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SPECIAL ISSUE

Managing Traumatic Brain Injury: Appropriate Assessment and a Rationale for Using Neurofeedback and Biofeedback to Enhance Recovery in Postconcussion Syndrome

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Impairments that may result from a mild traumatic brain injury (TBI) or concussion can be both severe and long-lasting. This article will list some of the common persisting symptoms that may occur and give a brief description of the neuropathological processes that can be triggered by TBI, including diffuse axonal injury and its effects on the mitochondrial Kreb's cycle and the production of adenosine triphosphate, the brain's source of energy. This is followed by a summary of a comprehensive assessment process that includes quantitative electroencephalography, evoked potentials, heart rate variability (HRV) measures, neuropsychological testing, and blood and urine analysis. Details concerning a neurophysiological approach to effective treatment are given. These include conventional single-channel neurofeedback (NFB), also called brain-computer interface training, low-resolution electromagnetic tomography z-score neurofeedback, HRV training, and counseling on diet, sleep, and exercise. The authors expand the discussion on their treatment approach to include a neuroanatomical explanation of why the practitioner should consider combining the NFB training with HRV training.

Introduction

This article provides an overview of how postconcussion syndrome is assessed and, for the most part, successfully treated even a number of years after injury. It provides a clinical rather than a research perspective and is based primarily on work carried out at the ADD Centre and Biofeedback Institute of Toronto with clients who have suffered traumatic brain injuries (TBI). Success is measured in terms of patients having sufficient amelioration of cognitive and emotional symptoms to be able to resume and succeed in their social, academic, and/or employment pursuits. To date, this has been achieved in all cases in which the person did sufficient training (40 or more sessions). Eventually, we will follow this descriptive article with a case series detailing outcomes. (See also the initial case study by Bhandari, Thompson, and Reid-Chung, 2013, in this issue of *Biofeedback*). Here we summarize the most common presenting symptoms and the assessment process, including a rationale for the use of 19-channel quantitative electroencephalogram (QEEG), evoked potentials (also called event-related potentials [ERPs]), heart rate variability (HRV) measures, biomedical analysis, and neuropsychological testing. Also included is a short description of the treatment approach. Interventions may include single-channel neurofeedback, low-resolution electromagnetic tomography (LORETA) z-score neurofeedback,[1] HRV training, and metacognitive strategies, plus counseling regarding diet and supplements, sleep hygiene, and exercise.

Symptoms

There is nothing mild about the impairments that may result from a mild TBI, which is commonly called a concussion. A lack of objective pathognomonic signs of concussion means that a physician's diagnosis often rests, for the most part, on the patient's report of subjective symptoms, such as headache or "seeing stars." In 95% of cases in athletes, concussion does not involve loss of

[1]LORETA is a widely used statistical technique to analyze surface EEG signals and determine which sites deeper in the cortex are the sources of the surface electrical activity. Today's LORETA software produces instantaneous displays of cortical processes, usable for neurofeedback training. LORETA z-score training involves training cortical processes toward normalcy, based on a comparison to a normative database.

consciousness (McCrory et al., 2013). Common presenting symptoms shortly after a concussion include dizziness, nausea, headaches, balance deficits, and blurred vision. The diagnosis of concussion should be made by a physician in the period immediately following the trauma, preferably within a few hours of the injury. A designated health care professional with appropriate training may make the diagnosis in remote areas where there is no physician available (Tator, 2013).

The longer-term problems are referred to as postconcussion syndrome when symptoms persist for weeks or months, and this is more common after repeated brain injuries. Symptoms include fatigue, sleep disturbance, slowed reaction time, plus cognitive and emotional changes that produce difficulties in everyday life. The cognitive problems include slowed processing speed, poor sustained attention, poor concentration, impaired short-term and long-term memory, and learning problems. Language problems, including word-finding difficulties as well as poor comprehension, are also frequently observed among patients with TBI. Emotional changes may include depression, anxiety, labile mood, irritability, and anger ("a short fuse"). Unfortunately, in some cases, family and friends notice a major personality change to which the patient may be oblivious because self-perception can also be affected. It is not at all uncommon for someone to present years after the injury with all of these difficulties present to varying degrees.

Pathology of a TBI

A variety of neuropathological processes can be triggered by damage caused when brain matter collides with the rough, ridged inside edges of the skull or the tough falx cerebri, which is an infolding of dura mater between the hemispheres. With rapid deceleration, as in an automobile crash or when a football player is running at full speed and is tackled, there is a coup injury, where the brain first hits the skull, and a contrecoup injury, as it rebounds and hits against the opposite wall of the skull (see Figure 1). The results of this type of injury are usually assessed in the hospital with the more commonly used imaging techniques. These techniques may include positron emission tomography (PET), single-photon emission computed tomography (SPECT), magnetic resonance imaging (MRI), and functional MRI (fMRI). A PET scan measures glucose metabolism, and PET images provide a visual display of brain activity by detecting where a radioactive form of glucose goes while the brain performs a given task. SPECT scans, which track perfusion (blood flow), are significantly less expensive than PET scans, in part because they are able to use longer-lived and more easily obtained radioisotopes than PET. Both of these measures are invasive in the sense that radioactive isotopes must be used. MRI and fMRI have the advantage of not requiring any injection or ingestion of radioactive material, but they involve a noisy environment, and they do require the patient to lie very still in the tube while the magnetic imaging is being done. Charles Tator, a neurosurgeon and concussion expert in Toronto, in a review article states that routine computed tomography and MRI scans almost always appear normal in concussion (Tator, 2013). Even PET, which indicates tissue metabolic activity by virtue of the regional glucose uptake; SPECT, which measures blood flow in the brain; and fMRI, which measures the blood-oxygen level–dependent response that will vary with oxygen uptake, may fail to detect a common type of injury associated with TBI called diffuse axonal injury (DAI; Thatcher, 2012; Thatcher, Walker, Gerson, & Geisler, 1989).

Diffuse Axonal Injury

DAI is a primary pathologic feature of brain injury in concussions at all levels of severity (Kushner, 2001; Thompson & Hagedorn, 2012). DAI occurs both with hitting the head and also when there is rapid acceleration/deceleration and/or quick rotation of the brain such as may occur with a whiplash or a blast injury. DAI is caused by the twisting and stretching of axons or, in severe head injuries, the tearing or shearing of axons (see Figure 2). An axon is a nerve connection that goes from the cell body of one neuron to the dendrite or cell body of another neuron. It connects to the next neuron at a chemical junction called a synapse. When a neuron fires, the electrical impulse travels down the axon, at times a long distance, to the end of the axon, where it causes the release of a chemical called a neurotransmitter. This neurotransmitter enters the synaptic space between the end of the axon and the receptor sites on the dendrite of the neuron that is receiving the "message." One such neurotransmitter is glutamate, which is important in this discussion and is shown in Figure 3. Clearly, damage to an axon will interfere with the transmission of messages from one neuron to another. Although the biochemical and structural changes caused by DAI may not be detectable by MRI, EEG is able to detect the electrophysiological results of the disruption in communication between neurons.

The rotational forces affecting the brain that result in DAI also set off a metabolic cascade that includes a flood of neurotransmitter release, including glutamate. Glutamate excess will stimulate neurons to fire incessantly. This results in an increase in neuronal permeability with an

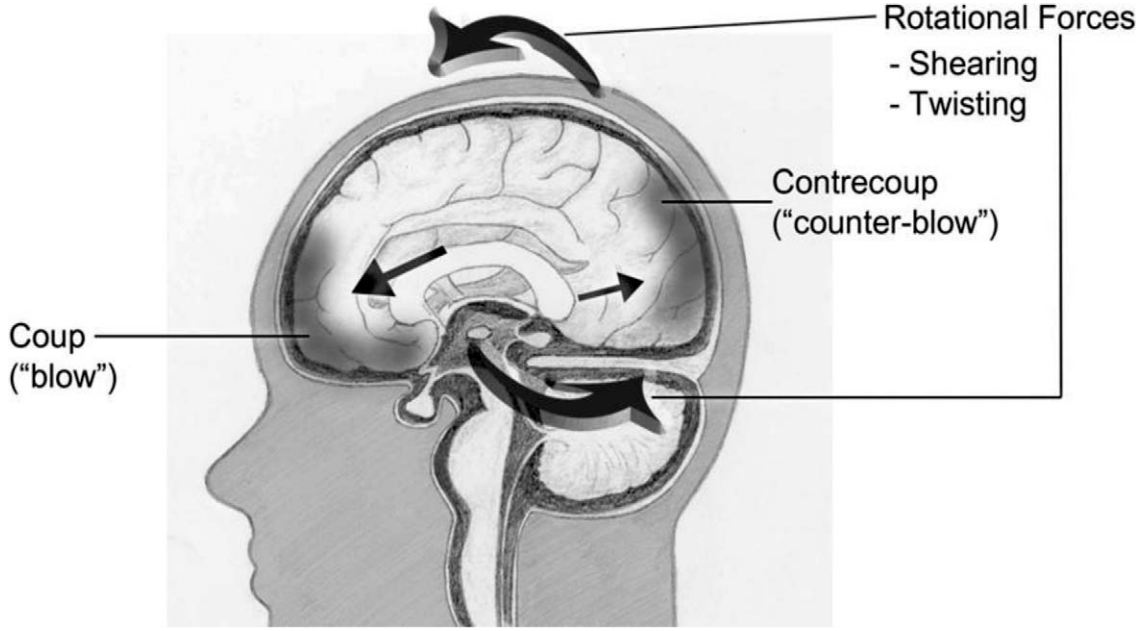

Figure 1. Example of sites of injury in traumatic brain injury. Drawing by Maya Berenkey from *The Companion to the Neurofeedback Book* (in press) using figures by Amanda Reeves in *The Neurofeedback Book*.

influx of sodium and calcium and a release of potassium. Both sodium (Na^{+}) and calcium (Ca^{2+}) excess can interfere with mitochondrial function. With normal blood flow and nutrition, the mitochondria can produce energy in the form of adenosine triphosphate (ATP) by means of the citric acid cycle (Krebs cycle). Energy is essential to life, and it is required in the cell membrane for the transport of ions against a concentration or electrical gradient. Without this

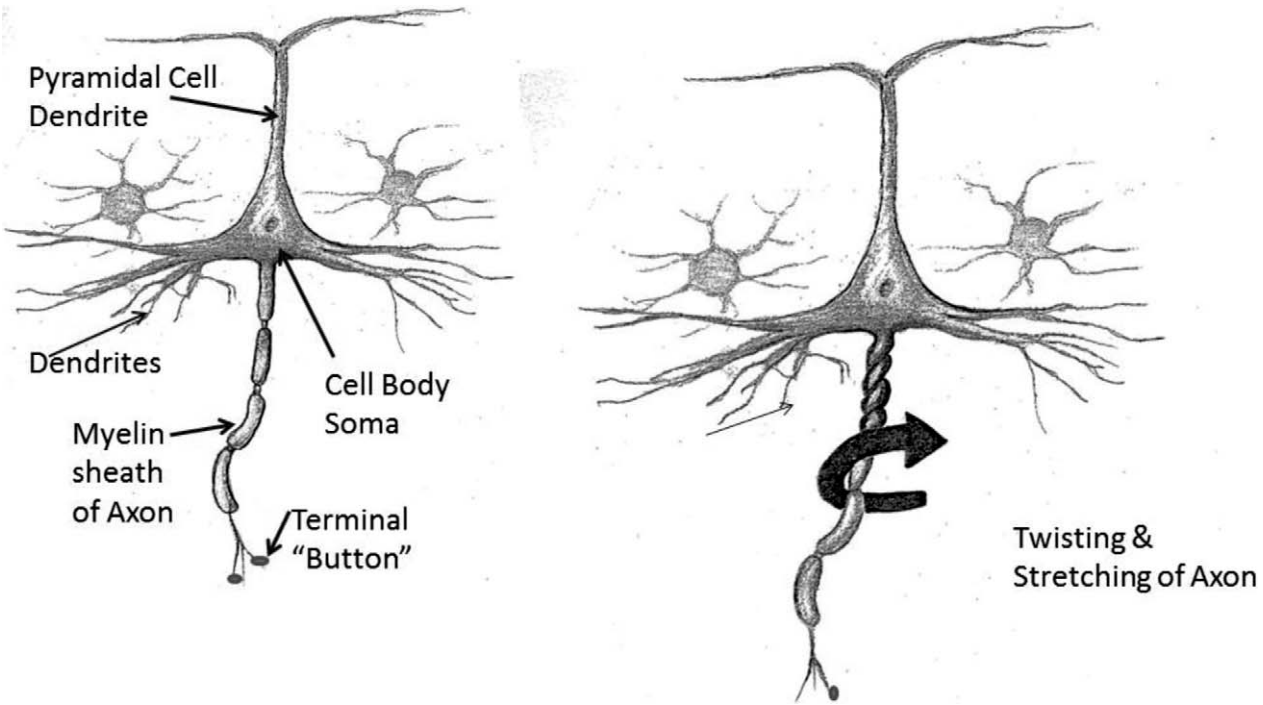

Figure 2. Structure of a neuron drawn by Maya Berenkey. The terminal button is the presynaptic link to the dendrite of the next neuron. This synaptic transmission is disrupted when there is diffuse axonal injury. (Note that this figure shows a pyramidal cell. Only pyramidal cells produce the extracellular currents that summate to produce the electroencephalogram.) Figure from *The Companion to the Neurofeedback Book* (in press).

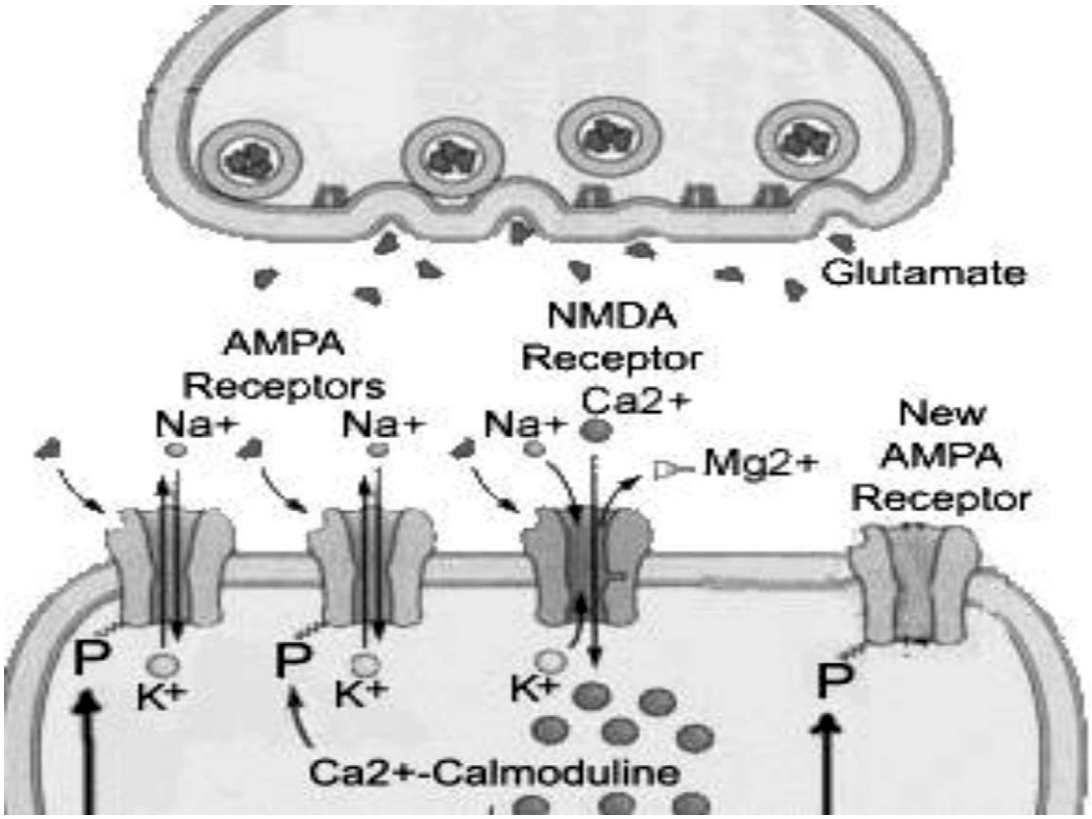

Figure 3. Schematic diagram of a synaptic junction. The figure shows, at the top, the presynaptic ending of the axon (called a "button" in Figure 2) and the postsynaptic receptor sites for glutamate: the α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor (AMPA), which is a glutamate receptor site, and the *N*-methyl-D-aspartate (NMDA) receptor site, which is initially blocked by magnesium ions. This figure is often used to explain long-term potentiation. It is illustrated here to show the synapse. Thanks are extended to Wikipedia Images; Bruno Dubuc, Website of Canadian Institutes of Health Research; Institute of Neurosciences; Mental Health and Addiction (NMHA); and to Copyleft; The content of the site The Brain from Top to Bottom is under copyleft. © is also from Nature Reviews, Neuroscience, Nature Publishing, 2005.

energy source, the cell cannot depolarize and repolarize normally. Cell death is the eventual result. With a shut down of the mitochondrial production of ATP, there is a reduction of energy production and a resulting energy crisis because, to keep firing, the neurons demand extra energy.

In addition to the direct interference with ATP formation in the mitochondria due to the influx of sodium and calcium, there is also an additional problem due to the vasoconstriction caused by the excess release of potassium. Potassium constricts the blood vessels and decreases blood flow to the compromised area in the brain. The mitochondrial Krebs cycle requires fuel to keep running. In simple terms, this fuel includes glucose, fats, amino acids for coenzymes, and vitamins for the enzymatic processes. However, the constriction of the blood vessels caused by the excessive release of potassium limits the supply of these necessary nutrients and oxygen to the cell.

The high-energy demand, restricted blood flow, and oxygen debt together create the aforementioned energy crisis that exhausts the neurons (see Figure 4). This will inevitably lead to symptoms such as mental confusion and failed memory, symptoms commonly seen in people who have had a concussion. The brain will take varying amounts of time to recover. The time to restore the chemical balance will vary between people and will depend on many variables including age, general health, and many other factors, such as nutrition. Children and females, for example, are more vulnerable to the effects of concussion, as mentioned by Tator (2013). Long-term potentiation is decreased, and long-term depression is increased. The membrane excitability is reduced.

The diagram in Figure 5, adapted after a figure from the UCLA Brain Research Centre, implies that within about 1 week, one may observe neurochemical recovery. However, time lines predicted by different authors do show a wide range. Because of changes and subsequent derangements in cerebral glucose metabolism, persons suffering from even minimal TBI may remain vulnerable to second-impact injury for periods of up to a few months (Giza & Hovda, 2001). Other authors note that hypermetabolism and an

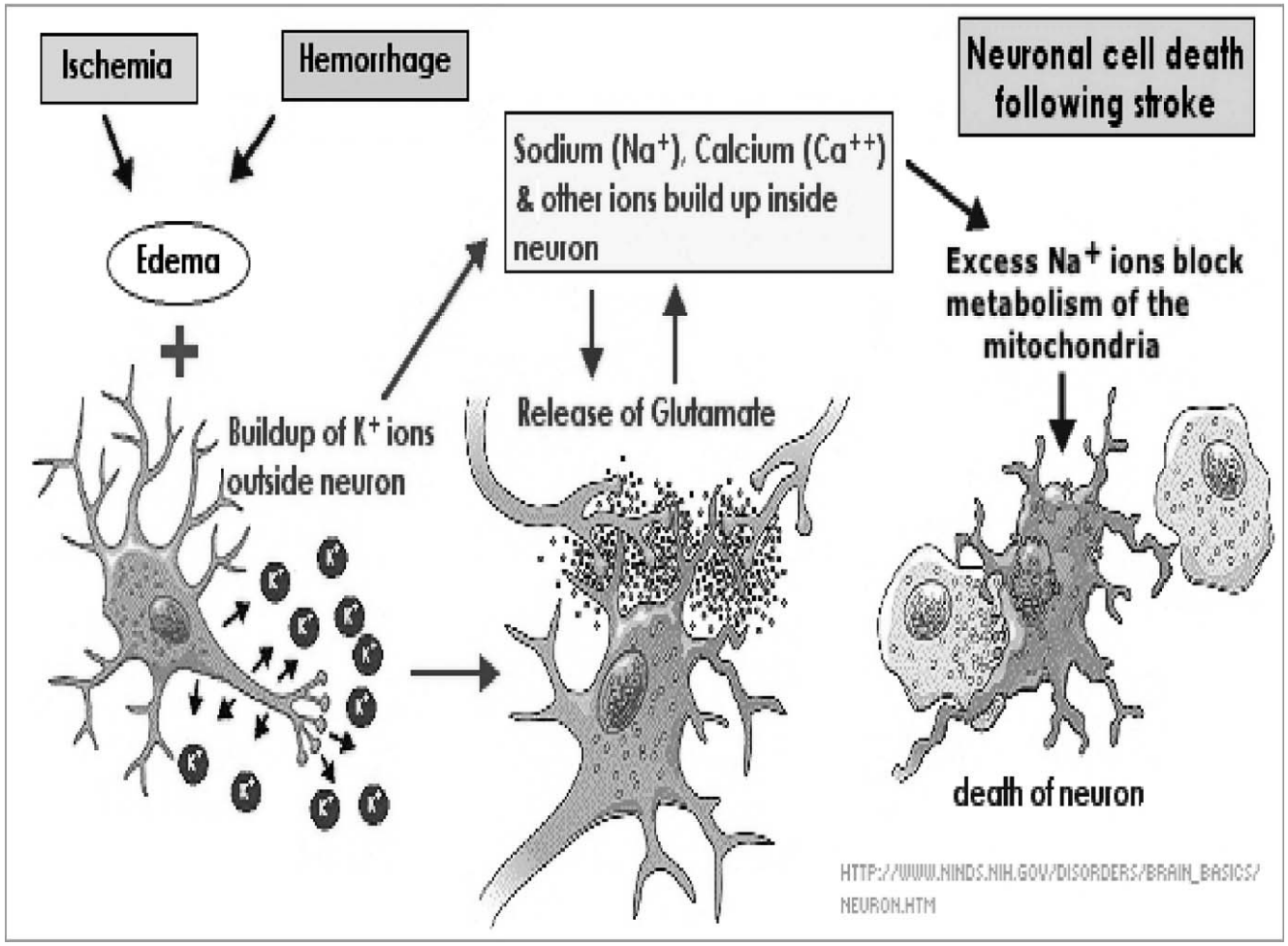

Figure 4. Steps contributing to neuronal cell death. National Institutes of Health (NIH) government document in public domain. Although originally used by the NIH to illustrate changes after a stroke, this diagram applies equally well to the energy crisis that ensues after traumatic brain injury. The next figure summarizes some of the factors that contribute to this energy crisis.

energy crisis lead to a secondary reaction with increased regional cerebral blood flow (rCBF; occurs after about 1 minute) to meet increased energy demands needed to restore ionic membrane balances (DeWitt et al., 1986; Meyer, Kondo, Szewczykowski, Nomura, & Teraura, 1970; Nilsson & Nordström, 1977). Shaw (2002) notes that this initial increase in rCBF is followed by a subsequent decrease of up to 50% in rCBF for 2 to 4 weeks depending on the severity of injury. Because of factors that include the gradual death of some neurons after DAI and the long-term decrease in CBF that means the energy crisis in cells cannot be resolved, it is possible for symptoms to worsen a few weeks after the concussion so that they are more serious than symptoms seen after initial neurochemical recovery during the first week. This may be a factor contributing to the susceptibility of athletes who have experienced one head injury to experience a second concussion in the period after an initial head injury. The results of subsequent head injuries can be more profound, even when the severity is less, and resolution of symptoms will take longer. *Second-impact syndrome* is a term used when the second injury occurs before there has been recovery from the first TBI, and it is a serious condition that involves cerebral swelling that results in death or major neurological deficits. This is why doctors have to be careful when making return-to-play decisions in athletes.

Assessment

General Considerations

Conventional imaging techniques may miss DAI in mild-to-moderate TBI. Further, conventional neuropsychological testing and symptom checklists, such as those traditionally used with injured athletes, have been found to normalize within a few days to a few weeks. For this reason, a more advanced paradigm is needed to detect this type of injury.

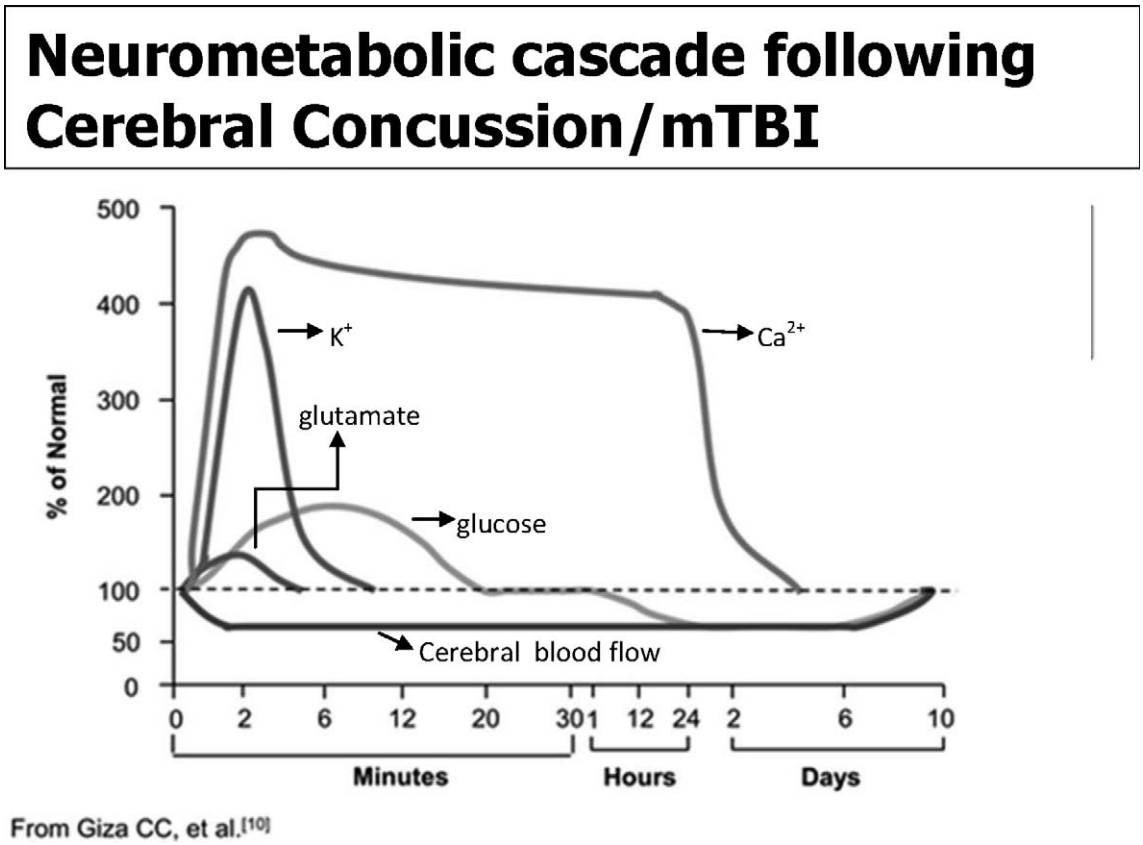

Figure 5. Chemical and metabolic changes after traumatic brain injury. Adapted from David A. Hovda, PhD, UCLA Brain Injury Research Center, and from a drawing in Giza and Hovda (2001).

In a half dozen points, the authors summarize research done with athletes who suffered a mild TBI. This research used comparisons of multiple testing domains and involved development of a new metric for assessing the sequelae of concussion and measurement of recovery (Thompson, Sebastianelli, & Slobounov, 2005):

1. SR (self-report) is sensitive within 24 hours.
2. NP (neuropsych) testing typically normalizes within 1 week.
3. Postural testing typically normalizes within 7 to 10 days.
4. EEG measures, when combined with postural tasks, are sensitive beyond the 10-day period.
5. Combining modalities is more sensitive and reliable than any single method.
6. LORETA is effective to localize cortical areas affected by the head injury.

Thompson found that no one test was sufficient, and he recommended the use of a testing paradigm that combines the most sensitive tests from each modality. Based on this research, and that of others in this field, a multimodal assessment method for the comprehensive measurement of brain functioning was developed. The primary tool for data collection is the Brain Injury Assessment Tool (BIAT), and this is used along with computerized neuropsychological testing and biochemical analysis (www.Evokeneuroscience.com). The BIAT instrument rapidly, with 20 minutes of data collection, measures 19-channel EEG, QEEG, ERPs, and HRV. The assessment employs a simultaneous continuous performance test during which ERPs are measured and also uses a standardized questionnaire. An additional balance assessment using a force plate is also available and can be integrated into this assessment. Biochemical testing of blood and urine can be added as deemed necessary. This is the methodology now used at the ADD Centre/Biofeedback Institute in Toronto. This testing is uniformly revealing abnormalities in our clients, even years after the primary injury.

Role of QEEG in Assessment

The principle findings in a QEEG, regardless of the time elapsed since the concussion, can include EEG slowing, decreased power, episodic discharges, and changes in

coherence. A slowed peak frequency, also referred to as the dominant frequency, is a common finding after a TBI (Angelakis, Lubar, Stathopoulou, & Kounios, 2004). Normal adult peak frequency, measured in the occipital region with eyes closed, is about 10 Hz. After a concussion, it may drop into the low alpha range, perhaps 8 Hz, or even fall into the theta range. A dominant frequency less than 8 Hz in an adult is considered abnormal. Another common finding is reduced overall power. The most consistent power findings after a concussion are reduced power in the higher-frequency bands (8–40 Hz) and increased slow waves (1–4 Hz; Slobounov, Sebastianelli, & Simon, 2002; Thatcher, 2009). There are long-term changes in the 1- to 4-Hz/8- to 12-Hz ratio (Korn, Golan, Melamed, Pascual-Marqui, & Friedman, 2005; Slobounov, Cao, & Sebastianelli, 2009). A change (increase) in this 1- to 4-Hz/8- to 12-Hz ratio activity is negatively correlated with patient outcome 6 months after injury (Leon-Carrion, Martin-Rodriguez, Damas-Lopez, Barroso y Martin, & Dominguez-Morales, 2009). Thatcher has suggested that power changes in the EEG following concussion result from dysfunctional ionic channels of neuronal membranes (e.g., Na+, K+, Ca++) and reduced average current flux (Thatcher et al., 2001). Decreased and/or increased coherence is a common finding. Coherence reflects communication between different sites in the brain and represents the amount of phase-locked activity in a particular frequency band. TBI will disrupt coherence. The most common finding is a decrease in coherence that is colloquially called a "disconnect." A less frequent but important finding may be episodic discharges. A dramatic example reported by M. Barry Sterman (personal communication, March 2012) was that one of his patients dropped his 6-month-old infant and was accused of abuse. Sterman determined that the probable cause of the accident in this very conscientious parent was a sudden episodic discharge with momentary loss of muscle tone and consciousness.

Addition of ERPs to Assessment Measures

Information from evoked potentials, also called event-related potentials (ERPs) because they are time locked to a specific event (sight or sound or even touch), can be used following concussion to determine the intactness of cortical pathways and speed of processing within these pathways (De Beaument et al., 2009; Dupuis, Johnston, Lavoie, Lepore, & Lassonde, 2000). ERPs reflect the neural activities associated with cognitive/behavioral demands and thus provide access to an improved understanding of brain functioning that goes beyond either neuropsychological testing or motor testing (Gosselin et al., 2010). ERPs have been shown to be resistant to practice effects and are therefore a hearty diagnostic assessment tool (Mendez, Hurley, Lassonde, Zhang, & Taber, 2005) that can be repeated as often as necessary. The population of people who have suffered a TBI demonstrate reduced amplitudes for the P3 components (P3a and P3b) of their ERPs. (For a detailed explication of P3a, P3b, and other ERP components, see Polich, 2007). When describing ERPs, "P" refers to positive-going waves and "N" to negative-going waves. The number, such as 300 (sometimes shortened to just 3), refers to the number of milliseconds after the stimulus when you expect the response to occur. This reduced amplitude can indicate diminished attention because the amplitude of the P3 has been shown to be related to the amount of attention allocated to information processing (Bernstein, 2002; Duncan, Kosmidis, & Mirsky, 2005). In addition to lower amplitude, the latency of the P3 response has been found to be greater, and this delay in conscious processing of information is called a slowed P3. This increased latency in the P3 response correlates with slowed cognitive processing (Lavoie, Dupuis, Johnston, Leclerc, & Lassonde, 2004). Earlier ERP responses are observed at less than 250 milliseconds, which is before there is conscious awareness of the stimulus. They reflect early brain sensory processing. In our experience, patients with TBI generally have sensory components (P1 and N2) that are longer latency and lower amplitude compared with non-TBI patients. P3 also tends to be longer latency and of lower amplitude (see Figure 6). P3a is an attention-orienting frontal component, whereas P3b reflects sustained attention and is more central-parietal, with the largest amplitude being observed at Pz. These observations of longer latencies with the P300 ERPs may help us understand why there are persistent problems with attention and concentration after concussion.

Addition of HRV to Assessment Measures

The observation that TBI impairs cardiac function has become increasingly apparent through work done with relatively young athletes and with clients who have had vehicular accidents (car, motorcycle, bicycle).

Figure 7 contrasts HRV in a healthy adult with that of an athlete who has suffered a concussion. It demonstrates that, after a concussion, even a healthy athlete can show significant HRV changes. The top electrocardiogram shows a 3-second sample, and below it is a tracing of variations in heart rate over a 300-second time period, both from the same healthy 36-year-old male. These demonstrate what one would expect in a healthy person: heart rate variations are even with high amplitude and steady rhythmicity. The

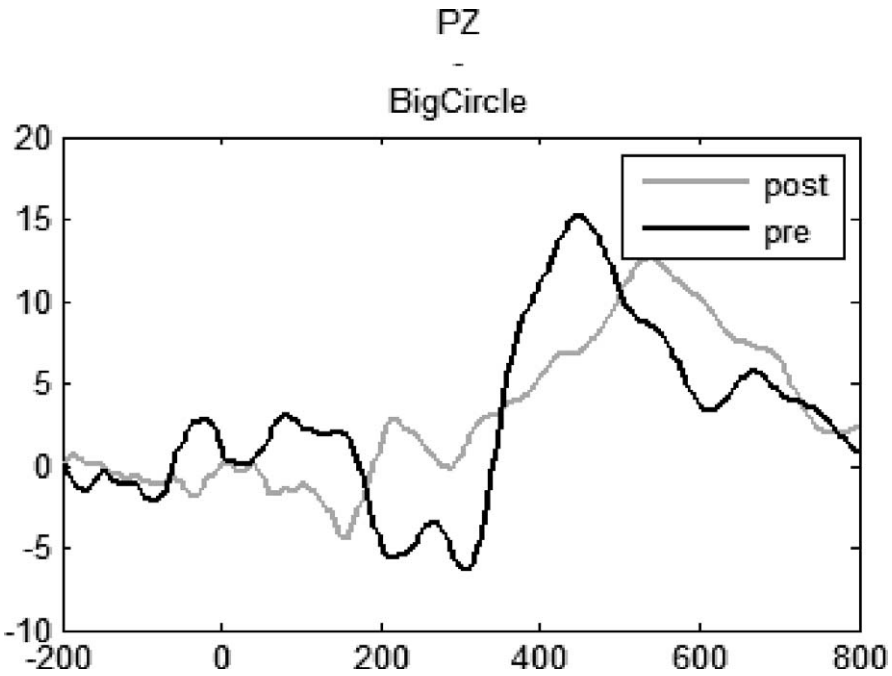

Figure 6. Pre (black) and post (gray) P300 event-related potential (ERP) recordings from the same subject comparing a baseline measurement and a postinjury measure. Latency of the P3 wave is delayed in the postconcussion test, and amplitude of the P3 wave is reduced following injury. ("Big Circle" refers to the target stimulus in this ERP test that is in the program from Evoke Neuroscience.) Figure from Thompson & Hagedorn in *The Companion to the Neurofeedback Book* (in press).

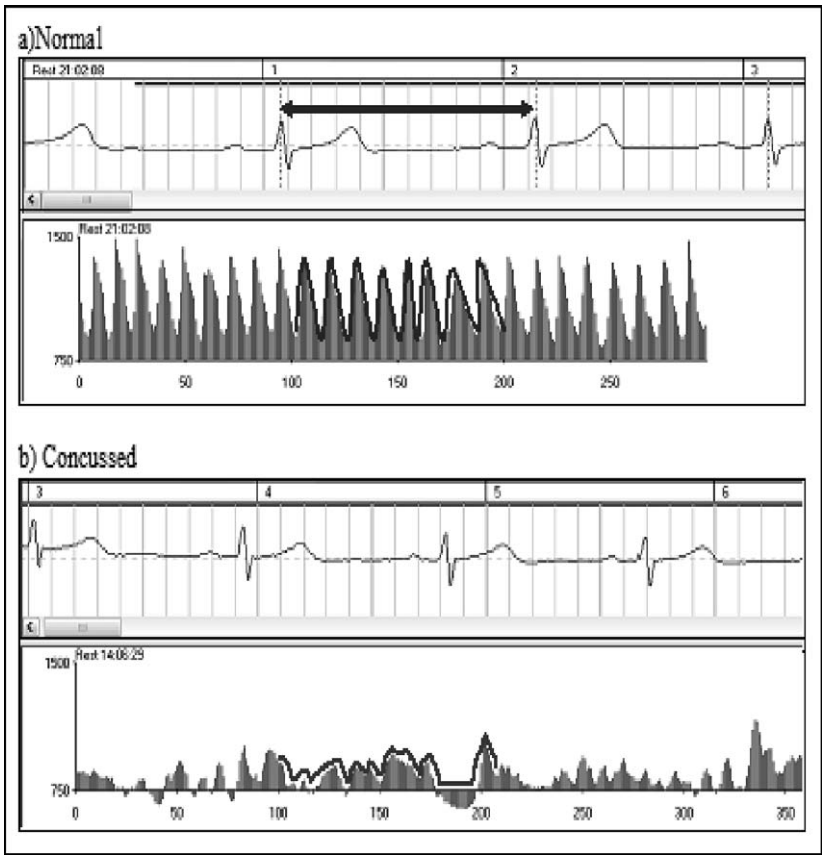

Figure 7. Heart rate variability profile from a healthy adult and from a concussed athlete. From Thompson and Hagedorn (2012).

figure below it shows data from a highly trained male athlete 2 weeks postconcussion. It demonstrates low amplitude and poor rhythmicity of heart rate variations. The amplitude of the variations in heart rate and the standard deviations of the heart rate variations after artifacts have been removed (SDNN) were also both lower than would be expected in a young athlete. These data provide an illustration of how brain injury affects not just the brain but also the heart, specifically that concussion has a negative effect on HRV. Similar observations are found postconcussion in clients at the ADD Centre and also have been published by researchers. One example is a study by Baguley, Heriseanu, Felmingham, and Cameron (2006). They compared HRV data on 16 TBI age-matched subjects with and without dysautonomia with that of 16 noninjured controls. The mean SDNN (milliseconds) for the control group was 60.2 with a standard deviation (*SD*) of 26.9, whereas the mean SDNN (milliseconds) for the TBI group was 40.6 with an *SD* of 16.4. The total power (ms^2/Hz) of the controls was 4065 ($SD = 4{,}082$) and in the TBI group only 1656 ($SD = 1{,}237$). The LF power (ms^2/Hz) of the control group was 1,304 ($SD = 1{,}484$) and in the TBI group only 189 ($SD = 152$). They demonstrated that the TBI group revealed significant differences in HRV parameters compared with controls.

Neuropsychological Testing

In a clinical as contrasted to a research setting, testing must be confined to that which can be useful to assist in diagnosis and, more importantly, to design an appropriate treatment program and track the patient's progress during rehabilitation. The Weschler Intelligence Scale may be used to obtain a profile of intellectual strengths and deficits with other testing carried out as appropriate for a particular patient. Problems with memory and concentration are among the common cognitive sequelae of TBI. In adolescents and adults with a TBI presentation, a computerized neuropsychological test battery is used at the ADD Centre. In the interest of brevity, an example from the first page of the report is given in Figure 8. Completion of this test battery takes the patient approximately 45 minutes to 1 hour, and the full report is delivered by computer within 1 hour after the testing is competed. The objective testing of aspects of response time and memory involves tests that can be quite difficult, and results, for the most part, do pinpoint the difficulties the patient is experiencing. This computerized methodology is simple to administer and can be repeated so the results can be used, in a clinical setting, to follow progress during treatment. The objective test procedures in this computerized neuropsychological test are complemented by subjective self-report questionnaires and validated neuropsychological behavioral and cognitive testing tasks. Questionnaires include an Alertness Rating Scale, the Zung Anxiety and Depression Self-Report Scales, an Adult ADHD Self-Report Scale, a Neuropsych Questionnaire Short Form (a series of questions about the patient's clinical state including mood, attention, sleep, fatigue, aggressiveness, and so on), and a 16-item Medical Outcomes Survey.

This computerized neuropsychological test battery is a cost-effective way to obtain objective and subjective information about the patient and track changes during training. It may be a particularly useful tool for clinicians who do biofeedback but who are not trained in the administration of psychological tests. It is also very helpful in the assessment of patients who could not afford individual testing done by a psychologist.

Vestibular/Balance Assessment

At the ADD Centre, we are considering adding an assessment tool that uses a force plate to quantify a person's balance, coordination, and vestibular system following a brain injury. This can be a very sensitive tool that, especially when combined with simultaneous EEG measurements, will pick up functional deficits well after the time that conventional instruments, such as self-report questionnaires and psychometric tesing, have returned to the normal range. The subject has his or her balance tested in various modes (both feet, one foot, eyes open and closed) while standing on a small rectangular force board that sends signals to the computer that evaluates subtle movements.

A Neurophysiological Approach to Effective Treatment

In the acute phase, rest from both physical and cognitive activities is the only treatment that is agreed upon for concussion (McCrory et al., 2013). Basic rehabilitation procedures, as are carried out in hospital settings and in rehabilitation centers, should be adhered to as well. These interventions are necessary but often not sufficient for full cognitive recovery. For later stages of treatment, both neurofeedback/biofeedback practitioners and nutritionists have important complementary techniques to apply. These are now being demonstrated to achieve further results that may be significant and, indeed, often life changing.

Normalizing the EEG deviations is a first step. This intervention must be designed while keeping in mind that not all deviations from a normative database will correspond to symptoms. Indeed, we have seen examples of genius correlating with significant deviations from a normal

Total Test Time: 37:43 (min:secs)	Online Version 1.0

Patient Profile	Percentile Range				> 74	25 - 74	9 - 24	2 - 8	< 2
	Standard Score Range				> 109	90 - 109	80 - 89	70 - 79	< 70
Domain Scores	Subject Score	Standard Score	Percentile	Valid Score**	Above	Average	Low Average	Low	Very Low
Neurocognitive Index (NCI)	NA	93	32	Yes		X			
Composite Memory	87	78	7	Yes				X	
Verbal Memory	44	72	3	Yes				X	
Visual Memory	43	92	30	Yes		X			
Psychomotor Speed	167	95	37	Yes		X			
Reaction Time*	640	100	50	Yes		X			
Complex Attention*	8	97	42	Yes		X			
Cognitive Flexibility	41	96	40	Yes		X			
Processing Speed	65	114	82	Yes	X				
Executive Function	43	97	42	Yes		X			

Domain Dashboard: Above average domain scores indicate a standard score (SS) greater than 109 or a Percentile Rank (PR) greater than 74, indicating a high functioning test subject. Average is a SS 90-109 or PR 25-74, indicating normal function. Low Average is a SS 80-89 or PR 9-24 indicating a slight deficit or impairment. Below Average is a SS 70-79 or PR 2-8, indicating a moderate level of deficit or impairment. Very Low is a SS less than 70 or a PR less than 2, indicating a deficit and impairment. Reaction times are in milliseconds. An * denotes that "lower is better", otherwise higher scores are better. Subject Scores are raw scores calculations generated from data values of the individual subtests.

VI** - Validity Indicator: Denotes a guideline for representing the possibility of an invalid test or domain score. "No" means a clinician should evaluate whether or not the test subject understood the test, put forth their best effort, or has a clinical condition requiring further evaluation.

Verbal Memory Test (VBM)	Score	Standard	Percentile	
Correct Hits - Immediate	8	67	1	Verbal Memory test: Subjects have to remember 15 words and recognize them in a field of 15 distractors. The test is repeated at the end of the battery. The VBM test measures how well a subject can recognize, remember, and retrieve words e.g. exploit or attend literal representations or attribute. "Correct Hits" refers to the number of target words recognized. Low scores indicate verbal memory impairment.
Correct Passes - Immediate	14	96	40	
Correct Hits - Delay	11	98	45	
Correct Passes - Delay	11	54	1	
Visual Memory Test (VSM)	**Score**	**Standard**	**Percentile**	
Correct Hits - Immediate	13	109	73	Visual Memory test: Subjects have to remember 15 geometric figures, and recognize them in a field of 15 distractors. The test is repeated at the end of the battery. The VIM test measures how well a subject can recognize, remember, and retrieve geometric figures e.g. exploit or attend symbolic or spatial representations. "Correct Hits" refers to the number of target figures recognized. Low scores indicate visual memory impairment.
Correct Passes - Immediate	9	80	9	
Correct Hits - Delay	13	113	81	
Correct Passes - Delay	8	81	10	
Finger Tapping Test (FTT)	**Score**	**Standard**	**Percentile**	
Right Taps Average	56	94	34	The FTT is a test of motor speed and fine motor control ability. There are three rounds of tapping with each hand. The FTT test measures the speed and the number of finger-taps with each hand. Low scores indicate motor slowing. Speed of manual motor activity varies with handedness. Most people are faster with their preferred hand but not always.
Left Taps Average	46	77	6	
Symbol Digit Coding (SDC)	**Score**	**Standard**	**Percentile**	
Correct Responses	65	113	81	The SDC test measures speed of processing and draw upon several cognitive processes simultaneously, such as visual scanning, visual perception, visual memory, and motor functions. Errors may be due to impulsive responding, misperception, or confusion.
Errors*	0	111	77	

Figure 8. The ADD Centre logs in to CNS Vital Signs (www.cnsvs.com) to run a computerized Neuropsychological Test Report (administered through Evoke Neuroscience). This 38-year-old client presented 15 months after experiencing a brief loss of consciousness in a fall. This very bright and capable individual has a doctoral degree, yet she was plagued by memory problems and even forgot appointment times, despite e-mail reminders the day before. Given past academic and work history, one would expect consistently above-average scores, but testing corroborated self-report of memory problems continuing more than 1 year after the head injury.

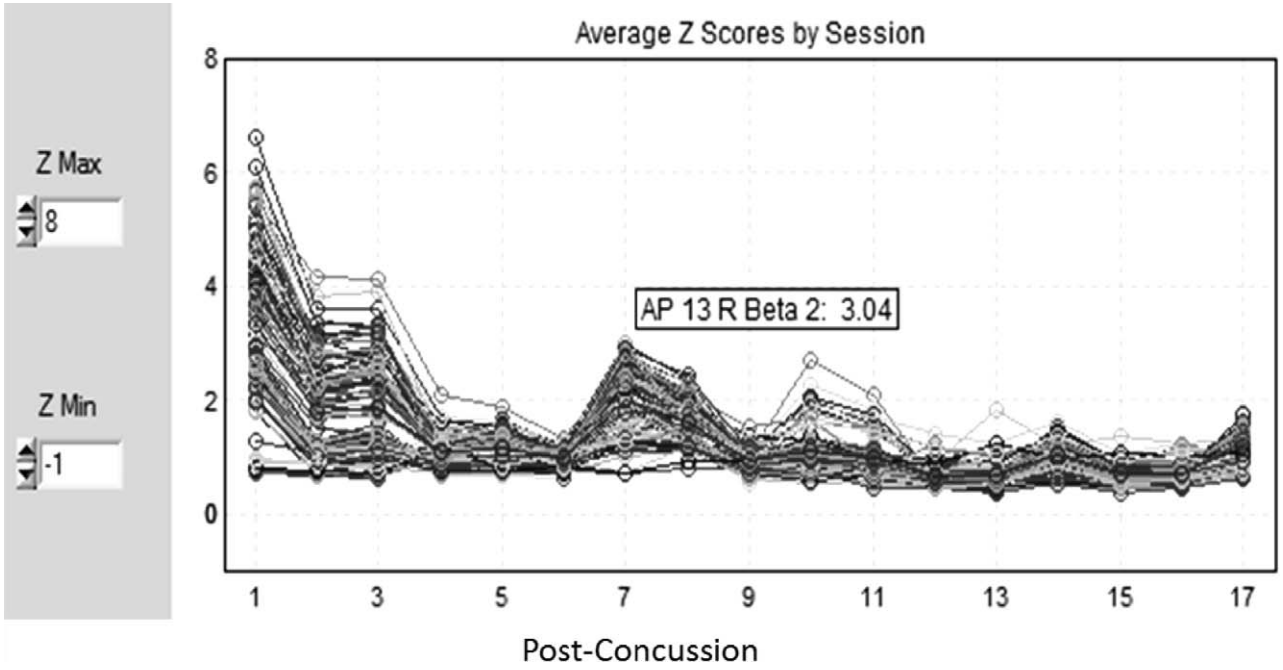

Figure 9. Neuroguide Program diagram of progress across 17 LORETA z-score neurofeedback training sessions. At 17 sessions, this client's z-scores had moved from >4–6 standard deviations (SD) to <2 SD at all the Brodmann Area sites that were chosen to be trained. The cursor has been placed on the highest SD site for the 7th session and the message that pops up is shown in the figure. This message indicates that this site represents the SD of amplitude (AP) for Brodmann Area 13 (the insula) on the right side (R) for Beta 2 (15–18 Hz in this program). The right insula will have major effects on heart rate variability. It is also involved in affect and executive networks. Figure from *The Companion to the Neurofeedback Book* (in press).

database. The person with deviations in the auditory cortex who has perfect pitch, for example, does not have the goal of normalizing that aspect of his auditory processing. Thus, the cautious practitioner will include only Brodmann areas with z-score deviations that correlate with a particular patient's symptoms of concern and will carefully assess during each session whether the patient's changes, when meeting the feedback criteria, correlate with symptom relief. Normalization should not induce any decrement in cognitive functioning. Training that addresses shifting brain functioning toward what is expected for the person's age is often done using LORETA z-score neurofeedback (LNFB). Nevertheless, we must note that we have had excellent results using single-channel neurofeedback training with clients who had suffered a TBI in past years. In some cases, we are doing one session of LNFB and one of regular, single-channel neurofeedback each week. LNFB is a logical choice of intervention when working with patients who have had concussions and show multiple deviations involving areas deep in the cortex. Single-channel training can affect these due to network properties in the brain's cortical-subcortical loops, but the LORETA training is hypothesized to have a more direct influence on these structures. The initial assessment in the majority of patients who have suffered a TBI typically shows both positive and negative deviations in amplitude (>2 *SD*) at multiple sites, often in central midline areas, accompanied by both coherence and phase differences as compared with database norms. LNFB allows the practitioner to address up to 24 parameters, correcting the activity at various sites at the same time for amplitude and/or coherence and/or phase according to the Neuroguide database. An example of a summary graph of LORETA neurofeedback training is shown in Figure 9.

The graph of the learning curve across 17 sessions of LORETA neurofeedback training in Figure 9 shows a sudden increase at Session 7. This was because the client had to leave the country for several months after six sessions of training. When he came back to Canada, some of his symptoms had returned, so he came back to complete his training and achieved good results.

Neurofeedback training is nearly always combined with HRV training and with monitoring of diet, sleep, and exercise. Exercise needs to be appropriate and not jarring to the spine and brain. In the immediate postinjury period, even before nutritional assessment is carried out, an increase in supplementation of omega-3 essential fatty acids (i.e., fish oil) to several grams per day is usually appropriate.

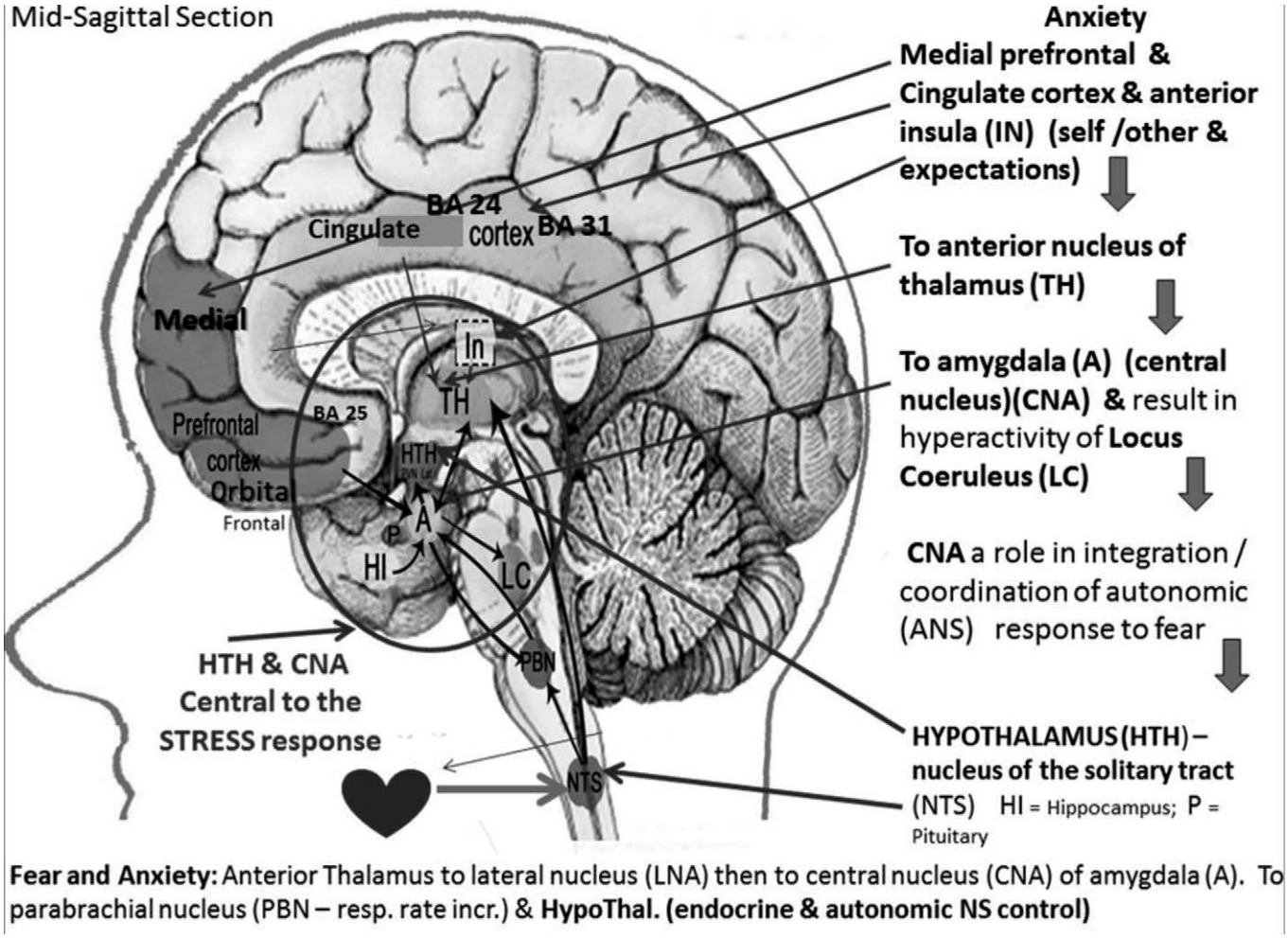

Figure 10. Figure by Maya Berenkey after drawings by Amanda Reeves from *The Companion to the Neurofeedback Book* (in press). Midsagittal representation of selected central midline cortical and subcortical structures (plus the insula, which in reality is not midline but more lateral).

At the ADD Centre, HRV training is carried out simultaneously with the neurofeedback. As reported in the literature (Thompson & Hagdorn, 2012) and in our clinical experience, even healthy athletes can show major cardiac changes postconcussion. Instead of heart rate variations that are even, with high amplitude, and steady rhythmicity, the picture becomes one of low amplitude and poor rhythmicity, as illustrated earlier in Figure 7. The standard deviation of RR intervals with artifacts removed (SDNN) has been observed to be unexpectedly low (usually <50 milliseconds) in concussed athletes before HRV training is done. In one controlled study concerning HRV after a TBI, the TBI group revealed significant differences in HRV parameters compared with controls (Baguley et al., 2006). At the ADD Centre, it is frequently observed that the insulae (right insular cortex as well as left insular cortex) show values that are outside the database norms when analyzed using QEEG and LORETA. The insulae are important regulators of autonomic balance, with the right insula affecting the sympathetic and the left the parasympathetic nervous system, in part through connections to the nucleus accumbens, which has a role in balancing the sympathetic and parasympathetic systems (Nagai, Hoshide, & Kario, 2010). There are reciprocal connections between the insula and the anterior cingulate gyrus, amygdala, entorhinal cortex, medialfrontal and orbitofrontal cortex, and the hippocampus. There are reciprocal connections with subcortical autonomic structures, including the lateral hypothalamic area, nucleus tractus solitarius, and the parabrachial nucleus. These areas are also reciprocally connected to each other. The medial prefrontal cortex (MPFC) normally gives a tonic inhibition of the amygdala (Thayer, Åhs, Fredrikson, Sollers, & Wager, 2012). Dysfunction of the ventral MPFC that results in hypo-

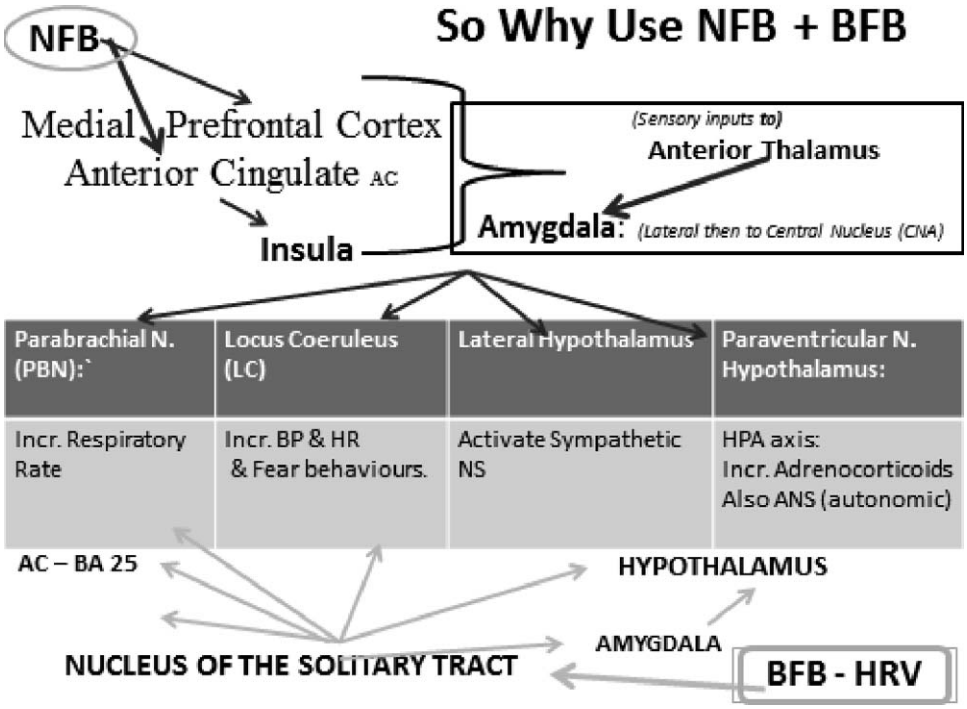

Figure 11. Schematic representation of structures to highlight the synergistic influences of neurofeedback and biofeedback–heart rate variability.

activation can result in amygdala excitation of the sympathetic system and results in changes in HRV. Figures 10 and 11 illustrate, in a summary format, influences of cortical and subcortical areas that can affect both the patient's responses to stress and to neurofeedback combined with HRV training.

With HRV training, the feedback from the baroreceptors goes directly to the nucleus tractus solitarius. We postulate that it is quite probable that, through these connections, HRV training may directly affect all of the aforementioned cortical and subcortical structures and thus have some influence on affect, stress, and executive networks in the brain. In a clinical setting, it is our responsibility to do all the interventions that may reasonably be expected to help our patients. We therefore combine the HRV training with neurofeedback. For a single-channel feedback session, one sample screen is shown in Figure 12.

Figure 12 shows conventional single-channel neurofeedback at FCz[2] combined with HRV training. This screen is often used as an example in teaching because it is a client favorite. It induces a sense of competition between the boats with the client attempting to have a green boat win. To do this they must focus, eliminate all unnecessary thoughts and ruminations, relax, and concentrate. In addition, clients are asked to maintain synchrony between the variations in heart rate (red line) and their breathing (inspiration and expiration shown by the blue line). Their SDNN and HRV amplitudes can be immediately calculated for them at the end of the session using the CardioPro program from Thought Technology. When LORETA neurofeedback is being done, the client may do the HRV training as the 19-channel cap is being put on. Clients can also do it during the session by using a second computer with the biofeedback sensors attached to another instrument such as the Infiniti from Thought Technology, as is being used in the foregoing example. From a subjective point of view, our patients have been unanimous in their opinion that one of the most important aspects of their training program has been HRV training followed by using their effortless diaphragmatic breathing when under stress in their everyday life.

At the ADD Centre, we also address nutrition. Given that nutrition is a complex area requiring an extensive background in biochemistry and physiology, how can the neurofeedback practitioner be of practical assistance to the patient? The simplest route is to have the blood and urine work ordered by their family practitioner and the results forwarded to a nutrition specialist. At the Biofeedback Institute, we usually do one of the following.

[2]The International 10–20 system defines scalp sites for EEG placements, in a grid organized around two axes. FCz is a midline location half way between Cz (the center or vertex of the scalp) and Fz (the next more frontal midline location).

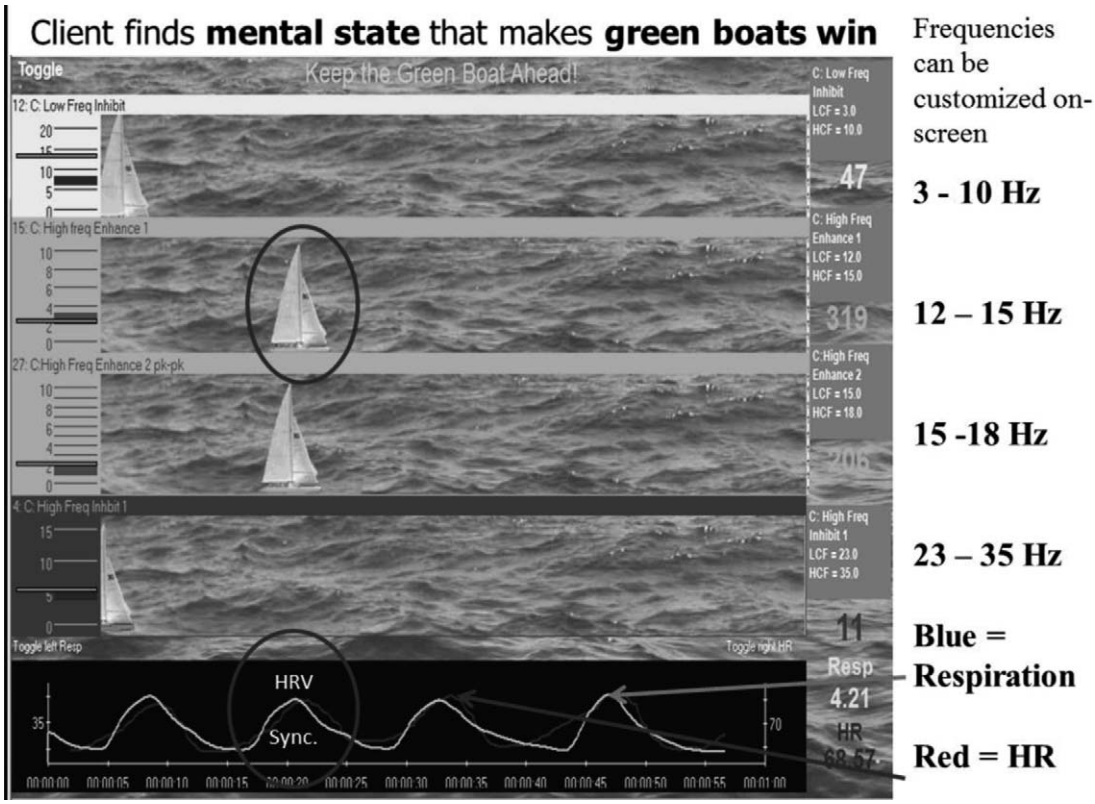

Figure 12. Screen from Thought Technology instruments and programs using the Thompson suite, *Setting up for Clinical Success* (www.BFE.org) for simultaneous neurofeedback and biofeedback training.

1. For regular clients (for example, with attention-deficit/hyperactivity disorder), we recommend OHIP (provincial health insurance)–covered blood and urine work as recommended by a PhD nutritionist/dietitian. This is ordered by the family practitioner, and the results are forwarded to the nutrition specialist.
2. For complex clients (depression, posttraumatic stress disorder, TBI), we recommend the TRIAD Profile from Metametrix in the United States, which is then interpreted for us by licensed specialists at EvokeNeuroscience. This analysis can go in progressively more advanced stages. It will look at markers for all the relevant nutrients plus toxins and will identify all major imbalances. There is a cost involved for the patient.

Conclusion

In our experiene, postconcussion symptoms may be sufficiently severe and long-lasting as to completely disrupt the academic pursuits or employment of the affected individual. This puts considerable strain on the patient's social and family life. The emotional and personality changes after a minor traumatic head injury, especially if it is not the first such injury, can be very severe. At the tragic end of the continuum are deaths from accidental overdose, second-impact syndrome in athletes, accidents, or even suicide. Traditional assessment procedures do not fully detect the problems, especially not DAI. Traditional rehabilitation can be helpful but may not fully ameliorate the patient's cognitive and emotional difficulties. Currently, we are able to report that all postconcussion patients who completed a neurofeedback plus biofeedback program at the ADD Centre have been able to return to work and have been successful in the amelioration of most cognition and affect symptoms. Clients have included an author who could barely compose a paragraph when first seen 2 years postinjury who subsequently authored three books, a student who had to take a greatly reduced course load all through his undergraduate years after suffering a head injury in first year of university who completed a demanding graduate program in actuarial science with absolutely no accommodations while concurrently doing neurofeedback, and a PhD candidate who had been stalled in his thesis work and unable to teach for 2 years after a whiplash injury who returned to his studies and subsequently completed his doctoral work on artificial intelli-

gence, sending us a copy of his graduation certificate along with a note crediting his recovery to the neurofeedback intervention. These are three examples of what is meant when we say that training can be life changing. A case study of another long-term patient, who had been in a coma and was deemed by insurance to have suffered a catastrophic injury, is described in an accompanying article.

The combination of neurofeedback with HRV training and nutritional interventions offers hope for better outcomes because it can result in significant improvements for patients who have suffered a TBI. As was stated at the outset, there is nothing "mild" about the impairments that result from a mild TBI, and individuals who suffer these injuries deserve the opportunity to receive modern treatment methodologies that have the potential to ensure positive outcomes.

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Competing Interests

James Thompson is the Chief Science Officer at Evoke Neuroscience Inc. Michael Thompson and Lynda Thompson are the authors of the *Setting up for Clinical Success Suite* available from the Biofeedback Federation of Europe.

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