and refer patients likely to have problems, whom they are trained to recognise, to health centres or hospitals. The hospital and health centre staff could not cope with the delivery of all women even if all women were willing to come to them.

In Sri Lanka I accompanied a group of "modern" postgraduates to visit a centre of the self help Sarvodaya movement. A doctor and a health inspector in the group had acute "traveller's tummy." An ayurvedic practitioner working with Sarvodaya gave them a medicine which gave a more rapid and lasting cure which was free from side effects than I have seen from any allopathic remedy.

Swapping anecdotes is no substitute for scientific study of the different systems, their theories, and their results. Such study can be facilitated by mutual respect. Respect does not mean approval of each other's practices or acceptance of each other's theories, but it does imply a willingness to try to understand these.

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SIR,—I agree with Dr Nadaraja Bathirunathan (20 November, p 1498) that many traditional medicine men are ignorant, greedy, and harmful, but not all are like this. Some have an excellent and most reassuring bedside manner so often pathetically lacking in our orthodox specimens. Some are excellent at massage. Some actually have excellent methods of treatment. During my time in Ghana between 1929 and 1936 I worked with some witch doctors in Accra to whom I sent many cases of tetanus neonatorum; they managed to cure many of these cases which I could not. Some of them even began to show me what materials they were using. Unfortunately, I could not persuade any of the money sources to take an interest in research associated with child health. I now think that one of the drugs used might have been something like rauwolfia. The text of a lecture on witch doctors which I gave at the Johns Hopkins Hospital in 1968 gives a more detailed account of my experiences.[1] I believe that with more regular contact and with regular collaboration with traditional medicine men we would benefit from their knowledge and we would recruit some able and genuinely interested medical assistants.

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Does low dietary and serum linoleic acid predispose to myocardial infarction?

SIR,—This question has been of interest for many years especially since the cross sectional Edinburgh-Stockholm Study[1] showed significantly less linoleic acid in adipose tissue, serum cholesterol esters, and triglycerides in the city with a high rate of coronary heart disease (Edinburgh) compared with the findings in Stockholm, where the rate of coronary heart disease is much lower in men aged 40. An earlier comparative study also indicated that adipose tissue linoleic acid is lower in patients with coronary heart disease than in controls.[2] Supportive evidence comes from a recent paper by Dr H C R Simpson and others (11 September, p 683) although the low linoleic acid found in that small study was primarily in the plasma triglyceride component and this might have been influenced by recent dietary consumption. Furthermore, the Oxford study might have been affected by other confounding factors—for example, in the Edinburgh-Stockholm Study[1] the percentage of linoleic acid in adipose tissue was significantly negatively correlated with cigarette smoking ($-0{\cdot}29$) and alcohol ($-0{\cdot}30$).

In a recent survey of a random sample of 448 men (aged 45-54) we found that in 28 men, in whom coronary heart disease was newly diagnosed, adipose tissue linoleic acid and its elongation product, dihomo-γ-linolenic acid, were lower than in 336 control subjects with no coronary heart disease (see table).[3] Preliminary analysis suggests that this difference remains significant when age, social class, cigarette smoking, alcohol consumption, blood pressure, high density lipoprotein cholesterol, and total cholesterol concentrations are each taken into account separately. We did not, however, find significant differences in the linoleic acid content of platelet phospholipids between the two groups.

An important Finnish study by Dr T A Miettinen and others (9 October, p 993) has shown prospectively that low serum phospholipid linoleic acid is an independent risk factor for coronary heart disease. In the Finnish study it was only the phospholipid fraction in which a significantly low linoleic acid was negatively correlated with coronary heart disease. To advance our understanding of this finding it would be helpful to know whether the fatty acid composition of individual phospholipid classes was also measured? Phosphatidyl-inositol, -ethanolamine, and -serine have all been incriminated in thrombogenesis, and it might be through this mechanism that the correlation operates. Additionally, was there an increase in the relative proportions of sphingomyelin in their patients with coronary heart disease? This has been observed earlier in patients with aortic atherosclerosis[4] and might have caused a false correlation between the low percentage of linoleic acid in total phospholipids and coronary heart disease because of the low content of linoleic acid in sphingomyelin.

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Does control of risk factors prevent coronary heart disease?

SIR,—Predictably, Dr R W D Turner (27 November, p 1573), as a leading evangelist for the prevention of coronary heart disease by controlling risk factors, finds the indecisive results of the Multiple Risk Factor Intervention Trial[1] disconcerting.

He emphasises that the important components of a really prudent diet are an increase in polyunsaturated fats and a greater reduction of dietary cholesterol than that obtained by reducing saturated fat of dairy and meat origin. One inconvenient aspect of the Multiple Risk Factor Intervention Trial, however, is that it was exactly such a diet that was recommended and apparently implemented. The polyunsaturated:saturated fat ratio of the participants in the special intervention group in the Multiple Risk Factor Intervention Trial was increased from 0·46 at baseline to 1·25, and the cholesterol intake was reduced to 250 mg day or less: but these changes produced a reduction in plasma cholesterol of only 0·31 mmol/l (12·1 mg/100 ml) (5%) and no change in high density lipoprotein cholesterol. This change would not be expected to lead to much reduction in incidence of coronary heart disease, and, in the event, did not. The difference in plasma cholesterol between the intervention and control groups was even less—0·11 mmol/l (4·2 mg/100 ml) (2%). It will be interesting to learn in due course whether there were any dietary changes in the control group.

Dr Turner believes that the high mortality from coronary heart disease in the UK is due to our failure to switch to unsaturated fats of vegetable origin, and he dismisses the usefulness of a reduction of saturated fat by saying that it "has little effect on blood cholesterol concentrations and was long ago found to be unacceptable and ineffective." In fact, its effectiveness against coronary heart disease has never been formally tested, but that is by the way. He cites the recent World Health Organisation report[2] in support of his views, but the first major dietary recommendation in this report is: "A reduction in saturated fat and dietary cholesterol, which together are the primary factors that raise plasma cholesterol; this can be assisted by replacing some of the saturated fat by monounsaturated and polyunsaturated fat." It cuts no ice whatever to state that: "Safety [of a really prudent diet] has been ensured by use in tens if not hundreds of millions of people in many different countries for periods of up to 25 years," since records are not available to identify either the extent to which this diet has been used or whether it has been beneficial or harmful.

Fatty acids (mean % ± SEM) in adipose tissue and platelets in patients with coronary heart disease and in controls

	Adipose tissue		Platelets	
	Patients (n = 28)	Controls (n = 336)	Patients (n = 28)	Controls (n = 336)
18-2n6 linoleic	7·89 ± 0·3*	8·9 ± 0·1	4·91 ± 0·18	5·06 ± 0·05
20:3n6 dihomo-γ-linolenic	0·08 ± 0·008*	0·11 ± 0·002	1·40 ± 0·05	1·46 ± 0·02
20-4n6 arachidonic	0·5 ± 0·05	0·6 ± 0·01	27·8 ± 0·35	28·0 ± 0·08

*p < 0·01. Two-sided Wilcoxon rank sum test.