Fz):

    3.1. Theta wave increases (is higher) progressively from parietal (Pz) to the frontal area (Fz), so we have a more slow wave in front than behind, when it should be the opposite. It is about dysfunctional-displaced Theta hyperactivity.

    3.2. Theta and also alpha are higher (amplitude) in the frontal area (Fz) than in the central area (Cz) when it should be the opposite. It is about dysfunctional-displaced Theta and Alpha hyperactivity.

    3.3. Theta values should decrease amplitude when tasks are performed, but this is not the case. It is about Theta hyperactivity.

    3.4. Beta 1 should have a higher amplitude in the frontal zone, but this is not the case. It is about Beta hypoactivity.

4. Comparison between hemispheres:

    4.1. The values (amplitudes) in the medial zone (Fz) should be higher than in the hemispheres only for slow waves. It is about dysfunctional-displaced slow-wave hyperactivity.

    4.2. Beta must be higher in the hemispheres (F3-F4, C3-C4, P3-P4) than in the medial zone (Fz, Cz, Pz). The opposite is abnormal. It is about dysfunctional-displaced Beta hyperactivity.

    4.3. Beta predominates in the right hemisphere in both the frontal area and the central and posterior area (F4, C4, P4, O2) instead of predominating (higher in amplitude) on the left hemisphere (F3, C3, P3,01). It is about dysfunctional-displaced Beta hyperactivity.

    4.4. Alpha dominates (higher in amplitude) in the left hemisphere (F3, C3, P3, T5, O1) instead of the right. Alpha dominates in the left hemisphere and not in the right as would be expected. Inverse Alpha frontal asymmetry. It is about dysfunctional-displaced Alpha hyperactivity.

    4.5. Alpha tends to be higher (amplitude) in the right hemisphere, and too little (amplitude) alpha in the right hemisphere is not expected. It is about Alpha hypoactivity.

4.6. Beta wave value (amplitude) in the occipital area (O1, O2) is higher than in the frontal area (F3, F4), where it should have the maximum value. The opposite is not expected. It is about dysfunctional-displaced Beta hyperactivity.

Each of the subjects in the sample (n=210) who completed the diagnostic protocol presented a specific registration pattern to a greater or lesser degree. Therefore, ≥ 13 registry anomalies are necessary to meet the diagnostic criteria of neurological dysfunction. We have been able to verify in each case the following: (a) the presence of at least three out of four criteria for points 1, 2, and 3, and (b) the presence of four out of six criteria for point 4.

A therapeutic intervention based on family therapy and Ericksonian hypnosis was performed. Post-testing, 6 months after treatment, indicated remarkable normalization of the neural activation. 56 out of 70 subjects in the low academic achievement group (80% response rate), 63 out of 70 subjects in the disruptive behavior group (90% response rate), and 65 out of 70 subjects in the somatic syndrome group (92.8% response rate) did not meet diagnostic criteria for electrophysiological dysfunction. The cases that responded in electrophysiology also did so in their clinical-behavioral conditions and vice versa. We define clinical remission as not meeting DSM-5 criteria. The results were maintained for three-month and at a one-year follow-up. After two years, follow-up testing indicated a slight regression in ten patients.

DISCUSSION

After treatment [33-35], results from brain activity are so dramatic that even the observational study results leave no room for doubt. It is a sufficient demonstration of its usefulness. First of all, it should be noted that the factorial analysis seems to confirm that fast and slow waves form two operating systems with their entity. Neither of these systems is fully identified with cognitive processing. On the contrary, one could postulate that the factors identified as brain waves mostly express emotional processing.

We have scientific literature to support the role of brain waves in brain normal function/dysfunction. As the results say, slow frequencies (theta 4-8 Hz, slow alpha 8–10 Hz) and fast frequencies (high alpha 11–12 Hz and beta 12- 20 Hz) appear as markers of neurological dysfunction. Only slow frequency delta does not appear as a marker of dysfunction. In short, the electrical pattern denotes a dysfunction consisting of a predominance of subcortical subconscious processing over cortical conscious processing. We speculate that the beta wave is expressed as a defensive activity triggered in response to the subcortical-subconscious hyperactivity typical of the sensitive processing of danger-fear (no self-confident). In this sense, cortical activity (beta) would explain an activity responsible for the conscious cognitive bias. In this case, beta is a response to a state of tension.

Growing developments in neuroscience have enabled elegant studies that investigate the neural electrophysiology underlying function /dysfunction. Concerning theta wave, music was associated with an increase in frontal midline (Fm) theta power, which reflects emotional processing. Therefore, Fm theta is taken to reflect emotional processing [38]. Emotional experiences have been significantly correlated with theta band power in the anterior and frontal midline region [39]. An increase in theta activity in the prefrontal cortex related to emotional experience has been associated with orbitofrontal cortex activation [40]. The higher theta responsivity in autistic spectrum disorder may indicate compensatory activity to a cognitively demanding condition as a stressor [41]. Delta (0–4 Hz) and theta (4–8 Hz) rhythms are associated with hypoactive cortical states, in contrast to alpha (8–12 Hz) which is associated with cortical activated states.

Although the delta wave does not appear as a marker in our study, the literature on it, supporting its role as an expression of subcortical processing, is relevant to our conception as it is a slow wave. Delta waves have been associated with panic attacks and sustained pain. Also, with subliminal perception. In this way, from an evolutionary perspective, they are associated with evolutionary old basic processes, among which is the emotional processing of danger [42]. Delta that is normal during slow-wave sleep can convey cognitive abnormalities in the awake state in the frontal and temporal lobes [7]. Delta oscillations dominate the EEG of waking reptiles. In humans, they are characteristic of developmental stages and during slow-wave sleep. This evidence shows that delta waves are associated with evolutionary old basic processes that reside in the subcortical areas as opposed to fast waves. The increase of delta power has been documented in a wide array of developmental disorders and pathological conditions, for instance, during panic attacks and sustained pain. However, the former processes increase activity when the latter are

dysfunctional [42, 43]. In one patient, single delta wave bursts persisted following the cardiac rhythm cessation [44]. Increases in the delta/ theta are associated with early cognitive impairment [45].

Concerning alpha wave, inverse frontal alpha asymmetry, that is, greater left-sided activation of frontal alpha (frontal asymmetry) band power (8-13 Hz), has been associated with emotion [46-48] and emotional decision-making [49].

From our conception, beta is an ambivalent wave in the sense of a double interpretation. It can translate cognitive cortical processing but also unspecific ‘‘irritable’’ cortex. A significant absolute power increase in beta at frontal (Fp1, F3, and F4) and central (C4) areas has been related to increased cortical activation [50]. The low beta frequency range (13–17 Hz) is associated with subsequent memory success, independent of stimulus modality [51]. Beta spindles are seen in conditions such as ADHD and bipolar depression, and epilepsy, suggesting an irritable cortex. ADHD is associated with an excess of theta and, in some cases, low alpha, that is, an excess of subcortical processing. The theta/beta ratio has FDA approval as a diagnostic marker in ADHD [52]. A consistent decrease in beta power measured at the brain stimulation site was correlated with a significant and sustained reduction in depressive symptoms [53].

Next, we will focus on the interrelationship between brain waves. An inverse relationship between fast and slow waves is practically constant. It has been reported that connectivity changes with development so that it increases from childhood to adolescence. The increase is between frontal hubs and frontal, parietal, and temporal regions, whereas connections decrease in the posterior part of the brain. This can be translated into less cortical activity in the posterior region of the brain [54, 55]. Therefore, it is expected that the theta wave will be more evident from the center towards the rear, and the beta wave will predominate in the frontal area compared to the posterior area.

An increase in alpha but a decrease in theta reflects cognitive activity [56]. CNS injuries are associated with increased theta and decreased alpha, which is compatible with less cognitive-cortical function [57, 58]. In the same sense, it has been reported that increases in slow frequencies, namely, delta, theta, slow alpha 8-10 Hz, and decreases in fast cognitive frequency alpha 10.5-13.5 Hz [59]. Regular meditation can increase alpha waves (relaxation brain waves) and reduce beta waves, the brain waves linked to thought and learning [60]. Chronic pain has been associated with an increase in theta and alpha power at rest [61].

Beta responds to cortical hyperactivity, whatever its nature, level of information processing, or irritative state. The association of lower levels of alpha and theta with an excess of fast beta has been reported in alcoholics and their children [62]. This is compatible with low subcortical activity linked to emotional life and high cortical activity, which in this context can be irritative. It is assumed that beta (19–30Hz) both on the left and right can be associated with an irritable state. Another pattern can be seen in various mental illnesses, dominant theta with high power in beta 12-30 Hz, which is compatible with high subcortical processing (emotional mind) and irritative cortical processing [63].

Attention lapses share a common physiological origin, the emergence of local sleep-like activity within the awake brain, i.e., sleep-like slow waves (within the delta (1–4 Hz) or theta (4–7 Hz) range), despite individuals being behaviorally and physiologically awake [65]. Being redundant on the subject, increasing alpha and theta with decreasing fast beta can be felt to produce a relaxed state [64]. Theta is linked to the inattentive state, and high alpha and beta are related to the attentive state (stress state). In the same sense, elevated delta and theta in the awake state in frontal, central, and temporal lobes can convey disrupted cortical information processing [66]. Traumatic brain injury is associated with increases in slow frequencies (delta, theta, slow alpha 8–10 Hz), decreases in fast frequency alpha (10.5–13.5 Hz), and increases in beta levels [57-59]. We can say CNS injury means less cortical processing. In our opinion, beta is expressing unspecified cortical irritative conditions not linked to information processing.

Individuals with learning disabilities and attention problems can show a deficiency of 13Hz activity in certain brain regions, which can affect the ability to perform sequencing tasks and math calculations easily. Increased slow-wave activities and reduced asymmetry between the two hemispheres have been associated with Alzheimer's disease (AD) patients. These patients can have increased theta and delta wave activity in the right parietal areas, which reflects their decreased cognitive brain function in these regions [67]. A significant increase in the activity of delta waves and theta waves in the frontal lobe of people with schizophrenia has been reported. In contrast, the alpha

waves indicated a decrease in the occipital lobe in all schizophrenic patients [68]. High alpha or low beta is associated with “active” intelligence.

This study has its limitations. While the true accuracy of a study is not known until it has been evaluated in one or more independent studies. Replications are needed. Possibly the biggest limitation is the lack of a control group, but there is a broad consensus on the criteria for normality in brain wave electrophysiology. The results may overestimate the benefit of the new diagnostic technology. Readers of research studies should always keep in mind potential confounding explanations. The interpretation of a research study depends on how well the reader has estimated and considered confounders and biases. In order to better understand the cognitive implication, it is important to evaluate the brain wave register and its relation to the results of a cognitive test. We are in the process of doing it with the Cognitive Assessment System to assess the PASS processes. For replication purposes, we refer to the bibliography [69].

CONCLUSION

In conclusion, this is a study in the real-world setting of routine clinical practice. The results are extremely encouraging and convincing evidence. This study suggests that miniQ register could be considered a simple and objective tool for the study of neurological-behavioral dysfunction. In addition, results show that miniQ probably is a relevant outcome measure for assessing changes in therapeutic intervention. These electrophysiological findings can be a "biomarker" for treatment optimization. Therapeutic intervention may correct these dysfunctions and bring the brain back to a more normative and efficient way of processing.

Moreover, these quantitative electrophysiological findings in children with dysfunctional behavior or/and learning have a clear relation to a psychological condition. In neurology and psychiatry, the common denominator is that the same circuits underpin different symptoms or behaviors. In the coming years, we will continue better understand the mechanisms that generate these distinct states of reversible dysfunction. These results suggest that miniQ may be a cheap and reliable method and a promising tool for the investigation in the field.

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AUTHOR CONTRIBUTIONS

All authors contributed toward data analysis, drafting, and critically revising the paper and agree to be accountable for all aspects of the work. They all approved the final version of the manuscript for submission and agreed to be accountable for all aspects of the work.

DISCLOSURE

The authors report no conflicts of interest in this work.

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