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REVIEW

Cocaine Revisited

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Insurance companies often test applicants for the presence of cocaine. Because a positive test may automatically preclude further consideration, such cases often never reach the medical director. The negative impact of cocaine use on insurability is reviewed.

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Cocaine is an alkaloid compound found in the leaves of a small tree native to the mountains of northern South America. Leaves are dissolved in hydrochloric acid to make cocaine hydrochloride, which can then be mixed with ether to form freebase cocaine or extracted with sodium bicarbonate and water to make crack cocaine. The name ''crack'' comes from the popping sound this form of cocaine makes when smoked. Production of crack cocaine is much easier and cheaper and thus significantly more profitable. A kilogram of cocaine converted to crack can yield 3 to 8 times the profit of powder cocaine. Smoking either form of cocaine rapidly yields high blood levels similar to those achieved with intravenous use. Generally, intranasal cocaine use yields a peak effect in 15–20 minutes and lasts 60–90 minutes, while smoking or injecting cocaine gives a peak effect in 5–10 minutes and lasts only 30 minutes. Because the user's ''high'' depends on the rate of rise and peak blood level, smoking cocaine produces rapid intense highs that are quickly followed by ''crashes,'' promoting frequent use to avoid these profound ''lows,'' often resulting in binging. Tolerance develops with repeated use, increasing the amount of cocaine needed to produce the same high. Users often add heroin to IV cocaine (a ''speedball''), as the effects from direct IV injection of cocaine may be too strong for most individuals. IV drug abusers in turn have been found to die at 7 times the rate of comparable age groups of non-drug users. Cocaine is metabolized by the liver into benzoylecgonine and ecgonine, which are in turn excreted in the urine. A single dose of cocaine can lead to positive urine drug screens for the next 24–36 hours. When the user also ingests alcohol, as many do, cocaine is metabolized into cocaethylene. This metabolite not only has a longer half-life, but may play a role in the increased risk of sudden death by users of alcohol and cocaine. Indeed, in users of both products, the risk of sudden death is increased 25-fold compared to users of cocaine only.

Cocaine is one of the most popular and dangerous illicit drugs used. The National Household Survey on Drug Abuse (NHSDA) estimates 1.5 million Americans, 0.7% of the population older than age 12, are current cocaine users. In the last decade, there was a

Table 1. 1999 Annual Poison Control Center Data*

Drug	# of Exposures	# of Deaths	% Deaths/Exposure
Cocaine	4286	70	1.6%
Heroin	1669	30	1.8%
Codeine	1669	6	—
Morphine	1218	10	0.8%
Oxycodone	5185	27	0.5%
Methadone	1047	17	1.6%
Benzodiazepines	40,299	65	0.1%
Barbituates	4443	8	0.2%
Methaqualone	38	0	—
Chloral hydrate	305	2	0.6%
Amphetamines	16,684	18	0.1%
LSD	1252	0	—
Marijuana	1930	0	—
Phencyclidine	465	1	0.2%
Gasoline (inhalant)	20,720	1	—
Lighter fluid (inhalant)	4011	1	—

* Numbers above may underestimate drug usage and deaths, as episodes may go unreported.
Source: Hals et al.[1] Reprint permission granted by Thomson American Health Consultants.

37% increase in new cocaine users. This drug remains the most common cause for drug-related emergency visits, and is the number 1 complaint of patients admitted to drug treatment centers. Alcohol is a well recognized factor in motor vehicle accidents, with 40%-70% of traffic fatalities related to its abuse. Yet illicit drug use is likewise highly correlated with traumatic injury and death; indeed, several studies have demonstrated positive illicit drug screens in 40%-80% of major trauma victims, with cocaine being the most common drug of abuse.

COCAINE'S IMPACT

Cardiovascular System

Cocaine use can cause multiple and sometimes lethal dysrhythmias in the user. Cocaine acts as a type 1a sodium channel blocker, like quinidine or procainamide, and prolongs the QT interval by prolonging action potential duration. It may also provoke dysrythmias through direct sympathetic stimulation and ischemia due to cocaine-induced coronary artery vasospasm. Ischemia may result not only via coronary vasoconstriction, but as a result of increased platelet aggregation and thrombus formation leading to myocardial infarction. Cocaine increases workload and demand for oxygen, thereby worsening ischemia, and may have direct myocardial toxicity. Patients with cocaine-associated myocardial infarction may not benefit from thrombolytics to the same degree as non-users experiencing myocardial infarction. Repeated cocaine use accelerates atherosclerotic development, as well as nonatherosclerotic plaque growth.

Central Nervous System

Cocaine-induced seizures are usually single and self-limited, may occur up to 12 hours after use, and typically occur in patients with no seizure history. Patients with a previous history of noncocaine-related seizures can experience repetitive focal motor seizures, and those using large amounts of the drug (2–8 grams) may experience status epilepticus. Cocaine is the most common cause of drug-associated stroke, and causes 90% of strokes in young adults (3rd and 4th decades of life). Stroke associated with co-

caine use can occur with first-time use or in chronic users. It may cause subarachnoid hemorrhage, intracerebral bleed, or ischemic/embolic stroke. There is also a strong connection with psychiatric diagnoses and cocaine abuse.

Respiratory System

Cocaine use has caused increased asthma symptoms, pneumothorax, pneumomediastinum, noncardiac pulmonary edema, and pulmonary hemorrhage/infarction. Upper airway burns can occur and lead to acute epiglottitis. A syndrome labeled ''crack lung'' has been described consisting of fever, dyspnea, hemoptysis, hypoxia, chest pain, infiltrates and sometimes respiratory failure developing 1–12 hours post crack use.

Other

Acute rhabdomyolysis is a well-known complication of cocaine abuse and may be seen in users with no other risk factors (trauma, seizures, hyperthermia, hyper/hypotension, etc); in fact, as many as 30% of such patients will progress to renal failure. Cocaine may also cause direct renal toxicity. It has caused bowel injury and infarction through vasospasm. Cocaine use during pregnancy is associated with a multitude of maternal and fetal complications, including eclampsia, placental abruption, and fetal death or anomalies.

Table 2. Accuracy of Urine Testing

Drug	Time Detectable After Last Use
Marijuana—Single Use	1–7 days
Marijuana—Chronic Use	1–4 weeks
Cocaine	1–4 days
Heroin	1–4 days
Codeine	1–2 days
Morphine	1–3 days
Amphetamine	8–24 hours
Methamphetamine	1–2 days
Alcohol	1 day
Barbituates	2–10 days
Benzodiazepines	2–7 days
Phencyclidine	1–7 days
LSD	8 hours
MDMA, GHB and Inhalants	Not detected

Source: Hals et al.[2] Reprint permission granted by Thomson American Health Consultants.

Tables 1 and 2 place cocaine testing and mortality in the context of other well-known drugs of abuse.

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