Chapter 2, “Wondrous Strange: The Neuropsychology of Abnormal Beliefs”

1. * Delusions are bizarre and not properly sensitive to evidence. Among other things, the monothematic nature of many delusions challenges the holism of belief. They offer real world versions of what would otherwise just be philosophical thought experiments that test theories of belief.

   * Young offers an account of the Capgras delusion that shows it to be an attempt to make sense of abnormal perceptual experiences. Data considered include subjective reports, brain injuries, and cognitive functioning. Young terms this approach *cognitive neuropsychiatry*.

2. * Classic examples of delusions include anosognosias, and Anton’s syndrome in particular. Young also describes several delusions that resulted from brain injury. Not all of these delusions fall under recognized types. These delusions can be quite circumscribed, and the subject can be aware of their absurdity.

3. * Sometimes the Capgras delusion leads to serious and even tragic action – see the chilling examples discussed on pp. 52-53. But most do not act on their delusions in a serious way. In fact, many accept the substitute and do not even inquire about the original.

Q: What do they believe when there is a significant mismatch between avowal and expected action?

4. * Psychiatrists posit psychiatric syndromes defined by co-occurring symptoms. The hope is that this will lead to the discovery of the relevant mental illnesses. Young prefers a more focused approach on symptoms.

5. * Delusional beliefs tend to be caused by damage to the right cerebral hemisphere. This suggests that the delusions aren’t to be accounted for purely in terms of emotions and psychodynamics, say. Organic factors will likely play a role as well.

6. 
Capgras patients typically cannot explain what is visually different about their loved one. They also perform worse than controls at face recognition. Young proposed that the Capgras delusion might be the reverse of prosopagnosia. Capgras patients might recognize the familiar face, but perhaps they do not experience the right affective (autonomic) response. The delusion then offers an explanation for this visual recognition without the feeling or emotion of familiarity. Later studies of skin conductance responses confirmed this hypothesis. The delusion also faded in some cases when the relatives were spoken to without being seen.

7.
* But why do they leap to this absurd conclusion? Well, many probably do not leap to this conclusion, but they are not as likely to be studied. There is some evidence of reasoning biases in these subjects, whereby they believe on scant evidence. Also, there is a tendency to attribute negative outcomes to external causes (attributional bias).

In some cases the anomalous experiences might play a larger role; in other cases the reasoning biases might. This account could also be extended to Cotard’s delusion, with an accompanying difference in attributional biases.

Chapter 3, “Towards an Understanding of Delusions of Misidentification: Four Case Studies”

**Delusions of misidentification:** cases in which a person has a mistaken, delusional belief “in the identity of oneself, other people, places or objects.” (75) This belief could involve doubles or impostors, for example.

--Note the discussion in footnote 1, and from the Young reading, about syndrome vs. symptom approaches.

* One interesting study about testing to see if the Capgras delusion persists across sense modalities:

  “Hirstein and Ramachandran (1997) demonstrated further that DS’s Capgras delusion was specific to the visual modality: he claimed his parents were impostors when looking at them, but not when speaking to them on the telephone.” (78)

* Interesting point on the rationality and awareness of Capgras patients:

  “Young (1998) reports that ‘if you ask (the Capgras patient) “what would you think if I told you my wife had been replaced by an impostor”, you will often get answers to the effect that it would be unbelievable, absurd, an indication that you had gone mad. Yet these patients will claim that, none the less, this is exactly what has happened to their own relative.’”
Capgras patients can be plotted on a continuum from “identified neurological lesion” to “no identified neurological lesion”. (82)

4 cases studies will be examined at 3 different levels: the neurological, cognitive, and phenomenological.

Mirrored-self misidentification:
--FE: Treated his own reflection as if it were another person.
“FE’s family tried on numerous occasions to dissuade him from his belief by providing him with evidence contrary to the delusion. FE would listen attentively to their arguments and often agreed with their logic, but his delusional belief remained steadfast.” (83)
Importantly, FE recognized Nora’s reflection as such.

--TH: He too treated his own reflection as if it were another person.
TH seems a bit more confused, or uncertain, about the status of the other person/reflection stating things like:
“I see my face in there, a reflection of it.” (88)
“He’d have to be [bald] wouldn’t he, or he wouldn’t, it wouldn’t…” (88)
“No. He’s only wearing a reflection of the clothes that I am wearing.” (90)
“I can see your [examiner’s] reflection and I can see Tom’s reflection.” (90)
And: “When asked where this person and his wife lived, TH said that they lived in an adjoining unit located at the back of TH’s home (there was no unit adjoining TH’s home).” (90)
Also, note TH’s “mirror agnosia”. (91-92)

Reduplicative Paramnesia
--DB: “…she clearly stated that her husband had died suddenly four years earlier and had been cremated (this was correct), but also that her husband was a patient on the ward in the same hospital.” (92)
“I’m not religious fortunately or I might be worried about it.” (93)

Reverse Intermetamorphosis
--RZ: 40 year old woman who believed she was her father (or grandfather).

From the neurological point of view these case differ greatly. DB’s DM was clearly the result of a stroke, FE and TH had a more gradual onset, and RZ had no evident neurological abnormalities (and her delusions came on at a much earlier age).

From the cognitive level: TH, DB, and FE all had problems processing sensory information. These same three patients also had a general tendency toward heightened affective responses. All DM patients also seem to fail to give proper weight to evidence against the deluded belief.

And from the phenomenological level: FE, TH, and DB each held to their delusional belief with great conviction:
“Their replies were immediate, and they never attempted to convince the interviewer of the validity of their delusional belief. If a question were put to them that they could not explain, they simply admitted it. For example, FE and TH both agreed that it was strange that the other person was in their own homes but neither man felt any obligation to provide an explanation for it.” (105)

“Neither man [FE or TH] developed a wide web of delusional beliefs in which their delusional misidentification became embedded. In both cases the delusional beliefs remained isolated from a more rational belief system which was operative in other cognitive domains” (105)

RZ shifted the content of her delusional belief, and sometimes reverted to a confused state when pressed (e.g. “I’m just confused because I don’t remember. It feels like they’ve taken my brain out and I don’t even have a brain.” (97)). The other three remained steadfast in their delusion.

None of the patients exhibited paranoia/suspiciousness or depersonalization/derealization issues.

Clearly, RZ is quite different from the other 3 cases.

Chapter 4, “Refining the Explanation of Cotard’s Delusion”

* Recall the Young hypothesis that Capgras and Cotard patients share an affective disorder, but differ in attributional style (e.g., external-biased vs. internal-biased).

--Gerrans rejects this picture:
“While is [sic] 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.” (112)
Instead, he argues that Cotard patients have a more serious reasoning problem – a reasoning deficit.
“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.” (112)

* Gerrans identifies the following 2 failures of rationality in the deluded:

“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.” (114)

* Distinguish: forming a delusion belief from maintaining a delusional belief.

--Perhaps a delusional belief is maintained because of the modularity, and cognitive impenetrability, of the mechanisms generating such beliefs. (115)

* Note the three different roles that depression might play in the Cotard delusion. Gerrans endorses 3b. (116-117)

--Importantly, Gerrans insists that a reasoning deficit must be present to explain the maintenance of this delusion:

“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.” (117)

--The depression of the Cotard patient is a more global affective disorder than the affective disorder that appears with Capgras.

“Depression is a global suppression of affect, which because of its complex interdependence on levels of chemicals like serotonin and norpinephrine 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 mechanism by which we achieve phenomenal awareness of our body state.” (118)