Potential QEEG Markers of Childhood Stuttering And Their Implications for Neurofeedback

by Christopher Fisher, PhD on May 16, 2009 in Disease | Disorders, Neurofeedback, QEEG

Researchers compared the quantitative EEGs (QEEG) of 26 children with a history of stuttering to 21 age matched controls with no stuttering and may have identified important “EEG markers” of pediatric stuttering*. The authors’ epidemiological review of pediatric stuttering finds that this disorder afflicts approximately 1% of prepubertal children typically between 2 to 7 years of age with an peak onset around 3 to 4 years old with boys being 3 times more susceptible to this disorder.

In this cross-sectional case control study, children aged 3-12 years received a QEEG using the International 10–20 system during rest and hyperventilation states. Frequency band were defined as: delta (0.5–3 Hz), theta (3.5–8 Hz), alpha (8.5–12 Hz), and beta (12.5–30 Hz). No subjects in experimental group (male = 20, average age = 6.9; age range 3-12) or control group (male = 14, average age = 8.8; age range 4-11) exhibited epileptiform activity.

Visual examination of the raw EEG found increased parieto-occipital slow waves in 4 participants with stuttering and 1 in the control group. One child with stuttering had obvious fronto-central asynchronic slow waves.

An overall summary of the QEEG analysis, irrespective of montage, was that children who stuttered exhibited significantly increased delta and theta and reduced alpha and beta when compared to controls. These differences were apparent during resting background activity and at hyperventilation, except that theta increased only during hyperventilation. QEEG analysis of hemispheric asymmetry at rest found significantly more delta in the right frontal regions (56.6 ± 16.2%) than in the left (49.8 ± 16.7%) for those who stutter. Compared to the control group, children who stutter exhibited increased delta in the right front regions (56.6 ± 16.2% versus 41.8 ± 17.7%, p = 0.004) and parietal regions (47.9 ± 25.5% versus 27.9 ± 15.7%, p = 0.003) at rest, and increased delta (45.3 ± 21.2% versus 34.3 ± 10.5%) in the right parietal region during hyperventilation. Children who stutter also had significantly lower alpha in the left and right frontal regions at rest and in the right frontal region during hyperventilation, as well as significantly lower beta in the left region during rest and in the bi-lateral temporal regions during hyperventilation.

The researchers summarized these results as:

“The results of QEEG analysis revealed increased slow wave, especially delta, activity both of the recordings from resting state and hyperventilation in the children with stuttering compared to the controls. These observations were supported by decreased alpha and beta activity all of the brain recordings,
especially from right frontal and bi-temporal regions. Our results suggested a possible role of right frontal….dysfunction during resting state and bi-frontal neuronal dysfunction during hyperventilation.” (pg. 278).

Although the authors did not discuss the treatment implications for neurofeedback, there is a wealth of data here that suggests that stuttering may be amenable to neurotherapy. It appears that neurotherapists could target the overall excessive delta/theta with appropriate inhibit protocols and deficient alpha/beta with relevant enhance protocols. I tend to use inhibit protocols first and only enhance as needed. Restoration of the significantly deviant delta asymmetries may represent an additional neurofeedback protocol. I am cautious to enhance delta so I might inhibit the right hemisphere before I would enhance on the left. Excessive delta in the right hemisphere (frontal/parietal regions) suggest a possible “disconnect” from the left brain and overall slow information processing. Additionally, deficient beta in the left hemisphere could be a particularly important target since this side the brain handles language for most persons.

Of course, the above neurotherapy protocols are speculative and need further research to support their use. Responsible neurotherapy should only be conducted based on the patient’s presenting symptoms and personal EEG, not generic protocols found on the internet.

Reference: