et al, recently reported 14 cases which they documented in this manner.[1]

Within a period of six months we have seen three babies with ALTE from three unrelated families. All three had been discovered limp, cyanotic, and apparently lifeless during their afternoon nap. Petechial haemorrhages were found on the face and neck of two of the babies on admission. No other episodes occurred during observation in hospital or the follow up period.

These would probably have remained unexplained unrelated cases were it not that when the third case was brought to the casualty department by the family doctor he was accompanied by the babysitter who had discovered the baby. She was recognised by one of the nursing staff as a regular attender at the casualty department with minor wounds that were suspected of being caused by automutilation and she had made several allegations of being attacked or raped.

In the ensuing discussions it came to light that this woman was the babysitter who had also been involved in the first two cases. She has since been investigated by the police. However, in spite of strong suspicions of imposed upper airway obstruction of the babies by her, there has been insufficient evidence to bring her to trial.

CVS as described by Southall *et al*[2] would have failed in these cases, because it is usually only the parents or very close relatives who are allowed to be continually present with the baby in hospital. Very thorough history taking especially concerning the surrounding circumstances remains extremely important in investigating every case of ALTE especially if imposed upper airway obstruction is suspected.

A M OUDESLUYS-MURPHY
W J VAN YPEREN
Department of Paediatrics, Zuiderziekenhus, Groene Hilledijk 315, 3075 EA Rotterdam, The Netherlands

1 Samuels MP, Mc Claughlin W, Jacobson RR, Poets CF, Southall DP. Fourteen cases of imposed upper airways obstruction. *Arch Dis Child* 1992;**67**:162–70.
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Anal abnormalities in childhood myotonic dystrophy

SIR,—The paper by Reardon and colleagues on abnormal anal signs seen in myotonic dystrophy is worrying on several counts.[1] The physical signs are described inadequately which makes interpretation of the paper difficult. The Royal College of Physicians report,[2] while referenced, is not heeded in the need for a consensus on definition and method of examination. Thus 'a reflex dilation of the anus was observed on parting the buttocks'. How was the child examined, for how long, and was this a dynamic sign and what degree of dilation was observed? It is noteworthy that only one child in six was said to demonstrate this sign but all had anal laxity.

The illustration shows a degree of anal laxity we have never seen, is this laxity or dilation? Was this degree of gaping achieved on merely parting the buttocks? Was the child constipated and demonstrating the 'visibly relaxed anus' of Clayden?[3] This child was 15 years old and had had a lifetime of soiling, what had earlier examinations revealed and to what treatments and manoeuvres had she been subjected?

Single physical signs are rarely diagnostic and in making a diagnosis of child sexual abuse the jigsaw must be carefully constructed.[4] To begin an investigation on the basis of a child with a known bowel dysfunction and anal laxity is clearly problematic but in the context of a girl who is alleging abuse it is, in child protection terms, quite proper. Advice to doctors stated 'It is important to take what the child says very seriously, and to spend time listening to what the child has to say.'[5] It is also evident that children with special needs are at risk of abuse and to recognise abuse in children with communication problems requires particular skill.[6] Children do need protection from the trauma of wrongful diagnosis but similarly physicians have until recently failed their patients by their inability to recognise maltreatment.

Finally, what proportion of children with myotonic dystrophy have bowel disorders or an abnormal anus on examination? What proportion have no abnormality? The association of two conditions does occur.

JANE WYNNE
CHRISTOPHER HOBBS
Community Child Health, Belmont House, 3/5 Belmont Grove, Leeds LS2 9NP

1 Reardon W, Hughes HE, Green SH, Lloyd Woolley V, Harper PS. Anal abnormalities in childhood myotonic dystrophy—a possible source of confusion in child sexual abuse. *Arch Dis Child* 1992;**67**:527–8.
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Drs Reardon, Hughes, and Green and Professor Harper comment:

The comments of Drs Wynne and Hobbs reflect the difficulties with which clinicians are obliged to grapple in attempting to confirm or refute a suspected diagnosis of child sexual abuse. Foremost among these is the non-specificity of single clinical signs. However, in many instances, abnormalities of anal physiology do contribute significantly to decisions with far reaching effects for patients and families. It is precisely because we absolutely agree with Drs Wynne and Hobbs that 'children need protection from the trauma of wrongful diagnosis' and because of the central role which anal abnormalities and interpretation thereof often has in reaching diagnoses of sexual abuse that we felt prompted to submit our report.

The report did not pretend to be a scientific treatise of anal sphincter dysfunction in myotonic dystrophy. Our hope in sharing these non-specific clinical observations is to make colleagues aware of added potential pitfalls which attend the diagnosis of child sexual abuse in myotonic dystrophy. We feel that shared clinical experiences such as ours are of value to clinicians in avoiding inappropriate diagnoses of child sexual abuse.

Prostacyclin concentrations in haemolytic uraemic syndrome after acute shigellosis

SIR,—Although the epidemiology of certain forms of the haemolytic uraemic syndrome (HUS) has become clearer in recent times, pathogenic details remain largely unresolved. The role, if any, of prostacyclin (PGI2) in the diarrhoea-associated forms of HUS (D+HUS) cannot be deduced from previous reports and the study by Alam *et al* on patients with shigella induced HUS adds to the confusion.[1] In the following we acknowledge, however, that in past European and American studies the aetiology of D+HUS is likely to be verocytotoxin producing *Escherichia coli*, which although similar is not homologous with shigella induced HUS.

Prostacyclin is probably not a circulating hormone in man: its plasma half life is brief and intact molecules cannot be measured directly in clinical practice. Thus the term 'prostacyclin concentration' is at best an extrapolation. Two main approaches have been used to identify abnormalities of PGI2 metabolism in disease states. The more direct of these is to measure stable degradation products such as 6-keto PGF-1α in biological fluids. The commonly used radioimmunoassay kits give plasma estimates which exceed those of the more specific gas chromatography and mass spectrometry. Blood sampling in itself induces artifacts, and although the urinary excretion of degradation products may be more consistent in health, this is unlikely to be a reliable method when renal function is rapidly changing. Both Stuart *et al*[2] and Turi *et al*[3] found plasma concentrations of prostacyclin metabolite to be increased in D+HUS at onset.

The second approach has been to determine whether plasma or serum stimulates generation of prostacyclin by endothelial tissue in vitro. Prostacyclin release into the supernatant medium is measured indirectly either by bioassay or as 6-keto PGF-1α. Published results are conflicting; Schlegel *et al*[4] and Levin[5] found that prostacyclin generation was promoted by plasma, whereas Turi *et al*[2] and Siegler *et al*[6] showed reduced production. Methodological differences may explain these inconsistent findings, but abnormalities of plasma stimulating factors or inhibitors have in any case not been shown to reflect disturbances of prostacyclin metabolism in vivo.

Unfortunately Alam *et al* did not make clear which method they used. The result section and abstract refer to plasma concentrations of 6-keto PGF-1α suggesting direct measurement. However, their method refers to radioimmunoassay for 'prostacyclin', but also to generation of 6-keto PGF-1α by rabbit aortic rings in response to patients' plasma.

C M TAYLOR
C J LOTE*
Department of Nephrology, The Children's Hospital, Ladywood Middleway, Ladywood, Birmingham B16 8ET
**Department of Physiology, University of Birmingham*

1 Alam AN, Abdal NM, Wahed MA, *et al*. Prostacyclin concentrations in haemolytic uraemic syndrome after acute shigellosis in children. *Arch Dis Child* 1991;**66**:1231–4.
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3 Turi S, Beattie TJ, Belch JJ, Murphy AV. Disturbances of prostacyclin metabolism in children with hemolytic uremic syndrome and in first degree relatives. *Clin Nephrol* 1986;**25**:193–8.

4 Schlegel N, Maclouf J, Loirat C, *et al.* Absence of plasma prostacyclin stimulating activity deficiency in hemolytic uremic syndrome. *J Pediatr* 1987;111:71–7.
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Interleukin-1α and soluble interleukin-2 receptor in atopic dermatitis

SIR,—Dr Agata and colleagues reported enhanced interleukin-2 (IL-2) activity in blood mononuclear cells from patients with food sensitive atopic dermatitis following allergen exposure.[1] Soluble interleukin-2 receptor (sIL-2R) is an indirect marker of T cell activation by IL-2 and raised concentrations have previously been described in adults with atopic dermatitis.[2] However, in vitro studies suggest that IL-2 secretion by T helper cells is not only antigen driven but it is also dependent on interleukin-1 (IL-1) expression by cells of the macrophage/monocyte series; IL-1 also upregulates the expression of high affinity receptors for IL-2 on T helper cells type 1.[3] In order to investigate this hypothesis, we measured IL-1α and sIL-2R using an ELISA technique. Twenty three children were assessed: 11 with atopic dermatitis and 12 non-atopic normal controls. The results are shown in the table.

	Controls	*Atopic dermatitis*	*Unpaired t p value*
Mean age (years)	9·2	8·6	NS
Range	4–14	4–16	
IL-1α (pg/ml)	163	369	p<0·02
95% CI	56 to 269	232 to 505	
sIL-2R (U/ml)	189	377	p<0·02
95% CI	75 to 301	262 to 492	

CI=confidence interval.

There was a strong direct correlation between IL-1α and sIL-2R ($r=0·67$, $p<0·001$) which suggests that expression of the two are dependent. While no apparent relationship existed between either cytokine concentration and disease severity using the criteria of Rajka and Langeland[4] or IgE concentrations, our results demonstrate that there is enhanced endogenous secretion of IL-1α, and increased stimulation of IL-2 receptors, in children with atopic dermatitis. These findings suggest that these cytokines may contribute to the inflammatory process in atopic dermatitis and are consistent with the observations of Dr Agata and colleagues.

P GREALLY
M J HUSSAIN*
J F PRICE
*Departments of Child Health, Thoracic Medicine, and Immunology** King's College Hospital, London SE5 9PJ*

R COLEMAN
Department of Dermatology, Hospital for Sick Children, Great Ormond Street, London WC1N 3JH

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Pulse oximetry reference values at high altitude

SIR,—Lozano *et al* in their article on pulse oximetry reference values at high altitude conclude that their reference values could be used for the interpretation of oxygen saturation at high altitude in Bogota and other cities at a similar altitude.[1] However, the authors have failed to take into account potential sources of error.

The fall in arterial oxygen saturation at altitude is due to the fall in barometric pressure rather than the gain in altitude *per se*. There is a corresponding fall in atmospheric oxygen tension (Po_2) with fall in barometric pressure (at 2000 m atmospheric $Po_2=124·9$ mm Hg (16·6 kPa) and at 3000 m $Po_2=110·2$ mm Hg (14·7 kPa)[2]) such that the author's proposed reference ranges are not suitable for use at altitudes other than that of Bogota (2640 m) as even small changes in altitude will alter the availability of atmospheric oxygen and the arterial oxygen saturation.

Furthermore, at a given altitude the barometric pressure changes with local variations in weather and to a greater extent with the season such that in mid-summer the barometric pressure may be 11 mm Hg (1·47 kPa) higher than in winter.[3] Moreover, there is an equatorial bulge in the atmosphere such that the barometric pressure at a given altitude near the equator is higher than at the same altitude nearer the poles by 17 mm Hg (2·27 kPa).[4] These pressure variations will affect the partial pressure of oxygen in the atmosphere. Although of little significance at sea level pressures, these changes in atmospheric Po_2 in the already 'desaturated' infant at altitude may significantly affect arterial saturation.

Taking these observations into account the authors may find that there are both seasonal and latitudinal variations in arterial oxygen saturation along with the changes in barometric pressure such that their proposed reference ranges should be intrepreted with caution in summer and probably not used at all at latitudes or altitudes distant from Bogota.

Before recognition of these changes in barometric pressure with season and latitude the ascent of Everest (8848 m) without supplementary oxygen was thought impossible. In 1978 Messner and Habeler proved the physiologists wrong.[5]

A J POLLARD
The Children's Hospital, Ladywood Middleway, Ladywood, Birmingham B16 8ET

1 Lozano JM, Duque OR, Buitrago T, Behaine S. Pulse oximetry reference values at high altitude. *Arch Dis Child* 1992;67:299–301.
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Randomised trial of nutrition for preterm infants after discharge

SIR,—This interesting study draws attention to a possible need for preterm infants to be fed a slightly higher protein, mineral, and energy-containing milk in the first nine months after birth.[1] However, the authors include detailed tables on feed tolerance, stool number, size and consistency, and skinfold thickness (which show no significant differences between diet groups) yet choose not to present any data on growth of weight, length, and head circumference on which they base their conclusions of a possible advantage for this specially designed formula. It is not enough to say that differences are apparent on visual inspection of the charts. In fact, looking at these it seems to me that if there are any differences at all they take place for length and head circumference only in the first few weeks: after this the curves virtually parallel each other. Weight does fall off in those fed the standard infant formula but only between about 6 and 18 weeks. Growth data have been meticulously collected and the statistical methods explained in detail but without giving information, on velocity or incremental growth, it is difficult to evaluate the findings of the study.

D P DAVIES
Department of Child Health, University of Wales College of Medicine, Heath Park, Cardiff CF4 4XN

1 Lucas A, Bishop NJ, King FJ, Cole TJ. Randomised trial of nutrition for preterm infants after discharge. *Arch Dis Child* 1992;67:324–7.

Drs Lucas, Bishop, and Cole comment:

Professor Davies seeks further information on growth in our study on postdischarge nutrition. The growth data collected at two weekly intervals are shown in our paper graphically (tabulation would have been cumbersome and the graphic data show centile placing). Given the small sample size, as expected, relatively few individual two week measurements showed a significant difference between groups. Nevertheless, in view of the consistently increased weight, length, and head circumference at every two week period up to 9 months' corrected age in the infants fed the fortified diet, we explored whether the overall growth trajectory was different between groups. This is a statistically robust procedure as, unlike the *t* test comparisons of individual two weekly data points, we were taking the entire growth data set for each diet group into account. As growth trajectory is not linear, but curvilinear, growth velocity was calculated from a quadratic fit of the data. The significant differences we reported in length and weight gains between feed groups would be difficult to describe numerically, short of presenting equations for the quadratic curves, and we elected not to. We emphasise, however, that our statistical analyses confirm a clear advantage in weight and length gain ($p<0·005$ and $p<0·01$ respectively) for infants fed the fortified formula. The difference between groups in rate of head growth did not achieve significance, because the early divergence between groups tended to diminish later in the study period. Nevertheless, the head growth issue needs further exploration especially in view of our unpublished analyses that show a trend towards higher gross motor development scores at 9 months in infants fed the fortified