

turn evoke behavioural abnormalities, represents a promising avenue for the further understanding of brain-behaviour relationships.

ARNOLD E. MERRIAM
ANGELA M. HEGARTY

Bronx Municipal Hospital Center
Room 1022
Bronx, NY 10461
USA

References

- MERRIAM, A. E., HEGARTY, A. & MILLER, A. (1989) A proposed aetiology for psychotic symptoms in white matter dementia. *Neuropsychiatry, Neuropsychology, and Behavioural Neurology* (in press).
- MILLER, B. L., LESSER, I. M., BOONE, K. *et al* (1989) Brain white matter lesions and psychosis. *British Journal of Psychiatry*, **155**, 73–78.
- PYCOCK, C. J., CARTER, C. J. & KERWIN, R. W. (1980) Effect of 6-hydroxydopamine lesions of the medial prefrontal cortex on neurotransmitter systems in subcortical sites in the rat. *Journal of Neurochemistry*, **34**, 91–99.
- ROBINSON, R. G., KUBOS, K. L., STARR, L. B. *et al* (1984) Mood disorders in stroke patients: importance of lesion location. *Brain*, **107**, 81–93.
- WEINBERGER, D. R. (1987) Implications of normal brain development for the pathogenesis of schizophrenia. *Archives of General Psychiatry*, **44**, 660–669.

Trichotillomania

SIR: In their interesting report of a female victim of incest with trichotillomania, Singh & Maguire (*Journal*, July 1989, **155**, 108–109) conclude that sexual conflicts must always be considered when assessing trichotillomania. I would like to add two points to this conclusion.

Firstly, the authors do not mention the presence or absence of trichophagia (hair eating) in their patient. This is essential to ascertain, because of the well-recognised complication of trichobezoar. Mere enquiry is insufficient, as evidenced by Grant *et al* (1979), who reported a trichobezoar occurring when trichophagia was denied. At operation there was incontrovertible evidence of recent hair ingestion. Enquiry into gastrointestinal symptoms, physical examination and further investigations where relevant should be considered when assessing trichotillomania.

Second is the well-worn question of the role of iron deficiency in the picas in general. This has been debated for decades, and there are many anecdotal reports of a dramatic response to iron therapy (e.g. Coleman *et al*, 1981). However, a controlled study in the mentally handicapped (Bicknell, 1975) did not support the conclusions of earlier investigators (e.g. MacDonald & Marshall, 1964) that iron deficiency is aetiologic. McGhee (1980) reported trichotillomania and trichophagia in two children with

iron deficiency anemia whose behaviour ceased when serum haemoglobin was restored to normal levels. One had a trichobezoar, one did not. Whatever the relationship – whether primary or secondary – the occurrence of iron deficiency in conjunction with trichophagia is sufficiently well documented to indicate that a full blood count and serum iron estimation should be considered when assessing trichotillomania.

CAROLINE SULLIVAN

Rampton Hospital
Retford
Notts DN22 0PD

References

- BICKNELL, D. J. (1975) *Pica: A Childhood Symptom*. London, Sydney: Butterworths.
- COLEMAN, D. L., GREENBERG, C. S. & RIES, C. A. (1981) Iron deficiency anaemia and pica for tomato seeds. *New England Journal of Medicine*, **304**, 848.
- GRANT, J. A., MURRAY, W. R. & PATEL, A. R. (1979) Giant trichobezoar – an unusual case. *Scottish Medical Journal*, **24**, 83–86.
- MACDONALD, R. & MARSHALL, S. R. (1964) Value of iron therapy in pica. *Paediatrics*, **34**, 558–562.
- MCGHEE, F. T. & BUCHANAN, G. R. (1980) Trichophagia and trichobezoar: aetiological role of iron deficiency. *Journal of Paediatrics*, **97**, 946–948.

Failure to convulse with ECT

SIR: Complete inability to convulse in ECT has occasionally been reported (Sharpe & Andrew, 1988). This issue has long been controversial (Freeman, 1988; Pippard & Russel, 1988). Electrical parameters such as electrode placement, stimulus waveform, duration of pulse train and amount of energy delivered have been discussed as possible contributing factors. In clinical practice, the most widespread ECT devices use sine-wave stimulus waveforms (often modified). Pippard & Russel (1988), referring to such instruments, argued that the effective amount of energy delivered is probably too low to induce seizure and a satisfactory clinical response, suggesting that a constant current stimulus of 275–325 mC lasting about 3.25 ms at pulse rate of 50–60 Hz could overcome the problems of a minority of patients with high seizure thresholds. Freeman (1988) agreed, proposing the use of an ECT device which is able to deliver 2000 mC at maximum setting for high threshold patients.

We report a clinical observation, with a modern brief-pulse, constant current device (Thymatron, Somatics) on a cohort of 115 patients (age range 18–78) for which ECT was prescribed by their consultant. Energy delivered ranged from 75.6 to 302.4 mC, with a duration of pulse train that ranged from 0.6