

# Depression and Treatment of Depression in Neurofeedback

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## Abstract

At the root of all our thoughts, emotions and behaviors lay the intricate networks of communication among the trillions of neurons within our brains. A measurement of this communication activity, like the rhythm or pulse of a flowing river, is the brainwaves. Brainwaves are tiny pulses of the electrical activity that are produced as the neurons communicate with each other. By influencing these electrical patterns, we can change the brains communication. This means that when there are patterns set up within the brain that are not working correctly there will be corresponding problems. These patterns are sometimes referred to as pathologically stable patterns. The pathological patterns can arise from a variety of possible stressors, i.e. abuse, physical trauma, emotional trauma, chronic continuous stress, worry, anxiety, etc. As a response to the perceived threat, the brain has adopted a protective patter in an effort to deal with the past (it can be just a memory) or present trauma. The brain is simply doing the best it can to protect us and enable us to deal with the real or unreal dangers and threats it perceives. A huge list of disorders can be traced to this underlying problem.

**Keywords:** depression; neurofeedback; neurofeedback treatment protocols.

## 1. Introduction

Depression is a state of low mood and aversion to activity that can affect a person's thoughts, behavior, feelings and sense of well-being [1,2].

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Depressed patients show relative hypo activation in the left prefrontal area, which differs from that found in normal people who show relative hyper activation in the left prefrontal area compared to the right frontal area [3,4]. This phenomenon was observed in remitted depressed patients [3] and children of a depressive mother [5]. This observation suggests that an impairment in the left prefrontal function may indicate susceptibility to depression [6]. 'Depression' is a broad and popular concept that has been widely used in reference to a state of diminished humor that stands beyond the relationship which one draws with specific stressors. The intensification and recurrence of this state gives birth to major depressive disorder (MDD), which is a much more specialized concept that psychologists and psychiatrists use to refer to a disabling condition that not only include the aforementioned (low) mood symptoms, but several other mental and organic manifestations [7], ranging from neuro vegetative abnormalities to cognitive and problem-solving deficits [8]. The most well-known models of MDD neurobiological basis focus on the neurochemical dysfunctions that accompany its phenomenological manifestations (of which the monoaminergic hypothesis is the most traditional: [9]). Recently, alternative models have been proposed; with a special emphasis on the disorder's electroencephalographic (EEG) correlates. This new perspective opened a venue to the use of EEG for diagnostic and prognostic purposes, producing a new hope and excitement among patients and health practitioners, commonly seen today. In 2003, an article published in a journal of the "Nature Group" pointed to the possibility of using an electroencephalographic marker, discriminated in REM sleep, as a predictor of therapeutic success of subsequent pharmacological treatment [10]. In 2006, another study demonstrated the possibility of using EEG to predict the potential effects of different antidepressants (and thus to define the pharmacological treatment of choice), 48 hours before any perceived effect [11]. Also, in 2008 it was found that a very simple electroencephalographic marker (Alpha asymmetry) could be used to predict the response to antidepressants before the beginning of the pharmacologic treatment, in such a sense that it could serve as an aid in the choice of treatment [12].

**Neurofeedback** Training the electrical activity and timing of the brain to improve brain functioning 'Neurofeedback is a complementary therapy that is based on the paradigm of operant conditioning to teach brainwave activity to increase or decrease' is a new therapeutic approach that has recently entered therapy and specialists in different fields of psychology, psychiatry and occupational therapy is used [13].

## **2. History of neurofeedback**

The first major neurofeedback study was done with cats by Dr. Barry Sterman. He trained cats to increase their SMR (12-15hz) through operant conditioning. Showed that cats could be trained to increase SMR. Published in Brain Research, 1967. Studied seizure thresholds for cats' exposure to rocket fuel. Some of the cats did not have seizures at the known threshold levels. Sterman checked his records and found the seizure resistant cats were the ones who had SMR training then decided to investigate whether neurofeedback would help humans with seizure disorders. His studies showed a decrease in seizure severity and frequency with SMR training [14].

## **3. Brain waves and their functions**

### **3.1 Slow Frequencies**

Internalized awareness. Awareness is deep inside.

DELTA (.5-4 Hz)

- Primary rhythm. Brainstem.
- Found during sleep
- During waking state = lesion or metabolic disturbance (CFS)
- 3 Hz surges = old emotional trauma
- Delta is original energy and absolutely essential for the restoration work. So don't skip a good night sleep.

THETA (4-8 Hz)

- Drowsy state
- Good vs. bad theta:

Good theta: intuition-hypnogogic; creativity.

Bad Theta: escape/spacing out; aversive using protection state.

- Theta is protecting you. It is a result of the Limbic system. Fight or flight response. However, protecting is not (always) serving you. Therefore training Theta down is getting used of a situation without protection. In other words, training theta down is asking to give up protection. In important task here for the practitioner.
- Hormone influence: just before menstruation cycle Alpha slows down and Theta gets dominant.
- 5 Hz surges = old cognitive distortions
- 7 Hz surges = integration of information & memory consolidation

### ***3.2 Middle Frequencies***

Internal awareness speeds, minimal processing & minimal thought.

ALPHA (8-12 Hz)

- Cortex.
- Relaxed state; Alpha is the expression of the resting brain.
- Mental stillness
- Auto-pilot state, resting state
- Awareness without processing
- 10 Hz dominant for adults; normal adult alpha 8-13 —background rhythm.

8 Hz for young children (till +/- 8)

- Elderly people back to 8 Hz again.

- Hormone influence: just before menstruation cycle Alpha slows down and Theta gets dominant.
- Left less alpha, right more alpha
- Range varies with age (young = 10Hz/ old = slower)
- Slow speeds for visualization
- Three types of alpha:
  - 1- High alpha when eyes closed means.
  - 2- High alpha with eyes open and with eyes closed can indicate a problem: disassociation.
  - 3- No alpha when eyes closed and eyes open can indicate a problem: high stress response; alcohol problem.

#### Low BETA (SMR) (12-15 Hz)

- Supplementary motor area. Cz .
- Sensory Motor Strip:

T3 C3 Cz C4 T4. All information passes this strips. Filtering and organizing is taken place.

When you train here, all communication left/right and front/back gets optimized.

- Physical stillness
- Body presence: heavy & warm, low muscle tone
- 14 Hz = sleep spindles and internal body rhythms
- SMR is identified by Stroman as the most important frequency for training.
- For most people, you train SMR —GOI. It's very beneficial.
- SMR processes all input (eye, ear, skin). When you optimize this, you are more than half way.
- SMR gives two things:

It gives a pause, so that you get the opportunity to think before you make a decision. It gives more freedom of choice; you are not \_auto-triggered\*.

It increases your inhibit possibilities.

- Result of SMR-training is: Locus of control. You become less influenced from the outside world. You become the

Pilot from your own being. It means also: true responsibility.

- SMR regulates a lot of other frequencies in the brain. For example it stimulates sleep spindles at night. This is necessary to be able to sleep and to wake up fresh.
- SMR by children is lower: to 9 Hz. Mostly 10 to 14 instead of 12 to 15 for adults.
- SMR can restore the menstruation-cycle. The same with Bulimia and Anorexia.

### 3.3 Fast Frequencies

Processing speeds, sequencing, language processing, faster = more, external awareness

BETA (15-18 Hz)

- Neocortex.
- Alert state
- Detail oriented processing, calculation and understanding meaning

BETA2 (19-23 Hz)

- extreme engagement, high focus
- can produce anxiety (21 Hz may disrupt old patterns – shear frequency)

HIBETA (23-38 Hz)

- Hyper vigilance, extreme anxiety,
- PTSD or abuse issues, neglect.
- High beta in the back:
- You get endless loop thinking.
- The backside must be —let go and leave the decision-making to the front part. The back is gathering info and the front for processing.

GAMMA

- 40 Hz integrative/binding frequency – found in all areas of the brain
- 40 Hz is like drinking a pot of strong coffee [15,16].

In 1994 Behr and Rosenfeld introduced their protocol to five depressed patients who were being treated with psychotherapy, and a sixth patient who was seen in another clinic. They agreed to participate in a study to assess the effectiveness of this approach. [17]-[18]. Using the alpha asymmetry protocol, they assessed and trained depressed patients to reallocate brainwave amplitude so that the amplitude of alpha was greater in the right frontal cortex than in the homologous left frontal cortex. The application of this protocol requires scalp electrodes at two active sites, F3 and F4, a reference at Cz, and the ground at Fz . (Most of the above reported studies have used a standard EEG montage utilizing 19 or more sites). Brief mood shifts brought about by happy and sad thoughts resulted in changes in frontal asymmetry in both depressed and normal control subjects [19]. Daily changes in frontal alpha asymmetry were shown to correlate with changes in affect in therapy [20]. Right frontal activation measured in a resting state was found in adults with current, remitted and past depressions [21,22,23].

Right frontal activation was found in non-depressed adolescent daughters of depressed mothers [24]. The frontal

asymmetry has also been found in infants under the age of one that were born to depressed mothers [25], even as young as at 3–6 months [26] and at one month of age [27]. This may result from either a genetic predisposition to depression that has been passed on, and/or it may result from an over- or under activation of brain areas that mediate different emotions in the infant whose frontal lobe begins to be increasingly active at about 8 months of age. However, genetic studies of twins provided only limited evidence of heritability of frontal asymmetry patterns [28,29]. For the most part, these studies imply that right frontal cortical EEG may predict both state and trait individual differences in affect, although not all studies confirm these results [30]. Allen [31] studied subjects over an 8- to 16-week period. They acknowledged that while trait-like aspects of alpha asymmetry were characteristic of depressed individuals, state changes also occurred. Changes were not related to changes in the severity of the depression. However, [6] found a strong correlation between alpha asymmetry scores and the Beck depression inventory ( $P < 0.0001$ ), and on the MMPI-II depression scale ( $P < 0.0001$ ). The roles of early experiences and plasticity as factors in patterns of asymmetry were explored by [32]. Reference [33] studied long-term stability for frontal EEG asymmetry in adults with a history of depression, and non- depressed controls. They found that resting asymmetry reflected a moderately stable condition in adults. Reference [34] found that depressed persons could respond to a brief cognitive restructuring task, with positive changes also occurring in alpha asymmetry. An asymmetry with more fast-frequency activity in the right hemisphere has even been found to remain in the architecture of sleep in depression [35]. Research has further suggested that the right hemisphere may be specialized for processing negative affect [36,37]. Along with the frontal electrophysiology findings in depression there also seems to be an inverse relationship between frontal alpha asymmetry and parietal asymmetries. More specifically, depressed patients without significant anxiety appear to have decreased right parietal activation (more alpha at P4 than at P3) [38]. These findings are also congruent with neuropsychological test findings that have consistently identified right parietotemporal deficits in functioning in depressed subjects [39]. Recent research [40] has also verified an inverse parietal alpha asymmetry in grandchildren of depressed parents and grandparents, compared with controls without a parental and grandparental depression history. Reference [15] utilized a protocol designed to increase left frontal beta activity (15–18 Hz and 12–15 Hz) in the left frontal cortex at electrode sites FP1 and F3 in a successful single case study. He later reported [41] on the successful treatment of seven of eight subjects using the same protocol. Neurofeedback training to increase alpha and theta, while inhibiting faster beta frequencies, has also been found to produce significant improvements in depression in alcoholic and post-traumatic stress disorder populations— in randomized, control group studies [42] as well as in a case series [43] populations where one may expect an excess of fast beta activity to often be prominent, and which is quite different from the EEG patterns usually seen in depression.

#### **4. Principles of neurofeedback**

Principles of neurofeedback training directly with an immediate and direct feedback signal from the brain is reflected. The method used to record electrical brain waves and give feedback to the person trying to make a kind of self-regulation to teach the subject. Typically through sound or image feedback presented to person and in this way, the person realizes that the appropriate changes in your brainwave activity has created or not [44].

Protocols and techniques discussed here is for the Brain master [45].

#### **4.1 In cases where can teach waves in neurofeedback or not:**

- i. Do not train up Beta (15-22) in the right hemisphere. (This may result in ‘\_manic attacks’).
- ii. Do not train up Lo beta (12-15) in the left hemisphere, except in mirror protocols.
- iii. Do not train up Hi beta (23-32) anywhere.
- iv. Do not train up Alpha (8-12) frontally.
- v. Do not train up Theta (2-6) frontally.
- vi. Only do the Alpha/Theta-training after working with other protocols.
- vii. Do not train the Alpha/Theta-training when Theta amplitudes are equal or higher than Alpha. First train Alpha to be at  
Alpha to be at
- viii. least 1.5 times higher than Theta.
- ix. In the beginning Neuro therapy training must always be slow and easy.
- x. Do not train after sunset (biorhythm).
- xi. Do not train by high fever.
- xii. Do not train when the body is fighting stress.

#### **5. Duration of neurofeedback**

1. Unless otherwise is mentioned by the specific protocols, start for each protocol with 12 minutes, gradually working up to 24 minutes maximum.
2. Note: This applies to all protocols except the Instability-subcategories ‘\_Blocking’ and ‘\_Disconnect’. Here it is 10 to max. 20 minutes. The shorter the better.
3. The protocol should be continued until patient feels ‘\_calm, relaxed and focused’ without getting sleepy, falling asleep or without getting hyper – usually 10-20 sessions.
4. Training twice a week is the minimum, especially in the beginning. In fact: the more intensive the better: two times per day is Okay. Except by rebounds! To find out whether there will be rebounds, never start with 2 times a day.

#### **5.1 bi-polar and mono-polar montage.**

Bi-polar montage:

- Electrode montage on the two mirror sites.
- Both places are in fact and active and reference.
- Note. When doing coherence training, than active and reference location is important!
- Ground goes to one of the ears.
- You are balancing things in bigger areas in the brain.

- Examples:
- Two-sided: T3T4 with A1 ground.
- One-sided: T4P4 with A1 ground. (autism; Asperger)

Mono-polar montage:

- You are training a very specific problem area with this montage.
- This type of training is very intense! BE CAREFUL!!
- Train shortly: only 3 minutes !!
- This type of training is very powerful ! It activates immediately!! When you, for example, quiet down this way on C4, you can really go into —slow-motion!!

- Examples:
- When beta is low on F3 (depression), you train beta up: electrode on F3, corresponding ear as reference so A1, and ground on the other ear so A2.
- The Alpha/Theta training is an example of mono-polar montage.

When there are brain problems found in the EEG, then you find here the roadmap towards the appropriate approach and protocols (see table1).

### ***5.2 Priorities for the category or categories***

- Always begin training protocols that is the same as those of category 1 INSTABILITIES
- The next priority protocols that are in those with type 2 LOCKING
- The next priority protocol for those who are on the left.

Marker analysis of EEG in 15 the rules are eyes closed ‘2-3-12 rule base that is related to depression

The rules are as follows: (see table2).

### ***5.3 Typical approach for instability-reversal***

- When Alpha is the problem:
  - Training alpha/theta ratio up toward 1,5 at P4 (rule 13) (note. When the ratio is low, do eyes closed!),



- or training alpha coherence up at P3P4 works well as a start,
- Finishing with a bit of beta up and theta (or alpha) down at F3.
  
- When Beta is the problem:
  - Training beta/theta ratio up toward 2,5 at F3 (rule 1).
  - Or training beta coherence up at F3F4 works as well as a start,
  - Finish with a bit of alpha up and theta (or beta) down at P3.

**Table1:** The 4 categories of Brain Problems with their EEG-characteristics

<b>INSTABILITY -1</b>		
Up and down surges of activity – high variability in standard deviation of EEG. Sometimes surges of Hi beta The subcategories are specific strategies of the brain for controlling specific types of emotional overwhelm		
Subcategory 1A <b>DISCONNECT Pattern</b>	Subcategory 1B <b>BLOCKING Pattern</b>	Subcategory 1C <b>REVERSAL Pattern</b>
Excessive temporal Beta and Hi beta	Excessive Beta and Hi beta at Fz	Alpha and Beta imbalance left/right, front/back.
Typical symptoms		
<b>Subcategory 1A DISCONNECT Pattern</b>	<b>Subcategory 1B BLOCKING Pattern</b>	<b>Subcategory 1C REVERSAL Pattern</b>
Can indicate early abuse or neglect. -Flat or regressive emotional responses. -Anxiety and/or depression—Disconnection from memories & emotional content	Tendency to intellectualize emotions. -Obsessive thinking. -Compulsive behaviors. (anorexia, bulimia) -Tendency to addiction. -Phobias.	-Emotion issues: anxiety, depression. -Driven character. -Sleep disturbances. -Anger outbursts with grudges. -Tendency to —crash from time to time. -Processing problems.
<b>LOCKING -2</b>		
High coherence between mirror sites F3F4, C3C4, P3P4, mostly in Beta and Hi beta.		
<b>HYPO AROUSAL -3</b>		
1. Excess frontal Theta and/or Alpha.2. Low frontal Beta.3. High frontal Theta to Beta ratio.		
<b>HYPER AROUSAL-4</b>		
1.Excess Beta or Hi beta at P4. 2.Very low levels of Lo beta at C3 and C4. 3. Surging activity up and down the spectrum		

**Table 2:** Rules Category brain problems

category	Symptoms	EEG indicators	rule
<b>Brain Problems</b>			
Instability- REVERSAL	Depression	Beta left/right. At F3 and C3, Beta 15-20 should be higher than at F4 and C4.	2
Instability- REVERSAL	Depression	Alpha left/right. At F4, Alpha should be at least 1,25 time higher than at F3.	3
Instability- REVERSAL  /or  HYPOarousal/  PROCESSING	Depression	Alpha left/right. At any left-sided placement where Alpha is more than 1,25 times Alpha at its mirror site, i.e. C3>C4, indicates a problem.	12

We use Symptoms of Instability-REVERSAL on the left and right brain in depression following the protocols (see table3).

#### 5.4 Typical protocol for instability-reversal

Two channel —mirror-montage<sup>4</sup>:

- ✓ Typical protocol if ALPHA-ratio is the problem in front (=more alpha left than right):
  - 2 channel example: F4 – A1 . Fz . A2 – F3
  - Ch1: Go ALPHA. Stop THETA.
  - Ch2: Go Nothing. Stop ALPHA and THETA or BETA.
  - Both channels: Stop HIBETA (23-32hz).
  
- ✓ Typical protocol if BETA-ratio is the problem in front (=more beta right than left):
  - 2 channel example: F3 - A1 . Fz . A2 - F4
  - Ch1: Go BETA. Stop THETA.
  - Ch2: Go Nothing. Stop BETA and THETA or ALPHA.
  - Both channels: Stop HIBETA (23-32hz)

Note. When symptoms are ameliorated Finish off with Alpha-Theta training.

**Table 3:** using Symptoms of Instability-REVERSAL on the left and right brain in depression following the protocols.

<b>Protocol</b>	<b>EEG indicators</b>	<b>Symptoms REVERSAL</b>
INrev1	BETA should be higher at F3 than at F4	rule 2a
INrev2	BETA should be higher at C3 than at C4	rule 2b
INrev6	ALPHA should be 25% higher at F4 than at F3	rule 3
INrev6	ALPHA should be 1,25 times less at F3 than at F4	rule 12a
INrev7	ALPHA should be 1,25 times less at C3 than at C4	rule 12b
INrev8	ALPHA should be 1,25 times less at P3 than at P4	rule 12c

**Table 4:** Application of neurofeedback protocols in depression

<i>protocols</i>	<i>EEG indicators</i>	<i>Electrodes</i>	<i>CHI act-ref</i>	<i>GND</i>	<i>CH2 ref-ac</i>	<i>DURATION minutes</i>	<i>EYES</i>
<i>INrev1</i>	<i>betaF3 should be &gt; betaF4</i>	<i>Electrodes</i>	<i>F3-A1</i>	<i>Cz</i>	<i>A2-F4</i>		
<i>INrev2</i>	<i>betaC3 should be &gt; betaC4</i>	<i>Electrodes</i>	<i>C3-A1</i>	<i>Cz</i>	<i>A2-C4</i>		
		<i>GO</i>	<i>B15-18</i>		<i>--</i>	<i>10to 20.</i>	<i>Open</i>

		<i>INHIBIT</i>	<i>T2-6 (or A8-12), H23-32</i>		<i>B15-18, T2-6, H23-32</i>	
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Remarks Sound: either one sound for both criteria or two sounds when both met.  
 Screen: thermo on left = ch1 = beta up, thermo on right = ch2 = beta down

<i>Protocol</i>	<i>EEG indicators</i>	<i>Electrodes</i>	<i>CH1 act-ref</i>	<i>GND</i>	<i>CH2 ref-act</i>	<i>DURATION minutes</i>	<i>EYES</i>
<i>INrev6</i>	<i>alphaF4 should be &gt; alphaF3</i>	<i>Electrodes</i>	<i>F4-A1</i>	<i>Cz</i>	<i>A2-F3</i>		
<i>INrev7</i>	<i>alphaC4 should be &gt; alphaC3</i>	<i>Electrodes</i>	<i>C4-A1</i>	<i>Cz</i>	<i>A2-C3</i>		
<i>INrev8</i>	<i>alphaP4 should be &gt; alphaP3</i>	<i>Electrodes</i>	<i>P4-A1</i>	<i>Cz</i>	<i>A2-P3</i>		
		<i>GO</i>	<i>A8-12</i>		<i>nothing</i>	<i>5 to 10</i>	<i>Open</i>
		<i>INHIBIT</i>	<i>T2-6 (or B15-18), H23-32</i>		<i>A8-12, T2-6, H23-32</i>		

Remarks Sound: either one sound for both criteria or two sounds when both met.

Screen: thermos on left = ch1 = beta up, thermos on right = ch2 = beta down

**Table 5:** Brain problem category3: HYPOAROUSAL-PROCESSING

<b>Symptoms</b>	<b>EEG indicators</b>
Inattentive ADD	1- rule 1: Theta/Beta ratio problem at F3, F4, Fz. F4 (=theta 2x's
- Depressed/hopeless	Beta amplitude)
- Chronic pain	2- rule 18: Excess (= 2x higher than anywhere else) frontal Theta.
- Fibromyalgia & CFS (Chronic Fatigue Syndrome)	
- Learning issues : reading, listening, writing, organizing,	

sequencing.	3- Beta amplitudes frontal are excess LOW.
- Mood disorders adults	4- Excess frontal Alpha
Processing <u>depression</u> .	6- rule 12: Alpha left-sided is more than 1.25 times Alpha rightsided.
	Consider in this situation also the INSTABILITYPROTOCOLS, INrev6, 7 and 8.

<i>Protocol</i>	<i>EEG indicators</i>	<i>Electrodes</i>	<i>CHI</i>	<i>GND</i>	<i>CH2</i>	<i>DURATION</i>	<i>EYES</i>
			<i>F</i>		<i>ref-act</i>	<i>minutes</i>	
Hypo1	1,2,3,4,	<i>Electrodes</i>	F3-A1	A2	--		
Hypo2	1,2,3,4,	<i>Electrodes</i>	C3-A1	A2	--		
Hypo3	,1,2,3,4	<i>Electrodes</i>	F7-A1	A2	--		
		GO	B15-20		--	12-> 24max	Open
		INHIBIT	T2-5 & H23-32		--		

Remarks: DO NOT USE THESE PROTOCOLS for FIBROMYALGIA and CFS. Use specific protocol instead for this.

## 6. Conclusion

Neurofeedback training in more depressive disorder based on education and training alpha and beta waves, alpha waves asymmetric position of f3, f4 with reference cz, fz in the treatment of depression has been successfully training fp2 beta in the treatment of people who use the drug resistance depression showed a significant reduction in depression. A robust body of research has validated that there is a biological predisposition to depression (and to becoming withdrawn) which is associated with a frontal asymmetry wherein there is less activity in the left frontal area. Although pharmacologic treatment for depression is widespread, reviews [47,48,49,50,51] have documented that antidepressants, on average, only have an 18% effect over and above placebo effects, and yet they are associated with significant side effects such as sexual dysfunction, insomnia, increased suicide risk, diarrhea, nausea, anorexia, bleeding, forgetfulness, and withdrawal syndromes. Thus alternatives are needed to the invasive treatments commonly utilized by “biological psychiatry,” namely medication, electroconvulsive therapy, transcranial magnetic stimulation, and neurosurgery, which are

commonly associated with side effects. Studies [52,53,54] that have compared psychotherapy with medication have found that treatment outcomes are generally comparable or better than pharmacologic treatment, and when drop-out rates are taken into account drug treatment alone produces worse outcomes than psychotherapy.

## References

- [1] Salmans, Sandra (1997). *Depression: Questions You Have – Answers You Need*. People's Medical Society. ISBN 978-1-882606-14-6.
- [2] Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5). American Psychiatric Association. 2013.
- [3] Gotlib IH, Ranganath C, Rosenfeld JP: Frontal EEG alpha asymmetry, depression, and cognitive functioning. *Cogn Emot* 1998; 12: 449–478.
- [4] Johnstone T, van Reekum CM, Urry HL, Kalin NH, Davidson RJ: Failure to regulate counterproductive recruitment of top-down prefrontal-subcortical circuitry in major depression. *J Neurosci* 2007; 27: 8877–8884.
- [5] Dawson G, Panagiotides H, Klinger LG, Spieker S: Infants of depressed and mothers exhibit differences in frontal nondepressed brain electrical activity during the expression of negative emotions. *Dev Psychol* 1997; 33: 650–656
- [6] Vuga M, Fox NA, Cohn JF, George CJ, Levenstein RM, Kovacs M: Long-term stability of frontal electroencephalographic asymmetry in adults with a history of depression and controls. *Int J Psychophysiol* 2006; 59: 107–115..
- [7] Lewis, A. J. (1934). Melancholia: a clinical survey of depressive states. *Journal of Mental Science*, 80(329), 277-378.
- [8] Hamilton, J. P., & Gotlib, I. H. (2008). Neural substrates of increased memory sensitivity for negative stimuli in major depression. *Biological Psychiatry*, 63(12), 1155-1162.
- [9] Delgado, P. L. (2000). Depression: the case for a monoamine deficiency. *Journal of clinical Psychiatry*, 61, 7-11.
- [10] Murck, H., Nickel, T., Kunzel, H., Antonijevic, I. A., Schill, J., Zobel, A., et al. (2003). State markers of depression in sleep EEG: dependency on drug and gender in patients treated with tianeptine or paroxetine. *Neuropsychopharmacology*, 28(2), 348-358
- [11] Hunter, A. M., Leuchter, A. F., Morgan, M. L., & Cook, I. A. (2006). Changes in brain function (Quantitative EEG Cordance) during placebo lead-in and treatment outcomes in clinical trials for major depression. *American Journal of Psychiatry*, 163(8), 1426-1432
- [12] Bruder, G. E., Sedoruk, J. P., Stewart, J. W., McGrath, P. J., Quitkin, F. M., & Tenke, C. E. (2008). Electroencephalographic alpha measures predict therapeutic response to a selective serotonin reuptake inhibitor antidepressant: pre- and post-treatment findings. *Biological Psychiatry*, 63(12), 1171-1177.
- [13] Demos JN. *Getting started with neurofeedback*: WW Norton & Company; 2005.3-22
- [14] Serman, M. B. and Kaiser, D. (2001). Comodulation: A new QEEG analysis metric for assessment of structural and functional disorders of the central nervous system. *Journal of Neurotherap.* 73. – 83 .4 (3), .

- [15] Hammond, D. (2004). Neurofeedback treatment of depression with the Roshi. *Journal of Neurotherapy*, 4(2), 45-56
- [16] Brown, v.s. (2000) the role of 40 hz activity and the journal of neurotherapy, 4(2), 100-101.
- [17] Baehr, E., Rosenfeld, J. P. and Baehr, R. (1997). The clinical use of an alpha asymmetry neurofeedback protocol in the treatment of depression: Two case studies. *Journal of Neurotherapy*, 293, 10 – 23.
- [18] Baehr, E., Rosenfeld, J. P., Baehr, R. and Earnest, C. (1999). Clinical use of an alpha asymmetry neurofeedback protocol in the treatment of mood disorders. In *Introduction to Quantitative EEG and Neurofeedback* (J. R. Evans and A. Arbarbanel, eds), pp. 181 – 203. New York: Academic Press.
- [19] Baehr, E. (2002). Frontal Asymmetry and Brief Mood Shifts in Normal and Depressed Subjects. Unpublished paper, Society for Neuronal Regulation, 10th Annual Conference. Scottsdale, AZ.
- [20] Rosenfeld, J. P., Baehr, E., Baehr, R., Gottlieb, I. H. and Ranganath, C. (1996). Preliminary evidence that daily changes in frontal alpha asymmetry correlate with changes in affect in therapy sessions. *International Journal of Psychophysiology*, 23, 137 – 141.
- [21]. 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 – 33.
- [22] Davidson, R. J., Abercrombie, H., Nischke, J. B. and Putnam, K. (1999a). Regional brain function, emotion and disorders of emotion. *Current Opinion in Neurobiology*, 9, 228 – 234.
- [23] Gotlib, I. H., Ranganath, C. and Rosenfeld, J. P. (1998). Frontal EEG alpha asymmetry, depression, and cognitive functioning. *Cognition and Emotion*, 12, 449 – 478.
- [24] Tomarken, A. J., Diehter, G. S., Garber, J. and Simien, C. (2004). Resting frontal brain activity linkages to maternal depression and socio-economic status among adolescents. *Biological Psychology*, 67, 77 – 102.
- [25] Dawson, G., Grofer Klinger, L., Panagiotides, H., Spieker, S. and Frey, K. (1992). Infants of mothers with depressed symptoms: Electroencephalographic and behavioral findings related to attachment status. *Development & Psychopathology*, 4, 67 – 80.
- [26] Field, T., Fox, N., Pickens, J. and Nawrocki, R. (1995). Relative right frontal EEG activation in 3- to 6- month-old infants of “depressed” mothers. *Developmental Psychology*, 26, 7 – 14.
- [27] Jones, N. A., Field, T., Fox, N. A., Lundy, B. and Davalos, M. (1997). EEG activation in 1-month-old infants of depressed mothers. *Developmental Psychopathology*, 9, 491 – 505.
- [28] Smit, D. J. A., Posthuma, D., Boomsma, D. I. and DeGeus, E. J. C. (2006). The relation between frontal EEG asymmetry and the risk for anxiety and depression. *Biological Psychology*, 74 (1), 26 – 33.
- [29] Anokhin, A. P., Heath, A. C. and Myers, E. (2006). Genetic and environmental influences on frontal EEG asymmetry: A twin study. *Biological Psychiatry*, 71 (3), 295 – 298.
- [30] Vuga, M., Fox, N. A., Cohn, J. F., George, C. J., Lenenstein, R. M. and Kovacs, M. (2006). Long-term stability of frontal electroencephalographic asymmetry in adults with a history of depression and controls. *International Journal of Psychophysiology*, 59 (2), 107 – 115.

- [31] Allen , J. J. B. , Urry , H. L., Hitt , S. K. and Coan , J. A. ( 2004a ) . The stability of resting frontal electroencephalographic asymmetry in depression . *Psychophysiology* , 41 , 269 – 280.
- [32] Askew, J. H. (2001). The diagnosis of depression using psychometric instruments and quantitative measures of electroencephalographic activity. Unpublished doctoral dissertation, University of Tennessee.
- [33] Davidson , R. J. ( 1994 ) . Asymmetric brain function, affective style and psychopathology: The role of experience and plasticity . *Development and Psychopathology* , 6 , 741 – 758 .
- [34] Deldin , P. J. and Chiu , P. ( 2005 ) . Cognitive restructuring and EEG in major depression . *Biological Psychology* , 70 ( 3 ) , 41 – 151 .
- [35] Armitage , R. , Hudson , A. , Trivedi , M. and Rush , A. J. ( 1995 ) . Sex differences in the distribution of EEG frequencies during sleep: Unipolar depressed outpatients . *Journal of Affective Disorders* , 34 , 121 – 129 .
- [36] Joseph , R. ( 1999 ) . Frontal lobe psychopathology: Mania, depression, confabulation, catatonia, perseveration, obsessive compulsions, and schizophrenia . *Psychiatry* , 62 , 138 – 172 .
- [37] Schwartz , G. E., Davidson , R. J. and Maer , F. ( 1975 ) . Right hemisphere lateralization for emotion in the human brain: Interactions with cognition . *Science* , 190 , 286 – 288 .
- [38] Tenke , C. E., Bruder , G. E., Towey , J. P. , Leite , P. and Sittis , J. ( 1993 ) . Correspondence between ERP and behavioral asymmetries for complex tones . *Psychophysiology* , 30 , 62 – 70 .
- [39] Jaeger , J. , Borod , J. C. and Peselow , E. ( 1987 ) . Depressed patients have atypical biases in perception of emotional faces . *Journal of Abnormal Psychology* , 96 , 321 – 324 .
- [40] Bruder , G. E., Tenke , C. E., Warner , V. and Weissman , M. M. ( 2007 ) . Grandchildren at high and low risk for depression differ in EEG measures of regional brain asymmetry . *Biological Psychiatry* , 62 .
- [41] Hammond , D. C. ( 2005b ) . Neurofeedback treatment of depression and anxiety . *Journal of Adult Development* , 12 ( 2/3 ) , 131 – 137 .
- [42] Peniston , E. G. , Marrinan , D.A., Deming , W. A. and Kulkosky , P. J. ( 1993 ) . EEG alpha–theta brainwave synchronization in Vietnam theater veterans with combat-related post-traumatic stress disorder and alcohol abuse . *Advances in Medical Psychotherapy* , 6 , 37 – 50 .
- [43] Saxby , E. and Peniston , E. G. ( 1995 ) . Alpha–theta brainwave neurofeedback training: An effective treatment for male and female alcoholics with depressive symptoms . *Journal of Clinical Psychology* , 51 ( 5 ) , 685 – 693.
- [44] Frank H. Duffy, MD' Associate Editor for Neurology, *Clinical EEG Journal*, 2000
- [45] A. Martin Wuttke' Neurofeedback therapy' Handbook for the practitioner' Part Number M-oo1 - Version 1.0 - August 1, 2004.
- [46] A. Martin Wuttke' Neurofeedback therapy' Handbook for the practitioner' Part Number M-oo1 - Version 1.0 - August 1, 2004
- [47] Antonuccio , D. O. , Danton , W. G. , DeNelsky , G. Y. , Greenberg , R. P. and Gordon , J. S. ( 1999 ) . Raising questions about antidepressants . *Psychotherapy & Psychosomatics* , 68 , 3 – 14 .



- [48] Greenberg , R. P. , Bornstein , R. F. , Greenberg , M. D. and Fisher , S. ( 1992) . A meta-analysis of antidepressant outcome under “blinder ” conditions . *Journal of Consulting & Clinical Psychology* , 60 , 664 – 669 .
- [49] Hammond , D. C. ( 2007a ) . Hypnosis, placebos, and systematic research bias in biological psychiatry . *American Journal of Clinical Hypnosis* , 50 ( 1) , 37 – 47 .
- [50] Kirsch , I. , Moore , T. J. , Scoboria , A. and Nicholls , S. S. ( 2002) . The emperor’s new drugs: An analysis of antidepressant medication data submitted to the U.S. Food and Drug Administration . *Prevention & Treatment* , 5 . Article 23. <http://www.journals.apa.org/prevention/>
- [52] Moncrieff , J. ( 2001) . Are antidepressants overrated? A review of methodological problems in antidepressant trials . *Journal of Nervous and Mental Disease* , 189 ( 5) , 288 – 295 .
- [53] Elkin , I. , Shea , T. , Watkins , J. T. , et al. ( 1989) . National Institute of Mental Health treatment of depression collaborative research program: General effectiveness of treatments . *Archives of General Psychiatry* , 46 , 971 – 982 .
- [54] Hollon , S. D. , Shelton , R. C. and Loosen , P. T. ( 1991) . Cognitive therapy and pharmacotherapy for depression . *Journal of Consulting and Clinical Psychology* , 59 , 88 – 99 .