a large true association. In contrast, a weak confounding effect is hard to detect and can plague weak associations—that is, associations with relative risks smaller than 2·0. It can either produce spurious associations or mask true associations.

Therefore, the warning against confounding bias raised by Smith and Phillips applies really to the study of weak associations. It is certainly true that weak associations should be interpreted with caution because there is no certainty that all confounding effects have been neutralised. Nevertheless, as long as the menace of confounding bias remains a purely theoretical exercise, there is no ground to give more credit to sceptical debunking of a suspected relative risk rather than to a postulated biological mechanism supporting its plausibility.

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1 Smith GD, Phillips AN. Confounding in epidemiological studies: why "independent" effects may not be all they seem. *BMJ* 1992;**305**:757-9. (26 September.)

Distinguishing inhalers to aid blind people

EDITOR,—Martyn Partridge points out the confusion engendered by the colour of a new asthma inhaler.[1] Consideration should also be given to those who have partial or no sight and are therefore unable to distinguish any colour at all on their inhalers.

Drug manufacturers at present pay little regard to this problem. The Ventolin inhaler has a V embossed on its side to help distinguish it from Becotide. Pulmicort and Bricanyl Turbohalers can barely be distinguished by small concentric rings embossed at their bases (and how blind asthmatic patients may realise when the Turbohaler is empty is not apparent). Other inhaler "pairs," such as Ventidisks and Becodisks, Aerolin and Aerobec, Ventolin and Becotide Rotacaps, and Bricanyl and Pulmicort metered dose inhalers, seem to have no obvious distinguishing features for blind people at all. In addition, not all practitioners agree that inhalation devices that combine both a bronchodilator and a steroid are useful in managing asthma.

Practical solutions to this problem may be devised, such as wrapping an elastic band around one of the devices to distinguish it from the other, or keeping the inhalers in containers of different shapes. It would be more appropriate, however, if the Department of Health and drug manufacturers were to agree on a common standard solution, such as simple embossed symbols or Braille lettering.

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1 Partridge M. Coloured inhalers. *BMJ* 1992;**305**:890. (10 October.)

Changes in drug treatment after discharge from hospital

EDITOR,—Rachel Ann Cochrane and colleagues examine why 45 out of 50 patients had their drugs changed after discharge from hospital.[1] They do not, however, discuss the problems of general practitioners having a prescribing policy. For example, in my practice we use the loop diuretic frusemide with monitoring of the potassium concentrations. Patients are discharged from our local hospital taking frusemide, co-amilofruse, or bumetamide, depending on the consultant they have been under. As soon as they come out of hospital they go back on frusemide.

The authors say that patients are discharged with five days' supply of drugs. They do not report having studied how many days it takes for a general practitioner to get the full discharge report. This is necessary for the general practitioner to plan how to merge the patient's existing drug treatment with the new treatment from the hospital; a brief list on a form is not enough. The best way to prevent interruption to the patient's treatment is to make sure that the patient is discharged with enough days' treatment to last until the general practitioner has received the necessary information.

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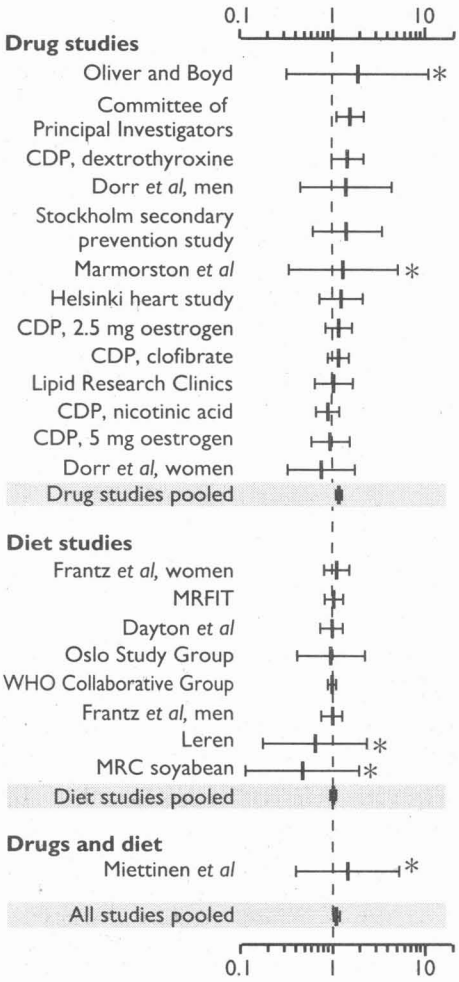
1 Cochrane RA, Mandal AR, Ledger-Scott M, Walker R. Changes in drug treatment after discharge from hospital in geriatric patients. *BMJ* 1992;**305**:694-6. (19 September.)

Cholesterol lowering treatment and mortality

EDITOR,—U Ravnskov's paper shows again that reducing coronary mortality with cholesterol lowering treatment does not result in a reduction in total mortality despite the considerable proportionate mortality of coronary heart disease and the high statistical power of a meta-analysis of 22 trials.[1] Because death from coronary heart disease accounts for a large proportion of total mortality (36% on the average in the primary prevention studies, 77% in the secondary prevention studies) the failure to reduce total mortality may suggest a lack of any net benefit of cholesterol lowering treatment.

Total and coronary mortality has been published for 22 of the 27 trials listed in table I of Ravnskov's paper. This allows the calculation of the "non-infarct mortality" (mortality from causes other than coronary heart disease—that is, "all deaths" minus "fatal coronary heart disease"). The figure displays the odds ratios and 95% confidence intervals of these 22 trials. The mean weighted odds ratio for all trials shows a significant increase in the non-infarct mortality (odds ratio=1·09 (95% confidence interval 1·02 to 1·17); p=0·007); this increase is even more significant in a separate analysis of only the trials that used drugs (odds ratio=1·27 (1·20 to 1·35); p=0·00009). The overall increase in non-infarct mortality seems to be the result of drug treatment alone as it is not observable in the studies that used diet (figure).

Table I shows a striking consistency in this increase in non-infarct mortality in all subgroups of the drug trials. Trials that used modern drugs as well as those that used obsolete drugs show this highly significant adverse increase in non-infarct mortality. The increase in non-infarct mortality



Odds ratios and 95% confidence intervals (logit method) for mortality other than that from coronary heart disease (non-infarct mortality) in all trials, grouped into drug and diet trials, and mean weighted odds ratios (Mantel-Haenszel method)
**Confidence intervals and p value approximate (χ^2 test for number of events < 5)*
CDP=Coronary drug project. MRFIT=Multiple risk factor intervention trial

TABLE I—*Mortality from causes other than coronary heart disease (non-infarct mortality). Mean weighted odds ratios and confidence intervals* in all trials and subgroups of trials*

	No	Odds ratios (95% confidence interval)	p Value
All trials	22	1·09 (1·02 to 1·17)	0·007
Unifactorial	18	1·19 (1·13 to 1·24)	<0·001
Multifactorial	4	1·02 (0·97 to 1·08)	>0·5
Primary prevention	12	1·07 (1·00 to 1·14)	0·08
Unifactorial only	8	1·18 (1·03 to 1·35)	0·017
Drugs only	5	1·42 (1·13 to 1·78)	0·003
Secondary prevention	10	1·20 (1·04 to 1·38)	0·012
Drugs only	8	1·22 (1·06 to 1·41)	0·006
Drugs	13	1·27 (1·20 to 1·35)	<0·001
Modern drugs only†	8	1·26 (1·09 to 1·45)	0·002
Excluding nicotinic acid	6	1·35 (1·14 to 1·61)	<0·001
Obsolete drugs only†	5	1·31 (1·06 to 1·63)	0·014
Primary prevention only	5	1·42 (1·13 to 1·78)	0·003
Secondary prevention only	8	1·22 (1·06 to 1·41)	0·006
Diet	8	1·03 (0·96 to 1·10)	0·5
Duration ≤ 5 years	15	1·17 (1·02 to 1·33)	0·022
Drugs only	9	1·30 (1·08 to 1·57)	0·006
Duration > 5 years	7	1·07 (0·99 to 1·16)	0·07
Drugs only	4	1·25 (1·07 to 1·47)	0·005

*Mantel-Haenszel method. Test for heterogeneity: p ≥ 0·08 for all analyses.
†Modern drugs: clofibrate, anion exchange resins, nicotinic acid, gemfibrozil. Obsolete drugs: oestrogen, dextrothyroxine.

TABLE II—*Mean weighted attributable risks* for mortality from coronary heart disease, total mortality, and non-infarct mortality in drug trials*

	Coronary mortlity		Non-fatal coronary disease		Non-infarct mortality		Total mortality	
	Attributable risk per 1000 annually (95% confidence interval)	p Value	Attributable risk per 1000 anually (95% confidence interval)	p Value	Attributable risk per 1000 annually (95% confidence interval)	p Value	Attributable risk per 1000 annually (95% confidence interval)	p Value
All	−0·72 (−2·09 to 0·77)	0·33	−1·56 (−2·57 to −0·46)	0·006	1·67 (1·24 to 2·14)	<0·001	1·30 (−0·45 to 3·16)	0·15
Primary prevention†	−0·44 (−0·86 to 0·11)	0·11	−1·91 (−2·36 to −1·44)	<0·001	0·96 (0·29 to 1·81)	0·003	0·53 (−0·28 to 1·49)	0·22
Secondary prevention‡	−0·68 (−3·31 to 2·18)	0·63	−0·60 (−2·51 to 1·54)	>0·5	2·11 (0·55 to 3·91)	0·006	1·74 (−1·52 to 5·27)	0·30

*Risk difference, calculated as (odds ratio − 1) × (average control group incidence).
Average coronary mortality and non-fatal coronary disease in control groups: †2·24 and 7·91 per 1000 annually, ‡31·9 and 18·3 per 1000 annually.

occurs in both primary and secondary prevention trials that used drugs.

Table II shows that the (significant) increase in non-infarct mortality is considerably greater than the (non-significant) reduction in coronary mortality, be it in primary or secondary prevention trials. The excess number of non-infarct deaths is even greater than the number of non-fatal infarctions prevented, the balance being especially unfavourable in secondary prevention. Prevention of about two coronary events is at the cost of 1000 treatment years. Thus the treatment of patients with hypercholesterolaemia over five years will benefit only one in 100. Ninety nine patients, without the chance of benefiting from treatment, are exposed long term to potentially adverse effects of drugs.

Various hypotheses for possible adverse effects of cholesterol lowering treatment have been advanced, but none has found general acceptance. The fact that the excess of non-infarct deaths equals the number of non-fatal infarctions prevented and far exceeds the number of fatal infarctions, however, may be more important than the lack of an accepted explanation for this strikingly consistent phenomenon. Patients are more concerned with not dying than with whether medicine can explain the mechanism of their dying.

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1 Ravnskov U. Cholesterol lowering trials in coronary heart disease: frequency of citation and outcome. *BMJ* 1992;**305**: 15-9. (4 July.)

Health checks for people over 75

EDITOR,—In his editorial on health checks for people over 75 Andrew Harris draws attention to the widespread misconception that the main objective of screening in elderly people is to identify previously unrecognised disease (although good clinical care remains the bedrock of this work). He draws attention to the need for programmes to focus more on functional disability.[1]

Harris makes one serious omission. Though it is true that the outcomes in controlled trials have generally been inconsistent owing (probably) to the heterogeneous nature of these studies, they have been consistent in one respect. All five trials looking at this indicator have shown that screened populations of elderly people spend less time in institutional care than control groups,[2-6] and in only one of these trials was the difference not significant.[3] This benefit alone would make properly organised screening of older people worthwhile since most old people dread spending their last years in a home or hospital. Furthermore, care of frail, dependent old people in an institution is very expensive, which is why the government has privatised this sector of the NHS.

Harris's article was subtitled "The doubt persists." This is true, and consequently the Medical Research Council hopes to do a cost-benefit evaluation of screening of elderly people in 108 practices in Britain, starting next year. Pending the results of this study, it is important for family doctors—who are often pessimistic about the value of screening elderly people—to recognise this established and important benefit which the editorial does not mention.

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EDITOR,—In his editorial on health checks for people over 75 Andrew Harris discusses methods of assessing the functional state and mental health of elderly people,[1] but by confining his attention to the clinical professions he misses an important point. Social services departments are required by the NHS and Community Care Act 1990 to revise their assessment procedures. Separate development could result in old people being interviewed by a succession of different agencies asking similar questions, each using different instruments to obtain and record the same information. This could be avoided by widespread adoption of standardised assessment scales that can be used by people with varying backgrounds and training.

A health check schedule devised in 1990 by the Oxford collaboration on assessment of elderly people (OxCASSE) incorporates the Barthel scale of activities of daily living[2] and the abbreviated mental test,[3] two scales recommended in a report[4] that was discussed in M J Bendall's editorial on improving the care of elderly people.[5] An evaluation by the King's Fund College ranked the OxCASSE health check in the top six of 54 assessment schedules rated for sensitivity to both health and social functions and for suitability of use with different care groups, by different agencies, and by assessors from a broad range of professions.[6]

Effective care of elderly people requires local and health authorities to work closely together. Whatever else they are intended to do, annual health checks should contribute to the development of a common language of assessment.

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1 Harris A. Health checks for people over 75. *BMJ* 1992;**305**: 599-600. (12 September.)
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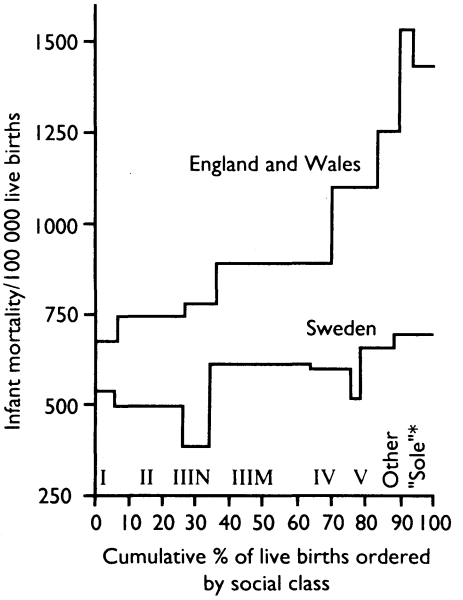
Social class differences in infant mortality

EDITOR,—D A Leon and colleagues provide an interesting comparison of the scale of socioeconomic differences in infant mortality in Sweden and in England and Wales.[1] Their conclusions, however, partly reflect the measures of inequality used. Simple comparisons of differences in mortality between contrasting classes in each country are vulnerable to the criticism that they compare mortality in dissimilar proportions of each country's population and take no account of how consistent the mortality gradient is across the population as a whole.

The best solution to this problem seems to be to follow Pamuk's use of the "slope index of inequality."[2] This is the slope of the linear regression of mortality across all classes weighted for the proportion of the population in each class. According to this measure, the degree of inequality in both neonatal and postneonatal mortality is appreciably smaller in Sweden than in England and Wales. This is true whether the mortality gradient is measured in terms of the absolute change in mortality across all classes or divided by the mean



*Scale of social class differences in infant mortality: Sweden compared with England and Wales. *In England and Wales this category comprised births registered by mother only. In Sweden it comprised principally births to mothers for whom no cohabitant could be identified*