Three Cases of Capgras' Syndrome

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Three cases of Capgras' syndrome were identified in one psychiatric hospital within a 1-year period. These are presented and their psychopathology discussed. The traditional assumption that this is a rare syndrome is challenged.

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Capgras' syndrome was described initially by Capgras & Reboul-Lachaux in 1923 and termed "L'illusion des sosies" (the illusion of doubles). It is defined as the delusional belief "that a person, usually closely related to the patient, has been replaced by an exact double", by Enoch & Trethowan (1979).

A number of authors have observed that this is a rare syndrome (e.g. Christodoulou, 1977; Enoch & Trethowan, 1979). The occurrence of three cases in one psychiatric hospital within the space of 1 year therefore provokes the question, how rare is rare?

Case reports

Case 1

An 83-year-old lady who lived with her husband was admitted for assessment owing to memory impairment and a stepwise decline in intellectual ability over the previous 6 months. The onset was sudden and possibly related to a cardiovascular event. There was no significant previous psychiatric history and her social circumstances were unremarkable. Her premorbid personality was described by her husband as cheerful, capable, and outgoing and their marriage was reported to be a happy one.

Over the preceding 6 months she had developed the firm belief that her husband was an impostor who was a double of her real husband. This belief was held with delusional intensity and she would act on it, e.g. making three cups of tea daily, including one for her 'missing' husband. If she and her husband went out she would surreptitiously return indoors to leave a note stating where she had gone. At times she would sob while in her house and ask to be taken home, claiming that while she recognised the furniture it was all a replica of her real home. She remained, however, passively acceptant of both the impostor and duplicated house.

Her dementia progressed over the following 2 years and she gradually lost her belief in the double of her husband. Sadly she subsequently became unable to identify him altogether. A few months later she died.

Case 2

A 55-year-old married housewife was referred to outpatients by her GP because she suddenly developed persecutory beliefs. In particular she believed that their house had been broken into and 'bugged'. She became convinced that her husband's personality had changed and that he had been replaced by a demonic double who was trying to poison her. The impostor could be distinguished from her real husband because he tied his shoe laces slightly differently.

Apart from taking an overdose of barbiturates at the age of 29 years, when her first marriage collapsed, this lady's past was unremarkable. There was no family history of psychiatric illness. Both parties denied any problems within the present marriage.

Weeks before the referral she had developed a severe chest infection for which antibiotics and high doses of prednisolone were prescribed. This medication was discontinued when she developed a marked allergic reaction. Her GP prescribed benzodiazepines for the ensuing anxiety. She became mildly toxic and confused and the delusions developed in this setting. There had been a number of recent stressful events: a house move, a myocardial infarction sustained by her husband, and an ongoing dispute with the local council about nearby building work.

Within 5 days of starting oral neuroleptics, the paranoid psychosis and delusional misidentification had resolved. Assessment 3 months later confirmed her complete recovery.

Case 3

A 45-year-old divorced unemployed woman was admitted believing that all the children in her street had been sexually molested. Her 14-year-old son was put on a 'place of safety' order as she repeatedly attempted to physically examine him because of these worries. Her behaviour came to light when she attacked her son at school because he did not want to return home with her. She then assaulted the headmaster and stabbed a builder near her home.

Her early life was promising until she suffered a psychotic illness at the age of 22 while studying for a sociology degree. There followed a 23-year history of acutely relapsing paranoid schizophrenia, with at least ten hospital admissions.

When visited by her son soon after her admission she denied that he was related to her, calling him an 'impostor'. She claimed that this visitor had different-coloured eyes, was not as big and brawny, and that her real son would not kiss her. On one occasion she claimed "They're as different as chalk and cheese". Following treatment with depot neuroleptics the paranoid symptoms abated, although the delusional misidentification continued.

Discussion

Capgras' syndrome, which is present in all three cases, is one form of delusional misidentification. Several other varieties have been described, including

delusional replacement of inanimate objects (Anderson, 1988) and reduplicative paramnesia (defined by Förstl *et al* (1991) as the "delusional belief that a physical location has been duplicated"). Reviews by Berson (1983) and Förstl *et al* (1991) show these varieties to be reported much less frequently than Capgras' syndrome, so it is of interest that in case 1 arguably both reduplicative paramnesia and delusional replacement of inanimate objects are also displayed. Of the two, reduplicative paramnesia seems more appropriate because her belief that the furniture had been substituted appears to be a rationalisation to reinforce her belief that the whole house had been duplicated. Förstl *et al* (1991) report that reduplicative paramnesia is most frequently associated with neurological disorders and our case confirms this finding. In addition, this lady later became completely unable to recognise her husband. Unfortunately, no specific tests were performed to demonstrate prosopagnosia, but the diagnosis is a compelling possibility. Prosopagnosia is distinguished from Capgras' syndrome in being a non-recognition rather than a misrecognition of a face. Anderson (1988) suggests that the two disorders may represent a dysfunction at different stages of the same neurological process of complete object identification (i.e. the integration of object perception with the feeling of familiarity). His theory is that the neuropathology of Capgras' syndrome lies in the visual recognition pathway, probably distal to the prosopagnosic lesions that are commonly found in the right occipitotemporal region. This view has proved to be controversial (Signer, 1989).

The aetiology of Capgras' syndrome remains unclear. The case for a psychodynamic basis (Berson, 1983) and that for an organic basis (Anderson, 1988; Doran, 1990) have both been argued eloquently. Published reports describe the syndrome arising in both functional and organic illness. This is supported by our case reports. In case 2 a clear organic illness is identified, as well as recent psychological stresses. How much each contributed to the paranoid psychosis and Capgras' syndrome is a matter of conjecture. A paranoid component is identified in cases 2 and 3; this frequently accompanies Capgras' syndrome, as Christodoulou (1977) and others have noted.

Both cases 2 and 3 identify small differences in physical appearance and behaviour that distinguish the impostor from the original person. This is not an unusual finding, as Todd *et al* (1981) highlight. In their convincing discussion they propose that the accepted definition, which requires replacement by an "exact double" (Enoch & Trethowan, 1979), should be modified to ". . . replaced by impostors with a close resemblance to the originals", to allow for minor differences. Two explanations are proposed for this specific psychopathology. One is that it is coincidental, occurring *ab initio*; the other is that it is the result of a secondary rationalisation process on the part of the patient, to support their belief that the original person has been replaced. The process of rationalisation, which Todd *et al* (1981) favour, is much more satisfactory.

The delusional misidentification in our third example persisted following recovery from acute psychosis. Although the syndrome usually subsides as the underlying illness responds to treatment, occasional persistence of the delusion has been noted (Enoch & Trethowan, 1979). This could be interpreted as evidence for an autonomous syndrome, but underlying functional or organic illness seems to us a more likely explanation.

In emphasising the rarity of Capgras' syndrome, Christodoulou (1977) pointed out that it took him more than 6 years to collect 11 cases. A number of reports have since been published that challenge this assumption. Fishbain (1987) conducted a 1-year prospective study and concluded that a psychiatric emergency-room psychiatrist should expect to encounter the Capgras' delusion in one or two patients per thousand examined. In their study of a Chinese population of 2000, Mak *et al* (1985) found eight patients with Capgras' syndrome, a prevalence rate of 0.4%. Förstl *et al* (1991) identified an increase in reported prevalence and commented that this could be due to enhanced awareness of the symptom. The three cases described in our report were diagnosed coincidently and not as the result of a systematic study. The prevalence of the syndrome within the catchment population of 300 000 could indeed be higher than our rate of 0.001%.

The prevalence issue is of more than academic interest, because of the frequency of violent behaviour secondary to misidentification syndromes. This has been demonstrated in many reports (Fishbain, 1987; De Pauw & Szulecka, 1988; Förstl *et al*, 1991) and is exhibited in case 3, although the violence occurs before Capgras' syndrome emerges. Specific enquiry about misidentification beliefs should be undertaken in the mental state examination, particularly where violence is a factor.

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Koro: Culture-Bound Disorder or Universal Symptom?

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A case report is presented of koro associated with a depressive illness in a 31-year-old male Briton. The specificity or otherwise of koro as culture bound and a distinct nosological entity is discussed.
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Koro was originally described as a psychogenic syndrome among the Chinese. It is characterised by the belief that the penis (breasts and labia in females) is shrinking, that it will disappear into the abdomen, and that this will result in death (Anderson, 1990). The cultural belief of the Chinese was considered as the basis of the symptom, hence it was regarded as a culture-bound syndrome (Yap, 1951). However, there have been questions about its specificity as a culture-bound syndrome and a distinct nosological entity. Apart from the Chinese of southern coastal China and among overseas Chinese in south-east Asia, it has been reported among individuals of various cultural backgrounds.

Various authors have attempted to distinguish between complete and incomplete forms of koro, as well as cultural and non-cultural forms. It is claimed that the Chinese exhibit the complete forms, fulfilling all the criteria of the symptom complex, while the non-Chinese have the incomplete type, characterised in most cases by the absence of the belief that the penis will disappear into the abdomen or cause death. The incomplete forms are regarded as the non-cultural forms of koro, while the complete form is the classical culture-bound type. The view has been expressed also that the complete koro occurs in otherwise healthy individuals, usually in the context of acute severe anxiety, while the incomplete form is said to occur as part of a recognisable primary psychiatric disorder. Berrios & Morley (1984) reviewed 15 non-Chinese cases of koro, and since then the number of reported non-Chinese cases has increased to well over 30. In some of these non-Chinese cases, the complete symptom complex was reported in the context of functional and organic psychiatric disorders as well as non-psychiatric physical conditions (Anderson, 1990; Holden, 1987). We present a case of koro in a patient with depressive illness.

Case report

The patient is a 31-year-old single white Briton who was admitted following worsening symptoms of depression. He gave a history of gradual onset of symptoms dating back to about 18 months. He was feeling depressed with loss of interest and drive, poor concentration, various aches and pains, disturbed sleep, and diminished appetite. A year after the onset of his symptoms, he presented for treatment and was managed as an out-patient. He responded favourably to antidepressant medication but discontinued treatment after 2 months, claiming that he no longer needed it. About 6 weeks after stopping his medication, he had a re-crudescence of his symptoms and began to deteriorate, culminating in his admission.

There is no family history of psychiatric illness. He has always been shy and sensitive, with difficulty making friends, and has tended to feel somewhat inadequate. He left school at 16 and thereafter has had various jobs. He was smoking 40 cigarettes