
Metamisery and Bodily Inexistence

A Reply to Ying-Tung Lin

Philip Gerrans

The difference between the Cotard Depersonalisation and Depersonalisation Disorder may consist, not only in the fact that the Cotard delusion is a response to prediction error affective/bodily information, but the level in the predictive processing hierarchy at which predictions about bodily information are violated.

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1 Prediction error and veridicality

My explanation of Depersonalisation Disorder (DPD) argued that the characteristic experience is shared by people who suffer from the Cotard Delusion (CD). The difference between the two conditions is that the person with DPD does not develop a delusional response to her experience of de-affectualisation. She simply reports as it is: “I *feel as if* my experiences do not belong to me”. The person with Cotard, however develops an explanation of that feeling and identifies with it “I no longer exist”. In commenting on this proposal Ying-Tung Lin opens up a range of new possibilities for cognitive the-

orizing. The first is that the predictive coding approach provides a new framework for cognitive theorizing which improves on “second factor” approaches to delusion. The second is that attention to the predictive nature of the processes which generate experience might suggest an important difference between the two conditions: namely the role of the Anterior Insular Cortex (AIC).

One way to approach the phenomenon would be to ask why the person with DPD seems to be able to understand that her experience is not veridical while the person with CD

does not (*modulo* all the *caveats* about the epistemic status of delusional attitudes). The CD patient for example does not say “It feels as if I don’t exist” she says “I don’t exist”. This way of approaching the problem fits with a now standard approach to delusion, that argues that there are (at least) two stages of cognitive processing involved in delusion formation. The first generates an anomalous experience and the second generates a delusional response to that experience.

Ying-Tung Lin however, following Hohwy and Clark, explains delusion in terms of the attempt by higher order control systems to account for surprisal in a predictive coding hierarchy. The radical aspect of these ideas is that neither the precipitating experience nor the delusional response need be conceived of as the result of cognitive malfunction. Because there is no intrinsic connection between error minimization and malfunction “certain misrepresentations can lead to error minimization; furthermore, it is possible for misrepresentation rather than veridical representation to lead to a generative model” (Lin this collection, p. 8).

Ying-Tung Lin’s commentary applies these ideas to the Cotard delusion, arguing that it is a model that mimimises the prediction error represented by depersonalisation experience. Her target is to describe a

cognitive architecture [that] could, in principle, explain CD in terms of its development from depersonalization, and what exactly are the underlying differences between patients suffering from the Cotard delusion and those suffering from depersonalization disorder (DPD) but free from the Cotard delusion? (Lin this collection, p. 2)

2 The sense of presence

Before I make some comments, I want to highlight the original aspects of her account and show how it can explain how experience acquires a quality of “mineness” or “sense of presence”, that is of belonging to a self. We can then use the predictive coding framework to ex-

plain how the sense of presence can go missing. Loss of the sense of presence signals a prediction error which then requires a higher-level system to build a predictive model that fits that error.

The first point to note is that on the most radical interpretation of predictive coding ideas the veridicality of representation is a corollary of cognition not its primary goal. The primary goal of a cognitive system is to predict its own informational states consequent on its actions (broadly construed to include internal regulatory actions). The point is not just that the objects of experience are constructed and hence may be illusory or misrepresented. Rather veridicality of experience is secondary to the accuracy with which cognitive process predicts the flow of information in sensory systems. As she says in the case of perception this means that “instead of aiming to answer the question ‘what is this?’ perception studies should answer the question ‘... what does this resemble?’” (Lin this collection, p. 6). This formulation captures the idea that the visual system, for example, is not passively registering retinal information and constructing a representation of the external world, but using a model which predicts the flow of information coming from the retina.

The first step is to apply the same idea to interoception. We see that the mind is not passively registering changes in body state and constructing a model of the body accordingly but predicting the flow of bodily information in cognitive context. Those contexts range from maintenance of homeostasis to the use of affective experience to inform decision-making and reflective cognition. Thus when I think about the past or future these episodes of retrospection or prospection are infused with affective significance.

The radical import for the understanding of pathologies of self-representation is very elegantly brought out by her discussion. Ying-Tung Lin in effect argues that the experience of the self in autobiographical episodes is no more direct than experience of the world in perception or of past events in memory. In each case no object is directly represented or experienced. Rather the relevant object in each case (object

of perception, remembered event, or self in the case of first person awareness) is *inferred* as a part of a process of optimizing predictive accuracy in specific cognitive contexts.

As many have argued the role of the Anterior Insular Cortex (AIC) is to integrate and represent affective information: i.e., those bodily states, which tell the organism how it is faring in the world, actual, imagine or remembered. The point to recall from Ying-Tung Lin's account is that the AIC is not representing a self but constructing and optimizing a model that predicts the flow of affectively-charged bodily information.

This is why when AIC is hypoactive the subject feels a loss of subjective presence, reported as depersonalization. In particular the patient has a loss of subjective presence for her own body: she registers changes in body state but they do not feel affectively significant for her. Because that lack of feeling is not predicted she then reports it in the vocabulary of DPD.

Why does the DPD patient not proceed to something like the Cotard delusion? According to Ying-Tung Lin whether a delusion is formed depends on the degree of precision assigned to the information produced by hypoactivity in the AIC.

In the case of Cotard delusion developed from depersonalization, when one has the expectation of high precision, the system tends to be driven by the bottom-up predictive error of unexpected hypoactivity of the AIC, rather than the prior model. One is, therefore, more likely to revise the model in order to explain away the surprisal resulting from the mismatch between the actual and predicted activation level of the AIC; that is, the systems of patients suffering from CD are driven by an urge to modify their top-down predictive models in order to conform to the loss of AIC activity. The construction of the model in CD is considered an attempt to minimize prediction error.

3 Conclusion

Reading over this account I wonder if there is an alternative interpretation available consistent with the predictive coding account. It is consist-

ent with the view that patterns of activity in the AIC are abnormal in CD, but unlike DPD those patterns are not the result of VLPFC-induced hypoactivity.

Ex hypothesi the CD patient is extremely depressed. Evidence suggests that circuitry centred on the amygdala is affected, which means that online affective responses are flattened.

The role of the AIC is to monitor for changes driven by affective processing. It thus predicts for example that a typically positive event would be processed as positive. Thus, when that event is processed as negative or neutral, the AIC detects an error, signaled in the form of an anomalous experience. The patient is in the position being able to detect and signal changes in her affective responses, which take the form of unpredicted absences in bodily response. Thus *her lack of felt bodily response is processed as affectively significant* in the Cotard delusion with the result that she experiences it. Thus she does not feel neutral she feels miserable. Or as we might put it *she feels metamisery* because the role the AIC is to enable the person to feel the affective significance of bodily changes *including the absence of predicted changes*. In Cotard delusion the patient feels the affective significance the unpredicted absence of positive changes.

In DPD, by contrast, the patient does not feel the significance of bodily information because her AIC is inhibited and hypoactive.

Thus the difference between the two conditions may consist, not only in the fact that the Cotard delusion is a response to lower level prediction error, but the level in the predictive processing hierarchy at which predictions about bodily information are violated.

References

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