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Advice on reducing cholesterol should be included

EDITIOR,—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,²³ 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.' In fact, the evidence for the efficacy of cholesterol reduction in the form of formal trials of both diet' and drug based interventions' 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.

> T REYNOLDS Consultant chemical pathologist

Clinical Chemistry Department, Burton Hospital, Burton on Trent DE13 0RB

A WIERZBICKI Lecturer in chemical pathology

Charing Cross and Westminster Medical School, Chelsea and Westminster Hospital, London SW10 9NH

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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.

Department of Cardiology, Western General Hospital, Edinburgh EH4 2XU

ANDREW RANKIN Senior lecturer

Consultant

IOHN MCMURRAY

University Department of Medical Cardiology, Royal Infirmary, Glasgow C31 2ER

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.

T M COOK Registrar in anaesthesia J HANDEL Senior registrar in anaesthesia

Department of Anaesthesia, Royal United Hospital, Bath BA1 3NG

1 Tham KY, Evans RJ, Rubython EJ, Kinnaird TD. Management of ventricular fibrillation by doctors in cardiac arrest teams. BMJ 1994;309:1408-9. (26 November.)

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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/1. 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/I 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