



# Psychiatric Diseases and Anxiety: Issues, Challenges, Signs, Symptoms, Etiology, and Pathophysiology

**Correspondance Authors:** <sup>1</sup>Mr. Manish Kumar, <sup>2</sup>Prof. Satish Sharma

1. Research Scholar, Glocal university, Saharanpur U.P.
2. Professor, Glocal university, Saharanpur U.P.

## ABSTRACT

Psychiatric disorders are diseases that affect cognition, emotion, and behavioral control and substantially interfere both with the ability of children to learn and with the ability of adults to function in their families, at work, and in the broader society. Mental disorders tend to begin early in life and often run a chronic recurrent course. They are common in all countries where their prevalence has been examined. Because of the combination of high prevalence, early onset, persistence, and impairment, mental disorders make a major contribution to total disease burden. Although most of the burden attributable to mental disorders is disability related, premature mortality, especially from suicide, is not insignificant. This review was done to assess the burden of Psychiatric Diseases and to study the various issues and challenges at community level. World Health Organization estimated that mental and behavioural disorders account for about 12 percent of the global burden of diseases. In India the burden of mental and behavioural disorders ranged from 9.5 to 102 per 1000 population.

## 1. INTRODUCTION

Psychiatric Diseases have complex etiologies that involve interactions among multiple genetic and nongenetic risk factors. Gender is related to risk in many cases: males have higher rates of attention deficit hyperactivity disorder, autism, and substance use disorders; females have higher rates of major depressive disorder, most anxiety disorders, and eating disorders. Biochemical and morphological abnormalities of the brain associated with schizophrenia, autism, mood, and anxiety disorders are being identified using approaches such as postmortem analysis and noninvasive neuroimaging. Major worldwide efforts under way to identify risk conferring genes for mental disorders are proving challenging, but initial results are promising. Identifying the gene or genes causing or creating vulnerability for a disorder should help us understand what goes wrong in the brain to produce mental illness and should have a clinical effect by contributing to improved diagnostics and therapeutics. Burden of Psychiatric Diseases seen by the world is only a tip of iceberg. Various studies had

shown that the prevalence of mental disorders were high in females, elderly, disaster survivors, industrial workers, children, adolescent and those having chronic medical conditions. There is need to have better living conditions, political commitment, primary health care and women empowerment. Burden of Psychiatric Diseases had risen over last few decades. Mental health is a state of well-being in which the individual realizes his or her own abilities, can cope with the normal stresses of life, can work productively and is able to make a contribution to his or her community. WHO estimated that globally over 450 million people suffer from mental disorders. Currently mental and behavioural disorders account for about 12 percent of the global burden of diseases. This is likely to increase to 15 percent by 2020. Major proportions of mental disorders come from low and middle income countries.<sup>1</sup> There are lacunae in psychiatric epidemiology due to intricacy related to defining a case, sampling methodology, under reporting, stigma, lack of adequate funding and trained manpower and low priority of mental health in the health policy.<sup>2</sup>

The evolution in medical sector is tremendous as medical solution for every diseases right from simple fever to ill-fated diseases have been invented and are readily available. There are no limitations to government hospitals and free medicines. Almost all the diseases are treated and cured and hence a very few deaths are encountered every year due to diseases, i.e. failure of treatments. Curing of diseases depends upon individual's age that certain diseases could not be treated because of old age. Treatments for people who are born with abnormalities or grown immature cannot be done in certain cases because human body should be in a position to absorb and respond to the provided medicines and treatment. Many people are unaware that diseases arise out of Psychiatric Diseases, when affected due to stress or anxiety. It is due to chemical imbalance of the brain that forces the persons to behave indifferently. When the human mind is stress-free and in harmony, various diseases can be prevented and even the prolonged diseases can be cured within days. According to the World Health Organization,<sup>3</sup> anxiety disorders are burdensome "common Psychiatric Diseases" to communities. These prevalent disorders are not communicable and affect approximately one in every five individuals of the world population.<sup>4,5,6</sup> Anxiety disorders present an early onset, even during childhood. Their enduring waxing and waning course deeply affects patients' functionality and interpersonal relationships throughout the lifespan.<sup>7</sup> Most pathological anxiety (specific phobias, social anxiety, generalized anxiety, separation anxiety, obsessive-compulsive, and panic disorder) is underrecognized, and patients seek treatment in outpatient settings, either in medical or specialized mental health-care contexts.<sup>8</sup> However, anxiety disorders receive less attention from clinicians when compared with major mental disorders, such as psychotic conditions and substance use disorders that require hospitalization. Moreover, anxiety is less reported in the media than depression and suicide attempts, which reduces the help-seeking behaviors of patients suffering from anxiety. Most experts advocate either psychotherapy and/or pharmacotherapy for alleviating or controlling symptoms of anxiety. The combination of psychological treatment with psychotropic drugs is recommended for patients with severe cases of disabling anxiety. Traditionally, several talk therapies are subsumed as techniques of psychological treatment and have been recommended to handle different degrees of anxiety.<sup>9</sup> Well-accepted but not always efficacious modalities

of psychotherapy vary from psychoanalytic, cognitive-behavioral, interpersonal, supportive, and group therapy to brief therapy.

## 2. ISSUE AND CHALLENGES OF PSYCHIATRIC DISEASES

Most strongly associated factors with Psychiatric Diseases are deprivation and poverty. Individuals with lower levels of education, low household income, lack of access to basic amenities are at high risk of mental disorder.<sup>10</sup> Lifetime risk of affective disorders, panic disorders, generalized anxiety disorder, specific phobia and substance use disorders is found to be highest among illiterate and unemployed persons.<sup>11</sup> Suicidal behavior was found to have relation with female gender, working condition, independent decision making, premarital sex, physical abuse and sexual abuse.<sup>12</sup> Ongoing stress and chronic pain heightened the risk of suicide. Living alone and a break in a steady relationship within the past year were also significantly associated with suicide.<sup>13</sup> Work environment, school environment and family environment plays important role in pathogenesis of Psychiatric Diseases.

Females are more predisposed to mental disorders due to rapid social change, gender discrimination, social exclusion, gender disadvantage like marrying at young age, concern about the husband's substance misuse habits, and domestic violence.<sup>14</sup> Divorced and widowed women are at slightly elevated risk of mental disorders.<sup>10</sup> In India domestic violence is a big problem. A survey done in Maharashtra reported that 23 percent of women had been beaten in the last six months and of these 12 percent had explicitly been threatened to be burned.<sup>15</sup> Poorer women are more likely to suffer from adverse life events, to live in crowded or stressful conditions, to have fewer occupational opportunities and to have chronic illnesses; all of these are recognized risk factors for common Psychiatric Diseases.<sup>16</sup> Psychological factors such as headache and body ache, sensory symptoms and nonspecific symptoms such as tiredness and weakness also makes people vulnerable to mental disorders. Biological factors affecting mental disorders are genetic origin, abnormal physiology and congenital defect.

Disasters are potentially traumatic events which impose massive collective stress consequent to violent encounters with nature, technology or mankind.<sup>17</sup> Stigma related to Psychiatric Diseases, lack of awareness in common people, delayed treatment seeking behavior, lack of low cost diagnostic test and lack of easily available treatment are the main hurdles in combating the problem of mental health in India. In addition factors pertaining to traditional medicine and beliefs in supernatural powers in community delays diagnosis and treatment. India had focused its attention mainly to maternal and child health and communicable diseases. This leads to lack of political commitment to non communicable diseases further aggravating the load of mental disorders.

### 3. PSYCHOTIC DISORDERS

A survey has been initialised which records around 27% are being suffered from mental disabilities. Out of this, patients affected by anxiety, mood and substance use disorders are found to be 15%, 9% and 6% correspondingly.<sup>18</sup> It is confirmed that approximately one-third of the world population has been affected by mental dysfunctions. Estimation reports reveal that around 165 million people are considerably retarded mentally. Insomnia, major depression, dementia and drug dependence are some of the disorders which occur more frequently.<sup>19</sup>

#### 3.1 Trauma and stressor related disorder

The psychiatric condition due to stress, depression and anxiety leads to trauma and stressor related disorder. Neurochemicals are often included in the therapeutic diagnosis.<sup>20</sup> Psychosocial and somatic approaches are carried out to intervene this type of disorder.<sup>21</sup> Usually, post-traumatic stress condition is associated with acute stress dysfunction, where, clinical evidence in preventing such cases seems to be rare and it is suggested to have more techniques to deal with.<sup>22</sup> Experiments are carried out in soldiers before and during their deployment and the results show that the risk of being affected by this type of stress problem is low at pre-deployment. It focuses on prevention strategies at these stages.<sup>23</sup> Post-traumatic stress disorder is compared with that of mild traumatic brain injury and the results are found to be positive, i.e. both the outcomes are almost similar, where rat models are considered for research purpose.

Cognitive behavioural (CBT) therapy has been advised to people who are suffering from post-traumatic disorder.<sup>24</sup> Aftermath of the cognitive behavioural therapy is dealt in-order to establish at what rate that this treatment has been worked out. Although this method is effective, it can be strengthened along with modern and inhibitory learning and emotional processing theories.<sup>25</sup> Cognitive behavioural conjoint therapy is opted for post-traumatic stress disorder.<sup>26</sup>

#### 3.2 Depressive disorder

Stress on daily basis are accumulated and resulted in negative effects which have been illustrated by multilevel structural equation modeling. Two goals namely, disabling distress and strengthening resilience are achieved based on its findings.<sup>27</sup> Depressive disorder normally affects cognitive functions and can be treated through neuro-modulation along with antidepressants and psychotherapy. All the treatments mentioned above are required as in the case of major depressive disorder or psychotic depression.<sup>28</sup> In around 16% of affected people, its effects are retained lifelong. Antidepressants should be continued for several months after the treatments so that relapse, that is, degeneration in health status can be prevented. In rare events, such major depressive condition cannot be treated.<sup>29</sup> Alternative treatments such as interpersonal and psychodynamic medications are suggested along with regular exercises including yoga, acupuncture and consumption of omega-3-fatty acids and S-adenosyl-L-methionine.<sup>30</sup> Major depressive disorders with psychotic characteristics are medicated with antidepressant and electro-convulsive therapy. As electro-convulsive therapy is limited due to its extreme relapse rate which deteriorates the illness soon after the treatment, it is not put into practice often. Hence

pharmacologic treatments are favoured. In such cases, sertraline-olanzapine, fluoxetine-olanzapine or venlafaxine-quetiapine can be recommended.<sup>31</sup>

### 3.3 Dissociative disorder

Dissociative disorder arises in people who tend to forget things that are happening around. Some may even forget their personal details or get detached from self. It is named to be depersonalisation and amnesia respectively. It develops due to over stress.<sup>32</sup>

### 3.4 Schizophrenia

Schizophrenia is a neurodegenerative disease which describes the impairment of neurons and its death. It's a mental disorder and people who are affected by this would also be the victims of other disorders including major depressive defect, anxiety or substance use disorders. It can be controlled by antipsychotic medication like clozapine along with counselling and rehabilitation. The consequences of schizophrenia include hallucinations, delusions and cognitive impairments.<sup>33</sup> Practices such as histocompatibility and molecular genetic evidence are performed to evaluate the risk of schizophrenia. In extreme cases, this may also results in bipolar defect. DNA sample is taken from blood and genetic information is categorised using birdseed algorithm followed by several tests and algorithm to determine the threat of such mental illness.<sup>34</sup> States that schizophrenia is the disease which persist for a longer term and affects all aspects of patient's livelihood.<sup>35</sup> Therapy is prescribed so as to provide better quality of life by lowering the symptoms and thus maintaining its recovery. A particular method is considered in the treatment process and analysed. In-case if the progress is not satisfactory, the therapeutic method should be changed and the outcomes are further analysed. Treatments vary depending upon the reason for the illness. Hence initial assessment prior to treatment is very essential to deter patient's status and behaviour. Periodical checking and planning contributes well in treatments. The treatment may be seemed to be effective immediately after the medical aid in certain situations. This cannot be concluded as that the patient is well cured or further medication can be avoided. Only when the entire family and clinical members continue to support, remarkable improvements could be noticed in the rest of their lives. This is named to be bifocal approach and is proved to be fruitful under meta-analysis test. Superior temporal gyrus abnormalities are common in schizophrenia.<sup>36</sup>

The diagrammatic representation is provided in A1. It shows that dorsolateral prefrontal cortex (DLPFC) is the most affected area. Cognitive therapy is applicable to treat psychosis and to prevent adverse effects of schizophrenia.<sup>37</sup>

### 3.4 Delusional Disorder

A psychotic disorder where the patients are mostly suffered from delusional beliefs and negative thoughts is a form of illness connected with neurocognitive system. The sufferers would normally show learning restriction and poor memory capacity.<sup>38</sup> When trail making test, stroop tests, wisconsin card sorting test and verbal learning test are being conducted among patients with delusional disorder, even-though they are not affected by cognitive deterioration, failed in cognitive test.<sup>39</sup> Glossodynia, a psychosomatic defect, is normally characterised as a pain disorder. Psychotropic drugs including valproic acid, tandospirone and sertraline had no

effect over patients when these medicines are injected to them. Instead, a small dose of milnacipran could be able to control this type of disorder. In general even antidepressants are used to treat this, if it is not so acute. Glossodynia might also be related to cerebrovascular dysfunctions.

### **3.5 Body Dysmorphic Disorder (BDD)**

Any defect in physical appearance can be distressing and the affected individuals may develop symptoms of body dysmorphic disorder.<sup>40</sup> Assessments are based upon standardised clinical ratings to check the progress under cognitive behavioural therapy. It is found to produce maximum results in all the patients who are subjected to test.<sup>41</sup>

### **3.6 Substance related dysfunction and eating disorder**

Some of the substances resulting in malfunction include alcohol, tobacco, inhalants and stimulants.<sup>42</sup> Mental retardation causes substance dependence and vice versa. Psychiatric disorders occur owing to drug abuse. Univariate and multivariate tests have been conducted to foresee the accumulation and existence of induced drugs causing mental disturbances. Maladaptive perfectionism is associated with eating disorder due to body image dissatisfaction and negative self-evaluations.<sup>43</sup>

### **3.7 Anxiety disorder**

It is normal for an individual to be anxious regarding his/her affairs and status but overwhelming and going on thinking about issues about past and future leads to anxiety where over reaction to present facts roots to fear. Such factors are categorised to a type of mental disorder that acts as a causative agent to physical sickness too. This is classified as below.

#### **Generalised Anxiety Disorder (GAD)**

Excess anxiety is the cause for GAD if it is present for a longer duration.<sup>44</sup> In generalised anxiety disorder, getting worried is the noted cause. The article presents an effective training method in overcoming worry by viewing pictures unrelated to their panic condition or through verbal practices. A few number of people are subjected to this kind of experiment and all the people showed positive outcomes within shorter interval of time.

#### **Panic disorder**

Panic disorder is contributed to breathlessness, irregular heart beat (palpitation) and chest pain in-turn to heart failure.<sup>45</sup> It often results in chest pain which leads to coronary artery disease and other defects too.<sup>46</sup> It is given that the reduction of stress hormones such as corticotropin, cortisol and prolactin are the causative factors for panic situation at the dorsal periaqueductal grey matter of the brain. Presents the staging model. Subclinical symptoms and acute manifestations of agoraphobia or GAD are grouped under stage 1 and 2 accordingly, where panic disorder with hypochondriasis comes under stage 3.<sup>47</sup> All the above disorders are considered to be major depressive and are categorised in stage 4. According to this classification, suitable medications are provided with respect to the residual symptoms.

Some of the useful recommended factors before the actual treatment include panic disorder severity scale provided along with anxiety disorder and clinical interview which motivate the patients to get well soon. Behavioural inhibition syndrome is often noticed among teenagers. Anxiety and posttraumatic stress disorders

are the common defects due to lower hippocampal mass. It is stated that chronic stress can be resulted in defective hippocampal functioning as well.<sup>48</sup>

### **Phobic disorder**

Down syndrome has been associated with phobic disorder, which is said to be a common neurodevelopmental dysfunction. CBT is adopted to heal such phobic victims with intellectual disability and to reduce behavioural defects. Researchers conclude that childhood phobia may lead to drug dependence in their later age.<sup>49</sup> Substance use disorder and anxiety disorders are related to phobia. In most cases, occurrence of phobia is family dependent. Hence family based analyses such as Fisher's exact tests, logistic regression test and random effects are being held to determine the effects of phobic disorder.<sup>50</sup> Agoraphobia arises due to intense fear as a result of defensive response of the body. To control this effect, strong behavioural capability is to be cultivated by the patients, only then the exposure treatments would work in them.<sup>51</sup>

### **Obsessive-Compulsive Disorder (OCD)**

The presence of undesired thoughts or images within the people makes them obsessive. These people are forced to do a particular task repeatedly by themselves. Eye tracking devices are used to measure this sort of over checking behaviour. Mechanisms of symptom change during exposure and response prevention are analysed. Exposure and response prevention is provided to patients through cross-lagged multilevel modeling and it could not produce anticipated results. It is reported to find out productive techniques as a solution to OCD. Treatment includes anti-depressants such as selective serotonin reuptake inhibitors and clomipramine.<sup>52</sup>

## **4. Signs and Symptoms of anxiety disorders**

A subjective experience of distress with accompanying disturbances of sleep, concentration, social and/or occupational functioning are common symptoms in many of the anxiety disorders. Despite their similarities, these disorders often differ in presentation, course and treatment. Patients often present with complaints of poor physical health as their primary concern. This may temporarily distract from the underlying anxiety symptoms. This is particularly common in panic disorder, which is characterized by a short period of intense fear and a sense of impending doom, with accompanying physical symptoms, such as chest pain, dizziness and shortness of breath.<sup>53</sup> When complicated by agoraphobia, the individual fears have a panic attack in a place that prevents escape. This results in the patient avoiding such situations, with subsequent disturbances in functioning. The ancient term agoraphobia is translated from Greek as fear of an open marketplace. Agoraphobia today describes severe and pervasive anxiety about being in situations from which escape might be difficult or avoidance of situations such as being alone outside of the home, traveling in a car, bus, or airplane, or being in a crowded area.<sup>54</sup>

Most people who present to mental health specialists develop agoraphobia after the onset of panic disorder. Agoraphobia is best understood as an adverse behavioral outcome of repeated panic attacks and the subsequent worry, preoccupation, and avoidance.<sup>55</sup> Generalized anxiety disorder (GAD) rarely occurs without a co-morbid psychiatric disorder, with the patient experiencing consistent worry over multiple areas of his or her life for at least 6 months.<sup>56</sup>

Social phobia describes fear and anxiety in social situations leading to avoidance of social interaction.<sup>57</sup> Specific phobia is characterized by similar symptoms and behavior, but is triggered by a specific object or situation, such as a fear of certain animals (especially snakes, rodents, and dogs); birds, insects (especially spiders and bees or hornets); heights; elevators; flying; automobile driving; water; storms; and blood or injections.<sup>58</sup> Post-traumatic stress disorder and acute stress disorder occur after a patient experiences a traumatic event with subsequent physiological arousal in the face of stimuli that trigger memories of the event; avoidance of such stimuli; and a sense of re-experiencing the event. The latter occurs in the short term, while the former describe a more chronic version of the disorder.<sup>59</sup>

Obsessive-compulsive disorder (OCD) is characterized by repeated behaviors (compulsions), which serve to reduce anxiety connected to unwanted, intrusive thoughts (obsessions). Commonly seen behaviors are cleaning or washing in response to concerns about contamination, or repeatedly checking to see if a stove is turned off in response to concerns over a fire starting. Some people repeatedly check work or seek excessive reassurance due to obsessive self-doubt.<sup>60</sup>

## **5. Etiology and psychological basis of anxiety**

The etiology of anxiety may include stress, physical condition such as diabetes or other co-morbidities such as depression, genetic, first-degree relatives with generalized anxiety disorder (25%), environmental factors, such as child abuse, and substance abuse. The anxiety disorders are so heterogeneous that the relative roles of these factors are likely to differ. Some anxiety disorders, like panic disorder, appear to have a stronger genetic basis than others, although actual genes have not been identified. Other anxiety disorders are more rooted in stressful life events.

It is not clear why females have higher rates than males of most anxiety disorders, although some theories have suggested a role for the gonadal steroids. Other research on women's responses to stress also suggests that women experience a wider range of life events as stressful as compared with men who react to a more limited range of stressful events, specifically those affecting themselves or close family members.<sup>61</sup>

What the myriad of anxiety disorders have in common is a state of increased arousal or fear. Anxiety disorders often are conceptualized as an abnormal or exaggerated version of arousal. Much is known about arousal because of decades of study in animals and humans of the so called fight-or-flight response,<sup>||</sup> which also is referred to as the acute stress response. The acute stress response is critical to understanding the normal response to stressors and has galvanized research, but its limitations for understanding anxiety have come to the forefront in recent years.<sup>62</sup>



## 6. Pathophysiology

The exact mechanism is not entirely known. Anxiety can be a normal phenomenon in children. Stranger anxiety begins at seven to nine months of life. Anxiety symptoms and the resulting disorders are thought to be due to disrupted modulation within the central nervous system. Physical and emotional manifestations of this dysregulation are the result of heightened sympathetic arousal of varying degrees.<sup>63</sup>

Several neurotransmitter systems have been implicated to have a role in one or several of the modulatory steps involved. The most commonly considered are the serotonergic and noradrenergic neurotransmitter systems. In very general terms, it is thought that an under activation of the serotonergic system and an over activation of the noradrenergic system are involved. These systems regulate and are regulated by other pathways and neuronal circuits in various regions of the brain, resulting in dysregulation of physiological arousal and the emotional experience of this arousal.<sup>64</sup> Many believe that low serotonin system activity and elevated noradrenergic system activity are responsible for its development. It is, therefore, selective serotonin reuptake inhibitors and serotonin-norepinephrine reuptake inhibitors that are the first-line agent for its treatment. Disruption of the gamma-aminobutyric acid system has also been implicated because of the response of many of the anxiety spectrum disorders to treatment with benzodiazepines. There has been some interest in the role of corticosteroid regulation and its relationship to symptoms of fear and anxiety. Corticosteroids may increase or decrease the activity of certain neural pathways, affecting not only behavior under stress, but also the brain's processing of fear-inducing stimuli. Cholecystokinin has long been viewed as a neurotransmitter involved in regulating emotional states.<sup>66</sup>

There is such careful orchestration between these neurotransmitters that changes in one neurotransmitter system invariably elicit changes in another, including extensive feedback mechanisms. Serotonin and GABA are inhibitory neurotransmitters that quieten the stress response.<sup>66</sup> All of these neurotransmitters have become important targets for therapeutic agents.

## 7. Biochemical Basis of Anxiety

An exciting new line of research proposes that anxiety engages a wide range of neurocircuits. This line of research catapults to prominence two key regulatory centers found in the cerebral hemispheres of the brain—the hippocampus and the amygdala. These centers, in turn, are thought to activate the hypothalamic-pituitary-adrenocortical (HPA) axis. Researchers have long established the contribution of the HPA axis to anxiety but have been perplexed by how it is regulated. They are buoyed by new findings about the roles of the hippocampus and the amygdala.

Anxiety differs from fear in that the fear producing stimulus is either not present or not immediately threatening, but in anticipation of danger, the same arousal, vigilance, physiologic preparedness, and negative affects and cognitions occur.<sup>67</sup> Different types of internal or external factors or triggers act to produce the anxiety symptoms of panic disorder, agoraphobia, post-traumatic stress disorder, specific phobias, and generalized anxiety disorder, and the prominent anxiety that commonly occurs in major depression. It is currently a matter of research to determine whether dysregulation of these fear pathways leads to the symptoms of anxiety disorders. It has now been established, using noninvasive neuroimaging, that the human amygdala is also involved in fear

responses.<sup>68</sup> Fearful facial expressions have been shown to activate the amygdala in MRI studies of normal human subjects.<sup>68</sup> Functional imaging studies in anxiety disorders, such as PET studies of brain activation in phobias,<sup>69</sup> are also beginning to investigate the precise neural circuits involved in the anxiety disorders.

What is especially exciting is that neuroimaging has furnished direct evidence in humans of the damaging effects of glucocorticoids. In people with post-traumatic stress disorder, neuroimaging studies have found a reduction in the size of the hippocampus. The reduced volume appears to reflect the atrophy of dendrites—the receptive portion of nerve cells in a select region of the hippocampus. Similarly, animals exposed to chronic psychosocial stress display atrophy in the same hippocampal region. Stress-induced increases in glucocorticoids especially corticosterone are thought to be responsible for the atrophy (McEwen, 1998). If the hippocampus is impaired, the individual is thought to be less able to draw on memory to evaluate the nature of the stressor.<sup>70</sup>

## CONCLUSION

It is also crucial to be confidential within the group that it might not affect the victim any longer. Since acute psychotic illness is linked to emotional distress, patients should not be left alone and continuous monitoring could be beneficial. These forms of rehabilitation strategies are encouraged so as to bring such illnesses under control world-wide. Since the therapies of cognitive behavioural approach prove to be efficient, all clinical strategies must emphasise in developing further innovative techniques in this field for the betterment of patients and their well-wishers. Rather than academic oriented, people must be set to live a balanced life ensuring health, relationship and mental peace. It can be concluded that anxiety is manifest by disturbances of mood, thinking, behaviour, and physiological activity and accompanying disturbances of sleep, concentration, social and/or occupational functioning. Also, it is associated with restlessness, feeling keyed up or on edge, being easily fatigued, difficulty in concentrating or mind going blank, irritability, muscle tension, and irritability. The etiology of anxiety may include stress, diabetes, depression, genetic, and environmental factors. Drugs to reduce anxiety have been used by human beings for thousands of years. The drugs were used to reduce anxiety, including the barbiturates and the carbamates

## REFERENCE

- [1] World Health Organization. The world health report 2001 - Mental Health: New Understanding, New Hope. World Health Organization 2001, Geneva.
- [2] Kessler RC. Psychiatric epidemiology: selected recent advances and future directions. Bull World Health Organ. 2000;78(4):464-74.
- [3] World Health Organization. Depression and other Common Mental Disorders: Global Health Estimates. WHO: Geneva, 2017.
- [4] Whiteford HA, Ferrari AJ, Degenhardt L, Feigin V, Vos T. The global burden of mental, neurological and substance use disorders: an analysis from the Global Burden of Disease Study 2010. PLoS One. 2015;10(2): e0116820. <https://doi.org/10.1371/journal.pone.0116820>
- [5] Wittchen HU, Jacobi F, Rehm J, Gustavsson A, Svensson M, Jönsson B, et al. The size and burden of

- mental disorders and other disorders of the brain in Europe 2010. *Eur Neuropsychopharmacol.* 2011;21(9):655-79.
- [6] Kessler RC, Aguilar-Gaxiola S, Alonso J, Chatterji S, Lee S, Ormel J, et al. The global burden of mental disorders: an update from the WHO World Mental Health (WMH) surveys. *Epidemiol Psychiatr Soc.* 2009;18(1):23-33. <https://doi.org/10.1017/S1121189X00001421>
- [7] Bandelow B, Michaelis S. Epidemiology of anxiety disorders in the 21st century. *Dialogues Clin Neurosci.* 2015;17(3):327-35.
- [8] Wang YP, Chiavegatto Filho AD, Campanha AM, Malik AM, Mogadouro MA, Cambraia M, et al. Patterns and predictors of health service use among people with mental disorders in São Paulo metropolitan area, Brazil. *Epidemiol Psychiatr Sci.* 2017;26(1):89-101. <https://doi.org/10.1017/S2045796016000202>
- [9] Craske MG, Stein MB. Anxiety. *Lancet.* 2016;388(10063):3048-59. [https://doi.org/10.1016/S0140-6736\(16\)30381-6](https://doi.org/10.1016/S0140-6736(16)30381-6)
- [10] Patel V, Kirkwood BR, Pednekar S, Weiss H, Mabey D. Risk factors for common mental disorders in women. Population-based longitudinal study. *Br J Psychiatry.* 2006;189:547-55.
- [11] Deswal BS, Pawar A. An Epidemiological Study of Mental Disorders at Pune, Maharashtra. *Indian J Community Med.* 2012;37(2):116-21.
- [12] Pillai A, Andrews T, Patel V. Violence, psychological distress and the risk of suicidal behavior in young people in India. *Int J Epidemiol.* 2009;38(2):459-69.
- [13] Manoranjitham SD, Rajkumar AP, Thangadurai P, Prasad J, Jayakaran R, Jacob KS. Risk factors for suicide in rural south India. *Br J Psychiatry.* 2010;196(1):26-30.
- [14] Patel V, Kleinman A. Poverty and common mental disorders in developing countries. *Bull World Health Organ.* 2003;81(8):609-615.
- [15] Jain D, Sanon S, Sadowski L, Hunter W. Violence against women in India: evidence from rural Maharashtra, India. *Rural Remote Health.* 2004;4(4):304.
- [16] Kermode M, Herrman H, Arole R, White J, Premkumar R, Patel V. Empowerment of women and mental health promotion: a qualitative study in rural Maharashtra, India. *BMC Public Health.* 2007;7:225.
- [17] Nandi PS, Banerjee G, Mukherjee SP, Nandi S, Nandi DN. A study of psychiatric morbidity of the elderly population of a rural community in West Bengal. *Indian J Psychiatry.* 1997; 39(2):122-9.
- [18] F.Jacobi, M.Hofler, J.Siegert, S.Mack, A.Gerschler, Scholl, Markus A.Busch, U.Hapke, U.Maske, I.Seiffert, W.Gaebel, W.Maier, M.Wagner, J.Zielasek and A.Wittchen, Twelve- Month Prevalence, Comorbidity and Correlates of Mental Disorders in Germany: The Mental Health Module of the German Health Interview and Examination Survey for Adults (DEGS1-MH), *International Journal of Methods in Psychiatric Research*, Vol. 23, No. 3, 2014, pp. 304-319, <http://dx.doi.org/10.1002/mpr.1439>.
- [19] H.U.Wittchen, F.Jacobi, J.Rehm, A. Gustavsson, M.Svensson, B.Jonsson J.Olesen, C.Allgulander, J.Alonso, C.Faravelli, L.Fratiglioni, P.Jennum, R.Lieb, A.Maercker, J.Os, M.Preisig, L.Salvador-Carulla, R.Simon and H.C.Steinhausen, The Size and Burden of Mental Disorders and Other Disorders of the Brain in Europe,

- European Neuropsychopharmacology, Vol. 21, No. 9, 2011, pp. 655-679, <http://dx.doi.org/10.1016/j.euroneuro.2011.07.018>.
- [20] I.P.B.Watson, M.Brune and J.Bradley, The Evolution of the Molecular Response to Stress and its Relevance to Trauma and Stressor-Related Disorders, Neuroscience and Biobehavioral, Vol. 68, 2016, pp. 134-147, <http://dx.doi.org/10.1016/j.neubiorev.2016.05.010>.
- [21] Jonathon R.Howlett and Murray B.Stein, Prevention of Trauma and Stressor-Related Disorders: A Review, Neuropsychopharmacology Reviews, Vol. 41, 2016, pp. 357-369.
- [22] H.J.Sorensen, S.B.Andersen, K.Inge Karstoft, and T.Madsen, The Influence of Pre-Deployment Cognitive Ability on Post-Traumatic Stress Disorder Symptoms and Trajectories: The Danish USPER Follow-Up Study of Afghanistan Veterans, Journal of Affective Disorders, Vol. 196, 2016, pp. 148-153.
- [23] D.R.Davies, D.Olson, D.L.Meyer, J.L.Scholl, M.J.Watt, P.Manzerra, K.J.Renner and G.L.Forster, Mild Traumatic Brain Injury with Social Defeat Stress Alters Anxiety, Contextual Fear Extinction, and Limbic Monoamines in Adult Rats, Frontiers in Behavioural Neuroscience, Vol.10, No.71, 2016.
- [24] Joo Eon Park, Hyun-Nie Ahn and Young-Eun Jun, Prevention and Treatment of Trauma and Stressor- Related Disorders: Focusing on Psychosocial Interventions for Adult Patients, Journal of Korean Neuropsychiatry Association, Vol. 55, No. 2, 2016, pp. 89-96.
- [25] Adele M.Hayes, Carly Yasinski, Damion Grasso, C.Beth Ready, Elizabeth Alpert, Thomas Mccauley, Charles Webb and Esther Deblinger, Constructive and Unproductive Processing of Traumatic Experiences in Trauma-Focused Cognitive- Behavioral Therapy for Youth, Behavior Therapy, 2016.
- [26] Philippe Shnaider, Iris Sijercic and G.W.K.Suvakcandice M.Monson, The Role of Social Support in Cognitive-Behavioral Conjoint Therapy for Posttraumatic Stress Disorder, Behavior Therapy, 2016
- [27] D.M.Dunkley, M.Lewkowski, Ihno A.Lee, K.J.Preacher, D.C.Zuroff, Jody-Lynn Berg, J.E.Foley, Gail Myhr and Ruta Westreich, Daily Stress, Coping and Negative and Positive Affect in Depression: Complex Trigger and Maintenance Patterns, Behavior Therapy, 2016, <http://dx.doi.org/10.1016/j.beth.2016.06.001>.
- [28] H.F.Zickgraf, D.L.Chambless, K.S .Mccarthy, R.Gallop, B.A.Sharpless, B.L. Milrod and J.P.Barber, Interpersonal Factors are Associated with Lower Therapist Adherence in Cognitive-Behavioural Therapy for Panic Disorder, Clinical Psychology & Psychotherapy, Vol. 23, No. 3, 2016, pp. 272-284.
- [29] Dorothy E.Stubbe, Defeating Depression: The Healing Power of the Therapeutic Relationship, The Journal of Lifelong Learning in Psychiatry, Vol. 14, No. 2, 2016, pp. 219-221,
- [30] G.Gartlehner, Non-pharmacological versus Pharmacological Treatments for Adult Patients with Major Depressive Disorder, The Journal of Lifelong Learning in Psychiatry, Vol. 14, No. 2, 2016, pp. 283-293.
- [31] Anthony J.Rothschild, Treatment for Major Depression with Psychotic Features (Psychotic Depression), The Journal of Lifelong Learning in Psychiatry, Vol. 14, No. 2, 2016, pp.207-209.
- [32] S.B.Renard, R.J.C.Huntjens, Paul H.Lysaker, A.Moskowitz, A.Aleman and Gerdina H.M.Pijnenborg, Unique and Overlapping Symptoms in Schizophrenia Spectrum and Dissociative Disorders in Relation to Models of Psychopathology: A Systematic Review, Schizophrenia Bulletin, 2016.

- [33] Pamela Sklar and Shaun Purcell Common Polygenic Variation Contributes to Risk of Schizophrenia that Overlaps with Bipolar Disorder, *Nature*, Vol. 460, No. 7256, 2014, pp. 748-752.
- [34] A.F.Lehman, J.A.Lieberman, L.Dixon, T.Mcglashan, A.Miller, D.O.Perkins and D.Regier, Practice Guideline for the Treatment of Patients with Schizophrenia, *American Journal of Psychiatry*, Vol. 161, No.2, 2004.
- [35] G.Walz, S.Leucht, J.Baum, Kissling and R.Engel, The Effect of Family Interventions on Relapse and Rehospitalization in Schizophrenia: A Meta-Analysis, *Focus-The Journal of Lifelong Learning in Psychiatry*, Vol. 2, No. 1, 2004, pp. 78-94.
- [36] H.Matsumoto, A.Simmons, Williams, M.Hadjulis, R.Pipe, R.Murray and S.Frangou, Superior Temporal Gyrus Abnormalities in Early-Onset Schizophrenia: Similarities and Differences with Adult-Onset Schizophrenia, *American Journal of Psychiatry*, Vol. 158, No. 8, 2001, pp. 1299-304.
- [37] Robert A.Gould, Kim T.Mueser, Elisa Bolton, Virginia Mays and Donald Goff, Cognitive Therapy for Psychosis in Schizophrenia: An Effect Size Analysis, *Focus-The Journal of Lifelong Learning in Psychiatry*, 2015.
- [38] I.Casas, E.Portugal, N.Gonzalez, Mckenney, J.M.Haro, J.Usall, Deficits in Executive and Memory Processes in Delusional Disorder: A Case-Control Study, *Plos One*, Vol. 8, No. 7, 2013.
- [39] K.Ukai, H.Kimura, M.Arao, B.Aleksic, A.Yamauchi, R.Ishihara, S.Iritani, K.Kurita, and N.Ozaki Effectiveness of Low-Dose Milnacipran for a Patient Suffering from Pain Disorder with Delusional Disorder (Somatic Type) in the Orofacial Region. *Psychogeriatrics*, Vol. 13, No. 2, 2013, pp. 99-102.
- [40] J.L.Greenberg, S.S.Mothi and Sabine Wilhelm, Cognitive Behavioral Therapy for Body Dysmorphic Disorder by Proxy, *Behavior Therapy*, Vol. 47, No. 4, 2016, pp. 515-526.
- [41] J.L. Greenberg, S.S. Mothi and Sabine Wilhelm, Cognitive-Behavioral Therapy for Adolescent Body Dysmorphic Disorder: A Pilot Study, *Behavior Therapy*, Vol. 47, No. 2, 2016, pp. 213-224
- [42] Richard N.Rosenthal, *Duel Diagnosis*, Bruner-Routledge, New York, 2015, pp. 1-43.
- [43] A.M.Bardone-Cone, Stacy L.Lin and Rachel M.Butler, Perfectionism and Contingent Self-Worth in Relation to Disordered Eating and Anxiety, *Behavior Therapy*, 2016.
- [44] Claire Eagleson, Sarra Hayes, Andrew Mathews, Gemma Permand and Colette R.Hirsch, The Power of Positive Thinking: Pathological Worry is Reduced by Thought Replacement in Generalized Anxiety Disorder, *Behaviour Research and Therapy*, Vol.78, 2016, pp.13-18.
- [45] Sergio Machado, Eduardo Lattari and Jeffrey P.Kahn, Possible Mechanisms Linking Panic Disorder and Cardiac Syndromes, *Panic Disorder*, 2016, pp. 185-202.
- [46] Luiz Carlos Schenberg, A Neural Systems Approach to the Study of the Respiratory-Type Panic Disorder, *Panic Disorder*, 2016, pp. 9-77.
- [47] Fiammetta Cosci, Staging of Panic Disorder: Implications for Neurobiology and Treatment, *Panic Disorder*, 2016, pp. 113-125.
- [48] J.Mallet, V.Guillard, O.Huillard, C.Dubertret and F.Limosin, Effectiveness of Cognitive Behavioral Therapy in the Treatment of a Phobic Disorder in a Patient with Down Syndrome and Early Alzheimer's Disease, *Vol.33*, 2016.

- [49] H.C.Steinhausen, H.Jakobsen,A.Meyer, P.M.Jorgensen and R.Lieb,Family Aggregation and Risk Factors in Phobic Disorders over Three- Generations in a Nation-Wide Study. *Plos One*, Vol. 11, No. 1, 2016, <http://dx.doi.org/10.1371/journal.pone.0146591>.
- [50] A.O.Hamm, J.Richter, C.Pane-Farre, D.Westphal, H.Wittchen, A.Elsebusch,A.L.Gerlach, A.T.Gloster, Andreas Strohle, T.Lang, Kircher, B.M.Gerdes, G.W.Alpers, Andreas Reif and Jurgen Deckert, Panic Disorder with Agoraphobia from a Behavioral Neuroscience Perspective: Applying the Research Principles Formulated by the Research Domain Criteria (RDoC) Initiative, *Psychophysiology*, Vol. 53, No. 3, 2016, pp. 312-322.
- [51] Marieke B.J.Toffolo, Marcel A.Van Den Hout, Iris M.Engelhard, Ignace T.C.Hooge and Danielle C.Cath, Patients with Obsessive-Compulsive Disorder Check Excessively in Response to Mild Uncertainty, *Behavior Therapy*, Vol. 47, No. 4, 2016, pp.550-559.
- [52] Y.J.K.Carpenter and H.S.Edna Foa, Cognitive Mediation of Symptom Change in Exposure and Response Prevention for Obsessive-Compulsive Disorder, *Behaviour Therapy*, Vol. 47, No. 4, 2016, pp. 474-486.
- [53] Markowitz, J. S., Weissman, M. M., Ouellette, R., Lish, J. D., & Klerman, G. L. (1989). Quality of life in panic disorder. *Archives of General Psychiatry*, 46(11), 984-992.
- [54] Magee, W. J., Eaton, W. W., Wittchen, H. U., McGonagle, K. A., & Kessler, R. C. (1996). Agoraphobia, simple phobia, and social phobia in the National Comorbidity Survey. *Archives of general psychiatry*, 53(2), 159-168.
- [55] Barlow, D.H. (1988). *Anxiety and its disorders: The nature and treatment of anxiety and panic*. New York: Guilford Press.
- [56] Schweizer, E. (1995). Generalized Anxiety Disorder: Longitudinal Course and Pharmacologic. *Psychiatric Clinics of North America*, 18(4), 843-857.
- [57] Ballenger, J. C., Davidson, J. R., Lecrubier, Y., Nutt, D. J., Bobes, J., Beidel, D. C., ... & Westenberg, H. G. (1998). Consensus statement on social anxiety disorder from the International Consensus Group on Depression and Anxiety. *The Journal of clinical psychiatry*.
- [58] Marks, I.M. (1969). *Fears and phobias*. New York: Academic Press.
- [59] Kessler, R. C., Sonnega, A., Bromet, E., Hughes, M., & Nelson, C. B. (1995). Posttraumatic stress disorder in the National Comorbidity Survey. *Archives of general psychiatry*, 52(12), 1048-1060.
- [60] Eddy, M. F., & Walbroehl, G. S. (1998). Recognition and treatment of obsessive-compulsive disorder. *American Family Physician*, 57(7), 1623- 8.
- [61] Maciejewski, P. K., Prigerson, H. G., & Mazure, C. M. (2001). Sex differences in event-related risk for major depression. *Psychological medicine*, 31(4), 593-604.
- [62] Barbee, J. G. (1998). Mixed symptoms and syndromes of anxiety and depression: diagnostic, prognostic, and etiologic issues. *Annals of Clinical Psychiatry*, 10(1), 15-29.
- [63] Kaplan, H.I., & Sadock, B.J. (1995). *Comprehensive Textbook of Psychiatry/VI*. 6th ed. Williams & Wilkins, Baltimore, Maryland. pp. 1244-48.
- [64] Ressler, K.J., & Nemeroff, C.B. (2000). Role of serotonergic and noradrenergic systems in the pathophysiology of depression and anxiety disorders. *Depress Anxiety*, 12(1), 2-19.

- [65] Korte, S.M. (2001). Corticosteroids in relation to fear, anxiety and psychopathology. *Neurosc. Biobehav. Rev.*; 25(1), 17-42.
- [66] Coplan, J.D., & Lydiard, R.B. (1998). Brain circuits in panic disorder. *Bio. Psychiatry.* 44; 1264–1276.
- [67] LeDoux, J. (1996). Emotional networks and motor control: A fearful view. *Prog. Brain Res.*, 107, 437–446.
- [68] Breiter, H.C., Etcoff, N.L., Whalen, P.J., Kennedy, W.A., Rauch, S.L., Buckner, R.L., Strauss, M.M., Hyman, S.E., & Rosen, B.R. (1996). Response and habituation of the human amygdala during visual processing of facial expression. *Neuron*, 17; 875– 887.
- [69] Rauch, S.L., Savage, C.R., Alpert, N.M., Miguel, E.C., Baer, L., Breiter, H. C., Fischman, A.J., Manzo, P.A., Moretti, C., & Jenike, M.A. (1995). A positron emission tomographic study of simple phobic symptom provocation. *Arch. Gen.Psychiatry*, 52; 20–28.
- [70] McEwen, B.S., & Magarinos, A.M. (1997). Stress effects on morphology and function of the hippocampus. *Annals of the New York Academy of Sciences*, 821; 271–284.

