Neurofeedback for Cerebral Palsy

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Cerebral palsy (CP) is a non-progressive motor disease occurring in young children after birth trauma or intrauterine pathology. Abnormal motor reflexes and abnormal motor development characterize it. You frequently see ataxia, hemiplegia, paraplegia, seizures and either muscle flaccidity or muscle spasticity. In athetosis cerebral palsy you see ceaseless slow, involuntary muscle movements occurring.

In an EEG study (Al-Sulaiman, 2001) of 151 children with cerebral palsy, 81 had seizures and 70 were seizure free. Epileptiform activity included spikes, sharp waves, and polyspike-wave complexes. In the CP children without seizures, there were synchronous slow waves. At the pediatric medical school in Nagoya, Japan, Maruyama and colleagues (2002) found that the EEG, if used on either the first or second day after birth, could predict the development of cerebral palsy in 295 children.

Since no two cerebral palsy cases are alike, controlled research with EEG feedback does not exist. I have treated individuals with cerebral palsy for over 25 years and have seen hundreds of these cases. I have done neurologically specific site feedback with either analog or Neuropathways all digital real time EEG and documented their EEGs. In these EEGs I have also seen excessive slow wave activity, mostly in the theta range, and when there is severe damage there are also delta waves.

Knowledge of the neurological reflexes is critical when using feedback with these individuals. This knowledge, in conjunction with real time feedback and MRI, CT and PET scans constitutes the armamentarium necessary to approach this clinical entity. Bipolar instrumentation is necessary to eliminate artifact from muscle spasticity. The EEG protocol is always neurologically based and site specific. For example, if the child can stand but not walk, we place the electrode over the leg areas on the sensorimotor cortex with one electrode on the sensory cortex and one electrode on the motor cortex, and the
ground on the ear. Remember that sensory input determines motor output. We then inhibit the abnormal theta activity and gradually lower the theta micro-voltage level until the EEG begins to normalize. I never enhance beta in cerebral palsy because as the beta increases, so does the delta and theta activity. I always monitor the beta by leaving the threshold at 0.4µV, which is lower than the beta voltage found in coma.

Tonic spasticity comes from the upper cerebral cortex, so focusing on neurological site specific inhibition of abnormal theta on the leg, arm and facial areas of the sensorimotor cortex brings improvement in motor development and function. Paradoxically, in cerebral palsy resulting in flaccid paralysis, inhibition of theta also brings more muscle flexion and more normal development. If there is clonic spasticity, which comes from the brain stem, I inhibit theta activity just below O1 and O2 (but still on the skull). The result is less athetosis. Cerebral palsy with epilepsy requires inhibition of theta on T4-C4 and T3-C3, F4-T4 and F3-T3. I spend 15 minutes on T4-C4 and 15 minutes on T3-C3, inhibiting theta activity all in the same session.

My best success story is a child named Jamie who came to me when he was nine years old. He was unable to talk, blind but unable to read Braille because he could not feel the letters and he had tonic spasticity. The school district had labeled him autistic, severely mentally retarded, cerebral palsy, and uneducable. After a period of inhibiting theta activity at neurological site specific areas such as legs, speech centers, arms and others, Jamie is now considered to be functioning at a genius level. He talks, feels, composes music, dances, and plays the piano. Now in his twenties, he composed an original classic song for me which he entitled, “The Wizard of Ayers.”

Sensorimotor inhibition can be obtained solely by inhibiting theta activity on the appropriate sensory motor areas, utilizing bipolar hookup and analog or all digital real time feedback. Cerebral palsy is one of the most rewarding neurological challenges in feedback.

REFERENCES
