in May 1977. She was receiving no other medication. At the end of 1977 she complained of pain and swelling in both wrists, which subsequently spread to involve the knees, ankles, hands, and elbows. In July 1978 there was clinically detectable synovitis of the proximal interphalangeal, metacarpophalangeal, and wrist joints. Her blood pressure was 150/80 mm Hg and her general examination was otherwise normal. There was no history suggestive of pleuropericardial disease or skin involvement. Investigations showed haemoglobin 9.6 g/dl, white blood cells $3.7 \times 10^9/l$, erythrocyte sedimentation rate 50 mm in the first hour, Coombs test negative, antinuclear antibody (ANA) test positive (titre 1/320), urea and electrolytes normal; the urine contained no protein, the Rose-Waaler test was negative, C3 and C4 levels were normal, DNA binding activity was <25 units/ml (normal), and the test for extractable nuclear antibodies was negative. Radiographs of the hands, feet, and chest were normal.

She continued to have evidence of a low-grade synovitis and in December 1978 she developed painful, pitting oedema of the right hand and left foot. The ANA titre remained at 1/320, the DNA binding was normal, and the leucopenia persisted. Labetalol was stopped in January 1979 and the patient was started on propranolol long-acting 160 mg daily. By February 1979 the symptoms had resolved; there was no evidence of synovitis and the ESR had fallen to 16 mm in the first hour. The ANA titre had then fallen to 1/80 and by April 1979 it was 1/20; and the patient remained asymptomatic.

We feel that this patient developed a lupus diathesis with synovitis, leucopenia, possible vasculitic oedema, and antinuclear antibodies during treatment with labetalol. There was resolution of the clinical signs and improvement of serological abnormalities on withdrawal of the drug. Our patient differs from the one described by Dr Bensaid and his colleagues in that pleuropericardial involvement was not present and at no stage was our patient's illness sufficiently severe to require corticosteroid therapy. Antibodies to double-stranded DNA were absent in both cases, as is usual in drug-induced lupus disorders. Although labetalol proved effective in controlling this patient's hypertension, it is probable that it induced a lupus syndrome and, as such, should be added to the list of drugs capable of provoking lupus-like illnesses.

IAN D GRIFFITHS

Department of Rheumatology,
Royal Victoria Infirmary,
Newcastle upon Tyne NE1 4LP

JOHN RICHARDSON

Ryton on Tyne, NE40 3LU

Sulphinpyrazone and warfarin after myocardial infarction

SIR,—Drs J A Tulloch and T C K Marr (14 July, p 133) ask for comments on their report of their experience with sulphinpyrazone (Anturan) and warfarin used early after myocardial infarction. I hope that the following remarks may be of some help.

At the present time evidence from controlled clinical trials of sulphinpyrazone following myocardial infarction is available only with respect to treatment being started approximately one month after the acute event and with a daily dose of 800 mg given in four divided doses. Indeed, the relevant current product licence for this product specifies both these conditions for its use.

The final results of the sulphinpyrazone reinfarction trial were presented at the recent

VIIth Congress on Thrombosis and Haemostasis in London and indicate a statistically significant reduction in "sudden death" in the sulphinpyrazone-treated group over the whole period of the trial (mean exposure time 16.1 months). No significant effect on reinfarction was noted. However, the benefit seemed to be due to an early effect which was maintained through the study, and it was therefore suggested that further controlled studies should be performed with earlier use of sulphinpyrazone following myocardial infarction.

At the present moment, however, little or no information is available on the use of this agent on admission of patients to the coronary care unit. Interpretation of results of therapeutic intervention in such patients is extremely difficult in view of the very labile natural history of the disease. For this reason, a comparative control group is essential in any trial attempting to assess new measures in this clinical setting. Preferably this comparison should be made on a double-blind basis. Unfortunately, it would seem that Drs Tulloch and Marr do not have such a comparative group and therefore an assessment of whether a particular event has occurred exceptionally frequently must have been made subjectively. Does "six recurrences . . . within a short period among 41 patients" represent a significant variation from the norm?

Even disregarding these methodological considerations, there are certain questions about this report which need to be answered before any attempt can be made to interpret the results. Did all patients admitted to the coronary care unit during the relevant period receive sulphinpyrazone? If not, how was the selection made? Similarly, were all patients admitted to the coronary care unit anticoagulated? What determined whether a patient received warfarin rather than heparin? Is any information available on the "coagulation status" of the patients following the withdrawal of warfarin? Were the recurrent episodes "fresh ischaemic episodes" or conventionally diagnosed myocardial infarctions?

We are not given any information as to the clinical course of patients during the hospital admission period, but it appears that there may have been only one death among the 78 patients following discharge from hospital. Subjectively, this figure would seem particularly encouraging.

Drs Tulloch and Marr have reported an interesting observation worthy of further investigation, but it would be unwise to draw conclusions from it regarding beneficial or adverse effects of sulphinpyrazone, alone or in combination.

JOHN DOMENET

Geigy Pharmaceuticals,
Macclesfield, Cheshire SK10 2LY

* * * We sent a copy of this letter to the authors, whose reply is printed below.—ED, *BMJ*.

SIR,—Dr Domenet's letter, while of interest, suggests that he has missed the reason for our letter. However, to answer the points he raises:

(1) It was and still is our intention to compare the progress over one year (initially) in patients admitted during the first half of 1979 who received sulphinpyrazone and in those admitted in the second half who will not receive it. (2) All patients admitted up to 30

June who had an acute infarct or acute ischaemic episode did receive sulphinpyrazone. (3) All of these patients were anticoagulated—those admitted under JAT receiving warfarin and those under TCKM subcutaneous heparin. No patient had an absolute contra-indication to anticoagulation. (4) We have no information on the "coagulation status" of the patients after warfarin was withdrawn. (5) No details of the clinical course of the patients were given as it did not seem relevant to the point we were raising. (6) The recurrent attacks were "ischaemic episodes" rather than major infarcts. In our experience six attacks among 41 patients in the course of a few months is unusually and alarmingly high.

J A TULLOCH
T C K MARR

Stracathro Hospital,
Brechin, Angus DD9 7QA

Sodium in peritoneal dialysis solutions

SIR,—We read with interest the paper by Dr P G Bisson and Mr K M Bailey on sodium in peritoneal dialysis solutions (19 May, p 1322). Dialysate sodium concentration is no doubt critical in the management of patients on regular dialysis treatment, whether they are on peritoneal dialysis or haemodialysis. Optimal dialysate sodium concentration in regular dialysis treatment remains a controversial subject. Whereas there are only two types of peritoneal dialysis solution available in the United Kingdom (Na 130 and 141 mmol(mEq)/l), we are alarmed by the fact that on our last count 21 haemodialysis solutions with varying sodium concentration (range 115–145 mmol/l) were commercially available in the United Kingdom.

The need for varying the type of dialysate for haemodialysis stems from the days when haemodialysis machines with fixed proportioning units were used. The modern generation of haemodialysis machines, and indeed the ones used during the past decade, have variable proportioning units, which deliver a desired dialysate sodium concentration, at site, and obviate the need for so many types of haemodialysis solutions.

In the light of these developments we suggest the reassessment of our requirements and standards of dialysis technology.¹

MARTIN DE SOUZA
RASHEED AHMAD

Sefton General Hospital,
Liverpool L15 2HE

¹ Ahmad, R, *Proceedings of European Dialysis and Transplantation Association*, 1979, 16 (in press).

Seizures after aminophylline by suppository

SIR,—A 19-year-old girl without a previous history of epilepsy presented in status asthmaticus. She had been ventilated on two previous occasions during the preceding nine years.

She weighed 36 kg and her arterial oxygen pressure (PaO₂) was 10.1 kPa and arterial carbon dioxide pressure (PaCO₂) 3.3 kPa. Within 12 hours of admission, following the failure of conventional treatment to prevent carbon dioxide retention, she was treated with positive-pressure ventilation for three days, intravenous salbutamol, hydrocortisone, and ampicillin. Off the ventilator on