Trial of acetylsalicylic acid in the secondary prevention of mortality from myocardial infarction

SIR,—Some years ago a report of a randomised controlled trial of aspirin and the secondary prevention of myocardial infarction was published in your journal.[1] The results were statistically inconclusive, though there was a reduction in total mortality of 12% at six months and 25% at 12 months after admission to the trial. In the report the fate of 113 subjects who had been omitted from the trial for various reasons was not given and the analysis presented was based on 1126 men who had continued on treatment up to the end of the trial.

We have now ascertained the fate of 108 of these men. The other five could not be traced. The accompanying table gives all-cause mortality for all the men ever admitted to the trial, excluding the five not traced. The data

All-cause mortality in the first MRC trial of aspirin in the secondary prevention of myocardial infarction, based on all men ever entered into the trial

Age	Aspirin Total No	Aspirin No (%) died	Placebo Total No	Placebo No (%) died
<55	264	16 (6)	286	25 (9)
55-65	263	23 (9)	270	30 (11)
65+	85	10 (12)	66	12 (18)
All ages	612	49 (8·0)	622	67 (10·8)

now suggest a reduction by aspirin in all-cause mortality of about 25·7% but this is not statistically significant at conventional levels ($p > 0.05$).

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[1] Elwood PC, Cochrane AL, Burr ML, *et al. Br Med J* 1974;i:436-40.

The split BMJ

SIR,—We wish to express our strong disapproval of the new format of the journal.

The essential point to us is that the new format is potentially divisive of the profession, making for further separation of general practice from specialist and hospital practice. Such separation if not halted will do nothing but harm to the profession.

We feel that the main aspect of this separation will ensue from the division of the advertisements; there are a number of doctors who like us have a foot in both camps, being interested in both GP and in hospital practice. This new format will be felt particularly by younger doctors who have not finally made their minds up about their exact careers, and who have a clear need for access to both hospital and GP-orientated advertisements. A special problem will fall on those who do not take up a straight GP training programme, and will for some time fall between hospital and general practice. Many doctors may well prefer to make up their own training programme. We have both, over most of our careers, made use of jobs in both general practice and in the hospital service, and feel that loss of access to one or other set of advertisements will be a problem to us.

Fortunately for us, we are both members of the Association and so can solve our problems by having you send us one of each version of the journal; similarly, partnerships may well be able to act in this way. However, as we suggest, the younger, loner type of doctor will be the one to suffer. We feel that some arrangement should be made to cater for this problem, unless the old format can be revived. We feel that the Association has an extra obligation as they in a sense have a monopoly of most NHS advertisements.

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**Most of the comments sent to the *BMJ* have welcomed our new split run for the extra coverage it provides for research and general practice features. In planning the change the *BMJ* recognised its responsibilities to readers as a general medical journal and decided to use the miniprint technique as a way of ensuring that hospital and general practice readers would have access to each other's special interest sections, while preserving the remaining regular features intact and in normal-size type. As an important part of the exercise is to keep the *BMJ* financially healthy—every year £2·5m worth of *BMJs* are sent to members at no cost to the BMA—it is necessary to publish advertisements in a more economical way, so we aim at putting all those advertisements likely to be of immediate interest to family doctors in the Practice Observed edition. By saving at least 26 million pages this new format should enable us to balance the books for the next few years. We appreciate that a few doctors might have difficulty in making a choice, and we sympathise with the special problem of trainees, but thank those who have arranged with a colleague, trainer, or postgraduate centre library to see the other edition of the *BMJ*.—ED, *BMJ*.

Impaired glucose tolerance and diabetes —WHO criteria

SIR,—As reported in your leading article under this title (6 December, p 1512), the WHO Expert Committee on Diabetes Mellitus has proposed[1] raising the degrees of hyperglycaemia necessary for a diagnosis of diabetes and creating a new category of "impaired glucose tolerance," which will not be regarded as diabetic. Patients in this category would thus be "spared the social, economic, and psychological stigmata that attach to the label of diabetes." It is pertinent to consider whether the proposal is justified or desirable.

A recent, very useful, and important prospective study[2] provided valid statistical confirmation of the pathogenetic importance of a raised blood glucose for the vascular complications of diabetes mellitus, establishing that microangiopathy mostly becomes overt at a two-hour capillary blood glucose of 11·1 mmol/l (200 mg/dl) and over, but that macrovascular disease (for example, coronary heart disease) significantly increases in incidence also beyond a mere 5·3 mmol/l (96 mg/dl) at two hours after a 50 g oral glucose load. This proof now warrants even drastic preventive measures.

All the above, however, confirms that the formerly adopted[3-5] two-hour blood glucose threshold figures for the diagnosis of diabetes were broadly[6] correct (and not unduly low); and that even two-hour capillary blood glucose figures now included under the term "impaired glucose tolerance"—namely, 11·05 mmol/l (96-199 mg/dl)— could be definitely harmful and productive of *organic* (notably vascular) complications, and hence deserve the name "disease" rather than "functional disorder."

Hence I cannot, academically or clinically, agree on not calling this stage by the disease name of diabetes mellitus. This phase of diabetes is simply an early one, but needs to be properly controlled just as the graver two-hour capillary blood glucose concentrations of 11 mmol/l and over, often associated with microangiopathy. This would be more consistent with our time-honoured practice in medical nomenclature with regard to other diseases (such as pulmonary tuberculosis and cancer), where early diagnosis and adequate treatment could make all the difference to the patient's life. No disease names were changed for these early stages with an incomplete clinical picture.

Early recognition and treatment of diabetes could thus prevent not only microangiopathy, but also atherosclerotic disease (for example, coronary heart disease), the chief cause of complications and mortality, especially in the maturity-onset diabetic. Hence in my opinion, in order not to underestimate unfairly the potential serious harm from even "impaired glucose tolerance," this phase should continue to be considered the early stage of diabetic disease, in the interests both of the patient and of consistency. A sound diagnosis and wise management should minimise any psychic or socioeconomic trauma—by far the lesser evil.

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[1] World Health Organisation Expert Committee on Diabetes. *Second report. WHO Technical Report Series* No 646. Geneva: WHO, 1980.
[2] Fuller JH, *et al. Lancet* 1980;i:1373-6.
[3] Butterfield WJH. *Proc R Soc Med* 1964;57:196-200.
[4] World Health Organisation. *Technical Report Series* No 310. Geneva: WHO, 1965.
[5] Zammit Maempel JV. *Lancet* 1965;ii:1197-1200.
[6] Zammit Maempel JV. *Lancet* 1965;i:205-6.

Alcoholism: an inherited disease?

SIR,—Your leading article on alcoholism (15 November, p 1301) drew attention to the high rate of alcoholism in adoptees who had biological parents one or other of whom had had an alcohol problem. As alcoholism in those adoptees is commoner than would be expected from the behaviour of the adoptive families, it is concluded that there is an inherited predisposition to develop alcoholism.

What is ignored is the considerable evidence that alterations in the chemical environment of the fetus in utero can subtly alter the finer details of brain development, and so influence eventual adult behaviour. It is therefore possible that if a mother drank during pregnancy the brain of her child might be altered in such a way as to give rise to a predisposition to alcoholism in later life. What we need to know is whether there is a predisposition to alcoholism among adoptees who had alcoholic fathers but abstemious mothers, and if such a predisposition is as great as among adoptees who had alcoholic mothers but abstemious fathers. Meanwhile, we should not conclude that there is an inherited predisposition to alcoholism.

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