Miss Hunt and her colleagues have postulated that vitamin D and to a lesser extent outdoor exposure are the two most important factors in the determination of the vitamin D status of Asians. If this is so any difference in the serum 25-OHD₃ levels of the five different religious groups should be explicable in terms of these two variables alone. Examination of the variation in serum 25-OHD₃ when the concomitant variation in outdoor exposure and vitamin D intake had been taken into account in the model mentioned above revealed that a significant difference in the serum 25-OHD₃ levels of the different religious groups still existed ($F=9.7$; 4,74 DF; $P<0.001$). On inclusion of chapatty intake in the model, no such difference between religious groups was detectable ($F=2.36$; 4,73 DF; $P>0.05$). It would therefore seem essential to include chapatty in any model of vitamin D status in Asians. The amount of variation in serum 25-OHD₃ explained by chapatty flour alone ($23.04\% = 100r^2$) is greater than the combined contributions of dietary vitamin D and outdoor exposure (19.2%).

These findings strongly support earlier observations which have incriminated chapatty consumption in the aetiology of Asian rickets.³⁻⁵

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¹ Dunnigan, M G, McIntosh, W B, and Ford, J A, *Lancet*, 1976, **1**, 1346.

² Wills, M R, *et al*, *Lancet*, 1972, **1**, 771.

³ Ford, J A, *et al*, *British Medical Journal*, 1972, **3**, 446.

Septic arthritis in rheumatoid disease

SIR,—In your leading article on this subject (6 November, p 1089) reference was made to the use of gentamicin in the treatment of staphylococcal infections.

We fully agree that gentamicin is often highly effective in staphylococcal infections which may fall into the group of life-threatening situations for which the manufacturers of the drug recommend its use. You refer to other workers who use gentamicin in combination with the penicillins, but the dose regimen cited bears no resemblance to any recommended scheme of which we are aware.

An eight- or six-hourly regimen is advocated in patients with severe infection when renal function is normal or slightly impaired. The interval between doses can be lengthened according to renal function and serum levels of gentamicin. The dose can be based on many formulae but a starting dose of 160 mg, with recurrent doses of 80, 120, or even 160 mg can be given conveniently. Doses of 100, 200, or 70 mg, however, are novel and would require complicated dispensing procedures at ward level during which unacceptable errors may occur. The Glasgow workers suggest that gentamicin should be kept in reserve, yet seven out of eight of their patients died. You reach the satisfactory conclusion that septic arthritis occurring in rheumatoid disease is rare but dangerous to life.

Finally, the statement that "gentamicin should not be used in the elderly or those with

evidence of renal or 8th nerve disease" is untrue. The initial doses of gentamicin should preferably be given intravenously by bolus injection. There are no contraindications to the use of gentamicin since dosage can be tailor-made to the patient's renal function.

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*.*In regard to dosage the essential need is to make sure the total daily intake is correct in relation to the seriousness of the disease, sensitivity of the organism, weight, other therapy (such as dialysis) or interacting drugs (such as ethacrynic acid or cephalosporins), and abnormal risks of toxicity on account of age, impaired renal function, nervous disease, or pregnancy. All regimens, whether or not designed with the aid of nomograms, must allow for divided doses from ampoules. Nurses ought therefore to regard all doses as novel and check each one against the prescription and instructions attached to the specific ampoules by the pharmacist.

In the absence of evidence of the relative effectivity and toxicity, or risks of toxicity, of the intravenous and intramuscular routes it is still a matter of opinion which is to be preferred even for loading doses.

We accept that the statement about usage of gentamicin is only partly true. It would have been better omitted or worded: "In patients with impaired renal function, or the elderly, the dosage of gentamicin should, of course, be suitably adjusted from the start and readjusted according to the serum concentration. It is probably best avoided altogether in pregnancy or with 8th nerve disease."—ED, *BMJ*.

Labelling of foodstuffs for patients with coeliac disease

SIR,—The Labelling of Food Regulations in Britain do not require the declaration of gluten in the ingredients panel of a food label. Aware of the potential hazards for coeliacs, the Coeliac Society of Great Britain and Northern Ireland launched the Gluten-free Food Labelling Project in 1970-1. A gluten-free symbol was designed and food manufacturers were requested to adopt the symbol when their foods fulfilled the criteria for the designation "gluten-free."¹ This was preferred to a statement that gluten features as an ingredient, as advocated by Dr J F Fielding (1 January, p 50), the reason being that gluten is a name often applied to the protein of all starchy seeds in the food industry and is therefore not exclusive to the gluten protein toxic to coeliacs. It is still my hope, as the director of that specific project six years ago, that the Food Standards Committee will advocate adoption of the symbol. This has already been done voluntarily by some food manufacturers.

In the meantime the WHO Codex Alimentarius Commission continues to work to achieve international agreement on the definition of a gluten-free food, along with other specific requirements for foods for special dietary uses. At the ninth session of the Codex Committee in September 1975 the draft

standards were returned to an earlier stage of the committee's proceedings because an agreement on these standards had not been possible. The subject continues to be discussed. Until these standards can be agreed, at least by the delegations from countries in the EEC, it would not appear possible to institute an EEC blanket directive to state the presence of gluten.

However, I support Dr Fielding in the principle that responsible labelling should be sought for the benefit of people who must follow a gluten-free diet. The designations of certain ingredients in manufactured foods—for example, cereal binder, starch, rusk, etc—do not convey sufficient information for those on a gluten-free diet.

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¹ Lacey, A E, *Nutrition*, 1971, **25**, 99.

Effect of acute infections on allergic conditions

SIR,—A recent contribution to "Any Questions?" (30 October, p 1065) concerned the improvement in hay fever symptoms during viral hepatitis. I think the explanation may be simpler than your expert suggests.

I had a severe attack of chickenpox during which my severe allergic rhinitis disappeared completely despite increased exposure to the causative house-dust mite. When, shortly afterwards, two separate patients commented on the improvement in their asthma while acutely ill I discussed this strange coincidence with my colleagues. It was pointed out that the probable explanation was the high level of endogenous corticosteroids produced by an acute illness. It would be interesting to know the therapeutic equivalent of the steroid levels produced by acute stress of this type.

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*.*Our expert writes: "This suggestion that improvement in allergic conditions such as asthma or allergic rhinitis during acute viral illnesses might be related to increases in endogenous steroid levels seems plausible.

"It is well recognised that endogenous cortisol secretion rates and plasma cortisol levels increase during acute infections, both viral and bacterial.^{1 2} From these studies both secretion rate and plasma cortisol levels increase by up to 300%, but these responses vary among subjects. It has been suggested that the stress response in plasma cortisol levels is similar in self-limiting viral infections to that in severe bacterial infection.² The increased steroid production may be related to the degree of fever or at least to pyrogen levels, since bacterial pyrogens stimulate corticotrophin production in rats, probably by stimulating the pituitary directly.³ Interestingly, the response in rats is potentiated by the addition of salicylate.³ The steroid response to acute infections seems to be similar to, though usually slightly less than, that to corticotrophin infusions. In therapeutic doses these usually induce increases of 300-700% in both cortisol secretion rates and plasma cortisol levels.^{4 5}

"Thus in some individuals the increase in endogenous steroid production as a result of