

JEROLD R. GOLD PH.D.

LEVELS  
OF  
DEPRESSION

DEPRESSIVE DISORDERS

# **Levels of Depression**

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# Levels of Depression

**Jerold R. Gold, Ph.D.**

Discussions of depression inevitably seem to convey concerns about the quantity and the topography of that disorder. Patients and clinicians are alike in their questions and descriptions, which include information about the “depth,” the “extent,” and the “severity” of the disorder. A cross-sectional study, formal or informal, of a group of depressed persons will quickly yield opinions about which patients are “more” or “less” depressed, and about “how much” those levels of depression are influencing the functioning of those individuals. Depressed persons tend to describe their past, present, and even future experiences in quantitative terms (“I’m less depressed than I was a month ago”), and topographic metaphors abound in their self-descriptions (“I’ve reached the depths,” “I hit bottom,” “I am down in the dumps”).

This chapter attempts to provide an overview of the quantity and topography of depressive phenomenology. The most typical and widely referred-to levels or types of depression will be described, as will the most salient and current conceptual and empirical concerns in the areas of nosology and diagnosis. Issues of development, etiology, course, and prognosis will be referred to but will receive only passing mention because these topics are discussed more fully and adequately elsewhere in this volume.

## ISSUES IN THE CLASSIFICATION OF LEVELS OF DEPRESSION

Depressive symptoms differ in intensity, severity, scope, and configuration from person to person. However, any account of the history of attempts to name and to classify depressive syndromes will mention the extensive debate between those who believe that depression is a single or unitary disorder, and those who accept the notion that there are two or more distinct, discrete syndromes which share certain signs and symptoms. This debate has been labeled the unitary/dualist debate (Beck, 1967), the monist/dualist controversy (Nemiah, 1975), and the one-factor/two-factor controversy (Levitt, Lubin, & Brooks, 1983), and it probably has other names in the literature as well.

The unitary or single-disorder position usually is linked to the work of Aubrey Lewis in England in the 1930s (Beck, 1967). Lewis is described as introducing the *spectrum* conception to the study of depression, which holds that the different presentations of depression, in terms of scope and severity, are merely varying surface manifestations of a continuous, single, underlying disorder. The binary or two-disorder position can be traced to the work of Kraepelin (1921), who identified two distinct and separate types of depression, which he named manic-depressive illness, and psychogenic depression.

Since Kraepelin's time, a plethora of dichotomies have been applied to describe and to name the specific levels or subtypes of depression. Beck (1967) listed four major dualities which have been used in the context of binary theory:

agitated vs. retarded depression, endogenous vs. exogenous depression, reactive vs. autonomous depression, and neurotic vs. psychotic depression. Levitt et al., (1983) added to this list the terms manic-depressive psychosis vs. psychogenic depression, vital vs. personal depression, and physiological vs. psychological depression. Rush (1986) extended this list further with the dimensions of psychotic vs. nonpsychotic depression, primary vs. secondary depression, anergic-hypoactive vs. anxious-agitated, and the triad of familial pure depression vs. depressive spectrum disorder vs. sporadic depression. This surely is not an exhaustive list of available names and dichotomies.

The official *Diagnostic and Statistical Manuals* (DSM) of the American Psychiatric Association (1952, 1968, 1980, 1987) introduced their own terminology into the general framework of the binary, two-disorder theory. The particular diagnostic labels used in these manuals will be discussed in detail in the next section of this chapter.

A substantial research literature has developed out of the unitary-binary controversy, in an attempt to determine the “true” nature of depression. A complete answer to this question, if in fact it does exist, has eluded students of depression to this date, although some authors insist that partial solutions have been found. These research efforts have been handicapped by the lack of consensual, validated, and reliable operational definitions of depression and of the various hypothesized subtypes which are the foci of the studies (Zung, 1977).

These research efforts have been hindered also by diagnostic biases across settings and examiners, and by instruments and interviews of questionable validity and reliability (Levitt et al., 1983).

Beck (1967) concluded a review of the research that was available in the mid-1960s by stating that these studies had failed to establish clear, qualitative differences between Type 1 depressions (endogenous, autonomous, psychotic) and Type 2 depressions (neurotic, reactive, exogenous), but that quantitative differences between these subtypes were robust and reliable, which Beck believed lent some credence to two-factor theory. Mendels and Cochrane (1968) focused on a group of factor-analytic studies of depressive symptomatology and found that these studies consistently yielded two orthogonal factors. These factors were judged to be consistent with the clinical stereotypes of reactive and endogenous depression, with the major differentiation being the loading of the latter on somatic symptoms. However, these studies have been critiqued and to some (Kendell, 1977), discredited, because of methodological and conceptual flaws inherent in the factor-analytic procedure.

An extensive cross-cultural study of the presentation and phenomenology of depression was conducted by the World Health Organization in the early 1980s (1983). This study included 1,209 patients drawn from psychiatric centers in Teheran, Iran; Basel, Switzerland; Montreal, Canada; Tokyo, Japan; and Nagasaki, Japan. The subjects were assessed via a series of interviews and with specially



constructed questionnaires, and the data were analyzed through a variety of factor-analytic and multivariate statistical procedures. The WHO researchers identified three distinct levels or types of depression which differed reliably with respect to history, symptoms, and severity. These types were named *Endogenous Depression*, which accounted for 57 percent of the sample; *Psychogenic Depression*, applicable to 37 percent of patients; and *Other Depression* (the remaining 6 percent). Psychogenic Depressions were more frequent than Endogenous Depressions in the Montreal center, while in all of the other sites the proportions of the two disorders were reflective of the numbers reported above. A core group of symptoms most typical of depression was noted; it included affects of sadness, joylessness, anxiety, tension, and guilt; cognitive symptoms of ideas of worthlessness, inadequacy, and failure, loss of the ability to attend and to concentrate, and self-criticism; and motivational symptoms of a loss of energy and of interest in others. Endogenous Depressions were distinguished by a reliable constellation of additional symptoms which included early morning awakening, diurnal mood variation, retardation in cognitive functioning, psychomotor retardation, and suicidal ideation. Psychogenic Depression was correlated with a higher frequency of symptoms of aggression and irritability, and the absence of the endogenous constellation. A multivariate factor analysis yielded three orthogonal factors which discriminated between the two levels. Endogenous Depression was found to load significantly on a factor labeled Anergia/Retardation, while Psychogenic Depression loaded significantly on the

factors of Premorbid Abnormal Personality and on Dejected Mood. An item analysis of the various questionnaires used in the study found that the presence of ongoing psychological stress and of a history of psychopathology in childhood or adolescence was typical of Psychogenic Depression, while items associated with Endogenous Depression referred to psychomotor retardation, early morning awakening, and repeated past depressive episodes. The WHO group concluded that these results were supportive of the traditional division of depression into reactive and endogenous types.

Rush (1986) reviewed and discussed the various dichotomies that have been used to differentiate depressive subtypes, and found that more questions than answers still remain. He noted—to look at one traditional duality—that we do not possess sufficient evidence to conclude that psychotic depressions differ in kind from nonpsychotic depressions, and that endogenous and reactive depression cannot be separated on the basis of an identifiable precipitant. Rush also noted that the premorbid personalities of patients with reactive and psychotic depressions have not been demonstrated to differ significantly. He indicated that the endogenous vs. reactive dichotomy does have some validity in differentiating patients on a variety of neuroendocrine, REM (rapid eye movement), and sleep variables, and in studies of clinical and blood plasma responses to antidepressant medication.

Cohen and Winokur (1988) wrote that the research which had attempted to

disprove or to demonstrate the existence of distinct types of depression was flawed by a lack of operational definitions, poor instrumental reliability, and investigator biases. They concluded that empirical support for the separation of Type 1 depressions (psychotic, endogenous, autonomous) and Type 2 depressions (reactive, neurotic, psychogenic) is minimal at best. However, they reported that recent work utilizing the Research Diagnostic Criteria (Spitzer & Endicott, 1978) and the Washington University Feighner criteria (Feighner, Robins, & Guze, 1972) has yielded four reliable subtypes of depression: endogenous depression, situational depression, psychotic depression, and incapacitating depression. However, they noted that most patients display symptoms typical of two or more of these categories.

This limited review of the literature cannot offer an exhaustive presentation of specific studies. However, the authors cited will highlight for the reader the general state of affairs in past and current efforts at establishing and validating a nosology of levels or subtypes of depression. The literature offers some support for the idea that different types of depression do “exist,” but this support is limited and many findings are contradictory and simply add to the debate.

As a result of this lack of an empirically validated and consensual system with which to identify specific levels of depression, the rest of this chapter will be organized around the traditional, clinically derived categories familiar to students of depression. Those levels of depression generally labeled as neurotic, psychotic,

chronic, masked, and secondary depressions will be examined in terms of diagnostic criteria, symptoms, and phenomenology. Where relevant and available, current empirical data about each level will be mentioned as well.

## LEVELS OF DEPRESSION

### Neurotic Depression

Also known as reactive depression, mild depression, depressive neurosis, and psychogenic depression, this level of depressive psychopathology was first identified and classified as a unique disorder by Kraepelin (1921), who coined the label psychogenic depression. Kraepelin excluded a particular form of depression from the group of depressive illnesses he had studied extensively (manic-depressive disorder, involuntional melancholia, and neurasthenic depression) on the basis of a presumed difference in etiology. The dysphoria, hopelessness, and associated behavioral symptoms of psychogenic depression were judged by Kraepelin to be reactions and responses to stressful, disappointing, or thwarting life experiences and/or environmental situations. Further, he observed that the course of psychogenic depression primarily was affected and channeled by subsequent experience and interaction with the interpersonal world, and that the depressive person's mood would and could vary considerably, even during the most acute, severe parts of the depressive episode, when the patient's attention was directed away from his or her disappointment or loss toward a more neutral or positive subject. The three types of depressive illnesses from which psychogenic depression was differentiated by Kraepelin shared, in his view, a biological etiology and were refractory to environmental influence and treatment.

Beck (1967) noted that until about 1950 the syndrome of reactive,

exogenous, or psychogenic depression was not linked specifically to the concept of neurosis. Instead, reactive or exogenous depressions were judged by many clinicians to occur in otherwise healthy individuals whose premorbid personality structure was unremarkable for the presence of neurotic psychopathology. These depressions were construed therefore as isolated but exaggerated responses to loss or disappointment, while the separate syndrome of neurotic depression was called upon to describe a psychogenic depression in an individual with a known or hypothesized history of neurotic maladjustment or disturbance. Since reactive depressions and neurotic depressions were highly similar, if not identical in symptomatology and course and in appearing to have a psychological origin with an environmental precipitant, the two concepts gradually became fused into one with a variety of interchangeable names. Also influencing the fusion of these categories into one syndrome (Beck, 1967) was the then dominant psychodynamic perspective on psychopathology, in which all nonpsychotic disorders are conceptualized as arising out of neurotic anxiety and conflict.

The first edition of the *Diagnostic and Statistical Manual* (DSM) of the American Psychiatric Association (1952) labeled this category of depression the Psychoneurotic Depressive Reaction, and described it as a reaction that has as a precipitant or stimulus some current situation in the patient's life. Most frequently this situation is a loss and is correlated with feelings of guilty responsibility and with self-deprecation and self-hatred for past actions and failures. This diagnostic category was designated as equivalent to "reactive depression" and was

differentiated from psychotic depressive reactions on the criteria of life history and absence of malignant symptoms. A depressed individual whose depression could be traced to a specific precipitant or precipitants, whose premorbid character structure was neurotic, who had a history free of reports of mood swings, and whose depression was *not* accompanied by such severe symptoms as suicidal rumination, delusions, hallucinations, severe psychomotor retardation, profound retardation of thought, stupor, intractable insomnia, or hypochondrial preoccupation, would be diagnosed as suffering from psychoneurotic depressive reaction. Patients whose clinical presentations included these symptoms, and whose history indicated repeated mood swings and a cyclothymic personality structure, would be diagnosed as suffering from the more severe type of depression, Psychotic Depressive Reaction.

DSM-II (1968) relabeled the disorder *Depressive Neurosis*, and described it as a “disorder . . . manifested by an excessive reaction of depression due to an internal conflict or to an identifiable event such as the loss of a love object or cherished possession” (p. 49). This description extends the range of possible precipitants to those occurring intrapsychically, but retains the essential point of view of DSM-I. Interestingly, both editions of DSM skimmed significantly on descriptions of the signs and symptoms of this (and other) disorders, a condition which the latest revisions have been designed to rectify (see below).

DSM-III and its later revision (American Psychiatric Association, 1980,

1987) do not contain a diagnostic label which is strictly equivalent or applicable to the level of neurotic or reactive depression. The diagnosis of Adjustment Disorder with Depressed Mood may be somewhat relevant. This label refers to a maladaptive response to a known psychosocial precipitant, which leads to either social and/or occupational impairment or excessive symptoms such as depressed mood, tearfulness, and hopelessness. Another diagnostic category mentioned by some authors as roughly equivalent to neurotic/reactive depression is Dysthymic Disorder. This diagnosis is made when the depressive periods present are marked by depressed mood or anhedonia, and by at least three of 13 common depressive symptoms (see "Chronic Depression" below). The absence of psychotic features also is required to establish this diagnosis. However, a fourth criterion is the requirement that the depressive symptoms have been present for at least two years, which would differentiate this disorder from depressions which in the past would have been labeled neurotic or reactive.

Most clinical descriptions of the presentation of neurotic/reactive depression overlap considerably, if not completely. A few authorities on this subject will be cited here to provide a general picture of the phenomenology of this level of depression.

Nemiah (1975) reported that neurotic/reactive depression almost invariably is a response to some life situation or intrapsychic event, the meaning of which is to lower or damage the person's sense of self-worth or self-esteem. In



those individuals whose self-esteem is relatively consistent or robust, a major, shattering event may be necessary to produce the requisite negative changes in self-evaluation. Persons with fragile self-esteem may lapse into depression following an experience or occurrence which seemingly is trivial in “objective” terms, but which has significant narcissistic meaning to the individual. The most frequent precipitants of neurotic/reactive depressions are separations from or losses of loved ones due to rejection, physical leave-taking, or death. However, any event, whether objective or purely “fantastic” (intrapsychic), can trigger a depression if the person construes the situation in such a way that his or her self-esteem is threatened or lowered.

Other writers (Fenichel, 1945; Kolb, 1973) also stressed the role of a precipitant in this level of depression but emphasized the role of anxiety rather than self-esteem as the critical intrapsychic issue. In this view, a loss, separation, or disappointment produces in the patient an unconscious wish and/or affect which is responded to with guilt, shame, sadness, and anxiety. The depressive symptoms thus are thought to be defenses or compromise formations which serve to mask and to unconsciously express the person’s conflicting wishes toward the lost person or object.

Central to the symptoms of neurotic/reactive depression are such affective phenomena as loneliness, sadness, and despair (Nemiah, 1975); issues of self-esteem such as self-criticism, self-deprecation, and self-hatred (Freud,

1917/1950); motivational and behavioral symptoms including helplessness, impotence, and resignation (Bibring, 1953; Seligman, 1975); and cognitive sets of pessimism, hopelessness, and poor self-evaluation (Beck, 1967). These psychological symptoms are usually accompanied by physical symptoms (Beck, 1967; Nemiah, 1975). Each area just mentioned will be described in greater detail below.

Although many cognitive theorists now dispute the etiological importance of affect in depression, phenomenologically and experientially, it is these symptoms that typically dominate the neurotic/reactive depressive's daily life, and which become a primary, if not the first, target for intervention by the clinician. Neurotic/reactive depression is typified by the emotions of sadness, despair, loneliness, and melancholy. Often, these feelings are accompanied by a significant level of anger, irritation, and contempt directed toward others and toward the world in general—affects that are usually secondary responses to the dysphoria and sense of isolation (Nemiah, 1975). The neurotic/reactive depressive often describes his or her inner world as being “gray,” “dead,” or “empty,” usually a symbolization of the affective states of emotional withdrawal, and experiences a loss of interest in and of ability to maintain affective ties to others (Freud, 1917/1950). His or her sadness, pain, and despair often are tinged with a sense of the self and the world having been altered or damaged so that something of significance is missing from life, and there often is a correlated sense of guilt and/or shame for being the locus or the cause of this emptiness and damage.

These emotions can intensify greatly in a neurotic/reactive depression and may persist for long periods of time, but the presence and severity of dysphoria are dependent upon and modifiable by both environmental and intrapsychic variables. As a result, considerable shifts in the type and intensity of affect experienced in a neurotic/reactive depression often are observed. Similarly, most patients with this type of depression retain some capacity to observe and to reflect upon their emotional suffering, and find these affects to be ego dystonic (Arieti & Bemporad, 1978) or ego alien, in that the patient recognizes and is able to acknowledge that his or her emotions are not justified entirely or are inappropriate to the objective circumstances of the present situation.

Freud (1917/1950) was among the first to describe poor self-esteem as a sphere of depressive phenomenology, and his work was followed and amplified by such writers as Bibring (1953), Beck (1967), and Arieti and Bemporad (1978), to name only a select few. These writers all emphasized the neurotic/reactive depressive's extreme tendency toward self-criticism, self-loathing, and self-hatred. The patient relies upon excessively harsh, rigid, demanding, and unrealistic standards and expectations to evaluate his or her thoughts, emotions, and behavior. Each experience is inspected and poked into for signs of failure or inadequacy, and any shortcomings, errors, incomplete acts, awkwardness, or related "sins" are responded to by the patient with violently hateful and attacking judgments. Often, a particular dimension of life and experience becomes the central issue upon which the self is evaluated, and the dimensions selected will

differ across patients. Attractiveness, intelligence, productivity, morality, social popularity, and success are among the issues that may be singled out or may occur in particular combinations as evaluative criteria; the negative evaluations and attacks on the self lead to a variety of negative views of the self and to a myriad of dysphoric affects, including guilt, shame, sadness, and despair (Beck, 1967). Often the neurotic/reactive depressive is able to evaluate the behavior of other people in a much more accepting and realistic way, and sometimes finds his or her severe and relentless deprecation to be alien, bizarre, and unjustified, but feels powerless to change or ameliorate these attitudes. The ability to comfort, forgive, and appreciate the self often is flawed or extremely fragile in this disorder. Self-love typically is highly conditional and based upon excessive, perfectionistic standards.

Motivational changes are a major characteristic of this level of depression. The neurotically depressed person sees no way out of his or her pain, and despite wishing for help, often becomes apathetic, lethargic, and passive. The patient often is reluctant to do things he or she knows will be ameliorative, and withdraws from social interaction, sexual and familial relationships, hobbies, sports, and domestic, occupational, or academic responsibilities. Often, grooming and appearance suffer as well. A general and pervasive sense of joylessness and anhedonia may loom large in the person's consciousness, though these and other motivational symptoms are not absolute and sometimes will respond to efforts from others. These symptoms may be reflective of what Seligman (1975) termed "learned helplessness," which is a conviction that one's behavior has been, is, and will be

ineffective with regard to obtaining life's gratifications and satisfactions. Such an outlook understandably leads to a sense of impotence and futility and to a reluctance or unwillingness to act.

Cognitive therapists, largely following Beck (1967), identify ideational symptoms as the primary causal factors in neurotic/reactive depression. Regardless of the validity of these hypotheses, the "depressive triad" identified by Beck (1967) figures largely in the phenomenology of most, if not all, neurotic depressions. This triad includes negative evaluations of the self (discussed in more detail above), negative evaluations of the environment, and a negative outlook for the future. Essentially, the patient castigates and finds fault within himself or herself; construes the world as depriving, frustrating, hostile, cold, empty, frightening, overwhelming, or unmanageable; and can see or admit to no chance for improvement or change in the future. Again, many neurotic/reactive depressives are more realistic in their evaluations of the lives and prospects of others, and experience their depressive cognitions as ego dystonic and unrealistic, but unavoidable and dominant in their psychologies.

Patients with neurotic/reactive depressions usually are thought (Nemiah, 1975) to be free of the more severe somatic and biological symptoms which are typical of more severe depressions (see the next section, "Psychotic Depressions"). However, such symptoms as lethargy and a loss of physical energy are common, as are uncomfortable but mild to moderate changes in sleep patterns, appetite, and

weight. Patients may complain of vague somatic distress, pain, or tension, and will present with unhappy, glum, and “weighted-down” appearance (Kolb, 1973).

The empirical status of neurotic/reactive depression is unclear. Certain studies, such as the WHO (1983) study cited earlier in this chapter, lent support for the existence of this level of depression as a separate disorder, typified by a history of neurosis or character pathology, an identifiable precipitant or precipitants, and symptoms primarily of a cognitive, affective, and motivational nature, which are less severe than in other types of depression. Cohen and Winokur (1988) cited recent studies of unipolar depressive disorders which have yielded a reliable, discrete subtype of depression that they labeled “depressive spectrum” disease:

Neurotic-reactive depression could be defined as a depression in a person with an unstable personality who has a tendency to react with depression, anxiety, and hostility when confronted with difficult life circumstances. These cases are associated with a family history of alcoholism in a number of studies. In a sense this closed the circle. The “Type 2,” or neurotic-reactive depression of characterological depression, or depression spectrum disease patient, is one who has a stormy life-style, attributes his/her illness to a life event, has lifelong personality problems and is more likely to have a family history of alcoholism. The criteria for a neurotic-reactive depression are equally as good as the criteria for an “endogenous” depression. In fact, they may be better in light of the fact that they utilize lifelong characteristics rather than evanescent symptoms.

(p. 91)

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In any event, it seems clear now that diagnostic criteria such as the Feighner criteria or the RDC or the DSM-III criteria in fact do define a syndrome but they do not define diseases. There do seem to be at least two separate valid entities within the unipolar depressive group, i.e., neurotic-reactive depression (depression spectrum disease) and endogenous depression (familial pure depressive disease).

(p. 92)

However, other authors believe that the separation of this level of depression is artifactual and is based primarily on the failure to study patients with neurotic/reactive depressions longitudinally and with procedures of sufficient validity and reliability. Fulwiler and Pope (1987), in a review of the literature, concluded that a neurotic/reactive type of depression could not be differentiated from endogenous depressions. Rush (1986) reported that a series of studies indicated that almost all depressions can be linked to an identifiable precipitant, thus canceling one of the most frequently cited discriminators between neurotic/reactive and endogenous depressions. Akiskal et al. (1979) reported that follow-up of patients with neurotic/reactive depressions indicated that at least 50 percent of these patients develop deeper, more serious depression which meets the criteria for endogenous depression. Akiskal (1983) also reported that when patients with DSM-II Depressive Neurosis diagnoses are compared to controls with DSM-II Cyclothymic Personality Disorder (the diagnosis used for patients with chronic or characterological depressions), the groups are more similar than they are different, in terms of course, history, and response to

treatment.

These and other differences in findings and opinions leave us to conclude that the jury is still out with regard to the empirical status of this level of depression.

### **Psychotic Depressions**

Depressions of this level of intensity, scope, and severity have been labeled and described variously as the depressive phase of manic-depressive illness, psychotic depressions, severe depressions, endogenous depression, and involuntional melancholias. Kraepelin (1921), in his pioneering work, identified the more severe, presumably endogenously derived affective illnesses to be a single disorder with three subtypes. The overall class of pathology he identified as Manic-Depressive Illness, and he included in this class the disorder itself as well as the variants of Involuntional Melancholia and Neurasthenic Depression. Kraepelin's theory became the focus of considerable and intense debate in descriptive psychiatry as various factions argued for or against a unitary grouping of the more severe depressions.

The original edition of the DSM (American Psychiatric Association, 1952) deviated from the unitary position and offered three diagnostic possibilities for severe depressions: Psychotic Manic-Depressive Reaction, Psychotic Depressive Reaction, and Involuntional Reaction.



The second edition of the DSM (American Psychiatric Association, 1968) retained the first two of these diagnoses and described them in the following ways. Psychotic Depressive Reaction was differentiated from the other types of severe depression on the basis of a presumptive psychogenic etiology. The criteria included a depressive mood attributable to an identifiable experience. The patient's history contained no evidence of repeated depressions or of mood swings, which, if present would have pointed to the diagnosis of manic-depressive illness. Psychotic Depressive Reaction was differentiated diagnostically from Neurotic Depressive Reaction on the basis of whether the depression caused a noticeable impairment in the patient's reality testing or functional adequacy. If such impairments were noted, a psychotic level diagnosis was indicated.

Manic-depressive illness, depressed type, was defined as a disorder comprised of exclusively depressive episodes marked by severely depressed mood, mental and motor retardation, and the possible appearance of illusions, hallucinations, and delusions which were attributable to the mood disorder. This syndrome was understood to be a biological or endogenous disorder, with no clear precipitant in the environment or on an intrapsychic level.

The Involutional Psychotic reaction of DSM-I was renamed Involutional Melancholia in DSM-I I (American Psychiatric Association, 1968) and was described as a disorder which had its onset in the involutional period and which had symptoms of worry, agitation, anxiety, and severe depression. Frequent and

delusionally proportioned feelings of guilt and preoccupations around somatic issues were features. The disorder differed from manic-depressive illness in the absence of previous occurrences; it differed from psychotic depressive reaction in that there was no identifiable precipitant.

Both manic-depressive illness, depressed type, and involuntional melancholia were grouped in DSM-II under the heading of major affective disorders, a group of psychoses which are:

characterized by a single disorder of mood, either extreme depression or elation, that dominates the mental life of the patient and is responsible for whatever loss of contact he has with his environment. The onset of the mood does not seem to be related directly to a precipitating life experience and therefore is distinguishable from psychotic depressive reaction and depressive neurosis.

(American Psychiatric Association, 1968, p. 37)

DSM-III and DSM-III-R (American Psychiatric Association, 1980, 1987) offer two possible diagnostic categories for severe depressions: Bipolar Disorder, and Major Depression. Bipolar Disorder has three subtypes, two of which, the mixed type and the depressed type, are potentially applicable to severe depressions. The essential differentiating feature between Bipolar Disorder and Major Depression is a history of at least a single manic episode. A positive history yields a diagnosis of one of the subtypes of Bipolar Disorder, while a negative history eventuates in the label of Major Depression. There are five criteria that lead to either diagnosis.

The first is dysphoric mood. The second is at least seven signs such as changes in appetite leading to weight loss or gain, insomnia, or hypersomnia; psychomotor agitation or retardation; loss of libido and/or loss of interest or pleasure in usual activities; loss of energy and fatigue; feelings of worthlessness, self-reproach, and/or excessive and inappropriate guilt; diminished or impaired cognitive processes, including poor attention and concentration, slowed thinking, and indecisiveness; and recurrent suicidal ideation, wishes to be dead, suicide attempts, and thoughts of death. A third criterion is the absence of preoccupation with mood-incongruent delusions and hallucinations, and absence of bizarre behavior, when an affective syndrome is absent. The fourth criterion is that the affective symptoms are not superimposed on schizophrenia, schizophreniform disorder, or paranoid disorder; and the last criterion is that the symptoms are unrelated to any Organic Mental Disorder or to Uncomplicated Bereavement.

Major depressive episodes in either Bipolar Disorder or in Major Depression can be further classified in DSM-III as occurring with Psychotic Features when symptoms include depressive stupor (the person is mute or unresponsive), hallucinations, delusions, or gross failure of reality testing. A subdiagnosis of Melancholia can be added when there is a Toss of pleasure and anhedonia, and three of the following: depressed mood, diurnal mood variation, early morning awakening, psychomotor agitation or retardation, anorexia or weight loss, or excessive or inappropriate guilt.

DSM-III does not contain a separate diagnostic category for Involutional Melancholia. Presumably, persons who would have received this diagnosis in the past will meet the current criteria for Bipolar Disorder or for Major Depression.

Despite these differentiations and etiologic considerations, a consolidation of labels has occurred with these syndromes similar to that discussed in the area of neurotic or reactive depression: many writers use the terms *endogenous depression*, *severe depression*, or *psychotic depression* interchangeably. With few exceptions, which will be noted below, the depressive episodes in the DSM-III diagnoses of Bipolar Disorder and Major Depression are thought to be equivalent as well, especially since the criteria for depression are shared by these two disorders.

The phenomenologies and symptoms of the three types of severe or psychotic depressions are essentially similar. Beck (1967), Gibson (1975), Arieti and Bemporad (1978), and Cohen and Winokur (1988) pointed out that these syndromes, if they are valid entities, require nonsymptomatic criteria to establish differential diagnoses. As a result, the following description of psychotic level depression will not be specific to any of the hypothesized subtypes.

Arieti and Bemporad (1978) suggested that severe or psychotic level depressions are typified by a classic and historically repetitive triad of psychological symptoms: a generalized and intense melancholic affect; disordered

and disturbed cognitive and perceptual processes, in which thinking is slowed, blunted, and blocked, and is marked by idiosyncratic and distorted content; and psychomotor retardation. These authors also noted that psychotic level depressions are typified by the appearance of a variety of severe somatic disturbances and dysfunctions, and that severe or psychotic depressions usually are ego syntonic:

The patient who is depressed to a psychotic degree has undergone predominantly a severe emotional transformation, but he believes that his way of feeling is appropriate to the circumstances in which he lives. Thus, he does not fight his disorder, as the psychoneurotic does, but lives within it. In many cases he even seems to nourish it. In this respect he resembles persons who are affected by character neuroses and do not even know the pathological nature of their difficulties. . . .

The severely depressed person may neglect feeding himself to the point of starvation; he may be so inactive as to be unable to take care of even the most elementary needs, he may think he is justified in believing that there is nothing good in life and death is preferable. He also considers any attempt to improve his life to be worthless, and in some cases he feels guilty in the absence of reasons which would make other people feel guilty. . . . He considers his mood consonant with what appears to him the reality of the situation. Thus he seems to have characteristics which would make appropriate the designation "psychotic." Only in a minority of cases do delusions, especially of guilt, and hallucinations occur.

(Arieti & Bemporad, 1977, pp. 59-60)

The melancholic mood in psychotic level depressions sometimes appears to be a gradual exacerbation of a chronic characterological pattern of sadness,

withdrawal, and dejection; for other patients, this dysphoria is quite different and distinct from the norm. The onset of the symptomatic mood usually is typified by unexplained and prolonged bouts of tearfulness, despairing dejection, wistfulness and nostalgia for lost happiness and for better times in the past, and extended and severe feelings of grief, loss, and emptiness. In some of these cases (presumably those of the psychotic depressive reaction type), there may exist an initial event which provided the stimulus for these feelings; in others (most likely depression as part of a manic-depressive or involuntional melancholic syndrome), a precipitant is absent or cannot be identified. Regardless of presence or absence of a precipitant, the dysphoric emotions remain at a severe and intense pitch for an extended period of time, and the person's mood is uninfluenceable by environmental or internal efforts. In fact, as time passes, the melancholia seems to become even more severe and disabling, and the patient experiences interference in his or her thought processes; loses the ability to concentrate, focus attention, or direct his or her thoughts; loses interest in and feels unable to work, sleep, or engage in sex, recreation, or familial/social activities. Often, the person is listless and agitated and becomes extremely anxious and confused when faced with minor decisions and seemingly insignificant frustrations.

The inner experience of this disorder frequently is described by patients with words such as "dead," "wasted," "diseased," and "consumed," to list just a few. The patient reports a severe anhedonia and a sense that previously valued persons, things, and objects have lost meaning. The self is experienced as empty,

unresponsive, frozen, or paralyzed, and as being hateful, disgusting, evil, unlovable, and deserving of punishment, destruction, and death. These ideas are often accompanied by severe, intense feelings of guilt, shame, humiliation, and embarrassment. The patient usually cannot cite particulars with regard to justifying this view of the self, but the ideas and emotions themselves, as mentioned above, are syntonetic and acceptable to the patient. Predictably, suicidal ideation is a frequent correlate of this melancholic mood, and suicide attempts occur with regularity. Arieti and Bemporad (1978) report that 75 percent of patients with severe depressions experience repetitive suicidal ideation, and at least 15 percent make serious attempts to end their own lives.

The melancholic mood in psychotic depressions is accompanied by profound and remarkable cognitive dysfunctions and symptoms. The content of the patient's cognitive activities is dominated by ideas and images of defeat, death, destruction, and an enduring outlook of futility, hopelessness, and pessimism. These ideas may be preceded or accompanied by obsessive or phobic preoccupations, or by aggressive and/or sexual ruminations. As the depression deepens, ideas of doom, gloom, and failure acquire greater permanence and importance. The patient is unable to think of anything but his or her failures, weaknesses, and flaws, and believes that great and terrible misfortunes will befall the patient, his or her family and friends, and even the world—all because of something the patient has/has not done in the past or will or will not be able to do in the future. In sum, the patient's thinking is dominated by the themes of failure,

guilt, self-blame and condemnation, hopelessness, and sin, and these contents, although unexplainable on the basis of facts, are experienced as congruent or true to the self by the patient. The patient cannot admit into consciousness any contradictory, pleasant ideas or turn his or her thoughts to neutral, ego-distant issues. Thinking in severe depression appears to lose its role and function as an adaptive, information-processing function. Instead, it becomes an internally oriented process, the role of which is to register and transmit mental pain (Arieti & Bemporad, 1978).

Along with these cognitive contents, there are noticeable dysfunctions in the processes of thinking and perception. Attention and concentration are disrupted and impaired, as is the person's ability to direct, guide, and focus his or her thinking. Reading, writing, thinking, and speaking often become cumbersome, slowed, or blocked entirely, and are experienced as requiring extreme effort. A certain subgroup of psychotic depressions is accompanied by gross delusions, hallucinations, and failures in reality testing. Brown (1988) noted that different studies report a rate of delusions in 5 to 30 percent of psychotically depressed patients. These delusions usually focus on contents of guilt and sin, ideas of reference and of persecution, somatic disease, poverty, or nihilism. Brown (1988) reported the example of a patient whose anorexic symptoms were caused by the delusion that her bowels, bladder, and body had rotted to the point that food and water would fall out of her. Another patient blamed himself for the starvation of his family because he had failed them financially, though they were in fact well fed



and economically secure. Hallucinations in this disorder are rare but include the types of contents just mentioned. Arieti (1974) noted that, in psychotic depressions, hallucinations usually are milder and less distinct than in schizophrenia, and are secondary and responsive to the patient's prevailing mood.

Severe/psychotic depressions are marked by changes in behavior; the most frequently observed is psychomotor retardation or retarded hypoactivity (Arieti, 1974; Arieti & Bemporad, 1978; Beck, 1967; Gibson, 1975; Nemiah, 1975). The patient simply stops behaving as frequently and as quickly as he or she did in the premorbid state. Talking and acting, even for the most basic tasks of life (eating, grooming, bathing) are slowed, postponed, or abandoned. The person is lethargic, indifferent, and disinterested in interpersonal situations; the frequency of spontaneous speech and interaction diminishes, often disappearing completely. In the most severe instances, *depressive stupor* may appear. This is thought to be (Arieti & Bemporad, 1978) the most severe form of depressive psychomotor and behavioral disturbance, and takes the form of total withdrawal from the environment, to the point that the patient becomes completely mute, unmoving, and unresponsive. Patients in this state cannot care for even their most basic needs, usually remain in bed, and require spoon feeding, or tubal or intravenous nutrition. They are prone to malnutrition and other severe physical complications.

The other correlated physical/behavioral symptoms of this disorder are changes in sleep patterns and appetite, weight loss or gain, and diurnal mood

variation. Most psychotic depressions are accompanied by insomnia, early morning awakening, and loss of appetite and weight.

Certain authors believe that the differential diagnosis of the various types of psychotic level depressions can be made by comparing symptoms on the behavioral and physical dimensions. In contrast to the similarities in affective and cognitive symptoms, these subgroups seem to differ on these variables. For example, Gibson (1975) reported that the phenomenology of depression in cases of involuntal melancholia is relatively equivalent to the depressions described above, with the exception that patients with involuntal disorders demonstrate high levels of psychomotor agitation and experience anxiety and psychological agitation as additional parts of their melancholic process.

Stokes (1988) suggested that although patients with bipolar illness who are depressed resemble patients with unipolar depression to a considerable degree, the depressed bipolar patients tend to have weight gains, increased food intake, and symptoms of hypersomnia, while unipolar patients become anorexic, lose weight, and typically complain of insomnia. Brown (1988) reported that psychotically depressed patients who report delusions among their symptoms differ from non-delusional psychotic depressives on several behavioral indices. He stated that a number of studies have indicated that delusional depressives experience greater degrees of guilt, and are more cognitively ruminative, display more behavioral agitation, and are more severely retarded psychomotorically.

Delusional depressives also demonstrate higher levels of depression on such measures as the Hamilton Depression Scale. At this point in time, such findings are useful in directing further research but do not as yet add up to a sufficiently substantial knowledge base for a conclusive portrait of unique subtypes of psychotic level depression.

Research in this area is not as conflicted and as contradictory as the research mentioned in the discussion of neurotic/reactive depression. Few would doubt the validity and reliability of a diagnosis of a depression of severe proportions, and the debated issues that are attached to this level of depression are concerned with more finely tuned diagnostic and classificatory questions. These efforts are exemplified by attempts to establish clear, reliable, and valid demarcations between unipolar and bipolar disorders (Stokes, 1988) and psychotic vs. nonpsychotic manifestations of major depressive disorder or of endogenous depression (Brown, 1988; Cohen & Winokur, 1988). At this point in time research has not yet yielded unanimity with regard to the definitive criteria for such distinctions.

### **Chronic Depression**

There is a subgroup of patients whose depressions do not follow the generally accepted rule that depression is a time-limited, self-correcting process. These patients may remain depressed for months, years, or decades without

significant change in mood, cognition, or affect. It is not uncommon to interview such a person and to have that patient state that he or she cannot remember a time when he or she was not depressed. These patients often trace the onset of their condition back to childhood or to adolescence. Interestingly, many persons with this type of depression report that they first realized that they were “depressed” only lately. Until the condition was diagnosed professionally or became recognizable to the patient in some other way, many such persons assumed that their dysphoria was “normal,” that is, that their depressed mood and thinking were universal.

This level of depression is an extremely controversial disorder, according to many authorities. The existence of a chronic form of depression has been discussed since the early part of this century, but the nature and features of the disorder have been the subject of disagreement until the present day. As Akiskal (1983) noted, chronic depression has had a very large number of names, including “depressive temperament,” “depressive personality,” “hysteroid dysphoria,” “characterological depression,” “neurotic depression,” and “chronic mild depression.” Chronic depression has been construed as a mild precursor to a full-blown manic-depressive disorder, as a type of personality disorder, or as a separate and unique affective disorder.

Kraepelin (1921) introduced the term depressive temperament to describe the condition of chronic depression which he understood to be a milder,

somewhat stable predecessor to manic-depressive illness. He described depressive temperament as sharing most, if not all, of the symptoms of the manic-depressive syndrome in form and type, with the symptoms occurring in attenuated quantities or levels.

An alternative conceptualization of chronic depression as a type of personality disorder or character problem was put forth by such psychoanalytic writers as Fenichel (1945) and Bemporad (1976). Schneider (1959) also deviated from Kraepelin's viewpoint, identifying chronic depression as a type of psychopathy (his term for personality disorder). He separated chronic depression from the other, biologically derived affective disorders. DSM-II (American Psychiatric Association, 1968) placed chronic depressions with Cyclothymic Personality Disorder, Asthenic Personality Disorder, or Neurasthenic Neurosis.

In the past two decades many investigators have classified chronic depression as an affective disorder without identifying it as a type of neurosis or of character disorder, or as a forerunner of any other form of affective disorder (Spitzer, Endicott, & Robins, 1978). This is the approach taken by the authors of DSM-III and DSM-III-R (American Psychiatric Association, 1980, 1987). These manuals place chronic depressions under the diagnostic label of Dysthymic Disorder. There are four defining characteristics of this syndrome. The first is a depressed mood for the majority of the day, on most days, for at least two years. The second criterion is the presence while depressed of two of six possible

depressive symptoms: poor appetite or overeating, insomnia or hypersomnia, low energy or fatigue, low self-esteem, poor concentration or difficulty in making decisions, or feelings of hopelessness. A third criterion for this diagnosis is that during the immediately preceding two-year period the patient does not report having been free of a depressed mood for longer than two months. The last criterion is the absence of clear evidence of a major affective disorder. These requirements have been judged by some (Elliot, 1989; Kocsis & Frances, 1988) to be overly similar to the diagnostic criteria for Major Depression, resulting in a blurring of boundaries between the two disorders. As Kocsis and Frances (1988) suggested, an empirical and theoretical consensus upon the nature and appearance of chronic depression is still elusive. There is at least one existing study (Hirschfeld, Klerman, Andreason, Clayton, & Keller, 1986) which concluded that there are two and possibly more patterns of life experience related to the development of chronic depression. In some patients, chronic depression was found to be the result of a failure to recover from an acute major depressive episode, while in others it was unrelated to an acute episode. In this latter group, chronic depression was in itself the major type of psychopathology present. These data were interpreted by Elliot (1989) as suggestive that chronic depression may not be a single disorder.

The patient who suffers from a chronic depression lives under the weight of his or her symptoms for an extended period of time. These depressive symptoms generally are rated as mild to moderate by patients and by clinicians and

researchers (Arieti & Bemporad, 1978; Bemporad, 1976; Kocsis & Frances, 1988) and tend to cluster around the cognitive and affective dimensions of depression. The more severe behavioral and somatic features found in psychotic depressions are absent or attenuated (Kocsis & Frances, 1987). Chronic depression seems then to be identifiable primarily by enduring negative and dysfunctional cognitive, perceptual, and attitudinal patterns or traits, and by correlated or resulting dysphoric affects and moods. Persons with chronic depression consistently view themselves, the future, and the world through the lenses of Beck's (1967) depressive triad: negative evaluations of the self, pessimism about the future, and a corresponding sense of the world as barren, depriving, depleting, or rejecting. In neurotic and psychotic depressions these cognitions are present but they are hypothesized to be latent or absent when the person is not in the midst of an acute episode. In other words, the depressogenic cognitions are believed to be atypical of the person's premorbid approach to life. The chronic depressive appears to construe experience through these sets in a pervasive and unremitting way, unchanged from any other previous state or time. The patient does not seem to have insight into the effect these ways of thinking have on his or her life, and demonstrates little effort at changing or correcting these attitudes. Other depressogenic beliefs are omnipresent and reside in the person's mind in an ego syntonic manner, including those ideas most expressive of the motivational issue of learned helplessness. As a result, the patient's affective life is dominated by gloom, hopelessness, disappointment, guilt, self-blame, poor self-esteem, and,

often, an unwillingness to harbor ambitions, take risks, or commit to goals because failure and disappointment always are anticipated. A full-blown depressive syndrome is not present and the person sometimes does not consider himself or herself to be depressed. These ideas, affects, and traits frequently are accompanied by feelings of anger, resentment, bitterness, jealousy, envy, and alienation, usually all correlated with the person's ideas about the unfairness of his or her inevitable deprivation and suffering. Chronic depressives often complain of fatigue and lack of energy. They feel overburdened and overwhelmed by familial, social, occupational, and recreational activities, which they usually partake of with diminished or absent pleasure, and at the subjective cost of considerable psychic energy.

Bemporad (1976) described chronic depression as distinguished by a stable background of dysphoric feeling on a day-to-day basis, usually activated by very minor frustrations or setbacks. The depressive symptoms are a part of the person's ongoing personality structure, so that there is a flattening of affect and a loss of optimism. Joy, excitement, and pleasure are systematically but unconsciously minimized or perverted by the depressive character structure. Akiskal (1983) identified a group of chronically depressed patients whom he labeled as having "early onset dysthymic disorders." These patients suffer from relatively permanent impairments of self-esteem and from depressogenic cognitive structures. Their ongoing functioning is marked by mild to moderate, and essentially stable, depressive affect. Elliot (1989), in an unpublished master's



thesis conducted under this writer's supervision, found that patients with DSM-III Dysthymic Disorder have lower self-esteem than do patients with Major Depression, but that Major Depressives have more severe cognitive dysfunctions (retardation, poor attention and concentration), higher levels of depressive affect, and more severe vegetative symptoms. Elliot (1989) placed the depressive cognitive triad at the "center of chronic depression." but concluded that chronic depression

... is a disorder that has measurable cognitive, personality, interpersonal, and familial characteristics. . . . These characteristics put serious constraints on the capacity of those who suffer from them. Dysthymics are severely limited in their capacity to cope. They appear to possess cognitive attributional styles which preclude them from engaging in adaptive, self-corrective behavior, and from reinforcing themselves for having completed any endeavor. Their dilemmas are not subjectively perceived to be of their own creation, nor do they believe that their situations will ever change.

(p.27)

Chronic depression has been discussed in the context of a newly identified level or type of depression, which is currently known as Double Depression (Keller & Shapiro, 1982). This level consists of a severe and acute depressive episode which is diagnosable as DSM-III Major Depression, and occurs in an individual who previously was diagnosed as suffering from Dysthymic Disorder. At the present time, the empirical status of this syndrome is uncertain. Miller, Norman, and Dow (1986) reported few distinguishing psychological

characteristics in groups of patients with double depressions or with major depressions. They found that double depressives suffered from the current episode of major depression for a longer time, and had more severe depressive symptoms, than did the control group. Klein, Taylor, Harding, and Dickstein (1988) found that double depressives had higher levels of self-criticism, negative cognitions, depressive personality traits, and stress reactivity, together with lower levels of social extraversion, than did a group of patients with major depression. There are no specific diagnostic guidelines or agreed-upon clinical portraits available at this time.

### **Masked Depression**

The idea of the existence of a syndrome of masked depression is a debatable and philosophically difficult construct for many students of depression. This label, applied synonymously with the diagnosis of *depressive equivalent*, refers to patients who have behavioral, psychological, or somatic symptoms which “mask” and symbolically express an underlying depression. Kennedy and Weisel (1946) reported on a phenomenon of “manic-depressive equivalents” in patients whose overt symptoms consisted of somatic dysfunctions, which hid a psychotic-level affective disorder. Berner, Katschnig, and Poldinger (1973) and Geisler (1973) reported that many psychophysiological and physical symptoms in actuality reflected underlying depressive pathology. The list of symptoms thought to be depressive equivalents included symptoms that resembled angina, nervous

dystonia, cardiovascular disorders, cholecystitis, colitis, diverticulitis, food allergy, neoplasm, and pernicious anemia. Braceland (1966/1978) noted that the most common symptoms of masked depression were general abdominal pain, fatigue, headache, anorexia, insomnia, and gastrointestinal distress. Stafford (1977) mentioned that a variety of psychodynamic, cognitive, behavioral, and biological studies provide some limited support for the existence of this syndrome. The evidence that Stafford reviewed is particularly applicable to addictive and alcoholic patients, whose substance abuse is thought to reflect an attempt to cover up and to ameliorate the dysphoria of depression. Clinically, this idea has gained a certain acceptance because of the observation that when addicts and alcoholics are prevented from obtaining chemical gratification, depression often results. Similarly, the recovery phase in these disorders often is marked by the emergence of depression. However, these observations are open to the opposite interpretation as well. The similarities in dynamics between substance abusers and depressives, and the presence of depression in the recovery phase, may be *caused* by the chemical dependency. If this latter explanation is more accurate, the depressions experienced by addicts and substance abusers might better be understood as *secondary depressions*, rather than as a “flowering” of a previously hidden depression.

Fulwiler and Pope (1987), in a review of the relationship of depression and personality disorder, suggested that many of the DSM-III Axis II personality disorders might better be looked at as “partial affective syndromes” or as

reflecting what Akiskal (1983) labeled a “subsyndromal affective illness.” Fulwiler and Pope (1987) pointed out that many of the diagnostic criteria for the various Axis II disorders overlap or are identical to the criteria for Dysthymic Disorder or for Major Depression. They believed that the abnormal behavior typical of personality disorders can and does obscure an additional and perhaps more basic affective disorder, particularly when that affective pathology is subsyndromal in extent and severity.

The validity of the construct of subsyndromal depression received some support from a group of studies wherein it was found that depressives, personality disorders, and alcoholics share similar family histories of drug abuse, alcoholism, and affective illness, and that these three groups often respond equivalently to antidepressant medication (Fulwiler & Pope, 1987). However, this concept and the older concept of masked depression are far from universally accepted. Critics suggest that the disorder is an artifact of methodological problems and of the confusion of correlative findings with causation.

### **Secondary Depression**

Depression as an affect, mood, or syndrome can accompany or follow almost any medical or psychiatric illness. It also may be the consequence of temporary biochemical alterations, such as the effects of medications, alcohol use, fatigue, prolonged sleep deprivation, or malnutrition. The symptoms and phenomenology

of secondary depressions are similar, if not identical, to one of the levels of primary depression which have been described above (Cohen & Winokur, 1988). Cognitive, affective, motivational, and vegetative changes may dominate the clinical picture singly or in combination, and the secondary depression may resemble a neurotic/reactive, psychotic, or chronic depression in severity and course. The presence of a primary medical or psychiatric illness, or of a biochemical derangement, or of the history of at least one of these precursors, therefore is necessary to establish the diagnosis of secondary depression (Cameron, 1987).

Certain secondary depressions may be related clinically and conceptually to the group of neurotic/reactive depressions, and might best be named Psychogenic Secondary Depression. These secondary depressions seem to result from the patient's reaction to, or appraisal of, his or her medical or psychiatric illness or the consequences of that illness. For example, a patient who has lost physical capabilities due to a stroke, or who has been disfigured by an accident, may respond to those changes with the cognitions typical of neurotic depression, thus eliciting depressive mood and behavior. In essence, the illness, psychopathology, or physical failing becomes the precipitant noted in the previous discussion of neurotic depression. Other secondary depressions may be more purely endogenous in that they are caused by biochemical processes associated with an initial disease process. Probably many secondary depressions are mixed, with regard to reactive and endogenous causation. A neurological or hormonal

disorder that biologically produces secondary depressive symptoms will yield in the patient a psychological response to both the original disease and the resulting disorder. This response may exacerbate the depression if it falls within the parameters of depressive appraisal and cognition, effectively adding a reactive element to a secondary depression which was begun by endogenous processes.

## SUMMARY

This chapter is concerned with the phenomenology and overt behavioral and psychological manifestations of the different types or “levels” of depressive psychopathology. Traditional typologies derived from clinical lore and observation are described and reviewed, as are the latest attempts to identify and delineate discrete classes of depression in DSM-III and DSM-III-R (American Psychiatric Association, 1980, 1987). The history of the unitary vs. dual disorder controversy is reviewed, as are current studies which attempt to test the utility of nosological work and to validate or disprove the commonly accepted differentiation of depression into such levels as neurotic-reactive, endogenous-psychotic, and characterological. Other traditional levels of depression, including masked depression, and secondary depression are discussed as well.

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