

the common sense conclusion that the prevention of sudden cardiac death and that of acute myocardial infarction are likely to be achieved by the same measures. Until primary prevention can abolish the problem we may save a substantial number of lives by investing more heavily in schemes for out of hospital resuscitation.

STUART M COBBE

Clinical Reader and Honorary Consultant Cardiologist,
John Radcliffe Hospital,
Oxford OX3 9DU

- 1 Armstrong A, Duncan B, Oliver MF, *et al*. Natural history of acute coronary heart attacks. A community study. *Br Heart J* 1972;34:67-80.
- 2 McNeilly RH, Pemberton J. Duration of the last attack in 998 fatal cases of coronary artery disease and its relation to possible cardiac resuscitation. *Br Med J* 1968;iii:139-42.
- 3 O'Doherty M, Tayler DI, Quinn E, Vincent R, Chamberlain DA. Five hundred patients with myocardial infarction monitored within one hour of symptoms. *Br Med J* 1983;286:1405-8.
- 4 Cobb LA, Baum RS, Alvarez H, Schaffer WA. Resuscitation from out-of-hospital ventricular fibrillation: 4 years' follow-up. *Circulation* 1975;51,52(suppl III):223-35.
- 5 Weaver WD, Lorch GS, Alvarez HA, Cobb LA. Angiographic findings and prognostic indicators in patients resuscitated from sudden cardiac death. *Circulation* 1976;52:895-900.
- 6 Ruskin JN, DiMarco JP, Garan H. Out-of-hospital cardiac arrest: electrophysiologic observations and selection of long-term antiarrhythmic therapy. *N Engl J Med* 1980;303:607-13.
- 7 DeWood MA, Spores J, Notske R, *et al*. Prevalence of total coronary occlusion during the early hours of transmural myocardial infarction. *N Engl J Med* 1980;303:877-902.
- 8 Roberts WC, Jones AA. Quantitation of coronary arterial narrowing at necropsy in sudden coronary death: analysis of 31 patients and comparison with 25 control subjects. *Am J Cardiol* 1979;44:39-45.
- 9 Spain DM, Bradess VA. Sudden death from coronary heart disease: survival time, frequency of thrombi and cigarette smoking. *Chest* 1970;58:107-10.
- 10 Davies MJ, Thomas A. Thrombosis and acute coronary-artery lesions in sudden cardiac ischemic death. *N Engl J Med* 1984;310:1137-40.
- 11 Friedman M, Manwaring JH, Rosenman RH, Donlon G, Ortega P, Grube SM. Instantaneous and sudden deaths: clinical and pathological differentiation in coronary artery disease. *JAMA* 1973;225:1319-28.
- 12 Janse MJ, Kleber AG. Electrophysiological changes and ventricular arrhythmias in the early phase of regional myocardial ischemia. *Circ Res* 1981;49:1069-81.
- 13 Mirowski M, Reid PR, Mower MM, *et al*. Termination of malignant ventricular arrhythmias with an implantable automatic defibrillator in human beings. *N Engl J Med* 1980;303:322-4.
- 14 Chamberlain DA, Jewitt DE, Julian DG, *et al*. Oral mexiletine in high risk patients after myocardial infarction. *Lancet* 1980;ii:1324-7.
- 15 Hondeghem LM, Grant AL, Jensen RA. Antiarrhythmic drug action: selective depression of hypoxic cardiac cells. *Am Heart J* 1974;87:602-5.
- 16 Ruskin JN, McGovern B, Garan H, Di Marco JP, Kelly E. Antiarrhythmic drugs: a possible cause of out-of-hospital cardiac arrest. *N Engl J Med* 1983;309:1302-5.
- 17 Hansteen V, Moienich E, Lorensten E, *et al*. One year's treatment with propranolol after myocardial infarction: preliminary report of Norwegian multicentre trial. *Br Med J* 1982;284:155-60.
- 18 Norwegian Multicentre Study Group. Timolol-induced reduction in mortality and reinfarction in patients surviving acute myocardial infarction. *N Engl J Med* 1981;304:801-7.
- 19 Beta-Blocker Heart Attack Trial Research Group. A randomized trial of propranolol in patients with acute myocardial infarction. *JAMA* 1982;247:1707-14.
- 20 Lipid Research Clinics. Coronary primary prevention trial results. 1. Reduction in incidence of coronary heart disease. *JAMA* 1984;251:351-64.
- 21 Sixty Plus Reinfarction Study Research Group. A double blind trial to assess long term oral anticoagulant therapy in elderly patients after myocardial infarction. *Lancet* 1980;ii:989-93.
- 22 Anonymous. Aspirin after myocardial infarction [Editorial]. *Lancet* 1980;i:1172-3.

Solvent misuse

The deliberate inhalation of volatile hydrocarbons by children and teenagers is a comparatively recent phenomenon, one of the first reports being by Merry and Zaccariadis in 1962.¹ The prevalence of solvent abuse in Britain is uncertain and is not restricted to any one area.² It occurs mainly in boys with a peak in those aged 13-15, and is usually a group activity.³⁻⁵ Boys may take up glue sniffing as a relief from boredom, in response to peer group pressure, out of curiosity, or in an attempt to gain status.⁵ Most such youngsters sniff solvents for a short time and on few occasions; only a few become dependent chronic users, and many of these mature out of the solvent habit.^{3,5,6} Solvent misuse may, however, also occur in adults.⁷ Press and Done found no relation between solvent abuse and socioeconomic factors,⁸ but a higher incidence in children from single parent families and families with paternal unemployment has been found in some studies,^{3,5} though not in others.⁹

The products that may be used include adhesives, dry

cleaning substances, petrol, lighter refills, acrylic paints, fire extinguishing agents, fingernail polish, antifreeze, paint thinners, cleaning fluids, and amyl nitrites.^{2,10} Adhesives containing toluene and acetone are the most commonly abused.^{4,5} All these products are readily available, inexpensive, easy to steal, legal, and act quickly; and their effects do not last long. There is no hangover—or the hangover is not as bad as that of alcohol.⁴ Glue is most often sniffed from a potato crisp bag; solvents may be directly inhaled from their containers or from a saturated rag.^{4,5}

Volatile solvents are central nervous system depressants. They act quickly because they are absorbed rapidly across the large surface area of the lungs and because of their solubility in lipid.¹¹ Subjective effects vary from person to person and depend on the amount of solvent inhaled and the duration of inhalation. The clinical features of solvent intoxication are similar to those of alcohol intoxication with initial stimulation followed by depression. Other symptoms may include euphoria, blurring of vision, tinnitus, slurring of speech, ataxia, feelings of omnipotence, headache, abdominal pain, anorexia, nausea, vomiting, jaundice, chest pain, bronchospasm, impaired judgment, irritability, and excitement. Less often a delirious state is seen with clouding of consciousness, illusions, and hallucinations.^{2,4,5} Many chronic users report transient symptoms of toxic psychosis, which often have an affective component.¹² Convulsions, status epilepticus,¹³ and coma² may occur. Self destructive and antisocial acts may be carried out by people under the influence of solvents.^{8,12} Psychological dependence and tolerance⁴ may develop, but physical dependence is rare.¹⁴ Solvent abuse is often associated with abuse of alcohol and multiple illicit drugs.¹² The effect can be potentiated by prior consumption of alcohol or cigarettes.¹²

Erythematous spots around nose and mouth ("glue sniffer's rash") may be observed when a plastic bag is used for inhalation.⁵ The physical sequels of prolonged solvent abuse have been reported to include aplastic anaemia¹⁵ and acute hepatic and renal damage.¹⁶ Irreversible neurological sequelae such as optic atrophy,¹⁷ encephalopathy,¹⁸ cerebellar degeneration,¹⁹ and equilibrium disorders²⁰ have been reported in adults who are chronic abusers. Toluene inhalation may cause encephalopathy in children and may lead to permanent neurological damage.¹¹ Peripheral neuropathy has been attributed to *n*-hexane inhalation.²¹ The electroencephalogram may show diffuse or focal slow or sharp wave complexes.^{11,18} Waldron has reviewed the effects of organic solvents as seen in industrial use.²²

In Britain from 1971 to 1981 some 140 deaths were identified from abuse of volatile substances.²³ Death rates were highest in conurbations and in Scotland, Northern Ireland, and northern England. The chief substances were butane (28%), solvents in adhesives (23%), other solvents (26%), aerosols (15%), and fire extinguishing agents (5%). Most deaths occurred alone at home. In 41% of cases death was only indirectly associated with solvent abuse (trauma 8%, plastic bag over the head 19%, and inhalation of stomach contents 14%). In nearly half death was attributed to the direct toxic effects of the substance; this proportion was highest with aerosols and lowest with solvents in adhesives.²³ An epidemic of 110 sudden deaths without plastic bag suffocation in American youths was reported by Bass.²⁴ Severe cardiac arrhythmias, intensified by hypercapnia, stress, or activity, are thought to be the most likely explanation for sudden death after inhalation of aerosols.²⁴ The lack of morbidity and mortality in a group of 300 "glue sniffers" seen at a Glasgow clinic is reassuring.⁵

Poor school adjustment and scholastic performance have been noted in chronic glue sniffers.⁸ "Sniffers" do not, however, appear to have less potential abilities than other students.^{25,26} Performance decrements of "sniffers" are more likely related to poor motivation.

There is no firm evidence to date that glue sniffing leads on directly to abuse of other substances.²⁷ In most cases solvent misuse is transient.^{5,27,28} Davies *et al* (p 109), however, highlight the fact that a few chronic solvent misusers do subsequently take illicit drugs and they are undertaking a much needed follow up study of a sample of solvent misusers to identify the characteristics of this minority group.

Toluene is the main solvent used in commercially available adhesives in Britain and is probably responsible for all the desired and toxic effects.¹¹ Toluene is metabolised in hepatic microsomes by oxidation to benzoic acid, which is conjugated with glycine to form hippuric acid and is eliminated in this form through the kidneys. The blood concentration of toluene may show a diphasic pattern, with an initial peak followed by a trough reflecting lipid binding and a second peak as toluene is slowly released in the blood.¹¹ In the investigation of a patient suspected of solvent abuse additional information may be obtained by measuring the urinary concentrations of benzoic acid and hippuric acids (metabolites of toluene) and of toluric acids (metabolites of xylene). The toluric acids are not normally present in urine, but hippuric acid (derived primarily from a metabolism of the food preservative sodium benzoate) is a normal urinary constituent. In general, a urinary hippurate to creatinine ratio of over 1 indicates recent exposure to toluene.²⁹

How can solvent abuse be prevented? Restriction on sales, reformulation of solvent products, and addition of aversive substances have all been found to be impracticable; some benefit has been shown from a voluntary, informed code of conduct practised by retailers.⁵ The controls of the Misuse of Drugs Act or similar legislature controls would probably be inappropriate.³⁰ The Solvent Abuse Scotland Act 1983 allows a child being found by more than one person to be under the influence of solvents to be referred to the children's panel as being perhaps in need of compulsory medical care. Two Glasgow shopkeepers who had endangered children by selling them "glue sniffing kits" were sentenced to three years' imprisonment; the judges held that such an abuse of young people fell within the ambit of a concept in Scottish common law of "Culpable and reckless conduct" causing real injury to others.³¹

Acute solvent intoxication is usually a brief and self limiting illness—but the child is best kept under observation in a place of safety. In chronic users a complete history, physical and neurological examination, and relevant laboratory investigations should be carried out. Particular attention should be paid to the lonely, dependent chronic abuser with emotional problems—who may also be using more dangerous substances. The problem is best dealt with by general practitioners, community physicians, social and youth workers, and community branch of the police.⁵ Health education, improved recreational facilities, and help to single parents and those with alcohol, physical, marital, and psychiatric problems have proved to be useful.⁵ Individual, family therapy,¹² behaviour modification programmes,⁵ and suggestion techniques³² may be useful in selected cases.

Glue sniffing appears to be essentially a socialised disturbance of conduct, and in only a few individuals is

there a mixed disturbance of conduct and emotions for which psychiatric intervention is warranted.⁵ Minimal, non-dramatic, non-alarmist intervention at an early stage, with the participation of parents, appears to be effective, and this is consistent with experience with other drugs, especially alcohol.^{5,33}

I SOURINDRHIN

Consultant Psychiatrist,
Dykebar Hospital,
Paisley PA2 7DE

- Merry J, Zaccariadis N. Addiction to glue sniffing. *Br Med J* 1962;ii:1448.
- Francis J, Murray VSJ, Ruprah M, Flanagan RJ, Ramsay JD. Suspected solvent abuse in cases referred to the poisons unit, Guy's Hospital, July 1980-June 1981. *Hum Toxicol* 1982;1:271-80.
- Masterton G, Sclare AB. Solvent abuse. *Health Bull (Edinb)* 1978;2:305-9.
- Watson JM. Solvent abuse: presentation and clinical diagnosis. *Hum Toxicol* 1982;1:249-56.
- Sourindrhin I, Baird JA. Management of solvent abuse: a Glasgow community approach. *Br J Addict* 1984;79:227-32.
- Cohen S. Glue sniffing. *JAMA* 1975;231:653-4.
- Hershey CO, Miller S. Solvent abuse: a shift to adults. *Int J Addict* 1982;6:1085-9.
- Press E, Done AK. Solvent sniffing—physiological effects and community control measures for intoxication from the intentional inhalation of organic solvents. *Pediatrics* 1967;39:451-61.
- Kaufman A. Gasolene sniffing among children in a Pueblo Indian village. *Pediatrics* 1973;50:1060-4.
- Oliver JS, Watson JM. Abuse of solvents "for kicks." A review of 50 cases. *Lancet* 1977;ii:84-6.
- King MD, Day RE, Oliver JS, Lush M, Watson JM. Solvent encephalopathy. *Br Med J* 1981;283:663-5.
- Skuse D, Burrell S. A review of solvent abusers and their management by a child psychiatric outpatient service. *Hum Toxicol* 1982;1:321-9.
- Alister C, Lush M, Oliver JS, Watson JM. Status epilepticus caused by solvent abuse. *Br Med J* 1981;283:1156.
- Watson JM. Glue sniffing: two case reports. *Practitioner* 1979;222:845-7.
- Powars D. Aplastic anemia secondary to glue sniffing. *N Engl J Med* 1965;273:700-2.
- O'Brien ET, Yeoman WB, Horby JAE. Hepatorenal damage from toluene in a "glue sniffer." *Br Med J* 1971;iii:29-30.
- Keane JR. Toluene optic atrophy. *Ann Neurol* 1978;4:390.
- Knox JW, Nelson JR. Permanent encephalopathy from toluene inhalation. *N Engl J Med* 1966;275:1494-6.
- Grabaski DA. Toluene sniffing producing cerebellar degeneration. *Am J Psychiatry* 1961;118:401-62.
- Sasa M. Equilibrium disorders with diffuse brain atrophy in long-term toluene sniffing. *Arch Otorhinolaryngol* 1978;221:163-9.
- Korobkin R, Asbury AK, Saumner AJ, Nielsen SL. Glue sniffing neuropathy. *Arch Neurol* 1975;32:158-62.
- Waldron HA. Effects of organic solvents. *Br J Hosp Med* 1981;26:645-9.
- Anderson HR, Dick B, Macnair RS, Palmer JC, Ramsay JD. An investigation of 140 deaths associated with volatile substance abuse in the United Kingdom (1971-1981). *Hum Toxicol* 1982;1:207-21.
- Bass M. Sudden sniffing death. *JAMA* 1970;212:2075-9.
- Massengale ON, Glaser HH, Lelievre RE, Dodds JB, Klock MH. Physical and psychological factors in glue sniffing. *N Engl J Med* 1963;269:1340-4.
- Mahmood Z. Cognitive functioning of solvent abusers. *Scott Med J* 1983;28:276-80.
- Herzberg JL, Wolkind SN. Solvent sniffing in perspective. *Br J Hosp Med* 1983;29:72-6.
- Watson JM. Solvent abuse by children and young adults: a review. *Br J Addict* 1980;75:27-36.
- Ramsay JD, Flanagan RJ. The role of the laboratory in the investigation of solvent abuse. *Hum Toxicol* 1982;1:299-311.
- Department of Health and Social Security. *Report of the Advisory Council on Misuse of Drugs. Treatment and rehabilitation*. London: HMSO, 1982.
- Anonymous. Abusing the statute book [Editorial]. *The Times* 1984 May 5:9 (cols 1-3).
- O'Connor D. The use of suggestion techniques with adolescents in the treatment of glue sniffing and solvent abuse. *Hum Toxicol* 1982;1:313-20.
- Orford J, Edwards G. *Alcoholism*. London: Oxford University Press, 1977.

Hypercarotenaemia

Yellow pigmentation in human skin may in some instances be due to jaundice but in others it may be due to no more than an unusually high consumption of carrots or other source of carotene. Hypercarotenaemia can be distinguished from jaundice by the fact that the sclerae retain their normal white colour.¹ With so much public interest in cult diets and extreme forms of vegetarianism some individuals may develop a passion for eating carrots and may consume up to 4 lb (1.75 kg) daily. Pigmentation occurs first on the palms and the soles and may extend to the nasolabial folds. The condition is harmless—for the body converts carotene to retinol (vitamin A) only in amounts as required; hypervitaminosis A cannot, therefore, occur from overconsumption of carotene.

When patients who have developed a passion for eating carrots are told that the condition is not harmful some may prefer to remain yellow and go on eating them. If the patient wants the colour to be dispersed, however, he or she need only reduce the number of carrots eaten to an ordinary level, and the pigmentation will disappear within a few weeks.