

Inhalant abuse in children and adolescents

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INTRODUCTION — Inhalants are substances with significant acute and chronic toxicity that are abused for their intoxicating properties. They have a wide variety of chemical structures with the majority being either hydrocarbons (aliphatic, aromatic, or halogenated), nitrites, or [nitrous oxide](#) [1]. Inhalants are similar in that they are all volatile substances, highly lipid soluble, and readily absorbed across the pulmonary bed.

Inhalant abuse is a common problem in adolescents due to several factors:

- Inhalants are readily accessible: an average home has between 30 and 50 products with abuse potential.
- They are inexpensive and legal to buy and possess.
- The perceived risk of use is low [2].

Because of these factors, inhalants are frequently the first drugs of abuse used by children and adolescents [1,3]. In addition to being a common practice, inhalant abuse is a serious problem as it is associated with permanent brain injury, cardiac dysfunction, liver toxicity, acute renal failure, and death.

The epidemiology, clinical manifestations, toxic effects, and management of inhalant use will be reviewed here. Substances that are snorted (eg, cocaine) or smoked (eg, tobacco, marijuana, cocaine, and opiates) are discussed separately. (See "[Cocaine: Acute intoxication](#)" and "[Cannabis \(marijuana\) use disorders: Clinical features and diagnosis](#)" and "[Cannabis \(marijuana\) use disorders: Epidemiology, comorbidity, and pathogenesis](#)" and "[Cannabis \(marijuana\) use disorders: Treatment, prognosis, and long-term medical effects](#)" and "[Opioid intoxication in children and adolescents](#)".)

EPIDEMIOLOGY — In the United States, inhalant abuse is common with more than 13 percent of middle school and high school students reporting having used inhalants in their lifetime [4]. An estimated 800,000 US children experiment with inhalants annually [5]. Unlike nearly all other classes of drugs, their use is most common among younger adolescents with use peaking between 7th and 9th grade [2].

The incidence of inhalant abuse in children 12 to 18 years old has fluctuated over the past 25 years based on surveys of adolescents. Incidence of use tripled between 1983 and 1993 [6], peaking in 1995, and then showed a steady decline until 2002. Since that time, incidence has gradually increased [2]. Unlike other drugs of abuse, male and female adolescents report a similar rate of inhalant abuse [5]. In the past, Native American children were at higher risk for inhalant abuse [7,8]. However, rates of abuse have fallen steadily in this group since 1985 and are now at levels comparable to the general population. Education on prevention and treatment is thought to be responsible for this trend [9]. A slightly higher incidence of inhalant abuse in rural communities continues to be noted [10,11].

Similar to national survey data, exposures in 12 to 17 year old patients reported to US regional poison control centers declined from 73 cases per million in 1993 to 33 cases per million in 2003, and the peak age of exposure was 14 years [11]. Among all patients, there were 167 deaths out of 35,453 exposures between 1993 and 2008. Butane, propane, and air fresheners were associated with the highest fatality rate. Males comprised 73 percent of all cases reported to poison centers suggesting that boys may have riskier inhalant abuse behaviors.

Numerous studies have demonstrated significant mental health and behavioral comorbidities among patients who are inhalant abusers. They are more likely to have an episode of major depression [5,12,13], suicidality [14], conduct disorder [12], and are at an increased risk for future drug abuse problems [15].

MECHANISM OF ACTION — Volatile hydrocarbons and **nitrous oxide** have a similar mechanism of action. Like **ethanol** and other inhalational anesthetic agents, they are highly lipid soluble. They are rapidly absorbed across the pulmonary bed into the bloodstream and are distributed throughout the body [16]. Neurons, which have a high lipid content, are particularly susceptible to the solvent properties of these compounds [17,18].

These inhalants act as central nervous system (CNS) depressants. CNS depression is thought to be mediated by alteration of neuronal membrane function at glutamate or gamma amino butyric acid receptors [19-21].

These inhalants produce an effect within seconds that typically lasts 15 to 45 minutes. There have been rare cases of prolonged symptoms when large quantities have been inhaled [22]. Initial euphoria is followed by lethargy. Judgment and coordination are

impaired [23]. Intoxication is maintained through repeated use. Symptoms of tolerance to the effects of inhalants and physiologic withdrawal have been described [1].

Nitrites produce their pleasurable effects by intense vasodilation which produces a sensation of heat and warmth. Absorption is rapid across the pulmonary bed leading to onset of hypotension and reflex tachycardia within seconds of inhalation. Effects are brief, lasting less than five minutes. Nitrites are also used to enhance sexual pleasure by prolonging penile erection and promoting anal sphincter relaxation.

TECHNIQUES (SNIFFING, HUFFING, OR BAGGING) — In the United States, the most frequently used inhalants include glue, shoe polish, or toluene (30 percent); gasoline or lighter fluid (25 percent); **nitrous oxide** or "whippits" (25 percent); and spray paints (23 percent) [5]. Each of these substances may contain more than one toxic compound (table 1) [1].

Inhalants may be sniffed directly from a container or sprayed directly on to a heated surface to enhance vaporization ("sniffing") [24]. Volatile liquid substances also may be inhaled from a saturated cloth that is held under the nose or near the mouth ("huffing"), or from a bag that is placed over the mouth, nose, or head ("bagging"). The risk of asphyxia is increased with bagging because the partial pressure of hydrocarbon displaces **oxygen** in the alveoli. The possibility of a suicide attempt should be considered in an individual who "bags" inhalants, particularly when the bag is placed over the head [25]. The concentration of the inhaled substance increases from sniffing, to huffing, to bagging.

Nitrous oxide is abused most commonly as "whippits". These are small, cylindrical metal bulbs with a pierceable end that contains compressed nitrous oxide, which are meant for use as a propellant for whipped cream makers. When misused, the end is pierced with a "cracker" and the escaping gas is captured in a balloon and then inhaled. Nitrous oxide can also be sniffed from whipped cream canisters.

The desired effects of inhalant abuse include euphoria, lightheadedness, and a general state of intoxication similar to that produced by alcohol or marijuana. The effects usually last for only 15 to 30 minutes, but can be sustained by continuous or repeated use [26].

RECOGNITION OF INHALANT ABUSE — Inhalant abuse frequently goes undetected. Clues to inhalant abuse include chemical odors on the breath, skin, or

clothes, and empty solvent containers or bags, rags, or gauze in the child's possession or trash [25]. "Glue-sniffer's rash" is an eczematoid dermatitis with erythema, inflammatory changes, and pruritus that occurs in the perioral area and extends to the midface. It is caused by the drying effects of hydrocarbons [27]. Symptoms and signs of inhalant use are listed in the table (table 2).

TOXICITY AND CLINICAL FINDINGS BY AGENT — Although the vast majority of children who abuse volatile inhalant substances do not seek or require medical attention, inhalant abuse is potentially life-threatening. Death may result from asphyxia, suffocation, choking on vomitus, careless or dangerous behavior in potentially dangerous settings, and sudden sniffing death [28,29] seen with hydrocarbon abuse, especially halogenated hydrocarbons. Diagnosis of inhalant abuse is primarily based on history or circumstance (eg, patient found unresponsive with toxic inhalant or inhalant apparatus nearby). (See 'Techniques (sniffing, huffing, or bagging)' above.)

Hydrocarbons — Household products abused as inhalants typically contain a mixture of hydrocarbons. They are categorized by structure: aliphatic, aromatic, and halogenated. (See "Hydrocarbon poisoning".)

Aliphatic compounds are straight-chain compounds and include butane, propane, kerosene, and mineral seal oil. Gasoline is a mixture of aliphatic hydrocarbons that also may contain other substances such as xylene, toluene, benzene, naphthalene, or lead [16,30].

Aromatic hydrocarbons are cyclic compounds containing a benzene ring and are used as industrial solvents; benzene, toluene, and xylene are encountered most commonly. Toluene is found in a large number of household products including glues, adhesives, acrylic paints, paint thinners, and automotive products [16].

Halogenated hydrocarbons include fluorinated hydrocarbons (freons) and chlorinated hydrocarbons (carbon tetrachloride, trichloroethylene, trichloroethane). They are used as solvents, degreasers, and spot removers, and in the dry cleaning industry. Freons are widely used as refrigerants and in fire extinguishers [31-33].

The most important toxicities are cardiac and neurological, although hydrocarbons are toxic to essentially all body systems. Certain agents are more closely associated with a particular toxicity.

CNS effects — Acute CNS effects include slurred speech, ataxia, disorientation, headache, hallucinations, agitation, violent behavior, and seizures [6,28,34-36]. Generalized CNS depression may involve the respiratory centers of the brain, rarely causing respiratory arrest and death [37]. Toluene intoxication can cause temporary or progressive cerebellar dysfunction and cranial neuropathies [1,6].

The long-term CNS effects are equally worrisome and include neurocognitive impairment, cerebellar dysfunction, and peripheral neuropathy [25,38]. The neurologic findings may be related to loss of brain mass or abnormal perfusion. Computed tomography (CT) and magnetic resonance imaging (MRI) of inhalant users demonstrates loss of brain mass and degeneration of white matter (also called "toxic leukoencephalopathy"), respectively [39-42]. Single photon emission computed tomography (SPECT) in long-term inhalant users demonstrates abnormal perfusion [43].

Abuse of gasoline can lead to persistent peripheral neuropathy and myopathy with myoglobinuria and creatine kinase elevation [44-47]. In addition, Parkinsonism can be caused by octane-enhancing additives [48], and lead poisoning may occur if lead-containing gasoline is inhaled. (See "[Childhood lead poisoning: Clinical manifestations and diagnosis](#)".)

Cardiovascular effects — Arrhythmias, myocarditis, or myocardial infarction may rarely occur with acute or chronic use [7,49]. Chronic heavy use of toluene has been reported to result in a statistically significant increase in QT dispersal, a marker for sudden death in a variety of clinical conditions [50].

"Sudden sniffing death" where a patient has cardiovascular collapse associated with inhalation of volatile compound has been reported with all classes of hydrocarbon, but is most commonly seen in children who abuse halogenated hydrocarbons [29,51]. Although a rare event, it is unpredictable and can occur in first time users. Commonly, it follows exertion or masturbation, both of which lead to increased catecholamine release [29,52]. It is thought to be due to sensitization of the myocardium to catecholamines that is possibly accentuated by hypoxia associated with inhalant abuse [1,27,29,37].

Other effects

- Pulmonary effects — Hypoxia may result from displacement of [oxygen](#) in the

alveoli, particularly with butane, isobutane, propane, and [nitrous oxide](#) [34], or suffocation when the patient huffs using a plastic bag. Pneumonitis with surfactant dysfunction, bronchospasm, or noncardiogenic or hemorrhagic pulmonary edema may occur [27,31,49,53]. Exposure to fluorocarbon may cause a reactive airway syndrome similar to asthma [54]. Pneumothorax may occur if gas is inhaled directly from a pressurized tank [25]. (See "[Spontaneous pneumothorax in children](#)".)

- **Gastrointestinal effects** — The gastrointestinal effects of inhalant use include nausea, vomiting, and abdominal cramps [34,49]. Anorexia and loss of weight may occur with chronic abuse [25]. Chlorinated solvents such as trichloroethylene and 1,1,1-trichloroethane are hepatotoxic [55].
- **Renal effects** — Volatile substance use may cause metabolic acidosis, urinary calculi, and glomerulonephritis [4,14,21,34]. Toluene, in particular, causes metabolic acidosis with profound potassium and phosphate wasting [56,57]. (See "[The \$\Delta\$ anion gap/ \$\Delta\$ HCO₃ ratio in patients with metabolic acidosis](#)", section on '[Toluene inhalation](#)' and "[Causes of hypokalemia](#)", section on '[Nonreabsorbable anions](#)'.)
- **Hematologic effects** — Inhalant abuse, particularly chronic abuse of benzene, may cause aplastic anemia and malignancy (eg, leukemia, lymphoma, multiple myeloma) [25,55,58-60]. Inhalation of methylene chloride (dichloromethane), which is metabolized to carbon monoxide, can result in a clinically important carboxyhemoglobin level [61].
- **Dermatologic effects** — "Glue-sniffer's rash" is an eczematoid dermatitis with erythema, inflammatory changes, and pruritus that occurs in the perioral area and extends to the midface. It is caused by the drying effects of hydrocarbons [27]. Burns may occur when a flammable inhalant ignites.
- **Musculoskeletal effects** — Heavy toluene abuse is associated with generalized muscular weakness (often to the point of quadriparesis) and is typically accompanied by metabolic acidosis, profound hypokalemia, hypophosphatemia, rhabdomyolysis, and elevated creatine kinase. These metabolic abnormalities are caused primarily by the conversion of toluene to hippuric acid, with the subsequent rapid excretion of hippurate in the urine [62]. (See "[The \$\Delta\$ anion gap/ \$\Delta\$ HCO₃ ratio in patients with metabolic acidosis](#)", section on '[Toluene inhalation](#)'.)
- **Pregnancy and postnatal effects** — Inhalant use during pregnancy may increase the risk of spontaneous abortion, premature delivery, or fetal malformation [63-65]. Toluene, in particular, is associated with oral clefts, micrognathia,

microcephaly, growth deficiency, and developmental delay [66]. Infants born to mothers who used inhalants during pregnancy may have symptoms of withdrawal [67].

- Miscellaneous — Trauma can result from falls, drowning, or motor vehicle accidents during the period of intoxication [68,69].

Nitrous oxide — **Nitrous oxide** (NO) is used as a propellant in canisters of whipped cream, as a power booster in automobiles and motorcycles, and as a sedative/amnestic agent for painful medical and dental procedures. It usually is inhaled from a balloon [23]. Neurologic, hematologic, and reproductive toxicity may result from exposure to NO.

- Neurologic effects — Neurologic toxicity is well documented in patients who abuse NO. Abuse is associated with a myriad of neurological findings including polyneuropathy [26,70-72], ataxia [71,73,74], and psychosis [73,75]. Neurotoxicity is potentially reversible with B12 supplementation and abstinence from NO.

The neurotoxicity of NO is due to its effects on **vitamin B12**. Vitamin B12 is a coenzyme of methionine synthase. **Nitrous oxide** converts vitamin B12 from the active monovalent form to the inactive bivalent form, thereby causing an irreversible inhibition of methionine synthase. Methionine synthase is a ubiquitous cytosolic enzyme that plays a crucial role in the generation of methyl groups for the synthesis of DNA, RNA, and myelin, among other products. The clinical effects are similar to the subacute combined degeneration syndrome associated with pernicious anemia [76]. Nitrous oxide is known to precipitate vitamin B12 deficiency when used on a chronic basis or acutely in patients with marginal stores of vitamin B12 such as the elderly or malnourished [74].

- Hematologic effects — NO has the potential for causing megaloblastic anemia on the basis of its interaction with B12. This has not been reported in patients who abuse NO, although it has been noted when NO was used as an anesthetic agent [77,78].
- Reproductive effects — Given NO effects on **Vitamin B12** and methionine synthase, it is plausible to assume that NO may have reproductive health risks. Animal studies have demonstrated fetotoxicity with prolonged exposure to NO. Human studies are difficult to interpret given the number of confounding variables [21]. However, a study of occupationally exposed healthcare workers has demonstrated a dose-dependent correlation between NO exposure and DNA

damage [79].

- Pulmonary effects — Pneumothorax may occur if gas is inhaled directly from a pressurized tank [25,80]. (See "[Spontaneous pneumothorax in children](#)".)

Nitrites — Alkyl nitrites (amyl, butyl, and isobutyl nitrites) have been used and abused for nearly 150 years [81]. [Amyl nitrite](#) is available by prescription for treatment of angina pectoris and as part of the "cyanide kit" for treatment of cyanide toxicity. It is available in a glass ampule encased in a woven absorbent covering. The ampule is broken or "popped" and the covering held close to the nose and inhaled. Butyl and isobutyl nitrites (also known as "Rush" and "Climax") are used as room deodorizers, particularly for locker rooms. They also are sold as "liquid incense." They were banned by the Anti-Drug Abuse Act of 1988, but are still available illegally, as are isopropyl and cyclohexyl nitrites [81]. They are typically sniffed directly from the container.

Inhalation of nitrate causes smooth muscle relaxation resulting in peripheral vasodilatation, flushing, and hypotension with reflex tachycardia. Dilation of cerebral blood vessels causes an increase in intracranial pressure (the "rush" reportedly experienced by users), headache, nausea, and syncope. Skin irritation, tracheobronchitis, and allergic reactions with wheezing and pruritus may occur [82,83].

Inhalation of nitrite may cause acute acquired methemoglobinemia, although this toxicity is much more common after oral ingestions [81,84]. (See "[Extrinsic nonautoimmune hemolytic anemia due to drugs and toxins](#)", section on 'Nitrites'.) Patients with acute acquired methemoglobinemia are symptomatic because the acute impairment of [oxygen](#) delivery to tissues does not allow sufficient time for compensatory mechanisms. Early symptoms include headache, fatigue, dyspnea, and lethargy. At higher methemoglobin levels (eg, greater than 70 percent), respiratory depression, altered consciousness, shock, seizures, and death may occur [16,81,85,86].

In addition, nitrite vapors are highly flammable, and serious burn injuries can occur if the substance comes into contact with a candle, cigarette, or other open flame.

ANCILLARY STUDIES — All patients with inhalant intoxication should have the following performed:

- Pulse oximetry
- Electrocardiogram and continuous cardiac monitoring for arrhythmias

- Urine screen for drugs of abuse

In addition, the following may be performed, especially in adolescents suspected of chronic abuse [17]:

- Complete blood count to evaluate for bone marrow suppression (benzene abuse)
- Serum electrolytes to identify hypokalemia and metabolic acidosis (toluene abuse)
- Liver enzymes (AST [aspartate aminotransferase] and ALT [alanine aminotransferase]), blood urea nitrogen, and serum creatinine to detect liver or renal impairment (halogenated hydrocarbons)
- Urine rapid dipstick and microscopic urinalysis to assess for renal tubular acidosis (chronic toluene use) or findings consistent with interstitial nephritis (halogenated hydrocarbons). (See "[Overview of renal tubular acidosis](#)".)

Methemoglobin levels should be obtained if nitrite abuse is suspected or if clinical features of methemoglobinemia are evident (eg, chocolate brown venous blood or cyanosis that does not resolve with supplemental oxygen). (See "[Clinical features, diagnosis, and treatment of methemoglobinemia](#)", section on 'Clinical features'.)

A venous blood lead level is appropriate in patients in whom volatile abuse using leaded gasoline is suspected.

A chest radiograph should be obtained in patients with hypoxemia, rales, or respiratory distress. Abnormalities on chest radiograph may develop as late as 24 hours after exposure in symptomatic patients [27]. They include increased bronchovascular markings, bibasilar and perihilar infiltrates, and pneumatoceles [27].

Although exposure to some hydrocarbons may be confirmed by detection of urinary metabolites (eg, trichloroethanol after chlorinated hydrocarbon exposure, hippuric acid after toluene exposure) or directly measured in the blood (eg, toluene), these laboratory studies are not rapidly available and do not change management priorities. Thus, the diagnosis of hydrocarbon exposure is based on clinical features.

DIFFERENTIAL DIAGNOSIS — Many other toxins may cause altered mental status or cardiac arrhythmias in overdose. Often, characteristic clinical findings can help differentiate these from inhalant abuse (table 3). (See "[Approach to the child with occult toxic exposure](#)".)

When hydrocarbons are inhaled, the characteristic sweet solvent odor of halogenated

hydrocarbons or the "glue" odor of toluene may be detectable on the breath.

TREATMENT — Management of acute inhalant intoxication is supportive. Maintenance of cardiorespiratory function and removal of the child from the source of the toxin (eg, bottle, rag or bag, or contaminated clothing) are of primary importance.

Supportive care — Supplemental 100 percent oxygen by a nonrebreather face mask is administered to treat hypoxia.

- Airway and breathing — Respiratory depression often responds transiently to tactile stimulation of the patient. However, clinicians should proceed with endotracheal intubation and mechanical ventilation if there is any doubt about the patient's ability to breathe adequately on their own or if pulmonary aspiration poses a significant risk.
- Circulation — Children with ventricular arrhythmias should receive countershock treatment according to standard protocols (algorithm 1 and algorithm 2). (See "Defibrillation and cardioversion in children (including automatic external defibrillation)".)

Children who have inhaled halogenated hydrocarbons may develop ventricular arrhythmias in response to parenterally administered epinephrine or other catecholamines (eg, norepinephrine) because these treatments can precipitate or worsen arrhythmias in the irritable myocardium [49,60]. In contrast, amiodarone has been successfully used to treat butane-induced ventricular fibrillation [87]. We suggest that these patients receive intravenous amiodarone (5 mg per kilogram) or lidocaine (1 mg per kilogram) instead of catecholamines.

For patients who persist with ventricular arrhythmias despite the use of Pediatric Advanced Life Support protocols, the clinician may attempt administration of propranolol or esmolol, which have reversed arrhythmias in patients poisoned with trichloroethylene [88,89].

Antidotes — Specific antidotes may be necessary to treat methemoglobinemia caused by nitrite exposure or lead toxicity due to inhalation of leaded gasoline:

- Patients with methemoglobinemia who are symptomatic should be treated with high-dose oxygen and intravenous methylene blue. (See "Clinical features, diagnosis, and treatment of methemoglobinemia", section on 'Acquired

methemoglobinemia'.)

- Lead toxicity may require chelation therapy, depending on the results of lead levels obtained from whole blood. (See "[Childhood lead poisoning: Treatment](#)".)

Psychiatric care — Acutely, patients with findings of inhalation abuse should undergo screening for depression and suicidality [14].

Long-term management includes referral for addiction treatment and a formal period of detoxification if indicated [25]. Chronic complications of abuse may resolve if the patient remains drug-free. Polydrug use and coexisting psychopathology are common occurrences, complicating the treatment of these children. Residential treatment may improve outcome. Systematic data comparing different approaches to treatment are lacking [1].

Disposition — Patients with significant toxicity marked by central nervous system findings (eg, coma, seizures) or cardiac arrhythmias warrant hospital admission to a unit with pediatric critical care capability.

Children who receive treatment for methemoglobinemia or lead toxicity may also require admission depending on the degree of toxicity.

Children who express suicidal thoughts need urgent psychiatric evaluation for possible admission to a mental health facility.

Patients who are asymptomatic in the emergency department or have mild symptoms (eg, lethargy) that quickly resolve may be discharged home as long as appropriate mental health and primary care follow-up are assured.

PREVENTION — The American Academy of Pediatrics encourages pediatricians to increase their awareness of the clinical features and complications of inhalant abuse and to promote education about the health hazards of inhalants to children, adolescents, parents, teachers, and vendors of volatile substances [28].

The Massachusetts Department of Public Health has developed a suggested action plan for prevention of inhalant abuse (www.state.ma.us/dph/inhalant). Their suggestions include the following:

- Review purchases of school supplies and substitute water-based products for solvent-based products whenever possible. Explain that the school is looking for

ways to reduce indoor air pollution. Describing solvent-based products as inhalants or drugs may arouse the curiosity of students.

- Closely monitor the use of solvent-based products and gases; in schools, such products should be checked out and in.
- Provide information to school faculty, staff, and nurses.
- Educate parents about the dangers of inhalant abuse.
- Review the appropriate use and consequences of misuse of solvents and gases whenever these products are used (eg, vocational programs, science, art).
- Develop a plan of action to treat students who use inhalants.

SUMMARY AND RECOMMENDATIONS — Inhalant abuse is common. Unlike nearly all other classes of drugs, their use is most prevalent among younger adolescents with use peaking between 7th and 9th grade among school children in the United States. (See 'Epidemiology' above.)

Abuse

- Volatile substances produce a rapid feeling of euphoria and inebriation because of their rapid absorption through the pulmonary vascular bed and their lipophilic properties that allow for quick deposition into the brain. (See 'Mechanism of action' above.)
- The most frequently used inhalants include glue, shoe polish, or toluene; gasoline or lighter fluid; **nitrous oxide** or "whippits"; and spray paints. Each of these substances may contain more than one toxic compound (**table 1**). Common methods of abuse involve sniffing, huffing, or bagging to concentrate the inhaled substance. (See 'Epidemiology' above and 'Techniques (sniffing, huffing, or bagging)' above.)

Clinical findings — Inhalant abuse frequently goes undetected. Clues to inhalant abuse include chemical odors on the breath, skin, or clothes and empty solvent containers or bags, rags, or gauze in the child's possession or trash. Children may also display characteristic skin changes (eg, glue sniffer's rash) (**table 2**). (See 'Recognition of inhalant abuse' above.)

- Inhalant abuse is potentially life-threatening. Death may result from asphyxia, suffocation, choking on vomitus, careless or dangerous behavior in potentially dangerous settings, and sudden sniffing death seen with hydrocarbon abuse. (See 'Toxicity and clinical findings by agent' above.)

- Cardiac arrhythmias and central nervous system dysfunction (inebriation, agitation, seizures) are the most concerning acute toxic effects of volatile inhalants, especially hydrocarbons. (See 'Toxicity and clinical findings by agent' above.)
- Additional findings or chronic toxicity varies by agent including (see 'Toxicity and clinical findings by agent' above):
 - - Methemoglobinemia (nitrites)
 - - Lead toxicity (leaded gasoline)
 - - Polyneuropathy and megaloblastic anemia (nitrous oxide)
 - - Muscle weakness, renal tubular acidosis (toluene)
 - - Carbon monoxide poisoning (methylene chloride)

Management

- Management of acute inhalant intoxication primarily consists of support of airway, breathing, and circulation (see 'Supportive care' above):
 - - Supplemental 100 percent oxygen by a nonrebreather face mask should be administered to treat hypoxia.
 - - Clinicians should proceed with endotracheal intubation and mechanical ventilation if there is any doubt about the patient's ability to breathe adequately on their own or if pulmonary aspiration poses a significant risk.
- Children with ventricular arrhythmias should receive treatment according to standard protocols (algorithm 1 and algorithm 2).

(See "Defibrillation and cardioversion in children (including automatic external defibrillation)".)

- Children with cardiac arrhythmias caused by inhalation of halogenated hydrocarbons may have worsening cardiovascular status in response to epinephrine or other catecholamines (eg, dopamine, norepinephrine). We suggest that these patients receive intravenous amiodarone (5 mg per kilogram) or lidocaine (1 mg per kilogram) instead of catecholamines (**Grade 2C**).
- Specific antidotes may be necessary to treat methemoglobinemia caused by nitrite exposure or lead toxicity due to inhalation of leaded gasoline. (See "Clinical features, diagnosis, and treatment of methemoglobinemia", section on 'Acquired

methemoglobinemia' and "Childhood lead poisoning: Treatment".)

- Acutely, patients with findings of inhalation abuse should undergo screening for depression and suicidality. Long-term management includes referral for addiction treatment and a formal period of detoxification if indicated. (See 'Psychiatric care' above.)