LETTERS TO THE EDITOR

Colposcopic genital findings in prepubertal girls assessed for sexual abuse

EDITOR,—We would like to respond to the commentary on our paper.¹ The diagnosis of child sexual abuse (CSA) is a jigsaw which includes physical signs and in Leeds is multi-disciplinary. The NHS, free at point of contact, health visiting, and case conference systems are important differences between British and US child protection practice.² In the UK, early referral of cases for paediatric examination and follow up (including re-examination) has taught us much about the progression of abuse and evolution of signs. Agreement on physical signs in CSA is likewise evolving here,³⁻⁴ utilising experience on both sides of the Atlantic. Peer review and a national paediatric child protection interest group are established. Earlier publications, with photographs, have described our population and practice.⁵⁻⁷ An atlas of physical signs is in press.⁸ Psychosocial information is part of all assessments (and research) and our case categories reflect this. Our references¹ indicate agreement with US colleagues on the significance of many findings. However, we take issue⁹ with papers¹⁰ which do not follow usual clinical practice but suggest that most children have 'normal' findings.

The commentary is misleading on a number of issues. For example, the statement: 'most [CSA] examiners would not agree that transverse hymenal diameter greater than 4 mm should be considered a sign of abuse', misconstrues the point. The Royal College report notes that 'the most commonly held view is that an orifice greater than 4 mm in the pre-pubertal girl is strongly correlated with abuse'.^{3, 11-13} The fact that there are considerable differences in hymenal diameters in studies of non-abused girls is not quoted and it is unclear why those of McCann *et al* have been favoured.¹⁴ Space does not allow a full analysis of every point but a further example of the commentary adopting an over inclusive and superficial approach is the statement that labial fusion is non-specific. It would be more informative to state that short labial adhesions may be common and innocent in infants in nappies but longer and thick fusion in older girls is much more unusual.

Our paper is a detailed descriptive study of children in whom sexual abuse was the major concern. It is worrying that the commentary suggests that, as there are no American published series describing gaping vaginal openings or dilated urethras, our observations are invalid. Why have such obvious findings not been described by other examiners? Further, a gaping opening appears to be associated with loss of hymenal tissue and should be important. Common sense dictates that if objects are repeatedly inserted into children, gaping or dilated orifices may result. There is also evidence cautiously emerging that a prolapsed urethra (and anus) may follow trauma.

While conflict remains in the literature on physical signs, it is hoped that those who would wish to put a strait jacket around this area of paediatric practice should not be

allowed to stifle careful observation based on sound clinical practice, which remains a vital part of good research. Physical signs will always have to be interpreted within the full clinical and social picture. We agree that there is still much for us all to learn.

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Transfer of critically ill patients with inhaled nitric oxide

EDITOR,—Inhaled nitric oxide (INO), a selective pulmonary vasodilator, has been shown to improve oxygenation and haemodynamic status in cardiorespiratory failure.¹⁻³ However, such patients often become dependent on this treatment during the first few days of administration, making the sudden discontinuation of the INO dangerous.

We have so far administered INO to nine patients (ages 6 hours to 4 years) during inter-hospital transport to our centre for extracorporeal membrane oxygenation (ECMO). Eight of these have been by road ambulance and one by air ambulance. Six of these patients were neonates with persistent pulmonary hypertension of the newborn and three were older infants and children with respiratory syncytial virus bronchiolitis, acute respiratory distress syndrome, and pulmonary hypertension complicating a cystic adenomatous malformation of the lung (CAM). Three of the patients were already receiving INO at their referring hospital and could not be weaned before transfer due to marked desaturation (arterial oxygen saturation <60%).

The other six patients were given a trial of INO at the referring hospital by the transport team in order to optimise oxygenation before transfer. All six had a significant response to a test dose of INO (20 ppm × 20 minutes) as defined by a greater than 10% improvement in arterial oxygen tension (mean (SE) before INO 5.2 (0.9) kPa and after INO 10.3 (3.2) kPa, $p < 0.05$).

There were no complications encountered during the transport of these patients on INO. Four of the patients transferred were placed on ECMO support; all four survived. The other five were left on INO treatment; three survived. The one patient who died had CAM of the lung and the other familial primary pulmonary dysplasia.

During the transports described above, the INO was introduced into the inspiratory limb of the ventilator (Draeger Bablog 2 or Oxylog).

With these ventilators gas flow changes with ventilator settings. Therefore changes in parameters such as peak inspiratory pressures, ventilator rate, minute volume and inspiratory: expiratory ratios will result in significant alterations to the concentration of INO. For this reason it is essential to monitor continuously the dose of INO being delivered. During transport we measure INO with a portable electrochemical nitric oxide analyser (Bedfont Scientific Instruments) with gas being sampled between the ventilator tubing and the endotracheal tube.

In conclusion, critically ill mechanically ventilated patients can be safely transported on INO. Its ease of administration, rapid clinical effect and apparent lack of toxicity, provided gas concentrations are monitored, make it an attractive additional drug for use during interhospital transfer of critically hypoxic patients. If INO becomes an established treatment then tertiary intensive care units, and in particular ECMO centres, will need to transport such patients on INO. We believe it is essential to monitor continuously INO concentrations during such transfers.

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Microcephaly and childhood non-Hodgkin's lymphoma

EDITOR,—Between 1968 and 1994 nine out of 194 newly diagnosed cases of non-Hodgkin's lymphoma seen at our department had microcephaly. The literature contains reports of associations between non-Hodgkin's lymphoma and immunodeficiency (congenital and acquired), chromosome instability syndromes, and malformations including microcephaly (Seemanova's syndrome).¹⁻⁵

Children with microcephaly and non-Hodgkin's lymphoma (NHL)

Case No	Sex	Age at diagnosis (years)	Type of NHL	NHL stage	Immunodeficiency	Family history	Mental development†	Outcome (survival)
1	F	8	*	II	IgA decreased	*	Retarded	Dead (4 months)
2	M	3	Probably B	III	*	Negative	*	Dead (2 weeks)
3	M	15	Probably T	III	Frequent respiratory infections	Positive for abnormalities‡	IQ 48	Dead, NED (15 months), pericarditis
4	M	7	T	III	*	Negative	IQ 61	Alive in I remission (38 months)
5	M	6	B	III	Decreased reaction to mitogen	Sister with cerebral palsy	IQ 76 (TM)	Alive in I remission (35 months)
6	M	10	T	IV	IgA decreased, low B cell, low T cell	Sister with microcephaly	IQ 77	Alive in II remission (28 months)
7	M	13	T	IV	IgG, IgA slightly decreased	Sister with microcephaly	IQ 64 (normal school)	Alive in I remission (29 months)
8	F	3	B	II	IgA slightly decreased	Negative	IQ 76 (TM)	Alive in I remission (18 months)
9	M	9	T	II	IgA decreased, low B cell low C4	Brother with hydrocephalus and foot malformation	IQ 87	Alive in I remission (4 months)

*Lack of information. †IQ measured on Wechsler scale or Termann-Merill (TM) scale.

‡Child from hospital for mentally disabled; lack of more precise information. NED=no evidence of disease.

The data of our nine patients are shown in the table. Familial microcephaly was seen in two patients. In three families other congenital malformations were found, and immunodeficiency was confirmed in six patients.

The outcome of treatment was not affected by the presence of microcephaly,⁶ despite treatment modifications to reduce toxicity (including omission of alkylating agents and/or irradiation of the central nervous system). Unfortunately, we were unable to perform complete immunological and cytogenetical examinations in our patients. However, there are reasons to believe that most of our cases were truly children with Seemanova's syndrome. It will be necessary to study more children to help explain the relation between congenital malformations, immunodeficiency, and malignancy.

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Ocular relapse in acute lymphoblastic leukaemia

EDITOR,—Ocular relapse associated with acute lymphoblastic leukaemia (ALL) is rarely seen nowadays as a result of improved treatment of childhood leukaemia. A 12 year old boy with ALL was treated in accordance with the recent UKALL protocol which did not include cranial irradiation. He developed leukaemic infiltration of his left eye while in remission and on maintenance chemotherapy, seven months after the original diagnosis. Lumbar puncture revealed central nervous system (CNS) relapse and he underwent local irradiation to the left orbit with a maximum dose of 800 cGy and subsequently reinduction chemotherapy and craniospinal irradiation. After initial partial resolution of signs, infiltrates persisted and 10 weeks later he underwent further craniospinal irradiation with 2400 cGy applied to the right and left cranium in 15 fractions with 6 MV x rays, resulting in a gradual improvement over a three month period and he remained in remission.

By six months only residual pigmentary changes were observed in the fundus and maintenance treatment was stopped due to neutropenia with the CNS remaining clear of blast cells. However, two weeks later, he developed further ocular recurrence which temporarily resolved on treatment, and subsequently further ocular and CNS relapse, and died 11 months after his original ocular presentation.

Ocular involvement in leukaemia is most commonly a consequence of associated haematological abnormalities and usually occurs when the patient is in relapse. Anaemia may precipitate a leukaemic retinopathy and hyperviscosity may give rise to microaneurysms, capillary closure, and retinal neovascularisation. Less frequently direct invasion of leukaemic cells may lead to ocular infiltration which carries a poor prognosis and is associated with CNS relapse.¹ A recent study revealed 96% of children with ALL died within 28 months of onset of ocular signs. Of those patients with ocular manifestations in ALL, 82% had CNS leukaemia.² It has been shown that intrathecal methotrexate

does not reach the eye, and its effect on tumour cells in the optic nerve is demonstrable only as far as the termination of the subdural space posterior to the globe.³

A multicentre study of leukaemic ophthalmopathy suggests that those leukaemic patients with ocular involvement, in first complete remission, may be cured with chemotherapy and high dose radiotherapy to the affected eye,⁴ and it is still possible to achieve remission after a second ocular relapse. If the eye is the sole sanctuary site, then enucleation may be indicated. Lo Curto and associates presented a case of isolated ocular relapse in which enucleation was undertaken after a third ocular relapse. The eye had previously been treated with chemotherapy and radiotherapy with a dose of 390 cGy at first relapse and 20 Gy at the second. The patient remained well 11 years after enucleation. It has been proposed that a dose of 20 Gy may be ineffective against ocular leukaemia, and that higher doses of over 30 Gy may be necessary to eradicate ocular leukaemic cells.⁴

Although ocular sanctuary is a well known but very rare form of relapse, its occurrence is associated with an extremely poor prognosis and high dose radiotherapy should be urgently instituted. As routine cranial irradiation is no longer mandatory for standard risk cases, it is important that such cases presenting with ocular relapse should be thoroughly assessed to ensure adequate evaluation of current treatment protocols.

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Guidelines for the establishment and operation of human milk banks in the UK

EDITOR,—Interest in milk banking (the collection, storage, and processing of donor mothers' breast milk), has lately reawakened. The last document to give guidance in this area was published by DHSS in 1981,¹ but there have been many changes since then, including the emergence of HIV and other viral infections. A milk banking symposium in March 1993 marking the closure of Sorrento Maternity Hospital, Birmingham (which had been in the forefront of milk banking in the UK²) highlighted the need to update UK guidelines. An *ad hoc* working party was therefore established and new guidelines drawn up similar to those published by the Human Milk Banking Association of North America.³

The selection of donor mothers was considered very carefully and the guidelines