

### References

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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 (Haupt, 1982; Evans, 1982). However, the majority of cases appear to be behavioural in origin (Haupt, 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 Raynaud'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.

We thank Dr M. K. Ward for allowing us to report this case.

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#### Hysterical Conversion States

DEAR SIR,

It has been generally accepted that the major hysterical conversion states which abounded in late nineteenth and early twentieth century psychiatric writings are much less prevalent today (APA, 1980). We have therefore been surprised by our recent experience of six such cases in a six week period, all seen by the Consultation Psychiatry Service in one of the three general hospitals in Newcastle upon Tyne. This represented one sixth of the cases seen during the period.

*Case i:* A 53 year-old man walked into the accident and emergency department complaining that he had lost his sight. Physical examination, including specialist ophthalmological examination, found no evidence of physical abnormality. A diagnosis of hysterical blindness was made. It was postulated that his hysterical symptomatology may in part have been his mechanism of dealing with the 'no alcohol' policy of the local alcohol and drug addiction unit to which he had been admitted as a day patient that morning. He did not wish to, or felt he could not, comply with this policy.

*Case ii:* A 39 year-old man was admitted to hospital having developed episodic muscular spasms following a minor accident at work. The muscular spasms were of 5–30 seconds duration and spread from his right arm to the rest of the body. Consciousness was not impaired. Neurological examination was normal, as was an EEG. Psychiatric examination confirmed a diagnosis of hysterical seizures. It was postulated that the underlying

mechanism for these seizures was the avoidance of unresolved difficulties at home. The seizures became less frequent and remitted within a few hours of admission.

*Case iii:* A 19 year-old girl was brought to the accident and emergency department in a mute state. She remained mute for the following 24 hours but over the subsequent three days her speech gradually returned. Neurological examination was normal. A diagnosis of hysterical mutism was made. Psychiatric examination revealed that her mutism was a way of communicating the pressure she felt herself to be under at home, where her mother was dependent on her in bringing up her six younger siblings.

*Case iv:* A 24 year-old man was brought to casualty having been found "unconscious" in the street. After overnight observation he was found to be mute. Neurological and ENT assessments revealed no abnormality. On enquiry it was learnt that he was to be interviewed by the police in connection with a charge of drunk and disorderly behaviour. It was thought that his hysterical mutism was a mechanism by which he sought to avoid this.

*Case v:* A 29 year-old shipyard worker was brought to casualty having had a sudden onset of bilateral paralysis in both arms and legs. Neurological examination was normal. Psychiatric examination confirmed the diagnosis of hysterical paralysis which was thought to be in response to the extreme anxiety generated by his marital problems and the threatened loss of his job. His paralysis resolved within a few hours of admission.

*Case vi:* A 32 year-old married man was admitted with a sudden onset of paralysis (with anaesthesia) of the left side of his body. Neurological examination did not reveal any abnormality. Enquiry showed that in the preceding few weeks he had developed a fear of AIDS after a recent extra-marital affair, which had generated great anxiety and guilt in him. After reassurance about his physical condition his hysterical paralysis and anaesthesia remitted over a period of 48 hours.

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