Orientation to Neurofeedback for Mental Health Professionals

3 hr Professional Development Workshop, Plaza P7, Brisbane Convention & Events Centre
Sunday 2nd July, 10.15am – 2.15pm

Presenter: Michelle G Aniftos
Clinical Psychologist & Neurofeedback Therapist
BSc; GradDipEd; PGDipEd; MEd; MPsych (Clinical); GradCertClinNeurophysiology

Michelle Aniftos is a Clinical Psychologist and Neurotherapist working in private practice in Toowoomba, Australia. She has been accredited by the Australian Health Practitioner Regulation Agency and the Australian Psychological Society’s Clinical College as a Supervisor of provisional and registered psychologists. For the past 12 years, Michelle has been providing mental health services for children and adults in her role as the Clinical Director of a well-regarded private mental health practice with offices in the Darling Downs, Western Downs and South Burnett regions of South-east Queensland. Michelle was an original facilitator for the Mental Health Professional Network and continued to lead two local groups, each for a three year term of office. As an Adjunct Lecturer of the University of Southern Queensland, Michelle works as a Clinical Psychologist Supervisor and has been associate supervisor of post-graduate research students, having previously held a three-year lecturing position in the Masters of Guidance and Counselling program. Michelle has been a member of the Australian Psychology Society since 2004 and a member of the APS College of Clinical Psychologists since 2008. Within the APS, Michelle has been an active contributor at local branch, state chapter and national interest group level, being an inaugural member, and current Convener, of the APS Neurofeedback Interest Group. Michelle has been accredited by Biofeedback Certification International Alliance and QEEG Certification Board as a mentor and educator in EEG–guided assessment and neuromodulation for improved self-regulation.

Affiliations:
Adjunct Lecturer, Psychology Department, University of Southern Queensland
Chair & Fellow, Biofeedback Certification International Alliance – Australia
Convener, APS Neurofeedback & Psychology Interest Group
Fellow & President-elect, Applied Neuroscience Society of Australasia
Diplomate, QEEG Certification Board International

Recommended Reading
• BCIA (2015). Blueprint of Knowledge Statements for Board Certification in Neurofeedback
About Neurofeedback Certification
Toward BCIA Certification, candidates must have: pre-requisite tertiary studies in neuroanatomy and psychophysiology; successfully complete 36 hours of didactic education aligned with Biofeedback Certification International Alliance Blueprint of Knowledge; 25 contact hours of BCIA-approved mentoring of clinical neurofeedback skills; and successfully complete the BCIA entry examination. See www.bcia.org for more information.

<table>
<thead>
<tr>
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<th>Hours</th>
<th>Neurofeedback Blueprint Modules</th>
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<tr>
<td>1. Orientation to Neurofeedback</td>
<td>4</td>
<td>6. Patient/Client Assessment</td>
<td>4</td>
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<tr>
<td>2. Basic Neurophysiology &amp; Neuroanatomy</td>
<td>4</td>
<td>7. Developing Treatment Protocols</td>
<td>6</td>
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<td>3. Instrumentation &amp; Electronics</td>
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<td>6</td>
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<td>5. Psychopharmacological Considerations</td>
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<td>10. Ethical &amp; Professional Conduct</td>
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N.B.: BCIA certification is not a substitute for a valid license or other registered credential to legally practise one’s profession as regulated by health care practices in your country.

Session One: 10.15 – 12.15, Orientation to Neurofeedback
- Definition of neurofeedback (EEG Biofeedback)
- History and development of neurofeedback
- Assumptions underlying neurofeedback
- Principles of human learning as they apply to biofeedback

Lunch 12.15 – 1.15 Plaza Ballroom Concourse & Exhibition Area
(Practical NFB demonstration here in P7 from 12.45 – 1.15)

Session Two: 1.15 – 2.15, Research Evidence for Neurofeedback
- Methodological and statistical criteria and procedures for determining levels of efficacy
- Key research studies establishing current efficacy levels of Neurofeedback Therapy

2.30pm - APS College of Clinical Psychologists National AGM, Plaza Ballroom

Workshop Aims (based on Modules 1 & 4 above)
1. Develop insight into the neural circuitry involved in psychopathology and the application of EEG biofeedback (neurofeedback) for modulation of brain states toward healthy self-regulation.
2. Identify the application of learning theory and principles to neurofeedback.
3. Review the research history and current evidence for neurofeedback.

Measure of Learning
- Define neurofeedback (EEG biofeedback)
- Define the following terms: Operant Conditioning, Neuroplasticity
- Psychological functions have physiological correlates within what system/s of the body?
- What are some of the common physiological symptoms of psychological distress?
- Which NFB researcher is associated with the following concepts?
  - *sleep & creativity* > *alpha-theta NFB psychotherapy*
  - *alpha-theta NFB protocol to treat alcoholism*
  - *efficacy study of SMR NFB for epilepsy*
  - *trained cats’ SMR > NASA-funded study > launched SMR NFB*
  - *open head trauma & TBI*
- Describe the efficacy and effectiveness levels indicated by current research for at least two of the following conditions: ADHD, Addictions, PTSD, Autism, TBI, Depression, & Anxiety.
Workshop Introduction
Neurofeedback (NFB) is emerging as a significant therapeutic intervention for a range of disorders including, ADHD, anxiety, autism, pain, and behaviour disorders. NFB also benefits healthy clients for optimal performance. As mental health practitioners, we focus on psychological processes influencing daily functioning. NFB enables both client and practitioner to have greater capacity to influence underlying physiological processes that contribute to perceptions and behaviour. This didactic education program introduces the concepts associated with EEG and EEG-guided behavioural intervention, addressing symptoms of disorder, their neurophysiological attributes, and the research evidence for neurofeedback intervention.

Orientation to Neurofeedback in Third Wave Psychotherapy
Our early behaviour therapy models were superseded by social-cognitive theories that promoted the influence of social interactions on behavioural change. While drawing on non-empirical philosophies and practice, the so-called third-wave behaviour therapies integrate social-cognition and emerging neuro-technologies to advance behaviour therapy into the modern era. Mental health professionals are now urged to better understand brain structures, functions and dysfunctions to influence both the externalized traits of the client and the internal states of their brain for sustained improvement in function. Mindfulness, for example, is a behavioural intervention that has been shown to change brain structures and neuronal functioning (Kleen & Reitsma, 2011).

Behavioural change is dependent on the capacity for learning which is reflected in activation and subsequent changes in neural circuitry and activity (neuroplasticity). In this respect psychotherapy can be considered as a specific form of neurotherapy and psychotherapists therefore as clinical neuroscientists (Cozolino, 2002). With more specificity, EEG biofeedback (aka neurofeedback) can be employed in an operant conditioning paradigm as a behaviour therapy to target neurophysiological measures of behavioural symptoms associated with mental disorder, e.g., reduce theta in ADHD.

Neurofeedback draws on theory and research from neurophysiology, neuropsychiatry, neurobiology, psychophysiology, and psychology to deliver evidence-based clinical practices to promote mental health via self-regulation. Research favours multi-modal responses to improve social, emotional and cognitive functioning. NFB may be an alternative or supplement to conventional approaches for treating the symptoms of brain-based (neuro-cognitive &/or neuro-behavioural) disorders.

<table>
<thead>
<tr>
<th>Modality</th>
<th>Method</th>
<th>Invasive</th>
<th>Biological basis</th>
<th>Specificity</th>
<th>Directedness</th>
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<tr>
<td>Talk/behavioral</td>
<td>Learning</td>
<td>No</td>
<td>Moderate (when neuroscience-driven)</td>
<td>Moderate (cognitive/emotional)</td>
<td>High (can focus on issue or problem)</td>
</tr>
<tr>
<td>Therapy</td>
<td>(various)</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Pharmaceutical</td>
<td>Altering</td>
<td>Yes</td>
<td>High (chemical change)</td>
<td>Moderate (neurotransmitters)</td>
<td>Low (widely distributed in brain, side effects and abstractions can occur)</td>
</tr>
<tr>
<td></td>
<td>(chemistry)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stimulation</td>
<td>Altering</td>
<td>Yes</td>
<td>High (electrical conduction)</td>
<td>Moderate (location on head)</td>
<td>Moderate (polarity, location)</td>
</tr>
<tr>
<td></td>
<td>(electrical)</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Neurofeedback</td>
<td>Learning</td>
<td>No</td>
<td>High (EEG and learning process)</td>
<td>High (site-specific or LORETA)</td>
<td>High (wide range of protocols, settings, sites)</td>
</tr>
<tr>
<td></td>
<td>(operant)</td>
<td></td>
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Source: Collura, 2014
Biopsychosocial Model

1. Biological - treating the brain
   • clinical investigation (Paediatrician/Psychiatrist)
   • manage symptoms (e.g., medication, surgery, ECT)

2. Psychological - treating the mind
   • develop therapeutic alliance
   • promote treatment compliance
   • employ psychoeducation
   • minimise distress of symptoms and treatment.

3. Social - facilitating function
   • family involvement
   • peer relationships – personal and professional
   • functional occupation, employment & education

Neuropsychological functions influencing self-regulation
Awareness > neuro-sensory stimulation & processing (parietal, midbrain)
Attention/Engagement > focus or filter (prefrontal cortex)
Motivation > identity, memory, appraisal (limbic, temporal)
Reaction/Response > integration of processes into response (basal ganglia, SMR)

Learning (neuroplasticity) = change in the brain, influencing performance & potential

Limitations of Traditional Cognitive-Behavioural Methods for Learning & Behavioural Change
- level of consciousness/arousal – influencing capacity for attention, concentration, conceptual tracking, and managing activity rate;
- executive functioning - initiative, goal formulation, planning, goal-directed performance;
- emotional states - anxiety impairs receptive understanding & memory function; depression dampens cognitive function;
- pain and sleep deprivation can affect cognitive function;
- medication for pain, sleep, anxiety, and some psychiatric conditions, sedate body and mind.

Improving Self-regulation
Mental health professionals can design interventions to improve self-regulation of the neurophysiological attributes of observable behaviours:

• State of the therapist
• Teaching/reinforcing new thoughts, feelings and behaviours – traditional psychotherapy
• EMDR, body work and energy medicine
• Meditation, yoga, mindfulness
• Improve chemical balance – medication, nutrition
• Influencing the biophysical measures – HRV, GSR, temp, HEG, EEG etc

A wide range of studies in applied neuroscience have demonstrated the clinical effects of EEG biofeedback therapy (a.k.a. neurofeedback therapy or neurotherapy - NFB). This workshop aims to introduce NFB and its potential to mediate improved self-regulation.
What is Biofeedback?
Feedback is provided to give immediate information about a biological function in order to improve an individual’s perception of their physiological events and to improve their self-regulation of the body’s internal regulatory system toward desirable states (see Cohen, 2013). In instrumental learning (operant conditioning), whether a response is learned and performed depends on whether that response is rewarded or punished and knowledge about the performance results (Miller, 1978). An athlete at the gym might believe she is training vigorously due to our generally poor perception of visceral events. A heart-rate monitor gives the athlete objective feedback cuing her to modify effort toward to her goal.

Peripheral biofeedback has long been incorporated into assessment and intervention e.g. heart rate and quality of breathing. Biofeedback can be a stand-alone intervention or supplementary to talk-based cognitive-behavioural therapy (CBT) for improving regulation of arousal state. In CBT, clients gain insight about their cognitive and behavioural perceptions. Through ‘talk’, clients learn to observe and influence their thoughts and feelings (‘mental state’) to shape their behaviours.

Biofeedback operates on the notion that we can develop a heightened perception of arousal state and function to voluntarily influence the automatic functions of our bodies (heart rate, temperature, breathing), through the exertion of will and mind (Figure 1). The control of “involuntary” responses (biofeedback) is now seen to be effective in the treatment of migraine headaches, asthma, ADHD, anxiety, epilepsy, enuresis, stress, and many other disorders.

Figure 1: Biophysical Components in the Client’s Environment (adapted from Collura, 2014)

EEG biofeedback therapy, or neurofeedback (NFB), is a form of personalised medicine or therapeutic training which promotes optimum mental health as individuals develop an awareness of their brain activity as measured by sensors on the scalp. As with all forms of biofeedback, monitoring devices record the moment-to-moment physiological data of the client (brain electrical activity) and present this information back to the client via visual (an EEG waveform or alternative graphic), auditory and/or tactile means (see Figure 2).

Figure 2: EEG biofeedback illustrated
Image by Prof. Josef Faber, www.mentis.ie/images/smr1_en.gif
When brain activity moves toward improved patterns of self-regulation, the display (depicted as a video game) advances, slowing or stopping as brain activity regresses, i.e., through operant conditioning, the trainee is soon able to induce physiological changes (changed brain wave patterns) thereby shaping mental states and subsequent behavioural functioning.

NFB enables remediation because all mental states - emotions, thoughts, behaviours - have a frequency dimension, correlating to arousal state, which can be depicted in the client’s environment and brought to his/her attention to increase self-awareness and therefore, improve the capacity for self-regulation of their central nervous system. Mental states, reflected as EEG parameters, can be voluntarily and selectively changed, and learned via neuroplasticity.

These changes lead to improved flexibility and general stability of the brain waves. Improved neurological functioning supports improved flexibility and stability of externalized behaviours in response to demands of daily living. With regular neurofeedback, therapy and practice, significant improvements seem to occur 75 to 80% of the time (Hammond, 2011).

**The Arousal Model**

Brain states are reflected in different parameters of brain functioning. The bands of EEG rhythms typically observed (see Figure 3), range in frequency from 0-70Hz including: direct current (DC) shifts <1.5Hz, delta (1-4Hz), theta (4-8Hz), alpha (8-13Hz); and beta (*including B1 13-21Hz, B2 to 30Hz, & Gamma >30Hz*).

![Figure 3: Representation of various EEG waves & states](image)

Pioneers in neurofeedback therapy claimed to remediate three underlying patterns of dysregulation improving brain resources and capacity for healthy functioning:

- Overactive: e.g. impulsiveness, hyperactivity, high anxiety, anger and rage reactions, obsessive-compulsive symptoms, tic disorders, and difficulty falling asleep at night.
- Underactive: e.g. inattention, low energy, depression and early morning awakening.
- Instability: e.g. seizures, migraine and panic.

In EEG biofeedback, for example, alpha waves are encouraged for relaxation and relief from anxiety, and perhaps epilepsy. Those with insomnia, psychomotor agitation, hyperactivity, or attention deficits are taught to control theta waves. Delta/theta excess and alpha/beta deficits are commonly encountered in children with learning disorders and this theta/beta ratio is often down-trained to help children with ADHD.
Traditional approaches to brain dysfunction have involved either a medical diagnosis and invasive (drug or surgical) treatments, or ‘mental’ diagnosis and psychotherapy e.g. increase insight re ‘unhelpful’ thoughts, feelings, actions; provide psycho-education & strategies to improve function. The biopsychosocial model, underpinning psychotherapy, promotes well-being as being mediated by biological, psychological and social influences and so our formulation and treatment plans generally consider all of these areas for strengths and vulnerabilities to determine the appropriate course of intervention for our clients.

Neurofeedback therapy has emerged in response to current research in applied neuroscience and the health demands of those seeking non-invasive interventions and/or for those whom psycho-stimulants are not effective (e.g. approximately 36% of inattentive-type ADHD are non-responsive to medication treatment). This intervention enables the practitioner to move away from the medical (crisis) models of treatment to a learning model that is conducive to a ‘strengths-perspective’ intervention (Matuszek & Rycraft, 2003).

In summary, a definition of Neurofeedback

Neurofeedback is employed to modify the electrical activity of the Central Nervous System including:
- electroencephalography (EEG),
- event related potentials (ERPs),
- slow cortical potentials (SCPs) and
- other electrical activity either of subcortical or cortical origin.

Neurofeedback is a specialized application of biofeedback of brainwave data in an operant conditioning paradigm. The method may serve as the basis for treatment of a clinical disorder or enhancement of normal functioning.
History and Development of Neurofeedback

- Pioneers in EEG and NFB
- Highlights of seminal studies
- Further developments in the field

Sources: Demos, 2005; Greenfield, et al., 2010; Finger, 2000

The Pioneers
- Hans Christian Oersted discovered the magnetic field surrounding a charged wire;
- André Ampère created a device to measure electric current;
- William Thomson refined the galvanometer to enable small deflections of an electrical current generated in the brain to be charted as a line on paper; and
- in 1875, Richard Caton (a British physician) was the first to record electrical potentials from the surface of the brain. He found interrupting the light transmitted to a dog’s eye altered the electrical waves detected from the opposite side of the brain.

Hans Berger, a German Professor of Neurology and Psychiatry, was the first to record human brain electrical potentials. In 1929 his EEG data distinguished larger [alpha] oscillations and smaller [beta] waves. Berger was interested in the physiological correlates of mental functions. He explored EEG correlates of attention, mental effort and consciousness (finding alpha attenuates in sleep and with eye-opening; and beta bursts occur with thinking and alertness); and EEG changes associated with cerebral injury such as from brain tumours and epilepsy.

Edgar Douglas Adrian won a 1932 Nobel Prize for his discoveries regarding neuronal functioning and the presence of electricity within nerve cells. His work contributes to our understanding of habituation i.e., that despite a constant stimulus, excitation decreases; while the sensory impulses passing along the nerves are constant in strength, they reduce in frequency over time, consequently diminishing sensory perceptions in the brain. Adrian’s work led to the idea of a somatosensory map, the homunculus. Adrian was inspired by Berger’s EEG studies and his research on the Berger [alpha] rhythm, proposed its occipital origin; the ability to maintain alpha by staring into an empty visual field; and despite eyes closed, the attenuation of alpha via problem solving with visual imagery. His findings advanced research in epilepsy.

Emergence of EEG Biofeedback (aka Neurofeedback)
Joseph Kamiya, a psychologist researching at the University of Chicago in the 1960s, trained volunteers to recognise alpha with verbal reinforcement, demonstrating a simple biofeedback loop. He found that 75% of subjects could learn to discriminate their alpha-state arousal by being given feedback about the characteristics of their brain’s electrical activity; and that those trained in alpha discrimination subsequently demonstrated superior capacity to selectively produce alpha (Frederick, 2012).

Clinical neurofeedback was advanced by Barry Sterman, University College of Los Angeles who demonstrated that EEG could be operantly conditioned. Researching the role of EEG in sleep onset in cats, Sterman (1967) trained the so-called sensorimotor rhythm apparent on the motor strip when cats became motor inhibited. Sterman (1972) was later testing the toxic effects of fuel for pilots. Of the 50 cats injected with hydrazine, 10 SMR-trained-cats were found to be seizure resistant. This ‘happy accident’ launched EEG biofeedback as a treatment of seizure disorders. Sterman (2000) began to treat severe epilepsy in patients with medication-resistant seizures. Of almost 200 patients treated with neurofeedback, over 75% had significant clinical improvement. Treatment efficacy for epilepsy was further demonstrated by Lubar and Bahler (1976).
While NFB originally explored brain waves in the range of 1 – 40 Hz, German researcher, Nils Birbaumer and his team, developed and used frequencies less than 1.5 Hz, giving rise to a new paradigm in neurofeedback training, the self-regulation of cortical DC (Birbaumer, et al., 1981). This research found that slow cortical potentials (SCPs) could also be trained with operant conditioning techniques and could impact cognitive functioning (Lutzenberger, et al., 1982).

Elmer Green established the Association for Psychophysiology and Biofeedback (AAPB) in 1969 and has contributed decades of work in biofeedback and self-regulation for healing the body and expanding consciousness (Stokes, 2012) particularly drawing on his studies of yogis in India.

During the 1970’s, EEG biofeedback was being explored for the treatment of (for examples):
- acquired brain injury (Gannon & Sternbach, 1971),
- headache (McKenzie et al., 1974),
- obsessive disorder (Mills, & Solyom, 1974),
- sleep (Bell, 1979; Feinstein, et al., 1975; Sterman, 1977),
- addiction (Cohen et al., 1977; Feinstein),
- pain and affective disorders (Melzack & Perry, 1975; Pelletier & Peper, 1977)
- anxiety (Hardt & Kamiya, 1978),

Lubar and Shouse provided the first empirical evidence for the efficacy of SMR biofeedback for the treatment of ADHD with a case study of a 9-year old boy whose EEG patterns and reduced ADHD symptomatology post-treatment were improved to the extent that the subject ended medication and continued to function well after the study. Joel Lubar’s EEG biofeedback method employed operant conditioning principles to reinforce increased production of electrophysiological activity within either the 12–15 Hz (SMR, beta 1) or 16–20 Hz (beta 2) ranges, while decreasing “slower” activity (4–8 Hz; theta), to reduce impulsivity and improve attention. This theta/beta protocol with recordings over the central front or sensorimotor regions continues to be a ‘staple’ for NFB treatment of ADHD (Monastra, Monastra, & George, 2002).

**Later Developments in Neurofeedback**

A broad range of effective training protocols emerged in subsequent clinical studies:
- Rosenfeld et al., (1996) found evidence that changes in frontal alpha asymmetry correlate with changes in affect in EEG biofeedback therapy sessions, based on evidence that depression is correlated with underactivity of the left frontal cortex (Davidson, 1995).
- Following her time working with Barry Sterman, Margaret Ayers went on to develop her own real-time EEG equipment and brought neurofeedback into the commercial arena. She specialized in the treatment of open and closed head brain injuries (1995) emphasizing the use of bipolar Beta/SMR montages across the sensorimotor strip (rewarding Beta 15-18 Hz at C3 and SMR 12-15 Hz at C4, while inhibiting excessive slow and fast wave brain activity).
- Tom Budzynski was another early research in EEG and EMG. He advanced the earlier work in alpha enhancement by developing an alpha-theta biofeedback system to study creativity, enhancement, and the hypnagogic (twilight) state, and proposing the utility of alpha-theta biofeedback for psychotherapy (Budzynski, 1976).
- Alpha-beta and psychotherapy was applied to the treatment of alcoholism and the prevention of relapse. The Peniston/Kulkosky protocol included desensitization training, peripheral biofeedback (temperature and breathing), guided imagery, visualization, and autogenic training utilizing alpha-theta neurofeedback (Peniston & Kulkosky, 1989; Saxby & Peniston, 1995), proving effective for increasing alpha-theta and decreasing symptoms of alcoholism.
Assumptions underlying Neurofeedback

To influence any system, one must first understand its purpose and functions. An understanding of EEG and neurofeedback technology requires an understanding of the structure and function of the brain and its neural circuitry. The CNS includes both the spinal cord and the brain which comprises the: - brainstem (basic processes of life - breathing, blood pressure, heart rate); - cerebellum (mediates movement coordination, balance and posture); - diencephalon (includes the thalamus and ventral hypothalamus); & - cerebral hemispheres (the cerebral cortex and deeper structures which play a key role in memory, attention, perceptual awareness, thought, language, and consciousness).

Figure 4: The central nervous system

The surface layer of the brain, the cerebral cortex, includes four lobes: - frontal: controls movement, problem solving, reasoning, speech, and emotions. - parietal: controls movement planning, recognition, orientation, and sensory perception. - occipital: controls visual processing; and - temporal: controls sensory perception, recognition, memory, hearing and speech.

The cortex is differentiated into six horizontal layers containing 10 billion neurons organised in vertical columns oriented perpendicularly to the brain surface. These vertical columns and are defined by their inputs i.e., neurons in a column driven by the same thalamo-cortical inputs behave as a unit - a microcircuit through which sensory input from the sensory periphery layer 1 of the skin, eyes and ears is sent to the thalamus and then processed in serial fashion:

1] middle layer 4 of excitatory cells which project to, 2] upper layers 2 & 3 of the cortex, which then project 3] to deep in layer 5, and following the axons 4] out of the cortex to layer 6, adjacent to the subcortical white matter, which also has a feedback loop to layer 4.
Deeper brain structures are critical for system regulation:

The Limbic System - is the route for all sensory information that wants to get to the cortex. It sets emotional tone, controls motivation and drive, holds emotional memories. The female limbic system is larger relative to the size of the brain than is the male.

Hypothalamus - facilitates homeostasis through its regulation of visceral functions of the ANS.

Amygdala - provides emotional content to language, intonation, sound of voice, social emotion, guilt, shame, mediates fear, sadness, depression, hostility & aggression via detection and judgment (evaluation & magnitude). Dysfunction shows as social disinhibition. Stores unconscious memories.

Hippocampus (beneath the temporal lobes)
Short and long term auditory and visual (emotional) memory (LH); sound-voice intonation, memory, and spatial-facial memory (RH).

Septal Nucleus - acts in conjunction with the hypothalamus and hippocampus particularly in relation to internal inhibition, exerting quieting and dampening influences on arousal and limbic functioning.

Cingulate Gyrus (midline) - shifts attention from one subject to another, i.e., mental flexibility and executive function, adapting to changing circumstances (e.g., co-operating in social contexts).

Our survival is dependent on complex physiological mechanisms regulating a stable internal environment (Breedlove & Watson, 2013). Two regulatory models are described (Schulkin, 2004) as:

- Homeostasis (stability through constancy) describes mechanisms that hold constant a controlled variable by sensing its deviation from a “setpoint” and feeding back to correct the error. Homeostasis has been the dominant model exhorting physicians to reason that when a parameter deviates from a set range of values (or setpoint), some internal mechanism must be in need of correction. However, homeostasis does not explain all disease and disorder; and

- Allostasis (stability through change) describes mechanisms that change the controlled variable by overriding local feedback, predicting and adjusting parameters to meet the anticipated demands, i.e. efficient resource management, preventing errors and minimizing costs.

The hypothalamus is the core driver of homeostatic regulation:
- sleep,
- hunger/thirst,
- responses to pleasure and pain,
- the hormonal system (via ANS) and
- the fight/flight responses of the sympathetic nervous system.

McEwen (2005) describes this allostatic system maintaining control through constant change, as standing in contrast to the mechanisms of homeostasis that maintain pH and oxygen levels within a narrow range. When the sensory cortex perceives threat, the CNS activates sympathetic nervous system and allostatic adaptations, preparing the body for action:
- accelerated heart rate & palpitations (to increase blood flow)
- muscle tension (as blood volume increases ready for action)
- rapid breathing (to increase oxygen levels)
- perspiration (to control temperature).
The Neurophysiological Burden of Stress
Chronic mood instability generates physiological stress with indicators such as headaches or dizziness and abdominal complaints (as blood flows to essential organs), fatigue, hypertension, depression, and sleep disturbance. Kapczinski et al., (2008) described “allostatic load” as the wear and tear of biological systems whereby physiological adjustment to prolonged exposure to stress produces neurotoxic effects. Sustained stress depletes dopamine, essential in pleasure and reward networks and also central to the executive functions of the prefrontal cortex. Stress compromises serotonin and norepinephrine which are essential to cognitive functioning (Sapolsky, 2013). Increased levels of glucocorticoid hormones and the hypothalamus–pituitary–adrenal (HPA) axis (a component of the stress response) is hyperactive in 50% of depressed patients (Anacker et al., 2011).

Neurophysiological Relief
To alleviate the depressive symptoms, antidepressants are prescribed to rectify the feedback regulation of the HPA axis by reducing the stress response (homeostatic model). However, neurophysiological activity (neuronal communication) is not only mediated by chemical but also by electrical activity. Alternatively, or as an adjunct approach to improve mood regulation, capacity for optimism may be associated with increased activation of the left frontal cortex via neurofeedback; and cardiovascular health could be promoted with improved heart rate variability (HRV) via controlled breathing.

A New Paradigm
- The brain organizes itself rhythmically
- Through the way it fires
- In the electrical or frequency domain
- It sets up oscillations
- These oscillations are the core of the brain’s self-organization and learning
- Default rhythms can be changed for new learning, behaviour, renewed capacity (Buzsaki, 2006).

EEG is a reflection of cortical self-regulation - excitatory and inhibitory neurons working harmoniously to promote healthy neuro-functioning. An excessive theta/beta ratio is commonly reported as a deficit parameter targeted by EEG biofeedback in ADHD (homeostasis model). Allostasis considers an unusual parameter value, not as a deficit but rather as a response to some prediction e.g., hyperarousal as a response to sustained neural signals arising from social stressors. Rather than focus on deviant parameters, the allostasis model would redirect therapy toward enhancing flexibility, to restore predictive fluctuations. Improved flexibility and general stability of brain activity contributes to improved flexibility and stability of behaviour in response to external demands. Neurofeedback can be thought of as a mechanism to establish additional goals, so that the brain learns to self-regulate in new ways, thus facilitating change (Collura, 2014).

Alterations in oscillatory patterns of EEG play a critical role in maintenance of brain functions and consequently may be used as a powerful tool neurodiagnostics and intervention. Kropotov (2009) proposes that oscillations may be not only the reflection of two opposite forces but may also contribute to the organization of neuronal networks. In the diseased brain, normal mechanisms of EEG rhythms may be impaired and the rhythm may:
1] become slower in frequency (EEG slowing);
2] appear in unusual places (e.g. alpha rhythms in the temporal area);
3] become higher in amplitude (hypersynchronization) and in more synchronicity with other areas (hypercoherence) indicating poor differentiation of brain structures and functioning;
4] in some cases, a separate slow rhythm in delta frequency may appear (e.g. disconnection of cortical areas from sub-cortical structures due to stroke, trauma or tumour); and
5] in some cases, normal synchronization may be enhanced and spike or spike/slow patterns appear indicating a so-called focus in the human brain (e.g. clinical or sub-clinical seizure).
There is a growing body of literature exploring measurable parameters, endophenotypes that reflect functioning of brain systems that may enable more accurate diagnosis of brain-based disease and disorder (Kropotov, 2009). EEG phenotypes may be recognised at semi-stable states of neurophysiological function observable in common psychopathologies (Johnstone, Gunkelman & Lunt 2005). These findings may contribute to predictions of treatment response, for example, to either medication and/or non-invasive interventions such as neuro or psychotherapies. EEG parameters can be displayed so that a client can observe and learn to control his/her brain activity (voluntary control over-riding automatic brain functions) to:
- enhance self-regulation,
- communicate with the external world, or
- for manipulation of biofeedback devices (Kropotov, 2009).

qEEG phenotypes (Johnstone et al., 2005)

<table>
<thead>
<tr>
<th>qEEG Profile</th>
<th>Description of Pattern</th>
<th>Medication</th>
<th>Neurofeedback</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diffuse slow activity, with or without low frequency alpha</td>
<td>Increased delta and theta (1-7 Hz) with or without slow posterior dominant rhythm</td>
<td>Stimulant</td>
<td>Inhibit midline fronto-central activity below 10 Hz, add reward anterior beta frequencies for increased effect</td>
</tr>
<tr>
<td>Focal abnormalities, not epileptiform</td>
<td>Focal slow activity or focal lack of activity</td>
<td>???</td>
<td>Inhibit slow activity (&lt;10 Hz) and reward higher frequencies (&gt;12 Hz)</td>
</tr>
<tr>
<td>Mixed fast and slow</td>
<td>Increased activity below 8 Hz, lack of alpha, increased beta frequency activity</td>
<td>Combine across classes, e.g., stimulant + anticonvulsant?</td>
<td>Inhibit slow frequencies, reward middle frequencies. Reward SMR</td>
</tr>
<tr>
<td>Frontal lobe disturbances</td>
<td>Frontally dominant excess theta or alpha frequency activity</td>
<td>Antidepressant, stimulant</td>
<td>Inhibit midline fronto-central activity below 10 Hz, add reward anterior beta frequencies for increased effect</td>
</tr>
<tr>
<td>Frontal asymmetries</td>
<td>Variable asymmetry L&gt;R or R&gt;L, primarily at F3, F4</td>
<td>Antidepressant</td>
<td>Reward F3 beta, inhibit F3 theta and alpha frequencies</td>
</tr>
<tr>
<td>Excess temporal lobe alpha</td>
<td>Increased alpha activity generated in temporal lobe</td>
<td>Stimulant</td>
<td>Inhibit 9-12 Hz activity over affected temporal region(s), + inhibit frontal slow activity</td>
</tr>
<tr>
<td>Epileptiform</td>
<td>Transient spike/wave, sharp waves, paroxysmal EEG</td>
<td>Anticonvulsant</td>
<td>Inhibit low and high frequencies over affected regions, central strip training, reward SCP</td>
</tr>
<tr>
<td>Faster alpha variants, not low voltage</td>
<td>Alpha frequency greater than 12 Hz over posterior cortex</td>
<td>???</td>
<td>Reward 9-10 Hz alpha at Pz, shift alpha frequency lower with alpha/theta protocol</td>
</tr>
<tr>
<td>Spindling excessive beta</td>
<td>High frequency beta with a spindle morphology, often with an anterior emphasis</td>
<td>Anticonvulsant</td>
<td>Inhibit beta frequencies, wide band inhibit</td>
</tr>
<tr>
<td>Generally low magnitudes (fast or slow)</td>
<td>Low voltage EEG overall theta/alpha</td>
<td>Metabolic support,</td>
<td>Reward alpha activity posteriorly</td>
</tr>
<tr>
<td>Persistent alpha with eyes open</td>
<td>Lack of appreciable alpha blocking with eye opening</td>
<td>???</td>
<td>Reward beta frequencies, inhibit alpha. Reward higher frequency alpha</td>
</tr>
</tbody>
</table>

Note: The above information is not intended as a substitute for professional consultation. See text for more complete descriptions.
Overview of Principles of Human Learning as They Apply to Biofeedback

Cognitive and behavioural theories dominate learning approaches, although the contributions of Vygotsky and Piaget have offered a constructivist approach that proposes learners create subjective meaning from reality. The stimulus-response/response-stimulus theories of behaviourists Pavlov and Skinner (classical versus operant conditioning) argue that learning can be explained without giving consideration to internal mental states, i.e., that is, behavioural change is evidence of learning a response to environmental stimuli. Skinner’s *radical behaviourism* accepted the mediating role of internal events such as emotions. Cognitivists shifted learning theory toward an information processing model - mental processes such as thoughts and feelings enabling learners to develop a schema or mental construction of their world that subsequently informs and influences behaviour.

In *operant conditioning*, a voluntary response is emitted by the organism. The behaviour (operant) produces consequences (*response-stimulus*) by acting upon the environment i.e., learning by trial & error/action and consequence. A rewarding or punishing stimulus is presented after the behaviour to promote or suppress the response (Sherlin, et al., 2011). Positive and/or negative reinforcements increase the behaviour being repeated while positive and/or negative punishments decrease its likelihood (see Table 1).

Table 1. Nature of Stimuli in Operant Conditioning

<table>
<thead>
<tr>
<th>After response, stimulus is</th>
<th>Pleasant</th>
<th>Aversive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Presented</td>
<td>Positive reinforcement (response increases)</td>
<td>Punishment I (response decreases)</td>
</tr>
<tr>
<td>Removed</td>
<td>Punishment II (response decreases)</td>
<td>Negative reinforcement (response increases)</td>
</tr>
</tbody>
</table>

Neurofeedback is a type of operant conditioning in which an individual receives feedback about their EEG activity, increasing their capacity to self-regulate the frequency, amplitude and other characteristics of the EEG, creating a feedback loop. Sterman, et al., (1972) trained cats by EEG operant conditioning, using food reward, to produce 11-15 Hz “alpha spindle” electrical activity. Neurofeedback is a non-invasive intervention enabling the practitioner to move away from dependency on deficit models of treatment to a learning enhancement. Responsibility for wellness rests with the client (Demos, 2005) who is free to explore their inner functions and to observe those micro-behavioural influences on their performance. Their therapeutic engagement toward goals is immediately rewarded via their own EEG data being represented moment-to-moment visuo- and audio-feedback to enable individuals to modify and regulate neurological activity to improve, for example, their executive functioning, learning ability, emotional regulation, and general functioning.

*Principles of Learning Theory Applied to Neurofeedback*

**Discrimination** enables an individual to identify various characteristics of an event and in order to determine relationships between behaviours and consequences. For example, in a clinical neurofeedback session, an individual can learn to distinguish the consequences of holding their body rigid versus relaxing body tension. The first behaviour may result in removal of auditory/visual rewards as high beta is activated while in the latter condition, auditory/visual reward may occur for calming their EEG activity, creating a feedback loop increasing the likelihood of self-regulation toward relaxation.

**Habituation** is a process that decreases a behavioural response to a recurring stimulus. While operant conditioning is an example of associative learning whereby the stimulus is presented with an event such as reward or punishment, habituation is a form of non-associative learning or adaptive behaviour.
Thompson (2009) records that modern interest in habituation as a fundamental form of behavioural plasticity began with research on habituation of EEG arousal where it was found that in response to a repeated stimulus, cortical EEG arousal becomes progressively shorter and finally disappears. After cessation of stimulation the arousal response exhibits spontaneous recovery over time. The human alpha-blocking response was shown to habituate to tactile, auditory, and visual stimulation by Sokolov (1960, cited in Thompson).

**Shaping** in operant conditioning is a method of positive reinforcement of behaviour patterns approximating the target behaviour. The neurofeedback clinician can widen frequency bands for the target EEG and increase rewards in order to ‘catch’ and reward behaviours moving in the direction of the clinical goals. As performance of the target behaviour increases, the frequency bands can be narrowed and the thresholds for reward can be increased in difficulty to shape a broad range of behaviours into a narrower, target range.

**Chaining** is an additional learning principle that requires reinforcement for the appropriate performance of each step within a sequence of behaviours toward a final target behaviour. In neurofeedback therapy, for example, a client first needs to sit calmly, refrain from touching electrodes, and attend to visual feedback on a monitor in order for reliable physiological data to be recorded and presented as feedback to the client. The comorbid behaviours may be shaped toward compliance via teaching expectations and selective reinforcement of segments of the target skills e.g., verbal cues “good sitting”, visual cues (smile, thumbs up), etc.

As the externalising behavioural goals are achieved, the focus of therapy can shift toward stabilizing the EEG activity. The total performance target may be to promote alertness (reduce slow wave activity), calmness (reduce excessively fast wave) and concentration (regulate SMR/Beta). The clinician could use chaining principles to first explain and teach strategies to improve relaxation and reinforce reduced fast wave; then require both calm and alert states, simultaneously reinforcing reductions of excessive fast and slow waves; before the total task training is introduced – reduce theta & Beta 2 while maintaining/increasing the amplitudes in the SMR/Beta 1 range.

**Generalization** is evidence of acquired learning in that it demonstrates the transfer of new skills to new situations and is essential for treatment/training outcomes to be taken beyond the clinic context for clients to experience improved functioning in their daily environments. Lubar (1976) reported maintenance of SMR neurofeedback outcomes beyond the treatment period. Clinicians need to develop techniques to facilitate generalisation of learning relevant to individual client and contextual features.

**Extinction** occurs when the frequency of a behaviour is reduced (potentially to zero) when that behaviour is not reinforced. This learning concept can work for and against therapeutic goals so clinicians must manage variables to benefit client outcomes. If performance goals are unrealistic or reward thresholds are unachievable then repeated efforts by the client to perform the target behaviour to receive the reward will eventually diminish. Similarly, novices undergoing neurofeedback may be restless and inquisitive with these behaviours undermining their capacity to be rewarded for generating a calm state of arousal. In moments of calm behaviour, they will be rewarded thus the calm state is reinforced and the restlessness is diminished via extinction.
Parameters for Optimal Learning

Miller (1978) recognised that to improve the utility of biofeedback in therapeutic applications, clients would need support for learning and performance. Biofeedback practitioners would also need knowledge and improved control over learning and performance processes. A review of neurofeedback and learning was conducted by a collective of elite mentors in applied neuroscience (Sherlin, Arns, Lubar, Heinrich, Kerson, Strehl & Sterman, 2011) who exhort adherence to seven principles of learning theory in neurofeedback (NF) research and/or intervention. These principles are listed below and then each is elaborated in response to research or clinical experience:

1. Speed of Reinforcement – delayed reinforcement decreases the strength of the conditioning;
2. Type of Reinforcement - a response–reinforcer association is developed in operant learning.
3. Shaping – the utility of this learning principle depends upon the feedback context.
4. Specificity – In operant conditioning, learning is promoted when characteristics of the target behaviour can be specified and discriminately reinforced.
5. Artifacts – treatment efficacy can be compromised by reinforcing artifacts in the EEG.
6. Secondary Reinforcement – rewards must be rewarding!
7. Generalisation – learning must be generalised beyond the clinic to the context of daily living in order for neurofeedback training to have any ecological validity.

Speed of Reinforcement

EEG is an analogue signal that must be converted into a digital form for computer analysis. The digital signal is obtained by sampling the analogue signal at discrete points in time to represent the electrode potentials at those moments. The sampling frequency must be high enough to preserve a clinically acceptable signal but not so high as to generate cumbersome data files. Filters edit unnecessary signal e.g., direct current < 1 Hz and extraneous noise >70 Hz. Notch filters may be applied to exclude artefact (e.g., 50 Hz AC line noise. All this processing takes time which may compromise the efficacy of reinforcement. Sherlin, et al., recommend Finite Impulse Response (FIR) filters to ensure the feedback latency does not exceed 250 to 350 milliseconds.

Type of Reinforcement

In addition to the timing of reinforcement, the learner must be aware of the contingent relationship between their behavioural response and the reinforcement. The strength of this association correlates with the speed of learning. In neurofeedback, complex audio and visual stimuli may overshadow the target response (EEG behaviour). Sherlin, et al., propose that discrete (versus continuous) feedback would be superior in its ability to “inform the learner whether the response was right or wrong and to what extent the brain signal changed” (p. 298).

Shaping

The continuous updating of reward thresholds in an a priori direction will promote learning where it is possible for the individual to perform the target behaviour (or its approximations). In some forms of neurofeedback, this is not the case because there is no target or ‘norm’ behaviour (e.g., slow-cortical potentials training). However, ‘auto-thresholding’ may be useful for double-blind research purposes contributing to null findings because an auto-threshold neurofeedback methodology will violate shaping as a fundamental learning component in neurofeedback training (Sherlin, et al., p. 299).

Specificity

This potential has previously been discussed with reference to the subjects of Kamiya who were able to selectively produce alpha. Sherlin, et al., provide an example for SMR neurofeedback training whereby “including metrics such as time above threshold or sustained reward period, the feedback will become more specific to the true SMR rhythm” (p. 300).
Artifacts
To minimize artifacts and to obtain acceptable EEG data, protocols are needed to: calibrate equipment; detect and rectify common artifacts in recordings; and to assess the acceptability of EEG recordings. Physiological artefacts are typically ocular (EOG), muscular (EMG) or cardiac (EKG) and may also be generated from state changes in the subject (such as sweating or drowsiness).

Secondary Reinforcement
In addition to, learner engagement and motivation for neurofeedback training can be further enhanced by associating the primary reinforcement (within-training response–reinforcer such as audio-tones or points on screen) with a secondary reinforcement for achieving the target behaviour. Clinical examples could include points or tokens that can be exchanged for a prize or purchase that is desirable to the individual.

Generalisation
Generalisation, both across time and across state, has been demonstrated in a range of studies and has also been facilitated by transfer trials, neurofeedback trials which provides information only at the end of the trial, rather than real-time feedback within session (Sherlin, et al., p. 301).

After skill acquisition, techniques that neurofeedback clinicians may incorporate in therapy to maximise generalisation, include:

- varying the frequency and power of rewards within sessions and perhaps adding tangible and social reinforcers (particularly skilling family members to reinforce target behaviours);
- varying the visual and auditory instructions, cues and stimuli given during neurofeedback;
- varying the setting of neurofeedback sessions or the extraneous variables such as lighting, noise, temperature, technician etc.; and
- behaviour schedules/journals to guide, record and reinforce ‘home practice and learning’.

****Mechanisms of Action Have Implications for Research and Application****

Not all neurofeedback practitioners employ operant conditioning methods as fundamental to their principles of neurofeedback practice. As brain-computer interface technologies become prolific, suppliers promote ways to exploit the market, professional and public consumers alike. To protect the integrity of our mental health professions and the integrity of this field of applied neuroscience, it is of paramount importance that we promote minimum standards of training, supervision and regulation to ensure neurofeedback in research and therapeutic contexts upholds best practice such as promoted by the Biofeedback Certification International Alliance – Australia (bciaaustralia@gmail.com).

Variations in neurofeedback methodologies were highlighted in a study by Gevensleben, Moll, Rothenberger & Heinrich (2014) who argued that the underlying models of feedback and control being assumed by NFB studies must be clearly articulated and aligned with the subsequent application of neurofeedback and its evaluation strategies. From their review of the literature on neurofeedback in ADHD, the authors identified a range of explicit and implicit mechanisms of action which, at the extreme poles, they determine to be Conditioning-Repairing and Skill-Acquisition models. These may align with models of homeostasis and allostasis and the implications for intervention (table 2) are significant: do we ‘fix’ the deficit or strengthen resources?
TABLE 2. Concurring assumptions and resulting ways of application regarding NF

<table>
<thead>
<tr>
<th>Assumptions</th>
<th>&quot;Conditioning-and-repairing model&quot;</th>
<th>&quot;Skill-acquisition model&quot;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indication</td>
<td>Specific neurophysiological deficit</td>
<td>No specific deficit</td>
</tr>
<tr>
<td>Mechanisms of learning (EEG regulation acquisition)</td>
<td>Automatic, unconscious (implicit) learning (operant conditioning of EEG pattern)</td>
<td>Controlled, effortful acquisition of regulation skills (explicit learning)</td>
</tr>
<tr>
<td>Significance of psychological and social variables and personality traits as</td>
<td>Susceptibility to basic learning mechanisms (operant conditioning), no higher-order cognitive</td>
<td>Effects moderated/mediated by cognitive-attributonal variables; generalization of effects</td>
</tr>
<tr>
<td>moderators/mediators</td>
<td>processes involved</td>
<td>moderated by social support, positive reinforcement of target behavior</td>
</tr>
<tr>
<td>Effects of the treatment</td>
<td>Automatic change in EEG-trait tonic change,</td>
<td>Change in EEG-state (phase changes), acquisition of self-regulation skills, enhancement of</td>
</tr>
<tr>
<td></td>
<td></td>
<td>neurophysiological functioning</td>
</tr>
</tbody>
</table>

| Ways of application                                                          |                                                                                                 |                                                                                           |
| Instructions, acquisition of self-regulation                                 | No active trainer, no specific instructions/no effort needed, passive participant                | Active coaching, support in the search for regulation strategies, active participant, effort to enhance self-regulation skills |
| Generalization                                                               | Automatic transfer to daily life → no effort necessary to support generalization                | Transferals; tasks for generalization of effects (e.g., homework)                           |
| Setting                                                                      | Unimodel treatment (Repairing the EEG deficit "normalizes" behavior.)                           | Module in a multimodal treatment; involvement of parents/teachers                            |
Research Evidence Base for Neurofeedback

Neurofeedback (NFB) draws on theory and research from neurophysiology, neuropsychiatry, neurobiology, psychophysiology, and psychology to deliver evidence-based and holistic clinical practices. Accountable practitioners need to balance internal and external validity with consideration to practice based on evidence and practice based on research (Barlow, 2010).

One of the criticisms of neurofeedback is the over-reliance of single-case or small clinical studies published in the literature. However, the single-case design conceptualizes the treatment of an individual as an experimental process that can be monitored over time and evaluated for effectiveness (Segool, et al., 2007). Treatment efficacy can be tested for a single patient and compared over time with subsequent patient findings. Positive and negative effects are readily recognised and managed. Negative effects from treatment are sometimes obscured as slower responses, less remission or recovery, higher rates of relapse or recurrence, or some combination of these (Barlow, 2010). Longitudinal studies might demonstrate durability and development of effects over time in treatment groups, data that might contrast well against the ‘wash-out’ of placebo effects in controls. Although the best reference condition might be an untreated control group, it might not be considered ethical to withhold an effective evidenced-based treatment for a patient at high-risk of harm without treatment.

The evidence-base for neurofeedback is founded on 3 scientific findings:
- research has demonstrated that neurological dysfunction, reflected in measurable EEG parameters, is indicated in particular disorders impacting mental health and functioning;
- with feedback about their EEG parameters, subjects can voluntarily change their EEG activity; and
- the brain can learn and hold this new state via neuroplasticity (Kropotov, 2009).

Although a non-invasive treatment modality, NFB resembles pharmacotherapy given its capacity to stimulate neurotransmission (Butnik, 2005). NFB offers a viable alternative and/or complementary treatment to the traditional medical model and is also considered to be the treatment of choice where “medication is ineffective, only partially effective, has unacceptable side effects, or where medication compliance is low” (Rossiter & La Vaque, 1995, p. 11). NFB treatment is also a cost effective alternative to the long term use of medication, for example, only 60-70% of individuals with ADHD do not outgrow symptoms and will therefore require some form of ongoing treatment (Rossiter & La Vaque). The assessment of treatment efficacy, specificity and clinical utility of neurofeedback interventions remains an ongoing challenge for researchers.

Resting state EEG measures (e.g., amplitude and frequency) at the scalp, can be subject to quantitative analysis (qEEG) of brain activity. Extensive reviews of well-designed EEG and qEEG studies revealed consistent findings among different diagnostic groups, suggesting there should be a strong positive recommendation for using EEG as a diagnostic tool for assessment and treatment of a range of clients including, for example, those with attention disorders for whom QEEG evaluation improves the rate of clinical success (Wright & Gunkelman, 1998; Hughes & John, 1999).

Accountable practitioners develop a therapeutic alliance with patients to design and implement knowledge-informed treatment plans relevant to the social, cultural and clinical context. Internal and external (ecological) validity need to be balanced with consideration to practice based on research and practice based on clinical experience and patient characteristics. This scientist-practitioner approach to psychological intervention is proposed by Barlow to be best practice for studies of efficacy and effectiveness, i.e., single-case experimental (idiographic) designs that are more closely aligned to clinical practice and which readily identify sources of inter-subject and intra-subject variability and manage these individual differences in order to promote the generality of findings (2010).
Current Evidence of Efficacy for Neurofeedback

For medical conditions, investigators have a high degree of certainty regarding the presence or absence of the disorder, while in psychiatry our diagnostic manuals are currently dependent on behavioural symptoms and subjective diagnoses. This will change as technologies advance to improve measures of the neurophysiological correlates of psychopathology. In an independent review of the literature, Duffy (2000) found that “the literature, which lacks any negative study of substance, suggests that ...EEG biofeedback... should play a major therapeutic role in many difficult areas.... if any medication had demonstrated such a wide spectrum of efficacy, it would be universally accepted and widely used.”

Schoenberg and David (2014) conducted a review of how biofeedback interventions have been used to treat a range of psychiatric disorders including anxiety, autistic spectrum disorders, depression, dissociation, eating disorders, schizophrenia and psychoses. EEG biofeedback was employed in 31.7% (n = 20) of all reviewed studies (the majority of NFB studies treating anxiety), the remainder incorporating EMG, HRV and/or other biofeedback or multi-modal methodologies. Fourteen studies (70.0%) reported statistically significant clinical amelioration following NFB. Multi-modal biofeedback appeared most effective in significantly ameliorating symptoms, suggesting that targeting more than one physiological modality for bio-regulation increases therapeutic efficacy:

- 14 of the NFB studies (70.0 %) included a comparison treatment (sham/placebo), a differing EEG parameter for feedback, another clinical intervention, or no treatment/wait-list control;
- 7 interventions (35.0 %) were randomized, four (20.0 %) nonrandomized, and for the remaining 9 (45.0 %) randomization was not feasible; and
- 23.7 was the mean number of sessions per study (range 5–69), with BF exposure lasting 28.7 min (range 14.6–60 min) on average per session.

Of all the biofeedback modes, NFB seems particularly promising for disorders where inducing particular states of conscious experience (through the alteration or regulation of cortical oscillatory activity) is a driving mechanism in alleviating symptomatology (Shoenberg & David, p. 125). In the treatment of at least one disorder, Attention-Deficit/Hyperactivity (ADHD), neurofeedback is considered to be the only type of treatment demonstrating sustained improvement of the central symptoms of pathology in the absence of stimulant therapy (Monastra, et al., 2002; Rossiter & La Vaque, 1995). Niv (2013) reviews the effectiveness of neurofeedback in ADHD, autism spectrum disorders, substance use, PTSD, and learning difficulties and found that neurofeedback emerged as superior or equivalent to either alternative or no treatment in many of the examined studies.

A special issue of Child and Adolescent Psychiatric Clinics of North America was devoted to emerging interventions that affect brain function, with neurofeedback featuring in seven of the ten chapters in the volume. About neurofeedback Hirshberg, et al., (2005) concluded, that EEG biofeedback meets the American Academy of Child and Adolescent Psychiatry (AACAP) ‘Clinical Guidelines’ criteria for treatment of ADHD, seizure disorders, anxiety (OCD, GAD, PTSD, phobias), depression, reading disabilities, and addictive disorders, whereby AACAP’s four-level scale is:

- **Minimal Standard** (MS) applies to recommendations backed up by rigorous empirical evidence, and/or an overwhelming clinical consensus;
- **Clinical Guidelines** (CG), recommendations based on strong empirical evidence and/or strong clinical consensus;
- **Option** (OP) applies to recommendations that are acceptable based on emerging empirical evidence or clinical opinion, but lack strong empirical evidence and/or clinical consensus;
- **Not Endorsed** (NE) applies to practices that are known to be ineffective.

In contrast, Sibley, et al., (2014) did not find evidence to support the AACAP position, their primary criticism being that NFB showed mild impact on ADHD symptoms but in particular, studies did not include measures of impact on impairment (e.g., academic, social and familial issues).
Much of the criticism (Loo & Barkley, 2005; Monastra, 2005) of neurofeedback studies has highlighted small sample sizes, randomization, inadequate control groups, and poor specificity of the treatment effects. Although single-case or uncontrolled studies dominate the literature, these clinical studies often inspire subsequent, more robust studies.

Two prominent international organisations (AAPB & iSNR), representing professionals engaged in applied neuroscience, collaborated in order to evaluate the neurofeedback research and to provide a template for scientific review of the evidence base for neurotherapies:

An essential function of both the Association for Applied Psychophysiology and Biofeedback (AAPB) and the (international) Society for Neuronal Regulation (iSNR) is the systematic evaluation of psychophysiological interventions that have been developed for the treatment of medical and psychiatric disorders (La Vaque, et al., 2002).

Table 3. Criteria for Levels of Evidence of Efficacy  (La Vaque, et al., 2002)

<table>
<thead>
<tr>
<th>Level</th>
<th>Descriptor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level 1</td>
<td>Not empirically supported (only anecdotal reports and/or non-peer reviewed case studies)</td>
</tr>
<tr>
<td>Level 2</td>
<td>Possibly Efficacious. At least one study of sufficient statistical power with well identified outcome measures, but lacking randomized assignment to a control condition internal to the study.</td>
</tr>
<tr>
<td>Level 3</td>
<td>Probably Efficacious. Multiple observational studies, clinical studies, wait list controlled studies, and within-subject and intra-subject replication studies that demonstrate efficacy.</td>
</tr>
</tbody>
</table>
| Level 4 | Efficacious: (a) In a comparison with a no-treatment control group, alternative treatment group, or sham (placebo) control utilizing randomized assignment, the investigational treatment is shown to be statistically significantly superior to the control condition or the investigational treatment is equivalent to a treatment of established efficacy in a study with sufficient power to detect moderate differences,  
(b) The studies have been conducted with a population treated for a specific problem, for whom inclusion criteria are delineated in a reliable, operationally defined manner,  
(c) The study used valid and clearly specified outcome measures related to the treated problem.  
(d) The data are subjected to appropriate data analysis,  
(e) The diagnostic and treatment variables and procedures are clearly defined in a manner that permits replication of the study by independent researchers, and  
(f) The superiority or equivalence of the investigational treatment have been shown in at least two independent research settings. |
| Level 5 | Efficacious and Specific. The investigational treatment has been shown to be statistically superior to credible sham therapy, pill, or alternative bona fide treatment in at least two independent research settings. |

According to this Efficacy Task Force (La Vaque, et al.), neurofeedback applications achieved Probably Efficacious (Level 3) with this finding being predominantly based on ADHD and NF research by Monastra (2002).

A meta-analysis of the ADHD research has since claimed Level 5 efficacy, demonstrating a large effect size for inattention and impulsivity (Arns, et al., 2009).
Research Evidence of Neurofeedback in Mental Disorder & Optimal Performance

In the rapidly growing body of literature, there is modest research evidence supporting neurofeedback therapies for a broad range of conditions. While not conclusive, there is sound evidence that warrants neurofeedback at least as an adjunct therapy for conditions such as ADHD, Seizure Disorders, Addictive Disorders, Anxiety and PTSD, particularly where clients are non-responsive to pharmaceuticals or experiencing abreaction to other treatment options.

Attention-Deficit/Hyperactivity Disorder (ADHD)

Although the clinical consensus holds that ADHD is comprised of three primary symptoms of impulsivity, inattention and/or hyperactivity, the co-morbidity of ADHD with disorders such as autism, mood or behavioural disorders complicates accurate diagnoses and treatment efficacy. In the recently released fifth revision of the diagnostic manual, the American Psychiatric Association has included ADHD in the section on Neurodevelopmental Disorders affirming the current conceptualisation of ADHD being the result of abnormalities in brain development (APA, 2013). However, biological testing is not routine for diagnosing ADHD.

Monastra, Monastra and George, 2002:
The effects of stimulant therapy, EEG biofeedback, and parenting style on ADHD were tested:
- 100 subjects, 6-19 years of age.
- All received stimulant medication, parenting and academic supports
- Half also received weekly NFB (parental choice) – average of 43 sessions
- Pre & post treatment measures: IQ, behaviour rating scales, TOVA, qEEG
  ➢ Both NFB and medication groups showed significant improvements in TOVA, rating scales, and qEEG post treatment
  ➢ Only NFB group maintained gains when reassessed after withdrawal from medication
  ➢ Positive treatment effects were measurable at 2 year follow up

Lévesque, Beauregard and Mensour, 2006:
Given mounting evidence that neurofeedback training (NFT) can significantly improve cognitive functioning in ADHD, research was conducted to examine the anterior cingulate cortex (ACC) which is involved in tasks requiring selective attention. An fMRI study measured the effect of NFB on neural substrates of selective attention in:
- 20 subjects, unmedicated children with ADHD, 8 – 12 years of age
- randomly assigned to NFB (15) or no-treatment control group (5)
- Pre and post fMRI while performing a Counting Stroop task
- Pre and post Digit Span, IVA, DSM clinical interview, Conners’ Parent ratings
- No differences in any measure between groups at Time 1.
- NFB: 40 sessions of NFB at CZ
  • 20 increase 12-15 HZ, decrease 4-7
  • 20 increase 15-18 HZ, decrease 4-7
  ➢ No change in controls on Conners, IVA, Digit Span, CST
  ➢ Significant improvements in NFB group on all measures
    • Post treatment, only NFB group showed new significant loci of activation in the right ACC (Brodman Area 32).
**ADHD continued...**

**Gevensleben, et al., 2009:**
To evaluate the clinical efficacy of neurofeedback in children with ADHD, a multisite randomised study was conducted using computerised attention skills training as a control condition for:
- 102 children with ADHD, aged 8 to 12 years
- 36 sessions of NF or computerized attention training — random assignment
- 2 blocks of NF (theta/beta then SCP)
- Pre/intermediate/post training ADHD symptom measures, parent & teacher rating scales.
- Parents assessed for attitude toward treatment group

- NF group ratings superior to controls post-training re reduction in symptom severity
- Measures of oppositional/defiant behavior also improved
- Comparable effects for the two NF protocols (theta/beta training, SCP training)
- Parental attitude did not differ between NF and control groups

**Steiner, Frenette, Rene, Brennan, & Perrin, 2014:**
To evaluate the efficacy of two school-based computer attention training systems, a randomised control study was conducted for:
- 104 children with ADHD, aged 7-11 years
- 40 sessions T/B neurofeedback or cognitive training (Captain’s Log) or control
- 6-month follow up measures of behaviour, executive function, use of medication

- NF group significant gains (Inattention effect size $[ES] = 0.34$, Executive Functioning ES = 0.25, Hyperactivity/Impulsivity ES = 0.23) i.e., significantly greater than gains in CT and control
- NF group maintained the same stimulant medication dosage, whereas participants in both CT and control conditions showed statistically and clinically significant increases (9 mg $[P = .002]$ and 13 mg $[P = .001]$, respectively).

**CONCLUSIONS:** Neurofeedback participants made more prompt and greater improvements in ADHD symptoms, which were sustained at the 6-month follow-up, than did CT participants or those in the control group.

**Holtmann, Pniewski, Wachtlin, Wörz & Strehl, 2014:**
To examine the efficacy of slow cortical potentials neurofeedback, a controlled multi-centre study (5 trial centres) was conducted using electromyographic (EMG)-feedback as an active control for:
- 144 children with ADHD, aged 6 to 10 years
- 25 sessions in 3 months of SCP-feedback or EMG control
- Pre/intermediate/post -test battery at 6 months, ADHD symptom measures, behaviour rating scales, psychometric tests and neurophysiological measures via qEEG & ERP.

**Strehl et al., 2017:**
A randomised control study was conducted to examine the efficacy of NF in comparison to electromyographic (EMG) feedback to control for unspecific effects of the treatment, and assessed self-regulation of slow cortical potentials (SCPs):
- 150 children with ADHD, aged 7-9 years (82% male; 43% medicated)
- Randomised to 25 sessions SCP neurofeedback or EMG feedback
- Compared pre-treatment parent ratings of ADHD symptoms and 4 weeks post-treatment

- NF proved significantly superior to EMG with an effect size (ES) of $d = 0.57$ without and 0.40 with baseline observation carried forward (BOCF).
- The sensitivity analysis confirmed the primary result. Successful self-regulation of brain activity was observed only in NF.
**ADHD continued...**

**Meta-analyses:**

- Arns et al., (2009) concluded that “NFB for treatment of ADHD is efficacious and specific (level 5) with a large effect size for inattention and impulsivity and medium effect size for hyperactivity”.

- Lofthouse, et al., (2012) reviewed 14 randomized published trials and unpublished conference presentations on the neurofeedback (NF) treatment of pediatric ADHD. Most studies used theta/beta midline protocols and demonstrated an overall ADHD mean effect size of $d = 0.69$, a medium effect, concluding that NF for paediatric ADHD is “probably efficacious.”

- Niv (2013) reviewed the effectiveness of neurofeedback in ADHD and found that neurofeedback emerged as superior or equivalent to either alternative or no treatment in many controlled studies with effect sizes ranging from 0.35 to 1.15. Protocols were typically increasing beta frequencies and decreasing theta frequencies, or modification of SCP responses. Given that four studies found comparable effects to a proven approach (assisted attention skills training), neurofeedback for ADHD qualifies as ‘**efficacious and specific**’.

- Mayer, et al., (2013) reviewed all published studies on slow cortical potentials (SCP) neurofeedback for the treatment of ADHD and reported that SCP is an efficacious and standardized neurofeedback protocol that yields moderate to large within group effect sizes for reduction of the core symptoms of ADHD.

- Hodgson, et al., (2014) conducted a meta-analysis of seven nonpharmacological treatments for ADHD including behaviour modification, neurofeedback therapy, multimodal psychosocial treatment, school-based programs, working memory training, parent training, and self-monitoring. Behaviour modification and neurofeedback treatments were most supported by the findings, however when their overall effect on outcome measures were examined, neurofeedback treatment was the most efficacious, being associated with the largest positive average weighted effect size, and thus greatest improvement in the treatment group.

- Arns, et al., (2014) reviewed studies published since 2009 which employed (1) semi-active, (2) active, and (3) placebo-control groups; and it concluded that standard protocols such as theta/beta, SMR and slow cortical potentials neurofeedback are well investigated and have demonstrated specificity.

- Micoulaud-Franchi, et al., (2014): meta-analysis of published Randomized Controlled Trials with semi-active control and sham-NF groups. Five identified studies met eligibility criteria. 263 patients with ADHD were included and 146 patients were trained with EEG-NF. This meta-analysis of EEG-NF in children with ADHD highlights improvement in the inattention dimension of ADHD symptoms. Future investigations need adequately blinded studies.
Seizure Disorders/Epilepsy

Seizure disorder is one medical condition which occurs when there is damage or disruption to the normal electrical function of the brain, with electrical discharges causing abnormal sensations and behaviours. Epileptiform activity is distinctive waves or complexes, distinguished from background EEG activity. These waves or complexes can appear as: isolated focal spikes, more generalized polyspikes, sharp waves, spike and wave activity, paroxysmal fast activity, and sometimes as abrupt rhythmic activity that heralds seizures (Greenfield, 2010). EEG biofeedback (later neurofeedback) has been explored as a treatment for epilepsy from as early as the 1970s, first in relation to modulation of the sensorimotor rhythm and later via regulation of the slow cortical potential (SCP).

There have been multiple controlled studies documenting changes in EEG following NF treatment providing evidence that NF for epilepsy is ‘efficacious’. For example:

Whitsett, et al., 1982:

To determine the effectiveness of EEG biofeedback for the control of seizures, a double-blind, ABA crossover investigation was conducted:
- 8 medically refractory epileptic patients
- SMR biofeedback treatment reinforced suppression of 3-7 Hz, enhancement of 12-15 Hz, or simultaneous suppression of 3-7 Hz and enhancement of 11-19 Hz activity
- polysomnographic recordings showed decreases in nocturnal 4- to 7-Hz activity correlated with decreases in seizure activity
- increases in 8- to 11-Hz activity were correlated with decreases in seizure activity
These findings strengthen the hypothesis that EEG biofeedback may produce changes in the sleep EEG that are related to seizure incidence.

Tan, et al., 2009:

Found 63 studies indexed from 1970 to 2005 which included measures of seizure frequency and responses to EEG biofeedback. 10 studies had adequate data for inclusion in the meta-analysis:
- N = 87
- 9 studies reinforced sensorimotor rhythms (SMR)
- 1 study trained slow cortical potentials (SCP)
- all studies reported an overall-mean decreased seizure incidence following treatment
- 64 patients (74%) reported fewer weekly seizures in response to EEG biofeedback
Significantly, all subjects were patients had seizures not controlled by medical therapies

Ayers, 1995:

Margaret Ayers reported to the 26th Annual Meeting of the Association for Applied Psychophysiology and Biofeedback regarding the first long-term follow up of patients. She had been following cases from 1978, utilizing EEG neurofeedback with absence seizures:
- 10 subjects aged 12 – 25 years
- 5 medicated, 5 non-medicated
- alternating T3C3 T4C4, NFB suppressing 4-7 Hz & increasing 15-18 Hz

- all 10 absence seizure cases became and remained medication-free and seizure-free.
Addictive Disorders

Addiction refers to the harmful consumption of all drugs (including alcohol). What addictions have in common is their neurological involvement in the reinforcement of behaviours and the production of memories. Addictions so intensely activate the reward system that normal activities may be neglected (DSM 5, 2013). Some individuals with lower levels of self-control, may be particularly predisposed to develop substance use disorders.

The Peniston/Kulkosky protocol incorporated neurofeedback (Peniston & Kulkosky, 1989; Saxby & Peniston, 1995) to effectively increase alpha-theta and decrease symptoms of alcoholism among subjects in a randomized and controlled study. There is an emerging body of evidence supporting alpha-theta and/or SMR/beta-training as ‘probably efficacious’ (Trudeau, 2005) for addictions, particularly to address the comorbid neuropathologies that are often evident (e.g., ADHD):

Scott, et al., 2005:

An RCT study of the effects of EEG biofeedback on 121 inpatients with substance abuse:
- Random assignment to control group or NF biofeedback for 40 -50 sessions:
  - beta/SMR protocol for attentional training
  - Peniston protocol (alpha/theta)
- outcomes measured: days sober, attention (TOVA), and personality (MMPI)
  - 46% percent of treatment as usual (TAU) subjects dropped out of the study
  - 24% of neurofeedback subjects dropped out
  - NF subjects remained in treatment 37 days longer
  - 77% percent of NF cases were sober at 1-yr follow-up
  - 44% of TAU cases were sober at 1-yr follow-up
  - NFB subjects, but not controls, exhibited significant reductions on personality scales
  - Effects of the training did not differ across different drugs of choice

Callaway & Bodenhamer-Davis, 2008:

A clinical trial provided a long-term follow-up of chemically dependent adult participants who were treated with the Peniston Protocol in a university outpatient clinic between 1993 and 1995:
- 16 participants, 10 who were probationers classified as ‘high-risk’ for rearrest
- pre/post measures of depression, personality, abstinence & re-arrest rates
  - depression scores reduced from mild/moderate Time 1 to within normal limits Time 2
  - MMPI results indicated less psychopathology following treatment
  - 81.3% (n = 13) subjects abstinent at follow-up (74-98 months)
  - Re-arrests/probation revocations for subgroup were lower than comparison group 40% : 79%
  - This study provides evidence of the durability of Peniston Protocol results over time.

Sokhadze, Cannon & Trudeau, 2008:

Reporting on three decades of publications examining EEG biofeedback and addictions and with reference to the AAPB and ISNR criteria for reviewing research, the authors summarised that “alpha theta training either alone for alcoholism or in combination with beta training for stimulant and mixed substance abuse and combined with residential treatment programs, is probably efficacious”.

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**Post-traumatic Stress Disorders (PTSD)**

PTSD has now been classified by the 5th edition of the DSM as belonging to the class of *Trauma- and Stressor-related Disorders* (including disorders of reactive attachment, disinhibited social engagement, PTSD, acute stress, & adjustment), all requiring *exposure to a traumatic or stressful event* as a diagnostic criterion, and separating the class from anxiety, obsessive-compulsive and dissociative disorders (APA, 2013).

**Peniston and Kulkosky, 1991:**

The authors conducted an RCT to compare treatment as usual (TAU group) with alpha/theta brainwave therapy (BWT group) to demonstrate the effect on PTSD symptoms, dependence on psychotropic medications for PTSD, and personality indicators as measured by the MMPI:

- 29 in-patient Vietnam veterans with PTSD volunteered to participate
- random assignment, 15 to experimental and 14 to control group
- BWT group received 30 x 30 mins NF sessions
- TAU group continued usual treatments

Results at end of treatment:
- all BWT subjects had reduced medication consumption
- only 1 TAU subject reduced medication, 2 = no change, 10 required additional medication
- both groups showed decreases on the MMPI schizophrenia scale
- only the BWT group showed reduced hypochondriasis, depression, hysteria, psychopathic deviation, paranoia, psychasthenia, hypomania, introversion, and PTSD symptoms

Results at 30-month follow up:
- all control group subjects had relapsed with PTSD symptoms
- only 3 of the 15 BWT subjects had relapsed.

**Anxiety Disorders**

Anxiety refers to the thoughts and bodily reactions a person has when they are presented with an event or situation that they feel they cannot manage or undertake successfully (Simpson, et al., 2010). Anxiety-related emotions are correlated with autonomic hyperarousal which can be difficult for patients to recognise and regulate. Symptom ambiguity contributes to diagnostic challenges.

While not conclusive, there is evidence that warrants neurofeedback at least as an adjunct treatment for symptoms of anxiety.

**Jarusiewicz, 2002:**

Although this author conducted a controlled study of the efficacy of NFB for children with ASD, the treatment group had a reduction in anxiety symptoms:

- 24 children aged 4–13 years
- NF treatment group versus waitlist Controls
- duration 6–8 months
- pre/post parent interview.

**Moore, 2000:**

A review of controlled studies of EEG biofeedback treatment of anxiety disorders found protocols for enhancement of alpha, theta and alpha-theta were most effective.
Peak (Optimal) Performance

Gruzelier (2014, 2014b) reviewed EEG neurofeedback for optimising performance, first in the context of cognitive and affective outcomes in healthy participants, and secondly for creativity.

Cognitive and affective outcomes:
- 23 controlled studies including correlations indicating gains being mediated by NF learning
- protocols include SMR & SMR ratio, beta1 & beta1 ratio, eyes closed alpha/theta ratio, upper-alpha, alpha desynchronisation, gamma & gamma ratio, frontal theta up-training, posterior theta up-training, and down-training theta EEG maxima.
- outcomes favoured: sustained attention, orienting and executive attention, the P300b ERP component, memory, spatial rotation, complex psychomotor skills, implicit procedural memory, recognition memory, perceptual binding, intelligence, and wide ranging aspects of mood and well-being.

NFB impact on creativity (especially in performing arts):
- protocols predominantly alpha/theta, SMR ratio, beta1 ratio, and HRV training
- outcomes favoured: as found above in cognitive and affective studies.

Quality research is needed to further examine the efficacy and effectiveness of neurofeedback for treatment of the following conditions, although preliminary findings are promising for:

- Depression
- Obsessive-Compulsive Disorder
- Traumatic Brain Injury
- Autism Spectrum Disorders
- Learning Difficulties
- Migraines
- Bipolar Disorder

Extensive bibliographies of NFB are available, for example, at:


ISNR, http://noviancounseling.wix.com/bibliography


References


Greenfield LJ, Geyer JD, Carney PR. Reading EEGs: A Practical Approach. Lippincott Williams & Wilkins


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Assessment Items for your Continuing Professional Development and Reflection

(You are not required to submit these responses. Please attempt to complete the tasks to consolidate your understanding of some of the key concepts addressed today and to identify some areas of interest for your future professional development.)

Workshop Aims
1. Develop insight into the neural circuitry involved in psychopathology and the application of EEG biofeedback (neurofeedback) for modulation of brain states toward healthy self-regulation.
2. Identify the application of learning theory and principles to neurofeedback.
3. Review the research history and current evidence for neurofeedback.

Measure of Learning
- Define neurofeedback (EEG biofeedback)
- Define the following terms: Operant Conditioning, Neuroplasticity
- Psychological functions have physiological correlates within what system/s of the body?
- What are some of the common physiological symptoms of psychological distress?
- Which NFB researcher is associated with the following concepts?
  - sleep and creativity > alpha-theta NFB psychotherapy
  - alpha-theta NFB protocol to treat alcoholism
  - efficacy study of SMR NFB for epilepsy
  - trained cats’ SMR > NASA funded-study > launched SMR NFB
  - open head trauma & TBI

  [Discuss: Sterman, Ayers, Peniston, Lubar, Budsynski]
- Describe the efficacy and effectiveness levels indicated by current research for at least two of the following conditions: ADHD, Addictions, PTSD, Autism, TBI, Depression, & Anxiety.

Item 1: Define Neurofeedback (EEG Biofeedback)

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Item 2: Describe how Operant Conditioning and Neuroplasticity form the philosophical and psychophysiological foundations for neurofeedback therapy.

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__________________________________________________________________________
Item 3: Considering the psychobiological viewpoint, describe how the psychological features of emotion have physiological correlates within the behavioural, autonomic and endocrine systems.

**Behavioural:**

**Autonomic:**

**Endocrine:**

Item 4: What are some of the common physiological symptoms of psychological distress?

Item 5: Match the NFB researcher by putting the correct LETTER label into the central column to link the person to the relevant concept.

<table>
<thead>
<tr>
<th>Sterman</th>
<th>A. sleep and creativity &gt; alpha-theta NFB psychotherapy</th>
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<tbody>
<tr>
<td>Lubar</td>
<td>B. alpha-theta NFB protocol to treat alcoholism</td>
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<tr>
<td>Birbaumer</td>
<td>C. efficacy study of SMR NFB for epilepsy</td>
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<tr>
<td>Rosenfeld</td>
<td>D. first to record human brain electrical potentials</td>
</tr>
<tr>
<td>Ayers</td>
<td>E. pioneering NFB study inspired by Zen monks</td>
</tr>
<tr>
<td>Budsynski</td>
<td>F. trained cats’ SMR &gt; NASA funded-study &gt; launched SMR NF</td>
</tr>
<tr>
<td>Peniston</td>
<td>G. slow-cortical potentials’ operant conditioning</td>
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<tr>
<td>Green</td>
<td>H. open head trauma &amp; TBI</td>
</tr>
<tr>
<td>Berger</td>
<td>I. alpha enhancement</td>
</tr>
</tbody>
</table>

Item 6: Describe the current evidence-base and level of efficacy for neurofeedback in the treatment of one area of mental disorder of interest to you in your clinical practice.

**Additional notes for your CPD journal:**
Feedback to Presenter re Learning Objectives – email michelle@msmh.com.au

1. Develop insight into the neural circuitry involved in mood, anxiety, attention, memory & sleep and the application of EEG biofeedback (neurofeedback) for modulation of brain states toward healthy functioning.

1a. To what degree was the learning outcome met? (please circle number to indicate your opinion)

Not very well met 1 2 3 4 5 Very well met
Comment:

1b. Rate to what degree this session was relevant to your learning need and/or current practice.

Not very relevant 1 2 3 4 5 Very relevant
Comment:

2. Identify the application of learning theory and principles to neurofeedback.

2a. To what degree was the learning outcome met?

Not very well met 1 2 3 4 5 Very well met
Comment:

2b. Rate to what degree this session was relevant to your learning need and/or current practice.

Not very relevant 1 2 3 4 5 Very relevant
Comment:

3. Review the research history and current evidence for neurofeedback.

3a. To what degree was the learning outcome met?

Not very well met 1 2 3 4 5 Very well met
Comment:

3b. Rate to what degree this session was relevant to your learning need and/or current practice.

Not very relevant 1 2 3 4 5 Very relevant
Comment: