Since you have chosen to reject this paper even after amendment and since you cannot publish my raw data I am wondering if you will extend to me the courtesy of your columns to ask for the "evidence" on which the committee's report and your leading article are based. Meanwhile, for anyone who wants it, my data are on file in the Communicable Diseases (Scotland) Unit at Ruchill Hospital. Our article will be submitted for information and publication in other quarters.

There is a further point of immediate importance. Because of continuing doubts and lack of "evidence" about the safety and efficacy of immunisation against whooping cough, immunisation of children is declining sharply. The decline is most marked in districts where, according to our "evidence," the risk of contracting infection is highest. Unfortunately, in parallel with diminishing use of pertussis vaccine there is a fall in other forms of immunisation in childhood, possibly because unanswered questions about pertussis vaccine are discouraging routine immunisations against diphtheria, poliomyelitis, and tetanus. For this reason also I trust that the "evidence" on which you clearly rely will not be unduly delayed in finding its way into your columns.

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The problem of rosacea

SIR,—It is true that, as reported in your leading article (15 November, p 366) I "took on the task of critically re-examining the main ideas in the light of [my] experience of 92 patients with rosacea; none could be supported." However, while agreeing that "there are still vacancies in the cowshed," I would point out that you made no reference to a suggestion that I have made previously as to a potential new occupant.

We have pointed out¹ that the upper dermis in rosacea is quite abnormal and shows evidence of both solar elastotic degeneration considerably in advance of what might reasonably be expected for a group of middle-aged Britishers and other dystrophic changes that are not easily categorised. Autoradiographs after injection of tritiated thymidine and enzyme histochemical tests have suggested² that small dermal blood vessels are also involved in rosacea (probably secondarily). It is my belief, based on these findings, that the primary disorder is a dermal dystrophy resulting from "weathering" (sun, wind, and cold) and an inherent

susceptibility to this process. The dermal attenuation produced in this way causes lack of dermal support for the subpapillary venous plexus, allowing these channels (and neighbouring lymphatics) to dilate enormously. The flushing seen in rosacea is in all probability the result of the vessel dilatation—not its cause. The dilated vessels could become incompetent in addition as a result of the persistent and extreme pooling seen in them and this in turn may lead to diffusion of injurious macromolecules and mediators of the inflammatory process into the dermis. This hypothetical process may be summarised in the diagram.

I would not wish the above suggested "occupant of the cowshed" to become a sacred cow. However, there is sufficient evidence for this hypothesis to be further tested and I hope that this letter will stimulate the cow's dissection rather than its perpetual milking.

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¹ Marks, R, *et al*, *Archives of Dermatology*, 1969, **100**, 683.

² Marks, R, *Proceedings of the Royal Society of Medicine*, 1973, **66**, 742.

Plasma free fatty acids and ventricular fibrillation

SIR,—Dr C A Sykes and his colleagues (27 December, p 735) report the relationship between plasma free fatty acid (FFA) concentrations in 23 diabetic and 24 non-diabetic patients with myocardial infarction and the incidence of ventricular fibrillation (VF). They showed that the plasma FFA levels were similar in diabetics who developed VF and in those who did not. But they are wrong in reaching the same conclusion for non-diabetics, as the following reassembly of their own data indicates.

Patients	Mean plasma FFA (mmol/l) $\pm$ 1 SD	Significance of difference
Non-diabetics with VF (5)	1.76 $\pm$ 0.61	P < 0.05
Non-diabetics without VF (19)	1.24 $\pm$ 0.40	
Diabetics with VF (5)	1.30 $\pm$ 0.59	Not significant
Diabetics without VF (18)	1.46 $\pm$ 0.62	

The highest mean plasma FFA is in the subgroup of non-diabetics who developed VF and the concentrations considerably exceed the "threshold level" of about 1.2 mmol/l, which approximates to a 2:1 molar binding ratio with albumin. Higher concentrations

are associated with less firmly bound FFA and, as we showed¹ and originally proposed,² with a greater incidence of serious ventricular arrhythmias. Therefore I regard the findings of Dr Sykes and his colleagues as contradicting one of their own conclusions and, at the same time, as additional supportive evidence to the other studies³⁻⁵ which have shown a positive correlation in non-diabetic patients with myocardial infarction between elevated plasma FFA or glycerol levels and ventricular arrhythmias.

The authors appear to be justified, however, in reaching their other conclusion that plasma FFA levels do not correlate with VF in diabetic patients. Why should this be? One explanation might be that the mean plasma FFA concentrations were not very greatly elevated in these particular patients at a mean level of 1.30 mmol/l. Another might be that the ischaemic myocardium is partly protected from the adverse effect of high plasma FFA because of the relatively high intracellular glucose concentrations likely to be present, particularly in those receiving insulin. The same authors⁶ have previously shown that VF is less common in insulin-treated diabetics compared with those on oral drugs but, unfortunately, it is not possible to determine from the recent report whether the five diabetics who developed VF were on insulin or oral drugs.

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¹ Oliver, M F, Kurién, V A, and Greenwood, T W, *Lancet*, 1968, **1**, 710.

² Kurién, V A, and Oliver, M F, *Lancet*, 1970, **1**, 813.

³ Gupta, D K, *et al*, *Lancet*, 1969, **2**, 1209.

⁴ Carlström, S, and Christensson, B, *British Heart Journal*, 1971, **33**, 884.

⁵ Carlström, S, and Gustafson, A, *British Heart Journal*, 1974, **36**, 996.

⁶ Soler, N G, *et al*, *Lancet*, 1974, **1**, 475.

Dietary fibre: search for the facts

SIR,—A letter from Mr C L Copeland, executive director of the Flour Advisory Bureau (15 November, p 404), was headed "Dietary fibre: search for the facts." Perhaps, as the reporter/producer concerned with the "recent BBC TV programme concerning food," I may be allowed to correct his facts.

Mr Copeland said that "once again, in a recent BBC TV programme concerning food, a commentator has had no hesitation in linking a list of serious diseases, including cancer of the colon, 'with all that lost fibre,' singling out white bread critically and claiming that some consumers *have* to obtain their fibre from a certain (named) proprietary pharmaceutical product." I am sure your readers will be able to make up their minds as to the correctness of Mr Copeland's synopsis if I quote what I did say.

The context of the programme was a comparison between 1875 (the date of the first effective food and drugs Act) and 1975. I said "compared to a century ago, the amount of roughage we get from bread has dropped dramatically, down to only one-sixth. That's the roughage that comes from the fibre of the wheat grain." Then came a sequence illustrating the baking of traditional white bread and comparing its water content with that of a modern plant-baked loaf.

I then said: "and what about all that lost fibre? There is a growing medical theory—

much challenged by the food manufacturers—that the loss of fibre is connected with some modern troubles of Western man. Like diverticular disease, which strikes at least one in four of us by the time we reach 70. And then there is appendicitis, cancer of the colon, obesity, piles, and constipation. So these days some of us have to take our fibre as medicine—like this, made by Reckitt and Colman, one of the biggest food and drug companies in Britain. Fybrogel selling well, overcoming some of the deficiencies associated with modern refined diets—that is what it says on the pack here.” The whole of the sequence was shot around an old-style privy with me (the reporter) poking my head out of the door and reading from the instructions on the packets. Yes, it was intended to “take the mickey” out of one of the “nonsenses” of modern society—taking something out of a natural food and replacing it with more expensive medicine.

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Treatment of meningitis and encephalitis

SIR,—Drs R J Fallon and J H Lawson (6 December, p 577) state that “intramuscular injection both in children and adults is perfectly adequate” in the treatment of meningococcal meningitis with penicillin. Indeed, they state that they “do not feel that there is any justification for intravenous therapy except on the grounds of causing less discomfort to the patient.”

Consider a typical 10-kg infant with meningococcal meningitis which one wishes to treat with a penicillin. Benzylpenicillin 400 000 U/kg daily will entail a total injection volume of 6-8 ml per day. Alternatively, ampicillin in a dose of 200 mg/kg daily requires a 6-ml injection volume per day (ampicillin 500 mg dissolved in 1.5 ml sterile water, given six-hourly). In addition, the infant may require hydrocortisone plus anti-convulsants.

An intravenous infusion is a wise “safety-line” in any ill child with bacterial meningitis. The administration of large intramuscular injection volumes to small infants requires frequent rotation of injection sites, occasionally produces sterile abscesses, and can cause considerable discomfort to the infant and to his attending nurses.

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Smoking in the elderly

SIR,—Your leading article (13 December, p 607) rightly stresses the difficulties in advising old people in the matter of smoking. The general feeling is one of laissez-faire in those who have survived well beyond the induction phase of bronchial cancer and are coping with their arterial degeneration. Nevertheless, there are three other reasons which I would advance in advising against smoking in older age groups.

There are many instances where the expense of the habit is the reason for de-

privation of other needs, such as food, clothing, and the provision of heat in winter.

Socially, many old people are unattractive to their children and grandchildren but remain unaware that this is due to the poor oral hygiene resulting from smoking. They are sometimes relieved to be told by the family doctor that this is a reason for refusing intimate contact with them and not a general rejection on account of age.

Finally, smokers in general are insufficiently aware of the effects of carbon monoxide contained in the inhalant. The anoxia produced by a small concentration of carboxyhaemoglobin can be equated to that produced by a diminution in barometric pressure. Susceptibility is increased in the presence of anaemia and when the lean body mass is reduced, as in old age. It is a habit which encourages several of the symptoms commonly attributed to age itself, such as weakness, syncope, and cerebral symptoms when increased activity is called for. I have encountered toxic amblyopia, spasticity, and incontinence of a reversible type when heavy smokers have been persuaded to stop smoking, and dysrhythmias on the ECG returning to normal.

Thus abandoning the habit is, as you point out, unlikely to increase longevity very much. It will, however, nearly always improve performance during the years of retirement and, presumably, reduce dependency.

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Free erythrocyte protoporphyrin

SIR,—We write with reference to the preliminary communication by Dr G M London and others (8 November, p 324) regarding free erythrocyte protoporphyrin levels and nerve conduction velocity in end-stage renal disease.

In the last paragraph of this paper there appears to be an inherent contradiction to which we wish to draw attention. While stating that “the free erythrocytic protoporphyrin level mainly reflects iron status” and that “iron deficiency . . . is common in dialysed patients,” the authors then go on to assert that there was an “absence of any real evidence of iron deficiency.” Their logic is difficult to follow and we believe that their discussion is confusing.

We have found comparable increases in erythrocyte protoporphyrin concentrations (that is, twice normal) in patients on maintenance haemodialysis, but the concentrations in our patients decreased to normal during intermittent iron therapy given parenterally over a period of nine months.¹ We would argue from this that an increase in erythrocyte protoporphyrin in patients on dialysis does indicate the presence of an iron deficiency component in their anaemia. The reason the erythrocyte protoporphyrin is not 10-20 times normal, as in non-renal iron deficiency anaemia, is possibly the lack in renal disease of an accompanying marrow stimulation commensurate with the degree of the anaemia.

However, we would not argue from this that there is any causal relationship between iron deficiency and changes in nerve conduction velocity. The biochemical picture in renal failure is a complex multiplicity of

increases and decreases in many disparate electrolytes and metabolites. In such circumstances it is not valid to argue either a direct relationship or a dependence on a common factor in any two without at least demonstrating that one varies specifically with the other under a common stimulus. A correlation of 0.5, as reported by Dr London and his colleagues, is not, after all, very convincing on its own.

Having ruled out iron deficiency in their patients (mistakenly, we believe) all the authors appear to be saying is that “the toxicity of uraemia” affects both protoporphyrin metabolism and nerve conduction velocity, which adds little to what is already known. To postulate defects in haem-synthetase activity and to suggest that this might be an underlying “common factor” hardly seems justified from their results.

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¹ Fleming, L W, Stewart, W K, and McConnell, J B, in abstracts of Proceedings of Scottish Society of Experimental Medicine, *Scottish Medical Journal*. In press.

Role of pelvimetry in active management of labour

SIR,—I thought the article by Mr D N Joyce and others (29 November, p 505) to be well conceived and showed well, to their surprise but not to mine, a clear correlation for both cephalic and breech presentation between obstetric conjugate and the size of the fetus that would deliver vaginally. What I could not understand, however, was their deduction from these findings that “antenatal pelvimetry is rarely justified except in breech presentation,” although they accept that the radiation risk to the fetus is only 1 in 30 000.

It is indeed true that the active management of labour has eliminated many of the problems associated with the prolonged labour that frequently occurs in the primigravida when the head is still above the brim at the onset of labour, and the better monitoring of labour and pain relief for the mother has made it possible to make correct decisions without the assistance of pelvimetry. This does not mean, however, because augmented labour delivers the baby in a reasonable time, that the pelvis cannot be contracted, and I think that cephalopelvic disproportion is one of the common causes of incoordinated uterine action. I would agree that such a labour should be augmented, but the conduct of that labour will be much safer for the fetus if we know accurately what difficulties it will have to negotiate. If the sacrum is flat and the outlet, as well as the inlet, small, too prolonged an augmentation of labour may result in a fetal head jammed in a pelvis that may make for difficulty in delivery upwards as well as downwards. In such cases we will be well advised not to indulge in fetal brinkmanship and delay caesarean section until there is evidence of fetal distress. If, however, the only narrowing is at the brim, with ample space below or no narrowing at all, we shall augment the labour with a lighter heart.

It is suggested by Mr Joyce and his