

Refining the Explanation of Cotard's Delusion

PHILIP GERRANS

Abstract: Recent work in cognitive neuropsychiatry explains the Capgras and Cotard delusions as alternative explanations of unusual qualitative states caused by damage to an affective component of the face recognition system. The difference between the delusions results from differences in attributional style. Cotard patients typically exhibit a style of internal attribution associated with depression, while Capgras patients exhibit the external attribution style more typical of paranoia. Thus the Cotard patient attributes her condition to drastic changes in herself and the Capgras patient attributes the same changes to alterations in the environment. I suggest three modifications to this explanation. Firstly, the nature of the affective deficit in Cotard cases may be more global than in Capgras cases, resulting from the diffuse effects of the neurochemical substrate of depression. Secondly, this explanation gives us additional insight into the content of the delusion. It is unsurprising that persons whose global affective responses were suppressed would explain their lack of response by saying that they had no bodily existence. Finally I suggest that in Cotard cases the delusion is produced by a reasoning deficit, rather than attributional style.

An elegant theory in cognitive neuropsychiatry explains the Capgras and Cotard delusions as resulting from the same type of anomalous phenomenal experience explained in different ways by different sufferers. 'Although the Capgras and Cotard delusions are phenomenally distinct, we thus think that they represent patients' attempts to make sense of fundamentally similar experiences' (Young and Leafhead, 1996, p. 168). On the theory proposed by Young and Leafhead, the anomalous experience results from damage to an information processing subsystem which associates an affect of 'familiarity' with overt recognition of faces, and, sometimes, scenes and objects. When the normal affect of familiarity is absent the subject experiences an unusual feeling of derealization or depersonalization. The Cotard and Capgras patients adopt different, delusional, explanations of this unusual qualitative state, for reasons to do with 'attributional style'.

It is part of this attribution hypothesis that delusional subjects, like normal people, interpret perceptual phenomena in the light of a set of background beliefs whose structure is a product of social/contextual influences and individual psychological dispositions. That structure predisposes people to reason in certain ways, to discount or reinterpret evidence and to favour certain hypoth-

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Address for Correspondence: Philosophy Department, Victoria University of Wellington, PO Box 600, Wellington, New Zealand.

Email: Philip.Gerrans@vuw.ac.nz.

eses: that is to have an attributional style. Perhaps what we encounter in delusion is extreme cases of attributional style in conjunction with bizarre qualitative states for which there is no precedent in normal experience. The Cotard patient, whose attributional style is introjective, interprets strange sensations of depersonalization or derealization in terms of a change in herself. The Capgras patient, whose attributional style¹ is to seek external explanations, interprets her unsettling experience in terms of a change in the external world.

I suggest that the difference between the Capgras and Cotard delusions cannot be explained solely in terms of different attributional styles applied to essentially the same affective deficit. The Cotard delusion, in its extreme form, is a rationalization of a feeling of disembodiment based on global suppression of affect resulting from extreme depression. In the Capgras case the affective deficit is more localized, confined to familiars. So, in that respect, their aetiologies and qualitative features differ significantly. While it is quite possible that someone whose global affective processes were suppressed would experience the local deficit which generates the Capgras delusion, we would not expect that local affective deficit to generate the Cotard response.

The attribution hypothesis seems closely tied to a view (explained in more detail below) that normal and delusional patients are reasoning in essentially the same ways. I suggest that, in Cotard patients at least, we are looking at seriously distorted reasoning processes which invite a version of the reasoning deficit hypothesis proposed by Langdon and Coltheart (this volume). The Cotard subject seems to have lost a very basic aspect of normal rationality, the ability to recognize oneself as the owner of one's experiences. As Langdon and Coltheart, and Gold and Hohwy (this volume) point out, schizophrenic delusions of thought insertion seem irrational for the same reason. The origin of the deficit in the Cotard delusion results from the dual role played by affective processing. Affective processes on which qualitative experiences depend signal, not only changes in body state, but that the experience is occurring *in one's own body*. Thus, when global affect is absent, flattened or distorted, the delusion of disembodiment, while irrational given one's background knowledge, is a natural way to explain the consequent derealization. The 'naturalness' or 'observational adequacy' of the explanation, however, does not indicate normality of the reasoning which generates it.

1. Routes to Recognition and Cognitive Impenetrability

In order to examine these ideas more closely, consider the explanation of these two delusions by Young and Leafhead. It builds on a theoretically elegant and

¹ Attributional style plays the same role here as the notion of bias in Langdon and Coltheart, this volume, who contrast bias and deficit explanations of delusional reasoning. They use the expression 'attributional bias' to capture a preference for types of explanation within a normally functioning reasoning system. The notion of deficit refers to abnormal function of the reasoning system.

empirically powerful neuropsychological theory of face recognition. According to that theory, developed over the last 15 years, face recognition involves an affective component together with a cognitive component that enables overt recognition to take place (Bauer, 1984; Ellis and Young, 1990; Young et al., 1993, 1994; Ellis et al., 1997).

The independence of the mechanisms responsible for overt recognition and affect is suggested by double dissociation effects following localized neurological damage. Young and collaborators refer to a series of cases in which subjects have overt recognition of faces but no affective response to them, or else intact affective response to faces without overt recognition (a result observed in some, though not all, cases of prosopagnosia). Of course, the mere occurrence of these types of dissociation does not, in itself, necessitate an explanation in terms of independent mechanisms of affect and overt recognition. In part the explanation depends on an overall theory of modular cognitive function into which the explanation of the dissociation phenomenon fits (Fleminger, 1992; Stone and Young, 1997; Shallice, 1988). On that model, dissociation of deficits is taken to indicate the independent (i.e. modularized) functioning of mechanisms whose damage produces the deficit.

The commitment of cognitive neuropsychiatry to the modular explanation of dissociation effects is shown, not just by its explanation of selective damage, but via its endorsement of Pylyshyn's (1984) thesis of cognitive impenetrability. That thesis states that the functioning of a modularized subsystem cannot be affected by the subject's beliefs. Although there is evidence of cross-talk, recurrence, feedback and feedforward interaction and top-down processing between and within modules, modules cannot be affected by the subject's acquisition or loss of beliefs (Coltheart, 1999). Modules deliver inputs to the belief system, their function being unaffected by its operations. Famously, the belief system is, in Fodor's words, a domain-general Central Processor whose functioning is 'Quinean and isotropic'. The CPU is holistic (domain-general) in its operations, whereas the modules which feed information to it are restricted in their operations to specific domains.

Capgras delusion arises in cases where the affective component of the face processing module is damaged, leaving overt recognition unimpaired. The subject sees someone who appears, in all respects, identical to the familiar person but the subject does not experience the normal affective response. Thus she has an uncanny phenomenal sensation based on the absence of an affect whose normal production is automatic and instantaneous. The Capgras delusion is an attempt to explain that uncanny feeling. The subject adopts the hypothesis that the familiar has been replaced by an identical replica, an inference which would explain why everything looks the same but 'feels' strange.

They [delusional subjects] receive a veridical image of the person they are looking at, which stimulates all the appropriate overt semantic data held about that person but they lack another, possibly confirming, set

of information which, as Lewis (1987) and Bauer (1986) have suggested, may carry some sort of affective tone. When patients find themselves in such a conflict (that is receiving some sort of information which indicates that the face in front of them belongs to X, but not receiving confirmation of this) they may adopt some sort of rationalisation strategy in which the individual before them is deemed to be an imposter, a dummy, a robot, or whatever extant technology may suggest (Ellis and Young, 1990, p. 244).

The operation of the affective component of the face processing subsystem, whether intact or impaired, is not cognitively penetrable by the rest of the agent's beliefs. In the Capgras delusion the subject believes that familiar persons have been replaced by identical replicas. The Capgras subject neither revises her other beliefs to make the new belief consistent with the others she already holds, nor rejects this inconsistent belief. Typically, Capgras delusion is associated with localized paranoia, but, once again, the paranoid beliefs do not prompt the type of systematic belief revision required by holistic rationality. As an example of this kind of inferential isolation, Stone and Young (1997) quote this record made by Alexander of a conversation with a Capgras patient:

- E: Isn't that [two families] unusual?
 S: It was unbelievable!
 E: How do you account for it?
 S: I don't know. I try to understand it myself, and it was virtually impossible.
 S: What if I told you I don't believe it?
 E: That's perfectly understandable. In fact, when I tell the story, I feel that I'm concocting a story . . . It's not quite right. Something is wrong.
 E: If someone told you the story, what would you think?
 S: I would find it extremely hard to believe. I should be defending myself.

(Alexander et al., 1979, p. 335)

Rationality is a normative constraint of consistency and coherence on the formation of a set of beliefs and thus is *prima facie* violated in two ways by the delusional subject. Firstly she accepts a belief which is incoherent with the rest of her beliefs, and secondly she refuses to modify that belief in the face of fairly conclusive counter-evidence and a set of background beliefs which contradict the delusional belief. Why not then explain delusion as a failure of rationality, of the system which adopts and maintains beliefs on the basis of their coherence with the rest of the agent's belief set? The answer in the case of Capgras, and other delusions of misidentification, is that the patient's irrationality, despite local paranoia, is confined to a single domain: the response

to familiars. In the rest of her life and cognitive activities, the patient's reasoning ability is undisturbed. Hence the idea that the delusion is best explained in terms of localized discrepant input to the reasoning system, not the malfunction of the reasoning system itself. Maher (1974, p. 103) summarizes the approach of cognitive neuropsychiatry thus:

Strange events, felt to be significant, demand explanation . . . In brief, then, a delusion is an hypothesis designed to explain unusual perceptual phenomena and developed through the operation of *normal cognitive processes*.

2. Are Delusional Subjects Reasoning Normally?

Note here that this way of describing things does not attend to an important distinction between forming a delusional belief ('belief formation') and failing to revise it once formed ('belief maintenance'). The account of the Capgras delusion just considered accords with the idea that the delusion is largely to be explained as the result of normal processes of belief formation. Stone and Young (1997) discuss the notion of an observationally adequate belief, one fixed on the basis of perception rather than extensive further theorizing. The Capgras patient has a veridical perceptual image and a 'feeling' of unfamiliarity. The replication hypothesis preserves the veridicality of both aspects of her experience. In that sense it is like an observationally adequate belief, one whose content is normally fixed on the assumption of veridical experience.

The notion of observational adequacy thus preserves the normality of belief formation. It does seem, though, that observational adequacy cannot explain the failure to revise a delusional belief. However, one way to resist this conclusion is to appeal to the notion of modularity. In cases of visual illusion, although the subject believes that her percept is non-veridical, she cannot alter it. Similarly, in the case of the Capgras delusion, the uncanny experience which generates the delusion, because it is the output of a modularized affective subsystem, is cognitively impenetrable. The patient therefore explains her intractable experience by generating a delusional observational belief consistent with that experience. Exactly which belief is generated depends on the patient's cognitive resources. In cultures like ours it is unsurprising that the beliefs would advert to robots or dummies, in other cultures perhaps one might expect hypotheses of possession or 'soul migration':

the content of the patient's perceptions and his thought will be determined by the psychology of the patient and his circumstances, but the original offending misperception appears likely to be triggered by an organic process (Spier, 1992, p. 283).

The idea that the exact content of the belief (i.e. which observationally adequate hypothesis is chosen) depends on psychological and sociological factors is the 'attribution hypothesis' and once again it preserves the normality of the cognitive function of the delusional subject. Normal reasoners also bring to bear background hypotheses which are a result of their own idiosyncratic personal and environmental histories. Everyone has some bias in their reasoning system which leads them to prefer some hypotheses, discount others and reinterpret evidence and, perhaps, the Capgras patient is merely bringing to bear her reasoning bias on a deeply disturbing and intractable phenomenal state. The attribution hypothesis together with the notion of observationally adequate belief and the impenetrability of the damaged system combine to give the most plausible possible explanation of the Capgras delusion within the framework established by Maher.

Within that framework, given that the deficit in the Cotard and Capgras cases is described by Young and Leafhead as 'essentially similar', the difference in the content of the delusions must be accounted for in terms of differences in the background psychology and circumstances of the patient, and this, following Cotard himself, is the explanation given by Young and Leafhead. The classic Cotard subject forms the belief that she is dead, a belief which *prima facie* is even more difficult to incorporate than the replication hypothesis of the Capgras patient. The crucial difference is that the Cotard patient is typically extremely (often psychotically) depressed (Berrios and Luque, 1995a). Noting this, Young and Leafhead pursue a theory (Beck, 1989; Candido and Romney, 1990) about attributional styles in depressive patients to the following conclusion:

while depressed patients tend to attribute negative events to internal causes, people with persecutory delusions tend to attribute them to external causes . . . the persecutory delusions and suspiciousness often noted in Capgras cases may therefore contribute to the patients mistaking a change in themselves for a change in others ('they must be imposters'), whereas people who are depressed exaggerate the effect of a similar change whilst correctly attributing it to themselves ('I must be dead') (Young and Leafhead, 1996, p. 167).

3. Cotard Delusion: Attribution or Deficit?

The involvement of depression in the Cotard delusion is extremely significant, so let us distinguish three possible ways of linking depression to the delusion:

- (1) Attribution Hypothesis. A person with a depressive tendency and associated attributional style suffers selective damage to a modularized

affect-of-familiarity-system, the experiential effects of which she then rationalizes in the way described above.

- (2) The localized damage and consequent loss of localized affect *cause* the depression and its associated tendency to internal attribution. After all, it is not inconceivable that someone who recognized her family members but no longer felt any of the familiar emotions in their presence would become depressed.
- (3) The neurochemical substrate of psychotic depression causes generalized loss of affect, including the local affect of familiarity. Here there are two possibilities:
 - (a) The global affective deficit is then rationalized using normal attributional style.
 - (b) Deficit Hypothesis. The global affective deficit is rationalized using reasoning processes which are abnormal. An intuitively plausible consideration in favour of this hypothesis over the Attribution hypothesis is given by Langdon and Coltheart (this volume, p. 202): 'If the formation of a delusional belief were just a question of bias, then surely the weight of counter-arguments would eventually tip the scales in favour of rationality'. They conclude that delusions maintained in the face of mounting counter-evidence must involve reasoning deficits as well as perceptual malfunction and attributional biases.

I shall try and show that, for Cotard cases, the deficit hypothesis (3b) is the best candidate. The abnormality or reasoning deficit is the subject's failure to acknowledge at the level of rationality that her experiences are her own. This is seen most clearly in the extreme cases described by Enoch and Trethowan (1991), but less extreme cases fall on a continuum from death to non-existence in which the patient seeks to explain her experience without implicating herself in it (Berrios and Luque, 1995b). Both Langdon and Coltheart, and Gold and Hohwy (this volume) discuss this 'ownership' issue in the context of schizophrenia. Langdon and Coltheart, for example, discuss the possibility that the failure in the schizophrenic case is the result of failure of a metarepresentational capacity, whereas I am claiming that in the Cotard case failure of affective processing is the causal origin of the deficit. However, in both cases, the failure to implicate oneself in one's experiences, evidenced by the nature of the delusions (thought insertion in schizophrenia, of bodily inexistence in Cotard), is evidence of a reasoning deficit, rather than a matter of attributional style.

Recall the important difference between the Cotard and Capgras cases. In the Capgras cases the paranoia is presented as a rationalization of localized feelings of unfamiliarity, depersonalization or derealization in relation to a particular person, place or object. The patient is otherwise unaffected and one is left with the impression that disruption of all affective responses has not

occurred. In the Cotard case, however, it seems likely that normal affective links to a wide range of perceptual and cognitive experiences have been suppressed. Furthermore, there are good reasons to think that the mechanisms of suppression may be the neurochemical mechanisms involved in depression, which have a global effect, not only on subtle affective responses like familiarity, but on more florid ones like elation and even, on some theories, on cognitive processes (Panskepp, 1982). In such a case, the lack of emotional responses, feelings of emptiness and derealization would be the result of global affective suppression or damage rather than of localized organic damage interpreted within a depressive mindset.

That affect can be suppressed globally as well as locally by neurochemical processes associated with depression is a well supported hypothesis. Mood states induced in this way have complex feedback relations with both cognitive and affective processing because they are

neurochemical states which act to modify the activity of broad areas of the central nervous system . . . [The] effect [of such a state] may be diffuse, affecting the whole system, or specific, affecting the various emotional responses differently. In addition to its effects on the affect program system in the limbic brain, the same chemical condition may affect higher areas of the brain and thus affect emotional phenomena involving higher cognition and higher cognitive phenomena generally (Griffiths, 1997, p. 256).

My suggestion is that in some Cotard cases the depression causes a global affective deficit with the type of diffuse effects cited by Griffiths. Our phenomenal experiences, whether subtle, persistent and global in the case of moods, or florid, instantaneous and localized as in fright, are instances of conscious awareness of processes, themselves inscrutable to introspection, which regulate body state. Depression is a global suppression of affect, which because of its complex interdependence on levels of chemicals like serotonin and noripinephrine is something which can occur in degrees. The point I wish to emphasize is that, at the limit, as in the Cotard cases, there is good reason to think that it would be experienced as disembodiment, because its physiological basis is global suppression of all mechanisms by which we achieve phenomenal awareness of our body state.

I note here that, in the course of a discussion of the role of affect, Ramachandran reaches a similar conclusion about the essential link between the global nature of affective suppression and the content of the delusion:

In Cotard's all the sensory areas are disconnected from the limbic system . . . I would predict that Cotard's syndrome patients will have a complete loss of GSR for all external stimuli—not just faces (Ramachandran and Blakeslee, 1998, p. 167).

Supporting evidence comes from those cases of extreme depression, accompanied by delusion, which can, apparently, occur without a history of the type of organic accident cited by Young and Leafhead. Cases of the Cotard delusion have been reported (Enoch and Trethowan, 1991) in which the subject proceeds beyond reporting her rotting flesh or her death to the stage of describing the world as an inert cosmos whose processes she merely registers without using the first-person pronoun.² In this type of case the patient conceives of herself as nothing more than a locus, not of experience—because, due to the complete suppression of affect, her perceptions and cognitions are not annexed to her body—but of the registration of the passage of events. She has effectively effaced herself from the universe: nothing which occurs is of any significance to her and, hence, she describes the world without implicating herself in that description. Few such cases are reported nowadays because that level of depression is recognized as preceding suicide and attracts pharmacological intervention, or, in extreme cases, ECT, at an earlier stage. However, their occurrence does strongly suggest that, in the absence of affective processing, perception and cognition have no bodily consequences and thus are not ‘felt’ at the phenomenal level to belong to the agent.³ The patient does not recognize experiences as significant for her because, due to the global suppression of affect, she has no qualitative responses to the acquisition of even the most significant information. These extreme cases of the Cotard delusion are those in which neural systems on which affect depends are suppressed and, as a consequence, it seems to the patient as if her experiences do not belong to her. Thus the patient reports, not changes in herself, but changes in the states of the universe, one component of which is her body, now thought of as another inert physical substance first decomposing and finally disappearing.⁴

If this is the case, then perhaps the Capgras and Cotard delusion may result from affective damage with quite different aetiologies. Of course this possibility does not invalidate the basic picture of normal and abnormal recognition. In both cases the patient generates a delusional belief to explain feelings of unreality or abnormality which arise following disruption to subpersonal mechanisms of affect. In the Capgras case, the absence of affect is confined to quite specific eliciting situations. In cases associated with psychotic depression, the suppression of affect is general and it is thus unsurprising that the Cotard

² Although it should be noted that in such cases organic damage of the type referred to by Young and Leafhead cannot be ruled out. The Enoch and Trethowan cases predate PET investigation for example.

³ My claim here is not that the mechanisms involved in bodily monitoring no longer function. The patient can still detect perceptual stimuli and the movements of her limbs, but the those mechanisms of detection no longer give rise to their normal consciously experienced phenomenal correlates.

⁴ Although note once again that these cases precede the ability to detect neurological damage by PET scan. Enoch and Trethowan give psychodynamic explanations which, as Stone and Young, 1997, point out, have been superseded.

delusion would be experienced initially as disembodiment and finally as absence from the physical world.

Exactly how different are the Capgras and Cotard delusions? Young (this volume) rightly warns of the danger that a dissociation effect (some people have the Capgras but not the Cotard and others have the Cotard but not the Capgras delusions) might lead us to assume that the Capgras and Cotard delusions are independent, conceptually and neurologically. Young reminds us of the cases of association, such as that described by himself and Leafhead. But although he is prepared to associate the delusions in some cases, Young would not go along with Ramachandran who describes the Cotard delusion as 'simply an exaggerated form of Capgras' syndrome and probably has a similar origin' (Ramachandran and Blakeslee, 1998, p. 167). If this is taken to mean that the loss of affect is global in the Cotard and local in the Capgras, then it is true. However, as stated, it implies that there are no significant differences in causal origin or reasoning strategy between the Capgras and Cotard patients.

I distinguished possible aetiologies of affective suppression and claimed that in the Cotard case there is an essential link between that aetiology and the content of the delusion. I also suggest that the neurochemical substrate of psychotic depression introduces more than normal bias into the subject's reasoning, it creates a deficit. It is part of normal rationality to self impute one's experiences, and the Cotard subject can no longer do this. Thus I incline toward the deficit hypothesis as an explanation of the Cotard delusion.

This endorsement of the deficit hypothesis requires elaboration to explain a type of case discussed by Young in support of the attribution hypothesis. He and his collaborators have reported a case of alternation between these delusions, following a neurological accident, which appeared to coincide with switches in attributional style (Young et al., 1993). As Young (this volume, p. 65) explains it:

the key factor seems to be the patients' moods—when in a suspicious mood, they think that other people are impostors, when depressed they think they are dead. The underlying basis of both delusions could therefore be quite similar; a delusional interpretation of altered perception (especially loss of affective familiarity).

Can the deficit hypothesis accommodate these types of case? The best explanation appeals to the distinction between forming and revising a belief. I said earlier that the attribution hypothesis accords best with the idea that the delusional and the normal subject are not reasoning differently. This may well be the case in the formation of the belief: a discrepant experience is rationalized according to attributional style, which may fluctuate.

However in psychotic depression the mood is not transient but deeply entrenched. And, characteristically, depressive hypotheses are quite immune to revision. Once depression has set in it functions to maintain the delusional

hypothesis in the face of pressure from the rest of the agent's beliefs and accumulating counter-evidence. My suggestion is that the maintenance of a belief like the Cotard delusion requires a reasoning deficit. And the effect of psychotic depression is to introduce that rigidity by making it impossible for the agent to feel as if her experience is her own.

This is not inconsistent with the alternation between the delusions in cases where the depression waxes and wanes. In those cases, if the depression is a matter of relatively transient mood it could account for fluctuation between attributional styles which suggest alternative observationally adequate hypotheses of death or replication. However, where depression is deeply entrenched and psychotic it produces a deficit of reasoning which makes it impossible for the subject to bring countervailing knowledge to bear. Thus I suggest that the deficit hypothesis as it applies to the Cotard case is best thought of as a deficiency in belief revision rather than formation.

*Department of Philosophy
Victoria University of Wellington*

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