Problems in Family Practice

The Depressed Patient

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Sadness and normal grief are distinguished from pathological grief and depression by intensity, duration, precipitating events, and the quality of psychopathological features. Depression is evaluated as a final common pathway of potential psychodynamic, genetic, psychosocial, physiological, and personality characteristics or events.

The clinical entity of depression is diagnosed by describing some of each of the affective, behavioral, and cognitive changes concomitant with depression. The clinical entity of depression is further differentiated for purposes of treatment into the categories of bipolar depression (manic-depressive illness), unipolar depression (psychotic depressive reaction or involutional melancholia), neurotic depression, and secondary depression (secondary to somatic disease, drugs, or to other psychiatric disorders).

The immediate treatment depends on the type of depression diagnosed. Unipolar and bipolar depressions respond to specific pharmacologic therapy and supportive care. Neurotic and characterologic depressions respond to supportive or insight psychotherapy with possible brief adjunctive anti-anxiety or hypnotic medication. All of the treatment modalities, with the possible exception of insight psychotherapy, can be effected very adequately by the primary care physician who is given clear diagnostic and assessment guidelines with specific treatment approaches.

Depression has been estimated to be the most common clinical condition that causes people to seek medical attention.[1] The majority of clinical depressions are treated by the family physician. One primary and continuing problem in treating depression is adequate and precise diagnosis. It may be difficult to recognize masked depression, in which symptoms of hypochondriasis, psychosomatic problems, anxiety, addictions, and/or acting-out behavior mask an underlying depression. Once the clinical entity of depression is recognized, the next problem is how to classify and treat it. An overview of the differentiation of normal sadness and grief from pathological grief and depression will be presented, as well as criteria by which to recognize each of the types of depression and what to do about each.

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Table 1. A Comparison of Normal and Pathological Grief

	Normal Grief	Pathological Grief
Intensity	Sensations of somatic distress.	Acquisition of the symptoms of the last illness of the deceased.
	Change in conduct patterns: restlessness, inability to organize activities or carry out social pleasantries.	Behavior destructive of patient's interest: foolish business deals or generosity, agitated depression.
Duration	Up to 4 or 8 weeks.	Longer than 6 months
Precipitating Events	Loss; preoccupation with image of deceased.	Loss which is *denied*; overactivity without a sense of loss.
Quality of Psychopathologic Features	Hostility toward others (or physician).	Marked hostility *without* grief.
	Guilt feelings; bereaved accuses himself of negligence.	Apathy.

Definitions

Sadness is a response to external events, particularly the reaction to unhappy events close to our personal lives. Sadness is appropriate when reactive to specific events, especially those involving some type of loss, and is something everyone experiences from time to time.

Normal grief is the acute affect or feelings experienced concomitant with a significant loss. Grief is not to be confused with mourning, the psychological work of gradually untying the emotional bonds to the lost person and making a memory of the relationship and all its meaning. Grief is usually precipitated by a specific loss and accompanied by a preoccupation with an image of the deceased. Possible manifestations of normal grief include sensations of somatic distress, guilt feelings with the bereaved accusing himself of negligence, perhaps hostility feelings toward physicians and others, and even a change in conduct patterns including restlessness and inability to organize activity or to carry out social pleasantries.[2] The average period of acute grief lasts four to eight weeks.

With sadness and grief, there are no distortions of events or persons, and thought centers around the actual loss which gradually is accepted. A reaction of grief has been associated with physical illness[3] and mutilating surgery[4] as well as with the loss of a significant person.

Pathological grief reactions indicate that for some psychological reasons the person is unwilling or unable to "give up" the lost or deceased person, refuses to commit him/her to memory, and engages in a pathological process around the loss. The precipitating event is usually a loss which is denied and around which there is little or no grief, with marked hostility or apathy in its stead. Con-

comitant with pathological grief may be the acquisition of the symptoms of the last illness of the deceased. The patient may engage in behavior destructive of his interest, such as foolish business deals, unwarranted generosity, or agitation.[5] Normal grief may extend to six months; pathological grief, eventuating in depression, usually lasts much beyond six months.

Sadness and normal grief are distinguished from pathological grief and depression by intensity, duration, precipitating events, and quality of psychopathologic features (Table 1).[6]

Etiology of Depressive Disorders

The physician must evaluate the physiological, psychological, and social components of any disease entity whether the disease is primarily organic or psychological. The following factors are considered important both etiologically and in considering the multiplicity of factors which form the clinical entity of depression.[7]

Psychodynamic: Early life experiences predispose a person to being sensitive to loss; for example, the early loss of a parent before development is complete may sensitize to further loss and may develop a proneness to depression on separation or other loss.

Genetic: Genetically determined hereditary predispositions have been specifically demonstrated in both bipolar and unipolar depression.

Psychosocial: Events in an adult life which overwhelm coping mechanisms either by the magnitude of the stressing factors or the diminished coping ability at a particular time in the individual have been shown to cause or worsen a depressive illness.

Physiological: Stressors of a physiological nature, such as medical illness, childbirth, or drugs, may increase susceptibility or cause a secondary depression.

Personality: Certain character traits, specifically those found in hysterical and obsessive-compulsive personalities, determine or modify the reaction of a person to stress.

The most significant factor accounting for depression in an adult is the previous position or vulnerability of the person based on a composite of the above five factors, with depression being a heterogeneous group of syndromes with multiple causations. Depressive illness is thus seen as a psychobiological final common pathway.[7]

Table 2. The Diagnosis of The Clinical Entity of Depression

1. Affect
- Sad with or without crying
- Discouraged
- Irritable

2. Behavior
- Anorexia and/or weight-loss
- Sleep disturbances (specifically, terminal insomnia)
- Diurnal mood swing
- Loss of energy and fatigue
- Decreased libido
- Multiple somatic complaints
- Alcohol and drug abuse
- Loss of interest in usual activities
- Loss of gratification from usual roles

3. Cognition
- Hopelessness and helplessness
- Poor concentration
- Decreased self-esteem
- Guilt and self-reproach
- Suicidal thoughts
- Paranoid ideation
- Loss of interest in usual activities
- Viewing of one's accomplishments as meaningless
- Delusions, specifically of a hypochondriacal, guilty, or impoverished nature.

Diagnosis

The Clinical Depressed State

By describing some of each of the following changes of affect, behavior, and cognition, a treat-

Table 3. Clinical Depressed State

Primary Affective Disorder		Neurotic Depression	Secondary Affective Disorder	
Unipolar	Bipolar		Other Psychiatric Disorder	Systemic Diseases
An episode of significant depression without evidence of any manic episode	Evidence of current or past manic episode	Reactive Depression	(a) Alcoholism (b) Schizophrenia	(a) Central nervous system disorders (b) Drugs (reserpine, birth control pills, steroids) (c) Endocrine disorders (d) Viral diseases (e) Postpartum depressions (f) Anemia (g) Organic brain disease

able clinical entity of depression is delineated (Table 2).

The affect is usually that of depression or irritability. The person may feel discouraged and irritable or sad, with or without crying.

The psychomotor or behavioral changes concomitant with depression include anorexia, possible weight loss, sleep disturbance, specifically terminal insomnia (ie, waking up at 3 or 4 AM and being unable to return to sleep), a diurnal mood swing, loss of energy, and decreased libido. The patient often presents to the primary care physician with multiple somatic complaints, frequently masking the depression initially. Drug and alcohol abuse should be specifically scrutinized.

The cognitive changes seen are thoughts of hopelessness and helplessness. It is to be remembered that the attitude of hopelessness (that the individual can no longer cope with his problems and there is no hope for change) is the single factor most correlated with successful suicide.[8] The patient may, in addition, show poor concentration, decreased self-esteem, guilt, and self-reproach as well as loss of interest in usual activities. Suicidal thoughts and paranoid ideation may be present here as well as in the more serious psychotic depression where there may be delusions of a hypochondriacal, guilty, or impoverished nature.

After the above present illness is elaborated, the past history is very important. The key concept in the history is a change from the usual. A history of present illness minimally includes when such changes began, the triad of ABC (affect, behavior, cognition), preoccupations, suicidal evaluation, and physiological concomitants. A past history is elicited including depression, mania, response to psychotropic drugs, and suicide attempts. A family history of each of these is also important, including alcohol abuse and antisocial trends, both of which cluster in the families of depressed patients.[9]

Once the clinical entity of depression is diagnosed, the next step is to differentiate the types of depression, as the specific type of depression is associated with a specific treatment approach (Table 3).

Differential Diagnosis of Depression (Table 4)

Bipolar Depression

The diagnosis of bipolar depression (manic-depressive illness) is made by showing evidence of a current or past manic episode. The clinical triad of a manic episode is elated but unstable mood, pressure of speech, and increased motor activity. Often the patient who is elated and hyperactive will go on spending sprees, and frequently have paranoid or grandiose ideation along with the mania. The triad of the depressive episode is a depressed mood, difficulty in thinking, and psychomotor retardation.

There are remissions and recurrences of cycling manic and depressive episodes. The first manic attack usually lasts three months, and the first depressive attack usually lasts about six months.[10] Often the patient can give a good account of the roller-coaster pattern of mania and depression in his/her life. Up to age 45 years, episodes are of about the same duration, and after age 45 years the individual episodes grow longer.

The incidence of manic-depressive illness is greater in the middle and upper class, and has its usual onset from ages 20 to 35 years. In first-degree relatives (father, mother, brother, sister), there is a 20 to 25 percent inheritance rate. With identical twins the inheritance rate approximates 100 percent, demonstrating a dominant inheritance pattern with incomplete penetrance, a fact to be considered in counseling families who have a manic-depressive member.

Unipolar Depression

The first clue to unipolar (sometimes referred to as endogenous) depression is that the ensuing depression is totally disproportionate to the magnitude of the precipitating cause, or a specifically delineated cause may not be found. There are two types of unipolar depression: psychotic depressive reaction and involutional melancholia.

A. *Psychotic Depressive Reaction.* A severely depressed mood is the primary sign and symptom of psychotic depressive reaction. This depression is referred to as psychotic because the depressive reaction is out of proportion with reality. The term does not mean that the patient is psychotic per se. The psychotic depressive reaction usually immediately follows a clear-cut precipitating event. The classical findings are delusions of guilt, poverty, or somatic preoccupation. The patient may present with the delusional idea that his stomach is rotting away or an incurable cancer is in his head.

B. *Involutional Melancholia.* A premorbid personality of obsessive-compulsive, schizoid, or hysterical features is associated with a greater incidence of involutional melancholia.[10] There is usually no history of previous psychiatric disease in this depression, which occurs during the "involutional" period. The primary symptom is anxiety, with concomitant symptoms of apprehension, guilt, agitation, insomnia, tension, and sometimes headache. Anxiety will be processed through the basic character structure, hence, an obsessive patient would appear more obsessive, and a hysterical patient even more dramatic or hypochondriacal.

Neurotic Depression

A neurotic depression indicates excessive reaction of depression to an internal conflict or to an identifiable event. There is no impairment of reality testing and no marked interference with functional adequacy. Patients with neurotic depression show self-pity, as opposed to the self-blame in psychotic depression.

Secondary Depressions

A. *To Somatic Disease.* The first symptom of depression may be a variety of somatic complaints; also, depression may be secondary to other disease entities by being either psychologically reactive or biologically reactive. Somatic diseases having a significant preponderance of associated secondary depressions include central nervous system disorders (eg, organic brain disease), endocrine disorders (eg, hypo and hyperthyroidism, pancreatic tumor), viral diseases (eg, influenza), and postpartum depression.

B. *To Drugs.* Intoxication or drug withdrawal states frequently manifest with depression. Depression is induced biologically by some drugs; of special note are reserpine (20 percent of hypertensives who use reserpine become clinically depressed),[11] birth control pills, and steroids.

C. *To Psychiatric Disorders.* Alcoholism, schizophrenia, and a number of other primarily psychological disorders may have a secondary or reactive depression associated with them. The treatment of these depressions is the treatment of

Table 4. Differential Features of Depressions[7]

	Clinical Features	Family History	Course	Pharmacology
Primary Depressions				
Bipolar	Classic clinical triads of either mania or depression	+mania +depression +suicide +alcoholism +2 generation affective illness	3 episodes per lifetime; episode lasts 3 to 6 months.	Lithium carbonate responsive (may switch to hypomania with tricyclics)
Unipolar	Agitated (or retarded) in psychomotor activity	−mania +depression	Usually 1 to 2 episodes per lifetime; episodes last 6 to 9 months	Tricyclic responsive (less likely to respond to lithium)
Secondary Depressions				
	Depression is *secondary* to other psychiatric or physical symptomatology (drugs, organic brain disease, central nervous system disease, hysteria, schizophrenia, viral diseases)	Depends on primary diagnosis	Usually no clear-cut episodes, transient or chronic	Treatment of primary disorder (tricyclics are not effective)
Neurotic Depressions				
	Depressive reaction to an identifiable event or internal conflict	May be early deprivation, suffering, or loss, with a history of depressive reactions to similar events	Remits within 6 months	Tricylics not effective. Adjunctive anti-anxiety or hypnotic (for insomnia) medication for brief period

the primary disorder itself rather than of the concomitant depression.

Treatment

Immediate Treatment Concerns

At an early point in the evaluation of the clinical depressed state, the immediate treatment concerns are of special importance. It is to be remembered that the mortality rate (completed suicides) of all depressions is five percent.[12]

Of a group of recently studied patients who completed suicide, three quarters of the patients had substantial evidence of depressive illness,[13] 91 percent who died by overdose had been under the recent care of a physician, and over half of these patients had been given a lethal prescription by a physician within a week of their committing suicide.[14] This speaks to the need to specifically ask about suicidal thoughts and plans. Conveying concern and instilling the appropriate amount of hope in a very depressed patient is of importance early in the evaluation process. To say to the patient, "Even though it may seem to you as if it's hopeless right now, I know you will get better," will be very comforting to the depressed patient, even to those patients who may feel "undeserving."

Treatment of Bipolar Depression

The treatment of bipolar depression or manic-depressive illness is lithium carbonate. Therapeutic blood levels of 0.6 to 1.2 mEq/liter are usually maintained on 300 mg given three times a day. The therapeutic and toxic levels are close and warrant monthly following by blood levels. It is important not to give a diuretic or advise a low-salt or no-salt diet concomitant with lithium treatment, as increased excretion of salt will enhance retention of lithium (a monovalent cation, as is sodium), causing dangerous buildup of lithium in the blood. Side effects of nausea, vomiting, tremor, ataxia, and diarrhea are especially noteworthy and warrant attention. Lithium carbonate in therapeutic blood level range has a prophylactic function for both manic and depressive episodes.

Since the onset of lithium carbonate effectiveness is 10 to 14 days, during an acute manic episode the patient may require a major tranquilizer such as chlorpromazine (Thorazine) initially for the first few days. Suicide may be a definite risk during the depressive phase of this illness.

Unipolar Depressions

Unipolar depression (major depressive episodes including psychotic depression and involutional melancholia) are treated by tricyclic antidepressants as a first choice. Electroconvulsive treatment is necessary only if the depression is so extreme that severe psychomotor retardation or suicidal ideation is present. Only if the patient is severely agitated would the addition for a brief time of a major tranquilizer be necessary.

Tricyclic Antidepressants: Treatment Considerations

A family history of depression and drug response is important. If a family member was depressed and took a particular tricyclic and it worked, the same tricyclic would be indicated (provided there were no contraindications as detailed below) for the patient due to inherited enzymatic and metabolic tendencies. The tricyclic antidepressants work by blocking reuptake of biogenic amines (norepinephrine or serotonin) at nerve endings, leaving greater amine quantities at the site of action. All tricyclics are dose-equated and equal in antidepressant effects at 150 mg, except protriptyline (Vivactil) which is dose-equated at 25 mg.

The choice of tricyclic is based on side effects. The most sedative tricyclics, and therefore the most useful for agitated and anxious depression, are amitriptyline (Elavil) and doxepin (Sinequan), and the least sedative is imipramine (Tofranil). "Activating" tricyclics are represented by protriptyline (Vivactil), and are thus the choice for a retarded depression. Doxepin has the least cardiac side effects and toxicity and might be considered with patients with cardiac pathology, especially in arrhythmia or history of myocardial infarction, for whom a tricyclic is not contraindicated.

The dosage is critical, in that most treatment failures are a failure of adequate dosage and duration.[15] At least 150 mg should be given for at least three weeks for an adequate clinical trial. Plasma concentration studies reveal a great variation of

tricyclic blood level. Tricyclics may be started at 25 mg twice a day plus 50 mg at night increased by 25 mg every other day, and at one week changing to an all-nighttime dosage, thus obviating an additional sleeping medication if the patient is alerted to the anticholinergic side effects of dryness of mouth, blurred vision, constipation, and sedation. These side effects are usually present for only about one week. The patient should be apprised of the two-to-three week delay of onset of antidepressant effectiveness of the drug. Effective maintenance of the tricyclic will be achieved by a dosage of 150 mg, given at night, with a reduced maintenance dosage to be considered in the geriatric population. If the patient has a suicidal potential, prescribe a smaller amount, as twenty 150 mg tablets are enough for a successful suicide.[15]

The overall improvement rate with tricyclics and properly diagnosed depression is 80 percent.[15]

Medical contraindications to the use of tricyclics include a history of recent myocardial infarction, arrhythmia, thrombophlebitis, glaucoma, and hyperthyroidism. Specific reactions of note are that tricyclics prevent guanethidine, bethanidine, and clonidine from reaching their site of action and therefore decrease their antihypertensive effects. Tetrabenazine and reserpine interact with tricyclics to produce an increased cardiovascular toxicity.

Neurotic Depression

Somatic treatments (medication, ECT) do not work in neurotic depression. Supportive or insight therapy produces the best results; the choice of support or insight as an approach would depend on the patient's ego strength (motivation, insight, intelligence) and desire for treatment. If the patient wants and has the ability to use insight treatment, this might be the kind of depression and patient for whom a referral to a psychiatrist would be warranted, if one is available.

Treatment Approach Overview

With unipolar and bipolar depressions, pharmacologic therapy and supportive care are indicated. With neurotic and characterologic depressions, tricyclics do not work and either supportive or insight psychotherapy involving dynamic antecedents of the depression are indicated. In acute reactive states, crisis intervention may be necessary, in which the patient's baseline or previous level of functioning is established with the goal of intervention not being to end the crisis but to maximize chances for an adaptive solution and minimize chances of maladaptive solutions. Understanding the meaning of precipitating stress to the individual is an important factor in crisis intervention as well as depressions. All of these modalities with the possible exception of insight psychotherapy, and including crisis intervention, can be done very adequately by the primary care physician who is given clear diagnostic and assessment guidelines with specific treatment approaches.

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