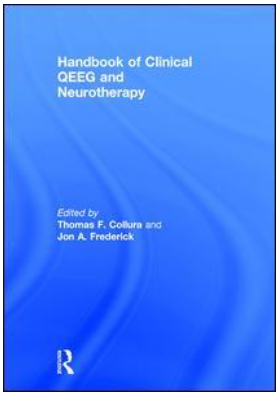

Handbook of Clinical QEEG and Neurotherapy

Thomas F. Collura, Jon A. Frederick

Neurotherapy for Clinicians in the Trenches

Publication details
https://www.routledgehandbooks.com/doi/10.4324/9781315754093.ch20
Paul G. Swingle
Published online on: 17 Nov 2016

20

NEUROTHERAPY FOR CLINICIANS IN THE TRENCHES

The ClinicalQ and Braindriving

Paul G. Swingle

Abstract

Neurotherapy marries perfectly with all health care disciplines by facilitating neurologically based diagnostics and guided treatment for a vast array of disorders. The reasons for the superiority of clinical data base guided diagnostics and treatment for clinical conditions such as the multiple varieties of depression are examined. Braindriving, a more aggressive treatment relative to instrumental conditioning based neurofeedback, has been found to facilitate more efficient neurological change. Practicing clinicians will find sufficient detail to be able to implement the ClinicalQ assessment procedure and to utilize basic braindriving protocols, all of which are guided by the intake clinical QEEG. Conditional probability concepts, relevant to the clinical context of expressivity of neurological predispositions, including differential susceptibility, conditional vulnerability, neurological diathesis and plasticity are reviewed.

Neurofeedback and the broader discipline, neurotherapy, are not stand-alone therapies—a misconception commonly made and, in some cases, perpetrated by non-clinicians, pseudo-clinicians and disenfranchised clinicians as well. Neurotherapy marries perfectly with all other therapeutic metaphors, providing no-nonsense, data driven and remarkably efficient treatment for a very wide range of disorders. This chapter is addressed to trained clinicians who have something to bring to the therapeutic context to blend with neurotherapy.

There are levels of engagement for the efficient blending of neurotherapy with other therapeutic methodologies. This chapter will focus on very basic levels of EEG intake assessment and treatment methods. These are rapid procedures that can be accomplished with very basic EEG encoders (clinical grade, of course). Basic does not mean compromised or limited, however. These are remarkably powerful procedures that, when combined with the professional health care clinician's skill set, can handle the majority of the disorders typically seen in practice. Problematic conditions, such as traumatic brain injury or psychoses, require not only enhanced neurotherapy assessment and treatment methods but more knowledgeable and experienced clinicians as well. Clinics that offer these more advanced procedures, however, are most efficient when the basic procedures described in this chapter are utilized for the general client populations. In fact, in many clinical contexts, the ClinicalQ is substantially more accurate than the normative data bases for reasons to be discussed. As will be discussed in detail, the ClinicalQ is an intake assessment QEEG based on measurements at five scalp locations. The obtained QEEG data are compared with a clinical data base to identify clients with

predispositions for specific clinical conditions. For example, several neurological profiles have been identified for clinical clients reporting "depression." ClinicalQ data matching one or more of these patterns identifies a client who is predisposed to these conditions and the clinical "probing" of these potentialities is the organizing concept for the clinical intake session. Again, as we will discuss, this process of bottom-up is a marked departure from usual clinical intake procedures and can have a remarkably favorable effect on the client's confidence in the efficacy of neurotherapy.

Clinical versus Normative Data bases

For clinicians, the most accurate data bases are clearly clinical. Normative data bases are far less accurate. The fundamental organizing concept of the normative data base for the clinical practitioner is, simply stated, logically incorrect.

The organizing concept for normative data bases 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 data base to identify those who are statistically deviant. The statistical departures from the normative data base define the anomalous neurological condition that is associated with the client's clinical condition. This concept is also logically incorrect.

The reason that normative data base 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 (similar data are available for other conditions as well such as vulnerability to PTSD and Bipolar Disorder). As the data in Table 20.1 (Ginsberg & Cancro, 1985; Gottesman, 1991; Gottesman & Shields, 1972) indicate, if one monozygotic twin has diagnosed schizophrenia, the probability that the second identical twin will have schizophrenia is about 50%. So, the twin with schizophrenia ends up in the ClinicalQ data base. 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 data bases! So clearly the organizing concept for normative data bases, at least for clinicians, is incorrect. Normative data bases 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 a trigger to "turn-the-key" to manifest a neurological predisposition.

These logic considerations are well known and surprisingly, at least to me, ignored by the developers of the normative data bases. If in the normative data base one has subjects with non-manifested

Table 20.1 Heritability of 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%

predispositions, then statistically one can expect very poor discrimination. That is, very poor discrimination between a client with a manifested predisposition compared with a data base containing like individuals with identical but unmanifested predispositions.

Conditional Probability Models

There are many conditional probability models associated with the concept of differential susceptibility. In mathematical game theory, the probability of a future event is predicated on present state. In chess, the probability of a specific 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 the 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 post-traumatic 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 it has not manifested. Hence, the latter individual is a candidate for normative data base whereas his or her cohort with the identical, but manifested, predisposition is in my office and hence in the clinical data base. Also, obviously, the normative data base is going to be statistically blind to many neurological conditions that are predispositions.

Where normative data bases 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) and secondary phenotypic (e.g., autonomic reactivity/sensory processing) are likely to be under the statistical discrimination thresholds for the reasons outlined above. However, most importantly, the normative data bases just simply miss neurological relationships found in brainwave activity for conditions that bring clients into the clinician's office.

The ClinicalQ is a clinical data base. The data base contains more than 1,400 clinical clients. The organizing logic is that clients who report a condition (e.g., depression) have specific neurological representations for the various forms 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 data base 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 data base.

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

Although the theoretical concepts associated with predispositions and vulnerabilities are of interest, for the purposes of this chapter the critical issue is that predispositions are just that, predispositions. It is also important to note 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.

Symptom Checklists

The absurdity of this approach is nicely captured by the diagnostic criteria used for applying the labels of ADHD (Inattentive Type) and ADHD (Hyperactive Type) advocated in the DSM-5 (American Psychiatric Association, 2013). As most readers know, the procedure is to rate the child if they have six or more of a list of symptoms such as "makes careless mistakes in schoolwork," "does not follow through on instructions," or the one I like the most (actually from the DSM-4; American Psychiatric Association, 1994), "runs/climbs excessively." Further, the child must be adjudicated to have manifested these symptoms for at least six months. Again, as most readers will recall, there are separate lists for inattentive and hyperactive types of ADHD.

So, here are a few questions about these criteria: What if you only have five? Or three? What if the parent notices these behaviors and has observed them for three months—not yet ADHD? Let Johnny fail his semester since we have to wait six months for it to be *real* ADHD? What if you only observe these behaviors in church? In this regard, it is interesting to note that there has been a marked increase in the NOS (not otherwise specified) diagnoses with children for exactly the reasons stated above. That is, children who do not meet all the criteria for the label are given the subthreshold (shadow) designations. This can be a problem, according to the authors, because of inappropriate off-label prescribing and creation of "vast heterogeneity" that, in turn, confounds intervention research (Safer, Rajakannan, Burcu & Zito, 2015). We also see this problem with misdiagnoses of the attention deficit disorders (Swingle, 2015a).

It is important to ask clients about their symptoms, of course. But, we must not base our neurological treatment solely on these self-reports, because they are often incorrect. Later in this chapter, the condition defined generally as "depression" will be reviewed. Clients report they are depressed for all sorts of reasons: poor sleep, loss of a pet, severe anxiety, emotional trauma, as well as neurological predispositions. Each of these conditions implicates different neurology, and to be strictly efficacious treatment must be neurology specific regardless of the self-reports. And given that the normative data bases are likely to miss the relevant neurology, for reasons already discussed, clinicians should be grateful that at least 50% of their effectiveness is placebo and hence canned protocols, either one-size-fits-all or symptom specific, have some efficacy.

The ClinicalQ

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 brainwave data. Clients are usually stunned by the accuracy of the description of their condition. The therapeutic value of this method is substantial. This experience is nicely captured by Susan Olding (2008, several paragraphs from pages 169 through 173) in her book *Pathologies*. In her book, Susan describes the ordeals of trying to find competent treatment for her child. A chapter in this book describes the ClinicalQ process. An excerpt that indicates the diagnostic power of the ClinicalQ is as follows:

> Desperate, determined, undeterred by cost or lack of insurance coverage, undismayed by the doubts of conventional physicians . . . I switched off my cell phone at the threshold of Dr. Swingle's office and carried my daughter across. . . .

> I had brought a medical and developmental history—the long litany of concerns that had brought us to his door—but Dr. Swingle waved the papers aside without even looking at them. Instead, he ushered Maia toward a computer screen . . . [and] fixed a couple of delicate wires to her ears. . . .
>
> Then Dr. Swingle sent Maia to the "treasure chest" in the waiting room. He stared at the printout in his hand. "Here," he said, and he pointed to an outline of the brain, "these numbers imply trauma." He shrugged, palms up, waiting for my response. I nodded. "And here," he continued, "too much Theta. This is the hyperactivity people associate with ADHD. . . ." There was more: extreme stubbornness, a tendency to perseverate, lapses of short-term memory, attachment disorder, inability to read social cues, emotional reactivity, tantrums, explosions. One by one he read the ratios, divining my daughter's character more quickly, more accurately than any professional I'd yet encountered.

The assessment described in this excerpt is based on 6.5 minutes of recording time. The data from the five brain-sites are compared to the clinical data base providing guidance for the clinician to probe the client regarding symptoms. In other words, bottom-up, not top-down. More importantly, the clinical data base gives guidance to location and substance of neurotherapeutic treatment to mitigate the client's symptoms.

The ClinicalQ Assessment Protocol

It is critical that the brainwave ranges be consistent with the clinical data base. Ground is right earlobe; reference is left earlobe. Recording sites are Cz, F3, F4, Fz and O1. The ClinicalQ procedure including the brainwave bandwidths, the administration protocol, the unremarkable and basic remarkable ranges is appended to this chapter. The listed remarkable ranges and related suggested clinical probes are basic. Recognizing that the number of combinations from only the two frontal regions, F3 and F4, is over 100, it requires a bit of practice to fully benefit from the diagnostic potential of the ClinicalQ. But soon, the experienced clinician will find that this procedure provides surprisingly accurate diagnostic data (Swingle, 2015b).

To illustrate the superiority of clinical norms, consider the following comparison with a normative data base. 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 immediately while the client was still hooked up. Many manufacturers of EEG platforms have software available for generating the ClinicalQ data and probes; however, following the outline in the Appendix, one can generate the ClinicalQ data and summary with any EEG platform with the aid of a desk-top hand calculator.

It is quite apparent that the ClinicalQ was far more accurate for this client. He reported sleep problems consistent with the low Theta/Beta ratio under eyes closed conditions at location O1. The marked imbalance in Alpha, frontally, with Alpha being considerably higher in amplitude in the right relative to the left, is the marker for emotional volatility. As this client reports: "I get angry easily." The client's complaints of problems with focus and attention are reflected in the elevated Theta/Beta ratios at location Cz, F3 and F4 as well as the elevated Delta and slow Alpha as measured at Fz.

We also see another marker that is not reported by the client. Beta is considerably greater in amplitude in the right relative to the left frontal cortex. This is a marker for depression. When probed about this, the client admitted to feeling "low" much more intensely and frequently than he believed was the case with his friends.

NAME: **M 21** DATE: 10/27/09

Please list the symptoms for which you are seeking treatment. Indicate the severity of the symptoms according to the following scale.

1 = A minor problem.
2 = Distressing, but I can usually ignore the problem.
3 = Serious and occasionally interferes with social or work activities.
4 = Very serious and often interferes with social or work activities.
5 = Completely interferes with work and social activities.

For example, if you experience sleep disturbances that occasionally leave you too fatigued to participate in social functions, but has never prevents you from working, your listing might be as follows:

Problem/Symptom	**Severity**
"Problem with falling into sleep and I wake often and find it difficult to get back to sleep"	3

Problem/Symptom	**Severity**
1. Problem falling asleep, and staying asleep	4
2. I get easily angry	4
3. I have trouble concentrating or staying focused	4

Figure 20.1 Client's (M21) self-reported conditions.

Table 20.2 Client M21. Full 19-site QEEG report from independent service using normative data base.

II. Conclusion:

The background alpha is seen at 9-10 Hz with eyes closed, with frontal theta seen maximally at the midline anteriorly overlying the cingulate, and with persistent EMG artifact fronto-temporally and posteriorly, as described. There is less beta power than expected. The frontal midline slower content is described in perseverative disturbances such as OCD/ODD and the over-focus associated with generalized anxiety disorder. The theta/beta ratio was increased significantly, as is commonly seen in AD/HD at the vertex. There is left mu noted, which is a normal neurological variant, though it is also reported disproportionately in those with mirror neuron disturbances. There is a slight increase in the relative slower content left temporally, suggesting a disturbance in areas involved in language comprehension and verbal memory.

III. Recommendation for therapy:

Based on these findings, the SMR training should be considered for stabilization and calming, and to help re-tune the alpha frequencies. The left temporal training may be added to address any language comprehension or verbal memory issues**. The frontal midline slower suppression with beta training may be used for stimulation, as well as to counter-act any under-activation noted due to the SMR type training. The faster beta suppression band is intended to catch the EMG activity noted during the recording.

The specific frequencies, montages, and the sequencing of sessions will likely require modification based on client response. Suggestions may be implemented differently depending on instrumentation used. All decisions regarding implementation of these suggestions are the responsibility of the practitioner. *I recognize the need for on-going consultation and welcome discussion concerning clinical progress of individual patients, or related questions and suggestions.*

	Active	Reference	train	inhibit
1	Cz	Linked ears	(12-15)	(5-11) + (22-30)
2	T5 **	Cz	(13-16)	(5-11) + (22-30)
3	Fz	Linked ears	(15-18)	(5-11) + (22-30)

** The T5 is for language comprehension, as well as verbal memory.

Tables 20.3a, 20.3b, 20.3c and 20.3d ClinicalQ for client M21.

(20.3a)

CZ	*Values*
EO Alpha	7.1
EC Alpha	10.2
% Change EO to EC Alpha	43.7
EO Alpha Recovery %	8.3
EO Theta/Beta	2.94
UT Theta/Beta	3.11
% Change T/B EO to T/B UT	5.8
Total Amplitude	33.6
Alpha Peak Frequency EC	9.3
Alpha Peak Frequency EO	9.1
Theta/SMR EC	2.45

(20.3b)

O1	*Values*
Alpha EO	9.2
Alpha EC	14.3
% Change in Alpha EO to EC	53.7
EO Alpha Recovery %	15.3
Theta/Beta EO	2.21
Theta/Beta EC	1.26
% Change T/B EO to T/B EC	75.4
Alpha Peak Frequency EO	9.6

(20.3c)

F3 & F4 (All EC)	*Values*		*% Difference F3-F4*
	F3	*F4*	
Alpha Amplitude	8.9	12.3	38.2
Beta Amplitude	6.8	8.4	23.5
Theta Amplitude	22.9	21.4	7.0
Theta/Beta	3.37	2.55	32.2

(20.3d)

FZ (All EC)	*Values*
Delta (2Hz)	17.2
HiBeta/Beta	0.48
Sum HiBeta + Beta	14.2
LoAlpha/HiAlpha	1.75
Alpha Peak Frequency	9.4

The ClinicalQ shows precisely where to treat these conditions and what to treat. Standard Theta/Beta training at locations Cz, and if necessary later at F3 and F4. Increasing the Theta/Beta ratio at O1, eyes closed, for the sleep problems. Speed up the Alpha Peak Frequency (or decrease the amplitude of low Alpha) and finally balance the frontal regions, F3 and F4 in the Alpha and Beta ranges. Rule of thumb—treat sleep problems first as restored sleep quality is likely to result in other improvements in brain functioning. There are many other general guidelines for how to approach developing a treatment strategy for the client (Swingle, 2015b).

It is also apparent that the QEEG report not only did not identify the client's complaints but the treatment strategy recommended is largely irrelevant to the client's problems. The possible exception is the recommended 12–15 Hz training at Cz. However, neurofeedback at almost any location is usually associated with client reports of improvement early in treatment.

So, it is obvious that the ClinicalQ is *not* a poor practitioner's substitution for the full 19-site QEEG. Many mini-Q systems are being marketed on exactly that basis. The purpose of using the ClinicalQ is to make neurotherapy much more efficient; because, again, the ClinicalQ is more accurate for clinical practice than the normative data bases. The intake procedure with the ClinicalQ is the first therapy session. Clients are strongly relieved that their complaints are understood, that there are identifiable neurological causes/corollaries of their condition and there is a precise "game plan" for treatment.

"Depression"

A common complaint of clients we all treat is "I'm depressed." The client has a huge array of options for receiving treatment for this amorphous condition including prescription medications, supplements, exercise, endless psychotherapies, R&R and, of course, an array of neurotherapies. In the array of neurotherapies we have those that are normative data base guided including: neurofeedback, the z-score zapping paradigms (brain-site specific frequency amplitude departures penetrating z threshold evoke an infinitesimal amp/gauss zap), and z-score neurofeedback; sLORETA; canned feedback protocols based on defined condition (i.e., "depression"); and franchises with proprietary symptom checklist driven canned protocol systems.

ClinicalQ based treatment is different. A few cases, described below, exemplify how treatment is guided by bottom-up assessment and verification. Neurotherapeutic protocols are then precisely targeted at these verified neurological inefficiencies. Because of space limitations, the ClinicalQs for the following cases will be presented in summary form rather than the full output, as shown above. In addition, only data relevant to the present discussion are included in the summary.

The fundamental neurological condition one finds in depression is an imbalance in the frontal cortex with the right (F4) being more active as compared with the left (F3). This imbalance can result from several neurological conditions as measured with the EEG. The Davidson (1995) pattern, identified years ago, is when Alpha has greater amplitude in the left relative to the right.

However, there are many other conditions that result in this imbalance. For example, the client shown in Figure 20.2 is what we might call "garden variety" depression. This client has an imbalance where Beta is greater in the right relative to the left. Clinically this appears to be the "genetic" predisposition for depression although it is found in clients who have recently experienced a loss. Figure 20.3 shows the Davidson depression marker of elevated Alpha in the left relative to the right frontal cortex. The client shown in Figure 20.4 is similar in that Theta is greater in the left relative to the right resulting in the right being more active than the left. Clinically the two patterns just described (low frequency amplitude greater in the left) are very frequently associated with reactive depression (exogenous). Finally in Figure 20.5, we see a pattern often found with a person with the predisposition to depression who has experienced a severe emotional stressor that has triggered the predisposition.

Emotional trauma, exposure to a severe emotional stressor or an accumulation of emotional stressors, is associated with a blunting of the Alpha response at locations Cz and O1. We understand that this marker is associated with incompletely processed emotional sequellae of the emotional event(s). Exposure to emotionally negative images (corpses) has been shown to temporarily blunt the Alpha response, and fortuitous exposure to severe emotional stress with clinical clients likewise revealed

Cz	Mθ/β	α↑%
	1.69	85.2

O1	θ/β O	θ/β C	α↑%
	1.62	1.74	110.9

	θ	α	β
F4	12.1	9.9	8.6
F3	11.3	9.2	7.1
%d	7.1	7.6	21.1

Fz	Dz	Hβ/β	Σ
	10.9	0.82	10.0

Figure 20.2 'Genetic' depression

Cz	Mθ/β	α↑%
	1.37	38.3

O1	θ/β O	θ/β C	α↑%
	2.20	2.24	55.8

	θ	α	β
F4	9.7	7.6	6.9
F3	9.2	9.3	6.3
%d	5.4	-22.4	9.5

Fz	Dz	Hβ/β	Σ
	8.9	0.52	11.2

Figure 20.3 Reactive depression (Alpha)

Cz	Mθ/β	α↑%
	1.48	59.3

O1	θ/β O	θ/β C	α↑%
	2.02	1.74	75.4

	θ	α	β
F4	9.7	9.1	6.3
F3	14.0	8.2	6.1
%d	-44.3	10.9	3.3

Fz	Dz	Hβ/β	Σ
	8.8	0.61	12.6

Figure 20.4 Reactive depression (Theta)

Cz	Mθ/β	α↑%
	2.24	10.9

O1	θ/β O	θ/β C	α↑%
	1.53	1.47	55.8

	θ	α	β
F4	11.8	7.7	8.9
F3	11.7	6.7	6.6
%d	1.0	14.9	34.8

Fz	Dz	Hβ/β	Σ
	7.5	0.58	7.5

Figure 20.5 Trauma triggered depression

Cz	Mθ/β	α↑%
	1.49	8.1

O1	θ/β O	θ/β C	α↑%
	1.55	1.64	15.4

	θ	α	β
F4	13.0	8.5	6.6
F3	14.4	8.9	6.7
%d	-10.8	-4.7	-1.5

Fz	Dz	Hβ/β	Σ
	11.7	0.74	13.6

Figure 20.6 Trauma based depression

Cz	Mθ/β	α↑%
	1.34	39.6

O1	θ/β O	θ/β C	α↑%
	1.06	0.87	64.8

	θ	α	β
F4	12.8	9.7	6.9
F3	12.0	8.9	10.6
%d	6.7	9.0	-53.6

Fz	Dz	Hβ/β	Σ
	7.3	0.77	11.4

Figure 20.7 Anxiety based depression

Alpha blunting. Alpha blunting is seen as restricted elevation of Alpha amplitude when clients close their eyes (Swingle, 2013). (See the parameters for this response in the Appendix to this chapter). The Alpha response is completely ignored in the normative data bases.

Occasionally one sees clients who report that they are depressed but there are no depression markers in the ClinicalQ. There are many profiles that are found but two are relatively common. The profile shown in Figure 20.6 shows no depression markers but both trauma markers. There are other details of clinical relevance in this profile but the critical point for this discussion is that unprocessed trauma can be manifested as reports of "depression." The lack of the reactive depression markers (e.g., Davidson, 1995) may indicate that the client is in the numb phase of post-traumatic exposure. However, although of interest to speculate on these matters, clinically one proceeds to release the Alpha and then utilize whatever therapy the clinician judges relevant to resolve the condition. It is with these trauma clients that the one-size-fits-all franchisers are the most destructive. Often one will hear comments about how to quiet an emotionally abreacted client who has been subjected to one of the canned protocols. Exactly the opposite of good clinical practice.

The profile shown in Figure 20.7 is also quite common. These are clients in severe states of anxiety who feel hopeless, frightened and out-of-control. They report being "depressed" because their lives are in shambles, or they feel they are going to decompensate, or they feel just plain helpless. Treating these conditions with antidepressants is a formula for creating a life-long problem. The ClinicalQ identifies the areas for neurotherapeutic treatment quite precisely. Again, there are several other aspects to this EEG profile of clinical relevance such as markers for cognitive perseveration, but for the purposes of the present discussion it is the two markers of deficient Theta/Beta ratio at the occipital location and elevated left frontal Beta that identify the anxiety state.

The markers for depression are only part of the profile. To illustrate, consider the child shown in Tables 20.4a and 20.4b. This child was brought for treatment of an attention problem. He was having

Tables 20.4a and 20.4b Nine-year-old male child—potential bully victim.

Cz	*Values*	*% Change*
EO Alpha	8.61	
EC Alpha	10.23	
% Change EO to EC Alpha > 30%		18.78%
EO Alpha Recovery	9.27	
% Change EO - Alpha Recovery		7.63%
Theta Amplitude EO	15.76	
Beta Amplitude EO	6.50	
EO Theta/Beta	2.47	
Theta Amplitude Under Task (UT)	13.69	
Beta Amplitude UT	5.89	
UT Theta/Beta	2.32	
% Change T/B EO to T/B UT		–6.45%
% UT Beta Increase		–10.29%
Total Amplitude	30.65	
Theta Aplitude preceding Omni	14.42	
Theta Amplitude with Omnl	13.15	
% Change In Theta with Omnl		–9.68%
Alpha Peak Frequency EC	10.00	
Alpha Peak Frequency EO	9.80	
Theta/SMR EC	3.15	

O1	*Values*	*% Change*
Alpha EO	6.16	
Alpha EC	12.06	
% Change in Alpha EO to EC		95.84%
EO Alpha Recovery	5.70	
% Change EO - Alpha Recovery		–8.16%
Theta Amplitude EO	10.09	
Beta Amplitude EO	5.17	
Theta/Beta EO	1.95	
Theta Amplitude EC	10.46	
Beta Amplitude EC	6.99	
Theta/Beta EC	1.50	
% Change T/B EO to T/B EC		–30.21%
Alpha Peak Frequency EC	10.00	
Alpha Peak Frequency EO	9.90	

Tables 20.5a and 20.5b Nine-year-old male child—potential bully victim.

F3 & F4 (All EC)	*Values*		*% Difference F3-F4*
	F3	*F4*	
Theta Amplitude EC	10.10	16.93	
Beta Amplitude EC	6.37	7.06	
EC Theta/Beta	1.59	2.41	
% Diff F3T/B - F4TB EC			50.93%
Theta Amplitude EC	10.10	16.93	
	13.47	9.28	
	0.75	1.83	
	29.95	33.26	
	6.37	7.06	10.74%
	13.47	9.28	–45.20%
	10.10	16.93	67.56%

Fz (All EC)	*Values*
Delta (2Hz)	10.05
HiBeta Amplitude	3.89
Beta Amplitude	6.15
HiBeta/Beta	0.63
Sum HiBeta + Beta	10.04
LoAlpha Amplitude	5.20
HiAlpha Amplitude	3.61
LoAlpha/HiAlpha	1.44
Alpha Peak Frequency	9.40

significant problems in school and was judged to have many of the symptoms associated with ADHD (inattentive type). The figures are the actual output from the ClinicalQ for this child.

Again, there are many features of this profile that are clinically important but we will limit the discussion to those associated with the suspected bullying. This child does show minor marker for ADD. The Theta/Beta ratios at Cz are a bit elevated. However, this child is showing a trauma marker at Cz (Alpha response of 18.78%), a marker for reactive depression at F3/F4 (Alpha is 45.2% greater in the left relative to the right), and a marker for emotional volatility (F4 Theta is considerably greater than at F3). So, the hypotheses are that this child has or is being exposed to significant emotional stressors, that he is experiencing a reactive depression (perhaps related to the emotional stressors) and that he is emotionally volatile (and hence a "sitting-duck" for a bully). This child, if these hypotheses are correct, cannot pay attention or do well in school because he is afraid! Probing the child and the parents revealed that the child was being severely bullied, he was afraid to tell his parents because of the bullies' threats and he was emotionally volatile (cried frequently over minor issues). The parent corrected the bully issue at school and we did some minor braindriving neurotherapy to improve the minor ADD problem.

Braindriving

As mentioned in the last case, braindriving was the neurotherapeutic treatment for the child's ADD. Braindriving is a very useful procedure that is based on classical rather than instrumental conditioning. Quite simply, it is based on concept of applying Unconditioned Stimuli (UCS) contingent on brainwave activity. For example, when the goal of treatment is to reduce the amplitude of Theta, an UCS for Theta reduction is presented when Theta amplitude exceeds training threshold. Conversely, if Theta enhancement is the goal, negative threshold crossings are contingent on application of a Theta enhancement UCS.

Classical conditioning of brainwaves was demonstrated by Herbert Jasper and Charles Shagass (1941). In a series of studies they demonstrated that Alpha blunting can be conditioned to a sound, periodic time intervals and verbal commands. For braindriving, we have found a number of UCS for conditioning brain activity including light at various wavelengths, frequencies and intensities; sounds; acupuncture sites (for electrical stimulation); and brain-sites for magnetic (milligauss) stimulation.

There are many combinations of UCS that can be used for braindriving. The details for combining UCS can be found in Swingle (2010). Protocols can include a Theta enhancement UCS that is presented when Theta amplitude drops below training threshold, plus an entraining UCS that is presented when the amplitude goes above threshold. For common ADD children, a Theta suppressant can be presented when Theta is above threshold (Swingle, 1996) and a sustaining UCS can occur when the amplitude is below the threshold. Because the lights UCS can be presented on eyeglass frames, around the periphery of the lenses, the child can be engaged in a relevant task such as reading, writing, math, etc. This has proven to be a very effective treatment for children with attention and learning difficulties.

Several examples of braindriving protocols are shown in Figures 20.8 through 20.11. Figure 20.8 shows the data associated with braindriving Beta down at location O1 for a client with a deficient Theta/Beta ratio. Common complaints associated with this condition as previously discussed include problems with stress tolerance, sleep quality and self-medicating behavior. Concentration can also be poor because of "brain chatter." As the data indicate, contingent stimulation of the Heart 6 (palmer ulnar surface slightly above the wrist crease) acupuncture meridian (bilateral) resulted in a decrease in the amplitude of Beta and an increase in the amplitude of Theta. The resulting increase in the Theta/Beta ratio after this 20-minute session was 76.1%. Subjective reports following this session was of profound quiescence. Recall, the efficacy of the UCS for this client is pretested before the braindriving session.

Figure 20.9 shows the data from an elderly client who was experiencing problems with memory and cognitive efficiency. Alpha slowing, measured in this case with the Alpha density ratio of lo-Alpha/hi-Alpha, can be an age related decline. These declines can be effectively treated with brain-brightening protocols such as those developed by the late Tom Budzynski (Budzynski, Budzynski, & Tang, 2007). Braindriving has also been shown to be particularly effective for these clients as the data shown in Figure 20.9 indicate. The protocol was to present both the OMNI harmonic (a blend of sounds that reliably suppresses Theta amplitude) plus 11 Hz visual stimulation whenever 8–9 Hz brainwave amplitude crossed the training threshold. As indicated the pre-treatment L/H Alpha ratio was 2.83, which dropped to 2.76 after the first two minutes of treatment. By the end of treatment the ratio was 1.78, which is a 35.5% decrease. Further sessions, either neurofeedback or braindriving, would be required to bring this ratio into efficient range (below 1.50). Once the L/H Alpha ratio is in an acceptable range, clients with persistent age related decline are assessed and treated between two and four times per year to maintain efficient Alpha Peak Frequency.

Figure 20.10 shows a braindriving session with a client with common ADD condition of elevated Theta amplitude as measured at location Cz. By the end of the session the Theta/Beta ratio had decreased by about 30%.

One very important use of braindriving is for treatment of emotional trauma. Obviously, this procedure should be used only by licensed providers experienced in dealing with clients affected by post-traumatic stress. The blunted Alpha response, discussed in the first section of this chapter, is a marker for unresolved emotional stress. There are several methods for releasing and processing this emotional state including EMDR, hypnosis, experiential psychotherapies—to mention but a few. Braindriving can markedly accelerate this process in a positively synergic manner.

The data shown in Figure 20.11 show emotional release with one of the "Alpha push" protocols. As the data indicate, as the Alpha amplitude starts to increase this client experienced an emotional release, lasting about eight minutes. Many therapists stop the braindriving at this point and continue with a procedure such as EMDR. In this case, the client continued with braindriving and started the recovery phase after about eight minutes. After the session, the client was probed regarding the experience. She reported an emotional episode which she described in some detail. Brief therapeutic intervention resulted in an emotional redefinition of the event, a desired outcome of such therapy.

Pre-treatment θ/β 0.85			
	θ	β	θ/β
START	14.3	12.4	1.09
END	17.8	9.4	1.92
%	24.5	–24.2	76.1

Figure 20.8 Braindriving Beta down @ O1 with H6 stimulated > T

Pre-treatment L/Hα = 2.83			
	Lα	β	θ/β
START	14.1	5.0	2.76
END	10.0	5.6	1.78
%	–29.1	+12.0	–35.5

Figure 20.9 Braindriving Low Alpha down @ Fz with OMNI and 11Hz visual > T

Pre-treatment θ/β under cognitive challenge = 3.29			
	θ	β	θ/β
START	23.5	7.4	3.17
END	18.8	8.4	2.23
%	–25.0	13.5	–29.6

Figure 20.10 Braindriving Theta down @ Cz with 16 Hz and OMNI > T

Trauma Release

Trial	θ	α	β	
1	12.5	11.5	8.2	
2	16.0	13.2	9.4	
3	13.1	15.5	9.9	← α release
4	8.4	12.6	9.3	
5	9.7	11.0	8.8	} emotional release
6	9.2	10.9	8.6	
7	8.8	10.1	8.1	
8	12.9	12.9	9.1	
9	15.8	13.4	9.5	} recovery
10	14.7	15.2	9.5	

Figure 20.11 Braindriving Alpha @ Pz with 11 Hz and Serene < T

Table 20.6 EEG of client going into sleep state.

Average Amplitudes: Trial #	*Theta Filter 1 uV*	*Alpha Filter 2 uV*	*Beta Filter 3 uV*
1	14.6	18.1	11.2
2	10.4	18.0	12.1
3	7.3	22.8	12.0
4	9.1	18.8	11.1
5	7.4	17.2	10.5
6	8.5	10.2	7.7
7	9.6	6.9	6.8
8	11.2	8.2	7.6
9	12.7	8.2	7.8
10	14.7	9.7	9.3
Session Avg.	10.5	13.8	9.6

Prior to all braindriving sessions the efficacy of the UCS should be assessed. Because braindriving is an aggressive therapy the changes are often sizable but one should expect some after session regression towards pre-treatment levels. The resulting after treatment level is usually above pre-treatment level. Often to stabilize the braindriving gains, the client is shifted to straightforward neurofeedback. Braindriving can also be added to regular neurofeedback protocols by having the UCS sound as the feedback with the client instructed to keep the sound "off." If the UCS lights are presented on goggles with look-though lenses, the child can be reading, writing, doing math, etc. while the implicated area of the brain is under braindriving treatment. This has been found to be very effective for facilitating skill acquisition (e.g., written output).

Finally, quieting braindriving protocols can be extremely soporific so the therapist should be attentive to brainwave activity indicating that the client is falling asleep. Table 20.6 shows just such a client where Alpha and Beta drop substantially while Theta starts to increase. The remarkable increase in the occipital Theta/Beta ratio in this case is attributable to the client's sleep state even though braindriving induced it!

References

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychological Association.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychological Association.

Belsky, J., & Pluess, M. (2009). Beyond diathesis stress: Differential susceptibility to environmental influences. *Psychological Bulletin, 135*, 885–908.

Budzynski, T., Budzynski, H. K., & Tang, H.-Y. (2007). Brain brightening: Restoring the aging mind. Chapter. In J. R. Evans (Ed.), *Handbook of neurofeedback* (pp. 231–265). Binghampton, NY: Haworth Medical Press.

Davidson, R. J. (1995). Cerebral asymmetry, emotion, and affective style. In R. J. Davidson & K. Hugdahl (Eds.), *Brain asymmetry* (pp. 361–388). Cambridge, MA: MIT Press.

Ginsberg, G. L., & Cancro, R. (1985). Schizophrenia: The epigenetic puzzle. *The Psychoanalytic Quarterly, 54*, 305–306.

Gottesman, I., & Shields, J. (1972). *Schizophrenia and genetics: A twin study vantage point.* Boston: Academic Press

Gottesman, I. I. (1991). *Schizophrenia genesis: The origin of madness.* New York: Freeman.

Ingram, R. E., & Luxon, D. D. (2005). Vulnerability-stress models. In B. L. Hankin & J. R. Z. Abela (Eds.), *Development of psychopathology: A vulnerability stress perspective* (pp. 32–46). Thousand Oaks, CA: Sage Publications Inc.

Jasper, Herbert, & Shagass, Charles. (1941). Conditioning the alpha rhythm in man. *Journal of Experimental Psychology, 28*(5), 373–388.

Lipton, B. H., (2005). *The biology of belief: Unleashing the power of consciousness, matter and miracles.* San Rafael, CA: Elite Books.

Oatley, K., Keltner, D., & Jenkins, J. M. (2006). *Understanding emotions.* Oxford, UK: Blackwell Publishing.

Olding, S. (2008). *Pathologies.* Calgary, AB: Freehand Books.

Safer, D. J., Rajakannan, T., Burcu, M., & Zito, J. M. (2015). Trends in subthreshold psychiatric diagnoses in youth in community treatment. *JAMA Psychiatry, 72*(1), 75–83.

Sigelman, C. K., & Rider, E. A. (2009). *Lifespan human development* (6th ed.). Belmont, CA: Wadsworth.

Swingle, P. G. (1996). Subthreshold 10Hz sound suppresses EEG theta: Clinical application for the potentiation of neurotherapeutic treatment of ADD/ADHD. *Journal of Neurotherapy, 2*, 15–22.

Swingle, P. G. (2010) *Biofeedback for the brain* (Revised paperback ed.). New Brunswick, NJ: Rutgers University Press.

Swingle, P. G. (2013). The effects of negative emotional stimuli on alpha blunting. *Journal of Neurotherapy, 17*(2), 133–138.

Swingle, P. G. (2015a). *When the ADHD diagnosis is wrong.* Santa Barbara, CA: Praeger.

Swingle, P. G. (2015b). *Adding neurotherapy to your practice.* New York: Springer.

Appendix A

ClinicalQ

Procedure

Epoch	*@Cz*	*@O1*	*@F4*	*@F3*	*@Fz*
1	EO	EO	EC	EC	EC
2	EO	EO	EC	EC	EC
3	EC	EC	EC	EC	EC
4	EO	EO	EC	EC	EC
5	(READ				
6	OR				
7	COUNT)				
8	EO	(EO = EYES OPEN)			
9	UCS	(EC = EYES CLOSED)			
10	TEST				

Technical Notes

1. Right ear ground and left ear reference
2. Epoch length 15 seconds, shorter if necessary
3. Recording @ Cz is usually one continuous run of 10 epochs
4. Recording @ O1, F3, F4, Fz is one run at each site of 4 epochs
6. Cognitive challenge is either reading or counting
7. UCS Test is testing of stimulus (e.g., sound)
8. Data are mean amplitudes unless artifacts indicate the use of medians
9. Unremarkable ranges, listed below, are normative guidelines; specific ranges may vary somewhat based on equipment, environmental conditions and certainly age of client
10. The ClinicalQ is not appropriate for assessment of stroke, seizure disorders or traumatic brain injury. ClinicalQ is often appropriate for first assessment of autistic spectrum to determine initial treatment protocols to be followed by full QEEG

Unremarkable Clinical Ranges

1. @Cz: mean Theta/Beta < 2.2; alpha increase EC > 30%; Theta/Beta ratio cognitive challenge < 2.2 but no marked difference from mean and Beta increase < 20%; sum of all mean amplitudes, total power, (TA) < 60.0
2. @O1: EO and EC Theta/Beta 1.80–2.20; Alpha increase EC > 50%
3. @F4 and F3: F4 = F3 in all bands, Theta/Beta ratios < 2.00; Theta/Alpha ratio 1.25–1.60; TP = and < 60
4. @Fz: 2Hz < 10.0; 28–40Hz/Beta 0.45–0.55; 28–40Hz and Beta < 15.0; 8–9Hz/11–12Hz < 1.50

Clinical Implications of Remarkable Ranges

The following clinical probes should be considered as suggestions for developing a behavioral profile of the client. Remarkable ranges do not validate a clinical diagnosis. Similar remarkable patterns can be associated with different clinical profiles. For example, developmental delay, fetal alcohol syndrome and some autistic spectrum profiles can have very similar remarkable QEEG patterns. It is important to keep in mind, therefore, that the remarkable ranges indicate inefficiencies and not necessarily clinical diagnoses. Unique remarkable patterns are associated with some specific conditions, such as Common Attention Deficit Disorder (CADD) (item 1 under CZ, with no other remarkable ranges). It is the treatment specificity afforded by identifying remarkable ranges rather than diagnostic labeling that makes the ClinicalQ a valuable rapid intake procedure. The following suggested clinical probes are not exhaustive. The experienced clinician will identify many patterns associated with specific client complaints.

@Cz

1. Mean Theta/Beta > 2.2 and under cognitive challenge > 2.2, probe for CADD
2. Mean Theta/Beta < 2.2, under cognitive challenge >2.2, probe for ADD and/or problem with poor reading, comprehension/retention
3. Mean Theta/Beta > 3.00, probe for AD(H)D
4. Limited or negative EC Alpha increase, probe for visual processing (memory) problem. Probe for exposure to traumatic stress, particularly if also negative @ O1
5. TA > 60.0, probe for developmental delay, autistic spectrum behavior, marked cognitive deficits

@O1

1. Theta/Beta EO < 1.80, probe for poor stress tolerance, "racing" thoughts, anxiety. If < 1.00, probe for addictive behavior, GAD and Stress Precipitated Depression
2. If Theta/Beta EC < EO, probe for sleep disturbance particularly sleep onset insomnia. If both EC and EO about = and < 1.50, also probe sleep disturbance
3. If Alpha EC increase minimal or negative probe for exposure to traumatic stress
4. Theta/Beta > 3.00, probe for cognitive inefficiencies. Also found in some Asperger's patterns

@F4 and F3

1. Theta/Beta > 2.2, probe for cognitive inefficiencies
2. Theta/Alpha < 1.00, probe for frontal Alpha ADD—problems with organization, sequencing, sustained focus. If Theta/Alpha < 0.80, also probe for fibromyalgia and sleep disturbance

3. F4 Beta > 15% of F3 Beta; F3 Alpha > 15% F4 Alpha; F3 Theta > 15% F4 Theta; F3 Theta/Beta > 20% of F4 Theta/Beta, probe for depression particularly in adults; also probe for impulse control problems in children
4. F4 Theta > 15% F3 Theta, probe for emotional volatility or conversely restricted emotional range. F4 Alpha > 15% F3 Alpha, also probe for emotional volatility, oppositional behavior in children, interpersonal problems with adults
5. TA > 60.0, probe for developmental delays, autism spectrum disorders, memory/cognitive deficits in adults
6. F4 Beta > 20% of F3 Beta and F4 Theta > 20% of F3 Theta, probe for fibromyalgia/chronic fatigue, particularly when O1 Theta/Beta < 1.50

@Fz

1. Delta (2Hz) > 9.0, probe for cognitive deficits
2. 28–40Hz/Beta < 0.45, probe for excessive passiveness
3. 28–40Hz/Beta > 0.55, probe for stubborn behavior, obsessive/compulsive behavior, perseveration in autistic spectrum behaviors; assume hot midline (anterior cingulate gryus) in treatment of autistic spectrum behaviors
4. Implications of ratios in 2 and 3 above apply only if sum of amplitudes of 28–40Hz & Beta < 15 If summated amplitudes > 15, but 28–40Hz/Beta is within normative range, probe for fretting and assume hot midline in treatment of autistic spectrum behaviors
5. 8–9/11–12 > 1.50, probe for cognitive inefficiency, age related deficits in memory and cognitive processing
6. 8–9/11–12 > 1.50, probe for developmental delay, marked cognitive deficits