Avoiding premature coronary deaths in Asians in Britain

Cultural factors are important

EDITOR,—An improved health service for all minority groups is to be applauded (even if the motives are predominantly economic). It may, however, be simplistic to suggest that the way to prevent coronary heart disease among Asians is to inform them of factors that would increase their likelihood of coronary heart disease and then expect them to change their lifestyle accordingly.¹

The assumption that each person appreciates health risks solely on the basis of information that he or she possesses ignores the large influence of cultural factors on the degree of importance that people ascribe to any risk.² The idea that people then analyse the costs of these risks and change their behaviour accordingly assumes, firstly, that they use an economic cost-benefit conceptual model to determine their behaviour³ and, secondly, that they will be in a position to change it.

The medical profession often assumes that if people do not correct their behaviour then they are not behaving in a reasoned way or are unable to attach appropriate priority to their health, and they may then be held responsible for having brought on their illness.⁴ Categorising a racial group as being at risk of a disease for which they may be blamed will marginalise them. This is particularly unnecessary when we are aware of the limited efficacy of health education campaigns, even among the supposedly rational, "health maximising" white middle classes.

I agree that coronary heart disease among Asians is an important issue that warrants funds for public

information and that we should not avoid our responsibilities as a profession to attend to the health requirements of minority ethnic groups merely because this is a sensitive issue. My point is that if we concentrate solely on the changes that should be made by individuals, from any minority group, to decrease their risk of illness, without regard to the complexity of factors that affect their conceptualisation of risk and behaviour, this will have consequences that may alienate them further.

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Several key facts need to be considered

EDITOR,—Sandeep Gupta and colleagues' urge actions by the "Asian" community (the term "Asian" here is misleading to international readers) and by the Department of Health. What actions? The authors' advice is to control central obesity and increase physical exercise. How much of a dent will these actions make? If central obesity is controlled and all Asians exercise will coronary heart disease in Asians be avoided? Obviously not.

Actions arising from the insulin resistance hypothesis must be components of a strategy.⁵ The influential view that the prevalence of risk factors for coronary heart disease is lower in south Asians in Britain (British residents whose origins lie in India, Pakistan, Bangladesh, or Sri Lanka) than in the white population may be simplistic: when non-biochemical risk factors were studied the balance went against south Asians in Glasgow.⁶ There are several key facts.

Firstly, the cause of coronary heart disease is poorly understood. The reasons why the rates rise and fall over time within populations are unclear. Among the ethnic majority population in Britain coronary heart disease is on the wane. Among ethnic minority groups similar rises and falls will surely occur.

Secondly, mortality from coronary heart disease in south Asians, whose overall socioeconomic status is low, is almost identical with that in members of social class V in Britain.⁷

Thirdly, the key questions for south Asians are why the rates are high and how to reduce them, not why they are higher than in those in the white population.

Fourthly, the priority is the control of risk factors that are agreed to be causal, including smoking, raised blood pressure, raised cholesterol concentration, obesity, and failure to exercise.

Fifthly, maintaining the low prevalence of smoking in south Asian women and Sikh men and reducing smoking in Bangladeshi and Pakistani men⁸ are priorities.

Sixthly, research is needed on the hypotheses based on insulin resistance and the role of uterine

and early childhood adversity and on socio-economic influences including stress.^{9,4} Research must not, however, overshadow preventive activity.

Finally, pending clearer understanding, an inclusive rather than a reductionist strategy is appropriate.

In the 1980s little health education material on coronary heart disease existed for ethnic minority groups.⁷ This deficit is being corrected, and the challenge is to ensure that a credible knowledge base is used for health promotion. In searching for knowledge researchers must explore the byways and cul de sacs; but the public, policymakers, and practitioners should stay on the main roads.

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General practitioners should identify risks opportunistically

EDITOR,—I am a general practitioner in Tower Hamlets in east London, an area with a substantial population of Bangladeshi origin, who are at documented high risk of premature coronary death. I support Sandeep Gupta and colleagues' call for the development and funding of an immediate prevention strategy with a strong base in the Asian communities at risk, while research about cause and effective interventions should be continued.¹

I would underline the importance of using consultations in general practice to identify and address risks opportunistically. To be more effective this needs the resources of time and readily available trained health advocates when doctors or nurses do not come from the community concerned. Well person clinics often reach only a small proportion of patients, particularly in deprived areas.

My concern with the editorial is that it quotes the highly speculative hypothesis of adaptation to famine² as an explanation for the excess risk in migrant Asians as if the hypothesis has been proved. This piece of reductionism is ahistorical and antievolutionary and, while not so intended, is open to racist interpretation. There is no reason to suppose differential exposure to famine among south Asians compared with other ethnic groups over an evolutionary timescale.

The current emphasis needs to be on modifiable risk and how to modify it. For example, in some

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of my Bangladeshi patients I find that their haemoglobin A_{1c} concentration consistently reverts to near normal during long holidays in Bangladesh and doubles inexorably in London; they attribute this to taking less exercise and eating fewer fresh vegetables in London. Group work in the community may be a more effective way of helping understanding and modifying risk for those at high risk.³ It must be recognised that the relation between genetics and past and present environment remains complex and unresolved in relation to an increased risk in south Asians.⁴

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Benefit from controlling blood pressure was understated

EDITOR,—Sandeep Gupta and colleagues press the case for coronary prevention among British Indo-Asians.¹ While we continue research into causes we also have to emphasise what we already know and may not be applying. The authors mention adequate detection and control of high blood pressure only in passing.

Results of controlled trials in groups at high risk, including people in the Medical Research Council's trial in older adults and its American equivalent in systolic hypertension, showed a dramatic benefit from low dose diuretics with or without a potassium retainer, which produced respectively 44% and 27% reductions in coronary events when compared with placebo. Even updated overviews hide this benefit because of the negligible impact of monotherapy with β blockers on coronary events. The issue is not only to detect hypertension and start treatment but then to achieve target blood pressures of <150/90 mm Hg.² Current evidence from surveys suggests that British Indo-Asians have a high rate of hypertension (roughly 30% in middle age), which combines with and adds to the excess risk of diabetes.³ Does that matter?

No direct evidence is available for Indo-Asians. The case is clearest in diabetes in a study of early middle aged Americans followed up for 10-12 years.⁴ There were some 170 coronary deaths and probably double that number of non-fatal events, totalling 512 coronary events per 1000 diabetic men with systolic blood pressure >160 mm Hg. As effective control of blood pressure reduces this figure by a minimum of 16%, some 80 such events could be prevented. If recent evidence from Manchester applies across the nation the wasted opportunity for coronary prevention by proper control of hypertension is considerable: only 14% (55/387) of known hypertensive people with diabetes had blood pressures below 150/90 mm Hg, indicating that some 70 of these 80 potential events would not be prevented. These are conservative estimates.

Once hypertension is established, reaching and maintaining the target of <150/90 mm Hg should be the goal for all patients (particularly Indo-Asians and those at excess risk) as well as nurses, doctors, and purchasers. We have the evidence that it is worth doing.

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Guidelines for pharmacological intervention are needed

EDITOR,—Rates of coronary artery disease in young Asian Indians aged under 40 are three to 10 times higher than those in other populations.¹ I applaud Sandeep Gupta and colleagues for advocating an "effective campaign of primary prevention" of coronary artery disease in Asian Indians² but doubt its efficacy unless there is a diligent search for and aggressive treatment of dyslipidaemias.¹

Among the one million Asian Indians settled in the United States 27 000 are physicians, including 2000 cardiologists. These physicians have made major changes in their lifestyle. In a study of 1478 Asian Indian physicians and their family members in the United States only 68 smoked any cigarettes and 18 smoked over 10 cigarettes a day. Obesity defined as >120% of ideal body weight was noted in 105 (E A Enas *et al*, proceedings of the international symposium on atherosclerosis, Rosemont, IL, US, 6-11 Oct 1991). These physicians have a low saturated fat intake of 14 g (8% of energy) and high levels of exercise (averaging 136 minutes a week).³ Nearly half of them are vegetarians.¹ Yet 110 of the 898 men in the study had coronary artery disease, which is a fivefold higher prevalence than that in American white subjects (12.2% *v* 2.5%) (E A Enas *et al*, proceedings of the international symposium on atherosclerosis, 1991). The rate of admission to hospital for coronary artery disease in Asian Indians in the United States is also four times higher.¹ Contrary to common belief, insulin resistance cannot account for this "Asian Indian paradox," since native Pima American Indians, who have the world's highest rate of glucose intolerance (79%) and a high prevalence of other conventional risk factors, have the lowest prevalence of coronary artery disease of all Americans (six in 546 men (1%))—"the American Indian paradox."

Higher serum lipoprotein(a) concentrations, which are a powerful, independent risk factor for premature coronary artery disease, have been reported in Asian Indians. This concentration is largely genetically determined and minimally influenced by diet and exercise. The pathological effects of high concentrations are augmented by high serum low density lipoprotein, high triglyceride, and low high density lipoprotein concentrations. This "lipid tetrad" best explains the malignant atherosclerosis in young Asian Indians and the resulting high morbidity and mortality from clinical events.¹ Recently a "comprehensive lipid tetrad index" has been proposed to quantify the total burden of dyslipidaemias.⁴ The index is much higher in Asian Indians and is derived by the product of total cholesterol, triglyceride, and lipoprotein(a) values divided by the high density lipoprotein value.

Since serum cholesterol concentrations greatly underestimate the total burden of dyslipidaemia in Asian Indians, specific guidelines that allow

pharmacological treatment to be started at a lower low density lipoprotein concentration and at a lower age are urgently needed. Pharmacological intervention similar to that in secondary prevention of coronary artery disease seems justified as primary prevention in Asian Indians at high risk, because of the persistent excess of coronary artery disease even after intense modification of lifestyle. This strategy may be far more cost effective than waiting and eventually paying for "tertiary prevention," such as coronary angioplasty and bypass surgery.

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β Blockers have important role in pulmonary oedema due to mitral stenosis

EDITOR,—I read with interest C A Morley and B A Lim's Lesson of the Week about previously undiagnosed mitral valve disease causing abrupt deterioration in pregnancy.¹ Colleagues and I have had similar experience of young Asian women with pulmonary oedema due to mitral stenosis.

Most of these patients are in sinus rhythm. As the authors say, the patients can develop pulmonary oedema rapidly as tachycardia reduces the time for left atrial emptying during diastole, but the authors fail to mention the important role of β blockers in management. The fall in stroke volume leads to a vicious spiral of a reflex increase in the heart rate, a further fall in stroke volume, and an ever increasing left atrial pressure, all of which can be reversed by β adrenergic blockade.

Giving a β blocker in sufficient dose to counteract the powerful adrenergic drive and slow the heart rate to 80 or 90 beats/min has an almost miraculous effect in reducing left atrial pressure and is followed by immediate improvement. The left ventricle is not in trouble so no inotropes are required (they are best avoided because they raise the heart rate), and only a small initial dose of diuretic is needed. This gives time to plan the best strategy and, if necessary, to transfer the patient safely. Most of these patients' valves are suitable for balloon valvuloplasty, but before this procedure was available closed mitral valvotomy was much safer for the fetus than open valvotomy during cardiac bypass.

Familiarity plus tachycardia was probably responsible for the admitting team's failure to recognise the auscultatory features of mitral stenosis. However, this coupled with the delay in performing chest radiography or echocardiography, let alone in getting a cardiologist, make this a lesson in missed diagnosis and consequent mismanagement.

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