Co-Occurring Addiction and Psychiatric Disorders
The focus of this chapter is to help clinicians understand, differentially diagnose, and treat a person with substance-induced psychiatric syndromes that mimic traditional psychiatric disorders such as depression, anxiety, and psychotic disorders. Because all substances of abuse work by altering the same kinds of neurotransmitters that are thought to be involved in these psychiatric disorders, it should not be surprising that many patients with substance abuse, dependence, or withdrawal may appear to be psychiatrically ill or at least appear to have significant psychiatric symptoms. The fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) of the American Psychiatric Association developed new definitions of the substance-induced mental disorders, which differ slightly from the guidelines offered in the revised third edition (DSM-III-R) and is unlikely to change in DSM-5, which is scheduled to be published while this text is in press. This chapter describes the diagnostic criteria in the DSM-IV, reviews the epidemiologic data, and discusses the clinical strategies needed to manage these disorders. Nine substance-induced mental disorders are given in the DSM-IV:

- Substance-induced delirium
- Substance-induced persisting dementia
- Substance-induced persisting amnestic disorder
- Substance-induced psychotic disorder (SIPD)
- Substance-induced mood disorder
- Substance-induced anxiety disorder
- Hallucinogen persisting perception disorder
- Substance-induced sexual dysfunction
- Substance-induced sleep disorder

Here, we will focus on those that are most confounding in terms of differential psychiatric presentation (substance-induced psychotic, mood, and anxiety disorders and hallucinogen persisting perceptual disorder). These will be referred to as substance-induced psychiatric disorders. Three others are referred to generically as “organic brain syndrome”: substance-induced delirium, substance-induced persisting amnestic dementia, and substance-induced persisting amnestic disorder and are not the focus of this chapter or are substance-induced sexual dysfunction and substance-induced sleep disorder (though substance-related sleep disorders are discussed in many of the chapters in this section and others).

### Prevalence of Substance-Induced Psychiatric Disorders

#### Prevalence of Substance-Induced Mood and Anxiety Disorders

Prevalence rates of substance-induced psychiatric disorders vary considerably depending on the study subjects (treatment-seeking populations vs. epidemiologic surveys) and the research diagnostic criteria used to define the disorders (e.g., how long is a substance-induced syndrome defined to last). Brown and Schuckit (1) reported that 42% of the male alcoholics presenting for treatment displayed depressive symptoms in a range comparable to that seen in individuals hospitalized for affective disorder (more than 19 points, which is in the moderate to severe range of the Hamilton Depression Rating Scale). The symptoms abated rapidly over the first 2 weeks of abstinence, with only 12% of the subjects still depressed at the end of the 2nd week. In light of this rapid abatement of depressive symptoms, it is significant that the subjects averaged more than 9 days of abstinence before the study began.

In a study of similar alcoholic subjects who had been sober for an average of 8 days, Brown et al. (2) reported that 33% of the primary alcoholics (with or without secondary affective disorder) scored in the moderate to severe range for depression, whereas 81% of the subjects with primary affective disorder did so at the end of week 1. By week 4 of the study, none of the patients with primary alcoholism were in the moderate to severe range, whereas 67% of the subjects with primary affective disorders were. This finding suggests that all of the subjects with primary alcoholism had alcohol-induced depressive disorder, a number quite...
comparable to the 30% reported in the earlier study by the same researchers. As with depressive symptoms, anxiety symptoms show similar changes over the early sobriety phase. Several studies have reported high rates of anxiety symptoms among alcoholics in withdrawal, with 80% of alcohol-dependent male subjects experiencing repeated panic attacks during alcohol withdrawal (3). In the same study, 50% to 67% of the alcohol-dependent subjects had high scores on the state anxiety measures, which resembled generalized anxiety and social phobia (3). Brown et al. (4) reported that 40% of recently detoxified alcohol-dependent men scored above the 75th percentile on the state anxiety subscale of the State Trait Anxiety Inventory. At discharge after 4 weeks, 12% scored that high, whereas at 3-month follow-up, only 5% remained above the 75th percentile. This finding suggests that 33% had an alcohol-induced anxiety disorder. Moreover, if 80% of alcoholic men report withdrawal panic attacks, is this alcohol withdrawal–induced anxiety disorder or is it an independent panic disorder evoked by alcohol withdrawal? Or is it merely anxiety symptoms caused by alcohol withdrawal?

Schuckit et al. (5) studied nearly 3,000 alcoholics to test three hypotheses related to substance-induced depression. They hypothesized that there would be more substance-induced depressive disorder (SIDD) than major depressive disorder (MDD), that those with SIDD would have more severe alcohol and drug histories, and that those with independent depression would have more first-degree relatives with affective disorder. All three hypotheses were supported by their findings. Among the study population, 15% had an independent depression and 26% reported information consistent with SIDD. Subjects with SIDD drank more alcohol per occasion, drank on more days per week, sought treatment more often, were more likely to have attended Alcoholics Anonymous meetings, and used more marijuana and stimulants than did those with co-occurring or no depression. Women were more likely to have co-occurring than substance-induced depression and less likely to not have depression. Those diagnosed with SIDD were more likely to have antisocial personality disorder. Subjects with co-occurring depression also had more anxiety disorders than did those with SIDD (8.4% vs. 4.8%) and the same percentage of mania (1.6% vs. 1.0%). More recent studies using the DSM-IV criteria and structured clinical interviews show a wider variety of results depending largely on the study population used. Kahler et al. (6) looked at substance-induced and independent MDD in treatment-seeking alcoholics and found that out of 166 alcoholic patients with elevated levels of depression, 122 met the Structured Clinical Interview for DSM-IV criteria for MDD. Of this group, 61.6% were found to have “pure” SIDD and 15.2% were found to have independent MDD. The remaining 23.2% had the diagnosis of SIDD with a history of independent MDD. In contrast to treatment-seeking populations, Grant et al. (7) in a major epidemiologic study found the prevalence of substance-induced mood and anxiety disorders to be extremely low in the general population (1.05% for current substance-induced mood disorders and 0.22% for substance-induced anxiety disorders). However, the prevalence of those with current independent mood disorders who reported having both independent and substance-induced mood disorders in the prior year was 7.35%. Of those with current independent anxiety disorders, 2.95% reported having both substance-induced and independent anxiety disorders in the prior year. Using the Psychiatric Research Interview for Substance and Mental Disorders (PRISM-IV) (8), which was designed to distinguish between substance-induced and independent psychiatric disorders in co-occurring populations, Langás et al. (9) interviewed first-time consecutively admitted patients aged 16 years or older who were admitted to addiction clinics and psychiatric outpatient or inpatient facilities. In this study, among those with comorbid addictive and psychiatric disorders (n = 54), 41% had psychiatric disorders considered to be independent of their substance use, 7% had substance-induced disorders only, and 38% had both independent and substance-induced disorders.

It can be very difficult to differentiate between substance-induced and independent depressive disorders, and the diagnosis may change if the patient is followed over time. Ramsey et al. (10) studied alcoholics with SIDD and found that over a course of a year, 26.4% of those diagnosed with SIDD were reclassified as having an independent MDD because of meeting full criteria for the diagnosis of major depression after 1 month of sobriety. In this study, those with a history of past independent major depression were five times more likely to be reclassified from substance-induced depression to independent depression. Patients who had lower severity of alcohol dependence were also more likely to be reclassified as having independent major depression. Nunes et al. (11) studied depressive disorders in patients with alcoholism admitted to an inpatient psychiatric unit using the PRISM-IV and found that 51% of patients had SIDD; however, after following them for a year, 32% of these patients were reclassified as having independent depression. Regarding cocaine-induced depressive disorders, Rounsaville et al. (12), in a study of patients addicted to cocaine, examined the current and lifetime prevalence of research diagnostic criteria disorders. They used both strict and less strict criteria in evaluating the depression diagnostic data from the Schedule for Affective Disorders and Schizophrenia—Lifetime. The less strict criteria allowed a diagnosis if the symptoms ever had been present, whereas the strict criteria allowed a diagnosis only if symptoms had persisted for 10 or more days after cessation of cocaine use. Using the less strict criteria, major depression was diagnosed in 59% of the subjects, whereas the more strict criteria yielded a 30% prevalence rate. This finding conservatively suggests a lifetime prevalence of about 30% for cocaine-induced depressive disorder. Further, the current rate of major depression was 4.7%, hypomania was 2%, and minor mood disorder was 38% (mania was 0%) (12). (The current diagnoses of minor mood disorders appeared to use...
the strict criteria, but this was not clarified in the report.) Rounsaville (12) also reported a 16% current rate and a 21% lifetime rate of anxiety disorders, but the investigators did not disclose what criteria were used to diagnose anxiety unrelated to cocaine use.

Methamphetamine users are also reported to have high rates of depressive symptoms and suicidal behavior during active use as well as during withdrawal and early abstinence. A large study of psychiatric symptoms among 1,016 methamphetamine users presenting for treatment revealed that depression was the most common symptom reported and 27% had attempted suicide in the past (13).

Prevalence of Substance-Induced Psychotic Disorders

Psychosis during intoxication is common among those abusing psychotomimetic drugs of abuse, which include cannabis, cocaine, amphetamines and related stimulants, hallucinogens, and dissociative drugs such as phencyclidine (PCP) and ketamine. Regarding the prevalence of SIPD, Brady et al. (14) evaluated individuals admitted for treatment of cocaine dependence and found that 53% reported transient cocaine-induced psychosis. Caton et al. (15) evaluated psychotic individuals with substance abuse presenting to a psychiatric emergency department in New York using PRISM-IV and reported a prevalence of 44% for SIPD, while the other 56% had primary psychotic disorder (PPD) with concurrent substance use. In a later study by Fraser et al. (16), the PRISM-IV differentially diagnosed 56% of first-episode patients with SIPD and 44% with PPD.

An Australian study done by McKetin et al. (17) to examine the prevalence of psychotic symptoms among 309 regular methamphetamine users not presenting for treatment found that 13% had psychotic symptoms and 23% had experienced clinically significant psychotic symptoms in the past year. This study reported that the prevalence of psychosis among current methamphetamine users was 11 times higher than among the general population. Although methamphetamine psychosis in general has a better prognosis than a PPD (18), studies conducted in Japan showed that chronic intravenous methamphetamine use is associated with increased rates of prolonged psychosis persisting for several months to over 2 years after abstinence that closely resembles paranoid schizophrenia (19,20).

Substance-Associated Suicidal Behavior

Substance-induced depression can dissipate rapidly, but it is as dangerous as MDD in terms of the risk of suicide and self-injurious behavior. When completed suicides are investigated, the rate of comorbidity is high. Henriksson et al. (21) reported that nearly half (43%) of a group of suicide victims in Finland had alcohol dependence and that 48% of the alcoholics had comorbid depression, 42% had a personality disorder, and 36% had a significant Axis III medical disorder. Salloum et al. (22) studied patients who had been hospitalized psychiatrically and found that more than half of the subjects in all three groups studied (with alcohol dependence, cocaine dependence, or alcohol plus cocaine dependence) had a history of suicide attempts. Zweben et al. (13) also found a high prevalence of prior suicide attempts (27%) in methamphetamine-dependent patients presenting for treatment. Elliott et al. (23) found that patients who made medically severe suicide attempts had a statistically higher rate of substance-induced mood disorder than did patients who made less severe suicide attempts. There was no difference between the two groups in the prevalence of alcohol abuse or dependence or in the prevalence of polysubstance abuse or dependence. Moreover, most of the patients with substance-induced mood disorder did not meet the criteria for substance dependence. This finding is consistent with the findings of Asnis et al. (24) and Murphy and Wetzel (25), who argued that alcohol dysregulates mood independent of use patterns, suggesting that some individuals are at risk of severe depression regardless of the chronicity of their alcohol use. Conner et al. (26) analyzed suicidal behavior in 3,729 alcoholic individuals and concluded that both independent and substance-induced depression are associated with suicidal ideation and planning, whereas alcohol-related aggression is correlated with suicide attempts. Aharonovich et al. (27) studied substance-dependent patients who had attempted suicide and found that patients with substance-induced depression were as likely as those with independent depression to have attempted suicide.

Ries et al. (28) studied acutely suicidal psychiatric inpatients with substance-induced psychiatric disorders and found that this subgroup had higher severity of suicidal ideation but improved more quickly than other patients and tended to have shorter lengths of stay. Among schizophrenics, however, Bartels et al. (29) found that it was the severity of the depression, not the substance abuse, which explained suicidal behavior. In contrast, Seibyl et al. (30) reported that schizophrenics who had used cocaine before admission exhibited increased suicidal ideation.

In summary, although a great deal of effort has gone into characterizing substance-induced psychiatric syndromes in both addictions and psychiatric populations, findings are widely variable. Results of prevalence studies in clinical populations are influenced by differences between the populations studied, the diagnostic criteria employed, and the type of interview used. In addition, despite the best methods of classification, a significant number of “substance-induced depression” cases turned out to have independent major depression over the following year. However, it is also clear that substance-induced states are common, and it is logical that clinical intervention for an alcohol-induced depression would focus on different issues (e.g., getting sober and into recovery) than typical major psychiatric depression (medications and psychotherapy). But what if a patient has both problems? Or a little of one, but a whole lot of the other? Ries et al. (31) have attempted to straddle the “either/or” problem by showing that clinicians...
can validly classify substance-induced syndromes in acute psychiatric patients admitted to an acute county hospital as having 0 = no substance-induced effect, 2 = mild substance effect (about 25% of the syndrome being substance related), 3 = moderate substance effect (about 50% of the syndrome being substance related), or 4 = major substance-induced effect (75% or greater substance related). They have suggested that rather than a separate diagnosis, using this system allows for a better clinical description of presenting patients, because in many cases, acute psychiatric patients have both conditions at the same time, for example, bipolar disorder with acute cocaine-induced paranoia.

**SPECIFIC SUBSTANCES: SUBSTANCE-INDUCED SYMPTOMS**

The occurrence of psychiatric symptoms as a result of legal and illegal drug use has been well documented. It is common medical knowledge that hallucinogens cause hallucinations, stimulants cause euphoria, and chronic sedative use can result in depression. It is common medical knowledge that in acute withdrawal, alcohol and sedatives cause anxiety. It is less obvious that a distinct set of symptoms appear when psychoactive substances are used over a long period. Symptoms reported for each of the major substances of abuse are reviewed below to establish a basis on which to understand the syndromes that can arise.

**Caffeine,** the most commonly used stimulant, is considered a benign drug by many consumers and professionals and is very popular worldwide. However, the abuse of high caffeine content “energy drinks” is increasing (32). Energy drinks can contain anywhere from 80 to 500 mg of caffeine (33). Symptoms of caffeine intoxication include anxiety, restlessness, insomnia, gastrointestinal upset, tremors, tachycardia, psychomotor agitation, and even death due to arrhythmias (38). It enhances dopaminergic actions indirectly via potentiation of dopaminergic neurotransmission through competitive antagonism at the adenosine receptors (33). Caffeine withdrawal occurs frequently although it is usually mild and is characterized by headaches, fatigue, drowsiness, impaired concentration, and depressed mood, which occur 12 to 24 hours after cessation of consumption and reach a peak after 20 to 48 hours. Withdrawal symptoms disappear shortly after ingestion of caffeine. Tolerance to the psychoactive effects of caffeine is also reported (32,33). Nicotine is the deadliest psychoactive drug, and the prevalence of depression among smokers has been estimated at three times that of nonsmokers (34). Some smokers experience relapse of depressive episodes during quit attempts (35).

**Alcohol** use is common among American adolescents and young adults. Although light consumption of alcohol is associated with a slight euphoria or “buzz,” moderate to heavy consumption may be associated with depression, suicidal feelings, or violent behavior in some individuals. With prolonged drinking, the incidence of dysphoria and anxiety rises, much to the distress of the drinker. In those who are physiologically dependent, one usually sees a hyperadrenergic state that is characterized by agitation, anxiety, tremor, malaise, hyperflexia, mild tachycardia, increasing blood pressure, sweating, insomnia, nausea or vomiting, and perceptual distortions. After acute withdrawal from alcohol, some persons suffer from continued mood instability, with moderate lows, fatigability, insomnia, reduced sexual interest, and hostility. A fewchronic heavy drinkers experience hallucinations, delusions, and anxiety during acute withdrawal, and some have grand mal seizures. Brain damage of several types is associated with alcohol-induced dementias and deliriums.

With **sedative–hypnotics,** particularly the benzodiazepines, acute use can produce a “high” similar to that seen with alcohol. The drug effects are perceived as relaxing, producing a social ease, but sedatives also can induce depression, anxiety, and even withdrawal-induced psychosis with prolonged use and dependence (36). Withdrawal symptoms include mood instability with anxiety or depression, sleep disturbance, autonomic hyperactivity, tremor, nausea or vomiting, transient hallucinations or illusions, and grand mal seizures. A protracted withdrawal syndrome has been reported to include anxiety, depression, paresthesias, perceptual distortions, muscle pain and twitching, tinnitus, dizziness, headache, derealization and depersonalization, and impaired concentration. These symptoms can last for weeks, and some (such as anxiety, depression, tinnitus, and paresthesias) have been reported for a year or more after withdrawal (36).

**Stimulants: Cocaine, amphetamine, and methamphetamine** use often is associated with an intense euphoria or “rush,” with hyperactive behavior and speech, hypersexuality, anorexia, insomnia, inattention, and labile moods. The route of administration and the dose alter the intensity of the experience. Depressive symptoms and cognitive problems as well as hypersomnia, decreased energy, and increased appetite commonly occur during a stimulant withdrawal phase. After a methamphetamine binge of several days, addicts will often be hostile and agitated, which is referred to as “tweaking” as they stop their use, or they may use other drugs such as benzodiazepines or alcohol to moderate the agitation. Individuals can become paranoid and delusional after prolonged heavy use of stimulants (37,38). Once abstinence is maintained for several weeks, the psychotic symptoms usually attenuate and resolve, but many stimulant addicts report a dysphoric state that is prominently marked by anhedonia and/or anxiety, but which does not meet the symptom severity criteria to qualify as a DSM-IV disorder. This anhedonic state can persist for weeks to months. Chronic and heavy methamphetamine users, particularly those who use intravenously, have an increased rate of psychosis and depression lasting several months or even years that closely resembles paranoid schizophrenia (19,20). Stimulant addicts frequently report hallucinatory symptoms that are visual (“coke snow”) and tactile (“meth mites” or formication). Sleep disturbances are prominent in the intoxicated and withdrawn states, as is sexual dysfunction. Methyleneoxymethamphetamine (MDMA),
more commonly known as “Ecstasy,” intoxication produces stimulant effects similar to cocaine and amphetamine as well as empathogenic effects of empathy, a sense of well-being, and sociability due to serotonergic activity (39). Withdrawal states are characterized by depression, hypersomnia, poor concentration, and fatigue. Chronic MDMA users may develop more severe long-term problems such as dysphoric states and cognitive impairments in memory, concentration, and executive functioning, which are thought to be due to serotonergic neurotoxicity (39).

Opiate use is characterized by a “high” or “rush” when the drug is used intravenously or smoked. Unlike the stimulants, opiate-induced euphoria usually is associated with some sedation and manifests as a mellow, sleepy state. If opiates are used for a long period, moderate to severe depression is common. The addict frequently experiences irritability, accompanied by craving, muscle aches, a flu-like syndrome, and gastrointestinal symptoms early in withdrawal from drugs such as heroin and morphine. More drug subsides the craving. In withdrawal, some opiate addicts are acutely anxious and agitated, whereas others report depression and anhedonia. Anxiety, depression, and sleep disturbance, in a milder form, can persist for weeks to months as a protracted withdrawal syndrome that gradually subsides.

Classical hallucinogens such as lysergic acid diethylamide (LSD), mescaline, psilocybin, and dimethyltryptamine produce visual distortions and frank hallucinations. All hallucinogens are associated with drug-induced panic reactions that feature panic, paranoia, and even delusional states in addition to the hallucinations. However, they are not associated with dependence or withdrawal syndromes. A few hallucinogen users experience chronic reactions, involving (a) prolonged psychotic reactions; (b) depression, which can be life threatening; (c) flashbacks; and (d) exacerbations of preexisting psychiatric illnesses. The flashbacks are symptoms that occur after one or more psychedelic trips and consist of flashes of light and afterimage prolongation in the periphery. The DSM-IV refers to flashbacks as “hallucinogen persisting perception disorder” and requires that they be distressing or impairing to the patient.

Marijuana (cannabis) and hashish or hash oil contain tetrahydrocannabinol (THC). Intoxication with THC augments appetite and causes sedation with euphoria. Some users experience a marked sense of time distortion and feelings of depersonalization. A cannabis withdrawal syndrome is described (40) that is generally mild and consists of anxiety, irritability, physical tension, depressed mood, decreased appetite, restlessness, and craving. Recent literature has emerged that early use of cannabis is a risk factor for the development of psychotic symptoms later in life (41).

Phencyclidine (PCP), an arylcyclohexylamine, and ketamine are N-methyl-D-aspartate (NMDA) antagonists and dissociative drugs that cause hallucinations and dissociative states. PCP is known for its dissociative and delusional properties. It also is associated with violent behavior and amnesia of the intoxication (42). Over the last decade, PCP use has increased in combination with cannabis.

Designer drugs and other emerging drugs of abuse often found on the Internet and advertised as “legal highs” have been appearing with increasing frequency over the past few years. Synthetic cathinones popularly known as “bath salts” are all synthetic analogs of cathinone derived from leaves of the khat plant (Catha edulis), a stimulant chewed for centuries in Yemen, Somalia, Eritrea, and Ethiopia (43). The cathinone derivatives found in bath salts are primarily 3,4-methylenedioxypyrovalerone (MDPV), mephedrone, and methylone, which are structurally similar to methamphetamine and MDMA and produce similar stimulant effects as well as psychotic effects in susceptible individuals (43). Other emerging designer drugs of which we have as yet little medical knowledge include methoxetamine, a “legal” ketamine analog that is an NMDA receptor antagonist that causes hallucinations (43), and the piperazine derivatives that have central serotonergic and amphetamine-like effects (43). These new arrivals to the club drug scene are becoming popular in Europe and other parts of the world such as New Zealand and Eastern Europe and are advertised as “research chemicals” that can be bought on the Internet.

Salvia divinorum, an herb in the mint family that has become more popular of late, is native to Southern Mexico and is used for its mind-altering effects. The plant is smoked, as yet considered legal in most of the United States and is widely available via the Internet or local “head shops.” Psychotropic properties are due to the chemical salvinorin A, a kappa opiate receptor agonist. Effects include hallucinations, visual distortions, perceptual disturbances, anxiety, confusion, and dysphoria (43). Some users describe synesthesia (i.e., hearing colors or smelling sounds), while others endorse an “out of body experience.” Effects can last as long as 24 hours (43).

Synthetic cannabinoids such as “Spice” and “K2” consist of inert plant material sprayed with chemical compounds originally designed by pharmaceutical companies as research drugs with cannabinoid receptor agonist activity (43,44). In early 2011, the U.S. Drug Enforcement Agency gave Schedule I status to several of the synthetic cannabinoids including JWH-018, JWH-073, CP 47, 497, and homologues (43). These new arrivals to the club drug scene are becoming popular in Europe and other parts of the world such as New Zealand and Eastern Europe and are advertised as “research chemicals” that can be bought on the Internet.

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**DIFFERENTIAL DIAGNOSIS AND TREATMENT**

Diagnosing and treating a substance-induced mental disorder is very much dependent on the attitude and training of the clinician. Is he or she attuned to the prevalence of alcohol and drug use? Without this awareness, there is less inclination to search for the problem. Does the clinician think that it is relevant to the current problem to take the time to elicit an alcohol and drug use history? Has the clinician received adequate training to counteract the therapeutic nihilism
acquired during medical school and residency training? Is he or she adversely affected by the distortions and denial that are exhibited by many alcoholics and drug addicts? Does the clinician routinely seek corroboration of an alcohol and drug use history from family or friends of the patient? Will the clinician order a drug screen? All of these questions hint at behaviors that can make the diagnosis more apparent or allow it to elude identification. Making the diagnosis of a substance use disorder (SUD) is the first step in the differential diagnosis and treatment of a substance-related problem. In the second step, the substance-induced symptoms must be differentiated from the symptoms of major psychiatric disorders. Finally, the substance-induced disorders must be differentiated from the dual disorders: substance abuse or dependence combined with a comorbid, nonsubstance Axis I disorder. The DSM-IV contains five criteria for substance-induced mood disorders:

1. A prominent and persistent disturbance in mood predominates, characterized by (a) a depressed mood or markedly diminished interest or pleasure in activities or (b) an elevated, expansive, or irritable mood.
2. There is evidence from the history, physical examination, or laboratory findings that the symptoms developed during or within a month after substance intoxication or withdrawal, or medication use is etiologically related to the mood disturbance.
3. The disturbance is not better explained by a mood disorder.
4. The disturbance did not occur exclusively during a delirium.
5. The symptoms cause clinically significant distress or impairment.

### Mood Disorders

Mood disorders may be the most common substance-induced disorders that clinicians need to consider in arriving at a differential diagnosis. It is important to consider, and make, the substance use diagnosis whenever it is pertinent. There are some guidelines that can help with these diagnoses. Because of denial, the patient may not understand what is happening in his or her life. If the clinician is aware of the prevalence of addictive disorders and the ways in which such disorders typically present, he or she is more likely to take a careful history and to seek confirmation of the history from collateral informants, especially family and friends, but also other health care professionals. Establishing whether there is a relationship between the use of psychoactive substances and the symptoms prominent at the moment is a crucial step. Chronic use of alcohol, sedatives, and opiates can cause depressed mood, as can withdrawal from stimulants and sedatives. Exploring the mood during periods of sustained abstinence from all depressant drugs is critical.

### Anxiety Disorders

For the substance-induced anxiety disorders, the criteria are almost identical. However, the first is different: Prominent anxiety, panic attacks, obsessions, or compulsions predominate. The remaining four criteria are the same as for mood disorder.

In making the diagnosis of SUD, it is helpful to order a drug screen. Even if the results come back hours after the clinical decision is made, they can be used to confirm the presence of a substance despite the patient's denial. Such a screen also can clarify the history in some future episode. Sometimes, patients report part of their history, but not all. For example, it may be useful to know that both alcohol and cocaine were used by a depressed patient. Although either substance can induce symptoms of anxiety (or depression), a slightly different treatment plan may be necessary for a patient dependent on both. A drug screen may be equally critical to the diagnosis of an SIPD especially since the subject may not have accurate knowledge of what substances were ingested.

### Substance-Induced Psychotic Disorders

Again, the criteria for SIPD in the DSM-IV are similar to diagnosis of substance-induced mood and anxiety disorders. Hallucinations or delusions must be prominent and are not counted if the individual has insight into the substance-induced nature of his or her cognitive problems. Among young people, substance use is common in first-episode psychosis, and the differentiation of SIPD from PPD is challenging and requires a careful psychiatric and substance use history, drug screen, and collateral information from family and friends. As yet, there has been surprisingly little research published on differentiating SIPD from PPD (47), and an even smaller amount has been published on the treatment, course, and prognosis of SIPD (48). The differential diagnosis of methamphetamine psychosis and PPD is difficult, and a recent multisite international study has concluded that the severity of psychotnic symptoms, including the negative ones, observed in methamphetamine psychotic and schizophrenic patients is almost the same (47). Fraser et al. (16) using the PRISM-IV studied individuals with first-episode psychosis admitted to a psychiatric ward and found that 56% had SIPD. Those with drug-induced psychosis had higher rates of substance use and SUD, had higher levels of insight, were more likely to have a forensic and trauma history, and had more severe hostility and anxiety symptoms compared to those with non-drug-induced psychosis. They reported that after logistic regression analysis, a family history of psychosis, trauma history, and current cannabis dependence were significant predictors for SIPD (16). A 12-month follow-up done by Caton et al. (18) found that those with SIPD were more likely to achieve psychotic remission compared to those with PPDs. Ali et al. (48) in a recent Multisite World Health Organization–sponsored study on SIPD reported on non-evidence-based treatment recommendations for SIPD and concluded that benzodiazepines to manage acute agitation during intoxication followed by atypical antipsychotic administration should the psychosis be severe or not resolve within 1 to 2 weeks after cessation of drug use. The time limit of substance-induced psychotic symptoms that must
persist before a PPD should be diagnosed is unclear as yet (18,47,48). It is however clear that most SIPD is short lived and resolves within a few days to 1 or 2 weeks with the exception of methamphetamine psychosis since persistent psychotic symptoms after heavy and/or long-term use has been well documented in several studies (17,19,20,47,48).

Case 1

Mr. B is a 46-year-old divorced white man who works as a house painter. He came to the emergency department because of suicidal ideas, which frightened him. He had become increasingly depressed over the preceding month and was afraid that he was “going crazy.” He had experienced episodes of depression over the preceding 7 years (since his divorce), but the episodes had not lasted more than a day or two. He also had experienced fleeting suicidal ideas, but had not hurt himself. In the past year, he occasionally had sat with his gun and considered ending it all. At those times, he felt momentarily hopeless. The suicidal and hopeless thoughts lasted for an evening, but were not continuously present for more than a day. He had never been treated psychiatrically for depression. The clinician gathers information suggestive of a depressive syndrome of some kind and must determine whether there are any organic causes, the most common of which would be substance-induced disorder. Mr. B has been hospitalized once, 4 years ago, to be detoxified from alcohol, but he received no treatment for alcoholism after the detoxification. Recently, his drinking has increased to about a case of beer a day. He reports that the alcohol use is the only way he can cope with his depression. He denies any loss of control, but admits to two arrests for driving under the influence (DUI) over the preceding 10 years. He is experiencing difficulty in getting to work on time since becoming depressed and is in trouble with his supervisor. He denies morning shakes and says that he never has experienced delirium tremens. Mr. B admits that his ex-wife complained about his drinking. He has had only one period of abstinence for more than a year, while on probation for his second DUI. He felt well during that time. He developed the depressive symptoms in his late 30s, whereas his heavy drinking began in his early 20s. He denies any ongoing medical problems or thyroid problems. He has had some weight loss in the past month because he has not been eating regularly with his heavy drinking, and he has experienced some nausea in the mornings, which made eating breakfast difficult. He denies any use of sedatives, barbiturates, cocaine, or opiates. On mental status examination, Mr. B is found to be a middle-aged white man, who looks more like 55 than 46 years old. He is thin, looks depressed, and smells of alcohol. He is vague about some details and specific about others. He is oriented to person, place, date, and purpose. He is tearful at some times and anxious at others. He seems bewildered about his predicament. He denies having problems with alcohol. He has suicidal ideas about shooting himself, but does not seem motivated to do so. He denies hallucinations and obsessions. He denies any manic episodes. His blood alcohol concentration is 200 mg%. A drug toxicology screen is negative for benzodiazepines, opiates, barbiturates, and cocaine.

Diagnostic Issues

Is there a reason to consider a diagnosis of alcohol dependence or abuse for Mr. B? Is depression a prominent symptom? Is there a reason to connect the depressive symptoms to alcohol or drug use or withdrawal? Is the intensity of depression more severe than is usually found with alcohol intoxication alone? Is the depressive mood better explained by a mood disorder? Did the depressed mood occur during a delirium?

Diagnostic Considerations

At this point, the clinician has enough information to diagnose alcohol dependence. Mr. B exhibits tolerance, alcohol withdrawal, use despite adverse consequences, impairment of personal relationships, and, possibly, impairment of occupational function, all related to his use of alcohol. His mood disturbance is prominent and is more severe than that experienced by social drinkers and most alcoholics. His depressive symptoms seem sufficiently severe to suggest major depression; however, there is no evidence of depression at the times that Mr. B is not drinking heavily, suggesting alcohol-induced depressive disorder. The alcohol dependence seems to be primary; that is, it began before the depressive symptoms. This sequence of symptoms also suggests alcohol-induced depressive disorder. It is possible that Mr. B has two independent disorders, alcohol dependence and major depression; however, there is no evidence of that at present. Finally, there is no evidence of a delirium.

Treatment Issues

Safety issues involve ongoing evaluation of the diagnosis, use of medications, and psychosocial therapy.

Treatment Considerations

A trial of abstinence is called for in a safe environment with a lot of support. The clinical challenge is to find a safe environment. The risk of alcohol withdrawal delirium and seizures is minimal, suggesting that Mr. B could be managed as an outpatient. However, there are other considerations in this decision. The ASAM patient placement criteria for the treatment of substance-related disorders, 2nd edition—revised (ASAM PPC-2R) (49) encourage the physician to evaluate the patient’s status in six different dimensions. The first is the potential for withdrawal. The second and third are the presence of medical and psychiatric comorbidities. (The seriousness of Mr. B’s suicidality, as well as the degree of his anergy and inability to mobilize because of depression, is relevant in this case. The medical comorbidity is minor [possibly gastritis] and should improve with abstinence.) The fourth dimension is the patient’s readiness for change. Mr. B appears to seek treatment; however, he may balk at inpatient or outpatient treatment, making his cooperation a major issue. The final two dimensions—the potential for relapse and the presence or absence of a supportive environment—are very pertinent to this patient. If Mr. B has no supportive friends
or family who can watch over him and be sure that he gets to therapy sessions, or to the emergency department if his condition worsens, then outpatient therapy becomes risky, particularly given the patient’s suicidal ideas. The patient’s potential for relapse, including his motivation for abstinence, craving for alcohol while abstinent, and history of prior attempts to quit, is crucial in determining the viability of outpatient treatment. Alcohol-induced depression should remit over the first 2 to 3 weeks of abstinence. Careful follow-up during this period is very important with outpatient treatment because of the severity of Mr. B’s symptoms and the possibility that the correct diagnosis is not alcohol-induced depression. However, if he cannot or will not stay sober as an outpatient, it is likely that he will remain depressed or become even more depressed. At this point, residential treatment is essential to break the cycle of addiction and allow the patient’s mood to improve. If his mood does not improve with abstinence, a major depression should be considered, and the patient should be given appropriate antidepressant treatment. Anticraving medications such as naltrexone, acamprosate and injectible naltrexone can be very helpful when the patient is cooperative, somewhat open to the idea of abstinence from alcohol, and willing to engage in some kind of psychosocial follow-up. The same is true of disulfiram. Patients with a firm commitment to sobriety may not need the assistance of such medications. Patients with a strong connection to Alcoholics Anonymous may not be motivated to take such medications because they believe that medications are not appropriate for sobriety; however, Alcoholics Anonymous produces a pamphlet that is quite supportive of both psychiatric care and the use of psychiatric medications.

Case 2

Mr. M is a 64-year-old African American veteran who is divorced and unemployed. He came to the evaluation area at 9:00 am because of a long-standing problem sleeping that has worsened over the past 2 months. He has been drinking more alcohol to fall asleep, but has been waking after only a few hours of sleep. He experiences hand tremors in the mornings, which resolve with a few shots of whiskey. He has become jumpy and irritable and tends to isolate himself from his friends.

Option A

Mr. M has no history of withdrawal seizures or withdrawal hallucinations. He has no history of panic attacks, chronic anxiety, or traumatic life events. His vital signs include blood pressure 148/90, pulse 96, temperature 98.6°F, and respirations 16. Mr. M was detoxified from alcohol within the past year, but refused to enter outpatient treatment. He complained that he was “not going to let the doctors treat him like a ‘guinea pig’ on the detoxification unit” and that “they just tried to lock up a Black man these days.”

Diagnostic Issues

Is there a prominent symptom? If so, is this symptom related to drinking? Is there any evidence of other drug use? Is this explained better by another DSM disorder? Did the symptoms occur during a delirium? Has this disorder caused symptoms beyond what normally is experienced during alcohol intoxication or withdrawal?

Clinical Considerations

At this point, the information available suggests alcohol withdrawal. There also is information suggesting a sleep disturbance that may be alcohol induced or related to alcohol withdrawal. Because of the cultural tensions already reported, an effort should be made to rule out an anxiety disorder, as Mr. M may be reluctant to report anxiety symptoms. Efforts should be made to rule out other organic causes of his anxiety and agitation, such as hyperthyroidism, stimulant intoxication, caffeineism, or medication-induced anxiety. A drug toxicology screen would be helpful to rule out stimulant intoxication, caffeineism, and opiate withdrawal. A phone call to a family member or friend may add confidence to the diagnosis and rule out chronic anxiety and paranoid disorders. The symptoms do not seem excessive for alcohol withdrawal, which is the likely diagnosis, given the available information.

Option B

Mr. M has no history of withdrawal seizures or withdrawal hallucinations. He has a history of panic attacks, which began after he returned from military service in Vietnam in 1971. He had been in combat for 6 months and had seen a lot of action, which he does not wish to discuss. He becomes more agitated as he talks about combat, eventually cutting off the conversation. He is angry about the way he was treated when he returned to the United States and reports a difficult transition to civilian life. He demonstrates recent heavy drinking, nightmares about combat, flashbacks, relationship problems, and a significant startle reaction to loud noises. He complains of racist treatment by white soldiers and officers in Vietnam and claims that his white superiors sent him on suicide missions.

Diagnostic Issues

Is there a prominent symptom? Is there a causal relationship with the drinking? Is there any drug use that could account for the disorder? Is this better explained by another DSM disorder? Did the symptoms occur during a delirium? Are the symptoms excessive for alcohol intoxication or withdrawal?

Diagnostic Considerations

The patient certainly evidences anxiety as a prominent symptom. However, it still is important to look for a substance dependence disorder and a substance-induced anxiety disorder. It is necessary to clarify the diagnosis of alcohol withdrawal. This patient seems to have a posttraumatic stress disorder (PTSD), alcohol dependence, alcohol withdrawal, and, possibly, substance-induced anxiety disorder. Because the engagement of this patient in treatment may depend on which combination of problems he has, it is important for the clinician to obtain collateral information and a toxicology screen. Because the patient’s denial may be convincing, it is not sufficient to dismiss substance dependence or a substance-induced disorder based on his history. If the patient’s report is the only
information available, the clinician must make a treatment
decision, while remaining aware that new information could
change the diagnosis and treatment plan. Mr. M’s vital signs
should be monitored to pick up signs of severe alcohol with-
drawal. A toxicology screen should be obtained to check for
drugs that can cause agitation or anxiety. A chronic history
of anxiety, tension, nightmares, and the like is excessive for
alcohol-induced anxiety or alcohol withdrawal alone. With
this information, the clinician may consider both PTSD and
alcohol withdrawal. It will be difficult to diagnose alcohol-
induced anxiety in combination with PTSD unless there is a
clear history of anxiety that recently has worsened without a
psychosocial trigger for the PTSD. More commonly, the clin-
ian will see an acute improvement with sobriety as a sign that
there was an alcohol-induced anxiety component.

**Treatment Issues**

Mr. M needs to be engaged in a detoxification setting (fol-
lowed by an alcohol rehabilitation setting), and his denial
needs management in order to expand his awareness
beyond the PTSD symptoms. His claims of racism should
be addressed as part of the engagement and assessment pro-
cesses. Mr. M’s anxiety should be managed without using
benzodiazepines. His sleep disorder should be managed and
the process of relapse prevention initiated.

**Treatment Considerations**

Treatment must be conceptualized in stages. The first stage
is detoxification from alcohol (and any other drugs that may
be present). With this patient, detoxification can be managed
through a careful outpatient regimen if he is able to remain
abstinent. If abstinence seems unlikely, if the patient fails at
outpatient detoxification, or if a comorbid problem arises
that cannot be monitored safely on an outpatient basis, then
residential detoxification must be considered. The next stage
is the maintenance of sobriety, a stabilization phase. During
this stage, the clinician should monitor Mr. M’s abstinence
and observe the course of the anxiety symptoms. A relapse
can increase his anxiety symptoms, which would interfere
with attainment of the treatment goals. Such a relapse
would require a treatment strategy that focuses on manage-
ment of denial, motivation, and relapse prevention. If, dur-
ing abstinence, the anxiety symptoms increase or stay the
same, the PTSD probably is severe, and medication will be
needed. If the anxiety symptoms diminish or are manageable
without medication, then counseling may be sufficient. The
use of benzodiazepines in alcoholics after detoxification is
achieved is controversial, even in the face of severe anxiety.
The anxiolytic properties of benzodiazepines are sustained
over time; however, many alcohol-dependent folks are sus-
cceptible to developing subsequent dependence on sedative–
hypnotics. Moreover, craving for drugs is greatest when the
drug, or a similar drug, is being used. Thus, there always is
a concern that benzodiazepines will stimulate the desire for
alcohol. Drinking in addition to benzodiazepine use can lead
to an “out of control” binge, as well as intoxicated behaviors,
which could exaggerate the PTSD symptoms of anxiety and
agitation. Disulfiram is a possible safeguard to prevent alco-
hol use during outpatient treatment, but the patient must
be willing to collaborate (i.e., take it regularly) if it is to be
effective. The use of antipanic medications such as the selec-
tive serotonin reuptake inhibitors and other antidepressants
would be a safer strategy. The same problem is encountered
with the complaints about insomnia. Avoiding sedatives is
important. Use of sedating antidepressants like trazodone
or mirtazapine can be very effective and avoids the abuse
potential of other sedative drugs.

**Option C**

Mr. M has no history of withdrawal seizures or withdrawal
hallucinations. He has had an episode (the day before com-
ing to the emergency department) in which he became
frightened, felt short of breath, felt his heart pounding, and
worried that he was having a heart attack. This episode was
not the first time he has experienced such an attack; he had
one 6 months earlier when he quit drinking. He had gone
to the emergency room the first time this happened, 5 years
ago. The physician then had checked his heart and told
him that he was having a nervous attack, not a heart attack.
Although Mr. M feels stressed at times, he does not have
these attacks regularly. When he stopped his drinking for a
year, he felt well and does not recall having a spell during
that time. He denies any major, traumatic life events. He was
embarrassed to be worried about these anxiety spells, but
also fearful that he might have been having a heart attack.

**Diagnostic Issues**

Is there a prominent symptom? Is this symptom related to
drinking? Is the symptom better explained by another DSM
diagnosis? Is the symptom in excess of the symptoms nor-
mally encountered during intoxication or withdrawal?

**Diagnostic Considerations**

There is a prominent symptom of anxiety in this case, and
it appears to occur only with drinking. The clinician must
think about alcohol dependence, alcohol withdrawal, and
alcohol withdrawal–induced panic disorder. The major anx-
xiety disorders should be ruled out, as should PTSD (although
certain events, like sexual abuse, may be denied initially). Some attempt to rule out cardiac disease as a cause of the
chest pain would be important. Organic causes of anxiety
also should be ruled out. It is possible that the patient has
a panic disorder that is in a prodromal phase, but this is
not the most likely diagnosis. Although patients frequently
experience anxiety during alcohol withdrawal, they usually
do not experience panic attacks or do they typically go to
the emergency department in a panic.

**Treatment Issues**

Mr. M should be engaged in a detoxification program and
his level of denial and motivation for treatment evaluated.
The denial needs to be evaluated and the problem redef-
ined as alcoholism, not panic or heart disease. The pos-
sibility of a comorbid anxiety disorder should be explored.
The patient then needs to be engaged in an alcohol
rehabilitation program. Medications that will enhance the likelihood of sobriety should be considered. The patient should be evaluated for relapse triggers and referred for relapse prevention as appropriate.

**Treatment Considerations**

Treatment should be designed to detoxify Mr. M safely from alcohol, to explore dependence on other drugs, and to keep him sober long enough to determine whether the anxiety disorder abates with sobriety, as an alcohol withdrawal-induced anxiety disorder would. The patient’s denial and motivation are important because he must understand the connection between his drinking and his panic. If his awareness of this connection is minimal, then the treatment may have to occur in a setting in which chest pain or panic is the primary focus. He may not accept the focus on his drinking. If this is the case, then referral to a rehabilitation program, either inpatient or outpatient, may not be possible at this time. Ongoing monitoring and working with his denial would be necessary before such a referral could be made. Benzodiazepines would be appropriate only during detoxification; medications that promote sobriety, such as disulfiram or naltrexone, would be appropriate if Mr. M is motivated and able to cooperate. Antipanic medications like the selective serotonin reuptake inhibitors probably would be used if the panic attacks persist with sobriety.

**Case 3**

Mr. S, a 20-year-old man, was brought by the police to the local emergency department after threatening his partner with a knife. He presented with acute agitation, hostility, paranoia, and delusions of reference. He expressed fear that his partner was a “necrophiliac” and a serial murderer, and over the past month, S had begun to arm himself with a knife in order to protect himself and others from possible attack. Other symptoms included frequent anger episodes, increased libido, insomnia, tachycardia, weight loss, and anxiety. On physical exam, he was found to have scattered scaring and sores on his face, arms, and legs. The patient reported having used methamphetamine on many occasions over the past year, smoked, and later injected over weekends to “party with friends.” During his partying experiences, he related repeated unsafe sexual practices, and he requested an HIV test. He described frequent withdrawal effects after a weekend of partying that included hypersomnia, increase appetite, poor concentration, and dysphoria that would last several days and culminate in craving until his next methamphetamine binge. In addition to methamphetamine use, the patient also reported frequent alcohol and marijuana use but denied abuse or withdrawal effects from those substances, and he denied opiate, cocaine, or club drug use.

**Diagnostic Considerations**

This case appears to be methamphetamine-induced psychotic disorder. However, given his young age, the possibility of a PPD cannot be ruled out. A drug toxicology screen should be obtained to rule out other drugs despite his denial of use since street drugs are often contaminated with other compounds. He appears delusional and dangerous with very poor insight as he has armed himself with a knife over the last month and believes his partner may be trying to kill him. Hostility is a frequent symptom of methamphetamine psychosis (50) but is also common in untreated paranoid psychosis. He does not appear to have negative symptoms of schizophrenia, but there may have been a short prodromal period of increasing delusional and paranoid thoughts, which is common for people who become psychotic from frequent use of methamphetamine. He also appears to have manic-like agitation, mood lability, and excessive sexual behavior accompanying his insomnia alternating with depressive periods and hypersomnia. This is characteristic of chronic stimulant users who binge and then withdraw. His weight loss, tachycardia, and mild hypertension are probably effects of the stimulant, but a thyroid disorder should be ruled out. His skin lesions are likely due to “meth mites” or formication and the chronic skin picking frequently seen in those who abuse methamphetamine. However, the lesions may also represent a parasitic infestation such as lice, scabies, or bed bugs or a sexually transmitted disease (STD) such as a rash or chancre associated with syphilis since he has had indiscriminate and unsafe sexual behavior that is frequently associated with recreational methamphetamine use. An HIV test as well as other STD tests should be done.

**Treatment Issues**

This patient is frightened but also hostile and delusional with poor insight and threatening behavior. He also has intense craving associated with his withdrawal periods, which likely will prompt him to leave against medical advice. Involuntary psychiatric hospitalization may be required as well as acute treatment of the agitation and psychosis with benzodiazepines and atypical antipsychotics. Mr. S will need extensive education about the origin of his psychosis followed by inpatient and then outpatient addiction treatment for relapse prevention. His partner should be warned if homicidal ideation is present and advised to seek a protective order. He will also need close follow-up after hospitalization to ensure that his paranoia attenuates with abstinence and antipsychotic treatment.

**Treatment Considerations**

Prognosis will depend primarily on his motivation and success in addiction treatment, and it is essential that he understand the role methamphetamine has played in his decompensation and the likelihood of worsening and prolonged psychosis should he relapse. If psychosis persists despite abstinence, a PPD must be considered. Subspecialized culturally appropriate addiction treatment for sexual minorities with methamphetamine dependence...
is available in most large urban areas and can include education about sexual addictions. Twelve-step groups such as "Crystal Meth Anonymous" and "Strength Over Speed" are very helpful if available, but "gay friendly" AA and NA groups will also suffice. As yet, there is no pharmacologic treatment for methamphetamine dependance.

Case 4

Ms. A is a 35-year-old, white divorced woman who came to the emergency department because of suicidal feelings. She reported feeling very despondent that day, with suicidal ideation, and thought that she needed to be admitted to the hospital to keep her safe. On questioning, Ms. A admits that she smokes crack cocaine, about $100 worth at a time. She recently came off a 4-day binge of crack use. She takes 4 to 6 drinks of vodka per day when she uses crack and also has used marijuana.

Diagnostic Issues

Is there a prominent symptom? Is this symptom related to drug use? Is this situation better explained by another DSM diagnosis? Did this situation occur exclusively during a delirium? Is this symptom more severe than usually encountered with intoxication or withdrawal?

Diagnostic Considerations

Ms. A has a prominent mood disturbance, which brought her to the emergency department. It is consistent with chronic cocaine use and temporally related to her recent binge. Although her history is not consistent with a primary mood disorder, it is important to evaluate the possibility. A careful history of mood swings also would be important. If she has been using crack for years, the clinician can expect that she has experienced transient episodes (never more than a few days) of intense depression, even suicidality, with the cessation of cocaine use. Because of the recent onset of this depression, it is not likely to be the result of metabolic problems; however, addicted individuals are not always aware of subtle changes in their bodies, which may be obscured by intoxication. A screening battery is recommended, because nutritional deficiencies often are found and, not infrequently, viral hepatitis (B or C). Unsafe sexual practices and needle sharing make HIV disease a concern. More relevant, the mood disturbance could be alcohol induced; however, this situation should not coincide with cocaine cessation. The cocaine-induced depression should last only a day or two, whereas alcohol-induced depression likely would last a few days longer with sobriety. Some alcoholics experience marked depression with suicidality during intoxication, which clears with sobriety. Although it is tempting to dismiss substance-induced depression as less significant than an MDD because the former resolves so quickly, it is important to remember that these episodes are frightening to the individual, that some people make serious suicide attempts, and that a few actually kill themselves. At this point, it appears that the patient has a cocaine withdrawal–induced mood disorder. It would be wise to obtain a toxicology screen to rule out benzodiazepine- or opiate-induced depression. With the history of depression, the clinician should look for manic episodes. Without a careful history of cocaine use and its relationship to the experience of intense moods, up or down, it would be easy to think of manic–depressive illness. Another diagnostic consideration is the possibility that Ms. A has had a social crisis and is homeless. The report of suicidality could be exaggerated to gain admission to housing or hospital. In such a case, there may be a pattern of similar behavior.

Treatment Issues

Patient safety and suicidality are the primary issues here. Ms. A needs to be engaged in drug rehabilitation, her denial managed, and her motivation for abstinence enhanced. She needs to be engaged in a comprehensive assessment, with her relapse triggers assessed and relapse prevention initiated as needed.

Treatment Considerations

Safety is the first treatment issue in this case because of the patient's depression (suicidality) and the possibility of alcohol withdrawal (delirium tremens). The clinician must assess the severity of the suicidal impulse and the social supports available before deciding whether a residential setting is appropriate. The clinician should assess what type of suicidal thoughts the person is having, whether he or she has formulated a plan to carry out the idea, whether he or she has the means to complete the plan, whether he or she has made prior attempts and, if so, if he or she was serious, whether there are other alternatives, and whether the patient is very agitated. A cocaine-induced depression usually is transient when abstinence ensues. Continued cocaine use sustains the cycle of addiction and depression. Outpatient treatment is viable only if the patient can refrain from using drugs and alcohol, and the ability to do so often depends on the degree of support in the patient's environment. Safety from alcohol withdrawal is a potential issue that should be considered; however, it is unlikely that a dangerous withdrawal will occur unless the patient has had delirium tremens or seizures in the past. Ongoing monitoring is the best precaution and can be handled on an inpatient, as well as an outpatient, basis, if the patient is cooperative. Assessing the patient's reliability (ability to follow through) may be difficult if she is previously unknown to the clinician. A variety of factors enter into this assessment, including motivation, denial, awareness, craving, relapse triggers, and availability of a supportive environment. Such an assessment is part of the safety management for this patient. Engagement in a drug and alcohol treatment program will depend on the patient's denial, motivation, and awareness of the centrality of drugs and alcohol, as well as the pull of other social relationships such as children, significant others, and family members who may be dependent on Ms. A. Attention to these psychosocial issues may be the key to engagement. Focusing on a comprehensive assessment, including relapse triggers,
can be the way to engage a difficult, ambivalent patient. Inclusion of the family sometimes facilitates engagement, as does admission to a treatment program if the patient is anxious about further drug use and its sequelae. If an individual has been through rehabilitation programs in the past, has relapsed, and has a commitment to abstinence as well as a capacity to remain abstinent, then a focus on relapse prevention may be the appropriate intervention. Such intervention remains part of the art of medicine and hinges on the physician’s style of practice, local resources, and managed care practices. It is a complex challenge each time and must be individualized to each situation.

**CONCLUSIONS**

The substance-induced mental disorders are common illnesses that often are associated with (but are not limited to) substance dependence. Although they frequently are short lived, these disorders are by no means clinically insignificant. Serious self-injury is reported with the substance-induced mood disorders, and safety is an important clinical issue. This situation can present a clinical dilemma in determining the proper level of care. Most patients with substance-induced mental disorders can be diverted away from traditional psychiatric inpatient treatment, either to dual diagnosis units or to inpatient or outpatient addiction treatment programs in which adequate assessment and appropriate treatment are available. Dual diagnosis clinics and residential units that specialize in substance-dependent patients who have a comorbid psychiatric illness play an important role when there is diagnostic confusion or when the patient does not respond (or has not responded in the past) to routine psychiatric treatment. Confusion about the diagnosis can delay interventions; therefore, achieving clarification through a comprehensive evaluation is the first order of business, after safety is addressed. Although abstinence is a critical factor in recovery from a substance-induced mental disorder, it is not always the only factor. Regular psychosocial treatments for substance dependence are relevant so long as the patient is behaviorally manageable and not psychotic or delirious. When the patient’s behavior is unsafe or wild, a psychiatric unit may be necessary until the patient’s behavior is less risky. If a specialized inpatient unit for dual diagnosis is available and can manage the patient’s behavior with seclusion, restraints, psychotropic medications, or a locked unit, it may be the best choice. Such patient–treatment matching should be done on an individual basis, depending on the patient’s needs, the resources available, and the skills and preferences of the clinicians involved.

**REFERENCES**


