somewhere between visual and limbic cortices causing visual images to become dissociated from the affective memories previously associated with them, may well be applicable to both sorts of disorder. This mechanism has certainly been suggested previously in attempts to explain Capgras' syndrome (Lewis, 1987) and reduplicative paramnesia (Staton *et al*, 1982).

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SIR: I was fascinated to read Dr Anderson's paper on the delusion of inanimate doubles (*Journal*, November 1988, **153**, 694–699) and its implications for Capgras symptoms. Dr Anderson suggests that the delusion of doubles for objects rather than people is a logical extension of the Capgras phenomenon, in the same way that the term prosopopagnosia has seemingly been extended to include rooms, buildings, and objects. There are two corollaries to this suggestion.

If the Capgras syndrome is more common than is suggested by the number of published case reports, then by extending it to cover the delusional misidentification of objects, it must be even more commonplace. I tentatively suggest that the majority of these cases must occur within the context of 'functional' illness. I have seen two cases which would fulfil Dr Anderson's criteria, both within the setting of paranoid schizophrenia.

Case reports: (i). A spinster of 74 first became ill at the age of 59, with a psychosis characterised by second-person auditory hallucinations and persecutory delusions. She is now maintained on injections of modecate. Living alone in a seaside bungalow with her elderly dog, she occasionally lapses into illness. She becomes acutely suspicious of neighbours and friends living in her road. She then rings the local police station to tell them that while she has been asleep these neighbours have been going through her belongings. She asserts that although her prized art book collection and paintings are still in the bungalow, and appear identical, they have been meticulously faked and replaced.

(ii) A widow of 55, again living alone, and with a diagnosis of paranoid schizophrenia, has become estranged from her family. In relapse she alleges that they persecute her, although the truth is somewhat the reverse of this. The widow sends them angry letters and plagues the police with unfounded accusations against them. When she is admitted to hospital she frets about her empty flat. After some leave at home she complained on return that her brother had been in her flat while she had been in hospital. She knew this because he had replaced a wardrobe and a chest of drawers with identical items, but that these lacked various marks of wear and tear that the originals had borne. She was convinced that her brother had gained access with her own keys, having stolen them from her while she was in hospital, after leaving a replica set in their place.

Neither of these patients admit to these ideas when well, and neither have any identified organic neurological disease. The delusion of inanimate doubles may thus be an intrinsic part of an elaborate delusional system in some cases of paranoid schizophrenia. The pure Capgras syndrome seems mainly to occur in 'functional' illness, in the absence of lesions detectable by computerised tomography imaging (Green, 1988; Green & Birchall, 1988).

Although macroscopic brain lesions do appear to account for a few cases of the delusion of doubles, the aetiology of the Capgras phenomenon seems to be rather wider than this, and it seems that we must not rely solely upon either a psychodynamic or organic explanation.

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Parasuicide in Young Asian Women

SIR: We were interested in the paper by Manium (*Journal*, August 1988, **153**, 222–225), which, like the paper by Merrill & Owens (1986), drew attention to the high parasuicide rate of young Asian women.

We analysed the attendances of patients aged 10–24 presenting with self-poisoning to the two casualty departments of The London Hospital during the years 1980–1984. This hospital serves an area with a large Bangladeshi population.

We used figures derived from the 1981 census and from ILEA statistics to estimate the proportion of

the Tower Hamlets population in these age groups that were of Asian origin, and compared Asian and non-Asian self-poisoners. We found no significant difference in the proportion of male Asians in the age group 10–24 who made parasuicide attempts; nor did we find an excess of Asian female patients in the age groups 10–14 and 20–24. However, of the 156 women aged 15–19 who presented with self-poisoning, 25 (16%) were Asian. Asians constituted only 7% of the female population in this age range, so this represents a significant excess ($P<0.005$).

Merrill & Owens (1986) suggested that unmarried adolescent girls face culture conflict around family discord over Asian versus Western lifestyles. Dr Manium draws attention to parents' disapproval of marriage in the predominantly Muslim population of his study, which can lead to conflicts in both these areas.

In contrast to the Malay study, where there was a high rate of suicide, there were no reported suicides of Asians aged 10–24 from North East London between 1980 and 1984.

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Castration and the Sexual Offender

SIR: Salzmann (*Journal*, August 1988, **153**, 270) recommends castration (orchidectomy) of persistent sexual offenders. His paradigm is "the Danish approach". He writes that "castration [is] voluntarily accepted by many inmates in Herstedvester as the price of release from indefinite detention". In that Dr Salzmann is wrong.

Due to fierce opposition within the public and the Danish Medico-Forensic Council, orchidectomy was abandoned in 1968. This procedure is an absolutely unthinkable approach to the sexual offender in Denmark today!

Currently, castration is only performed on a small number of transsexuals (2–3 men a year), this being a completely different issue.

On another note, we should add that the legal sanction "indefinite detention" within a treatment and rehabilitation perspective as epitomised by the Herstedvester model was rescinded in 1973.

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Melatonin and Sulphatoxymelatonin in Eating Disorder Patients

SIR: We read with interest the report by Bearn *et al* (*Journal*, March 1988, **152**, 372–376), in which urinary sulphatoxymelatonin (aMT6s) excretion was compared in underweight and weight-restored patients with anorexia nervosa and in age-matched female controls. No significant differences were found between groups at any time, and the authors concluded that melatonin output, as assessed by aMT6s, was not influenced by changes in weight, although they did not directly assess plasma melatonin levels, or consider the relative influence of depression within the patient group.

We have recently completed a comparative study involving both nocturnal serum melatonin and urinary aMT6s in female patients with anorexia nervosa ($n=15$), bulimia nervosa ($n=9$) and in control women ($n=10$) of similar age. All groups were compared according to weight and mood variables. Patients with anorexia nervosa had a significantly lower percentage of ideal body weight (76.4% ± 9.9%) than bulimia nervosa patients (102.1% ± 12.3%) and controls (105.2% ± 14.2%) ($F=22.2$, d.f. = 33, $P<0.001$). Both patient groups had significantly higher depression scores on the Hamilton Rating Scale for Depression (HRSD) (Hamilton, 1967) (for anorexia nervosa patients 19.8 ± 9.6; for bulimia nervosa patients 16.4 ± 8.4) compared with the control group (2.7 ± 2.6) ($F=14.9$, d.f. = 33, $P<0.001$).

There were no differences between patient groups and controls in night-time values of urinary aMT6s or serum melatonin values. A two-way analysis of variance for both urinary and serum results was carried out to examine the effects of weight and depression as independent variables among patients. Although weight did not influence either of these measures, a significant effect was found for depression. Patients who met DSM–III–R diagnostic criteria for major depression (American Psychiatric Association, 1987) and had HRSD scores equal to or higher than 17 had significantly lower melatonin output than the non-depressed group for both serum melatonin ($F=4.22$, d.f. = 1, $P<0.05$), and urinary aMT6s ($F=6.51$, d.f. = 1, $P<0.02$).