

SPECIAL ISSUE

The Disconnection Syndrome

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This article explores the disconnection syndrome and its historical development relevant to applications of interest to neurofeedback providers. Tracing the roots of this syndrome back to its discoverer, Norman Geschwind (1926–1984), the article examines the neural foundations of the syndrome. Geschwind's work provides the theoretical foundation for numerous neurofeedback applications, in particular the relatively new use of coherence training. Although Geschwind's initial findings were drawn from lesion studies, he also extended the concept to acquired functional disconnections. Studies using neurofeedback indicate improvement without regard to the etiology of the disconnection. The terms hyperconnectivity and hypoconnectivity, familiar to contemporary neurofeedback providers, are discussed with respect to their relationship to this syndrome. A case example illustrates how neurofeedback reduced symptoms in a young man with adult-onset schizophrenia. Future studies are likely to build on these foundational concepts and extend neurofeedback applications to treat various inherited, induced, and functional disorders rooted in disruption of neural connectivity.

Neural Foundations of the Disconnection Syndrome

Joel Lubar made the following comment during a lecture in the 1980s: "When you conduct neurofeedback with bipolar sensors at P3, which is roughly over Wernicke's area, and F3, which is roughly over Broca's area, you'll frequently notice improvements in speech and language." I recall Lubar's statement clearly and recall pondering the profound implications of that statement. At the time, I had completed my first of 2 years of neuropsychological assessment study, and I understood there to be a class of impairments called *disconnection syndromes*. The term *disconnection syndrome* was coined in 1965 by neurologist Norman Geschwind (1926–1984) to describe a condition in which information transfer between parts of the brain is interrupted or blocked.

Aphasia, and the many subtypes of this disorder, was the best documented disconnection syndrome. The various subtypes of this disorder express themselves as impairments

in ability to produce or understand language. Lubar was discussing a form of aphasia that involved neurofeedback remediation in a pathway of fibers called the arcuate fasciculus, which links Broca's area to Wernicke's area (see the Figure).

Damage from a lesion or infarct to the arcuate fasciculus yields a disconnection syndrome called conduction aphasia. This type of aphasia presents, for example, as patients knowing the meaning of a word but being unable to pronounce it correctly or displaying the inability to articulate words they hear. The pervasive belief at that time, which continues to the present time, was that this and other types of aphasia have no known cure.

Lubar's observation began the evolution of protocol developments to document improved language functioning using neurofeedback. This finding began the elucidation of a theoretical foundation for neurofeedback applications in the rehabilitation of disconnection syndromes. Because of the dedication and cooperation of many individuals who walk in the shoes of pioneers such as Barry Sterman, Joel Lubar, and Joe Kamiya, a number of studies supported by very little funding have continued to show efficacy in ameliorating speech and language disturbances, traumatic brain injury, and dyslexia through neurofeedback therapy, guided by quantitative electroencephalography (QEEG). Aided by the development of better diagnostic tools, more precise amplifiers, and continuing refinement of QEEG, research is providing increasing support for powerful and exciting applications of neurofeedback to treat disconnection-type disorders.

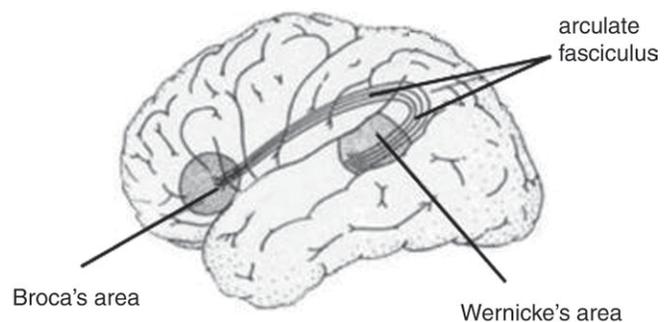


Figure. Broca's area, Wernicke's area, and the connecting fibers called the arcuate fasciculus.

The hypothesis relating disconnection syndromes and neurofeedback is based on a fundamental principle of neuronal activity known as Hebb's rule, named after Donald Hebb (1904–1985). His theories describe the basic mechanisms for the principle that the strength of neuronal connectivity is ever-changing as various neurotransmitters modulate delicate feedback loops within the central nervous system. This dynamic of interneuron communication helps us to understand how memory and learning occur and are essential to the understanding of neuroplasticity and the fact that experience changes the brain. Hebb is the source of the often quoted phrase "when neurons fire together they wire together." In *The Organization of Behavior* (1949), Hebb wrote, "Any two cells or systems of cells that are repeatedly active at the same time will tend to become 'associated', so that activity in one facilitates activity in the other" (p. 70). In addition, he stated that "when one cell repeatedly assists in firing another, the axon of the first cell develops synaptic knobs (or enlarges them if they already exist) in contact with the soma of the second cell" (p. 63). The reader is also referred to Zaidel, Zaidel, and Bogen (n.d.) for further discussion of disconnection syndromes.

Within the past 10 years, coherence-training neurofeedback has been added to the repertoire of the neurofeedback therapist. First emerging on most of our instruments as two-channel sum training, which rewarded the envelope of the summed signal, it has evolved to be a metric calculated with respect to the exact morphology, frequency, and amplitude of the respective brainwaves. Coherence training is a powerful tool that, properly used, can teach the brain to facilitate or, if desired, inhibit interneuron connectivity. Preliminary studies indicate that operant conditioning of the phase relationship in the EEG between pairs of sites in the cerebral cortex may yield the most dramatic and robust results seen in the relatively short history of our field. As QEEG-guided neurofeedback is validated as a tool to normalize both interhemispheric and intrahemispheric disconnection patterns, this could well be the application facilitating universal acceptance of neurofeedback as a neurorehabilitative tool.

As this technology advances and more studies demonstrate the efficacy of neurofeedback with respect to remediation of disconnection syndromes, proper funding will likely become available to support the evolution of this technology. This optimism is supported by two important facts. The first is the fact that traditional medicine and psychotherapeutic interventions are largely ineffective in remediating problems created by disconnection syndromes. Neuropsychological testing, assessment of function, and brain imaging may be

excellent at describing impairments, but the rehabilitation of them is thought to be nonexistent. The second related reason for optimism is that because there are no well-funded mainstream solutions, the chance that scientific research will get noticed and supported is greatly improved. As an example, look at the notoriety Rob Cohen has received for his breakthrough work with autistic spectrum disorder (Cohen & Padolsky, 2007), a study my office is currently replicating. I believe neurofeedback would be much further advanced had we not come up against established domains, as exist with neurology claiming ownership of the treatment of seizure disorder and psychiatry asserting a similar exclusive right to treat attention-deficit disorder (ADD). In both cases, well-established financial interests, as well as entrenched academics, have been successful in marginalizing neurofeedback applications with respect to either seizure or ADDs.

Not only does neurofeedback bring about improvement in functioning, but because of the primacy of cerebral connectivity to optimal brain function, the degree of improvement is large and, at times, profound. As our understanding of this application grows and we apply it to various impairments rooted in disconnection syndromes, we will likely see an expansion of the scope of disorders treated. Although preliminary, neurofeedback has shown some efficacy with respect to pilot or case studies in stroke, autism, traumatic brain injury, left-right or anterior-posterior disconnection syndromes, fetal alcohol syndrome, and learning disorders including dyslexia, dyscalculia, apraxia, and aphasia. Improvement is seen in diseases with known and unknown etiology. Improvement is not limited to one etiology; rather, it includes disconnection syndromes resulting from inherited disorders (such as dyslexia and central auditory processing disorder), those induced by trauma (such as traumatic brain injury, acquired aphasia), and those disorders arising from developmental exposure to toxins or disease (such as autism, stroke, or multiple sclerosis).

After years of study of the human brain, we remain at the beginning of our understanding of the intricate relationship between the areas of the brain known as the gray and white matter. The fact that we still call these by the color they appear to the human eye and that we call the tissue *matter* is indicative of the elementary level of our understanding. Neuroscientists once accepted as fact that higher cortical functions were exclusively the domain of the brain's gray matter. In the late 19th century, as we better understood multiple sclerosis, this understanding was replaced by the belief that it was the white matter that mediated and determined the degree of competency in a

wide range of brain functions. As we proceeded into the 20th century, the consensus was that the most pervasive disorders affecting cognitive performance and emotional stability were associated with disturbance in the heavily myelinated outer 3 mm of the brain called white matter.

It was not until the 1980s, when brain imaging methods such as magnetic resonance imaging and QEEG technology developed, that a more integrated and complex understanding emerged. Contemporary thinking is that the gray matter mediates and coordinates (gates) linkages of neuronal ensembles between regions of the brain's white matter as well as the deeper brain structures to produce a kind of coordinated symphony of behavior. Today we understand that many impairments in human behavior, cognition, and emotion stem from electrical or blood flow disturbances and/or disconnection patterns (lesions) in the gray or white matter as well as in deeper brain structures such as the basal ganglia, amygdala, thalamus, and even the brain stem and cerebellum.

One thing is clear: myelinated white matter connects cortical and subcortical regions as well as lateral interhemispheric regions via distributed networks that gate electrical brain activity in a way that supports higher cortical function. Impairments in specific areas of the brain that are critical to a particular ability (expressive speech, decoding, social prosody), or a breakdown or sheering in the critical pathways connecting these areas, will show up as a problem. Either of these can be a source of impairment affecting the degree of competency (fluency in speech, proficiency in reading, ability to make friends).

As stated earlier, Norman Geschwind was the first to describe the disconnection syndrome. He was a Harvard Medical School graduate, professor of neurology, and director of research at the Boston University Aphasia Research Center and is considered by many to be the father of contemporary behavioral neurology. His seminal work, titled "Disconnexion Syndromes in Animals and Man," was published in the journal *Brain* (Geschwind, 1965). Geschwind explained how disconnection syndromes are involved in disorders such as aphasia, dyslexia, and cerebral asymmetries and explored the control of seizures by severing neural pathways that facilitate kindling. His discoveries and those of his students influenced much of what we have come to accept as fact in terms of the importance of connectivity in the brain.

Perhaps of interest to the readers, Geschwind was a huge proponent of interdisciplinary research, including the work of speech and language experts, evolutionists, and cognitive and behavioral psychologists. Also, much of his work was accepted based on single case studies, an approach to research

that today is dismissed by the academic establishment as not having any scientific merit.

Geschwind defined the disconnection syndrome as higher function deficits that result from white matter lesions or lesions of the association cortices, the latter acting as relay stations between primary motor, sensory, and limbic areas. Although challenged, his theories have remained substantially foundational to the understanding of neural impairments. Of course, some of his statements have been discovered to lack a level of sophistication gained through further research and the development of better tools. Yet much of what he taught has been verified and expanded on. He was truly a man who deserves credit for so clearly demonstrating and teaching a fundamental principle of the brain: Disconnection yields dysfunction.

Disconnection syndromes in neurofeedback nomenclature are called hypo coherence abnormalities in the QEEG and are considered significant if they are abnormal by more than plus or minus two standard deviations when compared with the age-matched database (Z-score transform). However, one must not only look at values based on statistical analysis but also be aware that patterns of dysfunction can be present when regions or areas necessary for more complex neural competencies are merely borderline significant in abnormality. In the raw EEG, hypo coherence can be seen when two waveforms that should look very similar are not, according to age-matched norms.

An important development since the time of Geschwind's foundational work, and of importance to neurofeedback therapists, was the discovery of disorders related to hyperconnectivity or hypercoherence. Today we understand that the lack of proper (age-matched) differentiation in a brain also yields impairment in functioning. Hypercoherence is said to be present in the EEG when two brain sites or areas are overly connected, as indicated by the two waveforms at these different sites being more similar in terms of morphology than an age-matched normal subject. Hypercoherence can be thought of as a kind of immaturity wherein cortical areas do not specialize and take on specific abilities and thus appear overly similar to each other.

Although it may be helpful to think of hypercoherence as a type of immaturity, this impairment is not limited to developmental or maturational delays: It may be acquired as a result of trauma or toxic exposure. It is accepted by most experts in our field and related fields that hyperconnectivity can yield at least as serious an impairment, if not greater, as disconnection syndromes (hypoconnectivity). The commonly accepted nomenclature in our field is that we call disconnection syndromes *hypo coherent* and areas that are undifferentiated or overly connected *hyper coherent*.

Cautions

Coherence or comodulation training is the tool used by the neurofeedback clinician to remediate problems related to hypercoherence or hypocoherence and thereby normalize brain function. Unfortunately, nowhere in our field is our diversity more evident than in our lack of agreement as to a definition of coherence or the best way to train it. We also lack agreement as to how it is best measured and its clinical significance. I view this as a temporary but confusing issue, especially to people new to the field. As many of us have come to accept, our field is one that is evolving, and we should remain confident that these issues, like others in the past, will be resolved. An announcement was made at the International Society for Neurofeedback and Research (ISNR) held in San Antonio this past August that a new committee is being formed, and ISNR will invite the IEEE to become involved in assisting us to set standards not only in instrumentation but also nomenclature. The IEEE name was originally an acronym for the Institute of Electrical and Electronics Engineers, Inc. Today, the organization's scope of interest has expanded into many related fields. This will likely yield positive results in terms of decreasing the ambiguity existing in our data acquisition devices, assessment tools, and training instruments.

I do not recommend that anyone engage in coherence neurofeedback training without obtaining a QEEG to guide the treatment. Also, once a QEEG is done and indicates that hypercoherence or hypocoherence problems exist, it is important to know if the EEG training instrument one is using will train coherence in the same way the QEEG measured it and found it to be abnormal. This problem will hopefully be cleared up as experts in our field agree on definitions and measurements of coherence and build these into our equipment as defaults. I cannot emphasize enough the importance of being educated with respect to what it is you are doing and to apply this technique with great caution. Coherence training is many times more powerful than amplitude training, and therapists must exercise caution in its application. As powerful as this technique is for positive change, so is its misuse likely to show itself to be a technique to create dysfunction.

Although Z-score training claims to manage many concerns, especially that of overtraining or undertraining, this assertion has yet to be scientifically demonstrated to the satisfaction of many in the field. It may be good to remember that any Z-score training application can be no more reliable than the QEEG database on which it is derived. I do not believe there are any highly reliable QEEG databases readily available to us. This is not to suggest that the QEEG is not a helpful tool, but by standards typically

used by neuropsychologists to evaluate the validity and reliability of tests (called *psychometrics*), all of the U.S.-based QEEG databases fall short in many areas. This is particularly true for children whose brains develop capacities quickly, with differing competencies coming online for them within genetically programmed time frames that differ from family to family. Also lacking and not likely to be easily ascertained is the best method to apply Z-score training. Simultaneously applying powerful protocols to various regions of the brain to bring them into a range of statistical normality without regard to the interaction effect, or without attention to which subsystems of the brain this training may affect, may yield effects that are not desired. To illustrate my point, let us consider drug therapy. One would predict that a membrane-stabilizing drug, which reliably decreases seizures, when combined with an amphetamine that consistently increases attention in an individual with ADD, would yield both decreased seizures and improved attention. However, medications when taken in combination often do not yield predicted results. Given this knowledge, it is highly likely that coherence training applied simultaneously to different pairs of neuronal ensembles would yield unpredictable and, at times, undesirable effects. The brain is a complex system, and as chaos theory predicts, changes to one area likely affects multiple areas, making it that much more difficult to presume outcome when many changes are being asked of the brain simultaneously. Although the concept of Z-score training has a lot of face validity, careful study of its application is warranted. This is not a type of training I would advise independent clinicians to engage in without research support. In keeping with what I stated earlier, many of these issues will likely disappear as the science of this application evolves, our databases become more robust, and our instruments train in accordance with the QEEG metric that indicated impairment.

Also, we need a valid and reliable real-time measure of coherence that has universal acceptance within our field. This will solve the problem of not knowing how many sessions to administer, unless one requires every treatment to run 15 to 20 sessions, which often is not practical. Because of these and other issues, it is advisable to exercise caution when deciding to initiate coherence training and to do so only under the supervision of someone who has experience and is aware of the uses and limitations of each of the QEEG databases.

Historically, neurofeedback therapists have been in a field in which we have enjoyed many positive outcomes despite differing approaches, theories, and protocols. I attribute this to the miracle that is our brain. One group currently encourages rewarding 40 Hz along the sensorimotor strip,

another advises us to reward very low frequencies, and still another purports to achieve benefit from generally quieting and stabilizing all the frequencies available in the human EEG. Viewed through the eye of evolution, it would make sense that our ancestors who were most able to take in information (adaptation) and use it to enhance survival (accommodation) would add their genetic patterns to the gene pool. In keeping with this gift of evolution, it would stand to reason that just about anything we do in the amplitude domain could be reliably predicted to have either a positive effect or be ignored by the brain. This model elevates the brain to the status of an organ capable of knowing when feedback is useful, as opposed to when it is not, and capable of displaying different reactions depending on the frequency, intensity, and duration of the signal requesting it to change. However, as stated earlier, because proper cortical connectedness is so vital to higher functioning, and with coherence training we are focusing primarily on surface cortical to cortical white matter signals, even subtle changes to the phase relationship of these regions can affect brain function profoundly. As those of us who have been doing coherence training have discovered, the incorrect application of coherence training can have profound short- or long-term consequences. Therefore, although I encourage the appropriate and ethical use of coherence training, I advise proceeding with great caution and supervision is essential.

The Power of Coherence Training

With these recommendations and concerns in mind, once you initiate coherence training, you will likely experience the same thing I did: amazement. You will be surprised by the degree and rate of improvement, as well as the greater range in the disorders you will be able to treat.

Further support for our brain's being a miracle organ in terms of its ability to assimilate and accommodate to its environment is provided by the many neuroplasticity studies completed since the decade of the brain was initiated (1990–2000). It appears from the literature that our brain retains its plasticity, and given the right feedback, the brain that is impaired due to trauma or other deregulating event can continue on a pathway of genetically programmed health. Where windows of opportunity were once thought to close, we now see that they do not close completely if given the correct type of feedback. Unstable brains benefit from neurofeedback that emphasizes stability training, underactive brains benefit from neurofeedback that activates higher frequencies and inhibits lower ones, and overaroused, stuck, or disconnected brains likewise benefit from applications of neurofeedback. Clinicians and researchers using coherence training are demonstrating

several orders of magnitude higher rates of improvement in significantly less time than was typical with amplitude training.

Following the work of “Dr. Coherence,” the late Joe Horvat, as well as that of the neurologist Jonathan Walker, I initiated coherence training in my office about 7 years ago. I began by clearly indicating to the patients that this was an experimental approach. Starting first with dyslexic patients, I got results that were incredible, just as other clinicians have reported; I saw improvements in the ability to comprehend, to attend, and to read more rapidly. Post-QEEG testing indicates that hypercoherence patterns in the frontal lobes of autistic children improve with coherence training. With these brain-imaging improvements, the autistic child can go from babbling to speaking in sentences in as few as 10 weeks, with demonstrated decreases in hypercoherence in their QEEG. For example, I applied coherence training with an Asperger's patient. After eight sessions of training, he made his first friend and now has an understanding of depth and subtleties of relationships that he never comprehended before. One 16-year-old boy went from reading at the fourth-grade level to reading at the ninth-grade level over a 4-month period of training. He did a combination of coherence training neurofeedback and the Lindamood-Bell program. An orthopedic surgeon who had a left-posterior temporal lobe stroke had to stop doing surgery because he could no longer discriminate detail using the lighted magnifying scope. He was able to go back to doing surgery using the scope after 12 weeks of coherence training.

Coherence Training With ADD/ADHD

I had often wondered why some kids who were medication nonresponders, who had very few QEEG amplitude disturbances, would present with severe attention-deficit hyperactivity disorder (ADHD) symptoms. I examined closely the fact that many of these kids had coherence abnormalities apparent in their QEEG. When I attended to these coherence problems and applied neurofeedback coherence training within this subgroup, I saw significant benefits. We know that more than 70% of those diagnosed with ADHD have at least one co-occurring condition that, in the past, we were often unable to treat. Now, we can use the QEEG coherence maps to treat these other co-occurring conditions that appear as coherence abnormalities. I have come to appreciate the fact that these individuals will typically show hypercoherence or hypocoherence problems particularly in the anterior regions. This serves to amplify their apparent level of ADHD, but careful diagnosis often defines a co-occurring condition such as an impulsivity problem, a problem involving central auditory processing,

or other learning disorder whose etiology is the result of disconnection-type impairment. It has now become routine in my office, which specializes in neurofeedback treatment of ADHD, autism, and other developmental and learning disorders, to have two phases of treatment. The first phase attends to the amplitude abnormalities and the second remediates coherence issues.

Case Example

Lastly, I want to share an exciting application of neurofeedback I carried out with a patient who, because of adult-onset schizophrenia, had to drop out of college at the beginning of his senior year. I conducted five serial QEEGs on him and each time treated any significant disconnections that the NeuroGuide indicated. With each QEEG, I received validation that the areas to which I had applied coherence training went from an abnormal standard deviation to a normal standard deviation. The patient had previously taken increasing amounts of Abilify, and with the neurofeedback training, his psychiatrist was able to reduce his medication. He completed 535 sessions of neurofeedback over 1½ years, receiving treatment three to six times a week. He is now on the lowest dose or completely off Abilify, and he graduated from the University of Southern California in May 2008.

I am in the process of preparing this case for publication. A single case study may not get any serious consideration by skeptics, but perhaps it will inspire others to take the work further. I was inspired to attempt this application because new research has pointed to schizophrenia as a problem rooted in the creation of disconnection patterns. At least for this patient, I hypothesize that we stayed ahead of his encroaching schizophrenia and, with neurofeedback, he became more and more competent and exhibited fewer symptoms. I can only speculate as to his future, but we know that adult-onset schizophrenia has a very poor prognosis, and perhaps in this case, the neurofeedback was able to see him through a difficult period of vulnerability. Perhaps symptoms will return, and he will return to treatment. However, this remains a powerful example of how neurofeedback can be a tool to remediate disorders rooted in disconnection abnormalities and may one day be a treatment for this pervasive and debilitating disorder.

Conclusion

We remain at the beginning of the application of neurofeedback to remediate disconnection syndromes. Fortunately for the field of neurofeedback, most of these disorders have not been known to respond to medication. Thus, we have an opportunity to avoid the struggle that has consistently plagued neurofeedback economically and politically, that is, having a powerful, wealthy, and influential pharmaceutical industry that opposes any treatment that does not emphasize drug therapy. Because many disorders are in some sense disconnection syndromes, we can look to neurofeedback as a new tool in the rehabilitation of disease—and a powerful one, at that.

Note

Gary Schummer is faculty at EEG Spectrum, Inc.

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