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ABSTRACT

This document is one of seven publications contained in a series of materials for physicians on recognizing, intervening with, and treating adolescent alcoholism. The materials in this unit of study are designed to help the physician know the different classes of drugs, recognize common presenting symptoms of drug overdose, and place use and abuse in context. To do this, drug characteristics and pathophysiological and psychological effects of drugs are examined as they relate to administration, distribution, special concerns, drug-drug interactions, and tolerance. This unit of study will enable the physician to: (1) state the general principles of clinical pharmacology and pharmacokinetics as they relate to substance abuse; (2) describe the major neurophysiological effects of drugs belonging to the five major drug categories (sedatives, stimulants, narcotics, hallucinogens, and marijuana); (3) associate pathophysiological deleterious effects on systems other than the neurological system with the five major types of drugs; (4) indicate several factors which modify drug actions; (5) evaluate the additive, agonistic, and antagonistic interactions of drugs in combination with alcohol; (6) describe the common presenting symptoms of the five major drug groups in the acute/toxic, chronic, and withdrawal states; (7) list special concerns associated with each drug group; and (8) describe how, when, and where different classes of drugs are used. (NB)

Adolescent Alcoholism: Recognizing, Intervening, and Treating

(The titles and materials listed below are contained in this series.)

	Available Materials		
	Written	Audio	Video
1. Adolescents and Substance Abuse: An Overview	*	*	
2. The Physician's Role in Prevention	*	*	
3. Recognition and Diagnosis	*		*
4. Intervention with the Dependent Adolescent	*		*
5. The Physician's Role in Referral and Treatment	*		*
6. Alcohol and Other Chemicals	*		
Faculty Guide (regarding medical education, residency training, and continuing medical education)			*

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Adolescent Alcoholism:
Recognizing, Intervening, and Treating

6 Alcohol and Other Chemicals

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Introduction

It is widely known that large numbers of adolescents use and abuse alcohol and that many are alcoholics. The physician must be aware that such adolescents very often are co-addicted to other drugs which are taken frequently in combination with alcohol. To successfully treat the alcoholism, the other drug factors must be understood.

Recent studies have shown that there continues to be an increase in drug abuse and experimentation among adolescents. Drugs are readily available to most teenagers who desire them. It is apparent that we cannot depend solely on law enforcement agencies, schools, or parents to control problems with addictive substances. Therefore, it is the responsibility of the primary care physician to become involved in the recognition and treatment of an adolescent who has become addicted to alcohol or other drugs.

Knowledge of the actions of abused substances, beginning with their presenting symptoms, is essential to today's primary care physician. Although alcohol is the most commonly used/abused substance in the adolescent population, it is frequently used in combination with other street or prescription drugs. The resulting actions must be understood in order to correctly diagnose and to implement an appropriate management plan. A major purpose of this unit of study is to present the acute and chronic actions, as well as withdrawal symptoms, of the common street drugs. Becoming familiar with the general classes of drugs and their actions will aid the primary care physician in managing adolescents with these problems.

Goal

The goal of this unit of study is to help the physician know the different classes of drugs, recognize common presenting symptoms of drug overdose, and place use and abuse in context. To do this, pathophysiological and psychological effects of drugs are examined, in addition to drug characteristics, as they relate to administration, distribution, special concerns, drug-drug interactions, and tolerance.

Objectives

Upon completion of this unit of study, you will be able to:

1. *State the general principles of clinical pharmacology and pharmacokinetics as they relate to substance abuse.*
2. *Describe the major neurophysiological effects of drugs belonging to the five major drug categories: sedatives, stimulants, narcotics, hallucinogens, and marijuana.*
3. *Associate pathophysiological deleterious effects on systems other than the neurological system with the five major types of drugs.*
4. *Indicate several factors which modify drug actions.*
5. *Evaluate the addictive, agonistic, and antagonistic interactions of drugs in combination with alcohol.*
6. *Describe the common presenting symptoms of the five major drug groups in the acute/toxic, chronic, and withdrawal states.*
7. *List special concerns associated with each drug group; for example, special concerns during the toxic or withdrawal phases.*
8. *Describe how, when, and where different classes of drugs are used.*

General Principles

Diagnosing and managing drug abuse patients can be complicated by a variety of factors. The physician may find it difficult to find out which drug(s), and in what quantity, is influencing the patient. Below are some general principles that are helpful when one confronts the substance-abusing patient.

1. Psychological factors such as mental set, physical setting, and meaning of drug-taking to the individual are as important as the quantity, timing, and route of drug used.
2. The identity and quantity of the drug taken must be confirmed by dosage from markings or chemical analysis. Note: Counterfeit drugs may be purposely mislabelled.
3. Drug-taking histories are frequently inaccurate for a variety of reasons and should be used only as a point of departure for diagnostic and therapeutic decision-making. Additionally, users may be misinformed or mistaken about what they are taking.
4. There is no "street PDR" or "street FDA"; thus, the positive identity and contents of street drugs must be determined by chemical analysis.
5. Only careful questioning can determine the drug abuser's understanding (or lack thereof) of why he or she uses the drug(s).
6. Some useful and widely prescribed prescription drugs can present special problems for the chemically dependent adolescent; e.g., use of benzodiazepines may lead to a drinking binge in an alcoholic.
7. Use of any street drug by a chemically dependent person may initiate an uncontrolled bout of drug consumption.
8. It is occasionally necessary to administer a test dose of a known prescription drug in order to assess the patient's level of tolerance to a specific class of drugs; e.g., barbiturates.
9. It is essential to know whether one is treating acute, chronic, or withdrawal effects of single or multiple drugs.
10. Patients may manifest symptoms from the pharmacodynamic, toxicodynamic, or pathophysiologic actions of a drug. Pharmacokinetics may be very different in the abuse situation as compared to "normal" drug use.
11. Drug actions can be dramatically modified by drug/drug interactions.

In order to understand substance abuse problems, three spheres of the influences of drugs are frequently considered; i.e., biological, psychological, and social. All three levels are important in the diagnosis and treatment of substance abuse problems. A reality which is too often overlooked is that a symptom or event that manifests at one level (e.g., drug withdrawal symptoms at the biological level) may be significantly influenced at other levels by life events, mental set, physical settings, and cultural norms. Recognizing the interrelationship of these factors may be helpful in diagnosis and management, and the modification of these factors may be beneficial in the long-term treatment goals of the patient.

Definitions

Table 6-1 presents selected definitions with which one should become familiar to better understand the information provided within this unit of study.

Table 6-1. Useful Definitions in Clinical Pharmacology Relevant to Substance Abuse

Absorption - the process by which a drug gains access to the blood from its site of administration; e.g., oral, intravenous (IV), or pulmonary.

Accumulation - the process by which a drug builds up in the body as a result of repeated administration of a drug before the last dose is gone. In such a situation, the plateau plasma level is not reached until three to five half-lives have passed.

Biologic half-life - the time required for one-half of the biological activity of a drug to disappear; only pertains to drugs metabolized by first-order kinetics.

Detoxification - the process of slowly decreasing the dose of a drug so that severe symptoms of withdrawal can be avoided; also refers to simple elimination of a drug from the body by natural or artificial means.

Distribution - the process by and to the extent which the drug travels through the body and finds its way to the site of action (receptor) or to tissues where it may be stored or metabolized.

Elimination - the mechanisms by which the body rids itself of xenobiotics; i.e., chemicals which are foreign to the biological system.

Table 6-1. (cont)

First-order kinetics - a set of circumstances in which the rate of drug accumulation, elimination, or metabolism is proportional to the amount of drug in the body; true for most drugs at therapeutic levels.

Half-life - the time required for half of the initial amount of the drug absorbed to be eliminated. Kinetics must be first-order in order to have a "half-life."

Metabolism - the biochemical processes by which the body chemically modifies a drug.

Pathophysiology - the tissue or organ pathology caused by the use of a drug; e.g., cerebral atrophy from alcohol.

Pharmacodynamics - the actions produced by the drug when given in usual doses which terminate when the drug is eliminated from the site of action; e.g., sedation produced by barbiturates.

Pharmacokinetics - the Absorption, Distribution, Metabolism, and Elimination of a drug (ADME); what the body does to the drug.

Plasma half-life - the time required for the plasma level to fall to one-half of the peak level.

Toxicodynamics - the actions produced when the drug is given in toxic doses; the action usually terminates when the drug level at the site of action drops below the toxic level; e.g., tinnitis from aspirin usually occurs in blood levels over 20 mg%.

Zero-order kinetics - a set of circumstances in which the body metabolizes a fixed amount of drug per unit time regardless of the relative concentration of the drug; e.g., alcohol is metabolized at the rate of about one ounce per hour regardless of dose. Therefore, alcohol does not have a true "half-life" since it is metabolized by zero-order kinetics.

Use of This Unit of Study

This unit of study contains basic information about the five major classes of abused drugs: (1) sedative-hypnotics and depressants, (2) stimulants, (3) narcotics, (4) hallucinogens, and (5) marijuana. In addition, a brief discussion concerning caffeine and tobacco is included.

With each class of drugs, information will be presented that includes (1) nomenclature, (2) drug effects, (3) context of use, (4) common presenting symptoms, (5) pharmacological concepts, and (6) special concerns. Being familiar with this unit of study will be useful to the physician in working with drug-abuse patients.

Note: Reference numbers are listed according to the particular section in which they appear.

Sedative-Hypnotics and Depressants

Alcohol

Nomenclature

Generic (and Trade) Names	Street Names
Assorted	-------

Drug Effects

Pathophysiological: Alcohol is a multi-system toxin. The quantity and duration of alcohol abuse are important variables in determining the pathophysiological impact of alcoholism. A summary of the actions of alcohol on multiple body systems in the acute, chronic, and withdrawal stages of alcoholism is shown in Table 6-2. Most adolescent alcoholics are in the early or middle stages.

Psychological: Alcohol is psychologically addicting. Heavy use of alcohol in adolescence hampers normal developmental patterns. Chronic use of alcohol by the adolescent essentially stunts the psychological growth necessary for adulthood.

Context of Use

Alcohol use spans a broad spectrum. It is widely accepted in familial and social contexts. Widespread potential for abuse exists in these contexts as well as on the personal level.

Table 6-2. Common Presenting Symptoms of Alcohol

System	*Acute*	*Chronic*	*Withdrawal*
Nervous			
Brain	Profound CNS depression Mental impairment Constricted pupils	Wernicke's disease Korsakoff's psychosis Tolerance Chronic brain dysfunction	Delirium tremens Excitation Confusion Disorientation Generalized convulsions (Rumfits)
Spinal Cord	Depression of reflexes		Increased reflexes
Peripheral	Decreased sensitivity Hypothermia	Peripheral neuropathy	Asterixis
Digestive			
Oral	Alcohol on breath	Increased risk of head and neck cancer	
Esophagus	Inflammation	Esophageal varices with possible hemmorhage	
Stomach	Gastritis	Gastritis/ulcers	Increased motility
Pancreas	Pancreatitis with increased serum amylase	Pancreatitis with possible pseudocyst formation	
Liver	Increased SGOT/SPGT/GGT; SGOT usually greater than SGPT	Alcoholic hepatitis Alcoholic cirrhosis	
Respiratory	Respiratory depression Pulmonary edema (overdose)	Pneumonias Bronchitis	Increased respiration
Cardiovascular			
Myocardium	Arrythmias Increased cardiac output	Cardiomyopathy	Increased cardiac output Increased cardiac load
Vessels	Peripheral vasodilation	Increased blood pressure Caput Medusae	Hypertension
Urinary	Increased urine output	Renal damage Bladder tumors Urinary retention	
Hematopoietic		Macrocytic hypochromic anemia Coagulopathies	

Table 6-2. (cont) Common Presenting Symptoms of Alcohol

System	*Acute*	*Chronic*	*Withdrawal*
Skeletal	Traumatic fractures	Previous skeletal trauma	Trauma secondary to delirium tremens
Skin	Burns, trauma	Burns, trauma	
Muscular	Weakness Loss of coordination	Atrophy	Tremors Fasciculations Cramps
Endocrine			
Male	Decreased vasopressin release	Decreased testosterone Estrogen predominance Hyperlipidemia	Increased catecholamines
Female	Decreased vasopression release	Irregular menses Decreased feminization	Increased catecholamines
Psychological	Decreased awareness Agitation Hostility Euphoria Talkativeness Mood swings Increase in confidence Loss of inhibition Stuporousness		Paranoia Hostility Agitation

The above psychological states can also be correlated with the Laboratory Blood Alcohol content levels.

B.A.C. (Blood Alcohol Content) Status

0.05% = significant mental impairment (as shown below)
0.1% = legal intoxication in most states
0.2% = severe uncoordination
0.3% = stuporous condition
0.4% = semi-comatose to fatal

One fifth of 80-proof alcohol can be a lethal dose for a nontolerant 70 kg (155 lb) adolescent.

Drug Characteristics

Absorption: Orally

Absorption: Primarily absorbed in the small intestine, though also stomach and colon.

Distribution. Alcohol mixes readily in water, thus, it distributes into total body water.

Metabolism: Primarily in the liver by the enzyme alcohol dehydrogenase, where it is metabolized to acetaldehyde, which in turn is metabolized to carbon dioxide and water. Note that these enzymes are readily saturated, which allows the liver to pass ingested alcohol into the general circulation. This metabolism is zero-order kinetics.

Elimination: The actions of alcohol are terminated largely by metabolism, although small amounts are excreted via the lungs, skin, and kidneys. Even in large doses almost all alcohol is eliminated from the body in 12 to 24 hours.

Tolerance: Tolerance does develop with increased use by increasing the amount of enzymes available in the liver.

Approximate detection time in urine after use: From 1-12 hours.

Special Concerns

1. ALWAYS BE AWARE OF COEXISTING DRUG TOXICITIES.
2. Delirium tremons are MEDICAL EMERGENCIES associated with approximately 15% mortality rate if improperly treated.
3. Increased suicide.
4. Increased family violence.
5. Increased trauma - suspect broken bones, watch for central nervous system (CNS) damage; i.e., subdural hematoma.
6. In addition, suspect hypoglycemia, hyperglycemia, electrolyte imbalance, and stroke.
7. Hyperthyroidism and carbon monoxide poisoning may mimic alcohol into :ification.
8. Correct electrolytes and magnesium levels if needed.
9. Be aware of family abuse patterns.
10. Increased potential for birth defects (Fetal Alcohol Syndrome).
11. Always treat all suspected cases of alcohol abuse with thiamine on initial admission.
12. High association of alcohol abuse with motor vehicle accidents.

Table 6-3 shows the interactions and effects of drugs mixed with alcohol.

Table 6-3. Drug-Alcohol Interactions

Stimulants
- Increased hostility
- Increased blood pressure
- Increased mentation changes with amphetamine

Minor Tranquilizers
- Increased CNS depression[1]
- Increased absorption[2,3]
- Cross-tolerance
- Orthostasis

Major Tranquilizers
- Increased CNS depression
- Orthostatic hypotension
- Cardiac arrhythmias
- Akinesia and dystonia

Table 6-3. (cont) Drug-Alcohol Interactions

Marijuana
- Increased CNS depression
- Increased intoxication effects

Sedative Hypnotics
- Increased rate or absorption
- Inhibiting phenobarbital metabolism[4,5]
- Increased metabolism of barbiturates[1,6]
- Increased sedation, ataxia, uncoordination, feeling of drunkenness[7]
- Nystagmus, respiratory depression, coma, death (with large doses)
- Cross-tolerance
- Orthostasis

Tricyclic Antidepressants
- Increased sedation[8,9]
- Increased hepatic metabolism
- Increased cardiac arrhythmias
- Increased orthostatic hypertension

Disulfiram-type Reactions
- Disulfiram (Antabuse)
- Chloramphenicol (Chloromycetin, etc.)
- Chlorpropamide (Diabinese)
- Furazolidine (Furoxone)
- Griseofulvin (Fulvicin)
- Metronidazole (Flagyl)
- Phentolamine (Regitine)
- Quinacrine (Atabrine)
- Tolbutamide (Orinase)
- Moxalactam

Increased Effects - With Acute Alcohol Use

Antidiabetic
- Tolbutamide

Drug Action
- Barbiturates
- Phenytoin (Dilantin, etc.)
- Sedatives, tranquilizers
- Warfarin-like anticoagulants

CNS Depression
- Antihistamines
- Barbiturates
- Benzodiazepines

Table 6-3. (cont) Drug-Alcohol Interactions

Chloral hydrate
Major tranquilizers
MAO inhibitors
Marijuana
Meprobamate
Methyprylon
Narcotics
Tricyclic antidepressants

Decreased Effects - With Chronic Alcohol Use

Antidiabetic
Tolbutamide

Drug Action
Barbiturates
Isoniazid
Phenytoin
Sedatives, tranquilizers
Vitamin K-dependent syntheses
Warfarin-like oagulants

Barbiturates

Nomenclature

Generic (and Trade) Names	Street Names
Secobarbital (Seconal)	Reds, red devils, F-40's
Pentobarbital (Nembutal)	Yellows, yellow jackets
Secobarbital and Amobarbital (Tuinal)	Blues, blue birds, rainbows
Phenobarbital	Phennies, purple hearts
Amobarbital (Amytal)	Blues, downers

Also - mephobarbital, metharbital, aprobarbital, butabarbital, talbutal

Drug Effects

Pathophysiological: Barbiturates reduce postsynaptic cAMP and cGMP activity in the central nervous system. Strong physical dependence can develop. Most barbiturate effects subside a week or so after the last dose, although subtle psychomotor changes can still be detected up to six weeks after detoxification.

Psychological: Depression, relaxation, sedation, decreased inhibitions.

Context of Use

Recreational
Loner drug
Used with alcohol and other drugs

Common Presenting Symptoms

Acute
Ataxia
Respiratory depression
Depression
Uncoordination
Sedation
Euphoria
Drunkenness
Loss of judgement
Relaxation
Decreased blood pressure
Decreased urine formation

Chronic
Emotional debility
Clouded thinking
Fabrication
Denial
Excessive sleepiness
Irritability
Severe withdrawal sickness
Progressive cardiac failure
Fever

Withdrawal
Potentially life-threatening
Increased anxiety
Insomnia
Tremors
Delirium
Convulsions
Grand mal seizures
Loss of control
Hallucinations
Regressions
Decompensation
Death

Drug Characteristics

Administration: Usually oral, but can also be taken IV.

Absorption: Primarily in the small intestine.

Distribution. Barbiturates are bound to plasma proteins in varying degrees.

Metabolism: Primarily by the liver.

Elimination: Although some short-acting and intermediate-acting barbiturates are excreted by the kidneys, the only barbiturates which have significant renal excretion are phenobarbital and barbital.

Tolerance: Tolerance develops to both the effective dose and the lethal dose of barbiturates. However, tolerance develops more rapidly to the effective dose than to the lethal dose, and therefore it is easier for a fatal overdose.

Approximate detection time in urine after use: Over 30 days (phenobarbital).

Special Concerns

1. Barbiturates and alcohol are a lethal combination; death can result from the additive effects.
2. Barbiturate overdose and withdrawal can result in death if untreated.
3. Toxicokinetics: In overdose, the kinetics of barbiturates may go from first-order to zero-order, a phenomenon which is common in cases of massive drug exposure. Therefore, serum drug levels may prove helpful in treatment.

Diagnosing Barbiturate Addiction[1]

The most reliable way to assess the level of barbiturate or nonbarbiturate sedative-hypnotic addiction is to hospitalize the patient and administer a 200 mg dose of pentobarbital after a 24-hour drug-free period. If sedation occurs in this setting, the patient's level of addiction to sedative-hypnotics can be assumed to be trivial. If the patient manifests nervousness, agitation, anxiety, tremulousness, and hyperactive reflexes, an additional dose of 100 mg pentobarbital should be administered hourly until sedation occurs. The dose of pentobarbital can then be changed to the equivalent number of milligrams of phenobarbital, to be given as a t.i.d dose. At this point detoxification can begin; once the patient has stabilized, the doses can be decreased by 10 percent per day, remembering that withdrawal from barbiturates and nonbarbiturate sedative-hypnotics can be fatal.[1] See Table 6-4 for a summary of pharmacological actions and equivalencies.

Nonbarbiturate Sedative-Hypnotics

These drugs are taken for essentially the same reasons as barbiturates and have similar problems and risks associated with their abuse.

Nomenclature

Generic (and Trade) Names	Street Names
Chloral Hydrate (Noctec, etc.)	-------
Ethchlorvynol (Placidyl)	Jelly beans, Mr. Green Jeans, pickles
Glutethimide (Doriden)	Cibas, downers, dyls
Meprobamate (Equanil, Miltown, etc.)	Bams, Uncle Milties
Methaqualone (Quaalude)	Ludes, sopors, Q's, 714's

Table 6-4. Sedative-Hypnotic Properties of Barbiturates

Drug Name	*Duration of Action*	*Toxic Blood Level*	*Single Dose Equiv. to 100 mg Pentobarbital*	*Dose/24 hr Equivalency*
Phenobarbital	12-24 hr	8 mg %	30 mg	100-200 mg
Secobarbital	4-6 hr	3 mg %	100 mg	400-600 mg
Pentobarbital	6-8 hr	3 mg %	100 mg	-------
Glutethimide (Doriden)	4-8 hr	1-8 mg %	600 mg	300-600 mg
Ethchlorvynol (Placidyl)	4-6 hr	2 mg %	500 mg	2,000-3,000 mg

Note. Doriden and Placidyl will have extended duration of action in an overdose state because of increased protein binding which does not release drug for metabolism as rapidly.

Drug Effects

Pathophysiological and psychological effects are similar to barbiturates.

Context of Use

Similar to barbiturates. Quaaludes are also reported aphrodisiacs.

Common Presenting Symptoms

Same as barbiturates, except:

Acute
- Pupil dilation
- Nausea, vomiting
- Double vision

Chronic
- Gastric irritation
- Blood dyscrasias

Withdrawal
- Insomnia
- Irritability
- Convulsions

Drug Characteristics

Administration: PO.

Absorption: Primarily in the small intestine.

Distribution: Throughout the body; lipid-soluble and tend to be bound to tissues.

Metabolism: Primarily by liver metabolism.

Elimination: Largely excreted in the urine.

Tolerance: Glutethimide has cross-tolerance with barbiturates.

Approximate detection time in urine after use: Up to 4-24 days (methaqualone).

Special Concerns

As with barbiturates, in regard to toxicity and overdose.

Quaaludes are currently off the U.S. market and, therefore, presently tend to be either bootleg or foreign drugs. One popular foreign drug is called mandrax (mandies), which is a combination of methaqualone and diphenhydramine (Benadryl).

Counterfeit street ludes have been found to contain diazepam, phenobarbital, antihistamines, phencyclidine (PCP), and pain relievers (aspirin and acetaminophen, among others).

Major and Minor Tranquilizers

1. Major tranquilizers such as chlorpromazine (Thorazine) are commonly abused by the middle-aged/middle-class. These are not popularly abused drugs among adolescents. Therefore, we will discuss only the minor tranquilizers.
2. Minor Tranquilizers (Benzodiazepines) - Popular with adolescents in part due to wide-spread availability.

Nomenclature

Generic (and Trade) Names	Street Names
Diazepam (Valium)	Roches, pumpkin seeds, tranks, downers
Chlordiazepoxide (Librium)	Libs, tranks, downers
Flurazepam (Dalmane)	Tranks, downers
Lorazepam (Ativan)	Tranks, downers
Oxazepam (Serax)	Tranks, downers

Drug Effects

Pathophysiological: Increase GABA activity in the central nervous system; also act as glycine receptors.[1] Physical dependence is not as severe as with barbiturates.

Psychological: These are anxiolytic and hypnotic, and they cause sedation. Psychological dependence is not as severe as with barbiturates.

Social Context

Similar to barbiturates; they may be used in combination with other drugs; e.g., as a "come-down" method after stimulant use. Of concern is the widespread availability due to the large amount of prescriptions written for these drugs.

Common Presenting Symptoms

Acute, chronic, and withdrawal signs are similar to barbiturates[2]; however, rarely are the severe symptoms of barbiturates usage (neurological, behavioral) seen with benzodiazepines.

Drug Characteristics of Diazepam

Administration: PO, IM, IV.

Absorption: If PO, there is rapid gastrointestinal absorption; slow if IM.

Distribution: Lipid-soluble. Bound to plasma proteins.

Metabolism: Liver mitochondrial enzymes. Long half-life (20-50 hours).

Elimination: Slow.

Tolerance: Yes, to the pharmacokinetic effects. Cross-tolerance is seen with short-acting barbiturates.

Approximate detection time in urine after use: Up to 72 hours (as oxazepam metabolite).

Special Concerns

1. Diazepam is occasionally used with opiates; for example, heroin.
2. Combination with alcohol is dangerous due to additive CNS depression, with death occurring at times.
3. The benzodiazepines vary greatly in terms of their pharmacological properties; Table 6-5 lists the half-lives of selected benzodiazepines.

Table 6-5. Benzodiazepine Half-Lives

Drugs	*Hours*
Oxazepam (Serax)	3-21
Chlordiazepoxide (Librium)	5-30
Temazepam (Restoril)	8-12
Halazepam (Paxipam)	10-14
Lorazepam (Ativan)	10-20
Alprazolam (Xanax)	12-15
Diazepam (Valium)	20-50
Chlorazepate (Tranxene)	30-60
Flurazepam (Dalmane)	30-60
Prazepam (Verstran)	60-80

Inhalants

Solvents and Aerosols

Nomenclature

Generic (and Trade) Names	Street Names
Toluene	-------
Acetone	-------
Benzene	-------
Gasoline	-------
Rubber cement	-------
Paint thinner	-------
Freon	-------
Aerosol sprays	-------
Butane	-------
Xylene	-------
Fluorocarbons	-------

Drug Effects

Pathophysiological: Related to their lipid-solubility, these cause CNS depression by impairing membrane permeability and neuronal transmission.[1] Physical dependence occurs rarely, and no direct abstinance syndrome has been identified.

Psychological: Produce altered states of consciousness.

Social Context

A relatively cheap method of getting high; e.g., fumes are available from tubes of model airplane cement. This makes it particularly available to lower socioeconomic groups.

Common Presenting Symptoms

Acute

Altered state of consciousness
Mild intoxication
Disinhibition
Euphoria
High similar to all CNS depressants
Nausea
Vomiting
Chest pain
Nasal irritation

Bronchial irritation
Irritation of the eyes
Drowsiness
Lightheadedness, giddiness
Slurred speech
Ataxia
Impaired judgement

Later stages with continued use: deliriums, hallucinations, coma, convulsions, cardiorespiratory arrest, and sudden death.

Chronic

May lead to psychological dependence and tolerance, and habituation; compulsive chronic use has been reported.

Withdrawal

Chronic use may cause damage to the central nervous system, peripheral nerves, kidney, liver, and bone marrow that remains after withdrawal.

Drug Characteristics

Administration: Sniffing, usually in a closed container such as a plastic or paper bag.

Absorption: Through the respiratory alveolar membranes and into the pulmonary capillaries.

Distribution: They are lipophilic and can be taken up in the high lipid content of the central nervous system or fat tissue.

Metabolism: Unchanged and excreted by the lungs, or metabolized by the liver and excreted in the urine.

Elimination: Usually by exhalation.

Tolerance: The central nervous system effects are experienced by sniffers who use them regularly for long periods of time. This tolerance tends to be quite variable.

Approximate detection time in urine after use: Not applicable.

Special Concerns

Suffocation, accidental explosions, and increased incidence of trauma. Of primary concern are the multiple toxicities associated with the ingredients found in many inhalants; i.e., lead toxicity.

Nitrous Oxide

Nomenclature

Generic (and Trade) Names	Street Names
Nitrous oxide	Laughing gas

Drug Effects

Pathophysiological: Nitrous oxide is a general anesthetic and analgesic. In the blood it goes primarily into solution and is not decomposed; therefore, the oxygen in nitrous oxide is unavailable to support tissue respiration. It does not combine with hemoglobin.

Psychological: Used for a brief high; sometimes persons may experience deliriums, hallucinations, amnesia, or analgesia.

Context of Use

Large sociological use, especially at parties by medical personnel who have access to the pure form, especially anesthesiologists and dentists.

Common Presenting Symptoms

Usual presentations are giddiness, slight intoxication, floating sensation, analgesia, and in later stages coma, respiratory depression, and seizures.

Drug Characteristics

Administration: Respiratory, usually with an anesthesia mask.

Absorption: Through the alveolar membranes and into the pulmonary capillaries.

Distribution: Within the blood.

Metabolism: Primarily unchanged when excreted by the lungs, or metabolized by the liver and excreted via the urine.

Elimination: Primarily unchanged via the lung.[2]

Tolerance: Variable.

Approximate detection time in urine after use: Not applicable.

Special Concerns

1. Primarily asphyxiation, secondary to hypoxia resulting from nitrous oxide inhalation without oxygen.
2. There is a form of nitrous oxide in whippets, which are manufactured for use as propellants but are commonly available in "head shops."

3. Note that nitrous oxide is available in pressurized whipped cream containers from which, when tilted, the gas can be inhaled directly into the mouth from the can.
4. Respiratory arrest secondary to relaxation of throat and tongue muscles.

Volatile Nitrites

Nomenclature

Generic (and Trade) Names	Street Names
Amyl nitrite	Ames, pearls, poppers, snappers

Drug Effects

Pathophysiological: Potent smooth muscle relaxant and vasodilator. No long-term effects and no withdrawal have been noted.

Psychological: Changes in perception, thought, and behavior.

Context of Use

In the socially homosexual population, as a prelude to sexual activity and just prior to orgasm. Reputation is that it is an aphrodisiac.

Common Presenting Symptoms

Acute
- Lightheadedness
- Weakness
- Nausea
- Confusion
- Ataxia
- Headache
- Syncope

Chronic
- Sensory disturbances
- Impotence
- Sphincter dysfunction

Withdrawal
- NA

Drug Characteristics

As with the other inhalants previously mentioned.

Special Concerns

1. Suspect methemoglobinemia in patients with cyanosis which does not clear with oxygen therapy.
2. Death as been reported in patients with prolonged use.[3]

Stimulants

Amphetamine, Methamphetamine

Nomenclature

Generic (and Trade) Names	Street Names
Amphetamine (Benzedrine)	Bennies, black beauties, peaches, splash, uppers, Christmas trees
Dextroamphetamine (Dexedrine)	Dexies, uppers, oranges
Methamphetamine (Methedrine, Desoxyn)	Crystal, meth, speed, water, white crosses

Drug Effects

Pathophysiological: Essentially these are both alpha and beta receptor agonists, primarily affecting the CNS. Physiological dependency can develop.

Psychological: Psychologically addicting. Heightened awareness, mood elevation, decreased fatigue; increased performance, energy, labile effect, anxiety, delirium, euphoria, and floating feelings.

Social Context

- Getting high
- Cramming for exams
- Partying all night
- Counteract depressant effects

Common Presenting Symptoms

Acute
- Dilated pupils
- Tachycardia
- Talkativeness
- Anorexia
- Tremor
- Cardiac arrhthymias
- Dizziness
- Floating feeling
- Facial grimacing
- Hallucinations
- Hyperthermia
- Irritability
- Dry mouth
- Muscle spasms

Piloerection
Hyperactive reflexes
Restlessness
Skin picking
Paranoia
Euphoria
Delirium
Increased anxiety and emotional lability

Chronic
Emaciation
Cardiac arrhthymias
Psychosis and paranoia
Sleep disturbances
Withdrawal symptoms
Severe depression
Disorientation
Slow comprehension
Hyperphagia
Orthostatic hypotension
Rhinorrhea
Hypertension
Arterial inflammatory disease

Withdrawal
Slow comprehension
Coryza
Depression
Fatigue
Hypotonia
Sleep disturbances

Drug Characteristics

Description: Weak bases.
Administration: Nasal, IV, PO, or smoking.
Absorption: Primarily via the small intestine, with a peak absorption 1-2 hours.
Distribution: Water and lipid soluble with wide body distribution.
Metabolism: Primarily liver.
Elimination: Drug and metabolites are excreted by kidney.
Tolerance: Yes, dramatically up to 100 times the usual dose.
Approximate detection time in urine after use: Varied, according to drug taken.

Special Concerns

Drug-amphetamine interactions
Alcohol and amphetamines
Increased violence
Increased hypertension
Decreased mentation

Marijuana and Amphetamines[1,2]
Increased heart rate
Increased hypertension

Withdrawal Concerns
Severe emaciation
Severe depression*
Paranoia
Psychosis

**NOTE:* Depression in amphetamine withdrawal is not responsive to antidepressant therapy.

Cocaine

Nomenclature

Generic (and Trade) Names	Street Names
Cocaine hydrochloride	Crack, coke, snow, uptown, toot, blow, flake, nose candy, rich man's drug leaf

Drug Effects

Pathophysiological: Blocks neurotransmitter re-uptake, potentiating the effects of epinephrine and norepinephrine. It is a powerful CNS stimulant. Direct effects are upon the cortex and medulla. A local vasoconstrictor and a local anesthetic.

Psychological: Euphoric, psychologically addicting (addiction could be life-threatening), paranoia, aggressive behavior, megalomania, anxiety, psychosis, depression, suicide ideation.

Common Presenting Symptoms

Acute (Early)
Euphoria
Increased energy
Increased self-confidence
Increased awareness
Mood elevation
Increased physical performance
Increased concentration
Feelings of anxiousness, or being wired
Headache
Tremor
Dilated pupils
Hyperreflexia

Arrhythmias
Paranoia
Appetite suppression

Acute (Middle)
Cyanosis, dyspnea
Cheyne Stokes breathing
Grand mal seizures
Ventricular arrhythmias
Anoxia

Acute (Late)
Respiratory failure
Paralysis
Loss of consciousness
Ventricullar fibrillation
Circulatory collapse
Death

Chronic
Physically similar to acute phase
Intense psychological dependence
Preoccupation with drug use
Paranoia
Auditory, visual, tactile hallucinations
Emaciation

Context of Use

Primarily social; sharing is commonplace. Associated with higher social status ("Rich Man's Drug").

Drug Characteristics

Administration: PO, IV, smoking, intranasal (snorting).
Absorption: IV administration results in a rapid onset of action, within minutes. Intranasal administration results within minutes. When smoked, as a "free base," or "crack," the absorption is also within 5 minutes.
Distribution: Widely distributed into the lipid tissues of the body.
Metabolism: Cocaine is rapidly hydrolyzed by plasma and hepatic cholinesterase.
Elimination: Via the urine.
Tolerance: The issue of tolerance is not as clear as with the case of amphetamines, although larger doses of cocaine produce more cocaine effects, which can terminate in toxic states of excitation and convulsions.
Approximate detection time in urine after use: 4-6 days; 18-27 hours (as metabolites).

Special Concerns

1. Sudden cardiac death.
2. Nose bleeds, infection, and perforation of the nasal septum.
3. Speedballing, the injection of a mixture of heroin and cocaine, is difficult to treat and is potentially lethal. See Table 6-6 for other cocaine-drug interactions.
4. Free-basing is the ether extraction process used to make cocaine smokeable. Explosions and fire may result from this procedure.
5. Detoxification and Withdrawal: Cocaine can be suddenly discontinued without significant medical problems. There is a profound depression associated with withdrawal; however, it is not responsive to antidepressants. Suicide precautions may be necessary.
6. "Crack": A highly purified aqueous solution of cocaine HCl. Its use is reported to be epidemic in major metropolitan areas of the U.S. It is also known to be highly addicting, sometimes only after 1-2 doses.[3] Crack is so named because of the popping sound which is made when the crystals are heated. When smoked and absorbed by the pulmonary vascular bed, its effects are comparable to intravenous injection.[4] The highly addictive potential coupled with easy accessibility and low cost make crack a threat to teenagers everywhere. *The physician should be aware of its rapid growth and great potential for abuse.*

Table 6-6. Drug-Cocaine Interactions

1. Monoamine oxidase inhibitors (by decreasing catecholamine metabolism) increase the effects of cocaine (which prevents catecholamine re-uptake) and may result in hypertensive episodes.
2. Tricyclic antidepressants potentiate cocaine activity, as both inhibit catecholamine re-uptake.
3. Reserpine and methyldopa (by producing a "chemical denervation supersensitivity") potentiate cocaine actions, as all of these drugs increase responsiveness to catecholamines, as well as decrease antihypertensive effects of drugs.
4. Cholinesterase inhibitors interfere with cocaine hydrolysis by inhibiting plasma pseudocholinesterase.
5. Methadone does not block cocaine effects, making cocaine a favorite drug of methadone-maintained patients.

Look-Alike Stimulants

Nomenclature

Generic (and Trade) Names	Street Names
Caffeine	-------
Plenylpropanolamine	-------
Ephedrine	-------

Normally, look-alikes contain phenylpropanolamine, caffeine, and ephedrine. Over-the-counter diet pills are similar.

Drug Effects

Pathophysiological: CNS stimulant. Chronic caffeine abuse may be associated with renal or hepatic disease. Acute toxicity of these drugs can cause hypertension which can result in cranial bleeds, convulsions, and cardiac arrhythmias.

Psychological: Euphoria, excitement, and hyperactivity.

Context of Use

Legal and accessible. Known as a "poor man's speed." There may be a lot of adolescent peer pressure. Usage may be a shared social event. Perceived by adolescents to be safe.

Common Presenting Symptoms

Acute

Appetite suppression
Increased anxiety
Mild feelings of euphoria and paranoia
Possible psychosis with increased agression

Chronic

Poor nutrition
Loss of appetite
Gastritis, ulcers
Paranoid ideation

Withdrawal

No significant symptoms physically, although psychologically there may be paranoia, depression, and a return of appetite.

Drug Characteristics

Absorption: When taken PO, it is absorbed from the small intestine. Be aware that some products are sustained-released.

Distribution: Peak level in the plasma within ¾ to 2 hours.

Metabolism: All drugs are metabolized by the liver.

Elimination: Via the kidneys.

Tolerance: Not clinically significant with phenylpropanololamine, although tolerance may develop to the actions of caffeine and ephedrine, especially with chronic use.

Approximate detection time in urine after use: Varied, according to drug taken.

Special Concerns

1. Sudden death (Cerebral Vascular Accident).
2. Addiction withdrawal and detoxification: The drug can be withdrawn abruptly; however, hypotension may occur. Caffeine withdrawal is similar to amphetamine withdrawal and may cause headaches and depression.
3. A look-alike overdose is a medical emergency.

Narcotics

Nomenclature

Generic (and Trade) Names	Street Names
Heroin	H, Harry, horse, dogie, dope, scag, smack
Morphine	M, morph, morpho, Miss Emma, junk, hard stuff, Mary
Meperidine (Demerol)	Demmies
Methadone (Dolophine)	Meth, dollies
Codeine	Blue velvets
Opium	Black pills
Oxycodone with aspirin (Percodan)	Percs
Hydromorphone (Dilaudid)	Dils

Drug Effects

Pathophysiological: Analgesia. Effects are at CNS opiate receptors and medullary areas. Peripheral vasodilation and gastrointestinal depression may occur. Strong physical dependence develops.

Psychological: Strong psychological dependence develops. Profound classical addiction can occur so that the adolescent is totally dedicated to the procurement and use of narcotics. The physical addiction can become quite severe where the user will continue to take the drugs to avoid the discomfort of withdrawal sickness.

Social Context

Common Medical Use: Analgesics, cough suppressant, paregoric; related analgesics are Darvon and Talwin.

Narcotics are used by adolescents much more commonly by the oral than the intravenous route. Codeine is particularly popular because it is readily available. Cough syrup with codeine can be obtained without a prescription from pharmacists.

Intravenous narcotic use is found in increasing numbers in adolescents from middle class families.

Common Presenting Symptoms

Acute
- Mental clouding
- Drowsiness
- Mild to extreme euphoria
- Nausea
- Vomiting
- Itching sensation
- Pinpoint nonreactive pupils
- Severe respiratory depression due to collapse
- Decreased pulse and hypotensiveness
- Cold clammy skin
- Hypothermia
- Possible pneumonia and pulmonary edema

Chronic
- Physical addiction
- Lethargy
- Weight loss

Withdrawal
- Restlessness
- Irritability
- Tremors
- Loss of appetite
- Panic
- Chills
- Sweating
- Cramps
- Watery eyes
- Runny nose
- Nausea
- Vomiting
- Muscle spasms

Drug Characteristics

Administration: Usually PO, but IV or SQ also.
Absorption: Codeine taken orally is slowly absorbed, with a peak in 1-2 hours.
Distribution: Narcotics distribute into the CNS fairly rapidly once they are absorbed or injected into the blood.
Metabolism: Narcotics are metabolized by the liver.
Elimination: In the feces and urine.
Tolerance: Monumental tolerance can develop to narcotics in which a hundred times the normal dose is required to produce significant effects. There seems to be no upper limit to this tolerance.
Approximate detection time in urine after use: Up to 24 hours (heroin, as morphine metabolite); 48 hours (proxyphene).

Special Concerns

Detoxification can be accomplished in a number of ways:
a. Cold turkey, with short-lived, intense symptoms.
b. Use of clonidine. The average maintenance dosage during treatment is 0.2-0.8 mg/day.[1] Divide the doses into 24 hours, and continue for 2 weeks before discontinuing the narcotic, or until no longer deemed necessary.
c. Use of methadone. The patient can be transferred to methadone, then slowly detoxified by decreasing the dose that was agreed upon by himself and the physician.
d. Any narcotic can be used for detoxification.

Pentazocine (Talwin). This drug became popular when quality heroin was scarce.[2] Its use was complicated by needle disease, emboli, overdoses, chemically dependent neonates, and multiple other problems when used in combination with other drugs. Talwin now has fallen out of use because now it is marketed only as Talwin Nx (with Nalaxone) to reverse the narcotic effect.

Hallucinogens

LSD, Mescaline, Psilocybin, MDA, DMT

Nomenclature

Generic (and Trade) Names	Street Names
LSD	Acid, blotter, blue devil, mickies, window panes
MDA, MDMA	Ecstasy
DMT	Businessman's lunch, snuff
Mescaline	Peyote, buttons, mesc, moon
Psilocybin	Magic mushrooms, shrooms

Drug Effects

Pathophysiological: Altered state of consciousness, and psychedelic experiences with profound changes in body image and space/time perception, sensory mixing, as well as dissolution of ego boundaries. No known physical addiction. The phenylethylamine hallucinogens, like mescaline, have considerable sympathomimetic action. In overdose, they may produce tachycardias, arrhythmias, etc. LSD is relatively safe in overdose, with amounts up to 1,000 times the usual dose not being fatal to humans.

Psychological: Disillusion of the ego boundary and consequences thereof, and feelings of a mystical experience. Visual and sensory distortion, altered states of perception occur.

Social Context

To achieve religious or mystic experiences. More recently DMT, MDA, MDMA have become fad drugs, easily available to most adolescents.

Common Presenting Symptoms

Acute

Visual, sensory, and auditory hallucinations
Hypotension or hypertension
Hypothermia
Disorientation of person, place, and time
Emotional upsets
Mania

Chronic

As with acute phase

Drug Characteristics

Administration: Primarily orally; putting the drug into the eye.

Absorption: Can be absorbed in the stomach, through the cornea of the eye, or through the mucosa of the mouth.

Distribution: Weak bases distributed throughout the body.

Metabolism: Mostly metabolized by the liver.

Elimination: Excreted in the bile or the urine.

Tolerance: Develops in all of the hallucinogens within a period of 2 weeks; with daily administration of LSD the tolerance is significant with a cross tolerance to other hallucinogens.

Approximate detection time in urine after use: Varied, according to drug taken.

Special Concerns

A significant behavioral toxicity occurs with the drug actions which may lead patients to think they can fly, walk in front of cars, or have super-human powers. This means that custodial care of the patient intoxicated with hallucinogens is very important. The most serious effects of hallucinogens is the psychological toxicity rather than physiological toxicity. Excessive anxiety may persist; this is responsive to diazapam. Be certain to rule out other drug toxicities, with particular importance given to PCP or other delirients.

Withdrawal Concerns: By and large there are no medical opinions that there are chronic effects.

DMT, or dimethyltryptamine, gives an extremely potent and instantaneous hallucinogenic effect when administered.

MDMA or 3, 4-methylenedioxymethamphetamine, has recently appeared in the United States. It is popular among college students and is remarkably potent.

Treatments: Drug therapy is not recommended for patients under the influence of hallucinogens. A "talk-down" is most effective; however, if drugs are required due to excessive agitation, Valium/diazepam is recommended.

PCP

Nomenclature

Generic (and Trade) Names	Street Names
Phencyclidine	PCP, angel dust, crystal

Drug Effects

Pathophysiological: As with other hallucinogens.

Psychological: As above with other hallucinogens, although this is a delirient which can cause severe and horrible negative experiences.

Social Context

Personal and social use: Initially was sold as organic mescaline or THC.

Common Presenting Symptoms

Acute

Dream-like states
Considerable distortion of reality
Horrible nightmares
Paranoia in psychotics

States of agressive behavior
Mental confusion

Chronic
Decreased concentration
Sleeplessness
Psychosis

Drug Characteristics

Administration: Smoking, injection, as well as oral and nasal routes.
Absorption: Well-absorbed by all routes.
Distribution: A weak base; distributed in all body tissues, including fat and muscle.
Metabolism: PCP is metabolized in the liver.
Elimination: Excreted by the kidneys via the urine, and feces.
Tolerance: Tolerance to PCP has not been reported.
Approximate detection time in urine after use: Up to 200 hours.

Special Concerns

Violent psychotic behavior which can cause trauma.

Detoxification: PCP is a weak base and excreted via the urine. Excretion via the urine can be increased by acidifying the urine. Small amounts present no problems, and only environmental support may be necessary. In larger doses, be aware that the process may take weeks to complete as PCP is stored in fat. Also be aware that the enzymes metabolizing PCP become saturated. Further, the apparent psychotic behavior can be seen for weeks after treatment. As the patient begins to exercise vigorously, the increased perfusion to the fat tissue begins removing the PCP, and psychotic episodes have occurred at this point in time.

Belladona Alkyloids

Nomenclature

Generic (and Trade) Names	Street Names
Benztropine (Cogentin)	-------
Atropine	-------
Scopolamine	-------
Artane	-------
Jimson weed	-------
Antihistamines (Benadryl, Phenergan)	-------

These drugs comprise a group of anticholinergics which historically were used to produce delirium, mostly for mystical and religious purposes.

Drug Effects

Pathophysiological: They produce a state of anticholinergic toxic psychosis which may persist for long periods, greater than 24 hours. The effects in children are more serious than in adults or adolescents. In the latter two populations CNS toxicity can produce behavioral deaths, for example, drownings or accidents: also, they may produce convulsions. Peripheral toxicity produces tachycardia and secondary anticholinergic actions on the heart, and hyperthermia secondary to loss of the ability to sweat. Addiction does not occur.

Psychological. Anticholinergic delirium, agitation, and visual hallucinations.

Context of Use

The drugs are usually used by adolescents in combination with alcohol or solvents because they are not able to obtain more desirable psychoactive drugs.

Drug Characteristics

Absorption. Absorption is from the small intestine. Anticholinergic effects slow the gut and prolong absorption.
Distribution: These drugs distribute throughout the body and CNS.
Metabolism: Hepatic.
Elimination: Via urine and feces.
Tolerance: Does not occur.
Approximate detection time in urine after use: Varies according to drug.

Special Concerns

Patients with either significant CNS or peripheral toxicity from these compounds should be hospitalized.

Other Hallucinogens

Nutmeg

Nutmeg—contains lysergide, as well as eugenol, a potent hallucinogen. Approximately five milligrams of nutmeg contains enough LSD to keep the user hallucinating for 8-12 hours.[3]

Morning Glory Seeds

These common flower seeds contain hallucinogens such as d-lysergic acid and lysergol. It has been noted that an average-sized adult male who eats five seeds may hallucinate for approximately 16 hours.[3]

Marijuana

Nomenclature

Generic (and Trade) Names	Street Names
Tetrahydrocannabinol	Mary Jane, weed, grass, Acapulco Gold, Pot
Hashish (more potent)	Hash

Drug Effects

Pathophysiological: CNS and cardiovascular. Pulmonary effects are more severe than those from cigarette smoking. Physical dependence is not known to occur, and it is not physically addicting.

Psychological: Marijuana or hashish smoke, when inhaled, produces a high which is different from other drug highs; presents with variable psychological dependency.

Context of Use

Strong social drug, shared commonly at parties. Five percent of high school seniors use marijuana daily. Strong peer pressure may exist to prompt its use.

Common Presenting Symptoms

Acute
- Tachycardia
- Conjunctive injection
- Anxiety
- Paranoia
- Euphoria

Chronic
- Pulmonary irritation
- Cancer risk
- Amotivational syndrome

Drug Characteristics

Administration: Smoking, PO.

Absorption: Smoking is the usual route, although oral absorption is also utilized with a slow 1-6 hour peak, with only 5-20% being absorbed.

Distribution: When smoked, the THC is absorbed within minutes and circulated to the brain and peripheral tissues. THC is lipid-soluble and binds to most body tissues. It is also dissolved in the lipoproteins of the blood.

Metabolism: THC is metabolized by the liver.

Elimination. THC is eliminated in the feces and urine.

Tolerance: Yes, to the biological effects.

Approximate detection time in urine after use: 120 hours (single dose); 240 hours (daily use); 336 - 720 hours (chronic daily use)—metabolites detected in all.

Special Concerns

Adverse Effects
1. Inhalation of marijuana smoke probably carries a cancer risk at least approximate to an equal number of tobacco cigarettes smoked in a similar way.
2. Decreased sexual drive with chronic use.
3. Chromosomal changes in circulating white cells.
4. See Table 6-7 for THC-drug effects.

Table 6-7. Drug-THC Interactions

THC and Barbiturates[1,2,3]
- Initially antagonistic, later synergistic
- Decreased motor performance
- Decreased respiration
- Increased barbiturate sleeping time

THC and Diazepam[4,5]
- Increased drowsiness
- CO_2 dose-response curve shifts right
- Increased Diazepam sleeping time

THC and Amphetamines[6,7,8]
- Increased heart rate
- Increased blood pressure

THC and Atropine[9]
- Pressor response
- Increased heart rate

THC and Narcotics[10,11,12]
- Increased narcotic analgesia (animals)
- Increased morphine respiratory depression

Caffeine and Tobacco

Caffeine

The widespread acceptance and popularity of caffeine products hide the fact that it is a commonly abused CNS stimulant. It inhibits the phosphodiesterases, causing increased levels of cGMP and cAMP. Caffeine also causes increased gastric motility, diuresis, and gastric acid secretion.

Tolerance to caffeine develops easily, and physical dependence occurs in heavy users. Abrupt halt of caffeine use may result in withdrawal symptoms.

Chronic use of caffeine may cause disease of the kidney and/or liver. Lethal doses can occur, especially with children.

Overdose and Withdrawal

Anxiety
Arrhythmias, tachycardia
Headaches
Nausea
Abdominal cramping
Irritability
Increased yawning
Restlessness
Lethargy

Nicotine

Nicotine is another widely abused drug that acts as a CNS stimulant. It exerts its effects by transiently stimulating, then blocking, CNS receptors and autonomic ganglia. Most of its effects are peripheral.

Nicotine is physically and psychologically addicting, withdrawal syndromes may occur once the user quits.

Tobacco which contains nicotine can be smoked, "dipped," or sniffed through the nose. Smoking has been the focus of considerable attention, because of its risks for cerebrovascular, pulmonary, and gastrointestinal diseases. Smokeless tobacco, or "dip," is currently a popular trend; however, the practice of placing the tobacco between cheek and gum, or dipping, has been strongly associated with an increased incidence of oral cancer.

Withdrawal[1]

Irritability
Headache
Loss of concentration
Tremors
Decreased heart rate
Increased body weight

Summary

Drug and alcohol abuse among adolescents is one of the greatest health problems facing our nation today. The federal government has pledged full support in fighting this epidemic. As primary care practitioners we will continually be in a position to recognize and treat drug and alcohol abuse. It is important to keep up with not only new drugs but trends involving the use of these drugs.

Evaluation

Because of the nature of this unit of study, little can be done via evaluation activities to promote synthesis of the information presented. In many aspects this unit of study is a book of lists and a reference work. Rather than try to quiz you on the knowledge you have gained, we encourage you to seek ways to keep that knowledge current. Therefore, we suggest you pursue one of the two following activities or generate a similar awareness plan.

1. Organize (or participate in) a substance-abuse journal club. This activity should include as participants not only fellow physicians but also substance-abuse specialists, and perhaps individuals such as teachers, police, and others who are streetwise. The purpose of such a group would be to remain abreast of both current understandings of drug actions and treatment and also "what's on the street."
2. Begin a literature file containing medical and popular articles on current trends in drug use and methods of substance abuse treatment.

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Sedative-Hypnotics and Depressants

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Barbiturates

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