American Academy of Pediatrics
DEDICATED TO THE HEALTH OF ALL CHILDREN™

CLINICAL REPORT

# Inhalant Abuse

Guidance for the Clinician in Rendering Pediatric Care

**Janet F. Williams, MD, Michael Storck, MD, and the Committee on Substance Abuse and Committee on Native American Child Health**

## ABSTRACT

Inhalant abuse is the intentional inhalation of a volatile substance for the purpose of achieving an altered mental state. As an important, yet-underrecognized form of substance abuse, inhalant abuse crosses all demographic, ethnic, and socioeconomic boundaries, causing significant morbidity and mortality in school-aged and older children. This clinical report reviews key aspects of inhalant abuse, emphasizes the need for greater awareness, and offers advice regarding the pediatrician's role in the prevention and management of this substance abuse problem.

## TYPES OF CHEMICALS AND PRODUCTS ABUSED

The term "inhalant" encompasses a wide range of pharmacologically diverse substances that readily vaporize. Most other substances of abuse are classified by grouping together substances that share a specific central nervous system action or perceived psychoactive effect, but inhalant substances that are abused are grouped by having a common route of drug use. Inhalant abuse, sometimes referred to as solvent or volatile substance abuse, can be better understood when the expansive list of inhalants is classified into 3 groups on the basis of what is currently known pharmacologically: group I includes volatile solvents, fuels, and anesthetics; group II includes nitrous oxide; and group III includes volatile alkyl nitrites (Table 1). This classification is also consistent with reported differences in user populations, patterns of abuse, and associated problems seen clinically.[1–3] Drugs that do not readily vaporize at room temperature, such as cocaine, heroin, nicotine, or alcohol, can also be abused through inhalation, but characteristic pharmacologic properties distinguish these substances from inhalants.

Inhalant abusers use volatile products that are capable of producing a quick and generally pleasurable sensory experience, or "high," with rapid dissipation and minimal "hangover" symptoms. Inhaled substances are widely available, convenient, inexpensive, easily concealed, and legal for specific intended uses but are intentionally misused by abusers. Many of these qualities are important factors that promote use in a young age group, because children have less sophisticated resources for acquiring alternative substances of abuse. The most commonly abused inhalants are the group I aliphatic, aromatic, or halogenated hydrocarbons found in thousands of commonly used and readily available consumer products. Virtually any hydrocarbon can have mind-altering effects when inhaled in large doses. Nitrous oxide or "laughing gas" is diverted from medical or dental anesthesia use and sold in balloons for inhalation or is simply inhaled from whipped cream aerosol cans. Alkyl nitrites or "poppers" are also abused; prototypically, amyl nitrite ampules intended to treat angina are "popped" open and inhaled.

www.pediatrics.org/cgi/doi/10.1542/peds.2007-0470

doi:10.1542/peds.2007-0470



**Key Words**
substance abuse, inhalant abuse, inhalants, solvent abuse, inhaled nitrites, drug abuse

**Abbreviations**
NSDUH—National Survey on Drug Use and Health
MTF—Monitoring the Future
TESS—Toxic Exposure Surveillance System

PEDIATRICS (ISSN Numbers: Print, 0031-4005; Online, 1098-4275). 

TABLE 1 **Pharmacologic Classification of Inhalants: Selected Common Street Names and Chemical Content of Product Examples**

| |
|---|
| I. Volatile solvents, fuels, and anesthetics (air blast, discorama, hippie crack, medusa, moon gas, oz, poor man's pot) |
| Solvents: toluene, acetone, methylene chloride, ethyl acetate, TCE in paint thinner, paint and polish removers, correction fluid, and felt-tip marker fluid; TCE and tetrachloroethylene in degreasers, spot removers, and dry-cleaning fluids; toluene, hexane, TCE, ethyl acetate, and methyl chloride in glues and rubber cement; propellants and solvents such as butane, propane, chlorofluorocarbons, hydrocarbons in aerosol spray paint, computer/electronics-cleaning spray, spray deodorant, hair spray, vegetable-oil cooking spray, air-freshener spray, fabric-guard spray, and analgesic sprays |
| Fuels: butane or propane lighters or pressurized fuel tanks, gasoline, racing car octane boosters, refrigerants |
| Anesthetics: ether, halothane, enflurane, ethyl chloride |
| II. Nitrous oxide (laughing gas, buzz bomb, shoot the breeze): diverted medical anesthetic, whipped-cream dispenser charger (whippets), whipping-cream aerosol |
| III. Volatile alkyl nitrites (poppers, snappers, boppers, pearls, amys [isoamyl nitrite diverted from medical use], quicksilver, and brand/slang terms Rush, Bolt, Thrust, Climax, Locker Room): videocassette-recorder head cleaner, liquid aroma/liquid incense air fresheners or room odorizers (mostly cyclohexyl nitrite), isobutyl nitrite or butyl nitrite, isopropyl nitrite |

TCE indicates 1,1,1-trichloroethane.

## EPIDEMIOLOGY

Inhalant abuse occurs throughout the world, in industrialized nations as well as developing countries. Several studies have helped define the epidemiology of volatile substance abuse in the United States.[3–11] The peak age of inhalant abuse is 14 to 15 years, with onset in children as young as 5 or 6 years of age. Use typically declines by 17 to 19 years of age but can continue into adulthood. Use by adults may predominate under particular circumstances, such as when certain occupations make abusable solvents, propellants, or anesthetics readily available. Inhaled nitrites have a long history of being abused in certain social settings, particularly when men have sex with men.[3] The type, frequency, and method of volatile substance abuse vary widely in relation to age of the abuser, geographic region, and ease of availability.

The National Survey on Drug Use and Health (NSDUH), an annual survey of drug use in the general US noninstitutionalized civilian population 12 years and older, has documented inhalant abuse initiation by both adolescents and adults.[10] In 2005 and similar to previous years, 72.3% of the 877 000 new volatile substance abusers aged 12 to 49 years were younger than 18 years, with a mean age of 16.1 years. Since 2002, no significant change has occurred in the number of inhalant initiates, the average age of first use, or the rates of inhalant abuse by either youth or adults. This survey again showed no significant male-female difference in lifetime prevalence of inhalant abuse in the 12- to 17-year age group but confirmed a greater prevalence of inhalant abuse by men in the 18- to 25-year age group, suggesting that sustained use of inhalants is more common in males. Comparison of 2004 NSDUH findings with the Canadian Addiction Survey, a telephone survey in early 2004 of Canadian household residents 15 years and older, showed that US residents were 7 times more likely (9.5%) than Canadians (1.3%) to have ever used inhalants.[12]

According to the National Institute on Drug Abuse and the University of Michigan's annual Monitoring the Future (MTF) survey results[11]:

- Prevalence of lifetime inhalant use ("ever used") among 12th-graders has ranged between 10.3% in 1976 (when first included in the survey) and 18.0%, the 1990 peak. The 2006 rate of 11.1% has been stable since 2002.
- Since 1979, prevalence has additionally been reported after adjustment for the underreporting of amyl and butyl nitrite use. Adjusted lifetime prevalence figures remained at or above 17.0% until 1997 before steadily declining and stabilizing near the 2004 low of 11.4%. Adjusted lifetime and annual-use ("at least once in the past year") rates for 12th-graders (11.5% and 4.7%, respectively, in 2006) are among the lowest levels in survey history.
- Roughly similar declines in prevalence of inhalant use have been documented in the 8th- and 10th-grade age groups, which the MTF survey has included since 1991 but does not adjust for possible nitrite use.
- Recent data on perceived harmfulness may be portentous. Since 2001, the percentages of 8th- and 10th-graders who indicated that they "think people risk harming themselves (physically or in other ways) if they try inhalants once or twice" or "try inhalants regularly" have decreased. Past research has shown that decreasing perceived risk of using a drug often precedes an upswing in use of that drug.

MTF survey results have consistently shown that the reported prevalence of inhalant use by 8th-graders has been, on average, approximately 2% to 3% greater than that of 10th-graders, which runs approximately 2% greater than that of 12th-graders. This pattern, which is opposite that of nearly all other abused substances, may simply reflect that early experimentation with this easily acquired drug class is greater in younger age groups, that older students may fail to report inhalant use that occurred in earlier grades, or that many 8th-grade inhalant abusers subsequently drop out of school and are, therefore, no longer included in the survey population.[6,13,14] Research has shown that inhalant use often occurs in conjunction with other risk behaviors and that higher rates of inhalant abuse occur among children who have poor grades or have dropped out of school compared with classmates who remain in good standing at school.[6,13–15]

Although inhalant abuse is more prevalent among

geographically isolated and socioeconomically disadvantaged populations, it crosses all demographic boundaries and occurs in rural as well as urban settings and among all ethnic groups in the United States.[4,6–11] Important universal factors that promote initial experimentation with inhalants and their continued use include peer use and low perceived harm from use.[8,11,16] Inhalant use is often associated with impoverished living conditions, delinquency, criminal behavior, incarceration, depression, suicidal behavior, greater antisocial attitudes, family disorganization and conflict, or a history of abuse, violence, or other substance abuse, including injection drug use.[4,13,15–21]

MTF surveys have documented lower rates of past-year inhalant use among Hispanic compared with white individuals, with the lowest rates consistently among black individuals.[11] Similarly, most other studies have found rates of inhalant use by Hispanic youth to be the same as or lower than use by non-Hispanic white youth.[4–8,10,11] NSDUH data consistently show that rates of inhalant abuse by Asian American youth are among the lowest.[4,10] Inhalant use has been seen as a particularly serious problem among American Indian/Alaska Native youth for many years.[6,10,22] Research to discern factors contributing to inhalant abuse suggests that adverse socioeconomic conditions, including isolation and lower educational levels rather than racial or cultural factors per se, account for the reported higher rates among these minority populations. Eskimo children 10 to 19 years of age living in 14 isolated Bering Strait villages reported a lifetime prevalence of inhalant use of 48%.[23] American Indian youth living on reservations have been shown to have higher rates of inhalant abuse than do either American Indian youth living off reservations or non-Hispanic white youth.[22] Paralleling the decreasing inhalant abuse shown by the MTF studies until 2004, a promising downward trend has also been demonstrated since 1995 by the annual survey of American Indian students living on reservations.[8] Because use of other drugs during much of this time period also decreased, substitution of other drug use for solvent use was not felt to explain the trend. Despite these epidemiologic data, inhalants remain among the least studied groups of abused substances. Much research is needed to understand all aspects of inhalant use, including the neuropharmacologic effects and psychosocial correlates.

## MECHANISM OF ABUSE AND IMMEDIATE EFFECTS OF INHALANTS

Inhalants are abused through a variety of methods, and many "street" terms for this activity have been generated, such as (glue) sniffing, snorting, huffing, glading, and dusting. Product fumes are usually inhaled through the mouth (huffing) or nose (sniffing or snorting) from the original container. Abusers may also inhale vapors from a chemical-saturated rag held to the face or stuffed in the mouth, which is also called huffing. Some aerosols are sprayed directly into the mouth or nose, and volatile solvents can be applied onto the nasal mucosa or a nearby surface such as fingernails or a shirt collar or cuff and then inhaled. "Glading" refers to the inhalation of air-freshener aerosols, whereas a recently coined term, "dusting," refers to the abuse of aerosol computer and personal electronics cleaning products by placing the canister straw into the mouth or nose. Familiar and innocuous containers are often used to help conceal inhalant abuse (eg, inhaling spray paint fumes out of a soft drink can or nitrous oxide–filled balloons). A paper or plastic bag containing the inhalant can be held to the mouth and nose or over the head ("bagging").

Unusual fads in inhalant abuse products or methods have been reported, such as heating volatile substances and inhaling the released vapors, as has been done with certain fertilizers or "snotballs" of rubber cement.[24] Mothballs have been abused by bagging or chewing.[25] Products combining inhalants with nonrespirable toxic ingredients, such as antiperspirants containing the toxic compound aluminum chlorohydrate, can be bubbled through water.[26] Combined alcohol and inhalant abuse by drinking "ocean" has been a periodic problem on and near some southwest American Indian reservations. Mixing water or mouthwash with the contents of a hairspray can, usually AquaNet, containing ethanol, methanol, and a propellant, produces foamy "ocean water" and combined toxicities.[27]

Inhalants are readily absorbed through the lungs, with immediate and brief effects, and then relatively rapidly metabolized predominately through the cytochrome P450 system of the liver. Inhalants, except nitrites, are depressants that act directly on the central nervous system through a wide range of mechanisms yet to be completely elucidated.[1,28] As a group, inhalants most resemble alcohol, whereby different cellular mechanisms are responsible for myriad pharmacologic and toxicologic effects. Opiate receptor involvement likely plays a role in the analgesic effects of nitrous oxide, but there is evidence for γ-aminobutyric acid (GABA)–mediated behavioral effects.[1] Volatile hydrocarbons also have GABAergic effects and a possible role in the inhibition of glutamatergic neurotransmission involving *N*-methyl-D-aspartate (NMDA) receptors.

The immediate effects of inhaling volatile solvents, fuels, anesthetics, or nitrous oxide are similar to the early stages of anesthesia. The user feels an initial stimulating "rush," then is light-headed, disinhibited, excitable, and prone to impulsive behavior. Intoxication lasts only a few minutes but can be extended for several hours by breathing inhalants repeatedly. Slurred speech, dizziness, diplopia, ataxic gait, and disorientation occur as the inhalant dose increases. Euphoria is followed by drowsiness, a lingering headache, and sleep, particularly after repeated cycles of inhalation. Visual hallucinations

are possible with prolonged use. Coma is unusual, because as the user becomes drowsy, exposure to the inhalant is usually terminated before a large enough dose is absorbed to cause severe neurologic and respiratory depression. Mucous membrane irritation may manifest as rhinorrhea, epistaxis, sneezing, coughing, excess salivation, and conjunctival injection. Some patients experience nausea, vomiting, diarrhea, abdominal cramps, dyspnea, or wheezing.[28,29]

Nitrites significantly differ pharmacologically from other inhalants, because instead of direct central nervous system effects, they primarily cause vasodilation and smooth muscle relaxation. The sensations of floating and increased skin tactility as well as warmth and throbbing occur within 10 seconds of inhalation but diminish within 5 minutes. Nitrite abuse may result in tachycardia, flushing, blurred vision, headache, lightheadedness, significant hypotension, syncope, and sufficient methemoglobinemia to cause cyanosis and lethargy.[1,3,29] Other inhalants are used to alter mood, but nitrites are inhaled to enhance sexual feelings, penile engorgement, and anal sphincter relaxation to intensify sexual experience.[3,29]

## MORBIDITY AND MORTALITY

Patterns of inhalant abuse are similar to those of other substance abuse, and users can generally be described as experimenters, intermittent users, or chronic inhalant abusers. Similarly, morbidity and mortality increase as frequency of use increases, with the important exception that "sudden sniffing death syndrome" is a risk during any use, even during initial experimentation. In 1 study, 22% of inhalant abusers whose deaths were attributed to sudden sniffing death syndrome had no history of previous inhalant abuse.[30] Sudden sniffing death syndrome is the leading cause of fatality related to inhalant abuse.

Bass[31] originally described sudden sniffing death and elucidated its pathophysiology. Hydrocarbons and other inhalants "sensitize" the myocardium to epinephrine, and when this hormone is produced in response to any of a number of stimuli, most commonly sudden stress or fright, a fatal cardiac arrhythmia can result. Instead of truly sensitizing the cells, volatile substances stabilize myocardial cell membranes to depolarization. Because of variable individual myocardial cell response and the complex way that myocardial electrical impulses are propagated, greater cell stability actually blocks electrical impulse conduction and increases arrhythmia risk. During inhalant use, arrhythmias can occur even with normal epinephrine concentrations, but an adrenaline surge, such as when hallucinating or when discovered by or running from an authority figure, increases the risk.[32] Sudden sniffing death can occur during inhalation or in the subsequent few hours, because a volatile substance dissolved in lipid-rich cell membranes dissipates relatively slowly.[32] This unpredictable and unpreventable type of death leaves no specific macroscopic or microscopic postmortem features, so no cause can be identified at autopsy.

Death caused by inhalant abuse can also occur through a variety of other mechanisms but is usually attributable to an acute and related event, most likely suffocation, aspiration, or accidental injury (Table 2). From 1981 to 1985 in Britain, suffocation, aspiration, and accidental injury each accounted for approximately 15% of deaths attributable to inhalant abuse, and the remaining 56% of deaths were attributed to sudden sniffing death syndrome.[30] Suffocation occurs when the mode of use involves inhalation through the nose and mouth from a plastic bag, which may occlude the airway if the user loses consciousness. The risk of death caused by aspiration, usually of vomitus, is similar to that for alcohol or other depressants and is related to the combination of a decreased level of consciousness and the loss of protective airway reflexes. While under the influence of inhalants, users become less inhibited as well as less alert and oriented, which can promote their engaging in risk behaviors and lead to accidental injury and death, such as from a motor vehicle crash, drowning, fire, a jump or fall from heights, or hypothermia from exposure to the elements.

The United Kingdom, with a population approximately one fifth that of the United States, has been the only major part of the Western world to track in a systematic way deaths associated with volatile substance abuse. Since 1999 legislation banned the sale of butane cigarette lighter refills to youth younger than 18 years, there has been a significant drop in inhalant use deaths in both this age group and older individuals. The 2003 volatile substance abuse–related death total of 51 was the lowest number recorded for the United Kingdom since 1983. Of the 9 individuals younger than 18 years who died, 6 did so in relation to inhalation of butane lighter refills, compared with 15 of the 24 deaths in this age group in 2002.[33]

Three reports shed light on the US inhalant abuse mortality rate. The Toxic Exposure Surveillance System (TESS) database of the American Association of Poison Control Systems showed 63 deaths in 11 670 cases of

**TABLE 2 Causes of Death From Inhalant Abuse**

| |
|---|
| Acute |
| Direct causes: immediate or "postponed" sudden sniffing death syndrome; methemoglobinemia |
| Indirect causes: suffocation, aspiration, trauma, drowning, fire, other |
| Delayed |
| Cardiomyopathy |
| Central nervous system toxicity: toluene dementia and brainstem dysfunction |
| Hematologic: aplastic anemia, leukemia |
| Hepatocellular carcinoma |
| Renal toxicity: nephritis, nephrosis, tubular necrosis |

intentional inhalant abuse reported from 1996 to 2001 to poison-control centers nationwide.[9] Actual mortality rates are likely greater, as evidenced by extrapolation from 2 studies that examined state death records that mentioned inhalants as a contributing cause of death at any age.[34,35] These studies found 39 deaths in Virginia from 1987 to 1996 and 144 deaths in Texas from 1988 to 1998. In Virginia, 70% of those who died were 22 years and younger, and in Texas, 28.7% of victims were 8 to 17 years of age. Of the inhalant abuse cases reported to the TESS, 54% were in youth 13 to 19 years of age, 15% were in children 6 to 12 years of age, and 0.4% were in children 5 years and younger. The 63 fatalities occurred almost exclusively in adolescents and young adults. Three types of inhalants were associated with the majority of deaths reported to the TESS: gasoline (45%), air fresheners (26%), and propane/butane (11%). These same group I inhalants (Table 1) were associated with the majority of deaths in both Virginia and Texas, particularly fuels including refrigerants and various solvents.

There is as diverse a list of possible sequelae of chronic inhalant abuse as there is diversity in the types of volatile solvents, fuels, and anesthetics used and the dose and frequency of exposure. If chronic solvent abuse is terminated, there is remarkable reversibility of many of the pathologic effects, but compared with other organ systems, the nervous system has less regenerative capacity. Of all biological membranes, myelin has the highest fat content at 75%, and neuronal membranes may contain up to 45% lipid. The primary consequence of frequent and longer-term inhalant use over months to years is chronic nervous system absorption of these highly lipophilic substances and significant nervous system damage, resulting in muscle weakness, tremor, peripheral neuropathy, cerebellar dysfunction, chronic encephalopathy, and dementia, including mood changes[28,29,36–39] (Table 3). Loss of coordination, gait disturbance, and spasticity, particularly in the legs, have also been noted.[37–39] Computed tomography has demonstrated a loss of brain mass, and magnetic resonance imaging has shown white-matter degeneration and subcortical abnormalities, particularly in the thalamus, basal ganglia, pons, and cerebellum.[40] Cognitive impairment has been reported with deficits found in memory, attention, auditory discrimination, problem-solving abilities, visual learning, and visual-motor function.[29,38] A limitation of the few studies that have investigated cognitive and neuropsychiatric functioning of inhalant abusers is that most of them have not adequately demonstrated that the impairments were not premorbid deficits. Most of the acute neurologic, neuropsychiatric, and cognitive sequelae of volatile solvent abuse seem to be reversible, but the resolution of chronic symptoms is much slower and less complete.[29,38]

TABLE 3 **Major Neurotoxic Consequences of Inhalant Abuse**

| |
|---|
| Cerebellar ataxia |
| Cranial neuropathy: usually cranial nerves V and VII |
| Encephalopathy: acute or chronic |
| Multifocal: both cortical and subcortical central nervous system damage, both central nervous system and peripheral nerve effects |
| Optic neuropathy: visual loss |
| Parkinsonism |
| Peripheral neuropathy |

Other causes of morbidity and mortality are related to the specific volatile chemical(s) used, associated health risk behaviors, drug-drug interactions, or additional material(s) found in the various inhaled products. Toxic effects attributed to specific chemicals include an ichthyosis-like dermatitis on the extremities,[25] decreased visual acuity,[41,42] sensorineural hearing loss,[42] cardiomyopathy,[43] toxic hepatitis,[44] distal renal tubular acidosis,[45] metabolic acidosis,[46] leukemia,[47] and aplastic anemia.[48] There is evidence that tolerance, dependence, and withdrawal symptoms can occur, and reported morbidities also include toluene embryopathy and neonatal withdrawal.[49–52] Lung damage from paint pigments, lead poisoning from leaded gasoline, and other such toxicities have been reported when an inhalant contains another potential toxin.[53] Inhalant abuse is associated with the abuse of other substances, including pharmaceuticals, alcohol, tobacco, and illicit drugs, which can obscure the diagnosis of inhalant abuse and increase potential morbidity.[21,54,55] Combining other drugs with inhalants expands the potential for risk behaviors, altered drug metabolism, and drug-drug interactions, including potentiation of drug effects, particularly depressant effects. High flammability and accidental combustion of volatile agents have led to burns and other fire-related injuries.[3,24]

Chronic nitrous oxide abuse causes short-term memory loss and peripheral neuropathy, which subside with discontinuation of the abuse.[29] Peripheral neuropathy results from nitrous oxide inactivating vitamin $B_{12}$ and mediating a pernicious anemia-type syndrome, which includes anemia, leukopenia, sensorimotor neuropathy, and posterior/lateral column spinal cord disease.[29] Nitrite inhalation has been associated with hemolytic anemia in patients with glucose-6-phosphate dehydrogenase deficiency.[56] Because nitrites are abused mainly for their sensory and sexual effects, use may promote higher-risk sexual practices, facilitate transmission of sexually transmitted infections, and result in drug interactions, such as with sildenafil.[3] Chronic abuse of volatile alkyl nitrites has documented hematologic and immune system effects without associated cognitive deficits.[1,29,56]

## DETECTION OF INHALANT ABUSE

Inhalant abuse may not readily come to the attention of others, including pediatricians, because signs and symptoms of use are often subtle. Abuse of inhalants should

be suspected when a cache of a potential inhalant is discovered or when products with abuse potential are found stored in unusual locations, such as cans of gasoline or spray paint under a youth's bed. Changes in an adolescent's behavior, including apathy, malaise, poor appetite, a significant shift in choice of friends or activities, or an unexplained drop in school grades, can also be signs of inhalant abuse. Those who are chronic and heavy inhalant abusers may be identifiable because of their combination of poor hygiene and grooming, weight loss from decreased caloric intake, and chronic complaints of, for example, fatigue, rhinitis, conjunctivitis, recurrent epistaxis, and oral or nasal ulcerations. Chronic neuropsychiatric changes, such as confusion, poor concentration, depression, irritability, hostility, or paranoia,[28,29,57] may predominate. Symptoms of other organ system toxicities from long-standing inhalant use may also bring the abuser to medical attention.

Inhalant abusers may present with obvious intoxication and evidence of use, such as a conspicuous odor of the inhalant. This chemical odor is most often present because the abuser excretes a significant proportion of the absorbed dose when exhaling, and the odor can persist on the breath for many hours.[57] If the abused product spilled onto clothing during use or was intentionally put on clothing, the odor may also persist, and clothing stains or paint may be found. Paint or glitter may also be seen on the abuser's face or hands, or there may be a "huffer's rash," classically a perioral or perinasal dermatitis with pyoderma.[24,58] Contact with inhalants dries the skin and leads to small cracks, which allow bacteria to enter. The dermatitis may look like a nonspecific contact hypersensitivity reaction or perioral eczema or, with nitrite use, may have a yellow crust attributed to nitric acid, which can promote a xanthoprotein reaction.[58] Refrigerants and chlorofluorohydrocarbon propellants, such as those found in computer-cleaning aerosols, have been reported to cause frostbite on the face or in the nose or oral cavity that can lead to airway compromise.[59]

Similar to the approach to other substance abuse, providing appropriate medical care for any child or adolescent using inhalants results from a keen diagnostic awareness leading to detection, intervention, and treatment. Regular office screening for inhalant abuse as well as other substance abuse and health risk behaviors must be part of standard pediatric care. Inhalants are not detected by using routine urine drug screening, so detection relies on knowledgeable medical personnel who consistently include screening questions as part of conducting a thorough history and physical examination.[26] When considered in certain clinical contexts, abnormal nontoxicologic laboratory results, such as elevated liver enzymes, may arouse or confirm a suspicion of inhalant abuse. Blood and other tissues, usually brain or liver, can be tested by specific gas chromatography technique when inhalant detection is necessary, such as in a fatality.[26] Specific urine drug testing is sometimes useful as part of the treatment-compliance plan when benzene, toluene, or a similar agent has been chronically abused, because major urinary metabolites (phenol and hippuric acid, respectively) are detectable when there has been a high level of use.[26]

## INHALANT ABUSE PREVENTION AND MANAGEMENT CONSIDERATIONS

As with other types of substance abuse, the most effective way to curtail use is through broad prevention efforts, particularly primary prevention through education paired with skills-building. Developmentally and culturally sensitive educational strategies should be implemented, such as those implemented in many American Indian communities through a prevention initiative in conjunction with the American Indian Institute at the University of Oklahoma.[8]

Limiting the availability of volatile substances is impractical, because they constitute products that are universally available and legal and have legitimate uses. Restricting the availability of some of these products, such as the United Kingdom ban on the sale of butane lighter refills to youth, can be successful but may also promote the use of other more-available products or create a black market for the restricted products. Adding a noxious chemical to the product to prevent misuse was tried with plastic glue and found to be ineffective, because multiple products would require such adulterants, abusers switch products, and legitimate consumers and product efficacy might be adversely affected.[60] Reformulating the product by replacing the hydrocarbon with other chemicals has occurred when economically feasible and when product efficacy could be maintained. Product warning labels can alert the public to inhalant dangers but may also promote easy identification of abusable substances.[61] Most states have laws making the use of inhalants or sale to minors illegal, and although difficult to enforce and of yet-unproven efficacy, such laws serve as a reminder that society condemns inhalant abuse.

Most acutely intoxicated inhalant abusers do not seek medical attention, and only when intoxication is life-threatening or has led to serious injury will an abuser present to the emergency department. Acute medical management of inhalant abuse starts with applying the "ABCs" of life support to assess and stabilize the patient and address any specific acute injury or toxicity, such as combating methemoglobinemia by administering intravenous methylene blue. Hydration and cardiorespiratory status should be monitored closely. Myocardial sensitization by inhalants necessitates a calm and supportive environment in which the use of pressor medications and bronchodilators are relatively contraindicated. No medications reverse acute inhalant intoxication or have

been found to be helpful with dependence or withdrawal symptoms. Decontamination of the patient's clothing and skin may be indicated. Laboratory testing can help monitor oxygenation and hematologic status and detect other substances being abused. Testing for organ-system damage should be considered only when there is a history of regular and long-term inhalant use. After acute stabilization, comprehensive medical care includes documenting a detailed history and physical examination and specifically evaluating the patient's mental health, substance abuse history, and psychosocial needs so that appropriate inpatient or outpatient interventions can be initiated.[28,29,57]

Little research exists concerning treatment needs and successful treatment modalities specific to inhalant users, so clinicians rely on applying methods that are used to treat other addictive disorders, such as cognitive-behavioral therapy, multisystem and family therapy, 12-step facilitation, and motivational enhancement techniques,.[62] Inhalant abusers seem to respond best to a treatment program that includes an extended detoxification or "treatment readiness" period of 4 or more weeks, during which basic supportive care and general orientation are emphasized. If sufficient time is not allowed, individuals seem incapable of engaging in the treatment program.[63] Some treatment facilities have used a peer-advocate system for patients, which seems to offer a nonthreatening and supportive treatment approach.[64] Neuroleptics and other forms of pharmacotherapy are usually not useful in the treatment of inhalant abusers except to address comorbid conditions. Increasing personal and ethnic self-identity through role-modeling has been suggested as helpful in treating some groups of inhalant abusers, and positive cultural identification has been shown to be important in American Indian/Alaska Native populations.[65] Treatment challenges are posed by the diversity of abused inhalants and user populations, comorbid psychopathology, psychosocial problems, polydrug use, and the physiologic and neurologic effects of inhalant abuse.[62,63] Treatment of longer-term inhalant users is hindered by the fact that there are few programs designed specifically for inhalant abuse treatment, access to care may be limited, providers generally have a pessimistic view about users' neurologic damage and chance for recovery, and providers often lack sufficient knowledge and training about inhalant abuse, inhalant users, and their treatment needs.[64] Although the principles of effective substance abuse treatment in general apply to inhalant abuse treatment, any treatment regimen must address the many clinical, emotional, social, academic, pharmacologic, neurocognitive, cultural, and demographic factors that make this type of substance abuse unique. Treatment strategies are still under development, and additional research is needed to identify effective strategies for the treatment of children and adolescents who use inhalants.

## CONCLUSIONS AND ADVICE

The American Academy of Pediatrics has established recommendations[66] regarding the pediatrician's role in the prevention, identification, and management of substance abuse and advises the following to promote that role with regard to inhalant use by youth.

1. Pediatricians are encouraged to:
   - be aware that inhalant abuse occurs in all patient populations, including their own;
   - be knowledgeable about the epidemiology of inhalant abuse, particularly about local and regional trends, as well as resources, such as the telephone number 1-800-222-1222 to contact the nearest poison-control center;
   - be knowledgeable about health consequences of inhalant abuse and, in particular, about unique clinical features such as central nervous system damage and sudden sniffing death syndrome;
   - assist in educating children, adolescents, parents, teachers, media representatives, and vendors of volatile substances regarding inhalant abuse prevention and the health risks of inhalant use; and
   - serve as a community resource regarding inhalant use awareness, prevention, detection, and management using national and local community resources such as the National Inhalant Prevention Coalition (1-800-269-4237 or www.inhalants.com), an information and referral clearinghouse.
2. Inhalant abuse education can be included in all substance abuse prevention curricula in the primary and secondary grades, using approaches that effectively warn against the dangers of inhalant use yet do not inadvertently introduce youth to available substances with abuse potential.
3. Widespread accessibility and use of research-based resources such as National Institute on Drug Abuse publications (available at www.drugabuse.gov/DrugPages/Inhalants.html) are encouraged.
4. Increased research efforts to evaluate prevention and treatment approaches specific to inhalant abuse and to identify those with efficacy are needed.

**COMMITTEE ON SUBSTANCE ABUSE, 2005–2006**
Alain Joffe, MD, MPH, Chairperson
Marylou Behnke, MD
John R. Knight, MD
Patricia K. Kokotailo, MD, MPH
Tammy Sims, MD, MS
Janet F. Williams, MD*

## REFERENCES

1. Balster RL. Neural basis of inhalant abuse. *Drug Alcohol Depend.* 1998;51:207–214
2. Beauvais F, Oetting ER. Toward a clear definition of inhalant abuse. *Int J Addict.* 1987;22:779–784
3. Romanelli F, Smith KM, Thornton AC, Pomeroy C. Poppers: epidemiology and clinical management of inhaled nitrite abuse. *Pharmacotherapy.* 2004;24:69–78
4. Wu L, Pilowsky DJ, Schlenger WE. Inhalant abuse and dependence among adolescents in the United States. *J Am Acad Child Adolesc Psychiatry.* 2004;43:1206–1214
5. Neumark YD, Delva J, Anthony JC. The epidemiology of adolescent inhalant drug involvement. *Arch Pediatr Adolesc Med.* 1998;152:781–786
6. Edwards RW, Oetting ER. Inhalant use in the United States. In: Kozel N, Sloboda Z, DeLaRosa M, eds. *Epidemiology of Inhalant Abuse: An International Perspective*. Rockville, MD: National Institute on Drug Abuse 1995:8–28Publication No. NIH 95–3831
7. Spear S, Longshore D, McCaffrey D, Ellickson P. Prevalance of substance use among white and American Indian young adolescents in a Northern Plains state. *J Psychoactive Drugs.* 2005; 37:1–6
8. Beauvais F, Wayman JC, Jumper-Thurman P, Plested B, Helm H. Inhalant abuse among American Indian, Mexican American, and non-Latino white adolescents. *Am J Drug Alcohol Abuse.* 2002;28:171–187
9. Spiller HA. Epidemiology of volatile substance abuse (VSA) cases reported to US poison centers. *Am J Drug Alcohol Abuse.* 2004;30:155–165
10. Substance Abuse and Mental Health Services Administration. *Results From the 2005 National Survey on Drug Use and Health: National Findings.* Rockville, MD: Substance Abuse and Mental Health Services Administration Office of Applied Studies; 2006. Publication No. SMA 06–4194. Available at: http://oas.samhsa.gov. Accessed December 7, 2006
11. Johnston LD, O'Malley PM, Bachman JG, Schulenberg JE. Teen drug use continues down in 2006, particularly among older teens; but use of prescription-type drugs remains high [press release]. 2006. Available at: www.monitoringthefuture.org/pressreleases/06drugpr.pdf. Accessed January 18, 2007
12. Center for Substance Abuse Research. U.S. residents more likely than Canadians to use inhalants, cocaine, and stimulants; slightly less likely to use marijuana. 2005. Available at: www.cesar.umd.edu/cesar/cesarfax/vol14/14–46.pdf. Accessed April 18, 2006
13. Substance Abuse and Mental Health Services Administration, Office of Applied Studies; RTI International. Inhalant use and delinquent behaviors among young adolescents. Available at: www.oas.samhsa.gov/2k5/inhale/inhale.htm. Accessed April 18, 2006
14. Swaim RC, Beauvais F, Chavez EL, Oetting ER. The effect of school dropout rates on estimates of adolescent substance use among three racial/ethnic groups. *Am J Public Health.* 1997;87: 51–55
15. Beauvais F, Chavez EL, Oetting ER, Deffenbacher JL, Cornell GR. Drug use, violence, and victimization among white American, Mexican American and American Indian dropouts, students with academic problems and students in good standing. *J Couns Psychol.* 1996;43:292–299
16. McGarvey EL, Clavet GJ, Mason W, Waite D. Adolescent inhalant abuse: environments of use. *Am J Drug Alcohol Abuse.* 1999;25:731–741
17. Howard MO, Jenson JM. Inhalant use among antisocial youth: prevalence and correlates. *Addict Behav.* 1999;24:59–74
18. Grunbaum JA, Kann L, Kinchen SA, et al. Youth risk behavior surveillance: National Alternative High School Youth Risk Behavior Survey, United States, 1998. *MMWR CDC Surveill Summ.* 1999;48(7):1–44
19. Howard MO, Walker RD, Walker PS, Cottler LB, Compton WM. Inhalant use among urban American Indian youth. *Addiction.* 1999;94:83–95
20. Mackesy-Amiti ME, Fendrich M. Inhalant use and delinquent behavior among adolescents: a comparison of inhalant users and other drug users. *Addiction.* 1999;94:555–564
21. Sakai JT, Hall SK, Mikulich-Gilbertson SK, Crowley TJ. Inhalant use, abuse, and dependence among adolescent patients: commonly comorbid problems. *J Am Acad Child Adolesc Psychiatry.* 2004;43:1080–1088
22. Beauvais F. Comparison of drug use rates for reservation In-

dian, non-reservation Indian and Anglo youth. *Am Indian Alsk Native Ment Health Res.* 1992;5(1):13–31
23. Zebrowski PL, Gregory RJ. Inhalant use patterns among Eskimo school children in western Alaska. *J Addict Dis.* 1996;15: 67–77
24. Center for Substance Abuse Treatment. Inhalants. *Subst Abuse Treat Advis.* 2003;3(1):1–8
25. Feuillet L, Mallet S, Spadari M. Twin girls with neurocutaneous symptoms caused by mothball intoxication. *N Engl J Med.* 2006;355:423–424
26. Broussard LA. The role of the laboratory in detecting inhalant abuse. *Clin Lab Sci.* 2000;13:205–209
27. DeViore WJ. Dynamics of drinking hairspray-water mix[t]ure. "Ocean" Awareness Project. Available at: http://wellbriety-nci.org/Publications/ocean.htm. Accessed April 18, 2006
28. Lorenc JD. Inhalant abuse in the pediatric population: a persistent challenge. *Curr Opin Pediatr.* 2003;15:204–209
29. Brouette T, Anton R. Clinical review of inhalants. *Am J Addict.* 2001;10:79–94
30. Ramsey J, Anderson HR, Bloor K, Flanagan RJ. An introduction to the practice, prevalence and chemical toxicology of volatile substance abuse. *Hum Toxicol.* 1989;8:261–269
31. Bass M. Sudden sniffing death. *JAMA.* 1970;212:2075–2079
32. Shepherd RT. Mechanism of sudden death associated with volatile substance abuse. *Hum Toxicol.* 1989;8:287–291
33. Field-Smith ME, Butland BK, Ramsey JD, Anderson HR. *Trends in Death Associated With Abuse of Volatile Substances 1971–2003*. Report 18. London, England: St George's, University of London, Division of Community Health Sciences; 2005. Available at: www.vsareport.org. Accessed April 18, 2006
34. Bowen SE, Daniel J, Balster RL. Deaths associated with inhalant abuse in Virginia from 1987 to 1996. *Drug Alcohol Depend.* 1999;53:239–245
35. Maxwell JC. Deaths related to the inhalation of volatile substances in Texas: 1988–1998. *Am J Drug Alcohol Abuse.* 2001; 27:689–697
36. Lolin Y. Chronic neurological toxicity associated with exposure to volatile substances. *Hum Toxicol.* 1989;8:293–300
37. Meadows R, Verghese A. Medical complications of glue sniffing. *South Med J.* 1996;89:455–462
38. Maruff P, Burns CB, Tyler P, Currie BJ, Currie J. Neurological and cognitive abnormalities associated with chronic petrol sniffing. *Brain.* 1998;121:1903–1917
39. Tenenbein M, deGroot W, Rajani KR. Peripheral neuropathy following intentional inhalation of naphtha fumes. *Can Med Assoc J.* 1984;131:1077–1079
40. Rosenberg NL, Grigsby J, Dreisbach J, Busenbark D, Grigsby P. Neuropsychologic impairment and MRI abnormalities associated with chronic solvent abuse. *J Toxicol Clin Toxicol.* 2002;40: 21–34
41. Keane JR. Toluene optic neuropathy. *Ann Neurol.* 1978;4:390
42. Ehyai A, Freemon FR. Progressive optic neuropathy and sensorineural hearing loss due to chronic glue sniffing. *J Neurol Neurosurg Psychiatry.* 1983;46:349–351
43. Wiseman MN, Banim S. "Glue sniffer's" heart? *Br Med J (Clin Res Ed).* 1987;294:739
44. Kaplan HG, Bakken J, Quadracci L, Schubach W. Hepatitis caused by halothane sniffing. *Ann Intern Med.* 1979;90:797–798
45. Taher SM, Anderson RJ, McCartney R, Popovtzer MM, Schrier RW. Renal tubular acidosis associated with toluene "sniffing." *N Engl J Med.* 1974;290:765–768
46. Fischman CM, Oster JR. Toxic effects of toluene: a new cause of high anion gap metabolic acidosis. *JAMA.* 1979;241: 1713–1715
47. Austin H, Delzell E, Cole P. Benzene and leukemia: a review of the literature and a risk assessment. *Am J Epidemiol.* 1988;127: 419–439
48. Powars D. Aplastic anemia secondary to glue sniffing. *N Engl J Med.* 1965;273:700–702
49. Keriotis AA, Upadhyaya HP. Inhalant dependence and withdrawal symptoms. *J Am Acad Child Adolesc Psychiatry.* 2000;39: 679–680
50. Pearson MA, Hoyme HE, Seaver LH, Rimsza ME. Toluene embryopathy: delineation of the phenotype and comparison with fetal alcohol syndrome. *Pediatrics.* 1994;93:211–215
51. Arnold GL, Kirby RS, Langendoerfer S, Wilkins-Haug L. Toluene embryopathy: clinical delineation and developmental follow-up. *Pediatrics.* 1994;93:216–220
52. Tenenbein M, Casiro OG, Seshia MM, Debooy VD. Neonatal withdrawal from maternal volatile substance abuse. *Arch Dis Child Fetal Neonatal Ed.* 1996;74:F204–F207
53. Engstrand DA, England DM, Huntington RW III. Pathology of paint sniffers' lung. *Am J Forensic Med Pathol.* 1986;7:232–236
54. Dinwiddie SH, Reich T, Cloninger CR. The relationship of solvent use to other substance use. *Am J Drug Alcohol Abuse.* 1991;17:173–186
55. Young SJ, Longstaffe S, Tenenbein M. Inhalant abuse and the abuse of other drugs. *Am J Drug Alcohol Abuse.* 1999;25: 371–375
56. Brandes JC, Bufill JA, Pisciotta AV. Amyl nitrite-induced hemolytic anemia. *Am J Med.* 1989;86:252–254
57. Meredith TJ, Ruprah M, Liddle A, Flannagan RJ. Diagnosis and treatment of acute poisoning with volatile substances. *Hum Toxicol.* 1989;8:277–286
58. Fregert S, Poulsen J, Trulsson L. Yellow stained skin from sodium nitrite in an etching agent. *Contact Dermatitis.* 1980;6: 296
59. Kurbat RS, Pollack CV Jr. Facial injury and airway threat from inhalant abuse: a case report. *J Emerg Med.* 1998;16:167–169
60. Akerman HE. The constitution of adhesives, and its relationship to solvent abuse. *Hum Toxicol.* 1982;1:223–230
61. Liss BI. Government, trade and industry and other preventive responses to volatile substance abuse. *Hum Toxicol.* 1989;8: 327–330
62. Reidel S, Hebert T, Byrd P. Inhalant abuse: confronting the growing challenge. In: *Treating Alcohol and Other Drug Abusers in Rural and Frontier Areas*. Rockville, MD: Center for Substance Abuse Treatment; 1995. Technical assistance publication series #17. Available at: www.treatment.org/taps/tap17/tap17toc.html. Accessed April 18, 2006
63. Jumper-Thurman P, Plested B, Beauvais F. Treatment strategies for volatile solvent abusers in the United States. In: Kozel N, Sloboda Z, DeLaRosa M, eds. *Epidemiology of Inhalant Abuse: An International Perspective*. Rockville, MD: National Institute on Drug Abuse 1995:250–259Publication No. NIH 95–3831
64. Beauvais F, Jumper-Thurman P, Plested B, Helm H. A survey of attitudes among drug user treatment providers toward the treatment of inhalant users. *Substance Use Misuse.* 2002;37: 1391–1410
65. Thurman PJ, Green VA. American Indian adolescent inhalant use. *Am Indian Alsk Native Ment Health Res.* 1997;8(1):24–40
66. Kulig JW; American Academy of Pediatrics, Committee on Substance Abuse. Tobacco, alcohol, and other drugs: the role of the pediatrician in prevention, identification, and management of substance abuse. *Pediatrics.* 2005;115:816–821