only against the way it is sometimes presented. The paramount need is for balance, and to those who suggest that that is a dangerous, or unenforceable, concept we would reply that the parliament enforces it on television programmes in Norway (surely no less democratic than Britain). Much more use needs to be made of medical advisers, whose brief should be extended to cover all medical television programmes. There is also a good case for establishing a small committee to monitor programmes, as in Scotland.

Starting this summer, the BMJ hopes to introduce a long-planned feature—a new column “Medicine and the Media”—to review the best and the worst of the features on television and radio and in the press. Nevertheless, such reviews are unlikely to alter the attitude of the invincibly ignorant or prejudiced. In his article our special correspondent quoted Cromwell, and possibly the attitude of the BMJ may be entirely wrong. One thing is certain, however: mere debate is unlikely to settle the issue. So may we suggest a return to scientific method? Sir Michael Swann, FRS, the chairman of the BBC, is a distinguished man of science who understands the need for freedom and open discussion on the one hand, and for accuracy and fairness on the other. Let him collect a panel of, say, 20 uncommitted people and show them videotapes of the Horizon programme on induction of labour, the Panorama programmes on practolol and ECT, and the Tomorrow's World programme on breast cancer. Then let him ask them what conclusions they drew from the programmes. He will find that there is a striking gap between the impression given by the programmes and a consensus of reasonable medical opinion.

Hitherto in Britain our main guide to the application of SI in medicine has been the recommendations prepared by a working party of chemical pathologists, haematologists, and others for the DHSS, the substance of which was published in a paper by Baron et al in the Journal of Clinical Pathology.¹ WHO has now produced its own booklet, *The SI for the Health Professions*, which is described as “the most authoritative account of the subject that is available for the medical and allied professions.” Comparison of the two guides provides one or two interesting pointers to the ways in which medical SI has developed in the past three years and may be expected to develop in future. The newly agreed units of radiation are included, but how soon they will be generally adopted in clinical radiology remains to be seen; an increased emphasis on the kilopascal as the measure of blood pressure will hardly be welcomed, though the concession allowing the millimetre of mercury to be used is retained, albeit relegated to secondary position; while confusion will be caused by a change from the previously allowed expression of mass concentration of haemoglobin as g/dl to g/l and by a recommendation that “substance concentration (mmol l⁻¹) may be used provided it is specified whether the monomer—Hb (Fe⁺)—or the tetramer—Hb (4Fe⁺)—is used.”

More important, perhaps, is the failure to eliminate some of the more rigidly academic and practically unnecessary and irritating requirements. Foremost among these for the clinician is the insistence on the use of negative exponents in place of the familiar solids (¹) or “per” in complex symbols such as mg. kg⁻¹.d⁻¹. This sort of expression is ugly, difficult to comprehend, a nuisance to typists and printers, and intended to avoid an ambiguity that rarely if ever can arise in clinical medicine. It is one of the rules that can well be ignored—and indeed we already ignore it in the pages of the BMJ. Another absurdity is the rule that “concentration” must always be qualified by “mass” or “substance” even though the nature of the units that follow makes it obvious which is intended; the suggested abbreviations “mase” and “subst” are not ones that will commend themselves to medical writers, editors, or readers.

The greatly extended list of conversion units in clinical chemistry will be welcomed, but one serious omission from the new guide—as from the old—is a really clear exposition of the mathematics whereby a conversion factor that is not given in the table can be derived from the molecular weight (or “relative molecular mass”—another unnecessary change of traditional and well-understood terminology) of the substance concerned and of the necessity to know exactly which substance is concerned. Conversions from mass to substance concentrations may have to be undertaken by relatively junior laboratory or editorial staff, and a failure to understand the exact nature of the substance whose concentration is being measured may lead to a calamitous mistake. An unfortunate example of this possibility and the confusion resulting from it that has recently been experienced in this office is brought to light this week in our correspondence columns (p 716).

Linked with this is another serious omission—the lack of clear guidance on the use of SI units in clinical pharmacology. Drugs may be administered in the form of the base itself or of a salt or other compound of the base, but when assayed in the blood or urine the substance measured may be the base or its compound, a conjugate, a metabolite, or a mixture of these, so that it may be difficult or impossible to decide what the molecular weight should be for the purposes of calculation.

For this and other reasons, and because there is little likelihood as yet of drug dosages being expressed in moles,

### SI, moles, and drugs

Whether we like it or not, there has clearly been no relaxation in the determination of the World Health Organisation and other international bodies to secure the worldwide acceptance in the medical sciences of the Système International d'Unités (SI) and its corollary, the use of the mole, unit of substance, in place of the gram, the metric unit of mass, for the reporting of concentrations in clinical chemistry. Last year the 13th World Health Assembly noted that “the change to the use of SI units in medicine has already taken place or is now under way in several countries.” The latest recruit is Canada, and there are signs that opposition may be beginning to weaken even in those countries, notably the United States, in which it has hitherto been most stubborn. Universal acceptance seems most unlikely for a few years, however, and in the meantime we must continue to live in a world of duplicate sets of values, conversion factors, and, at the worst, confusion. Fortunately the clinical disasters that were widely prophesied in our correspondence columns and elsewhere when SI was introduced into the NHS in 1974-5 do not seem to have materialised—or at least have not been reported—but undoubtedly the past two years have been a trying and exhausting time for clinicians, who have had to familiarise themselves with new normal ranges and new standards of abnormality, all to no obvious practical advantage.

¹ British Medical Journal, 1978, 1, 323.
clinical pharmacologists in general seem averse to using substance concentrations; yet it is not unusual for clinicians to receive from their hospital laboratory the results of, say, serum digoxin estimations in nmol l. Some agreement here is long overdue, and the present uncertainty will not be dispelled by the thoroughly inadequate advice given in the section on “Pharmaceutically” in the WHO booklet or by the absence from the table of conversion factors of any drugs other than salicylate, barbiturates, and sulphonamides.

Altogether, we cannot believe that this booklet will do much to commend the SI to those who have not already adopted it or had it thrust upon them, while the statement that “it has been written in a style that makes it suitable for use by all health professionals—specialists, general practitioners, clinical chemists, nurses, and laboratory technicians, to name but a few” is at the very least a little optimistic.


Diet and asthma

Some foods may provoke asthma. In young children wheezing may be caused by immediate IgE-mediated allergic reactions after eating eggs, fish, nuts, and chocolate, but it is more common due to respiratory infections or inhaled allergens.1–4 Positive skin tests are found in about 15%, of children with asthma,6 less often in adults. Allergy to foods is largely an occupational problem for adults with asthma: sensitised workers may develop symptoms while handling grain, flour, coffee, and castor beans.5 About 25%, of positive skin tests to foods seem to be clinically relevant,1 but food allergy is a dominant factor in less than 1% of childhood asthma.5 The decline in importance with age of allergy to ingested foods may be due to the development of specific secretory IgA antibodies at the gastrointestinal mucosal surfaces, which prevent the absorption of offending proteins.6 Another mechanism is the development of effective blocking antibodies in the blood leading to systemic tolerance.7

In adults asthma can also be provoked by colouring agents and preservatives in food and drink. Tartrazine, an azo dye widely used for yellow colouring in foods, beverages, and medicines,9 may provoke asthma in sensitive individuals.10 Reaction to tartrazine seems to be especially common in asthmatics who are also sensitive to acetylsalicylic acid.10 Sodium benzoate, used as an antibacterial and antifungal agent in food and drink, may also provoke asthma.12 The pathogenesis of these reactions is uncertain, but they are commoner in non-atopic people, and no recognisable immunological mechanisms have been identified.

In a recent survey of 272 patients with asthma 30 gave a history of wheezing caused by orange drinks but only one wheezed with fresh oranges.15 Proprietary orange drinks commonly contain tartrazine with sodium benzoate and sulphur dioxide as preservatives. When 14 of the affected patients were given provocation tests the forced expired volume in one second (FEV1) was reduced after drinking solutions of sulphur dioxide in eight cases, after sodium benzoate in four, and after tartrazine in one. Three patients were sensitive to both sulphur dioxide and sodium benzoate. Patients who reacted to sulphur dioxide tended to be young, with extrinsic atopic asthma, while those reacting to sodium benzoate were older and non-atopic. Reactions to sulphur dioxide occurred within 1–2 minutes of ingestion. The effects of sodium benzoate tended to come on after 10 to 20 minutes, as occurs with tartrazine and salicylates. In four patients prior inhalation of sodium cromoglycate protected against reactions to sulphur dioxide, tartrazine, and sodium benzoate.

Exposure to sulphur dioxide may cause an immediate increase in airway resistance in normal persons,16 and this substance was thought to be the cause of an increased incidence of episodes of asthma recorded during periods of high atmospheric pollution.17 The speed of reaction after ingestion of sulphur dioxide solution is so fast that inhalation rather than gastrointestinal absorption may be the route of sensitisation (though rapid asthmatic reactions have been recorded18 with sublingual challenge tests to other substances).

In managing children with asthma the doctor seldom needs to resort to exhaustive skin tests, provocation tests, or trials of exclusion diets—offending foods have usually already been identified and excluded by the patient or parents.13 Adults with persistent or troublesome asthma may sometimes benefit from a diet free of additives, and a suitable dietary for use in Britain has recently been published.19 The importance of tartrazine as a provoking or exacerbating agent in asthma, especially in adults with aspirin sensitivity and nasal polyposis, requires further investigation. Treatment with sodium cromoglycate is worth a trial in patients with asthma in whom reactions to food additives may be acting as provoking factors.

Antibiotic-associated colitis: a progress report

Both doctors and patients often accept diarrhoea as a natural accompaniment of antibiotic treatment—a price that has to be paid for the benefits of science. The danger of this complacency is that diarrhoea may herald the onset of more serious disturbance. Staphylococcal overgrowth may be associated with a fulminant enterocolitis,1,2 a condition (fortunately rarely seen nowadays) which often had maximum impact on the lower small intestine. At sigmoidoscopy or necropsy some of these patients showed pseudomembrane covering the mucosa of the bowel, but staphylococcal enterocolitis and pseudomembranous colitis are now thought to be separate entities, and the resistant staphylococcus seems to play no part in causing pseudomembranous colitis.3 Nevertheless, this latter