difference in volume of distribution of the drug rather than solely from a true difference in hepatic microsomal function. It is stated that some expansion of the extracellular fluid volume remained in the oedematous patients despite three days' treatment with frusemide, and this is confirmed by the lower estimated mean plasma antipyrine concentration at time zero in this group (6·18 μmol/l compared with 6·44, 6·39, and 6·48 μmol/l in groups 1-3 respectively). Thus it is possible that there was a spuriously prolonged half life in the presence of a normal hepatic antipyrine clearance in these patients, and we feel that this possibility deserves fuller consideration.

Finally, we question the validity of administering to oedematous patients a dose of antipyrine calculated from the body weight.

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Management of hypertension

SIR,—Granted that blood pressure is not recorded as often as it might be, both in general practice and in hospital (see Dr R F Heller and Professor G Rose, 4 June, p 1441), we could at least publicise the reading once known. All printed hospital discharge and casualty notes as well as registrar's summaries should include a box for BP. Not only would this enter the GP's record folder, but it might also remind the GP of his obligation to include abnormal readings in his referral letters to hospitals.

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Drug compliance in hypertensive patients

SIR,—Drs A J Marshall and D W Barritt (14 May, p 1278) comment that it is appropriate to test carefully whether patients are more likely to take tablets prescribed only once daily than in daily divided doses and whether calendar packing is feasible and preferable. In their study of atenolol they failed to obtain consistent tablet taking for one-third of the time.

We studied 42 hypertensive patients for 16 weeks—four weeks on conventional oxprenolol three times daily, eight weeks on slow oxprenolol once daily, and a final four weeks back on conventional oxprenolol. Patients were assessed at two-weekly intervals. Conventional oxprenolol was dispensed loose in bottles and slow oxprenolol was dispensed in the manufacturer's calendar packs; a variable number of surplus tablets was supplied each time.

70% of 39 patients took all their tablets, as judged by tablet counting, while on slow oxprenolol. Only three took less than 90%; two took 86% and one 88% of the correct dosage, one of these also complying poorly on conventional oxprenolol. Thus compliance was good on slow oxprenolol in 92% (36 of 39 patients). Two patients withdrew from the study shortly after starting on slow oxprenolol, owing to dizziness and abdominal distension respectively. One patient withdrew for domestic reasons. Thirty-three of 41 patients (80%) preferred slow oxprenolol to conventional oxprenolol.

All patients found that the calendar pack was helpful, particularly in enabling them to determine whether or not they had taken their tablets.

As pointed out in your leading article (26 March, p 793) and by Marshall and Barritt, tablet counting may be misleading and may give an overestimate of compliance. Even allowing for this, however, it is probable that the majority of patients on slow oxprenolol exceeded the 80% value which may be designated as compliance. The full results of this study will be reported elsewhere.

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Learnt voluntary control of the heart

SIR,—I was interested in your article on voluntary heart control (11 June, p 1491) because 50 years ago I discovered that I could make my pulse speed up and my pupils dilate while absolutely motionless, much to the amusement of some fellow undergraduates. I had completely forgotten about this but find I have retained the facility.

The technique of learning is rather like that for contracting individual muscles of the leg where one has to transfer a joint-movement volition to a relatively abstract muscular association. Pupil dilatation and heart acceleration were at first achieved by autoinduction of repressed excitement. This is associated with a sort of descending wave of excitement travelling down the body from beneath the sternum, and I suppose one developed a direct ability to provoke that without recourse to any of the original psychological process. I see your references date back to 1971, but I had never heard of anything else of this nature until reading your leading article.

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Sulphinpyrazone in acute stuttering hemiparesis

SIR,—What does a physician do when confronted by a patient with an acute stuttering hemiparesis? An ischaemic process may be thought likely, but the retrospective diagnosis of transient ischaemia or of completed slow-onset stroke must await the passage of time. The imminent danger of a complete disabling hemiplegia may prompt one to therapeutic intervention, but no treatment is established. Published controlled trials of anticoagulant therapy are not convincing of benefit[1 2] and anticoagulant treatment of cerebral vascular disease is associated with a high risk of intracranial bleeding,[2 4] partly because cerebral haemorrhage or tumour cannot be excluded by clinical examination and routine investigations. On the basis that platelet thromboemboli play a role in cerebral ischaemia drugs affecting platelet function, such as sulphinpyrazone, have been studied in patients with transient ischaemic attacks with promising initial results.[5] We wish to report a patient with an acute stuttering hemiparesis which resolved after initiation of sulphinpyrazone therapy.

A 67-year-old housewife was admitted to hospital six hours after the sudden onset of right-sided weakness involving the face, arm, and leg. The initial episode lasted 30 min and resolved completely, but weakness recurred one hour later and the patient experienced six episodes of similar duration on the day of admission, four of which were documented by physicians. On admission intermittent dysarthria, lower right facial weakness, and flaccid weakness of the right arm and leg were noted. The optic fundi were normal. An aortic systolic murmur was heard but there were no carotid bruits. Blood pressure on admission was 200/100 mm Hg, falling to 140/80 mm Hg. Investigations did not reveal any relevant pathology. Shortly after admission she was started on sulphinpyrazone 200 mg thrice daily; 18 h later she had only mild subjective right-sided weakness, and by 36 h after admission she was asymptomatic. There was no recurrence of symptoms over the next seven days and she was discharged home on sulphinpyrazone. She was reviewed some six weeks after discharge and was still symptom-free.

Controlled trials of sulphinpyrazone in the management of patients with transient ischaemic attacks and of patients with reasonable recovery from a completed stroke are currently in progress. In addition, controlled trials of antiplatelet agents in patients with unstable cerebral ischaemia, such as we have described, would seem worth while despite the difficulties of definition and the relative rarity of such patients. To prevent completion of an evolving stroke would be desirable, as would early treatment of transient ischaemic attacks. In a recent community study the greatest risk of completed stroke was in the first month after development of transient ischaemic attacks.[6] Experience with sulphinpyrazone so far suggests that it is a less hazardous treatment than anticoagulants or carotid artery surgery.

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[1] Carter, A B, *British Medical Journal*, 1961, **2**, 70.
[2] Baker, R N, *et al*, *Neurology*, 1962, **12**, 823.
[3] Marshall, J, and Shaw, D A, *Lancet*, 1960, **1**, 995.
[4] Hill, A B, Marshall, J, and Shaw, D A, *British Medical Journal*, 1962, **2**, 1003.
[5] Evans, G, in *Proceedings of Eighth Conference on Cerebral Vascular Diseases, New York*, 1973, ed F H McDowell and R W Brennan, p 297.
[6] Cartlidge, N E F, Whisnant, J P, and Elveback, L R, *Mayo Clinic Proceedings*, 1977, **52**, 117.

Colony-forming cells and myeloblasts in chronic granulocytic leukaemia

SIR,—The demonstration by Dr A J Barrett and his colleagues (14 May, p 1259) that a relationship exists between colony-forming cells (CFUc) and blast-cell numbers in the peripheral blood of a patient undergoing treatment with splenic irradiation for chronic granulocytic leukaemia (CGL) is of great interest because a relationship between myeloblasts and their presumed progenitor cell can be predicted on kinetic grounds. Their failure to demonstrate a relationship between CFUc and total leucocyte numbers must, however, be a temporary phenomenon reflecting an initially selective effect of splenic irradiation on immature granulocytes since we and others have demonstrated such a relationship in untreated patients, in busulphan-treated patients, and in a patient treated by long-term leucopheresis alone.[1-4]

In most patients with CGL whose leucocyte counts have been reduced or restored to normal during treatment with busulphan blast cells have disappeared from the blood and CFUc numbers are low (but not absent). Such a trend is seen also in Dr Barrett's radiotherapy-treated patient. This