SPECIAL ISSUE: POSITION PAPER

BEYOND A SINGLE AREA: MOTOR CONTROL AND LANGUAGE WITHIN A NEURAL ARCHITECTURE ENCOMPASSING BROCA'S AREA

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ABSTRACT

In this position paper, we discuss a neural architecture comprising three major cortical systems: the inferior frontal cortex (including Broca's area), the rostral part of the posterior parietal cortex, and the superior temporal cortex. This network of areas is critical to imitation and to language. What are the functional properties of the network that make it possible for imitation and language to co-exist within the same neural architecture? We propose that this network implements cortical forward and inverse modeling for actions and speech sounds of self and others.

Key words: imitation, aphasia, mirror neurons, inverse models, forward models

Broca's area is a classical language area. Recent evidence has suggested that the functions of Broca's area are not confined to language and encompass some aspects of motor behavior and imitation. To understand how it is possible that a cortical region implements shared functions between behaviors seemingly so different, it is necessary to consider Broca's area not in isolation, but as part of a larger network of neural systems. The network of the human brain we discuss here is comprised of the inferior frontal cortex (including Broca's area), the rostral part of the posterior parietal cortex, and the superior temporal cortex.

Single-cell studies in macaques have suggested the existence of evolutionary precursors of these neural systems. In the inferior frontal cortex of the macaque, area F5 contains mirror neurons that fire during the execution of goal-directed actions but also during the observation of similar actions made by others (Di Pellegrino et al., 1992; Gallese et al., 1996; Kohler et al., 2002; Umilta et al., 2001). Neurons with similar properties are also observed in area PF, in the rostral sector of the inferior parietal lobule of the macaque (Rizzolatti et al., 2001). Moreover, in the superior temporal sulcus (STS) of the macaque there are neurons that fire to the sight of goal-directed actions (Jellema et al., 2000; Perrett and Emery, 1994; Perrett et al., 1989), though unlike the mirror neurons in F5 and PF, these are not motor neurons. STS and PF are anatomically connected (Seltzer and Pandya, 1994), and PF and the sector of F5 containing mirror neurons are also reciprocally connected (Rizzolatti and Luppino, 2001). These anatomical and physiological data delineate a whole circuitry for coding actions of self and others composed by STS, PF, and F5 in the macaque brain.

In the human brain, the homologues of STS (superior temporal cortex), PF (rostral posterior parietal cortex), and F5 (inferior frontal cortex including Broca's area) have been recently associated with imitative behaviors in a series of functional magnetic resonance imaging (fMRI) (Carr et al., 2003; Iacoboni et al., 1999, 2001; Koski et al., 2002, 2003) and transcranial magnetic stimulation (TMS) studies (Heiser et al., 2003). The available evidence suggests that the superior temporal cortex provides a higher order description of the observed action (Puce and Perrett, 2003), the rostral part of the posterior parietal cortex provides 'somatosensory' information (Decety et al., 1997) (broadly conceived, maybe even of kinesthetic and kinematic type) (Kalaska et al., 1983, 1990) associated with the observed and to-be-imitated action, and the inferior frontal cortex codes the goal of the observed and to-be-imitated action (Heiser et al., 2003; Koski et al., 2002). Single-unit studies in macaques have likewise suggested that area F5 represents the goal of actions, since neurons in this region are sensitive to the presence versus absence of a target object even when that object is obscured from view (Umilta et al., 2001). From the inferior frontal cortex, efferent copies of motor plans are sent back to the superior temporal cortex (Iacoboni et al., 2001). These efferent copies allow a prediction of the sensory consequences of planned imitative actions. In the superior temporal cortex there would be a matching process between the higher order visual description of the observed action and the predicted sensory consequences of the planned imitative action. When this matching process converges, imitation occurs (Figure 1). While most studies of imitation have focused on hand actions, imitation of facial expressions has

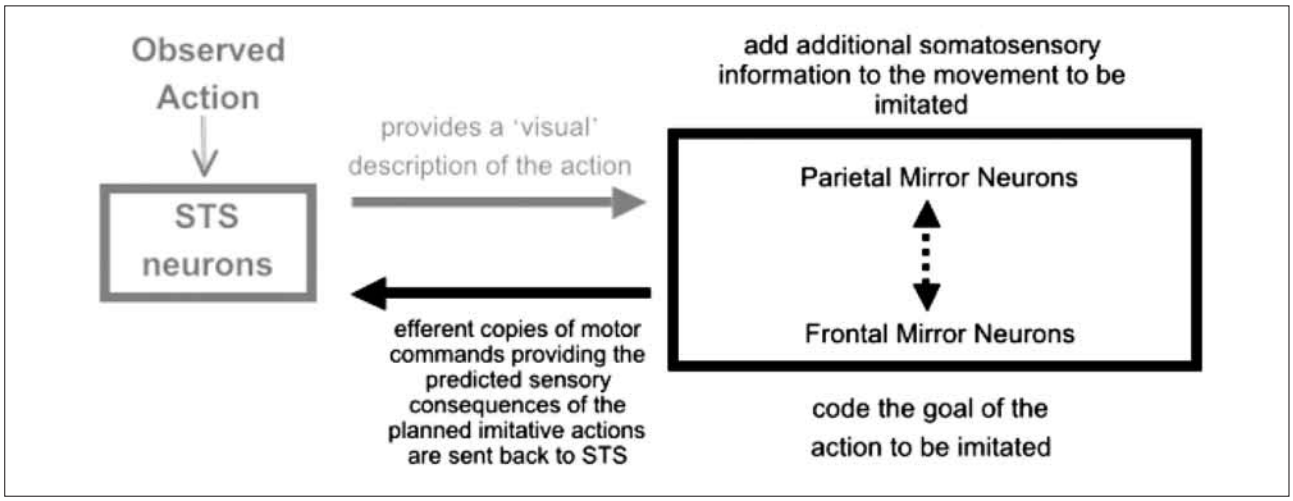

Fig. 1 – *Schematic representation of the neural architecture for imitation. The inverse model goes from superior temporal sulcus (STS) to parietal to frontal mirror neurons, whereas the forward model goes from frontal to parietal mirror neurons to STS. The grey part of the schema is "visual", the black part is "somatomotor".*

also been shown to rely on a similar neural network (Carr et al., 2003). An interesting open question is whether imitation of actions involving the leg or foot would involve the same premotor areas, given that these body parts are not thought to be represented in Broca's area.

Let us now address general functional mechanisms of motor control and learning that may play a role in imitation. A critical aspect of motor control is the ability to retrieve the motor plan necessary to reach a desired sensory state (an 'inverse model') (Wolpert and Ghahramani, 2000). During imitation, the desired sensory state (the observed action) is represented in superior temporal cortex, thus this area provides the input to the inverse model. Inverse modeling is implemented in the connections between superior temporal cortex and parieto-frontal mirror areas, such that the output of the inverse model consists of the motor commands necessary to imitate the observed action, which are coded in inferior frontal cortex.

Another kind of internal model is also thought to be important in motor control: the "forward model", which is a prediction of the sensory consequences of a planned action (Wolpert and Ghahramani, 2000). We propose that forward models are implemented by projections from inferior frontal cortex, which codes the intended motor plan, to superior temporal areas (via parietal cortex). So in superior temporal cortex, the output of the forward model, which is the predicted sensory consequences of the motor commands, can be compared to the original input to the inverse model, which is the visual description of the action to be imitated. The role of the rostral posterior parietal areas would be to reinforce specific forward-inverse model pairs under specific contexts when the predicted sensory consequences are confirmed by re-afferent feedback. This reinforcement is referred to as a responsibility signal (Haruno et al., 2001).

Let us now discuss in greater detail how the pairing of forward and inverse models can occur within the network for imitation, that is, how the principles outlined above can be implemented in a circuitry that seems dedicated to the processing of actions. Under widely different visual circumstances STS neurons fire at the sight of the same kind of action (Jellema et al., 2002). PF and F5 neurons also respond to a relatively large class of observed actions under different visual conditions. With regard to their motor properties, however, *mirror* neurons in PF and F5 often have a narrow tuning, coding a specific type of action (Gallese et al., 1996). It is possible that a large variety of visual stimuli in the form of observed actions activate non-selectively a large number of neurons in superior temporal, posterior parietal, and inferior frontal cortex. This visual activation, however, is mapped efficiently only onto a few specific motor outputs coded in inferior frontal cortex. When a less efficient motor output is selected, the forward model generates a large error signal, and the pairing is not reinforced. When the appropriate motor output is selected, a small error signal assigns "high responsibility" to the pair of forward and inverse model for that given action (Haruno et al., 2001).

Within the neural architecture described here, the aspects of internal models we have discussed in the last few paragraphs are general aspects of motor control that should not be considered specific to imitation, whereas the neural mechanisms associated with "mirroring" are, if not entirely exclusive to the imitative process, certainly more specific to imitation than to motor control in general.

How is this network of areas important for imitation relevant to language? It is quite striking that the three key brain regions identified for imitation are all considered crucial for language in classical neurolinguistic models. Although it is increasingly clear that the Wernicke-Lichtheim-Geschwind model represents only a broad outline

of a more complicated reality (Dronkers et al., 2000), the important roles of inferior frontal cortex, superior temporal cortex, and the supramarginal gyrus in linguistic processing have been confirmed in numerous studies (Caplan, 2003; Bates et al., 2003). Let us now address how the functional mechanisms for motor control described above may plausibly account for language disorders associated with lesions in the network.

Lesions to Broca's area generally result in articulatory deficits (Caplan, 2003), which would follow from the role of Broca's area in generating the output of the inverse model (necessary for control of the articulatory output). Broca's patients also show *conduite d'approche*, that is, the patient gets closer and closer to the target word through repeated attempts (Nadeau, 2003). This would be in line with the role of Broca's area in generating the input of the forward model (necessary for prediction, important when planning articulatory sequences). In fact, if the forward model cannot be used as predictor of the planned motor output, the only correction to a motor plan can be provided by overt feedback, as in conduite d'approche.

The superior temporal cortex is a multi-sensory integration area responding to both visual and auditory stimuli, and integrating them when necessary (as when we see the lips of somebody else moving and simultaneously hear her speech) (Calvert et al., 2000). Wernicke's area may perform in language an analogous function to superior temporal cortex in imitation, i.e. it may provide a perceptual representation of the input. Although patients with Wernicke's aphasia are fluent, they do have severe production problems, ranging from paraphasias in which phonemes are incorrectly selected, to semantic errors in which words are incorrectly selected, to neologisms and jargon (Goodglass, 1993). These symptoms can be accounted for by a disordered input to the inverse model: no longer can a motor plan be appropriately recovered to achieve a desired perceptual outcome. Furthermore, imaging evidence suggests that the superior temporal cortex receives efferent copies of articulatory motor plans (Paus et al., 1996). This is consistent with the notion of Wernicke's area as the site of the output of the forward model in speech.

With regard to the rostral part of the posterior parietal cortex, lesions to the supramarginal gyrus are often associated with conduction aphasia (Green and Howes, 1978), characterized by relatively preserved comprehension, impaired repetition, and paraphasic and otherwise disordered speech. The relatively preserved comprehension in conduction aphasia might be a consequence of the sparing of superior temporal areas. The deficits in repetition and production can be readily understood in terms of the functional properties of the neural architecture for imitation. An inappropriate pairing of forward and inverse model due to deficits in the "responsibility signal", should result in jumbled speech and failed repetition arising from the inability to update the inverse model on the basis of the forward model. These patients also show *conduite d'approche* (Nadeau, 2003). If the inverse model cannot be updated by the appropriately paired forward model, the only way these patients can correct themselves and finally say a word correctly is by receiving sensory feedback of their speech output.

Given the proposed overlap between regions crucial for imitation and areas important for language, it is notable that many aphasic patients are also apraxic, and apraxia often involves deficits in imitation (Heilman and Valenstein, 2002). The lesion correlates of apraxic deficits are not well understood, but several recent studies have suggested that damage to "mirror" areas is predictive of deficits in conceptual knowledge for actions (Tranel et al., 2003) and action recognition (Saygin et al., 2004). It has been shown, however, that aphasia and apraxia can be dissociated in a minority of patients (Papagno et al., 1993). How do we explain the existence of this double dissociation in a minority of patients? Double dissociations in neuropsychological literature are often considered evidence of independent functional architecture of the cognitive domains that are dissociated after brain damage (see for instance Caramazza, 1986; Caramazza and Badecker, 1991; Caramazza and McCloskey, 1988). However, an alternative interpretation of double dissociations in cognitive neuropsychology is provided by the recent approach of probabilistic mapping of the human brain (Mazziotta et al., 2001). The human brain shows large inter-subject variability in practically all the descriptors used in the neurosciences, including the most obvious ones, that is, macroscopic and microscopic anatomy. Thus, it is possible that the minority of patients showing double dissociations in language and higher order motor functions represent the two tails of a distribution in a population in which the majority of brains have shared neural systems for language and motor control.

To conclude, to better understand the role of Broca's area at the intersection between language and motor control, we believe it is necessary to consider it not as an isolated neural system, but as part of a larger network. By doing so, we can have a better grasp on the flexible functional properties that allow the co-existence of seemingly different behaviors within the same neural circuitry.

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