LETTERS TO THE EDITOR

Prevalence of behaviour disorders in low birthweight infants

EDITOR,—We wish to comment on some methodological and conceptual ambiguities in the report by Pharoah *et al* on behaviour disorders in low birthweight infants.[1]

First, in their methods section, dealing with the assessment of hyperactivity, the authors refer to 'the Conners modification, which used the Rutter teacher questionnaire only'. The Conners paper to which they refer does not cite Rutter's work, describes no modification of the Rutter teacher scale, and does not mention the hyperactivity cut off employed in the authors' study.[2] It therefore remains unclear which instrument was actually used in this study, and how the hyperactivity cut off was derived. In the results section, we discover that the Wechsler Intelligence Scale for Children was also employed. While we would accept that the authors describe their full battery of measures elsewhere, it is important to list the instruments used in the appropriate methods section and to give a brief indication of results. Failing this, one is left wondering whether the authors trawled their data bank and reported only positive findings.

Second, in their discussion section, the authors fail to make the necessary distinction between *diagnosis* of cases with disorder/disease and *identification* of cases with possible behaviour problems. The former is a function of judgment by a clinician, often drawing on a variety of sources of information, while the latter is all that screening tests can do. Screening tests do not diagnose.

The clinical management of hyperactivity often requires collaboration across a number of disciplines: the same holds true for its assessment, and consultation with colleagues in psychology and psychiatry might have led to a more meaningful report from this study. The information given is inadequate for discussion of the clinical and social implications of the findings reported.

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1 Pharoah POD, Stevenson CJ, Cooke RWI, Stevenson RC. Prevalence of behaviour disorders in low birthweight infants. *Arch Dis Child* 1994; **70:** 271–4.
2 Conners CK. A teacher rating scale for use in drug studies with children. *Am Psychiatry* 1969; **126:** 884–8.

Professor Pharoah and coauthors comment:
Drs MacMillan and Morton are correct in stating that the Connors paper does not cite Rutter's work and describes no modification of the Rutter teacher scale. We used a modified Connors' scale and should have cited Taylor and Sandberg (1984)[1]; this was an unfortunate omission on our part. Our failure to mention the use of the Wechsler Intelligence Scale for Children in the methods was because it was stated in the immediately preceding paper in the same issue of the journal. The comment that we trawled our data bank and reported only positive findings is unwarranted and MacMillan and Morton are welcome to inspect the raw data.

The second point raised is that we fail to make the distinction between *diagnosis* of cases and *identification* of those with possible behaviour problems. This is not so. The opening part of the discussion states that 'The Rutter parent and teacher scales are screening instruments' and that 'The validity of any screening test is determined by its sensitivity ... and its specificity'. We also indicate, in the second paragraph, that there is only a moderate correlation in behaviour ratings at ages 7, 11, and 16 years, as determined by these screening questionnaires. We clearly state that the long term morbidity needs to be determined. The important point that we wish to stress, is that there is a significant difference between the cases and controls as shown by behaviour screening instruments.

The final point made is that we failed to consult with colleagues in psychiatry and psychology. This is also not so, we consulted with colleagues in both of these disciplines in Liverpool and London. Furthermore, two of the research workers on the project were themselves graduates in psychology.

1 Taylor E, Sandberg S. Hyperactive behavior in English schoolchildren: a questionnaire survey. *J Abnorm Child Psychol* 1984; **12:** 143–56

Studies on the cure rates in acute lymphoblastic leukaemia in children from urban and rural areas

EDITOR,—Acute lymphoblastic leukaemia (ALL) is at present cured in approximately 70% of children.[1 2] Successful treatment is very costly and consequently cure rates in ALL are much lower in developing countries than in affluent countries.[2] There are almost no data indicating whether children from urban and rural areas within one country or one region have the same chances for being cured of ALL.[3–7] For many years in Poland there were great differences between urban and rural areas in accessibility to doctors. When the present studies were started medical care for rural children had greatly improved.

The studies were carried out at least four years after the completion of treatment for ALL and included 417 children treated in the years 1967–86. The group included 191 children from urban and 226 patients from rural areas. The cure rates from ALL among urban and rural children were analysed. Comparative studies were carried out in three periods (see table) in which markedly different treatment protocols had been used, and the observations were completed on 31 December 1993. The shortest maintained remission after cessation of treatment was 58 months. The comparative analysis was based on the χ^2 test, with the significance level accepted as $p<0.05$.

Cure rates (%)

Period of treatment (years)	*Urban area*	*Rural area*
1967–74	8·9	17·5
1975–81	34·2	29·2
1982–6	66·1	46·6
Total	36·1	32·0

The percentage of children cured from ALL was higher among children from urban than from rural areas, the differences being statistically significant only in the third period. Other observations and comparative studies are necessary to explain the differences in cure rates among children from urban and rural areas. Doctors should make further efforts, particularly in improving the cooperation of parents, so that children with leukaemia have similar chances for a cure, irrespective of their place of residence.

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1 Armata J, Cyklis R, Balwierz W, Pekacki A. Curability of acute lymphoblastic leukemia in children treated in years 1975–1984. *Nowotwory* 1991; **41:** 234–9.
2 Pinkel D. Lessons from 20 years of curative therapy of childhood acute leukaemia. *Br J Cancer* 1992; **65:** 148–53.
3 Curtiss CP. Trends and issues for cancer care in rural communities. *Nurs Clin N Am* 1993; **28:** 241–51.
4 Harris R, Leiniger L. Preventive care in rural primary care practice. *Cancer (Suppl)* 1993; **72:** 1113–8.
5 Anonymous. Not all US children with cancer receive optimal care. *Ann Oncol* 1992; **3:** 251.
6 Papas G, Queen S, Hadden W, Fisher G. The increasing disparity in mortality between socioeconomic groups in the United States, 1960 and 1986. *N Engl J Med* 1993; **329:** 103–9.
7 Robinowitz HK. Recruitment, retention, and follow-up of graduates of a program to increase number of family physicians in rural and underserved areas. *N Engl J Med* 1993; **328:** 934–9.

Renal scarring after acute pyelonephritis

EDITOR,—Dr Jakobsson and colleagues have confirmed the value of technetium-99m dimercaptosuccinic acid (DMSA) imaging in urinary tract infection (UTI) in children both on initial presentation and follow up.[1] The authors highlight the sensitivity of DMSA imaging in the detection of renal cortical defects; however, we feel that the apparent 37% incidence of renal scarring following an episode of acute pyelonephritis in their study is over pessimistic.

We feel it is extremely important, when describing DMSA abnormalities, to state whether the abnormality is associated with or without loss of the normal cortical outline.[2] The definition of DMSA abnormality used in Jakobsson's report was 'one or more areas of decreased cortical uptake with or without preservation of the cortical outline'. It is therefore unclear to us why it was assumed that all the DMSA abnormalities found on follow up were felt to be consistent with cortical scarring.

We found a relatively high proportion of children presenting with a first documented UTI have reduced uptake on DMSA but with preservation of the cortical outline.[2] In the majority of patients, these changes resolved completely; however, we have anecodotal evidence of these changes persisting up to 36 months after an episode of UTI.

There is no doubt that DMSA imaging will enable the development of a more complete understanding of the natural history of UTI, however, if as is likely, this imaging modality

becomes more popular, the use of an accurate definition of cortical scarring is essential.

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1 Jakobsson B, Berg U, Svensson L. Renal scarring after acute pyelonephritis. *Arch Dis Child* 1994; **70:** 111–5.
2 Tappin DM, Murphy AV, Mocan H, *et al.* A prospective study of children with first acute symptomatic E-coli urinary tract infection: early 99M Tc DMSA appearances. *Acta Paediatr Scand* 1989; **78:** 923–9.

Drs Jakobsson, Berg, and Svensson comment: Drs Beattie and MacKenzie raise an important question. In our paper we have used the same criteria for renal scarring as used by Rushton *et al.*[1] The results of both studies are similar. We consider a defect, still present on a DMSA scan after two years as a thinning or flattening of the renal parenchyma, and on the same site as the original insult, to be a sign of irreversible renal damage. Moreover, a scar situated on the front or on the back of the kidney will not disturb the cortical outline when only a posterior projection is used, as was the case in our study. It is difficult to believe that proximal tubular cells not functioning after two years from the insult will return to normal function. The anecdotal experience described by Dr Beattie and Dr MacKenzie may be explained by hypertrophy of remaining healthy proximal tubular cells resulting in an improvement of DMSA uptake, obscuring the original defect.

As the use of DMSA scan is becoming more popular, one will find signs of permanent renal damage in children after acute pyelonephritis more frequently than previously when intravenous urography was used. We will not know for some time how we should interpret many of these defects seen on a DMSA scan but not on intravenous urography in terms of risk for later complications. The term 'renal scarring' is well defined as it appears on intravenous urography and the term 'parenchymal reduction' is used for kidneys with signs of parenchymal thinning only. Maybe we should proceed in the same manner with regard to findings on DMSA scan, reserving the term renal scarring for kidneys with reduced DMSA uptake without preservation of the cortical outline, and parenchymal reduction or thinning for those with reduced uptake with preservation of the cortical outline. In both cases, however, the kidney is permanently damaged.

1 Rushton HG, Majd M, Jantausch B, Wiedermann BL, Belman AB. Renal scarring following reflux and nonreflux pyelonephritis in children: evaluation with ^{99m}technetium-dimercaptosuccinic acid scintigraphy. *J Urol* 1992; **147:** 1327–32.

Acute hepatitis due to brucella in a 2 year old child

EDITOR,—Liver involvement is frequent in acute and chronic brucellosis. Usually an increase in transaminase values and a mild hepatosplenomegaly occur, sometimes an acute hepatitis develops, but it is rarely the only clinical manifestation of the infection.[1] We report a case where hepatitis was the only manifestation of acute brucellosis.

The patient, a 2 year old boy from Sicily, with an unremarkable history, was found by chance to have hepatosplenomegaly and high transaminase values (aspartate aminotransferase (AST) 220 IU/l; alanine aminotransferase (ALT) 570 IU/l). Because of the persistence of this clinical picture, he was admitted to our department one month later. Clinical examination showed good general condition, apyrexia, hepatosplenomegaly, inguinal and laterocervical microadenia, and normal neuropsychic development. Laboratory tests showed haemoglobin 122 g/l, platelet count 155×10^9/l, white cell count $5{\cdot}3\times10^9$/l, erythrocyte sedimentation rate 5 mm/hour, AST 793 IU/l, ALT 1184 IU/l, alkaline phosphatase 805 IU/l, γ-glutamyltransferase 72 IU/l, and lactate dehydrogenase 1314 IU/l. Abdominal ultrasonography showed moderate hepatosplenomegaly, without any structural or morphological changes. Common causes of viral, metabolic and immunological liver damage were excluded by specific assays. Because of a further increase in transaminase values (AST 1065 IU/l, ALT 1552 IU/l), liver biopsy was performed. The biopsy specimen showed a picture of a specific reactive hepatitis with microgranulomas. The possible causes of liver granulomatosis were investigated.[2] Mantoux intradermal reaction and serological test for chlamydia, mycoplasma, and rickettsia were negative. The Wright reaction was positive (*Brucella abortus* 1:640, *Brucella melitensis* 1:160). The diagnosis was confirmed on a liver biopsy specimen by indirect immunofluorescence, using specific monoclonal antibody ISS/32.[3]

A more accurate study of the patient's history showed intake of homemade fresh cheese prepared by local shepherds.

After recommended treatment,[4] liver function tests returned to within the normal range and the titre measured by the Wright reaction decreased (*B abortus* 1:80, *B melitensis* 1:20).

This case shows that acute hepatitis may represent the first and the only manifestation of brucellosis. Brucellosis must be considered in the differential diagnosis of hepatitis, especially in patients coming from endemic regions.

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1 Titone L, Scarlata F, Giordano S. Epatic involvement of brucellosis. *Giornale Malattie Infettive e Parassitarie* 1987; **39:** 46–8.
2 Harrington PT, Gutitèrrez JJ, Ramirez-Ronda CH, Quinones-Soto R, Bermudez RH, Chaffey J. Granulomatous hepatitis. *Rev Infect Dis* 1982; **4:** 638–55.
3 Adone R, Ciuchini S, Pistoia C, Marcon G, Piccininno G. Protective activity to murine experimental brucellosis conferred by monoclonal antibody ISS/32 anti-B abortus. *Journal of Veterinary Medicine* 1991; **38:** 397–400.
4 Lubani MM, Dudin KI, Sharda DC. A multicenter therapeutic study of 1100 children with brucellosis. *Pediatr Infect Dis J* 1989; **8:** 75–8.