THE NEUROTHERAPEUTIC TREATMENT OF DEPRESSION

(Published in BC Psychologist: The Journal of the BC Psychological Association. Winter 2015)

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INTRODUCTION

A recent advertisement for an antidepressant medication shows depression as a dark little pesky fellow who seems to whimsically make you feel more or less glum. You can take him on a picnic with you and can make him better behaved if you take the supplementary antidepressant offered by the pharmaceutical company. There are many wrong messages conveyed in this advertisement. A few of which include: First, there is nothing funny or cute about depression. Second, if the depression is appearing and fading ephemerally, then behavioural modification not chemicals is warranted. Third, depression is not just bad humour and if you are taking antidepressant medication for bad humour then you are popping "happy pills."

We have many terms for depression meant to convey qualitative differences in just how one experience various states of despair, gloominess, melancholy, misery, disinterest, sadness, hopelessness, and dejection. We also have technical terms to define the functional differences among these states such as the presumed cause of the condition, and the tendency for the condition to fluctuate between sadness and elation or vary according to time of year.

Depression is a neurological disorder that can be effectively treated with neurotherapy. Depression that results from an event such as death of a loved one, or even the depression of a loved one, is also reflected in identifiable and therefore treatable anomalies in brainwave activity. However, such reactive depression states are usually more appropriately left untreated or treated psychologically such as with CBT. These event-related disturbances in mood are generally transient and one gets on with life. Neurotherapeutic or pharmacological treatment of "reactive" depression can often exacerbate these conditions (sedate rather than process) leading to long-term mood disorder.

Neurological predisposition to depression is genetic in nature although some event such as severe stress is usually associated with "turning on" the state of depression. Neurologically-based mood disorders persist and predispose one to have more prolonged or more intense reactions to emotional events. Hence, when a client has a neurological predisposition to depressed mood states, treating these neurological predispositions with a client in a reactive depressed state is appropriate.

All of the mood disorders have identifiable brainwave patterns that permit very precise diagnoses and more importantly point out precisely how the neurotherapist should proceed to correct the problem

The traditional method for dealing with depression is for the client/patient to describe the subjective symptoms during an initial visit with a health care provider. After describing the symptoms the client may be asked to complete questionnaires that help define the symptom complex. Treatments vary, of course, depending on the training/expertise/field of practice of the health care provider and the specifics of the depression as defined by the client/patient.

SYMPTOM CHECKLISTS

The absurdity of this approach is nicely captured by the diagnostic criteria used for applying the label of "Major Depressive Disorder" advocated in the DSM-5. As most readers know, the procedure is to rate the client if they have 5 or more of a list of symptoms such as "Depressed mood most of the day…," "…diminished interest or pleasure in all, or almost all, activities most of the day…," "Insomnia or hypersomnia nearly every day" Further, the client must be adjudicated to have manifested these symptoms every day for at least 2 weeks.

So, here are a few questions about these criteria. First: What if you only have four? Or three? What if you only observe these behaviors in church? What if the patient wakes up wanting to hang himself every Friday but is OK by Monday?

It is important to ask clients about their symptoms, of course. But, we must not base our neurological treatment on these self-reports, because they are often incorrect. Later in this chapter, the conditions 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.

Where neurotherapy differs substantially from these traditional methods is in the diagnosis and treatment of the cause rather than treatment of the symptom. People can have symptoms of depression for many different neurological and experiential reasons. Neurometrics identifies those causes and predispositions and localizes regions of the brain for efficient treatment. Neurotherapy does not necessarily replace the other treatment methods and is often used in complement with the other therapeutic procedures.

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 (Swingle, 2010) 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 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 ½ minutes of recording time. The data from the 5 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 (Swingle, in press).

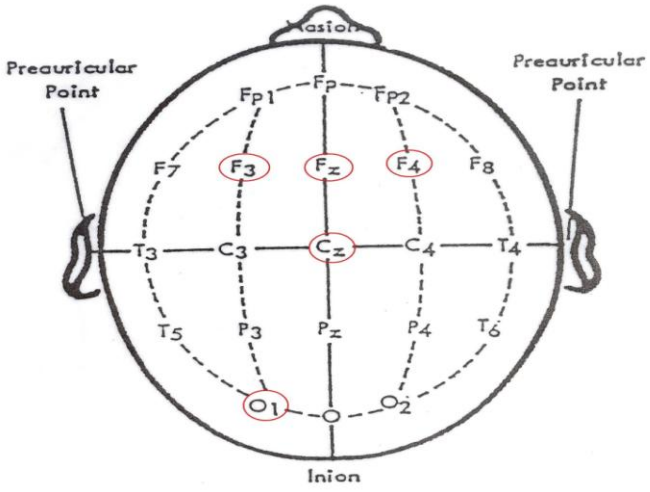

Figure 1: 10-20 international EEG site location system.
The five point ClinicalQ locations are noted in red.

THE NEUROMETRICS OF DEPRESSION

The fundamental neurological condition associated with depressed mood is when the right frontal region of the brain is more active than the contralateral left side. There are several ways this can occur. The neurological condition associated with this disparity in activity level is linked to differences in the qualitative features of the depression. Common neurological patterns of depression include those associated with genetic predisposition and exposure to negative emotional experience. In addition, neurological predisposition to poor stress tolerance is also often misconstrued as "depression" but is in actuality an anxiety pattern not a depression pattern. These common patterns are shown below. The EEG patterns have been identified statistically from a clinical data base of over 1400 clinical clients (see Swingle, in press, for details).

ClinicalQ based treatment is different in that 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 neurological patterns will be presented in summary form rather than the full output. 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; Henriques and Davidson, 1990) 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 2 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 client's having recently experienced a loss. Figure 3 shows the Davidson depression marker of elevated Alpha in the left relative to the right frontal cortex. The client shown in Figure 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. In Figure 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 Alpha blunting. Alpha blunting is seen as restricted elevation of Alpha amplitude when clients close their eyes (Swingle, 2013).

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 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 "neurofeedback" 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 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. This common form of depression results from exhaustion associated with severe chronic anxiety, poor sleep quality or persistent mental chatter. In such circumstances the person is worn out, sleep deprived and "at the end of their tether." Commonly associated with "burn-out," this form of depression is characterised by profound feelings of fatigue, lack of interest in activities and avoidance of social interaction. This condition is better thought of as a fatigue condition caused by a neurological predisposition to poor stress tolerance. The neurological marker for this condition is a low ratio of the amplitude of theta brainwaves (3-7 Hz) to beta brainwaves (16-25 Hz) at location O1. At the Swingle Clinic we see many clients with decades of pharmaceutical and "psychotherapy" treatment for "depression" who obviously are not doing well. They are not doing well because the wrong condition is being treated. 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. There are some qualitative differences in this condition depending on whether the deficient theta/beta ratio results from elevated Beta amplitude or from deficient Theta amplitude, or both. Knowing which form of anxiety based depression the client is experiencing is critical to providing effective neurological treatment for this disorder.

Cz	M Θ/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 2: 'Genetic' depression. Beta (16–25 Hz) amplitude is > 15% higher in the right (relative to left) frontal cortex.

Cz	M Θ/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 3: Reactive depression (Alpha). Alpha (8-12 Hz) amplitude is > 15% higher in the left (relative to the right) frontal cortex.

Cz	M Θ/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 4: Reactive depression (Theta) That (3-7 Hz) amplitude is > 15% higher in the left (relative to the right) frontal cortex.

Cz	M Θ/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 5: Trauma triggered depression. Alpha (8-12 Hz) amplitude response when eyes closed is < 30%. Beta (16-25 Hz) amplitude is > 15% higher in the right (relative to left) frontal cortex.

Cz	M Θ/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 6: Trauma based depression. Alpha (8-12 Hz) amplitude response is < 30% at Cz and < 50% at O1.

Cz	M Θ/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 7: Anxiety based depression. Theta/Beta amplitude ratio < 1.80 at location O1, and Beta amplitude > 15% higher in the left (relative to right) frontal cortex.

SUMMARY

Depression is a neurological disorder that can be effectively treated with neurotherapy. Depression that results from an event such as death of a loved one, or even the depression of a loved one, is also reflected in identifiable and therefore treatable anomalies in brainwave activity. Predisposition to depression is also a neurological condition that is genetic in nature although some event such as severe stress is usually associated with "turning on" the state of depression.

There are many factors that contribute to depression and mood disorders**.** Events such as loss of a job, death of a friend, family member or a pet, break-up of a relationship, dissatisfaction with one's performance, etc. can all

cause periods of depressed mood states. These event-related disturbances in mood are generally transient and one gets on with life. Neurologically-based mood disorders, on the other hand, persist and predispose one to have more prolonged or more intense reactions to emotional events. Some depressed mood states are characterized by intense feelings of sadness or hopelessness, others by burn-out and fatigue and still others by feelings of disinterest, emptiness and flat emotional states.

All of the mood disorders have identifiable brainwave patterns that permit very precise diagnoses and more importantly point out precisely how the neurotherapist should proceed to correct the problem. Some forms of depression are associated with imbalances in the frontal regions of the brain such as when beta amplitude is considerably greater in the right frontal cortex relative to the left, as shown above. Other forms of depression are associated with deficits of slow frequency brainwave amplitude in the back of the brain. Bipolar conditions are usually associated with brainwave anomalies in both the front and the back of the brain. A brainwave assessment by a qualified neurotherapist is an effective first step toward treating depression.

REFERENCES

Henriques, J.B., and Davidson, R.J. (1990) Regional brain electrical asymmetries discriminate between previously depressed and healthy control subjects. *Journal of Abnormal Psychology, 99, 22-31.*

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