et al, recently reported 14 cases which they documented in this manner. 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 unreported if the babies had not been investigated. However, in one of the cases it was accompanied by relaxed anus' which prompted the baby's mother to take it to the hospital. It is important to take to what the child says very seriously, and to spend time listening to what the child has to say. It is also evident that children with special needs are at risk of abuse and to recognize abuse in children with communication problems requires particular skill. Children do need protection from the trauma of wrongful diagnosis but similarly physicians have until recently failed their patients by their inability to recognize maltreatment.

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

JANE WYNNE CHRISTOPHER HOBBS
Community Child Health,
W Belforne Street,
35 Belgrave Grove,
Leeds LS2 9NP


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, or 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 disease 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. In the following report from the last 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 PG12 metabolism in disease states. The more direct of these is to measure stable metabolites of prostacyclin such as 6-keto PGF-1a 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 and Turi et al found plasma concentrations of prostacyclin metabolites to be increased in D+HUS at onset.

Our 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-1a. Published results are conflicting; Schlegel et al and Levin2 found that prostacyclin generation was promoted by plasma, whereas Turi et al and Siegler et al showed reduced generation. 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.

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-1a suggesting direct measurement. However, their method refers to radioimmunoassay for 'prostacyclin', but also to generation of 6-keto PGF-1a by rabbit aortic rings in response to patients’ plasma.

C M TAYLOR
C J LOTE
Department of Paediatrics and Reproduction
The Children’s Hospital,
Loddonwood Midstowery,
Loddow, Birmingham B16 8ET
*Department of Physiology,
University of Birmingham

1 Alam AN, Abdal NM, Wahed MA, et al. Prosta-
