haemolytic streptococci (2.9×10^5 organisms/ml) which were serologically group B (the organisms were, unfortunately, not typed). Pasteurisation at 62°C for 30 min in a purpose-built Holder pasteuriser (Oxford¹) eliminated the organisms, as predicted from their known heat sensitivity.

The early-onset, septicaemic form of group B streptococcal infection is thought to originate from colonisation in passage through the birth canal; there is little information, however, on the pathogenesis of the less common, late-onset, meningitic form of the disease. Kenny and Zedd² and Schreiner and Coates³ have suggested that in the latter disease form breast milk may be a source of infection; these authors have reported three cases of infection in neonates whose mothers' milk has grown group B streptococci. In our own studies it is not certain that the organism originated from the milk itself, but, whatever the source, the profuse growth of group B streptococci in a human milk pool again raises the important and unresolved question as to the bacteriological safety of using untreated pooled breast milk for feeding premature infants.

The advantages of untreated milk, with its live cells and intact protective properties, may be outweighed by its bacteriological disadvantages when using pooled milk from a milk bank, where cross-contamination and pathogenic bacterial overgrowth are inevitable problems. Optimal procedures in milk banking have yet to be defined, but at present pasteurisation would seem a satisfactory method of processing human milk. There is evidence for considerable preservation of the protective properties in milk with this form of heat treatment⁴ and our own preliminary data suggest that pasteurisation satisfactorily destroys most common pathogenic bacterial.

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¹ Gibbs, J H, et al, *Early Human Development*, 1977, **1**, 227.

² Kenny, J F, and Zedd, A J, *Journal of Pediatrics*, 1977, **91**, 158.

³ Schreiner, R L, and Coates, T, *Journal of Pediatrics*, 1977, **91**, 159.

⁴ Ford, J E, et al, *Journal of Pediatrics*, 1977, **90**, 29.

Changing mortality from ischaemic heart disease

SIR,—Dr C du V Florey and his colleagues (11 March, p 635) point to the associations between the recent slight decrease in mortality due to coronary disease in men and a number of concurrent changes in diet and in cigarette smoking. They draw only tentative conclusions from these associations, and this is particularly understandable in relation to a condition that has many causes.

In regard to diet the hesitation of the authors is even better founded than they themselves realise. The figures they use for food intake are those of the National Food Survey,¹ which measures only that which is consumed in the home and also omits entirely a range of items that include confectionery, ice cream, soft drinks, and alcoholic drinks. As a result the intake of some dietary constituents is considerably underestimated. For example, the survey assesses current average sugar intake at about 12 ounces (340 g) a week, but the real

total is more than twice as much as this, at around 30 ounces (850 g) a week. More importantly, the survey records a continuing smooth fall in sugar intake of some 30% between 1970 and 1976 (apart from the exceptional fall due to the world shortage of 1975), whereas total intake has changed very little over that period: less than 6% between the extremes if we again omit the figure for 1975.

To accept figures from the National Food Survey as accurately representing the amounts of food that people eat is as logical as to accept attendances at cinemas as accurately representing the number of films that people watch while ignoring those that are seen on television.

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¹ Ministry of Agriculture, Fisheries and Food, National Food Survey Committee, *Household Food Consumption and Expenditure for the Years 1968-76*. London, HMSO, 1970-7.

SI, moles, and drugs

SIR,—I read with interest your leading article on this subject (18 March, p 668). My experience is that most doctors are intelligent people who, on the whole, have made it. SI units may be "trying" initially, but "exhausting" seems an overstatement for all but the most tired.

Symbols such as kg^{-1} , meaning "per kilogram," appear straightforward. To suggest that they are ugly or even worse than they are "difficult to comprehend" could give rise to misinterpretation of the calibre of the profession by an outside observer.

The new units certainly require some work by the clinician. The difficulties outlined in the leading article are probably not insurmountable, and with a more positive attitude the new units will, like any other hurdle, be jumped successfully by the vast majority.

G V RIGBY

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SIR,—Your leading article (18 March, p 668) will restimulate interest in SI unit practice, which is good. However, I am now convinced you are wrong in opposing the recommended change in the method of indicating concentration per litre, from "/l" to $\text{"l}^{-1}\text{"}$. "/l" is perfectly safe when typed or in print, but about one in five of our young staff find it almost impossible to make the solidus ($/$) diagonal and they write it vertical, which is very dangerous next to a figure 1. It is not difficult to manipulate a typewriter in full flood to introduce the symbol for the inverse power of a litre, that is, l^{-1} . A slight turn of the left wrist and simultaneous tapping upon the - followed by the 1 (not, of course, by the l) makes a quick neat job of this symbol. If necessary a key can be made to do it in one jab. All the youngsters from school and university nowadays are trained in the use of this symbol. Where safety and speed matter we older ones must try to catch on.

Another point. We in this laboratory have used grams per litre for haemoglobin concentrations for several years now and are delighted that it is to be recognised as more desirable than grams per decilitre with its decimal point and general ability to confuse among the indices. We recognise that milli-

moles may come when a way round the monomer/tetramer controversy is thought out. A tip when changing over the grams per litre from grams per decilitre: always state the unit in full every time a haemoglobin concentration is mentioned, never just the figure. It's dangerous to answer, "What's the haemoglobin?" with "a hundred"; but perfectly safe if you say the unit out in full, "grams per litre."

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SIR,—I would like to correct an unfortunate error which crept into my letter (1 April, p 853), in which I stated that 513.5 mg of chlormethiazole edisylate and 161.65 mg of chlormethiazole base both provide 10 mmol of chlormethiazole (as edisylate or base). This should, of course, be 1.0 mmol. As I stated, care is needed in conversion!

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Serum IgE in mycosis fungoides

SIR,—After reading the paper by Dr P L Amlot and Mr L A Green on IgE in Hodgkin's disease and other lymphomas we considered it of relevance to collect the knowledge available of the relation between IgE and mycosis fungoides. Mycosis fungoides is a T-cell lymphoma originating in the skin, sometimes disseminating to lymph nodes and viscera. In three reports an elevation of serum IgE concentration (>1000 U/ml) has been found in mycosis fungoides, occurring in about 20% of cases.¹⁻³ However, the numbers of patients reported on were rather small—14, 22, and 23 respectively.

In the Scandinavian Mycosis Fungoides Study Group we have collected data on serum IgE levels in cases of mycosis fungoides since July 1974. Our findings are presented in the accompanying table. Among 75 patients the serum IgE level exceeded 1000 U/ml in only eight cases, and four of these had levels above 4000 U/ml. Neither a family or personal history nor clinical evidence of atopy (eczema, hay fever, or asthma) was recorded in these cases. In this series no relation could be found between the clinical type, stage, or response to treatment and the occurrence of elevated serum IgE concentration. In one of the cases the IgE level increased during treatment (from 1200 to 5200 U/ml) and in another case the level decreased (from 4000 to 300 U/ml); both patients were treated with psoralen and long-wave ultraviolet light (PUVA) to complete remission. No correlation was found in the series between IgE and the other immunoglobulins (IgA, IgG, IgM). At present we are not able to draw any conclusions from these

Serum IgE concentrations in mycosis fungoides

Stage of MF	Serum IgE concentrations (U/ml)			Total
	<500	500-1000	>1000	
Plaque stage	42	10	7	59
Tumour stage	8	2	1	11
Tumour stage with extracutaneous involvement	5	—	—	5
Total	55	12	8	75