CORRESPONDENCE

Because we receive many more letters than we have room to publish we may shorten those that we do publish to allow readers as wide a selection as possible. In particular, when we receive several letters on the same topic we reserve the right to abridge individual letters. Our usual policy is to reserve our correspondence columns for letters commenting on issues discussed recently (within six weeks) in the BMJ.

Letters critical of a paper may be sent to the authors of the paper so that their reply may appear in the same issue. We may also forward letters that we decide not to publish to the authors of the paper on which they comment.

Letters should not exceed 400 words and should be typed double spaced and signed by all authors, who should include their main degree.

Halothane and the liver

SIR,—The recent statement by the Committee on Safety of Medicines on halothane and the liver and its recommendation that the minimum interval between halothane anaesthetics be extended from 28 days to three months[1] concern us because we fear that some may see them as a signal that the use of halothane in routine practice should be substantially reduced or abandoned. On present evidence such a change in anaesthetic practice would not only be without justification but might be harmful to patients.

Epidemiological studies over 25 years have failed to yield firm evidence on the factors predisposing to halothane hepatitis. All have concluded that the condition is rare: the incidence is estimated to be 1/10 000 to 1/35 000 halothane anaesthetics. The possible role of repeated exposure to the drug has been recognised at least since the report of Walton *et al* in 1976.[2] Nevertheless, cases have occurred after single exposures and after exposure intervals longer than 28 days or even three months. Special attention has been focused on unexplained fever and severe nausea and vomiting after halothane as possible indicators of a likely future adverse reaction to the drug. The diagnosis is made only by excluding other likely causes of hepatitis—a difficult task in the complex conditions of the postoperative period, especially after multiple operations, a circumstance which may make for over-reporting of halothane hepatitis to the CSM.

We recognise that the CSM has a duty to monitor reports of adverse reactions to halothane and to offer guidance on the basis of its findings. The CSM cannot indicate the totality of the risk associated with particular drugs and techniques.

For almost 30 years halothane has enjoyed a reputation as a reliable and usually safe anaesthetic. It is more potent than enflurane or isoflurane and less irritant to the respiratory tract than isoflurane. These properties may confer an important advantage when there is a need to deepen the level of anaesthesia quickly, given that the maximum output of most vaporisers is 5% (vol/vol) nominal. Inability to control the level of anaesthesia effectively is always an immediate threat to a patient's life if regurgitation or airway obstruction supervenes. Such problems are not, of course, normally reported to the CSM.

On the present evidence we believe that it is desirable for there to be a choice of volatile anaesthetics: halothane, enflurane, and isoflurane. That choice should be a clinical decision, exercised responsibly with due regard to the patient's anaesthetic history and with careful recording of untoward effects, whatever the drugs used.

Finally, it has been suggested that halothane liver injury will be increasingly likely to attract actions for litigation (28 June, p 1691; 2 August, p 335), although, to our knowledge, no case has been pursued successfully as a civil action in the United Kingdom. An anaesthetist who departed from the CSM recommendations on the interval between halothane anaesthetics, having carefully considered the options and the patient's condition and recorded appropriate reasons for the choice of halothane, would not, in our opinion, be acting negligently. A more rigorous view than that would be expensive and nonsense. On the basis of its statement we believe that the CSM would concur with that view.

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Is there an ideal body weight?

SIR,—Professor R J Jarrett (23 August, p 493) rediscovered that the risk of premature death is not a simple direct function of relative body weight. Apparently that fact, well established from various prospective studies, is not yet common knowledge in the medical profession, let alone the general public. This deficiency prevails in all "developed" countries; in the least developed countries the popular view is the reverse: obesity is prized as a sign of "health." A brief review of the facts is overdue.

Professor Jarrett attributes to me information about the role of the late Dr Louis Dublin and the Metropolitan Life Insurance Company in creating body weight standards.[1] Labelled "ideal weight" in 1943, the Metropolitan tables, revised in 1959 and more modestly termed "desirable weight," covered three "frame" types—small, medium,

large or light, medium, heavy.[2] There were no data on how to classify people in those categories. Dr Dublin realised that individuals differ in skeletal type. Since no measurements were available he simply arrayed the body weights at given height and divided the array into thirds. To this day there is no agreement about frame type, how to classify people, or its actuarial significance. In 1959 the Society of Actuaries adopted Dublin's tables, including the "frame types."[3] No actuarial experience concerning frame types has been reported; with no measurements no analysis is possible.[4]

The idea that the risk of premature death is directly related to relative body weight came from the insurance industry and the evidence mainly from the mortality experience of policy holders who had to pay extra premiums because they were overweight. More than 30 years ago I expressed doubts about that evidence.[5 6] Commonly, insurance companies have demanded extra premiums from applicants more than 20 or 25% heavier than average applicants of the same age, sex, and height. The mortality experience of such overweight policy holders justified the extra premiums.[7] Only some 2% of policy holders are in the extra premium class but all surveys indicate that around 7% of the population are equally overweight.[7 8] The implication is that insurance applicants willing to pay the extra premiums demanded have special reasons (not disclosed to the company) for wanting life insurance. They select against the company.

For many years proponents of the view that relative body weight is a major risk factor paid no attention to opposing evidence from studies outside the insurance industry. Yater *et al* showed that 866 soldiers who died from coronary heart disease did not differ in relative weight from their fellows killed in military accidents nor were they any heavier than the rest of the soldiers when they entered the army.[9] Patients entering hospital with acute myocardial infarction included no excess of obese persons, but the fatter patients had the best prognosis.[10] Among 1356 men of the DuPont Company who had myocardial infarctions 30% were 20% or more above the "desirable weight" standards; among healthy men of the same age and occupation the percentage was 29·7%.[11 12]

Fortunately there is now no lack of data from prospective studies free from the selection bias of the insurance companies and they are generally characterised by better entrance examination data. The "coronary problem" was the focus of most of those studies. Ten years ago we reviewed the studies available and showed little relation between relative weight and the subsequent incidence of myocardial infarction or death from coronary heart disease. Only the Framingham study reported a significant relation between relative weight and incidence of coronary heart disease but relative weight was given only a minor role.[13] Later, the Framingham group reported "minimum mortality around the average weight with increasing mortality for persons weighing more or less than the average."[14]

With longer periods of follow up time in prospective studies it was possible to examine mortality and with that end point three facts emerge. Firstly, no study has reported a direct linear relation between relative weight and mortality from all causes or from coronary heart disease. Secondly, using the multiple logistic model with attention to age, blood pressure, and serum cholesterol value as well as relative body weight, several investigators found a negative linear relation between relative weight and mortality. Thirdly, more detailed analyses commonly found a curvilinear relation with excess mortality at both ends of the relative weight array.[15] The solution to the linear multiple regression equation showed mortality decreasing with increasing relative body weight, but the relationship was curvilinear and a much better fit was found with the quadratic model using the square of the body mass index. The 10- and 15-year experience of the Seven Countries study was similar.[1 16 17] The picture seems to hold for much longer follow up, as in the 25 year experience of the Finnish cohorts in the Seven Countries study (Pekkanen J, personal communication, 1986).

With the quadratic model the solution of the multiple logistic equation may be used to find the relative body weight with the lowest probability of premature death. With this approach applied to 2571 American men aged 40 to 59 the least risk of death within 15 years seems to be when body mass index is around 26 (kg/m^2). That figure is slightly above the average for middle aged men in the United States.

Data on women are scanty but there is no indication that, except for extreme obesity, they should be concerned about relative body weight as a major risk factor for premature death. Losing weight is very big business in the United States and some other Western countries. While the advertisements emphasise health, the real appeal is cosmetic. Among side effects of the current mania is an increase in anorexia nervosa and an explosion of bulimia. In decadent Rome vomiting was self induced to make room for more food. Self induced vomiting to lose weight is at least as deplorable and perhaps more dangerous to health.

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Glue ear and speech development

SIR,—Mr A G D Maran and Ms Janet Wilson (20 September, p 713) quoted a 99% incidence of glue ears in a screening clinic for educational underachievement. I was working in that clinic at the time and would like to correct this statement. Only a few had glue ears when tested in mid-childhood. Many had low and fluctuant middle ear pressures without actual fluid but with histories of auditory inattention. Some had scarred or thin eardrums indicating previous chronic low middle ear pressures. Some had histories or symptoms of previous otitis. Thus almost all had clear evidence of past or present ear disease. Certainly there was nothing to refute the idea that the main cause of dyslexia is fluctuant conductive deafness in infancy.

The authors ask what level of deafness causes language impairment. But why should there be any such quantitative relation or cut off point? Language impairment is not closely related to degree of deafness in congenital sensorineural impairment. My observations suggest that low and fluctuant middle ear pressures, age of onset, stability, asymmetry and laterality of deafness, and associated symptoms such as tinnitus, audiosensitivity, imbalance, and clumsiness are far more important than pure tone hearing loss.

It is a great pity that professionals do not listen to parents,[1] as they can resolve the conflicting opinions about a link between otitis media and speech development. If surgeons are uncertain about whether to take the authors' advice not to seek out children under 2 years old for myringotomy all they need to do is ask parents for observations when grommets have been inserted in such children. They would be left in no doubt that the effect is often dramatic. Parents notice an immediate improvement in speech or behaviour,[2] often without having noticed any impairment of or change in hearing. The decision to treat young children with glue ear will be determined not by further research but by pressure from consumers who see for themselves that grommets work. Inevitably this demand will be led by the middle classes, who are most likely to have realised that early ear disease might account for their child's problems[1] and to have the confidence to insist on treatment against the advice of professionals.

The main reason for proposing a link between ear disease and dyslexia is the simple fact that auditory processing and verbal intelligence quotients, and not visuomotor skills or non-verbal IQs, are impaired in children with dyslexia,[3] the opposite pattern to that in children with brain damage. If not deafness, what environmental factor is selectively depressing verbal IQs in children with dyslexia of above average ability from good homes?

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SIR,—I am sure I am not alone in expressing my dismay at the leading article by Mr A G D Maran and Ms Janet Wilson.

Glue ear is not a middle class disease, nor is speech and language deficit. Glue ear is common and probably more common in lower social class children, but the parents are less likely to recognise a problem and they accept lower standards of speech and language development. Also they are less likely to bring their children to clinic appointments for screening.

Why should a sensorineural hearing loss and not a conductive loss affect the child's speech and language development? The child with true glue ear does not have a hearing loss of 15 dB or 25 dB; it is usually nearer 30-40 dB. I suggest the authors should try learning a foreign language with plugs or fingers in their ears and they might then understand what it is like for a small child learning his or her own language.

Research is difficult. How is anyone going to measure environmental stimulation and language experience in the home? Audiological assessment in young children is possible, but shortage of trained staff to measure even such aspects as speech and language development is bad enough and I am sure this is the reason for such a scarcity of evidence at present. The evidence, however, is there. The parents of the children will recount the dramatic improvement in their child's efforts to talk after surgery.

To say that research has not been carried out is no reason to withhold treatment for a problem