Paul G. Swingle

Adding Neurotherapy to Your Practice

Clinician's Guide to the ClinicalQ, Neurofeedback, and Braindriving

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Overview

The purpose of this book is to encourage clinicians from all licensed healthcare disciplines to consider adding basic neurotherapeutic assessment and treatment to their practices. The data are compelling, indicating that even very basic neurotherapy can markedly enhance the efficacy of most all therapeutic methods and metaphors. This book is structured to help licensed professionals learn the basic methods and principles to enable a disciplined introduction of these most efficacious therapies into their patient care modalities. The book is also focused on helping licensed professionals from falling victim to the one-size-fits-all franchise and franchise-like operations.

Neurotherapy is a data-driven methodology that is straightforward, logical, and validated by compelling research from many quarters. The procedures are based on the fact of brain plasticity, a concept that physicians and psychologists did not fully comprehend even a few short years ago. What we were taught about the brain's potential for recovery in medical and graduate schools, when I took my training, is simply wrong. Hence, many of our treatment concepts are rooted in the belief that the brain has limited capacity for functional change.

The paradigm shift implicated in the concept of neurotherapy is firmly grounded in the recent research on the plasticity of the brain. The rationale is direct and empirical: measure the functional anomalies in the brain so that one knows what symptoms the client/patient is likely to manifest. Then, treat those symptoms by normalizing the anomalous brain activity.

The procedures described in this book are quite specific and limited. Neurotherapy is a broad field with applications to many disorders. The more specialized applications are not addressed in detail, other than identifying circumstances in which one may want to refer the patient to a clinician specializing in neurotherapy.

This guide is based on a single-channel clinical grade EEG. Additional channels may be useful but are not necessary for the ClinicalQ or for the treatment that follows from the ClinicalQ analysis. Some EEG feedback systems do a very bad job of measuring the higher-frequency brain waves and others have very cumbersome software.

To proceed with introducing neurotherapy into your practice, as I am advocating in this book, you will need some basic, clinical grade equipment and the necessary

basic training on the use of the equipment. My strong recommendation is that you purchase your equipment from a supplier or manufacturer that offers the basic hands-on training. Many manufacturers and suppliers have developed "turn-key" systems for the procedures presented in this book. You can contact the Biofeedback Certification International Alliance (BCIA) for a list of manufacturers and suppliers and those that offer approved hands-on training. Distance training over the net is available, but many prefer hands-on training specifically with the equipment you intend to use. Start small and work up, not the other way around. Be sure to query the supplier on the weaknesses of the system relative to the ClinicalQ requirements that are described in this book. Specifically, verify that the system measures brain wave frequencies, reliably, up to 40 Hz.

This book is divided into several sections. After a general introduction to neurotherapy in Chap. 1, Chap. 2 describes the ClinicalQ in detail. In this chapter, the conditions associated with the various brain wave patterns are presented, as well as the statistical data on the research validating the procedure. A detailed description of the diagnostic application of the ClinicalQ is emphasized in this chapter as well. Chapter 3 focuses on some of the conditions where traditional medicine and psychology have not done well following a "try this" approach to treating the labeled condition as opposed to the putative cause(s) of the client's complaints. Chapter 4 reviews the basics of neurofeedback, the "backbone" of neurotherapy. Chapter 5 focuses on methods for potentiating brain wave changes. This chapter includes the research data on identifying unconditioned stimuli for braindriving procedures, discussed in Chap. 6, as well as for procedures used for home treatment. Chapter 6 focuses on the more therapeutically aggressive braindriving techniques that use classical conditioning methods for changing brain wave activity. Chapter 7 gives a very abbreviated review of biofeedback for the peripheral systems such as thermal, muscle, and heart rate biofeedback that are very important adjunctive treatments with neurotherapy. The Appendices include the details for using the ClinicalQ in practice and evaluative questionnaires that can be used for clients. In this section as well is a review of some of the highly positively synergic interactions between pharmaceutical and neurotherapeutic approaches to treatment.

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Chapter 1
Introduction

The purpose of this book is to encourage clinicians to introduce neurotherapy into their practice. Neurotherapy blends synergically with every therapeutic metaphor. Whatever your discipline, neurotherapy will markedly enhance your efficacy. As will be discussed in detail later on in this book, clinicians such as psychologists and psychiatrists, for example, will find neurotherapy markedly efficacious for treating all levels of the detrimental sequellae of exposure to severe emotional stressors.

Physicians will find that they have alternative methods for dealing with conditions such as anxiety and depression as well as facilitating greater specificity of pharmacological treatments. In the latter situation, for example, the ClinicalQ can identify the forms of ADHD that respond well to stimulants such as methylphenidate and those that do not respond well or are exacerbated by such medications. Of course, they can also incorporate neurotherapy to treat the ADHD adjunctively with medication in many instances.

This Clinician's Guide introduces clinicians to basic neurotherapy. The procedures are applicable to a broad range of patients/clients seeking treatment for a very wide array of conditions. These are basic procedures designed to augment the clinician's skill set in whatever therapeutic metaphor practiced within the jurisdictional guidelines for the professional discipline. This guide does not include more specialized areas such as full brain QEEG, z-score therapies, sLORETA, and the like. Hence, conditions such as traumatic brain injury, epilepsy, and Parkinson's require more specialized neurotherapy, although these basic procedures may be helpful in such conditions for treating adjunctive conditions such as sleep disorder, depression, and anxiety.

This guide is the sister volume to my book *Biofeedback for the Brain* (2010). The latter book is for the general public and this guide provides the technical details for clinicians. This book presents evidence from our database for the efficacy of the diagnostic procedure referred to as the "ClinicalQ." The ClinicalQ uses a limited number of EEG brain sites but provides a wealth of information about the conditions and symptoms presented by clients and patients. This procedure is not

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"diagnostic" in the usual understanding of that term. Rather, the ClinicalQ identifies brain functioning anomalies associated with the client's symptoms and behaviors that direct the practitioner to exact brain locations and brain wave ranges that need to be treated. It is not a labeling procedure. There is ample evidence to indicate that using the QEEG to guide treatment markedly increases the efficacy of treatment in contrast to simply relying on behavioral diagnosis (Gunkelman 2006).

Definitely NOT Business as Usual

I do not ask clients why they have come to see me. I tell them why they are seeking treatment. The level of precision of the ClinicalQ is such that, with experience, one can describe the client's condition based exclusively on the brain wave data. Clients are usually stunned by the accuracy of the description of their condition. The therapeutic value of this method is substantial. The methods have been refined over the last 20 years to the point that clients usually do not elaborate on my description of their condition.

Imagine a client who has been to many clinicians. She has told her story many times and obviously has not had much success in getting relief from her condition; otherwise, she would not be sitting in my office. She is often angry, disillusioned, depressed, and feeling hopeless. Before she can start telling her tale of woe, I say, "Do you know what I do?" I explain that I look at how the brain is functioning. I am looking for areas of inefficiency in brain activities that are, in turn, directly related to symptoms. Once identified, I correct the brain inefficiency that in turn reduces symptom intensity. I then explain that I will be looking at a few spots on the brain. She will not feel anything; it is measurement only. After I collect the brain wave data, I will do some calculations and go over the results in detail to be sure that what the brain is telling me is consistent with her personal experience. This procedure can help to commit the client to treatment. Clients are impressed by the accuracy of the diagnostic procedures and gain optimism regarding the potential efficacy of the treatment.

Many "one-size-fits-all" practitioners, many of whom are not licensed to practice any healthcare profession, treat with relaxation-focused feedback protocols. Clients may have some benefit in the short term, in terms of feeling more relaxed, but seldom achieve relief from the causes of their difficulties. As Hammond (2006b, p. 32) has pointed out, "A 'one-size-fits-all' approach that is not tailored to the individual will undoubtedly pose a greater risk of either producing an adverse reaction or of simply being ineffective." Hammond goes on to stress that anyone doing neurotherapy should be a bona fide licensed healthcare provider in the relevant jurisdiction. Hence, this book is written specifically for the licensed practitioner who wishes to add this technology to those available within her or his healthcare professional discipline.

One of my goals is to bring this effective set of therapeutic tools into the primary healthcare context so that clinicians have a broader array of options to treat

many of the disorders seen on a day-to-day basis. I will describe, in precise detail, how one uses the ClinicalQ for client assessment and will show the data that validates the interpretative process. I will then proceed to describe, in detail, exactly how one does the various forms of neurotherapy to correct the conditions identified with the ClinicalQ. Neurotherapy offers the possibility to correct the problem at the source so the orientation in the treatment of some disorders like depression, for example, shifts from coping and symptom control to correcting the cause of the problem.

The term "neurotherapy" refers to a number of treatment methods that alter brain functioning. In this book we will examine many different methods for correcting brain wave anomalies. The core treatment method within this array is neurofeedback or brain wave biofeedback. All clinicians, by now, have at least a passing understanding of brain wave biofeedback. The procedure has been in use for over four decades with compelling evidence for the efficacious treatment of many disorders including epilepsy, ADHD, and depression. As we shall see, neurotherapy can be an effective alternative for the treatment of a very large array of disorders.

Neurofeedback is an operant conditioning procedure. When the brain is responding as desired, the client receives a rewarding stimulus. This can be a tone indicating positive changes in brain wave activity. The reward can also be icons moving on a computer screen so that an ADHD child, for example, is playing a video-like game with his brain. We can also create treatment preparations in which the child can keep an electric train moving with her brain. The "reward" in other words is a stimulus indicating success.

More aggressive treatment protocols include "braindriving" which is a treatment incorporating the classical conditioning processes. The classical conditioning of brain wave amplitude was demonstrated in the 1940s at McGill University in Canada by Herbert Jasper and Charles Shagass (1941). The basic preparation is to present an unconditioned stimulus contingent on brain wave activity such as amplitude. Thus, for example, when Alpha amplitude exceeds a training threshold, a flashing light is presented to the client's eyes that ceases as soon as the Alpha amplitude drops below the training threshold. Flashing light is an unconditioned stimulus for Alpha amplitude suppression. Flash a light in someone's eyes and Alpha amplitude drops. By making this process contingent on the Alpha EEG amplitude, lower amplitude can be conditioned. Much of this book will be focused on identifying the classical conditioning paradigm and the unconditioned stimuli that can be used for different brain waves at various locations.

Although clinicians may have some understanding of brain wave biofeedback, that understanding, I have found, is frequently limited. A common belief is that brain wave biofeedback is a good, but costly, method to help patients relax. More efficient than meditation, it nonetheless has a limited benefit of a temporary change in brain wave activity, similar to drowsiness that helps clients find a relaxing state. Inherent in this conception is that brain wave biofeedback does not affect permanent change in brain wave functioning. Thus, clinicians with this misconception of brain wave biofeedback are likely to dismiss neurotherapy as simply another form of relaxation.

Neurotherapy treats conditions that have been considered untreatable. These methods can provide more effective ways for treating many of the depression and anxiety disorders than conventional psychology and medicine have offered to date. But neurotherapy does not replace these traditional methods; rather, it offers opportunities for synergy among the treatment methods.

Neurotherapeutic treatment starts with an assessment of brain wave activity. We have an understanding of what that brain wave activity should look like under normal circumstances. Departures from those normative values are indicative of some level of inefficiency in brain functioning. These inefficiencies in brain functioning in turn are associated with symptoms. By interpreting these departures from normative values, the clinician can identify the symptoms that brought the patient to seek treatment. It is very different from the usual procedure of the client describing the problems to the clinician. After verifying that the symptoms suggested by the brain wave anomalies are those for which the client seeks treatment, the clinician proceeds to outline, to the patient, the exact nature of the treatments designed to normalize the brain wave activity that in turn leads to symptom improvement.

An initial decision that must be taken at the outset is whether or not the patient needs to have a full head electroencephalography (all 19 sites, called a FullQ, or full map) or if the limited ClinicalQ will be adequate. Conditions of traumatic brain injury, including head trauma and stroke, certainly would require the FullQ. Similarly, conditions such as seizure disorders and psychoses likewise should be assessed with the FullQ. Conditions more commonly seen, including the anxiety disorders, the various forms of depression, attention problems in children, panic disorders, irritable and inflammatory bowel disorders, sleep quality issues, addictions, fibromyalgia, chronic fatigue, and the sequellae of emotional trauma, all would be appropriate to assess with the ClinicalQ.

Brain Wave Biofeedback

Brain wave biofeedback, also called neurofeedback, is based on a simple premise that clients can alter a brain wave activity if they receive immediate feedback on brain wave state changes. We have known since the mid-1960s that rodents can be taught to change their heart rate and blood pressure (Dicara and Miller 1969). And from around that same time, we have known that cats can be taught to change their brain wave activity (Sterman 2000). Evidence that such changes are stable over time, an indicator of brain plasticity (LeDoux 2002), was reported some years later (Lubar 1991).

If clients can be taught to change their brain wave activity, and such changes are stable over time, then symptoms associated with an abnormal brain wave activity should be treatable by normalizing brain waves. The clinical procedures are remarkably logical and straightforward: measure the brain wave activity; find the anomalies; ask the patient if the symptoms associated with the identified anomalies are present; and if the client admits to the symptoms, then help the client learn to correct

the anomalies with brainwave biofeedback. When the anomalies are corrected (brain wave activity within normative ranges), the patient should report symptom elimination or improvement.

We will be looking at a limited number of brain wave ranges for diagnostic purposes. The important ranges are Delta (2 cycles per second, Hz), Alpha (8–12 Hz), Theta (3–7 Hz), Beta (16–25 Hz), and high Beta/Gamma (28–40 Hz). Some other more limited ranges will be considered as well. As will become obvious, the significance of each of these brain wave ranges depends on the brain location. For example, elevated Theta in the front part of the brain may be associated with poor cognitive processing, whereas similar amplitude of Theta in the back of the brain may be associated with feelings of calm and well-being. Similarly, too much Alpha in the right frontal cortex may be associated with defiance in a child, whereas similar amplitude in the left may be associated with depressed mood state.

Once the problematic areas are determined, the practitioner selects the appropriate brain wave ranges that are to be treated and sets the treatment parameters so the patient receives feedback for the desired brain wave changes in real time. For example, a client with an alcohol problem who has a brain wave deficiency in the back of the brain would have the electrode placed over the occipital region. In such cases the problem is often a deficiency of Theta amplitude (brain waves between 3 and 7 Hz) or an excess of Beta amplitude (brain waves between 16 and 25 Hz), so the practitioner would set the training parameters to give a tone feedback whenever the amplitude of Theta is increasing and/or the amplitude of Beta is decreasing. Patients generally do this kind of brain wave biofeedback with eyes closed.

Stimulated EEG (Braindriving)

All stimulation changes brain waves. Stimulation, such as sound, can have a specific effect on brain waves, and this specificity can be used systematically to condition brain activity. One of the most important developments in neurotherapeutic treatment procedures, stimulated EEG procedures, called braindriving, can rapidly modify brain wave activity. These procedures are often used for emergencies to calm distressed clients and are particularly effective with autistic spectrum disordered clients. In addition, braindriving is used in remediation protocols, in which brain stimulation is occurring simultaneously with a task such as reading or writing.

The basic principle of braindriving is that stimulation is contingent on brain wave activity, in contrast to a static procedure of stimulation independent of brain wave activity. For example, one can listen to music, a static situation, and measure changes in EEG activity associated with the stimulation. In a braindriving preparation, the stimulating music would be presented only when a specific brain wave condition prevailed. The music would shut off if the specific brain wave condition were not present.

For example, severely distraught clients often need immediate relief. These clients may be in states of anxiety, panic, fatigue, despair, or physical unease. A psychologist might attempt to provide relief by guiding the client in a relaxation exercise or by administering a quieting procedure such as hypnosis, craniosacral therapy, bilateral stimulation, experiential therapy, microamperage stimulation of the head or of acupuncture points, or one of the several energy psychology procedures. A physician might also consider medicating the patient. Generally, these procedures are used to quiet the patient, after which the patient is engaged in some therapeutic procedures to correct the cause of the distress.

Braindriving offers an effective method for bringing immediate relief in such situations, as exemplified in the following case of a client who was experiencing severe anxiety and deep depression. She reported that she was crying "continuously," could not sleep, and was having panic episodes. The condition had come on suddenly and she was frightened and considered going to an emergency room at a psychiatric hospital. She reported a history of depression and always felt that she was an anxious person.

When using braindriving in this acute context, it is critical that the client understands that the procedure is designed to give significant but temporary relief and that they must commit to engaging in therapy to deal with the neurological and psychological causes of distress. When dealing with urgent situations of clients in severe distress, the usual ClinicalQ brain wave evaluation is abbreviated. The clinician reads the data and moves directly to the treatment without providing assessment information to the client. Clients in states of severe distress are not in a position to benefit from a detailed explanation of their brain wave patterns, and eliminating it provides more time for the emergency treatment. The brain wave data are explained in detail to the client at the next session, after the client has regained some calm and composure. Acute decompensation is routinely associated with a frontal lobe imbalance and a deficiency in the slow brain wave frequencies and/or an excess of fast-frequency amplitude in the back of the brain.

After verifying that these brain wave conditions prevail, the clinician moves directly to administering the emergency treatment to calm the client. In the present case, the frontal Alpha was found to be imbalanced with the left, having 26.9 % greater Alpha amplitude relative to the right, and the Theta/Beta ratio at location O1 in the occipital region was 0.66. As will be discussed in detail later in this book, these values are considerably outside the clinical normative ranges. The emergency braindriving combined the treatment of both the frontal lobes and the occipital region. At the end of the 40-min treatment, the frontal lobes were balanced and the ratio in the back of the brain had more than doubled. Even though still deficient in the Theta/Beta ratio at the back, the client reported considerable relief from her distress and was able to resume her day-to-day activities while she proceeded with psychological treatment. She was scheduled for a return visit in 2 days at which time her data would be reviewed, and a brief version of the emergency treatment administered to help sustain the improved calm.

At the core of my approach to neurotherapy is the ClinicalQ. Simply stated, the ClinicalQ is a very efficient intake assessment methodology. It provides rapid

diagnostic data that permits remarkably accurate descriptions of the client's complaints without asking the client to explain anything. Of critical importance to any clinician treating disorders of the central and autonomic nervous systems, the rapport with the client/patient is profoundly strengthened with the use and interpretation of the ClinicalQ during the intake session.

The ClinicalQ is rapid, requiring only 6½ min of recording time when using a single EEG channel and considerably less time when using multiple channels. It is data driven and logical and offers not only diagnostic insights but also serves as a guide to precise treatment and a method to evaluate patient progress as treatment proceeds. We turn now to a detailed discussion of the ClinicalQ and the research supporting the interpretations of the data obtained from this efficient assessment procedure.

Chapter 2
The ClinicalQ

Clinical Versus Normative Databases

For clinicians, the most accurate databases are clearly clinical. Normative databases are far less accurate. The fundamental organizing concept of the normative database for the clinical practitioner is, simply stated, wrong.

The organizing concept for normative databases is that one can identify a group of individuals who are symptom free and therefore have "normal" functioning neurology. This group of symptom free individuals then serves as the comparative database to identify those who are statistically discriminant. The statistical departures from the normative database define the anomalous neurological condition that is associated either causatively or exacerbatitively with the client's clinical condition. This concept is wrong.

The reason that normative database treatment recommendations are so often incorrect is because the fundamental premise is wrong. Symptom free individuals may well have predispositions to conditions that have not manifested. The data are quite clear and we have definitive evidence for this that spans decades.

Let us simply take the example of heritability data for schizophrenia. As the data in Table 2.1 indicate, if one monozygotic twin has been diagnosed with schizophrenia the probability that the second identical twin will have schizophrenia is about 50 %. But, the interesting statistic is that 50 % will not! Where do we find the 50 % without manifested schizophrenia, but obviously with the same genetic load? In the normative databases! So clearly the organizing concept for normative databases, at least for clinicians, is incorrect. Normative databases so constituted ignore basic psychopathology and basic biology. Every person has predispositions. Predispositions to anxiety, depression, emotional volatility, and the like. However, many of these predispositions are not manifest at any particular time. In general, clinicians understand that one needs an experiential trigger to "turn-the-key" to manifest a neurological predisposition.

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Table 2.1 Heritability statistics on schizophrenia

Genetic predispositions	
Monozygotic twins	30–50 %
Dizygotic twins	15 %
Siblings	15 %
General population	1 %

Adopted-biological relatives with Schizophrenia	
Adoptee with Schizophrenia	13 %
Adoptee without Schizophrenia	2 %

Source: Gottesman (1991) *Schizophrenia Genesis: The Origin of Madness*. New York: Freeman.

These logic considerations are well known and surprisingly, at least to me, ignored by non-clinicians that develop the normative databases. If in the normative database one has subjects with non-manifested predispositions, then statistically one can expect very poor discrimination.

Conditional Probability Models

There are many conditional probability models associated with the concepts of differential susceptibility. In mathematical game theory, the probability of future actions is predicated on present state. In chess, the probability of Queen move is markedly different if Queen Pawn has advanced. This is considered a state conditional probability.

In optimal performance contexts, conditional probability theories consider both vulnerability as well as resilience markers. The markers can be direct, or primary, such as the genetic serotonergic system inefficiency affecting stress tolerance. The concept of "preparation for duty" for military and police personnel is premised on reducing vulnerability to work stress (e.g., combat) by increasing the neurological basis for stress tolerance.

Secondary markers may be introversion that reduces probability of development of social relationships that in turn is negatively synergic with the primary marker. Hence, in the latter case the individual who has experienced severe stress may be more vulnerable to negative posttraumatic sequellae if the secondary marker impeded the development of a social support network.

Obviously, in the clinical context, individuals who present themselves for treatment have a manifested susceptibility factor. Individuals who do not present for treatment may have the same neurological predisposition but has not manifested. Hence, the latter individual is a candidate for normative database whereas his cohort with the identical, but manifested, predisposition is in my office and hence in the clinical database. Also, obvious, the normative database is going to be statistically blind to many neurological conditions that are predispositions.

Where normative databases have strength are determinant neurological abnormalities such as those associated with epilepsy, autism, structural damage, and

progressive neurological deterioration. Conditions associated with primary genetic (e.g., dopamine/serotonin), secondary endophenotypic (e.g., autonomic reactivity) and phenotypic (e.g., sensory processing), and tertiary endomorphic (e.g., body mass) are likely to be under the statistical discrimination thresholds. However, most importantly, the normative databases just simply miss neurological relationships found in brainwave activity for conditions that bring clients into the clinician's office.

The ClinicalQ is a clinical database. The database contains 1,508 clinical clients. The organizing logic is that clients who report a condition (e.g., depression) have a neurological representation of that condition. Based on the diathesis vulnerability model, the condition reported by the client is one that is associated with a neurological predisposition that has manifested. A normative database is likely to miss this entirely since this clinical client, before becoming depressed, had the same neurological predisposition but would be considered "normal" (i.e., symptom free) and eligible for the normative database.

The important concepts of the vulnerability or conditional probability models for the clinician include conditional vulnerability (cf., Ingram and Luxton 2005), diathesis (Sigelman and Rider 2009; Belsky and Pluess 2009) and that although neurological predispositions are stable across the lifespan, they are not unchangeable (Lipton 2006; Oatley et al. 2006).

Although the theoretical concepts associated with predispositions and vulnerabilities are of interest, for the purposes of this guide, the critical issues are that predispositions are just that, predispositions. It is also important that predisposition does not mean inevitable. People can have a multitude of predispositions but may be fortunate enough to never have them triggered and therefore be even more fortunate to never need our services.

Finally, expressivity of the predisposition in neurology is analogous to severity of a condition in clinical medicine. The severity of the EEG condition is not directly associated with the severity of the symptom. In general, the more severe the EEG condition, the more pronounced the symptomatology in terms of several parameters including chronicity, intensity, treatment resistance, and qualitative manifestation. However, many variations occur so that clinically one uses the ClinicalQ to identify clinical conditions that should be probed/explored with the client. The qualitative features of the symptoms may well be poorly correlated with the magnitude of the ClinicalQ markers. This is especially true of ClinicalQ markers associated with experiential factors as compared to genetic predispositions.

The ClinicalQ

Introduction

To illustrate the superiority of clinical norms, consider the following comparison with a normative database (Figs. 2.1, 2.2, and 2.3). Both the ClinicalQ and the 19-point full EEG were obtained simultaneously. The normative report was generated by one of the best known services whereas the ClinicalQ was generated