Twenty two percent of these children with CDH were still not walking at the age of 18 months. Thus the commonly taught axiom that CDH does not delay the onset of walking is incorrect. Indeed, in a later study that I made in 1962, into the way in which the diagnosis came to be made in 244 cases of CDH (average age 19 months), 'late walking' was the fourth most common form of presentation (11%), after 'abnormal gait' (53%), 'short leg' (29%), and 'associated deformity' (21%).⁴ No correction was made for gestational age but then CDH is uncommon among preterm infants.^{4,5} In conclusion, while the late diagnosis of CDH is becoming less common, the failure of a term infant to walk at 18 months remains a useful marker for this condition.

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Drs Johnson, Goddard, and Ashurst comment: While acknowledging Dr Dunn's point that some degree of delay in achieving motor milestones is to be expected among very preterm infants, the high rate of impairment among the late walkers in this group means that 'immaturity' should not be too readily accepted as the sole reason for late walking. As so often in clinical practice, a balance needs to be achieved between causing unnecessary anxiety to parents and yet remaining aware of the risk of associated impairment in all late walkers, particularly those who were born very preterm.

There was just one infant among the late walkers who had associated congenital dislocation of the hip. This had been detected, however, before the age of 18 months.

Lichen sclerosis

SIR,—As a paediatrician who has encountered eight new cases of lichen sclerosis in young children over a period of 18 months, I would like to add my observations to those of Drs Priestley and Bleehan and Dr Harrington.^{1,2}

The earliest of these cases was a girl aged 6 years, who presented with dysuria and bleeding after a visit to a male neighbour—a regular babysitter. The girl gave a clear account of repeated sexual abuse involving frictional interlabial trauma. The neighbour admitted to these offences as well as to those on other children has since served a prison sentence. The findings were of gross lichen sclerosis with pallor, friability, and haemorrhage, with the hymen showing partial disruption at one site.

Of the other seven cases seen, a further two were associated with clear histories of sexual abuse. In another child, extensive hymenal damage was associated with behavioural and family factors, and abuse remains a worrying possibility.

Lichen sclerosis has therefore been noted by me in three children where sexual abuse was described, as well as in children presented by protective agencies, because of this and other behavioural symptomatology.

The suggestion that the presence of lichen sclerosis in some way excludes sexual abuse must be unacceptable. Indeed, if I were to use the statistical reasoning of Dr Berth Jones *et al* in terms of my own limited experience,³ the presence of these changes would be linked to a sexual abuse diathesis in such significant numbers (50%), as to turn this argument on its head with equal frailness.

If lichen sclerosis is the outcome of chronic trauma in some children, then it must follow that every case requires careful study. Perhaps we should also be considering the possibility that these changes may relate to a local immune response to the recurrent presence of substances like semen, or contraceptive lubricant, both in adults and children.

My personal approach is to accept lichen sclerosis in children as a spontaneous unexplained phenomenon. I would not accept hymenal disruption, sexualised behaviour, or a child's disclosure as part of this phenomenon. The presence of lichen sclerosis in these circumstances can neither be considered 'spontaneous' nor 'unexplained'.

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Surveillance for anaemia: risk factors in pattern of milk intake

SIR,—We were interested to read Mills' study from east London, of the prevalence of anaemia in early infancy,¹ and fully agree with his conclusion that iron status is closely related to the type of milk consumed.

Prompted by a high reported incidence of iron deficiency in China, and concern in Hong Kong that babies were having a poor iron intake during weaning, in the mid 1980s we investigated the iron status of a sample of full term healthy Hong Kong Chinese babies. These babies were being followed up to monitor their growth and nutritional state and most were formula fed. Of our initial cohort of 174 babies only 8, 4, and 2% were breast fed at 2, 4, and 6 months respectively.²

Mean (SD) daily iron intakes at 6, 12 and 18 months were 8(5), 9(3), and 8(3) mg respectively, comfortably within the WHO dietary allowance of 5-10 mg. The mean (SD) haemoglobin concentration in 123 babies, at 18 months was 124 (8.9) g/l. Eleven had a haemoglobin concentration <110 g/l. Of these, seven had β thalassaemia trait, two showed a satisfactory response to iron treatment (2 mg/kg/day) for three months, one had

an unchanged haemoglobin, and one defaulted further follow up. Three of 112 babies with haemoglobin of >110 g/l had a mean corpuscular volume <70 fl. Mean (SD) serum ferritin concentration at 18 months in 128 infants was 155.9 (24.8) μ g/l, with only one having a concentration <7 μ g/l.

We attributed this very low (~2% excluding β thalassaemia trait) incidence of iron deficiency to infant feeding practices, which provide: (i) an adequate iron intake during the weaning period from fortified milk formulas, iron fortified cereals, meat, and fish and (ii) a negligible consumption of pasteurised cows' milk with its low iron content and risks of causing occult intestinal blood loss.

These practices contrast with those described in Mills' study,¹ where, with an incidence of anaemia of 22%, continued breast feeding, with the early giving of pasteurised milk, were risk factors for anaemia. But the value of prolonged formula feeding, with iron fortified milks that comply with international standards, as one means of lessening the incidence of iron deficiency in early childhood, was evident from both our investigations.

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Collecting 24 hour urine samples from children

SIR,—Collection of urine over several hours for metabolic investigations in infants and toddlers is notoriously awkward: disposable napkins frequently produce contaminated samples, and modified cots are only practicable for small infants.^{1,2} The following modified technique imposes few restraints on the patient, and is therefore metabolically accurate, and may be used for accurate collections of up to 72 hours.³

A bag is constructed with a narrow tube inlet and a larger outlet. The Hollister 24 hour paediatric urine collection bag is particularly appropriate, having a reinforced outlet provided. The inside pocket of the bag is removed and the bag resealed using a heated bar sealer. A nasogastric tube of approximately 8F gauge is inserted 2 cm through a small hole in the top corner of the bag, and sealed in place using spray on plastic adhesive (for example, Dow Corning) and plastic adhesive tape.

The air diaphragm pump (for example, the Charles Austen series), collection vessels, and tubing are attached to provide an air flow of some 3 l per minute, with positive pressure directed into the top of the collection bag, and

Urine collection system.

negative pressure through the collection vessel to its lower end (see fig). For trace element analysis, the tubes, bag, and collection vessel should be rinsed in 0.01N hydrochloric acid, followed by deionised water. Rubber bungs should be avoided as they contain both zinc and iron.

The collection bag is applied with spray on plastic adhesive (Dow Corning). For boys, the penis is drawn into a round hole in the side of the bag, which is then sealed over the scrotum and pubis. For girls, an oval opening is more successful; the seal should include the perineal body. The inlet tube should be secured plastic tape around the lower abdomen, and the free lower end of the bag should be wrapped with a strip of absorbent material, which is changed when the patient passes stool.

The most common complication encountered is urinary leakage, usually the result of the patient playing with the collection bag or tubes. A well fitting nappy and toys attached to the tubes may prevent this complication.

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Glucose polymer regimens and hyponatraemia

SIR,—In response to the recent report of hyponatraemia associated with the use of glucose polymer feeds in the management of metabolic decompensation,¹ we would like to report our practice in the management of such cases.

It is well documented that feeds used in metabolic decompensation should not have an osmolality greater than 500 mmol/l.² While a generous intake of energy is desirable, fluid and osmolality tolerance will restrict energy provision.

In mild to moderate metabolic stress we advise volumes of oral rehydration solution (Dioralyte, Rorer) and separate drinks of up to 15% strength (w/v) glucose polymer solution. The use of separate solutions eliminates the problems of hyperosmolar feeds and yet provides adequate fluid and a sufficient energy intake over the temporary period of metabolic decompensation. This is generally well tolerated and after 12 to 24 hours the appropriate feed regimen is reintroduced, initially at a reduced strength with regrading to full strength achieved usually by 24 hours.

The authors' reminder of the hazards of

hyperosmolar feeding during incidental illness in children with metabolic disease is timely. While glucose polymers have improved the treatment of metabolic disease, their inappropriate use can lead to further complications.

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Ozone in medical gases

SIR,—Ozone is a powerful oxidant and is recognised as causing lung fibrosis if over-exposure occurs in adults. We investigated the possibility that concentrations of ozone in medical gases could be such as to be a factor in the development of fibrotic change in lungs in the neonate. Ozone is constantly forming in the atmosphere, particularly in the stratosphere where energetic extraterrestrial photons interact with molecules in the air. Ozone decays continually, normally giving a ground level concentration of around one part per billion.

Cylinders of medical oxygen were analysed using a Dasibi Environmental Corporation model 1108 UV photometric analyser. The limit of detection of the instrument is one part per billion. Six D size oxygen cylinders were analysed, some filled the same day as the analysis, as well as cylinders filled on previous days. In all cylinders no ozone was detected, indicating concentrations of less than one part per billion. During the course of the analysis the ozone concentration of the ambient air in the laboratory was also monitored and readings in the range of 0-6 parts per billion were obtained.

Failure to detect ozone in any of the oxygen cylinders is reassuring, but is not unexpected due to the high reactivity of ozone.

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