

# Inhalant Abuse

Committee on Substance Abuse and Committee on Native American Child Health

Inhalant abuse is the intentional inhalation of a volatile substance for the purpose of achieving a euphoric state. It is also known as solvent abuse, volatile substance abuse, glue sniffing, sniffing, and huffing. Beginning with children as young as 6 years of age, it is an underrecognized form of substance abuse with a significant morbidity and mortality. This statement reviews important aspects of inhalant abuse and makes several recommendations involving prevention and education strategies to address this problem.

## EPIDEMIOLOGY

As with other types of substance abuse, precise epidemiologic data on inhalant abuse are not available. The peak age of inhalant abuse is 14 to 15 years, with onset occurring in those as young as 6 to 8 years. Use declines typically by 17 to 19 years of age; however, some users may continue into adulthood.

Since 1975, the National Institute on Drug Abuse annual survey of high school seniors (Monitoring the Future) has documented a lifetime incidence of inhalant abuse of 15% to 20%, with 5% to 10% of seniors using inhalants during the previous year.<sup>1</sup> This survey underestimates the true prevalence, because school dropouts, who have a relatively higher incidence of substance abuse, are not included. Although there has been a general decline in the use of most other mind-altering substances, the relative incidence of inhalant abuse has increased. Since 1988, eighth graders also have been surveyed, disclosing that inhalant abuse has increased recently and has surpassed marijuana use within this group.<sup>2</sup> Nationwide mortality data are not collected; however, the United Kingdom (with a population approximately one fifth of that of the United States and the only major country in the western world that tracks deaths caused by inhalants) has documented two deaths per week.<sup>3</sup>

Patterns of inhalant abuse are similar to patterns of the abuse of other types of substances. There are experimenters, intermittent users, and chronic inhalant abusers. Although morbidity and mortality are associated with the frequency of use, the so-called "sudden sniffing death syndrome,"<sup>4</sup> which is the

most common cause of death from inhalant abuse, can occur in first-time users.

## TYPES OF CHEMICALS AND PRODUCTS ABUSED

Products selected by inhalant abusers are volatile, capable of producing a pleasurable sensory experience rapidly, available, convenient, and inexpensive. The latter three qualities are important, because children have less sophisticated resources for acquiring substances that are abused.

Virtually any hydrocarbon can have mind-altering effects when inhaled in large doses. The most commonly abused inhalants are aliphatic, aromatic, or halogenated hydrocarbons. Chemicals from these groups are found in thousands of commonly used and readily available consumer products (Table). Furthermore, almost all pressurized aerosol products can be abused, because their propellants are volatile hydrocarbons.

## CAUSES OF INHALANT ABUSE

Important factors that contribute to the initial experimentation and continued use of inhalants include peer pressure and dysfunctional families.<sup>5</sup> Peer pressure is a powerful force contributing to initial experimentation. Children of dysfunctional families that accept violence and substance abuse as parts of their lives are more likely to engage in drug experimentation that escalates to chronic abuse. Although inhalant abuse is more prevalent among the poor, it crosses all socioeconomic boundaries. While it is seen in all ethnic groups in the United States, it is especially prevalent among Hispanic and Native American children and adolescents,<sup>6,7</sup> perhaps because of an increased likelihood of membership in drug-using peer clusters and a perception of a lack of opportunities.<sup>8,9</sup>

## MECHANISM OF ABUSE AND IMMEDIATE EFFECTS OF INHALANTS

The fumes of the product may be inhaled directly from a container, plastic bag, or saturated rag. Inhalation is usually through the mouth, with several deep inspirations required to produce euphoria.

TABLE. Commonly Abused Products

Liquids	Aerosols
Model glue	Paints
Gasoline	Butane fuel
Contact cement	Cooking sprays
Laquers	Cosmetics
Dry cleaning fluids	Toiletries

This statement has been approved by the Council on Child and Adolescent Health.

The recommendations in this statement do not indicate an exclusive course of treatment or serve as a standard of medical care. Variations, taking into account individual circumstances, may be appropriate.

PEDIATRICS (ISSN 0031 4005). Copyright © 1996 by the American Academy of Pediatrics.

Inhalants are depressants and are pharmacologically related to anesthetic gases. In fact, some anesthetic gases, such as ether and nitrous oxide, are also abused. The immediate effects of inhalant abuse are similar to the early classic stages of anesthesia. The user is initially stimulated, uninhibited, and prone to impulsive behavior. Speech becomes slurred, and the user's gait becomes staggered. Euphoria, frequently with hallucinations, is followed by drowsiness and sleep, particularly after repeated cycles of inhalation. Coma is unusual, because as the user becomes drowsy, exposure to the inhalant is terminated before a large enough dose is absorbed.

#### MORBIDITY AND MORTALITY

Significant morbidity and mortality are associated with inhalant abuse. Morbidity is psychosocial as well as organic. Ongoing inhalant abuse is associated with failure in school, delinquency, and an inability to achieve societal adjustment.<sup>10,11</sup> There is evidence that withdrawal symptoms can occur<sup>12</sup> and that inhalant abuse can lead to the abuse of other substances.<sup>13,14</sup>

The chief organic morbidity, a consequence of chronic abuse, is central nervous system damage, resulting in dementia and cerebellar dysfunction.<sup>15-17</sup> Typically there is a loss of cognitive and other higher functions, gait disturbance, and loss of coordination. Computed tomography demonstrates a loss of brain mass,<sup>15</sup> and magnetic resonance imaging shows white matter degeneration.<sup>16,17</sup> Inhalants are used commonly both in industry and by consumers as fat solvents; thus, because the brain is a lipid-rich organ, chronic solvent abuse dissolves brain cells.

Chronic use of any inhalant increases the risk of brain injury. Other organic effects are related to specific chemicals found in some but not all products. The strength of the association ranges from definite through likely to speculative. Definite associations include peripheral neuropathy (hexane),<sup>18</sup> deafness (toluene),<sup>19,20</sup> and metabolic acidosis (toluene).<sup>21,22</sup> Likely morbidities include embryopathy (toluene),<sup>23,24</sup> neonatal withdrawal (multiple agents),<sup>25</sup> and lung damage (paint pigments).<sup>26</sup> Speculative morbidities include cardiomyopathy,<sup>27</sup> toxic hepatitis (chlorinated hydrocarbons),<sup>28,29</sup> decreased visual acuity (toluene),<sup>20,30</sup> aplastic anemia (benzene),<sup>31,32</sup> and leukemia (benzene).<sup>31</sup> Because the latter complications are hypothetical, routine laboratory testing for their presence is not indicated.

Death caused by inhalant abuse can occur by several mechanisms, including asphyxia, suffocation, dangerous behavior, aspiration, and sudden sniffing death syndrome. Asphyxia is probably only a theoretical concern, occurring when the partial pressure of the inhalant is so high that oxygen is displaced. In Britain, suffocation, dangerous behavior, and aspiration each account for approximately 15% of deaths caused by inhalant abuse, whereas sudden sniffing death syndrome is responsible for the remaining 55%.<sup>33</sup> 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 loss of inhibition while

under the influence of inhalants may cause dangerous behavior and may result in risks such as drowning, jumps or falls from heights, hypothermia, and, particularly, fire-associated deaths because of the flammability of most inhalants. The risk of death caused by aspiration 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.

Sudden sniffing death syndrome was described originally by Bass.<sup>4</sup> Death occurs suddenly after the user is startled during inhalation (usually as a consequence of being discovered by an authority figure or of having a particularly stimulating hallucination). The pathophysiology of sudden sniffing death syndrome has been elucidated.<sup>4</sup> The hydrocarbons of inhalants sensitize the myocardium to epinephrine, and the sudden surge of this hormone produced by the startle reflex results in a fatal cardiac arrhythmia. No cause of death is found at autopsy. Sudden sniffing death can result during the initial experimentation or from any episode of inhalant abuse. In one study of deaths attributed to inhalant abuse, 22% of the abusers who died had no history of previous inhalant abuse.<sup>33</sup>

#### DETECTION OF INHALANT ABUSE

Chronic, heavy inhalant abusers may be identifiable because of poor hygiene and grooming, frequent, obvious episodes of intoxication, weight loss from decreased caloric intake, and, most importantly, the conspicuous odor of the inhalant.<sup>34</sup> This odor is often present, because a significant proportion of the absorbed dose exits the body by the same route that it enters (via the lungs); therefore, the odor can persist on the breath for many hours.<sup>34</sup> The product also may be spilled onto clothing during use, resulting in another source of odor. Additional clues include stained clothing, flecks of paint or glitter on the face, and perioral pyodermas<sup>34</sup> related to inhalants drying the skin and resulting in small cracks, which serve as portals of entry for bacteria. Finding products of abuse stored in unusual locations, such as a can of gasoline under a child's bed or a large cache of a potential inhalant, often suggests inhalant abuse. Finally, deviation from normal behavior can be an indicator of potential dysfunction in the adolescent and can be a sign of inhalant abuse. Urine drug screens do not detect these chemicals; laboratory testing for organ dysfunction should be considered only in chronic abusers.

#### PREVENTION OF INHALANT ABUSE

As with other types of substance abuse, the most effective way to curtail use is through prevention. Although chronic users must not be ignored, their treatment is very difficult, expensive, and not highly effective.

Of the many potential methods of prevention, education is seen as the most effective.<sup>35</sup> Limiting the availability of inhalants is impractical, because they constitute a large group of products that are universally available and licit and have legitimate uses. Restricting the availability of some of these products

merely results in a shift to the use of other products or creates a black market for the restricted products.<sup>35</sup> Adding a noxious chemical to the product to prevent misuse is also ineffective,<sup>35,36</sup> because there are multiple products that would require such adulterants, and the result is often unacceptable to the legitimate consumer. Reformulating the product by replacing the hydrocarbon with other chemicals is not practical, because this usually results in a less-effective product. Warning labels on packages may be counterproductive, because they allow children to identify sniffable substances easily.<sup>35,37</sup> More than 40 states have laws making the sale or use of inhalants illegal, and, although difficult to enforce, such laws clearly make the point that society condemns inhalant abuse. Education is considered the most effective preventive strategy,<sup>33</sup> particularly if initiated before the usual age of experimentation. A progressive, school-based inhalant abuse curriculum beginning in kindergarten with developmentally appropriate modules throughout elementary school is seen as the most efficient strategy and should be implemented particularly in areas where inhalant abuse is prevalent. The provision of alternate activities and role models for high-risk children and adolescents, especially with trained and knowledgeable staff, also may be an effective prevention strategy.

The treatment of inhalant users is difficult because of the many pharmacologic, clinical, cultural, and demographic factors that make this type of substance abuse unique. Such treatment requires specific rather than generic substance abuse treatment approaches and may be most effective when conducted by reformed inhalant abusers.

#### CONCLUSION AND RECOMMENDATIONS

The American Academy of Pediatrics is extremely concerned about the practice of inhalant abuse among children and adolescents and wishes to draw attention to this underrecognized and dangerous practice.

1. Pediatricians are encouraged to increase their awareness of the unique clinical aspects and complications of inhalant abuse, particularly central nervous system damage and sudden sniffing death syndrome.
2. Pediatricians need to promote education about the health hazards posed by substance abuse to children, adolescents, parents, teachers, media representatives, and vendors of volatile substances.
3. Inhalant abuse should be included in all substance abuse prevention curricula in the primary grades and should be emphasized especially in areas where inhalant abuse is endemic.
4. Research efforts to identify and evaluate prevention and treatment approaches that are specific for this type of substance abuse should be increased.

COMMITTEE ON SUBSTANCE ABUSE, 1995 TO 1996  
 Richard B. Heyman, MD, Chair  
 Hoover Adger, Jr, MD  
 Trina M. Anglin, MD  
 Paul G. Fuller, Jr, MD  
 Edward A. Jacobs, MD

Rizwan Z. Shah, MD  
 Milton Tenenbein, MD

#### LIAISON REPRESENTATIVES

Marie Armentano, MD  
 American Association of Child and Adolescent Psychiatry  
 Gayle M. Boyd, PhD  
 National Institute of Alcohol Abuse and Alcoholism  
 Dorynne Czechowicz, MD  
 National Institute on Drug Abuse

#### COMMITTEE ON NATIVE AMERICAN CHILD HEALTH, 1995 TO 1996

Lance Chilton, MD, Chair  
 Joann Bordurtha, MD, MPH  
 Bernadette T. Freeland-Hyde, MD  
 John Goodrich, MD, PA  
 David Grossman, MD  
 Jonathan Jantz, MD  
 Frederick Mandell, MD

#### LIAISON REPRESENTATIVES

F. W. Baker, MD, FRCP(C)  
 Canadian Paediatric Society  
 Joseph Bell, MD  
 Association of American Indian Physicians

#### CONSULTANT

Harry L. Wilson, MD

#### REFERENCES

1. Johnston LD, O'Malley PM, Bachman JG. Prevalence of drug use among 8th, 10th and 12th grade students. In: *National Survey Results on Drug Use From Monitoring the Future Study, 1975-1992*. Rockville, MD: National Institute on Drug Abuse, US Dept of Health and Human Services; 1993
2. Edwards RW. Drug use among 8th grade students is increasing. *Int J Addict*. 1993;28:1613-1621
3. Ashton CH. Solvent abuse. *Br Med J*. 1990;300:135-136
4. Bass M. Sudden sniffing death. *JAMA*. 1970;212:2075-2079
5. American Academy of Pediatrics, Committee on Substance Abuse. Role of the pediatrician in prevention and management of substance abuse. *Pediatrics*. 1993;91:1010-1013
6. Young TJ. Inhalant use among American Indian youth. *Child Psychiatry Hum Dev*. 1987;18:36-46
7. Beauvais F. Comparison of drug use rates for reservation Indian, non-reservation Indian and Anglo youth. *Am Indian Alsk Native Ment Health Res*. 1992;5:13-31
8. Beauvais F. Characteristics of Indian youth and drug use. *Am Indian Alsk Native Ment Health Res*. 1992;5:51-67
9. Mail PD, Johnson S. Boozing, sniffing, and toking: an overview of the past, present, and future of substance abuse by American Indians. *Am Indian Alsk Native Ment Health Res*. 1993;5:1-33
10. Chadwick O, Yule W, Anderson R. The examination attainments of secondary school pupils who abuse solvents. *Br J Educ Psychol*. 1990;60:180-191
11. Chadwick OF, Anderson HR. Neuropsychological consequences of volatile substance abuse: a review. *Hum Toxicol*. 1989;8:307-312
12. Evans AC, Raistrick D. Phenomenology of intoxication with toluene-based adhesives and butane gas. *Br J Psychiatry*. 1987;150:769-773
13. Davies B, Thorley A, O'Connor D. Progression of addiction careers in young adult solvent misusers. *Br Med J*. 1985;290:109-110
14. Altenkirch H, Kindermann W. Inhalant abuse and heroin addiction: a comparative study on 574 opiate addicts with and without a history of sniffing. *Addict Behav*. 1986;11:93-104
15. Fornazzari L, Wilkinson DA, Kapur BM, Carlen PL. Cerebellar cortical and functional impairment in toluene abusers. *Acta Neurol Scand*. 1983;67:319-329
16. Rosenberg NL, Spitz MC, Filley CM, Davis KA, Schaumburg HH. Central nervous system effects of chronic toluene abuse—clinical, brainstem evoked response and magnetic resonance imaging studies. *Neurotoxicol Teratol*. 1988;10:489-495
17. Filley CM, Heaton RK, Rosenberg NL. White matter dementia in chronic toluene abuse. *Neurology*. 1990;40:532-534

18. Tenenbein M, deGroot W, Rajani KR. Peripheral neuropathy following intentional inhalation of naphtha fumes. *Can Med Assoc J.* 1984;131:1077-1079
19. Pryor GT, Rebert CS, Dickinson J, Feeney EM. Factors affecting toluene-induced ototoxicity in rats. *Neurobehav Toxicol Teratol.* 1984;6:223-238
20. Ehyai A, Freemon FR. Progressive optic neuropathy and sensorineural hearing loss due to chronic glue sniffing. *J Neurol Neurosurg Psychiatry.* 1983;46:349-351
21. 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
22. Fischman CM, Oster JR. Toxic effects of toluene: a new cause of high anion gap metabolic acidosis. *JAMA.* 1979;241:1713-1715
23. 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
24. Arnold GL, Kirby RS, Langendoerfer S, Wilkins-Haug L. Toluene embryopathy: clinical delineation and developmental follow-up. *Pediatrics.* 1994;93:216-220
25. Tenenbein M, Casiro OG, Seshia MMK, Debooy V. Neonatal withdrawal from maternal inhalant abuse. *Arch Dis Child.* In press
26. Engstrand DA, England DM, Huntington RW. Pathology of paint sniffers' lung. *Am J Forensic Med Pathol.* 1986;7:232-236
27. Wiseman MN, Banim S. "Glue sniffer's" heart. *Br Med J.* 1987;294:739
28. Kaplan HG, Bakken J, Quadracci L, Schubach W. Hepatitis caused by halothane sniffing. *Ann Intern Med.* 1979;90:797-798
29. Hutchens KS, Kung M. "Experimentation" with chloroform. *Am J Med.* 1985;78:715-718
30. Keane JR. Toluene optic neuropathy. *Ann Neurol.* 1978;4:390
31. Aksoy M. Benzene as a leukemogenic and carcinogenic agent. *Am J Ind Med.* 1985;8:9-20
32. Powars D. Aplastic anemia secondary to glue sniffing. *N Engl J Med.* 1965;273:700-702
33. 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
34. Meredith TJ, Ruprah M, Liddle A, Flannagan RJ. Diagnosis and treatment of acute poisoning with volatile substances. *Hum Toxicol.* 1989;8:277-286
35. Kerner K. Current topics in inhalant abuse. *NIDA Res Monogr.* 1988;85:8-29
36. Akerman HE. The constitution of adhesives and its relationship to solvent abuse. *Hum Toxicol.* 1982;1:223-230
37. Liss BI. Government, trade and industry and other preventive responses to volatile substance abuse. *Hum Toxicol.* 1989;8:327-330