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A Modular Activation/Coherence Approach to Evaluating Clinical/QEEG Correlations and for Guiding Neurofeedback Training: Modular Insufficiencies, Modular Excesses, Disconnections, and Hyperconnections

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A Modular Activation/Coherence Approach to Evaluating Clinical/QEEG Correlations and for Guiding Neurofeedback Training: Modular Insufficiencies, Modular Excesses, Disconnections, and Hyperconnections

Jonathan E. Walker, MD Gerald P. Kozlowski, PhD Robert Lawson, MS

ABSTRACT. Current approaches to QEEG-guided neurofeedback involve efforts to normalize the abnormalities seen, without reference to the functional localization of the cortical areas in-
volved. Recent advances in cortical neurophysiology indicate that specific brain areas are devel-
oped to perform certain f between modules, particularly during a learning situation. For example, the left prefrontal "activation module" must cooperate with one or both occipital "visual modules" to attend *and* see something on a chalkboard. To remember what has been seen, both temporal "memory modules" must cooperate with the visual modules for the image to be retained in short-term memory. If the connections between these modules are not functioning optimally, visual learning will be impaired. Decreased coherence (hypocoherence) indicates a decrease in functional connectivity between these modules, and increased coherence (hypercoherence) indicates an increase in functional connectivity between the modules. Neurofeedback can be used to normalize coherence between these modules, thereby improving the efficiency of their cooperation in the learning process. If coherence is less than normal, it is trained up. If coherence is more than normal, it is trained down. Three cases are presented where this approach has succeeded in remediating the client's symptoms. doi:10.1300/J184v11n01_03 *[Article copies available for a fee from The Haworth Document Delivery Service: 1-800-HAWORTH. E-mail address: <docdelivery@haworthpress.com> Website: <http://www. HaworthPress.com>* © *2007 by The Haworth Press, Inc. All rights reserved.]*

KEYWORDS. Activation, coherence, connectivity, neurofeedback, QEEG, disconnections, excess, hyperconnections, insufficiencies, module

INTRODUCTION

This paper provides motivation and a detailed rationale for the use of power and coherence metrics in the assessment and training of a variety of clinical cases, and presents individual case outcomes. Our findings provide a foundation for further development and application of coherence and related metrics in practical clinical scenarios, based upon a functional model of the brain and EEG.

There are four major ways in which information is coded and processed in the cerebral cortex:

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- 1. Frequency coding (cycles/second)
- 2. Intensity coding (amplitude)
- 3. Spatial coding (connections)
- 4. Tim binding (simultaneous or asynchronous activation)

The only technology that gives us information with which to adequately evaluate cortical function is the quantitative electroencephalogram (QEEG). Further, the time course of EEG information (milliseconds) is the only technologythatisinrealtime,i.e.,whatishappeningas it is happening. Localization of brain functions basedonthestudyofbrainlesionsisatime-honored tradition in neurology (Mesulam, 2000). Gradually over time the concept of modules subserving distinct brain processes has gained widespread acceptance (Fodor, 1983). With the advent of QEEG it has become possible to evaluate localized brain dysfunctions, and to correlate those abnormalities with neuropsychological test abnormalities (Shenal, Rhodes, Moore, Higgins, Harrison, 2001). A problem with this approach is that there may be several functions associated with a given area delimited by the 10/20 system (e.g., FP2). On the other hand, a functional module may involve several areas of the 10/20 system. For example, the process of reading involves FP1, 01, 02, T3, T5, and P3 (at a minimum), as well as connections between those areas (Walker & Norman, 2006). The commercially available QEEG databases (Lubar, 2003) are restricted to the 10/20 system, so we cannot train all the elements of such complicated modules at the same time. However, we can evaluate the connectivity of the different areas represented in the 10/20 system. These areas may be viewed as having a central role in the various brain processes. Neurofeedback can then normalize the connections with coherence training. If the modules are under-activated or over-activated, neurofeedback can restore normal activation. Once the modules are activated and connections are normalized, normal brain activity can take place.

DEFINITIONS FOR THIS PAPER

1. Module–an area of the cerebral cortex, lying under an electrode location defined by the 10/20 system, which has a characteristic or principal function (e.g., 01, which has the principal function of analyzing visual information from the right half of visual space). There may be other functions within that module (e.g., color perception). Several modules may be needed to subserve complex brain functions, such as reading.

2. Coherence–thedegree of cooperationbetween two brain areas (modules). Normal coherence leads to optimal cooperation. Decreased coherence results in less cooperation than normal, leading to reduced efficiency, longer processing time, and mistakes. Increased coherence leads to excessive cross-talk between the two areas involved and less cooperation with other brain areas, leading to stereotypic or stuck responses, decreased flexibility, and decreased creativity in cortical processing.

Table 1 is information we gathered from our clinical experience and from other resources (Brownback et al., 2003; Joseph, 1990; & Mesulam, 2000). It indicates the principal functions of the different modules, as delineated by the 10/20 system. Other functions in which the modules seem to be important are listed in the third column. Table 2 indicates the coherence pairs involved in functions requiring cooperation of activity between those two sites to produce that activity (Walker, 2003).

This model emphasizes the roles of specialized areas (modules) and their connections in normal brain function. Brain disease commonly results in modular insufficiencies, modular excesses, disconnections, and hyperconnections. Neurofeedback training to normalize these abnormalities is proving to be an effective way to normalize the functions of the cerebral cortex. At this point, only a few examples of each type of abnormality have been found, but this approach is proving to be a reliable way to restore normal brain functions in patients with stable deficits involving cortical areas and their connections, as assessed by QEEG.

TABLE 2. Coherence Pairs Involved in Specific Functions

FPI Coherences

FP2 Coherences

F7 Coherences

F3 Coherences

F4 Coherences

F8 Coherences

T3 Coherences

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C3 Coherences

C4 Coherences

T4 Coherences

T5 Coherences

P4 Coherences

T6 Coherences

O1 Coherences

O2 Coherences

Midline Coherences

UNDERLYING ASSUMPTIONS

- A. The QEEG data bases (using the 10/20 system) represent a reasonable estimate of the optimal (normal) modular activity (amplitude) and connectivity (coherence).
- B. The brain can learn to normalize the abnormalities with the use of neurofeedback.
- C. Resolution of the abnormalities will result in remediation of the symptoms and normalization of brain functions.
- D. Modules and connections not evaluated with available data bases are not likely to be detectedon QEEG, nor to be improved by QEEG-based neurofeedback.

PATTERNS OF ABNORMALITIES ON QEEG

The six patterns so far delineated include:

- 1. Modular insufficiencies–Excessive slow activity or diminished fast activity in a module. The classical example is reduced verbal expression (fluency) with increased amplitudes of slow frequencies (delta, theta, alpha) in module F7 (Broca's area). Training to decrease slow frequencies at F7 would be associated with improvement in speech fluency. A second example: an increase in the amplitude of slow frequencies at FP1 is a common finding in attention deficit disorder (inattentive type). Training to decrease the amplitude of slow frequencies usually results in improved attention (Othmer & Othmer, 2005).
- 2. Diffuse insufficiencies–Excessive slow activity or diminished fast activity diffusely. This is seen with toxic encephalopathies, mental retardation, and severe (diffuse) head injuries. Normalizing these abnormalities results in improved cognitive functions.
- 3. Modular excesses–Excessive beta activity. For example, if there is an excess of beta activity at FP1, this is also likely to produce attentional difficulty, but of the hyper-focused or anxiety associated type

rather than the inattentive type. A second example is tics, which are associated with excessive beta at C3 and C4. Training the beta down improves these problems.

- 4. Diffuse amplitude excesses–Excessive beta activity diffusely. This is seen in alcoholism and various anxiety disorders, including obsessive compulsive disorders. Training the beta down reduces anxiety, obsessive compulsive behavior, and craving for alcohol.
- 5. Disconnections–Decreased connectivity between two brain areas (modules). An examplewould be conductionaphasia, as elucidated by Geschwind (1965). The QEEG would show hypocoherence between F7 (Broca's area) and T5 (Wernicke's area). Training to increase coherence between those two modules would be expected to resolve the conduction aphasia. This kind of abnormality is commonly responsible for dyslexia, which is associated with one or more disconnections between left hemisphere language locations. Reading ability usually improves markedly with neurofeedback training to normalize coherence between these areas (Walker & Norman, 2006).
- 6. Hyperconnections–Increased connectivity between two brain areas (modules). The idea that hyperconnection between different areas could result in brain dysfunction is relatively new (Catani & ffythe, 2005). Rather than difficulty using two areas simultaneously, there is difficulty in getting and giving information from other brain areas. As a result, there is a decrease in flexibility and creativity secondary to less connection with other brain areas required to make varied approaches or responses. An example would be hyperconnection between FP1 (logical attention module) and F3 (motor planning module for the right upper extremity). This would result in inflexible or stereotyped responses to attentional stimuli (see Patient 3 below).

Table 3 lists other examples of disorders that have been successfully treated using this model, as well as disorders based on "off the map" modules.

TABLE 3. Quantitative EEG abnormalities and associated disorders.

* FPO2 (frontopolar/orbital) = right medial orbit just below eyebrow (Fisher, 2003) (Blum, et al 2005)
** FPO1 (frontopolar/orbital) = left medial orbit just below eyebrow
*** I01, I02 (inferior occipital left, right = bel

neurofeedback therapy. It suggests that more sessions of neurofeedback may be necessary to help these people with their addictions, which are

largely determined by their insensitivity to reward (Blum et al., 2000). It also suggests that initial training to activate the reward module

(decrease 2-7 Hz/ at FPO2 and FPO1) should make reward-based therapies, such as other neurofeedback protocols, more effective in

ameliorating other problems in such patients (such as excessive high frequency beta). FPO1 and FPO2 beta training (decrease 2-7 Hz, increase

15-18 Hz) should also help with other addictions (drugs, food, sex, gambling, etc.) by sensitizing these individuals to cognitive (FPO1) and non-

verbal (FPO2) reward, thereby reducing the amount of these rewards required to make them satisfied with the rewards into the normal range.

QEEG would not be helpful in diagnosing reward deficiency syndrome, since the nucleus accumbens, where the abnormal dopamine receptors in these individuals is located, does not generate sufficient rhythmic activity to be detected with scalp electrodes. Still treatment with neurofeedback

should be effective, since activity in the nucleus accumbens can be regulated by orbital frontal cortex (FPO1 and FPO2) (Kalivas, 2005).

This model also predicts that states of fear cannot be detected by QEEG, since these states are generated by amygdalar activity (LeDoux, 2003). Nevertheless, excessive amygdalar activity should be down-regulated by FPO1 alpha training for cognitive fears and FPO2 alpha training for non-verbal fears (including phobias).

METHODS

EEG's were recorded with a Cadwell® system (model Easy II) using standard recording techniques. QEEGs were evaluated with the Thatcher Neuroguide database®. Neurofeedback was done on Brainmaster® equipment (model 2.5 SE) using auto-thresholding.

Examples from Our Clinic

Patient #1 –15 y/o boy

Training: 5 sessions to increase coherence of theta F3/01 5 sessions to increase coherence of theta F3/02 Result: Marked improvement in school performance Improved shooting ability when hunting

> Batting average improved from .250 to .500

Discussion: This case represents a relatively simple disconnection syndrome involving the left motor planning module (F3) and both right and left visual processing areas (01 and 02). This disconnection resulted in a visual/ motor learning difficulty and a performance difficulty. Both were rapidly remediated with neurofeedback. Visual/motor improvements resulted in better reading, better copying from the chalk board, improved accuracy in rifle memory" (excess rumination)

3) Hypocoherence beta C4/ F4–disconnection between sensorimotor interaction modulefor the left upper extremity and the motor planning module for the left hand, resulting in clumsiness of the left hand and performance errors

4) No excess of delta, theta, or alpha power–This implies the patient does not have classical ADD, which is associated with excess theta or alpha at FP1. Classical neurofeedback training to decrease theta and/or alpha probably would not have helped this child.

Training: 5 sessions to decrease beta power at FP1

5 sessions to decrease beta power at T3

5 sessions to increase coherence of theta C4/F4

Results: Doing well in school and at home

Discussion: This case represents a combination of problems. First is excess beta at FP1, an indicator of anxiety-associated attentional difficulty. The second is excess beta at T3, an indicatior of excess rumination. Third, there is a disconnection between the sensorimotor integration and motor planning areas for theleftupperextremity,resultinginclumsiness and slowed reaction time with the left hand. Each problem was rapidly remediated with training to normalize each.

Patient #3–T.R., 10 y/o

2) Excess absolute alpha power P4 ($Z = 2.43$)

3) Hypocoherence of delta $T3/T5 (Z = 2.56)$

4) Hypocoherence of beta $O1/F3 (Z = 2.54)$

5) Hypocoherence of alpha $T4/T6 (Z = 3.11)$

6) Hypercoherence of alpha $FP2/F4 (Z = 2.32)$

7) Hypercoherence of alpha $FPI/F3 (Z = 3.23)$

8) Hypercoherence of alpha $O2/F4 (Z = 2.52)$

9) Hypercoherence of theta $FP2/F4 (Z = 2.63)$

QEEG/Clinical

Correlations: 1) Excess alpha at C3 (sensorimotor integration module for right upper extremity)–modular insufficiency, resulting in clumsy right hand, poor handwriting

> 2) Excess alpha at P4 (perceptual/cognitive processing module of the right hemisphere)–modular insufficiency,resultingin mathematics difficulty

> 3) Hypocoherence of delta at T3/T5 (disconnection between the verbal memory/ phoneme recognition module and the verbal understanding/comprehension module)– resulting in difficulty with phoneme recognition and verbal memory (a left hemisphere auditory processing problem). This probably accounted for part of the child's difficulty reading.

> 4) Hypocoherence of beta at O1/F3 (right visual/right

motor upper extremity disconnection)–resulting in increased visual motor reaction time

5) Hypocoherence of alpha at T4/T6 (emotional memory/emotional understanding disconnection)–resulting in slow auditory/emotional processing, errors (right hemisphere auditory processing problem)

6) Hypercoherence of alpha at FP2/F4 (emotional attention/motor planning left upper extremity hyperconnection)–resultingindecreased flexibility and creativity in emotional attention/motor planning with left upper extremity

7) Hypercoherence of alpha at FP1/F3 (logical attention/ motor planning right upper extremity hyperconnection)– resulting in decreased flexibility and creativity in logical attention/motor planning with right upper extremity

8) Hypercoherence of alpha at O2/F4 (visual processing left visual field/motor planning left upper extremity hyperconnection)–resulting in decreased flexibility and creativity in visual/motor processing to the left

9) Hypercoherence of theta at FP2/F4 (emotional attention/motor planning right upper extremity hyperconnection)–resulting in decreased flexibility and creativity in emotional/motor processing

Training: 55 sessions:

1) Decrease alpha amplitude at C3 (10 sessions) to improve fine motor coordination with right hand and to improve handwriting

2) Decrease alpha amplitude at P4 (10 sessions) to improve visualization of mathematical problems and cognitive processing of them (reasoning)

3) Increase beta coherence atO1/F3 (5 sessions) tointegrate visual processing of right visual information with motor planning for the right upper extremity and speed visual motor reaction times and reduce visual/motor errors

4) Increase alpha coherence at T4/T6 (5 sessions) to integrate emotional memory with emotional understanding and improve auditory processing and reading

5) Increase delta coherence at T3/T5 (5 sessions) to integrate verbal memory and phonological processing and improve auditory processing

6) Decrease alpha coherence FP2/F4 (5 sessions) to improve flexibility and creativity in coordinating emotional attention and motor activities of the left hand

7) Decrease alpha coherence at FP1/T3 (5 sessions) to improve flexibility and creativity in coordinating attention and verbal memory. This would be expected to improve reading.

8) Decrease alpha coherence O2/F4 (5 sessions) to improve flexibility and creativity in coordinating visual processing of right visual field information with motor planning for the left hand (for example, mimicking)

9) Decrease theta coherence FP2/F4 (5 sessions) to improve flexibility and creativity in coordinating emotional attention and judgment with motor planning for the left hand

Result: No improvement in reading ability with amplitude training alone

> Reading at grade level after amplitude plus coherence training

Pre: reading at 1st grade level

Post: reading at 5th grade level (in 3 months)

Normally attentive

Not hyperactive or impulsive

CONCLUSION

A modular coherence model is presented, based on modern concepts of distributed networks and their role in cerebral dysfunctions. The model presented here has proven successful in using the QEEG to guide neurofeedback training in clients with static brain dysfunctions involving the cerebral cortex and the corticocortical connections. These include learning disabilities, residual problems from closed head injury, epilepsy, and autism.

The QEEG is less useful in guiding training in disorders with prominent subcortical pathology. These types of cases may respond better to empirical symptom-based protocols, such as those used by the Othmers (2005) for remediation of symptoms.

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