government's plans and 73% being opposed to them. We are aware, however, that moving from such a high profile campaign to an intensive less public operation, aimed at achieving amendments to the government's proposals by means of the democratic parliamentary process, is likely to give rise to the perception that we have given up the battle. Nothing could be further from the truth. We shall continue to do all we possibly can to safeguard the future of the NHS and to ensure that patients continue to have available locally a comprehensive health care service. We are taking steps to increase the amount of information we are giving to our members about these activities.

We are already taking steps to establish intelligence gathering systems to monitor the effects of changes within the NHS."—ED, *BMJ*.

Medicine and politics

SIR,—In his letter Dr C Chantler states that "hospitals managed by trusts within the NHS are simply a means to improve management within the provider hospitals."[1] I would like to take issue with this simplistic view of the functions of NHS trust hospitals.

The original review of the health service stated that trust hospitals would have the ability to "hire or fire." As an accredited representative for Lewisham Hospital I have been endeavouring to discover the contractual position for medical staff within the hospital if it were to be opted out of the NHS. In response to my queries the hospital manager for Guy's Hospital has replied that if consultants' contracts 'are changed after they have been transferred to a trust hospital they will lose their entitlement to terms and conditions of service. He explained that normal employment law will apply but failed to comment that provisions under normal employment are grossly inadequate for senior professional staff. Furthermore, he stated that, although trust hospitals may provide a contract similar to the national terms and conditions of service for medical staff appointed after the starting date for the trust, there will be no right of appeal to the secretary of state if these doctors are subsequently dismissed. I suggest that the general managers' insistence on contractual change for doctors working within trust hospitals provides a far better recognition of what will happen when competitive tendering is established within the NHS than Dr Chantler's simplistic view.

According to the Department of Health and Social Security's service performance indicators for 1987-8 Lewisham and North Southwark Health Authority is one of the 10 most expensive health districts in which to have an operation. It is probable, therefore, that some competitive tenders covering existing work may not be awarded to units in Guy's Hospital or Lewisham Hospital. The manager then has two choices: either to seek to have the work done at a lower cost, perhaps by replacing consultants with partially trained specialists for simpler procedures, or to accept that the tenders will never be competitive and simply make redundant the staff whose work has now left the health district. In either case the removal of consultants' rights will simplify the processes which the manager may wish to utilise. Additionally, the removal of the right of medical staff to speak freely about their work, which exists in the present terms of service, may render it impossible to publicise a lowering in standards within the units. Already one service provided to the health district by consultants has been replaced by clinical assistants in order to free money for other service developments.

Though I am an admirer of the system of clinical management that Dr Chantler has pioneered at Guy's and Lewisham Hospitals, I regard the present proposals for trust hospitals as potentially extremely dangerous and suggest that they should not receive the support of medical staff.

J M CUNDY

Lewisham Hospital,
London SE13 6LH

1 Chantler C. Medicine and politics. *Br Med J* 1990;**300**:124. (13 January.)

Rationalising laboratory services

SIR,—Greater Glasgow Health Board serves a population of 940 000, with acute general services other than obstetrics being provided on 14 separate sites. In a review of this provision it has proposed concentrating all acute activity in five general hospitals plus the paediatric and obstetric units. The detail of these proposals is currently under considerable debate, but the principle of concentrating acute beds in a small number of sites is generally accepted.

During the meeting of the Joint Consultants Committee[1] it was reported that these reviews of clinical services have now been followed by a review of laboratory services that seeks to rationalise provision on a basis of "meeting the needs of the health service in Glasgow, providing a cost effective service, and delivering a quality service to the users." It is proposed to achieve these ends by centralising laboratory services in two sites—the Western Infirmary and the Royal Infirmary. The only facilities available elsewhere will be those for emergency haematological and biochemical tests such as cross matching and estimations of arterial gas tension and electrolyte concentrations along with histological examination of frozen sections if arranged in advance. Thus a hospital such as the Victoria Infirmary, which serves a population of 220 000 and has a major teaching commitment at both undergraduate and postgraduate level, will have no bacteriology, histopathology, routine biochemistry, or haematology departments, no dedicated laboratory medical staff, and no close liaison between clinicians and laboratory specialists. Each of the two main sites will have an overall managing director of laboratory services and a director of each individual laboratory specialty. These directors will have control over laboratory finance and staffing.

As consultants who are to lose laboratory support we are appalled by the proposal. We have little faith in the prospect of the normal consultative process affecting Greater Glasgow Health Board's decisions and are therefore attempting to enlist public support in opposition to its plans. The board already has a timetable for introducing these changes within a few months. There must be a strong likelihood that if applied in Glasgow the same centralising process will occur elsewhere. Such a process may be appropriate for small units, but to deprive general hospitals with between 500 and 1000 acute beds of on site laboratory services is potentially dangerous and educationally disastrous. It should be opposed wherever it is attempted.

A G HUTCHISON JAMES R LOUDEN
PATRICK TANSEY JAMES MOWAT
DAVID BALLANTYNE H B HOWIE
ANGUS C SMITH

Victoria Infirmary,
Glasgow G42 9TY

1 Beecham L. MPs to be told of JCC's worries on NHS bill. *Br Med J* 1990;**300**:334-5. (3 February.)

Emergency services in Rhodes

SIR,—Having read the news item concerning the handling of accident and emergency cases around the world,[1] I felt compelled to describe my recent experience while on holiday in Rhodes. As Greece is a member of the European Community and a country committed to tourism (about half a million tourists visited Rhodes alone last year) you would expect a reasonable level of emergency facilities to be available in all parts of the country. Having participated in the attempted resuscitation of an 18 year old youth who was injured after a motorcycle accident, I can confirm that this most certainly is not the case.

The youth in question was the 20th person to die on the island in a road traffic accident in 1989, and yet the ambulance, having taken 20 minutes to arrive, contained an oxygen mask (no oxygen) and three bandages as the resuscitation equipment. The ambulanceman was completely untrained, and the patient would have been left to asphyxiate in the back of the vehicle had I not been present. The hospital facilities were little better for a centre receiving a similar number of patients with severe injuries as a busy accident and emergency department in the United Kingdom.

I would have hoped that the European Community would seek assurances from all member countries concerning the adequacy of medical facilities in popular tourist areas. The travel companies are equally culpable as they seem to pay scant attention to what health facilities are available. It would be more responsible of them to warn potential customers of how things really are than to gloss over the inadequacies. I have no doubt that this knowledge would make little difference to the numbers of people visiting such resorts, but there is no place for the complacency that currently exists, which merely prolongs this unsatisfactory state of affairs.

SIMON STOCK

Morpeth,
Northumberland NE61 2TY

1 Various authors. Emergency services elsewhere. *Br Med J* 1990;**300**:285-7. (3 February.)

Drug Points

Acute parkinsonism associated with flurbibrofen

Drs T P ENEVOLDSON and C M WILES (Department of Neurology, St Thomas's Hospital, London SE1 7EH) and Dr G V SAWLE (PET Section, MRC Cyclotron Unit, Hammersmith Hospital, London W12 0HS) write: We describe a patient who developed a severe parkinsonian syndrome acutely one week after starting flurbiprofen.

A 52 year old West Indian man was prescribed flurbiprofen 50 mg three times a day for a painful knee. The pain settled, but one week later he awoke to find himself virtually unable to walk despite no loss of power or sensation. Friends confirmed the abrupt onset and that his gait had previously been normal. He was taking no other drugs. General examination was normal. On neurological examination there was symmetrical and severe bradykinesia of his face and limbs, increased tone in his neck and trunk, mild rest tremor of his hands, and a typical parkinsonian gait.

Serum copper and caeruloplasmin concentrations and the results of electroencephalography and autonomic function tests were normal. Results of serological testing were negative for HTLV-I antibody. Tests on blood and cerebrospinal fluid were consistent with previous but not active treponemal infection, and the cerebrospinal fluid was otherwise completely normal. Cranial computed tomography showed some cerebral atrophy and calcification of the basal ganglia (thought to be coincidental) but no other abnormality. Positron emission tomography using ^{18}F-6-L-fluorodopa was performed eight months after the onset and showed that uptake in the putamen was below the normal mean bilaterally.

The flurbiprofen was stopped. Over the next

three weeks the patient's parkinsonism improved somewhat but he remained disabled. Although clinical and laboratory evidence argued against neurosyphilis, a two week course of doxycycline was then given, but the patient did not improve over the next four weeks. His symptoms subsequently improved considerably on increasing doses of levodopa with carbidopa.

This man developed a severe symmetrical parkinsonian syndrome responsive to levodopa whose striking feature was its sudden onset. The only precipitant apparent in the history and from investigations was the flurbiprofen. There are reports of extrapyramidal syndromes in patients taking non-steroidal anti-inflammatory drugs[1-3] but few specifically of parkinsonism. Parkinsonian symptoms have appeared in a few patients taking indomethacin (Merck Sharp and Dohme, personal communication) and been worsened by sulindac in a patient with idiopathic Parkinson's disease already receiving a levodopa and carbidopa preparation.[4] Conversely, diflunisal improved symptoms in six patients with Parkinson's disease.[5] Flurbiprofen has been associated with various motor disorders including ataxia (five reports), tremor (four reports), and myoclonus, akathisia, hypertonia, and extrapyramidal disorder (one report each). (Boots; Committee for the Safety of Medicines, personal communications). To these should now be added the akinetic rigid syndrome.

It is unknown how flurbiprofen mediated its effects, but they were only partially reversible. The findings in the positron emission tomography scan indicated nigrostriatal cell loss, as is seen in idiopathic Parkinson's disease,[6] in contrast with the normal appearance seen in some patients with parkinsonism induced by neuroleptic drugs (D J Brooks, personal communication). We suspect that this patient had subclinical nigral cell loss, which rendered him susceptible to a superimposed drug effect.

1 Wood N, Pall HS, Williams AC, Dieppe C. Extrapyramidal reactions to anti-inflammatory drugs. *J Neurol Neurosurg Psychiatry* 1988;51:731-2.
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5 Anderson CB, Larson EJ. Diflunisal in idiopathic Parkinson's disease. *Neurology* 1984;34:400.
6 Leenders KL, Palmer AJ, Quinn N, *et al*. Brain dopamine metabolism in patients with Parkinson's disease measured with positron emission tomography. *J Neurol Neurosurg Psychiatry* 1986;49:853-60.

Gynaecomastia induced by angiotensin converting enzyme inhibitor

Drs YUJI NAKAMURA, KEIKO YOSHIMOTO, and SHIGEKI SAIMA (Department of Internal Medicine, National Medical Centre Hospital, Tokyo, Japan)

write: Gynaecomastia may be induced or aggravated by a variety of drugs. We report a case of breast enlargement secondary to the administration of angiotensin converting enzyme inhibitor.

A 72 year old man with mild essential hypertension had been receiving captopril 75 mg a day since October 1987. He had never suffered from liver disease, diabetes mellitus, or alcoholism and had not taken any kind of drug. Four months later he noticed a painful gynaecomastia (the size of an egg) on the left side. No urological abnormalities were found. Liver function and renal function were normal, and serum concentrations of triiodothyronine, thyroxine, thyroid stimulating hormone, prolactin, and oestradiol and 24 hour urinary excretion of 17-ketosteroid and 17-hydroxycorticosteroid were all within the normal range. In May 1989 captopril was stopped and nifedipine 30 mg a day was started. One month later the breast enlargement and mamillary pain vanished, but he complained of flushing and foot oedema. Enalapril 5 mg a day was given once a day, but he complained of painful gynaecomastia again within two weeks and refused to take this drug. We changed the antihypertensive drug to prazosin 2 mg a day in August 1989, and gynaecomastia disappeared thereafter.

Markkusse and Meyboom first reported gynaecomastia associated with the use of captopril and suggested that gynaecomastia might be induced by drugs containing sulphydryl. Our patient had breast enlargement while receiving not only captopril but also enalapril, which does not contain the sulphydryl group. Therefore we suggest that angiotensin converting enzyme inhibitor itself induced gynaecomastia.

1 Markkusse HM, Meyboom RHB. Gynaecomastia associated with captopril. *Br Med J* 1988;296:1262.

Severe magnesium toxicity after magnesium sulphate enema in a chronically constipated child

Drs M R ASHTON, D SUTTON, and M NIELSEN (Princess Anne Hospital, Southampton SO9 4HA)

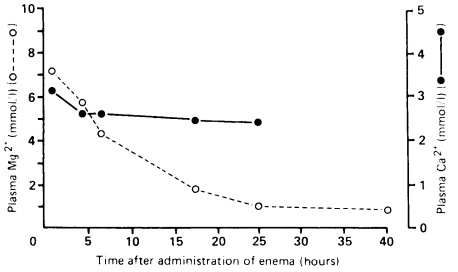
write: The dangers of iatrogenic magnesium toxicity have been recognised in adults in the management of hepatic coma (rectally administered),[1,2] pre-eclampsia (intravenously administered),[3,4] and self poisoning (used as a cathartic).[5] The use of magnesium sulphate enemas in managing hyaline membrane disease in premature infants was discontinued after reports of magnesium induced cardiorespiratory arrest.[6] Eleven years ago Brown and Campbell reported a near fatal case of magnesium toxicity after administration of a magnesium sulphate enema to a chronically constipated child.[7] We report a second such case.

A 25 month old girl with a long history of constipation, treated with lactulose, senna, suppositories, and weekly enemas, presented to the casualty department about one hour after the district nurse had administered what was thought to be a soap and water enema. She was atonic and areflexic, with shallow respirations. Her pupils reacted to light, her blood pressure was 150/60 mm Hg, and her pulse 80 beats/min. Over five minutes her pupils became fixed and dilated and she became apnoeic. There were no doll's eyes or corneal reflexes, though her fundi were normal. She was intubated (with no gag reflex noted) and ventilated. A presumptive diagnosis of raised intracranial pressure was made and 100 ml of 20% mannitol was given before computed tomography of the brain. The scan was normal. The child was transferred to the intensive care unit for continued support.

The district nurse had meanwhile been contacted and said that half a Fletcher's magnesium sulphate enema (about 32·5 g magnesium sulphate) had been given and retained. Acute magnesium toxicity was diagnosed and confirmed by analysis of a blood sample taken on arrival; magnesium 7·1 mmol/l (normal 0·70-0·95 mmol/l) calcium 3·1 mmol/l. The sample also showed hypokalaemia (1·8 mmol/l). An electrocardiogram showed the changes typical of magnesium toxicity—a prolonged PR interval and QRS widening. Electrical stimulation of the ulnar nerve produced no twitches of the forearm muscles, confirming complete neuromuscular blockade.

A forced calcium diuresis was not used in view of her hypokalaemia, the absence of cardiovascular compromise, the previous administration of mannitol, and her rapidly improving clinical state. As the mannitol induced diuresis was established, the child's conscious state steadily improved and her magnesium values fell (figure). Eleven hours after presentation she was extubated. Two days later she was discharged home, neurologically normal.

Urine was collected for 24 hours after admission.



Plasma magnesium and calcium values during the first 40 hours

Analysis showed 33·2 mmol magnesium in the 21·5 hours (normal 1·7-6·8 mmol/24 h), which was only 12·5% of the rectal dose given (about 4 g of the 32·5 g load). Probably not all of the rectal dose was retained and not all of it absorbed.

Magnesium is usually absorbed in the small bowel, but the intact rectum and colon can absorb considerable quantities.[8] In adult cases of magnesium poisoning, magnesium was generally administered over a long period. In both our case and that previously reported[7] a single large dose of magnesium was given (32·5 g and 65 g respectively). Both children were chronically constipated, and chronically dilated rectum and colon probably provided a greater surface area for absorption.

The mechanisms for regulating magnesium concentrations are efficient, with the kidney acting as the main homoeostatic organ under parathyroid hormone control. Physiologically, calcium and magnesium can be regarded as antagonists. This fact, together with the rapid urinary excretion of excess magnesium and the concurrent fall in plasma calcium concentration that should occur in hypermagnesaemia, has led to the use of forced calcium diuresis in managing magnesium poisoning. In our case the mannitol provided the diuresis, and the calcium value was never depressed—an unusual, though previously reported, phenomenon.[1]

The neurological action of magnesium is complex and thought to entail both presynaptic and postsynaptic blockade of the neuromuscular junction. In our case, as well as neuromuscular blockade, central effects appeared to be present, with abolition of brain stem reflexes, the recovery of which correlated with the fall in magnesium values.

We question whether the administration of magnesium sulphate enemas to chronically constipated children is ever justified. They should certainly be avoided in children with renal insufficiency or Hirschsprung's disease. When enemas are to be given in the community the type should be specifically prescribed and carefully checked (the packaging of Fletcher's phosphate and of magnesium enemas is very similar). Children should be observed for 30-60 minutes after the enema has been administered.

We thank Professor I C S Normand and Mr J Atwell for their help in preparing this report.

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