

Inpatient Psychiatry

Diagnosis and Treatment

Lloyd I. Sederer, M.D.
EDITOR

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To my son.

preface

Two quite separate considerations prompted the writing of this text. First was my interest in providing a theoretical and pragmatic basis for the evaluation and treatment of the hospitalized psychiatric patient. In my teaching responsibilities I witnessed a multitude of students and clinicians of varied professional disciplines come to learn and practice on an inpatient unit and find no *specific* body of knowledge or reference text to which they could turn.

There has emerged, in the past few decades, what must be considered a subspecialty practice in psychiatry. The decline of state mental hospitals coupled with the increasing affiliation of psychiatry with medicine, as well as economic incentives, has resulted in the rapid growth of inpatient units in general hospitals. In the 1940's there were a few dozen such units; there are now 1700. Though general hospital units most clearly represent the development of short to intermediate stay inpatient evaluation and treatment facilities, these units are also readily found in private, governmental and, occasionally, state psychiatric hospitals. It is the vigorous emergence of these subspecialty inpatient units that was the second motivation for this manuscript. This text represents an effort to begin to define and academically organize the subspecialty of Inpatient Psychiatry as it is currently and commonly practiced.

The work for this book occurred as the *Diagnostic and Statistical Manual of Mental Disorders*, ed. 3, of the American Psychiatric Association (DSM-III) was published and popularized. DSM-III is an effort to achieve diagnostic reliability through explicit diagnostic criteria. Furthermore, DSM-III introduces a multiaxial system of diagnosis that emphasizes how personality traits and disorders (Axis II) may coexist with and influence a major psychiatric disorder (Axis I). The additional three axes (medical, stressors, and highest level of adaptation) complement the psychiatric and personality axes in a manner that permits further individual specificity and greater prognostic accuracy. DSM-III is a descriptive document in the tradition of Kraepelin. It is not the only system of understanding, nor is it the bearer of Platonic truth; it is, however, the single most important advance in contemporary nomenclature and, as such, has been utilized throughout this text.

The reader will encounter a somewhat overlapping yet rather different multiaxial or, better, multiconceptual perspective in the first section of the text, Inpatient Diagnosis and Treatment. The authors have endeavored to present etiological and therapeutic considerations from biological (disease), psychological (person), and social perspectives (circumstance) to the extent they are known or are applicable to the specific disorders in this section. Underlying this presentation is a philosophical adherence to a multiconceptual schema for understanding and action in psychiatric practice.

The second section of the text, Specific Aspects of Inpatient Psychiatry, aims

to be more microscopic. Herein lies a more detailed account of the treatments, populations and resources unique to the subspecialty of Inpatient Psychiatry.

Throughout the text, the authors have used the pronoun *he* (or his) to refer to male or female. The he/she construction may help reduce gender bias but I find it linguistically awkward. In the absence of a more suitable alternative, I have chosen to follow standard English practice.

My acknowledgments begin with the patients, trainees, permanent staff and consultants of the Inpatient Psychiatric Service of the Massachusetts General Hospital. Together we have created a laboratory for learning to heal, the essence of which this book seeks to capture.

Nancy Regan, my secretary, has been an assistant *par excellence*. Her fine work and kind demeanor were most welcome throughout the book's production.

My fortunate proximity to Dr. Aaron Lazare, whose outpatient text stands as a testimony to his creative industry and spirit, served as an important inspiration to me. To Dr. Robert Seidenberg I will be forever grateful, for it was his love of writing and teaching, and his encouragement, that were instrumental to my development as an author.

I wish to thank my friends, whose camaraderie helped to bring me to the point of this adventure, and Mr. James Sangston, my Editor at Williams and Wilkins, for the trust he held in me to proceed. To Dr. Jane Thorbeck go my greatest thanks for her unyielding insistence on clarity and accuracy and for her critical intellectual assistance. Finally, I would like to thank my contributors, who taught me how to become an Editor.

L. I. S.

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part 1

inpatient diagnosis and treatment

depression

Lloyd I. Sederer, M.D.

DEFINITION

Depression is the most common of psychiatric disorders. It has been estimated that in excess of 15% of all adults will experience a depressive episode at some point in the course of their lives.^{1,2} Depression is the most common cause for psychiatric hospitalization³ and is the “bread and butter” of outpatient psychiatric practice. Furthermore, depression is widely found among medically and surgically hospitalized patients.

When depression is severe, persistent, and disabling of everyday bodily and social functioning, it is easily discernible. At other times the distinction between a normal fluctuation of mood and a depression may not be very clear. Feelings of sadness, blueness, frustration, and discouragement are part of the normal range of human emotions. These fluctuations in mood, however, tend to be short-lasting, do not become overwhelming in their experience, do not impair reality testing, and do not generate suicidal thoughts or behavior. In addition, normal mood fluctuations do not produce persistent disturbances in sleep, appetite, or motoric activity.

The central feature of clinical depression generally is a subjective experience of sadness, despondency, hopelessness, or gloom. This feeling of depressed mood is accompanied by a loss of interest and pleasure in life and its activities and responsibilities. Some patients will present with anxiety as the prominent mood disturbance, whereas other patients may present with the experience of agitation as their major subjective emotional complaint. In some cases, patients will report no mood disturbance, despite the presence of a host of other symptoms and clear cause for a mood disturbance.

A feeling of lowered self-esteem is common, as are feelings of helplessness. Depressed patients show an inability to perform even the simplest of daily tasks. They are frequently preoccupied (sometimes obsessed) with work, family, money, and their own health. They approach these matters with marked pessimism and hopelessness. In some patients, hopelessness, pessimism, extremely low self-esteem, and guilt, in concert, prompt thoughts of death and suicide.

The predominance of depressed patients show a loss of appetite and consequent weight loss. Some patients, many of whom are younger and show milder depressions, present with symptoms of increased appetite with consequent weight gain.

Sleep disturbance is a very common symptom of depression. Disturbances of sleep are described according to whether they present as difficulty falling asleep, difficulty remaining asleep, or early morning awakening (initial, middle, and terminal insomnia).

Many patients have psychomotor disturbances. Those patients with an increase in psychomotor activity are described as having agitation. They may be unable to remain still; instead they pace about, wring their hands, bite their nails, smoke, or talk incessantly. Those patients showing decreased psychomotor activity are described as psychomotorically retarded. These patients typically complain of lethargy and fatigue. Objectively, their body movements are slowed and limited and there is a poverty, monotony, and latency to their speech. In some severely retarded patients, a clinical syndrome approaching catatonia may occur. In these cases, the patient is virtually mute and is without spontaneous movement.⁴ Self-care slips away and the patient shows no interest in eating or taking care of his bodily needs.

Decreased libido is a common finding in depression. The loss of libido reflects a general loss of cathexis, or energy, for living. Work, play, friends, and family are all neglected.

Difficulty with concentration and memory may occur. Thinking may be slowed and indecision frequent. These cognitive disturbances can become so marked as to appear like dementia. Patients in whom the primary disturbance is depression and who demonstrate a severe, cognitive disorder that mimics dementia are said to have depressive pseudodementia.

Furthermore, many depressed patients complain of bodily disturbances involving almost every organ in the body. Gastrointestinal disturbances, headache, backache, and urinary difficulties are common. Often, patients with a mild, pre-existing bodily disorder will present with an exacerbation of this symptomatology. In these cases, the patients come to the attention of their physicians because of their physical complaints. Upon more careful examination the diagnosis of depression aggravating their pre-existing disorder can be made.

In a small number of depressed patients, disturbances in reality functioning may occur. These are called delusional or psychotic depressions. These patients show delusions and/or hallucinations that tend to reflect the person's sense of self-reproach or pessimism. Examples include somatic delusions or auditory hallucinations that are highly critical.

In essence, depression is a syndrome characterized by a persistent, severe and abnormal disturbance of mood, with neurovegetative symptomatology, and with or without psychosis. The syndrome of depression has varied etiologies which are discussed later in this chapter.

DIAGNOSIS

The *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III) of the American Psychiatric Association⁵ offers the following diagnostic criteria for a major (clinical) depression:

1. A dysphoric (depressed, blue, sad, irritable, hopeless) mood or loss of interest in most daily activities. This dysphoria does not show transient shifts from one mood to another. The mood disturbance of depression is per-

sistent and prominent.

2. At least four of the following symptoms that have been present daily for at least 2 weeks:
 - a. decreased appetite and weight loss or increased appetite and weight gain
 - b. insomnia or hypersomnia
 - c. psychomotor agitation or retardation
 - d. a loss of interest or pleasure (anhedonia); decreased libido
 - e. loss of energy and fatigue
 - f. feelings of worthlessness, self-reproach, or excessive and inappropriate guilt (which may reach delusional proportions);
 - g. subjective complaints or objective evidence of decreased ability to think or concentrate; memory difficulties and indecisiveness without the presence of loosened associations or incoherence
 - h. recurrent thoughts of death, suicidal ideation, wishes to be dead, or a history of attempted suicide
3. This symptom picture is not superimposed on schizophrenia, a schizophreniform disorder, or a paranoid disorder.
4. This disorder is not due to any organic mental disorder or uncomplicated bereavement.

In summary, the DSM-III defines depression as a particular constellation of symptoms lasting for at least 2 or more weeks. DSM-III does allow for distinctions between psychotic and nonpsychotic depressions, but it does not concern itself with specific syndromal or etiological differences between psychotic and nonpsychotic depressions.

Nosological research in psychiatry over recent years has attempted to develop valid and reliable systems for classifying affective disorders. Figure 1.1 shows a widely accepted nosology of the affective disorders and of depression in particular. This diagnostic system provides a distinction between primary and secondary affective disorders. Primary affective disorders (whether they are depressive or manic in nature) have no previous history of another psychiatric disorder and are not secondary to any systemic medical disease. Within the primary affective disorder group is the dis-

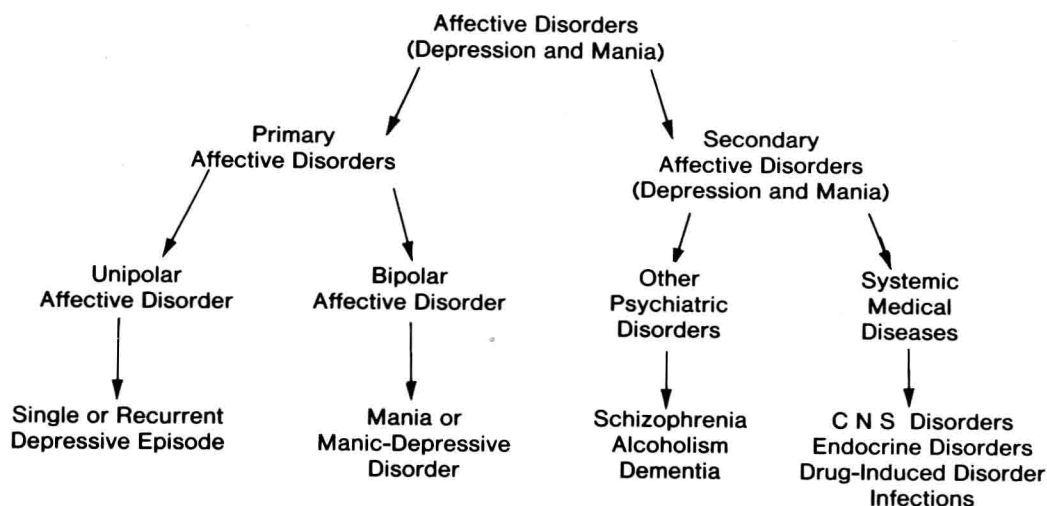


Figure 1.1. Nosology of depression.^{6,7}

inction between bipolar and unipolar affective disorders. Bipolar affective disorders are mood disorders in which the patient demonstrates either mania or a history of both manic and depressive episodes. A unipolar affective disorder is the contemporary nomenclature for a primary depressive illness.

The primary-secondary diagnostic distinction grew out of efforts to enhance research of the depressive disorders.^{7,8} This distinction allows the researcher to examine the depressive syndrome on the basis of etiologic differences. Questions of reactive versus endogenous depression and psychotic versus neurotic depression do not pertain to this diagnostic scheme. The unipolar and bipolar distinctions have become increasingly important as greater specificity in the treatment of the mood disorders has been gained. Research in psychopharmacology indicates significant differences between bipolar and unipolar patient response to lithium and to the antidepressant drugs.

Over half a century ago Gillespie⁹ proposed a nosology of depression in which reactive and endogenous terms were applied. These are terms that remain alive, though they are less popular and do create some semantic confusion. Klerman¹⁰ has suggested that the term endogenous depression signifies more than a lack of precipitating event or external cause. He has suggested that endogenous depressions

show certain "state" characteristics as well as an autonomy to the disorder that renders it unresponsive to environmental alterations. Furthermore, there is data to support abnormalities in neurophysiology and specific differences in response to organic treatment for the so-called endogenous depressions.¹¹⁻¹³ What is clear is that the biological treatment of depression is symptom specific. The presence of certain neurovegetative signs (especially early morning awakening, weight loss, and psychomotor retardation) all auger a good response to antidepressant medication or electroconvulsive therapy (ECT). Furthermore, in support of this observation, patients with endogenous depression are responsive to combined treatment with medication and psychotherapy but are not responsive to psychotherapy alone. On the other hand, patients with reactive (situational) depression have been shown to be responsive to either tricyclic medication or psychotherapy alone.¹⁴

The neurotic-psychotic distinction in the nosology of depression bears a few words. It is of crucial importance to diagnose the presence of psychotic features because this calls for the use of antipsychotic medications or ECT rather than the use of tricyclics or psychotherapy alone. The term neurotic depression has fallen into disuse and appropriately so. It has come to mean so many different things to so many different people that it has lost its clinical utility.¹⁵

Recent nosological efforts in the study of depression offer some fascinating possibilities for advancing the understanding of the unipolar depressions. The work of Andreasen and Winokur¹⁶ suggests that genetic or familial methodology allows for increased nosological specificity. Figure 1.2 details a proposed classification for subtypes of unipolar depression.

This proposed classification suggests a continuum from nonfamilial to pure to depressive spectrum disorders. Nonfamilial (or sporadic) depressive diseases show no family history of alcoholism, antisocial personality, or depression. Pure depressive disease is diagnosed in the depressed patient who has a family history only of affective disorder. Depression spectrum disease is a disorder in which the depressed person has a family history of alcoholism or antisocial personality, disorders which appear to form a continuum with depression.^{17, 18}

The core symptom picture of mood and neurovegetative symptomatology occurs primarily in pure and nonfamilial depressive disease. Depression spectrum disease tends to show features of helplessness, anxiety, and transient psychosis, which suggests that this disorder is a less "pure" form of depression with features not uniquely characteristic of a depression. The psychopathology often resembles that of a personality disorder. An important finding is that patients with nonfamilial and pure depression show treatment response to antidepressants, whereas those patients with depression spectrum disease are less responsive to antidepressants and, interestingly, show a response to neuroleptic medication. The depression spectrum patients may be more responsive to treatment with monoamine oxidase inhibitors (MAOI) than to tricyclic antidepressants (TCA). These are early suggestive findings and not definitive nosological and treatment statements. Hopefully, future research will further clarify these clinical questions.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of depression requires a thorough knowledge of organic and functional (nonorganic) disorders that can present looking like depressive syndromes. Failure to separate an organic disorder will result not only in the patient's failure to respond to treatment for his ersatz depression but will also allow another disorder to progress without diagnosis and specific treatment. The failure to separate a depressive illness from a variety of other functional psychiatric disorders will result in lack of specificity of treatment, with a consequent effect on course and prognosis.

Organic Disorders that Can Mimic Depression

The first order of business for the inpatient psychiatrist is to rule out any organic disorder that presents mimicking a depression. The clinician also must keep in mind that a functional depressive illness may accompany a medical illness. As a consequence, it is important not to let the presence of a powerful precipitating event or a past history of depression bias the clinician against considering an organic etiology for the current depressive episode.

Table 1.1 presents an extensive differential diagnosis for the organic causes of depression. Careful history-taking with additional, collaborative information from family members, a full review of systems, a complete physical and neurological examination, as well as appropriate laboratory tests (see pages 10-12), will inform the clinician of the possibility of an organic disorder.

Functional Disorders that May Be Confused with Depression

There are a variety of functional, or non-organic, disorders that may present as depression.

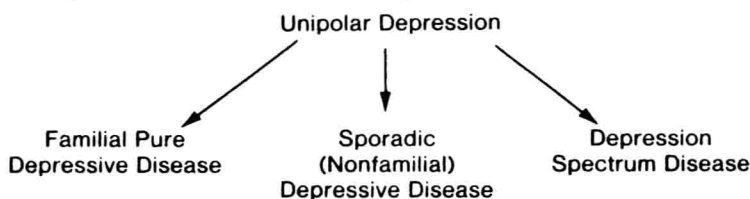


Figure 1.2. Proposed classification for unipolar depression.^{16, 65}

Table 1.1
Organic Causes of Depression*

I. Drugs and Poisons	
Amphetamine	Other sedatives
Cocaine	Bromides
Reserpine	Digitalis
Methyldopa	Steroids
Alcohol	Oral contraceptives
Antabuse	Lead poisoning
Propanolol	Other heavy metals
Opiates	Carbon disulfide
Barbiturates	
II. Metabolic Disturbances and Endocrine Disorders	
Hyperthyroidism	Diabetes
Hypothyroidism	Uremia
Hyponatremia	Hypopituitarism
Hypokalemia	Porphyria
Cushing's disease	Hepatic disease
Addison's disease	Hyperparathyroidism
Pernicious anemia	Wernicke-Korsakoff syndrome
Pellagra	
Severe anemia (any cause)	Wilson's disease
III. Infectious Diseases	
TB	Hepatitis
Subacute bacterial endocarditis	Brucellosis
Lues	Encephalitis
Mononucleosis	Postencephalitic states
IV. Degenerative Diseases	
Parkinson's disease	
Huntington's disease	
Alzheimer's disease	
Multiple sclerosis	
Other CNS degenerations	
V. Neoplasia	
Carcinomatosis	
Cancer of the pancreas	
Primary cerebral tumor	
Cerebral metastasis	
VI. Miscellaneous Conditions	
Pancreatitis	
Lupus and other collagen disorders	
Chronic pyelonephritis	
Chronic subdural hematoma	
Normal pressure hydrocephalus	
Postconcussion syndrome	
Postpartum syndrome	
Meniere's disease	

* Reproduced with permission from WH Anderson: Depression, in Lazare, A (ed): *Outpatient Psychiatry: Diagnosis and Treatment*, p. 259, Baltimore, Williams & Wilkins, 1979.

Schizophrenia

Schizophrenic patients often demonstrate depressive affect or symptomatology. Further, depressed patients can have psychotic symptoms.

In psychotically depressed patients, the affective symptoms occur first. The psychotic symptoms then follow as the disorder increases in severity. In schizophrenia, the psychotic symptoms precede the mood disturbance and the mood symptoms are of shorter duration than the psychosis. Premorbid and family histories are very helpful in differentiating schizophrenic patients from those with an affective disorder. Furthermore, the autognostic, or subjective, experience of the clinician is one of distance and difficulty in comprehending the schizophrenic patient. In contrast, the depressed patient tends to evoke feelings of sadness and empathy. Finally, schizophrenia is a diagnosis that is made only when there are persistent thought disordered symptoms lasting more than 6 months (see Chapter 3).

Schizoaffective Disorder

This disorder may be a subset of the affective disorders, though the diagnosis has come under considerable question.¹⁹ Family history, acute treatment with lithium and/or neuroleptics, and long-term outcome all support considering this an affective disorder. However, until greater nosological clarity is obtained, it may be best to avoid this diagnosis.

Dysthymic and Cyclothymic Disorders

The Dysthymic Disorder in the DSM-III is what was previously called a depressive neurosis. Though the dysthymic disorder or depressive neurosis resembles a depression, the signs and symptoms are not as severe nor as persistent as those of a depressive episode. It does happen, however, that patients with dysthymic disorders can develop a depressive episode. In these cases the diagnosis is dysthymic disorder with a unipolar depression.

Cyclothymic disorder is a chronic mood disorder in which the patient demonstrates repetitive episodes of depression and hypomania. Like the dysthymic disorder, the periods of depression do not meet the criteria for a clinical depression. Hypomania, by definition, is less severe than mania.

Other personality disorders may present with depressive features. For example, obsessive-compulsive patients can show signs of depression when esteem is low or when a loss has occurred. In all cases of person-

ality disorder, the basis for making the diagnosis of a full depressive syndrome is demonstrating the presence of a constellation of depressive symptoms that have been severe and persistent.

Uncomplicated Bereavement

Persons who have suffered the death of an intimate demonstrate a normal human response called bereavement. Bereavement may be indistinguishable in its symptomatology from a depression. Sadness and neurovegetative disturbances are common. Unlike a depression, bereavement does not tend to be associated with worthlessness and self-reproach. In addition, uncomplicated bereavement is a state that improves over the course of several months.

Bereaved patients who show persistent symptomatology for more than several months or who show severe self-reproach and persistent social disability may have developed a depression superimposed on their bereavement.

Unresolved grief is a disorder that has been recognized as a disabling syndrome. Lazare²⁰ has written articulately about this disorder.

EPIDEMIOLOGY

Depression is the most common adult psychiatric disorder. It has been estimated that in excess of 15% of adults are at risk to develop a clinical depression during the course of their lives.^{1, 2} Some estimates suggest that these figures are low because they are based on a research definition of depression. If dysthymic or neurotic depressions are included, the risk for depression in the course of a lifetime appears to rise as high as 20–30%.²¹ Furthermore, only a portion of patients with clinical depression seek out medical or psychiatric care. As a consequence, these figures may be falsely low because of a high number of unreported cases.²²

Incidence

The incidence of a disorder is the number of new cases in a given population in 1 year. Estimates for first admissions to a psychiatric facility with a diagnosis of affective disorder range from about 10–20 per 100,000. This is an incidence of 0.01–0.02%. Because approximately 10–20% of these

cases are manias, the adjusted incidence for depression would be that much less.

Prevalence

The period prevalence of a disorder is the total cases that exist in a population in a given year.

DSM-III⁵ indicates that epidemiological figures for the United States and Europe approximate that 18–23% of adult females and 8–11% of adult males have had a depressive episode at some time in their lives. It is also estimated that the depression was severe enough to require hospitalization in 6% of the females and 3% of the males.

Age, Sex, and Race

Depression may occur at any age. Admissions to a hospital for depressive disorders peak in the age group from 40 to 60.²³ Any major psychiatric disorder (excluding dementia and delirium) that presents for the first time in a person's life after the age of 35 is likely to be a depression.

Depressive disorders are twice as prevalent in women as they are in men.²⁴ This appears to be true of the range of depressive disorders, with a larger difference for less severe forms of depression. Weissman and Klerman²⁵ have demonstrated that the differences in the prevalence of depression in men and women are true findings, without methodological error or significant differences in health-seeking behavior. They begin to hypothesize that these differences are multifactorial with biological influences (either genetic or endocrinological), and psychosocial elements (such as sex discrimination or learned helplessness) acting to render the female more vulnerable to depression.

Differences in the racial distribution of depression have not been established.

Socioeconomic and Marital Status

At one time manic-depressive illness was considered a disorder of the upper and middle classes. Recent epidemiological research, however, has shown no highly valid or reliable data to indicate that depression is found in one particular socioeconomic stratum.²⁶

Several authors have hypothesized that the status disadvantage a married woman experiences is related to her vulnerability to depression.^{25, 27–30} Their data indicate