CHAPTER 7: Mood Disorders and Suicide

Chapter Overview/Summary

Mood disorders (formerly called affective disorders) are those in which extreme variations in mood—either low or high—are the predominant feature. We all experience such variations at mild to moderate levels in the natural course of life, but for some people the extremity of moods in either direction becomes seriously maladaptive, even to the extent of suicide.

The vast majority of people with mood disorders have some form of unipolar depression—dysthymia or major depression. In these disorders, the person experiences a range of affective, cognitive, motivational, and biological symptoms including persistent sadness, negative thoughts about the self and the future, lack of energy or initiative to engage in formerly pleasurable activities, too much or too little sleep, and gaining or losing weight.

Unipolar depression may have multiple causes; traditional biological explanations have increasingly been shown to interact with more psychosocial factors. Among biological causal factors for unipolar depression, there is evidence of a moderate genetic contribution to the vulnerability for major depression, but probably not for persistent depressive disorder. Moreover, major depressions are clearly associated with multiple interacting disturbances in neurobiological regulation, including neurochemical, neuroendocrine, and neurophysiological systems. Disruptions in circadian and seasonal rhythms in depression are also prominent features of depression.

Among psychosocial theories of the causes of unipolar depression are Beck’s cognitive theory and the reformulated helplessness and hopelessness theories, which are formulated as diathesis-stress models where the diathesis is cognitive in nature (e.g., dysfunctional beliefs and pessimistic attributional style, respectively) and stressful life events are often important in determining when those diatheses actually lead to depression.

Personality variables, such as neuroticism, may also serve as diatheses for depression. Psychodynamic and interpersonal theories of unipolar depressions emphasize the importance of early experiences (especially early losses and the quality of the parent-child relationship) as setting up a predisposition for depression.

In the bipolar disorders (cyclothymia and bipolar I and II disorders), the person experiences episodes of both depression and hypomania or mania. During manic or hypomanic episodes, the symptoms are essentially the opposite of those during a depressive episode.

For bipolar disorders, biological causal factors probably play an even stronger role than for unipolar disorders. The genetic contribution to bipolar disorder is among the strongest of any psychiatric disorders. Biochemical imbalances, abnormalities of the hypothalamic-pituitary-adrenal axis, and disturbances in biological rhythms all play a role in bipolar disorder. Stressful life events may be involved in precipitating manic or depressive episodes. It is unlikely that they cause the disorder, but rather affect the timing and frequency of episodes of illness.

Treatment of unipolar depression may be successfully accomplished through cognitive-behavioral therapy (CBT), behavioral activation therapy, interpersonal therapy, antidepressant drugs, and electroconvulsive therapy (ECT). The biologically based treatments are more likely to lead to negative side effects, sometimes severe, and to result in greater chance of relapse or recurrence. Married couples in which one partner is experiencing depressions are probably best treated with marital therapy. Bipolar disorder is most frequently treated with mood stabilizing drugs such as lithium or one of the newer anticonvulsant drugs. Increasingly, however, psychosocial treatments are also being used with good effectiveness, especially in reducing the incidence of relapse or recurrence.

Suicide is a constant danger with depressive syndromes of any type or severity. Accordingly, an assessment of suicide risk is essential in the proper management of depressive disorders. A small minority of suicides appears unavoidable—chiefly those where the person really wants to die and uses a highly lethal method. However, a substantial amount of suicidal behavior (e.g., taking nonlethal or slow-acting drugs where the likelihood of discovery is high) is motivated more by a desire for indirect interpersonal communication than by a wish to die.

Somewhere between these extremes is a large group of people who are ambivalent about killing themselves and who initiate dangerous actions that they may or may not carry to completion, depending on momentary events and impulses.

Suicide prevention (or intervention) programs generally consist of crisis intervention in the form of suicide hotlines. Although these are undoubtedly effective in some cases in averting fatal suicide attempts, the long-term success of treatment aimed at preventing suicide in those at high risk is much less clear at the present time.

Detailed Outline
I. Mood Disorders: An Overview
   A. Types of Mood Disorders
      1. Unipolar depressive disorders.
      2. Bipolar disorders.
      3. Major depressive episode.
      4. Manic episode.
      5. Mood disorders—severe alterations in mood for long periods of time.
   B. The Prevalence of Mood Disorders
      1. Mood disorders occur with alarming frequency, at least 15–20 times more frequently than schizophrenia at almost the same rate as anxiety disorders.
      2. Lifetime prevalence rates of unipolar major depression are nearly 17%.
      3. Women are overly represented in unipolar depression 2:1. This is found cross-culturally

II. Unipolar Depressive Disorders
   A. Other Forms of Depression
      1. Loss and the grieving process
         a. Bowlby identified four phases of normal response to the loss of a spouse or close friend.
         b. Four phases include: numbing and disbelief; yearning and searching for the dead person; disorganization and despair; reorganization.
         c. Depressive symptoms tend to peak 2–6 months after the loss.
         d. Recent studies of bereaved individuals have found that about 50% exhibit genuine resilience.
         e. DSM-IV bereavement exclusion dropped
      2. Postpartum “blues”
         a. May occur in new mothers or fathers.
         b. It was believed postpartum major depression in mothers was common but more recent evidence states that only “postpartum blues” are common.
         c. Symptoms include changeable mood, crying easily, sadness, and irritability, often intermixed with happy feelings.
         d. 50%–70% of women experience the “blues” within 10 days of giving birth
         e. Rare to have accompanying psychotic features.
         f. Especially likely if new mother lacks social support, has difficulty adjusting to new demands, or if there is a history of depression.
         g. Hormonal readjustment and alterations in serotonin and noradrenaline functioning may play a role in the onset of postpartum blues and depression.
   B. Dysthymic Disorder (Persistent Depressive Disorder)
      1. Depressed mood of mild to moderate intensity.
      2. Primary hallmark is chronicity
         a. Average duration is four to five years.
         b. Chronic stress increases the severity of symptoms.
         c. Must be persistently depressed mood for most of the day for more days than not.
         d. Is common, with 2.5%–6% lifetime prevalence rates.
         e. Begins in adolescence, 50% of those seeking treatment have an onset before the age of 21 and 74% recover within 10 years.
      3. Half relapse usually occurs at 71% after the 3-year follow up.
   C. Major Depressive Disorder
      1. Cognitive, motivational, and biological symptoms
         a. Intense symptoms marked with sadness, insomnia, diminished cognitive capacity, and low self-esteem.
         b. Also known as major depression.
         c. To receive a diagnosis one must be experiencing a major depressive episode.
         d. Lethargy and lack of motivation.
         e. Anxiety symptoms are common.
         f. Must experience a depressed mood most of everyday and must also have three
to four of the following symptoms: feelings of worthlessness, guilt, thoughts of suicide, fatigue, physical agitation, changes in appetite and sleep.

2. Depression as a recurrent disorder
   a. Single (initial) vs. recurrent (preceded by one or more previous) episodes.
   b. Recurrence vs. relapse.
   c. 40%–50% may experience recurrence.

3. Depression throughout the life cycle

4. Specifiers for major depression
   a. **Major depressive episode with melancholic features** (see Table 7.1 for chart).
   b. Specifiers—different patterns of symptoms or features.
   c. **Severe major depressive episode with psychotic features:**
      (1) Mood congruent
      (2) Mood incongruent
   d. **Major depressive episode with atypical features.**
   e. **Major depressive episode with catatonic features.**
   f. **Recurrent major depressive episode with a seasonal pattern.**
   g. **Seasonal affective disorder.**
   h. Double Depression
      (1) Major depression coexists with dysthymia.
      (2) May be extremely common among those with dysthymia.

III. Causal Factors in Unipolar Mood Disorders

A. Biological Causal Factors

1. Genetic influences:
   a. Two to three times more common among blood relatives.
   b. Hippocrates hypothesized that depression was caused by an excess of black bile (400 B.C.).
   c. The monozygotic twin of a person with unipolar depression is twice as likely to develop unipolar depression as is a dizygotic twin.
   d. 31%–42% of the variance in unipolar depression is due to genetics.
   e. Serotonin transporter gene, the short allele (s) and the long allele (l).
   f. Genotype-environment interaction.

2. Neurochemical factors:
   a. Focus on two neurotransmitters of the monoamine class: norepinephrine and serotonin—monoamine hypothesis.
   b. Failure of research to support monoamine hypothesis.
   c. Focus now is on the interaction of neurotransmitters and how they affect cellular functioning.

3. Abnormalities of hormonal regulatory and immune systems:
   a. Hypothalamic-pituitary-adrenal (HPA) axis
      (1) Blood plasma cortisol levels elevated in 20%–40% of depressed outpatients and 60%–80% of severely depressed hospitalized patients.
      (2) Elevations in cortisol may be due to failure of feedback mechanisms—dexamethasone.
      (3) Recent evidence suggests that dexamethasone nonsuppression may be a general indicator of mental distress rather than specific to depression.
   b. Hypothalamic-pituitary-thyroid axis
      (1) 20%–30% of depressed people who have normal thyroid functioning show dysregulation in this axis.
      (2) Using drugs to increase thyroid hormone levels can lower depression in these individuals.
      (3) Prolonged elevations of cortisol lead to cell death in the hippocampus.
      (4) Hypothyroidism, which is low levels of thyroid, is often discovered.

4. Neurophysiological and neuroanatomical influences
   a. Depressed individuals show lower levels of EEG activity in the left hemisphere and higher levels in the right hemisphere.
b. May be able to use this to identify persons at risk for unipolar depression.
c. Several regions: orbital prefrontal cortex, dorsolateral prefrontal cortex, hippocampus, anterior cingulated cortex, and the amygdala have all been shown to play a role in depression.

5. Sleep and other biological rhythms
   a. Sleep: Sleep problems range from early morning awakening, periodic awakening during the night, and difficulty falling asleep.
      (1) Enter the first period of REM after only 75–80 minutes and show greater amounts of REM sleep.
      (2) Five states of sleep: stages 1 to 4 of non-REM sleep, and REM sleep make up a sleep cycle.
      (3) Rapid Eye Movement (REM) Sleep—rapid eye movements, dreaming, and bodily changes.
      (4) Patients who are depressed have early morning waking, periodic waking, and difficulty falling asleep.
   b. Circadian rhythms
      (1) Size or magnitude of the circadian rhythm may be blunted.
      (2) Circadian rhythm becomes desynchronized.
      (3) These control sleep, body temperature, REM sleep, and secretion of cortisol, thyroid, and growth hormone.
   c. Sunlight and seasons
      (1) Typically show increased appetite and hypersomnia.
      (2) Clear disturbances in circadian rhythm.
      (3) Majority of people diagnosed with seasonal affective disorder become depressed in the fall and winter, and normalize in the spring and summer.
      (4) Therapeutic use of light therapy.
   d. Biological explanation for sex differences
      (1) For females, hormones play a crucial role with the onset of puberty, before menstrual cycles, postpartum period, and menopause.
      (2) Women have a greater genetic predisposition.

6. Summary of biological causal factors
   a. Moderate genetic contribution is mediated by environmental factors.
   b. Stress response system is chronically overactivated.
   c. Severe depression is linked to multiple interacting disturbances in neurochemical, neuroendocrine, and neurophysiological systems. Those with less severe depressions may show few, if any, biological abnormalities.

B. Psychological Causal Factors
   1. Stressful Life Events as Causal Factors
      a. Stressful life events involved in precipitating depression include: loss of loved one, serious threats to important relationships or one’s occupations, severe economic or health problems, events involving humiliation.
      b. Independent life events vs. dependent life events.
      c. Severely stressful life events play a causal role in about 20%–50% of cases.
      d. Mildly stressful events and chronic stress.
      e. Minor events may play more of a role in the onset of recurrent episodes than in the initial episode.
      f. Vulnerability and invulnerability factors in response to stressors.
         (1) Vulnerability and invulnerability factors:
            (a) Genetics.
            (b) Living in poverty.
            (c) Chronic life stress.
   2. Different Types of Vulnerabilities for Unipolar Depression
      a. Personality and cognitive diathesis
         (1) Neuroticism or negative affectivity.
         (2) Introversion.
         (3) Negative patterns of thinking: internal, stable, and global.
b. Early adversity and parental loss as a diathesis
   (1) Parental loss when followed by poor care.
   (2) Early environmental adversity such as abuse, family turmoil, parental psychopathology, and so on.
   (3) Low self-esteem and insecure attachment with a caregiver.
   (4) Can lead to “stress inoculation” if adversity is moderate rather than severe and mediated by strengthening socio-emotional and neuroendocrine resistance.

c. Summary of different types of vulnerabilities.

3. Psychodynamic theories
   a. 1917—Mourning and Melancholia.
   b. Regression to the oral stage followed by incorporation of the lost person—anger turned inward.
   c. Later psychodynamic theorists such as Klein and Jacobson emphasized the quality of the early mother-infant relationship.
   d. Bowlby—attachment theory.

4. Behavioral theories
   a. Depression occurs when an individual’s responses no longer produce positive reinforcement or when the rate of negative reinforcement increases.
   b. Not very influential as an etiological theory.

5. Beck’s cognitive theory (See Figure 7.4 for a model of Beck’s Cognitive Model of Depression)
   a. Depressogenic schemas/dysfunctional beliefs
      (1) Beliefs predispose a person to depression.
      (2) Develop during childhood and adolescence as a function of one’s negative experiences with parents and significant others.
      (3) Activated by current stressors or depressed mood creating a pattern of negative automatic thoughts.
      (4) Negative cognitive triad: self, one’s experiences and the surrounding world, one’s future (see Figure 7.5 for model of the negative cognitive triad).
   b. Negative cognitive biases or errors maintain the negative cognitive triad
      (1) Dichotomous or “all or none” reasoning.
      (2) Selective abstraction.
      (3) Arbitrary inference.
   c. Evaluating Beck’s theory as a descriptive theory
      (1) Nondepressed people show a large positivity bias in attributions.
      (2) Stressors are not necessary to activate negative cognitive triad; simple depressed mood can activate negative cognitive triad.
      (3) Teasdale—vicious cycle of depression.
   d. Evaluating the causal aspects of Beck’s theory
      (1) Results mixed.
      (2) Recent studies suggest that those with high levels of dysfunctional attitudes and high stress are more likely to develop major depression than those with low stress or low dysfunctional attitudes and high stress.

6. The Helplessness and Hopelessness Theories of Depression
   a. Learned helplessness in laboratory dogs proposed as useful model of human depression.
   b. Reformulated Helplessness Theory
      (2) Pessimistic attributional style associated with depression.
      (3) Mixed results in testing whether this causes depression.
      (4) Many studies demonstrated that depressed people do indeed have a more pessimistic attributional style.
c. The hopelessness theory of depression
   (1) Revision of reformulated helplessness theory.
   (2) Hopelessness expectancy: one has no control over what will happen and something bad will happen.
   (3) Internal/external dimension not important.
   (4) Proposed two new dimensions: other likely negative consequences will occur and negative inferences about the implication of the event for the self-concept.
   (5) Initial research supports this conceptualization.

d. The ruminative response styles theory of depression
   (1) Focusing intently on how you feel and why you feel that way often leads to periods of depression more so than those who don’t feel that way.
   (2) Rumination—involves a pattern of repetitive and relatively passive mental activity.
   (3) Those with negative cognitive styles tend to ruminate and more likely to develop depression.
   (4) 7.1 Developments in Research Why Do Sex Differences in Unipolar Depression Emerge During Adolescence?

7. Interpersonal Effects of Mood Disorders
   a. Lack of social support and social skills deficits.
   b. The effects of depression on others.
   c. Marriage and family life
      (1) High correlation between marital dissatisfaction and depression.
      (2) Marital distress increases relapse for depression.
      (3) Parental depression increases problems for children.
      (4) 7.2 Developments in Thinking: Comorbidity of Anxiety and Mood Disorders.

IV. Bipolar and Related Disorders
A. Distinguished from unipolar disorders by the presence of manic or hypomanic episodes (See Figure 7.2 for a description of the manic-depressive spectrum.

B. Cyclothymic Disorder
   1. Cycles between hypomania and depression are signs of cyclothymia.
   2. Cyclothymic disorder—a less serious version of the full-blown bipolar disorder
   3. May be a mild form of major bipolar disorder.

C. Bipolar Disorders (I and II)
   1. Kraepelin—1899—manic-depressive insanity
   2. Bipolar I disorder
      a. One episode of mania or a mixed episode is needed for diagnosis.
      b. Mania shows elevated mood, irritability, increases in activity, and a “flight of ideas.”
      c. A mixed episode, once thought rare but increasing in occurrence, is characterized by symptoms of both full-blown manic and major depressive episodes for at least 1 week, where the symptoms are mixed or alternating rapidly every few days.
   3. Bipolar II disorder
      a. Hypomanic episodes and major depressive episodes with atypical features.
      b. More common than bipolar I, 2%–3% of the U.S. population.
   4. Features of bipolar disorder:
b. Symptoms during depressive episodes of bipolar disorder are identical to depressive symptoms in unipolar major depression.

c. Suicide attempts may be more common than in unipolar.

d. Average age of onset is 18–22 years old.

e. May be misdiagnosed until first manic episode appears.

f. Some drugs used to treat unipolar depression may actually trigger manic episodes in patients who have been misdiagnosed.

g. Rapid cycling in 5%–10% of patients.

h. 24% relapse within 6 months; 77% have a new episode within 4 years; 82% by 7 years.

V. Causal Factors in Bipolar Disorder

A. Biological Causal Factors

1. Genetic Influences

   a. Genes show that 8%–10% of the first-degree relatives of a person with bipolar I can be expected to also have bipolar I.

   b. About 70% of the genetic liability for bipolar is distinct from unipolar.

   c. Polygenic transmission; have not identified precise genes.

2. Neurochemical Factors

   a. Monoamine hypothesis of unipolar disorder extended to bipolar.

   b. Increased levels of dopamine may be related to manic symptoms.

   c. Abnormalities in how ions (such as sodium) are transported across the neural membranes (lithium may substitute for sodium ions).

3. Abnormalities of Hormonal Regulatory Systems

   a. Similar abnormalities as in unipolar with the hypothalamic-pituitary-adrenal axis and the hypothalamic-pituitary-thyroid axis.

   b. PET scans reveal that blood flow to the right frontal and temporal regions is reduced during manic episodes.

   c. More changes in subcortical structures than in unipolar, including enlarged basal ganglia and amygdale.

   d. Disturbances in circadian rhythms.

4. Neurophysiological and Neuroanatomical Influences

   a. Blood flow to the left prefrontal cortex is decreased during depression and increased during mania.

   b. Positron emission tomography (PET) has shown variations in the brain glucose metabolic rates in depressed and manic states of individuals.

   c. As with unipolar, there are deficits in the anterior cingulated cortex.

   d. In bipolar the basal ganglia and the amygdala are enlarged; in unipolar they are decreased in size.

   e. The decrease in the hippocampus found in unipolar is absent in the case of bipolar.

   f. Increased activation in the subcortical brain regions.

5. Sleep and Other Biological Rhythms

   a. During manic episodes individuals sleep very little, in depressed states, too much sleep.

B. Psychosocial Causal Factors

1. Stressful life events.

2. Other psychological factors in bipolar disorder:

   a. Low social support,

   b. Personality and cognitive variables: neuroticism, high levels of achievement striving, increased sensitivity to rewards in the environment, pessimistic attributional style.

VI. Sociocultural Factors Affecting Unipolar and Bipolar Disorders

A. Cross-Cultural Differences in Depressive Symptoms
(See Figure 7.8 for the prevalence rates for depression across several nations)

1. China and Japan—psychological symptoms of depression are low; somatic and vegetative manifestations are higher.
2. As Asian cultures have incorporated Western values, rates of depression have increased.
3. Adolescents from Hong Kong were shown to have higher rates of depression than adolescents in the United States.

B. Cross-Cultural Differences in Prevalence
1. Varies tremendously; Taiwan—1.5%, whereas United States and Lebanon—17% to 19%
2. Need to identify risk factors in each culture.

C. Demographic Differences in the United States
1. No large racial differences identified.
2. Inversely related to socioeconomic status in unipolar depression; bipolar is more common in higher socioeconomic classes.
3. Mania or hypomania may facilitate the creative process; intense negative emotional experiences of depression provide material for creative activity.
4. African Americans might display chronic course of depression compared to white Americans.
5. Native Americans have a higher rate of depression than white Americans.

VII. Treatments and Outcomes
A. Only about 40% of people with mood disorders receive minimally adequate treatment, with the other 60% receiving no treatment or inadequate care.

B. Pharmacotherapy
1. First category of antidepressant drugs, developed in the 1950s, were the monoamine oxidase inhibitors (MAOIs).
2. Treatment of choice from 1960s–1990 was one of the tricyclic antidepressants such as imipramine.
3. Selective Serotonin Re-uptake Inhibitors (SSRIs)
4. Recently several atypical antidepressants (Wellbutrin, Serzone, Remeron) have become popular because they have fewer side effects or are more effective with severe depression.
5. The course of treatment with antidepressant drugs:
   a. Require 3–5 weeks to take effect.
   b. 50% do not respond to first drug tried.
   c. Discontinuing drug may lead to relapse.
   d. 25% of patients relapse while on drugs.
6. Lithium and other mood-stabilizing drugs
   a. Lithium
      (1) 75% of patients show at least partial improvement on lithium.
      (2) Some unpleasant side effects can be seen such as lethargy, decreased motor coordination, and GI difficulties; long-term use can cause kidney damage.
      (3) Compliance is a major problem.
   b. Anticonvulsants (carbamazepine, divalproex, and valproate)
      (1) Often effective in those who do not respond to lithium.
      (2) Risk for attempted and completed suicide increases 2 to 3 times on anticonvulsants compared to lithium.

C. Alternative Biological Treatments
1. Electroconvulsive Therapy
a. Severely depressed patients who present an immediate and serious suicidal risk would be most appropriate for ECT.
b. Typical treatment involves 6–12 sessions administered every other day.
c. Varying levels of amnesia may persist.
d. Has also been found to be of use in manic episodes.

2. Transcranial Magnetic Stimulation (TMS)
   a. Noninvasive technique allowing focal stimulation of the brain in awake patients.
   b. Brief, but intense, pulsating magnetic fields which induce electrical activity in certain parts of the cortex are delivered.
   c. Some studies have shown TMS to be more effective than antidepressants without the side effects of ECT.

3. Deep Brain Stimulation
   a. Used for individuals with refractory depression who have not responded to other treatments such as medication, psychotherapy, and ECT.
   b. Involves implanting an electrode in the brain and then stimulating that area of the brain with an electrical current.

4. Bright Light Therapy
   a. Originally used only for seasonal affective disorder.
   b. Recently shown to be effective for nonseasonal depression.

D. Psychotherapy
   1. Cognitive-behavioral therapy
      a. Focuses on here-and-now problems; teaches people to evaluate their beliefs and negative automatic thoughts systematically as well as to challenge their underlying depressogenic assumptions.
      b. Equally, or more, effective as antidepressants and more effective in preventing relapse.
      c. Cognitive therapy, originally developed by Beck and colleagues.
   2. Behavioral activation treatment is relatively new
      a. Focuses on getting patients to become more active and engaged with their environment and their interpersonal relationships.
      b. May be even more effective, and easier to administer, than CBT.
      c. Modified form of CBT may be effective with bipolar as well.
      d. Mindfulness-based cognitive therapy: developed for highly recurrent depression; involves training in mindfulness meditation techniques aimed at increasing awareness of unwanted thoughts and feelings and sensations so they are accepted as simply thoughts and not reality; found effective in two studies.
   3. Interpersonal therapy (IPT)
      a. Not as extensively studied or used.
      b. Research suggests it is as effective as CBT or antidepressants.
      c. Focuses on current relationship issues, trying to help the person understand and change maladaptive interaction patterns.
      d. Adapted for treatment of bipolar disorders by focusing on stabilizing daily life
   4. Family and marital therapy
      a. For some types of bipolar disorder, reducing the level of expressed emotion or hostility and increasing coping information has been shown to be effective in preventing relapse.
      b. For unipolar, focusing on marital discord is as effective as CBT.
      c. Stressors in the patient’s life may lead to recurrent depression and require longer treatment.

5. Conclusions

VIII. Suicide: The Clinical Picture and the Causal Pattern

A. General Information
   1. Depressed individuals are 50%–90% more likely to commit suicide than nondepressed individuals.
   2. Ranks among the 10 leading causes of death in most Western countries.
3. 90% of people who either attempted or committed suicide had some psychiatric disorder.
4. Most people who commit suicide are ambivalent and are alone and in a state of severe psychological distress and anguish.
5. **Suicide**—taking one’s own life.

**B. The Clinical Picture and the Causal Pattern**

1. **Who Attempts and Who Commits Suicide?** (See Figure 7.11 for chart of U.S. suicide rates by age, gender, and race).
   a. Until recently the peak age for suicide attempts was 25–44; now the peak age is 18–24 with three times as many women attempting suicide compared to men.
   b. Highest rate of completed suicides is in the elderly (65 and over), particularly those who are divorced, widowed, or suffer from chronic illness.
   c. Methods of suicide vary between the genders.
   d. 7th leading cause of death for men and 15th leading cause of death for women.
   e. High-risk groups include the depressed, those suffering from schizophrenia, alcoholics, divorced persons, certain professionals (highly creative or successful scientists, physicians and psychologists, businessmen, composers, writers, and artists), people living alone, and people from socially disorganized areas.

2. **Suicide in Children**
   a. Rates have been increasing—suicide under the age of 10 is rare.
   b. Ages 10–14: suicide is 5% of deaths.
   c. Increased risk if child has lost a parent or has been abused.

3. **Suicide in Adolescents and Young Adults**
   a. Third most common cause of death in ages 15–19.
   b. For people ages 15–24 suicide has tripled from the mid-1950s to the mid-1980s.
   c. Second leading cause of death among college students.
   d. Increases in suicide rates for adolescents observed worldwide.
   e. Known risk factors for adolescent suicide:
      (1) Mood disorder, conduct disorder, and substance abuse (especially alcohol).
      (2) Treatment with antidepressants.
      (3) Media exposure to suicides.
      (4) For college students, the combined stressors of academic demands, social interaction problems, and career choices.
      (5) 7.3 The World Around Us: Warning Signs for Student Suicide.

4. **Other Psychosocial Factors Associated with Suicide**
   a. Personality traits such as impulsivity, aggression, pessimism, and negative affectivity.
   b. Associated with negative life events.
   c. Hopelessness about the future may be a good predictor of suicide within one to two years.
   d. Predictors of immediate suicide among the depressed: severe psychic anxiety, panic attacks, severe anhedonia, global insomnia, delusions, and alcohol abuse.
   e. Shneidman—suicide is an escape from pain.
   f. Psychosocial causes: combination of family psychopathology, child maltreatment, and family instability associated with low self-esteem, hopelessness, and poor problem-solving skills.

5. **Biological Causal Factors**
   a. **Concordance rate in monozygotic twins about 3 times higher than in fraternal twins.**
   b. Reduced serotonergic activity.
   c. People who are hospitalized with low levels of serotonin are 10 times more likely to kill themselves in the next year than are those without low serotonin levels.
   d. Having one or two copies of the short allele.

6. **Sociocultural Factors**
   a. Whites have much higher suicide rates than African Americans except among young males where rates are similar.
   b. United States: 11 per 100,000 people.
c. Rates vary from one society to another (Hungary is the highest: 40 per 100,000 people).

d. Western countries with high suicide rates (20 per 100,000) are Switzerland, Finland, Austria, Sweden, Denmark, and Germany.

e. 30% of suicides worldwide occur in China and India.

f. Religious beliefs are important determinants of suicide rates (Catholics and Islamic countries are low).

g. Japan is one culture that sanctions certain suicides; Muslim extremists are another.

h. Subgroup differences exist within societies.

i. Durkheim’s view of group cohesiveness as a factor in suicide.

IX. Suicidal Ambivalence

A. Ambivalence

1. Some wish to communicate a message to others.
2. Some are seemingly intent on ending their lives.
3. Some are ambivalent and leave death to chance.
4. Following an attempt there is a reduction in emotional turmoil.
5. In the year after a suicide attempt repetition occurs in 15%–25%.

B. Communication of Suicidal Intent

1. 40% communicate suicidal intent in very clear and specific terms; an additional 30% had talked about death and dying.
2. 50% had never seen a mental health professional.
3. Among inpatients hospitalized for suicidal ideation or intent, nearly 80% denied suicidal ideation the last time they spoke with a clinician.

C. Suicide Notes

1. Only about 15%–25% leave notes.
2. Suicide notes are typically coherent and legible; some include statements of love and concern; occasionally suicide notes contain very hostile content.
3. Most notes do not provide significant insights into the suicidal mind.

X. Suicide Prevention and Intervention

A. Treatment of Mental Disorders

B. Crisis Intervention

1. Suicide prevention centers try to avert an actual suicide attempt.
2. Primary objective is to help people regain their ability to cope with their immediate problems as quickly as possible.
3. Emphasis is placed on:
   a. Maintenance of supportive contact with person.
   b. Helping the person to realize that distress is impairing judgment.
   c. Helping the person see that present distress is not endless.
   d. Establishment of suicidal hotlines since the 1960s—not much information to document success in reducing suicide rates.

D. Focus on High-Risk Groups and Other Measures

1. Broad-based programs are needed to alleviate life problems.
2. Involving older men in social and interpersonal activities that help others.
3. 10 sessions of cognitive therapy for those who have already attempted suicide once has proved beneficial in reducing further attempts.

I. Unresolved Issues: Is There a Right to Die?

A. Ethical Issues in Suicide Prevention
   1. Should people be allowed to take their own lives?
      a. Ancient Greeks provided hemlock to those who had received permission to commit suicide due to illness.
      b. Some Western European countries allow terminally ill people to commit suicide.
      d. Dr. Jack Kevorkian (1928–2011).
   2. Terminally ill people are asking for the “right to commit suicide.”

B. Suicide Intervention is a Neutral Moral Stance
   1. Refers to interceding without the implication of preventing the act.
   2. May encompass the possibility of facilitating the suicidal person’s objective

C. Moral problems associated with involuntary hospitalization.
   1. Personal items removed; medication may be forcibly administered.
   2. Practitioners take a cautious and conservative path to avoid legal repercussions on both sides.
   3. This may lead to some people being institutionalized based upon limited clinical justification.

Key Terms

- attributions
- behavioral activation treatment
- bipolar disorder with a seasonal pattern
- bipolar disorders
- bipolar I disorder
- bipolar II disorder
- chronic major depressive disorder cognitive-behavioral therapy (CBT) (cognitive therapy)
- cyclothymic disorder
- depression
- depressogenic schemas
- double depression
- dysfunctional beliefs
- dysthymic disorder
- electroconvulsive therapy (ECT)
- hypomanic episode
- interpersonal therapy (IPT)
- learned helplessness
- lithium
- major depressive disorder
- major depressive episode
- major depressive episode with atypical features
- major depressive episode with catatonic features
- major depressive episode with melancholic features
- mania
- manic episode
- mixed episode
- monoamine oxidase inhibitors (MAOIs)
- mood congruent
- mood disorders
- negative automatic thoughts
- negative cognitive triad
- persistent depressive disorder
- pessimistic attributional style
- rapid cycling
- recurrence
- recurrent major depressive episode with a seasonal pattern
- relapse
- ruminations
- seasonal affective disorder
- selective serotonin reuptake inhibitor (SSRI)
- severe major depressive episode with psychotic features
- specifiers
- suicide
- tricyclic antidepressants
- unipolar depressive disorder