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# Solvent abuse and the heart

The deliberate inhalation of volatile substances, usually halogenated or unsubstituted hydrocarbons, to obtain a "high" is common among teenage boys and causes important morbidity and mortality[1 2]—80 deaths in Britain in 1983.[3] Solvent abuse may cause both acute and chronic cardiotoxicity, and this issue carries three reports of serious cardiac problems associated with exposure to volatile hydrocarbons (pp 727 and 739).

In 1970 Bass reported an epidemic of sudden deaths associated with solvent sniffing in the United States.[4] He proposed that these deaths were caused by an arrhythmia and suggested that volatile hydrocarbons might sensitise the heart to the arrhythmogenic effects of endogenous catecholamines. There is now much evidence to support this hypothesis. Firstly, solvent abuse has been followed by documented ventricular fibrillation.[5-7] Secondly, sudden death related to solvent abuse has occurred often in circumstances associated with intense cardiac sympathetic stimulation—physical exertion, particularly running,[4 8] and various forms of autoerotic behaviour.[3 9] The case of myocardial infarction, possibly caused by coronary artery spasm, and ventricular fibrillation described by Cunningham *et al* (p 739) fits the same pattern. Thirdly, well controlled studies in dogs have shown that adrenaline given after many different inhaled volatile hydrocarbons may produce serious ventricular arrhythmias including ventricular fibrillation.[10 11] Furthermore, ventricular tachycardia has been seen in conscious dogs subjected to a loud noise or made to run on a treadmill after inhaling volatile hydrocarbons.[10 12] Hypoxia,[10] hypokalaemia,[13] and alcohol[14] may all increase the risk of an arrhythmia after solvent abuse, and the cases described by McLeod *et al* (p 727) suggest that there may also be an adverse interaction with halothane.

Studies of acute toxicity have shown that inhaled volatile hydrocarbons can also induce bradyarrhythmias[15 16] and hypotension[17]; these observations were made, however, in animals under general anaesthesia and may not therefore be relevant to those who sniff glue or inhale from aerosol cans.

The relative toxicity of the many chemicals inhaled is not known, largely because we do not know the prevalence of abuse of each agent. Nevertheless, all of these substances may be cardiotoxic. Their effects on the heart are probably caused by non-specific physicochemical actions and seem to occur at doses similar to those that affect the central nervous system.[11] This implies that any sniffer who obtains a "high" also runs the risk of developing an arrhythmia, particularly if he or she then exercises.

Rhabdomyolysis, renal and hepatic damage, and various neurological, psychiatric, and metabolic syndromes have been attributed to habitual solvent abuse.[1 2 13] Now Wiseman and Banim (p 739) and McLeod *et al* (p 727) report three cases of dilated cardiomyopathy associated with chronic solvent abuse or in one case heavy occupational exposure to 1, 1, 1-trichloroethane. Although there are one or two similar case reports,[18 19] the evidence that volatile hydrocarbons may cause a dilated cardiomyopathy is still only anecdotal. Nevertheless, physicians should consider chronic solvent exposure when treating patients with dilated cardiomyopathy, and anaesthetists should be aware of the potential hazards of using halothane or similar agents in patients who may be solvent sniffers.

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# Drums begin to beat in the waiting list jungle

Radical change in medical practice is thought to be necessary to shorten the time patients wait for treatment and investigation in the National Health Service. Just last month the government allocated another £25m to help health authorities reduce their waiting lists (28 February, p 590), and last week saw surgeons and managers meeting together at the King's Fund to discuss what should be done (p 783). If quicker is better—and in some cases present treatment delays mean no treatment[1 2]—are doctors willing to alter the referral system and assist their patients to earlier treatment? Will general practitioners refer patients away from the local hospital and named consultant on to others who can see, diagnose, and treat more quickly? Can consultants be persuaded to send their patients on to colleagues within their specialty who can offer quicker treatment?

There is a practical difficulty. Despite modern communications, advances in technology, and the Körner reports on information systems in the NHS, there is no system within

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the health service that tells doctors which hospital and consultant can see their patients most quickly. Has outside industry anything to teach the health service? For instance, could the system be organised like the travel business so that customers (patients) are linked to flights (theatre sessions) and hotels (hospitals) to provide holidays (treatment)? As part of the national waiting list initiative North West Thames Regional Health Authority and North Hertfordshire District Health Authority are jointly piloting an information service that will explore and identify the sort of information that will help general practitioners to obtain earlier treatment for their patients.

Enthoven proposed an "internal market model" to solve the present ills of the NHS.[3 4] He visualised that an authority would provide and pay for the health care of its residents but be able to obtain further payments at a fixed rate for the emergency care of non-residents and to negotiate an even higher fee for a non-emergency service. This conjures up the possibility of health authorities competing with one another and perhaps the private sector to provide certain forms of health care; and patients would travel from one end of Britain to another for treatment. This has already begun. Gloucester Health Authority recently contracted with Victoria (now Riverside) Health Authority to provide total hip replacement operations at £1200 a hip, and several authorities have contractual arrangements with the private sector.[5] There is also an opportunity to test some ideas. The projects funded by the national waiting list initiative will allow authorities to compare and contrast the cost, treatment, and care provided for similar cases by the private sector, by locally expanded NHS facilities, and by other health authorities using their spare capacity.

Health care is provided free at the time of use, and waiting lists are a way of controlling the access that patients have to treatment. In other countries, such as the United States, the income of the patient is the rationing mechanism. Waiting lists are misunderstood by both doctors and the public. Doctors have used them to show a need for resources and to manage patients whose illness will improve with time; politicians and the public view them as the measure of government performance on the delivery of health care. Yet waiting lists for treatment are put together in many different ways. They are subject to the working practices of particular consultants, whose systems may be quite different from those of their colleagues in the same specialty as well as from those in other specialties. The data collection systems are just as inconsistent.[2]

Some health authorities have tried to shorten their waiting lists by getting agreement among doctors,[2 6] by having common waiting lists and preassessment clinics, by encouraging weekend operating sessions, and by providing managers to organise and coordinate outpatient attendances, admissions, and the number of beds and theatre sessions in a specialty.[7 8] Others believe that waiting times would shorten if doctors were more efficient, adopted new techniques, and increased their throughput of patients. This they might do by increasing the numbers of day and short stay cases and the amount done in outpatient clinics.[2] The Royal College of Surgeons of England suggests that the amount of day care could be increased,[9] but national and professional edicts require local will and facilities (including adequate retraining) to happen.

The national care system does require more resources to provide better facilities and modern equipment and to maintain adequate numbers of nurses, technicians, and junior doctors. It also needs scope to test and explore some of these new ideas. There is a risk that district health authorities and hospitals will give higher priority to non-emergency patients for whom they receive additional payment than to local residents and that the patients who live further away will be less likely to be referred.

After almost 40 years of the same referral systems and waiting practices, is it not time for consultants, general practitioners, and managers to test out alternative ways to provide health care free at time of use for their patients?

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# Overuse of monitoring of blood concentrations of antiepileptic drugs

Monitoring serum or plasma concentrations of antiepileptic drugs has become routine for largely historical reasons: phenytoin has a non-linear relation between the dose and the serum concentration[1] and a dose related neurotoxicity (drowsiness, ataxia, dysarthria, and nystagmus). This results in a narrow therapeutic window, and monitoring is necessary to avoid neurotoxicity in patients who continue to have seizures. The concept of the "therapeutic" or "optimal" range for phenytoin has been extended to other antiepileptic drugs, and many laboratories now routinely estimate serum concentrations of drugs other than phenytoin. This is a questionable practice.

A single measurement will give a good approximation of the steady state for drugs with long half lives (phenytoin and phenobarbitone) but not for drugs with short half lives. Measurements of sodium valproate concentrations from specimens taken at random during the day are virtually uninterpretable as they may represent unpredictable peak, trough, or intermediate concentrations.[2] Collecting early morning specimens for measuring troughs is, however, rarely practicable.

Doctors must be aware of what is measured during routine estimations of blood concentrations of antiepileptic drugs and, perhaps more importantly, what is not measured. Some drugs have metabolites that seem to contribute to the therapeutic effect but which are not routinely monitored. These include the 10, 11-epoxide of carbamazepine[3] and phenylethylamonamide derived from primidone.[4] Most laboratories determine the drug concentration in whole

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