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Understanding the neurobiological bases of psychiatric disorders is a huge challenge for the neurosciences. It has been repeatedly said that the lack of animal models that truly resemble the different psychiatric conditions is one of the main obstacles for progress in this field. Although this paucity of models is certainly a problem, it is hardly the whole story. Owing to the advent of imaging techniques on the one hand and the astounding progress in genomics on the other, it is now more feasible than ever to investigate the neurobiological roots of psychiatric disease in the patients themselves. What, then, might be the limiting step?

We tend to think about psychiatric disorders as sharply defined entities, conditions with precise boundaries that leave little room for confusion. But the truth is that there is significant phenotypic heterogeneity among the people that are affected by each disease, making it necessary to refine our existing nosology. In other words, if we identify single specific phenotypes that define true boundaries between groups of patients and establish the neurobiological underpinning of each phenotype, we might end up with a better understanding of mental disorders.

The study of spontaneous confabulation, which Armin Schnider reviews in this issue (p. 662), provides a fascinating example of the power of this approach. Instead of thinking about confabulation as a single entity, the definition of a specific phenotype with predictive value is giving us clues about how to treat this condition and, in broader terms, about the way in which our brain adapts thought to ongoing reality.

Although it can be argued that the search for phenotypes might lead to an extreme fractionation of psychiatric disease, understanding each phenotype should be extremely useful to subsequently solve the larger puzzle that these illnesses represent.