imposed, quite rightly, by the editor seems to have been the cause of that disagreement.

The authors may feel they have been unjustly reviewed. However, taking their title and the definitions of health education used not only by me but by the health education movement itself, I hope they have not been too unfairly treated. They have, perhaps, confused my criticism of the context in which the book is written, health education as it is, which is unfavourable, with my criticism of the content of the book, which is favourable. Authors have to write books for a subject as it is. Laudable though it is to write books for the subject as it should be, few people read them.

Finally, health education should be grateful to the *BMJ* not only for reviewing this book for teachers in the first place, but, by requiring reviewers to reveal their names, thus introducing an element of accountability into an ever less accountable world, for stimulating this debate on that neglected topic "health education." So few doctors are interested in this topic, yet education is inherent in all of medical practice—inherent but inefficient, as non-compliance studies reveal. May many more doctors write, debate, and disagree about health education, so that we can approach a consensus, and may many more practise it, to bring preventive medicine nearer to its potential.

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- ¹ Bernstein, B, *Class Codes and Control*, p 38. London, Routledge and Kegan Paul, 1975.
- ² Department of Education and Science, *Handbook of Health Education*. London, HMSO, 1969.
- ³ Department of Education and Science, *Health Education in Schools*. London, HMSO, 1977.
- ⁴ Office of Health Economics, *Prospects in Health*, paper No 37. London, OHE, 1971.
- ⁵ Schools Council, *Health Education in Secondary Schools*, Working Paper No 57, p 37. London, Evans/Methuen Educational, 1976.
- ⁶ Committee on Child Health Services, *Fit for the Future*, para 10.27. London, HMSO, 1976.

Teratogenic effects of waste anaesthetic gases

SIR,—In the Birmingham conurbation there are approximately 75 anaesthetists who have families with children of school age or below. The size of the families range from one to five children, the total number of children currently is 135, but the number of families in Birmingham varies according to the turnover and rotation training programmes of the junior anaesthetists. In five of these families children have been born (four of the children were born in Birmingham) with serious congenital abnormalities. A distinct pattern of abnormality has emerged. The defects are confined only to the musculoskeletal or nervous system—see table—and there have been no abnormalities of the other major physiological systems—for example, cardiovascular, respiratory, genitourinary, alimentary, etc. None of the children suffered severe birth trauma nor were they all born in Birmingham. Not all the defects were reported in a previous survey¹ since either the defect was not manifest or the children were born subsequent to that survey.

In only one case was the mother an anaesthetist. Although another mother had worked in the operating theatre, this had been discontinued long before the birth of the affected child. The remaining three mothers had no connection with hospital work. All other

children born to the respective parents are healthy. The inclusion of one child with ependymoma is because that tumour originates from deranged embryonic tissue.² Curiously, all the children were female (P less than 0.05) and this accords with an earlier report that there is an excess incidence of females among the children of anaesthetists although this latter finding was not confirmed in the United Kingdom.

These results of five affected children in 75 families must be the result of statistical clustering, yet the probability of such a cluster increases if the incidence rises above the natural background incidence. For example, the probability of finding by chance in a small group of children just one case of ependymoma, one of aqueduct stenosis, and one of kyphoscoliosis is less than one in a million, given the natural occurrence rate of these conditions (from 1 in 1000 to 1 in 10 000 live births each)—and this ignores the other congenital anomalies found. The emergence of a particular pattern of disorders and the absence of the more common major congenital abnormalities—for example, cleft palate or congenital heart disease—would seem to make any conclusion that these observations are due to a chance cluster of the normal congenital abnormality hazard highly unlikely.

Since the only feature that is common to all the affected children was that the father was an anaesthetist this implies that there is an occupational hazard. An excess incidence of congenital abnormalities to the children of anaesthetists has been previously reported³ but not confirmed in the United Kingdom.¹ Significantly, in that report the excess abnormalities were also confined to the musculoskeletal system, although the abnormalities were less severe than in this report and it was the mothers who were exposed. An excess incidence of tumours was also noted⁴ which included a cerebral tumour.

Abortion has been officially recognised as a hazard to staff exposed to waste anaesthetic gases,⁵ although some believe that this is due to stress.⁶ The latter is an unquantifiable hypothesis incapable of disproof but difficult to reconcile with the congenital abnormality problem if it is the father who has been exposed to the stress. The alternative hypothesis thus becomes more compelling—that abortion represents one end of a spectrum which ranges from immaturity (underweight),⁷ through teratogenicity to fetal death and so abortion that is the feature of a drug response affecting cell division. In this case the presumptive drug must be waste anaesthetic gases.

The observations that the anti-pollution measures recommended by the DHSS⁸ are not particularly effective⁹ in all clinical circumstances would seem to give urgency to finding a real solution to the problem if more damaged children are to be avoided. Two questions suggest themselves. Should we expect anaesthetists (and perhaps other staff) who are in their reproductive years to continue to work in unventilated or inadequately ventilated

areas—for instance, some operating theatres, x-ray departments, casualty departments, etc? Should the DHSS as a model but essentially monopolistic employer be asked to keep a register of the health of the children of the staff at risk and to accept the responsibility for any affected children?

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- ¹ Knill-Jones, R P, *et al*, *Lancet*, 1975, 2, 807.
- ² Jones, P G, and Campbell, P E, *Tumours of Infancy and Childhood*, p 251. London, Blackwell Scientific Publications, 1976.
- ³ Corbett, T H, *et al*, *Anesthesiology*, 1974, 41, 341.
- ⁴ Tomlin, P J, *Anaesthesia*, 1977, 32, 578.
- ⁵ *Pollution in Operating Departments by Waste Anaesthetic Gases*, DHSS, HC(76)38. London, HMSO, 1976.
- ⁶ Melka, S, and Burton, P, *Lancet*, 1976, 2, 695.
- ⁷ Rosenberg, P, and Kirves, A, *Acta Anaesthesiologica Scandinavica*, Suppl, 1973, 53, 37.
- ⁸ Howarth, P H, *Hospital Engineering*, October 1976, p 19.
- ⁹ Thompson, J M, *et al*, *British Journal of Anaesthesia*, 1977, 49, 1171.
- ¹⁰ Kurlund, L T, Kurtzke, J F, and Goldberg, I O, in *Epidemiology of Neurologic and Sense Organ Disorders*, p 196. Cambridge, Mass, Harvard University Press, 1973.

Inheritance of Marfan's syndrome

SIR,—I was alarmed to see Marfan's syndrome included in your leading article (3 December, p 1438) "Dominant inheritance of mental handicap," as the latter is not a feature of this syndrome. Any patient in whom mental retardation is associated with arachnodactyly and lens dislocation is suffering from homocystinuria. The distinction has important implications for counselling, as homocystinuria is inherited as an autosomal recessive.

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*.*As Dr Burn says, attention in Marfan's syndrome is usually focused on the physical anomalies. Indeed, Smith¹ in his atlas states categorically that "these individuals are of normal intelligence." Penrose² is less definite: "Mental defect is not considered to be an essential feature, but numerous instances of its concurrence with arachnodactyly are known—mental grades vary greatly from one patient to another." Summarising extensive experience with mentally handicapped children, Crome and Stern³ attribute "occasional mental retardation" to Marfan's syndrome. This difficulty may lead to repeated requests, with negative results, to the chemical laboratory for investigation for homocystinuria.

Congenital deformities in children of anaesthetists in Birmingham

Parents	Sex of child	Abnormality	Treatment required
Father anaesthetist Mother ex theatre staff	Female	Dislocated hip, thoracic scoliosis	Hip spica Operation Hamilton Road
Father anaesthetist Mother anaesthetist	Female	Bilateral dislocated hips, ? hydrocephalus*	Surgical correction drainage operation
Father anaesthetist Mother no connection	Female	Hydrocephalus due to aqueduct stenosis**	Drainage operation
Father anaesthetist Mother no connection	Female	Ependymoma	Craniotomy (died)
Father anaesthetist Mother no connection	Female	Bilateral talipes, diffuse cerebral abnormalities***	Too young Under investigation

* Interpretation complicated owing to an episode of neonatal meningitis.

** WHO expected incidence less than 1 in 1000 live births.¹⁰

***These include marked motor and EEG abnormalities.

This syndrome has been recognised only since 1963 and before this confusion with Marfan's syndrome must have occurred, but it is now unlikely in countries with well-developed laboratory services. It is still probable that Marfan's syndrome is genetically heterogeneous.¹—Ed, *BMJ*.

- ¹ Smith, D W, *Recognizable Patterns of Human Malformation*, p 258. Philadelphia, Saunders, 1970.
- ² Penrose, L S, *The Biology of Mental Defect*, 4th edn, p 145. London, Sidgwick and Jackson, 1972.
- ³ Crome, L, and Stern, J, *Pathology of Mental Retardation*, p 281. Edinburgh, Churchill Livingstone, 1972.
- ⁴ McKusick, V A, in *Birth Defects: Original Article Series*, vol 11, No 6, ed D Bergsma, pp 1-13. Alan R Liss, New York, 1975.

Paracetamol overdose

SIR,—The report by Dr R D Hughes and others on a controlled trial of cysteamine and dimercaprol after paracetamol overdose (26 November, p 1395) ends with a plea for a further controlled clinical study with methionine.

In view of the critical need for glutathione to protect the body from the hepatotoxic effects of intermediary metabolites of paracetamol¹ and the well-documented role of cysteine and acetyl cysteine conjugation in the detoxication mechanism^{2,3} it would perhaps be more useful to base further control studies on the experience gained so far with *N*-acetyl cysteine,^{4,5} especially as it would appear that cysteamine and *N*-acetyl cysteine might not be acting by the same mechanism.^{6,7}

Since the time factor after overdose is so important in determining the outcome of treatment with SH-donor substances⁸ the danger of inducing a biochemical lesion could be minimised or avoided altogether if pharmaceutical preparations of paracetamol incorporated *N*-acetyl cysteine.

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- ¹ Brodie, B, *Intra-Science Chemistry Reports*, 1974, 8, Nos 1-3, p 1.
- ² Gillette, J R, in *Drug Metabolism from Microbe to Man*, ed D V Parke and R L Smith, p 147. London, Taylor and Francis, 1977.
- ³ Knox, J H, and Jurand, J, *Journal of Chromatography*, 1977, 142, 651.
- ⁴ Piperno, E, and Berssenbruegge, D A, *Lancet*, 1976, 2, 738.
- ⁵ Prescott, L F, et al, *Lancet*, 1977, 2, 432.
- ⁶ *Lancet*, 1975, 2, 1189.
- ⁷ *British Medical Journal*, 1977, 2, 481.

SIR,—We read with interest the report by Dr R D Hughes and others (26 November, p 1395) of a controlled trial of cysteamine and dimercaprol following paracetamol overdose but noted that it suggests that cysteamine is inconvenient to use.

In their report the authors mention that the cysteamine infusions used in the trial were freshly prepared and then filtered through a Millipore filter. One assumes that this method of preparation was used because of the initial problems with cysteamine stability.^{1,2}

It has been demonstrated that cysteamine hydrochloride is stable to autoclaving and storage.^{3,4} In order to make the injection easier to use we have, since 1976, produced the solution as an initial dose of 2 g in 120 ml isotonic solution in a 125-ml transfusion bottle, with subsequent doses in 10-ml ampoules. This provides a convenient dosage form for ease of use which can easily be produced by any hospital sterile manufacturing unit.

We cannot therefore understand the need

for freshly prepared solutions when such a proved and convenient method of use is available.

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- ¹ Prescott, L F, et al, *Lancet*, 1974, 1, 588.
- ² van Heijst, A N P, Douze, J M C, and Pikaar, S A, *Nederlandsch Tijdschrift voor Geneeskunde*, 1976, 120, 1151.
- ³ Brouwers, J R B, and Vermeij, P, *Pharmaceutisch Weekblad*, 1976, 111, 204.
- ⁴ Prior, F G R, *Lancet*, 1977, 1, 315.

Confidentiality and life insurance

SIR,—I should be grateful if you would allow me to comment on the letter (10 December, p 1544) from Dr T M Pickard regarding confidentiality and life assurance. His conclusions, reflected in the last paragraph of his letter, demonstrate the misunderstanding of life assurance and the work of the underwriter which is all too prevalent—not only in the medical profession. Perhaps I can clarify the situation.

The principal objectives of a life assurance office are to give cover and to pay claims. The protection of life assurance for family commitments, etc, is much sought after, as is evidenced by the considerable total of premiums paid to life offices during each year. The cost of this protection is calculated from the mortality statistics of assured lives, and if offices allowed proposers to have cover at the normal rates of premium when their mortality was likely to be substantially greater than the standard, then the office would make a loss. It is the underwriter's job to assess those who propose for life assurance and assess the extra mortality of any who do not fit into the standard of the life office. Each office has its own standard, so the number not accepted at the ordinary rate of premium will vary, but is likely to be between 5% and 10% of the total.

Dr Pickard has drawn attention to the fact that many people who fill in forms do not read them, nor do they understand their implications. I do not think anyone would disagree with him here. Unfortunately, this fault is not confined to the completion of proposals for life assurance, nor is it confined to those outside the medical profession. However, Dr Pickard's objection is to the position of the underwriter as it affects his relationship with his patient and casts the life assurance office as the villain.

An underwriter is conscious of the very special doctor-patient relationship and does all he can to preserve it. One of the main difficulties arises when a doctor makes a clinical judgment not to tell his patient the full implication of his troubles, perhaps placing the underwriter in the position of having to decline the proposal for no apparent reason. The natural reaction is for the patient to ask his doctor what is wrong with him. The only help an underwriter can give is to pay for an unnecessary independent examination in the hope that this will offer some protection to the doctor. Unfortunately Dr Pickard has illustrated his comments by referring to mental disturbances. This is probably the most difficult disorder for any doctor to combat and it is no less difficult for the underwriter. It is made more difficult for the underwriter because of the loose terminology used. Words like "depressed" and "anxious" have different meanings to the layman and the clinician.

If an underwriter is faced with a proposal in which he is told "I saw my doctor for depression" what is he to believe? It could mean anything between "I felt run down" or "I didn't feel up to it" to the proposer being a manic-depressive. The treatment could be anything between 1% tranquillisers plus 99% reassurance and regular treatment with lithium carbonate. The underwriter cannot possibly make a balanced judgment without help from the medical adviser and any report requested is for the benefit of the patient and certainly not to "be used in evidence against him".

It is understandable that doctors may feel their patients are unaware of the implications of giving authority to the doctor to furnish reports. It is a not uncommon practice in such circumstances for the doctor to request separate written authority from the patient. This is of course expensive and time-wasting, but if the doctor feels the circumstances warrant his patient being more forcibly reminded of the disclosure he is authorising, then it is a very worthwhile precaution. A photocopy of the permission already granted, as suggested by Dr A J Caro (7 January, p 50), might, however, serve this purpose.

The comments I have made do not apply solely to mental disturbances, but to the whole scope of medicine. I hope they have at least put the underwriter's point of view that nothing he does is against the patient but entirely for his benefit. If it were not this way round we, of course, would soon be out of business.

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Dietary cholesterol and atherosclerosis

SIR,—It is good that Professor John Yudkin (3 December, p 1477) should remind us of the pioneering work of Anitschkow, who established the principle of what he called "dietary atherosclerosis." Anitschkow's meticulous work describing how he tracked down the culprit cholesterol in egg yolk and later in brain is still fascinating to read. Time marches on and his findings have been confirmed by many workers in many different species—in fact in all in which the plasma cholesterol concentration is raised,¹ including our nearest relatives, the non-human primates. The stronger the stimulus, the more cellular is the response, but with a lesser stimulus continued for a longer time progressively, even in rabbits, the changes more closely resemble those occurring in man. Wissler and his colleagues in Chicago,² by feeding actual Western diets to rhesus monkeys, which are closely similar to man in their anatomical, physiological, and biochemical features and not spontaneously prone to atherosclerosis, have produced severe arterial changes similar to those seen in humans.

Anitschkow noted, as must have most physicians in their patients, that hypercholesterolaemia (by conventional standards) is not always present. This is not surprising because other factors, including in particular the permeability of the arterial wall and even mild elevation of blood pressure, are also important. Many pathologists, including in particular Oxborn,³ have shown that pathological changes in the coronary arteries are frequently present in children and pre-atheromatous changes even in infancy. Ex-