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A Clinical Study of Adult Coprophagia

DEAR SIR,

In their study of adult coprophagics, Ghaziuddin and McDonald (1985), suggest that the absence of thiamine deficiency in their series appears to differentiate it from coprophagia observed in animals.

They base their assumption on a single study of experimentally induced thiamine deficiency in beagle dogs. These animals developed coprophagia during the course of the experiment (Read & Harrington, 1981). It does not follow that clinical cases of coprophagia in adult dogs are inevitably due to thiamine deficiency.

Coprophagia is a relatively common condition in dogs and various causes have been postulated including pancreatic deficiency, parasitic burden and nutritional deficits (Houpt, 1982; Evans, 1982). However, the majority of cases appear to be behavioural in origin (Houpt, 1982). Puppies occasionally eat their own faeces but this trait disappears in adolescence. Adult dogs eat herbivore faeces as part of their natural behaviour but coprophagia of canine faeces is abnormal. This vice is thought to be acquired through boredom and perhaps in an attempt to avoid punishment for defaecation in the house. Behavioural methods including aversion therapy often provide a permanent cure (Evans, 1982). Dr Haupt (1982) describes the case of an adult coprophagic collie which was otherwise well behaved. The dog was left on its own for long periods and the problem was alleviated by giving it more attention, subjecting it to less isolation and providing toys to relieve its boredom.

The evidence suggests that there are actually similarities between the condition in dogs and humans. Thus behavioural treatments might prove useful when coprophagia is found in adult patients.

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Cognitive Deficits in Systemic Sclerosis

DEAR SIR,

We wish to report the case of a patient with systemic sclerosis in whom cognitive dysfunction was a feature. Central nervous system involvement is well recognised in systemic lupus erythematosus (Johnson & Richardson, 1968) and in periarteritis nodosa (Ford & Siekert, 1965), but up till now has not been reported in systemic sclerosis.

A 55 year old married women was admitted with renal failure in January 1985. For several months she had complained of pain, stiffness and swelling in her hands associated with Rayneud's phenomenon. She had a past history of carcinoma of the breast in 1972 for which a left mastectomy had been performed. In November 1984 local recurrence in the left axilla was diagnosed by computerised tomography and treated by aminoglutethimide. The clinical impression of systemic sclerosis was confirmed by renal biopsy which showed severe intimal thickening of the small arteries and arterioles with areas of cortical necrosis. There was no recovery of renal function and the patient has been successfully treated by intermittent peritoneal dialysis and latterly by continuous ambulatory peritoneal dialysis.

She was referred for psychiatric assessment in March 1985 because of the discrepancy between her medical and psychological status. The objective improvements found in her medical condition were not reflected in her psychological functioning. On examination, she was found to have a labile mood, gross temporal disorientation, and impaired short-term memory. There was evidence of marked dysgraphia and dyscalculia, both out of keeping with her previous occupation as an accounts clerk. Her comprehension of spoken and written language were intact. These deficits have all remained stable over a six month period.

This is the first case of systemic sclerosis in which a cerebral involvement has been documented. The nature of the cognitive deficits suggests a multi-focal cortical involvement, affecting both parietal and temporal lobes, reminiscent of multi-infarct dementia. We were able to exclude cerebral metastases as the cause of her cognitive dysfunction by computerised tomography and radio-isotope brain scans. In our opinion, these deficits are based on a vascular pathology similar to those found at renal biopsy.