Sri Lakshmi Narayana Institute of Medical Sciences

Date: 01.06.2020

From
DR.V.R Sridhar
Professor and Head,
Department of Psychiatry,
Sri Lakshmi Narayana institute of medical sciences,
Bharath Institute of Higher Education and Research,
Chennai.

To
The Dean,
Sri Lakshmi Narayana Institute Of Medical Sciences,
Bharath Institute of Higher Education and Research,
Chennai.

Sub: Permission to conduct value-added course: Anxiety Disorders and its management

Dear Sir,

With reference to the subject mentioned above, the department proposes to conduct a value-added course titled: **Anxiety Disorders and its management** on 1/07/2020. We solicit your kind permission for the same.

Kind Regards

Dr.V.R. Sridhar

FOR THE USE OF DEANS OFFICE

Names of Committee members for evaluating the course:

The Dean: **Dr. Rajeshekaran. K**

The HOD: **Dr. Sridhar**

The Expert: **Dr. Arun Seetharaman.**The committee has discussed about the course and is approved.

Dean
(Sign & seal)

Prof. S. RAJASEKARAN, M.S., (Gen.)
DEAN
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Agaram Post, Pondicherry-605 502.

Subject Expert
(Sign & Seal)

Dr. ARUN SEETHARAMAN, MD.,
Reg. No: 91440
Associate Professor, Psychiatry
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Kudapakkam, Puducherry-605 502.

HO
(Sign & Seal)

Dr. V. R. SRIDHAR, MD.,D.P.M.,
Reg. No: 30995
Professor & HOD, Psychiatry
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Kudapakkam, Puducherry-605 502.

OFFICE OF THE DEAN

Sri Lakshmi Narayana Institute of Medical Sciences

OSUDU, AGARAM VILLAGE, VILLIANUR COMMUNE, KUDAPAKKAM POST,
PUDUCHERRY - 605 502.
[Recognised by Medical Council of India, Ministry of Health letter No. U/12012/249/2005-ME (P -II) dt. 11/07/2011]
[Affliated to Bharath University, Chennai - TN]

Circular

15.06.2020

Sub: Organising Value-added Course: Awareness, Identification and Classification of Anxiety Disorders and Its Management for final year students -6 months

With reference to the above mentioned subject, it is to bring to your notice that Sri Lakshmi Narayana Institute of Medical Sciences, **Bharath Institute of Higher Education and Research** is organizing **"Awareness, Identification and Classification of Anxiety Disorders and Its Management for final year students"**.

The application must reach the institution along with all the necessary documents as mentioned. The hard copy of the application should be sent to the institution by registered/ speed post only so as to reach on or before 30 June, 2020. Applications received after the mentioned date shall not be entertained under any circumstances.

Dean

Dr.Rajasekar

Prof. S. RAJASEKARAN, M.S., (Gen.)
DEAN
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Agaram Post, Pondicherry-605 502.

Encl: Copy of Course content

Course Proposal

Course Title: **Awareness, Identification And Classification Of Anxiety Disorders And Its Management** .

Course Objective:

Awareness on the importance of anxiety disorders
Awareness On The Contributing Factors To Anxiety Disorders Bio-psychosocial Model Of Approach
Identify diagnostic criteria for anxiety disorders

Course Outcome:

Course Coordinator: Dr.V.R. Sridhar
Course Faculties with Qualification and Designation:
1.Dr.V.R.SRIDHAR, Professor & HOD
2.Dr.Arun, Assistant Professor
Course Curriculum/Topics with schedule (Min of 30 hours)

SlNo	Date	Topic	Faculty	Time	Hours
1.	1.07.2020	Awareness on the importance of anxiety disorders	Dr.Arun	4-5p.m	1
2.	3.07.2020	Awareness On The Contributing Factors To Anxiety Disorders Bio-psychosocial Model Of Approach	Dr.Arun	2-3p.m	1
3.	6.07.2020	Identify diagnostic criteria for anxiety disorders	Dr.Arun	4-6p.m	2
4.	8.07.2020	Awareness on the role of physicians & HCWs in anxiety disorders	Dr.Arun	4-6p.m	2
5.	10.07.2020	Recognize the influence of society on patients with anxiety disorders	Dr.Arun	4-6p.m	2
6.	13.07.2020	**Module 1** – Introduction to Anxiety Disorders	Dr.Arun	4-5p.m	2
7.	15.07.2020	**Module 2** – Generalized Anxiety Disorder	Dr.Arun	4-5P.M	1
8.	17.07.2020	**Module 3** – Panic Disorder	Dr.Arun	4-5p.m	1
9.	20.07.2020	**Module 4** – Phobic Anxiety Disorder	Dr. Shridhar	4-6p.m	1
10.	22.07.2020	**Module 5** – Obsessive Compulsive Disorder	Dr.Arun	4-6p.m	2
11.	24.07.2020	**Module 6** – Other Anxiety Disorders	Dr.Arun	4-6p.m	1
12.	27.07.2020	**Module 7** – Unspecified Anxiety Disorders	Dr.Arun	4-6p.m	2
13.	29.07.2020	Pre course and Post Course evaluation, Feedback analysis from Likert	Dr.Arun	2-5p.m	3

		scale			
		Practical Class I	Dr. Shridhar		
13.	3.08.2020	Cognitive Behaviour Therapy	Dr. Shridhar	2-3 PM	1
14.	5.08.2020	Graded Relaxation	Dr. Shridhar	2-3 PM	1
15.	7.08.2020	Hypnotherpy	Dr. Shridhar	2-4 PM	2
16.	10.08.2020	Yoga And Breathing Techniques	Dr. Shridhar	2-4 PM	2
17.	12.08.2020	Biofeedback	Dr. Shridhar	2-4p.m	2
		Total			30 hrs

REFERENCE BOOKS:

1.Comprehensive textbook of PSYCHIATRY by Kaplan and Saddocks.

2. Barlow, D. H. (2002). *Anxiety and its disorders: The nature and treatment of anxiety and panic (2nd ed.)*. New York: Guilford Press.

3. DSM 5 AND ICD 10

4.Beck, A. T. & Emery, G. (1985). *Anxiety disorders and phobias: A cognitive perspective*. New York: Basic Books.

5.Borne, E. J. (2000). *The anxiety & phobia workbook (3rd ed.)*. Oakland: New Harbinger Publications.

Websites

- Anxiety disorders Association of America
- Association for Behavioral and Cognitive Therapies
- National Institute of Mental Health

VALUE ADDED COURSE

1. Name of the programme & Code

An Introduction And Assessment Of Various Types Of Anxiety Disorders Along With Its Management - PSYC01

2. Duration & Period

30 hrs & July– December 2020

3. Information Brochure and Course Content of Value Added Courses

Enclosed as Annexure- I

4. List of students enrolled

Enclosed as Annexure- II

5. Assessment procedures:

Assessment Evolution by MCQ method - *Enclosed as Annexure- III*

6. Certificate model

Enclosed as Annexure- IV

7. No. of times offered during the same year:

1 time, July– December 2020

8. Summary report of each program year-wise

Value Added Course- July– December 2020					
Sl. No	**Course Code**	**Course Name**	**Resource Persons**	**Target Students**	**Strength & Year**
1	**PSYC01**	Anxiety Disorders Along With Its Management	**Dr. Sridhar**	IInd YEAR	(15) July– December 2020

9. Course Feed Back

Enclosed as Annexure- V

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RESOURCE PERSON

1. **Dr. V.R. Sridhar**
2. **Dr. C. Arun Seetharaman**

COORDINATOR

V.R. Sridhar

ANXIETY DISORDERS

PARTICIPANT HAND BOOK

BIHER SLIMS

COURSE DETAILS

Particulars	Description
Course Title	Anxiety disorders
Course Code	PSYC01
Objective	**1.** Classification **2.** Epidemiology **3.** Etiology **4.** Psychological test **5.** Laboratory tests **6.** Pathophysiology and brain studies **7.** Differential Diagnosis **8.** Course and prognosis **9.** Treatment
Further learning opportunities	Diagnosis and treatment of anxiety disorders
Key Competencies	On successful completion of the course the students will have skill in diagnosing and treating anxiety disorders.
Target Student	IInd year MBBS Students
Duration	30hrs Every July 2020– December 2020
Theory Session	20hrs
Practical Session	10hrs
Assessment Procedure	Multiple choice questions

Classification

There are 11 diagnostic types of anxiety disorders ranging in the fifth edition of Diagnostic Statistical Manual of Mental Disorders (DSM-5) from separation anxiety disorder to unspecified anxiety disorder. They are among the most common groups of psychiatric disorders. Each disorder is discussed separately below.

A. Separation anxiety disorder. Fears of separation from loved ones (not commensurate with appropriate development) (at least 3 of the following for >1 month if under 18, and >6 months if an adult): Anxiety when separating from home or loved ones leaving the home because it will entail such separation sleeping away (different room) from loved ones Along with feelings of worry about harm to loved ones worry about separation (even if not impending) from loved ones fear about isolation from loved ones Including nightmares physical symptoms

B. Selective mutism. Inability to speak in certain social situations despite the ability to speak in others (occurring for at least 1 month in the absence of a separate disorder and impeding social/educational/occupational functioning). Specific phobia. Marked and disproportionate anxiety about a specific thing (e.g., horses, heights, needles) or situation. This fear must be consistently and persistently present. The person experiences massive anxiety when exposed to the feared object and tries to avoid it at all costs. Up to 25% of the population has specific phobias.

C. Social anxiety disorder (social phobia). Social anxiety disorder is an irrational fear of public situations (e.g., speaking in public, eating in public, using public bathrooms [shy bladder]). May be associated with panic attacks. It usually occurs during early teens but can develop during childhood. Effects up to 13% of persons. Equally common in men and women.

D. Panic disorder. Panic disorder is characterized by spontaneous panic attacks. It may occur alone or be associated with agoraphobia (fear of being in open spaces, outside the home alone, or in a crowd). Panic may evolve in stages: subclinical attacks, full panic attacks, anticipatory anxiety, phobic avoidance of specific situations, and agoraphobia. It can lead to alcohol or drug abuse, depression, and occupational and social restrictions. Agoraphobia can occur alone, although patients usually have associated panic attacks. Anticipatory anxiety is characterized by the fear that panic, with helplessness or

humiliation, will occur. panic disorder often have multiple somatic complaints related to autonomic nervous system dysfunction, with a higher risk in females.

HISTORY

The idea of panic disorder may have its roots in the concept of irritable heart syndrome, which the physician Jacob Mendes DaCosta (1833– 1900) noted in soldiers in the American Civil War. DaCosta's syndrome included many psychological and somatic symptoms that have since been included among the diagnostic criteria for panic disorder. In 1895, Sigmund Freud introduced the concept of anxiety neurosis, consisting of acute and chronic psychological and somatic symptoms.

EPIDEMIOLOGY

The lifetime prevalence of panic disorder is in the 1 to 4 percent range, with 6-month prevalence approximately 0.5 to 1.0 percent and 3 to 5.6 percent for panic attacks. Women are two to three times more likely to be affected than men, although underdiagnoses of panic disorder in men may contribute to the skewed distribution. The differences among Hispanics, whites, and blacks are few. The only social factor identified as contributing to the development of panic disorder is a recent history of divorce or separation. Panic disorder most commonly develops in young adulthood—the mean age of presentation is about 25 years—but both panic disorder and agoraphobia can develop at any age. Panic disorder has been reported in children and adolescents, and it is probably underdiagnosed in these age groups.

COMORBIDITY

Of patients with panic disorder, 91 percent have at least one other psychiatric disorder. About one-third of persons with panic disorders have major depressive disorder before onset; about two-thirds first experience panic disorder during or after the onset of major depression. Other disorders also commonly occur in persons with panic disorder. Of persons with panic disorder, 15 to 30 percent also have social anxiety disorder or social phobia, 2 to 20 percent have specific phobia, 15 to 30 percent have generalized anxiety disorder, 2 to 10 percent have PTSD, and up to 30 percent have OCD. Other common comorbid conditions are hypochondriasis or illness anxiety disorder, personality disorders, and substance-related disorders.

ETIOLOGY

Biological Factors Research on the biological basis of panic disorder has produced a range of findings; one interpretation is that the symptoms of panic disorder are related to a range of biological abnormalities in brain structure and function. Most work has used biological stimulants to induce panic attacks in patients with panic disorder. Considerable evidence indicates that abnormal regulation of brain noradrenergic systems is also involved in the pathophysiology of panic disorder. These and other studies have produced hypotheses implicating both peripheral and central nervous system (CNS) dysregulation in the pathophysiology of panic disorder. The autonomic nervous systems of some patients with panic disorder have been reported to exhibit increased sympathetic tone, to adapt slowly to repeated stimuli, and to respond excessively to moderate stimuli. Studies of the neuroendocrine status of these patients have shown several abnormalities, although the studies have been inconsistent in their findings. The major neurotransmitter systems that have been implicated are those for norepinephrine, serotonin, and GABA. Serotonergic dysfunction is quite evident in panic disorder, and various studies with mixed serotonin agonist–antagonist drugs have demonstrated increased rates of anxiety. Such responses may be caused by postsynaptic serotonin hypersensitivity in panic disorder. Preclinical evidence suggests that attenuation of local inhibitory GABAergic transmission in the baso-lateral amygdala, midbrain, and hypothalamus can elicit anxiety-like physiological responses. The biological data have led to a focus on the brainstem (particularly the noradrenergic neurons of the locus ceruleus and the serotonergic neurons of the median raphe nucleus), the limbic system (possibly responsible for the generation of anticipatory anxiety), and the prefrontal cortex (possibly responsible for the generation of phobic avoidance). Among the various neurotransmitters involved, the noradrenergic system has also attracted much attention, with the presynaptic α2 -adrenergic receptors, particularly, playing a significant role. Patients with panic disorder are sensitive to the anxiogenic effects of yohimbine in addition to having exaggerated MHPG, cortisol, and cardiovascular responses. They have been identified by pharmacological challenges with the α2 -receptor agonist clonidine (Catapres) and the α2 -receptor antagonist yohimbine (Yocon), which stimulates firing of the locus ceruleus and elicits high rates of panic-like activity in those with panic disorder. Panic-Inducing Substances. Panic-inducing substances (sometimes called panicogens) induce panic attacks in most patients with panic disorder and in a

much smaller proportion of persons without panic disorder or a history of panic attacks. So-called respiratory panic inducing substances cause respiratory stimulation and a shift in the acid–base balance. These substances include carbon dioxide (5 to 35 percent mixtures), sodium lactate, and bicarbonate. Neurochemical panic-inducing substances that act through specific neurotransmitter systems include yohimbine, an α2 -adrenergic receptor antagonist; mCPP, an agent with multiple serotonergic effects; m-Caroline drugs; GABAB receptor inverse agonists; flumazenil (Romazicon), a GABAB receptor antagonist; cholecystokinin; and caffeine. Isoproterenol (Isuprel) is also a panic-inducing substance, although its mechanism of action in inducing panic attacks is poorly understood. The respiratory panic-inducing substances may act initially at the peripheral cardiovascular baroreceptors and relay their signal by vagal afferents to the nucleus tractus solitarii and then on to the nucleus paragigantocellularis of the medulla. The hyperventilation in patients with panic disorder may be caused by a hypersensitive suffocation alarm system whereby increasing PCO2 and brain lactate concentrations prematurely activate a physiological asphyxia monitor. The neurochemical panic-inducing substances are presumed to primarily affect the noradrenergic, serotonergic, and GABA receptors of the CNS directly. Brain Imaging. Structural brain imaging studies, for example, MRI, in patients with panic disorder have implicated pathological involvement in the temporal lobes, particularly the hippocampus and the amygdala. One MRI study reported abnormalities, especially cortical atrophy, in the right temporal lobe of these patients. Functional brain imaging studies, for example, positron emission tomography (PET), have implicated dysregulation of cerebral blood flow (smaller increase or an actual decrease in cerebral blood flow). Specifically, anxiety disorders and panic attacks are associated with cerebral vasoconstriction, which may result in CNS symptoms, such as dizziness, and in peripheral nervous system symptoms that may be induced by hyperventilation and hypocapnia. Most functional brain imaging studies have used a specific panic-inducing substance (e.g., lactate, caffeine, or yohimbine) in combination with PET or SPECT to assess the effects of the panic-inducing substance and the induced panic attack on cerebral blood flow. Mitral Valve Prolapse. Although great interest was formerly expressed in an association between mitral valve prolapse and panic disorder, research has almost completely erased any clinical significance or relevance to the association. Mitral valve prolapse is a heterogeneous syndrome consisting of the prolapse of one of the mitral valve leaflets, resulting in a midsystolic click on

cardiac auscultation. Studies have found that the prevalence of panic disorder in patients with mitral valve prolapse is the same as the prevalence of panic disorder in patients without mitral valve prolapse. Genetic Factors Various studies have found that the first-degree relatives of patients with panic disorder have a four- to eightfold higher risk for panic disorder than first-degree relatives of other psychiatric patients. The twin studies conducted to date have generally reported that monozygotic twins are more likely to be concordant for panic disorder than are dizygotic twins. At this point, no data exist indicating an association between a specific chromosomal location or mode of transmission and this disorder. Psychosocial Factors Psychoanalytic theories have been developed to explain the pathogenesis of panic disorder. Psychoanalytic theories conceptualize panic attacks as arising from an unsuccessful defence against anxiety-provoking impulses. What was previously a mild signal anxiety becomes an overwhelming feeling of apprehension, complete with somatic symptoms. Many patients describe panic attacks as coming out of the blue, as though no psychological factors were involved, but psychodynamic exploration frequently reveals a clear psychological trigger for the panic attack. Although panic attacks are correlated neuro-physiologically with the locus ceruleus, the onset of panic is generally related to environmental or psychological factors. Patients with panic disorder have a higher incidence of stressful life events (particularly loss) than control subjects in the months before the onset of panic disorder. Moreover, the patients typically experience greater distress about life events than control subjects do. The hypothesis that stressful psychological events produce neurophysiological changes in panic disorder is supported by a study of female twins. Separation from the mother early in life was clearly more likely to result in panic disorder than was paternal separation in the cohort of 1,018 pairs of female twins. Another etiological factor in adult female patients appears to be childhood physical and sexual abuse. Approximately 60 percent of women with panic disorder have a history of childhood sexual abuse compared with 31 percent of women with other anxiety disorders. Further support for psychological mechanisms in panic disorder can be inferred from a study of panic disorder in which patients received successful treatment with cognitive therapy. Before the therapy, the patients responded to panic attack induction with lactate. After successful cognitive therapy, lactate infusion no longer produced a panic attack. The research indicates that the cause of panic attacks is likely to involve the unconscious meaning of stressful events and that the pathogenesis of the panic attacks may be related to neurophysiological factors

triggered by the psychological reactions. Psychodynamic clinicians should always thoroughly investigate possible triggers whenever assessing a patient with panic disorder.

DIAGNOSIS

A panic attack is a sudden period of intense fear or apprehension that may last from minutes to hours. Panic attacks can occur in mental disorders other than panic disorder, particularly in specific phobia, social phobia, and PTSD. Unexpected panic attacks occur at any time and are not associated with any identifiable situational stimulus, but panic attacks need not be unexpected. Attacks in patients with social and specific phobias are usually expected or cued to a recognized or specific stimulus. Some panic attacks do not fit easily into the distinction between unexpected and expected, and these attacks are referred to as situationally predisposed panic attacks. They may or may not occur when a patient is exposed to a specific trigger, or they may occur either immediately after exposure or after a considerable delay. Panic Disorder Some community surveys have indicated that panic attacks are common, and a major issue in developing diagnostic criteria for panic disorder was determining a threshold number or frequency of panic attacks required to meet the diagnosis. Setting the threshold too low results in the diagnosis of panic disorder in patients who do not have an impairment from an occasional panic attack; setting the threshold too high results in a situation in which patients who are impaired by their panic attacks do not meet the diagnostic criteria.

CLINICAL FEATURES

The first panic attack is often completely spontaneous, although panic attacks occasionally follow excitement, physical exertion, sexual activity, or moderate emotional trauma. Clinicians should attempt to ascertain any habit or situation that commonly precedes a patient's panic attacks. Such activities may include the use of caffeine, alcohol, nicotine, or other substances; unusual patterns of sleeping or eating; and specific environmental settings, such as harsh lighting at work. The attack often begins with a 10-minute period of rapidly increasing symptoms. The major mental symptoms are extreme fear and a sense of impending death and doom. Patients usually cannot name the source of their fear; they may feel confused and have trouble concentrating. The physical signs often include tachycardia, palpitations, dyspnea, and sweating. Patients often try to leave whatever situation they are in to seek help. The attack generally lasts 20

to 30 minutes and rarely more than an hour. A formal mental status examination during a panic attack may reveal rumination, difficulty speaking (e.g., stammering), and impaired memory. Patients may experience depression or depersonalization during an attack. The symptoms can disappear quickly or gradually. Between attacks, patients may have anticipatory anxiety about having another attack. The differentiation between anticipatory anxiety and generalized anxiety disorder can be difficult, although patients with pain disorder with anticipatory anxiety can name the focus of their anxiety. Somatic concerns of death from a cardiac or respiratory problem may be the major focus of patients' attention during panic attacks. Patients may believe that the palpitations and chest pain indicate that they are about to die. As many as 20 percent of such patients actually have syncopal episodes during a panic attack. The patients may be seen in emergency departments as young (20s), physically healthy persons who nevertheless insist that they are about to die from a heart attack. Rather than immediately diagnosing hypochondriasis, the emergency department physician should consider a diagnosis of panic disorder. Hyperventilation can produce respiratory alkalosis and other symptoms. The age-old treatment of breathing into a paper bag sometimes helps because it decreases alkalosis.

Associated Symptoms Depressive symptoms are often present in panic disorder, and in some patients, a depressive disorder coexists with the panic disorder. Some studies have found that the lifetime risk of suicide in persons with panic disorder is higher than it is in persons with no mental disorder. Clinicians should be alert to the risk of suicide. In addition to agoraphobia, other phobias and OCD can coexist with panic disorder. The psychosocial consequences of panic disorder, in addition to marital discord, can include time lost from work, financial difficulties related to the loss of work, and alcohol and other substance abuse.

DIFFERENTIAL DIAGNOSIS

Panic Disorder

Medical Disorders Panic disorder must be differentiated from a number of medical conditions that produce similar symptomatology. Panic attacks are associated with a variety of endocrinological disorders, including both hypo- and hyperthyroid states, hyperparathyroidism, and pheochromocytomas. Episodic hypoglycemia associated with insulinomas can also produce panic-like states, as can primary neuropathological processes. These include seizure

disorders, vestibular dysfunction, neoplasms, or the effects of both prescribed and illicit substances on the CNS. Finally, disorders of the cardiac and pulmonary systems, including arrhythmias, chronic obstructive pulmonary disease, and asthma, can produce autonomic symptoms and accompanying crescendo anxiety that can be difficult to distinguish from panic disorder. Clues of an underlying medical aetiology to panic-like symptoms include the presence of atypical features during panic attacks, such as ataxia, alterations in consciousness, or bladder dyscontrol; onset of panic disorder relatively late in life; and physical signs or symptoms indicative of a medical disorder. Mental Disorders Panic disorder also must be differentiated from a number of psychiatric disorders, particularly other anxiety disorders. Panic attacks occur in many anxiety disorders, including social and specific phobia, Panic may also occur in PTSD and OCD. The key to correctly diagnosing panic disorder and differentiating the condition from other anxiety disorders involves the documentation of recurrent spontaneous panic attacks at some point in the illness. Differentiation from generalized anxiety disorder can also be difficult. Classically, panic attacks are characterized by their rapid onset (within minutes) and short duration (usually less than 10 to 15 minutes), in contrast to the anxiety associated with generalized anxiety disorder, which emerges and dissipates more slowly. Making this distinction can be difficult, however, because the anxiety surrounding panic attacks can be more diffuse and slower to dissipate than is typical. Because anxiety is a frequent concomitant of many other psychiatric disorders, including the psychoses and affective disorders, discrimination between panic disorder and a multitude of disorders can also be difficult. Specific and Social Phobias Sometimes it is diffcult to distinguish between panic disorder, on the one hand, and specific and social phobias, on the other hand. Some patients who experience a single panic attack in a specific setting (e.g., an elevator) may go on to have long-lasting avoidance of the speciOc setting, regardless of whether they ever have another panic attack. These patients meet the diagnostic criteria for a speciOc phobia, and clinicians must use their judgment about what is the most appropriate diagnosis. In another example, a person who experiences one or more panic attacks may then fear speaking in public. Although the clinical picture is almost identical to the clinical picture in social phobia, a diagnosis of social phobia is excluded because the avoidance of the public situation is based on fear of having a panic attack rather than on fear of the public speaking itself.

COURSE AND PROGNOSIS

Panic disorder usually has its onset in late adolescence or early adulthood, although onset during childhood, early adolescence, and midlife does occur. Some data implicate increased psychosocial stressors with the onset of panic disorder, although no psychosocial stressor can be definitely identified in most cases. Panic disorder, in general, is a chronic disorder, although its course is variable, both among patients and within a single patient. The available long-term follow-up studies of panic disorder are difficult to interpret because they have not controlled for the effects of treatment. Nevertheless, about 30 to 40 percent of patients seem to be symptom free at long-term follow-up, about 50 percent have symptoms that are sufficiently mild not to affect their lives significantly, and about 10 to 20 percent continue to have significant symptoms. After the first one or two panic attacks, patients may be relatively unconcerned about their condition; with repeated attacks, however, the symptoms may become a major concern. Patients may attempt to keep the panic attacks secret and thereby cause their families and friends concern about unexplained changes in behavior. The frequency and severity of the attacks can fluctuate. Panic attacks can occur several times in a day or less than once a month. Excessive intake of caffeine or nicotine can exacerbate the symptoms. Depression can complicate the symptom picture in anywhere from 40 to 80 percent of all patients, as estimated by various studies. Although the patients do not tend to talk about suicidal ideation, they are at increased risk for committing suicide. Alcohol and other substance dependence occurs in about 20 to 40 percent of all patients, and OCD may also develop. Family interactions and performance in school and at work commonly suffer. Patients with good premorbid functioning and symptoms of brief duration tend to have good prognoses.

TREATMENT

With treatment, most patients exhibit dramatic improvement in the symptoms of panic disorder and agoraphobia. The two most effective treatments are pharmacotherapy and cognitive-behavioral therapy. Family and group therapy may help affected patients and their families adjust to the patient's disorder and to the psychosocial difficulties that the disorder may have precipitated. Pharmacotherapy Overview. Alprazolam (Xanax) and paroxetine (Paxil) are the two drugs approved by the US Food and Drug Administration (FDA) for the treatment of panic disorder. In general, experience is showing superiority of the selective serotonin reuptake inhibitors (SSRIs) and clomipramine (Anafranil)

over the benzodiazepines, monoamine oxidase inhibitors (MAOIs), and tricyclic and tetracyclic drugs in terms of effectiveness and tolerance of adverse effects. Some reports have suggested a role for venlafaxine (Effexor), and buspirone (BuSpar) has been suggested as an additive medication in some cases. Venlafaxine is approved by the FDA for treatment of generalized anxiety disorder and may be useful in panic disorder combined with depression. β-adrenergic receptor antagonists have not been found to be particularly useful for panic disorder. A conservative approach is to begin treatment with paroxetine, sertraline (Zoloft), citalopram (Celexa), or fluvoxamine (Luvox) in isolated panic disorder. If rapid control of severe symptoms is desired, a brief course of alprazolam should be initiated concurrently with the SSRI followed by slowly tapering use of the benzodiazepine. In long-term use, fluoxetine (Prozac) is an effective drug for panic with comorbid depression, although its initial activating properties may mimic panic symptoms for the first several weeks, and it may be poorly tolerated on this basis. Clonazepam (Klonopin) can be prescribed for patients who anticipate a situation in which panic may occur (0.5 to 1 mg as required). Selective Serotonin Reuptake Inhibitors. All SSRIs are effective for panic disorder. Paroxetine and paroxetine CR have sedative effects and tend to calm patients immediately, which leads to greater compliance and less discontinuation, but this must be weighed against its weight gain potential. Citalopram, escitalopram (Lexapro), fluvoxamine, and sertraline are the next best tolerated. Anecdotal reports suggest that patients with panic disorder are particularly sensitive to the activating effects of SSRIs, particularly fluoxetine, so they should be given initially at small dosages and titrated up slowly. At therapeutic dosages—for example, 20 mg a day of paroxetine—some patients may experience increased sedation. One approach for patients with panic disorder is to give 5 or 10 mg a day of paroxetine or 12.5 to 25 mg of paroxetine CR for 1 to 2 weeks and then increase the dosage by 10 mg of paroxetine or 12.5 mg of paroxetine CR a day every 1 to 2 weeks to a maximum of 60 mg of paroxetine or 62.5 mg of paroxetine CR. If sedation becomes intolerable, then taper the paroxetine dosage down to 10 mg a day of paroxetine or 12.5 mg of paroxetine CR and switch to fluoxetine at 10 mg a day and titrate upward slowly. Other strategies can be used based on the experience of the clinician. Benzodiazepines. Benzodiazepines have the most rapid onset of action against panic, often within the first week, and they can be used for long periods without the development of tolerance to the antipanic effects. Alprazolam has been the most widely used benzodiazepine for panic disorder, but controlled studies have

demonstrated equal efficacy for lorazepam (Ativan), and case reports have also indicated that clonazepam may be effective. Some patients use benzodiazepines as needed when faced with a phobic stimulus. Benzodiazepines can reasonably be used as the first agent for treatment of panic disorder while a serotonergic drug is being slowly titrated to a therapeutic dose. After 4 to 12 weeks, benzodiazepine use can be slowly tapered (over 4 to 10 weeks) while the serotonergic drug is continued. The major reservation among clinicians regarding the use of benzodiazepines for panic disorder is the potential for dependence, cognitive impairment, and abuse, especially after long-term use. Patients should be instructed not to drive, abstain from alcohol or other CNS depressant medications, and avoid operating dangerous equipment while taking benzodiazepines. Whereas benzodiazepines elicit a sense of well-being, discontinuation of benzodiazepines produces a well-documented and unpleasant withdrawal syndrome. Anecdotal reports and small case series have indicated that addiction to alprazolam is one of the most difficult to overcome, and it may require a comprehensive program of detoxification. Benzodiazepine dosage should be tapered slowly, and all anticipated withdrawal effects should be thoroughly explained to the patient. Tricyclic and Tetracyclic Drugs. At the present time, SSRIs are considered the first-line agents for the treatment of panic disorder. Data, however, show that among tricyclic drugs, clomipramine and imipramine (Tofranil) are the most effective in the treatment of panic disorder. Clinical experience indicates that the dosages must be titrated slowly upward to avoid overstimulation and that the full clinical benefit requires full dosages and may not be achieved for 8 to 12 weeks. Some data support the efficacy of desipramine (Norpramin), and less evidence suggests a role for maprotiline (Ludiomil), trazodone (Desyrel), nortriptyline (Pamelor), amitriptyline (Elavil), and doxepin (Adapin). Tricyclic drugs are less widely used than SSRIs because the tricyclic drugs generally have more severe adverse effects at the higher dosages required for effective treatment of panic disorder. Monoamine Oxidase Inhibitors. The most robust data support the effectiveness of phenelzine (Nardil), and some data also support the use of tranylcypromine (Parnate). MAOIs appear less likely to cause overstimulation than either SSRIs or tricyclic drugs, but they may require full dosages for at least 8 to 12 weeks to be effective. The need for dietary restrictions has limited the use of MAOIs, particularly since the appearance of the SSRIs. Treatment Non-response. If patients fail to respond to one class of drugs, another should be tried. Recent data support the effectiveness of venlafaxine. The combination of an SSRI or a

tricyclic drug and a benzodiazepine or of an SSRI and lithium or a tricyclic drug can be tried. Case reports have suggested the effectiveness of carbamazepine (Tegretol), valproate (Depakene), and calcium channel inhibitors. Buspirone may have a role in the augmentation of other medications but has little effectiveness by itself. Clinicians should reassess the patient, particularly to establish the presence of comorbid conditions such as depression, alcohol use, or other substance use. Duration of Pharmacotherapy. When it becomes effective, pharmacological treatment should generally continue for 8 to 12 months. Data indicate that panic disorder is a chronic, perhaps lifelong, condition that recurs when treatment is discontinued. Studies have reported that 30 to 90 percent of patients with panic disorder who have had successful treatment have a relapse when their medication is discontinued. Patients may be likely to relapse if they have been given benzodiazepines and the benzodiazepine therapy is terminated in a way that causes withdrawal symptoms. Cognitive and Behavior Therapies Cognitive and behavior therapies are effective treatments for panic disorder. Various reports have concluded that cognitive and behavior therapies are superior to pharmacotherapy alone; other reports have concluded the opposite. Several studies and reports have found that the combination of cognitive or behavior therapy with pharmacotherapy is more effective than either approach alone. Several studies that included long-term follow-up of patients who received cognitive or behavior therapy indicate that the therapies are effective in producing long-lasting remission of symptoms. Cognitive Therapy. The two major foci of cognitive therapy for panic disorder are instruction about a patient's false beliefs and information about panic attacks. The instruction about false beliefs centers on the patient's tendency to misinterpret mild bodily sensations as indicating impending panic attacks, doom, or death. The information about panic attacks includes explanations that when panic attacks occur, they are time limited and not life threatening.

E. Agoraphobia.

Agoraphobia refers to a fear of or anxiety regarding places from which escape might be difficult. It can be the most disabling of the phobias because it can significantly interfere with a person's ability to function in work and social situations outside the home. In the United States, most researchers of panic disorder believe that agoraphobia almost always develops as a complication in patients with panic disorder. That is, the fear of having a panic attack in a public place from which escape would be formidable is thought to cause the

agoraphobia. Although agoraphobia often coexists with panic disorder, DSM-5 classifies agoraphobia as a separate condition that may or may not be comorbid with panic disorder.

HISTORY

The term agoraphobia was coined in 1871 to describe the condition of patients who were afraid to venture alone into public places. The term is derived from the Greek words agora and phobos, meaning "fear of the marketplace."

EPIDEMIOLOGY

The lifetime prevalence of agoraphobia is somewhat controversial, varying between 2 to 6 percent across studies. According to the DSM-5, persons older than age 65 years have a 0.4 percent prevalence rate of agoraphobia, but this may be a low estimate. The major factor leading to this wide range of estimates relates to disagreement about the conceptualization of agoraphobia's relationship to panic disorder. Although studies of agoraphobia in psychiatric settings have reported that at least three fourths of the affected patients have panic disorder as well, studies of agoraphobia in community samples have found that as many as half the patients have agoraphobia without panic disorder. The reasons for these divergent findings are unknown but probably involve differences in ascertainment techniques. In many cases, the onset of agoraphobia follows a traumatic event.

DIAGNOSIS AND CLINICAL FEATURES

The DSM-5 diagnostic criteria for agoraphobia stipulates marked fear or anxiety about at least one situation from two or more of five situation groups:

(1) using public transportation (e.g., bus, train, cars, planes),

(2) in an open space (e.g., park, shopping center, parking lot),

(3) in an enclosed space (e.g., stores, elevators, theaters),

(4) in a crowd or standing in line, or

(5) alone outside of the home.

The fear or anxiety must be persistent and last at least 6 months Patients with agoraphobia rigidly avoid situations in which it would be difficult to obtain help. They prefer to be accompanied by a friend or a family member in busy

streets, crowded stores, closed-in spaces (e.g., tunnels, bridges, and elevators), and closed-in vehicles (e.g., subways, buses, and airplanes). Patients may insist that they be accompanied every time they leave the house. The behavior can result in marital discord, which may be misdiagnosed as the primary problem. Severely affected patients may simply refuse to leave the house. Particularly before a correct diagnosis is made, patients may be terrified that they are going crazy.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis for agoraphobia includes all the medical disorders that can cause anxiety or depression. The psychiatric differential diagnosis includes major depressive disorder, schizophrenia, paranoid personality disorder, avoidance personality disorder, and dependent personality disorder.

COURSE AND PROGNOSIS

Most cases of agoraphobia are thought to be caused by panic disorder. When the panic disorder is treated, the agoraphobia often improves with time. For rapid and complete reduction of agoraphobia, behavior therapy is sometimes indicated. Agoraphobia without a history of panic disorder is often incapacitating and chronic, and depressive disorders and alcohol dependence often complicate its course.

TREATMENT

Pharmacotherapy- Benzodiazepines. Benzodiazepines have the most rapid onset of action against panic. Some patients use them as needed when faced with a phobic stimulus. Alprazolam (Xanax) and lorazepam (Ativan) are the most commonly prescribed benzodiazepines. Clonazepam (Klonopin) has also been shown to be effective. The major reservations among clinicians regarding the use of benzodiazepines are the potential for dependence, cognitive impairment, and abuse, particularly with long-term use. However, when used appropriately under medical supervision, benzodiazepines are efficacious and generally well tolerated. The most common side effects are mild dizziness and sedation, both of which are generally attenuated by time or change of dose. Caution must be exercised when using heavy or dangerous machinery or when driving, especially when first starting the medication or when the dose is changed. Benzodiazepines should not be used in combination with alcohol because they can intensify its effects. Benzodiazepines are also best avoided in individuals

with histories of alcohol or substance abuse unless there are compelling reasons, such as failure to respond to other classes of medications. Selective Serotonin Reuptake Inhibitors. SSRIs have been shown to help reduce or prevent relapse from various forms of anxiety, including agoraphobia. Effective doses are essentially the same as for the treatment of depression, although it is customary to start with lower initial doses than in depression to minimize an initial anxiolytic effect, which is almost always short lived, and to titrate upward somewhat slower toward a therapeutic dose. The main advantages of SSRIs antidepressants include their improved safety profile in overdose and more tolerable side-effect burden. Common side effects of most SSRIs are sleep disturbance, drowsiness, light headedness, nausea, and diarrhea; many of these adverse effects improve with continued use. Another commonly reported side effect of SSRIs is sexual dysfunction (i.e., decreased libido, delayed ejaculation in men, delayed orgasm in women), which rarely improves with time or switching among SSRIs (or from an SSRI to a serotonin-norepinephrine reuptake inhibitor [SNRI]). Proposed strategies to combat sexual dysfunction in patients taking SSRIs include adjunctive use of yohimbine (Yocon), bupropion (Wellbutrin), or mirtazapine (Remeron); dose reduction; or adjunctive use of sildenafil (Viagra). Another issue to be considered when prescribing an SSRI is the possibility of a discontinuation syndrome if these medications are stopped abruptly. Commonly reported symptoms of this condition, which tend to occur 2 to 4 days after medication cessation, include increased anxiety, irritability, tearfulness, dizziness or lightheadedness, malaise, sleep disturbance, and concentration difficulties. This discontinuation syndrome is most common among SSRIs with shorter half-lives (e.g., paroxetine [Paxil]). Tricyclic and Tetracyclic Drugs. Although SSRIs are considered the first-line agents for treatment of panic disorders with or without agoraphobia, the tricyclic drugs clomipramine (Anafranil) and imipramine (Tofranil) are the most effective in the treatment of these disorders. Dosages must be titrated slowly upward to avoid overstimulation (e.g., “jitteriness” syndrome), and the full clinical benefit requires full dosages and may not be achieved for 8 to 12 weeks. Therapeutic drug monitoring (TDM) may be useful to ensure that the patient is on an adequate dose of medication while avoiding issues of toxicity. The other adverse effects to these antidepressants are related to their effects on seizure threshold, as well as anticholinergic and potentially harmful cardiac effects, particularly in overdose. Psychotherapy Supportive Psychotherapy. Supportive psychotherapy involves the use of psychodynamic concepts and a therapeutic

alliance to promote adaptive coping. Adaptive defenses are encouraged and strengthened, and maladaptive ones are discouraged. The therapist assists in reality testing and may offer advice regarding behavior. Insight-Oriented Psychotherapy. In insight-oriented psychotherapy, the goal is to increase the patient's development of insight into psychological conflicts that, if unresolved, can manifest as symptomatic behavior. Behavior Therapy. In behavior therapy, the basic assumption is that change can occur without the development of psychological insight into underlying causes. Techniques include positive and negative reinforcement, systematic desensitization, flooding, implosion, graded exposure, response prevention, stop thought, relaxation techniques, panic control therapy, self-monitoring, and hypnosis. Cognitive Therapy. This is based on the premise that maladaptive behavior is secondary to distortions in how people perceive themselves and in how other perceive them. Treatment is short term and interactive, with assigned homework and tasks to be performed between sessions that focus on correcting distorted assumptions and cognitions. The emphasis is on confronting and examining situations that elicit interpersonal anxiety and associated mild depression. Virtual Therapy. Computer programs have been developed that allow patients to see themselves as avatars who are then placed in open or crowded spaces (e.g., a supermarket). As they identify with the avatars in repeated computer sessions, they are able to master their anxiety through deconditioning.

F. Generalized anxiety disorder. Involves excessive worry about everyday life circumstances, events, or conflicts. The symptoms may fluctuate and overlap with other medical and psychiatric disorders (depressive and other anxiety disorders). The anxiety is difficult to control, is subjectively distressing, and produces impairments in important areas of a person's life. Occurs in children and adults with a lifetime prevalence of 45%. Ratio of women to men is 2:1.

G. Substance/medication-induced anxiety disorder. Panic attacks or anxiety developing during or soon after intoxication of or withdrawal from a substance/medication that is known to produce such symptoms. (Red flags include delirium and panic attacks at baseline.) A wide range of substances can cause anxiety symptoms that are often associated with intoxication or withdrawal states. Most common contributing drugs include amphetamine, cocaine, and caffeine, as well as LSD and MDMA. Primary treatment includes removal of casually involved substance.

H. Anxiety disorder due to another medical condition. Many medical disorders are associated with anxiety. It is fairly a common condition and DSM-5 suggests that clinicians specify whether the disorder is characterized by symptoms of generalized anxiety or panic attacks. A wide range of medical and neurologic conditions can cause anxiety symptoms.

I. Other specified anxiety disorder. Anxiety disorder not meeting full criteria (e.g., short duration, culturally specific symptoms, etc.).

J. Unspecified anxiety disorder. This condition does not meet criteria for any of the above-listed anxiety disorders despite resulting in significant social and occupational dysfunction. There is incomplete information for diagnoses. This may include the following conditions.

1. Adjustment disorder with anxiety. This applies to the patient with an obvious stressor in whom excessive anxiety develops within 3 months and is expected to last no longer than 6 months. It may occur as a reaction to illness, rejection, or loss of a job, especially if it is experienced as a defeat or failure.

2. Anxiety secondary to another psychiatric disorder. Seventy percent of depressed patients have anxiety. Patients with psychoses— schizophrenia, mania, or brief psychotic disorder—often exhibit anxiety (psychotic anxiety). Anxiety is common in delirium and in dementia (catastrophic reaction).

3. Situational anxiety. Effects of a stressful situation temporarily overwhelm the ability to cope. This may occur in minor situations if it brings to mind past overwhelming stress. 4. Existential anxiety. This involves fears of helplessness, aging, loss of control, and loss of others in addition to the fear of death and dying.

5. Separation anxiety and stranger anxiety. Regressed adults, including some who are medically ill, may manifest anxiety when separated from loved ones or when having to react to staff in a hospital. Separation anxiety disorder occurs in some young children when going to school for the first time. It is a normal reaction in infants and children until about 2½ years of age.

6. Anxiety related to loss of self-control. In circumstances in which control must be surrendered, such as medical illness or hospitalization, patients with a need to feel in control may be very threatened. Loss of autonomy at work can precipitate anxiety.

7. Anxiety related to dependence or intimacy. If past dependency needs were not met or resolved, a patient can be anxious being in a close relationship, which involves some dependence, or being a patient in a hospital, which involves giving up control.

8. Anxiety related to guilt and punishment. If a patient expects punishment for imagined or real misdeeds, he or she may feel anxiety and the punishment may be actively sought or even self-inflicted.

K. Mixed anxiety–depressive disorder. This disorder describes patients with both anxiety and depressive symptoms that do not meet the diagnostic criteria for either an anxiety disorder or a mood disorder. The diagnosis is sometimes used in primary care settings and is used in Europe; sometimes called neurasthenia.

III. Epidemiology

The anxiety disorders make up the most common group of psychiatric disorders. One in four persons has met the diagnostic criteria for at least one of the above-listed anxiety disorders, and there is a 12-month prevalence rate of about 17%. Women are more likely to have an anxiety disorder than are men. The prevalence of anxiety disorders decreases with higher socioeconomic status. An epidemiologic overview of anxiety disorders as well as obsessive–compulsive disorder and posttraumatic stress disorder.

IV. Etiology

A. Biologic

1. Anxiety involves an excessive autonomic reaction with increased sympathetic tone.

2. The release of catecholamines is increased with the increased production of norepinephrine metabolites (e.g., 3-methoxy-4- hydroxyphenylglycol).

3. Decreased rapid eye movement (REM) latency and stage IV sleep (similar to depression) may develop.

4. Decreased levels of γ-aminobutyric acid (GABA) cause central nervous system (CNS) hyperactivity (GABA inhibits CNS irritability and is widespread throughout the brain).

5. Alterations in serotonergic system and increased dopaminergic activity are associated with anxiety.

6. Activity in the temporal cerebral cortex is increased.

7. The locus coeruleus, a brain center of noradrenergic neurons, is hyperactive in anxiety states, especially panic attacks.

8. Recent studies also suggest a role for neuropeptides (substance P, CRF, and cholecystokinin), but currently there are no agents available for these targets.

9. Hyperactivity and dysregulation in the amygdala may be associated with social anxiety.

B. Psychoanalytic. According to Freud, unconscious impulses (e.g., sex or aggression) threaten to burst into consciousness and produce anxiety. Anxiety is related developmentally to childhood fears of disintegration that derive from the fear of an actual or imagined loss of a love object or the fear of bodily harm (e.g., castration). Freud used the term signal anxiety to describe anxiety not consciously experienced but that triggers defense mechanisms used by the person to deal with a potentially threatening situation.

C. Learning theory

1. Anxiety is produced by continued or severe frustration or stress. The anxiety then becomes a conditioned response to other situations that are less severely frustrating or stressful.

2. It may be learned through identification and imitation of anxiety patterns in parents (social learning theory).

3. Anxiety is associated with a naturally frightening stimulus (e.g., accident). Subsequent displacement or transference to another stimulus through conditioning produces a phobia to a new and different object or situation.

4. Anxiety disorders involve faulty, distorted, or counterproductive patterns of cognitive thinking.

D. Genetic studies

1. Half of patients with panic disorder have one affected relative.

2. About 5% of persons with high levels of anxiety have a polymorphic variant of the gene associated with serotonin transporter metabolism.

V. Psychological Tests

A. Rorschach test

1. Anxiety responses include animal movements, unstructured forms, and heightened color.

2. Phobic responses include anatomic forms or bodily harm.

3. Obsessive–compulsive responses include over attention to detail.

B. Thematic apperception test

1. Increased fantasy productions may be present.

2. Themes of aggression and sexuality may be prominent.

3. Feelings of tension may be evident.

C. Bender–Gestalt

1. No changes indicative of brain damage are apparent.

2. Use of small area may be manifested in obsessive–compulsive disorder.

3. Productions may spread out on the page in anxiety states.

D. Draw-a-Person

1. Attention to head and general detailing may be noted in obsessive–compulsive disorder.

2. Body image distortions may be present in phobias.

3. Rapid drawing may be evident in anxiety disorders.

E. Minnesota Multiphasic Personality Inventory-2. High hypochondriasis, psychasthenia, hysteria scales in anxiety.

VI. Laboratory Tests

A. No specific laboratory tests for anxiety.

B. Experimental infusion of lactate increases norepinephrine levels and produces anxiety in patients with panic disorder.

VII. Pathophysiology and Brain-Imaging Studies

A. No consistent pathognomonic changes.

B. In obsessive–compulsive disorder, positron emission tomography (PET) reveals decreased metabolism in the orbital gyrus, caudate nuclei, and cingulate gyrus.

C. In generalized anxiety disorder and panic states, PET reveals increased blood flow in the right parahippocampus in the frontal lobe.

D. Magnetic resonance imaging (MRI) has shown increased ventricular size in some cases, but findings are not consistent.

E. Right temporal atrophy is seen in some panic disorder patients, and cerebral vasoconstriction is often present in anxiety.

F. Mitral valve prolapse is present in 50% of patients with panic disorder, but clinical significance unknown.

G. Nonspecific electroencephalogram (EEG) changes may be noted.

H. Dexamethasone suppression test does not suppress cortisol in some obsessive–compulsive patients.

I. Panic-inducing substances include carbon dioxide, sodium lactate, methyl-chloro

phenyl-piperazine (mCPP), carbolines, GABAB receptor antagonists, caffeine, isoproterenol, and yohimbine (Yocon).

ICD-10 criteria Generalized anxiety disorder

A. A period of at least six months with prominent tension, worry and feelings of apprehension, about every-day events and problems.

B. At least four symptoms out of the following list of items must be present, of which at least one from items (1) to (4).

Autonomic arousal symptoms

(1) Palpitations or pounding heart, or accelerated heart rate.
(2) Sweating.
(3) Trembling or shaking.
(4) Dry mouth (not due to medication or dehydration).

Symptoms concerning chest and abdomen

(5) Difficulty breathing

ICD-10 criteria Generalized anxiety disorder Cont:

Symptoms concerning brain and mind

(9) Feeling dizzy, unsteady, faint or light-headed.

(10) Feelings that objects are unreal (derealization), or that one's self is distant or "not really here" (depersonalization).

(11) Fear of losing control, going crazy, or passing out.

(12) Fear of dying.

General symptoms

(13) Hot flushes or cold chills.

(14) Numbness or tingling sensations.

Symptoms of tension

(15) Muscle tension or aches and pains.

(16) Restlessness and inability to relax.

(17) Feeling keyed up, or on edge, or of mental tension.

(18) A sensation of a lump in the throat, or difficulty with swallowing.

Symptoms of a Panic Attack

- Sweating, shortness of breath or hyperventilation
- Racing heart, chest pain or discomfort
- Feeling dizzy, light-headed or faint
- Choking or smothering sensation
- Numbness or tingling
- Chills or hot flashes
- Trembling or shaking
- Nausea or upset stomach
- Feeling unreal or detached from one's surroundings
- Fear of losing control, "going crazy" or dying

E MS

VIII. Differential Diagnosis

A. Depressive disorders. Fifty percent to 70% of depressed patients exhibit anxiety or obsessive brooding; 20% to 30% of primarily anxious patients also experience depression.

B. Schizophrenia. Schizophrenic patients may be anxious and have severe obsessions in addition to or preceding the outbreak of hallucinations or delusions.

C. Bipolar I disorder. Massive anxiety may occur during a manic episode.

D. Atypical psychosis (psychotic disorder not otherwise specified). Massive anxiety is present, in addition to psychotic features.

E. Adjustment disorder with anxiety. Patient has a history of a psychosocial stressor within 3 months of onset.

F. Medical and neurologic conditions. A secondary anxiety disorder is caused by a specific medical or biologic factor. Undiagnosed hyperthryroidism is a frequent cause.

G. Substance-related disorders. Panic or anxiety is often associated with intoxication (especially caffeine, cocaine, amphetamines, hallucinogens) and withdrawal states

H. Cognitive disorder. Severe anxiety may interfere with cognition and impairments may occur; however, they remit when the anxiety is diminished, unlike the cognitive defects in dementia.

IX. Course and Prognosis

A. Separation anxiety disorder

1. Starts as early as 1 year of age.

2. Periods of exacerbations and remissions.

3. Adults may have social and occupational dysfunction.

4. Overall good prognosis with 96% remission.

B. Selective mutism

1. Shy, anxious, and risk for depression.

2. Academic difficulties.

3. Increased risk for comorbid anxiety disorders.

4. Good treatment response to SSRIs.

C. Specific phobia

1. The course tends to be chronic.

2. Phobias may worsen or spread if untreated.

3. Agoraphobia is the most resistant of all phobias.

4. Prognosis is good to excellent with therapy.

D. Social anxiety disorder (social phobia)

1. Onset in late childhood or early adolescence.

2. Chronic but symptoms may remit.

3. Disruption in the individual's life.

4. Good prognosis with pharmacotherapy and psychotherapy.

E. Panic disorder

1. The course is chronic, with remissions and exacerbations.

2. Panic attacks tend to recur two to three times a week.

3. Patients with panic disorder may be at increased risk for committing suicide.

4. The prognosis is good with combined pharmacotherapy and psychotherapy.

F. Agoraphobia

1. Frequently caused by panic disorder.

2. Without panic disorder is chronic and incapacitating.

3. Comorbid alcohol dependence and depressive symptoms.

4. Treatment approach requires pharmacotherapy with CBT and virtual therapy.

G. Generalized anxiety disorder

1. Course is chronic; symptoms may diminish, as the patient gets older.

2. With time, secondary depression may develop. This is not uncommon if the condition is left untreated.

3. With treatment, prognosis is good; over 70% of patients improve with pharmacologic therapy; best when combined with psychotherapy.

X. Treatment

The treatment of anxiety disorders involves both a psychopharmacologic approach as well as psychotherapy (CBT, psychodynamic, time-limited, group, and family therapies).

A. Pharmacologic.

1. Benzodiazepines. These drugs are effective in reducing anxiety generally. In panic disorder, they reduce both the number and intensity of attacks. They are also useful in social and specific **phobia**. Because of concern about physical dependence, benzodiazepines are not prescribed by physicians as often as they should be. With proper psychotherapeutic monitoring, however, they can be used safely for long periods of time without being abused. Discontinuation (withdrawal) syndromes may occur in patients who use these drugs for long periods, but, if the medication is properly withdrawn, signs and symptoms of withdrawal are easily managed. Commonly used drugs in this class include alprazolam (Xanax), clonazepam (Klonopin), diazepam (Valium), and lorazepam (Ativan). Alprazolam is effective in panic disorder and anxiety associated with depression. Alprazolam has been associated with a discontinuation syndrome after as little as 6 to 8 weeks of treatment.

2. Selective serotonin reuptake inhibitors (SSRIs). There are six SSRIs available in the United States that are effective in anxiety disorder: fluoxetine (Prozac), citalopram (Celexa), escitalopram (Lexapro), paroxetine(Paxil), sertraline (Zoloft), and venlafaxine (Effexor). Paroxetine is especially useful for the treatment of panic disorder. SSRIs are safer than the tricyclic drugs because they lack anticholinergic effects and are not as lethal if taken in overdose. The most common side effects are transient nausea, headache, and sexual dysfunction. Some patients, especially those with panic disorder, report an initial increase in anxiety after starting these drugs, which can be controlled with benzodiazepines until the full SSRI effect is felt, usually within 2 to 4 weeks. SSRIs are used with extreme caution in children and adolescents because of reports of agitation and impulsive suicidal acts as side effects of the medication in that population.

3. Tricyclics. Drugs in this class reduce the intensity of anxiety in all the anxiety disorders. Because of their side effect profile (e.g., anticholinergic effects, cardiotoxicity, and potential lethality in overdose [10 times the daily recommended dose can be fatal]), they are not first-line agents. Typical drugs in this class include imipramine (Tofranil), nortryptaline (Aventyl, Pamelor), and clomipramine (Anafranil).

4. Monoamine oxidase inhibitors (MAOIs). MAOIs are effective for the treatment of panic and other anxiety disorders; however, they are not first-line agents because of a major adverse side effect, which is the occurrence of a hypertensive crisis secondary to ingestion of foods containing tyramine. Certain medications such as sympathomimetics and opioids (especially meperidine [Demerol]) must be avoided because if combined with MAOIs, death may ensue. Common drugs in this class include phenelzine (Nardil) and tranylcypromine (Parnate).

5. Other drugs used in anxiety disorders

a. Adrenergic receptor antagonists (beta blockers). Drugs in this class include propranolol (Inderal) and atenolol (Tenormin), which act to suppress the somatic signs of anxiety, particularly panic attacks. They have been reported to be particularly effective in blocking the anxiety of social phobia (e.g., public speaking) when taken as a single dose about 1 hour before the phobic event. Adverse effects include bradycardia, hypotension, and drowsiness. They are not useful in chronic anxiety, unless it is caused by a hypersensitive adrenergic state.

b. Venlafaxine (Effexor). This drug has been found to be effective in the treatment of both generalized anxiety disorder and panic disorder. Because it also acts as an antidepressant, it is of use in mixed states. Its major indication is for the treatment of depression.

c. Buspirone (Buspar). This drug has mild serotonergic effects and is most effective in generalized anxiety disorder rather than in acute states. It is not cross-tolerant with benzodiazepines and cannot be used to treat discontinuation syndromes. It has a slow level of onset and may produce dizziness and headache in some patients.

d. Anticonvulsant anxiolytics. Typical drugs in this class used in the treatment of anxiety disorders include gabapentin (Neurontin), tiagabine (Gabitril), and valproate (Depakene, Depakote). Reports of their efficacy are few and anecdotal; however, they deserve consideration in the treatment of these disorders, especially if panic attacks are present.

B. Psychological

1. Supportive psychotherapy. This approach involves the use of psychodynamic concepts and a therapeutic alliance to promote adaptive coping. Adaptive defenses are encouraged and strengthened, and maladaptive ones are discouraged. The therapist assists in reality testing and may offer advice regarding behavior.

2. Insight-oriented psychotherapy. The goal is to increase the patient's development of insight into psychological conflicts that, if unresolved, can manifest as symptomatic behavior (e.g., anxiety and phobias). This modality is particularly indicated if

(1) anxiety symptoms are clearly secondary to an underlying unconscious conflict,

(2) anxiety continues after behavioral or pharmacologic treatments are instituted,

(3) new anxiety symptoms develop after the original symptoms have resolved (symptom substitution), or

(4) the anxieties are more generalized and less specific.

3. Behavior therapy. The basic assumption is that change can occur without the development of psychological insight into underlying causes. Techniques include positive and negative reinforcement, systematic desensitization, flooding, implosion, graded exposure, response prevention, stop-thought, relaxation techniques, panic control therapy, self-monitoring, and hypnosis. Virtual Therapy Treatment (VRT) uses augmented reality and virtual immersion experience to desensitize the patient against anxiety.

a. Behavior therapy is indicated for clearly delineated, circumscribed, maladaptive behaviors (e.g., panic attacks and phobias).

b. Most current strategies for the treatment of anxiety disorders include a combination of pharmacologic and behavioral interventions.

c. Current thinking generally maintains that although drugs can reduce anxiety early, treatment with drugs alone leads to equally early relapse. The response of patients who are also treated with cognitive and behavioral therapies appears to be significantly and consistently better than the response of those who receive drugs alone.

4. Cognitive therapy. This is based on the premise that maladaptive behavior is secondary to distortions in how people perceive themselves and in how others perceive them. Treatment is short-term and interactive, with assigned homework and tasks to be performed between sessions that focus on correcting distorted assumptions and cognitions. The emphasis is on confronting and

examining situations that elicit interpersonal anxiety and associated mild depression.

5. Group therapy. Groups range from those that provide only support and an increase in social skills to those that focus on relief of specific symptoms to those that are primarily insight-oriented. Groups may be heterogeneous or homogeneous in terms of diagnosis. Homogeneous groups are commonly used in the treatment of such diagnoses as posttraumatic stress disorder, in which therapy is aimed at education about dealing with stress.

Assessment Procedure

Multiple choice questions based assessment after successful completion of theory and practical sessions

SRI LAKSHMI NARAYANA INSTITUTE OF MEDICAL SCIENCE

VALUE ADDED COURSE STUDENT DETAILS

CLASSIFICATION OF ANXIETY AND ITS TREATMENT METHODS- 2ND YEAR STUDENTS

S.No	Register No	Students List	Department	SIGNATURE
1	U13MB261	SHANMUGAVEL.L.N.	Psychiatry	
2	U13MB262	SHEELA PRIYA DARSHINI. N	Psychiatry	
3	U13MB263	SHEIK MOHAMMED YASAR	Psychiatry	
4	U13MB264	SIVASANKARI. B	Psychiatry	
5	U13MB265	SNEHA. T (DOB- 10.12.1994)	Psychiatry	
6	U13MB266	SNEHA. T (DOB- 15.01.1996)	Psychiatry	
7	U13MB267	SOBICA.D	Psychiatry	
8	U13MB268	SOWNDARAVEL.S	Psychiatry	
9	U13MB269	SRI SAKTHI PRIYA.N	Psychiatry	
10	U13MB270	SRILEKHA.D.J	Psychiatry	
11	U13MB271	SRIMUKESH.A	Psychiatry	

12	U13MB272	STEPHEN.A	Psychiatry	A. Steph..
13	U13MB273	SUGANYA.P	Psychiatry	Suganya.
14	U13MB274	SUGESH CHANDRAN.V	Psychiatry	Sugesh chandran
15	U13MB275	SURIYAKUMAR.G	Psychiatry	Suriya.

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Annexure - IV

An Introduction And Assement Of Various Types Of Anxiety Disorders Along With Its Management

MULTIPLE CHOICE QUESTIONS

CANDIDATE AND ASSESSOR INFORMATION

Candidate Name		Assessor Name	
Date of Assessment		Assessor Position	

Course Code: PSYCO1

I. ANSWER ALL THE QUESTIONS

1) An anxiety disorder is:

a) An emotional state identified by panic attacks.

b) An emotional condition classified by excessive checking.

c) Disordered thinking.

d) An excessive or aroused state characterized by feelings of apprehension, uncertainty and fear.

Check your answer

2) Which of the following are common aspects of co-morbidity in anxiety disorders?

a) Physiological symptoms of panic are found not only in panic disorder, but also in the reactions to phobic stimuli in specific phobias.

b) Cognitive biases - such as information processing biases that tend anxious people to selectively attending to threatening stimuli (Mathews & McLeod, 1994) - are common to almost all anxiety disorders.

c) Certain specific early experiences can be found in the aetiology of a number of different anxiety disorders (e.g. physical or sexual abuse during childhood), and experiences such as these may increase an individual's risk of developing several anxiety-based problems.

d) All of the above.

3) Specific phobias are defined as:

a) Excessive worry bouts triggered by a specific object or situation.

b) An abnormal sensitivity to light.

c) An excessive, unreasonable, persistent fear triggered by a specific object or situation.

d) A persistent fear of social situations.

Check your answer

4) In phobia individuals acquire a strong set of phobic beliefs which:

a) Information about why they think the phobia is threatening.

b) How to react when they are in the phobic situation.

c) Appear to control their fear.

d) All of the above.

Check your answer

5) Psychodynamic theory as developed by Freud saw phobias as:

a) Repressed Ego.

b) Repressed Id impulses.

c) Repressed superego.

d) Repressed defence mechanisms.

Check your answer

6) In the famous “Little Albert” study by Watson & Rayner, they attempted to condition in him, a fear of his pet white rat. This was done by:

a) Pairing the unconditioned stimulus (UCS) with unconditioned response (UCR) to produce the conditioned stimulus.

b) Pairing the conditioned response (CR) with conditioned stimulus (CS) which produced the unconditioned stimulus (UCS).

c) Pairing the rat - the conditioned stimulus (CS) with the unconditioned stimulus (UCS) which produced the unconditioned response (UCR) and subsequently conditioned response (CR).

d) Pairing the unconditioned response with the unconditioned stimulus (UCS) to produce the conditioned response (CR).

Check your answer

7) According to conditioning theory Incubation is a phenomenon that should lead to:

a) Distinction.

b) Extinction.

c) Annulation.

d) Conflagration.

Check your answer

8) Which of the following is a predominant evolutionary theory of phobias?:

a) Non-associative fear acquisition.

b) Learned fear responses.

c) Biological preparedness.

d) Specific phobia acquisition.

Check your answer

9) Recent evidence suggests that at least some phobias are closely associated with the emotion of:

a) Anger.

b) Disgust.

c) Exhilaration.

d) Sadness.

Check your answer

10) The disease-avoidance model of animal phobias (Matchett & Davey, 1991) is supported by which of the following?

a) Evidence that a medical approach supports psychological disturbances.

b) Findings that sick individuals avoid animals.

c) All spiders spread disease.

d) Findings that high levels of disgust sensitivity is a vulnerability factor for animal phobias.

Check your answer

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Annexure - IV

An Introduction And Assement Of Various Types Of Anxiety Disorders Along With Its Management

MULTIPLE CHOICE QUESTIONS

CANDIDATE AND ASSESSOR INFORMATION

Candidate Name	SNEHA T	Assessor Name	DR ARUN
Date of Assessment	16 11 2020	Assessor Position	ASSISTANT PROFESSOR

UNIVERSITY REG NO:- 013MB266

6/10

Course Code: PSYCO1

I. ANSWER ALL THE QUESTIONS

1) An anxiety disorder is:

- a) An emotional state identified by panic attacks.
- b) An emotional condition classified by excessive checking.
- c) Disordered thinking.
- d) An excessive or aroused state characterized by feelings of apprehension, uncertainty and fear.

Check your answer

2) Which of the following are common aspects of co-morbidity in anxiety disorders?

- a) Physiological symptoms of panic are found not only in panic disorder, but also in the reactions to phobic stimuli in specific phobias.
- b) Cognitive biases - such as information processing biases that tend anxious people to selectively attending to threatening stimuli (Mathews & McLeod, 1994) - are common to almost all anxiety

-1-

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AND RESEARCH

disorders.

c) Certain specific early experiences can be found in the aetiology of a number of different anxiety disorders (e.g. physical or sexual abuse during childhood), and experiences such as these may increase an individual's risk of developing several anxiety-based problems.

d) All of the above.

3) Specific phobias are defined as:

a) Excessive worry bouts triggered by a specific object or situation.

b) An abnormal sensitivity to light.

c) An excessive, unreasonable, persistent fear triggered by a specific object or situation.

d) A persistent fear of social situations.

Check your answer

4) In phobia individuals acquire a strong set of phobic beliefs which:

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c) Appear to control their fear.

d) All of the above.

Check your answer

5) Psychodynamic theory as developed by Freud saw phobias as:

SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON
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(a) Repressed Ego.

b) Repressed Id impulses.

c) Repressed superego.

d) Repressed defence mechanisms.

Check your answer

6) In the famous "Little Albert" study by Watson & Rayner, they attempted to condition in him, a fear of his pet white rat. This was done by:

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c) Pairing the rat - the conditioned stimulus (CS) with the unconditioned stimulus (UCS) which produced the unconditioned response (UCR) and subsequently conditioned response (CR).

(d) Pairing the unconditioned response with the unconditioned stimulus (UCS) to produce the conditioned response (CR).

Check your answer

7) According to conditioning theory, incubation is a phenomenon that should lead to

a) Distinction.

(b) Extinction.

c) Annulation.

d) Conflagration.

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Check your answer

8) Which of the following is a predominant evolutionary theory of phobias?

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- c) Biological preparedness.
- d) Specific phobia acquisition.

Check your answer

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- c) Exhilaration.
- d) Sadness.

Check your answer

10) The disease-avoidance model of animal phobias (Matchett & Davey, 1991) is supported by which of the following?

- a) Evidence that a medical approach supports psychological disturbances.
- b) Findings that sick individuals avoid animals.

SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

Annexure - IV

An Introduction And Assement Of Various Types Of Anxiety Disorders Along With Its Management

MULTIPLE CHOICE QUESTIONS

CANDIDATE AND ASSESSOR INFORMATION

Candidate Name	SNEHA .T	Assessor Name	DR. ARUN
Date of Assessment	16.12.2020	Assessor Position	ASSISTANT PROFESSOR

UNIVERSITY REG NO:- U13MB265

5/10

Course Code: PSYCO1

I. ANSWER ALL THE QUESTIONS

1) An anxiety disorder is:

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- b) An emotional condition classified by excessive checking.
- c) Disordered thinking.
- d) An excessive or aroused state characterized by feelings of apprehension, uncertainty and fear.

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-1-

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disorders.

c) Certain specific early experiences can be found in the aetiology of a number of different anxiety disorders (e.g. physical or sexual abuse during childhood), and experiences such as these may increase an individual's risk of developing several anxiety-based problems.

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c) Appear to control their fear.

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Check your answer

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- b) Repressed Id impulses.
- c) Repressed superego.
- d) Repressed defence mechanisms.

Check your answer

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- b) Pairing the conditioned response (CR) with conditioned stimulus (CS) which produced the unconditioned stimulus (UCS).
- c) Pairing the rat - the conditioned stimulus (CS) with the unconditioned stimulus (UCS) which produced the unconditioned response (UCR) and subsequently conditioned response (CR).
- d) Pairing the unconditioned response with the unconditioned stimulus (UCS) to produce the conditioned response (CR).

Check your answer

7) According to conditioning theory Incubation is a phenomenon that should lead to:

- a) Distinction.
- b) Extinction.
- c) Annulation.
- d) Conflagration.

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Check your answer

8) Which of the following is a predominant evolutionary theory of phobias?:

- a) Non-associative fear acquisition.
- b) Learned fear responses.
- c) Biological preparedness.
- d) Specific phobia acquisition.

Check your answer

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- c) Exhilaration.
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- a) Evidence that a medical approach supports psychological disturbances.
- b) Findings that sick individuals avoid animals.

Sri Lakshmi Narayana Institute of Medical Sciences

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(Deemed to be University under section 3 of the UGC Act 1956)

CERTIFICATE OF MERIT

This is to certify that SHEELA PRIYADARSHINI N. has actively participated in the Value Added Course on **Awareness, Identification and Classification of Anxiety Disorders and Its Management for final year students** held during July 2020 – Dec 2020 Organized by Sri Lakshmi Narayana Institute of Medical Sciences, Pondicherry- 605 502, India.

Dr. Arun Seetharaman

RESOURCE PERSON

Dr. ARUN SEETHARAMAN, MD.,

Reg. No. 91440

Associate Professor, Psychiatry

Sri Lakshmi Narayana Institute of Medical Sciences

Osudu, Kudapakkam, Puducherry-605 502.

Dr. V.R.Sridhar

Dr. V. R. SRIDHAR, MD.,D.P.M.,

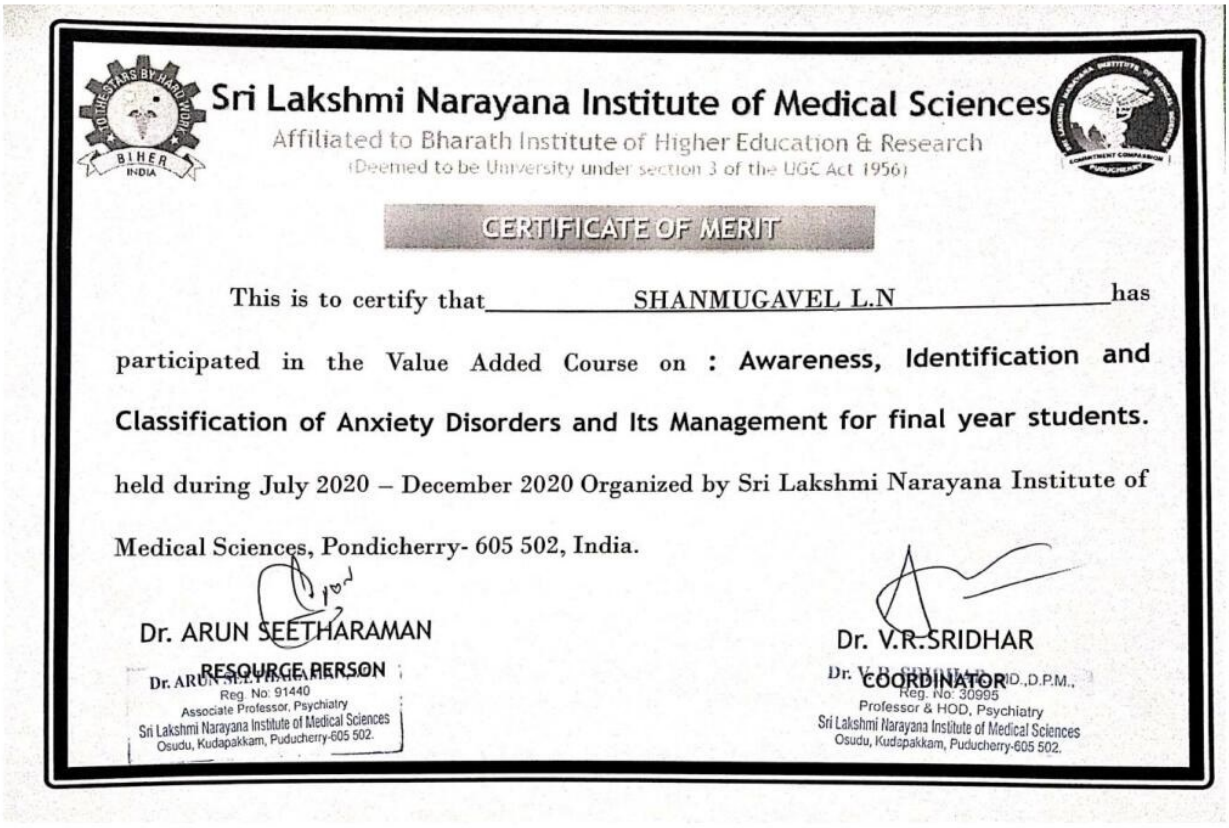
Reg. No. 90[illegible]

COORDINATOR

Professor & HOD, Psychiatry

Sri Lakshmi Narayana Institute of Medical Sciences

Osudu, Kudapakkam, Puducherry-605 502.



Sri Lakshmi Narayana Institute of Medical Sciences

Affiliated to Bharath Institute of Higher Education & Research

(Deemed to be University under section 3 of the UGC Act 1956)

CERTIFICATE OF MERIT

This is to certify that SHANMUGAVEL L.N has participated in the Value Added Course on : **Awareness, Identification and Classification of Anxiety Disorders and Its Management for final year students.** held during July 2020 – December 2020 Organized by Sri Lakshmi Narayana Institute of Medical Sciences, Pondicherry- 605 502, India.

Dr. ARUN SEETHARAMAN
RESOURCE PERSON
Reg. No: 91440
Associate Professor, Psychiatry
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Kudapakkam, Puducherry-605 502.

Dr. V.R.SRIDHAR
COORDINATOR
Reg. No: 30995
Professor & HOD, Psychiatry
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Kudapakkam, Puducherry-605 502.

SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

Student Feedback Form

Course Name: **ANXIETY DISORDERS**
Subject Code: **PSYC01**

Name of Student: _____ Roll No.: _____

We are constantly looking to improve our classes and deliver the best training to you. Your evaluations, comments and suggestions will help us to improve our performance

Sl. NO	Particulars	1	2	3	4	5
1	Objective of the course is clear					
2	Course contents met with your expectations					
3	Lecturer sequence was well planned					
4	Lectures were clear and easy to understand					
5	Teaching aids were effective					
6	Instructors encourage interaction and were helpful					
7	The level of the course					
8	Overall rating of the course	1	2	3	4	5

**** Rating: 5 – Outstanding; 4 - Excellent; 3 – Good; 2– Satisfactory; 1 - Not-Satisfactory***

Suggestions if any:

SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

Student Feedback Form

Course Name: **ANXIETY DISORDERS**
Subject Code: **PSYC01**

Name of Student: SIVASANKARI - B Roll No.: U13MB264

We are constantly looking to improve our classes and deliver the best training to you. Your evaluations, comments and suggestions will help us to improve our performance

Sl. NO	Particulars	1	2	3	4	5
1	Objective of the course is clear				✓	
2	Course contents met with your expectations					✓
3	Lecturer sequence was well planned				✓	
4	Lectures were clear and easy to understand					✓
5	Teaching aids were effective					✓
6	Instructors encourage interaction and were helpful				✓	
7	The level of the course					
8	Overall rating of the course	1	2	3	4 ✓	5

** Rating: 5 – Outstanding; 4 - Excellent; 3 – Good; 2– Satisfactory; 1 - Not-Satisfactory*

Suggestions if any:

Sivasankari
Signature

Date: 31 12 . 2020

SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

Student Feedback Form

Course Name: **ANXIETY DISORDERS**
Subject Code: **PSYC01**

Name of Student: SHEIK MOHAMMED YASAR Roll No.: U13MB263

We are constantly looking to improve our classes and deliver the best training to you. Your evaluations, comments and suggestions will help us to improve our performance

Sl. NO	Particulars	1	2	3	4	5
1	Objective of the course is clear					✓
2	Course contents met with your expectations				✓	
3	Lecturer sequence was well planned					✓
4	Lectures were clear and easy to understand					✓
5	Teaching aids were effective				✓	
6	Instructors encourage interaction and were helpful				✓	
7	The level of the course					✓
8	Overall rating of the course	1	2	3	4	✓5

** Rating: 5 – Outstanding; 4 - Excellent; 3 – Good; 2– Satisfactory; 1 - Not-Satisfactory*

Suggestions if any:

SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

Signature

Date:

Date: 31-12-2020

From

Dr.V.R.Sridhar
Professor and Head,
Department of Psychiatry,
Sri Lakshmi Narayana Institute of Medical Sciences
Bharath Institute of Higher Education and Research,
Chennai.

Through Proper Channel

To

The Dean,
Sri Lakshmi Narayana Institute of Medical Sciences
Bharath Institute of Higher Education and Research,
Chennai.

Sub: Completion of value-added course: <u>Bipolar mood disorders , classification and its management</u>

Dear Sir,

With reference to the subject mentioned above, the department has conducted the value-added course titled: **<u>Bipolar mood disorders , classification and its management</u>**

We solicit your kind action to send certificates for the participants that is attached with this letter. Also, I am attaching the photographs captured during the conduct of the course.

Kind Regards,

Dr. Sridhar

Dr. V. R. SRIDHAR, MD.,D.P.M.,
Reg. No. 30995
Professor & HOD, Psychiatry
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Kudapakkam, Puducherry-605 502.

Encl: Certificates

Photographs


PSYCO1 -ANXIETY DISORDERS AND ITS MANAGEMENT JULY – DECEMBRE 2020
ANXIETY DISORDER
AND ITS
MANAGEMENT