CHAPTER 20

DELUSIONAL EXPERIENCE

JENNIFER MUNDALE
AND SHAUN GALLAGHER

One of the central debates concerning delusion has to do with the role played by faulty hypothesis formation and other cognitive errors. Top-down models assign causal power to such errors in generating delusions, whereas bottom-up models regard these same cognitive errors as at least secondary to (and in some cases derivative from) the delusional experience. We support a bottom-up model of delusion, one that holds that delusional experiences are immediate and noninferential. With respect to the noninferential character of delusion, our approach is similar to that espoused by Gold and Hohwy (2000) in which delusions are referred to as “disorders of experience.” At the same time, however, we also acknowledge the explanatory appeal of top-down models of delusion, in which delusions are thought to derive from predictable, cognitive errors. Rather than accept that delusions are the result of higher-order cognitive mistakes, however, we argue that the kinds of errors to which such top-down models typically appeal may themselves be understood, in certain crucial respects, in a bottom-up way or as part of the immediate experience of the delusional subject. This view is supported by Kapur’s (2003) work in which schizophrenic delusion is understood in terms of aberrant salience, which in turn is explained at the neurological level as a disorder of the dopaminergic system. Thus, our model of delusion provides an integrated approach in which aberrations at the neurological level are directly related to a “disorder of experience,” at the phenomenological level, without recourse to mistaken inferences at the cognitive level. Though we acknowledge that delusions are, of course, associated with higher order cognitive effects, we argue that these are not the proper locus for the explanation of the delusional experience itself.
To further contrast bottom-up versus top-down views of delusion, a typical example of the top-down approach is the two-factor or two-stage model of delusion. In a two-stage model, delusions are seen as an erroneous, cognitive attempt to explain an anomalous experience. The first stage consists of the anomalous experience, such as a hallucination, and the second stage consists of the cognitive or reasoning error that, in attempting to explain the anomalous experience, generates the delusion.

Ramachandran’s (1998) explanation of Capgras’s delusion illustrates this top-down, two-stage approach. In his account, the first stage is the anomalous perceptual experience of seeing one’s parents or other loved ones without simultaneously experiencing an emotional response to them. This sets the stage for the second factor, which is the attempt to rationalize this experience by regarding the loved one as an imposter. The second factor generates the delusional component, and it is either immune or very resistant to revision.

Not all two-stage models consist purely of neurological and cognitive deficits. McKay, Langdon, and Coltheart (in press) have recently argued for including motivational factors in the two-stage model, either as stage 1 or stage 2 factors, noting that “desires are powerful doxastic forces.” Though we disagree with certain features of the two-stage model, we support the view that desires, appetites, wishes, and other motivational factors have something important to contribute to our understanding of delusion, perhaps especially in understanding the theme and maintenance of a delusion.

Alternatively, the key feature of bottom-up theories, sometimes referred to as empiricist theories, is the denial of the claim that delusions are cognitively derived, second-order processes. Gold and Hohwy (2000) have recently offered an especially innovative example of a bottom-up model, one that provides a useful starting point for the discussion of our own account. One of the central features of Gold and Hohwy’s model has to do with what they call “experiential irrationality,” something they regard as “a new department of rationality.” In their view, this new category of rationality is needed because neither the standard categories of content rationality nor procedural rationality adequately capture the irrational nature of schizophrenic delusion. As they explain:

We claim…that the source of thought insertion and related delusions is the experience itself of the schizophrenic subject, and, in particular, its alien quality. The elaboration of the delusion in hypotheses and ancillary beliefs should be understood to be derivative from, or secondary to, this experience. Thus the violation of egocentricity does not merely produce strange experiences that form the basis of delusional beliefs as the result of pathological processes of thought or reasoning. Rather, it is the experience of non-egocentric thought as alien that is the delusion itself. The alien quality of the delusional experience is part of its content, and it is the content of experience that is the locus of the delusion and thus of the irrationality. At least some delusions, therefore, are best explained as disorders of experience rather than disorders of belief, desire, or reasoning. (Gold and Hohwy, 2000, p. 160)

“Experiential irrationality” itself is an intriguing concept, as it suggests the non-derivative immediacy of at least some forms of delusion, an approach we find not
only empirically convincing but intuitively appealing as well. As Gold and Hohwy further note, the subject is not led into a deluded hypothesis, his experience is itself delusional. Subjects experience certain thoughts as intrinsically alien. In contrast with top-down models, they insist that “A hallucination is an unusual form of experience, but no subsequent judgment is required on the part of the subject for the experience to become delusional” (2000, p. 162). This insistence on the first-order immediacy of the delusion is one of the key features of the bottom-up model we advocate and to which we now turn.

On this bottom-up model, we can distinguish between (1) first-order experience, that is, the phenomenological level of immediate, prereflective, lived-through experience of the world; and (2) higher order cognition, a reflective experience that supports the ability to make attributive judgments about one’s own first-order experience. Both first-order experience and second-order cognition depend on a third level of the cognitive system, (3) the nonconscious, subpersonal processes that are best described as neuronal or brain processes. As Gallagher has previously argued (2000a, 2004, 2007), schizophrenic delusions, such as thought insertion, alien control, and other misattributions of agency, are experienced by the subject at the first-order phenomenological level. They are immediate, noninferential, and nonintrospective. This is especially clear starting with prodromal symptoms, that is, early symptoms that precede the characteristic manifestations of the fully developed illness. Though the higher order, cognitive report of the delusional experience may be confused, it is not necessarily mistaken; the subject is merely recounting what he or she is experiencing at the phenomenological level.

Furthermore, the problems that manifest themselves at the level of first-order experience are not explained by misguided beliefs, dysfunctional introspective processes, or pathological self-referential narratives, as top-down models would suggest (e.g., Graham and Stephens, 1994; Hoffman, 1986), rather, they are the result of dysfunctions at the neuronal level. For example, in schizophrenia, neurological problems may cause the tacit sensory-motor processes that are normally implicit in first-order phenomenal experience to become abnormally explicit (Sass, 1998, 2000; Sass and Parnas, 2003). Accordingly, what is normally the tacit integration of cognitive, emotional, and motivational factors is disrupted at the level of first-order experience; the implicit unity of the self breaks down; and one begins to feel alienated from one’s thoughts and actions.

On this kind of account, for example, problems with self-agency that manifest themselves in the first-order phenomenology of thought insertion and delusions of control are generated on a neurological level. Farrer and Frith (2002; Farrer et al., 2003), for example, have shown contrasting activation in the right inferior parietal cortex for perception of action caused by others, and in the anterior insula bilaterally when action is experienced as caused by oneself. The role of the anterior insula in providing a sense of self-agency involves the integration of three kinds of signals generated in self-movement: somatosensory signals (sensory feedback from bodily movement, e.g., proprioception), visual and auditory signals, and corollary discharge associated with motor commands that control movement. They suggest that
a “close correspondence between all these signals helps to give us a sense of agency” (Farrer and Frith, 2002, 602). That is, a disruption in one or more of these signals, a disruption in their integration, or some other kind of malfunction in the anterior insula or the right inferior parietal cortex may generate a loss of the sense of self-agency or a sense of alien control at the level of first-order experience. Indeed, in schizophrenic patients, the feeling of alien control (delusions of control) during a movement task has been associated with an increased activity in the right inferior parietal lobe (Spence et al., 1997). There may be a more general or basic disruption of neuronal processes that affect not just the sense of agency for motor action but also disrupt the sense of agency for cognitive processes, resulting in symptoms of thought insertion. The sense of agency for thought may depend on the anticipatory aspect of working memory (Gallagher, 2000b, 2004), something that may also malfunction in schizophrenic subjects with delusions of control (see Daprati et al., 1997; Franck et al., 2001; Singh et al., 1992; Vogeley, Kurthen, Falkai, and Maier, 1999).

The delusional experiences (of control and thought insertion) may then motivate second-order introspective processes, which may be playing a defensive role in an attempt to explain or justify the alien experience. In this way, higher order processes are reiterating and perhaps enhancing problems first manifested at the experiential level. Thus, a key point of contrast between bottom-up and top-down models concerns the location of causal power. According to the top-down approach, the misattribution of agency to someone or something else is caused by a cognitive error, which becomes the explanans rather than the explanandum (Gallagher, 2007). According to the bottom-up approach, the primary cause of delusional experience is to be found in the brain pathology; at best, whatever higher order cognition does is secondary to the alien experience.

Although we deny that the delusional experience is primarily the product of faulty reasoning at the cognitive level, we also acknowledge the heuristic appeal of various mental errors commonly associated with delusion. Unlike top-down models, however, we do not locate the primary influence of these errors at the cognitive level (as far as the delusional experience is concerned). Thus, one can retain the explanatory value of certain kinds of mental errors within a bottom-up model of explanation. More specifically, we claim that there are very basic kinds of mental errors that, though often referred to as cognitive errors, are so deeply entrenched in us and so automatic that they are an integral part of our immediate, phenomenological experience. Well-known examples of such errors may include salience effects, attribution errors, primacy and recency effects, contrast effects, and various other biases and distortions. Salience effects pertain to causal attribution: observers consistently attribute greater causal influence to those objects (and persons) in their immediate environment that are most salient, not necessarily the most causally influential (Taylor and Fisk, 1975). Attribution errors come in many forms, but the most common—the so-called “fundamental error of attribution”—consists of overattributing the cause of others’ behavior to internal, dispositional factors, such as character, abilities, and motivations, and underattributing their behavior to external, situational factors (Ross, 1977). More is said below about salience, attribution,
and the relation between them. Such biases are commonly regarded as having a top-down influence and to be operative primarily at the cognitive level. Although no one is denying that these can have higher order, cognitive influences, biases such as these also exert a bottom-up or background influence on first-order experience. In other words, their influence can be precognitive and noninferential. If this is so, then it would allow for some of the appeal of the cognitive approaches to delusion, while preserving the position, which we think is fundamentally correct—that the delusional experience is immediate and not derived from faulty inference.

Salience effects and their role in misattribution, for example, are prime examples of the sort of effect that, in the case of schizophrenic delusion, exerts important precognitive, or background effects. The association between schizophrenic symptoms and dopaminergic dysfunction has been studied and generally accepted for decades, though there is still considerable controversy over the exact role played by dopaminergic excess. Drawing from the contributions of such researchers as Bindra, Toates, Panksepp, DiChiara, and others, Kapur (2003) expands on the so-called motivational salience hypothesis, that is, the view that dopamine mediates the qualitative (attractive or aversive) “valence” that attaches to an external stimulus. Overall, Kapur seeks to explain schizophrenic delusion as a state of abnormal salience. He arrives at this conclusion by combining a modified version of the motivational salience hypothesis with the dopamine hypothesis, that is, the hypothesis that schizophrenia is associated with the faulty dopamine regulation.

For Kapur, the motivational salience hypothesis helps explain the heightened significance that schizophrenics may attach to ordinary objects, sensations, events, or ideas. He speculates that prior to a psychotic episode, patients experience an abnormal dopamine release, which in turn leads to an experience of the exaggerated and contextually unmotivated importance of ordinary stimuli. In the psychotic state, Kapur insists, it is not just that dopamine mediates salience but that the rush of dopamine “becomes a creator of saliences, albeit aberrant ones” (2003, p. 15). As he points out, this fits well with first-person reports of the psychotic experience. Patients report such things as having a new awareness of their surroundings, a heightened sense of consciousness, unusually sharp sensory abilities, newfound clarity of thought, and so on. This exaggerated sense of significance is the catalyst for the delusion. Motivational salience may also help explain the problem of specificity (Gallagher, 2004), that is, the fact that in the case of thought insertion, specific kinds of thought contents (but not all kinds) appear to be inserted. In terms of content, these experiences are very specific and are often associated with specific others. In effect, schizophrenics may experience a certain semantic or content consistency amid the agentive inconsistency of their inserted thoughts. It is difficult to explain the problem of specificity in purely subpersonal terms (e.g., Frith’s [1992] suggestion about the dysfunction of a subpersonal comparator). Saliency effects at the first-order level of experience, however, may contribute to an explanation.

Somewhat surprisingly, at this point, Kapur largely invokes a two-stage, top-down process for completing the full-blown generation of the delusion, claiming that “once symptoms are manifest, delusions are essentially disorders of inferential logic”
and that delusions are “a 'top-down' cognitive explanation that the individual imposes on these experiences of aberrant salience in an effort to make sense of them” (2003, p. 15). It is not clear, however, where Kapur divides the cognitive from the precognitive influences on the delusion, in that he accounts for the variable form of expression of the delusion partly in cultural terms. For example, he notes that a patient in one context may impute evil intentions to a local shaman, whereas in another, more modern context, the patient may implicate the local police (2003, p. 15). We don't dispute the cultural variability that one sees in the expression of delusions, or the cognitive contributions that may later reverberate through the patient’s altered epistemological framework, coloring each psychotic episode. Rather, we argue that these cognitive factors are not, in the first instance, responsible for the delusional experience itself.

To recap Kapur’s contribution, however, and to return to our main focus, he argues that (1) schizophrenics suffer from aberrant dopamine regulation, (2) dopamine mediates salience, and therefore, (3) schizophrenics suffer from aberrant salience effects. Additionally, Kapur notes that psychotic schizophrenics suffer from “alterations in attributional styles,” among other biases and distortions, which is not surprising if salience mediates attribution. Next, we examine this link between causal attribution and salience, adding another link between lower order, neurological phenomena and first-order, phenomenological experience.

Several authors have touched on the relation between salience and attribution. Heider (1958) is often credited as the earliest, but the work of Taylor and Fisk (1975, 1978) is widely cited as the most central and detailed work in substantiating the relation between salience and attribution. In one well-known experiment, Taylor and Fisk (1975) set up a two-man conversation, watched by six silent observers arrayed in different positions around the two men. The two men in the center (call them A and B) sat facing each other as they conversed, and the observers were arranged around them as follows: two observers sat to the north of the conversation, behind A, with a direct view of B’s face (and no view of A’s face); two observers sat to the south of the conversation, behind B with a direct view of A’s face (and no view of B’s face); one observer sat to the east of the conversation, with an equal view of both A and B; and one sat to the west of the conversation, also with an equal view of both A and B. Thus, during the conversation, two people observed only A’s face, two observed only B’s face, and two had equal views of both men’s faces. Though the six observers were naive participants, the two conversationalists were trained to engage in a standard, scripted conversation that lasted approximately 5 minutes. During this conversation, A and B made roughly equal contributions, exchanging “the same amount of information of approximately equivalent social desirability” (Taylor and Fisk, 1975, p. 441). After the conversation, the observers were asked to rank A and B according to the role each had taken in setting the tone of the conversation, influencing the course and content of the conversation, and causing the other man to behave as he did. As Taylor and Fisk predicted, perceptions of causal agency varied according to the position of the observers. Those who could see the face of A judged him to have exerted more causal influence than B; those who could see the face of B judged him to have more causal influence than A; those with an equal view of both
judged that each had displayed roughly equal causal influence over the conversation. Salience, in this case, was a matter of which man’s face each observer could see; other things being equal, an actor whose face we can see is more salient to us than an actor whose face we can’t see. That which is more salient, in turn, is judged to have greater causal effect. As Taylor and Fisk conclude,

Perceptually salient information is subsequently overrepresented when one imputes social or causal meaning to one’s perceptual experience. Thus a perceiver, even a highly sophisticated adult perceiver, is to some extent bound by the literal nature of the sensory experience he seeks to transcend when he is interpreting the environment of which he is a part. (1975, p. 445)

Coupling this with Kapur’s work, if psychosis is a state of aberrant salience, and salience governs causal attributions, it is not surprising that psychotics should exhibit certain kinds of attribution errors. Furthermore, this matter of being “bound” by our sensory experience, in this context, may help explain the stubborn, nearly inescapable nature of errors of attribution, as well the precognitive level at which their influence operates.

In an appropriately titled paper, “Swimming Upstream against the Fundamental Attribution Error,” Pietromonaco and Nisbett (1982) show how attribution errors are resistant to change, even for normals (nonpsychotics), even under circumstances in which subjects are aware of the pitfalls of misattribution. In an experiment that has since become a classic, Pietromonaco and Nisbett employed another famous experiment, the Darley and Batson (1973) study concerning the helping behavior of seminary students. In this 1973 study, seminary students were asked to give a short talk on the parable of the Good Samaritan. While on their way to their talk, they were lead by an experimental assistant down an alley where they encountered a shabbily dressed man, slumped over and groaning in distress (actually, an actor planted by the experimenters). Darley and Batson then observed whether the seminary students would stop to help. They found that the extent to which the students helped was related to how hurried they were to get to their talk. If the experimental assistant rushed them along, telling them they were running late and so better hurry, they were less likely to help or even acknowledge the man in the alley. But if they weren’t rushed along, and told they had a few minutes to spare, they were more inclined to assist the man. The fact that the students were giving a talk on the parable of the Good Samaritan seemed to have no effect on their behavior. The lesson of this study, for the purposes of the later Pietromonaco and Nisbett experiment, was that helping behavior was related to the external situation of hurriedness, rather than on the internal or dispositional factor of religiosity.

Pietromonaco and Nisbett used this earlier study in the following way: They presented about half of their subjects with a summary of it, but withheld it from the other half. They then presented all subjects with a scenario similar to the one described in the Darley and Batson study, and asked them to predict the helping behavior of the seminary students. Not surprisingly, the group that had not read about the Darley and Batson study relied on religiosity, a dispositional factor, in predicting helping
behavior, thus repeating the fundamental error of attribution. What was surprising, however, was that the students who had read the Darley and Batson study—those who were already aware of the tendency toward misattribution—did the same thing! In fact, they were not significantly less inclined to repeat the error than the naive group. These and similar experiments support our view that attribution errors can and do exert a precognitive, noninferential influence on our experience.

Through following out the neurological and phenomenological underpinnings of misattribution, we hope to have given a plausible, bottom-up account of the genesis of schizophrenic delusions, particularly those involving misattributions of agency. In our account, there is no need to appeal to any cognitive-level mistakes in reasoning to explain how delusions arise. Though we do grant that many cognitive-level effects are commonly observed in schizophrenic psychosis, we deny that they should be regarded as the causal catalyst for the delusion.

NOTE

1. In later work, Hohwy (2004) appears to favor a combination of bottom-up and top-down models and thinks neither is adequate alone. But he does not appear to revoke his claim about the nonderivative nature of (at least some) forms of schizophrenic delusion.

REFERENCES


