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What can the neural architecture of the language system tell us about schizophrenia...and vice versa?

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“I always liked geography. My last teacher in that subject was Professor August A. He was a man with black eyes. I also like black eyes. There are also blue and grey eyes and other sorts, too...”
(Bleuler, 1911/1950).

This is an example of language produced by some patients with schizophrenia—a common neuropsychiatric disorder that affects 1% of the adult population. This type of disorganized speech is usually attributed to a ‘thought disorder’ or a ‘loosening of associations’, which influences not only the production of language but also comprehension and other aspects of higher-order cognition in schizophrenia patients. It is usually assumed that thought disorder reflects a *qualitative* abnormality in the neurocognitive mechanisms engaged in language processing. The assumption is that healthy individuals first retrieve the meaning of individual words, combine these words syntactically to form sentences, and then combine sentences with other sentences to construct whole discourse. In contrast, thought disorder has often been viewed as a separate disturbance of memory—stored associations between single words and whole events intrude upon normal language comprehension and production mechanisms.

Our lab has carried out a series of cognitive neuroscience studies in both patient and control populations that challenge these assumptions. We are using multimodal neuroimaging techniques—event-related potentials, functional MRI and magneto-encephalography—to probe the time-course and neuroanatomical networks engaged in language processing. Our findings suggest that we use information stored in memory all the time to facilitate neural processing of incoming words in the anterior temporal cortex between 300-500ms of their onset. Sometimes we even use memory to *predict* upcoming words. This leads to a further enhancement of neural facilitation if these predictions are confirmed. If, however, we encounter words that *disconfirm* our predictions, we appear to recruit quite a different neural system at a slightly later stage of processing (between 500-800ms)—one that is closely linked to core aspects of cognitive control. We propose that schizophrenia is characterized by a failure to recruit this system in response to disconfirmed predictions, and that this leads to language and thought that is dominated by memory-based associations.

Seen within this broad framework, thought disorder does not reflect a qualitative abnormality in how language is processed; rather, it is best conceptualized as reflecting an *imbalance* of a tight reciprocal relationship between the memory-based predictive and cognitive control neural systems that contribute to normal language processing. In this way, the study of the neural architecture of the language system can give important insights into how thought and communication break down in neuropsychiatric disorders such as schizophrenia, and vice versa.