reflex anal dilatation occurs exclusively in cases of sexual abuse.

In a recent experience at this hospital, a 7 year old boy was admitted with non-specific abdominal pain, vomiting, and perianal soreness. Physical examination showed redening of the anus, an anal fissure, and marked reflex anal dilatation; a diagnosis of sexual abuse was considered on the basis of these findings. There were no other features to support the diagnosis, however, and the child seemed a cheerful, well adjusted boy who enjoyed an excellent relationship with his parents.

Before voicing our suspicions, further questioning showed recent onset of mild diarrhoea containing mucus. A barium follow through examination subsequently showed extensive changes of Crohn’s disease in the terminal ileum. Colono-scopically mucosal ulceration in this region, and histology of a biopsy specimen was consistent with Crohn’s disease. The child was started on an elemental diet, and his symptoms and signs resolved quickly.

Drs Hobbs and Wynne point out that they never base their diagnoses of abuse on the anal dilatation reflex alone, but in conjunction with other signs of anal damage such as ‘...fissures, veins, thickened perianal skin, scars...’ etc. These abnormalities, however, are all common findings in the 46% of children with Crohn’s disease who have anal involvement at time of presentation. Presumably, reflex anal dilatation occurs in some of these children in response to the pain often associated with perianal lesions in Crohn’s disease.

This case emphasises the importance of not relying on anal signs alone when diagnosing child sexual abuse. Crohn’s disease occurs in approximately 1 child per 10,000, and, although uncommon, does appear to be increasing in incidence. It would, therefore, benefit paediatricians and general practitioners to bear the condition in mind when diagnosing child sexual abuse on the basis of abnormal anal findings.

References

C M Evans and J A Walker-Smith
Department of Paediatric Gastroenterology,
St Bartholomew’s Hospital,
London EC1A 7BE

Anal dilatation and anal dilatation reflex associated with severe haemorrhagic colitis

Sir,

There is no doubt that dilatation of the anus and the anal dilatation reflex can be associated with penetrating anal abuse. Recent controversy has focused on these two features, particularly the latter, among the many manifestations of child sexual abuse. Clearly doubt exists regarding the validity of these two signs as sole indicators of anal sexual abuse in children. They have been described also in Crohn’s disease and constipation (the passage of a very large formed stool). It remains part of the controversy whether other local irritant or inflammatory conditions such as threadworm and rectal candidiasis can be associated with these signs.

We recently admitted a toddler, aged 13 months, with a three day history of watery diarrhoea. For the two days before admission his stools were bloody and frequent, he was lethargic and unwell but not febrile. He had not received any medication, and his parents denied administering rectal preparations or using rectal thermometers. His bowel motions had previously been regular and soft, without pain or bleeding.

On examination the perianal skin was reddened, and the anus was 2 cm patent, showing red anal and rectal mucosa with oedema blurring the pectinate line. Gently parting the buttocks resulted in reflex anal dilatation by approximately a further centimeter. There were no abnormal neurological signs. Careful questioning of both parents independently yielded the same story: the anus had become patulous on the previous day, from which time his diarrhoea had been “running out of him”.

During his admission he developed the colitis associated haemolytic uraemic syndrome. No pathogens or toxins were isolated from his stools, although it is thought that most patients with this form of the disease have been infected with verotoxin-producing Escherichia coli. Six weeks later his anus was entirely normal, and remained so on a subsequent admission.

We conclude that rectal inflammation may be associated with unexpected dilatation of the anus and the anal dilatation reflex. It would be pertinent to ask “how little (or how much) inflammation or irritation is required?” and “for how long after the inflammation has clinically settled can these signs persist?”

References

A R MAGNAY and J INSLEY
Birmingham Children’s Hospital,
Ladywood Middleway,
Birmingham B16 8ET

Immunisation in the immunosuppressed child

Sir,

We read with interest the annotation on immunisation in immunosuppressed patients. The author highlighted the problems associated with viral infections and the need to determine immune state. Chickenpox is a common infectious illness which can lead to major complications in immunosuppressed children. Unfortunately previous illness
either before or during the period of immunosuppression does not necessarily confer protection. This is illustrated by two of our patients.

Case reports

Case 1. A boy, aged 3 years 5 months, first presented with a history of a progressive petechial rash. Physical examination showed pallor, bruising, and hepatosplenomegaly. A bone marrow aspirate confirmed a diagnosis of acute lymphoblastic leukaemia. We were unable to obtain a history of chickenpox infection and viral titres were negative for varicella zoster virus. He was started on chemotherapy. After 18 months of treatment he was admitted with shingles affecting the ophthalmic division of the right fifth cranial nerve. He had a full course of acyclovir and recovered well. The titre for varicella zoster was less than 16. He was then restarted on maintenance chemotherapy but returned four months later with a severe illness associated with a typical chickenpox rash. He was again treated with acyclovir, and at the end of this illness his varicella zoster titre had risen to 128.

Case 2. A 4 year old boy presented with a short history of bruising, pallor, and lethargy. A diagnosis was made of acute lymphoblastic leukaemia. He had had a chickenpox illness during the first year of life and shingles six weeks before the onset of his acute leukaemia. Physical examination showed scarring in the right lumbar region consistent with shingles. Varicella zoster titre was negative. He was started on chemotherapy but had a relapse of his leukaemia two and a half years later. At this time his varicella zoster titre was 1 in 16. He was restarted on chemotherapy and three months later developed chickenpox with a typical rash which was managed successfully with acyclovir. He developed another mild bout of chickenpox two and a half years later, six months after completing chemotherapy.

These two patients illustrate the problems associated with varicella zoster infections and the difficulty in assessing immune state. It is therefore necessary to be vigilant and protect immunosuppressed chickenpox contacts with zoster immunoglobulin even in the presence of a history of exposure.

Conductive education for motor disorders

Sir,

As a research orientated physician and the father of a baby with cerebral palsy, I would like to comment on the recent controversy about conductive education, which you have recently highlighted in your journal.1 2 Dr Beach suggests that the parents of children who are severely handicapped or who are making only slow progress are drawn towards such ‘alternative forms of treatment’ because of suggestions by ‘family and friends, professionals, parents’ groups, or by the media’. In some instances this may well be true, but I suspect that most parents do so, at least in part, because they have come to realise that their children’s future is being compromised by the limitations of our health service. Should parents really be expected to accept one or two hours physiotherapy per week (in some instances not even that) as being sufficient for their children? What would you do? No parent would dispute Dr Beach when he suggests that ‘teams of dedicated professionals’ look after their children.1 The rub is that there are simply not enough of them to go around! While there are justifiable doubts about the value of conductive education, I fear that there can be no doubt that the British system of care for children with cerebral palsy is grossly underfunded, and I cannot

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The highly talented child

Sir,

I was most interested to read the article by Dr Lask, but wish to take issue with him on one point. In the first paragraph he comments ‘a 12 year old studying for an honours degree at Oxford University is clearly misplaced.’ I fear that Dr Lask is falling into the trap of confusing

gifted and highly gifted children. I agree that, if a child has an IQ of ‘only’ 140 or 150, she would be misplaced at such an institution at such an age. Many researchers have found, however, that the highly gifted, that is with an IQ of 170 and over, are peculiarly handicapped in the true meaning of the word: they have learning and social difficulties and are children with special educational needs.

In a famous experiment at Harvard, several children with exceptionally high IQs were admitted as young as 12, and in the report following their graduation the principal, Professor Eliot, stated that these students had shown fewer psychological problems, been happier, studied better and more effectively, and shown just as good results in their examinations as the ordinary students. He felt that the experiment had been a great success.

In the Stanford University’s Longitudinal Studies of Giftedness it was clear that if a gifted child was allowed full freedom to progress at his or her own rate he or she achieved their full potential and there were no psychological ‘hang-ups’ to sort out later. It has also become clear that, contrary to popular misconception, the highly gifted do not burn themselves out early, but continue to show superior mental powers right through into old age—as, indeed, one would expect.

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Reference