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Capgras syndrome: a novel probe for understanding the neural representation of the identity and familiarity of persons

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SUMMARY

Patients with Capgras syndrome regard people whom they know well such as their parents or siblings as imposters. Here we describe a case (DS) of this syndrome who presents several novel features. DS was unusual in that his delusion was modality-specific: he claimed that his parents were imposters when he was looking at them but not when speaking to them on the telephone. Unlike normals, DS's skin conductance responses to photographs of familiar people, including his parents, were not larger in magnitude than his responses to photographs of unfamiliar people. We suggest that in this patient connections from face-processing areas in the temporal lobe to the limbic system have been damaged, a loss which may explain why he calls his parents imposters. In addition, DS was very poor at judging gaze direction. Finally, when presented with a sequence of photographs of the same model's face looking in different directions, DS asserted that they were 'different women who looked just like each other'. In the absence of limbic activation, DS creates separate memory 'files' of the same person, apparently because he is unable to extract and link the common denominator of successive episodic memories. Thus, far from being a medical curiosity, Capgras syndrome may help us to explore the formation of new memories caught *in flagrante delicto*.

1. INTRODUCTION: FACING REALITY

The Capgras delusion is one of the rarest and most colourful syndromes in neurology (Capgras & Reboul-Lachaux 1923; Ellis & Young 1990). The most striking feature of this disorder is that the patient—who is often mentally quite lucid in other respects—comes to regard close acquaintances, typically either his parents, children, spouse or siblings, as 'imposters', i.e. he may claim that the person in question 'looks like' or is even 'identical to' his father, but really isn't. Although frequently seen in psychotic states, over a third of the documented cases of Capgras syndrome have occurred in conjunction with traumatic brain lesions, suggesting that the syndrome has an organic basis (Signer 1994).

Capgras syndrome should be distinguished from a related class of disorders called prosopagnosia, characterized by an inability to recognize people's faces (Damasio 1985; Farah 1990). Prosopagnosia is usually caused by bilateral lesions in the inferior temporal lobes (IT), regions of the brain thought to be at least partially specialized for face recognition (Damasio *et al.* 1982). When given a mixture of photographs of unfamiliar (novel) and familiar faces and asked to sort them into two piles, prosopagnosics are unable to do so; yet remarkably, they register a stronger skin conductance response to familiar faces (as do normals), implying that the face-processing machinery is still connected to the limbic system (Bauer 1984, 1986;

Tranel & Damasio 1985, 1988; Bruyer 1991; Dennett 1996). Apparently there are two components to the visual recognition of a familiar face, one of which is responsible for conscious recognition of the face and the recall of associated semantic information, whereas the other is responsible for the limbic-mediated emotional arousal—the emotional valence, including the feeling of familiarity, that accompanies the conscious recognition of a familiar face. A dissociation between these two would not only explain states such as *déjà vu* (familiarity without recognition) and *jamais vu* (recognition without familiarity), that are sometimes associated with temporal lobe seizures (Bancaud *et al.* 1994), but would also explain why it is possible to get a skin conductance response even in the absence of conscious recognition of a face.

Adapting an explanation by Bauer (1984, 1986), Ellis & Young (1990) have suggested that the Capgras syndrome represents a 'mirror image' of prosopagnosia in that the ventral route from the visual centres to the temporal lobes may be preserved (so as to allow overt, conscious face 'recognition'), but the dorsal visual route responsible for giving the face its emotional significance is damaged. Perhaps the only way the patient can make sense of the absence of this emotional arousal is to form the belief that the person he is looking at is an imposter.

This explanation leaves two questions unanswered, however. First, why is the phenomenon specific to close relatives? One possibility is that only with one's parents or spouse does one expect a glow of arousal, and

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therefore its absence leads to a confabulatory delusion that one's parent is an imposter. With an emotionally neutral person on the other hand, such as one's mailman, one does not expect such arousal, and therefore there is no incentive for generating a delusion.

A second related question is, why does the mere absence of this emotional arousal lead to such an extraordinarily far-fetched delusion? Why doesn't the patient just think, 'I know that is my father but I no longer feel the warmth'? One answer is that some additional lesion, perhaps in the right frontal cortex, may be required to generate such extreme delusions. In trying to explain a completely different syndrome (anosognosia) we have argued elsewhere that the left hemisphere seeks to preserve consistency at all costs by explaining away any discrepancies, whereas there may be a global 'consistency-checking' mechanism in the right hemisphere which ordinarily serves to counter-balance this tendency (Ramachandran 1995). Damage to such a mechanism may explain the extreme confabulations that one often sees in anosognosics following a right hemisphere stroke: the implausible explanation produced by the left hemisphere is no longer censored by the right hemisphere. It may be, therefore, that to develop a full-blown Capgras delusion one needs a *conjunction* of two lesions: one which affects the brain's ability to attach emotional significance to a familiar face, and one which affects the global consistency-checking mechanism in the right hemisphere.

We recently had the opportunity to study an intelligent and mentally lucid young man who developed Capgras syndrome following a head injury. One of our goals was to test the hypothesis that his face-recognition mechanisms were intact, but disconnected from limbic areas. Our prediction was that unlike prosopagnosics he should experience no difficulty in distinguishing familiar faces from unfamiliar ones, but should show a loss of the normal skin conductance response to familiar faces. We tested this directly in our patient (DS), and in the course of doing so discovered some novel and hitherto undescribed aspects of this syndrome (Ramachandran 1996). One phenomenon in particular, DS's occasional inability to link successive episodic memories of the same person, may illuminate how new memories are formed and organized.

2. CASE DESCRIPTION

DS was a 30-year-old Brazilian man who had been in a coma for three weeks following a head injury (right parietal fracture) sustained in a traffic accident. During the subsequent year, he made remarkable progress in regaining speech, intelligence, and other cognitive skills. He was brought to us by his parents principally because of his tendency to regard them as imposters. When we first saw him he appeared to be an alert and fairly intelligent young man who was not obviously hysterical, anxious or dysphoric. A 'mini' mental status exam (serial sevens, three objects, writing, orientation in time and place, etc.) revealed no

obvious deficits in higher functions, and there was no evidence of dementia.

The most striking aspects of his disorder were that he regarded his father as an 'imposter' and he had a similar, although less compelling, delusion about his mother. When asked why he thought his father was an imposter his response was 'He looks exactly like my father but he really isn't. He's a nice guy, but he isn't my father, Doctor'. The following dialogue then occurred:

Experimenter (E)

'But why was this man pretending to be your father?'

Patient (P)

'That is what is so surprising, Doctor—why should anyone want to pretend to be my father? Maybe my father employed him to take care of me—paid him some money so that he could pay my bills...'

According to his parents he had entertained these delusions for the two years since he had been discharged. Interestingly, they related that DS never treated either parent as an imposter when speaking with them over the telephone—the difficulty arose only in the visual modality. This is inconsistent with a recent suggestion by Luaute & Bidault (1994) that Capgras is similar to hemineglect in being supramodal. The unimodality of DS's delusion is important, for it implies that the patient is not simply 'crazy'; his delusions are confined to the visual modality, as one might expect from our inferotemporal-limbic disconnection hypothesis. The finding also implies that DS does not simply have retrograde amnesia with regard to his parents. A similar dissociation is often seen in prosopagnosia—the patient cannot recognize familiar people by sight, but can do so when listening to their voices.

According to the parents, other people who were once familiar to DS were readily recognized by him and did not provoke the delusion that they were imposters, a finding typical of Capgras patients. Interestingly, DS also had the delusion with regard to pictures of himself:

E: (pointing to photograph of DS from two years ago when he had a moustache): 'Whose picture is this?'

P: 'That is another DS who looks identical to me but he isn't me—he has a moustache.'

This delusion could not be provoked in DS simply by any visual image of himself, for instance it did not occur when DS looked at himself in a mirror.

DS's tendency to 'duplicate' himself—that is to regard himself as a distinct person from a former DS—also sometimes emerged spontaneously during conversation. For instance, on one occasion he volunteered, 'Yes they sent a check, but they sent it to the other DS.' Again, other than this very odd remark there was nothing in his conversation to suggest psychopathology. DS usually did not regard objects as duplicates but on some occasions he would run his fingers through his own hair and call it a 'wig—not my hair', partly because his scalp felt odd and unfamiliar as a result of scars from the neurosurgery he had undergone. On rare occasions, DS also duplicated countries, claiming at one point that there were two

Panamas (where he had recently gone to for a family reunion) and two United States.

One final point is worth noting. DS's father had seen one of us (V.S.R.) on television demonstrating 'that the brain could be tricked by simply using a mirror' to relieve phantom-limb pain (Ramachandran *et al.* 1995; Ramachandran & Rogers-Ramachandran 1996). Although he realized that phantom limbs were unrelated to the Capgras delusion, DS's father began to wonder whether a similar 'simple trick' could be used on his son to help him get rid of his delusion. To achieve this his father adopted the following extraordinary procedure: He walked into his son's room one day and announced, 'the man who you have been with all these days is an imposter—he isn't really your father. I have sent him away to China. I am your real father—it's so good to see you son.' DS's delusion seemed to abate slightly after this 'treatment,' as evidenced by the following dialogue:

E: 'Who is the man who brought you today?'

P: 'My father.'

E: 'Who was taking care of you?'

P: 'That guy has gone back to China. He looked very similar to my father, but he is gone now.'

Yet during a subsequent interview a week later DS had reverted to his original delusion, claiming that the imposter had returned. Also, his father told us in confidence that although DS had accepted him now as his father 'intellectually', he had not yet done so emotionally. We are now planning to try variants of this 'treatment' on other Capgras patients to see whether a more permanent improvement can be achieved.

3. LESION ANALYSIS

A CT scan without contrast revealed that all ventricles were enlarged without change, with the right lateral ventricle being especially large. Bilateral areas of encephalomalacia were noted, most prominent bifrontally. Of course, a subtle disconnection between IT and the limbic system of the kind we have postulated here would not be visible on a CT scan, but we are planning future imaging studies on this patient using fMRI and PET. Previous studies on Capgras patients have found lesions predominantly in the temporal (Signer 1994) and right fronto-parietal (Benson 1994) cortices, which is consistent with our two-lesion (IT-limbic, and right fronto-parietal) hypothesis.

4. RESULTS

(a) *Experiment 1: electrodermal response to familiar versus unfamiliar faces*

(i) *Rationale*

The Capgras delusion may be a functional mirror image of prosopagnosia, in that the Capgras patient visually 'recognizes' a familiar face, but lacks the emotional 'glow' that such a face would normally evoke (which is associated with the skin conductance response one sees in prosopagnosics when they are shown familiar faces).

(ii) *Procedure*

Electrodermal activity was measured as skin conductance magnitude between the middle phalanges of the index and middle finger of the subject's left hand. Dermal contact areas were cleaned with alcohol and lightly abraded. Silver/silver-chloride electrodes were then affixed with velcro straps. Skin conductance response signals went first to a UFI model 2701 skin conductance meter, which also displayed skin conductance level, then into a MacIntosh computer, which amplified and displayed the data.

The sets of photos seen by the subjects contained images of people DS had delusions about (mother, father, self, grandfather) and well-known faces (Bill Clinton, Albert Einstein, Elizabeth Taylor, Michael Jordan, etc.), randomly interleaved with pictures of unfamiliar people taken from people's family photographs. The control group consisted of six college undergraduates. Before the experiment, subjects were told that they would be shown pictures of faces, some of which would be familiar to them, some of which would be unfamiliar. After the electrodes were attached, subjects were given a period to establish baseline SCR activity, then each photograph was shown for 2 s, with the experimenter waiting between photos for SCR levels to return to baseline, typically from 15 to 25 s, prior to showing the next image. Following Tranel & Damasio (1988) only the largest skin conductance response within a latency window of 1–5 s was recorded. DS was tested on three separate occasions, with three different photograph sets, over a six-week period with this procedure, referred to as DS I, DS II, and DS III in table 1. Responses to familiar faces were compared with responses to unfamiliar faces using the Mann–Whitney *U*-test (Siegel 1956), a nonparametric test that can be used on data from distributions which are not normal (SCR distributions are non-normal (Venables & Christie 1980)).

(iii) *Results*

See table 1.

(b) *Experiment 2: accuracy of judgements of gaze direction*

(i) *Rationale*

Some Capgras patients have been shown to have other problems processing faces (Young *et al.* 1993), and amygdalotomy has been shown to affect ability to judge gaze direction (Young *et al.* 1995). Also, Teske (1988) has shown that normal individuals are more inclined to judge, mistakenly, that a person is looking at them if that person is familiar. Given this link between gaze direction and familiarity, we wondered whether gaze hyperacuity—the ability to judge where a face in a photograph is looking (Gibson 1966; Anstis *et al.* 1969)—would be impaired in our patient.

(ii) *Procedure*

Three different series of images were prepared. In the first series, the same model looked either directly at the camera lens, or at a point 4 or 8 cm to the

Table 1. *Skin conductance response magnitudes (in microSiemens). Numbers in ‘mean’ columns correspond to mean response magnitudes; percentages of non-zero responses are included under ‘frequency’*

	familiar					unfamiliar						
	n	frequency	mean	s.d.	U	n	frequency	mean	s.d.	U		
		%					%					
DS												
I	7	71	0.12	0.18	21	$p > 0.05$	8	50	0.03	0.03	36	$p > 0.05$
II	3	100	0.64	0.41	17	$p > 0.05$	13	100	0.55	0.32	15	$p > 0.05$
III	7	86	0.46	0.32	80.5	$p > 0.05$	16	81	0.72	0.42	31.5	$p > 0.05$
control group												
S1	3	100	0.29	0.05	3		24	25	0.03	0.08	2.55†	$p < 0.01$
S2	6	83	0.35	0.27	17		17	82	0.08	0.08		$p < 0.01$
S3	5	100	0.19	0.23	17.5		18	82	0.07	0.10		$p < 0.05$
S4	3	100	0.76	0.40	4.5		17	100	0.15	0.27		$p < 0.05$
S5	5	80	0.54	0.42	34		24	36	0.27	0.40	1.50†	
S6	6	83	0.35	0.35	28		22	48	0.07	0.10	2.21†	$p < 0.05$

† z values.

Table 2. *Accuracy judging gaze direction*

degrees of deviation ...	3.3	6.6	9.9
DS	4/24 (17%)	9/23 (39%)	3/6 (50%)
controls (n = 7)	67/96 (70%)	79/80 (99%)	9/9 (100%)

right or left of the lens. Since the model was 0.7 m from the camera lens, the 4 cm offset corresponded to 3.3° of visual angle, and the 8 cm offset to 6.6°. The second series also contained images of models diverting their eyes an additional 4 cm from centre (9.9° of visual angle). Finally, a third series was administered two weeks later.

(iii) *Results*

See table 2. For the first series of 30 photographs, DS answered that the model was looking at him for every one, hence he was only correct on the seven trials in which the model actually was looking at him. Only in a second set of trials, where the model's eyes were sometimes 9.9° off centre, did DS's accuracy improve.

Remarkably, on the eighth trial of the first series of 30, DS claimed that the identity of the model had changed, even though all the photographs were, of course, of the same person. 'This one is older' he asserted, adding that this was a picture of a different woman from the one depicted in the earlier trials ('This is a lady, the other one is a girl'). Later in the testing, DS made another partition, so that now there were three models according to him, which he described as an old one, a young one, and a second young one, slightly younger than the other. When questioned at the end of testing, he was consistent and stated that there had been three models. During a subsequent gaze direction test with a new model, DS again claimed that there were two different models, making the partition at about the same trial as he did during the first test. This test had fewer trials, so perhaps DS did not have time to create a third identity for the model.

DS showed the same tendency to assign many identities to a single model when he was retested two weeks later.

(c) *Experiment 3: accuracy of face discrimination*(i) *Rationale*

Is it possible that DS simply had a problem in discriminating faces? This seemed unlikely from the fact that he recognized famous faces such as those of Clinton and Einstein, but we also conducted a more formal experiment.

(ii) *Procedure*

The stimuli consisted of nine pairs of photographs of college students not known to DS. Each pair was of the same person taken from two different points of view. In each trial, DS was simultaneously shown either two photographs of different people, or two (different) photographs of the same person, and he was to say whether the photographs depicted the same person or not. This test consisted of 16 trials.

(iii) *Results*

DS was correct on 14/16 trials. Four normals were tested with this set of photos, and all achieved scores of 16/16. DS tended to look closely at the details of the faces, rather than taking in the whole face as a 'gestalt'.

(d) *Experiment 4: perception of emotions in others*(i) *Rationale*

Perhaps the failure of DS's brain to produce a larger SCR when he sees close relatives is part of a larger

disturbance in his emotional ability. To rule this out, we tested DS's ability to sense emotions in others with the following experiment.

(ii) *Procedure*

A series of eight pairs of digitized images of models posing basic emotions such as fear, anger, and happiness (from Ekman 1975) were used. In each pair, the two images were either of the same model or of two different ones, and the emotions expressed were either the same or different. Each time, DS was asked what emotion the models were expressing, and whether they were expressing the same emotion.

(iii) *Results*

DS was accurate in saying which emotion was expressed, and whether the two models were expressing the same emotion or not. Thus DS's ability to discriminate emotions is largely spared; what is compromised is his ability to link particular familiar faces with the appropriate affect in his memory.

6. DISCUSSION: THE UNBEARABLE LIKENESS OF BEING

'One *can't* believe impossible things.'

'I daresay you haven't had much practice,' said the Queen. 'When I was your age I always did it for half-an-hour a day. Why, sometimes I've believed as many as six impossible things before breakfast.'

Lewis Carroll, *Through the Looking Glass*

During the last three decades, considerable progress had been made in understanding the neural basis of memory formation, but most of this research falls into two categories: (1) investigation of the actual synaptic changes (e.g. LTP) involved—studied elegantly in aplysia (Matzel *et al.* 1992; Skehel *et al.* 1995) and in *in vitro* hippocampal slices (Oliver *et al.* 1989; Sejnowski *et al.* 1989); and (2) investigation of anterograde amnesia results from medial temporal lobe/hippocampal damage (Milner *et al.* 1968; Mishkin 1978; Squire 1987; Weiskrantz 1987; Schachter 1995). Surprisingly, the equally important narrative or 'constructive' aspects of human memory, for example, the mechanisms of retrieval, the creation of new categories and a tacit taxonomy of these categories (e.g. tokens versus types), the encoding of spatial and temporal context, and the binding of objects across successive episodic memories, have rarely been studied experimentally—although their importance was recognized as far back as Bartlett (1932). The vast psychological literature on this topic is largely uninformative; in particular, we have no idea of what the neural substrates of these elusive mechanisms might be. Our research suggests that Capgras, and other allied delusional misidentification syndromes, such as Fregoli syndrome (in which the patient tends to misidentify several strangers as a single prototype whom he knows already, Courbon & Fail (1927)), might provide a valuable opportunity to experimentally probe these enigmatic aspects of human memory.

Bauer (1984, 1986) suggested that the reason that prosopagnosics register a skin conductance response

when they see familiar faces is that this response is mediated by the dorsal stream of visual processing leaving the occipital lobe, which is intact, whereas the ventral stream, which is presumably responsible for conscious recognition of faces, is damaged. Ellis & Young (1990) adapted this approach to Capgras syndrome, making the ingenious suggestion that Capgras is a mirror image of prosopagnosia: damaged dorsal stream, intact ventral stream. There are several problems with this idea, however: (1) postcentral lesions in Capgras patients are more often located in the temporal lobes than in the occipital or parietal lobes (Signer 1994); (2) the dorsal visual stream primarily contains information from peripheral vision, whereas our visual interactions with people would seem to involve mainly focal vision, a ventral stream function; (3) the ventral stream has dense reciprocal connections to the amygdala (Amaral *et al.* 1992), which is strongly implicated in the skin conductance response (although apparently not necessary for it, Tranel & Damasio 1989; Lee *et al.* 1989), whereas the dorsal stream does not; (4) Some Capgras patients sometimes also have face-processing impairments (Young *et al.* 1993), another ventral stream function. We propose instead that the principal cause of Capgras is a failure of communication between areas of ventral stream processing in the temporal lobe (e.g. IT and other face-sensitive areas around the superior temporal sulcus) and the limbic complex, especially the amygdala. This failure of communication leads to disturbances in memory 'management' of the kind seen in DS, specifically a relatively intact or even exaggerated ability to individuate different episodic memories, but a deterioration in the ability to generate enduring categories (e.g. 'my father') by extracting and linking a common denominator across successive episodes.

The Capgras syndrome is often regarded merely as a face recognition problem, but we would like to suggest that it is really part of a more general memory management problem. When you or I meet a new person, our brains open a new file, as it were, into which go all of our memories of interactions with this person. When DS meets a person who is genuinely new to him, his brain creates a file for this person and the associated experiences, as it should. But if the person leaves the room for 30 minutes and returns, DS's brain, instead of retrieving the old file and continuing to add to it, sometimes creates a completely new one. Why this should happen is unclear, but it may be that the limbic/emotional activation from familiar faces is missing and the absence of this 'glow' is a signal for the brain to create a separate file for this face (or else the presence of the 'glow' is needed for developing links between successive episodes involving a person). Alternatively, there may be a more basic flaw in DS's ability to extract and integrate the common denominator of successive episodic memories—a sort of temporal binding or mnemonic 'figure-ground' problem. Whatever the reason, DS's response to this failure is to assert that he is meeting a new person, one who looks very much like another person he just met. It is crucial to point out that DS has not lost his earlier

episodic memories of the person; he remembers the 'other' person, but simply behaves as if he has met two different people, and creates separate files for them. Again, consistent with the IT-limbic disconnection hypothesis, his problem is confined to the visual modality; DS can recognize his parents over the phone. Since the amygdala receives polymodal sensory input, the disconnection might be specifically between IT and the amygdala rather than say, amygdala and the hippocampus. A problem of this kind may underlie not only Capgras syndrome but also its parent category, reduplicative paramnesia (Pick 1903)—the belief that objects have been replaced by duplicates—as well as the occasional memory duplications seen in Korsakoff's patients (Zangwill 1941). Finally, one wonders whether a strengthening of these very same connections might underlie other delusional states. For instance, kindling in the amygdala of the kind that occurs in temporal lobe seizures might form the basis of interictal religious experiences, with the patient ascribing deep meaning and cosmic significance to everything around him. Alternatively, if the strengthening was less global and occurred for a single face node, the result might be deClerambault's syndrome (deClerambault 1942; Anstis, personal communication), an overwhelming romantic obsession that some young women develop with a single older man.

DS's memory partitioning problem showed itself in an extreme form while we were testing his ability to judge gaze direction. Recall that the test consisted of 30 digitized images of the same model looking either directly at the camera or 8 cm to the right or left of the camera lens. For each trial, the subject merely had to tell us whether or not the model's eyes were directed at him, but his response was to assert repeatedly that the identity of the model had changed. This finding nicely complements the intriguing observation of Teske (1988) who found that when normal people are asked to judge gaze direction, they are more likely to assert that familiar people are looking at them.

DS's tendency to 'split' people into multiple copies occurred only on some occasions, and more often with photographs of a person than in the person's presence. Such a waxing and waning of symptoms, of course, is only to be expected given that what we are dealing with here is not a 'pure' or classical anatomical disconnection of the kind Geschwind (1965) postulated for alexia without agraphia but, rather, a miscommunication between 'face' cells (or 'person-' or 'object-identity nodes' (Gross 1992; Tovee *et al.* 1996)) in the temporal lobe, and the temporal 'chunking' and binding mechanisms in the hippocampus and the amygdala (consistent with this view, 'face cells' in the amygdala are thought to be involved in linking successive views of the same face across time (Rolls 1995)). Our theory of partial functional disconnection would also explain why DS tended to duplicate more often with memories or photographs of people than in the presence of the people themselves. For example, on some occasions, when shown a photograph of his father, DS would identify him correctly, but in recalling the photograph a few minutes later claim that it was of 'that Jewish gentleman who looks like my

father.' When retrieving the memory trace, DS presumably dredges up a somewhat impoverished representation that fails to evoke limbic arousal, causing him to label its object an imposter, whereas when he is in the presence of his father there are a sufficient number of salient cues to adequately activate the limbic areas. (This would also explain why he regarded old photographs of himself as 'another DS' but never experienced the delusion while looking in a mirror.) On this hypothesis, one would expect a great deal of variability across different Capgras patients, depending on the extent of 'disconnection'. It would be interesting to test Capgras patients systematically to see whether the delusion is evoked more reliably by memories of a person, or perhaps with degraded photographs, than by the actual person.

DS was impaired in neither his perception of emotions in others, nor in expressing his own emotions. He was able to gauge correctly when two different models were expressing the same emotion or a different one, and to say which emotion the models were expressing. During the course of testing, DS expressed happiness, laughed and expressed impatience, and also showed the normal skin conductance responses to rapid, deep breathing and sudden loud noises, which seems to indicate that the limbic system itself is intact. Taken together with the fact that his ability to discriminate faces (experiment 3) is only mildly impaired, this would again seem to indicate that the communication between IT and the limbic system is impaired. In our view, this hypothesis is at least as seductive as the psychoanalytical view (Franzini & Grossberg 1995) that the Capgras delusion arises because of an 'unmasking' of latent, anxiety-provoking Oedipal impulses toward the mother (and sexual jealousy toward the father) followed by an attempt to resolve anxiety by calling them imposters (if she is my mother, why am I sexually attracted to her?). We find this latter theory to be highly implausible, given that there are documented instances of Capgras patients who believe that their pet dog has also been replaced by a duplicate!

In each of three tests, conducted over a six-week period, DS failed to show a difference between his skin conductance response to familiar people and his response to unfamiliar people. Of course, given that this is a single case study, and given the inherent difficulties of the SCR technique, this result should be regarded as highly tentative and as requiring confirmation in additional patients. But taken at face value the results are broadly consistent with the IT-limbic disconnection hypothesis—that it is the failure of faces to evoke the appropriate affective memories that leads DS to regard his parents as imposters. Interestingly, on some trials there were long-latency (between 4.5 and 6 seconds) SCR responses suggesting that DS sometimes relies on an alternative, more inferential means of identifying people, with an SCR delay reflecting this more circuitous route to the limbic system.

DS also occasionally appeared to have a general problem with visual categories—a 'taxonomy' problem. All of us have certain covert taxonomies of events and objects in our brains, e.g. ducks and geese are

birds, but rabbits are not. We did not specifically test this in DS but his remarks sometimes hinted at this possibility. For instance, he had an almost obsessive preoccupation with Jews and Catholics, and he tended to label a disproportionate number of recently encountered people as Jews. Is it conceivable that a similar Fregoli-like confusion can occur in otherwise normal brains, forming a basis for racist stereotypes? It is noteworthy that racism is so often directed at a single physical type (e.g. Blacks, Asians, etc.). Perhaps a single unpleasant episode with one member of a visual category sets up a limbic connection that is inappropriately generalized to include all members of that class and is notoriously impervious to 'top-down' intellectual correction. Indeed, one's intellectual views may be coloured (no pun intended) by this emotional knee-jerk reaction.

Perhaps the oddest aspect of DS's mnemonic problems is his tendency to regard himself as a double, a tendency for which there are two possible explanations (which are not mutually exclusive). First, photographs of himself from the past did not evoke limbic activation and warm feelings, and the person in the photographs is therefore to be rejected as 'another DS' (however bizarre that may seem to us with our intact brains). Second, his loss of emotional contact with people who matter to him most, such as his own parents, may lead him to say to himself: 'The reason I don't experience warmth is that they don't recognize me, and that in turn must be because I'm not the real DS.' Indeed, on one occasion he made the following poignant remark to his mother: 'Mother, if the real DS ever returns do you promise that you will still treat me as a friend and love me?'

Philosophers have often emphasized that if there is any aspect of our own lives that we can regard as axiomatic and beyond question, it is our own personal identity. This sense of a single, unified 'self' runs like a golden thread through the whole fabric of our experience. The Capgras patient, on the other hand, inhabits a strange no-man's land between illusion and reality where even this sense of an enduring, unitary self can no longer be assumed. Studying these patients may therefore not only allow us to observe the formation of new memories 'in slow motion' so to speak, but also may give us insights into how the brain creates a sense of seamless unity from a lifetime of diverse sensory experiences.

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