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Discussion forum

One or two things I know about apraxia

*Raffaella Ida Rumiati**

SISSA Trieste, Italy

In *Apraxia* (2013) Goldenberg combines a very reach clinical experience, reflected in many interesting observations and treatment of apraxic patients, with a unique scholarly knowledge of the classical studies on apraxia as it can be appreciated in the first three chapters but also throughout the book. This combination allowed him to cover a lot of what we wish to know about high-level motor deficits. In my commentary, however, I will focus on Goldenberg's explanations of what goes wrong in limb apraxia as assessed by asking patients to imitate meaningless gestures and to use objects and tools. Indeed, in their present formulation, these accounts are still susceptible to some further discussion.

1. Imitation

A neurologist by training, Goldenberg opened up very soon in his career to a cognitive view of apraxia and adapted it to his own taste. Let us first consider action imitation (chapter 6). The impairment at imitating gestures, a key symptom of ideomotor apraxia, was hypothesized to be caused by a damage either to a vision-to-action conversion mechanism (also called sub-lexical route), necessary for imitating novel, meaningless gestures, or to a lexical-semantic mechanism applicable to the imitation of gestures already known to the patient (see the model of action production by Rothi, Ochipa, & Heilman, 1991). Goldenberg and Hagmann (1997) described two patients who were worse at imitating meaningless than meaningful gestures as well as at reproducing them on a manikin. The authors argued that this pathological performance was caused not only by a faulty vision-to-action conversion mechanism, as originally hypothesized by Rothi et al. (1991), but also by an impaired *body structural description* that codes the spatial relations between body parts, and subserves the reproduction of one's own gestures as well as of the body postures on the manikin (see also chapter 7). This selective deficit at imitating meaningless gestures and reproducing them on a manikin has been observed at least in another patient (Peigneux et al., 2000). The possible interaction between action imitation and a supramodal representation of the body has been acknowledged also by other authors (Buxbaum, Giovannetti, & Libon, 2000; Buxbaum, 2001; Schwoebel, Buxbaum, & Coslett, 2004), to date there is no general agreement as to whether the body representation engaged during action imitation is indeed is the body structural description, as suggested by Goldenberg, or as suggested by Buxbaum, Coslett and colleagues, the *body image*, that is a dynamic representation that codes the position of body parts in movement (see Rumiati, Carmo, & Corradi-Dell'Acqua, 2009, for a discussion on this issue). What needs to be clarified is whether the presence of damage to either body representation is necessary for an apraxic deficit in gesture imitation to occur or whether the two deficits can simply co-occur.

2. Tool use

Goldenberg extensively studied ideational apraxia defined here as the patients' reduced ability to use common objects and tools. Where does the functional breakdown lie in patients who fail to use objects correctly? He argues that it depends on the kind of knowledge that is destroyed by brain damage. First, the functional knowledge about objects and tools associates them to different important aspects of their use, including their purpose, recipient and typical action. A loss of this knowledge affects only the use of objects and tools that already belong to patients' repertoire. On the other hand, individuals are also endowed with manipulation knowledge that, in Goldenberg's

* *Corresponding author*. Neuroscience and Society Laboratory, SISSA, Via Bonomea 265, 34136 Trieste, Italy.
E-mail addresses: rumiati@sissa.it, raffaella.rumiati@sissa.it.
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view, coincides with a mechanical problem solving ability, a generative system that specifies the functional properties of an object's parts. As such, this ability allows us to use novel objects, as specific functional properties can be derived from the objects' perceptual structure. Recently Goldenberg and Spatt (2009) showed that lesions in five patients with a defective retrieval of functional knowledge, but intact problem solving lesions, overlapped in the temporal cortex, while in five patients with the reverse pattern they overlapped in the parietal cortex. However, patients who failed on both tasks had problems with the use of common tools like hammer or scissors (see also Goldenberg & Hagmann, 1998). Goldenberg concludes that in order to efficiently use tools it is necessary that either functional knowledge or mechanical problem solving is intact (pp. 134–135), but that damage to the latter disrupts tool use more than the former.

This conclusion is partially in contrast with the neuropsychological evidence that degraded functional knowledge does not prevent patients from using tools appropriately (see Negri, Lunardelli, Gigli, & Rumiati, 2007; Silveri & Ciccarelli, 2009). We argued that the errors typically committed by apraxic patients when using common tools and objects in everyday activities occur at the level of action selection and are not due to a loss of functional semantic knowledge (Rumiati, Zanini, Vorano, & Shallice, 2001). In addition, when Cooper (2007) applied a computation model previously developed (Cooper & Shallice, 2000) to five standard multiple object tasks, he obtained error patterns similar to those committed by the two ideational apraxic patients studied by Rumiati et al. (2001). More specifically, tool use errors were found to arise from a generalised disturbance of object representations in triggering action representations (or schemas).

Errors, especially omissions, committed in performing naturalistic actions as those employed in our study (Rumiati et al., 2001), have been argued not to be caused by a selective damage to a particular mechanism or subsystem necessary for using tools (see Schwartz et al., 1998; Humphreys & Forde, 1998; Forde, Humphreys, & Remoundou, 2004; and see Goldenberg, 2013, chapter 9). In this view, the cause of errors is not deficit-based but due to the resource limitation (see Goldenberg, 2013, chapter 9, p. 141). I do not share this view and I argue that errors that relate to both the sequential organisation of an action as well as the misuse of the appropriate tools are qualitatively the same whether the object use is tested with objects in isolation or in a naturalistic action (De Renzi & Lucchelli, 1988; Rumiati et al., 2001). I do not discard the possibility that a diminished top down attentional control may account for some of the errors observed in patients performing naturalistic actions, as also it has demonstrated been by the simulation study of Cooper and Shallice (2000), but I would maintain that the deficit affecting the tool use in left-brain damaged patients can be pin down on a faulty mechanism in selecting actions from object representation (Cooper, 2007).

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