CORRESPONDENCE

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Increase in deaths from deliberate inhalation of fuel gases and pressurised aerosols

SIR,—The monitoring of deaths from deliberate inhalation of volatile substances is made difficult by the variety of substances abused, the various modes by which death may occur, and the lack of an appropriate code under the International Classification of Diseases. Since 1983, supported by the Department of Health, we have systematically ascertained deaths in the United Kingdom by three methods: a press clippings service; regular letters to coroners; and liaison with the Office of Population Censuses and Surveys, who have provided access to their associated digit coding system. From Scotland data are obtained direct from the Crown Office. Once ascertained, further details are obtained from necropsy and toxicology reports and inquest proceedings.[1] We wish to draw attention to important recent trends in the nature of this problem.

For analysis we have followed accepted practice in classifying the abused products into four categories: (*a*) gas fuels, which contain butane and propane and are abused from products such as gas lighter refills and cannisters for camping stoves and blowtorches; (*b*) pressurised aerosols, which contain halons (with increasing use of butane) as propellants; (*c*) solvents in glues, which mainly comprise toluene; and (*d*) other substances, which comprise a wide range of products and chemicals including principally the solvent 1,1,1-trichoroethane, which is found in thinning and cleaning agents and surgical plaster removal.[2] Trend analysis using a poisson regression model and weighting for the population at risk was carried out using the statistical package GLIM.[3]

The numbers of deaths in the United Kingdom following volatile substance abuse from 1983 to 1988 are shown in the table. Overall the upward trend is highly significant, and in the most recent year the number was the highest ever at 134. The death rate in males aged 10-19, among whom over 60% of deaths occur, doubled from 11·2 to 20·7 per million over this time, and in 1988 deaths from volatile substance abuse accounted for 4% of all deaths and 8% of deaths from injury and poisoning.

Number of deaths associated with volatile substance abuse in the United Kingdom, 1983-8, by type of substance

Year	Gas fuels	Aerosols	Solvents in glue	Other	Not known	Total
1983	19	13	24	26	0	82
1984	31	10	15	28	0	84
1985	30	20	37	31	0	118
1986	37	23	18	22	2	102
1987	37	33	21	22	1	114
1988	53	46	16	17	2	134
Trend p	<0·001	<0·001	NS*	NS*		<0·001

*Not significant.

When the data were divided by the type of substance, there was evidence of a significant trend over time ($p<0·001$). Looking at each substance separately, there were highly significant upward trends in deaths associated with both gas fuels and pressurised aerosols ($p<0·001$) while there was little or no change in deaths associated with solvents in glue and with other substances. To control more tightly for changes in population structure, the trend analysis was repeated for the 10-24 age group—among whom 88% of deaths occur—and similar results were obtained.

Our methods have not changed over the six years to 1988 and it is unlikely that many deaths have been missed where there was some mention of volatile substance abuse. Although the overall increase might be explained by a progressively greater awareness, we believe that it is more likely that such deaths are increasing. If so it will be in spite of the considerable efforts which have been made to control the practice through education and legislation. It may be relevant that those substances for which there has been an increase are all gaseous under ambient conditions and thus may be easily inhaled at unexpectedly high concentrations. The recent trend of substituting halons in pressurised aerosol products with butane (either wholly or in part) is unlikely to make them safer and may add to the risk of fire. One interpretation of these trends could be that efforts to control glue sniffing and solvent abuse have been relatively successful, at least in containing mortality. The unwanted side effect of this may have been a shift towards abuse of pressurised products, which are more dangerous, if only because the user finds the dose more difficult to control. Whether or not this is the case, it is now important that efforts to educate the population emphasise the dangers of inhaling gas fuels or aerosol products.

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1 Anderson HR, Macnair RS, Ramsey JD. Deaths from abuse of volatile substances: a national epidemiological study. *Br Med J* 1985;**290**:304-7. (3 February.)
2 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**:261-9.
3 Payne CD, ed. *The GLIM system manual. Release 3.77.* Oxford: Numerical Algorithms Group, 1985.

Aerosol inhalers

SIR,—I would like to add a word of caution to Dr Stephen P Newman's editorial before we start turning to devices such as the Turbohaler or Diskhaler in the possibly mistaken belief that they are environmentally friendly.[1] They contain more plastic and are more complicated in their construction than their predecessors, the metered dose aerosol inhalers. Before shifting to the newer devices for environmental reasons, I would have to be reassured that the energy required in obtaining the raw materials and manufacturing the devices was not doing just as much harm by the production of carbon dioxide as do the propellants in the older type of inhalers.

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1 Newman SP. Aerosol inhalers. *Br Med J* 1990;**300**:1286-7. (19 May.)

End tidal carbon dioxide detector for monitoring cardiopulmonary resuscitation

SIR,—We have recently assessed the function of the disposable end tidal carbon dioxide detector (FEF; Fenem) described by Dr D Higgins and colleagues.[1] The device distinguishes oesophageal from tracheal intubation[1,2] and was promoted by Dr Higgins and colleagues as a simple instrument for assessing cardiac output during resuscitation attempts. It may also be of use for estimating end tidal carbon dioxide concentrations when capnometry is not available.

This non-aqueous colorimetric carbon dioxide monitor uses a chemical pH indicator, metacresol purple, to detect the presence of carbon dioxide in expired respiratory gas.[2] The colours mauve, tan, and yellow are claimed by the manufacturers to approximate to the ranges of end tidal carbon dioxide concentration of 0·03%-<0·5%, 0·5%-<2·0%, and 2·0%-5·0% respectively (product data sheet, Fenem, New York). The colour should vary between expiration and inspiration as the end tidal carbon dioxide concentration rises and falls. This reversible colour change allows for up to two hours' usage in the absence of active humidification.[3]

For this device to be of use it must function reliably and consistently and should detect carbon dioxide specifically. Of greatest importance is that the function of the device should not be altered by contaminants that might be encountered under the circumstances in which it will be employed.

During a recent resuscitation attempt an FEF end tidal carbon dioxide detector confirmed the clinical impression of asystole during external cardiac massage, correct endotracheal tube placement having been confirmed visually. In keeping with the recently revised recommendations of the British Resuscitation Council, adrenaline solution was instilled directly into the endotracheal tube.[4]

On resuming ventilation an immediate and irreversible colour change from mauve to yellow occurred in the carbon dioxide detector. Consequently, the device no longer predicted either correct tube placement or efficacy of resuscitation efforts.

We have further investigated this phenomenon in vitro by instilling drugs into the device. An identical and permanent change to yellow occurs with the other drugs—lignocaine, adrenaline, and atropine—used intratracheally in patients suffering a cardiac arrest and with gastric aspirate. No significant colour change occurred with sterile water, physiological saline, or pulmonary oedema fluid.

Our findings suggest that the FEF device would not necessarily detect oesophageal intubation if the colour indicator became contaminated with gastric acid and remained permanently yellow. During cardiopulmonary resuscitation the device could no longer be used as an indicator for the effectiveness of resuscitation manoeuvres once lignocaine, adrenaline, or atropine had been instilled into the endotracheal tube and an irreversible colour change had occurred. Additionally, if the device is used in a breathing system that allows rebreathing—for example, a Bain or Mapleson B system—the usual breath to breath colour change of the carbon dioxide detector is not seen.

In conclusion, it is clear that the FEF end tidal carbon dioxide detector is not specific for carbon dioxide and its use must be tempered with caution. We have informed the device's manufacturers of our findings.

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1 Higgins D, Hayes M, Denman W, Wilkinson DJ. Effectiveness of using end tidal carbon dioxide concentration to monitor cardiopulmonary resuscitation. *Br Med J* 1990;**300**:581. (3 March.)
2 Goldberg JS, Rawle PR, Zehnder JL, Sladen RN. Colorimetric end-tidal carbon dioxide monitoring for tracheal intubation. *Anesth Analg* 1990;**70**:191-4.
3 Feinstein R, White PF, Westerfield SZ. Intraoperative evaluation of a disposable end-tidal Co_2 detector. *Anesthesiology* 1989; **71**:A461.
4 Chamberlain DA. Advanced life support. Revised recommendations of the Resuscitation Council (UK). *Br Med J* 1989; **299**:446-8.

AUTHORS' REPLY,—We wish to add our comments to those of Dr J D Muir and colleagues. Our experience of using the FEF carbon dioxide detector[1] during cardiopulmonary resuscitation had alerted us to the problems of gastric acid contamination and the effects of rebreathing when inappropriate circuitry is used.

Soiling with gastric acid produces a deep orange colour on the dome of the device, which is permanent and quite different from the yellow colouration produced by carbon dioxide. The use of an inappropriate gas delivery system during resuscitation attempts, which results in rebreathing to the extent that the detector does not return to the expected purple colour during inspiration, is an event that causes concern. There is an urgent need for junior medical staff to be aware of the basic principles of all gas delivery systems so that they are used appropriately during cardiopulmonary resuscitation. The FEF detector does have a useful role here in that it will indicate rebreathing.

The effect of intratracheal instillation of drugs on the detector during cardiopulmonary resuscitation has been highlighted by Dr Muir and colleagues and is further cause for concern. We investigated the effects of both nebulised drugs and the same drugs in direct contact with the detector. The table gives the results.

The yellow colouration produced by a 1:10 000 solution of adrenaline was identical with that produced by carbon dioxide and that shown on the dome of the detector. This colour was permanent and occurred despite the presence of a heat and moisture exchanger filter in the circuit between the nebuliser and the detector. We believe that the manufacturers of this device should issue a warning about this effect (we have advised them of our findings). Interestingly, in our study of 100 intubations during which the larynx and trachea were sprayed with 4% lignocaine the detector remained responsive to carbon dioxide.[2]

In conclusion, those who use this device during cardiopulmonary resuscitation must look for a cyclical colour change from purple to yellow during respiration to indicate effective ventilation and perfusion of the lungs. A fixed orange colour indicates contamination with acid. A fixed yellow colour indicates either an inspired carbon dioxide concentration above 2% or contamination of the device with tracheally administered drugs. A fixed purple colour indicates either inadequate perfusion or failed intubation. We still believe that this simple apparatus provides considerable information during cardiopulmonary resuscitation which is of great value.

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1 Higgins D, Hayes M, Denman W, Wilkinson DJ. Effectiveness of using end tidal carbon dioxide concentration to monitor cardiopulmonary resuscitation. *Br Med J* 1990;**300**:581. (3 March.)
2 Denman W, Hayes M, Higgins D, Wilkinson DJ. The Fenem carbon dioxide device. *Anaesthesia* 1990;**45**:465-7.

Effect of drugs on colour change shown by FEF carbon dioxide detector when nebulised and when in direct contact with detector

Drugs	pH	Permanent colour change with nebulised drugs	Permanent colour change with drugs in direct contact
Adrenaline (1:10 000)	3·33	Yellow	Yellow
Atropine	4·80	No effect*	Light purple
Lignocaine (4%)	1·72	Yellow discolouration at the edges*	Orange/yellow
Salbutamol (5 mg/ml)	3·28	No effect*	Light purple
Physiological saline	5·80	No effect*	No effect

*The detector remained responsive to carbon dioxide.

Vitamin B-12 and folate deficiency presenting as leukaemia

SIR,—I was surprised by the content of the Lesson of the Week by Dr I S Dokal and colleagues, who report on two patients with megaloblastic anaemia who were initially mistakenly diagnosed as having acute myeloblastic leukaemia. They state that the haematological findings were not typical of the correct diagnosis. With the exception of the raised white cell count in case 1 all of the features described are characteristic of patients with megaloblastic anaemia.

It is unusual not to be able to aspirate bone marrow from these patients, but if this is impossible a good trephine biopsy sample will give adequate cellular morphology. The typical appearances in megaloblastic anaemia may, as Dr Dokal and colleagues show, be frightening to those unfamiliar with them. It is standard practice in the Northern region for photographs of such trephine biopsy samples to be used in training sessions for young haematologists, and good photographs are available in current atlases of haematology.[2]

The bone marrow appearances in case 2 were more unusual, but, rarely, precursors of red cells are lost almost completely from the marrow and confusion with acute myeloid leukaemia can occur.[3]

I find it disturbing that chemotherapy was avoided in case 1 only because technical difficulties led to delays in getting treatment started. I think that there are lessons to be learnt from this paper but, unfortunately, I do not think that they are those that the authors intended.

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1 Dokal IS, Cox TM, Galton DAG. Vitamin B-12 and folate deficiency presenting as leukaemia. *Br Med J* 1990;**330**:1263-4. (12 May.)
2 Hoffbrand AV, Pettit JE. *Sandoz atlas of clinical haematology*. London: Gower Medical Publishing, 1988.
3 Weatherall DJ, Ledingham JGG, Warrell DA. *Oxford textbook of medicine*. Oxford: Oxford University Press, 1987.

AUTHORS' REPLY,—We agree with Dr Anne Lennard that in case 1 there were some features that are typical of megaloblastic anaemia. As she herself points out, however, this patient had a greatly increased white cell count ($24{\cdot}4\times10^9$/l), she had a normal mean corpuscular volume (89 fl), and aspiration of bone marrow proved to be impossible. On the basis of the bone marrow trephine biopsy a diagnosis of acute myeloid leukaemia was made by experienced haematological morphologists (including DAGG). It seems likely that the coexistent chest infection contributed to the atypical morphology, and we are not convinced that examination of a trephine roll would have given the correct diagnosis.

In case 2 acute myeloid leukaemia was considered as one of the possible diagnoses because of the presence of numerous promyelocytes showing heavy granulation. Megaloblastic anaemia was considered from the outset and was substantiated by the low serum folate concentration and response to treatment. In this case again the presence of infection may have been responsible for the unusual morphology.

In this Lesson of the Week we intended to emphasise the diagnostic difficulties posed by these two patients. We have seen several similar cases over the years but were not able to include them in our account because some essential information could not be found. We are pleased that Dr Lennard is confident that she can recognise these cases, but we have not found clear descriptions of their atypical clinical and laboratory features and fear that mistakes will continue to be made.

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Antihypertensive and adverse biochemical effects of bendrofluazide

SIR,—Perhaps Professor Peter Sever could explain the apparent contradiction between the views he expressed as a member of the British Hypertension Society working party: "First line treatment with a diuretic or β-blocker is equally acceptable: other agents may be necessary when these drugs are contraindicated or ineffective,"[1] and the view expressed in his letter that "the use of diuretics as