**Cocaine poisoning**

**Cocaine**,is a natural alkaloid found in coca plant, it is a strong [stimulant](https://en.wikipedia.org/wiki/Stimulant) commonly [snorted](https://en.wikipedia.org/wiki/Insufflation_(medicine)), inhaled as smoke, or as a solution injected into a [vein](https://en.wikipedia.org/wiki/Vein). Mental effects may include [loss of contact](https://en.wikipedia.org/wiki/Psychosis) , an [intense feeling of happiness](https://en.wikipedia.org/wiki/Euphoria), or [agitation](https://en.wikipedia.org/wiki/Psychomotor_agitation). Physical symptoms may include a [fast heart rate](https://en.wikipedia.org/wiki/Tachycardia), sweating, and [large pupils](https://en.wikipedia.org/wiki/Mydriasis). High doses can result in very [high blood pressure](https://en.wikipedia.org/wiki/Hypertension) or [body temperature](https://en.wikipedia.org/wiki/Hyperthermia).

it is well absorbed following contact with the oral, nasal, gastrointestinal, rectal, and vaginal mucosa; by the pulmonary alveoli following inhalation; and by intravenous injection. Intravenous and inhalational use of cocaine produces very rapid distribution of cocaine to both the central nervous system and the systemic circulation. Cocaine is rapidly hydrolyzed to its major metabolite, ecgonine methyl ester (EME) by liver and plasma esterases. The biologic half-life of cocaine is 0.5–1.5 hours. A relatively minor amount is excreted unchanged in the urine.

**Pathophysiology**

The effects of cocaine are related to its sympathetic nervous system effects, central nervous system stimulation, and local anesthetic effects. The initial effect of cocaine on the cardiovascular system is vagotonic, producing a transient bradycardia; however, the increased sympathetic stimulation rapidly produces tachycardia and hypertension. In the peripheral nervous system, cocaine inhibits the reuptake of both epinephrine and norepinephrine and stimulates the presynaptic release of norepinephrine. The resulting increased concentration of norepinephrine at the postsynaptic α and β receptors accounts for the stimulatory effects of cocaine. In the central nervous system, the stimulatory effect may be due to enhance the release of norepinephrine and excitatory amino acids and/or blockade of neuronal reuptake of dopamine, serotonin, and excitatory amino acids.

The vascular effects of cocaine include arterial vasoconstriction, in situ thrombus formation, platelet aggregation, and accelerated atherosclerosis that lead to ischemia, in addition to hypertension, tachycardia, and left ventricular hypertrophy and increase myocardial oxygen demand. Most cocaine-using patients are also cigarette smokers and the combination of cocaine and nicotine has a synergistic effect on coronary vasoconstriction.

**Clinical presentation**

Cocaine toxicity is characterized by a sympathomimetic toxidrome with hyperthermia, hypertension, tachycardia, tachypnea, altered mental status, seizures, mydriasis, diaphoresis, and hyperactive bowel sounds.

**Cardiovascular Effects**

Acutely, cocaine causes arterial vasoconstriction and enhanced thrombus formation. Chronic cocaine use can result in accelerated atherosclerosis,and left ventricular hypertrophy. These effects provoke myocardial ischemia or infarction. Supraventricular and ventricular dysrhythmias may occur.

**Neurologic Effects**

Most cocaine-intoxicated patients are anxious or agitated, seizures may occur.

**Vascular Ischemia**

In addition to cardiovascular and cerebrovascular ischemia, ischemia of the mesenteric, renal, pulmonary, and ophthalmic arteries can occur.

**Rhabdomyolysis**

Skeletal muscle injury may result in rhabdomyolysis. Most patients present without signs and symptoms of muscle injury, and the diagnosis of rhabdomyolysis is made by demonstration of myoglobinuria and/or measurement of serum creatine kinase. Patients with severe rhabdomyolysis may have lactic acidosis.

**Obstetric and Neonatal Effects**

Obstetric complications secondary to cocaine use include premature labor as a result of increased uterine contractility as well as abruption placenta. Symptoms of neonatal cocaine withdrawal usually begin within 24–48 hours of birth. Withdrawal results in infants with irritability, and vigorous sucking. In utero cocaine exposure may also result in infants with a small head circumference and low birth weight. Rapid diagnosis and treatment of neonatal withdrawal are important because of the risk of seizures and cardiovascular collapse. Benzodiazepines and phenobarbital are effective agents for the treatment of stimulant withdrawal.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of cocaine toxicity can be subdivided into three areas of consideration: the sympathomimetic toxidrome; specific complaints directly related to cocaine; and organic disease processes masked or confounded by cocaine.

* Sympathomimetic and anticholinergic toxidromes are both characterized by hyperthermia, hypertension, tachycardia, tachypnea, altered mental status, seizures, and mydriasis. Diaphoresis and hyperactive bowel sounds suggest a sympathomimetic toxidrome, while urinary retention and dry skin characterize an anticholinergic toxidrome.
* Specific complaints directly related to cocaine may include such signs and symptoms as chest pain, abdominal pain, and shortness of breath. chest pain may be caused by chest wall rhabdomyolysis, or myocardial ischemia resulting from cocaine use, or it may be caused by pneumonia unrelated to cocaine. A history of recent cocaine use significantly increases the likelihood of serious etiologies for many otherwise common complaints.
* **organ disease,** serious medical problems should not be falsely attributed to cocaine without excluding underlying organic pathology. For example, patients with an altered mental status may still need to undergo computerized tomography to exclude a subdural hematoma that may not have been caused directly by cocaine. Mental status changes caused by cocaine are short-lived. Failure to return to a completely normal mental status within 1–2 hours after exposure requires a complete evaluation for serious etiologies such as subarachnoid hemorrhage or stroke.

**LABORATORY STUDIES**

Most patients with mild cocaine toxicity do not require laboratory evaluation. When ordered, serum electrolytes and hyperglycemia, can be measured in severe cases, in addition to metabolic acidosis due to increased lactate. The serum creatinine level may be elevated in cases of rhabdomyolysis, renal failure, or renal infarction.

Computerized tomography (CT) should be used to detect cerebrovascular events. Urine immunoassays for cocaine metabolites generally detect the major metabolite of cocaine, benzoylecgonine at or above concentrations of 300 ng/mL. Usually, the presence of cocaine or its metabolites can be detected for 48–72 hours after use.

**Treatment**

Initial treatment should focus on the ABCs: Maintain or establish a patent airway; assess breathing, circulation, and neurologic status.

* If the patient is agitated, sedation with benzodiazepines is needed.
* Hyperthermic patients should be cooled with restricted activity, administraton of [diazepam](https://en.wikipedia.org/wiki/Diazepam) or [lorazepam](https://en.wikipedia.org/wiki/Lorazepam) to enhance muscle relaxation and decrease sympathetic outflow from the central nervous system, iced water baths may be used, antipyretics are not effective.
* Hypotensive patients should receive intravenous fluids. If pressor support is necessary, norepinephrine should be used. Hypertension and sinus tachycardia are generally transient and do not require specific treatment in most cases. When treatment is necessary for mild to moderate hypertension, benzodiazepines can be used. For malignant or refractory hypertension, IV nitroglycerin, nitroprusside, or phentolamine can be used. Beta-adrenergic blockade has not been demonstrated to be useful, and may exacerbate hypertension and myocardial ischemia by facilitating unopposed α-adrenergic stimulation.
* Supraventricular tachycardia can be treated with benzodiazepines followed by either calcium-channel antagonists or adenosine. Ventricular dysrhythmias may be managed with benzodiazepines to decrease central sympathetic stimulation and with lidocaine as antidysrhythmic agents.
* Ischemic chest pain should be treated with benzodiazepines, aspirin, and sublingual nitroglycerin. Patients who do not respond to this regimen can be treated with phentolamine or verapamil. Patients with unstable angina should receive heparin according to standard protocols. Beta-adrenergic antagonists and mixed alpha-beta antagonists should not be used in patients with cocaine-induced myocardial ischemia because they increase coronary artery vasoconstriction and decrease myocardial blood flow. Pulmonary edema should be treated with diuretics, and nitroglycerin according to standard protocols.
* Seizures are usually brief and transient. If necessary, seizures can be managed with benzodiazepines, and phenobarbital.