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The Capgras Syndrome Following Head Injury

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The Capgras syndrome, first described by Capgras and Rebould-Lachaux (1923), is the name applied to a peculiar delusional system in which the patients come to believe that persons well known to them—usually close relatives—are impostors who have assumed the exact appearances of those whom they have supplanted. The majority of examples of this condition have been observed in schizophrenic patients but, like most eponymous titles in medicine, the term was applied to a disease pattern that failed to fit conveniently into existing classification. Most branches of medicine, with the gradual accumulation of new information, pass through an 'identification' phase when new syndromes are being discovered. A further 'synthesizing' phase is eventually reached when researchers, equipped with greater knowledge regarding likely aetiology, pattern of symptoms and treatment, are able to appreciate the wider implications and ramifications of the problem, as well as uncertainties surrounding the initially identified disease pattern. In psychiatry, titles that emphasize specific patterns of symptomatology tend to be the rule rather than the exception, but advances in psychopathology, neuropsychology and neuropathology imply a need for reassessing the contribution of a purely phenomenological approach to the problem. Such an approach may well be overdue as far as the Capgras and other syndromes are concerned. The following case is presented in the hope that the condition, when it is recognized, will be evaluated in terms of its aetiology, and that attention will be paid to the more fundamental questions of psychopathology and related patterns of neuropsychological function.

Because of the excellent review of this condition by Enoch, Trethowan and Barker (1967), who added three cases of their own, any further extensive examination of the literature would be superfluous. Three cases reported by Gluckman (1968), Ball and Kidson (1968) and Minns (1970) have since been added. Gluckman's case is interesting in that it appears to be an example of the syndrome occurring in a setting of apparent organic brain disease. Admittedly the evidence for an underlying dementia was somewhat equivocal, and, as Gluckman himself points out, the patient appeared to be suffering fundamentally from paranoid schizophrenia, the diagnosis most commonly given to cases in which the Capgras syndrome has been diagnosed. Indeed, it appears that in virtually all reported cases in the literature the patients have been suffering from schizophrenia or severe manic-depressive psychosis, and as far as is known the occurrence of this syndrome in the presence of unequivocal brain disease or damage has not previously been described. Enoch (1963), who considered that the central symptom occurs in the setting of clear consciousness, observed in his second case the development of dementia some seven years after the onset of an illness which initially was diagnosed as paraphrenia and chronic alcoholism. However, as this writer observed, 'the basic mechanism in the Capgras syndrome is fundamentally different from the misidentification which occurs in the organic (psychoses) states where there is an impaired sensorium.' It follows, therefore, that a description of the syndrome following a serious head injury in an individual with no previous history of psychiatric disturbance may be of value in enabling us to understand more clearly the nature of the symptoms and their aetiology.

Description of Case

The patient was a single male, apprentice plumber, aged 20, who was first seen on 5 July 1969 when he was referred for psychiatric assessment following a serious head injury that had occurred on the previous

27 March. He attended accompanied by his mother, who described his birth, childhood and upbringing as normal and uneventful without any episode of serious illness. He was on good terms with all members of his family and had just completed his apprenticeship in a satisfactory manner. There was no family history of mental illness, and the patient had not, to our knowledge, experienced any psychiatric symptoms prior to his accident.

On 27 March 1969 he was injured while driving his own car, which was largely demolished by the impact. He was admitted to hospital in an unconscious state; there was a deep left parietal laceration with associated bleeding from the left ear. Skull X-rays showed a fracture of the left petro-squamous temporal bone, extending from the internal auditory canal upwards and backwards at an angle of 45° near the parieto-temporal suture. During the first 24 hours his condition fluctuated, with some signs of deterioration. A left carotid angiogram performed on 30 March showed no significant shift of the mid-line vessels, although there was some slight elevation of the middle cerebral artery, consistent with a diagnosis of temporal lobe contusion or of a small extra-dural haematoma on the floor of the left middle fossa. He was treated conservatively, and over the next four weeks there was a gradual return to consciousness. By 15 April he was able to respond to questions, but was noticeably restless and had some difficulty in swallowing. This last symptom had disappeared by 22 April, and a programme of rehabilitation was started. He had, therefore, been semicomatose for one week, and this had been followed by a four week period of disturbed consciousness associated with irritability, restlessness and disorientation.

On 9 May he was noted to have some ptosis of the left upper eyelid and a degree of left divergent squint with apparent diplopia. Eye movements appeared full and normal; the left pupil was slightly larger than the right, although both reacted to light and accommodation. His visual acuity was 6/8 right and 6/9 left, and he showed a left homonymous hemianopia. He was diagnosed as having suffered a third nerve lesion on the left side mainly on the evidence of the ptosis.

He was discharged home on 12 May, but on 20 May his mother reported difficulties over his management at home, and for this reason he was referred for psychiatric assessment.

When first seen on 5 July in the Department of Psychological Medicine, the patient's only complaint was some difficulty with reading and a poor memory. He had no recollection of his accident, and it became apparent that he refused to believe that he had been involved in one or sustained a head injury. He also had no apparent memory of events since his accident and failed to recognize that he was amnesic for this period of time. When questioned later, his mother was of the opinion that full consciousness had not in fact been restored until shortly before his discharge. Certainly the patient had no recollection of his stay in hospital between the end of March and the middle of May.

On examination, he appeared reasonably cheerful, but at times was slightly aloof and mildly disinhibited in speech and manner. When asked about his mother (who had accompanied him to hospital), he referred to her as 'that old woman who looks after me'. When further questioned about his mother, he said that all his family had been killed by Chinese communists and that the persons now claiming to be his parents and siblings were impostors, who despite their *exact* resemblance to members of his own family were in fact other persons who had assumed their outward appearance. Attempts to get him to see matters differently were brushed aside somewhat irritably. It became apparent that he believed that he had been involved in some catastrophic war-time experience. He recalled a particularly vivid hallucinatory experience whilst in hospital during which he had seen his parents and siblings taken out and shot by Chinese communists and had then had to assist in their burial. To the patient this was a dream in which he was a member of the Australian armed forces fighting the Chinese on the Chinese mainland. When he recovered full consciousness in hospital he concluded that he was in China waiting for a boat to take him back to Australia.

He mentioned having difficulty in seeing objects on his left, and when this was explained as being the result of a severe accident he denied the possibility of such an event on the grounds that he was such an excellent driver: 'I still don't believe I had an accident, I must have got in a fight, someone probably hit me over the head with a chair. The family says I did have an accident, but of course they're lying.' Formal testing showed a poor capacity to memorize facts given to him. At times he appeared to use words incorrectly, and there was evidence of some degree of nominal dysphasia. However, he was able to write and draw simple pictures without any obvious difficulty. His mother commented particularly on his tactlessness and irritability, and his general increase in weight on account of a voracious appetite. Electroencephalographic studies carried out on 1 August and 22 December were both essentially normal.

He was seen again on 12 September, when his main complaint was difficulty with reading. As he

put it: 'I can't see when I'm reading it. I see a word and go on to the next, and I can't see the word I've just read.' He claimed he was feeling well, was living with his mother and father, and 'another boy and two girls' (his siblings). He admitted that he had been involved in a smash, not only because he had been told so, but also because he had seen the remains of his wrecked vehicle. He appreciated that he had some visual defect, as he remarked that he had not driven since his accident on the grounds that this would be dangerous if one could not see properly. His mood was jocular and cheerful, and he tended to be rather boastful. He was less irritable, a fact confirmed by his mother, who had observed a general improvement in his behaviour. Even so, she felt he was still uncertain whether she and her husband were his true parents, and his remark about his siblings showed that this uncertainty extended to them also. On 18 December 1969 he was more concerned about his visual field defects. The peripheral fields to the left appeared satisfactory, but he had what amounted to a left central scotoma. He had himself observed that when he was being driven in a car he could see the left traffic indicator of a vehicle ahead, but failed to see brake lights and other signals nearer the mid-line of his vision. He was trying to refresh his memory regarding his trade as a plumber, but still had difficulty in comprehending and remembering what he read. Other aspects of memory functioning had improved, he could recall the day of his discharge from the neurosurgical ward, but he could remember only isolated events occurring during his stay in hospital. He still showed some uncertainty about the identity of his parents.—'I watch them—they do the same things as my parents did—I know they must be my parents because they do the same things, and they've been so kind to me. They wouldn't be so kind if they weren't my parents, would they?' His general improvement was confirmed by his mother, although she still felt that he would have some difficulty in coping with his work if he returned to full employment.

The visual field defect, first noted as a left homonymous hemianopia was confirmed by Dr. R. F. Wood, Honorary Ophthalmologist at the Royal Brisbane Hospital, who noted that in fact he now had two left para-central scotomata, the one in the left eye somewhat larger than that on the right. Although the clinical signs showed that injuries were sustained primarily on the left side of the skull, the presence of an incongruous left homonymous hemianopia with macular sparing would appear to indicate involvement of the right temporal radiation (Guillaumat *et al.*, 1959). Steady improvement in his vision during a period of twelve months was characteristically associated with awareness on his part of a unilateral visual defect. He appeared, in summary, to have some features of a frontal lobe syndrome together with evidence of bilateral temporo-parietal damage, a severe memory defect and mixed dysphasic involvement, all in a setting of generalized intellectual impairment. He also showed the characteristic symptoms of the Capgras syndrome.

During the period 18 July to 18 December, he was seen for psychological assessment on four separate occasions. On the 18 July he obtained a Full Scale I.Q. of 80 in the Wechsler Adult Intelligence Scale, and his performance on a battery of psychological tests indicated the presence of generalized intellectual impairment in an individual who was basically of at least average intellectual ability. He had a marked short-term and recent memory deficit, mixed dysphasic impairment and general cognitive slowing. When last seen on the 18 December, there was evidence of some recovery of intellectual functions, the patient obtaining a re-test Wechsler Adult Intelligence Scale I.Q. of 95. However, there was very little improvement in memory functioning, and there was still some evidence of residual dysphasic impairment with associated confabulation.

Discussion

An understanding of the development of the Capgras syndrome in this patient can probably be gained by considering the sequelae of his cerebral injury. However, four aspects of our patient's symptoms need to be considered beforehand.

(1) Their relationship to anosognosia.

(2) The role of prosopagnosia.

(3) The role of post-traumatic delirium and memory loss in the production of a delusional belief.

(4) The fact that the Capgras syndrome most frequently occurs in a setting of schizophrenic illness.

Some aspects of the Capgras syndrome undoubtedly resemble the paranoid reactions associated with anosognosia accompanying parietal lobe damage, and particularly behaviour presenting in somatoparaphrenia, originally described by Gerstmann (1942) and discussed by Critchley (1953). Critchley quotes an example of a negress who denied ownership of a paralysed arm. To use her own words when confronted by the true facts: 'My eyes and my feelings don't agree and I must believe

my feelings. I know they (referring to her hand and arm) belong to me, but I can feel that they're not, and I can't believe my eyes.' There was some evidence that our patient had sustained parietal lobe damage, and there is a striking resemblance between his unwillingness to believe the evidence of his senses and the anosognosic patient who observed the conflict between her feelings and the facts revealed by visual examination.

Our patient's refusal to acknowledge the true identity of his family members led us to consider the relationship of the phenomenon of prosopagnosia to the Capgras syndrome. Bondamer (1947) suggested that the inability to recognize faces, either of oneself or of other people, was a quite specific variant of visual agnosic defect for which he proposed the term 'prosopagnosia'. Critchley (1953) considered the possible role of reduction in visual efficiency, and particularly constriction of the visual fields, in the production of this symptom. Our patient certainly had a bilateral homonymous hemianopia with retention of macular vision, but it was at no time possible to elicit satisfactory evidence of prosopagnosia. In short, the Capgras syndrome appears to be the exact antithesis of prosopagnosia, but how far perceptual difficulties contributed to our patient's symptoms—as they apparently do in the case of prosopagnosia—is uncertain. Undoubtedly during the period of impaired consciousness following his head injury perceptual defects occurred, and these may have contributed to the subsequent presenting condition. Perceptual misidentification during a period of clouding of consciousness would at an early stage create initial doubt as to the identity of his visitors, particularly as he considered them to be dead. However, it seemed to us that, irrespective of visual defects, of far greater importance to the aetiology of our patient's symptoms was the profound memory disturbance and the hallucinatory experience that occurred during the post-traumatic delirium.

In the first place, the patient's loss of memory was global and complete for all events relating to his accident; secondly, he presented with an anterograde amnesia of several weeks' duration. The clinical picture was, therefore, one of generalized reduction in intellectual functioning, disorientation for time and place, mixed dysphasic impairment, perceptual defects and generalized cognitive slowing; together with an inability to retain information received after the time of trauma. He did not believe that he had been involved in an accident, and he tended to become evasive and to confabulate, though *without* a catastrophic reaction. When closely questioned about the incident, the patient showed a rather unusual pattern of ambivalent denial, which did not, however, extend into a full anosognosic denial presentation. He was, for example, able to discuss two quite incompatible statements without at the same time conveying any degree of doubt.

In trying to understand the patient's fixed belief that all his family had been killed, the nature of the incident which preceded this 'delusion' is obviously of the greatest importance. Our patient constantly referred to this experience as a 'dream', but quite obviously he was not able to appreciate the unreal quality of his hallucinations in the manner customary with dreams. For him, the 'dream' was intense, frightening, real and quite convincing, and was in all probability a visual hallucinatory experience occurring in the course of a post-traumatic delirious state. The ability to recall such phenomena following delirium is not unusual. Lipowsky (1967) has observed: 'After recovery from delirium there is usually a partial or total amnesia . . . but clear memory for the hallucinatory episodes is not infrequent. Some patients recall delirium as a chaotic dream, or a nightmare.' The principal difference between our patient and others recovering from delirium was the quite profound memory disturbance that led to a total incapacity to relate his 'dream' to a severe road traffic accident, the occurrence of which he strenuously denied. In the absence of such reintegrative knowledge one can begin perhaps to understand the origin of his delusional beliefs about the death of his family and their replacement by impostors.

In conjunction with his memory disturbance there was also some degree of intellectual impairment, and these taken together in

combination with the vivid recall of an emotionally charged single event would have provided a restitutive and explanatory solution for the patient when faced with his parents and siblings in a hospital environment. If, as he firmly believed, his family had all been killed and he was in a hospital in China, how was he to explain their sudden re-appearance? The general disturbance of intellectual functioning would have made it difficult for him to perform a *volte face*, whereas denial and confabulation enabled him satisfactorily to structure an otherwise chaotic and inexplicable set of circumstances. His behaviour is strongly reminiscent of Bartlet's (1932) use of the phrase 'effort after meaning.'

The common occurrence of dysmnesia, confabulation, disorientation for time and place, reduplicative paramnesia and misidentification of persons and objects in patients emerging from coma has been previously noted by Weinstein and Lyerly (1968). They further noted that confabulation was frequently elicited in response to questions about the patient's disability, his stay in hospital and, furthermore, in relation to close relatives. The emotional impact and complex nature of confabulation and the use of denial mechanisms (although not involving symptoms of the Capgras kind) as seen through the eyes of a doctor-patient have been fully documented by La Baw (1969). Paterson and Zangwill (1944) drew attention to the tendency of patients, during post-traumatic confusional states, firstly to exhibit generalized disorientation, and secondly to maintain a bizarre state of 'double orientation' (Henderson and Gillespie, 1940), which provides them with a means of reconciling incompatable circumstances. One of their patients who normally lived in Grimsby was being treated in hospital in Scotland following a head injury. They remarked: 'We obtained good evidence from the previous record that the patient was exceptionally anxious to return home. In the early stages of his post-traumatic confusion he behaved as though he was actually at home. A little later, while recognizing that he was not at home, he was insistent that the hospital was in Grimsby and made repeated attempts to walk home.' It will be remembered that our patient considered he was in hospital on the Chinese mainland.

Such states of disorientation tend to be transitory, but if persisting could be justifiably viewed as having delusional status. Wernicke (1906) described 'delusions of explanation', which served to resolve otherwise inexplicable experiences, and it is of interest that Slater and Roth (1969) report that following post-traumatic delirium 'residual delusions' occur in rare cases after delirium has subsided and where the patient obstinately maintains a belief in the reality of an impressive hallucination.

As already mentioned, in the majority of patients the Capgras symptoms appear in the course of a schizophrenic illness, and in those cases showing eventual organic brain deterioration the preferred diagnosis when the Capgras symptoms first developed was schizophrenia or paraphrenia. It could, of course, be argued that our patient had developed a schizophrenic illness following his head injury, and it is perhaps noteworthy that, as Davison and Bagley (1970) have recently shown, schizophrenic-like illnesses following cerebral trauma most commonly occur when damage has been sustained by the temporal lobe. Certainly there is reason to believe that in our particular case temporal lobe damage had occurred, but it would be most difficult to justify a diagnosis of schizophrenia in this case. It seems more economical to explain our patient's symptoms in terms of underlying neuropsychological variables, without having to involve the development of a schizophrenic psychosis that cleared spontaneously without recourse to specific drug therapy.

Up to the present time all cases of the Capgras syndrome have occurred in patients diagnosed as suffering from a functional psychosis. Enoch *et al.* (1967) emphasized the importance of paranoid features in their patients and the development of the syndrome in a setting of clear consciousness. Some confusion exists over differences between 'l'illusion des sosies'—the term first used by Capgras and Rebould-Lachaux—and 'le phénomène des sosies'. Critchley (1953)—erroneously we believe—refers to the 'illusion des sosies' as occurring

usually in states of exhaustion and delirium, whereas he equates the 'phénomène des sosies' with the Capgras syndrome. It might be argued, therefore, that our patient was exhibiting the phenomenon of doubles, although the persistence of his delusions long after full consciousness had been restored seems to speak against this diagnosis. On the other hand, there was no clear evidence of premorbid paranoid traits and the symptoms undoubtedly developed at a time when consciousness was grossly impaired. We could not discover any pre-existing psychopathology that might have played a part in the development of the syndrome in this particular case. As time has gone by, with gradual recovery of psychological functioning, reduced confabulation and continuing presentation of contradictory experiences, the Capgras symptoms have faded, although at the last interview it was apparent that if closely questioned the patient still had some reservations about the true identity of members of his family. He was not able, if pressed, to voice complete certainty as to the true identity of his parents. To use his own words: 'They must be my parents—they behave as my parents would—people wouldn't be so kind to me if they weren't my parents, would they? I can't believe the war did happen, but sometimes feel it could have and might have—it still could, couldn't it? It's about twenty per cent possible, I suppose, that it did happen.' He was, however, quite satisfied that he had been involved in a road traffic accident, as his attendance in Court appeared to provide him with irrefutable evidence of its occurrence.

Needless to say, on the basis of what we now know about our patient and other patients suffering from the Capgras syndrome, it is not possible to present a satisfactory neuropathological correlate for these symptoms when they occur in patients with functional psychoses. In our patient, the level of consciousness and the role of a major emotional experience were obviously crucial. However, previous reports have paid little attention to memory functioning or to the role of a major emotional experience, and one might wonder to what extent these factors are important in patients who developed the characteristic features of the Capgras syndrome in the course of a schizophrenic or manic-depressive psychosis. Evidence of damage to the parietal and temporal lobe in our patient suggests the possibility that failure to integrate memory, perception and affect may play a part in the aetiology of the Capgras syndrome when this occurs in a setting of functional psychosis.

A full explanation of the syndrome cannot be offered, but it is now apparent that it can occur in a number of different psychiatric disorders, including organic brain syndromes. It follows, therefore, that there is little reason for treating the syndrome as a distinct entity with a special title. We agree with Enoch that there is more to be said in favour of referring to Capgras *symptoms* rather than a Capgras *syndrome*, and we further suggest that the time may have come for the abandonment of this particular eponymous title in psychiatry. Many schizophrenic symptoms are bizarre in the extreme, but in general we do not consider them as being essentially different from other symptoms and signs of the disease. Greater understanding of such symptoms will only be gained by greater comprehension of neuropsychological, neuropathological and psychopathological mechanisms underlying the phenomena. In the meantime, little purpose is served by singling out one class of delusional belief as essentially different from others occurring in the course of functional and organic psychoses.

Summary

This paper describes the characteristic features of the Capgras syndrome occurring in a twenty-year-old man following a severe head injury. His delusional beliefs about his family members appeared to be related to a profound memory disturbance and the recollection of a vivid hallucinatory experience that took place during the period of post-traumatic delirium. As his general condition improved, the Capgras symptoms gradually faded, although it is not possible to be certain that they have wholly vanished although a year has passed since his accident. The neuropathological and psychopathological aspects of this case and their relationship to the Capgras syndrome occurring in a setting of functional psychosis are discussed.

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