reviewer claims to have read material in the book that in fact does not exist in it. The probability of accepting a null hypothesis is never mentioned. The acceptance of a null hypothesis is a common fallacy (p 63) arising from the misinterpretation of the result of a significance test. In addition, tests in the book are not described as "best," whether related to the t test or χ^2 test.

(4) The reviewer states that a description of what to do with small two by two tables is called for. If by "small" he refers to the fact that the table has only two rows and two columns, then a full description of what to do is provided (pp 91-92). If on the other hand "small" refers to small expected frequencies in the two by two table, then it is stated (p 91) that Fisher's exact test is warranted and a reference is provided for a full discussion of the test. This is generally adequate for the non-specialist, as Fisher's exact test is cumbersome and is rarely performed without the aid of a computer, so that it is not deemed necessary to include details of the method in an elementary text.

(5) Finally, the reviewer states that the comparison between three or more groups is never mentioned. Clearly he failed to read p 118, where precisely this situation is discussed.

Unfortunately the reviewer saw fit to consider only these five points in relation to my book. The remainder of his review, although topical, is not specific to the book.

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*We sent a copy of this letter to the reviewer, whose reply is printed below.—ED, *BMJ*.

SIR,—The basic difference between the author and myself is akin to the difference between physiology and pathology. As a consultant statistician a high proportion of the "cases" I see concern the abuse of statistics. My fundamental criticism of the book is that it is unsuitable for its audience. To answer the author's points serially:

(1) The vital link is omitted. The "excellent table" does not mention the connection between type of variable and type of test, nor do the chapters on estimation and hypothesis testing state anywhere that nominal and ordinal data should not be pumped into calculations whose basic assumption is that the variable is at least continuous without great care. This is a very common mistake and one to which the medical profession, with its use of classifications and rating scales, is especially prone. A book on *medical statistics* should warn the reader, particularly any book written after the publication of one of the standard works on the application of non-parametric test; I agree that pp 87 and 132 give the appropriate uses for these tests, but my point is the other way round.

(2) In my typescript and the proof there was a comma between "briefly" and "once," the omission of which does change the meaning and I sympathise with the author. I did not in fact say that the example was wrong. My point was that saying that small is "less than 20" (p 73) and using sample sizes of 12 and 13 in an example (p 83) "can only confuse the non-specialist." I believe it does. Incidentally, pretesting for equality of variances is still

controversial and is not necessarily to be recommended as routine in a book at this level.

(3) The words "the probability of accepting a false null hypothesis" do not appear in the book, but p 57 contains a too brief discussion of type II error, which is the same thing. I do apologise for substituting "best" for "good," which is the word in the book, but surely my point still stands. The χ^2 test is not a "good" test in the sense used and no warning of its dangers is given. Since the power of the tests to be used is a very important consideration in medical research²⁻⁴ one would have hoped that a book aimed at medical research workers (p ix) would contain more on the subject.

(4) With the advent of pocket calculators with memories Fisher's exact test is very easy to perform³ and in a book published in 1978 I would argue that a description of it is at least desirable, especially for the non-specialist; χ^2 for two by two tables with small frequencies is too conservative with Yates's correction and too unconservative without it.

(5) I overlooked the statement on p 118 because it is in the wrong chapter. It appears under "Linear Regression and Correlation," but surely its proper place is in the chapter dealing with t tests. Again, my point is that the repetitious use of t tests without the proper precautions is a too frequent error by medical writers.

In conclusion, may I add that I did not like writing as critical a review as I thought (and still think) the book deserves. I wrote three separate drafts before committing myself. But the book is still an inappropriate text for its target market.

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¹ Siegel, S, *Nonparametric Statistics for the Behavioral Sciences*. New York, MacGraw-Hill, 1956.

² *British Medical Journal*, 1978, **2**, 1318.

³ Freiman, J A, et al, *New England Journal of Medicine*, 1978, **299**, 690.

⁴ Clarke, I, *British Medical Journal*, 1978, **2**, 1497.

⁵ Swinscow, T D V, *Statistics at Square One*. London, British Medical Association, 1976.

Postanaesthetic oxygen

SIR,—Your leading article (25 November, p 1452) seems to make heavy weather of a situation which appears relatively straightforward. It is first necessary to distinguish the overt causes of postoperative hypoxaemia, which include underventilation and pulmonary shunting. These are largely avoidable by careful attention to anaesthetic technique. However, even with impeccable anaesthetic technique a considerable proportion of patients without cardiorespiratory disease have arterial hypoxaemia during the first few hours after even trivial surgery. Most cases cannot be explained by underventilation¹ and the most likely explanation is the reduction of functional residual capacity shown by Alexander *et al.*²

Although hypoxaemia is more pronounced in older patients, its incidence is unpredictable in the individual patient. In a survey of 169 patients arterial P_{O_2} levels were in the range 5-7 kPa (37-6-52-6 mm Hg) in 14 patients aged 39-70.³ This is not a degree of hypoxaemia which should go untreated when it is so safe and simple to raise the inspired oxygen concentration above about 35%, which gives satisfactory results.⁴ Selection of patients at risk is not really feasible and routine

administration of oxygen in the early post-operative period seems sensible. Simple disposable masks can be used as the precise concentration of oxygen is not important.

In a very small number of patients who have lost their carbon dioxide sensitivity there is a danger of hypoventilation when the hypoxic drive to respiration is withdrawn. Such patients must be identified and carefully monitored in the postoperative period. Venturi masks should be used since precise control of oxygen concentration is mandatory in these patients.

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¹ Nunn, J F, and Payne, J P, *Lancet*, 1962, **1**, 631.

² Alexander, J I, et al, *British Journal of Anaesthesia*, 1973, **45**, 34.

³ Nunn, J F, *Lancet*, 1965, **2**, 466.

⁴ Drummond, G B, and Wright, D J, *British Journal of Anaesthesia*, 1977, **49**, 789.

Diagnosis of breast tumours

SIR,—With reference to the letter from Mr Peter R Simpson and others (18 November, p 1430) we have calculated the cost of screening a woman for breast cancer in our London clinic so far this year at £13.80. This includes, in just over half, x-ray mammography, and the cost takes into account staff, x-ray and other materials, rent, rates, heating, lighting, and a depreciation charge on capital equipment and a contribution towards staff training, appointments, medical secretariat, and administration. This appears cheaper than the University College Hospital clinic, which is surprising, particularly as in our accounting we include overheads which the NHS often does not.

The difference in cost highlights two important points. Firstly, we rely heavily on paramedical staff who are specially trained in specific techniques, and a considerable saving in cost is thereby achieved. Secondly, a screening unit needs to see several thousand patients a year so that the unit cost is reduced (in our central unit we have already screened 5000 patients this year). The unit cost is higher in our mobile units, where the number of women screened is much lower.

A paper on the cost of detecting breast cancer is in preparation.

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Use of the telephone in consultant practice

SIR,—I would like to comment on your leading article (21 October, p 1106) from the standpoint of the consultant. Consultant practice in Hamilton is closer in its habits to the British than the usual North American in that patients are seen only by referral. Initially I was diffident about the advice I received to use the telephone freely for two reasons. Firstly, I thought it would be time-wasting and impractical because of the difficulty of getting hold of the referring doctor and the annoyance my interruption of his work would cause. Secondly, I thought that use of the telephone weakened the letter as a record of the consultation. I soon found that both fears were groundless and it is now rare for me not to

make a telephone call as part of the consultative relationship. The family doctors are rapidly accessible and value the personal contact so highly that they take almost all such calls immediately. They are of course usually in their office practice particularly in the afternoons because both they and most of the consultants have evolved the habit of doing hospital and other such work in the mornings, so that both parties do their "office" work in the afternoons. Moreover, I find that I write a better letter (that is, more useful to the referring doctor) after discussing the problem and its management with him.

The information available on first seeing the patients varies from (alas, all too often) virtually none to a detailed account of the problem on which my opinion is being sought. Often in the case of patients coming from a distance ("out of town") the referring doctor will telephone before referring the patient and this will expedite matters—for example, by leading to our agreement that to save the patient time he had better be admitted rather than try to complete the relevant investigations as an outpatient. As with most specialists the usual consultation requires two visits, one to assess the problem and the second to review the findings on investigation and "wrap up the consultation" with the patient. It is my usual practice to phone the referring doctor after the first visit to discuss what I have found and plan to do and then the definitive letter after the second visit can usually be prepared without a further telephone call. However, very commonly, particularly with complicated problems in which the history or the attitude and understanding of the patient is particularly important, I find it valuable to try to have all three parties "present" and then I will call the referring doctor while the patient is still with me and both he and the referring doctor hear my every word.

A further advantage is in deciding who does what. Shall I hang on to the patient until I resolve the problem (for example, the asthmatic whose drug regimen may take several visits to sort out) or may I see the patient for follow-up and if so at what intervals?

From what I have said it should be clear that this personal contact develops and grows one's relationships with the referring doctors and leads both parties to recognise each other's style. I could go on and on but will close with a final commendation from the standpoint of the teacher. I am sure the resident profits greatly as a trainee consultant by hearing these telephone consultations and even participating in them on a telephone extension. He then writes a better letter on my behalf—and to a doctor who knows that I am responsible.

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Paracetamol poisoning

SIR,—Mr J G Harvey and Dr J B Spooner (16 September, p 832) suggest that "the recorded number of paracetamol deaths considerably exceeds the actual number which can be directly attributed to paracetamol." The following account of 14 such cases reported to coroners in the north-east of England in 1974-6 further supports such view.

During 1974-6 six patients referred to the North-east Regional Paracetamol Overdose Service died in acute hepatic failure

complicated by severe disturbance of the clotting mechanism and a variable degree of renal dysfunction. The paracetamol overdose was in the region of 50 g or over. Indeterminate amounts of other non-hepatotoxic drugs and small quantities of alcohol were also taken. Only one of these patients was admitted early enough for the treatment with cysteamine (8 h after the overdose). Necropsy revealed "massive" hepatic necrosis and acute renal tubular necrosis of varying severity in all six patients. The majority of them also sustained major gastrointestinal bleeding and showed extensive petechial haemorrhage in the gastric and small-intestinal mucosa, with true superficial erosion in two cases. The prothrombin time ratio was severely altered before death and was in a range of 9-21%. In no instance could death be attributed solely to acute renal failure.

Further study of post-mortem protocols, coroners' files, and forensic laboratory records disclosed an additional eight cases of death in the region which had been reported to coroners as due to paracetamol overdose. When all the available data were scrutinised I came to the conclusion that only one of these patients died of pure paracetamol overdose. His death occurred in hospital four days after ingestion of the drug and was due to acute hepatic failure.

Of these eight cases of "sudden death" reported to coroners, one occurred within a few hours of alleged paracetamol overdose but on post-mortem examination it was shown to have been due to aspiration of gastric contents; a high blood alcohol level was detected. Six patients were found dead at home and empty bottles with missing known amounts of tablets were discovered by relatives or police by their bedside. Two of these patients had ingested large amounts of Distalgesic (dextropropoxyphene and paracetamol), one had swallowed a large, toxic dose of Lobak (chlormezanone and paracetamol), and in the remaining three cases only therapeutic blood levels of paracetamol were detected which could not have contributed to death. The findings in all 14 apparent cases of fatal paracetamol self-poisoning are summarised in the table.

Role of paracetamol as a cause of death

Drugs ingested	Interval between overdose and death	No of patients
Paracetamol alone	4-5 days	2
Paracetamol + small amounts of alcohol and non-hepatotoxic drugs	3-6 days	5
Paracetamol + large amount of alcohol	?	1
Distalgesic, Lobak	(found dead)	3
	(found dead)	3
Unrelated mode of death + paracetamol in therapeutic dose (found dead)	?	3

Since the first report of two fatal cases of paracetamol overdose in 1966 by Davidson and Eastham¹ it has become widely accepted that a large overdose of paracetamol may cause subtotal hepatic necrosis leading to death in acute hepatic failure at least several days after the overdose. In Clark's series² 12 deaths after paracetamol overdose occurred 4-18 days after ingestion of the tablets. Only one case of fatal renal tubular necrosis in the absence of hepatic failure was recorded by Prescott³ in 1971. Since paracetamol is a common ingredient of many analgesics which may have a depressive effect on the central nervous

system (for example, Distalgesic), if an overdose of any of these is taken death may occur after several hours rather than days and may be erroneously attributed to paracetamol rather than to the other ingredient.

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- ¹ Davidson, D G D, and Eastham, W N, *British Medical Journal*, 1966, **2**, 497.
² Clark, R, *et al*, *Lancet*, 1973, **1**, 66.
³ Prescott, L F, *et al*, *Lancet*, 1971, **1**, 519.

Changing social-class distribution of heart disease

SIR,—In his letter (18 November, p 1431) Professor T R E Pilkington questions the explanation by Dr M G Marmot and others (21 October, p 1109) of the excessive rise in coronary heart disease after 1951 among men in social classes IV and V. He says that a more likely explanation is to be found in that "a large number of classes IV and V in the younger age groups can be expected to be immigrants from overseas and Ireland."

Although the proportion of immigrants is higher among men in social classes IV and V at ages 35-44 (18% as opposed to 13% for all men), mortality rates are similar to those of men born in England and Wales. As the table shows, exclusion of immigrants—that is, persons born outside England and Wales—does not make a difference.

Hypertensive disease and ischaemic heart disease (ICD 400-414): deaths/million/year in men aged 35-44

Social class	All countries of birth	Excluding those born outside England and Wales
I and II	525	513
IV and V	877	884
Ratio I and II IV and V	0.598	0.58

Mortality of immigrants by social class is discussed in the latest decennial supplement.¹

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¹ Office of Population Censuses and Surveys, *Occupational Mortality 1970-72*, Series DS No 1, pp 184-8, London, HMSO, 1978.

Selection of patients for dialysis and transplantation

SIR,—With reference to your leading article (25 November, p 1449) I too have difficulty in reconciling the epidemiological aspects of terminal renal failure—40 patients per million per annum—with the fact that only 16 patients per million per annum receive treatment in Britain.

There seem to be two possible explanations: either the epidemiology is incorrect or criteria for exclusion from dialysis are too harsh. The prevalence of chronic renal failure in the British population is not dissimilar to that in Europe, so presumably the epidemiological