

CE FEATURE

Medical Complications of Cocaine Addiction: Clinical Implications for Nursing Practice

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Cocaine abuse is an important public health problem, with almost two million users in the United States alone. Cocaine abuse kills thousands annually. According to medical examiners, cocaine is the most frequent cause of drug-related deaths. As the use of cocaine remains pervasive so have cocaine-related medical problems. The most serious medical consequences of cocaine abuse are associated with the drug's potent vasoconstrictor properties that can cause life-threatening cocaine-related medical problems such as myocardial ischemia, cardiomyopathies, strokes, renal failure, respiratory arrest, neuronal destruction, and sudden death.

Addiction nurses are challenged to not only understand the complexity of cocaine addiction and its potential and actual deleterious effects on major body systems but also implement interventions that reduce health risks and facilitate recovery. This article focuses on cocaine-related medical complications, medical stabilization, and drug rehabilitation. The role of the nurse is discussed along with strategies and evidence-based approaches to treating patients presenting with cocaine related medical and psychiatric consequences.

Keywords Anhedonia, Cocaine, Cravings, Dopamine, Vasoconstriction, Ventral Tegmental Area (VTA)

Cocaine is one of the most addictive recreational drugs available and its use during the 1980s and 1990s soared. Easy access, relative inexpensiveness, and myths about its safety increased the popularity of cocaine during this era. Since the turn of the century cocaine abuse has declined (The National Institute of Drug Abuse [NIDA], 2006; Substance Abuse and Mental Health Services Administration [SAMHSA], 2001). Despite a lull in cocaine abuse since the 1990s, cocaine-related medical complications have increased dramatically.

Cocaine kills thousands annually and it remains the most frequently encountered illicit drug used in individuals presenting in emergency departments (Office of Applied Sciences [OAS],

2002b). According to medical examiners, cocaine is the most common cause of drug related deaths (OAS, 2002a). Cocaine-related deaths are due primarily to the drug's potent vasoconstriction properties and subsequent deleterious effects on major body systems. Despite the detrimental effects of cocaine, its powerful addictive properties make it difficult for patients to control cravings and compulsive drug consumption. Understanding the underpinnings of addiction, particularly cocaine's effects on the brain's reward system and intervening to reduce medical and psychiatric complications is a daunting task for addiction nurses and other health care providers. This paper presents a compendium of factors associated with cocaine-related medical complications and implications for nursing practice.

PREVALENCE

Although cocaine use has declined in the past decade, it remains a serious public health concern. In 1999, approximately 25 million Americans admitted that they used cocaine at least once; 3.7 million had used it within the previous year; and approximately 1.5 million Americans were regular users (Office of Applied Sciences: National Household Survey of Drug Abuse [NHSDA], 2000). The health care costs of treating individuals with cocaine-related medical complications continues to rise (Hollander & Henry, 2006; Weber, Chudnofsky, Boczar, Boyer, Wilkerson, & Hollander, 2000). In 2000 about 175,000 patients were seen in the emergency department for cocaine-related medical problems (Office of Applied Studies, 2002b), 40% of which complained of chest pain. Fifty-seven percent of patients with chest pain were hospitalized up to 3–4 days (Weber et al., 2000).

NEUROBIOLOGICAL EFFECTS OF COCAINE

Cocaine is a strong central nervous system stimulant that interferes with the reabsorption of dopamine, a neurotransmitter associated with pleasure, memory, and movement. Primary dopaminergic pathways are located in the striatum. The striatum is an essential target site of cocaine action. Structurally, the striatum consists of limbic, associative, and sensorimotor

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pathways, which mediate emotional, cognitive, and motor function. Dopamine projections ascending from the midbrain modulate important input to striatal brain regions. Cocaine blocks the presynaptic reuptake of norepinephrine (NE) and dopamine (DA) resulting in excess neurochemicals at the postsynaptic neuron site (Harvey, 2004; Leshner, 1996; Thompson, Levitt, Standwood, 2005). Acute blockage of dopamine reuptake produces a transient rise in DA concentration in the synaptic cleft, which subsequently expands neurotransmission in the reward pathways within the brain called the ventral tegemental area (VTA). Neurons in the VTA extend to the region of the brain known as the nucleus accumbens, one of the brain's primary pleasure centers. The VTA lies in the mesolimbic dopaminergic system and plays a primary role in mediating the euphoric and rewarding effects of cocaine. The release of dopamine in these neuronal pathways plays a direct or indirect role in the characteristic craving and impulsive behaviors of addiction (Grimm, Hope, Wise, & Shaham, 2001; Macey, Smith, Nader, & Porrino, 2003; Shiffer, Lee, Brodie, & Dewey, 2005).

Prolonged or chronic cocaine consumption increases the risk of tolerance and its addictive (e.g., craving) properties. Craving is a cardinal feature of addiction and is significant because of its potential to trigger cocaine use and relapse. Sun & Rebec (2006) noted that the significance of cravings is their propensity to activate a cascade of neural circuitry and behavioral responses that reinforce the drug's addictive properties. Neural circuitry and behavioral responses involve the limbic, striatal brain regions including structures involved in stimulus-reward recollection (amygdala); incentive motivation (subcallosal gyrus/nucleus accumbens; and anticipation (anterior cingulate cortex). Together these properties make cocaine one of the most addictive drugs and difficult substance-related problems to treat.

PHARMACOLOGICAL EFFECTS

Cocaine (benzoylmethylecgonine) is an alkaloid extracted from the leaf of the *Erythroxylon coca* leaf that has three prominent actions: (1) a powerful local anesthetic, (2) potent vasoconstrictor, and (3) a strong addictive stimulant that has intense reinforcing properties. It is a powerfully addictive stimulant that directly modifies the brain's chemistry and neuroanatomical structures. It is available in two forms: cocaine hydrochloride (powder) and cocaine base (freebase, rock, or "crack" form). Cocaine base is heat stable and thus smokable. Smokable cocaine delivers large amounts of cocaine to the lungs, producing very brisk absorption and physiologic effects. In contrast, cocaine hydrochloride, the powder form, is non-smokable and usually snorted (NIDA, 2006a). Cocaine's addictive or reinforcing properties are related to their effects at the nerve endings of dopaminergic, serotonergic, and noradrenergic neurons. The most powerful effects of cocaine occur at dopaminergic receptor sites, which account for compulsive drug use or bingeing and relapse (Jaffe, Rawson, & Ling, 2005).

A significant area of interest in cocaine's reinforcing properties is the relationships among euphoria, rate of absorption, method of consumption, and addiction. For example, the faster cocaine is absorbed, the more intense its effects and "high." Following the highs or euphoric state withdrawal ensues as evidenced by anxiety, depression, and paranoia. Rapid mood changes that occur between cocaine intoxication and withdrawal intensify cravings as an attempt to regain the "euphoric" state. Higher doses (e.g., 1 to 2 milligrams per kilogram of body weight) produce toxic symptoms such as sleep disturbances, intense anxiety, hypervigilance, delusions, agitation, bizarre and violent behaviors, and, in extreme cases, seizures and pyrexia. Symptoms may persist for several weeks after the drug is stopped.

The euphoric effects of cocaine vary according to the method of consumption or administration. The effects of cocaine depend upon method of administration, level of euphoria, and plasma levels. Compulsive cocaine use occurs more rapidly when the drug is smoked than when it is snorted. When cocaine is snorted cocaine plasma levels rise more slowly and produce less intense effects. When it is used intravenously (IV) or smoked, cocaine plasma level rise rapidly and produce intense effects. Snorted cocaine lasts 15 to 30 minutes, while the smoked form lasts for 5 to 10 minutes. Recurrent use often reduces the period of stimulation (NIDA, 2006a). The serum half-life of cocaine is 45 to 90 minutes; only 1% of the parent drug can be detected in the urine after its metabolites are measurable in blood or urine several hours after its use. However, its metabolites are detectable in blood or urine for 24 to 36 hours after consumption.

A comprehensive discussion of the pharmacologic effects of cocaine is beyond the scope of this article. However, the previous discussion provides salient points about the neurobiology and pharmacology of cocaine, the risk of compulsive and chronic use, as well as the risk of medical and psychiatric complications.

MEDICAL COMPLICATIONS OF COCAINE

Cocaine-related medical complications are vast and have been extensively researched. The most deleterious pharmacologic effect of this potentially addictive drug is due to its vasoconstriction properties (Mittleman et al., 1999; Satran et al., 2005). Apart from vasoconstriction-related medical complications and neuronal death, cocaine has been implicated in the spread of HIV, hepatitis B and C, and various sexually transmitted diseases (STDs). The following section focuses on specific cocaine-related medical and psychiatric complications.

Cardiovascular

Cocaine-related cardiovascular complications are often life-threatening and related to vasoconstriction, enhanced platelet aggregation, thrombus formation, tachycardia, and increased blood pressure. Vasoconstriction is due to activation of norepinephrine (NE) release that heightens myocardial oxygen demands. Increased oxygen demands expand heart rate, systemic arterial

pressure, and left ventricular contractility. These effects produce hypertension, angina pectoris, myocardial infarction (MI), vasospasm, aortic dissection, and arrhythmias (e.g., sinus tachycardia, ventricular irritability, and lower threshold for fibrillation). Cocaine-associated hypokalemia heightens the risk of arrhythmias. Hypokalemia results from the adrenergic properties of cocaine that cause potassium to shift to the intracellular compartment and generally manifests as generalized muscle weakness and sometimes paralysis (O'Leary, 2001). Sudden death results from arrhythmias associated with prolongation of the QT interval, ventricular fibrillation or asystole, cardiomyopathies, and myocardial ischemia (Kanani, Guse, Smith, Barnett & Ellinwood, 1998).

Normally, ischemia occurs within minutes to 36 hours after cocaine use (Jaffe, Rawson, & Ling, 2005, Stein, 1999). Most cocaine users with myocardial ischemia or infarction present with chest pain within one-hour post use, at which time drug serum levels are at a peak. In some cases vasoconstriction occurs several hours after using cocaine. This action is thought to arise from delayed breakdown of cocaine metabolites (e.g., benzoylecgonine and ecgonine methyl ester) (Mittleman et al., 1999). This explains why myocardial ischemia or infarction sometimes occurs several hours after cocaine use.

Cocaine accounts for about 25% of acute non-fatal myocardial infarction in patients 18–45 years of age (Quershi, Suri, Guterman, & Hopkins, 2001). Moreover, an estimated 6% of patients who seek treatment in the emergency department with cocaine-induced chest pain have enzymatic evidence of myocardial infarction (Hollander & Henry, 2006; Weber et al., 2000). Although a large number of cocaine users seek treatment in emergency departments with cocaine-related chest pain, a high percentage of patients continue to use cocaine post hospitalization (Hollander & Henry, 2006). Persistent cocaine use results in chronic insults to the myocardium and heightens the risk of cardiomyopathies.

Respiratory

Cocaine-related respiratory complications are common and appear most often when cocaine is freebased or smoked. Because the lungs are the principal organs exposed to smoked or freebased cocaine, diffuse damage of the alveolar wall and capillary destruction occurs as a result of vasoconstriction (Ali, Krugar, & Houghton, 2002). This damage often results in pulmonary edema, acute respiratory distress syndrome (ARDS), and exacerbation of wheezing and asthma.

“Crack lung” or “eosinophilic pneumonitis” is a respiratory complication whose clinical symptoms involve pain, hemoptysis, black sputum, cough, diffuse wheezing at lung apices, hyperthermia, dyspnea, and diffuse alveolar infiltrate (Ali, Krugar, & Houghton, 2002; Hirche, Lambrecht, & Wagner, 2002). Diagnostic findings associated with this condition include apical infiltrates and pleural effusions on chest xrays and CT scan and leukocytosis with elevated eosinophils and neutrophils. Tox-

icology screens are usually positive for cocaine (Strong et al., 2003). The pathogenesis of this lung condition is unknown, but researchers postulate that it may be a hypersensitivity to cocaine or its diluents. Pulmonary edema is also connected with freebased cocaine, possibly due to alterations in capillary permeability and barotrauma, attributing to a pneumothorax and increasing the risk of bacterial pneumonia (Jaffe, Rawson, & Ling, 2005; Stein, 1999).

Dissimilar to freebased or smoked cocaine, medical complications associated with snorted cocaine include atrophy of the nasal mucosa, rhinitis, rhinorrhea, bleeding, loss of the sense of smell, and vasculitis. Snorted cocaine also is implicated in the vasculitis, which is believed to result from an environmental insult that activates cerebral necrotizing granulomatous syndrome and causes nasal destruction and oronasal fistula in chronic users (Gertner & Hamlar, 2002). In severe cases, necrosis and perforation of the nasal septum have occurred. Chronic snorting also can cause rebound rhinitis and increase the use of over the counter decongestants. Destruction of nasal tissue results from local ischemia produced by chronic, cocaine-induced vasoconstriction (Jaffe, Rawson, & Ling, 2005).

Neurovascular

Neurotoxic effects of cocaine are vast, profound and potentially debilitating and fatal. Cocaine's direct effects on smooth muscle and elevated catecholamine and platelet aggregation are believed to be the most life-threatening and damaging effects of cocaine on the central nervous system. Compulsive cocaine use causes recurrent and persistent elevations in blood pressure; progressive weakening of the vessel wall; and ultimately aneurysm instability or cerebral vascular accident (CVA) (McEvoy, Kitchens, & Thomas, 2000). Additional cocaine-related neurovascular complications include transient ischemic attacks (TIAs), cerebral atrophy, seizures, cerebral vasculitis, infarctions, hyperpyrexia, alterations in neurotransmitter systems, movement disorders (e.g., extrapyramidal side effects [EPS]), and death (Jaffe, Rawson, & Ling, 2005; Tolat, O'Dell, Golamco-Estrler & Avella, 2000). Patients with co-occurring psychiatric disorders, such as schizophrenia are at a risk of developing EPS (Potvin et al., 2006). The pathophysiology of cerebral vasculitis involves a surge in blood pressure after ingestion (Fessler, Esshaki, Stankewitz, Johnson, & Diaz, 1997).

Seizures account for 3–8% of cocaine-related emergency room visits. Cocaine-related seizures normally manifest as single occurrences and may be misdiagnosed as partial complex status epilepticus. A previous history of seizures and high doses of cocaine accentuate the risk of cocaine-induced seizures (Jaffe, Rawson, & Ling, 2005).

Cocaine has a direct regulatory effect on the brain's thermoregulatory centers as evidenced by chills that precede the onset of pyrexia. Chills indicate that the body is adjusting its temperature to a higher level (e.g., 105° F). The potent pyrogenic properties of cocaine have been implicated in the pathogenesis of

life-threatening hyperthermic syndrome. Cocaine-induced hyperthermia is an ominous feature of cocaine toxicity or poisoning (Jaffe, Rawson, & Ling, 2005). This serious and life-threatening syndrome produces severe muscle activity and stimulation, as well as extreme temperature elevation; these symptoms worsen and are exacerbated by heat production. Incidentally, vasoconstriction properties of cocaine also reduce heat loss and expand its pyrogenic features. Because of the potential gravity of toxic pyrexia, it necessitates early recognition, evaluation, and rapid medical stabilization.

Neurochemistry

Analysis of postmortem brain tissue indicates that chronic cocaine users have a marked reduction in striatal vesicular monoamine transporter 2 (VMAT2) binding sites. These data demonstrate the role of cocaine in the destruction or loss of dopamine neurons in the human striatum and reduction of brain dopamine (DA) (Little et al., 1999). The striatum is an important brain region with the greatest density of DA neurons. The VMAT2 is present in brain monoamine neuron, primarily DA, and it plays a key role in pumping DA molecules from the cytoplasm and regulating the size of vesicular DA for synaptic release and storage pools in the CNS (Patel et al., 2003). Alterations in the storage of DA and other monoamines, such as serotonin and norepinephrine, result in a rapid rise in these neurotransmitters in the synaptic cleft. Chronic cocaine use eventually causes damage to or loss of DA neurons in the striatum. Dopamine depleted neurons are unable to reuptake and recover metabolized or excreted dopamine in the synapse. The inability to reuptake dopamine at this juncture can result in striatal hypertrophy and intensify the risk of movement disorders (Jacobsen, Giedd, Gottschalk, Kosten, & Krystal, 2001; Tolat et al., 2000). In addition, depleted dopamine in brain striatum has been found in cocaine users diagnosed with cocaine-related mood disorders at the time of their death (Milne, 2003).

Neurocognitive

Residual and acute neurocognitive effects of cocaine are more common than previously thought. Neurocognitive deficits result from the effects of cocaine on prefrontal and temporal function as evidenced by executive function, problem solving, concentration, visuospatial abilities, word production, attention, learning, and memory deficits (Cunha et al., 2004). Accumulating data indicate neurocognitive deficits are common in acute cocaine withdrawal and are caused by increased noreadrenergic or NE activation (Kelley, Yeager, Pepper, & Beversdorf, 2005). Activation of NE during withdrawal states heightens anxiety and cognitive changes (Brown dyke et al., 2004). However, recent studies demonstrate that these deficits extend beyond acute withdrawal states and are known to persist beyond a year of abstinence (Toomey et al., 2003). Implications from these data indicate the importance of neurological examination as part of the comprehensive physical and psychiatric

evaluation of patients seeking treatment for cocaine abuse or dependence.

Gastrointestinal

Cocaine use has been found to cause gastrointestinal complications. Vasoconstriction has been reported to be a major contributing factor in the pathogenesis of GI complications, which includes gastric ulceration, enterocolitis and perforation, retroperitoneal fibrosis, and intestinal ischemia. Primary symptoms of ischemic colitis are localized single quadrant abdominal pain (proximal colon) and tenderness and bloody diarrhea. Symptoms of enterocolitis usually occur within three days of cocaine consumption (Ellis & McAlexander, 2005). This life-threatening complication should be suspected when a younger or middle age client, especially in the absence of systemic diseases, complains of GI symptoms (Linder et al., 2000; Papi, et al., 1999).

Renal

Vasoconstriction properties of cocaine are linked to serious and potentially fatal renal injury. Renal injury evolves from alterations in hemodynamic function and glomerular injury and conceivably accelerated renal arteriosclerosis and rhabdomyolysis. Rhabdomyolysis is associated with vasoconstriction and muscle injury. Major symptoms of rhabdomyolysis include myoglobinuria, reddish brown pigmentation of urine, elevated blood urea nitrogen (BUN), delirium, and dehydration. Because of the high mortality rate linked to this life-threatening syndrome, aggressive supportive interventions, such as fluid and electrolyte replacement and hemodialysis, may be necessary.

HIV Disease and Hepatitis C Virus

HIV disease is one of the most devastating and life-threatening health consequences of illicit drug use, particularly IV drug use. Cocaine is a cofactor in the AIDS epidemic (Word & Bowser, 1997). Approximately two-thirds of AIDS cases in women and more than 50% of AIDS in children in the United States are related to IV drug use by women and their IV drug user sexual partners (Leshner, 1999). Intravenous drug use has been implicated in an estimated one-third of all HIV and more than half of all hepatitis C cases in the United States (NIDA, 2006). Acute and chronic liver disease, particularly hepatitis C, may be worsened by other comorbid disorders, such as alcoholism (Campbell et al., 2006). Intravenous drug use also widens the transmissional bacterial pneumonia prior to the AIDS epidemic (Mientjes, Spijkerman, & van Ameijden, 1996). The effects of cocaine must be distinguished from HIV disease because of similar presentations (e.g., weight loss and fatigue). High risk of sexually transmitted diseases, such as HIV, also is linked to needle sharing and unsafe sex practices. Unsafe sex practices generally involve unprotected sex and payment of sex for cocaine that can result in transmission of sexually transmitted diseases (STDs).

NEURODEVELOPMENTAL COMPLICATIONS

The precise effect of cocaine on neurodevelopment continues to be debated. However, some researchers submit that cocaine use during pregnancy magnifies the risk of neurodevelopmental impairment and life-long behavioral problems. Known neurodevelopmental conditions associated with cocaine use during gestation include impaired cognitive abilities, especially auditory information processing and receptive and expressive language development (Potter, Zelazo, Stack, & Papageorgiou, 2000). Subtle and varying neurobehavioral consequences of neurodevelopmental account for the difficulty making a definitive diagnosis and the relationship between drug use and subsequent life neurodevelopmental deficits. For this reason, symptoms must be evaluated within the context of substance use disorders and the child’s environment (Frank, Augustyn, Knight, Pell, & Zuckerman, 2001).

In summary, cocaine-related medical complications are serious and potentially fatal. Addiction nurses are challenged to identify vulnerable populations and high-risk behaviors and to collaborate with other providers to initiate preventive measures, including health education, and make accurate diagnoses and initiate appropriate interventions.

TREATMENT CONSIDERATIONS FOR COCAINE-ASSOCIATED MEDICAL CONDITIONS

Medical stabilization is paramount when treating cocaine-related medical and psychiatric complications. Patient evaluation and medical stabilization involve establishing rapport; making an accurate diagnosis and implementing appropriate interventions are pivotal components of holistic medical and psychiatric stabilization.

Medical Stabilization

Typically, a person with a cocaine-related medical problem seeks emergent medical attention exhibiting life-threatening symptoms. Characteristic medical presentations include chest pain, hyperthermia, ataxia, fluid and electrolyte imbalance, muscle weakness, severe agitation, paranoid delusions, respiratory distress, abdominal pain, or oliguria. Regardless of the cause, astute clinical skills to recognize cocaine-induced symptoms are crucial to making an accurate diagnosis and initiating appropriate medical management (Antai-Otong, 2004).

Medical stabilization begins with establishing rapport, performing a comprehensive physical examination and mental status exam, and ordering appropriate laboratory and diagnostic studies, including toxicology screens. Given that the serum-half-life of cocaine is 45 to 90 minutes, it is detectable in the blood and urine several hours after use and its metabolites are detectable 24 to 36 hours after use. (See Table 1). Data collection also must include neurological status and history of drug and psychiatric symptoms and treatment. The neurological evaluation must include cranial nerves, motor and sensory function, gait, coordination, and involuntary movements. Questions about

TABLE 1
Major Components of Physical Examination

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| Chief complaint(s) |
| Vital signs, including temperature |
| General appearance |
| Mental status examination |
| Neurological and nutritional status |
| History of present symptoms |
| Past psychiatric and substance use history |
| Toxicology screens |
| Blood alcohol level (concentration) |
| ECG |
| Urinalysis |
| Blood urea nitrogen (BUN) |
| Metabolic panel |
| Pregnancy test |
| Cardiac enzymes (e.g., serum cardiac troponin T [cTnT]) |
| Erythro sedimentation rate (ESR) |
| Liver panel |
| Chemistry panel |
| Complete blood count with differential |

current drug use, last use, past medical complications, and treatment along with duration of symptoms are invaluable and can be used to guide the evaluation and treatment process.

The high prevalence of cardiac related deaths induced by cocaine, particularly among younger clients, merits suspicion of cocaine-induced cardiac ischemia, especially with non-traumatic chest pain. Nurses must inquire about cocaine use, particularly in younger patients who complain of chest pain. Most studies indicate that the incidence of cocaine-induced chest pain or cardiac ischemia is seldom indicative of a myocardial infarction. However, a definitive diagnosis is difficult for two reasons. First, there is a high failure rate of electrocardiograms to accurately diagnose cocaine-induced myocardial infarction (MI). Secondly, serum creatine kinase (CK) concentrations are unreliable as cardiac markers for cocaine-related myocardial infarction. Consequently, researchers recommend the serum cardiac troponin T (cTnT) because it is a more sensitive cardiac marker than the CK levels in determining a cocaine-induced MI (Hollander et al., 1998; Sayre et al., 1998).

Cocaine induced vasoconstriction of the coronary arteries can be reversed with phentolamine, an alpha-adrenergic antagonist. Propranolol, a beta adrenergic blocking agent, must be avoided in cocaine-related chest pain because it exacerbates cocaine induced vasoconstriction (Hollander & Henry, 2006). Symptomatic treatment must be instituted to treat cardiac, metabolic, fluid, electrolyte, and related medical conditions.

Apart from the physical evaluation, the mental status examination is a pivotal component of the comprehensive evaluation. The client’s mental status must be closely monitored throughout treatment. Common findings from the MSE include cognitive deficits, abnormal movements, agitation, disordered mood (e.g.,

depression), paranoid delusions, and suicidal and aggressive behaviors (Antai-Otong, 2004). Due to the high co-occurrence of cocaine and psychiatric disorders, it is imperative to distinguish psychiatric conditions from medical problems. In the case of dual diagnosis, both conditions must be treated with an initial focus on managing acute symptoms. Measures to ensure staff and patient safety are a critical component of the evaluation and treatment process. Treatment varies and may be necessary for several days or longer depending upon the client's medical condition, psychiatric status, and response to treatment. Although the primary focus of this article has been cocaine-related medical complications, drug rehabilitation is crucial in the prevention of these life-threatening conditions.

Drug Rehabilitation

Because of the complexity of cocaine addiction, a drug rehabilitation model that integrates psychosocial strategies, psychopharmacological intervention for comorbid psychiatric conditions, cultural considerations, and client preferences is essential. Successful clinical outcomes require resolution of denial along with acceptance of the addiction and motivation to make adaptive changes and participate in treatment. Major principles that can guide and facilitate positive outcomes include:

1. A readiness to participate in treatment and maintain abstinence
2. Access to treatment
3. An integrated and comprehensive health model that focuses on medical and psychosocial needs and involves coordination of services
4. Psychotherapy that focuses on behavioral and cognitive concepts that address issues of motivation, self-efficacy, building effective coping and stress management skills, and improve problem solving abilities.
5. Health education about cocaine, safe sex practices to avoid STDs, including HIV and hepatitis C; prenatal dangers of drug and alcohol use
6. Pharmacological interventions to manage psychiatric and medical conditions
7. Continuous monitoring for signs of relapse (e.g., drug screens, missed appointments)
8. Gender-specific and culturally-based interventions
9. Appropriate community referrals, such as 12-step groups (e.g., Cocaine Anonymous), family therapy, crisis, and intervention

The addictive nature of cocaine and high relapse rate necessitate an intensive (e.g., twice a week) outpatient treatment program that utilizes an individualized and integrated model of care. Intense cravings and anhedonia are common during the early phase of treatment.

Patients may benefit from antidepressants such as SSRIs and novel antidepressants, such as bupropion, and mood stabilizers (e.g., lamotrigine) because of their anti-craving and mood stabilizing qualities.

SUMMARY

Cocaine addiction is a major public health problem. It is associated with serious and potentially fatal medical and psychiatric complications. Understanding pathogenesis of cocaine-related medical complications enables the addiction nurse to collaborate with the patient, family, and members of the interdisciplinary team to make an accurate diagnosis, implement evidence-based strategies, and facilitate medical stabilization. Once medical stabilization is attained, the client's motivation for treatment must be evaluated and the nurse must make appropriate referrals to prevent future medical complications and facilitate recovery. This paper has offered salient points concerning cocaine-related medical complications and implications for nursing practice.

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Answer Sheet for Continuing Education Credit

Article 1: Medical Complications of Cocaine Addiction: Clinical Implications for Nursing Practice

Journal of Addictions Nursing (17:4)

Continuing Education Credit: 1.5 contact hour

Purpose: To identify factors associated with cocaine-related medical complications and the implications for nursing practice.

At the completion of the article and the posttest, the reader should be able to:

1. List at least four cocaine-related medical complications.
2. Discuss four treatment strategies for patients with cocaine-related disorders.
3. Identify three nursing interventions that could be utilized in providing care to this population.

Posttest Answer Sheet: (Please circle selected response.)

Select **a** for true and **b** for false in true-false questions.

- | | | | | | |
|-----|---|---|---|---|---|
| 01. | a | b | c | d | e |
| 02. | a | b | c | d | e |
| 03. | a | b | c | d | e |
| 04. | a | b | c | d | e |
| 05. | a | b | c | d | e |
| 06. | a | b | c | d | e |
| 07. | a | b | c | d | e |
| 08. | a | b | c | d | e |
| 09. | a | b | c | d | e |
| 10. | a | b | c | d | e |

Program Evaluation

| | Strongly Disagree | | Strongly Agree | | |
|---|-------------------|---|----------------|---|---|
| Objective 1 was met. | 1 | 2 | 3 | 4 | 5 |
| Objective 2 was met. | 1 | 2 | 3 | 4 | 5 |
| Objective 3 was met. | 1 | 2 | 3 | 4 | 5 |
| Rate the effectiveness of the teaching/learning resources | 1 | 2 | 3 | 4 | 5 |
| Were the objectives relevant to the overall purpose/goal(s) | 1 | 2 | 3 | 4 | 5 |
| Rate the difficulty of this test: 1 = easy and 5 = hard. | 1 | 2 | 3 | 4 | 5 |

How long did this program and posttest take to complete? _____ hours.

Registration Form for Continuing Education Credit

JOURNAL OF ADDICTIONS 17:4 NURSING CE CREDIT

Article 1: Medical Complications of Cocaine Addiction: Clinical Implications for Nursing Practice

(A)—(see below)

CE Instructions:

1. Read the article.
2. Locate the answer sheet and posttest questions following the article.
3. Complete the posttest questions and program evaluation by circling the selected responses on the answer sheet.
4. Fill out the registration form.
5. Send registration form, answer sheet, and a check for \$15.00 to:
 - Continuing Nursing Education
 - The University of Texas at Arlington
 - Box 19407
 - Arlington, TX 76019-0407
6. Send before August 31, 2007

Within three weeks after receipt of your posttest and registration, you will be notified of your results. A passing score is 70%. If you pass, your CE certificate will be forwarded to you. If you do not pass, you will be notified and may repeat the test once at no cost.

The University of Texas at Arlington is an approved provider of continuing nursing education by the Texas Nursing Association, an accredited approver by the American Nurses Credentialing Center's Commission on Accreditation. This activity meets Type 1 criteria for mandatory continuing nursing education toward relicensure as established by the Board of Nurse Examiners for the State of Texas. A copy of your evaluation of this CE offering may be submitted directly to the Iowa Board of Nursing by any licensed nurse.

Registration Information:

Name: _____

Address: _____

City/State/ZIP: _____

Social Security Number: _____

State(s) of Licensure: _____

Telephone Number: _____

Please check the program you are completing. This 1.5 contact hour program is \$15.00.

_____ **Article 1: Medical Complications of Cocaine Addiction: Clinical Implications for Nursing Practice**

(A) Continuing Education Credit: 1.5 contact hours

Posttest Questions for Continuing Education Credit

Article 1: Medical Complications of Cocaine Addiction: Clinical Implications for Nursing Practice

Journal of Addictions Nursing (17:4)

Continuing Education Credit: 1.5 contact hours

CE Questions: Please circle your response on the answer sheet.

Posttest Questions for Continuing Education Credit

- Cocaine-related deaths are caused primarily from:
 - Suicide
 - Cocaine's potent vasoconstriction properties
 - The body's inability to manufacture adequate dopamine
 - Cocaine's ability to exaggerate the death instinct
- The most prevalent presentation in the emergency department for a patient with a cocaine-related medical problem is:
 - Respiratory insufficiency
 - Chest pain
 - Severe constipation
 - Severe diarrhea
- The neurotransmitter MOST associated with cognition is:
 - Dopamine
 - Serotonin
 - Norepinephrine
 - Acetylcholine
- Which of the following is NOT a pharmacological action of cocaine:
 - It is a reinforcing anxiolytic
 - It is a powerful local anesthetic
 - It is a potent vasoconstrictor
 - It is a strong addictive stimulant
- Cocaine has been found to exert its effects at all the major neurochemical receptor sites. However, the most powerful effects of cocaine occur at:
 - Serotonergic receptor sites
 - Adrenergic receptor sites
 - Dopaminergic receptor sites
 - Glutaminergic receptor sites
- Death from fatal cardiac arrhythmias is a result of cocaine-associated:
 - Hyperadrenalism
 - Hypercortisolemia
 - Hypogonadism
 - Hypokalemia
- Cocaine-related respiratory complications are more common when the cocaine is:
 - Injected
 - Smoked
 - Snorted
 - Swallowed
- From a public health viewpoint, addictions nurses' most important role is to:
 - Collaborate with other providers to initiate preventive measures
 - Develop individualized treatment plans for the cocaine-addicted population
 - Identify seroconversion rates for patients with comorbid cocaine dependence and Hepatitis C
 - Make accurate diagnostic formulations
- Characteristic medical presentations for the patient presenting with cocaine-related medical problems include:
 - Chest pain, hyperthermia, and ataxia
 - Muscle weakness, severe agitation, and paranoid delusions

- c. Respiratory distress, abdominal pain, oliguria
 - d. All of the above
10. A successful drug rehabilitation model includes all of the following EXCEPT:
- a. A program-defined length of treatment
 - b. Psychosocial strategies that incorporate a recovery management component
 - c. Psychopharmacological interventions for comorbid psychiatric conditions
 - d. Cultural considerations

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