lies in defining high risk. There was general agreement that patients with established coronary heart disease and hyperlipidaemia (cholesterol concentration >6.5 mmol/l), asymptomatic patients with familial hyperlipidaemia (>7.8 mmol/l) and a strong family history of premature death under 60, and asymptomatic middle aged men and women with multiple risk factors and a similarly malign family history are strong candidates for lipid lowering drugs.

The association's guidelines also recommended lipid lowering drugs for patients with established coronary heart disease and a serum cholesterol concentration exceeding 5.2 mmol/l (the great majority), for asymptomatic men with hypercholesterolaemia (>7.8 mmol/l) as their only risk factor, and for asymptomatic women with hypercholesterolaemia (7.8 mmol/l) and a low ratio (<0.2) of high density to low density lipoprotein but no other risk factors. Given the doubts about the costs and benefits of lowering cholesterol concentration, these indications for starting patients on lifelong treatment are debatable. How many numerate participants in the primary prevention trials of cholestyramine and gemfibrozil would contemplate lifelong drug treatment with equanimity if informed that these drugs had reduced the incidence of cardiovascular events by only 1.7% and 1.4% respectively when the treatment groups were compared with the control groups.² Among control subjects 90.2% in the cholestyramine trial and 95.9% in the gemfibrozil trial did not suffer a cardiovascular event. Participants might also be deterred by being told that their risk of dying had not been significantly reduced over the duration of the trial and that information on mortality beyond the trial period was unavailable or, in the case of gemfibrozil, the subject of controversy.¹²

The use of lipid lowering drugs is increasing rapidly in most developed countries, driven mainly by promotional techniques and advertising literature that concentrate on the modification of surrogate biochemical end points.^{13 14} The epidemiological content of promotional literature is often inadequate or unbalanced, promoting drugs that either have not been the subject of randomised controlled trials or, as in the case of the statins, are currently being studied in randomised controlled trials whose results are unknown. Unfortunately, the sample sizes of current trials of the statins may lack the statistical power necessary for mortality to be evaluated as an end point.¹⁵

The larger "mega" drug and diet intervention trial proposed by Peto and colleagues would therefore be valuable and cost effective.¹⁵ Some 20 000 participants with an

anticipated 2000 deaths from all causes over five years would ensure sufficient statistical power to evaluate all cause mortality in high risk patients with established vascular disease. This trial, however, will not address the risks and benefits of treating most asymptomatic patients aged under 65 with primary hypercholesterolaemia as the annual all cause mortality in the control arms of the three largest primary prevention trials was less than a quarter of that expected in the proposed "mega" trial. About 80 000 trialists would be required to evaluate all cause mortality in this class of patient with a trial of comparable statistical power. The likelihood of such a trial being carried out is extremely small.

Without definite data on all cause mortality and with current unresolved concerns about excess deaths from non-cardiac causes in randomised controlled trials, decisions to embark on lifelong lipid lowering drug treatment in most patients with primary hypercholesterolaemia depend on the doctor's interpretation of available evidence. As in other situations in which certainty is illusory, this varies from evangelical enthusiasm for lowering lipid concentrations to therapeutic nihilism. Most doctors pursue a somewhat uncertain middle way, which attempts to balance putative risks and benefits for the individual patient. Further clarification may prove elusive in the foreseeable future.

MATTHEW G DUNNIGAN
Consultant physician

Stobhill General Hospital,
Glasgow G21 3UW

- 1 Peto R, Yusuf S, Collins R. Cholesterol-lowering trial results in their epidemiologic context. *Circulation* 1985;72(suppl III):45.
- 2 Oliver MF. Might treatment of hypercholesterolaemia increase non-cardiac mortality? *Lancet* 1991;337:1529-31.
- 3 Muldoon MF, Manuck SB, Matthews KA. Mortality experience in cholesterol-reduction trials. *N Engl J Med* 1991;324:922-3.
- 4 Brett SA. Treating hypercholesterolaemia. How should practising physicians interpret the published data for patients? *N Engl J Med* 1989;321:676-9.
- 5 Jacobs D, Blackburn H, Higgins M, Reed D, Iso H, McMillan G, et al. Report of the conference on low blood cholesterol: mortality associations. *Circulation* 1992;86:1046-60.
- 6 Davey Smith G, Shipley MJ, Marmot MG, Rose G. Plasma cholesterol concentration and mortality. The Whitehall study. *JAMA* 1992;267:70-6.
- 7 Hulley SB, Walsh JMB, Newman TB. Health policy on blood cholesterol. Time to change directions. *Circulation* 1992;86:1027-9.
- 8 Holme I. Relation of coronary heart disease incidence and total mortality to plasma cholesterol reduction in randomised trials; use of meta-analysis. *Br Heart J* 1993;69(suppl):542-47.
- 9 Davey Smith G, Song F, Sheldon TD. Cholesterol lowering and mortality: the importance of considering level of risk. *BMJ* 1993;306:1367-73.
- 10 Davey Smith G, Pekkanen J. Should there be a moratorium on the use of cholesterol lowering drugs? *BMJ* 1992;304:431-3.
- 11 Betteridge DJ, Dodson PM, Hughes EA. Detection and management of hyperlipidaemia: guidelines of the British Hyperlipidaemia Association. *Postgrad Med J* (in press).
- 12 Newman TB. Possibly disappointing results of treatment with gemfibrozil. *N Engl J Med* 1993;328:139-40.
- 13 Magnani HN, ed. *Scrip's 1991 hypolipaeic report*. Richmond, Surrey: PJB Publications, 1992.
- 14 Sheldon TA, Davey Smith G. Consensus conferences as drug promotion. *Lancet* 1993;341:100-2.
- 15 Collins R, Keech A, Peto R, Sleight P, Kjeldsen S, Wilhelmsen L, et al. Cholesterol and total mortality: need for larger trials. *BMJ* 1992;304:1689.

The GMC: size and public accountability

Shrink it drastically but increase lay members as a proportion

One of the problems of the General Medical Council (GMC) is its size. Another problem is the perception that it has insufficient lay representation and hence is not sufficiently accountable to the public. These two problems come together in proposals to reconstitute the council, which will be debated at its meeting next week.

The GMC started with 24 members but now has 102. Organisational psychologists think that the optimum size of a group that takes decisions rather than debates and rubber stamps them is about eight. Inevitably, as the GMC's size has grown the influence of the full council has declined. This problem is seen in many organisations as they try to balance

representativeness against effective decision making, and many don't get it right. They end up with large, expensive, ineffective talking shops, and the real decisions are made elsewhere by often unelected cabals. Doctors may be particularly at risk of ending up with councils that are too large because of their suspicions of those who try to lead. Many members of the GMC would like to see a smaller council, but most are reluctant to give up their own seats.

The belief that the council ought to have more lay members has been growing for a long time both in the broader community and within the council. This is because the lay members are much appreciated and are being worked harder

and harder as they take on more tasks and because the leaders of the GMC are anxious to head off any proposal to scrap the council and introduce a new organisation dominated by lay people.

At the moment there are 11 lay members—appointed by the Privy Council on the advice of the health departments. Trying to spot the reasoning behind the appointments is a favourite game, but by custom rather than law two members in recent years have been members of parliament. One radical possibility would be to make lay members the majority, but the council is not ready for this. The council is thus likely to go for a much more modest increase—to something like 20. The question that immediately arises is whether these people should be appointed in the traditional way or whether they should be elected. If the aim is increased accountability it would obviously make sense to have the lay members elected rather than plucked from Hampstead dinner tables or from among Bradford butchers. But how? Tacking a poll on to public elections seems impractical, and electing members from unelected bodies like the Association of Community Health Councils or the Patients Association might decrease rather than increase representativeness. But elected lay members would surely be better than appointed ones.

Once the council has increased its lay members it must think whom to discard if it is to shrink rather than expand. The choice lies among the 21 members from the universities, the 14 from the other bodies producing registrable qualifications (the royal colleges and faculties and the Society of Apothecaries), the two chief medical officers, and the 54 elected members who are needed to make sure that they have a

comfortable enough majority not to need a byelection every time one of the members resigns or dies.

It's a matter of arithmetic, and the officers of the council and its members have been occupying themselves with it for some time. The rules of the game are that there must be an overall majority of doctors who have been elected (the idea that history might be rolled back and the elected members reduced to a minority was quickly killed), an increase in lay members, somebody from the government, somebody representing those granting registrable qualifications, and as small a council as possible. Another complication is that there should surely be at least somebody representing the consumers of undergraduate medical education—medical students. Most university authorities now have students represented, and the BMA's council includes students. At least two students should be elected: they would need mutual support.

One possible formula would be 20 lay members, one chief medical officer, four representatives from the universities and two from the other bodies producing registrable qualifications, two medical students, and 32 elected doctors—a total of 61. Better still might be to reduce the lay membership to eight and shrink the overall council even further, meaning that lay membership would increase proportionately. This could lead to a council of 30, saving doctors money (at a time when the retention fee looks set to explode) and increasing the effectiveness of the council.

RICHARD SMITH
Editor

BMJ

Bone gain and loss in premenopausal women

Physical activity, calcium nutrition, and sex hormone status are important

Low bone mass is an important determinant of age related fractures, and public health strategies to reduce the burden of these fractures currently aim at maximising the bone mass of elderly people. Bone mass in later life depends on both the peak attained during growth and the subsequent rate of loss. Although the risk factors for bone loss have been extensively studied,¹ the determinants of peak bone mass have only recently received the attention they deserve. We highlight three developments in our understanding of peak bone mass: the recognition that environmental factors have a role in its attainment; evidence that certain interventions during youth are capable of increasing bone gain during growth; and better information about the hormonal influences on skeletal status from studies of pathological conditions affecting premenopausal women.

Twin and family studies provide clear evidence that there is a substantial genetic contribution to peak bone mass. The size of this contribution is difficult to quantify, however, as estimates of heritability tend to underestimate the influence of environmental factors. The most recent data suggest that as much as half of the variation in peak adult bone mass among women is non-genetic.²

If environmental factors determine peak bone mass, what are they and can they be beneficially manipulated? Recent studies implicate physical activity, calcium nutrition, and sex hormone status as the three most important determinants. Cross sectional studies have shown associations between indices of physical activity and bone mass, especially in the proximal femur, among children and young adults. In a study

of 6–14 year old children those who spent more than two hours a day in weightbearing activities had around 10% greater wrist, spine, and hip bone mineral density than those who spent less than an hour each day in such activities.³ Data from a British national birth cohort study suggest that patterns of activity may track from school years into adult life, so that the benefit obtained from maintaining a high level of physical activity in childhood might be preserved in later decades.⁴ Finally, the only randomised controlled trial of exercise in premenopausal women showed that exercise significantly increased the mineral density of the young female skeleton.⁵

The relation between calcium nutrition and peak bone mass is less consistent. Observational studies have reported an association between the intake of dietary calcium in childhood and bone mass in adulthood, but studies in growing children have failed to replicate this finding. Nevertheless, a three year, double blind, placebo controlled trial of calcium supplementation in 22 twin pairs aged less than 14 years recently showed significantly greater bone gain in the forearms and lumbar spines of prepubertal twins who received a daily supplement of 1 g calcium compared with those receiving placebo.⁶

The greatest absolute rate of mineral accretion in the skeleton occurs during puberty. Numerous metabolic and hormonal changes occur at this time, including increases in the production of growth hormone, gonadotrophins, and sex steroids. Precisely how they influence skeletal development, however, remains elusive. Clues to this hormonal regulation have emerged from studies in older, though still premeno-