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Recent Advances in Quantitative EEG as an Aid to Diagnosis and as a Guide to Neurofeedback Training for Cortical Hypofunctions, Hyperfunctions, Disconnections, and Hyperconnections: Improving Efficacy in Complicated Neurological and Psychological Disorders

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Abstract Recent advances in QEEG-databases have enabled improvements in interpretation, which in turn have led to more effective neurofeedback interventions. These improvements relate mostly to evaluations conducted in the high frequency beta range (21–30 Hz) evaluation and in single Hz bins, which more specifically address which frequencies need to be trained to most quickly and effectively normalize their dysfunctions and remediate their difficulties. Use of the modular activation/coherence model (Walker et al. in J Neurother 11: 25–44, 2007) provides a framework for correcting the slow or fast modular dysfunctions, as well as normalizing connectivity using coherence training.

Keywords EEG · QEEG · Neurofeedback

Recent advances in neuroimaging (fMRI, PET, SPECT, and diffusion tensor topography) have led to a re-evaluation of the relationship between localization of cortical functions and the association pathways that connect them (Catani and ffytche 2005). The original conceptions of cortical localization by Broca, Wernicke, and their associates were based on studies which indicated that focal lesions of certain areas resulted in hypofunction (decreased function) of that area. For example, a lesion of Broca's area (roughly correlating with F7 in the 10/20 system) resulted in an expressive speech problem. A lesion in Wernicke's area (roughly correlating with T5) resulted in a receptive speech disorder. Over the years, other areas were discovered, which have come to be correlated with other specific brain functions (Walker et al. 2007). These are listed in Table 1, where the behavioral correlate for each lesion is noted in the last column. Note, there is some overlap in these functional areas. For example, an excess of slow activity at 1–10 Hz (hypofunction) at F3 may affect attention, an excess of slow activity at F4 (hypofunction) may be associated with poor judgment and inadequate restraint of impulses, while lesions at F3 and F4 may also be involved in production of symptoms of ADHD. There are also important areas of localization which are not detected by the databases using the 10/20 system. For example, Oz is an area that is important in regulating bladder control, oppositional behavior, and balance.

In our experience, these hypofunctions can oftentimes be managed with 5–7 sessions of neurofeedback, although we have not formally assessed this. This appears to be true no matter the etiology (the most common being congenital, head injury, and stroke in our practice).

The second category of dysfunction is hyperfunction. The development recently of databases which include normal values of high frequency beta activity (21–30 Hz) has enabled the recognition of these hyperfunctions. Whatever the localization of the excess high frequency beta, it is usually associated with anxiety, irritability, and reduced tolerance to stress. In addition, the excess high frequency beta appears to interfere with function of the area involved. Table 2 indicates the correlates of hyperfunction in the various areas with clinical symptoms. Hyperfunctions are addressed with neurofeedback by rewarding inhibition of 21–30 Hz activity and facilitation of 10 Hz activity. In our clinical experience, 5–7 sessions are usually sufficient to address the hyperfunction (again, we have not collected any systematic data on this).

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Table 1 Hypofunctional abnormalities

Brain area	10/10 Designation	Behavioral correlate of that area
Left prefrontal	FP1	Attention
Right prefrontal	FP2	Judgment, response inhibition
Left frontal	F3	Motor planning right hand
Right frontal	F4	Motor planning left hand
Left lateral frontal	F7	Expressive speech
Right lateral frontal	F8	Expression of emotions
Left mid-temporal	T3	Verbal memory
Right mid-temporal	T4	Emotional memory
Left central	C3	Sensorimotor integration right hand
Right central	C4	Sensorimotor integration left hand
Left parietal	P3	Cognitive processing of language
Right parietal	P4	Cognitive processing of spatiotemporal information
Left posterior temporal	T5	Verbal understanding, word recognition
Right posterior temporal	T6	Emotional understanding, word recognition
Left occipital	O1	Visual processing right hemifield
Right occipital	O1	Visual processing left hemifield
Mid-frontal	Fz	Working memory
Mid-central	Cz	Sensorimotor integration midline legs
Mid-parietal	Pz	Cognitive processing, Interhemispheric (thinking speed)

Table 2 Hyperfunctional abnormalities

Brain area	10/10 Designation	Clinical symptom which correlates
Left frontopolar	P1	Hyperfocused ADD
Right frontopolar	FP2	Hypersensitivity, impulsive behavior
Left frontal	F3	Spasticity, right upper extremity
Right frontal	F4	Spasticity, left upper extremity
Left lateral frontal	F7	Excessive speech activity
Right lateral frontal	F8	Excessive emotional responses
Left mid-temporal	T3	Rumination (ideas), auditory hallucinations
Right mid-temporal	T4	Rumination (emotions, music) auditory hallucinations
Left central	C3	Tics, tremors, obsessive/compulsive behavior
Right central	C4	Tics, tremors, obsessive/compulsive behavior
Left parietal	P3	Complex hallucinations (auditory and visual), increased tone right upper extremity
Right parietal	P4	Complex hallucinations (auditory and visual), increased tone left upper extremity
Left occipital	O1	Visual hallucinations
Right occipital	O2	Visual hallucinations
Mid-frontal	Fz	Spasticity lower extremities
Mid-central	Cz	Tics, tremors, obsessive/compulsive behavior
Mid parietal	Pz	Complex hallucinations (auditory and visual), increased tone of legs, gait difficulty

Many of these localized hyperfunctions had been previously identified using fMRI or other imaging procedures before being correlated with an excess of high frequency beta activity in that area

The third type of abnormality which is clearly delineated with QEEG is disconnection. Geschwind (1965a, b) introduced this terminology to describe clinical syndromes resulting from lesions of association pathways that spared the connected brain areas. The first such syndrome he called conduction aphasia, which resulted from a stroke affecting the connections of Broca's area (roughly F7) and Wernicke's area (roughly T5). He also described sensory-motor, sensory-limbic, and sensory-Wernicke's disconnections (tactile anomia, pure word deafness, pure alexia without agraphia, and modality-specific agnosias). These disconnections can now be recognized using QEEG, and

they can probably be addressed, using coherence training, to attempt to normalize the coherence and re-establish the disrupted connections.

One result of performing QEEG's in a variety of disorders has been the finding that disconnections are quite common and numerous in several common clinical disorders (Walker et al. 2007), congenital learning disabilities, closed head injury (Walker 2007), dyslexia, (Walker and Norman 2006). These include epilepsy (Walker and Kozlowski 2005), and autism (Coben 2007). Our experience has been that one must normalize the power abnormalities (both hypofunctional and hyperfunctional) and the coherence abnormalities to most effectively address these disorders. In our clinical experience, standard protocols using one or two electrode placements and reward bands have not been very helpful for these disorders.

The fourth type of abnormality commonly seen using QEEG is hyperconnection. This type of abnormality could not be recognized with the older imaging modalities, but they are usually recognized with QEEG, resulting in hypercoherence. Developmentally, these hyperconnections may arise from failure to prune connections or they may occur after brain injury of one kind or another, where the involved connections are spared, but there are disruptions (disconnections) from other brain areas. An example would be the combination of frontofrontal hyperconnectivity with frontal disconnection from other brain regions in autism (Courchesne and Pierce 2005). We believe one must address the hypofunctions, the hyperfunctions, the disconnections, and the hyperconnections to best approach a complicated disorder such as autism. We again have found 5–7 sessions adequate for addressing these types of abnormalities.

The addition of single Hz bins has helped interpretation in two ways. First, some patients who had normal activity in a given frequency band (delta, theta, alpha, beta) have now been found to have an abnormality restricted to one or two single Hz bins, which was averaged out when one looked at the whole band. For example, the only two patients who did not respond to our training for dyslexia (Walker and Norman 2006) were first examined with the John database, which indicated normal delta activity (1–3 Hz). When we retested those two patients using the new Neuroguide database, they were found to have excess 1 Hz in several areas, but normal 2 and 3 Hz activity. When we then trained them to normalize the excessive 1 Hz activity, they were soon reading at grade level. The second advantage of the single Hz bins is that more specific training can be done to target only those frequencies that are abnormal. Our impression is that training in this way goes more quickly and more effectively than training entire frequency bands.

References

Catani, M., & ffytche, D. H. (2005). The rises and falls of disconnection syndromes. *Brain, 128*, 2224–2239.

Coben, R. (2007). *Autistic spectrum disorder: A controlled study of EEG coherence training focused on social skill deficits*. Presented at the annual meeting of the International Society for Neurofeedback and Research. San Diego, CA.

Courchesne, E., & Pierce, K. (2005). Why the frontal cortex in autism might be talking only to itself: Local overconnectivity, but long-distance disconnection. *Current Opinion in Neurobiology, 15*, 225–230.

Geschwind, N. (1965a). Disconnexion syndromes in animals and man. I. *Brain, 88*, 237–294.

Geschwind, N. (1965b). Disconnexion syndromes in animals and man. II. *Brain, 88*, 585–644.

Walker, J. E. (2007). A neurologist's experience with qeeg-guided neurofeedback following brain injury. In J. R. Evans (Ed.), *Handbook of neurofeedback* (pp. 353–361). Binghamton, NY: Haworth Press.

Walker, J. E., & Kozlowski, G. P. (2005). Neurofeedback treatment of epilepsy. *Child and Adolescent Psychiatric Clinics of North America, 14*, 163–176.

Walker, J. E., Kozlowski, G. P., & Lawson, R. (2007). A modular activation/coherence approach to evaluating clinical/QEEG correlations and for guiding neurofeedback training: Modular insufficiencies, modular excesses, disconnections, and hyperconnections. *Journal of Neurotherapy, 11*, 25–44.

Walker, J. E., & Norman, C. A. (2006). The neurophysiology of dyslexia: A selective review with implications for neurofeedback remediation and results of treatment in twelve consecutive patients. *Journal of Neurotherapy, 10*, 45–55.