stimulant and euphoric effects and remains a common practice in east Africa that has persisted for centuries. Cathinone is structurally similar to amphetamine, and mephedrone is structurally similar to methamphetamine. Cathinones, like amphetamines, inhibit dopamine, serotonin, and norepinephrine transporters to varying degrees, and this probably accounts for variations in the behavioral effects observed. The effects of cathinone derivatives are often described as similar to the effects of MDMA or Ecstasy. Synthetic cathinones can be inhaled, snorted, injected, or taken orally. These drugs may be taken repeatedly over several hours in episodes lasting for hours or days. The onset of effects after oral ingestion is relatively rapid for MDPV (15–30 min) and slightly slower for mephedrone and methylone (30–45 min). The duration of action varies from 2 to 5 or 7 h. After mephedrone inhalation, effects occur within minutes and only last for 1 h or less, but mood changes may persist for several days. Evaluation of the neurotoxic effects of prolonged synthetic cathinone abuse is just beginning, and the long-term consequences are unknown.

The reported positive subjective effects of synthetic cathinones include euphoria, improved energy, alertness, sociability, and increased sensitivity to music and other sensory experiences. The reported negative subjective effects include agitation, visual and auditory hallucinations, anxiety and panic attacks, paranoid delusions, disorientation, depression, and suicidal ideation. Observers report irritability, aggression, violent behavior, tremors, and seizures. Medical evidence of adverse effects includes cardiovascular dysfunction and cardiac arrest, hypertension, hyperthermia, nausea and vomiting, and anorexia. There is no specific antagonist for synthetic cathinone intoxication. Patients with severe hyperthermia, seizures, and arrhythmia are medical emergencies and should be treated in a hospital. Sedation with benzodiazepines can be useful for managing agitation, seizures, aggression, and other related symptoms. Antipsychotic medications may be necessary for management of severe and persistent psychiatric symptoms.

LYSERGIC ACID DIETHYLAMIDE (LSD)

Discovery of the psychedelic effects of LSD led to an epidemic of LSD abuse during the 1960s. Imposition of stringent constraints on the manufacture and distribution of LSD (classified as a Schedule I substance by the U.S. Food and Drug Administration [FDA]) and public recognition that psychedelic experiences induced by LSD were a health hazard have resulted in a reduction in LSD abuse. LSD still remains popular among adolescents and young adults, and there are indications that LSD use among young persons has been increasing in some areas in the United States. In 2011, an estimated 358,000 persons used LSD, whereas 200,000 and 271,000 persons reported LSD use in 2003 and 2007, respectively.

LSD is a very potent hallucinogen; oral doses as low as 20 μg may induce profound psychological and physiologic effects. Tachycardia, hypertension, pupillary dilation, tremor, and hyperpyrexia occur within minutes following oral administration of 0.5–2 μg/kg. A variety of bizarre and often conflicting perceptual and mood changes, including visual illusions, synesthesias, and extreme lability of mood, usually occur within 30 min after LSD intake. These effects of LSD may persist for 12–18 h, even though the half-life of the drug is only 3 h.

Emergency ward visits involving LSD totaled nearly 5000 in 2011. The most frequent acute medical emergency associated with LSD use is a panic episode (the "bad trip"), which may persist up to 24 h. Management of this problem is best accomplished by supportive reassurance ("talking down") and, if necessary, administration of small doses of anxiolytic drugs. Adverse consequences of chronic LSD use include an enhanced risk for schizophreniform psychosis and derangements in memory function, problem solving, and abstract thinking. Treatment of these disorders is best carried out in specialized psychiatric facilities.

Tolerance develops rapidly for LSD-induced changes in psychological function when the drug is used one or more times per day for >4 days. Abrupt abstinence following continued use does not produce withdrawal signs or symptoms. There have been no clinical reports of death caused by the direct effects of LSD.

PHENCYCLIDINE (PCP)

PCP, a cyclohexylamine derivative, is widely used in veterinary medicine to briefly immobilize large animals and is sometimes described as a dissociative anesthetic. PCP binds to ionotropic *N*-methyl-D-aspartate (NMDA) receptors in the nervous system, blocking ion current through these channels. PCP is easily synthesized and is abused primarily by young people and polydrug users. It is used orally, by smoking, by snorting, or by IV injection. It is also used as an adulterant in THC, LSD, amphetamine, or cocaine. The most common street preparation, *angel dust*, is a white granular powder that contains 50–100% of the drug. Low doses (5 mg) produce agitation, excitement, impaired motor coordination, dysarthria, and analgesia. Physical signs of intoxication may include horizontal or vertical nystagmus, flushing, diaphoresis, and hyperacusis. Behavioral changes include distortions of body image, disorganization of thinking, and feelings of estrangement. Higher doses of PCP (5–10 mg) may produce profuse salivation, vomiting, myoclonus, fever, stupor, or coma. PCP doses of ≥10 mg cause convulsions, opisthotonus, and decerebrate posturing that may be followed by prolonged coma.

In 2011, more than 75,000 emergency ward admissions involved PCP. The diagnosis of PCP overdose is difficult because the patient's initial symptoms (anxiety, paranoia, delusions, and hallucinations) may suggest an acute schizophrenic reaction. Confirmation of PCP use is possible by determination of PCP levels in serum or urine. PCP assays are available at most toxicologic centers. PCP remains in urine for 1–5 days following high-dose intake.

PCP overdose requires emergency life-support measures that may involve treatment of coma, convulsions, and respiratory depression in an intensive care unit. There is no specific antidote or antagonist for PCP. PCP excretion from the body can be enhanced by gastric lavage and acidification of urine. Death from PCP overdose may occur as a consequence of some combination of pharyngeal hypersecretion, hyperthermia, respiratory depression, severe hypertension, seizures, hypertensive encephalopathy, and intracerebral hemorrhage.

Acute psychosis associated with PCP use is a psychiatric emergency because patients may be at high risk for suicide or extreme violence toward others. Phenothiazines should not be used for treatment because these drugs potentiate PCP's anticholinergic effects. Haloperidol (5 mg IM) has been administered on an hourly basis to induce suppression of psychotic behavior. PCP, like LSD and mescaline, produces vasospasm of cerebral arteries at relatively low doses. Chronic PCP use has been shown to induce insomnia, anorexia, severe changes in behavior, and, in some cases, chronic schizophrenia.

SALVIA DIVINORUM

This naturally occurring herb is a recent entry into the spectrum of hallucinogens. Like PCP and ecstasy, this drug can produce profound alterations in mood, hallucinations, and distorted perceptions. This drug is available on the Internet and is known by a variety of names including *magic mint, mystic sage, Mariana Pastora*, and *purple sticky*. The drug was first added to the annual National Surveys on Drug Use and Health in 2006, and its use is increasing. Between 2006 and 2011, the number of estimated users in the United States nearly tripled to more than 5000.

The active ingredient is salvinorin A, a selective kappa opioid receptor agonist that has a range of effects including hallucinations, sedation, analgesia, and depression. The hallucinatory symptoms may be associated with intense anxiety and severe agitation that can be managed with benzodiazepines. Importantly, this kappa opioid receptor agonist does not produce respiratory depression, and no significant change in blood pressure or heart rate was reported in a clinical study with healthy subjects.

Salvinorin A extract or crushed leaves of the *Salvia divinorum* plant can be chewed and absorbed through the buccal membrane or inhaled during smoking. The onset of the acute "high" is within 5–10 min after chewing and 30 s after inhalation. The duration of the effect is relatively brief, usually 15–20 min. However, if the drug is taken with alcohol or other hallucinogens, the duration and intensity of adverse