is based on two false premises—that is, dosage related to body weight and a single safe maximum dose for all procedures—and is therefore of little value.

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[1] Braid DP, Scott DB. The systemic absorption of local anaesthetic drugs. *Br J Anaesth* 1965;**37**:394-404.
[2] Tucker GT, Moore DC, Bridenhaugh PO, *et al.* *Anesthesiology* 1965;**37**:277-87.

**We sent copies of these letters to the authors, who reply below.—ED, *BMJ*.

SIR,—In reply to Dr Wildsmith's letter we would like to reiterate the main purpose of our short paper—namely, to highlight the erratic way in which lignocaine is commonly used and to offer a simple guide to safer practice.

We accept that the correlation between body weight, total dose injected, and plasma concentration produced is not always high; in our study, however, most doctors did not use any objective physiological variables to guide their use of local anaesthetics. To suggest that they calculate a safe maximum dose according to factors other than body weight is, we feel, unrealistic.

Our chart covers body weights up to 70 kg only, and we would not recommend extrapolation to include obese patients. We accept that the caption may be misinterpreted in the manner described, though in Britain intravenous regional analgesia should be practised only by specialists, whom we hope would not consider that possibility.

Although we agree with Dr Dennison that the site of injection and type of procedure are important factors in the production of toxic plasma concentrations of lignocaine, we would like to point out that our article was aimed at a general medical readership, and 95% of junior hospital doctors questioned were using lignocaine for local infiltration or topical analgesia. Few of our respondents were anaesthetists, and we hope that doctors attempting the specialist procedures he mentions would be aware of the many other factors involved in drug toxicity.

We do not suggest that the use of 3 mg/kg of lignocaine produces predictable plasma concentrations, but we do believe that if the total dose is kept at or below those derived from the chart then toxic plasma concentrations are unlikely to be achieved. This is borne out by Dr Dennison's statement that even in the vascular sites used in intercostal, interscalene brachial plexus, and epidural blocks, toxic concentrations (of greater than 5 mg/l) are not found in total doses of less than 200 mg in a man weighing 70 kg.

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Out of hours calls in general practice

SIR,—The interesting and important article of Dr T Cubitt and Dr Gabriela Tobias (2 July, p 28) illustrates a fundamental aspect of general practice: the effect of the attitude of the doctor on the work.

Over the years I have noticed a considerable variation in diagnosis, treatment, and workload between adjacent practices and between different doctors in the same practice. There is little doubt that differences and changes in attitudes play an important part in determining how we view and treat the patient and the nature and extent of our workload. For example, the reported incidences of psychiatric illness[1 2] more than doubled between 1958 and 1974, with a corresponding increase in psychiatric prescribing, as a result of changes in attitudes.

At present, thanks to our long tradition of independent practice, our disease orientated medical training, and the frequent exhortations of the Royal College of General Practitioners to make a diagnosis in terms of physical, psychological, and social criteria,[3] we tend to see everyone as ill and requiring treatment. Thus we encourage ill health and dependency.

The great mass of minor illness with which we are kept so busy in our surgeries will not be reduced by more or new physical or psychological treatments, but by changes in the attitude of doctors. Changes that involve sharing realities with the patient and attempting to increase his confidence and independence.

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[1] General Register Office. *Studies of medical population. No 14. Morbidity statistics from general practice.* London: HMSO, 1958.
[2] Office of Population Censuses and Surveys and the Royal College of General Practitioners. *Morbidity statistics from general practice.* London: HMSO, 1974.
[3] Royal College of General Practitioners. *The future general practitioner. Learning and teaching.* London: British Medical Association, 1972.

Statistical guidelines for contributors to medical journals

SIR,—I agree entirely with the reply by Dr Altman and others to the letter of Dr Corea and others (9 July, p 132) but I think that there are points which, though not strictly statistical, need to be made explicit.

Validation studies are experiments in measurement and are necessary to determine the discrepancies that occur between the measurement methods employed. Repeated measurements by the same method produce discordant results, and the statistical null hypothesis is that these are due to chancelike processes. These discrepancies are reduced by calculating an arithmetic mean which minimises the squares of the deviations of the observations from the mean (the residuals). Similarly the correlation coefficient indicates how good a fit is obtained to a line which minimises the residuals of points derived from two sets of measurements. If these are measuring the same thing the correlation coefficient is unlikely to indicate a discrepancy in the data. But it is just these discrepancies that need to be measured in order to assess the value of the method.

An analysis of variance with partition of all common sources of variation and a reasonable number of repeated measurements by the same method in each cell is a compact and efficient design[1] and allows estimation of both random and systematic errors. Calculation of a correlation coefficient will irrelevantly indicate good agreement of the measurements but more importantly the intercept of the line may reveal a systematic error between methods and the slope may show a correlated error in one method. Further experiments are necessary to determine the source of these errors.

It is unfortunate and due to historical accident that the theory of errors and practical experimentation have developed separately,[2] and it is difficult for the average worker to get coherent advice as the subjects of practical experimentation, theory of errors, design of experiments, the theory of similitude, modelling, and the theory of measurement are all treated as separate topics of study, often using the same terms in different senses or applying different terms to the same operation or concept.

A particularly pervasive and troublesome source of difficulty is the assumption that there is a so called "true" value which can be known separately from an act of measurement—that is, that there are biological parameters. This is due to confusing the statistical model (the normal curve with its parameters of mean and standard deviation) with the real world in which a more or less good estimate of some variable is made by measurement and described by using the parameters as summary statistics.

Dr Corea and others point out that several biological variables vary together—that is, in correlated fashion—and it is this which invalidates in many cases the use of the correlation coefficient to test the goodness of a measurement technique. For example, if one compared methods of assessing bone age of hand bones over the period from birth to say 20 years of age it would be extremely unlikely that a low correlation coefficient would be found because of the large changes with age; thus one might easily assume falsely that all methods were equally good because they show close association.

If it is desired to test a method of measurement it is the discrepancies which are important both practically and philosophically and the correlation coefficient minimises these. It is unfortunate that medical men try to confirm their hypotheses rather than refute them. This is because it is not widely realised that while in general a diagnosis in a patient can be confirmed but not excluded a scientific hypothesis can be refuted but not confirmed.

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[1] Davies P. The errors of linear measurements using ultrasonic B scanners. *Br J Radiol* 1982;**55**:380-1.
[2] Fisher RA. *The design of experiments.* 6th ed, Edinburgh and London: Oliver and Boyd, 1957.

Luxuskonsumption, brown fat, and human obesity

SIR,—Dr G R Hervey and Dr G Tobin (25 June, p 2060) comment on your review article on luxuskonsumption (28 May, p 1684). With their great experience of metabolic work on rats they show that the evidence that it exists in this species is unsatisfactory. Evidence of it being present in *Homo sapiens* is equally unconvincing.

The crux of the hypothesis is stated in the review. "It is also generally agreed that normal people taking a diet with a low energy content show a decrease in resting metabolic rate and that the same occurs with obese patients on a reducing diet. Most workers agree that the converse is true—that after a period of overfeeding the resting fasting metabolic rate increases." The first sentence is correct. For evidence for the second the review refers to an article by Garrow.[1] This contains a table which lists the answers given in 10 papers to the question, "Is resting metabolic rate increased by overfeeding in man?" The answer is yes in nine of them and no in only one. Three of the papers in which the answer is given as yes are by myself and various colleagues. The last of these[2] gives a summary of all our results and shows that our answers should be no. My reading of the other papers quoted is that in most of them the authors' answers are also no and that not one provides sufficient evidence to justify an answer of yes.

The idea that luxuskonsumption is a metabolic

control regulating body weight after dietary excess has been around for 80 years. If it existed it should have been unequivocally demonstrated by now. That such a mechanism should arise and persist through natural selection is unlikely. During the evolution of *Homo sapiens* men and women had to be physically active to secure a limited food supply, and the need to dispose of the surplus dietary intake seldom arose. Not until the twentieth century did large sections of the population have access to a surplus of food, and at the same time mechanisation of agriculture, industry, and transport reduced greatly the need for physical work. Only then did *Homo sedentarius* appear in large numbers and obesity become widely prevalent.[3]

Luxuskonsumption has now ceased to be a tenable hypothesis and has become a myth. Myths arise from attempts by honest and hard thinking people to explain obscure observations. Common and useful, if not essential, in religious literature, they are also well known in science and medicine. The history of nutrition and dietetics has many examples. The view that spinach had special nutritive properties was believed not only by Popeye but by reputable nutritionists in the second world war.[4] In the USA megavitamin treatment continues to be used by the general public on a large scale and by some doctors, despite the fact that controlled clinical trials have shown that it has no effect in those diseases for which it is commonly advocated. Faith in myths is strong. It is neither surprising that they persist nor necessary to be harsh on those who continue to strive to find evidence to support them.

Fortunately, the concept of luxuskonsumption has had no effect on the management of obesity and I agree entirely with the approach to this outlined in the final paragraph of the review. Unfortunately, the concept continues to be put on television and in the popular press, and this does not help those who try to present nutrition as a science.

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[1] Garrow JS. *Energy balance and obesity in man.* 2nd ed. Amsterdam: Elsevier/North Holland, 1978: 92.
[2] Strong JA, Shirling D, Passmore R. Some effects of overfeeding for four days in man. *Br J Nutr* 1967;**21**:909-19.
[3] Passmore R. How many calories? *Lancet* 1964;ii: 853-4.
[4] Hamblin TJ. Fake! *Br Med J* 1981;**283**:1671-3.

Effect of magnesium on blood pressure

SIR,—Dr T Dyckner and Dr P O Wester (11 June, p 1847) examined "the effects of magnesium supplementation on electrolytes in patients receiving long term diuretic treatment for hypertension." The results of their study show that magnesium supplementation (15 mmol/day) did not have an effect on plasma or urinary concentrations of magnesium or other electrolytes. Despite this the authors attempt to explain the appreciable decrease in supine and upright blood pressure (without a change in heart rate) by changes in calcium, potassium, or magnesium.

The major portion of magnesium, given orally, is excreted in faeces without being absorbed. Thus a magnesium dose of 15 mmol/day given by mouth may have been too small to cause any effect. We have previously shown in hypokalaemic patients that oral administration of magnesium chloride in doses as high as 60 mmol/day, which caused substantial increases in both serum magnesium and potassium concentrations and a substantial decrease in urinary potassium excretion, had no appreciable effect on blood pressure or heart rate.

Finally, in view of the absence of an effect of 15 mmol/day magnesium aspartate hydrochloride on plasma and urinary electrolyte concentrations, one might consider the possibility that the decrease in blood pressure noted by Dr Dyckner and Dr Wester was mediated by a placebo effect.

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Electrocardiographic chest wall mapping in the diagnosis of coronary artery disease

SIR,—Of the 150 patients of Dr A M Salmasi and others (2 July, p 9), 41 were classified as having exercise tests that showed coronary artery disease on the basis of pathological Q waves on the electrocardiograph at rest. A standard 12 lead electrocardiogram would have been a far simpler method of detecting coronary disease in these patients. The inclusion of this group is thus inappropriate, and rather than increasing the accuracy of the test, only confuses the evaluation of its role in the clinical assessment of coronary disease.

I was disappointed that ST segment elevation was not mentioned, as this is as accurate a sign of myocardial ischaemia on exercise as is ST segment depression.[1]

The ability of this version of precordial electrocardiogram mapping to identify lesions of individual coronary arteries is exciting. The predictive accuracy of a positive or negative result, however, was only 70% in a population with a high prevalence of coronary disease, suggesting that this test is unlikely to be especially useful if used alone to assess coronary disease in less selected populations.[2]

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[1] Fox KM, Jonathan A, Selwyn A. Significance of exercise induced ST segment elevation in patients with previous myocardial infarction. *Br Heart J* 1983;**49**:15-9.
[2] Epstein SE. Value and limitations of the electrocardiographic response to exercise in the assessment of patients with coronary artery disease. *Am J Cardiol* 1978;**42**:667-74.

The behaviour, development, and health of the young child

SIR,—The paper by Dr Martin Bax and others (4 June, p 1793) is an interesting contribution to the case for developmental paediatric surveillance in general practice. I do not believe, however, that the case has been made for a new sort of specialist, the consultant community paediatrician.

I believe that the tasks are already being performed by general practitioner paediatricians who have made themselves responsible for the child population of the practice in which they work, in many cases unpaid. It is a retrograde step to suggest that this work, which should properly be done within general practice, should be fragmented and the work divided so that sickness care can be carried out by general practitioners and the well care by some other organisation. Thorough surveillance as practised by general practitioner paediatricians in this country has shown the true worth of this work and their competence to carry it out. The real problem for many general practitioner paediatricians working in this field is one of inadequate staffing of the specialist referral agencies. It is difficult to work effectively where there is no speech therapist, where the audiologist has a waiting list extending into months, and where consultant ear, nose, and throat surgeons are satisfied with the results of one hearing test performed on a child's good day and will not see the child again.

The appointment of a new sort of consultant paediatrician will make no difference to this situation. It is, of course, a disgrace that there is no extra money for this work, but the General Medical Services Committee's activities in attempting to persuade the Department of Health and Social Security to fund item of service payments for serial examinations of the well child is not the answer. We believe that it is far more important that sums of money should be available to recompense properly those practices that organise and run successful whole practice surveillance programmes. Only when this is done will the service begin. It is important, however, that adequate training for this work is organised quickly with realistic aims and objectives.

The priorities are simple (*a*) adequate recompense for what is to me a 20% increase in workload compared with my partners' workloads; (*b*) increase in staffing in referral agencies, audiology, speech therapy, orthoptic services, and so on; and (*c*) a realistic general practitioner based training programme.

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Antiemetics and cytotoxic drugs

SIR,—Professor J R Trounce (29 January, p 328) cited our investigation using high doses of intravenous metoclopramide. Additional trials from several centres since our 1981 report[1] have further defined the role of metoclopramide in the control of acute emesis induced by cisplatin and are of importance to doctors considering using this agent. Over the past two years, published studies, using a double blind design with placebo control,[2] open trials,[3–5] and comparison with other antiemetics,[6 7] have confirmed the effectiveness of high doses of intravenous metoclopramide in controlling emesis following cisplatin. Additionally, a double blind study has shown the superiority of high dose intravenous metoclopramide over oral tetrahydrocannabinol in the management of emesis induced by cisplatin.[8]

Professor Trounce also noted the personal communication of P L Amlot and stated that many patients had dystonic reactions following metoclopramide. This anecdotal statement may be misleading unless the antiemetic treatment regimen, the number of patients treated, and their ages are taken into account. Only five acute dystonic reactions were noted among the 177 patients reported by others in the published trials above. All five reactions were promptly controlled with a single injection of 25-50 mg of parenteral diphenhydramine. Among 452 patients initially receiving high doses of intravenous metoclopramide for the control of emesis induced by chemotherapy we noted acute dystonic reactions in 14 (3%), usually consisting of torticollis or trismus. We have reported a