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Face Recognition Impairments [and Discussion]

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Face recognition impairments

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SUMMARY

Face recognition impairments are often found in the context of brain injury involving the right cerebral hemisphere. Recognition impairments can be dissociated from impairments affecting the processing of other types of information carried by the face, such as expression. The face recognition impairments themselves take different forms, corresponding to idealized stages or levels of recognition. These types of error can also arise as transitory phenomena in normal everyday life.

From these observations, psychologists have proposed functional models that characterize the organization of the face processing system in schematic form. Such models provide useful ways of summarizing what is known. More importantly, they also allow new findings to act as tests of each model's usefulness by the extent to which they can be readily accommodated or force revision.

Examples of this are briefly considered, including delusional misidentification, impaired learning of new faces, disordered attention to faces, 'covert' recognition in prosopagnosia, and unawareness of impaired face recognition.

1. INTRODUCTION

Investigations of face recognition impairments caused by brain injury have revealed a number of important features of the human face processing system. One of the first of these to be shown was the involvement of the right cerebral hemisphere, which was suggested in the 1950s, confirmed in several studies reported in the 1960s, and perhaps over-enthusiastically embraced in the 1970s. I mention the possible over-enthusiasm because there was always an undercurrent of reports suggesting that the right hemisphere was not exclusively responsible for face recognition, and thus pointing clearly toward one of the themes of 1980s work, which was the need for a more sophisticated analysis of which aspects of face processing primarily involve the right hemisphere (H. Ellis 1983; Rhodes 1985; Young 1988).

Posing the question in this way focuses attention onto the issue as to how one can fractionate the face processing system into its component parts, and map out their relation to each other. Hence, a number of us began to apply the cognitive neuropsychological approach, by comparing the different types of face recognition impairment observed after brain injury to an explicit functional model intended to acount for both normal and impaired performance, and then using the patterns of breakdown caused by brain injury to modify and refine this model. The approach involves carefully contrasting the effects of brain injury across people who show different patterns of impairment (A. Ellis & Young 1988; Shallice 1988; McCarthy & Warrington 1990). This paper takes stock of what we have learnt.

2. HIGHER-ORDER IMPAIRMENT

First, we must consider the relation of face perception impairments to impairments affecting more 'basic' visual abilities. Table 1 summarizes details concerning three ex-servicemen with unilateral missile wound injuries from the series studied by Newcombe *et al.* (1989*a*). Spatial contrast sensitivity functions have been plotted for each person, and performance on a face perception task (reaction time to decide whether or not a stimulus is a properly organized face). Cases T.C. and B.S. (both with left hemisphere lesions) showed impaired contrast sensitivity without a corresponding impariment of face perception, whereas P.G. (who had a right hemisphere lesion) performed very poorly on the face perception task and yet had unimpaired contrast sensitivity for all spatial frequencies that were tested.

Comparable findings have been reported for cases with impaired face recognition, where contrast sensitivity may show little impairment (Rizzo *et al.* 1986) or impairments that are no greater than for matched controls with poor vision but no problems in face recognition (Young & H. D. Ellis 1989).

The likely reason for these essentially negative findings concerning spatial contrast sensitivity is that higher-order representations that can pool information derived from a range of spatial frequencies are involved in face perception. However, it is not claimed that there is no relation at all between 'basic' and 'higher-order' visual impairments. For example, T.C. (see table 1) had very poor contrast sensitivity for all spatial frequencies and, although his performance of the face perception task was not significantly

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Table 1. *Cases drawn from Newcombe* et al.*'s (1989a) study, showing dissociable impairments of face perception and contrast sensitivity*

reaction times (ms) for face perception task		contrast sensitivity (2-$\log_{10}$ contrast) 00.72 c.p.d.	01.43 c.p.d.	02.88 c.p.d.	05.80 c.p.d.	10.10 c.p.d.	17.40 c.p.d.
T.C.:	1443	158.6**	187.6***	185.5***	159.7***	100.3*	000.0***
B.S.:	854	151.7***	192.1***	217.2*	208.5	120.0*	031.5*
P.G.:	2220***	191.6	255.1	287.6	278.9	225.0	150.6
control subjects ($n=20$):							
mean	1098	189.5	236.0	263.8	238.5	188.5	119.5
s.d.	210	11.3	13.7	20.5	19.4	31.5	33.7

(c.p.d. = cycles per degree; asterisked scores are significantly worse than the control mean: *$p<0.05$, **$p<0.01$, ***$p<0.001$.)

impaired, it did seem to be compromised to some degree. This is as one might expect if basic visual abilities 'feed' higher-order representations. Yet cases like B.S. (table 1), who had a less severe contrast sensitivity impairment and could perform the face perception task with ease, show that this is only a loose linkage.

3. FUNCTIONAL MODELS

Higher-order impairments of face processing can take a number of different forms, which makes it essential to have some way of achieving an economical description of how these different forms of impairment relate to each other. Hay & Young (1982) introduced a simple schematic representation, in the form of a 'functional model', which was further developed by Bruce & Young (1986). This (Bruce & Young 1986) model is shown in figure 1. It claims that recognition proceeds in parallel with expression analysis, lipreading ('facial speech'; see Campbell, this symposium), and directed visual processing. Recognition itself involves sequential stages of perceptual classification (by domain-specific face recognition units), semantic classification (involving domain-independent person identity nodes which can access previously learnt semantic information from the person's face, voice, or name), and name retrieval. This is only meant as an idealised sequence, and would be compatible with a 'cascade' mode of operation.

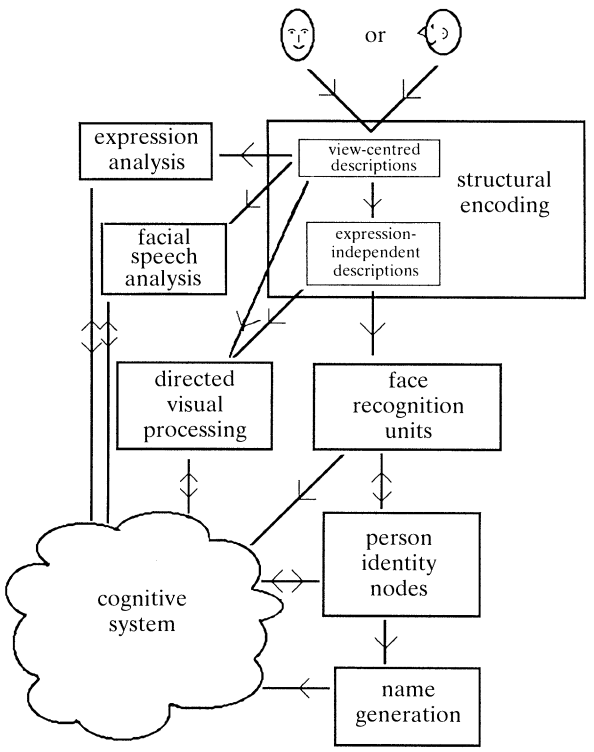

Figure 1. Functional model proposed by Bruce & Young (1986).

A model of this type does not solve the problem of how we recognize faces. But it does have its uses. It provides a convenient way of summarizing what is known. More importantly, it can predict hitherto unreported types of deficit, and allow new findings to act as tests of the model by the extent to which they can be readily accommodated or force revision. It is also possible to check the model's utility by extending its range of application, as we have recently done by developing accounts of delusional misidentification (H. D. Ellis & Young 1990; Young *et al.* 1990*c*).

The most well-known forms of delusional misidentification are the Capgras delusion (Capgras & Reboul-Lachaux 1923), in which close relatives are felt to have been replaced by 'dummies' or impostors; the Frégoli delusion (Courbon & Fail 1927), in which a cunningly disguised persecutor is claimed to be following the patient about; and intermetamorphosis, in which a particular person's appearance seems to transform into that of someone else. Our interest in these conditions was triggered by the observation that each can occur after brain injury, and we thus sought to extend the scope of the Bruce & Young (1986) model to encompass them. H. D. Ellis & Young (1990) pointed out that the Frégoli delusion would involve malfunction of the person identity nodes and cognitive system (see figure 1), whereas intermetamorphosis arises at the face recognition unit level. This approach has potential value in organising clinical observations and suggesting what should be tested.

The Capgras delusion is less easy to relate to the Bruce & Young (1986) model, but can be accommo-

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dated by using Bauer's (1984) suggestion that separable neurological 'routes' mediate overt recognition of familiar faces and reactions to their emotional significance. The basis of the Capgras delusion may thus lie in damage to neuro-anatomical pathways responsible for appropriate emotional reactions to familiar visual stimuli (see also Lewis (1987); Anderson (1988)). Because substantial parts of these pathways are in close proximity to those involved in visual recognition (Bauer 1984), one would expect that few brain lesions will compromise emotional reactions to visual stimuli without also affecting other visual functions involved in recognition to some extent. Hence most Capgras patients will show co-occurrent defective face processing abilities, as has been noted in several studies (reviewed by H. D. Ellis & Young (1990)) and our own observations.

4. RECOGNITION AND OTHER FACE PROCESSING ABILITIES

The central claims of the Bruce & Young (1986) model are the heterarchic relation between face recognition and the processing of other types of information carried by the face (such as expression), and the hierarchic organization of the recognition system itself.

Several reports suggest that recognition impairments can dissociate from other impairments of face processing, with independent impairments affecting the recognition of familiar faces, matching of unfamiliar faces, and processing of facial expressions. However, Young & Bruce (1991) noted that the evidence is not yet completely convincing.

For example, task requirements usually vary between familiar face recognition, unfamiliar face matching, and facial expression tasks. This problem has been corrected in a study by Parry *et al.* (1991), in which all three abilities were tested by means of forced-choice tasks with two alternatives; familiar face recognition by asking which of two faces belonged to a person with a specified occupation, unfamiliar face matching by asking which of two three-quarter view face photographs showed the same person as a full-face target, and expression recognition by asking which of two faces had a specified expression? Fifteen patients with closed head injuries were tested by Parry *et al.* (1991). As a group, their performance was worse than that of controls, and some of the patients showed dissociable impairments affecting one of the tasks only. Three such cases are presented in table 2.

Table 2. *Head injury cases with impairments of familar face recognition, unfamiliar face matching, and expression recognition from Parry* et al.*'s (1991) study*

	familiar face recognition (max = 60)	unfamiliar face matching (max = 60)	expression recognition (max = 60)
A.B.:	53***	57	57
V.S.:	59	51*	56
J.P.:	60	57	53*
control subjects ($n = 15$):			
mean	59.07	57.00	57.27
s.d.	1.28	2.65	1.94

(Asterisked scores are significantly below the control mean: *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.)

The data presented in table 2 show that differences in task demands are not in themselves a sufficient explanation of dissociable impairments. However, the impairments were not severe, and other potential difficulties remain. As Newcombe (1979) pointed out, some patients use idiosyncratic strategies to compensate for their problems, especially with unfamiliar face matching tasks. They can then reach normal levels of performance on accuracy scores by abnormal means, and this needs further investigation.

5. LEVELS OF RECOGNITION

The second important claim made by Bruce & Young (1986) is that there are different types of recognition impairment, which correspond to breakdown at differ-

Table 3. *Recognition of highly familiar people in line-up tasks by P.H. (de Haan* et al. *1987), S.P. (Young* et al. *1990a), K.S. (A. Ellis* et al. *1989), M.E. (de Haan* et al. *1991a) and E.S.T. (Flude* et al. *1989); these cases are chosen to illustrate contrasting patterns of impairment*

	faces line-up			names line-up	
	familiarity (1–7 rating scale)	occupation (max = 20)	name (max = 20	familiarity (1–7 rating scale)	occupation (max = 20)
P.H.:	1.2***	0***	0***	6.0	19
S.P.:	3.1***	8***	5***	6.5	20
K.S.:	4.2***	10***	6***	4.7**	16***
M.E.:	5.7	7***	7***	6.2	8***
E.S.T.:	5.2	17	3***	6.9	19
	Control subjects ($n = 28$):				Control subjects ($n = 12$):
mean	5.98	18.86	16.25	6.27	19.66
s.d.	0.51	1.15	2.81	0.63	0.84

(Asterisked scores are significantly below the control mean: * $p < 0.05$, **$p < 0.01$, ***$p < 0.001$.)

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ent stages or levels of recognition. To investigate this, we have developed 'line-up' tasks in which faces or names are presented one at a time, and subjects are asked to rate familiarity, provide information about the person's identity (such as occupation), and (for faces only) give the person's name. The tasks include faces and names of 20 highly familiar famous people, 20 less familiar people, and 20 people who are unknown to the observer.

Table 3 presents data from the highly familiar faces and names in these line-up tasks for five cases with contrasting patterns of impairment. P.H. (who had occipito-temporal lesions caused by a closed head injury; de Haan *et al.* 1987) and S.P. (subarachnoid haemorrhage from a right middle cerebral artery aneurysm; Young *et al.* 1990*a*) both show the 'prosopagnosic' pattern of impaired recognition of faces with relatively well-preserved recognition of familiar people from their names (Bodamer 1947), although we will see later that there are other differences between them. K.S. (history of epilepsy, and right anterior temporal lobectomy; A. W. Ellis *et al.* 1989) was poor at recognizing people from face or name; her problem is best considered one of person rather than face recognition. M.E. (vasculitic disorder; de Haan *et al.* 1991*a*) showed a different form of person recognition impairment, in which the sense of familiarity was well-preserved (this was confirmed by reaction time studies) but access to semantic information and name retrieval (from the face) were severely compromised. For E.S.T. (surgery to remove a left temporal lobe tumour; Flude *et al.* 1989) only name retrieval seemed to be affected.

The published reports on these cases present other data to substantiate the patterns seen in table 3, but even on the basis of table 3 alone certain points stand out. The faces line-up data are consistent with a hierarchy of impairments corresponding to Bruce & Young's (1986) idea that familiarity, occupation, and name retrieval involve sequential stages, in that order. There are breakdowns at each level, but those at the earlier levels affect later stages: without a sense of the face's familiarity, occupation and name cannot be retrieved (P.H., S.P., K.S.); if the face is familiar but the occupation cannot be retrieved, then it can't be named either (M.E.); and name retrieval impairments can exist when familiarity and occupation are available (E.S.T.). The same patterns of error also arise as transitory phenomena for normal people in everyday life (Young *et al.* 1985).

Also important are patterns of impairment which do not occur. For example, we have never found a brain-injured person for whom name retrieval was normal from seen faces but access to occupations was impaired. Such a case would clearly violate the proposed hierarchy, and therefore be of considerable theoretical importance.

Turning to the names line-up, some patients show the same pattern of impairment as for faces (K.S. and M.E.) whereas others do not (P.H. and S.P.). Hence there is a difference between impairments which primarily affect the recognition of faces, and those which seem to involve person recognition regardless of the input domain. In line with Bruce & Young's (1986) suggestion that it is the earlier stages of recognition that are domain-specific, when there is a problem with recognition from faces and not names all stages of face recognition (familiarity, occupation, and name retrieval) are affected.

Although it is clear that different forms of recognition impairment can occur, I have not yet addressed the issue as to whether these can be specific to face or person recognition?

In most cases, there are other problems. For example, P.H. was poor at recognizing stimuli from other visual categories with many similar exemplars (cars and flowers; de Haan *et al.* 1987; de Haan *et al.* 1992). M.E. showed long-term memory deficits on several tasks, though her other cognitive abilities were well preserved (de Haan *et al.* 1991*a*), and E.S.T. had severe word-finding difficulties (Flude *et al.* 1989). However, it is always risky to infer that one deficit causes the other in cases with co-occurring neurological impairments, and more powerful evidence comes in the form of fractionation of deficits which often co-occur, showing that their association is not inevitable (A. W. Ellis & Young 1988; Shallice 1988; McCarthy & Warrington 1990).

Such fractionations have been reported for face and person recognition impairments. De Renzi (1986) and De Renzi *et al.* (1991) described prosopagnosic patients whose problems did seem to affect faces only, and K.S. showed impaired recognition of familiar people despite good performance of many memory tests (A. W. Ellis *et al.* 1989). Semenza & Zettin (1988, 1989) described cases of impaired name retrieval for proper names only, and McKenna & Warrington (1980) reported a case of impaired ability to retrieve people's names with relatively well-preserved retrieval of other proper names. Thus there is some evidence that these impairments can take face or person-specific forms, although how this should be interpreted remains controversial (Farah 1990, 1991). My own view is that species that depend heavily on social interaction may develop specialized recognition and memory systems to underpin the need to be able to interact differently with different individuals, according to what one knows about them.

6. MODIFICATIONS TO EARLIER MODELS

The findings discussed thus far are mostly readily assimilated to the Bruce & Young (1986) model, but there are also reasons to reconsider it.

Minor modifications have been proposed by Burton *et al.* (1990; see Bruce *et al.*, this symposium), to achieve a workable computer implementation of the Bruce & Young (1986) model. Burton *et al.* (1990) proposed that the feeling of a face's familiarity involves the person identity nodes (see figure 1; the person identity nodes provide a domain-independent means of accessing previously learnt semantic information from the person's face, voice, or name), rather than being based only on the outputs of face recognition units as Bruce & Young (1986) had implied. This leads to the prediction that when a face and a name

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are recognized as 'familiar only', it should still be possible to decide whether or not they belong to the same person if the sense of familiarity is due to activation of a person identity node (Burton *et al.* 1990), whereas this would not be possible if familiarity involves domain-specific face or name recognition units (Bruce & Young 1986). Studies of M.E., who found many faces and names familiar only (see table 3), showed the correctness of Burton *et al.*'s (1990) view that ability to match faces to names would still be preserved (de Haan *et al.* 1991*a*).

Burton & Bruce (1992) have also suggested that there may be no need for a separate process of name retrieval, and that difficulties in accessing names could be due to the fact that names are mostly unique, whereas much semantic information is shared by many individuals. This predicts that patients like E.S.T. (Flude *et al.* 1989) would have trouble with semantic information if tested on knowledge that is unique to the person. This remains to be tested.

Although they have enhanced the precision and explanatory power of the Bruce & Young (1986) model, the modifications proposed by Burton *et al.* (1990) and Burton & Bruce (1992) still leave a need for more wide-ranging theoretical developments, including the introduction of a learning mechanism (see also Bruce *et al.*, this symposium).

Ross (1980) drew attention to brain-injured patients who show poor learning of new visual information but do not suffer from more general memory impairment. E.L.D. (subarachnoid haemorrhage from a right middle cerebral artery aneurysm; Hanley *et al.* 1990) had this problem. She was severely impaired on tests of unfamiliar face memory, and showed poor recognition of faces of people who had become famous since her illness in 1985. Her ability to recognize people who had been familiar to her for some time before her illness was normal, and she could also recognize people who had only been familiar since 1985 from their names and perform normally on tests of verbal memory.

E.L.D.'s spontaneous complaints concerned her inability to learn new faces, and problems in learning her way around in new environments. Further testing showed an impairment of visuo-spatial working memory (Hanley *et al.* 1991), as would be predicted by Baddeley's (1986) model.

The Bruce & Young (1986) and Burton *et al.* (1991) models have little to say about cases like E.L.D., as they do not incorporate an explicit learning mechanism, and this will need to be remedied in the future.

An interesting group of neuropsychological impairments involve distortions or breakdown of awareness (Young & de Haan 1990). Again, the Bruce & Young (1986) model did not give any detailed consideration to this possibility. For example, K.L. (right hemisphere stroke; Young *et al.* 1990*b*) experienced difficulty in recognizing the left sides of faces (i.e. the side falling to his left) but was able to recognize the left sides of objects. Young *et al.* (1990*b*) considered that this reflected impairment of an attentional mechanism for faces, producing a domain-specific form of unilateral neglect.

Just as striking have been findings of 'covert' recognition in prosopagnosia, where there is no global alteration of consciousness, but one specific aspect (awareness of recognition of familiar faces) is lost. Table 4 shows reaction times in an associative priming task for M.S. (encephalitis; Newcombe *et al.* 1989*b*) and P.H. (Young *et al.* 1988), and their accuracies for faces and names in forced-choice familiarity decision tasks (Young & de Haan 1988; Newcombe *et al.* 1989*b*). In the familiarity decision tasks, the familiar face or name had to be selected from two simultaneously presented alternatives (one familiar, one unfamiliar). Both patients showed chance-level performance for faces, reflecting severe impairments of overt recognition. Recognition of names in the equivalent task was much better.

In the associative priming task, target names had to be classified as familiar or unfamiliar, and a face or a name prime was presented before each target name. Three types of prime were used: related (for example, John Lennon's face or name as a prime for the target name 'Paul McCartney'), neutral (an unfamiliar prime followed by a familiar target name), or unrelated (for example, Ronald Reagan as a prime for the target name 'Paul McCartney').

Table 4. *Reaction times (in ms) for correct responses to target names of familiar people preceded by related, neutral, or unrelated face or name primes for M.S. (Newcombe* et al. *1989b) and P.H. (Young* et al. *1988), and their accuracies in forced-choice familiarity decision tasks involving faces and names (Young & de Haan 1988; Newcombe* et al. *1989b)*

	associative priming task reaction times			forced-choice familiarity decision accuracies	
	related	neutral	unrelated	faces	names
M.S.:					
face primes	1260	1276	1264		
name primes	1178	1370	1439		
				67/128	116/128
P.H.:					
face primes	1016	1080	1117		
name primes	945	1032	1048		
				65/128	118/128

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As the data in table 4 show, both M.S. and P.H. showed priming from names (faster reaction times in the related condition), but only P.H. showed priming from faces. Hence P.H. showed associative priming from faces he did not recognize overtly. Lack of overt recognition was confirmed by a separate test, in which P.H. could only identify two out of the 20 familiar faces used, even though they had been presented many times in the experiment. The associative priming effect was tested across all the primes, so the result is not just due to the two faces that could be recognized overtly. Thus in some cases (such as M.S.) there seems to be a genuine breakdown of all levels of recognition, whereas in others (e.g. P.H.) it is only awareness of recognition that is lost.

P.H.'s response latencies were quite long, but slow responding is a common consequence of certain types of brain injury (van Zomeren & Deelman 1978). The pattern of his reaction times across conditions was the same as that found for normal people, with faster reaction times in the related condition regardless of whether face or name primes were employed (Young *et al.* 1988).

Findings of covert recognition have also been made with a number of other procedures (Bauer 1984; de Haan *et al.* 1987; Young & de Haan 1988; Sergent & Poncet 1990; de Haan *et al.* 1992).

There is a parallel between P.H.'s preserved abilities and those aspects of recognition that operate automatically for normal people. We cannot look at a familiar face and decide not to recognize it; the mechanisms responsible for visual recognition are not open to conscious control in this way. It seems that some of these automatic aspects of recognition continue to function in some cases of prosopagnosia, even though the patients themselves do not realise this.

Covert recognition cannot be explained simply by proposing that the effects depend on weak degrees of overt recognition. P.H. performed at chance level in the forced-choice face familiarity decision task (see table 4), whereas weak overt recognition would have produced above-chance performance; a pattern we have reported for another case, N.R. (parieto-temporal lesions due to closed head injury; de Haan *et al.* 1991*b*). A simulation of P.H.'s problems can be made by weakening the connections between face recognition units and person identity nodes (Burton *et al.* 1991).

Another form of breakdown of awareness involves unawareness of impairment (anosognosia). Young *et al.* (1990*a*) investigated S.P., who had severe and stable face processing impairments but showed lack of insight into her face recognition difficulties. S.P. was very poor at recognizing familiar faces (see table 3), yet she maintained that she recognized faces 'as well as before', even when directly confronted with her failure to recognize photographs of familiar faces.

In contrast, S.P. showed adequate insight into other physical and cognitive problems produced by her illness. Her lack of insight into her face recognition impairment involved a deficit-specific anosognosia. Such deficit-specific anosognosias may reflect impairment to mechanisms needed for monitoring performance in everyday life (Young *et al.* 1990*a*).

7. CONCLUSION

Functional models have stimulated research into face recognition impairments. We know a lot more about these disabling conditions than we did ten years ago. Much of the work has shown that the Hay & Young (1982) and Bruce & Young (1986) models were reasonable first approximations, and that they can usefully be extended into new areas (such as H. D. Ellis & Young's (1990) account of delusional misidentification). The development of an implemented version by Burton *et al.* (1990) has also brought enhanced explanatory power. However, empirical work continues to throw up phenomena which challenge these models, including impaired learning of new faces and disorders involving impairments of awareness. These have not yet proved fatal to the enterprise; for example, Burton *et al.* (1991) have produced a simulation of covert recognition. Hence there are grounds for optimism that the interplay between observed patterns of impairment and explicit functional models will continue to enhance our understanding.

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Discussion

D. I. Perrett (*Department of Psychology, University of St Andrews, U.K.*). For any prosopagnosic patient, it must be very strange and worrying to fail to recognize the face of a very close friend and yet to find the same friend's voice, clothes, mannerisms all to be recognizably familiar. In such bizarre circumstances the assumption that the friend is being impersonated by an impostor/alien/actor is one rationalization of lack of familiarity with the face. Rather than speculating that Capgras syndrome represents damage to a specific system attaching emotional feeling to the facial appearance, is it not more parsimonious to interpret the syndrome as a breakdown of face recognition equivalent to that in prosopagnosia but perhaps with the patient having some additional and understandable problem in interpreting the disorder? From this account one would expect Capgras patients to be impaired in standard face recognition tests, just as Professor Young has shown.

A. W. Young. The logic of this suggestion is impeccable, and I am grateful for the opportunity to clarify our views. The face recognition impairments found in Capgras cases are not as severe as those typically reported in prosopagnosia. For example, even though they may be poor at recognizing a proportion of familiar faces, most Capgras patients can still recognize the faces of the 'dummies' who they claim have replaced their relatives. Conversely, prosopagnosic patients do not usually report feelings akin to those involved in the Capgras delusion. Hence we think that the two conditions are not as directly related as Dr Perrett's interpretation implies.

D. I. Perrett. The patients with Capgras syndrome that Professor Young and others have described appear concerned that only one familiar individual is being impersonated. If Capgras reflects damage to an entire system for processing faces (or generating affect in response to faces), then one would predict that patients would suffer delusions about many familiar individuals (perhaps including the patient him or herself). Why do the patients not suffer a general paranoid feeling, as expressed in the theme of 'Invasion of the body snatchers'?

A. W. Young. Quite a lot of Capgras patients do report that more than one person is being impersonated, and the original French literature had emphasized that the number of alleged impersonators tends to increase while the condition persists. I agree that our hypothesis is that when complaints are only made about one impersonator there will be 'peculiar' feelings about several other people, but we suspect that the patients initially only voice concern about those cases where the discrepancy is perceived to be greatest, which will usually be for the closest relatives.

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