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Capgras' Syndrome and Cerebral Dysfunction

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Summary. Two cases of Capgras' syndrome in association with coarse brain disease are presented. The authors suggest that prosopagnosia (face non-recognition) may be the primary expression of a specific cerebral dysfunction which forms the basis for a delusional elaboration resulting in Capgras' syndrome.

Introduction

Capgras' syndrome (Capgras and Reboul-Lachaux, 1923) is considered a rare, colourful illness in which the patient believes that a person, usually a close relative, has been replaced by an exact double or impostor and maintains this false belief despite evidence to the contrary. Although it often occurs as an isolated finding, the Capgras syndrome has usually been described as a manifestation of schizophrenia. Enoch, Trethowan and Barker (1967) assert that, since the syndrome occurs in a clear sensorium, 'it is obvious that there is no organic basis to the condition; but it is rather a functional illness and as such is explained on a psychodynamic basis'. Other authors (Vogel, 1974; Todd, 1957; Haslam, 1973) have also stressed this viewpoint.

However, in recent years an increasing number of reports have appeared describing the Capgras syndrome as a manifestation of organic brain disease. Weston and Whitlock (1971) presented a case of Capgras' syndrome in a 20-year-old male who was unconscious following head injuries sustained in an automobile accident. He had subsequently developed evidence of bilateral temporo-parietal damage, a frontal lobe syndrome, a severe memory defect with mixed dysphasic involvement, and generalized intellectual impairment. The presence of ptosis of the left upper eyelid, left divergent squint with diplopia, and anisocoria with the left pupil larger than the right indicated a left third nerve lesion. Further, an incongruous left homonymous hemianopia with macular sparing suggested involvement of the right temporal radiation.

MacCallum (1973) presented five cases of Capgras' syndrome in association with the following primary conditions: (a) toxicity and cerebral anoxia secondary to bronchopneumonia; (b) right-sided hemiplegia secondary to basilar migraine; (c) an alcohol paranoid state; (d) poorly controlled diabetes mellitus, with a buttock infection and folate deficiency; and (e) possible malnutrition. In each of these cases resolution of the primary organic illness resulted in remission of Capgras' symptoms. In the second case with right-sided hemiplegia secondary to basilar migraine, an EEG revealed bilateral theta waves more prominent on the left side. In the fourth case (diabetes, infection, and folate deficiency) spatial disorganization was elicited by drawing tests (not specified). This suggests non-dominant hemisphere dysfunction (Wheeler and Reitan, 1962).

Hay, Jolley and Jones (1974) discussed a case of Capgras' syndrome which developed after a course of ECT in a middle-aged mentally retarded woman with pseudohypoparathyroidism. EEG revealed a background of medium voltage dominant theta activity, with low voltage delta activity spreading into all areas, more marked on the left. A subsequent EEG resembled the initial tracing, with some evidence of focal abnormality in the left temporal region. Interestingly, the EEG dysrhythmia coincided with the florid Capgras' symptoms and improved as the symptoms resolved.

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Faber and Abrams (1975) presented the case of a 47,XYY male with symptomatic schizophrenia who, in addition to mild mental retardation, displayed Capgras' syndrome. EEG revealed predominantly left-sided slow activity.

To alert physicians to the frequency of brain disease in patients with Capgras' syndrome we report the following two cases in which the syndrome was associated with organic brain disease; the cases were seen during a four-month period on an acute treatment adult general psychiatric unit.

Case Reports

Case 1

(T.J.) This 37-year-old, white, divorced engineer was admitted to the University Psychiatric Service with a two-year history of misidentification of familiar faces. He insisted that his ex-wife, siblings, and physicians were impostors, and claimed he could detect slight differences between the 'actual' individuals and these impostors.

He had been well until seven years before the index admission, at which time he suffered a rupture of capillary telangiectases of the right frontal lobe, resulting in a huge right fronto-parietal, right lateral ventricular, and right basal ganglia hematoma, demonstrated at open surgical evacuation. He recovered, but was left with a persistent left spastic hemiparesis and thalamic hemisensory syndrome, minimal disorientation, and mild difficulty in performing sequential tasks. Psychological testing a year later showed a normal intelligence with some difficulty in performing higher mathematics. At this time he had the first of his left brachio-facial focal motor seizures, which were subsequently controlled with diphenylhydantoin and phenobarbital. During the next year delusional ideas developed and these eventually led to three admissions to psychiatric hospital within a two-year period, and a year later to the index admission.

On admission he appeared as a friendly, alert man without blunting of affect and with euthymic mood. His speech was tangential and he used the stock word 'wedlock' repeatedly. There was no other evidence of formal thought disorder. He had the delusional idea that he was a capacitor for electromagnetic waves. He also thought his parents had been replaced by impersonators and that they had conversations with famous people. Incomplete auditory hallucinations were the only perceptual disturbance present. No first rank symptoms of Schneider were present, and cognitive functioning and orientation were complete. Admission physical findings included left hemiplegia and hemianaesthesia. An EEG revealed a right-sided temporo-parietal slow focus. A Reitan-Indiana Aphasia Screening Test (Wheeler and Reitan, 1962) showed the presence of constructional apraxia. His WAIS was abnormal in that his performance IQ was 88 while his verbal IQ was 100. There was no family history of mental illness. The patient showed a moderate response to haloperidol, 30–50 mg daily; he also received diphenylhydantoin, 100 mg t.i.d. and phenobarbital, 30 mg b.i.d. He was discharged to his own care, improved, but with residual mild Capgras' syndrome.

Case 2

(R.S.) This 51-year-old, white, widowed mother of three was admitted to the University Psychiatric Service with the delusional idea that her two sons, who lived with her, were actually 'boarders', that her real sons were dead, and that three years previously someone had killed her daughter.

She had been well until four years before the index admission, at which time she was admitted to hospital for similar symptoms, responding well to neuroleptics. At age 16 she was in an automobile accident and suffered brief loss of consciousness. About 20 years before the index admission she used to drink a bottle of rum fortnightly and continued to do so for five years.

She was an endomorphic woman and showed lability of mood but no blunting of affect, mild impairment of recent and remote memory, poor judgement and insight, and disorientation to time. Thought disorder was not present. She expressed several delusional ideas: she thought that her neighbours could 'listen in' on her conversation and that someone was trying to 'break in' to her home. She said that Richard Nixon lived nearby under the name of Richard

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Smith. Intelligence was estimated as borderline. Physical examination revealed bilateral extinction of the hand stimuli on double simultaneous tactile discrimination and a left palmomental reflex. She was obese and mildly hypertensive. Waking EEG showed symmetrical biparietal slow activity in the theta range. The Reitan-Indiana Aphasia Screening Test showed constructional apraxia. Skull X-rays, brain scan and CSF were normal. Blood testing revealed mild diabetes. A sister was confined to a state mental hospital because of epilepsy; but there was no other family history of mental illness.

The patient's misidentification of her children persisted despite a four-week course of haloperidol, 20–45 mg a day. Marked urinary retention made it necessary to discontinue drug therapy. This was followed within one week by disappearance of the Capgras syndrome and improvement in her other delusions. She was discharged to return to her family; but her sudden improvement remains unexplained.

Discussion

Although Capgras' syndrome has been equated with schizophrenia, neither of our patients would satisfy rigorous criteria for this diagnosis because of the presence of signs and symptoms of coarse brain disease. A 'released' psychosis is unlikely because of the absence of identifiable predisposing factors such as a family history of schizophrenia or a premorbid schizoid personality.

There was evidence for right-sided cerebral dysfunction in both cases. The first patient had demonstrable right-sided brain damage with consistent EEG and behavioural abnormalities. In the second patient, right cerebral dysfunction was inferred from the presence of a left palmomental reflex and constructional apraxia, and the aetiology was presumed to be head injury. Pincus and Tucker (1974) suggest that a specific type of visual agnosia, non-recognition of faces, originates in a single retro-rolandic parietal lobe lesion and occurs three times more frequently with lesions of the right cerebral hemisphere than with lesions in the left. Bodamer (1947) suggested that the inability to recognize familiar faces was a quite specific variant of visual agnosia which he called *prosopagnosia* (i.e. 'face non-recognition').

Meadows (1974) gathered 42 cases of prosopagnosia from published reports over a period of 44 years, in 8 of which necropsy was performed. He reported that the right occipito-temporal region is always involved, though in each case there has been some lesion in the left hemisphere too. Commenting on Meadows' study, *The Lancet* (1974) tentatively concluded 'that the essential damage is of cortex adjoining the striate and parastriate areas, and also of the underlying tracts, among which is the optic radiation'. The non-dominant hemisphere might also be implicated in prosopagnosia because of its role in the recollection of pattern and topography.

We suggest that in our patients prosopagnosia may have been the primary expression of a specific cerebral dysfunction and that this formed the basis for a delusional elaboration resulting in Capgras' syndrome. This process could be considered analogous to the development in patients with Korsakov's psychosis of confabulations, which then function to mask the amnesic defect. With these points in mind, we further suggest that physicians encountering Capgras' syndrome should examine their patients for underlying organic brain disease, and more particularly for prosopagnosia.

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