

orientated and prevent many acute admissions, those patients who do require admission are more seriously ill and often more disturbed. This, combined with the lowering of the denominator of bed numbers, means that this ground will have an increasing impact on acute ward occupancy. This is supported by Dean (1993) who reported two NLS patients blocking a third of the beds in their home treatment service (23 beds per 100 000 population) with stays in excess of three years. We felt that many of the prolonged in-patient stays in Nottingham could have been reduced by use of alternative existing services. Acute care was, however, needed for prolonged periods to deal with treatment resistant conditions and to contain high levels of risk. We would agree with Lelliott that an urgent assessment of facilities and management strategies is needed to avoid blocking acute beds with patients who could be better managed by alternative services. This is particularly pertinent in Nottingham as acute occupancy has increased from 75% to 93% in the last four years.

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STEFFAN DAVIES
SIMON PAYNE
JANE JENKINS

*Mapperley Hospital
Porchester Road
Nottingham NG7 4GL*

The definition of anorexia nervosa

SIR: The editorial of Drs Parry-Jones (*BJP*, September 1994, **165**, 287–292) demonstrated that whereas the historical syndrome of bulimia bears little consistent resemblance to modern bulimia nervosa, historical data derived from a sizable group of non-fat phobic anorexic patients provides “substantial evidence of continuities” with its modern version, as defined in DSM–III–R or ICD–10. They cautioned against the rigid application of the diagnostic criterion of fat phobia, which has been accorded paramount importance in contemporary nosology.

For several years I have argued for the same thesis – that the patterning of anorectic complaints and the biomedical process of diagnosing anorexia

nervosa are both time- and context-dependent. Unlike historical data, which are finite and often incomplete, cross-cultural evidence grows that we may be seeing in non-Western societies a parallel evolution of eating disorder that had historically taken place in the West. A substantial number of non-fat phobic patients fail to fulfil the current diagnostic criteria for anorexia nervosa (Lee *et al*, 1993).

There is thus an exciting convergence of longitudinal historical and horizontal cross-cultural data. The ideas of Drs Parry-Jones are supported by an increasing number of Western experiments. Yager & Davis (1993) wrote that “many chronic patients seem far more able to acknowledge that they are too thin”, and “we are also very aware of how clinically difficult it can sometimes be to elicit a fat phobia in patients who have evolved a different type of attribution regarding why they are not eating”. By conceding that “the same sort of phenomenological heterogeneity occurs in Western anorexia nervosa patients as well”, they testified to the intracultural variability of anorexia nervosa in contemporary North America. Steiger (1993) similarly stated that “anyone who works with large numbers of anorexia nervosa sufferers knows that this disorder is not uniformly about a desire to be thin. Rather, the apparent pursuit of thinness or weight phobia seen in anorexia is often explicable in terms idiosyncratic to each case.”

Palmer (1993) criticised the central role given to weight concern in the modern diagnosis of anorexia nervosa, as this may have led to “a premature closure” of the inquiry into the nature of the eating disorders in general. Halmi (1994) reiterated that “psychiatric diagnostic categories are constrained by history as well as biology”, and even proposed that “the psychobiological vulnerability factors that induced the development of irreversible starvation in medieval saints are similar to those inciting the emergence of anorexia nervosa and bulimia in twentieth-century young women”.

The DSM–IV criterion that all anorectic subjects must insist on feeling fat, no matter how emaciated they are, may represent not so much their lived experience as the particular viewpoint of empowered nosologists, and may hamper research work. The bias of any classificatory system is not nearly so bad as the illusion that it is unbiased.

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SING LEE

*Department of Psychiatry
Prince of Wales Hospital
Shatin, Hong Kong*

Capgras, Fregoli and Cotard's syndromes and Koro in folie à deux

SIR: We wish to report the concurrence of Capgras, Fregoli and Cotard's syndromes and Koro and folie à deux in a man with cognitive impairment and a 15-year history of paranoid psychosis.

Case report. A 58-year-old unemployed man presented to an accident and emergency department with his wife. He believed that: his house had been burgled; his wife had been drugged by neighbours and replaced with a double (Capgras syndrome); his head and penis were shrinking; and that his penis would disappear inside his abdomen at which time he would die (Koro-like symptoms). He misidentified tradesmen as imposters trying to gain entry to kill him and his wife (Fregoli's syndrome). Both he and his wife believed that their neighbours were murderers and were part of a council conspiracy; both saw blood coming through their ceiling and heard noises coming from their neighbour's house which they said were the sounds of bodies being cut up and the screams of innocent victims (folie à deux).

Neither the patient nor his wife had a personal or a family history of psychiatric disorder. Psychometric testing of the patient revealed evidence of cognitive impairment, including impairment of facial recognition. He was treated initially with antipsychotic medication, which led to the disappearance of the misidentification symptoms, but his condition worsened and he became depressed with prominent nihilistic delusions, believing that parts of his body did not work and that he was seriously physically ill (Cotard's syndrome). These latter symptoms

improved with ECT and antidepressants. Our patient's wife initially became worse after separation and she only improved with addition of medication and supportive psychotherapy.

We know of no other reports of all these syndromes appearing in the same person (or of folie à deux involving visual hallucinations). There are reports of combinations of two of these syndromes occurring together (Enoch & Trethowan, 1991). Capgras, Fregoli and other misidentification syndromes have been described in a number of psychiatric disorders, especially schizophrenia and in organic conditions including dementia, and they have been linked with an underlying right hemisphere dysfunction (Cutting, 1991). Koro-like symptoms have been described as part of other primary disorders including affective disorders, schizophrenia and organic disorders (Joseph, 1986; Devan & Hong, 1987; Durst & Rosca-Rebaudengo, 1988).

Although it is appealing to try to find a unifying hypothesis in this case – that dementia has led to all the symptoms present – we believe that the patient had a long-standing paranoid psychosis which was shared by his wife in a folie à deux. He may have begun to experience misidentification phenomena and become depressed with growing cognitive impairment. The Koro-like syndrome seemed to be a consequence of his recent depression.

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GEOFFREY WOLFF
KWAME MCKENZIE

*Institute of Psychiatry
DeCrespigny Park
London SE5 8AF*

CORRIGENDUM

The editorial by R. P. Snaith in the November issue (*BJP*, November 1994, **165**, 582–584) quoted a personal communication attributed to Dr Henry Rollin. This should have been a specific reference.

The correct reference is:
ROLLIN, H. R. (1990) *Festina Lente: A Psychiatric Odyssey*. Memoir Club Series. London: British Medical Journal.