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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

explained on analytical grounds as lipid analyses performed in different laboratories give consistent results.^{1,3} Also, the men in the present study served as an internal control. The influence of diet and parity cannot be excluded as major contributors to this phenomenon, but Gupta and colleagues do not provide good data on these points. The endocrine control of plasma high density lipoprotein cholesterol concentrations has been widely studied, and the effect of insulin resistance in this group may be important.⁴

Gupta and colleagues' study adds weight to the increasing body of evidence that Asian women are at increased risk of coronary heart disease. We may expect an increase in the incidence of coronary heart disease in the British Asian population in the next decade, and further studies are urgently required.

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Temperature and cardiovascular mortality

EDITOR,—As Peter Wilmshurst points out, international rates of cardiovascular disease generally become higher as the temperatures become colder¹; this pattern holds true even within many countries. Scotland, however, shows the opposite, as colleagues and I found in our studies of geographical patterns of mortality from various diseases in Scotland.^{2,4} We examined the standardised mortality ratios for diseases during three quinquenniums (1959-63, 1969-73, and 1979-83) for the communities described in the annual reports of the registrar general for Scotland: 231 and 234 communities (cities, large and small burghs, and landward areas) in the first two quinquenniums respectively and the 56 local government districts in the last quinquennium.

The table shows the percentages of communities with standardised mortality ratios for coronary heart disease (*International Classification of Diseases* (ICD), seventh revision, codes 420-422; eighth and ninth revisions, codes 410-414) significantly

($P < 0.05$) high or low in the regions in the three categories of latitude (the corresponding numbers are given in the references). Because the diagnoses of coronary heart disease and cerebrovascular disease are liable to be confused, the pattern for heart disease would have been explicable as a diagnostic artefact if a reverse trend for cerebrovascular disease had been found. The comparable data for cerebrovascular disease (ICD, seventh revision, codes 450-468; eighth and ninth revisions, codes 430-438) showed a pattern broadly similar to that for heart disease, though less pronounced (table).

A knowledge of the locations of the regions or a view of the maps of these diseases² also shows a tendency for mortality to rise from east to west. Thus the milder climate in the southwest and, indeed, along the whole west coast, due to the Gulf Stream seems also to promote higher mortality from cardiovascular diseases in the communities there. By contrast, the colder and bleaker environment in the northeast seems to keep people's hearts warm longer. What genetic (Celtish or Pictish) or dietary differences overrule the climatic gradient to cause this Scottish paradox?

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Diagnosing pulmonary embolism

EDITOR,—In his editorial on diagnosing pulmonary embolism Kenneth M Moser expresses the desire for a test—ideally rapid, non-invasive, and widely available—that “would rule in or rule out the diagnosis of venous thromboembolism with a high degree of certainty.”¹ A (nearly) exclusionary test would be valuable, especially when combined with other well studied non-invasive tests for deep vein thrombosis. Such a test exists and deserves wider recognition and use: liquid crystal thermography of the lower limbs.^{2,3} A normal result of this procedure excludes deep vein thrombosis with a high degree of certainty (97-100%),^{2,4} and the test is rapid and non-invasive. It is not widely available, but there is no reason why it should not be. It is simple to perform, free of risk, and cheap in both capital and revenue costs.

Deep vein thrombosis is detected by liquid

crystal thermography because of increased skin temperature plus loss of the normal temperature gradient in the affected lower limb due to changes in the capillary skin circulation mediated by vasoactive amines released from the site of thrombus formation. The small percentage of false negative results is due to thrombi that are not propagating or undergoing thrombolysis; they are not likely to be a potential source of emboli and are therefore not of immediate clinical importance. A normal result of liquid crystal thermography thus effectively excludes deep vein thrombosis and saves the patient from unnecessary further investigation or treatment with anticoagulant drugs.

Liquid crystal thermography is non-specific, so confirmatory tests for deep vein thrombosis are indicated when the result is positive. Deep vein thrombosis can, however, be confidently excluded in over one third of patients in whom it is clinically suspected, and thus these patients can be spared further investigation.³ This results in considerable saving in time and effort with regard to conventional investigations and in revenue savings, no matter what method of investigation of deep vein thrombosis is usually used. In institutions where lower limb venography remains the usual investigation for deep vein thrombosis, use of liquid crystal thermography has the added advantage that the risks due to ionising radiation or allergy to contrast medium, which arise in 36% of patients, are avoided.⁵

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Interpretation of electrocardiograms

Interpretation of the ECG is as much an art as a science

EDITOR,—The ability to measure the PR and QT intervals accurately is useful for preclinical medical students. It might also reasonably be expected of cardiologists. It is not, however, equivalent to the intellectual process of interpretation of the electrocardiogram, as the title of Hugh Montgomery and colleagues' short paper suggests.¹ Interpretation often involves a doctor making a clinical diagnosis such as acute myocardial infarction, “old” myocardial infarction, left ventricular hypertrophy, or atrial fibrillation. The electrocardiogram is used extensively by the wide range of doctors who treat the commonest heart condition—that is, coronary artery disease. Accurate interpretation of the electrocardiogram in a patient with coronary artery disease is often difficult, and in many patients the abnormalities of the ST segment or t wave can be described only as non-specific. Measurement of the PR or QT interval is rarely an important component of interpretation of the electrocardiogram in patients seen by general surgeons, cardiothoracic surgeons, or accident and emergency physicians.

Interpretation of the electrocardiogram is as much an art as a science. It is vital, for instance, for appropriate administration of thrombolytic treatment and deserves further investigation by more

Percentages of communities in three categories of latitude in Scotland with significantly high or low standardised mortality ratios (SMRs) for coronary heart disease and cerebrovascular disease

Region	Significantly high SMR			Significantly low SMR		
	1959-63	1969-73	1979-83	1959-63	1969-73	1979-83
Coronary heart disease						
North (Highlands and Islands, Grampian)	0, 3	12, 3	18, 0	28, 17	28, 23	0, 80
Middle (Tayside, Central, Fife, Lothian)	10, 7, 8, 5	0, 13, 4, 5	0, 33, 66, 0	33, 27, 12, 30	24, 13, 15, 25	0, 0, 33, 50
South (Strathclyde, Borders, Dumfries and Galloway)	43, 24, 16	36, 6, 16	63, 0, 75	6, 6, 11	11, 18, 0	11, 25, 0
Cerebrovascular disease						
North (Highlands and Islands, Grampian)	8, 17	24, 6	27, 20	28, 26	28, 29	9, 60
Middle (Tayside, Central, Fife, Lothian)	14, 0, 12, 15	10, 13, 8, 25	0, 33, 66, 25	24, 0, 31, 10	33, 7, 12, 25	33, 0, 33, 50
South (Strathclyde, Borders, Dumfries and Galloway)	23, 6, 21	29, 12, 42	42, 25, 50	11, 12, 11	4, 35, 5	21, 25, 25