oestrogens to be statistically significantly better than placebo at relieving hot flushes. Sound and well-argued criticism of any scientific work is valid but the gross misrepresentation of our studies by Dr Mulley and Professor Mitchell is inexcusable.

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Campylobacter enteritis

SIR,—We have followed with great interest the correspondence on campylobacter enteritis since the original article by Dr M B Skirrow (2 July, p 9). During a three-month period, from the end of June 1977, we have examined 563 faecal samples for potential intestinal pathogens. Of these, 280 were from patients with recent acute gastrointestinal symptoms, 117 from contacts of these cases, and 156 from asymptomatic patients. The table below summarises our results.

Campylobacters of the *C jejuni-C coli* group were isolated on the selective blood agar medium incubated at 43°C under microaerophilic conditions as described by Dr Skirrow. Our isolation rate of 14% in patients with symptoms is higher than previously reported in your columns. We found that 38% of our patients with campylobacters were children under 10 years of age; five patients had another intestinal pathogen isolated in association with the campylobacter. In addition, over the same period examination of 63 chicken carcases from a common source yielded 39 (62%) positives for the *C jejuni-C coli* group by direct culture technique; enrichment methods were not employed. Similarly, 167 caecal samples from apparently previously healthy poultry gave 114 (68%) positive isolates. Antibiotic disc diffusion tests of the human and avian strains were identical for aminoglycosides and erythromycin (all sensitive), and trimethoprim and cephaloridine (all resistant); but they differed in the case of ampicillin (25 μg disc). Here we consistently found that the avian strains were resistant, while 29 of 33 human strains were sensitive.

The epidemiology of campylobacter enteritis remains uncertain, although chickens have been considered as a primary source, and indeed serum from one of our patients with symptoms agglutinated both human and avian strains, thus suggesting a common antigen. Our results show poultry are a reservoir for campylobacter, but until a reliable scheme of strain identification is developed the place of avian strains in the epidemiology of human campylobacter enteritis may not be easily established. Our high isolation rate may be explained by the fact that Herefordshire is predominantly a rural area and people may more frequently be exposed to campylobacters from a variety of potential animal reservoirs, or indeed from the environment.

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Potential pathogen isolated	No (%) positive samples from		
	280 patients with symptoms	117 contact patients	156 asymptomatic patients
Campylobacter sp	39 (13·9)	2 (1·7)	1 (0·6)
Salmonella sp	12 (4·3)	2 (1·7)	0
Shigella sonnei	11 (3·9)	14 (12·0)	1 (0·6)
Enteropathogenic *E coli*	5 (1·8)	0	2 (1·3)
Parasites	8 (2·9)	0	0

SIR,—While campylobacters are increasingly reported as a cause of gastroenteritis the epidemiology remains unclear, though the finger of suspicion is pointed regularly at chickens and other birds as sources of the organisms.[1 2] We investigated a series of cases of gastroenteritis due to campylobacter occurring after a wedding reception. So far as we are aware this is the first report of a common-source outbreak.

Of 29 people from different parts of the country who attended a wedding breakfast, five fell ill with colicky abdominal pain and profuse watery diarrhoea. Three of them were also pyrexial and had rigors. The diarrhoea persisted for about three days. The incubation periods ranged from 48 to 84 h, with a mean of 62 h. A campylobacter was isolated from one patient at the height of her illness, but by the time the extent of the outbreak had become apparent all other patients had fully recovered and the organism was not isolated from their stools. Blood taken one month after the illness from four of those affected, including the patient from whom a campylobacter had been isolated, was tested for agglutinating antibodies. The index case showed a titre of 1/320, as did one of the others, while the other two had titres of more than 1/640, suggesting that these patients had all been infected by the same organism.

The wedding breakfast consisted of several types of cold cooked meats, salmon, salad, cheesecake, fruit, and sweets. The meats were chicken, ham, beef, tongue, and Scotch eggs. No food was available for examination and no single dish was common to all sufferers. The chicken was supplied as uncooked portions from a large wholesaler on the day before the reception, refrigerated overnight, cooked early the following morning, and allowed to cool before serving. It had not been deep-frozen. A similar pack of chicken from the same manufacturer received a month later was opened on arrival at the restaurant and a portion taken for culture. A campylobacter was isolated from the chicken skin but not from the meat. Agglutinins to this organism were not detected in the serum of our patients, but it is likely that there is wide distribution of serotypes in the chicken population[3] and the human sera were therefore tested against four other isolates of campylobacter obtained from local chickens. With two of the chick isolates titres were just as high as against the human strains; with the others no cross-reaction was demonstrable. The evidence for the involvement of chickens in the outbreak, though presumptive, is strong.

Cooking should kill these organisms and it is unlikely that the chicken was itself the sole final vehicle for infection in this outbreak, as one of those taken ill had not eaten any. Before being cooked the chicken pieces had been skinned on a working surface upon which other cooked meats were later prepared. It therefore seems likely that in this case cross-contamination had occurred between uncooked and cooked food and several different dishes may have finally become contaminated.

Henceforth campylobacter must be considered as a possible cause of food poisoning.

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SI units and acidity

SIR,—I hope that many correspondents have already suggested the most obvious solution of the problem described by Dr P J Tomlin (24 September, p 833)—the discrepancies that are found when the same laboratory measurement is expressed in different units and then the mean and standard deviation are used in determining clinically "normal" values. The appropriate method of presenting the measurements is the old but often neglected percentile technique, which "is a simple way of obtaining understandable information from any sample of measurements or counts."[1] It requires only the arrangement of the observed values in rank order, which would be the same whether the SI or the "old" units had been used, and it does not depend on any information or assumption regarding the shape (Gaussian or other) of the frequency distribution.

Readers who are not familiar with the applications and potentialities of the method could find much discussion in the textbook just cited and in three more recent publications[2-4] which discuss clinical norms. Those who find it difficult to transfer their allegiance from the mean and standard deviation to the median and percentiles may be helped by knowing how the mean-SD allegiance probably came about. Dr Feinstein[2] suggests that it arose because the mean and variance (or its square root, SD) are fundamental to the *t*-test and related tests of "statistical significance." Having been involved in medical statistics since 1928 and at one time a worshipper of *t* and its relatives, I agree with Dr Feinstein's diagnosis. Indeed, I think it is possible to detect a time of change in the writings of Sir Ronald Fisher. An article[5] that was published in 1921 discussed the variation among individual measurements without invoking Gaussian-curve assumptions, but in 1925 there appeared the first edition of the book[6] that was the starting-point for the wide

dissemination of Gaussian-based "significance" tests.

If or when the percentile method of expressing interindividual differences has been widely adopted, there will still remain two much more difficult problems: (*a*) the collection, from healthy and diseased subjects, of data that are reliable and are applicable to the particular patient under investigation; and (*b*) the question of where to make the cut-off points between "normal" and "abnormal"—what risks of error (false-positives and false-negatives) are acceptable. For the balancing of these risks—often a different balance with respect to different disorders, different consequences of error, and even different patients—we must look to clinicians, not to biochemists or other laboratory workers, or to statisticians as such.

It would be interesting if the SI units compensated somewhat for their adverse effects by promoting the use of a sensible method of expressing intermeasurement variation and by allowing us to focus attention on how to collect data and apply them to individual patients.

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Surgical treatment of coronary disease

SIR,—The Veterans Administration co-operative study of the surgical treatment of coronary artery disease represents probably the most valuable contribution to an understanding of the role of surgery that is available at present. Minerva (15 October, p 1032) therefore does a grave disservice in misquoting the conclusions of a recent interim report from that study.[1] This showed no difference in mortality between the medically and surgically treated groups over a three-year period. However, the prime indication for surgery is angina pectoris and the prime objective is the relief of this symptom. By reporting the study as showing that "patients treated by bypass for chronic stable angina had done no better than those given beta blockers" Minerva implies that pain relief was not achieved. In fact, this aspect was not included in the report, although elsewhere in the same journal Braunwald states that coronary artery bypass grafting reduces the incidence and severity of angina.[2]

The failure to detect a difference in survival between the two groups may reflect a true failure of surgery to improve prognosis, but in view of the high graft occlusion rate (12% of the patients had no patent grafts at restudy) it could also point to the need to develop techniques whereby graft occlusion can be minimised. No analysis was made of the relation between graft patency and death, and since 56% of deaths in the surgical group occurred in the first 30 days the status of the grafts in most of the surgical deaths is not known.

The Veterans Administration study surely makes three points abundantly clear. Firstly, the prognosis is related to the distribution of the coronary artery lesions, which can be assessed only by angiography.[3] Secondly, many problems remain to be solved in relation to the intraoperative and postoperative management of patients treated surgically to improve graft patency. Thirdly, the relation between graft patency and survival is not yet known. This in no way detracts from the fundamental point, which is that coronary bypass surgery is an effective method of alleviating angina pectoris and at the very least does not diminish longevity. Surely the relief of an often incapacitating symptom is valuable in itself.

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Hyperglycaemia and complications of diabetes

SIR,—One of us (RR) has recently found that maintenance of a normal blood glucose concentration in streptozotocin-diabetic rats prevents the development of basement membrane thickening in the renal glomeruli.[1]

Dr C J Fox and others (3 September, p 605) studied "the effects of diabetic control on basement membrane thickening in rats" and concluded that there is a "strong relation between basement membrane thickening and hyperglycaemia." They add that "these findings should offer encouragement not only to those who treat diabetic patients with the aim of maintaining good blood glucose control but also to those who search for improved methods of achieving this aim."

Unfortunately, however, the above conclusion cannot be drawn from their results. It is based on a regression analysis including the non-diabetic rats, a procedure which, of course, is quite unacceptable. The lack of relation between hyperglycaemia and basement membrane thickness is revealed by the values obtained in the four groups of diabetic rats. It appears that there were no consistent differences in basement membrane thickness between diabetic groups with average plasma glucose levels of 9·8, 19·8, 21·5, and 27·4 mmol/l (176, 356, 387, and 493 mg/100 ml) respectively.

There are other unacceptable features in their paper, but it is not necessary to discuss them here.

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**The authors sent a copy of this letter to Dr Fox and his colleagues, whose reply is printed below.—ED, *BMJ*.

SIR,—Measurements of hyperglycaemia and basement membrane thickness, like most biological data (for example, blood pressure and serum cholesterol), form a continuum from normal through slightly deranged to grossly abnormal. To establish the relationship between these two variables it is necessary to look at both ends of the spectrum, and of course data from non-diabetics have to be included in the analysis.

If streptozotocin per se were to affect the basement membrane, then results from diabetic rats could not be grouped together with those from non-diabetic controls. That this is not the case has been shown by Dr Rasch herself. Thus in the paper quoted above streptozotocin-diabetic rats carefully controlled on insulin for six months have no increase in glomerular basement membrane thickness over normal controls.

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Control of hyperglycaemia in young diabetics

SIR,—Recent reports[1-3] have indicated that good control of hyperglycaemia may decrease the microvascular complications of diabetes mellitus.

At a recent summer camp for adolescent diabetics an opportunity arose to determine the views of the children on the control of their diabetes. Eighteen diabetics (mean age 15½ years) from most areas of England attended the camp; all were insulin-dependent and the mean duration of disease was 8 years. Only nine were being treated with a twice-daily regimen of soluble insulin together with an intermediate-action insulin, a regimen which many diabetologists would consider essential for good control of hyperglycaemia. Three of the group were being treated with a morning injection of Monotard insulin ranging from 20 to 80 U/day.

Ten of the patients performed urine tests on two or more occasions each day, but six children tested only once each day or less; the remaining two patients had low renal thresholds and so did not rely on urine testing. Only two of the group accepted urine tests showing no sugar as good control and four patients were satisfied with mainly negative reactions with occasional ¼% sugar, while 10 of the children preferred persistent glycosuria in their urine tests, five maintaining urine tests showing ½-¾% sugar. One of the patients with low renal threshold used Dextrostix and maintained her blood sugar between 10 and 13 mmol/l (180 and 240 mg/100 ml); the other patient did not use Dextrostix and did not test her urine.

Only seven of the group said that they would be happy if all their urine tests showed no sugar. Six patients would decrease their insulin dosage in this situation and a further three would take more carbohydrate exchanges. It is distressing that seven of the children (excluding the two with low renal threshold) felt that it was impossible to have continuous negative urine tests without frequent hypoglycaemic attacks. Despite the above findings 17 out of the 18 patients felt that good control was important for their future wellbeing and it was encouraging that seven of the group felt that the amount of animal fat in their diet might be of importance, six of