physically harmful consequences of substance abuse. To accept less stringent criteria on the grounds that false reports of toxic effects do not matter because they may deter only drug abusers while false reports of adverse effects do matter because they may deter drug prescribers is to adopt a dangerous double-standard on a very shaky foundation. For, firstly, the deterrent effect of scares about toxic effects is far from established, and, secondly, a whole segment of medical data becomes usable only by those who share the assumptions of those who collected it.

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**We sent this letter to the authors, who reply below.—ED, *BMJ*.

SIR,—We should like to reply to the criticisms raised by Mr Woodcock and coworkers regarding our report on solvent encephalopathy (5 September. p 663). Their letter highlights the difference in approach between clinicians, who are faced with the individual, and epidemiologists, who look at the whole. We published our report in the clinical topics section of the *BMJ* to bring to the attention of other clinicians our experience that toluene intoxication should be considered as a cause of what is commonly described as "unexplained encephalopathy." Encephalopathy is commonly used to describe a potentially reversible state of cerebral dysfunction, which may be due to a variety of causes—for example, uraemia, diabetes, hypertension, etc. If the state persists or is severe enough, however, structural damage may follow. We were concerned that one of the children studied had a cerebellar ataxia one year after the acute illness, and five others appeared not to have returned to their normal mental state at two weeks to three months at their latest follow-up. This reaction might be considered to fall into type A described by Dr G R Venning (23 January, p 249)—that is, that which is consequent on the pharmacological action of a drug—as toluene is known to affect cerebral function.[1] We hoped that our report might increase awareness of toluene abuse as a cause of unexplained encephalopathy and lead to early confirmation of the diagnosis by assay and close follow-up so that a fuller pattern of the toxic effects might emerge. We are at present engaged on such a study.

One of the reasons for the lack of details on duration and intensity of exposure is the difficulty in obtaining reliable information from the children. To answer, however, a few specific points:

(1) As far as we can ascertain, the duration of glue sniffing in the children we reported was from three to 24 months.

(2) Symptoms persisted in those children who recovered completely for periods ranging from three to 14 days. In five children parents continued to be concerned about a change of behaviour and personality up to the time they were lost to follow-up (two weeks to three months). In these children formal psychometric testing showed poor concentration, short attention span, and slow reaction time.

(3) It was established in 14 cases that a limited group of three proprietary brands of adhesives, all containing 80% toluene, were abused. It was shown in table 1 that toluene assay, by gas chromatography, was performed in seven cases and showed toluene concentrations ranging from 0·8 $\mu$g/g to 8 $\mu$g/g of blood. A wide range of other solvents were also looked for,[2] with negative results.

(4) Our comment about "epidemic proportions" stems from the fact that in Scotland alone in 1980, 1300 new cases of solvent abuse were reported to police from a secondary school population of almost half a million.[3]

Our report was intended to highlight the medical aspects of a growing social problem. We are fully aware of the limitations of retrospective studies and in particular of anecdotal reports, but we feel that our report provides evidence for serious concern about the long-term sequelae of solvent abuse and hope it stimulates well planned prospective studies.

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## Xylene-induced epilepsy following innocent glue sniffing

SIR,—We were interested in the report of severe status epilepticus in a 15-year-old glue sniffer (31 October, p 1156). We encountered recently an adolescent boy whose major and minor seizures regularly followed aeromodelling using a xylene-based glue but not when a methyl-ethyl-ketone-based glue was used.

This boy presented at this hospital after a generalised grand mal seizure in September 1979. He had a history of absence seizures over the previous two years, which were easily controlled with ethosuximide from his GP. An electroencephalogram showed occasional sharp wave bursts in the parietal leads consistent with epilepsy. His mother had noticed earlier that his brief absences, which had been abolished when ethosuximide therapy was started, relapsed over a 24-hour period after he had spent an evening making a model aircraft. This was repeated on several occasions. The parents made some experiments and found that Linka, a more expensive glue, could be safely used, but a cheaper glue (Stephen's) was regularly followed by absence attacks.

The evening before his severe generalised grand mal attack, he had run out of Linka glue and had reverted to Stephen's glue, which he had used all evening. At 6 40 pm the next evening (24 hours later) he had his grand mal fit. A month later he had a further series of absences, again 24 hours after using Stephen's glue. A month later he had further absences, this time not associated with exposure to glue. Anticonvulsant therapy was changed to sodium valproate, and he has remained well to date. We repeated his electroencephalogram after sniffing Stephen's glue but no acute changes were noticed. Although the parents did not keep a diary of these events, they were exceptionally good witnesses, and we accept their observations at their face value.

Inquiry to the manufacturers revealed that the solvent in Stephen's glue is xylene (a hydrocarbon solvent of the same family as toluene). The solvent in Linka glue is a mixture of methyl ethyl ketone and tetrahydrofuran, both central nervous system depressants. Xylene, benzene, and toluene are widely used in industry and are recognised as safe solvents —except on exposure to overwhelming concentrations, when respiratory and central nervous system symptoms have occurred.[1] But in only one case has a probable epileptic fit been described (following exposure to xylene in paint fumes).[2]

There is now an extensive literature on the biomedical consequences of solvent abuse.[3] Three cases of epilepsy with Evo-stik (toluene) were reported in 1979[4] and another three cases of convulsions in 1981 (5 September, p 663). All these case reports have concerned toluene, but xylene is pharmacologically close to toluene and it seems likely to carry a similar risk.

We are grateful to Dr J A Hoskins, MRC Toxicology Unit, Carshalton, Surrey, for his help.

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## Vaccination against tick-borne encephalitis

SIR,—The viral disease, tick-borne encephalitis, in Europe has recently received wide publicity in the press and on radio. The disease is transmitted to man by the tick *Ixodes ricinus*, which has a wide distribution in Europe and Great Britain. The disease is absent in Great Britain, although the related louping ill, affecting sheep and cattle and transmitted by the same species of tick, is a serious veterinary but not public health problem.

While we do not believe that tick-borne encephalitis is actually spreading westwards in Europe, there has been an increase in its incidence due to increased exposure to infected ticks in forested areas in Europe during the course of leisure activities such as walking, picnicking, and camping, as well as to an increased awareness on the part of clinicians. The increased incidence is a cause for concern but not alarm.

We have had inquiries from intending travellers to Austria and Czechoslovakia about protection against the disease. A safe and effective vaccine is produced by the Public Health Laboratory Service at the Centre for Applied Microbiology and Research under agreement with Immuno Limited. Limited stocks are held by Immuno Ltd, Arctic House, Rye Lane, Dunton Green, Nr Sevenoaks, Kent TN41 5HB (telephone 0732-458101), and can be obtained from them by giving the name of the person to be vaccinated and the name of the prescribing doctor. Two doses spaced four to six weeks apart and a "booster" after 12 months are recommended. The two initial doses should be taken well in advance of the date of travel.