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."

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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.

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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.

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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

if radiation therapy is to be the primary treatment. If the spleen is already enlarged and radiotherapy is to be the primary treatment, then laparotomy may be necessary because of the difficulties in encompassing a large spleen safely within the radiation field. The real question is apparent when one is considering patients who are thought to have stage I and II Hodgkin's disease. If an abdominal operation reveals negative results, in some institutions radiotherapy will be withheld from below the diaphragm. Obviously in this case a serious error would be made if laparoscopy only is used for staging, since it is already known that one-third of perfectly normal-appearing spleens may contain Hodgkin's disease. Therefore, in patients with stage I and II disease whose liver biopsies are negative at laparoscopy, if no further staging is to be done, radiation therapy must be given to the lymph nodes below the diaphragm and to the spleen. Since one would not rely on results of a negative splenic aspiration it need not, and should not, be performed under these circumstances.

At this institute we prefer this approach to the treatment of most patients with stage I and II disease. It should be emphasised that not only is the complication rate for routine splenic aspiration high but a patient with stage I and II disease who has a negative aspirate must still either have his or her spleen irradiated or removed at subsequent laparotomy. My concern is that while routine laparoscopy is to be encouraged as a screening procedure prior to consideration of staging laparotomy, routine aspiration of the spleen is too hazardous to recommend; it should not be included as part of the initial laparoscopy work-up.

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Medical and surgical emergencies in divers

SIR,—The problem of serious emergencies occurring in divers under pressure has recently become the subject of concern and was discussed at a conference arranged by the Society of Underwater Technology in London in November 1975 (leading article, 6 December, p 541). In a modern saturation diving system it may be impossible to bring a diver back to atmospheric pressure in less than several days whatever the medical emergency may be. Access to the diver may be difficult because of the location of the decompression chamber and because it may take many hours to compress a doctor or rig medical assistant to the ambient pressure. For this reason mobile pressure chambers in which the diver could be transferred from a barge or rig to a shore facility with transfer under pressure arrangements, where the necessary medical attention can be provided, have been proposed. As you pointed out, at the present time there is no combination of helicopter and light-weight chamber available for this kind of transfer, and in any case it seems likely that the need for such an elaborate procedure would only rarely occur.

It seems to us that it would be more profitable to consider what serious medical or surgical emergencies may arise and what measures should be taken to treat such emergencies at the site of operations. For further

Emergency	Treatment
Trauma	
Lacerations	Suturing
Fractures	Splinting
Burns	Dressings, transfusion
Bleeding	Pressure on bleeding point, transfusion
Ruptured spleen, liver, or kidney	Transfusion
Ruptured hollow viscus	Transfusion, gastric aspiration
Pneumothorax or haemopneumothorax	Pleurocentesis, transfusion
Ruptured urethra	Suprapubic needle drainage
Head injury	Observation alone in most cases
Medical emergencies	
Haematemesis or melaena	Intravenous drip, gastric aspiration, sedation
Myocardial or pulmonary infarction	Analgesia, O ₂ , drugs
Cerebral haemorrhage	Sedation, artificial respiration
Respiratory obstruction	O ₂ , drugs, tracheotomy
Pulmonary or meningeal infections	Antibiotics
Surgical emergencies	
Acute appendicitis	Fowler's position. Antibiotics
Intestinal obstruction, perforated ulcer	Intravenous drip, gastric aspiration. Analgesics

discussion, therefore, we have listed surgical and medical emergencies which we feel are most likely to occur and which would demand urgent treatment, and the treatment of which could be applied in a pressure chamber, if necessary by medical assistants or divers well trained in first aid.

With few exceptions we feel that it should be possible for a well-trained medical assistant or diver to carry out most of these procedures under radio telephone instruction from a doctor, who would carry ultimate responsibility for the treatment. In many cases these procedures would be carried out before the arrival of a medically qualified practitioner, who might then go into the chamber. In our view all but a very small minority of serious emergencies could be effectively dealt with by the application of relatively simple procedures while decompression takes place until transfer to hospital at atmospheric pressure by helicopter is possible.

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R A F COX

Radiology and endoscopy in acute upper gastrointestinal bleeding

SIR,—It has been suggested by Dr K F R Schiller and his colleagues (14 February, p 393) that we have been attacking endoscopy. This has never been our intention, nor could it have been when we have shown that among 61 endoscopy examinations a lesion missed by barium meal was demonstrated in no less than nine cases (31 January, p 270).

Dr Schiller and his colleagues pose four questions to which they are entitled to an answer. Firstly, the relatively small number of cases in our series in a population of some 300 000 is because there are four hospitals with acute medical beds serving the area. There was no selection of cases. Secondly, as Dr W Sircus has pointed out (28 February, p 522) all but a few patients were admitted to the general medical wards in this hospital and the decision whether to send the patients for

radiology or endoscopy was made by the physician in charge of the case. Thirdly, in the majority of cases radiology was performed first and the barium-meal findings were available to the endoscopist. Fourthly, as Dr Sircus has very rightly pointed out, the majority of the barium meals were performed within 48 hours of the haemorrhage and the majority of endoscopies after 48 hours, but in those cases where endoscopy and radiology were both performed within the first 48 hours there was no significant difference in the number of positive findings.

Dr Sircus makes a perfectly valid criticism when he states that we, as radiologists, by comparing radiology with endoscopy have set ourselves up as both prosecutor and jury—although we feel “judge and jury” would be a more appropriate term. Equally, endoscopists who have themselves compared radiology with endoscopy cannot escape similar criticism. With this in mind it is of interest that in a recent study carried out by a radiologist, admittedly in a series containing only a small number of cases of acute upper gastrointestinal bleeding, Cumberland¹ has shown that a firm diagnosis was made at endoscopy in only 13 of 21 cases (62%).

The 10 cases of chronic peptic ulcer in our series which were demonstrated radiologically but not seen at endoscopy must confirm the supplementary nature of the two methods of examination. Surely no one will deny that it is for the physician in charge of the case to decide for himself whether to send for the radiologist or endoscopist and, as Dr W G Scott-Harden has indicated (6 March, p 583), in those hospitals without an endoscopy service it can only be the radiologist.

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¹ Cumberland, D C, *Clinical Radiology*, 1975, **26**, 223.

Smoking tables for carbon monoxide

SIR,—The reasons are not clear to me why Dr Wald and his colleagues (21 February, p 434) object so persistently to the view that the carbon monoxide (CO) yields of cigarettes should be included in the Government tar and nicotine tables. When we suggested this in 1973¹ Dr Wald stated that such action then was premature because there was no direct evidence in man implicating CO as a cause of smoking-related disease.² Now that the case against CO is stronger Dr Wald and his colleagues object on the grounds that this “would make the tables confusing to smokers who wished to select a less harmful brand, as some cigarettes with high CO yields have low tar and nicotine yields, and vice versa.”

Firstly, this statement is incorrect. None of the brands listed in the Government table's “low tar” group have been shown to have a high CO yield. These brands have ventilated (perforated) filters and their CO yields are therefore likely to be very low. Furthermore, none of the brands in the “high tar” group have been shown to have CO yields as low as those of the “low tar” brands.

Secondly, Dr Wald and his colleagues base their conclusion on findings reported in their paper and also presented elsewhere a month before^{3 4} which suggest that the CO yields of