can be considered valid in the case of aged people, so that the results of our studies cannot be read from the literature but from our series alone.

We did not learn from Drs Jowsey and Riggs's letter whether their studies—which in general are based on a very small number of patients—included any control series. If there was one, were the studies done blind and did the possible control series also receive calcium and vitamin D, which are indeed very interesting and potent drugs even used separately? If not we cannot be very impressed with their results regarding the effect of fluoride but rather horrified because they observed three fractures in a series of eight patients during the first year and still continued the administration of fluoride. Drs Jowsey and Riggs claim that fluoride might have a beneficial effect on the bones beginning from the second year. It is only unfortunate that with each patient we cannot avoid the first year, during which according to their and our results the fluoride might just do harm. As an end result of treatment changes in bone biopsy and in quantitative radiology might be of a certain value but from the point of view of the patient and of the practising physician the most interesting thing to know is whether the bones break or not and that must also be the main guide for treatment.

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Nutritional protein needs

SIR,—I was astounded to read in the *BMJ* (30 August, p 547) what purported to be a review of a book by Francis Aylward and Mogens Jul on "Protein and Nutrition Policy in Low-income Countries" but which was in fact only a diatribe expounding the reviewer's one-sided approach to malnutrition.

In it Mr P R Payne dismisses the general need for protein by asserting that "there is now no general acceptance that protein deficiency even exists outside the laboratory." To substantiate this sweeping statement he cites only a Ministry of Overseas Development report which he coauthored. As an American scientist working in Britain I can attest that his conclusion is totally incorrect, as evidenced by the current US National Science Foundation study of protein resources, in which almost 100 of the country's food and nutrition experts are participating.

Mr Payne criticises Aylward and Jul for claiming that protein alone is the answer to world nutrition problems. This is a view they neither hold nor even imply in their book. He further goes on to say that malnutrition is merely "a symptom of poverty," a simplistic statement that offers little in the way of a solution to the extremely complex real problems. In fact, regarding nutrition there is only one acceptable position: all the elements which go to make up a nutritionally balanced diet for a population are equally important, and this must include protein.

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Abingdon

Measuring blood pressure

SIR,—Your leading article on measuring blood pressure (15 November, p 366) mentions preliminary assessment of the systolic blood pressure by palpation but fails to stress fully the importance of this procedure.

If blood pressure is taken by auscultation alone one may miss the "silent gap," which can occur during phase I or phase II Korotkoff sounds. The true systolic blood pressure will be that detected by palpation and may be as much as 30 mm Hg greater than that measured by auscultation.

Furthermore, at any level of diastolic blood pressure cardiovascular mortality increases in proportion to the associated systolic blood pressure.¹ Isolated systolic hypertension is associated with increased morbidity and mortality from cardiovascular disease.² Thus accurate measurement of the systolic blood pressure is as important as measuring diastolic blood pressure. The silent gap can be reduced by inflating the sphygmomanometer cuff quickly and by not having the arm hanging down.³

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Serum α -fetoprotein in cystic fibrosis

SIR,—In a recent article Professor R K Chandra and his colleagues (29 March, p 714) reported abnormally high levels of α -fetoprotein (AFP) in the serum of 18 Canadian patients suffering from cystic fibrosis (range 56–8825 μ g/l); furthermore, they claimed that all 16 heterozygotes tested also had elevated serum AFP levels (mean 178 μ g/l). Their results were contradicted by three British studies (Dr J C Wallwork and others and Dr D J H Brock and others, 17 May, p 392, and Dr J S Fitzsimmons and others, 30 August, p 544) but confirmed by another North American series (Dr J A Smith, 17 May, p 392). In view of this geographical discrepancy and because of the potential importance of the reported finding, we attempted to clarify this controversy.

Sera from 40 well-confirmed cystic fibrosis patients and 60 of their parents were obtained through two Canadian (Chicoutimi and Quebec) and one United States (San Diego) cystic fibrosis clinics. AFP was measured by two methods: a double antibody enzyme immunoassay¹ and a sodium sulphate precipitation radioimmunoassay.² Both assays had a sensitivity limit of 3 μ g/l when referred to the World Health Organisation AFP standard (kindly supplied by Dr P Sizaret, International Agency for Research on Cancer, Lyon, France). The reagents involved in each technique had been independently prepared by LB's (enzyme assay) and SS's (radioimmunoassay) groups.

All sera yielded AFP values below 20 μ g/l (upper normal limit). In order to eliminate the possibility of false-negative results the sera were reassayed by the enzyme method after the addition of 100 μ g/l of purified AFP. The analytical recovery ranged between 85 and 117 μ g/l. Sera also failed to react by

counterimmunoelectrophoresis (sensitivity limit 150 μ g/l) against anti-AFP serum.

Our results, obtained from subjects from three North American geographical areas and determined by two different, well-characterised methods, thus confirm that serum AFP levels are normal in cystic fibrosis. The results obtained by Professor Chandra and his colleagues and by Dr Smith might be explained by the reactivity of their antisera towards another antigen; the preparation of their antisera is not documented well enough to rule out this possibility. Therefore sera from cystic fibrosis patients may actually contain elevated concentrations of an antigen other than AFP which constitutes a marker of the disease.

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Penicillamine therapy, antistriational antibody, and myasthenia gravis

SIR,—In view of recent reports of myasthenia gravis apparently induced by D-penicillamine¹⁻³ and our own experience of a patient with Wilson's disease complicated by myasthenia gravis, thymic hyperplasia, and anti-acetylcholine receptor antibody while on penicillamine we have reviewed 34 patients treated with penicillamine for rheumatoid arthritis. In particular, we have examined sera for the antistriational antibody found in association with thymomata and myasthenia gravis.⁴ Six of these patients (18%) developed antistriational antibody at some stage during the course of therapy. One further patient was found to have antistriational antibody before and during therapy. The titre of the antibody was found to vary somewhat from time to time and in this respect the results are similar to those that we have found in the case of antinuclear factor apparently induced by penicillamine. In the six patients who developed antistriational antibody the latent period after starting penicillamine was 6–24 months. The period of follow-up after the development of the antibody ranges only from one to four months, but none of these patients has manifested overt myasthenia gravis.