Sri Lakshmi Narayana Institute of Medical Sciences

Date: 01.12.2020

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.

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: Classification of depression and its treatment methods

Dear Sir,

With reference to the subject mentioned above, the department proposes to conduct a value-added course titled: **Classification of depression and its treatment methods** on 4/01/2021. 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. Rajasekhar. K**

The HOD: **Dr. Sridhar.V.R**

The Expert: **Dr. Arun.S**

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]
[Affiliated to Bharath University, Chennai - TN]

Circular

14.12.2020

Sub: Organising Value-added Course: Awareness, Identification and Classification of depression and its treatment methods.

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 depression and its treatment methods for final year students."** The course content and registration form is enclosed below."

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 31 December 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 depression and its treatment methods**

Course Objective:

Awareness on the importance of depression
Awareness On The Contributing Factors To Depression Bio-psychosocial Model Of Approach
Identify diagnostic criteria for depression

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	Resource person	Time	Hours
1.	4.01.2021	The Definition of Depression	Dr.Arun	4-5p.m	1
2.	6.01.2021	Symptomatology of Depression	Dr.Arun	2-3p.m	1
3.	8.01.2021	Psychotic vs. Nonpsychotic Depression	Dr.Arun	4-6p.m	2
4.	11.01.2021	EXPERIMENTAL ASPECTS OF DEPRESSION Biological Studies of Depression	Dr.Arun	4-6p.m	2
5.	13.01.2021	Psychological Studies: Tests of Psychoanalysis	Dr.Arun	4-6p.m	2
6.	18.01.2021	PART III THEORETICAL ASPECTS OF DEPRESSION Theories of Depression	Dr.Arun	4-5p.m	2
7.	20.01.2021	Cognition and Psychopathology	Dr.Arun	4-5P.M	1
8.	22.01.2021	Development of Depression	Dr.Arun	4-5p.m	1
9.	25.01.2021	Course and Prognosis	Dr. Shridhar	4-6p.m	1
10.	27.01.2021	Pharmacological treatment	Dr.Arun	4-6p.m	2
11.	29.01.2021	Somatic Therapy	Dr.Arun	4-6p.m	1
12.	1.02.2021	Psychotherapy	Dr.Arun	4-6p.m	2
13.	3.02.2021	Evaluating Depression	Dr.Arun	2-5p.m	3

		Practical	Dr. Shridhar		
13.	5.02.2021	Cognitive Behavior Therapy	Dr. Shridhar	2-3 PM	1
14.	8.02.2021	Graded Relaxation	Dr. Shridhar	2-3 PM	1
15.	11.02.2021	Cognitive Hypnotherapy	Dr. Shridhar	2-4 PM	2
16.	13.02.2021	Supportive psychotherapy	Dr. Shridhar	2-4 PM	2
17.	15.02.2021	Cognitive therapy	Dr. Shridhar	2-4p.m	2
		Total			30 hrs

REFERENCE BOOKS:

- **Comprehensive textbook of psychiatry – Kaplan & Saddock**
- **Oxford Textbook Of Psychiatry**
- **Synopsis - Kaplan & Saddock**
- **Stahl's essential psychopharmacology**

VALUE ADDED COURSE

1. Name of the programme & Code

Classification of depression and its treatment methods

2. Duration & Period

30 hrs January – June 2021

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 by Multiple Choice Questions - *Enclosed as Annexure- III*

6. Certificate model

Enclosed as Annexure- IV

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

1 time January – June 2021

8. Year of discontinuation: 2021

9. Summary report of each program year-wise

Value Added Course- January – June 2016					
Sl. No	**Course Code**	**Course Name**	**Resource Persons**	**Target Students**	**Strength & Year**
1	**PSYC02**	Classification of depression and its treatment methods	**Dr. Sridhar**	IInd YEAR	15 January – June 2021

10. Course Feed Back

Enclosed as Annexure- V

Resource Person

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

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

COORDINATOR
V.R. Sridhar

Dr. V. R. SRIDHAR, MD.,D.P.M.,
Reg. No: 30998
Professor & HOD, Psychiatry
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Kudapakkam, Puducherry-605 502.

CLASSIFICATION OF DEPRESSION AND ITS TREATMENT METHODS

PARTICIPANT HAND BOOK

BIHER SLIMS

COURSE DETAILS

Particulars	Description
Course Title	Depression
Course Code	PSYC02
Objective	1. Epidemiology 2. Etiology 3. Laboratory, brain imaging & Pschychological tests 4. Clinical Assessment 5. Differential diagnosis 6. Course & prognosis 7. Treatment
Further learning opportunities	Depression & its management
Key Competencies	On successful completion of the course the students will have skill in handling patients with depression and Suicidal ideation
Target Student	IInd YEAR MBBS Students
Duration	30hrs Every JANUARY 2021– JUNE 2021
Theory Session	20hrs
Practical Session	10hrs
Assessment Procedure	Multiple choice questions

HISTORY

The Old Testament story of King Saul describes a depressive syndrome, as does the story of Ajax's suicide in Homer's Iliad. About 400 BCE, Hippocrates used the terms mania and melancholia to describe mental disturbances. Around 30 AD, the Roman physician Celsus described melancholia (from Greek melan ["black"] and chole ["bile"]) in his work De re medicina as a depression caused by black bile. The first English text entirely related to depression was Robert Burton's Anatomy of Melancholy, published in 1621. In 1854, Jules Falret described a condition called folie circulaire, in which patients experience alternating moods of depression and mania. In 1882, the German psychiatrist Karl Kahlbaum, using the term cyclothymia, described mania and depression as stages of the same illness. In 1899, Emil Kraepelin, building on the knowledge of previous French and German psychiatrists, described manic-depressive psychosis using most of the criteria that psychiatrists now use to establish a diagnosis of bipolar I disorder. According to Kraepelin, the absence of a dementing and deteriorating course in manic-depressive psychosis diPerentiated it from dementia precox (as schizophrenia was then called). Kraepelin also described a depression that came to be known as involutional melancholia, which has since come to be viewed as a severe form of mood disorder that begins in late adulthood.

Depression

A major depressive disorder occurs without a history of a manic, mixed, or hypomanic episode. A major depressive episode must last at least 2 weeks, and typically a person with a diagnosis of a major depressive episode also experiences at least four symptoms from a list that includes changes in appetite and weight, changes in sleep and activity, lack of energy, feelings of guilt, problems thinking and making decisions, and recurring thoughts of death or suicide.

I. Epidemiology

A. Incidence and prevalence. In the most recent surveys, major depressive disorder has the highest lifetime prevalence (almost 17%) of any psychiatric disorder. The annual incidence (number of new cases) of a major depressive episode is 1.59% (women, 1.89%; men, 1.10%).

B. Sex. Major depression is more common in women depressive episodes are more common in men.

C. Sociocultural. Depressive disorders are more common among single and divorced persons compared to married persons. No correlation with socioeconomic status. No difference between races or religious groups.

II. Etiology

A. Neurotransmitters

1. Serotonin. Serotonin has become the biogenic amine neurotransmitter most commonly associated with depression. Serotonin depletion occurs in depression; thus, serotonergic agents are effective treatments. The identification of multiple serotonin receptor subtypes may lead to even more specific treatments for depression. Some patients with suicidal impulses have low cerebrospinal fluid (CSF) concentrations of serotonin metabolites (5-hydroxyindole acetic acid [5-HIAA]) and low concentrations of serotonin uptake sites on platelets.

2. Norepinephrine. Abnormal levels (usually low) of norepinephrine metabolites (3-methoxy-4-hydroxyphenylglycol [MHPG]) are found in blood, urine, and CSF of depressed patients. Venlafaxine (Effexor) increases both serotonin and norepinephrine levels and is used in depression for that reason.

3. Dopamine. Dopamine activity may be reduced in depression. Drugs that reduce dopamine concentrations (e.g., reserpine [Serpasil]) and diseases that reduce dopamine concentrations (e.g., Parkinson's disease) are associated with depressive symptoms. Drugs that increase dopamine concentrations, such as tyrosine, amphetamine, and bupropion (Wellbutrin), reduce the symptoms of depression. Two recent theories about dopamine and depression are that the mesolimbic dopamine pathway may be dysfunctional in depression and that the dopamine D1 receptor may be hypoactive in depression.

B. Psychosocial

1. Psychoanalytic. Freud described internalized ambivalence toward a love object (person), which can produce a pathologic form of mourning if the object is lost or perceived as lost. This mourning takes the form of severe depression with feelings of guilt, worthlessness, and suicidal ideation. Symbolic or real loss of love object is perceived as rejection.

2. Psychodynamics. In depression, introjection of ambivalently viewed lost objects leads to an inner sense of conflict, guilt, rage, pain, and loathing; a pathologic mourning becomes depression as ambivalent feelings meant for the introjected object are directed at the self. In mania, feelings of inadequacy and worthlessness are converted by means of denial, reaction formation, and projection to grandiose delusions.

3. Cognitive. Cognitive triad of Aaron Beck: (1) negative self-view ("things are bad because I'm bad"); (2) negative interpretation of experience ("everything has always been bad"); (3) negative view of future (anticipation of failure).

4. Learned helplessness. A theory that attributes depression to a person's inability to control events. Theory is derived from observed behavior of animals experimentally given unexpected random shocks from which they cannot escape.

5. Stressful life events. Losing a parent before age 11 is the life event most associated with later development of depression.

IV. Laboratory, Brain Imaging, and Psychological Tests

A. Dexamethasone suppression test. Nonsuppression (positive test result) represents hyper secretion of cortisol secondary to hyperactivity of hypothalamic–pituitary–adrenal axis. Abnormal in 50% of patients with major depression but is of limited clinical usefulness owing to frequency of false-positives and false-negatives. Diminished release of TSH in response to thyrotropin-releasing hormone (TRH) reported in both depression and mania. Prolactin release decreased in response to tryptophan. Tests are not definitive.

B. Brain imaging. No gross brain changes. Enlarged cerebral ventricles on computed tomography (CT) in some patients with psychotic depression; diminished basal ganglia blood flow in some depressive patients. Magnetic resonance imaging (MRI) studies have also indicated that patients with major depressive disorder have smaller caudate nuclei and smaller frontal lobes than do control subjects.

C. Psychological tests

1. Rating scales. Can be used to assist in diagnosis and assessment of treatment efficacy. The Beck Depression Inventory (BDI) and Zung Self-rating Scale are scored by patients. The Hamilton Rating Scale for Depression (HAM-D), Montgomery Asberg Depression Rating Scale (MADRS).

2. Rorschach test. Standardized set of 10 inkblots scored by examiner —few associations, slow response time in depression.

3. Thematic apperception test (TAT). Series of 30 pictures depicting ambiguous situations and interpersonal events. Patient creates a story about each scene. Depressives will create depressed stories,

V. Bipolar Disorder There are two types of bipolar disorder: bipolar I characterized by the occurrence of manic episodes with or without a major depressive episode and bipolar II characterized by at least one depressive episode with or without a hypomanic episode.

A. Depression (major depressive episode).

1. Information obtained from history

a. Depressed mood: subjective sense of sadness, feeling "blue" or "down in the dumps" for a prolonged period of time.

b. Anhedonia: inability to experience pleasure.

c. Social withdrawal.

d. Lack of motivation, little tolerance of frustration.

e. Vegetative signs. (1) Loss of libido. (2) Weight loss and anorexia. (3) Weight gain and hyperphagia. (4) Low-energy level; fatigability. (5) Abnormal menses. (6) Early morning awakening (terminal insomnia); approximately 75% of depressed patients have sleep difficulties, either insomnia or hypersomnia. (7) Diurnal variation (symptoms worse in morning).

f. Constipation.

g. Dry mouth.

h. Headache.

2. Information obtained from mental status examination

a. General appearance and behavior. Psychomotor retardation or agitation, poor eye contact, tearful, downcast, inattentive to personal appearance.

b. Affect. Constricted or labile.

c. Mood. Depressed, irritable, frustrated, sad.

d. Speech. Little or no spontaneity; monosyllabic; long pauses; soft, low monotone.

e. Thought content. Suicidal ideation affects 60% of depressed patients, and 15% commit suicide; obsessive rumination; pervasive feelings of hopelessness, worthlessness, and guilt; somatic preoccupation; indecisiveness; poverty of thought content and paucity of speech; mood-congruent hallucinations and delusions.

f. Cognition. Distractible, difficulty concentrating, complaints of poor memory, apparent disorientation; abstract thought may be impaired.

g. Insight and judgment. Impaired because of cognitive distortions of personal worthlessness.

3. Associated features

a. Somatic complaints may mask depression: in particular, cardiac, gastrointestinal, and genitourinary symptoms; low back pain, other orthopedic complaints.

b. Content of delusions and hallucinations, when present, tends to be congruent with depressed mood; most common are delusions of guilt, poverty, and deserved persecution, in addition to somatic and nihilistic (end of the world) delusions. Mood-incongruent delusions are those with content not apparently related to the predominant mood (e.g., delusions of thought insertion, broadcasting, and control, or persecutory delusions unrelated to depressive themes).

4. Age-specific features. Depression can present differently at different ages.

a. Prepubertal. Somatic complaints, agitation, single-voice auditory hallucinations, anxiety disorders, and phobias.

b. Adolescence. Substance abuse, antisocial behavior, restlessness, truancy, school difficulties, promiscuity, increased sensitivity to rejection, poor hygiene.

c. Elderly. Cognitive deficits (memory loss, disorientation, confusion); pseudodementia or the dementia syndrome of depression, apathy, and distractibility

B. Depressive disorders

1. Major depressive disorder. Can occur alone or as part of bipolar disorder. When it occurs alone it is also known as unipolar depression. Symptoms must be present for at least 2 weeks and represent a change from previous functioning. More common in women than in men by 2:1. Precipitating event occurs in at least 25% of patients. Diurnal variation, with symptoms worse early in morning. Psychomotor retardation or agitation is present. Associated with vegetative signs. Mood-congruent delusions and hallucinations may be present. Median age of onset is 40 years, but can occur at any time. Genetic factor is present. Major depressive disorder may occur as a single episode in a person's life or may be recurrent

2. Other types of major depressive disorder

In addition to the severity, psychotic, and remission descriptions, additional symptom features (specifiers) can be used to describe patients with various mood disorders. With Psychotic Features. The presence of psychotic features in major depressive disorder reflects severe disease and is a poor prognostic indicator. A review of the literature comparing psychotic with nonpsychotic major depressive disorder indicates that the two conditions may be distinct in their pathogenesis. One difference is that bipolar I disorder is more common in

the families of probands with psychotic depression than in the families of probands with nonpsychotic depression. The psychotic symptoms themselves are often categorized as either mood congruent, that is, in harmony with the mood disorder (“I deserve to be punished because I am so bad”), or mood incongruent, not in harmony with the mood disorder. Patients with mood disorder with mood-congruent psychoses have a psychotic type of mood disorder; however, patients with mood disorder with mood-incongruent psychotic symptoms may have schizoaffective disorder or schizophrenia. The following factors have been associated with a poor prognosis for patients with mood disorders: long duration of episodes, temporal dissociation between the mood disorder and the psychotic symptoms, and a poor premorbid history of social adjustment. The presence of psychotic features also has significant treatment implications. These patients typically require antipsychotic drugs in addition to antidepressants or mood stabilizers and may need ECT to obtain clinical improvement. With Melancholic Features. Melancholia is one of the oldest terms used in psychiatry, dating back to Hippocrates in the 4th century to describe the dark mood of depression. It is still used to refer to a depression characterized by severe anhedonia, early morning awakening, weight loss, and profound feelings of guilt (often over trivial events). It is not uncommon for patients who are melancholic to have suicidal ideation. Melancholia is associated with changes in the autonomic nervous system and in endocrine functions. For that reason, melancholia is sometimes referred to as “endogenous depression” or depression that arises in the absence of external life stressors or precipitants. The DSM-5 melancholic features can be applied to major depressive episodes in major depressive disorder, bipolar I disorder, or bipolar II disorder. With Atypical Features. The introduction of a formally defined depression with atypical features is a response to research and clinical data indicating that patients with atypical features have specific, predictable characteristics: overeating and oversleeping. These symptoms have sometimes been referred to as reversed vegetative symptoms, and the symptom pattern has sometimes been called hysteroid dysphoria. When patients with major depressive disorder with atypical features are compared with patients with typical depression features, the patients with atypical features are found to have a younger age of onset; more severe psychomotor slowing; and more frequent coexisting diagnoses of panic disorder, substance abuse or dependence, and somatization disorder. The high incidence and severity of anxiety symptoms in patients with atypical features have sometimes been correlated with the likelihood of their being misclassified as having an anxiety disorder rather than a mood disorder. Patients with atypical features may also have a long-term course, a diagnosis of bipolar I disorder, or a seasonal pattern to their disorder. The DSM-5 atypical features can be applied to the most recent major depressive episode in major depressive disorder, bipolar I disorder, bipolar II disorder, or dysthymic disorder. Atypical depression may mask manic symptoms as in the

following case. With Catatonic Features. As a symptom, catatonia can be present in several mental disorders, most commonly, schizophrenia and the mood disorders. The presence of catatonic features in patients with mood disorders may have prognostic and treatment significance. The hallmark symptoms of catatonia—stuporousness, blunted aPect, extreme withdrawal, negativism, and marked psychomotor retardation—can be seen in both catatonic and noncatatonic schizophrenia, major depressive disorder (often with psychotic features), and medical and neurological disorders. Clinicians often do not associate catatonic symptoms with bipolar I disorder because of the marked contrast between the symptoms of stuporous catatonia and the classic symptoms of mania. Because catatonic symptoms are a behavioral syndrome appearing in several medical and psychiatric conditions, catatonic symptoms do not imply a single diagnosis. Catatonia is discussed in detail in Section 7.5. Postpartum Onset. DSM-5 allows the specification of a postpartum mood disturbance if the onset of symptoms is within 4 weeks postpartum. Postpartum mental disorders commonly include psychotic symptoms. Postpartum disorders are discussed in Section 26.1, Psychiatry and Reproductive Medicine. Rapid Cycling. Patients with rapid cycling bipolar I disorder are likely to be female and to have had depressive and hypomanic episodes. No data indicate that rapid cycling has a familial pattern of inheritance; thus, an external factor such as stress or drug treatment may be involved in the pathogenesis of rapid cycling. The DSM-5 criteria specify that the patient must have at least four episodes within a 12-month period. Seasonal Pattern. Patients with a seasonal pattern to their mood disorders tend to experience depressive episodes during a particular season, most commonly winter. The pattern has become known as seasonal affective disorder (SAD), although this term is not used in DSM5. Two types of evidence indicate that the seasonal pattern may represent a separate diagnostic entity. First, the patients are likely to respond to treatment with light therapy, although no studies with controls to evaluate light therapy in nonseasonally depressed patients have been conducted. Second, research has shown that patients evince decreased metabolic activity in the orbital frontal cortex and in the left inferior parietal lobe. Further studies are necessary to differentiate depressed persons with seasonal pattern from other depressed persons. This disorder is discussed further in Section 16.2 on Sleep-Wake Disorders. Non–DSM-5 Types. DSM-5 specifiers for depressive disorders are included in

Other systems that identify types of patients with mood disorders usually separate patients with good and poor prognoses or patients who may respond to one treatment or another. They also differentiate endogenous-reactive and primary-secondary schemes. The endogenous-reactive continuum is a controversial division. It implies that endogenous depressions are biological and that reactive depressions are psychological, primarily on the basis of the presence or absence of an identifiable precipitating stress. Other symptoms of

endogenous depression have been described as diurnal variation, delusions, psychomotor retardation, early morning awakening, and feelings of guilt; thus, endogenous depression is similar to the DSM-5 diagnosis of major depressive disorder with psychotic features, melancholic features, or both. Symptoms of reactive depression have included initial insomnia, anxiety, emotional lability, and multiple somatic complaints. Primary depressions are what DSM-5 refers to as mood disorders, except for the diagnoses of mood disorder caused by a general medical condition and substance-induced mood disorder, which are considered secondary depressions. Double depression is the condition in which major depressive disorder is superimposed on dysthymic disorder. A depressive equivalent is a symptom or syndrome that may be a forme fruste of a depressive episode. For example, a triad of truancy, alcohol abuse, and sexual promiscuity in a formerly well-behaved adolescent may constitute a depressive equivalent.

3. Persistent depressive disorder (Dysthymia).

The most typical features of dysthymia, also known as persistent depressive disorder, is the presence of a depressed mood that lasts most of the day and is present almost continuously. There are associated feelings of inadequacy, guilt, irritability, and anger; withdrawal from society; loss of interest; and inactivity and lack of productivity. The term dysthymia, which means “ill humored,” was introduced in 1980. Before that time, most patients now classified as having dysthymia were classified as having depressive neurosis (also called neurotic depression). Dysthymia is distinguished from major depressive disorder by the fact that patients complain that they have always been depressed. Thus, most cases are of early onset, beginning in childhood or adolescence and certainly occurring by the time patients reach their 20s. A late onset subtype, much less prevalent and not well characterized clinically, has been identified among middle-aged and geriatric populations, largely through epidemiological studies in the community.

(1) low-grade chronicity for at least 2 years;

(2) insidious onset, with origin often in childhood or adolescence; and

(3) a persistent or intermittent course.

The family history of patients with dysthymia is typically replete with both depressive and bipolar disorders, which is one of the more robust findings supporting its link to primary mood disorder. Epidemiology Dysthymia is common among the general population and aPects 5 to 6 percent of all persons. It is seen among patients in general psychiatric clinics, where it aPects between half and one-third of all patients. No gender diPerences are seen for incidence rates. The disorder is more common in women younger than 64 years of age than in men of any age and is more common among unmarried and young

persons and in those with low incomes. Dysthymia frequently coexists with other mental disorders, particularly major depressive disorder, and in persons with major depressive disorder, there is less likelihood of full remission between episodes. The patients may also have coexisting anxiety disorders (especially panic disorder), substance abuse, and borderline personality disorder. The disorder is more common among those with first-degree relatives with major depressive disorder. Patients with dysthymia are likely to be taking a wide range of psychiatric medications, including antidepressants, antimanic agents such as lithium (Eskalith) and carbamazepine (Tegretol), and sedative-hypnotics. Etiology Biological Factors. The biological basis for the symptoms of dysthymia and major depressive disorder are similar, but the biological bases for the underlying pathophysiology in the two disorders differ.

SLEEP STUDIES

Decreased rapid eye movement (REM) latency and increased REM density are two state markers of depression in major depressive disorder that also occur in a significant proportion of patients with dysthymia.

NEUROENDOCRINE STUDIES

The two most studied neuroendocrine axes in major depressive disorder and dysthymia are the adrenal axis and the thyroid axis, which have been tested by using the dexamethasone-suppression test (DST) and the thyrotropin-releasing hormone (TRH)- stimulation test, respectively. Although the results of studies are not absolutely consistent, most indicate that patients with dysthymia are less likely to have abnormal results on a DST than are patients with major depressive disorder. Psychosocial Factors. Psychodynamic theories about the development of dysthymia posit that the disorder results from personality and ego development and culminates in difficulty adapting to adolescence and young adulthood. Karl Abraham, for example, thought that the conflicts of depression center on oral- and anal-sadistic traits. Anal traits include excessive orderliness, guilt, and concern for others; they are postulated to be a defense against preoccupation with anal matter and with disorganization, hostility, and self-preoccupation. A major defense mechanism used is reaction formation. Low self-esteem, anhedonia, and introversion are often associated with the depressive character.

FREUD

In Mourning and Melancholia, Sigmund Freud asserted that an interpersonal disappointment early in life can cause a vulnerability to depression that leads to ambivalent love relationships as an adult; real or threatened losses in adult life then trigger depression. Persons susceptible to depression are orally dependent and require constant narcissistic gratification. When deprived of love, affection,

and care, they become clinically depressed; when they experience a real loss, they internalize or introject the lost object and turn their anger on it and thus on themselves.

COGNITIVE THEORY

The cognitive theory of depression also applies to dysthymia. It holds that a disparity between actual and fantasized situations leads to diminished self-esteem and a sense of helplessness. The success of cognitive therapy in the treatment of some patients with dysthymia may provide some support for the theoretical model.

Diagnosis and Clinical Features

The DSM-5 diagnosis criteria for dysthymia (Table 8.2-1) stipulate the presence of a depressed mood most of the time for at least 2 years (or 1 year for children and adolescents). To meet the diagnostic criteria, a patient should not have symptoms that are better accounted for as major depressive disorder and should never have had a manic or hypomanic episode. DSM-5 allows clinicians to specify whether the onset was early (before age 21 years) or late (age 21 years or older). DSM-5 also allows specification of atypical features in dysthymia. The profile of dysthymia overlaps with that of major depressive disorder but differs from it in that symptoms tend to outnumber signs (more subjective than objective depression). This means that disturbances in appetite and libido are uncharacteristic, and psychomotor agitation or retardation is not observed. This all translates into a depression with attenuated symptomatology. Subtle endogenous features are observed, however, including inertia, lethargy, and anhedonia that are characteristically worse in the morning. Because patients presenting clinically often fluctuate in and out of a major depression, the core DSM-5 criteria for dysthymia tend to emphasize vegetative dysfunction; however, cognitive symptoms are often present. Dysthymia is quite heterogeneous. Anxiety is not a necessary part of its clinical picture, yet dysthymia is often diagnosed in patients with anxiety and phobic disorders. That clinical situation is sometimes diagnosed as mixed anxiety depressive disorder. For greater operational clarity, it is best to restrict dysthymia to a primary disorder, one that cannot be explained by another psychiatric disorder. The essential features of such primary dysthymia include habitual gloom, brooding, lack of joy in life, and preoccupation with inadequacy. Dysthymia then is best characterized as long-standing, fluctuating, low-grade depression, experienced as part of the habitual self and representing an accentuation of traits observed in the depressive temperament. The clinical picture of dysthymia is varied, with some patients proceeding to major depression and others manifesting the pathology largely at the personality level.

Dysthymic Variants

Dysthymia is common in patients with chronically disabling physical disorders, particularly among elderly adults. Dysthymia-like, clinically significant, sub-threshold depression lasting 6 or more months has also been described in neurological conditions, including stroke. According to a recent World Health Organization (WHO) conference, this condition aggravates the prognosis of the underlying neurological disease and therefore deserves pharmacotherapy. Prospective studies on children have revealed an episodic course of dysthymia with remissions, exacerbations, and eventual complications by major depressive episodes, 15 to 20 percent of which might even progress to hypomanic, manic, or mixed episodes post puberty. Persons with dysthymia presenting clinically as adults tend to pursue a chronic unipolar course that may or may not be complicated by major depression. They rarely develop spontaneous hypomania or mania. When treated with antidepressants, however, some of them may develop brief hypomanic switches that typically disappear when the antidepressant dose is decreased.

Differential Diagnosis

The differential diagnosis for dysthymia is essentially identical to that for major depressive disorder. Many substances and medical illnesses can cause chronic depressive symptoms. Two disorders are particularly important to consider in the differential diagnosis of dysthymia— minor depressive disorder and recurrent brief depressive disorder. Minor Depressive Disorder. Minor depressive disorder is characterized by episodes of depressive symptoms that are less severe than those seen in major depressive disorder. The difference between dysthymia and minor depressive disorder is primarily the episodic nature of the symptoms in the latter. Between episodes, patients with minor depressive disorder have a euthymic mood, but patients with dysthymia have virtually no euthymic periods. Recurrent Brief Depressive Disorder. Recurrent brief depressive disorder is characterized by brief periods (less than 2 weeks) during which depressive episodes are present. Patients with the disorder would meet the diagnostic criteria for major depressive disorder if their episodes lasted longer. Patients with recurrent brief depressive disorder differ from patients with dysthymia on two counts: They have an episodic disorder, and their symptoms are more severe.

Double Depression

An estimated 40 percent of patients with major depressive disorder also meet the criteria for dysthymia, a combination often referred to as double depression. Available data support the conclusion that patients with double depression have a poorer prognosis than patients with only major depressive disorder. The treatment of patients with double depression should be directed toward both disorders because the resolution of the symptoms of major depressive episode

still leaves these patients with significant psychiatric impairment. Alcohol and Substance Abuse. Patients with dysthymia commonly meet the diagnostic criteria for a substance-related disorder. This comorbidity can be logical; patients with dysthymia tend to develop coping methods for their chronically depressed state that involve substance abuse. Therefore, they are likely to use alcohol, stimulants such as cocaine, or marijuana, the choice perhaps depending primarily on a patient's social context. The presence of a comorbid diagnosis of substance abuse presents a diagnostic dilemma for clinicians; the longterm use of many substances can result in a symptom picture indistinguishable from that of dysthymia.

Course and Prognosis

About 50 percent of patients with dysthymia experience an insidious onset of symptoms before age 25 years. Despite the early onset, patients often suffer with the symptoms for a decade before seeking psychiatric help and may consider early-onset dysthymia simply part of life. Patients with an early onset of symptoms are at risk for either major depressive disorder or bipolar I disorder in the course of their disorder. Studies of patients with the diagnosis of dysthymia indicate that about 20 percent progressed to major depressive disorder, 15 percent to bipolar II disorder, and fewer than 5 percent to bipolar I disorder. The prognosis for patients with dysthymia varies. Antidepressive agents and specific types of psychotherapies (e.g., cognitive and behavior therapies) have positive effects on the course and prognosis of dysthymia. The available data about previously available treatments indicate that only 10 to 15 percent of patients are in remission 1 year after the initial diagnosis. About 25 percent of all patients with dysthymia never attain a complete recovery. Overall, however, the prognosis is good with treatment.

Treatment

Historically, patients with dysthymia either received no treatment or were seen as candidates for long-term, insight-oriented psychotherapy. Contemporary data offer the most objective support for cognitive therapy, behavior therapy, and pharmacotherapy. The combination of pharmacotherapy and some form of psychotherapy may be the most effective treatment for the disorder. Cognitive Therapy. Cognitive therapy is a technique in which patients are taught new ways of thinking and behaving to replace faulty negative attitudes about themselves, the world, and the future. It is a short-term therapy program oriented toward current problems and their resolution. Behavior Therapy. Behavior therapy for depressive disorders is based on the theory that depression is caused by a loss of positive reinforcement as a result of separation, death, or sudden environmental change. The various treatment methods focus on specific goals to increase activity, to provide pleasant experiences, and to teach patients

how to relax. Altering personal behavior in depressed patients is believed to be the most effective way to change the associated depressed thoughts and feelings. Behavior therapy is often used to treat the learned helplessness of some patients who seem to meet every life challenge with a sense of impotence. Insight-Oriented (Psychoanalytic) Psychotherapy. Individual insight-oriented psychotherapy is the most common treatment method for dysthymia, and many clinicians consider it the treatment of choice. The psychotherapeutic approach attempts to relate the development and maintenance of depressive symptoms and maladaptive personality features to unresolved conflicts from early childhood. Insight into depressive equivalents (e.g., substance abuse) or into childhood disappointments as antecedents to adult depression can be gained through treatment. Ambivalent current relationships with parents, friends, and others in the patient's current life are examined. Patients' understanding of how they try to gratify an excessive need for outside approval to counter low self-esteem and a harsh superego is an important goal of this therapy. Interpersonal Therapy. In interpersonal therapy for depressive disorders, a patient's current interpersonal experiences and ways of coping with stress are examined to reduce depressive symptoms and to improve self-esteem. Interpersonal therapy lasts for about 12 to 16 weekly sessions and can be combined with antidepressant medication. Family and Group Therapies. Family therapy may help both the patient and the patient's family deal with the symptoms of the disorder, especially when a biologically based sub-affective syndrome seems to be present. Group therapy may help withdrawn patients learn new ways to overcome their interpersonal problems in social situations. Pharmacotherapy. Because of long-standing and commonly held theoretical beliefs that dysthymia is primarily a psychologically determined disorder, many clinicians avoid prescribing antidepressants for patients; however, many studies have shown therapeutic success with antidepressants. The data generally indicate that selective serotonin reuptake inhibitors (SSRIs) venlafaxine and bupropion are an effective treatment for patients with dysthymia. Monoamine oxidase inhibitors (MAOIs) are effective in a subgroup of patients with the disorder, a group who may also respond to the judicious use of amphetamines.

Hospitalization.

Hospitalization is usually not indicated for patients with dysthymia, but particularly severe symptoms, marked social or professional incapacitation, the need for extensive diagnostic procedures, and suicidal ideation are all indications for hospitalization.

4. Cyclothymic disorder. Less severe disorder, with alternating periods of hypomania and moderate depression. The condition is chronic and nonpsychotic. Symptoms must be present for at least 2 years. Equally common in men and women. Onset usually is insidious and occurs in late adolescence or

early adulthood. Substance abuse is common. Major depressive disorder and bipolar disorder are more common among first-degree relatives than among the general population. Recurrent mood swings may lead to social and professional difficulties. May respond to lithium

5. Disruptive mood dysregulation disorder. The disorder is a new category included in the DSM-5 to prevent over diagnosis of bipolar disorder in children. The pertinent symptoms include; acute and recurrent angry outbursts that are inconsistent with the developmental age and manifest as irritability, anger, and occur frequently three or more times a week. Of note the diagnosis should not be made before age 6 or after 18 years.

6. Premenstrual dysphoric disorder. This disorder has been reclassified in DSM-5 and categorized under depressive disorders. Is a distinct condition that responds to treatment that begins after ovulation and remits early in the menstruation phase. The hallmark symptoms are; begins in the final week of menstrual cycle, with mood lability (ups and downs, sudden tearfulness, and sense of rejection), irritability, depressed mood, hopelessness, anxiety, poor concentration, fatigue, changes in appetite, changes in sleep pattern and physical symptoms (breast tenderness, swelling, and bloating).

CLINICAL FEATURES

Depressive Episodes A depressed mood and a loss of interest or pleasure are the key symptoms of depression. Patients may say that they feel blue, hopeless, in the dumps, or worthless. For a patient, the depressed mood often has a distinct quality that differentiates it from the normal emotion of sadness or grief. Patients often describe the symptom of depression as one of agonizing emotional pain and sometimes complain about being unable to cry, a symptom that resolves as they improve. About two-thirds of all depressed patients contemplate suicide, and 10 to 15 percent commit suicide. Those recently hospitalized with a suicide attempt or suicidal ideation have a higher lifetime risk of successful suicide than those never hospitalized for suicidal ideation. Some depressed patients sometimes seem unaware of their depression and do not complain of a mood disturbance even though they exhibit withdrawal from family, friends, and activities that previously interested them. Almost all depressed patients (97 percent) complain about reduced energy; they have difficulty finishing tasks, are impaired at school and work, and have less motivation to undertake new projects. About 80 percent of patients complain of trouble sleeping, especially early morning awakening (i.e., terminal insomnia) and multiple awakenings at night, during which they ruminate about their problems. Many patients have decreased appetite and weight loss, but others experience increased appetite and weight gain and sleep longer than usual. These patients are classified as having atypical features. Anxiety, a common

symptom of depression, affects as many as 90 percent of all depressed patients. The various changes in food intake and rest can aggravate coexisting medical illnesses such as diabetes, hypertension, chronic obstructive lung disease, and heart disease. Other vegetative symptoms include abnormal menses and decreased interest and performance in sexual activities. Sexual problems can sometimes lead to inappropriate referrals, such as to marital counseling and sex therapy, when clinicians fail to recognize the underlying depressive disorder. Anxiety (including panic attacks), alcohol abuse, and somatic complaints (e.g., constipation and headaches) often complicate the treatment of depression. About 50 percent of all patients describe a diurnal variation in their symptoms, with increased severity in the morning and lessening of symptoms by evening. Cognitive symptoms include subjective reports of an inability to concentrate (84 percent of patients in one study) and impairments in thinking (67 percent of patients in another study). Depression in Children and Adolescents. School phobia and excessive clinging to parents may be symptoms of depression in children. Poor academic performance, substance abuse, antisocial behavior, sexual promiscuity, truancy, and running away may be symptoms of depression in adolescents. Depression in Older People. Depression is more common in older persons than it is in the general population. Various studies have reported prevalence rates ranging from 25 to almost 50 percent, although the percentage of these cases that are caused by major depressive disorder is uncertain. Several studies indicate that depression in older persons may be correlated with low socioeconomic status, the loss of a spouse, a concurrent physical illness, and social isolation. Other studies have indicated that depression in older persons is underdiagnosed and undertreated, perhaps particularly by general practitioners. The under recognition of depression in older persons may occur because the disorder appears more often with somatic complaints in older, than in younger, age groups. Further, ageism may influence and cause clinicians to accept depressive symptoms as normal in older patients

MENTAL STATUS EXAMINATION

General Description

Generalized psychomotor retardation is the most common symptom of depression, although psychomotor agitation is also seen, especially in older patients. Hand wringing and hair pulling are the most common symptoms of agitation. Classically, a depressed patient has a stooped posture; no spontaneous movements; and a downcast, averted gaze. On clinical examination, depressed patients exhibiting gross symptoms of psychomotor retardation may appear identical to patients with catatonic schizophrenia.

Mood, Affect, and Feelings Depression is the key symptom, although about 50 percent of patients deny depressive feelings and do not appear to be particularly

depressed. Family members or employers often bring or send these patients for treatment because of social withdrawal and generally decreased activity.

Speech Many depressed patients have decreased rate and volume of speech; they respond to questions with single words and exhibit delayed responses to questions. The examiner may literally have to wait 2 or 3 minutes for a response to a question.

Perceptual Disturbances Depressed patients with delusions or hallucinations are said to have a major depressive episode with psychotic features. Even in the absence of delusions or hallucinations, some clinicians use the term psychotic depression for grossly regressed depressed patients—mute, not bathing, soiling. Such patients are probably better described as having catatonic features. Delusions and hallucinations that are consistent with a depressed mood are said to be mood congruent. Mood-congruent delusions in a depressed person include those of guilt, sinfulness, worthlessness, poverty, failure, persecution, and terminal somatic illnesses (such as cancer and a "rotting" brain). The content of mood-incongruent delusions or hallucinations is not consistent with a depressed mood. For example, a mood-incongruent delusion in a depressed person might involve grandiose themes of exaggerated power, knowledge, and worth. When that occurs, a schizophrenic disorder should be considered. A 42-year-old civil servant said that she was so paralyzed by depression that she felt that she had no personal initiative and volition left; she believed that some malignant force had taken over her actions and that it was commenting on every action that she was undertaking. The patient recovered fully with thymoleptic medication. There is no reason to believe that, in this patient, the feelings of somatic passability and running commentary indicated a schizophrenic process. Thought Depressed patients customarily have negative views of the world and of themselves. Their thought content often includes non-delusional ruminations about loss, guilt, suicide, and death. About 10 percent of all depressed patients have marked symptoms of a thought disorder, usually thought blocking and profound poverty of content. Sensorium and Cognition Orientation. Most depressed patients are oriented to person, place, and time, although some may not have sufficient energy or interest to answer questions about these subjects during an interview. Memory. About 50 to 75 percent of all depressed patients have a cognitive impairment, sometimes referred to as depressive pseudodementia. Such patients commonly complain of impaired concentration and forgetfulness. Impulse Control About 10 to 15 percent of all depressed patients commit suicide, and about two-thirds have suicidal ideation. Depressed patients with psychotic features occasionally consider killing a person as a result of their delusional systems, but the most severely depressed patients often lack the motivation or the energy to act in an impulsive or violent way. Patients with depressive disorders are at increased risk of suicide as they begin to improve and regain the energy needed to plan and carry out a suicide (paradoxical suicide). It is usually

clinically unwise to give a depressed patient a large prescription for a large number of antidepressants, especially tricyclic drugs, at the time of their discharge from the hospital. Similarly, drugs that may be activating, such as fluoxetine, may be prescribed in such a way that the energizing qualities are minimized (e.g., be given a benzodiazepine at the same time). Judgment and Insight Judgment is best assessed by reviewing patients' actions in the recent past and their behavior during the interview. Depressed patients' descriptions of their disorder are often hyperbolic; they overemphasize their symptoms, their disorder, and their life problems. It is difficult to convince such patients that improvement is possible. Reliability In interviews and conversations, depressed patients overemphasize the bad and minimize the good. A common clinical mistake is to unquestioningly believe a depressed patient who states that a previous trial of antidepressant medications did not work. Such statements may be false, and they require confirmation from another source. Psychiatrists should not view patients' misinformation as an intentional fabrication; the admission of any hopeful information may be impossible for a person in a depressed state of mind.

Objective Rating Scales for Depression

Objective rating scales for depression can be useful in clinical practice for documenting the depressed patient's clinical state. Zung. The Zung Self-Rating Depression Scale is a 20-item report scale. A normal score is 34 or less; a depressed score is 50 or more. The scale provides a global index of the intensity of a patient's depressive symptoms, including the affective expression of depression. Raskin. The Raskin Depression Scale is a clinician-rated scale that measures the severity of a patient's depression, as reported by the patient and as observed by the physician, on a 5-point scale of three dimensions: verbal report, displayed behavior, and secondary symptoms. The scale has a range of 3 to 13; a normal score is 3, and a depressed score is 7 or more. Hamilton. The Hamilton Rating Scale for Depression (HAM-D) is a widely used depression scale with up to 24 items, each of which is rated 0 to 4 or 0 to 2, with a total score of 0 to 76. The clinician evaluates the patient's answers to questions about feelings of guilt, thoughts of suicide, sleep habits, and other symptoms of depression, and the ratings are derived from the clinical interview.

DIFFERENTIAL DIAGNOSIS

Major Depressive Disorder Medical Disorders. The diagnosis of mood disorder due to a general medical condition must be considered. Failure to obtain a good clinical history or to consider the context of a patient's current life situation can lead to diagnostic errors. Clinicians should have depressed adolescents tested for mononucleosis, and patients who are markedly overweight or underweight should be tested for adrenal and thyroid dysfunctions. Homosexuals, bisexual

men, prostitutes, and persons who abuse a substance intravenously should be tested for acquired immune deficiency syndrome (AIDS). Older patients should be evaluated for viral pneumonia and other medical conditions. Many neurological and medical disorders and pharmacological agents can produce symptoms of depression. Patients with depressive disorders often first visit their general practitioners with somatic complaints. Most medical causes of depressive disorders can be detected with a comprehensive medical history, a complete physical and neurological examination, and routine blood and urine tests. The workup should include tests for thyroid and adrenal functions because disorders of both of these endocrine systems can appear as depressive disorders. In substance-induced mood disorder, a reasonable rule of thumb is that any drug a depressed patient is taking should be considered a potential factor in the mood disorder. Cardiac drugs, antihypertensives, sedatives, hypnotics, antipsychotics, antiepileptics, antiparkinsonian drugs, analgesics, antibacterials, and antineoplastics are all commonly associated with depressive symptoms.

NEUROLOGICAL CONDITIONS.

The most common neurological problems that manifest depressive symptoms are Parkinson's disease, dementing illnesses (including dementia of the Alzheimer's type), epilepsy, cerebrovascular diseases, and tumors. About 50 to 75 percent of all patients with Parkinson's disease have marked symptoms of depressive disorder that do not correlate with the patient's physical disability, age, or duration of illness but do correlate with the presence of abnormalities found on neuropsychological tests. The symptoms of depressive disorder can be masked by the almost identical motor symptoms of Parkinson's disease. Depressive symptoms often respond to antidepressant drugs or ECT. The interictal changes associated with temporal lobe epilepsy can mimic a depressive disorder, especially if the epileptic focus is on the right side. Depression is a common complicating feature of cerebrovascular diseases, particularly in the 2 years after the episode. Depression is more common in anterior brain lesions than in posterior brain lesions and, in both cases, often responds to antidepressant medications. Tumors of the diencephalic and temporal regions are particularly likely to be associated with depressive disorder symptoms.

PSEUDODEMENTIA.

Clinicians can usually differentiate the pseudodementia of major depressive disorder from the dementia of a disease, such as dementia of the Alzheimer's type, on clinical grounds. The cognitive symptoms in major depressive disorder have a sudden onset, and other symptoms of the disorder, such as self-reproach, are also present. A diurnal variation in the cognitive problems, which is not seen in primary dementias, may occur. Whereas depressed patients with cognitive

difficulties often do not try to answer questions ("I don't know"), patients with dementia may confabulate. During an interview, depressed patients can sometimes be coached and encouraged into remembering, an ability that demented patients lack. Mental Disorders. Depression can be a feature of virtually any mental disorder, but the mental disorders listed in Table 8.1-8 deserve particular consideration in the differential diagnosis.

OTHER MOOD DISORDERS.

Clinicians must consider a range of diagnostic categories before arriving at a final diagnosis. Mood disorder caused by a general medical condition and substance-induced mood disorder must be ruled out. Clinicians must also determine whether a patient has had episodes of mania-like symptoms, indicating bipolar I disorder (complete manic and depressive syndromes), bipolar II disorder (recurrent major depressive episodes with hypomania), or cyclothymic disorder (incomplete depressive and manic syndromes). If a patient's symptoms are limited to those of depression, clinicians must assess the severity and duration of the symptoms to differentiate among major depressive disorder (complete depressive syndrome for 2 weeks), minor depressive disorder (incomplete but episodic depressive syndrome), recurrent brief depressive disorder (complete depressive syndrome but for less than 2 weeks per episode), and dysthymic disorder (incomplete depressive syndrome without clear episodes).

OTHER MENTAL DISORDERS.

Substance-related disorders, psychotic disorders, eating disorders, adjustment disorders, somatoform disorders, and anxiety disorders are all commonly associated with depressive symptoms and should be considered in the differential diagnosis of a patient with depressive symptoms. Perhaps the most difficult differential is that between anxiety disorders with depression and depressive disorders with marked anxiety. An abnormal result on the dexamethasone-suppression test, the presence of shortened REM latency on a sleep electroencephalogram (EEG), and a negative lactate infusion test result support a diagnosis of major depressive disorder in particularly ambiguous cases.

UNCOMPLICATED BEREAVEMENT.

Uncomplicated bereavement is not considered a mental disorder even though about one-third of all bereaved spouses for a time meet the diagnostic criteria for major depressive disorder. Some patients with uncomplicated bereavement do develop major depressive disorder, but the diagnosis is not made unless no resolution of the grief occurs. The differentiation is based on the symptoms' severity and length. In major depressive disorder, common symptoms that

evolve from unresolved bereavement are a morbid preoccupation with worthlessness; suicidal ideation; feelings that the person has committed an act (not just an omission) that caused the spouse's death; mummification (keeping the deceased's belongings exactly as they were); and a particularly severe anniversary reaction, which sometimes includes a suicide attempt. In severe forms of bereavement depression, the patient simply pines away, unable to live without the departed person, usually a spouse. Such persons do have a serious medical condition. Their immune function is often depressed, and their cardiovascular status is precarious. Death can ensue within a few months of that of a spouse, especially among elderly men. Such considerations suggest that it would be clinically unwise to withhold antidepressants from many persons experiencing such an intense mourningSchizophrenia. Much has been published about the clinical difficulty of distinguishing a manic episode from schizophrenia. Although difficult, a differential diagnosis is possible. Merriment, elation, and infectiousness of mood are much more common in manic episodes than in schizophrenia. The combination of a manic mood, rapid or pressured speech, and hyperactivity weighs heavily toward a diagnosis of a manic episode. The onset in a manic episode is often rapid and is perceived as a marked change from a patient's previous behavior. Half of all patients with bipolar I disorder have a family history of mood disorder. Catatonic features may be part of a depressive phase of bipolar I disorder. When evaluating patients with catatonia, clinicians should look carefully for a past history of manic or depressive episodes and for a family history of mood disorders. Manic symptoms in persons from minority groups (particularly blacks and Hispanics) are often misdiagnosed as schizophrenic symptoms. Medical Conditions. In contrast to depressive symptoms, which are present in almost all psychiatric disorders, manic symptoms are more distinctive, although they can be caused by a wide range of medical and neurological conditions and substances. Antidepressant treatment can also be associated with the precipitation of mania in some patients.

COURSE AND PROGNOSIS

Studies of the course and prognosis of mood disorders have generally concluded that mood disorders tend to have long courses and that patients tend to have relapses. Although mood disorders are often considered benign in contrast to schizophrenia, they exact a profound toll on affected patients.

Major Depressive Disorder Course

ONSET.

About 50 percent of patients having their first episode of major depressive disorder exhibited significant depressive symptoms before the first identified episode. Therefore, early identification and treatment of early symptoms may

prevent the development of a full depressive episode. Although symptoms may have been present, patients with major depressive disorder usually have not had a premorbid personality disorder. The first depressive episode occurs before age 40 years in about 50 percent of patients. A later onset is associated with the absence of a family history of mood disorders, antisocial personality disorder, and alcohol abuse.

DURATION

An untreated depressive episode lasts 6 to 13 months; most treated episodes last about 3 months. The withdrawal of antidepressants before 3 months has elapsed almost always results in the return of the symptoms. As the course of the disorder progresses, patients tend to have more frequent episodes that last longer. Over a 20-year period, the mean number of episodes is five or six.

DEVELOPMENT OF MANIC EPISODES.

About 5 to 10 percent of patients with an initial diagnosis of major depressive disorder have a manic episode 6 to 10 years after the first depressive episode. The mean age for this switch is 32 years, and it often occurs after two to four depressive episodes. Although the data are inconsistent and controversial, some clinicians report that the depression of patients who are later classified as having bipolar I disorder is often characterized by hypersomnia, psychomotor retardation, psychotic symptoms, a history of postpartum episodes, a family history of bipolar I disorder, and a history of antidepressant-induced hypomania. Prognosis. Major depressive disorder is not a benign disorder. It tends to be chronic, and patients tend to relapse. Patients who have been hospitalized for a first episode of major depressive disorder have about a 50 percent chance of recovering in the first year. The percentage of patients recovering after repeated hospitalization decreases with passing time. Many unrecovered patients remain affected with dysthymic disorder. About 25 percent of patients experience a recurrence of major depressive disorder in the first 6 months after release from a hospital, about 30 to 50 percent in the following 2 years, and about 50 to 75 percent in 5 years. The incidence of relapse is lower than these figures in patients who continue prophylactic psychopharmacological treatment and in patients who have had only one or two depressive episodes. Generally, as a patient experiences more and more depressive episodes, the time between the episodes decreases, and the severity of each episode increases.

PROGNOSTIC INDICATORS.

Many studies have focused on identifying both good and bad prognostic indicators in the course of major depressive disorder. Mild episodes, the absence of psychotic symptoms, and a short hospital stay are good prognostic indicators. Psychosocial indicators of a good course include a history of solid

friendships during adolescence, stable family functioning, and generally sound social functioning for the 5 years preceding the illness. Additional good prognostic signs are the absence of a comorbid psychiatric disorder and of a personality disorder, no more than one previous hospitalization for major depressive disorder, and an advanced age of onset. The possibility of a poor prognosis is increased by coexisting dysthymic disorder, abuse of alcohol and other substances, anxiety disorder symptoms, and a history of more than one previous depressive episode. Men are more likely than women to experience a chronically impaired course.

VIII. Treatment

Treatment of patients with mood disorders should be directed toward several goals. First, the patient's safety must be guaranteed. Second, a complete diagnostic evaluation of the patient is necessary. Third, a treatment plan that addresses not only the immediate symptoms but also the patient's prospective well-being should be initiated. Although current treatment emphasizes pharmacotherapy and psychotherapy addressed to the individual patient, stressful life events are also associated with increases in relapse rates. Thus, treatment should address the number and severity of stressors in patients' lives. Overall, the treatment of mood disorders is rewarding for psychiatrists. Specific treatments are now available for both manic and depressive episodes, and data indicate that prophylactic treatment is also effective. Because the prognosis for each episode is good, optimism is always warranted and is welcomed by both the patient and the patient's family. Mood disorders are chronic, however, and the psychiatrist must educate the patient and the family about future treatment strategies. Hospitalization The first and most critical decision a physician must make is whether to hospitalize a patient or attempt outpatient treatment. Clear indications for hospitalization are the risk of suicide or homicide, a patient's grossly reduced ability to get food and shelter, and the need for diagnostic procedures. A history of rapidly progressing symptoms and the rupture of a patient's usual support systems are also indications for hospitalization. A physician may safely treat mild depression or hypomania in the office if he or she evaluates the patient frequently. Clinical signs of impaired judgment, weight loss, or insomnia should be minimal. The patient's support system should be strong, neither overinvolved nor withdrawing from the patient. Any adverse changes in the patient's symptoms or behavior or the attitude of the patient's support system may suffice to warrant hospitalization. Patients with mood disorders are often unwilling to enter a hospital voluntarily and may have to be involuntarily committed. These patients often cannot make decisions because of their slowed thinking, negative Weltanschauung (world view), and hopelessness. Patients who are manic often have such a complete lack of insight into their disorder that hospitalization seems absolutely absurd to them. Psychosocial Therapy Although most studies indicate—and most clinicians and

researchers believe—that a combination of psychotherapy and pharmacotherapy is the most effective treatment for major depressive disorder, some data suggest another view: Either pharmacotherapy or psychotherapy alone is effective, at least in patients with mild major depressive episodes, and the regular use of combined therapy adds to the cost of treatment and exposes patients to unnecessary adverse effects. Three types of short-term psychotherapies—cognitive therapy, interpersonal therapy, and behavior therapy—have been studied to determine their efficacy in the treatment of major depressive disorder. Although its efficacy in treating major depressive disorder is not as well researched as these three therapies, psychoanalytically oriented psychotherapy has long been used for depressive disorders, and many clinicians use the technique as their primary method. What differentiates the three short-term psychotherapy methods from the psychoanalytically oriented approach are the active and directive roles of the therapist, the directly recognizable goals, and the end points for short-term therapy. Accumulating evidence is encouraging about the efficacy of dynamic therapy. In a randomized, controlled trial comparing psychodynamic therapy with cognitive behavior therapy, the outcome of the depressed patients was the same in the two treatments. The National Institute of Mental Health (NIMH) Treatment of Depression Collaborative Research Program found the following predictors of response to various treatments: low social dysfunction suggested a good response to interpersonal therapy, low cognitive dysfunction suggested a good response to cognitive-behavioral therapy and pharmacotherapy, high work dysfunction suggested a good response to pharmacotherapy, and high depression severity suggested a good response to interpersonal therapy and pharmacotherapy.

Cognitive Therapy.

Cognitive therapy, originally developed by Aaron Beck, focuses on the cognitive distortions postulated to be present in major depressive disorder. Such distortions include selective attention to the negative aspects of circumstances and unrealistically morbid inferences about consequences. For example, apathy and low energy result from a patient's expectation of failure in all areas. The goal of cognitive therapy is to alleviate depressive episodes and prevent their recurrence by helping patients identify and test negative cognitions; develop alternative, flexible, and positive ways of thinking; and rehearse new cognitive and behavioral responses. Studies have shown that cognitive therapy is ePective in the treatment of major depressive disorder. Most studies found that cognitive therapy is equal in efficacy to pharmacotherapy and is associated with fewer adverse effects and better follow-up than pharmacotherapy. Some of the best controlled studies have indicated that the combination of cognitive therapy and pharmacotherapy is more efficacious than either therapy alone, although other studies have not found that additive effect. At least one study, the NIMH Treatment of Depression Collaborative Research Program, found that

pharmacotherapy, either alone or with psychotherapy, may be the treatment of choice for patients with severe major depressive episodes. Interpersonal Therapy. Interpersonal therapy, developed by Gerald Klerman, focuses on one or two of a patient's current interpersonal problems. This therapy is based on two assumptions. First, current interpersonal problems are likely to have their roots in early dysfunctional relationships. Second, current interpersonal problems are likely to be involved in precipitating or perpetuating the current depressive symptoms. Controlled trials have indicated that interpersonal therapy is effective in the treatment of major depressive disorder and, not surprisingly, may be specifically helpful in addressing interpersonal problems. Some studies indicate that interpersonal therapy may be the most effective method for severe major depressive episodes when the treatment choice is psychotherapy alone. The interpersonal therapy program usually consists of 12 to 16 weekly sessions and is characterized by an active therapeutic approach. Intrapsychic phenomena, such as defense mechanisms and internal conflicts, are not addressed. Discrete behaviors—such as lack of assertiveness, impaired social skills, and distorted thinking—may be addressed but only in the context of their meaning in, or their ePect on, interpersonal relationships.

Behavior Therapy.

Behavior therapy is based on the hypothesis that maladaptive behavioral patterns result in a person's receiving little positive feedback and perhaps outright rejection from society. By addressing maladaptive behaviors in therapy, patients learn to function in the world in such a way that they receive positive reinforcement. Behavior therapy for major depressive disorder has not yet been the subject of many controlled studies. The limited data indicate that it is an effective treatment for major depressive disorder. Psychoanalytically Oriented Therapy. The psychoanalytic approach to mood disorders is based on psychoanalytic theories about depression and mania. The goal of psychoanalytic psychotherapy is to effect a change in a patient's personality structure or character, not simply to alleviate symptoms. Improvements in interpersonal trust, capacity for intimacy, coping mechanisms, the capacity to grieve, and the ability to experience a wide range of emotions are some of the aims of psychoanalytic therapy. Treatment often requires the patient to experience periods of heightened anxiety and distress during the course of therapy, which may continue for several years. Family Therapy. Family therapy is not generally viewed as a primary therapy for the treatment of major depressive disorder, but increasing evidence indicates that helping a patient with a mood disorder to reduce and cope with stress can lessen the chance of a relapse. Family therapy is indicated if the disorder jeopardizes a patient's marriage or family functioning or if the mood disorder is promoted or maintained by the family situation. Family therapy examines the role of the mood-disordered member in the overall psychological wellbeing of the whole family; it also examines the role of the

entire family in the maintenance of the patient's symptoms. Patients with mood disorders have a high rate of divorce, and about 50 percent of all spouses report that they would not have married or had children if they had known that the patient was going to develop a mood disorder.

Vagal Nerve Stimulation

Experimental stimulation of the vagus nerve in several studies designed for the treatment of epilepsy found that patients showed improved mood. This observation led to the use of left vagal nerve stimulation (VNS) using an electronic device implanted in the skin, similar to a cardiac pacemaker. Preliminary studies have shown that a number of patients with chronic, recurrent major depressive disorder went into remission when treated with VNS. The mechanism of action of VNS to account for improvement is unknown. The vagus nerve connects to the enteric nervous system and, when stimulated, may cause release of peptides that act as neurotransmitters. Extensive clinical trials are being conducted to determine the efficacy of VNS.

Transcranial Magnetic Stimulation

Transcranial magnetic stimulation (TMS) shows promise as a treatment for depression. It involves the use of very short pulses of magnetic energy to stimulate nerve cells in the brain. It is specifically indicated for the treatment of depression in adult patients who have failed to achieve satisfactory improvement from one prior antidepressant medication at or above the minimal effective dose and duration in the current episode. Repetitive transcranial magnetic stimulation (rTMS) produces focal secondary electrical stimulation of targeted cortical regions. It is nonconvulsive, requires no anesthesia, has a safe side effect profile, and is not associated with cognitive side effects. The patients do not require anesthesia or sedation and remain awake and alert. It is a 40-minute outpatient procedure that is prescribed by a psychiatrist and performed in a psychiatrist's office. The treatment is typically administered daily for 4 to 6 weeks. The most common adverse event related to treatment was scalp pain or discomfort. TMS therapy is contraindicated in patients with implanted metallic devices or nonremovable metallic objects in or around the head. Sleep Deprivation Mood disorders are characterized by sleep disturbance. Mania tends to be characterized by a decreased need for sleep, but depression can be associated with either hypersomnia or insomnia. Sleep deprivation may precipitate mania in patients with bipolar I disorder and temporarily relieve depression in those who have unipolar depression. Approximately 60 percent of patients with depressive disorders exhibit significant but transient benefits from total sleep deprivation. The positive results are typically reversed by the next night of sleep. Several strategies have been used in an attempt to achieve a more sustained response to sleep deprivation. One method used serial total sleep

deprivation with a day or two of normal sleep in between. This method does not achieve a sustained antidepressant response because the depression tends to return with normal sleep cycles. Another approach used phase delay in the time patients go to sleep each night, or partial sleep deprivation. In this method, patients may stay awake from 2 AM to 10 PM daily. Up to 50 percent of patients get same-day antidepressant effects from partial sleep deprivation, but this benefit also tends to wear off in time. In some reports, however, serial partial sleep deprivation has been used successfully to treat insomnia associated with depression. The third, and probably most effective, strategy combines sleep deprivation with pharmacological treatment of depression. A number of studies have suggested that total and partial sleep deprivation followed by immediate treatment with an antidepressant or lithium (Eskalith) sustains the antidepressant effects of sleep deprivation. Likewise, several reports have suggested that sleep deprivation accelerates the response to antidepressants, including fluoxetine (Prozac) and nortriptyline (Aventyl, Pamelor). Sleep deprivation has also been noted to improve premenstrual dysphoria.

Phototherapy

Phototherapy (light therapy) was introduced in 1984 as a treatment for SAD (mood disorder with seasonal pattern). In this disorder, patients typically experience depression as the photoperiod of the day decreases with advancing winter. Women represent at least 75 percent of all patients with seasonal depression, and the mean age of presentation is 40 years. Patients rarely present older than the age of 55 years with seasonal affective disorder. Phototherapy typically involves exposing the affected patient to bright light in the range of 1,500 to 10,000 lux or more, typically with a light box that sits on a table or desk. Patients sit in front of the box for approximately 1 to 2 hours before dawn each day, although some patients may also benefit from exposure after dusk. Alternatively, some manufacturers have developed light visors, with a light source built into the brim of the hat. These light visors allow mobility, but recent controlled studies have questioned the use of this type of light exposure. Trials have typically lasted 1 week, but longer treatment durations may be associated with greater response. Phototherapy tends to be well tolerated. Newer light sources tend to use lower light intensities and come equipped with filters; patients are instructed not to look directly at the light source. As with any effective antidepressant, phototherapy, on rare occasions, has been implicated in switching some depressed patients into mania or hypomania. In addition to seasonal depression, the other major indication for phototherapy may be in sleep disorders. Phototherapy has been used to decrease the irritability and diminished functioning associated with shift work. Sleep disorders in geriatric patients have reportedly improved with exposure to bright light during the day. Likewise, some evidence suggests that jet lag might respond to light therapy. Preliminary

data indicate that phototherapy may benefit some patients with OCD that has a seasonal variation.

1. Psychopharmacologic.

Major Depressive Disorder. The use of specific pharmacotherapy approximately doubles the chances that a depressed patient will recover in 1 month. All currently available antidepressants may take up to 3 to 4 weeks to exert significant therapeutic effects, although they may begin to show their effects earlier. Choice of antidepressants is determined by the side effect profile least objectionable to a given patient's physical status, temperament, and lifestyle. That numerous classes of antidepressants (Table 8.1-10) are available, many with different mechanisms of action, represents indirect evidence for heterogeneity of putative biochemical lesions. Although the first antidepressant drugs, the monoamine oxidase inhibitors (MAOIs) and tricyclic antidepressants (TCAs), are still in use, newer compounds have made the treatment of depression more "clinician and patient friendly."

GENERAL CLINICAL GUIDELINES.

The most common clinical mistake leading to an unsuccessful trial of an antidepressant drug is the use of too low a dosage for too short a time. Unless adverse events prevent it, the dosage of an antidepressant should be raised to the maximum recommended level and maintained at that level for at least 4 or 5 weeks before a drug trial is considered unsuccessful. Alternatively, if a patient is improving clinically on a low dosage of the drug, this dosage should not be raised unless clinical improvement stops before maximal benefit is obtained. When a patient does not begin to respond to appropriate dosages of a drug after 2 or 3 weeks, clinicians may decide to obtain a plasma concentration of the drug if the test is available for the particular drug being used. The test may indicate either noncompliance or particularly unusual pharmacokinetic disposition of the drug and may thereby suggest an alternative dosage.

DURATION AND PROPHYLAXIS.

Antidepressant treatment should be maintained for at least 6 months or the length of a previous episode, whichever is greater. Prophylactic treatment with antidepressants is effective in reducing the number and severity of recurrences. One study concluded that when episodes are less than 21/2 years apart, prophylactic treatment for 5 years is probably indicated. Another factor suggesting prophylactic treatment is the seriousness of previous depressive episodes. Episodes that have involved significant suicidal ideation or impairment of psychosocial functioning may indicate that clinicians should consider prophylactic treatment. When antidepressant treatment is stopped, the drug dose should be tapered gradually over 1 to 2 weeks, depending on the half-

life of the particular compound. Several studies indicate that maintenance antidepressant medication appears to be safe and effective for the treatment of chronic depression. Prevention of new mood episodes (i.e., recurrences) is the aim of the maintenance phase of treatment. Only patients with recurrent or chronic depressions are candidates for maintenance treatment.

INITIAL MEDICATION SELECTION.

The available antidepressants do not differ in overall efficacy, speed of response, or long-term effectiveness. Antidepressants, however, do differ in their pharmacology, drug–drug interactions, short- and long-term side effects, likelihood of discontinuation symptoms, and ease of dose adjustment. Failure to tolerate or to respond to one medication does not imply that other medications will also fail. Selection of the initial treatment depends on the chronicity of the condition, course of illness (a recurrent or chronic course is associated with increased likelihood of subsequent depressive symptoms without treatment), family history of illness and treatment response, symptom severity, concurrent general medical or other psychiatric conditions, prior treatment responses to other acute phase treatments, potential drug–drug interactions, and patient preference. In general, approximately 45 to 60 percent of all outpatients with uncomplicated (i.e., minimal psychiatric and general medical comorbidity), nonchronic, nonpsychotic major depressive disorder who begin treatment with medication respond (i.e., achieve at least a 50 percent reduction in baseline symptoms); however, only 35 to 50 percent achieve remission (i.e., the virtual absence of depressive symptoms).

TREATMENT OF DEPRESSIVE SUBTYPES.

Clinical types of major depressive episodes may have varying responses to particular antidepressants or to drugs other than antidepressants. Patients with major depressive disorder with atypical features may preferentially respond to treatment with MAOIs or SSRIs. Antidepressants with dual action on both serotonergic and noradrenergic receptors demonstrate greater efficacy in melancholic depressions. Patients with seasonal winter depression can be treated with light therapy. Treatment of major depressive episodes with psychotic features may require a combination of an antidepressant and an atypical antipsychotic. Several studies have also shown that ECT is effective for this indication—perhaps more effective than pharmacotherapy. For those with atypical symptom features, strong evidence exists for the effectiveness of MAOIs. SSRIs and bupropion (Wellbutrin) are also of use in atypical depression.

COMORBID DISORDERS.

The concurrent presence of another disorder can affect initial treatment selection. For example, the successful treatment of OCD associated with depressive symptoms usually results in remission of the depression. Similarly, when panic disorder occurs with major depression, medications with demonstrated efficacy in both conditions are preferred (e.g., tricyclics and SSRIs). In general, the nonmood disorder dictates the choice of treatment in comorbid states. Concurrent substance abuse raises the possibility of a substance-induced mood disorder, which must be evaluated by history or by requiring abstinence for several weeks. Abstinence often results in remission of depressive symptoms in substance-induced mood disorders. For those with continuing significant depressive symptoms, even with abstinence, an independent mood disorder is diagnosed and treated. General medical conditions are established risk factors in the development of depression. The presence of a major depressive episode is associated with increased morbidity or mortality of many general medical conditions (e.g., cardiovascular disease, diabetes, cerebrovascular disease, and cancer).

THERAPEUTIC USE OF SIDE EFFECTS.

Choosing more sedating antidepressants (e.g., amitriptyline [Elavil, Endep]) for more anxious, depressed patients or more activating agents (e.g., desipramine) for more psychomotor-retarded patients is not generally helpful. For example, any short-term benefits with paroxetine, mirtazapine, or amitriptyline (more sedating drugs) on symptoms of anxiety or insomnia may become liabilities over time. These drugs often continue to be sedating in the longer run, which can lead to patients prematurely discontinuing medication and increase the risk of relapse or recurrence. Some practitioners use adjunctive medications (e.g., sleeping pills or anxiolytics) combined with antidepressants to provide more immediate symptom relief or to cover those side effects to which most patients ultimately adapt. A patient's prior treatment history is important because an earlier response typically predicts current response. A documented failure on a properly conducted trial of a particular antidepressant class (e.g., SSRIs, tricyclics, or MAOIs) suggests choosing an agent from an alternative class. The history of a Orst-degree relative responding to a particular drug is associated with a good response to the same class of agents in the patient.

ACUTE TREATMENT FAILURES.

Patients may not respond to a medication, because (1) they cannot tolerate the side effects, even in the face of a good clinical response; (2) an idiosyncratic adverse event may occur; (3) the clinical response is not adequate; or (4) the wrong diagnosis has been made. Acute phase medication trials should last 4 to 6 weeks to determine if meaningful symptom reduction is attained. Most (but not all) patients who ultimately respond fully show at least a partial response (i.e., at

least a 20 to 25 percent reduction in pretreatment depressive symptom severity) by week 4 if the dose is adequate during the initial weeks of treatment. Lack of a partial response by 4 to 6 weeks indicates that a treatment change is needed. Longer time periods—8 to 12 weeks or longer—are needed to define the ultimate degree of symptom reduction achievable with a medication. Approximately half of patients require a second medication treatment trial because the initial treatment is poorly tolerated or ineffective.

SELECTING SECOND TREATMENT OPTIONS.

When the initial treatment is unsuccessful, switching to an alternative treatment or augmenting the current treatment is a common option. The choice between switching from the initial single treatment to a new single treatment (as opposed to adding a second treatment to the first one) rests on the patient's prior treatment history, the degree of benefit achieved with the initial treatment, and patient preference. As a rule, switching rather than augmenting is preferred after an initial medication failure. On the other hand, augmentation strategies are helpful with patients who have gained some benefit from the initial treatment but who have not achieved remission. The best-documented augmentation strategies involve lithium (Eskalith) or thyroid hormone. A combination of an SSRI and bupropion (Wellbutrin) is also widely used. In fact, no combination strategy has been conclusively shown to be more effective than another. ECT is effective in psychotic and nonpsychotic forms of depression but is recommended generally only for repeatedly nonresponsive cases or in patients with very severe disorders. A new therapy involves the use of the anesthetic agent ketamine, which has been shown to be ePective in treatment resistant depression. It has a mechanism of action that inhibits the postsynaptic glutamate binding protein N-methyl-D-aspartate (NDMA) receptor. Because abnormalities in glutamatergic signaling have been implicated in major depressive disorder, this may account for its eQcacy. Patients usually receive a single infusion of ketamine over a 30-minute period at a concentration of 0.5 mg/kg. A positive response is usually seen within 24 hours, and improved mood lasts for about 2 to 7 days. The most common side ePects are dizziness, headache, and poor coordination, which are transitory. Dissociative symptoms, including hallucinations, may also occur.

COMBINED TREATMENT.

Medication and formal psychotherapy are often combined in practice. If physicians view mood disorders as fundamentally evolving from psychodynamic issues, their ambivalence about the use of drugs may result in a poor response, noncompliance and probably inadequate dosages for too short a treatment period. Alternatively, if physicians ignore the psychosocial needs of a patient, the outcome of pharmacotherapy may be compromised. Several trials of

a combination of pharmacotherapy and psychotherapy for chronically depressed outpatients have shown a higher response and higher remission rates for the combination than for either treatment used alone

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 DEPRESSION AND ITS TREATMENT METHODS

S.No	Register No	Students List	Department	SIGNATURE
1	U14MB311	SHALINI. T. C.	Psychiatry	T. C. Shalini
2	U14MB312	SHANMUGA RAJA. A	Psychiatry	Shanmuga Raja. A
3	U14MB313	SHANMUHA PRIYA. S	Psychiatry	S. Shanmuha
4	U14MB314	SHAREEFA AKHTAR.S	Psychiatry	Shareefa Akhtara. S.
5	U14MB315	SHEMBIYAN. R.M.	Psychiatry	Shembiyan. R.M.
6	U14MB316	SIKKANDAR. A	Psychiatry	Sikkandar. A.
7	U14MB317	SINDHU. M	Psychiatry	Sindhu.
8	U14MB318	SIVARAJ. S	Psychiatry	
9	U14MB319	SOUNDARYA. S	Psychiatry	
10	U14MB320	SOWMYA DEVI. N	Psychiatry	
11	U14MB321	SOWWMYA. S	Psychiatry	

12	U14MB322	SOWMYAMANJA MADHA.I	Psychiatry	
13	U14MB323	SREEDEVI. B	Psychiatry	
14	U14MB324	SREEPADMA PURUJITH. S.S.	Psychiatry	
15	U14MB325	SRI JAYALAKSHMI. A	Psychiatry	

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MULTIPLE CHOICE QUESTIONS

Classification of depression and its treatment methods

Course Code: PSYC02

Candidate Name		Assessor Name	
Date of Assessment		Assessor Position	

I. Multiple Choice Question

2) Depressed individuals exhibit which of the following symptoms?

- a) Behavioural symptoms.
- b) Physical symptoms.
- c) Cognitive symptoms.
- d) All of the above.

Check your answer

3) Which of the following is a behavioural symptom exhibited by individuals suffering unipolar depression?

- a) Unpredictable and erratic behaviour.
- b) Compulsive checking.
- c) Stay in bed for long periods.
- d) Ritualised behaviour.

Check your answer

4) DSM-IV-TR criteria for a major depressive episode includes which of the following?

a) Symptoms cause clinically significant distress or impairment in social functioning.

b) Symptoms are not due to physiological effects of substance misuse.

c) Symptoms are not accounted for by bereavement.

d) All of the above.

Check your answer

5) Which of the following is a chronic mood disturbance that can cause depressive symptoms, but does not disrupt normal functioning?

a) Cyclothymic Disorder.

b) Dysthymic Disorder.

c) Dissociative disorder.

d) Personality disorder.

Check your answer

7) What percentage of adults who live in temperate climates are affected by Seasonal Affective Disorder (SAD)?

a) 1-3%.

b) 5-8%.

c) 10-12%.

d) 15-20%.

Check your answer

8) Physical symptoms of Chronic Fatigue Syndrome (CFS) include which of the following?

a) Extreme fatigue.

b) Muscle pain.

c) Chest pain.

d) All of the above.

Check your answer

9) In DSM-IV-TR Bipolar I disorder includes which of the following symptoms?

a) Currently (or most recently) in a Manic Episode.

b) The previous occurrence of at least one Major Depressive Episode, Manic Episode or Mixed Episode.

c) Mood episodes are not better accounted for by psychotic disorders.

d) All of the above.

Check your answer

10) In Bipolar II Disorder, major depressive episodes alternate with periods of:

a) Hyperventilation.

b) Hypomania.

c) Hypothermia.

d) Hypoxia.

Check your answer

11) In Major Depression, which of the following is a significant neurotransmitter?

- a) Serotonin.
- b) Dopamine.
- c) Betacarotine.
- d) Acetylcholine.

Check your answer

XX.

12) Which of the following neurotransmitters is associated specifically with Bipolar Disorder:

- a) Serotonin.
- b) Norepinephrine.
- c) Dopamine.
- d) Acetylcholine.

Check your answer

13) Lower levels of activation in the Prefrontal Cortex results in:

- a) Failure to regulate emotions.
- b) Deficit in the will to change.
- c) Failure to anticipate incentives.
- d) Inability to understand the context of affective reactions.

SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

MULTIPLE CHOICE QUESTIONS

Classification of depression and its treatment methods

Course Code: PSYC02

Candidate Name	SIKKANDAR. A	**Assessor Name**	DR. ARUN
Date of Assessment	16.5.2016	**Assessor Position**	ASSISTANT PROFESSOR

UNIVERSITY REG NO:- U14MB316

11/13

I. Multiple Choice Question

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- ✓ c) Stay in bed for long periods.
- d) Ritualised behaviour.

Check your answer

SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

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- d) All of the above.

Check your answer

5) Which of the following is a chronic mood disturbance that can cause depressive symptoms, but does not disrupt normal functioning?

- a) Cyclothymic Disorder.
- b) Dysthymic Disorder.
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- d) Personality disorder.

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SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

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- b) Hypomania.
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SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

- d) Hypoxia.

Check your answer

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- c) Failure to anticipate incentives.

SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

MULTIPLE CHOICE QUESTIONS

Classification of depression and its treatment methods

Course Code: PSYC02

Candidate Name	SEMBIYAN.R.M	Assessor Name	DR. ARUN
Date of Assessment	16.5.2016	Assessor Position	ASSISTANT PROFESSOR

UNIVERSITY REG NO:-U14MB315

12/13

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SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

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- c) Symptoms are not accounted for by bereavement.
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SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

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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 SREEPADMA PURUJITH. S.S. has actively participated in the Value Added Course on **Classification Of Depression And Its Treatment Methods** held during January - June 2021 Organized by Sri Lakshmi Narayana Institute of Medical Sciences, Pondicherry- 605 502, India.

Dr. ARUN SEETHARAMAN

RESOURCE PERSON

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Dr. V.R.SRIDHAR

COORDINATOR

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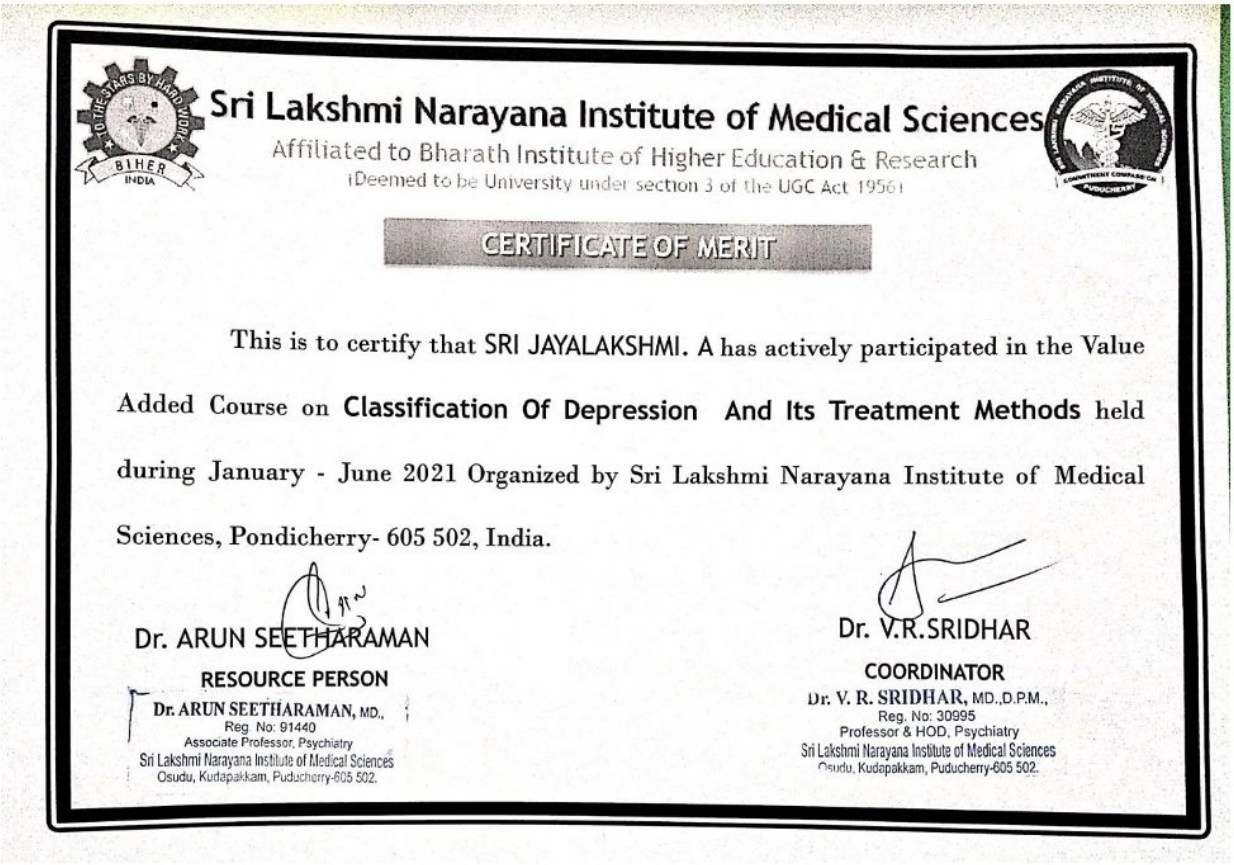
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Kudapakkam, Puducherry-605 502.



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Dr. ARUN SEETHARAMAN

RESOURCE PERSON

Dr. ARUN SEETHARAMAN, MD.,
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Dr. V.R.SRIDHAR

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SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

Student Feedback Form

SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

Course Name: **DEPRESSION**

Subject Code: **PSYC02**

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:

Signature

Date:

SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

Student Feedback Form

Course Name: **DEPRESSION**

Subject Code: **PSYC02**

Name of Student: SEMBIYAN · R · M Roll No.: U14MB315

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: **DEPRESSION**

Subject Code: **PSYC02**

Name of Student: SIKKANDAR . A Roll No.: U14MB316

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:

Signature

Date: 30-6-2016

SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

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: Identification And Classification Of Depression And Its Management

Dear Sir,

With reference to the subject mentioned above, the department has conducted the value-added course titled: **Awareness, Identification And Classification Of Depression And Its Management**. 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

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