Electroencephalographic Applications

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Introduction

The goal of this module is to acquaint the reader with the exciting and rapidly expanding field of electroencephalographic (EEG) biofeedback. Terms that are synonymous with EEG biofeedback include neurofeedback, brain wave biofeedback, operant conditioning of brain waves, and neurotherapy. “Neurotherapy” sometimes refers to a broader range of interventions that can include audiovisual stimulation as well as operant feedback procedures (La Vaque, 2003) and “therapy” implies an intervention that targets the symptoms of a disorder. The more general terms “EEG biofeedback” or “neurofeedback” can be utilized for describing interventions for clients with a broad range of goals, from ameliorating a disorder to optimizing performance.

This module is not a how-to manual for neurofeedback assessments and training, which require hands-on demonstration and practice as well as theoretical knowledge. The objective here is to cover some of the principles of neuroscience that underlie the use of EEG biofeedback, discuss the frequencies that are trained, mention the effects of medication on the EEG, and outline the evidence for the use of EEG biofeedback with clients who present with a variety of conditions. The list of applications concludes with optimal performance, which ranges from dealing with athletes and executives to recent research that found an association between neurofeedback training and improved memory in medical students and enhanced musical performance in conservatory students (Gruzelier, 2003).

More than three decades ago, it was established that operant conditioning of brain wave activity in mammals was possible (Wyrwicka & Sterman, 1968) and that humans could consciously control alpha activity (Kamiya, 1968). Applying this work to humans, numerous publications show the efficacy of neurofeedback for treating seizure disorders and attention-deficit disorder (ADD). Because the precise mechanisms by which the changes are made and how they are sustained is not known, neurofeedback is an empirical field based on outcome research. The clinical results, however, are impressive; for example, children with ADD who learn to shift their brain wave patterns show improved management of symptoms, increased academic performance, decreased use of medication, and gains on intelligence test scores of ten or more points (Linden, Habib, & Radojevic, 1996; Lubar
Neurofeedback has become an established field with a basic text *The Neurofeedback Book: An Introduction to Basic Concepts in Applied Psychophysiology* (Thompson & Thompson, 2003) published by the Association for Applied Psychophysiology and Biofeedback (AAPB). EEG Biofeedback Certification that is separate from General Biofeedback Certification is available through the Biofeedback Certification Institute of America.

Practitioners from many disciplines are using neurofeedback in clinical practice to complement other modalities of treatment. Combining EEG biofeedback with peripheral biofeedback leads to effective interventions that underscore the unity—rather than the dichotomy—of mind and body. As William James wrote in *The Principles of Psychology* ([1890] 1981), “The great thing, then, in all education, is to make our nervous system our ally instead of our enemy” (vol. 1, p. 126).

### Neuroscience and EEG Biofeedback

#### Relevance of Structures in the Central Nervous System

The central nervous system (CNS) consists of the brain and the spinal cord. This is the control center for the rest of the nervous system, known as the peripheral nervous system, and indeed for the rest of the body. The neurons (nerve cells), which handle communication within the CNS, are numerous (about 100 billion) and complex (about fifty different kinds). One type of neuron, called a pyramidal cell, has the properties of a tiny battery with positive and negative charges at opposite ends. These cells produce most of the electricity that we measure as the electroencephalogram. It was established in the 1960s that EEG activity could be operantly conditioned in cats (Wyrwicka & Sterman, 1968). From that discovery grew applications to humans (Sterman & Friar, 1972) and the field of EEG biofeedback was born.

Though the brain weighs only 2–3 pounds, it uses 25 percent of the oxygen and 20 percent of the glucose when the body is at rest. That such a high proportion of the body’s resources is allocated to the brain attests to its importance. Influencing the brain can influence all other parts of the body. Communication pathways and patterns of activation or rest in the brain
relate to psychological states and activities including sleep-wakefulness; affect and emotion; pain; motor function; and executive functions such as attention, planning, and inhibition. There is no real mind-body distinction but, rather, a flow of information with complex feedback systems and great interdependence.

The brain is comprised of three major parts: hindbrain, midbrain, and forebrain (see Figure 1). Looked at phylogenetically, we have a reptilian brain that developed first and is surrounded by the limbic system, which in turn is wrapped in the neocortex. In broad general terms, the hindbrain is responsible for survival instincts and automatic responses; the midbrain for emotions and motivations; and the forebrain for higher reasoning. This module gives just the briefest overview concerning the complex topic of neuroanatomy. Only the main components related to the production of certain types of electrical activity in the brain or to particular behaviors will be mentioned.

Figure 1. Schematic diagram showing structures in the brain.
When we measure electrical activity with sensors placed on the scalp, we measure cortical activity. Each sensor, called a standard electrode, measures the activity generated by columns of pyramidal cells that lie below it. Although it originates in the surface layers of the cortex, this activity is influenced by deeper structures. Thus it is important to understand the various structures within the brain and their connections.

**Forebrain: Cerebral hemispheres.** As illustrated in Figure 2, the brain looks like a cauliflower with its lumpy ridges (called gyri) and valleys (called sulci or fissures if they are deeper). The two main divisions, the left and right hemispheres, lie on either side of the longitudinal cerebral fissure. Though fairly symmetrical, these two hemispheres have different functions. The left is mainly involved in sequential processing (such as generating language) and the right does faster, more simultaneous processing (such as expressing and reading emotions). The aspects of intelligence measured on an intelligence test depend primarily on left brain functioning, whereas “street smarts” (expressing emotion and navigating the social scene) depend more on right brain functions. Division of labor within the brain is complex and requires fine-tuned communication links among neurons; for example, although most language functions are in the left hemisphere, the emotional aspects of language—such as the meaning conveyed by voice intonation—are processed in the right hemisphere.

*Figure 2. Lateral view, left cerebral hemisphere, showing Broca’s and Wernicke’s areas (adapted from Thompson & Thompson [2003]).*
On each side the hemispheres are further divided into frontal, parietal, temporal, and occipital lobes of the cortex with the basal ganglia and the limbic system beneath. A central sulcus separates the frontal and parietal lobes. The lateral fissure separates the frontal and parietal lobes from the temporal lobe.

The EEG that we record comes from the cortex or gray matter on the surface of the brain. White matter is found at the lower levels and has a white color due to myelinated fibers. Myelin is a fatty coating along a neuron (similar to insulation) that allows for faster transmission of an electrical signal. The neocortex in mammals has six layers. The pyramidal cells that produce the electricity measured as EEG constitute the largest part of the third, fifth, and sixth layers.

The left and right hemispheres are connected in a number of ways, although there is more communication within than between hemispheres. Axon connections unite homotropic (same site) areas in the right and left cerebral hemispheres; for example, Wernicke’s area in the left hemisphere, which is involved in understanding language, is linked to the same site on the right side where the processing of the emotional content of language takes place. Connections are seen structurally as large bands of white matter or commissures, the largest of which is called the corpus callosum. These fibres are myelinated and therefore white in color.

Myelination in the brain occurs not just in the early years of life (there are spurts of myelin growth just before a baby learns to walk, for example), but also in the teenage years when the amount of myelin doubles. The latest discoveries in neuroscience indicate that the brain has more plasticity at all ages than previously thought possible. Plasticity means the ability to change, such as growing new cells and connections while also pruning existing connections. For a readable account of changes during adolescence, refer to The Primal Teen (Strauch, 2003), which was written by a journalist who reviewed the neuroscience research pertaining to that stage of development. For an inspiring review of changes in the elderly brain, read Aging with Grace (Snowden, 2001), which gives an account of the research findings from the so-called “Nun Study” and reviews the neuroscience research related to Alzheimer’s disease. This book also discusses
factors related to successful aging and describes many nuns who were leading productive lives when well over one hundred years old.

The central sulcus divides the motor (toward the front) and the sensory cortex. This part of the brain sends motor commands and receives sensory information (touch, temperature, pain) that has been relayed from the periphery to the cortex by way of specific areas in the thalamus. Pain has dual pathways, one of which goes through the thalamus and the other directly to the cortex. The visual cortex is located in the occipital area and the auditory cortex in the temporal lobes near the lateral fissure. The remainder of the cortex is called association cortex. In the frontal lobes, the association cortex is involved in cognitive functions, including executive functions such as planning. The frontal lobes are the last to come online, both phylogenetically and developmentally, and are still developing throughout the second decade of life.

**Forebrain: The limbic system.** The limbic system consists of the hippocampus, amygdala, and septum in the telencephalon and the anterior thalamic nuclei and mammillary body in the diencephalon. The amygdala, which is located deep within the temporal lobe, receives inputs from the thalamus, hypothalamus, midbrain, and temporal lobe. The amygdala has connections to all the areas concerned with emotions, the autonomic nervous system, and the endocrine system. It is an integral part of the system that controls autonomic and endocrine responses to emotional states. Animal experiments involving electrical stimulation of the amygdala result in aggression, whereas bilateral removal of the amygdala can result in a tame animal that appears indifferent to danger.

The amygdala is also involved in having affect (emotional expression) toward others and is important in memory. It appears to lay down unconscious memories that activate the autonomic nervous system state that accompanied the emotions evoked by an event (Carter, 1998). This phenomenon is particularly evident with traumatic memories. This is in contrast to the hippocampus, which is involved in laying down conscious memories. It is interesting to note that there is growth in myelin in many areas, including the links between the cingulate gyrus and the hippocampus, during the toddler and teen years. While these links are under construction, people are more prone to intense emotional reactions.
Forebrain: The basal ganglia. The structures that comprise the basal ganglia include the globus pallidus, caudate nucleus, and putamen. The basal ganglia-thalamic system is involved in the selection of actions. For appropriate executive actions, this system must flexibly select sensations, cognitions, and appropriate motor actions and also inhibit inappropriate sensations, motor actions, or irrelevant thoughts. The basal ganglia are involved in the motor system and are important in neurofeedback because of their importance in movement disorders, including Tourette’s syndrome, Parkinson’s disease, and dystonia. They also play a role in learning.

Forebrain: The diencephalon. The diencephalon lies between the telencephalon and the midbrain and contains the thalamus and the hypothalamus.

The name “thalamus” derives from the Greek word for “inner chamber” according to some and “antechamber” according to others. Both meanings are appropriate because it is an inner structure and, like an antechamber, things pass through the thalamus before going elsewhere. All sensory information (except smell) passes through the thalamus before moving on to the cortex. The neurons within the thalamus project to specific areas of the cerebral cortex. The connecting fibers, called projection fibers or axons, comprise feedback loops that are important for understanding EEG rhythms. The thalamus seems to set the slower rhythms, especially theta. Fewer than 5 percent of the connections in the brain are thalamo-cortical, but they have an influence that far exceeds their number because they set rhythms that are then relayed among cortico-cortical connections. Without the thalamus setting slow rhythms, we would not be able to turn off activation in the brain and fall asleep.

The hypothalamus, which lies under the thalamus and on either side of the third ventricle, is important in biofeedback because it is involved in the control of the autonomic nervous system. It is also a keystone in the control of the endocrine system. The pituitary gland is attached to its base and thus, as Carlson (1986) humorously puts it, this system is responsible for “the survival of the species — the so called 4 F’s: fighting, feeding, fleeing, and mating” (p. 104). Even maternal behavior may be controlled by genetically determined brain structures. The medial (on the side toward the middle) preoptic area, which contains estrogen receptors, is important for nurturing the young and is linked to the hypothalamus.
**Midbrain (Mesencephalon).** Following are the main points about structures in the midbrain.

- The reticular formation contains over ninety nuclei (a nucleus is a network of neurons) extending down into the brain stem. It receives sensory information and communicates with the thalamus, cortex and spinal cord. It is crucial to our understanding of sleep, arousal, and attention.

- The red nucleus assists our understanding of how increasing the sensorimotor rhythm of the EEG may help clients who are hyperactive and also those who suffer from movement disorders (Tourette’s syndrome, dystonia, and Parkinson’s disease). Sensorimotor rhythm activity is associated with suppression of cell discharge rates in the red nucleus (Sterman, 2000a). (Sensorimotor rhythm activity is discussed in detail later in this module.)

- The substantia nigra projects fibers to the caudate nucleus. Its production of dopamine is reduced in Parkinson’s disease.

- The periaqueductal gray matter is involved in fighting and mating in animals.

- The ventral tegmental area secretes dopamine and projects to the basal forebrain (medial forebrain bundle). It is involved in learning and has been implicated in schizophrenia. Dopamine is an important neurotransmitter in many disorders, such as attention-deficit disorder.

- The midbrain is the origin of the third and fourth cranial nerves, which control movements, pupillary dilation, and accommodation.

**Hindbrain (Metencephalon, Myelencephalon).** The metencephalon (anterior portion of the hindbrain) comprises the cerebellum and the pons. The cerebellum, like the cerebrum, is covered with cortex. It is involved in coordination and motor performance. Damage results in jerky, exaggerated movements on the same side of the body as the lesion. The pons is involved in sleep and arousal.

The myelencephalon (posterior portion of the hindbrain) consists of the medulla oblongata. It is involved in the functioning of the lungs and heart and is also implicated in the maintenance of skeletal muscle tone. An
important nucleus found in the hindbrain, which has connections to the forebrain, is the *locus coeruleus*. It is vital in the production of the neurotransmitter norepinephrine, which is thought to be important in attention-deficit disorder.

The pons and medulla contain the origins for cranial nerves 5–12, which are responsible for sensory, motor, and autonomic functions in the head region and face in particular.

**The Role of Neurotransmitters**

Reference has been made to communication among neurons and the electricity that is produced as this communication takes place. The electricity, which we usually measure from the scalp (although it can also be measured with electrodes implanted within the cortex), can be displayed as a line graph plotting amplitude against time—the electroencephalogram. (This is analogous to measuring the electrical activity of the heart and displaying it as the electrocardiogram [ECG].) At the neuronal level, axons and dendrites are like little feelers or branches: axons are little feelers that carry impulses from the cell body and dendrites are little feelers that carry impulses to the cell body. Any neuron among the billions of neurons in the nervous system can communicate with up to one thousand or more other neurons and each connection can be made by way of four or fewer synapses (Poeppel, 2002). Picture a magnificently complex road system with intersections and neurotransmitters controlling the traffic flow at each intersection.

The synapse is a gap or space between one neuron’s axon and another neuron’s dendrite and the signal has to be carried across that synaptic cleft. If you use the analogy of information being a car traveling on a highway, the car comes to the end of the road at a river and has to cross by ferry to the road that continues on the other side. The substance that ferries the information across is a neurotransmitter. The neurotransmitter is released (due to an influx of calcium) on the presynaptic side of the synapse, from the end of one cell’s axon, and goes across the gap and lands at a receptor site on the other side (the postsynaptic side). The receptor site is on the dendrite of an adjacent neuron and these sites are specific for particular neurotransmitters. In most instances the receptor site governs whether a transmission will be *excitatory* or *inhibitory*.
Researchers have identified over two hundred neurotransmitters. Following is a brief discussion of the four major groups of neurotransmitters and their clinical significance.

*Acetylcholine* is the most common neurotransmitter. It is the excitatory neurotransmitter at neuromuscular junctions but may be either excitatory or inhibitory in the CNS. It is involved in recording memories in the basal forebrain and the hippocampus and is deficient in Alzheimer’s disease. In the reticular activating system, acetylcholine has a role in attention and arousal. It is also involved in the control of the stages of sleep.

The *biogenic amines* group includes the much researched catecholamine dopamine. Schizophrenia, Tourette’s syndrome, and obsessive-compulsive disorder may involve an excess of dopamine and Parkinson’s and addictions are related to a reduction in dopamine. Dopamine is the principle neurotransmitter in the brain’s reward or pleasure circuit, which involves the medial forebrain bundle. Norepinephrine (the common neurotransmitter in the sympathetic portion of the autonomic nervous system) and dopamine are derived from the amino acid *tyrosine*. Dopamine is generally excitatory; norepinephrine can be either excitatory or inhibitory.

Amphetamines and cocaine are catecholamine agonists. They block the reuptake of dopamine and norepinephrine from the synaptic cleft and thus increase the availability of these transmitters to the postsynaptic neuron. When this happens in the nucleus accumbens, there is the experience of a “high.” Alcohol, nicotine, and caffeine can also increase dopamine in the nucleus accumbens.

Norepinephrine, produced in the locus coeruleus, has an excitatory function in the CNS related to arousal and attention. It is released during stress; may be a part of the “fight or flight response”; and is involved in emotions such as fear, anxiety, and possibly mania. It is also thought to have a role in learning and the formation of memories. Too little norepinephrine may be associated with depression and too much with mania. It may be in excess in some anxiety disorders but may be depleted in patients who have had chronic stress.

Serotonin, primarily an inhibitory neurotransmitter, is produced in the brain stem. It is involved in the regulation of pain, mood, appetite, sex drive,
and sleep; it may also be involved in memory. It is a precursor for melatonin, which in turn is important in biological rhythms. Low levels of serotonin are thought to be related to a number of psychiatric disorders including depression, obsessive-compulsive disorder, and aggression. Selective serotonin reuptake inhibitors (SSRIs) are used to treat these conditions.

The amino acids group includes the two inhibitory transmitters: gamma amino butyric acid (GABA) and glycine. It also includes glutamate and aspartate, which are excitatory transmitters. The anxiolytic medications (benzodiazepines such as Valium), alcohol, and barbiturates may exert their effects by potentiating the responses of GABA receptors. GABA is possibly the most important inhibitory neurotransmitter in the central nervous system because the entire CNS is a system in which, when a neuron is stimulated, a feedback loop is activated to inhibit or stop that neuron from continuously firing. Thus GABA mediates the braking and stabilizing mechanism of the CNS.

Glutamate is essential in learning and memory and in an important process called long-term potentiation. Long-term potentiation is the process whereby a postsynaptic cell is enhanced in response to episodes of intense activity across the synapses. It is thought to be crucial to memory storage.

Neuropeptides are short chains of amino acids that are responsible for mediating sensory and emotional responses. Among them is Substance P, which mediates the perception of pain. Measurements of Substance P in the cerebral spinal fluid are assisting clinicians in the diagnostic workup of persons suffering from fibromyalgia. The endorphins are also neuropeptides: they are the body’s natural opiates and function at the same receptor sites that receive heroin and morphine.

Some functions of neurotransmitters. Neurotransmitters can produce membrane depolarization, which leads to the generation of an action potential. Polarization means that you have two poles, one positive and one negative, such as the opposite poles of a battery (or, in this case, a negative charge within the cell and a positive charge outside the cell). If the difference becomes greater—i.e., the inside becomes even more negative—then
it has hyperpolarized. If the difference becomes less—i.e., the inside becomes less negative and starts to move toward a positive charge—then it has depolarized. If there is sufficient depolarization, then an action potential is generated and propagated along an axon to the next synapse.

Action potentials are all or nothing: if the resting level of about −70 millivolts depolarizes enough to reach the threshold for excitation of about −55 millivolts, an action potential is produced (the cell “fires”). If the threshold for excitation is not reached, no action potential is generated. The electrical change, if it does occur, is a temporary reversal in charge along the cell membrane. The charge changes from negative to positive (jumping from the critical level of −55 millivolts to about +110 millivolts) and lasts about one millisecond. There has to be a summation of inputs at the section of the neuron called the axon hillock that is sufficient to generate an action potential. The axon hillock is located where the axon joins the cell body.

The amount of neurotransmitter available at the synapse is one of the variables that determines whether there is sufficient summation (temporal, spatial, or both) to produce depolarization that reaches the threshold for excitation. This is why drugs that produce changes in neurotransmitter release and availability, such as SSRI medications, are so powerful and widely used. It also explains why it takes a few weeks for their effects to be felt. There must be time for changes to occur at the receptor sites that are due to the prolonged increase in availability of particular neurotransmitters.

Other types of medications that affect neurotransmitters (such as Ritalin, which affects dopamine levels) are short acting. When given to children with ADD, the effects of Ritalin are seen within an hour and the half-life is only four hours (slow release forms are designed for eight hours) and thereafter the child returns to his or her previous behavior. Perhaps the child’s behavior is even a little worse if he or she experiences a rebound effect as the drug wears off. See *The A.D.D. Book* (Sears & Thompson, 1998) for a review of medication effects, a discussion of diet and neurotransmitters, and a chapter on neurofeedback for ADD.

Remember that it is not the electricity from an action potential that is documented when we measure the EEG. The electrical changes of an action potential are very brief (one millisecond) and occur across the cell mem-
brane. As discussed below, the EEG is recording extracellular potentials that have a longer duration (15–200 milliseconds) and slower frequencies (< 50 hertz).

**Brain-Behavior Links**

An excellent book on the topic of how brain function relates to brain structure is *Mapping the Mind* (Carter, 1998), written by a journalist who did an extensive review of research related to brain function. She also consulted with a well-known British neuroscientist, Christopher Frith, to ensure that her simplified explanations were consistent with the complex research findings. It is a popular book that offers a readable account of brain-behavior links without being overly technical. In a similar vein, Strauch (2003) focuses on brain changes in adolescence. If you are interested in the question of how human emotions relate to brain structure and function, consult *A General Theory of Love* (Lewis, Amini, & Lannon, 2000), jointly written by three psychiatrists of different generations. The authors span the evolving viewpoints from psychoanalytic through psychopharmacological and biobehavioral understandings of emotions, and eloquently link traditional psychiatric views of emotional functioning with more recent knowledge gleaned from neuroscience research.

Although we do not know the mechanisms by which neurofeedback works, there is speculation that there are structural changes—perhaps an increase in the number of dendritic connections among neurons, changes at the synapse with respect to the transmission of signals between neurons, or changes in neurotransmitter receptor sites. Neurofeedback is an empirical field where changes can be observed without knowing the underlying causation. This intervention seems to produce changes that last after training is complete if enough training has been done to alter the electrical patterns. The research evidence for this comes from Lubar’s work training both students and patients who exhibited the symptoms of attention-deficit disorder. Patients trained between the ages of eight and twelve who successfully shifted the amount of slow wave activity in their EEG patterns (i.e., lowered the theta/beta ratio) showed a corresponding decrease in their ADD symptoms and improvement continued when telephone follow-up was done ten years later (Lubar, 2003a).
Source of the EEG

How the EEG Is Displayed

The EEG is an alternating voltage and is displayed as a line that rises and falls according to whether the voltage is positive or negative at each moment in time: by convention, a downward deflection represents a positive deviation. The display that you watch on a computer screen or that is drawn on paper is the electroencephalogram. This electrical activity is measured with an instrument called an electroencephalograph. In principle the representation is no different than looking at an ECG showing the heart’s electrical activity, but in practice there are two differences. First, since the heart produces about ten to one hundred times as much electricity as the brain, the amplitudes are higher. Second, the ECG signal is very regular with the same wave forms repeating about once per second, whereas the EEG signal is highly variable depending on what the brain is doing from moment to moment.

The EEG also resembles an SEMG, but the frequencies are usually much faster when you are dealing with muscle contractions (100–200 hertz is a common bandwidth, whereas the EEG is usually recorded at < 50 hertz) so the waveform looks different. The SEMG is a complex signal since muscles produce frequencies between about eight and one thousand hertz; this results in overlap in the frequency ranges between the EEG and SEMG. Muscle contractions can be a major source of artifact in EEG recordings, which will be discussed later in this module.

In Figure 3 you can look at the raw EEG and count about six big waves in the first second. Thus the dominant activity for this sample was 6 cycles per second or 6 hertz. There are also faster waves riding on those slow 6-hertz waves and some even slower waves (below 6 hertz) underlying them, so it is a complex signal. The spectral array at the bottom simplifies the analysis and shows the activity for each frequency in the 2–62 hertz range. The information displayed also includes boxes that provide information about the amplitude (in microvolts) for particular frequency ranges, such as 4–8 hertz. Frequencies and particular mental states associated with different frequency ranges will be discussed in a subsequent section.
Traditionally the EEG was displayed on paper with pens moving up and down to produce the tracings, each line representing the electrical activity from one site where an electrode was placed. When doing neurofeedback (and also in recent times for most hospital applications using the EEG), the display is on a computer screen. Neurologists usually use at least nineteen locations on the scalp to conduct a clinical EEG. Often a 19-lead assessment is done before neurofeedback begins, although it is also possible to do a single-channel quantitative assessment. With a single active lead, the site most often used is the vertex or top of the head (CZ in the International 10–20 Electrode Placement System [see Figure 4]).

The naming of sites according to the International 10–20 System uses a letter to represent the area of the cortex (frontal [F], central [C], parietal [P], temporal [T], occipital [O]) followed by a number (even numbers on the right and odd numbers on the left) or a Z, which represents the mid-line locations. In an update to the nomenclature, the American Clinical Neurophysiology Society has renamed the sites above and behind each ear: T3 is now T7, T5 is now P7, T4 is now T8, and T6 is now P8 (see Fisch, 1999, Appendix 2). Thus the sequence on the left for the lower electrodes is F7–T7–P7.
This system is known as the “10–20 system” because placements are derived by measuring from front to back and then using 10 percent or 20 percent of that distance as the spacing between adjacent electrodes. Bony landmarks on the skull that are easily identified by feel are used as the endpoints. At the front it is called the nasion (feel the ridge at the top of your nose where the skull indents) and at the back of the head it is called the inion (the ridge or bump where the skull indents). A measurement is also made from side to side, using points just in front of the ear canal called the preauricular notch. To feel this notch, open and close your jaw and notice the notch where the jaw is hinged. Usually the measurement from front to back and from side to side is about equal. A common measurement for adults is 36 centimeters.

Amplitude refers to the size of a wave, usually measured from peak to peak (that is, from the top of one wave to the trough of the next) and the unit is microvolts (millionths of a volt). To eliminate the negative values, a mathematical transformation (the Fast Fourier Transformation) is used to describe the waves in terms of sine and cosine values. The resulting spectral array gives values for the power—measured in picowatts—at each fre-
quency. This allows one to view the EEG in the frequency domain for a given time period rather than viewing the spontaneous EEG in the time domain. Mathematically, microvolts can be converted to picowatts by squaring the ratio of the magnitude (average microvolts over time) of two frequency bandwidths. Power ratios that calculate the amount of theta (4–8 hertz activity) divided by the amount of beta activity (13–21 hertz) have been published that discriminate between individuals diagnosed with ADD and non-ADD individuals (Monastra et al., 1999).

**How the EEG Is Measured**

The electroencephalogram is defined as the difference in voltage between two different recording locations plotted over time (Fisch, 1999). You are measuring a potential difference and that requires three electrodes, two active electrodes and one reference electrode. The procedure is noninvasive. You simply monitor electrical activity and do nothing to the client other than give him or her information about his or her neuronal activity.

Before placing the electrode on the scalp or earlobe, the site is cleaned and prepared using a mildly abrasive gel in order to remove skin oils, dead skin cells, or anything else (e.g., hair spray) that could act as an insulator. After cleaning, use a conductive gel between the electrode and the skin. It is imperative that adequate preparation is done and that enough conductive gel is used to obtain low impedance readings between each pair of electrodes. This follows from Ohm’s Law, which states that voltage is the product of current multiplied by resistance (V = I x R). Impedance is defined as resistance to the flow of an alternating current and is measured in ohms. Research criteria are that the impedance readings be below five kohms and within one kohm of each other. The reference electrode is typically placed above the mastoid bone immediately behind the ear or on an earlobe.

If you use a referential (formerly called “monopolar”) placement, you have your active electrode over a site where you expect electrical activity to be generated—that is, on the scalp at one of the sites described above in the 10–20 electrode placement system. The reference electrode is then placed on a site that is relatively inactive with respect to electrical activity, such as an earlobe. In this case the third electrode (the ground) is usually placed on the ear lobe that is not being used as the reference for the active electrode.
When the referential placement is used, any changes in voltage that you observe are mainly due to changes in cortical activity below the active (positive) electrode.

If you use a *sequential* (formerly called “bipolar”) placement, both active electrodes are placed on sites on the scalp where you expect there to be electrical activity. For example, if you want to assess the amount of slow wave (theta) activity in a child who has symptoms of ADD, you use a central, mid-line location. You would place one electrode halfway between CZ and FZ and the other electrode halfway between CZ and PZ. (If you were using a referential placement, you would use CZ referenced to one ear.)

With sequential placement you are not sure which site has the higher amplitude; you just know that there is a difference. If you see a change over time, you cannot be sure whether one site had an increase, the other site had a decrease, or the two sites changed in terms of the waves being more (or less) in phase. Lubar (2003a) has used both sequential and referential placements to obtain good results with clients who have ADD, and advocates using the placement that initially gives the clearest picture of the increased slow wave activity. He has remarked that although the referential placement gives a more straightforward explanation of the change when you get a decrease in theta, the sequential placement may give the brain more ways to learn the task of changing the brain wave pattern.

Other researchers, such as Sterman in his work with those who have seizure disorders (Sterman, 2000a), prefer a referential placement to help better gauge the site where they are having the effect. For increasing sensorimotor rhythm, a pattern that is protective against seizures, Sterman would train some sessions at C3 and some at C4—thus staying across the sensorimotor strip—and train for increased sensorimotor rhythm to decrease the likelihood of unwanted motor activity (i.e., a seizure).

**The Importance of Artifacting**

When doing neurofeedback you want to obtain the most accurate information possible, which means that you must minimize artifact in the EEG. After you collect the EEG data for an assessment, you need to remove artifacts before doing any analysis. During a training session, however, you are feeding back information that is based on the spontaneous EEG. Therefore
you must do what you can to eliminate or reduce sources of electricity that are not EEG. When recording the EEG, an artifact is defined as electrical activity that is not of cortical origin. The most common artifacts that can fool you are thinking that you have beta activity when it is really the result of muscle tension, and interpreting eye blinks and eye movement as if they were delta or theta activity.

Hammond and Gunkelman (2001) give an excellent discussion and more than 75 pages of examples in their text *The Art of Artifacting*. Following is a list of the types of artifacts that they discuss.

- SEMG (muscle) artifacts.
- Movement artifacts (wires moving, body movements).
- Eye blink and eye movement artifacts.
- ECG and pulse artifacts.
- Tongue movement and swallowing artifacts.
- Artifacts associated with drowsiness.
- Sweat or electrodermal artifact.
- Salt bridge between electrodes.
- Breach rhythm due to a skull defect.
- Electrode artifact (e.g., poor impedance).
- Evoked potentials and transients.
- Artifacts from environmental sources (cell phones, lighting, etc.).

Before starting with clients, it is a good idea to catalog their most common artifacts by asking them to blink and roll their eyes so that you can see how those eye movements affect the EEG. Similarly, ask them to clench their jaws—both strongly and lightly—and see how that affects the waveforms. Educating your clients about artifacts will enable you to enlist their help in reducing them, and thus provide a good-quality signal on which to base the feedback.

**How the EEG Is Generated**

The pyramidal cells that produce the electrical potentials that we measure as EEG are aligned in columns at right angles to the surface of the cor-
tex. (This usually means at a 90-degree angle to the scalp, but because of the folds in the surface of the cortex, this assumption is not always true.) One way to produce a positive postsynaptic potential is to have an excitatory stimulus from another neuron’s axon come in near the top of a dendrite (the end nearest the surface of the scalp) and produce changes in the cell membrane that make it more permeable to sodium. Sodium—which has a positive charge—rushes into the cell, thereby leaving the extracellular space with a negative charge. At the other end of the dendrite, a positive charge is created outside the cell because the pyramidal cells behave like little batteries: if one end is negative, the other end is positive. (There are other changes too, such as the permeability to potassium ions, but the main effect is from movement of positively charged sodium.)

If there are enough pyramidal cells in the same vicinity (a macrocolumn of cells) and all are receiving the same type of stimulus at the same level of the dendrite at the same time, then you will measure a potential on the surface of the scalp at that moment in time. Note that four conditions must be met.

- The cells are aligned in the same direction.
- The inputs occur at the same level (near the top for an excitatory stimulus that leaves a negative charge near the scalp).
- The inputs happen at the same time.
- The inputs are the same kind of stimulus (excitatory or inhibitory).

If the excitatory stimulus had come in at the other end of the dendrite, near the cell body rather than near the surface of the scalp, then the negative charge would be at that end and the positive charge would be near the surface of the cortex and you would record a positive potential from the scalp. With inhibitory inputs the effect is to further polarize the cell (make it more negative within) so that it is less likely to reach the threshold for firing an action potential.

What you record is the sum of the electrical activity—the sum of the excitatory and inhibitory postsynaptic potentials—at that particular location on the scalp. Think of it as an algebraic sum: EPSP + IPSP = EEG. Each standard electrode measures the activity below it, picking up electricity from an area of about six square centimeters of scalp.
In neurofeedback there is an initial assessment involving either one location or nineteen locations. The latter is often called a *full-cap assessment* because to locate the sites, a cap with the nineteen electrodes built into it is often used. Some research laboratories now do dense arrays of up to 256 electrodes. When training is done, it is usually at one or two sites.

**Summary: Physiological Basis of the EEG**

The EEG is generated by the synchronous activity of postsynaptic inhibitory and excitatory potentials involving large groups of cortical pyramidal cells. These pyramidal cells’ postsynaptic potentials form an extracellular dipole layer, like a layer of tiny batteries.

The postsynaptic potentials have a long duration (15–200 milliseconds). These potential changes summate (algebraic sum of EPSP + IPSP) and the EEG records the resulting potential (positive or negative) traveling toward the electrode on the surface of the scalp.

Action potentials (electrical activity traveling down the axons of these cortical cells) have a very short duration (one millisecond) and do not significantly contribute to the EEG. The EEG is recorded as changes in voltage over time. The voltage is the potential difference between two electrodes at different locations on the head.

**EEG Waveforms and Their Behavioral Correlates**

**Frequencies, Waveforms, Phase, and Synchrony**

**Cortical loops and EEG frequencies.** Frequency refers to how often a wave occurs in one second—for example, five complete waves in one second is five cycles per second, usually designated as five hertz. Different frequency ranges have traditionally been assigned Greek letter names. From the lowest to highest frequencies, they are *delta*, *theta*, *alpha*, *beta*, and *gamma*. Waves that run at about 8–12 cycles per second (8–12 hertz) with fairly high amplitude and a regular shape stand out clearly and dominate the EEG of an adult with eyes closed. These waves received the name of the first letter in the Greek alphabet (alpha) when Hans Berger first wrote...
about the EEG in humans in the late 1920s. The faster, desynchronized activity above 12 hertz he called beta. These designations are still used today.

There are different generators for different frequencies and this is a complex topic that is still being researched. There can even be different generators for the same frequencies. For example, alpha waves in the occipital region may have a different source than alpha waves in the frontal region. Lubar (1997) has discussed the general principle that the cortex produces frequencies according to the distance between the neurons that are communicating. When communication is between macrocolumns that are local (close together, like roads in the same neighborhood), you have fast frequencies in the gamma range above 30 hertz. If the communication is regional, between macrocolumns that are several centimeters apart (main roads between towns), you have the intermediate frequencies called alpha and beta. If the communications are long distance, between macrocolumns that are global (interstate highways) such as frontal-parietal or frontal-occipital, you have the slow frequencies known as delta and theta.

These three connections can operate spontaneously or be influenced by thalamic pacemakers. Although about 97 percent of what we record as EEG originates in the cortex, it is modulated by the less than 3 percent of the connections that run between the thalamus and the cortex. If there were no thalamo-cortical connections, you would see only delta in the EEG (as Sterman demonstrated in some of his early research with cats). The important thing about thalamo-cortical loops is that “changes in the cortical loops as a result of learning, emotion, motivation, or neurofeedback for that matter, can change the firing rate of thalamic pacemakers and hence change their intrinsic firing pattern” (Lubar, 1997, p. 116).

**Waveforms (morphology).** Morphology, or waveform, refers to the shape of a wave. Some waves are regular and sinusoidal whereas others constantly change their duration and shape. Slower, regular waves (for example, alpha waves) are sometimes described as *synchronous activity*; this signals that the brain is resting or in standby mode (see Figure 5).
A complex is a sequence of two or more waves that is repeated and recurs with a reasonably consistent shape (Fisch, 1999). The kind of complex that might be seen, and that would indicate the need for an immediate referral to a neurologist, would be spike and wave complexes that are indicative of seizure activity (see Figure 6).

Figure 5. Alpha activity (eyes open) in a 20-year-old with ADD and Asperger’s syndrome.

Figure 6. Example of a spike and wave complex recorded at CZ from an 8-year-old girl who had been referred as having ADHD, Inattentive Type.
Sinusoidal waves such as alpha or spindle waves (e.g., sleep spindles or the similar looking sensorimotor rhythm waves) are described as being rhythmic. Because the skull attenuates fast frequencies more than slower frequencies, it can be difficult—using electrodes placed only on the scalp—to see the spindle-like pattern as the waves increase in amplitude and then decrease. The original work by Sterman on sensorimotor rhythm, a term he coined for low beta frequencies when they occur across the sensorimotor strip, was done in cats using electrodes implanted in the brain. The amplitude is much higher (2–58 times higher according to Fisch [1999]) with recordings from the cortex than those from scalp electrodes.

*Phase* simply refers to whether the troughs and peaks of the waves in one area are occurring at the same time as the troughs and peaks of waves in another area of the cortex. It thus relates to the timing and polarity of waves. Waves that are *in phase* have troughs and peaks that occur at the same time. If they do not coincide, they are *out of phase.*

**Dominant Frequency and Age**

As mentioned above, alpha is the dominant frequency in adults in the occipital region with eyes closed measurements of the EEG. There is a correlation between a person’s dominant alpha frequency and his or her intellectual ability, with higher peak alpha being associated with better cognitive performance. Thus someone whose peak alpha is 10.5 hertz is probably brighter than someone whose peak alpha is 9 hertz. In the elderly the peak alpha frequency tends to decline, although that observation may result from sampling bias due to measurements of people who were in retirement homes.

The dominant frequency found in eyes closed recordings has a developmental aspect. In infants slow delta activity dominates. In young children through age five, theta is dominant. The peak frequency continues to move higher: by adolescence alpha is the dominant rhythm, and the peak alpha frequency increases a bit through the second decade of life. The term “pediatric alpha” is sometimes used when waveforms with the morphology and spatial distribution of alpha are found at frequencies below the usual 8–12 hertz frequency range.

The developmental aspects of the EEG must be kept in mind when working with different age groups. What would be considered excess theta in a 12-year-old client with ADD would be quite normal in a 4-year-old.
The theta to beta ratios published for children with ADD reflect these developmental changes (Monastra et al., 1999) and show that the relative amounts of slow and fast wave activity measured at the vertex (CZ) in adults with ADD approximate the ratios of young children without ADD. These ratios have nothing to do with intelligence—there are many people with ADD who have IQs in the gifted range—but a high ratio is associated with a short attention span for boring or repetitive activities.

**Correlation of Frequency Ranges and Mental States**

**Historical context.** In 1929 Hans Berger, a German physician, became the first researcher to publish on the human EEG. EEG activity in animals (such as rabbits) had been researched and written about by Richard Caton in England in 1875. Berger’s observations that slower waves (alpha) were associated with a resting state—or standby mode as we think of it today—and that faster waves (beta) were associated with cognitive processing have held true. A milestone in terms of relating EEG patterns to mental states came with experiments reported by Joe Kamiya in the 1960s (Kamiya, 1962, 1968). Kamiya observed in his first experiment in 1956 that people could recognize when they were producing alpha activity, even though they could not say how they produced that mental state.

Approaching the issue from a different perspective, sleep researcher Barry Sterman stumbled on the discovery of sensorimotor rhythm when he was investigating reciprocal inhibition as a process associated with animals going to sleep when they were in a conflict situation and could not make a decision. After establishing that particular brain wave frequencies could be operantly conditioned (see Wyrwicka & Sterman, 1968), Sterman went on to demonstrate that by increasing these same frequencies, animals increased their resistance to seizures. This led to clinical applications in humans with epilepsy.

Joel Lubar extended this work to children with attention problems and hyperactivity, publishing the first paper on the successful treatment of a child with ADD (see Lubar & Shouse, 1976). Lubar also spearheaded the clinical application of low resolution electromagnetic tomography, a mathematical technique for solving the question of source localization—that is, inferring from activity measured from the scalp what is happening in deeper structures in the cortex (Pascual-Marqui, Michel, & Lehmann, 1994).
**Frequency bandwidths.** A bandwidth is simply a range of frequencies. Common bandwidths, such as 4–8 hertz or 8–12 hertz, are given names (theta and alpha, respectively). The four bandwidths commonly used by neurologists and electroencephalographers are:

- Delta frequency bandwidth: below 4 hertz.
- Theta frequency bandwidth: 4–8 hertz.
- Alpha frequency bandwidth: 8–12 hertz.
- Beta frequency bandwidth: 13 hertz and above.

Note that these bandwidths are somewhat arbitrary and overlap. Alpha, for example, is sometimes regarded as being 8–13 hertz. Note also that the field of neurofeedback refers to the lower beta frequencies when they are measured across the sensorimotor cortex as the *sensorimotor rhythm* (SMR) rather than using a Greek letter designation. Sometimes in children it is 12–15 hertz that is uptrained for sensorimotor rhythm; in adults it would usually be 13–15 hertz. This is consistent with the frequency ranges moving higher with age for the same type of waveform.

The name “sensorimotor rhythm” was chosen by Sterman because he observed that the spindle-like rhythm occurring in the lowest beta frequencies, which resembled sleep spindles, was associated with a particular kind of behavior. In Sterman’s early work with cats, the SMR was produced by an alert cat that was waiting to perform an action that would produce food. The cats had to inhibit a bar press until a light signaled that the bar press would produce a mixture of milk and chicken broth. In later experiments they were rewarded for merely producing the SMR without a bar press. The cats learned to produce these rhythms for a food reward.

Humans learn operant conditioning of brain waves with information about their success serving as the reward. The production of SMR reflects inhibitory processes in the brain and is associated with a decrease in sensory input as well as a decrease in motor output. Increasing SMR is thus helpful in conditions such as ADD since you want the child to be less distracted (pay less attention to irrelevant sensory input) and less hyperactive. It is also helpful in seizure disorders, a condition that exemplifies a particular kind of unwanted motor activity.
Frequencies above 30 hertz are referred to as gamma. Frequencies in the 40-hertz range are called Sheer Rhythm, named after Daniel Sheer who did research on these frequencies in the 1970s (Sheer, 1975). Increasing 40-hertz activity appears to benefit children with learning difficulties. It is considered a binding rhythm that helps consolidate brain functions. Note that is difficult to work with 40-hertz activity because the amplitude of the waves is so small and the effects of muscle artifact can be great.

The spectral array. A spectral array, or EEG spectrum, is a way of showing EEG data. Rather than plotting amplitude (measured in microvolts) against time (measured in seconds), as in the case of the spontaneous EEG, a power spectrum takes the data from a particular period of time (e.g., one second) and represents it as a plot of power (measured in picowatts) across the different frequencies. The Fast Fourier Transformation is one mathematical method used to translate the data from the time domain to the frequency domain. When you square the Fourier coefficients, you create a power spectrum, a common way of looking at data. The relationship between amplitude and magnitude when dealing with ratios, such as the theta/beta ratio, is such that you can square the microvolt value to determine the picowatt value.

A power spectrum allows you to quickly see the amount of activity at each frequency, and watch how the relative amounts of different frequencies change from moment to moment depending on the person’s mental state. When the brain is resting, for example, the slow frequencies increase in the occipital region when the eyes are closed; no visual processing need be done and the waves appear synchronous. When the brain becomes engaged in a task (for example, opening the eyes so that the occipital region becomes activated to process the visual information), there is a reduction in that activity and an increase in faster waves that are desynchronized. This is referred to as event-related synchronization and event-related desynchronization.

Remember that there is no substitute for looking at the raw EEG. Brain maps and spectral arrays are excellent ways to represent data using quantitative EEG techniques, but the mathematical transformations lose information about waveforms. If you look only at the power spectrum of someone
with absence seizures (petit mal epilepsy), you would see very high power at three hertz; it is only by looking at the raw EEG, however, that one recognizes this information as intermittent spike and wave activity. You might otherwise think that there was simply high delta activity or suspect eye blink artifact or movement of electrodes, both of which may also mimic delta at that frequency.

**How frequencies relate to various mental states.** Delta activity (0.5–3 hertz) dominates the EEG during Stage IV sleep and is also found in the awake EEG in conjunction with learning disabilities and brain injury. Eye movement mimics delta activity so careful artifacting must be done before you conclude that there is real delta activity present. It is the dominant frequency in infants.

Theta waves (4–7 or 4–8 hertz) are seen in drowsy states where creative thoughts may also occur. In addition, the theta band seems to be related to the encoding and retrieval processes of a complex working memory system (Klimesch et al., 1997). Theta is the normal dominant activity in young children.

Low alpha (8–10 hertz) is found in dissociative states, some kinds of meditation, and tuning out from external stimuli (e.g., daydreaming). A person’s dominant posterior rhythm, measured in the eyes closed condition in the occipital region, moves into the alpha range in childhood around age ten, and moves slightly higher with age until about age twenty.

High alpha (11–12 or 11–13 hertz) can be found associated with creative reflection and long-term memory as well as relaxed, calm states of optimal performance. When you perform a well-practiced maneuver that is overtrained and automatic, such as a professional golfer at the moment when he or she hits a golfball off a tee, you are on automatic pilot in the high-alpha range. The amateur who is still in beta and thinking about whether his or her knee bend is correct will not perform as smoothly and effectively.

SMR frequencies (13–15 hertz) are found when a person is calm (not fidgeting or moving) and alert to the outside world— for example, in reflection before action.
Beta waves (16–20 hertz) are associated with singular focus, external orientation, and problem solving, while higher beta frequencies may be found in association with anxiety and rumination (bursts of activity in the 20–32 hertz range).

Slow waves refer to the activity below 13 hertz (delta, theta, and alpha) and fast waves are at and above 13 hertz. Flexible people can shift quickly between frequencies according to task demands. The comparison has been made to gears in a car: you need them all but they are used appropriately at different times, and you want to be able to shift smoothly.

**Drug Effects Related to EEG Measures**

For a detailed account of medication effects, the reader is referred to Bauer and Bauer (1999). Suffice it to say that medications may alter the EEG and not always in the way that one would expect. Benzodiazepines, barbiturates, and tranquilizers (for example) usually increase beta activity—particularly beta over 20 hertz—and there may also be a slight decrease in alpha; they also increase sleep spindles (Fisch, 1999, p. 417). Neuroleptics, on the other hand, are found to generally increase alpha power and reduce beta power (Hughes & John, 1999).

When dealing with adolescents and young adults, one must be vigilant about asking about marijuana use if there is excess alpha activity present in central and frontal regions in the eyes open state. The increased alpha found in association with smoking marijuana may last for a day or two. The person is less aware of the world around him or her and more detached from everyday concerns, as reflected in the brain being more in a resting than an activated state. (Alpha is an inverse indicator of activation; that is, more alpha means less activation.)

Antidepressants will usually decrease alpha activity and lithium—which is used for bipolar disorder—will produce a slowing of alpha, theta, and delta. Selective serotonin reuptake inhibitors increase beta activity and may decrease alpha. Phenothiazines, haloperidol, and rauwolfia derivatives may slow alpha and produce asynchronous slow waves even at nontoxic doses. There may also be increased synchrony. Alcoholics often show a low voltage, fast EEG with increased beta and decreased theta and alpha activity. Alpha increases with ingestion of alcohol.
Stimulants, such as methylphenidate (Ritalin) and amphetamines, may produce some increase in beta and possibly a decrease in theta activity. However, one usually sees minimal (if any) effect in the brain wave patterns of children with ADD when they are taking such medication. There is no contraindication to training a child who is taking these medications, but one should instruct parents to watch for changes in their child’s behavior and in his or her response to the medication: children may start to look like they are on too high a dose as self-regulation of brain waves is learned during the course of training. Most children will reduce, or even eliminate, the need for stimulant medication during the course of 40–60 sessions of neurofeedback training. In one case series (Thompson & Thompson, 1998), 80 percent of the children taking Ritalin when they began a neurofeedback intervention that decreased theta and increased SMR were drug free after forty sessions of training.

Cocaine is a fast-acting and therefore addictive stimulant. Both cocaine and LSD increase fast wave activity; LSD, however, will decrease alpha whereas cocaine tends to increase alpha. Heroin and morphine will increase low alpha initially but this is followed by a decrease in alpha and an increase in theta and delta.

Caffeine and nicotine are commonly used drugs that will suppress alpha and theta. Morning coffee is a way to wake up your brain. Withdrawal from these substances may result in an increase in alpha and theta frontally. One reason given by people who are reluctant to quit smoking is that they do not wish to lose the mental sharpness that smoking a cigarette provides.

Withdrawal from medications may have an effect on the EEG. This is especially true if the withdrawal is sudden and if the patient has taken that medication for an extended period. Medications that take some time to elicit their effect (such as antidepressants) must be withdrawn very slowly or you risk a discontinuation syndrome. Short-acting medications such as methylphenidate, which shows its effects in about one hour and has a half-life of approximately four hours, have a faster onset and withdrawal is usually less problematic.

Knowledge of the medications that your client is taking will influence your interpretation of what you see in the EEG. If you see high beta activity (above 20 hertz), for example, it may be a medication effect rather than...
an indicator that the person tends toward being anxious or ruminating. If the client is taking antibiotics or an antihistamine for allergies, you can expect his or her slow wave activity (theta) to be higher: this may be the effect of the medication or simply the body’s response to the bacterial infection or the allergen for which the medication was prescribed. One requires more sleep when fighting an infection and increased theta is associated with sleepiness. The most important thing is that in EEG work, the practitioner must ask about medication and drug use and consider the possible drug effects when analyzing the EEG and doing training.

**Clinical Uses and Efficacy of EEG Biofeedback**

The AAPB/International Society for Neuronal Regulation (ISNR) joint guidelines for evaluating research on the efficacy of neurofeedback interventions (La Vaque et al., 2002) distinguish between validated applications—i.e., those with some support—and those that are experimental. Using these criteria, a new AAPB publication (Yucha & Gilbert, 2004) places seizure disorders and attention-deficit disorder in the first group. All other applications should be treated as experimental: even though there is some support from clinical outcome studies, there is not yet enough controlled research. Treatment of mood disorders, treatment of alcoholism and addictions, helping those with closed head injuries/traumatic brain injury, treatment of anxiety, and work with children who have learning disabilities are in the latter group.

Applications that look promising due to emerging clinical reports of improvement—but that are not yet verified and should be considered experimental—include Tourette’s syndrome and other movement disorders (Parkinson’s disease and dystonia), Asperger’s syndrome and autistic spectrum disorders, “brain brightening” in the elderly, and obsessive-compulsive disorder. When anxiety is part of the symptom picture or pain is a major symptom (headaches, fibromyalgia), it makes sense to combine general biofeedback with neurofeedback (Thompson & Thompson, 2003).

The reader is encouraged to consult the ISNR website for a bibliography of published research, organized by disorders treated and compiled by Corey Hammond. The list of conditions for which there is research (including controlled studies published in peer-reviewed journals) is limited; in
addition, the field has been criticized for lack of double-blind, placebo-controlled studies. Sham control groups, however, are not easily designed in research on EEG biofeedback—not only because of questions concerning what constitutes an appropriate placebo, but also due to ethical concerns associated with giving “phony feedback” for as many as forty sessions. Both the subject and experimenter are readily aware of whether feedback reflects the subject’s mental and physiological states and thus it is not possible to maintain double-blind conditions. Awareness of SEMG artifact, for example, is essential for quality EEG feedback and subjects can easily check if moving their jaw stops feedback. If there is no change with jaw muscle contraction, they know that they are not securing accurate feedback.

Rather than employing experimental designs used in drug studies, it may be more appropriate to use an analogy to exercise. Just as physical exercise can be beneficial in cardiac rehabilitation or the treatment of obesity using outcome studies and comparison groups, exercising the brain using EEG biofeedback can be beneficial in behavioral and cognitive performance. Another approach is to use a comparison group that receives an intervention that has already been established as efficacious, such as a drug-treated group versus a neurofeedback-treated group for ADD research. There should be random assignment of subjects to the neurofeedback and comparison groups. This approach is endorsed by the World Medical Association as being preferable to placebo-controlled designs when there is an established treatment available.

**Attention-Deficit Hyperactivity Disorder**

Attention-deficit hyperactivity disorder (ADHD) is the most widely studied and most frequently diagnosed neurobehavioral disorder in children. There is an EEG signature of excess slow wave activity (theta in the 4–8 hertz range) in the majority of people who qualify for a diagnosis. Helping people manage the symptoms of ADHD is a widespread application of neurofeedback because the number of people who could benefit is huge: at least 5 percent of the school-age population is affected and about 3 percent of American children are taking stimulant medication to treat this condition.

The fourth edition of the American Psychiatric Association’s *Diagnostic and Statistical Manual of Mental Disorders*, published in 1994, uses the
term “attention-deficit hyperactivity disorder” with three subtypes: Inattentive Type, Hyperactive-Impulsive Type, and Combined Type. The core symptoms are problems with sustained attention for boring or slow-paced tasks; distractibility; impulsivity; poor organization/losing things; and (in some but not all cases) restless, fidgety, or hyperactive behavior. The symptoms should have been evident before age seven, must occur in more than one setting, and must impair functioning to a clinically significant degree. They continue into adulthood in the majority of cases, although the symptom picture may change over time—for example, a decrease in hyperactivity with the onset of puberty.

People with ADHD typically have excellent attention for things that interest them, which may include watching television, playing video games, participating in sports, or doing creative activities. Thom Hartmann’s (2002) analogy of the “Hunter Mind” is an apt comparison that also recognizes some positive features of this style. Nevertheless, the symptoms usually lead to academic underachievement and difficulties in social relationships affecting home, school, and work environments.

Applications of EEG biofeedback for decreasing hyperactivity and symptoms of attention-deficit disorder have been in use for more than a quarter-century. This approach was originally tried because it had been observed that patients with epilepsy who were successfully treated using operant conditioning of brain waves not only showed a reduction in the frequency and severity of seizure activity, but also became calmer. (Hyperactivity and restlessness are frequently found in association with epilepsy.) There are over fifty publications, including case series and controlled studies, so this application meets the criteria of an intervention that is validated as being a useful part of a multimodal approach to managing the symptoms of ADHD.

Other things that should be considered when treating ADHD include diet, sleep, and exercise, all of which have an influence on behavior and brain function. Educational interventions and good parental management techniques are also important. For a discussion of and suggestions in these areas, consult *The A.D.D. Book: New Understandings, New Approaches to Parenting Your Child* (Sears & Thompson, 1998), the first book written for parents that included a chapter on neurofeedback.
The rationale for using EEG biofeedback rests not only on the observation that hyperactivity is reduced in conjunction with increasing SMR activity (12–15 hertz), but also on EEG studies that show excessive slow wave activity in central and frontal regions in individuals with ADHD (Jantzen et al., 1995; Lubar, 2003b). These EEG findings are cross-validated by findings using other imaging techniques, such as decreased glucose metabolism (PET scans) in frontal regions in those with ADHD (Zametkin et al., 1990) and decreased blood flow (SPECT scans) in frontal regions in boys with ADHD when doing a math task (Amen, Carmichael, & Thisted, 1997).

The EEG profile may be obtained using a single channel with placement at CZ, a reference electrode on one ear and the ground on the other ear. When a sequential placement is used, Lubar recommends using FZ and CZ for adults and sites halfway between FZ and CZ (FCZ) and between CZ and PZ (CPZ) for children. Multiple channels, as with a 19-lead full-cap assessment, may also give a more complete idea of activation.

The most frequently used training approaches involve rewarding the client for reducing the amount of slow wave activity (such as 4–8 hertz) while encouraging faster wave activity (12–15 hertz) in those who are hyperactive or 15–18 hertz in those who are inattentive without restlessness and fidgeting. The frequency ranges are based on the person’s EEG findings; for example, 6–9 hertz may be the range for slower frequencies that are excessive or 13–15 hertz may be the range for faster waves. You customize for each client. Common sites for training are CZ or C4, the latter frequently used when the hyperactive component is present.

A well-established finding, and the most common pattern found, is increased slow wave activity (4–8 hertz) in frontal and sensorimotor cortex. This excess theta is seen at CZ in children. In adults you may find the pattern more frontally at FZ. A review of quantitative EEG patterns is found in Sterman’s (2000b) review article.

The field of neurofeedback applications for ADHD has benefited from the publication of norms for theta to beta power ratios at different ages (Monastra et al., 1999; Monastra, Lubar, & Linden, 2001). These studies indicate that you can identify ADHD with 93 percent accuracy. Recently published research shows that improvements in ADHD symptoms achieved
with neurofeedback plus medication continued after both training and medication were withdrawn, whereas improvements with stimulant medication alone were not sustained when the drug was withdrawn (Monastra, Monastra, & George, 2002).

**Seizure Disorders**

The application of EEG biofeedback to seizure disorders generally relies on reducing slow wave frequencies while increasing the SMR. Following is an overview of the research on seizure disorder treatments and their rationale, which was reviewed in detail by Sterman (2000a).

Some cells fire in an inappropriate fashion (epileptiform activity, for example, spike and wave at three hertz.). If these cells recruit nearby cells to also fire at that rate, there is kindling of a seizure that can lead to a full, generalized seizure where the whole brain begins to fire at the same rhythm. The approach using neurofeedback is to discourage the epileptiform activity by inhibiting those slow frequencies while, at the same time, encouraging frequencies that are protective against seizures (such as sensorimotor rhythm). Placement is across the sensorimotor strip alternating between sessions at C3 and C4. With newer equipment one could do two-channel training at both sites simultaneously. (Note that this is not the same as a sequential placement across the two hemispheres using a single channel.) If there is a focus to the seizure, training can also be done at that site to discourage the slow wave activity. Sterman explains that it is a two-pronged attack: you discourage the kindling (inhibit epileptiform slow waves) and you also build a firewall (encourage SMR).

Sterman (2000a) reviews 18 different studies on the clinical effects of sensorimotor EEG operant conditioning treatment of epilepsy that were published in peer-reviewed journals between 1972 and 1996. Patients showed clinical improvement in 82 percent of the reported cases (N = 174) and EEG improvement in 66 percent of them. Sterman concludes that the research “documents both immediate and sustained functional changes that are consistent with the elevation of seizure thresholds” (p. 53). In the majority of cases, both the frequency and severity of seizures was reduced, and in some cases patients became seizure free.
Anxiety and Affective Disorders

Herbert Benson, author of the well-known book The Relaxation Response (1975), has expressed the view that understanding alpha is key to understanding the relaxation response because—regardless of the technique used to elicit the response—it is associated with the production of alpha. Increased alpha production is associated not only with meditation but also with rapid learning, stress reduction, lessened perception of pain, enhanced creativity, personal growth, and mystical experiences.

Yet “increased alpha” is a very broad phrase. It is important to understand where the increased alpha is and what alpha represents. Alpha is the normal dominant frequency in adults measured with eyes closed. It is found primarily at the back of the head (occipital region) when the eyes are closed and is a sign that the occipital region is resting or, at least, in standby mode. Since visual processing is done in the occipital region, it can rest when there is no visual input; hence alpha is produced.

This same concept of alpha as an area of the brain that is not doing active processing is also important in helping to manage depression. The patterns found in depression reported by Richard Davidson (2004) indicate that the left frontal cortex is underactive in those who are depressed. Left frontal region activation is associated with positive thoughts and approach behavior, whereas right frontal activation is associated with a focus on negative thoughts and avoidance behavior. Neurofeedback interventions thus have a goal of activating the left frontal regions. (Of course one should measure differences at F3 and F4 to ensure that your client does show this pattern before you proceed.)

A novel approach is the alpha asymmetry protocol developed by Rosenfeld (2000). He used a formula that measures alpha activity at F3 and F4—both referenced to CZ—and compares which side has more activation. (Remember that alpha is used as an inverse indicator of activation: the more alpha, the less activation.) The formula is R – L / R + L; when the outcome is greater than one, the person is presumed to be less depressed.

Substance Abuse

There has been considerable research on alcohol dependency and substance abuse, starting with work undertaken at the Veterans Administration hospital in Topeka in 1973 (Goslinga, 1975). This alpha-theta brain wave
biofeedback grew out of Elmer Green’s observations at the Meninger Clinic on patterns seen in practiced meditators. As summarized by Trudeau (2000), “Brain wave biofeedback was first conceptualized as a way to augment insight and motivation through guided imagery in alcoholics, and the focus of initial implementation was on achieving very relaxed hypnagogic states using occipital alpha wave feedback” (p. 13).

Peniston’s work with inpatients at the Fort Lyons (New Jersey) VA was the first randomized and controlled study of alcoholics, and subsequent alpha-theta work has often been referred to as the Peniston Protocol (see Peniston & Kulkosky, 1990). The technique employed by Peniston involved initial sessions in which the subjects were taught hand warming for relaxation (five sessions involving autogenic phrases and skin temperature biofeedback) and also imagery exercises (for example, imagining themselves in situations that were happy and that did not involve drinking) prior to initiating EEG biofeedback.

The EEG work involved placement over the occipital region at O1 and rewarding slow wave activity in the theta range when that activity exceeded the alpha activity. The work is done eyes closed in a relaxed condition and an induction script is used to help the patient sink into reverie. Alpha is the usual dominant activity with eyes closed. When theta exceeds alpha, the person is moving toward drowsiness and entering a hypnagogic state in which he or she is highly suggestible.

The 30-minute sessions of eyes closed EEG biofeedback were followed by a therapy session in which the therapist explored the imagery and any abreacts that were experienced. There were 15 sessions of alpha-theta biofeedback. Peniston and Kulkosky reported reductions on the Beck Depression Inventory among the experimental group; in addition, 13-month follow-up data showed significant protection against relapse and changes in personality variables in the treated group compared to controls who received traditional treatment.

Successful work has also been done with patients whose substance abuse involved stimulant drugs. Scott and Kaiser (1998) started with 10–20 SMR-beta (12–18 hertz) neurofeedback sessions and then did 30 alpha-theta sessions during residential treatment with 48 experimental subjects (43 control subjects). Experimental subjects improved on attention and per-
sonality measures and had lower relapse rates, and there was better treatment retention (fewer dropouts).

It remains an open question whether the therapeutic benefits of alpha-theta training are specific to the addition of brain wave training or are mainly related to the learning of relaxation. For an extensive discussion of research performed in the 1990s and suggestions for further areas of investigation, see David Trudeau’s (2000) review in an issue of the journal Clinical Electroencephalography that was devoted exclusively to reviewing applications of EEG biofeedback.

Note that alpha-theta therapy is designed to be used by experienced psychotherapists since the patient is highly suggestible and may experience an abreaction. The therapist must be trained to help the patient deal with the emotional material that emerges while in the hypnagogic state. This is quite different from the eyes open, improved attention (increase SMR-beta), educational approach employed with conditions such as ADHD. The EEG biofeedback is adjunctive to the other therapeutic techniques employed and does not stand alone.

Mild Closed Head Injuries

There is a substantial literature delineating the functions of various parts of the brain, much of it based on the observed changes in behavior that accompany brain trauma of various kinds (stroke, head injury, brain surgery for tumors or epilepsy, etc.). To effectively utilize EEG for looking at brain changes after head injuries, an understanding of what happens when there is an impact is critical.

In addition to causing local damage in the area of the blow, an impact may also result in damage directly opposite to that site as the brain is shaken and hits the skull on the side opposite the blow (coup–contra-coup damage). There is also breakage along axonal connections because, when there is a blow to the head, the gray and white matter will move at different speeds due to their different densities. (White matter, having more fat due to myelination, will be less dense than gray matter.) The resultant sheer forces lead to diffuse axonal injury. This type of injury can be detected in the EEG but may not be documented by such brain imaging techniques as magnetic resonance imaging (MRI). The EEG also has better temporal res-
olution than MRI. (On the other hand, MRI and other imaging techniques have better spatial resolution and can look at deeper structures.) After head injuries there is more delta activity in the EEG.

Although there are encouraging clinical case reports, the literature on successful treatment of head injury using neurofeedback is still sparse. Interventions are guided by findings on the quantitative EEG undertaken with a 19-channel assessment. Various databases can be used for the analysis. Robert Thatcher and his associates (1989) developed a Traumatic Brain Injury Score based on correlations between EEG and MRI data from Veterans Administration hospitals across the United States. Thatcher (2000) also wrote a review article for the special issue of the journal *Clinical Electroencephalography*.

**Optimal Performance**

This is the newest frontier of EEG biofeedback training and the one that may eventually have the widest application. Athletes, students, executives, and performers may benefit but the training has to be tailored to individual needs. The interventions could be considered educational rather than therapeutic, although a professional such as a psychologist may do the initial evaluation. Training that combines neurofeedback with peripheral biofeedback usually aims to produce calm and relaxed yet alert and focused concentration. A diverse population may benefit from this type of work since difficulties with attention span, concentration, or being a bit impulsive interferes with students using their full intellectual potential or with executives and athletes reaching their top functioning.

For students the training would be similar to what is done for ADHD. Many of the best candidates would be those characterized by Thom Hartmann’s (2002) description of the “Hunter Mind.” They can hyperfocus when there is something of interest that they want to pursue but have difficulty with time management, organization, and concentration on things that are boring or slow paced.

Sports require both intense concentration and an ability to shift mental states quickly. Golfers, for example, must analyze the shot that they need to make by taking a myriad of factors into consideration (wind, lie of the ball, distance to the green or the hole, etc.). This requires beta activity, but they
must shift into alpha to release their shot effortlessly. Training can help an athlete find the zone where performance in his or her sport seems effortless and automatic. In line with this approach, an article in a recent edition of Reader’s Digest reported on Wesley Sime’s use of neurofeedback with athletes, especially mentioning improvement in golfers (Johnson, 2004).

Executives are good candidates because they often work under intense pressure and tight deadlines and need to be able to handle stress and work efficiently. This is achievable through a combination of general biofeedback and neurofeedback; for example, breathing techniques quickly produce a calm state. It is a great asset to be able to choose your mental state: calm and reflective, or energetic and enthusiastic, depending on the kind of interaction that you want to have.

Controlled research done by Gruzelier’s group (see Gruzelier, 2003) at the Imperial College in London has shown the specificity of training different frequencies. In one study, enhanced fast wave activity correlated with improved memory in normal subjects (medical students). In a series of controlled studies conducted over the course of two years, it was shown that increasing slow wave activity enhanced musical performance (Egner & Gruzelier, 2003). The latter work involved rewarding the subjects, who were students at the Royal College of Music, with a pleasant tone when theta activity exceeded alpha activity (P1 was the site used).

The subjects were told only that the training was designed to produce deep relaxation. There were significant improvements in musical interpretation in those subjects receiving the alpha-theta training in contrast to no change in groups receiving beta training, mental strategies, exercise, or the “Alexander technique” (a type of body work frequently done with musicians).

Gruzelier’s research has received a great deal of media attention in England and is raising awareness about the possibilities of neurofeedback for nonclinical populations. In a similar vein, Simon Hanselmayr (2003)—a graduate student in Wolfgang Klimesch’s neuroscience laboratories in Salzburg, Austria—reported on enhanced cognitive functioning in students on a mental rotation task after neurofeedback training. In that case the training was designed to increase individual alpha power.
It is likely that the public’s demand for brain wave training for peak performance will increase as a result of such exposure. Remember that optimal performance is considered an experimental application of neurofeedback since it does not yet have sufficient controlled research to be considered an established application.

**Conclusion**

The existing information on various applications of EEG biofeedback, used alone and in combination with other interventions, suggests that it is a robust field that is reaching a critical mass in terms of consumer awareness and supporting research. The onus is on practitioners to do the training well so that the field will continue to grow, thereby improving the quality of life for increasing numbers of people.
References


Continuing Education Appendix

A. Continuing Education Instructions

B. Learning Objectives
A. Continuing Education Instructions

Some readers have purchased this module to obtain continuing education credit for psychologists or for BCIA recertification. If you are interested in receiving continuing education credit, please read the following information and instructions.

Continuing Education for Psychologists
AAPB is approved by the American Psychological Association to offer continuing education for psychologists. AAPB maintains responsibility for the program. Credit is provided for the term of the program; i.e., one hour of independent study provides one hour of credit. This module offers four hours of credit.

Biofeedback Certification Institute of America
This independent study module is accredited by BCIA to provide four hours of Category A continuing education. If you would like more information about continuing education credit through BCIA, please see its website at www.bcia.org.

Other Continuing Education Credit
Please check with your certification/licensure provider to determine specific requirements for CE credit for nursing, physician, physical and/or occupational therapy, social workers and all other CE requirements.
Module 6: Electroencephalographic Applications

B. Learning Objectives

This module is designed to help you:
1. Explain the scientific underpinnings of EEG biofeedback (neurofeedback), including the source of the electrical activity and the identification of the neuroanatomical structures that are important in understanding thalamo-cortical dynamics.
2. List the EEG frequency ranges (in hertz), using Greek letter designations where applicable, and identify the associated mental states and/or behavior that corresponds to activity that occurs in those frequency ranges.
3. Outline the effects of various classes of medications on the EEG.
4. Identify the conditions that may benefit from EEG biofeedback intervention and distinguish between those that are validated by sufficient controlled research published in peer-reviewed journals, and those that are still considered experimental.