improvement as any more esoteric methods of management [not defined]."

My own non-esoteric common sense tells me that severely disabled stroke patients need and respond to intensive rehabilitation, started on the day of onset of the stroke and provided by an expert and committed team of doctor, nurse, physiotherapist, occupational therapist, speech therapist, and social worker.¹ Your reference to "physical treatment . . . unenthusiastically applied at infrequent intervals over a long period in a vain attempt to achieve unrealistic goals" certainly does not describe what happens in the all-too-few stroke rehabilitation units and in the many departments of geriatric medicine where stroke rehabilitation is taken seriously; nor do the staff of such units need to be reminded that "contractures, stiff and painful shoulders, bed sores, and loss of morale may be preventable." (And you might have added incontinence, loss of balance, muscle wasting, mental confusion, and loss of family support.)

The severely disabled stroke patient needs two or three months of intensive team work in hospital and prolonged follow-up in day hospital and community, with the active participation of his family, if he is to have a fair chance of regaining his full potential for recovery. If the acute medical units to which most such patients are admitted in the first instance are unable to provide this level of care—and it is understandable that most units should find this difficult—then it is time that collaborative arrangements were made with departments willing and able to do so.

Your article appeared one week before a heavily oversubscribed conference organised by the Chest and Heart Association on stroke clubs—an indication of how eager the community is to respond to the needs of stroke patients. You rightly quote the Royal College of Physicians' report² as having stressed that stroke causes more severe disability than any other medical condition. You might well have quoted their conclusion that "the resources available for . . . treatment and management should be better concentrated and co-ordinated in health assessment."

BERNARD ISAACS

University Department of Medicine,
Queen Elizabeth Hospital,
Birmingham

¹ Isaacs, B, et al, *The Two Stage Management of Stroke*. In press.

² The Royal College of Physicians of London, *Report of the Geriatric Committee Working Group on Strokes*. London, RCP, 1974.

Cross-sensitivity between practolol and other beta-blockers?

SIR,—During 1974 and 1975 the Swedish Adverse Drug Reaction Committee received 95 reports of adverse reactions involving the skin and eyes following treatment with practolol. The causal relationship between the condition described in the reports and practolol was classified as "probable" or "not excluded" by the committee. When the possibility of a cross-reaction between practolol and other beta-blockers started to be discussed¹ inquiries were sent to the reporting physicians asking if their patients had been treated with other beta-blocking agents. Eighty-six answers were sent in and indicated that 64 of the 86 patients had received another beta-blocker to replace practolol. All beta-

blocking agents on the Swedish market had been used, alprenolol in 26 cases, propranolol in 17, pindolol in 17, metoprolol in seven, and oxprenolol in three (six patients had received two drugs).

In six instances the symptoms thought to have been provoked by practolol either reappeared when another beta-blocker was administered (two patients) or failed to disappear in spite of the change of drug (four patients). Of the first two patients, one, a 70-year-old woman, got macular skin changes which had developed when she was taking practolol, disappeared when this drug was discontinued, but came back when alprenolol was administered one month later. The other, a man aged 56, had psoriasiform skin eruptions and complained of dry eyes. Both complaints vanished when practolol was discontinued. The eye symptoms reappeared when treatment with metoprolol was started some weeks later. Five months later a challenge test with metoprolol provoked the same eye symptoms. A test for antinuclear factor was negative. In the remaining four cases the symptoms remained in spite of a change from practolol to another beta-blocker (alprenolol, pindolol (two patients), or propranolol). Irritation of the eyes was described in three patients, one of whom also had cutaneous symptoms. The fourth patient had psoriasiform changes of the skin which did not improve when her medication was changed from practolol to pindolol.

The possibility of a cross-reaction between practolol and other beta-blockers is suggested by the first two cases, in which there was an interval with no symptoms between the periods of treatment. The chances of making even a tentative diagnosis are much less when there was a direct change from one drug to another. Four of these six patients had eye symptoms. Only one of them had undergone a specialist's examination, which had revealed no abnormalities. In three cases the causal relationship between drug(s) and suspected adverse reaction must be regarded as highly dubious, especially since Wright² has pointed out how common reduced tear flow with variable hyperaemia and vascular engorgement is in this age group.

About a year ago Felix *et al*³ reported on 32 patients with cutaneous reactions to practolol, all of whom tolerated treatment with other beta-blocking agents. Our inquiry confirms the impression that serious side effects of the type induced by practolol are uncommon with other beta-blockers, even in patients who do not tolerate practolol.

A-K FURHOF

Secretary,
Swedish Adverse Drug Reaction Committee

MARGIT NORDLANDER

CURT PETERSON

Section of Adverse Reactions,
Department of Drugs,
National Board of Health and Welfare,
Stockholm, Sweden

¹ Lyall, J R W, *British Medical Journal*, 1975, 2, 747.

² Wright, P, *British Medical Journal*, 1975, 4, 577.

³ Felix, R H, Ive, F A, and Dahl, M G C, *British Medical Journal*, 1975, 1, 626.

Recoverable organic psychosis after hypopituitary coma

SIR,—The case reported by Dr R R Parker and others (17 January, p 132) is very similar to that reported by myself and Dr Isaac Marks in 1961.¹ Our patient suffered from Addison's disease and was originally admitted to hospital in coma with a blood sugar of 1.1 mmol/l (20 mg/100 ml). She recovered from this but an organic confusional state persisted for two months and she was admitted to the Maudsley Hospital. She had been treated with steroids

by mouth and her blood sugar before admission had been 5.5 mmol/l (100 mg/100 ml) on two occasions. The steroids were continued after admission and we found when we made numerous random blood sugar estimates that it could drop as low as 2.9 mmol/l (52 mg/100 ml).

It was long ago pointed out² that symptoms of hypoglycaemia might develop at higher levels of blood sugar in patients with Addison's disease than in normals and it seems likely that the same might be true in hypopituitary patients. We thought that in our patient the blood sugar may not have been maintained at a consistently normal level for long enough for the brain to recover after a prolonged period of hypoglycaemia. We therefore gave her regular and frequent glucose drinks and by the end of a week she was quite normal and remained so during follow-up. It seems possible that because of the confusional state she had not fed herself properly and had probably not taken her medication regularly so that there was a vicious circle tending to perpetuate the hypoglycaemia and confusion. It is possible that during the confusional state she might not have been normally responsive to hunger and that once her mental state became normal she remained well because she responded normally. The recovery of the patient reported by Dr Parker and his colleagues seems to have been rather slow and I think it is possible that she might have recovered more quickly had more emphasis been placed on the correction of hypoglycaemia.

SAMUEL I COHEN

Psychiatric Department,
The London Hospital,
London E1

¹ Cohen, S I, and Marks, I M, *Journal of Neurology, Neurosurgery, and Psychiatry*, 1961, 24, 366.

² Thorn, G W, et al, *Journal of Clinical Investigation*, 1940, 19, 813.

Laparoscopy and laparotomy in staging of Hodgkin's disease

SIR,—I was pleased to see the article by Dr P Spinelli and others (6 December 1975, p 554) on this subject. Our previous work indicated that laparoscopy with multiple biopsies by needle was as likely to be useful in diagnosing Hodgkin's disease of the liver as biopsy is at time of surgery. However, I think it should be further emphasised to those who might attempt to stage patients using peritoneoscopy that splenic aspiration carries a considerable risk for the low yield. Dr Spinelli and his colleagues found that only 38% of patients ultimately found to have disease in the spleen at the time of laparotomy had identifiable disease at aspiration. This low yield is no surprise since spotty involvement of the spleen has been demonstrated in all laparotomy studies. Obviously if one relies on laparoscopy to sample the spleen a significant number of false-negative results will be achieved. There were two episodes of bleeding requiring surgical intervention which, in my view, is an unacceptably high complication rate.

The main value of laparoscopy in staging is the identification of disease in the liver. If disease is demonstrated this may change the entire approach to the therapy of the patient regardless of what is ultimately found in the spleen. If disease is not demonstrated in the liver and the patient is already known to have stage III disease the spleen, if it is of normal size, can be encompassed in a radiation field