LETTER TO THE EDITOR

Language functions in right-hemisphere damage and schizophrenia: apparently similar pragmatic deficits may hide profound differences

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Sir, In a recent article discussing the cognitive roots of schizophrenic communicative behaviour, Mitchell and Crow (2005) emphasized the importance of full access to right hemisphere language functions to ensure successful social communication. Based on the literature describing communicative deficits among right-hemisphere-damaged (RHD) patients, as well as on the literature reporting functional neuroimaging studies of healthy individuals, these authors suggested that ‘the core deficit in psychosis is a failure of segregation of right from left hemisphere functions’ (p. 963). In other words, Mitchell and Crow (2005) argue for a reduction of the language lateralization in schizophrenia (SZ). The role of so-called ‘RH functions’ in the pathophysiology of SZ proposed by Mitchell and Crow (2005) could prove crucial to an understanding of SZ from a neurobiological point of view; this proposal may also account for the presence of social communication deficits in SZ. However, the apparent similarities noted by Mitchell and Crow (2005) between the pragmatic deficits following RH damage and those present in patients with SZ seem to be only at the surface level. Indeed, the underlying cognitive dysfunctions appear to be quite different in these two populations.

As Mitchell and Crow (2005) rightly remind us in their article, language requires more than the classical syntax, phonology and morphology processing levels. It also relies on pragmatics, which refers to language use in context including non-literary uses of language such as metaphor, indirect requests or irony. According to the literature on RHD individuals (Joanette et al., 1990), an intact RH is required to allow for the normal processing of non-literary utterances that goes beyond the comprehension of syntax and vocabulary. Despite their ability to understand simple sentences, RHD patients with pragmatic deficits—which do not tend to affect left-hemisphere-damaged patients—experience significant communicative disabilities in their everyday life.

However, the RH cannot be viewed as nesting ‘pragmatics’ per se. Many different cognitive processes are required for such a complex social behaviour, some of them possibly depending upon RH-based neural networks. It should first be mentioned that the evidence in favour of a major role for the RH in the processing of non-literal language remains controversial. Contrary to Bottini et al. (1994), who reported a right lateral temporal and frontal PET activation during the processing of metaphors, Lee and Dapretto (2006) as well as Rapp et al. (2004) suggested that the RH’s involvement in the processing of metaphorical language might reflect mainly the increased complexity of non-literal language rather than an RH specialization for understanding metaphors. Others, such as Mashal et al. (2005), argue in favour of a specific contribution by the RH. They suggest that the RH may specialize in the processing of metaphors, for example, because of its greater sensitivity to the processing of less salient linguistic material. Consequently, there are still very few data that directly address the neural substrate of pragmatics. These existing data are not convergent.

Over and above the factors mentioned above, the quest for a single cognitive dysfunction that would account for the impaired processing of all non-literary utterances is probably naïve (Champagne-Lavau et al., 2006). Indeed, pragmatics depends on a multitude of high-level interactive cognitive processes. In a meta-analysis of studies with different clinical populations (e.g. right-hemisphere damage, traumatic brain injury, autism), Martin and McDonald (2003) concluded that many different cognitive dysfunctions may result in an impairment of the processing of non-literary utterances. Consequently, trying to identify a single specific cognitive mechanism as the sole cause of pragmatics difficulties is pointless. Thus, similar pragmatic deficits may result from the acquired dysfunction of many different cognitive processes which compose the pragmatic system. The large
number of disorders (e.g. RHD, traumatic brain injury, SZ, autism) characterized by pragmatic impairments may reflect a heterogeneous range of underlying functional deficits (Eales, 1993).

Our group has generated data which tend to concur with such a view. We have thus recently investigated (Champagne et al., 2005) pragmatic abilities and executive functions of 16 patients with SZ, 15 RHD patients and 25 normal control participants—all matched for age and educational level. The goal of this study was to identify the different cognitive dysfunctions accompanying apparently similar pragmatic deficits in RHD and SZ patients. This study showed a clear dissociation between RHD and SZ patients concerning their understanding of indirect versus direct requests and the types of executive dysfunction they experience. Thus, SZ patients showed specific problems understanding indirect requests as opposed to direct requests, whereas the inverse pattern was found in RHD patients (cf. Fig. 1). They also showed difficulties with both non-idiomatic and idiomatic metaphors while RHD patients showed difficulties only for non-idiomatic. In addition, this pragmatic deficit seems to be associated with a lack of inhibition in RHD individuals (Champagne and Joanette, 2004), whereas a lack of flexibility may well explain SZ patients’ deficits. These results clearly demonstrate that apparently similar pragmatic deficits can express the impairment of very different cognitive components, namely here a lack of flexibility in SZ patients versus a lack of inhibition in RHD patients. Thus, RHD patients would correctly activate contextual information but their lack of inhibition would prevent them from inhibiting irrelevant information in a given situation, leading them to over-attribute intention to the protagonist and to understand literal utterances as non-literal ones. Conversely, SZ patients’ lack of flexibility would prevent them from activating relevant contextual information, leading them to access only the literal meaning of the utterance in a given context.

Overall, as suggested by Eales (1993), our recent findings support the claim that, although SZ patients seem to have pragmatic deficits that are apparently similar to those reported in individuals with RHD patients, different cognitive deficits appear to underlie and account for these deficits, thus raising doubts about the reduced lateralization hypothesis proposed by Mitchell and Crow (2005). Our results rather suggest that in SZ and RHD populations, specific cognitive processes are malfunctioning, rather than a global pragmatic dysfunction. Here, like in many steps of medical history, appearance can be misleading. It is clearly impossible to conclude with Mitchell and Crow (2005) that the pragmatic deficits in those two very different populations express a convergence of the basic underlying mechanisms.

References
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