Succinic acid poisoning in dermatological treatment
SIR,—We read with interest the article on the successful treatment of a harlequin fetus.1 We note that the baby was initially treated with topical liquid paraffin containing 1% succinic acid every three hours. This resulted in significant percutaneous succinic acid absorption and systemic toxic manifestations. We have had similar problems with succinic acid toxicity after the topical application of succinic acid containing preparations for the management of skin disorders in two children.

One was an infant born at 38 weeks' gestation and weighing 2500 g. His skin was covered by a collodion like membrane which within 24 hours started to crack and peel in large plaques. Topical 2% succinic acid in aqueous cream was applied every three to four hours. On day 3 the baby vomited feeds and had a persistent metabolic acidosis. His succinate concentration was 3.1 mmol/l. Topical succinate treatment was stopped and a high fluid intake ensured by intravenous administration. He made a complete recovery.

The second patient was a 12 year old boy with severe ichthyosis. Treatment was started with topical 2% succinic acid in simple cream applied to the whole body twice daily. The succinate concentration was increased to 5% on day 3 of treatment and 10% on day 5. On day 8 he developed symptoms of succinate toxicity. His blood succinate concentration was 3.3 mmol/l. Topical succinate treatment was stopped. Intravenous fluids and bicarbonate were given and complete clinical and biochemical recovery was achieved after two days.

Topical succinate acid in paraffin or simple cream is supported by several authors as treatment in a number of skin disorders.2-4 Our two cases and the one described by Ward and Jones show that significant percutaneous succinate acid absorption can occur not only in newborn infants but also in older children, especially when succinate preparations of increasing strength are used. We suggest that all topical succinate treatment should be routine ly monitored with succinate blood concentration especially the first few days after onset or after any changes in treatment.

P GALEA
K M GOEL
Royal Hospital for Sick Children, Edinburgh
Glasgow G3 8ST

Lichen sclerosus
SIR,—In response to the article on lichen sclerosus by Dr Berth-Jones et al, I would like to take them to task on their quoting 'that there is absolutely no evidence to support any link between lichen sclerosus and sexual abuse.' I disagree that the chances of these two problems occurring together would seem very remote. I have had my care over 400 cases of lichen sclerosus and am well versed in the appearance of lichen sclerosus lesions at sites of trauma—for example, where wrist bands and bra straps cause friction. What is more, some patients report extra genital lesions occurring at sites of trauma—for example, oven burns.

It is my feeling that vulval trauma may precipitate vulval lichen sclerosus. I have seen two cases, a girl aged 4 and another aged 6, with definite lichen sclerosus of the vulva and they have also been sexually abused. Another 8-year-old girl with a 5-year history of ano-genital lichen sclerosus had probably also been sexually abused at the age of 3.

It is therefore important to accept that lichen sclerosus does occur at sites of trauma and that the trauma may be sexual abuse.

C J HARRINGTON
Royal Hallamshire Hospital, Glossop Road,
Sheffield S10 2JF


Lichen sclerosus and sexual abuse
SIR,—Dr Berth-Jones et al refer to the potential for misdiagnosis of severe anogenital lichen sclerosus as sexual abuse. They state that 'there is absolutely no evidence to support any link between lichen sclerosus and sexual abuse' and the 'chances of these two problems occurring together would seem very remote indeed'. We wish to report such a case.

We have treated 20 girls aged 4-11 years with lichen sclerosus over a two and a half year period. One girl, aged 7 years, presented with a four month history of intense diurnal frequency of micturition and wetting, and a sore vulva. The appearances were suggestive of early lichen sclerosus with patchy pallor and inflammatory plaques with minor fusion of the labia minora. Subsequently the changes became much more marked with characteristic thickened scrofulous white plaques, wrinkling, areas of acute infection, excoriation, and purpura. Substantial improvement occurred with topical clotobate. There have, however, been psychological problems and concern about possible abuse has persisted.

The girl has a complex social background, her stepfather being a single I offender (physical injury in a different family). Careful social and psychological investigation initially failed to support a diagnosis of sexual abuse by the next year, coincident with worsening of her lichen sclerosus, she developed frequent encopresis, not associated with faecal retention.

She eventually disclosed sexual abuse by her stepfather and much of her story was validated independently by her young brother.

A diagnosis of lichen sclerosus does not exclude sexual abuse, the diagnosis of which, as always, depends on non-verbal statements of the child than upon physical findings. There is the additional possibility that chronic trauma in the presence of low grade skin infection may on occasion lead to the changes of lichen sclerosus.

B L PRIESTLEY
S BLEHLEN
The Children's Hospital, Western Bank,
Sheffield S10 2TH


Fragile X mental retardation
SIR,—It is not only in the context of mental retardation without dysmorphic features that the general paediatrician should consider fragile X syndrome. It can present as a connective tissue dysfunction which mimics Ehlers-Danlos or Marfan's syndromes with joint hyperextensibility so severe as to lead to dislocation, high arched palate, and cutis hyperelastica. There may also be mild dysmorphic features such as epicantic folds and large malformed auricles. Cardiovascular features include mitral valve prolapse and dilatation of the ascending aorta.1 We agree that fragile X is an important diagnosis to exclude, and accept the need to be selective in deciding which patients should be screened, but would suggest that the screening criteria might usefully be broadened to include patients with connective tissue or cardiovascular abnormalities with non-specific developmental delay. We also consider that full neurological assessment should be performed in proved cases, because of the importance of diagnosing mitral valve prolapse for the need to appropriate prophylaxis against infective endocarditis in these patients.4

A REDINGTON
A BUSH
Department of Paediatric Cardiology, Brompton Hospital, Fulham Road, London SW3 6HP


Fetal ascites: an unusual presentation of Niemann-Pick disease type C
SIR,—We read with interest the paper of Maconochie et al we have recently seen a case of Niemann-Pick disease type C with an
Fragile X mental retardation.

A Redington and A Bush

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