Health of juvenile delinquents

Intermediate treatment centres were established in 1969 to enable delinquent adolescents to remain within their communities under supervision, avoiding the imposition of a custodial sentence.[1] This study compares the physical health of a group of delinquent boys who attended Parkway, an intermediate treatment centre in a deprived inner city area, with that of non-delinquents.

Subjects, methods and results

Twenty four delinquents admitted during one year were eligible for the study. They were matched with 24 boys who were at the same school and in the same year and who lived in the same postal district. A medical history was obtained, general physical examination was performed, and hearing and vision were assessed. Student's t and the χ^2 test were used as appropriate.

The table shows the physical and social characteristics of the boys. There was no difference in birth weight, the incidence of perinatal problems, or the number of hospital admissions between the two groups. Urinalysis showed normal results in all cases. Smoking was more common among delinquents ($p<0.01$). The

Physical and social characteristics of delinquent and non-delinquent boys. (Figures are means (SD))

	Delinquent boys (n=24)	Non-delinquent boys (n=24)
Age (years)	15·3 (0·7)	15·7 (0·6)
Weight (kg)	54·4 (7·7)	58·6 (11·0)
Height (cm)	168·4 (8·4)	167·1 (7·5)
White	16 (67%)	19 (79%)
Mixed race	6 (25%)	3 (13%)
Afrocaribbean	1	2 (8%)
Asian	1	
Single parent	9 (38%)	8 (33%)
Children in family	4·0	3·0
Unemployed "breadwinner"	9 (38%)	6 (25%)
Smokers	20 (83%)	2 (8%)*
Failed vision test	9 (38%)	7 (29%)
Spectacle wearers	1 †	5*
Failed hearing test	8 (33%)	4 (17%)

*$p<0.05$.
†Spectacles lost.

offences that led to the admission of boys to Parkway were robbery or mugging, or both (six), theft (six), offences at school (four), offences with motor vehicles (four), drug related offences (two), being drunk and disorderly (one), and carrying an offensive weapon (one). There were no significant differences in social class, numbers of single parent families, or unemployed heads of households. The delinquents came from slightly, but not significantly, larger families.

Boys were tested with spectacles if they had been prescribed. Nine delinquents and seven non-delinquents failed the vision test, but only one of the delinquent boys was aware that he had a vision problem, although he never wore his spectacles. By contrast, five of the non-delinquents wore spectacles ($p<0.05$). Hearing was tested with pure tone audiometry 500-4000 Hz. All the boys who failed the test failed to hear at ≥ -30 db on at least two frequencies. Eight delinquents failed compared with four controls.

Three boys in the control group had medical problems: one suffered from recurrent urinary tract infections and two had mild asthma, easily controlled by bronchodilator treatment. Four boys in the delinquent group had asthma: one had been prescribed treatment that he was using inappropriately and the three others were unaware that they suffered from a potentially remediable condition. One of the delinquent boys, diagnosed as having febrile fits aged 3, had been prescribed phenytoin and phenobarbitone prophylactically. When sent to Parkway aged 16 he was still taking these anticonvulsant drugs four years after his last fit. After a lecture on the side effects of drugs he threw his pills away and subsequently described himself as feeling "much better, much less aggressive." His behaviour was exemplary after drugs were stopped.

Comment

The high prevalence of smoking and being a member of a large family have previously been recognised among delinquents.[2-4] Smoking may be an easy way to react against society,[2] and a large family may be a potential cause for neglect.[3,4]

The most striking finding of this study was the potentially remediable, but hitherto unrecognised, ailments among delinquent boys. We think that it is unlikely that these boys' disabilities contributed to their delinquency.[5] Rather we would suggest that the lack of recognition of physical disabilities and delinquency share a common cause—namely, neglect and deprivation during earlier childhood. School health screening programmes, which are aimed at younger children, should pay particular attention to those who fail to attend for examination and should arrange for them to be urgently and vigorously followed up.

Children who attend an institution such as Parkway should be offered a routine medical examination.

We thank the school nursing and community paediatric services of central and southern Manchester for their help and Professors Leck and Boyd for helpful discussion.

1 Nicholas P. *Intermediate treatment*. Gloucester Social Services (Cheltenham Area), 1983.
2 Tamir A, Wolff H, Epstein L. Health-related behaviour in Israeli adolescents. *J Adolesc Health Care* 1982;2:261-5.
3 Glueck S, Glueck ET. *Unravelling juvenile delinquency*. Cambridge, Massachusetts: Harvard University Press, 1950.
4 Fischer DG. Family size and delinquency. *Percept Mot Skills* 1984;58:527-34.
5 Penner MJ. The role of selected health problems in the causation of juvenile delinquency. *Adolescence* 1982;17:347-68.

(Accepted 11 November 1985)

Department of Paediatrics, Saint Mary's Hospital, Manchester M13 0JH
A M WEINDLING, BSC, MRCP, senior registrar
F N BAMFORD, MD, FFCM, reader

Parkway Intermediate Treatment Centre, Manchester M15 5PA
R A WHITTALL, BA, CQSW, senior social worker

Correspondence to: Dr Weindling, Department of Child Health, Liverpool Maternity Hospital, Liverpool L7 3BN.

Profound hypophosphataemia in patients collapsing after a "fun run"

Profound hypophosphataemia, with plasma phosphate concentrations below 0·32 mmol/l (1 mg/100 ml) is associated with generalised muscle weakness; respiratory failure; cardiomyopathy; signs of central nervous disorder including irritability, numbness, dysarthria, confusion, seizures, and coma; and death.[1-4] Here we report it in runners who collapsed at the end of the annual Great North Run, a half marathon from Newcastle upon Tyne to South Shields.

Patients, methods, and results

In four races the Great North Run has attracted over 100 000 entrants, of whom 34 men collapsed near the finishing line and were either unconscious or confused and disorientated. They were usually weak, dehydrated, and hyperthermic with rectal temperatures over 40°C. Two of the runners who collapsed had a grand mal fit; neither had hypoglycaemia or a history of seizures.

As glucose is known to lower plasma phosphate concentrations[2] venous blood samples were obtained shortly after the runner was brought into the medical tent and immediately before the start of any treatment. The specimens were centrifuged on site and the separated plasma stored for analysis the next day. Samples were also obtained from a control group of 63 men who finished uneventfully.

The plasma phosphate concentrations in the group who collapsed were significantly lower than those in the control group ($p<0.001$; table). Fifteen of the runners who collapsed had subnormal concentrations, and seven of these were profoundly hypophosphataemic with concentrations immediately after the run as low as 0·17 mmol/l (0·5 mg/100 ml).

Of the two who had seizures, one with an initial plasma phosphate concentration of 0·48 mmol/l (1·5 mg/100 ml) had collapsed in a previous half marathon race. The other was deeply unconscious, hypotensive (blood pressure 76/50 mm Hg), and dehydrated with a rectal temperature of 40°C. He was unresponsive to painful stimuli and had dilated and unreactive pupils. Intravenous infusion of dextrose and saline and ventilation with 100% oxygen were without satisfactory effect, and he was transferred to a hospital intensive care unit, where he began to breathe spontaneously after two hours. His plasma phosphate concentration (0·29 mmol/l (0·9 mg/100 ml) at the end of the race) fell to 0·06 mmol/l (0·2 mg/100 ml) while he was in the intensive care unit but rose slowly, without specific phosphate replacement, to 0·6 mmol/l (1·9 mg/100 ml) after 48 hours. When visited six months later he was none the worse for this episode.

Two of the other severely hypophosphataemic men who collapsed were seen in their homes 24 hours later. Both were still subjectively unwell and were unable to return to work that day. One, who had had a plasma phosphate concentration of 0·27 mmol/l (0·8 mg/100 ml) immediately after the event, was found to be normophosphataemic (1·13 mmol/l (3·5 mg/100 ml)). The other, who had been disorientated for 30 minutes after finishing the race, with a phosphate concentration immediately after the run of 0·17 mmol/l (0·5 mg/100 ml) (and 0·3 mmol/l

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 (μmol/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 μmol/l≈ 11·3 μg/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[2] 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.

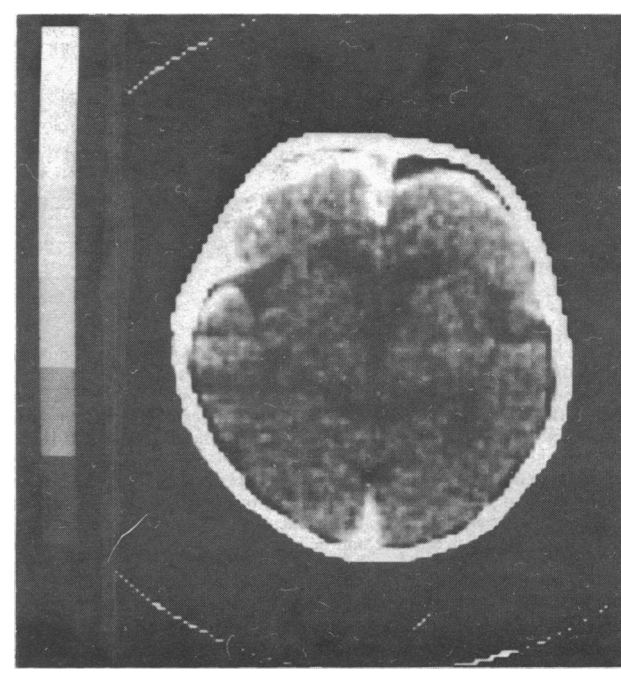
Addition of rifampicin in persistent Haemophilus influenzae type B meningitis

Meningitis due to *Haemophilus influenzae* type B (haemophilus) is the second most common form of bacterial meningitis in the United Kingdom.[1] Despite the wide availability of effective antimicrobials the mortality and morbidity of this illness remain appreciable.[2] 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 2×10^9/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 75×10^6/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 500×10^6/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 500×10^6/l (95% polymorphs). A computed tomography (CT) scan with iothalamate (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 24×10^6/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