the scheme is to reduce drug related harm to health.

After discussions between general practitioners and the health board clinical standards were defined and a review body appointed to supervise the administration of the scheme. General practitioners seeking approval have to agree to register from five to 20 patients with opiate dependence and a current or recent history of injecting drugs; to provide a drug counsellor at clinics; to attend educational meetings; to use oral methadone mixture as the only opiate substitute treatment; not to prescribe dihydrocodeine or temazepam; to arrange for methadone to be consumed under the supervision of a local community pharmacist; and to complete contact forms for every consultation, and a six monthly opiate treatment questionnaire⁴ for each patient, to allow evaluation of the scheme.

The scheme started on 1 May last year, and during the first six months 42 general practitioners were approved and 759 patients registered. This represents 9% of the estimated population of injecting drug misusers in Glasgow and 6% of principals in general practice. The results of evaluation are awaited, but the scheme seems to be popular with general practitioners and patients. It provides an opportunity for raising the standard of management in general practice of this potentially difficult group of patients while compensating general practitioners for the costs of doing so.

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it was routine in three units and done in high risk cases in eight; and for antibodies to hepatitis C it was routine in only one unit, with four other units requesting testing in some cases. Prophylactic procedures for hepatitis B and HIV infection after sharps injuries also varied. Hepatitis B specific immunoglobulin was given more widely than is recommended by the Public Health Laboratory Service,² including in circumstances in which the status of the source patient was not known (two units).

At the Royal Free Hospital all known source patients are approached for testing for hepatitis C antibodies, although this goes beyond recent guidelines from the Public Health Laboratory Service.³ We have found hepatitis C antibodies in 14% of source patients in reported incidents of exposure to blood, with a quarter of these infections being previously unknown.⁴ The decision whether to test source patients routinely for hepatitis C virus and other bloodborne viruses will depend on the rates of infection in each hospital and the resources available for counselling and testing. Many occupational health units in our region do not have access to testing for hepatitis C antibodies in source patients.

The audit exercise in the region has been helpful, allowing units to compare their practices and providing support for units requesting additional resources. The level of provision of occupational health care varies too much at present to allow the development of formal guidelines that all can follow, but we hope that this may be possible in the future.

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Auditing incidents of exposure to blood

EDITOR,—Ruth R White and Elisabeth J Ridgway highlight variations in the management of reported sharps injuries in Mersey Regional Health Authority.¹ The management of common conditions, especially when there is believed to be room for variation, is a good topic for audit. The North Thames (East) Regional Occupational Health Audit Group, which has representatives from all the occupational health units in the region, recently audited the management of reported incidents of exposure to blood.

Of the 17 occupational health units, 15 participated in the audit. All were involved in the management of reported exposure to blood in some way; four were not responsible for the initial management. Only one unit ran an out of hours service for such incidents; for several others this was provided by another department. Eleven units routinely stored a sample of blood from the staff member involved. The policies about approaching source patients to request tests for bloodborne viral infections varied considerably: for hepatitis B surface antigen this was routine in four units and done in high risk cases in eight; for HIV antibody

Treatment of myocardial infarction and angina

Review underestimated the benefit of early thrombolysis

EDITOR,—In their article on the treatment of myocardial infarction and angina John McMurray and Andrew Rankin perpetuate a serious error, underestimating the benefit of earlier thrombolysis in acute myocardial infarction.¹ Their figure 1 shows the loss of benefit within five weeks to be 1.6 per 1000 patients per hour of delay and is reproduced from the overview by the Fibrinolytic Therapy Trialists' Collaborative Group of all randomised trials of more than 1000 patients.² The graph shows the absolute reduction in mortality with thrombolytic treatment by delay from onset of symptoms to randomisation.

The severity of infarction, however, is negatively related to the delay by the patient, which constitutes a major portion of the time from onset of infarction to thrombolysis. The outcome of myocardial infarction treated at different times therefore depends on the balance between greater severity of infarction with earlier presentation and greater

efficacy of thrombolysis with earlier administration. Thus the benefit of earlier thrombolysis is underestimated when based on subgroup analyses of placebo controlled clinical trials in which patients are treated as they present.

The magnitude of the benefit of earlier thrombolysis can be determined only by a trial in which patients are randomly allotted treatment on presentation or after a delay. The three largest trials of this design are the European myocardial infarction project,³ the myocardial infarction triage and intervention study,⁴ and the Grampian region early anistreplase trial.⁵ Mortality at one month is reduced with prehospital thrombolysis by 1.7% absolute ($P=0.03$) when the results of all three trials are combined, though was not significantly reduced in any one of the trials.

The figure shows mortality at one month plotted against median times to the start of thrombolytic treatment in groups in whom it was started before and after admission to hospital; the lines linking the two groups represent gradients of benefit.

Mortality at one month plotted against median time at which thrombolytic treatment was started in prehospital and hospital groups in three trials. Figures in graph are benefit gradients. EMIP=European myocardial infarction project. MITI=Myocardial infarction triage and intervention study. GREAT=Grampian region early anistreplase trial

Although none of the comparisons is significant and there are large confidence intervals around each point, the figure gives three rough estimates of the magnitude of the time dependent benefit that are consistent with each other. Perhaps the best way to average these gradients is to draw the line of best fit between the six points: the resulting slope is 2.3% an hour. Over the period one to four hours from the onset of symptoms 23 more lives would be saved per 1000 patients treated per hour of earlier treatment. Giving confidence intervals for this estimate would give a spurious impression of reliability. Although tentative, this is so far the best estimate of the size of the time effect; it is more than 10 times that of the Fibrinolytic Therapy Trialists' Collaborative Group and substantially greater than that given in McMurray and Rankin's article.

Follow up of the Grampian region early anistreplase trial shows a separation of the survival curves, so that the reduction in mortality with prehospital thrombolysis is twice as great at one year as at one month. The true benefit of earlier thrombolysis may therefore be even greater than the estimate at one month given above.

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Advice on reducing cholesterol should be included

EDITOR,—John McMurray and Andrew Rankin's review of treatment of myocardial infarction and angina and subsequent advice on lifestyle modifications does not mention the advisability of reducing cholesterol concentrations.¹ This is interesting given that large studies have shown cholesterol reduction to be effective in inducing the regression of atheromatous plaques,^{2,3} from which a reduction in subsequent deaths related to coronary heart disease might be expected.

On the day that this paper was published the final results of the Scandinavian simvastatin survival study were announced.⁴ They showed that after six years of follow up the relative risk of death related to coronary heart disease had fallen to 0.58 (confidence interval 0.46 to 0.73). The trial design followed the European Atherosclerosis Society's guidelines of reducing total cholesterol concentration to less than 5.2 mmol/l.⁵ In fact, the evidence for the efficacy of cholesterol reduction in the form of formal trials of both diet² and drug based interventions⁴ is far greater than that for stopping smoking in secondary prevention.

In keeping with the scientific evidence, we contend that advice on reducing cholesterol concentrations cannot be omitted from any comprehensive care plan for coronary heart disease. Medical litigation over this omission may become more common in the future because the provision of strategies to reduce plasma lipid concentrations varies considerably at present despite the importance of the control of hyperlipidaemia in secondary prevention.

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Authors' reply

EDITOR,—We agree with the principle of John Rawles's point. If every patient could receive thrombolysis immediately at the onset of myocardial infarction the potential number of lives saved would be enormous. The difficulty is calculating the likely benefit in the more typical situation of trying to give thrombolysis perhaps an hour earlier than usual. Rawles's approach to this calculation is interesting. We believe, however, that drawing a line of best fit might not be the best way of getting a true estimate of the likely typical benefit of early treatment. We think that the

Effect of secondary prevention after myocardial infarction*

- Aspirin: 16 deaths, myocardial infarctions, or strokes
- β Blocker: 13 deaths, 5 myocardial infarctions
- Angiotensin converting enzyme inhibitor: Myocardial infarction complicated by left ventricular failure: 45 deaths
- Myocardial infarction complicated by left ventricular dysfunction: 12 deaths, 9 myocardial infarctions, 16 cases of chronic heart failure
- Stopping smoking: 27 deaths
- Hydroxymethyl glutaryl coenzyme A reductase inhibitor: 6 deaths, 12 myocardial infarctions, 11 coronary artery bypass graftings or percutaneous transluminal coronary angioplasties

*No of problems prevented per 1000 patient years of treatment

results of the three trials of early treatment should be weighted according to the number of patients in each trial. As the European myocardial infarction project (n=5469) was by far the largest trial (Grampian region early anistreplase trial, n=311; myocardial infarction triage and intervention study, n=360), it may give the best estimate of benefit. The average time saving in the European project (60 minutes) was also probably more realistic, in terms of usual urban practice, than that obtained in the Grampian trial (139 minutes). As the benefit gradient in the European project was 1.4% per hour our estimate is not unreasonable.

T Reynolds and A Wierzbicki are correct to point out that the Scandinavian simvastatin survival study is a landmark clinical trial that shows, for the first time, that reducing cholesterol concentration after myocardial infarction significantly reduces all cause mortality. Unfortunately, it was published about two months after our review was submitted for publication. While reducing a total serum cholesterol concentration of >5.5 mmol/l after infarction is of substantial benefit, the reduction in total mortality may not be as great as that seen after stopping smoking (table). The magnitude of benefit seems to be more like that obtained with treatment with aspirin and β blockers. The benefit of lowering cholesterol seems, however, to be additional to that obtained with aspirin and a β blocker and is probably also additional to that of stopping smoking.

Much greater attention will undoubtedly have to be paid in Britain to reducing cholesterol concentrations as an effective secondary prevention strategy after myocardial infarction. Benefit is also likely in other patients with established coronary heart disease.

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Management of ventricular fibrillation by cardiac arrest teams

EDITOR,—K Y Tham and colleagues report the results of a telephone survey regarding the management of ventricular fibrillation by doctors in cardiac arrest teams.¹ While the responses given to a telephone survey may be accurate, they may not correlate with actual management at cardiac arrests. We are auditing the management of

cardiac arrests in our hospital. We will establish how frequently the European Resuscitation Council's guidelines of 1992 on advanced life support are available² and how often they are followed. We hope to determine whether arrest calls are appropriate, who responds to them, and whether airway management is appropriate and effective. We have audited 45 arrest calls so far.

Our interim results show that in seven of the 45 calls the call was inappropriate. Cardiac output was lost in 34 cases and re-established in 15 of these. Initial airway management was inappropriate or ineffective in 14 of the 40 cases in which it was necessary (35%, 95% confidence interval 21% to 52%). Guidelines on resuscitation were not available at 16 of the 45 arrest calls (36%, 22% to 51%). Guidelines were not followed in six of the 34 cases in which they were appropriate (18%, 7% to 35%). We believe that this shortfall in the availability and application of guidelines on resuscitation is unacceptable. Initial airway management was effective or appropriate in only two thirds of cases.

We agree with Tham and colleagues' conclusion that leaders of cardiac arrest teams should be certified providers of life support techniques, but courses teaching these skills remain oversubscribed. This trust has recently appointed a resuscitation training officer; we believe that audit such as ours is essential to outline areas of deficiency so that training can be appropriately directed. Repeat audit later will determine if that training attains its goals.

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Risk of coronary heart disease in Asian women

EDITOR,—Rajeev Gupta and colleagues' study of risk factors for coronary heart disease in a rural Asian population highlights the link between educational status and risk.¹ Closer scrutiny of the lipid variables in the women studied, however, shows an important finding that the authors do not comment on and that may provide insight into mechanisms responsible for the high incidence of coronary heart disease in Asian women. A high proportion of the women (41%) had abnormally low serum concentrations of high density lipoprotein cholesterol—that is, <0.9 mmol/l. This was twice the proportion of men with such low concentrations, while the overall mean concentration of high density lipoprotein cholesterol in the men and women was identical. This pattern is completely contrary to that observed in white populations, in whom women have been reported to have higher high density lipoprotein cholesterol concentrations than men at all ages after puberty.² These higher concentrations have been used to explain the relative protection from coronary heart disease enjoyed by premenopausal women.

Interestingly, it has been reported that Asian women living in Britain show a similar lipoprotein pattern to that described by Gupta and colleagues. The mean high density lipoprotein cholesterol concentrations in Asian women were 0.18 mmol/l lower than those of their white counterparts and similar to those in Asian men.³

Why do Asian women have lower high density lipoprotein cholesterol concentrations than white women? This observation cannot simply be