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The Integration of Psychodynamic Theories and Biological Aspects in the Development of Anxiety and Anxiety Disorders

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Additional information is available at the end of the chapter

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Abstract

Anxiety is a normal human reaction to stressful and threatening events in their surroundings, but if it does not correlate with inducible stimulus with respect to intensity and duration and if it permanently impairs a person's ability to function normally, then we are dealing with pathological anxiety, that is to say, a symptom of one of the anxiety disorders classified in 9th revision of the International Classification of Diseases (ICD-10), or in American Psychiatric Association, DSM-IV classification. We may consider the aetiology of anxiety from psychodynamic, biological and neuroscientific aspect. Finally, certain genes have been located, the variability of which in the expression of "visceral brain" neurons modulates remembrance of fear and somatic reactions to anxiety. These genes also represent the potential focal points for future pharmacotherapeutic solutions for the treatment of anxiety and anxiety-connected psychic disorders.

Keywords: anxiety, aetiology, psychodynamics, neuroscience, biological aspects, neuroscientific aspects

1. Introduction

Throughout evolution each species has been armed with certain defence mechanisms to help the organisms cope with internal or external threats. These defence mechanisms are highly individual from one species to another.

The term anxiety can also be used to describe normal reactions to danger, which can then be differentiated from fear because anxiety is much weaker and lasts longer than fear; however,

anxiety can also be a pathological state of mind, because constant fear of unknown, undefined or unreal comes from inside the person.

Fear is the best activator of stress response because it has a vast array of peripheral and neuroendocrine changes which help us survive. Fear also impacts the brain, altering the way emotional and cognitive systems process information [1]. Even though all these mechanisms help us when needed, they are a later threat because of chronic activation which when triggered can cause post-traumatic stress disorder (PTSD) and even some physical symptoms [2].

Fear is an emotion which has a central role in evolution and natural selection. It is also one of few emotions which we share even with lower mammals. Because of this connection, it is easy to research fear and its effect on animals and compare the result with human behaviour.

Psychoanalysis provided a highly influential theory for the development of anxiety also showing the way anxiety was maintained. Psychodynamic notions of anxiety have since lost favour to the more parsimonious and empirically grounded behavioural theories. These theories have a strong ground in treatment of anxiety. On the other hand, the main problem of behavioural theories is that they cannot be applied to explain all cases of anxiety.

Every person throughout their lifespan has experienced the feeling of anxiety. Fear and anxiety have become such strong synonyms that people are having a hard time differentiating between the two. According to Freud, anxiety is the fear of unknown, when a person cannot describe what they are afraid of. When a person is able to describe exactly what they are afraid of, the term fear is used.

Emotions affect our behaviour; every day we experience a lot of emotions most of which do not have a great effect on our behaviour. But when the intensity of emotions shifts up, they can have a huge impact on our behaviour [3].

The primary emotions are anger, fear, pleasure, sadness and disgust [4]. Emotions help us find balance in our surrounding, and they also help with adapting our behaviour to the situation we are faced with. They can be divided into two groups: positive and negative. Negative emotions express an attempt or intention to exclude. Strengthening one's own position at the expense of others. Keeping bad stuff away, destroying what is perceived as a threat. Negative emotions are fueled by an underlying fear of the unknown, a fear of the actions of others, and a need to control them or stop them to avoid being harmed.

Positive emotions express an attempt or an intention to include. Taking the whole into consideration. Working on learning more viewpoints, interacting more with others, enjoying making things better. Positive emotions are fueled by an underlying desire for enjoyment and unity.

Anxiety is a sign of life and experience, and it does not always have to be a part of an illness. Anxiety is a psychological, physiological and behavioural state induced in animals and humans by a threat to well-being or survival, either actual or potential. In biology, the term anxiety is associated with awareness and stimuli of the conscious on danger. When a living organism faces danger, it reacts in only two ways: fight and flight. When a human faces danger, the first sign is anxiety. Anxiety can stimulate a human being to fight, retreat, exhibit hyper-

activity and awareness. If anxiety activates awareness and readiness, then the person can overcome all the difficulties that they are faced with, in order to achieve their aim. This type of anxiety is called normal, and we benefit from normal anxiety. It helps us become aware and ready for all the problems we have to deal with, and it also awakes a sense of satisfaction and joy when the task is complete [5].

To understand anxiety on the biological level, it is necessary to be familiar with the physiology and anatomy of the brain and neurotransmitters. Pharmacological research shows us that in anxiety there is hyperactivity of the noradrenergic nuclei, locus coeruleus and noradrenergic pathways. Also it was noticed that lower levels of serotonin and gamma-hydroxybutyric acid (GABA) neurons cause anxiety disorders. Technical breakthroughs help us pinpoint certain regions and structures of the central nervous system which are responsible for conditioning and integrating the feeling of fear and producing an adequate response. These neuroscientific insights are based on the Pavlovian (classical) conditioning. Modern neuroimaging techniques have located Papez circuit, limbic system and the complex of amygdala nuclei, which is the primary structure responsible for the processes of learning and fear conditioning [6].

2. Psychodynamic aspects of anxiety

Glen Gabbard provides a modern understanding of the psychodynamic aspects of anxiety disorder [7]. One can only admire the insights he derives from Freud's seminal work on the subject, *Inhibitions, Symptoms and Anxiety* [8]. Many of Freud's ideas are now being proven correct by neuroscientists. It has been proven that there is a memory system for anxiety responses in the amygdala that processes stimuli without any reference to conscious memory. Gabbard points out that panic attacks may not just happen without any reason but are more likely to happen because of stress memory activation which is specific to each patient. He also discusses panic disorders which find cause in childhood. These can be compared to scientific finding on animals which were separated in early life from their parents showing later in life as an anxiety prone adult animal [7].

The creator of the psychoanalytic paradigm is Sigmund Freud. The psychoanalytic model, which is based on Freud's works and later expanded by his followers and other psychoanalysts of the modern times, now includes the ideas of all those who reviewed and tried to change his mind. The model is based on the assumption that human behaviour is determined by intrapsychical impulses, desires, motives and conflicts.

For Freud, there are three stages in the development of the concept of anxiety. The first period (works between 1893 and 1895) is in connection with the neurosis of fear and its relationship with sex life [9]. In the second period (between 1909 and 1917), Freud elaborated the relationship between anxiety and repressed libido [10–13], and in the third period (between 1926 and 1932), he discusses the relationship of anxiety with the mental apparatus [8, 14, 15].

In his major work, "Inhibition, symptom and anxiety", Freud gave his final views on the theory of anxiety [8].

The alarm signal is a form of anxiety that occurs at provoking some old situations of danger and is a manifestation of the ego, which is used in order to start defensive measures against drives coming from the id or its representations. Defence mechanisms of the ego, no matter how much unskilled they are, condition the symbolic activity of a similar opinion.

Freud elaborated the relationship between anxiety, pain and grief for the object. Pain is a personal reaction to the loss of an object, while anxiety is a reaction to the danger that implies that loss and, through shifting, a reaction to the threat of the loss. The loss of the object causes the pain to penetrate insurmountable amount of excitation to the ego, which is experiencing anxiety because of fear of helplessness. In order to prevent this occurrence of pain and fear of helplessness, anxiety signal precedes the disaster and warns ego to employ defensive measures that will be able to cope with danger [14].

To sum up, this theory is based on the concept of danger and “dangerous situations” and is based on two main pillars: (1) anxiety occurs as a response signal for the purpose of preparing a person to a dangerous situation and (2) the ego is the centre of anxiety and sometimes can even be the cause, whether it is repeated anxiety for their own account (eroticized anxiety) or as a signal of impending danger instinctively. Functional anxiety is determined by two aspects:

1. historical aspect, since the anxiety as a signal represents a repetition of infantile anxiety experiences, which it plays, creating at the same time some protection from the return of the repressed;
2. symbolic aspect, since it is a function of anxiety at the same time and symbolic because it represents in itself symbolically a dangerous situation.

As a universal reaction of the human being, anxiety frequently occurs as a situational conditioned disorder, and only further observation usually allows us to distinguish normal, neurotic and psychotic anxiety.

2.1. Object relations theory

Beside Freud’s theory, in the later development of psychoanalysis, a second significant theory arises—object relations theory. Without this theory, we would not be able to explain the necessity to communicate with other individuals [16–18].

Object relations theory stresses the importance of the earliest interactions with other people from our surrounding as a building stone in the construction of the id, ego and superego. More specifically, from birth onwards, our relations with others are highly influenced by strong emotions which form specific memories and integrate us in the society by our behaviour. Positive experience helps us form ourselves as mature individuals, and the same goes for negative experience. Each experience is individually processed and stored in our memory [19].

Simply put, we can say that we are born with a certain capacity of perception, memory and establishing a representation of our perception and to gradually develop symbolic thought, or the capacity of abstract thinking and intelligence. Let us imagine that the ego is a computer that absorbs, stores and integrates information and learns how to sort and classify the specific pattern of priorities. It also has to distinguish the important from the unimportant, good from

bad. Gradually, we learn and differentiate what is inside from what is outside, and so, overtime, our inner world is built. The largest part of it remains in the unconscious memory, or preconscious area (reservoir of information that we do not use all the time, but can access), while only a small part goes into the realm of conscious. The rest is stored in an even deeper level, the dynamic unconscious or id [20].

3. Biological aspects of anxiety

Neurochemistry is the only way to understand and cure anxiety disorders. Pharmacological treatment is based on neurochemistry. Neurons communicate with each other with neurotransmitters; at least three neurotransmitters can be associated with anxiety. In recent studies, there is a sign that certain large neurotransmitters also play a role in anxiety and they are called neuropeptides. These neuropeptides are not only used in communication between neurons, but can also regulate them and cause hormonal misbalance. This is because of their chemical structure which biologically has a strong effect on the human body. Research on neuropeptides is still at its beginnings, so it will not be mentioned any more through this chapter because there is no clinical finding which can be used to help us. For now, we will base ourselves on gamma-hydroxybutyric acid (GABA). When this neurotransmitter's balance is disrupted in the body, we can note a relationship with anxiety. Benzodiazepines, drugs that exert their effect through GABA receptors, are used to help people suffering from acute anxiety [21].

Serotonin is greatly important in anxiety. It is a monoamine neurotransmitter involved in controlling a wide range of behaviours by affecting the neural system. These behaviours include emotions which are connected with fear and anxiety. All serotonin neurons emerge from the raphe nuclei. The raphe nuclei have a part called median raphe which acts as a connection with the septo hippocampal system as well as the cortex. Because of these connections, it has an important role in emotional cognition [22].

Studies on animals have successfully proved the connection between different neurotransmitters and anxiety. These types of studies are called knockout studies, because a certain receptor in the animal genetic code is knocked out. Animals which have had their serotonin re-uptake transporters knocked out show in case studies abnormal response to fear and anxiety in a number of behavioural conflict tests. This confirms the role of this receptor in modulating anxiety [23].

On the other hand, animals which had their serotonin receptor knocked out showed an increased heart rate and anxiety in a large variety of tasks such as eating and locomotion. Foot shock on animals with the serotonin receptor removed showed longer freezing and increased tachycardia.

Together these data show the importance of serotonin in modulating the levels of fear and anxiety in our brain.

As stated above, the median raphe nuclei (MRN) provide the necessary input to the neural circuits within the brain causing fear and anxiety modulation. MRN lesion showed us that short-term fear is independent from MRN, while long-term fear depends on MRN [24].

In human research, it was found that people with short serotonin transport acquired faster fear than people with longer serotonin transport. All these findings are consistent with the findings in animal models.

However, the best proof of the function of the serotonergic system in fear and anxiety is the pharmacological evidence. Drugs that change the function of serotonin have beneficial effects on various forms of anxiety. The best pharmacological treatment of anxiety is serotonin re-uptake inhibitors which allow greater levels of serotonin to accumulate and in that way help in treatment of anxiety [22].

Noradrenalin is also an important neurotransmitter in anxiety. Neurons which carry noradrenalin rise from the locus coeruleus (LC), and these are also a centre associated with warnings or alarms. The LC secretes directly into the brain causing immediate response. Increased levels of noradrenalin cause higher levels of anxiety. In pharmacology, noradrenalin blockers lower the levels of noradrenalin and do the same to a patient as do serotonin re-uptake blockers. Among adults, agents that alter noradrenergic functioning are powerful anxiolytics. Similarly, agents, such as yohimbine, that increase firing of the locus coeruleus are potent anxiogenic compounds [25].

3.1. The visceral brain

At the beginning of the twentieth-century, scientists have identified the hypothalamus as a key structure in the control of the autonomic nervous system [26]. Based on these assumptions, there was a so-called hypothalamic theory of emotion, which contained three main strands: first—the hypothalamus in a way valued events in our environment; second—the expression of emotional response is proportional to the outbreak of the pulses from the hypothalamus in the brain stem; and the third—hypothalamic projections into the cortex allow conscious perception of emotion [27, 28]. American neurologist James Papez in 1937 proposed that the circuit connecting the hypothalamus to the limbic lobe was the basis for emotional experiences. Papez circuit (or medial limbic circuit) is a neural circuit for the control of emotional expression [29]. Papez theory was later reconceptualized and expanded by an American neuroscientist Paul D. MacLean [30]; MacLean redefined the circuit as the “visceral brain”, which consisted of the limbic lobe and its major connections in the forebrain—hypothalamus, amygdala and septum. Overtime, the concept of a forebrain circuit for the control of emotional expression has been modified to include the prefrontal cortex. The amygdale, complex nuclei (central, medial and cortical basolateral, which is still divided on the lateral, basal and accessory basal nucleus) located in the medial temporal lobe was also part of MacLean’s theory of the limbic system, but not of any importance, until the 1956 publication by the British psychologist Lawrence Weiskrantz. In this work, he tried to explain the causes of clinical manifestations of the Kluver-Bucy syndrome (a set of behavioural disorders due to the bilateral damage to the temporal lobes usually after temporal lobotomy).

Weiskrantz suggested that the main cause of such a clinical picture is just the damaged or removed amygdala. For years after its publication, numerous studies have focused on the amygdala function and its relationship with other parts of the central nervous system; however, greater progress in understanding the role of the functions of the amygdala and its link with emotions, including fear, has been in the 70s and 80s, when the scientists returned Pavlov method of classical conditioning [31].

3.2. The amygdala in fear conditioning process

Evolutionary man has developed a number of coping mechanisms, some of which are based on the ability to anticipate and avoid dangerous, potentially life-threatening situations. Neural circuits involved in the processes of memory and processing emotions play a key role in these mechanisms; each new stimulus is incorporated and based on the nature of that stimulus and appropriate behavioural, psychological and somatic reaction that, in addition to character, must match the intensity of the resulting stimulus. The scientists used this knowledge in research of pathways related to emotions, while the main tool used was the simple method of classical conditioning, similar to that used by Pavlov in his experiments. In it he explored the mechanisms by which animals predict a dangerous or a pleasant event. In these studies, emotionally related stimulus (unconditioned stimulus), which was the unpleasant, threatening, associated with fear, or rewarding, and reward-related event, preceded by an emotionally neutral (s conditioning) stimulus, mainly sound or light pulse [32–35].

After a period of learning (conditioning), animals are using these conditioned stimuli to predict the magnitude and time of occurrence of the desired or undesired events. In further analyses of conditioned fear, the researchers were able to map the entire neural path from the input of sensory stimuli to output behavioural responses [36–38]. Studies have shown that just amygdala, a complex structure of the temporal lobes, plays a key role in the coordination of behavioural and psychosomatic reactions associated with fear [37–42]. When sound (neutral stimulus) arrives to organ of Corti to signal danger, impulse to the amygdala comes from two ways. The first is through the thalamus, through nucleus corpora geniculata medial, responsible for fast processing of sound information that allows quick preparation of the body to potentially dangerous and threatening event before sound region of the cerebral cortex of the temporal lobe processes the audio information and discern whether it is a real danger or “false alarm” [40]. The second path, which leads from the sound region temporal cortex to the amygdala, is slower, but also more complex because it integrates functions of the higher centres of the central nervous system. Electrophysiological studies have shown that conditioned stimuli that preceded those unfavourable fear conditioning, then the fear associated, strengthens synaptic connections between the auditory thalamus and lateral nuclei of the amygdala. The stimulus is passed into the central amygdala nuclei that project axons to different regions of the hypothalamus, activating the sympathetic system and inducing the secretion of stress hormones such as CRH (corticotropin-releasing hormone). The secretion of CRH in the periventricular nuclei of the hypothalamus results in an increased secretion of adrenocorticotrophic hormone (ACTH) in adenohypophysis and consequently increases the secretion of glucocorticoids from the adrenal cortex, by which the body leads to a catabolic state. The central

nucleus of the amygdala plays an important role in emotional behavior. Nociceptive inputs through the spino-parabrachio-amygdala pathway probably contribute to pain-induced changes in affective behavior, and the projections of the amygdala to the PAG-RVM (rostromedial medulla) circuitry may be involved in mediating the influence of emotions on pain. Stressful situations like physical exercise, exposure to extreme temperatures, fight, fear and pain may induce a decrease in pain sensitivity. However, if these mechanisms are deregulated, it creates a strong etiological factor backing the development of anxiety disorders [43].

4. Case study: example of connection psychodynamic and neuroscientific approach to treatment of anxiety

In recent years, there are numerous mental disorders as the consequences of extreme stress; they are the official classifications of anxiety and PTSD. Post-traumatic stress disorder (PTSD) develops in some people after exposure to an extreme stress or traumatic event. It is characterized by three distinct types of symptoms consisting of re-experiencing of the event, avoidance of reminders of the event and hyper arousal, which continue for a substantial period of time. These symptoms indicate dysfunction in acquisition and extinction of fear conditioning, and the hippocampus has been the most consistently implicated brain region.

Our clinical experience has shown that hyper arousal symptoms and especially those associated with impulsive aggressiveness, such as hyperirritability, hyper excitability, hypersensitivity, aggressive acting outs, outbursts of anger, present one of the dominant disturbances for chronic PTSD patients. This kind of symptoms can be understood in the perspective of loss of the affective modulation, which can explain the PTSD patients' lack of capacity to use effect states as cues to attend to incoming information. The elevated—arousal—state is likely to precipitate flight-or-fight reactions in these patients, so they often go immediately from stimulus to response without psychologically assessing the meaning of the event. This makes them prone to freeze or, alternatively, to overreact in response to minor provocations [44].

The aim of our study was to assess possible changes in brain activation in PTSD patients after 2 years of intensive psychotherapy in form of closed-door group analysis in a heterogeneous group specially designed in our clinic for treatment of PTSD patients.

4.1. Method and participants

Participants in our study were selected from all PTSD patients treated at the Clinic for Psychological Medicine, University of Zagreb Medical School, in the period from April to July 2001. We selected 25 participants from the total of 82 patients treated in the day hospital unit at the clinic over the period. All the participants in the study, as the most of our patients, were Croatian war (1991–1995) veterans who actively participated in the combat and were exposed to multiple traumatic experiences such as witnessing the death of the fellow soldiers and sudden air-rides or artillery attacks. The patients who experienced other kinds of traumatic

experiences such as prisoners of war and concentration camp detainees were not included in this study (because they have difficult symptoms, and they need psychopharmacological therapy, and other reason was ethical because we do not want to retraumatize these vulnerable patients). They all signed informed consent to participate in the study.

The group evaluation was performed over 2-week group psychotherapy, as described above, encompassing four group sessions and consisted of monitoring of patients' symptom manifestation. The same co-therapist couple performed the selection for all the participants in the study. Individual evaluation was organized in the form of Structured Clinical Interview for DSM-IV (SCID-CV) [45]. The interview was held during the first week of patients' treatment at the Clinic and was conducted by the same therapist for all the participants. All therapists conducting the selection were clinicians with at least 3 years of experience in working with psych traumatized patients. Group therapists were not informed of the evaluation performed by the individual therapist, conversely.

All patients selected for the investigation had chronic, combat-related PTSD with severe hyper arousal symptoms, impulsive aggressiveness and no other Axis-I and/or Axis-II diagnosis in accordance with DSM-IV criteria (American Psychiatric Association, 1994) [46]. Severity of hyper arousal symptoms was assessed on the basis of anamnesis and heteroanamnesis and also on the basis of symptoms they manifested in group therapy and individual psychiatric interview. Only the patients who continuously over the two-week observation presented all hyper arousal symptoms as defined in DSM-IV, and impulsive aggressiveness symptoms were selected for the study. Impulsive aggressiveness symptoms were defined as: hyperirritability, aggressive acting outs and outbursts of anger. Only patients who manifested these symptoms in both individual interviews and all four group sessions over the observation period, and who had positive anamnesis and heteroanamnesis for hyper arousal symptoms in the month preceding the hospitalization, were included in the study. This selection design had to be implemented because no psychometric instrument for assessing hyper arousal and/or impulsive aggressiveness symptoms as such, qualitatively or preferably quantitatively, was available in Croatia.

The patients selected for the study received no other than benzodiazepine pharmacotherapy for at least 6 months preceding the study. Also, 4 weeks before the study entry the patients received no psychotropic medication at all. After the psychiatric evaluation, neurological examination was conducted for all participants to exclude those with possible neurological comorbid conditions, head trauma or loss of consciousness in the year preceding the study.

All patients included in the study were male, right-handed, of similar age (mean = 42 years, SD = 2) and had no history of alcohol or drug abuse or dependence.

Single photon emission computed tomography (SPECT) using Tc^{99m}-ECD functional brain imaging was organized in the week after the two-week observation period immediately after the first group session in that week to establish possible alterations in regional cerebral blood flow (rCBF). The procedure was organized in the following way: patients who manifested at least one and no more than three aggressive acting outs during the pre-SPECT session were asked to remember the specific situation in the group that provoked the last aggressive acting

out. The ultimate of no more than three aggressive acting outs during the designated session was a preset criteria to enable remembering of the specific situation that provoked the last acting out, but was not used because none of our subjects actually manifested more than three aggressive acting outs during the pre-SPECT session. The subjects were asked to try to remember the exact situation in the group that provoked their aggressive acting out and to describe it, and they were included in the further procedure. Once again, all of our subjects fulfilled these criteria and all declared very distressed when remembering the situation. Following this, subjects were introduced into SPECT procedure, and the scans were obtained in 50–60 min after the end of the session. Once again, immediately before the SPECT brain scan was made, each subject was asked to remember the situation that provoked his last aggressive acting out in the pre-SPECT session.

SPECT scans were started 20 min after the administration of 740 MBq of ^{99m}Tc -ECD in resting state (“white noise” environment, without any auditory or visual combat stimuli provocation). Measurements were made with IRIX triple-headed rotating scintillation gamma camera fitted with high-resolution collimators, using a 360° circular orbit and step and shoot mode (20-s image each 3°), so that the acquisition lasted 40 min. Within-subject correlated slices were obtained.

Following regions of interest (ROI) were considered: temporal, frontal, orbitofrontal and occipital cortices, cingulate gyrus, amygdala, thalamus, basal ganglia (caudate, putamen, ventral basal ganglia) and cerebellum. The irregular ROIs were outlined in the right hemisphere and mirrored in the left. The same investigator, blinded for all clinical data, placed ROIs. For each ROI, mean counts per pixel in two consecutive slices were averaged to minimize partial volume effects. In each patient, studies were normalized to the mean whole-brain uptake, so relative hypo/hyper perfusion was established for each ROI. The percentage of asymmetry between two homologous regions was calculated as $200 \times (\text{right-left}/\text{right+left})$, where right and left indicate mean average counts per pixel in the ROIs of the respective hemisphere. The negative values indicate thus lower perfusion in the right hemisphere. Percentages of interhemispheric asymmetry between homologous brain regions were used to identify abnormalities.

4.2. Therapy

Patients were subdued to a group psychotherapy used in the form of closed-door group analysis with once a week 90-min session rate, focusing on patients’ interpersonal and social relations and communication, specially designed in our clinic for treatment of PTSD patients. The group consisted of 10 members: six men (Croatian war veterans) and four women (partners of the war veterans, who were not related to the men in the group). During the psychotherapy period, the patients consumed no pharmacotherapy, except anxiolytic (benzodiazepine) therapy over the first 3 weeks of the therapy in order to alleviate acute symptoms and enable the psychotherapeutic process.

People whose reactions to early traumas have become integrated into the totality of their personalities are apt to repeat aspects of the trauma and defensive reactions to it in their relationships with other people. In heterogeneous group, transference-oriented trauma

groups, the social expressions of those trauma-related affects and cognitive schemata are inevitably expressed on a social level and became readily observable.

Long-term heterogeneous groups can be particularly helpful to people with previous individual therapy, where they had an opportunity to develop a language in which they can identify their feelings and a basic curiosity about how they may contribute to their problems in interpersonal relationships themselves. Patients “fitting” for the group analysis should have the ability to observe their own problems, the ability to react emotionally as well as a certain capacity to save somebody else’s emotions and the capacity for insight [47].

The therapeutic environment should be created in that way to enable individuals who are similar in some way to understand and feel with others, but in the same time different enough so they could watch and help other group members from different perspectives and positions. The best group composition is if the members are similar in terms of ego development and different in terms of interpersonal style. Groups that focus on the relationships between the members allow new losses to be experienced, as object losses with concomitant grief and sadness, rather than with narcissistic injuries accompanied by feelings of helplessness, numbing and vengeful rage. As heterogeneous trauma groups mature, they gradually make that transition. When that happens, the group members start getting the full benefit of the strengths that traumatized persons have developed to survive catastrophic trauma.

4.3. Results

Based on the studies of normal subjects, it is well documented that the normal cerebral blood distribution is bilaterally symmetric, with percentages of interhemispheric asymmetry never exceeding 12% for any brain region [48, 49]. In our study, SPECT brain scans at the beginning of the psychotherapeutic treatment, with two-week period between, showed unilateral (dominant brain hemisphere) 20% or more increase in the regional blood flow (rCBF) in the projection area of ventral basal ganglia (VBG).

After 2 years of group psychotherapy, PTSD patient’s symptoms diminished, and they regained adequate impulse control. Control SPECT scans, of the same group of subjects, were performed in two separate occasions with 3-week period in-between. No differences from normal pattern were found. Because of that reason, we do not need control group, because subjects acted as their own control (before and after group psychotherapy).

5. Conclusion: the integration knowledge of psychodynamics and biological aspects in treatment of anxiety disorder

In our study, we found unilaterally increased perfusion in the projection area of ventral basal ganglia (VBG) in the dominant brain hemisphere, in our PTSD subjects with severe hyper arousal symptoms and impulsive aggressiveness, and after 2 year of group psychotherapy, our patients show no differences from normal pattern. Possible conclusion is that SPECT findings are associated with the symptoms of PTSD. They receded together with the clinical manifes-

tation of symptoms, so it could be used as neurobiological indicator in order to make psychotherapeutic intervention focused on symptoms and clinical remission yet neurobiologically verified, as in our study.

Trauma is biochemically encoded into the brain in a variety of ways, including changes in the availability and effects of neurotransmitters and neuromodulators.

Neuroanatomical encoding occurs through changes in structures like the hippocampus, through coordination and integration of neural network functioning. The hyper arousal symptoms and persistent re-experiencing of the traumatic event suggest abnormalities in emotion and memory regulation implicating thus limbic brain regions as possibly associated with the disorder.

As scanning techniques become more precise and the hardware more affordable, they will without doubt become incorporated into treatment of PTSD and other disorders. As a part of initial assessment, they could help therapists pinpoint areas of neural activation and inhibition.

Treatment planning will come to include specific psychotherapeutic and pharmacological intervention to enhance growth and integration of affected networks.

Regular scans during the course of therapy may someday replace psychological tests as ways of fine-tuning the therapeutic process and measuring treatment success. The psychiatrist must be thought to think about the whole person first, to appreciate that each one is interesting and unique, not simply a composite of symptoms that are used to make an ICD or DSM diagnosis and provide treatment according to a standard algorithm.

It is possible to conclude that SPECT findings are associated with the symptoms of PTSD.

They receded together with the clinical manifestation of symptoms, so it could be used as neurobiological indicator in order to make psychotherapeutical intervention focused on symptoms and clinical remission yet neurobiologically verified, like as in Seedat study [50]. Functional brain imaging has opened a window to the living human brain in the acts of motor tasks, experiencing symptoms, imagining a feared situation or telling a lie.

An examination of areas activated during different tasks has enhanced our understanding of which neural networks participate in various functions [51].

Recent neurobiological research has confirmed Freud's original observation that there are essentially two forms of anxiety: one largely determined by psychological issues and another driven by autonomous biological factors outside the realm of psychological content. Research of locus coeruleus has been productive in defining the biological dimensions of anxiety. Whereas Freud viewed the ego as the psychological seat of anxiety, modern neurobiological researchers have identified the locus coruleous as the biological seat of anxiety. Neural pathways enter this nucleolus from every level of the central nervous system, and efferent pathways lead to all the major psychological systems involved in anxiety.

The existence of a neural mechanism for anxiety does not, however, preclude the usefulness of psychotherapeutic techniques. Effective psychotherapy most likely results in long-term

structural and functional changes in the brain that are related to changes in the expression of genes.

Contemporary approaches to the treatment of mental disorders are not distinguished from older ones by emphasis on drugs as by an emphasis of effective treatment. Psychotherapy is fundamentally a learning process for its patients and as such is a way to rewire the brain. In this sense, psychotherapy ultimately uses biological mechanisms to treat mental illness. This does not mean, however, that psychotherapy involves learning, while drug therapy involves something else, like correction of genetically dictated chemical imbalance. Even if chemical imbalance was to account for mental disorders, the imbalance could result purely from environmental factors, like some intensely stressful experience, or from environmental events that trigger and amplify a genetic predisposition. The therapist's job, whether using drugs or not, is to restore mental well-being [52].

At the centre of psychotherapy is an understanding of mutual interweaving of nature and education, successful and unsuccessful development and its impact on the healthy functioning. When psychotherapy results in a reduction in severity of symptoms, the brain is, in some ways, changed [52].

Fundamental premise was "that any form of psychotherapy is successful to the degree to which it enhances positive experiential change and underlying neural networks, growth and integration". All forms of psychotherapy are successful to the extent they enhance change in the relevant neural circuits. The brain is an organ of adaptation and built by experience during development and rebuilt during psychotherapy. At the neural level, integration and communication of neurons are associated with feelings, cognition and behaviour. On the psychological level, integration is marked with the ability to experience important life situation to be dismissed with the inclusion of minimum defence.

From the perspective of neuroscience, psychotherapy can be understood as a special form of enriched environment created to facilitate the growth of neurons and the integration of neural connections. The therapeutic environment is, in fact, an environment tailored individually, according to the symptoms and the patient's needs [52].

Historically speaking, before the Freud's treatment of psychiatric disease was the area of neurology. Even Freud, who was a neurologist, believed that most psychiatric disorders have an organic substrate and anticipated application medication. Freud wrote in 1914: "We must be aware of the fact that our provisional ideas in psychology probably one day be based on organic substructures". In the article, "The draft of psychoanalysis" Freud says: "Maybe in the future we learn to direct, through chemical substances, influence the quantity of energy and its distribution in the mental apparatus" [53].

In the years that followed, the more emphasized the difference in the two approaches became, but its peak is experienced in the 50s when the current psychodynamic psychiatry equated with intellectual and better educated grouping of pharmacologically oriented psychiatrists. Neuroleptics and antidepressants, which were discovered in the late fifties, have not been as effective in the treatment, but research has increasingly intensified, and the drugs have become

more efficient, which resulted in the DSM-III-R classification of diseases that did not include psychodynamic knowledge in diagnosis mental illnesses [54].

Our neural web is determined by two types of effects: first one is genetics: this part is individual from person to person; and the second one is external factors: it includes the environment which moulds our genetics. This process of moulding genetics is to actually activate or inactivate certain genes that are not used causing parts of the neural network to stop working due to the inactivation. Moulding is dependent on multiple life style factors, like diet and exercise [54].

Synaptic connections and communications do not go away. Psychotherapy works in such a way that it uses the brains plasticity to cause forming of new synaptic connections which will be stronger than the earlier ones and in cause be used more. Psychotherapy can also inhibit the usage of undesired synaptic connections. Of course, for Stahl, this is ideally achieved through the synergy with drugs and psychotherapy. He also states that medication can help the facilitation process and enhance the health situation [55].

All this research of learning and the way we memorize does not just help us understand psychotherapy, but it also gives us a new perspective of anxiety disorders involving the inability to control fear, obsessions, compulsions and delusions. To end this paper, it should be stated that the greatest leap forward would be a better collaboration between neurobiologists and psychotherapists in trying to find links between neurobiology and psychotherapeutic processes [56].

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