Correspondence

Kawasaki disease

Sir,

I read with interest the letter of Morgan and Lynch (*Archives*, 1978, 53, 916), and should like to report 2 more white children in the UK—one Welsh and one English.

A 6-year-old boy from Anglesey was admitted in June 1978; 2 weeks previously he had developed fever and a blotchy red rash over the face with red macules on the trunk. He was seen by his doctor, diagnosed as having tonsillitis and scarlet fever, and given a 5-day course of ampicillin. His condition did not improve. On the 12th and 13th days of his illness (two days before admission) he developed peripheral limb pain, particularly around fingers, wrists, ankles, and toes. He was febrile, 38.6°C. There was bilateral cervical lymphadenopathy. The throat was inflamed. There was a grade-1 systolic murmur. Swelling of the fingers, hands, and wrists was noted. At this stage no firm diagnosis was made, but 3 days later prominent tongue papillae, oedema proceeding to skin peeling of the hands and feet, and a high ESR (100 mm in the 1st hour) were noted, and the diagnosis of mucocutaneous lymph node syndrome (MLNS) was made. Hb was 10.6 g/dl. Other investigations gave normal results: white blood count, urine analysis and culture, ASO, throat swab, chest x-ray, Paul Bunnell test, blood culture, and ECG.

After 18 days' illness his temperature remitted. The skin continued flaking from the periphery for another 2 weeks, but otherwise he was well, and has remained so for the subsequent 5 months.

A 3½-year-old girl from Merseyside on holiday in Wales presented with a 4-day history of fever for which a doctor had prescribed ampicillin. She had experienced a sore throat, a slight cough, and a few loose stools. The day before admission she had developed an erythematous rash with peeling on the face, trunk, and nappy area.

On examination she had a fever of 39.5°C. There was cervical lymphadenopathy, and an erythematous rash on the face, upper trunk, and nappy areas, with peeling around the nappy area. The tongue papillae were unusually and markedly prominent. The throat and ear drums were injected. There was no heart murmur. Hb was 10.8 g/dl, ESR 106 mm in the 1st hour, urine trace albumin, nil sugar, microscopy and culture negative, CSF normal, throat swab negative, ASO 605 units, blood culture negative, plasma Na 128 mmol/l, K 4.8 mmol/l, Cl 98 mmol/l, plasma urea 20 mg/100 ml (3.3 mmol/l), ECG normal, chest x-ray normal, C3 complement 1.16 g/l (raised).

Initially the diagnosis was thought to lie between toxic epidermolysis, due to a staphylococcal toxin, and MLNS. Treatment was started with IV fucidin to no effect, the child remaining febrile and ill. On the 8th day

of illness, oedema of the extremities developed and peripheral joint arthralgia. On the 9th day the child was transferred to Alder Hey Children's Hospital, Liverpool, under the care of Professor Harris to be closer to her home. The oedema and arthralgia of the periphery subsided quickly, but the skin of the periphery continued to peel for 2 or 3 weeks. She subsequently recovered completely (4 months later).

R. H. DAVIES
St David's Hospital,
Bangor,
Gwynedd LL57 4SL

Acquired toxoplasmosis and acute hemisindrome in childhood

Sir,

In an article (*Archives*, 1978, 53, 414) McNicholl and Flynn reported on 9 cases of acquired toxoplasmosis in childhood, 2 of whom contracted an acute hemisindrome with persisting sequelae. We observed a similar case in a 5-year-old boy who gradually developed hemiplegia over 24 hours (9-10 December 1977). The boy had been treated with penicillin for an otitis media during the preceding week. On admission he had a complete left-sided hemiparesis, but had neither lymphoglandular enlargement nor chorioretinitis. An acute right carotid angiography, isotope, and computerised axial tomography (CAT) scans were negative. A repeat CAT scan 6 months later was still normal. CSF findings were inconclusive. Improvement was initially rapid but subtotal. Currently, one year later, only a slight left-sided dysfunction and spasticity remains.

The patient is a farmer's boy and had been playing with his cat. Toxoplasma antibody titres in the patient and cat were as shown in the Table:

	1978	5 January	February 7 14	31 May	7 August
<i>Patient</i>					
Immunofluorescence					
IgM		128	64		
IgG		1024	1024		
Complement fixation		128	128	32	32
Dye test	1000		4000	500	500
<i>Cat</i>					
Immunofluorescence			1024		
Dye test			≥ 250		

As both a significant increase in dye test titre and toxoplasma antibodies of the IgM-class were demonstrable,

the findings indicated that the patient had ongoing toxoplasmosis. It appears most likely that the disease was acquired from the cat which had high serum levels of toxoplasma antibodies. Trimethoprim-sulphafurazole treatment (6 February–15 May) may have contributed to the rather rapid fall in antibody titres.

Acquired toxoplasmosis has occasionally been identified in acute hemiplegia in childhood (Sabin, 1941; Hedenström *et al.*, 1958; Lelong *et al.*, 1960). Isler (1971) reported only one case of toxoplasmosis (a 12-year-old boy with a left-sided parietal lobe syndrome with a complete restitution) among 116 acute hemisyndromes in children.

Acquired toxoplasmosis may thus occasionally be the cause of an acute childhood hemiparesis. A focal encephalitis may not be envisaged on the CAT scan and the diagnosis should accordingly be looked for by serological and microbiological methods.

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- M. KYLLERMAN
Department of Pediatrics II,
Östra Sjukhuset,
S-416 85 Gothenburg, Sweden
Ö. STRANNEGÅRD
Department of Microbiology,
Sahlgren's Hospital,
S-413 46 Gothenburg, Sweden

Dose of clonidine in alternating hemiplegia

Sir,

My fellow authors and I wish to point out that in our paper (*Archives*, 1978, **53**, 656–659) there is an error (page 658) in the dose of clonidine given to one of the patients. The dose quoted should have read 2.5 µg/kg per day (not milligrams).

GWILYM HOSKING
The Ryegate Centre,
(The Children's Hospital),
Sheffield S10 5DD