

Biochemical data (mean (SD)) on control group and group who collapsed at end of half marathon

	Control group (n=63)	Group who collapsed (n=34)	p*	Laboratory reference range
Age (years)	31.1 (8.4)	27.9 (7.4)	NS	
Sodium (mmol/l)	143.3 (1.7)	144.1 (2.7)	NS	134-147
Potassium (mmol/l)	4.25 (0.45)	4.24 (0.52)	NS	3.5-5.0
Chloride (mmol/l)	103.9 (2.6)	103.3 (2.6)	NS	96-106
Total carbon dioxide (mmol/l)	21.7 (2.4)	18.7 (3.0)	<0.001	22-29
Anion gap (mmol/l)	21.8 (2.3)	26.3 (4.0)	<0.001	12-20
Urea (mmol/l)	6.40 (1.08)	6.76 (1.33)	NS	1.5-7.0
Creatinine ( $\mu$ mol/l)	137 (19)	164 (31)	<0.001	53-125
Glucose (mmol/l)	4.77 (1.35)	4.62 (1.22)	NS	3.5-5.0
Lactate (mmol/l)	3.7 (1.9)	7.1 (3.7)	<0.001	0.6-2.4
Calcium (mmol/l)	2.57 (0.14)	2.63 (0.23)	NS	2.25-2.75
Phosphate (mmol/l)	1.02 (0.20)	0.69 (0.33)	<0.001	0.65-1.30
Alkaline phosphatase (U/l)†	78 (18)	73 (17)	NS	30-130
Total protein (g/l)	78.5 (3.4)	79.2 (7.5)	NS	60-80
Albumin (g/l)	47.5 (2.1)	48.8 (5.0)	NS	34-50
Aspartate transaminase (U/l)†	31 (9)	30 (8)	NS	<37
Creatine kinase (U/l)†	245 (110)	266 (95)	NS	<175
Lactate dehydrogenase (U/l)†	517 (83)	618 (178)	<0.005	<430

\* Student's *t* test with Cochran's correction for unequal variances.

† Measured at 37°C.

Conversion: SI to traditional units—Sodium, potassium, chloride, total carbon dioxide: 1 mmol/l=1 mEq/l. Urea: 1 mmol/l=6 mg/100 ml. Creatinine: 1  $\mu$ mol/l=11.3  $\mu$ g/100 ml. Glucose: 1 mmol/l=18 mg/100 ml. Lactate: 1 mmol/l=9 mg/100 ml. Calcium: 1 mmol/l=4 mg/100 ml. Phosphate: 1 mmol/l=3.1 mg/100 ml.

(0.9 mg/100 ml) after 50 minutes) was still hypophosphataemic (0.54 mmol/l (1.7 mg/100 ml)) 24 hours later but was able to return to work on the second day after the run.

#### Comment

This incidence of hypophosphataemia is worrying, particularly as the values seen are those usually associated with serious or even fatal disease. As these runners had the clinical and biochemical features of profound hypophosphataemia we conclude that this contributed substantially to their collapse.

The explanation for this hypophosphataemic collapse is not immediately apparent. Although as a group those who collapsed had significantly higher lactate concentrations than the controls (table), there was a positive correlation between the plasma lactate and phosphate concentrations in the group who collapsed ( $r=0.51$ ,  $p<0.005$ )—that is, those with the highest lactate concentrations were not those with profound hypophosphataemia. The condition appears to be transient, the plasma phosphate concentrations returning to normal within 24-48 hours without specific phosphate treatment. Because of the dangers of phosphate infusions<sup>2</sup> and the fact that the assays were not carried out on the spot we thought it inadvisable to administer parenteral phosphate as a first aid measure.

We thank Miss Ann Weddell and Mr R Ellis, biochemists, and the technical staff of the department of clinical biochemistry, Newcastle General Hospital, for help with documentation, preparation of samples, and analyses; Mr B Johnson for the computer software enabling us to analyse the results; and Miss E Wiffen for preparing the manuscript.

1 Anonymous. Treatment of severe hypophosphataemia. *Lancet* 1981;ii:734.

2 Knochel JP. The pathophysiology and clinical characteristics of severe hypophosphatemia. *Arch Intern Med* 1977;137:203-20.

3 Storm TL. Severe hypophosphataemia during recovery from acute respiratory acidosis. *Br Med J* 1984;289:456-7.

4 Young GB, Amacher AL, Paulseth JE, Gilbert JJ, Sibbald WJ. Hypophosphataemia versus brain death. *Lancet* 1982;i:617.

(Accepted 4 November 1985)

Department of Clinical Biochemistry, Newcastle General Hospital, Newcastle upon Tyne NE4 6BE

G DALE, MD, senior lecturer and consultant chemical pathologist  
J A FLEETWOOD, PHD, top grade biochemist

Department of Anaesthetics, Fleming Memorial Hospital for Sick Children, Newcastle upon Tyne NE2 3AX

J S INKSTER, FFARCS, consultant anaesthetist

Department of Surgery, University of Newcastle upon Tyne Medical School, Newcastle upon Tyne NE2 4HH

J R C SAINSBURY, FRCS, senior registrar

Correspondence to: Dr Dale.

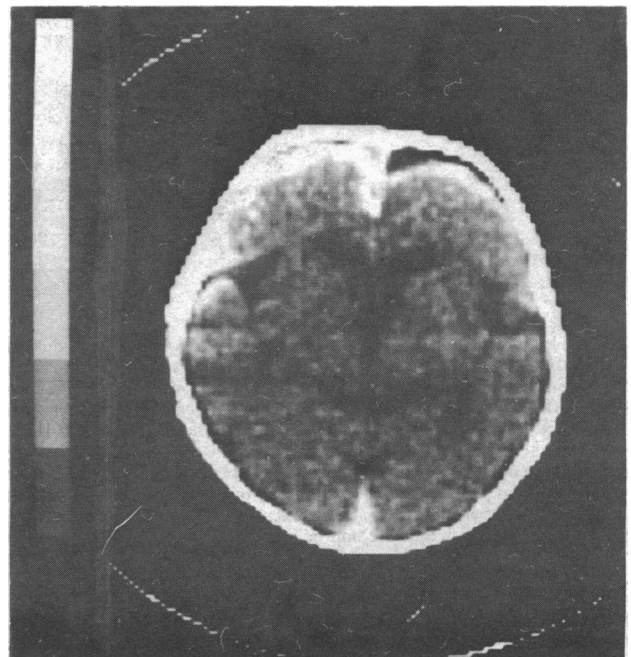
## Addition of rifampicin in persistent *Haemophilus influenzae* type B meningitis

Meningitis due to *Haemophilus influenzae* type B (haemophilus) is the second most common form of bacterial meningitis in the United Kingdom.<sup>1</sup> Despite the wide availability of effective antimicrobials the mortality and morbidity of this illness remain appreciable.<sup>2</sup> We report a case of haemophilus meningitis with ventriculitis in a 3 month old infant refractory to conventional treatment despite in vitro sensitivity. It resolved promptly when rifampicin was introduced.

#### Case report

A thriving and developmentally normal 3 month old Bangladeshi boy was admitted to hospital with a 48 hour history of fever and malaise. There were no focal signs of infection. The lumbar spinal cerebrospinal fluid was turbid, with a white cell count of  $2 \times 10^9/l$  (95% polymorphs). Culture yielded *H influenzae* type B, sensitive to ampicillin and chloramphenicol.

The infant was treated with intravenous chloramphenicol (100 mg/kg/24 h). Serum concentrations were satisfactory at 8 mg/l trough and 32 mg/l peak. On the seventh day after admission he had two short convulsions. Plasma glucose, electrolyte, and calcium concentrations were normal. Lumbar cerebrospinal fluid showed a white cell count of  $75 \times 10^6/l$ , with no organisms found on microscopy or culture. On the ninth day he was still feverish and irritable. A right ventricular tap yielded cloudy cerebrospinal fluid with a white cell count of  $500 \times 10^6/l$  (90% polymorphs). Culture was sterile. The cerebrospinal fluid chloramphenicol concentration was 7 mg/l. Daily intraventricular chloramphenicol was begun.



CT scan showing enhancement over frontal and temporal lobes.

During the tap on day 11 a pocket of thick, yellow, purulent fluid was entered and 6 ml pus removed. Culture remained sterile. There appeared to be loculated infection. The antibiotic regimen was changed to ampicillin (400 mg/kg/24 h) and co-trimoxazole (96 mg/kg/24 h), both given intravenously, plus daily intraventricular ampicillin.

After initial improvement the fever and irritability recurred on day 18 and the ventricular cerebrospinal fluid white cell count again rose to  $500 \times 10^6/l$  (95% polymorphs). A computed tomography (CT) scan with iohalamate (Conray) enhancement showed a mixed density filling defect in the left lateral ventricle and striking enhancement over the surfaces of the frontal and temporal lobes. The appearances were consistent with loculation of pus in the ventricle and widespread active meningitis (figure). Oral rifampicin (20 mg/kg/24 h) was therefore added.

The infant showed a prompt and dramatic improvement. The fever disappeared. The white cell count in the ventricular cerebrospinal fluid fell to  $24 \times 10^6/l$  within three days of beginning rifampicin. Intraventricular antibiotics were stopped on day 25 and intravenous antibiotics stopped on day 32. Oral rifampicin and co-trimoxazole (48 mg/kg/24 h) were given until day 40. A repeat CT scan after treatment was normal.

At 1 year of age the child was showing normal general and developmental

progress. Results of full audiometric examination were normal. His head circumference was growing along the 10th centile line.

### Comment

Rifampicin kills a wide variety of micro-organisms, including haemophilus.<sup>3</sup> In our patient its use led to the rapid resolution of all his symptoms and signs of disease. Drainage of the infected material was difficult and reaccumulation rapid. Chloramphenicol readily enters the brain and cerebrospinal fluid. The concentration in the cerebrospinal fluid with intravenous treatment alone was adequate. Ampicillin and co-trimoxazole both penetrate the cerebrospinal fluid in the doses used. The combination of intravenous and intraventricular administration would have produced more than adequate cerebrospinal fluid concentrations for both chloramphenicol initially and ampicillin with co-trimoxazole subsequently. Failure of treatment was presumably secondary to sequestration of organisms in loculated pus, making them inaccessible.

Rifampicin has the unique ability both to penetrate pus and kill phagocytosed organisms.<sup>4</sup> Bactericidal concentrations are achieved in brain,<sup>3</sup> cerebrospinal fluid,<sup>3</sup> and pus<sup>5</sup> after oral administration. It has been used successfully in staphylococcal meningitis and subdural empyema,<sup>5</sup> enterococcal meningitis, and neonatal flavobacterial meningitis.

We suggest that, in combination with another antibiotic, rifampicin should be considered as second line treatment in haemophilus meningitis not responsive to conventional agents.

- 1 Goldacre MJ. Acute bacterial meningitis in childhood. Incidence and mortality in a defined population. *Lancet* 1976;i:28-31.
- 2 Sell SHW, Merrill RE, Doyno EO, Zimsky EP. Long term sequelae of Haemophilus influenzae meningitis. *Pediatrics* 1972;49:206-11.
- 3 Farr B, Mandell GL. Rifampicin. *Med Clin North Am* 1982;66:157-68.
- 4 Mandell GL. Interaction of intraleucocyte bacteria and antibiotics. *J Clin Invest* 1973;52:1673.
- 5 Vichyanond P, Olsen LC. Staphylococcal CNS infections treated with vancomycin and rifampicin. *Arch Neurol* 1984;41:637-9.

(Accepted 19 November 1985)

### The Children's Hospital, Western Bank, Sheffield S10 2TH

MALCOLM A LEWIS, MB, MRCP, paediatric registrar  
BETTY L PRIESTLEY, MB, FRCP, consultant paediatrician

Correspondence to: Dr Malcolm A Lewis, Research Registrar, Department of Endocrinology, Royal Manchester Children's Hospital, Pendlebury, Manchester M27 1HA.

## Cause of severe head injury and risk of complications

Mortality and morbidity after head injury can be reduced by the early identification of patients at high risk of intracranial complications and their prompt transfer to a neurosurgical unit.<sup>1,2</sup> On the other hand, transfer can be made hazardous by failure to diagnose and treat the major extracranial injuries that often accompany serious head injury<sup>3</sup> and that are more difficult to diagnose when consciousness is impaired.<sup>4</sup> The aim of this study was to establish whether the cause of injury—which is usually easily determined—influences the relative risks of intracranial haematoma and major extracranial injury and thus to resolve the potential conflict between the urgency of transfer to a neurosurgical unit and the value of an extended period of detailed assessment and stabilisation.

### Patients, methods, and results

Since 1974 data have been collected prospectively on all head injured patients admitted to the Glasgow neurosurgical unit. Using this database, we studied

retrospectively 891 consecutive patients treated during 1974-83 inclusive, all of whom were in coma (not obeying commands, not opening the eyes, and not uttering recognisable words) for at least six hours after injury and none of whom was conscious between injury and admission to the neurosurgical unit.

A road traffic accident was the cause of injury in 581 cases (65%), the 310 other patients being injured in falls; assaults; accidents at home, at work, or at sport; or in other ways. An intracranial haematoma had been evacuated by craniotomy in 320 patients (36%), and 343 patients (38%) had multiple injuries, in that a serious head injury was accompanied by at least one major extracranial injury meriting admission in its own right. Victims of road traffic accidents more often had multiple injuries, either in isolation or combined with an intracranial haematoma, than patients injured by other causes (table). The presence of multiple injuries decreased the risk of a haematoma only slightly in victims of road traffic accidents (from 26% to 21%) but from 65% to 31% in the others. A skull fracture had been diagnosed clinically or radiologically in 535 patients (60%) and increased the risk of a haematoma from 12% to 33% in victims of road traffic accidents and from 41% to 66% in the others.

### Comment

These results show that an unconscious patient with an injury sustained at a high velocity, such as in a road traffic accident, has a substantial risk of a intracranial haematoma but an even higher risk of multiple injuries. Although a patient with an injury sustained at a low velocity, such as a drunk who has fallen, may seem to have sustained a less severe injury than the victim of a road traffic accident, he is at greater risk of life threatening but potentially remediable intracranial complications. One in 10 of these patients too, however, will have major extracranial injuries.

When a head injured patient is in coma it is natural to focus attention on the need for urgent neurosurgical assessment. More and more head injured patients are being transferred to neurosurgical units, and more rapidly than before, to offer the maximum chance of recovery to those who may have intracranial haematomas. On the other hand, all authorities agree that the elimination of shock and respiratory insufficiency must take priority over transfer, which otherwise exposes the patient to grave risks. In our recent studies all patients who were both hypotensive and hypoxaemic when they arrived at the neurosurgical unit died,<sup>3,5</sup> irrespective of their intracranial condition. Shock is rarely due to a head injury alone, and an unconscious patient with an injury sustained at a high velocity who has any degree of hypotension should be assumed to have internal injuries until proved otherwise. Transfer of such a patient to a neurosurgical unit should not be done until these have either been detected and treated or else eliminated as far as is possible.

- 1 Mendelow AD, Teasdale GM, Jennett B, *et al.* Risks of intracranial haematoma in head injured adults. *Br Med J* 1983;287:1173-6.
- 2 Teasdale GM, Galbraith SL, Murray L, *et al.* Management of traumatic intracranial haematoma. *Br Med J* 1982;285:1695-7.
- 3 Gentleman D, Jennett B. Hazards of inter-hospital transfer of comatose head injured patients. *Lancet* 1981;ii:853-5.
- 4 McLaren CAN, Robertson C, Little K. Missed orthopaedic injuries in the resuscitation room. *J R Coll Surg Edinb* 1983;28:399-401.
- 5 Kohi YM, Mendelow AD, Teasdale GM, *et al.* Extracranial insults and outcome in patients with acute head injury—relationship to the Glasgow coma scale. *Injury* 1984;16:25-9.

(Accepted 4 November 1985)

### Department of Neurosurgery, Institute of Neurological Sciences, Southern General Hospital, Glasgow G51 4TF

DOUGLAS GENTLEMAN, BSC, FRCS, registrar  
GRAHAM TEASDALE, MRCP, FRCS, professor  
LILIAN MURRAY, MSC, statistician

Correspondence to: Mr Gentleman.

### Correction

#### Dietary practices of Asian diabetics

In the legend to the table in the article "Dietary practices of Asian diabetics" (18 January, p 171) "unsaturated oil" should have read "unsaturated margarine."

Relation between cause of injury and risks of multiple injuries and intracranial haematoma

Cause of injury	No (%) of patients	No (%) with multiple injuries alone	No (%) with intracranial haematoma alone	No (%) with both complications	No (%) with neither complication
Road traffic accident	581 (65)	232 (40)	76 (13)	60 (10)	213 (37)
Other	310 (35)	35 (11)	168 (54)	16 (5)	91 (29)
All causes	891 (100)	267 (30)	244 (27)	76 (9)	304 (34)