conclusion is the author's "impression that the increased incidence of peanut or nut allergy is real" and the statement that "there has been a considerable increase in the rate of referrals for food allergy." Even more disturbingly, Hugh A Sampson cites this study in his editorial in support of his conclusion that "the prevalence of peanut and nut allergy is increasing."[2]

While the incidence of nut allergy may indeed be rising, we believe that authors have a responsibility not to overstate their case, particularly on issues that are likely to be of interest to the media. Ewan should provide us with the evidence that led her to conclude that nut allergy is becoming common so that we can decide on this important issue, for ourselves and for our patients.

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Reduced exposure might increase allergic sensitisation

EDITOR,—Pamela W Ewan makes the important statement that the incidence of peanut and nut allergy is rising and that sensitisation seems to occur early in life.[1] Regrettably, she does not provide any evidence to back her recommendation that "young allergic children should avoid peanuts and nuts to prevent the development of this allergy" and her extraordinary suggestion that avoidance should be practised until the age of 7. Hugh A Sampson, in the accompanying editorial, makes similar recommendations and further suggests that mothers who are breast feeding should eliminate peanuts from their diet.[2]

Firstly, there is no evidence that avoiding foods during lactation or early childhood prevents allergic sensitisation to these foods. Indeed, in certain cultures that consume large quantities of peanuts, peanut allergy seems to be less of a problem than it is in Britain. Secondly, allergic sensitisation may occur in utero, but no advice is given on maternal diet during gestation. Thirdly, exposure to peanuts and other food allergens during lactation and childhood may be important in the development of immunological tolerance and may prevent allergic sensitisation to these foods. Finally, avoidance measures would serve only to reduce exposure to peanuts to low levels, and this could paradoxically increase allergic sensitisation to peanuts: low dose exposure to allergens (rather than high dose exposure) favours production of IgE,[3] and as little as 1 μg of inhaled allergen a year may be sufficient to induce allergic sensitisation via the airways.[4]

Prospective data comparing consumption of peanuts by children who are allergic to them and by atopic controls are required before broad policy recommendations are made. History contains far too many examples of uninformed health policies that were based on insufficient data and achieved unintended effects.

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Author's reply

EDITOR,—I am aware that various creams (for eczema, cracked nipples, and massage) contain arachis (peanut) oil. While these are possible sources of sensitisation, it has not yet been established whether this oil is allergenic. One study showed no effect of giving arachis oil orally to patients who were allergic to peanuts,[1] whereas another showed that it exacerbated eczema.[2] Such products have exacerbated eczema in some of my patients. More data are needed, and research is in progress. The makers of chamomile ointment are reformulating their product without arachis oil.

John A Wilson and Sheila Jones and Ian Jones question my suggestion that the incidence of peanut allergy has increased. This is based on 16 years' experience in major allergy centres. The rise in referrals began in the early 1990s. Studies are under way to measure prevalence, but one difficulty will be that no previous data exist. If Wilson has population based data on prevalence then he should publish them. Some of the rise will be due to increased public awareness, but I believe that a real change has also occurred. I have data showing that the age at sensitisation is falling, and most of the 62 patients on whom I reported had become allergic by the age of 2—that is, the cases were of recent onset.

Wilson questions the value of diagnosis and management. At the allergy clinic our approach is two pronged. Avoidance is the key, and expert advice is essential since peanuts and nuts are now often hidden in foods. Many of the children who died knew that they were allergic (exactly as Wilson describes), practised avoidance, but had not had professional advice. Secondly, we provide drugs for self treatment of reactions after inadvertent ingestion. This does not always mean adrenaline for injection (unpublished data).

My advice was that young atopic children (not all children) should avoid peanuts and nuts because of the strong association (96%) with other atopic disease. I agree with Gideon Lack and Jean Golding, however, that more studies are needed. I postulate that factors that are important in the increase in peanut allergy are the increase in atopic disease and early and increased exposure to peanuts. Avoidance can reduce sensitisation to food allergens,[3] but the effects of the dose of antigen on the production of cytokines are complex (S M Hugh *et al*, unpublished findings).[4] Genetic and other factors are clearly important in induction of the Th2 phenotype.

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Sesame allergy is also a problem

EDITOR,—Hugh Sampson's editorial on managing peanut allergy omits one important point[1]: medical identification bracelets should be worn at all times. Unsurprisingly, attention focuses on peanuts,[2] but sesame allergy, although less common than peanut allergy, can be every bit as severe. Sesame is used extensively in the food industry, and the seeds present a danger because of their versatility.[3] I report here my most recent allergic reaction to sesame. I was looking forward to an evening out with my daughter in law: a meal in a restaurant and then a visit to a theatre. I telephoned the restaurant to advise it of my serious allergy and then packed my "survival kit" (injectable adrenaline, an adrenaline inhaler, and a note that backs up my Medic-Alert bracelet). After my first anaphylactic shock in 1981 I was issued with an American kit containing a pre-filled adrenaline syringe and tablets of chlorpheniramine maleate. Eventually, this was replaced with the standard injectable adrenaline that is issued by the NHS. I had never felt comfortable with this: it had to be assembled before use, and I wondered how I would cope in an emergency.

I reminded the restaurant staff about my allergy; I always feel a bit uneasy when eating out. A glass of champagne calmed my nerves, and then the soup arrived. I tend to avoid soup when eating out,[4] but this was made in house and I was assured that it did not contain sesame. It did. Within seconds my mouth started to tingle, my ears burnt, my neck flushed, and my hands started to itch—characteristic signs of an allergic reaction. I rinsed out my mouth and tried to assemble the syringe. Impossible! Could anyone, in such a stressful situation? I cursed the syringe, abandoned it, and used my inhaler instead. The restaurateur was frantic: "Is there a doctor in the house?" There wasn't.

We sat outside and waited for an ambulance. I was gasping for breath and wanted to be sick. I was. A warm glow came over me, and everything just faded away. In the ambulance the paramedics clamped an oxygen mask on my face. My son, who must have driven like Fangio, arrived at the hospital just as the casualty officer was preparing an injection. I smiled when I heard his voice: "Excuse me, my mother is allergic to sesame; it's used in some drugs."[5] We had done our homework.

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Value of ECGs in identifying heart failure due to left ventricular systolic dysfunction

EDITOR,—We wish to reply to the letters[1] about our short report.[2]

We are pleased to learn that Suresh Khandekar and colleagues are following our example in using electrocardiography to identify heart failure due to left ventricular systolic dysfunction, but we do not understand why they use an automated report for interpreting electrocardiograms. While we appreciate Kamlesh Khunti and Robert McKinley's concerns about the interpretation of electrocardiograms in general

practice, we do not suppose that automated reporting represents an acceptable alternative gold standard. Furthermore, we made a specific distinction between major abnormalities, which are generally easily distinguished,[3] and minor abnormalities, which are generally harder to distinguish. We also pointed out that patients with a murmur will require echocardiography because patients with valvar heart disease may have a normal echocardiogram; this may explain part of the difference between Khandekar and colleagues' and our findings (in our service, referrals for assessment of murmurs were not encouraged and few such referrals were received).

We accept Simon Sanderson's comments about predictive values and prevalence, although we think that his qualifications strengthen our case. One of our chief findings was the exceptional negative predictive value of the electrocardiogram (that is, a normal electrocardiogram virtually excludes chronic heart failure secondary to left ventricular systolic dysfunction). This was subsequently confirmed by data from the coronary artery surgery study's registry.[4] Sanderson's recalculation of our results on the basis of a lower prevalence yields an even more exceptional negative predictive value of 99.9%.

We are pleased to see that Gregory Y H Lip and colleagues are so far in agreement with our findings that they have extended them to acute heart failure in hospital practice. One of our reasons for publishing our findings in a general medical journal was the fact that, while they may do little more than "confirm a longstanding clinical impression," they do confirm it and provide evidence for it. They are therefore of interest to a wider audience than general practitioners alone.

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Inequality in income and mortality in US

Lower mortality in Hispanic population may have affected findings

EDITOR,—In their study on inequality in income and mortality in the United States, George A Kaplan and colleagues discuss the fact that many of the states in which the proportion of total household income received by the less well off 50% was low were those with a considerable proportion of African American residents.[1] Since African Americans have a higher overall mortality they questioned whether the strong correlation that they found between inequality of income and mortality simply reflected those states having a higher proportion of African American residents. They dismiss this as a possible confounder, however, since they found virtually identical correlations between household incomes and mortality in both the white and African American populations.

What the authors failed to address was the possible effect that the Hispanic population in a state had on this correlation. Although a higher proportion of the Hispanic population than the non-Hispanic population in the United States lives in poverty, the Hispanic population has a lower mortality.[2,3] Thus the correlation between inequality of income and mortality could be affected in states with a high proportion of Hispanic residents. We note that the seven states with the highest percentage of Hispanic residents (New Mexico (38.2), California (25.8), Texas (25.5), Arizona (18.8), Colorado (12.9), New York (12.3), and Florida (12.2)) had among the lowest age adjusted mortality per total household income in the study, as shown in the authors' figure 1.[4]

This suggests that the Hispanic population, which is overwhelmingly immigrant, may have some sociocultural characteristics that somehow blunt the effects that inequality of income has not only on mortality but on various other health outcomes. If this is so then the overall solution to the poorer outcomes associated with the inequality of income must include not only policies that address distribution of income and wealth but also actions that preserve protective sociocultural traits.

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Authors' reply

EDITOR,—Patrick T Dowling and Joseph M Allevato suggest that our report of an association between inequality of income and mortality in the United States may be incomplete because we did not consider the proportion of Hispanic people in each state. We recognise the apparent protected mortality status of Hispanic people, although controversies about the validity of the numerator, denominator, and mortality linkage used in calculating Hispanic mortalities are unresolved.[1,2]

While the states that Dowling and Allevato mention as having the highest proportion of Hispanic residents do tend to have a mortality below the median (five of the seven do so), eliminating these states from the analyses makes little difference to the results. For all states, the correlation between our measure of inequality of income and all cause mortality in 1990, adjusted for median income, was –0.59 ($P<0.0001$); when the seven states with the highest proportions of Hispanic residents were excluded the correlation was –0.65 ($P<0.0001$). Contrary to Dowling and Allevato's suggestion, the association between inequality of income and mortality was considerably higher when only those seven states were considered, although one should be cautious in interpreting a correlation based on only seven points.

We agree that it is important to examine sociocultural characteristics that protect groups against health risks. It is also important, however, to note that macroeconomic factors and their consequences may make it more or less difficult for groups to maintain these protective characteristics over time. It will be interesting to see whether the well documented increasing inequality of income in the United States[3] will put at risk the protective traits to which Dowling and Allevato refer.

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Monitoring may need to be prolonged in patients given warfarin and amiodarone

EDITOR,—B Cheung and colleagues report a case from Hong Kong illustrating a potential latent interaction between warfarin and amiodarone.[1] They detail the pharmacokinetics of amiodarone and how these may have contributed to the problem but do not discuss the pharmacodynamics of warfarin, which may also be relevant.

Warfarin has a relatively long elimination half life (average about 35 hours for the racemate) and takes at least a week to reach steady state plasma concentrations.[2] The half life for the biological effect varies for the different vitamin K dependent coagulation factors, and changes in the activity of these factors lag behind changes in the warfarin dose by one to three days. Chinese patients are more sensitive to the anticoagulant effect of warfarin, and the mean maintenance dose is about 3.0 mg daily in Chinese patients,[3,4] compared with 6.1 mg in white patients. The reason for this is unclear and may be a combination of both pharmacokinetic and pharmacodynamic effects. The metabolism of many drugs differs between Chinese and white patients, but detailed studies have not been conducted with warfarin. There are also racial differences in clotting factors: fibrinogen concentrations, for instance, are about 20% lower in Oriental than white people.

Recent studies have shown that the warfarin dose required in Chinese patients, as in white patients, has a negative correlation with age, decreasing from a mean of 4.0 mg daily in patients aged 31-40 to 2.3 mg daily in patients aged over 70.[5] As Cheung and colleagues indicate, less than half the usual dose of warfarin may be required with concomitant amiodarone treatment, so it could be predicted that the elderly patient on whom they report would require a maintenance dose of ≤ 1 mg daily if he were Chinese.

In Chinese patients we usually give a loading dose of 5 mg warfarin daily for two days and then continue with an average of 2-3 mg daily depending upon the international normalised ratio on day 3. Cheung and colleagues do not seem to have used a loading dose in their patient, and a steady state is unlikely to have been reached after only five days' treatment (fig 1). Similarly, the appropriate maintenance dose