LETTERS TO THE EDITOR

Haemophilus parainfluenzae

EDITOR,—We would like to describe the first report of *Haemophilus parainfluenzae* urinary tract infection occurring in the paediatric population, a case which highlights the importance of looking for more fastidious organisms in selected patients.

Case report

A 2 year old boy was admitted with dysuria, intermittent vomiting, and abdominal pain. Ten weeks earlier he had undergone a distal hypospadias repair which was complicated postoperatively by mild penile inflammation; a wound swab grew low numbers of *Pseudomonas aeruginosa* but the inflammation settled spontaneously and was not treated with antibiotics. Two weeks later he developed persistent dysuria. On examination the hypospadias wound was clean with no evidence of cellulitis. He had not received antibiotics before admission and was started on cefuroxime.

Microscopic examination of a clean catch urine showed neither white nor red cells, but there were a few white cell casts and many small Gram negative bacilli. No growth was seen on CLED agar, however, in view of the microscopy results, a sample of urine was plated out on to chocolate and blood agar. After incubation the chocolate plate revealed a heavy pure growth ($>10^5$/ml) of bacteria, subsequently identified as *H parainfluenzae*, based upon a growth requirement for factor V (but not X) and a positive ALA (δ-aminolaevulinic acid) disk test (MAST Diagnostics) and which was sensitive to cefuroxime. A blood culture taken on admission showed no growth. He has made a complete recovery clinically but is to be reviewed on an outpatient basis.

Although *H parainfluenzae* has recently been associated with IgA nephropathy,[1] infections within the urinary tract itself are rare. The only study that has attempted to determine the prevalence of *Haemophilus* spp within the urinary tract has shown that, while urethral colonisation is possible, infection is probably very uncommon within the general paediatric population.[2] A review of the literature revealed only five reports of this organism being isolated in significant numbers from urine,[3–7] all in the adult population. Four of the five patients had chronic urinary tract problems and, as in this case, two had had instrumentation to the urinary tract (transurethral resection/lithotropy) in the weeks before the onset of infection. The prevalence of infection within this apparent at-risk group is unknown, but given the lack of growth on conventional isolation media and the usual antibiotic sensitivity of this organism, the number of infections is likely to be underestimated. The first reported urinary isolate[3] was able to grow rapidly in spiked samples of urine in contrast to other *H parainfluenzae* isolates from the upper respiratory tract, and certainly our isolate survived in high numbers in urine stored at 4°C for two days, so that there may be urine adapted strains.

For selected patients, those with chronic urinary tract disease and recent instrumentation in particular, the use of more nutritious agars should be considered where the results of standard culture do not accord with clinical symptoms and the results of urine microscopy.

A F MAGGS
D C OLD
Department of Medical Microbiology

P FOWLIE
Department of Child Health, University of Dundee Medical School, Ninewells Hospital, Dundee DD1 9SY

1 Suzuki S, Nakatomi Y, Sato H, Tsukada H, Arakawa M. Haemophilus parainfluenzae antigen and antibody in renal biopsy samples and serum of patients with IgA nephropathy. *Lancet* 1994; **343:** 12–20.
2 Schuit KE. Isolation of haemophilus in urine cultures from children. *J Pediatr* 1979; **95:** 565–6.
3 Ruhen RW, Genat RJ. Haemophilus parainfluenzae in urine. *Med J Aust* 1977; i: 756.
4 Blaylock BL, Baber S. Urinary tract infection caused by Haemophilus influenzae. *Am J Clin Pathol* 1980; **73:** 285–7.
5 Bäck E, Carlsson B, Hylander B. Urinary tract infection from Haemophilus parainfluenzae. *Nephron* 1981; **29:** 117–8.
6 Golledge C. Urinary infection and haemophilus species. *Med J Aust* 1987; **147:** 103.
7 Morgan MG, Hamilton-Miller JMT. Haemophilus influenzae and H parainfluenzae as urinary pathogens. *J Infect* 1990; **20:** 143–5.

Skull fractures in infancy

EDITOR,—Skull fractures in infants are frequent findings in victims of non-accidental injury,[1] and may also occur during childbirth. There are reports in textbooks that skull fractures may remain radiologically detectable for a period up to six months.[2] In a leading article in 1992 in this journal radiological dating of fractures was discussed in detail,[3] but it did not mention the healing of skull fractures. A skull fracture due to abuse in a child under 2 years is associated with very high mortality and morbidity.[4]

Paediatricians may need to know whether a skull fracture in say a 6 week old infant was sustained at birth or more recently; or indeed whether what appears to be a fracture really is one. Therefore it will be of great benefit to general paediatricians and radiologists in district hospitals if the following questions could be answered:

(1) How long do skull fractures take to heal radiologically?

(2) Does the healing process of skull bones differ from healing of long bones? If so how could these fractures be dated?

(3) When is a skull fracture not a fracture?

AMIT SARMAH
Ravensbourne NHS Trust, Phoenix Centre, Beckenham Hospital, Croydon Road, Beckenham, Kent BR3 3QL

1 Meservy CJ, Towbin R, McLaurin RL, Myers PA, Ball W. Radiographic characteristics of skull fractures resulting from child abuse. *American Journal of Radiology* 1987; **149:** 173–5.
2 Rudolph AM, ed. *Pediatrics.* 16th Ed. Appleton – Century Croft, 1977.
3 Chapman S. The radiological dating of injuries. *Arch Dis Child* 1992; **67:** 1063–5.
4 Brown JK, Minns RA. Non-accidental head injury with particular reference to whiplash shaking injury and medicolegal aspects. *Dev Med Child Neurol* 1993; **35:** 849–69.

Dr Wilson, paediatric radiologist, comments:

The time it takes a skull fracture to heal is so variable that it is in practice impossible to date skull fractures from radiographs. Part of the difficulty is that they heal by a somewhat different process from that seen in other bones and do not form visible periosteal reactions. Also, the bone is too thin for one to be able to see whether the fractured edge has been resorbed early in the healing process. Most do heal in a few days to months; a few fail to react or undergo slow, continuous absorption of the fractured edges – the so-called 'expanding fracture', thought to be due to leakage of cerebrospinal fluid from a torn dura, and requiring surgical repair. There are a few guidelines that point towards a non-accidental cause for a skull fracture: these include a widely displaced fracture suggesting extreme violence, or a stellate fracture suggesting a blow from a blunt instrument. In distinguishing birth trauma from non-accidental injury, it is often necessary to look for other radiological or clinical evidence of trauma in order to date the incident.

The child's skull is notorious for the number and variety of the normal radiolucent lines that may run across it. Even the sutures vary a good deal in prominence and position, and there are also arterial and venous vascular markings, synchondroses, and so on.

In general terms, fractures are unilateral, and darker for their width than nearby sutures (which do not pass through both tables of the skull in the same place) or blood vessels (which only indent the skull). When the fractured edges overlap, a thin white line may be produced. They may have sharp corners, unlike blood vessels they tend to taper even when not branching, and if they are displaced as well as angled, the width may appear to vary on different limbs of the zigzag.

In particular, anyone who has to decide upon the presence or absence of skull fractures on radiographs should have to hand a copy of a good atlas of normal appearances such as the one by Theodore Keats,[1] and not be embarrassed to ask for a second opinion if in doubt: it is well known that a consensus opinion in radiology is more reliable than that of a single radiologist.

BRENNAN WILSON
Department of Radiology, Booth Hall Children's Hospital, Charlestown Road, Blackley, Manchester M9 2AA

1 Keats TE. *Atlas of normal roentgen variants that may simulate disease.* 5th Ed. Chicago: Mosby Year Book Publishers, 1991.

Central nervous system tumours lack national studies

EDITOR,—The Childhood Cancer Research Group demonstrated that survival from acute non-lymphocytic leukaemia was improved for those children entered in national studies.[1] The same group showed this in acute lymphoblastic leukaemia in 1989.[2] In a population based study in the South West region from 1976–85, 245 children were identified with leukaemia and 54% of these were entered in national studies. A large percentage would have then benefited from the survival advantage of being entered. This contrasts sharply with the situation for children with central nervous system (CNS) tumours in the same period when of 164 children identified only 5% were entered on trials. A major reason for failure to enter these children into studies for CNS tumours was the lack of available national studies for which they were eligible. This situation still exists today. All children with acute leukaemia are eligible for the different national studies.