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Cocaine Abuse

The abuse of cocaine in sports first attracted national attention in 1986 with the cocaine-related sudden deaths of basketball star Len Bias and football star Don Rogers. The use of cocaine at the collegiate athlete level peaked in the mid-1980s at about 17 percent, and fell dramatically over the ensuing decade to less than two percent. While many factors are at play motivating an athlete to use cocaine, several points are particularly noteworthy. First, cocaine is generally not used to enhance performance. Second, athletes are thought to be vulnerable to recreational substance abuse because of some combination of the following variables: fame, fortune, free time and a feeling of invincibility.

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Current Comments

Report on Cocaine
Abuse in Sports



Cocaine's Effects on the heart

Cocaine's effects on the heart are varied. They include increasing the heart's demand for oxygen, raising blood pressure, causing spasm of the coronary arteries that nourish the heart muscle, producing clots in the coronary arteries, promoting premature arteriosclerosis, and in some cases causing heart attacks, particularly in cigarette smokers. In addition, athletes abusing cocaine may experience palpitations, anxiety, shortness of breath and chest pains. Chronic snorters of cocaine may experience recurrent bouts of sinusitis, perforate their nasal septum, and/or be bothered by a chronic nasal drip.

Written for ACSM
By Gary I. Wadler, M.D., FACSM.

ACSM Current Comment

There are numerous adverse health effects associated with the abuse of cocaine. However, there are certain ones that are of particular concern to athletes. Most notable is sudden death. The mechanism of sudden death may be attributable to rhythm disturbances of the heart, seizures, or bleeding into the brain.



Detection

The metabolites of cocaine are readily detected in the urine at very low concentrations. The International Olympic Committee, the United States Olympic Committee, the NCAA and professional sports leagues specifically ban cocaine use.

Cocaine Abuse in Sports

The abuse of recreational drugs, both licit (e.g., alcohol and nicotine) and illicit (e.g. cocaine, marijuana) by athletes far outweighs their abuse of performance-enhancing drugs (e.g. anabolic steroids). The situation is not unique to athletes; many factors contribute to recreational drug use. They include age, genetics, family influences, peer pressure, education and mental health factors.

An ACSM Report

Cocaine Use

Derived from the coca plant, cocaine is a naturally occurring ecgonine alkaloid. Cocaine hydrochloride is the form of the drug that is inhaled. However, it decomposes when heated. Freebased cocaine and crack cocaine (both lacking the hydrochloride), are heat stable, and therefore are smoked.

Cocaine's effects on the brain are complex and share many similarities with amphetamine. Many of its effects relate to its ability to interfere with the neurotransmitters in the brain, particularly dopamine and norepinephrine.

Most abusers of cocaine inhale, or "snort," the drug. Recreational users may snort as much as one to three grams per week. Smoking crack or free-based cocaine is the fastest way of getting the drug to the brain. Euphoria can occur within three to five minutes and last for less than ten minutes. Addicted individuals can "binge" as frequently as every ten minutes to maintain a sensation of intense euphoria.

Most observers report athletic deterioration with the use of cocaine; there is no evidence of its being a performance enhancer.

Sometimes behavioral changes are observed with athletes, who may show up for practice too early or too late, miss practice altogether, or have altercations with their teammates. Feelings of grandiosity may distort the athlete's perception of actual performance. At normal room temperature, cocaine reduces the body's core temperature. At higher ambient temperatures, especially during exercise, cocaine causes a rise in body temperature, so the athlete exercising in the heat may be susceptible to hyperthermia. This may be related to cocaine-induced changes in the body's thermoregulatory set point and to a decrease in heat loss secondary to peripheral vasoconstriction.