rise in antibody titre at all. In a small minority of these individuals there may be evidence of reinfection. This usually manifests as an entirely subclinical event with only a 4-8-fold rise in antibody titre, all the antibody being of the IgG class. There is no evidence that such events represent any risk to the fetus. Very rarely there is evidence of clinically apparent reinfection^{1,2} and in the following case this appeared to be related to vaccine.

A 22-year-old staff member donated serum for rubella antibody screening on 13 January 1975. The rubella HI titre was 1/16 and all this activity fractionated as IgG on gel filtration with Sephadex G-200. On 18 February she was given rubella vaccine (Almevax) and on 24 February she developed a typical rubella-like illness with a rash. A serum sample taken on 4 March had a titre of 1/4096, and 50% of this activity was of the IgM class. The clinical features and the serological response in this case were similar to those occurring in primary infections. The patient described by Dr Snijder and his colleagues did not have a rash, but the magnitude of the serological response was again similar to that of a primary infection. Their failure to find specific IgM antibody in the serum taken in the 38th week does not prove that an IgM response did not occur, since specific rubella IgM antibody may become undetectable within 3-4 weeks of the acute infection.³

Thus it would seem possible that reinfection with evidence of a viraemia (the occurrence of a rash in the case described here and of congenital infection in the case described by Dr Snijder and his colleagues) may occur. However, whether these are reinfections or primary infections depends entirely on the significance of the low-titre rubella HI activity in the early sera. Haukenes and his colleagues have shown that all classes of lipoprotein can be non-specific inhibitors of rubella haemagglutination⁴ and that some sediment in the IgG-containing fractions in sucrose density gradients.⁵ The work of Freeman and Smith⁶ suggests that some α -lipoproteins will fractionate with IgG on gel filtration with Sephadex G-200. Thus it is possible that rubella HI activity which fractionates as IgG may in reality be non-specific inhibitor.

It is likely that in the vast majority of cases rubella HI titres of 1/16 represent specific antibody and immunity to reinfection with viraemia. Very occasionally such low titres may represent residual non-specific inhibitors and this should be taken into account when investigating persons with low rubella HI titres as possible cases or contacts of rubella.

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¹ Haukenes, G. and Haram, K O, *New England Journal of Medicine*, 1972, **287**, 1204.

² Northrop, R L, Gardner, W M, and Geittman, W P, *New England Journal of Medicine*, 1972, **287**, 615.

³ Pattison, J R, and Mace, J E, *Journal of Clinical Pathology*, 1975, **28**, 377.

⁴ Blom, H, and Haukenes, G, *Medical Microbiology and Immunology*, 1974, **159**, 271.

⁵ Haukenes, G, Haram, K, and Solberg, C-O, *New England Journal of Medicine*, 1973, **289**, 429.

⁶ Freeman, T, and Smith, J, *Biochemical Journal*, 1970, **118**, 869.

Marathon running and atherosclerosis

SIR,—Your leading article on the prevention of coronary disease (10 April 1976, p 853) correctly calls for modification of risk-related traits in the prevention of coronary heart disease. Marathon running is being used in several rehabilitation centres in the United States and

Canada with this in mind. The life style necessary to cover the 42 km on foot is a very demanding one and has few risk factors. Forty-three cardiac patients, graduates from formal exercise programmes, finished the 12 December Honolulu marathon and earned "non-competitive" trophies from the Hawaii Heart Association. Among the patients were cases of double and triple vessel disease, with and without infarctions, with and without bypass grafts. Physicians and nurses from the rehabilitation centres also ran.

Cardiac patients have been running marathons since the mid-1960s. They are so numerous that it is difficult to find a race without at least one. In some cases the clinical improvement on this new life style is quite remarkable. One bypass patient ran the 24 October New York marathon only four months after his heart surgery. Our surveillance of these distance runners, patients and non-patients, suggests that covering 42 km on foot is associated with a very high level of "protection" against atherosclerosis. To date we have been unable to substantiate a single case of necropsy-proved fatal atherosclerosis among these marathon runners.

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Units of measurement

SIR,—Dr Eva Lester (8 January, p 102) is to be congratulated on drawing attention to the geographical absurdities and the international isolation of the UK in respect of the recent changes in laboratory reporting. The controversy is not a moribund duck, however, as it still vigorously quacks on in Brighton and other areas in the country where, sensibly it now seems, those changes have not been made which are now described in your leading article (1 January, p 5) as "a futile and wasteful exercise."

As it is only a year since the implementation date was imposed by the DHSS irritated clinicians might request through their committee structure that a return be made to the more convenient and uniform traditional system of reporting which conforms with the majority internationally and the *Système International*.

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SIR,—While agreeing with the tone of your leading article about the SI (1 January, p 5) I must say I always felt the same about the old units as well. Seldom-used values have to be looked up each time; the precise significance of borderline results are obscure and the range of each and every value is different.

Would it not be beyond the wit of our "computers-in-medicine" men to "normalise" biochemical and other pathological data? The application of a formula to each value could result in a conventional normal of 100 (say), with 90 and 110 being placed one standard deviation from the normal and 80 and 120 two standard deviations away. Some data cannot be handled this way.

Normalisation would make for much greater intelligibility in laboratory reports and

be an aid to communication between doctors and different disciplines and reduce (however minimally) the obscurantism which bedevils our profession.

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SIR,—I would like to see the authors in your journal, on your instructions (1 January, p 6), giving scientific measurements in either SI or traditional units followed by the normal range of values, in the same units, in parentheses. I submit that scientific measurements should never be given verbally or in print without stating the normal range of values. At the most the omission of the appropriate normal range can lead to severe injury or death of a patient and at the least infuriate the reader who has mislaid his list of normal values. At the best it would allow your readers, wherever they are in the world, to appreciate the significance of the scientific result immediately, without recourse to conversion tables. There is no need to increase the printed space by giving two sets of units. When HM Government accepted the *Système International d'Unités* our hospitals and surgeries were saturated with conversion tables.

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Chapatty intake, vitamin D status and Asian rickets

SIR,—We read with interest the paper by Miss Sandra Hunt and her colleagues on the dietary intake and vitamin D status of British Asians (4 December, p 1351). Because of our mutual interest in Asian rickets Miss Hunt generously provided us with her findings some months ago. After noting the magnitude of the correlation between chapatty intake and serum 25-hydroxycholecalciferol (25-OHD₃) we carried out a more detailed statistical analysis.

Although the data on chapatty intake, vitamin D intake, and outdoor exposure were not normally distributed, transformation to a logarithmic scale allowed the results to be adequately fitted using a linear model. The results of the analyses presented below were derived from these logged values. The parametric correlations of serum 25-OHD₃ with chapatty intake, vitamin D intake, and outdoor exposure were -0.480, +0.348, and +0.345 respectively. Although the correlation of chapatty intake with serum 25-OHD₃ was the largest in magnitude, this might have partly reflected the correlation with vitamin D intake. The independent contributions of the three variables to the variation in serum 25-OHD₃ were therefore assessed by multiple regression analysis.

The multiple regression of serum 25-OHD₃ on outdoor exposure and vitamin D intake explained 19.2% of the variation in serum 25-OHD₃ levels, whereas inclusion of chapatty flour in the model increased the amount explained by 15.5% to 34.7%. This increase is significant at the 0.1% level as assessed by an F test in the corresponding analysis of variance ($F=13.7$; 1,77 DF; $P<0.001$). Hence chapatty intake provides a contribution to the serum 25-OHD₃ level which is additional to and independent of the effect of dietary vitamin D and outdoor exposure.

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