BMJ
1840-1990

LONDON, SATURDAY 20 JANUARY 1990

Solvent abuse

Little progress after 20 years

Solvent abuse (volatile substance abuse, "glue sniffing") may no longer be headline news, but the problem has not gone away. A recent special issue of *Human Toxicology* has reviewed the subject, updating a similar review in July 1982. The prevalence, associated mortality, and sociological and toxicological aspects of the practice are now well documented, but efforts at prevention have made little impact.[1]

Recreational inhalation of volatile substances has enjoyed periodic vogues in the past, but the current epidemic spread to the United Kingdom from the United States in about 1970. Between 3·5% and 10% of current adolescents have at least experimented with solvents, and 0·5% to 1% of the secondary school population are currently abusers. The overall mortality is low, but deaths in the United Kingdom have increased from two in 1971 to over 100 each year since 1985.[1]

Typical solvent abusers are teenagers, though they may be younger.[2,3] Boys somewhat outnumber girls, and 90% of the deaths are in male abusers, of whom almost three quarters are under 30.[1] Abusers may be from any socioeconomic class, though children from ethnic minorities, lower socioeconomic classes, and disrupted families are overrepresented. No particular personality type seems to be specially vulnerable: users may be anxious, depressed, and psychopathic or happy-go-lucky, outgoing, and extravert. Abuse may be experimental (once or twice only) or recreational (a social group practice) or may become habitual in about 10% of those who start.[2,4] Most abusers eventually give up, but some continue through adulthood, and a few progress to abuse of alcohol and other drugs.[2,5]

Compounds abused include aliphatic, aromatic, or mixed hydrocarbons, esters, ethers, halogenated compounds, ketones, and anaesthetic gases. These are contained in many easily obtained "household" products that are cheap and conveniently packaged; indeed, the modern practice of solvent abuse has developed alongside the chemical industry.[1] Adhesives (such as rubber repair cement) contain several volatile components, including toluene and xylenes; aerosols (such as air fresheners, deodorants, and hair lacquer) contain butane and halons; dry cleaners and domestic stain removers include trichloroethylene or tetrachloroethylene; cigarette lighter refills and fuel for camping stoves contain *n*-butane and isobutane; and typewriter correcting fluid and thinners contain trichloroethane. Other common sources of volatile substances are petrol, paint stripper, fire extinguishers, and nail varnish remover. Amyl and butyl nitrites are sometimes inhaled for their vasodilator properties in homosexual encounters, and anaesthetists occasionally abuse volatile anaesthetics.

The solvent vapour may be inhaled by various techniques designed to maximise the available concentration—such as inhaling from a plastic bag or empty potato crisp packet (viscous products) or from a handkerchief or plastic bottle (liquids). Cigarette lighter refills are sometimes sprayed directly into the mouth.[1]

The first effect of inhalation is euphoria with excitatory effects probably due to disinhibition, but as the dose is increased the next stages are confusion, perceptual distortion, hallucinations (ecstatic or terrifying), and delusions, which may lead to aggressive and risk taking behaviour. With small doses the onset and recovery from intoxication are rapid—an advantage for the child who "sniffs" after school but wants to return home sober.[1] Experienced users, however, can maintain a "high" for up to 12 hours by judicious repeated sniffing.[6] Larger doses cause depression of the central nervous system with ataxia, nystagmus, dysarthria, drowsiness, coma, and occasional convulsions. Vomiting, coughing, sneezing, and increased salivation may occur at any stage.

Sudden death from solvent abuse may result from anoxia, vagal inhibition, respiratory depression, cardiac arrhythmias, or trauma.[1,7] Anoxia may be caused by inhalation of vomit (about a fifth of deaths) or placing a plastic bag over the head (about a tenth).[1] Vagal inhibition occurs reflexively from laryngeal stimulation and is particularly associated with butane and aerosol propellants sprayed into the throat. Respiratory depression is a direct result of general depression of the central nervous system. Cardiac arrhythmias account for over half the deaths; the mechanism is probably sensitisation of the myocardium to adrenaline and sympathetic stimulation.[7] Once arrhythmia develops the victim is resistant to resuscitation; the risk of sudden arrhythmia remains for some hours after inhalation. Any type of user is at risk, and each session is equally dangerous. Death from trauma follows accidents and suicide attempts; inexplicably it accounts for half of the deaths associated with toluene (mainly adhesives) but only 2-3% of deaths from other volatile substances.[1]

Chronic toxicity from organic solvents may cause irreversible damage to several organs. Toluene, trichloroethane, and trichlorethylene damage the kidney, liver, heart, and lungs.[8] Neurological effects include peripheral neuropathy (mainly associated with *n*-hexane and methyl-*n*-butyl ketone),

cerebellar disease, chronic encephalitis, and dementia (all mainly associated with toluene).[9] Dose-response relations are not clear, but the tenth of experimental sniffers who become chronic abusers are most at risk. Neuropsychological impairment is unlikely in volatile substance abuse as practised by schoolchildren.[10] Perioral eczema and chronic upper respiratory tract inflammation may result from repeated contact, especially with adhesives, and those who inhale petrol may in addition develop lead poisoning.[11] Solvent abuse in late pregnancy may result in neonatal depression, and solvents may possibly be teratogenic.[12,13] A degree of tolerance develops with daily use, and symptoms similar to alcohol withdrawal have been described in people who stop abruptly.[14] Organic solvents have a dependence potential, and (like other drugs that are abused) have reinforcing properties in animals.[15]

The treatment of acute solvent toxicity consists of standard measures, with cardiorespiratory resuscitation when necessary, conventional treatment of arrhythmias, and intensive supportive treatment.[4] The management of long term abusers is difficult: a combination of counselling and psychotherapeutic techniques is often needed.[4,16] Prevention is clearly a better strategy, but that is easier said than done. Voluntary guidelines issued by manufacturers in 1984 and the Intoxicating Substances (Supply) Act of 1985 placed some restrictions on the sales of solvents to people under 18, though solvent abuse is not illegal.[17] Most observers believe that the best approach is through education in public health with the provision of family and community counselling, residential care, and alternative recreational facilities.[18,19] Such measures are also applicable to smoking, alcohol, and other drug abuse—but they have done little yet to reduce the use of or the mortality from any of these drugs.

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Nibblers, gorgers, snackers, and grazers

Eating little and (very) often is beneficial to health

The belief that one should eat three "proper" meals a day and not between meals is deeply ingrained in the British public's mind[1] despite the evidence from studies of eating patterns that the traditional way is rapidly disappearing in favour of eating many times a day. Indeed, some people nowadays eat virtually continuously in a meal pattern that the marketing men have labelled "grazing."

Late last year a study was published showing that increasing meal frequency may have some important and potentially beneficial effects.[2] Professor David Jenkins and his colleagues have spent years (first in Oxford and now in Toronto) describing the metabolic effects of the rate of entry into the blood of absorbed carbohydrates and have argued for the benefits of slow release (lente) carbohydrates for patients with diabetes.[3]

Their recent paper may be seen as an extension of this concept; but instead of attenuating digestion and absorption by the choice of foods, the changes were made to the eating process itself.[2] In the study the effects of consuming a standardised 2500 kcal (10·46 MJ) diet in three meals were compared with those of the same intake divided into 17 portions of roughly equal size and composition eaten hourly throughout the day. Seven men took part in a randomised crossover design with each pattern being followed for two weeks. On the penultimate day of each regimen venous blood was taken while the men were fasting and further specimens taken at two hourly intervals for 12 hours for measurement of concentrations of hormones and other metabolic markers. On the final day of the diet a glucose tolerance test was carried out, and on the day after that the metabolic responses to a standardised breakfast were measured. The analyses were designed to examine the effects of the eating patterns on glucose, cholesterol, and lipid metabolism.

The result that attracted most attention was the fall in total serum cholesterol concentration with the 17 portions a day regimen, which was greater than that with the more conventional meal pattern. This fall was also accompanied by a significant decrease in low density lipoprotein cholesterol and apolipoprotein B concentrations. Serum insulin concentrations in the three meals a day period showed a pronounced cyclical pattern, whereas those for the 17 portion period rose a little from the overnight fasting value and remained virtually constant through the day. Similar differences were seen with the patterns of C peptide and free fatty acid concentrations in serum.

The authors think that the lower serum cholesterol concentrations in the 17 portion period may be due to the lower insulin concentrations in this period compared with the period for the conventional meal pattern. This resulted in reduced hepatic synthesis, which in turn is compatible with an increase in the number of low density lipoprotein