ARA-C (total dose 6 mg/kg). This treatment was repeated three weeks later and he had passed into full remission by January 1971. Remission was maintained by weekly immunotherapy with BCG and allogeneic leukaemia cells and monthly courses of ARA-C. The chemotherapy consisted of five daily injections of ARA-C 1-2 mg/kg together with 1-5 mg/kg or 6-thioguanine 2 mg/kg per day for five days. After four such courses he developed burning sensations in his feet which persisted in spite of a subsequent course of treatment but gradually improved between courses. Because this occurred every four weeks the treatment was restricted to the relapse, he continued to complain of intermittent burning or tingling sensations in his feet, starting in the arch of the foot and spreading to the toes. These symptoms slowly improved, but he also complained of persistent or intermittent burning or tingling sensations in his hands. This was probably due to the use of ARA-C, since he had not previously complained of similar symptoms.

Case 2—A 52-year-old man with acute myelogenous leukaemia (weight 70 kg) had similar induction chemotherapy to Case 1 and after 6 mg/kg of ARA-C he complained of numbness and paraesthesiae of the fingers and hands. After four courses of treatment he passed into full haematological remission and was then given eight weeks of therapy consisting of vincristine and cyclophosphamide. This was immediately followed by a single intravenous injection of 100 mg daunorubicin and 130 mg ARA-C. He then stopped all chemotherapy and was maintained in complete remission on intermittent treatment for a period of 1-5 years. Thereafter he relapsed after seven months and had intermittent burning in the fingers and was treated with two courses of intravenous ARA-C (total 20 mg/kg) followed by a single intravenous injection of daunorubicin (2 mg/kg). These courses were given 10 days apart and following the second dose his paraesthesiae became worse. Thereafter aggravation of his neurological symptoms was always associated with ARA-C treatment. He relapsed only once during the period his symptoms improved, though he still complained of painful intermittent paraesthesiae in a glove-and-stocking distribution extending to the toes and occasionally affecting the fingers. There has been no evidence of any objective neurological abnormality and his symptoms have continued to improve slowly, but they are still present 2 years after stopping chemotherapy.

Case 2A—A 52-year-old woman with acute myelogenous leukaemia (weight 70 kg) had similar induction chemotherapy to Case 1 and after 6 mg/kg of ARA-C he complained of numbness and paraesthesiae of the fingers and hands. After four courses of treatment he passed into full haematological remission and was then given eight weeks of therapy consisting of vincristine and cyclophosphamide. This was immediately followed by a single intravenous injection of 100 mg daunorubicin and 130 mg ARA-C. He then stopped all chemotherapy and was maintained in complete remission on intermittent treatment for a period of 1-5 years. Thereafter he relapsed after seven months and had intermittent burning in the fingers and was treated with two courses of intravenous ARA-C (total 20 mg/kg) followed by a single intravenous injection of daunorubicin (2 mg/kg). These courses were given 10 days apart and following the second dose his paraesthesiae became worse. Thereafter aggravation of his neurological symptoms was always associated with ARA-C treatment. He relapsed only once during the period his symptoms improved, though he still complained of painful intermittent paraesthesiae in a glove-and-stocking distribution extending to the toes and occasionally affecting the fingers. There has been no evidence of any objective neurological abnormality and his symptoms have continued to improve slowly, but they are still present 2 years after stopping chemotherapy.

Cutaneous and Occlusive Reactions to Proctolol

SIR—I was very interested to read the paper by Dr. R. H. Felix and others (9 November, p. 321). I would like to bring up to date a quotation from a publication of mine which appeared in their discussion section.

I reported an incidence of 3-4% of positive antinuclear factor (A.N.F.) titre in patients on propranolol treatment. A retrospective survey of 67 patients taking propranolol for angina found that seven had an A.N.F. titre greater than 1/40, an incidence of 11%. In a prospective survey of 71 patients who were treated with propranolol for angina or hypertension, the A.N.F. titre was greater than 1/40 before treatment in only one (1-4%). After an average of six months of treatment this had risen to five (11%) and one of these patients developed a rapidly disseminated lupus erythematosus (D.L.E.).

A prospective study of 50 patients receiving oxprenolol for the treatment of hypertension revealed, instead of a rise in A.N.F. titre before or after treatment for a period of three months. The same was true for 24 patients taking propranolol for hypertension. There have been no instances of D.L.E. syndrome in these two groups of patients.

These observations suggest that propranolol is unique among the commonly used beta-blocking agents in producing the D.L.E. syndrome and is introducing a rising A.N.F. titre. I am, etc.,

E. B. RAFFERTY
Northwick Park Hospital,
Harrow, Middlesex.

Impaired Colour Vision in Diagnosis of Digitalis Intoxication

SIR,—Measurement of the serum level of digoxin provides an accurate estimate of the diagnosis of digitalis intoxication.1 Measurement are, however, time-consuming and need expensive instrumentation. Simple methods for the confirmation of digitalis intoxication would be most desirable.

Various visual complaints are frequently seen as initial signs of digitalis toxicity.2 3 We have tested colour vision in five consecutive ambulant patients in whom the serum level of digoxin was low, because of clinical signs such as loss of appetite, nausea, abdominal discomfort, extrasystoles, or unexplained feeling of sickness. In two patients barium meal examination, carried out because of loss of weight and constant abdominal discomfort, had revealed no signs of organic disease. In the examination of colour vision Ishihara tables (24 plates, 1974) were used. The patients (two male, three female, aged 61-78) were receiving 0-15 to 0-75 mg of digoxin daily and two with heart failure were also receiving frusenide with potassium chloride; four had atrial fibrillation. The serum potassium level was normal (4-2-4 6 mmol/l) in all five. The serum digoxin was only 1-3 ng/ml in one patient but ranged from 2-1 to 3-2 ng/ml in the other four. Dr. Oliver’s report is most interesting and pertinent. In others the diagnosis was made on the strength of a clinical finding of a diastolic pressure that remained at a level of 100 mm Hg or more during a period of ten minutes rest on one occasion. A single such reading in women aged up to 45 may suggest but cannot justify a diagnosis of hypertension. Still less is it relevant that in the remainder of this group the pressure was found to have risen into similar levels within a year after the infarction.

It is said of the allegedly hypertensive patients who sustained infarcts that 25% also had left ventricular hypertrophy. The implication being that here was further proof of pre-existing hypertension. We are not told how the hypertrophy was recognized. But in patients who had survived, or were about to, the acute infarction the assumption would appear to be precarious based. Radiologically it is seldom possible to make an exact distinction between minor cardiac enlargement caused by hypertension and that also found in normotensive patients with myo-cardial change secondary to coronary artery disease. Nor do electrocardiographic tracings afford any precise means of distinguishing between the two, especially in terms of T-wave changes and T-wave inversion, the only abnormalities that are cited.

That hypertension after infarction was later found to be a “potent risk factor” is scarcely surprising since the obvious bias is an exact strain upon a myocardium that was already damaged, a simple sequence of cause and effect unrelated to those obscure and diverse influences that determine the slow development of coronary atherosclerosis with its allied, yet nonetheless distinct, occasional complication of myocardial infarction—I am, etc.,

J. McD. G. STEWART
Victoria Hospital,
Blackpool.

Ischaemic Heart Disease in Young Women

SIR,—In his analysis of ischaemic heart disease in young women Dr. M. F. Oliver (2 November, p. 253) claims that there had been a hypertensive “risk factor” in 40% of those who suffered myocardial infarction. This figure, I would suggest, is misleading since the evidence, much of it retrospective, is insufficient to establish that these women were hypertensive before their infarctions.

An unstatistified proportion of them were “recorded as having hypertension” for no better reason than that they had been under treatment for this condition apparently at the hands of their family doctor, there being no reference to any earlier attendance. Dr. Oliver’s finding that he found the diagnosis was made on the strength of a clinic finding of a diastolic pressure that remained at a level of 100 mm Hg or more during a period of ten minutes rest on one occasion. A single