disease it is important to recognise new primary tumours and their possible influence upon survival. The appearance in a single patient of more than one of the malignancies associated with cigarette smoking is further evidence of the hazard of this habit.

We wish to thank the Medical Research Council for their continuing support (SD, MIS, and ML) and our colleagues in surgery and pathology in district hospitals in the North-west Thames Region.

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Canadian sheet dermatitis

During 1971-2 there was an epidemic of dermatitis in the Ottawa area of Canada caused by Wabasso polyester-cotton bed sheets.¹ We have seen several patients with a similar dermatitis over the past 18 months. All had used Wabasso bed sheets.

Present series

Since April 1975 we have seen nine patients with widespread dermatitis who had recently purchased cheap Canadian bed sheets. Three days to two weeks after starting to use the sheets they had developed severe itching and burning sensations, especially at night. When pillow-cases had also been purchased, redness of the ears and puffy eyelids were early complaints. In some cases several members of one family were affected, and in others one bed partner was spared.

Clinically the cases looked similar and were recognisably different from other forms of contact dermatitis from fabrics. The most characteristic feature was red follicular lesions, especially on the limbs. There was wide variation in the severity of the rashes, the most severely affected patients having large, continuous areas of redness and oedema (see figure) and swollen faces with puffy eyelids. The axillae and other skin folds were spared, and nightwear gave some protection. Patients had often been diagnosed as having drug eruptions, scabies, or toxic erythemas.

Wabasso bed sheets can often be recognised on sight or from the patient's description. They are brightly coloured, either uniformly or, more often, in large abstract or floral patterns employing several colours. Mauve, pink, blue, yellow, and green are the most common. The patient may remember that the packet the sheets were in was printed in French as well as English or was stamped "Made in Canada."

Patch tests with the patients' own sheets produced negative results in five out of eight cases tested, and there were no positive patch test results with formaldehyde. Only one patient reacted to a standard formaldehyde clothing resin patch test. Laundering the sheets helped in only one case, in which tolerance was reported after three washes. Once the patients stopped using the sheets the rash usually resolved in three to four weeks.

Comment

When patients approach us with suggestions about the cause of their ailments our response depends on our previous conditioning. In this instance an identical dermatitis had already been reported in Canada.^{1 2} The dermatitis first occurred shortly after a sheet manufacturing company changed its processing methods in the permanent-press resin treatment of polyester-cotton sheets from a slow cold process to a hot flash process.³ The offending agent was shown to be a fragmentation product of permanent-press resin polymer that was rendered unstable in the presence of heat and moisture.²

Follicular lesions on buttocks becoming confluent over back.

The epidemic in Canada halted when the company discontinued the implicated processing method. All our patients bought the sheets from street markets or similar outlets rather than from high-street stores. It would be interesting to know how sheets made unsaleable in Canada because they produce a dermatitis appear on sale in Britain and perhaps in other countries. Correct diagnosis of Canadian sheet dermatitis rests on the triad of its follicular, clinical appearance, its involvement of usually more than one member of the family, and the frequently negative results on patch testing with the offending sheets.

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Paroxysmal brain stem dysfunction as presenting feature of multiple sclerosis

Paroxysmal disturbances are differentiated from other transient episodes in multiple sclerosis by their explosive, short-lived, repetitive occurrence and by their localisation to the brain stem or cervical cord.¹ They have been reported in this disease but not as the sole presenting feature.¹⁻⁴ We have recently seen a patient whose paroxysmal attacks

preceded the development of the classical clinical features of multiple sclerosis by almost four years.

Case report

A 36-year-old man began to have paroxysmal attacks of dysarthria, ataxia, and weakness of his legs in February 1972. Each attack would last up to 10 seconds and he would experience at least a dozen such episodes each day. They would occur more frequently after a hot bath or any sudden exertion. In between his attacks he was free of symptoms and signs but during them he had bilateral horizontal nystagmus, dysarthria, and ataxia of his legs. In view of his unusual symptoms he was investigated in detail. Among the normal results of tests were cerebrospinal fluid, air encephalography, and four-vessel arteriography.

Treatment with carbamazepine resulted in the complete cessation of his attacks. He was followed up at monthly intervals and eventually, in January 1976, developed numbness of both lower limbs and ataxia of gait. Examination then disclosed bilateral horizontal nystagmus, cerebellar and pyramidal signs in all four limbs, with extensor plantar responses and absent abdominal reflexes. Examination of his cerebrospinal fluid then showed a protein of 0.45 g/l of which 23% was IgG. The Lange colloidal gold curve was 1232100000.

Comment

The diagnosis of multiple sclerosis, although presumed, could be established only when this patient developed the classical features of this condition four years after his presentation with a paroxysmal brain stem disturbance. The dramatic response of these paroxysmal symptoms to carbamazepine has been noted by others.^{3,4}

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Unusual cerebral inclusions in Reye's syndrome

We wish to report on unusual inclusion bodies, revealed by electron microscopy, in the brain of a patient with Reye's syndrome.¹

Case report

A 13-year-old boy was admitted with a two-day history of confusion and drowsiness after an influenza-like illness lasting three weeks. He was unresponsive to painful stimuli and had irregular respiration with periods of apnoea. The arms were areflexic, but reflexes in the legs were brisk with extensor plantar responses. The serum contained persistently raised levels of aspartate aminotransferase (67-121 IU/l) and bilirubin (37-61 μ mol/l). The serum calcium concentration fell from normal to 1.4 mmol/l causing tetany. The cerebrospinal fluid was normal. Serial EEGs showed high amplitude repetitive complexes at 2 Hz initially but became isoelectric later. Carotid angiography showed no intracranial blood flow. Viral studies for herpes simplex, measles, and influenza A and B were negative. Treatment was supportive but assisted ventilation was finally discontinued and the patient died on the fourth day after admission.

At necropsy the liver was found to be enlarged to 2610 g. The brain weighed 1655 g and showed signs of severe oedema: the gyri were flattened, the sulci obliterated, and the cerebellar tonsils herniated. Midline structures (septum pellucidum and subependymal areas of the third ventricle), the thalamus, and the lentiform nuclei showed liquefactive necrosis—a feature of the respirator brain death syndrome.²

Light microscopy showed abundant microvesicular fatty change affecting all liver cells, but no inflammation or necrosis. Similar changes were also present in other parenchymatous organs, notably in the renal tubular epithelium, pancreas, and myocardium. The brain showed non-specific cellular changes, which had resulted from oedema and anoxia. Thus the criteria of Reye's syndrome—namely, encephalopathy and microvesicular fatty change of viscera—were fulfilled.

Electron-microscope examination of the cerebral cortex revealed a spectrum of extensive neuronal damage ranging from mild degeneration to necrosis. The neuronal mitochondria were badly affected: their matrices were swollen and their cristae disrupted. Myelin sheaths had lost their structural integrity: there were widened, irregular spaces between individual lamellae. Astrocytic cytoplasm appeared to be swollen.

Discussion

All these changes, described already,³ may be due to hypoxia. They also may be artefactual, resulting from delayed fixation. It was, however, interesting to find large, complex inclusion bodies in some neurones and vascular pericytes. These inclusions were irregular and occupied most of the cytoplasm in many cases. They consisted of lipid droplet-like areas and masses of electron-dense granules. In the neurones they resembled an agglomerate of lipofuscin granules, while in the pericytes they were larger and more complex in structure (see figure). The pericytic inclusions were similar to residual bodies found

A large inclusion in the cytoplasm of a vascular pericyte. L=lumen ($\times 7465$).

in the hepatocytes of patients with Reye's syndrome.⁴ Osmiophilic dense bodies, different from those described here, were found in pericytes and were thought to reflect a mild disturbance of lipid metabolism.⁵ Thus probably these inclusions in pericytes, together with the lipofuscin-like bodies in neurones, are of greater importance in the diagnosis of the cerebral lesions of Reye's syndrome than the other changes, all of which can be produced by anoxia.

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