Medical Hypotheses (2007) **69**, 1165–1168

medical hypotheses

http://intl.elsevierhealth.com/journals/mehy

**Editorial**

# Apraxia, metaphor and mirror neurons

**Summary** Ideomotor apraxia is a cognitive disorder in which the patient loses the ability to accurately perform learned, skilled actions. This is despite normal limb power and coordination. It has long been known that left supramarginal gyrus lesions cause bilateral upper limb apraxia and it was proposed that this area stored a visual-kinaesthetic image of the skilled action, which was translated elsewhere in the brain into the pre-requisite movement formula. We hypothesise that, rather than these two functions occurring separately, both are complementary functions of chains of ''mirror neurons'' within the left inferior parietal lobe. We go on to propose that this neural mechanism in the supramarginal gyrus and its projection zones, which originally evolved to allow the creation of a direct map between vision and movement, was subsequently exapted to allow other sorts of cross-domain mapping and in particular those sorts of abstract re-conceptualisation, such as metaphor, that make mankind unique.


## Introduction

### Apraxia

Apraxia is defined as a cognitive motor disorder in which the patient loses the ability to accurately perform learned, skilled actions [1,2]. This inability does not arise from an impairment of motor power, co-ordination or sensation. In other words, although the patient has both the desire and apparent physical ability to perform the action and can verbally describe its purpose, he is simply unable to adroitly execute it. Although, the word ''apraxia'' is nowadays attached to several different acquired and developmental neurological conditions, we use it in its original sense; that of acquired limb apraxia. What we are most interested at looking at here is the commonest and most archetypal form of that condition: ideomotor apraxia [1–4].

Ideomotor apraxia has been described as ''an impairment in the timing, sequencing and spatial organisation of gestural movements'' [3]. Therefore, when asked to pantomime an activity, especially a transitive activity (using a tool or instrument), a patient with ideomotor apraxia may incorrectly position or move his upper limb with regard to the imagined object. He might also reverse the correct order in a sequence of movements (e.g. locking a door) or incorporate a body part as an object (e.g. use a hand as a hammer), despite being asked not to. Similarly, he might perseveratively repeat a previous pantomime when a new one is requested. The ability of these patients to correctly use the actual objects themselves is also impaired [2–4] although less so than when pantomiming. It's as if pantomiming requires the conjuring up of an especially vivid visual-kinaesthetic ''image'' of the action.

Although the first description of apraxia is credited to Steinthal in 1881 [5], it was Liepmann [3,6,7] who was to do more than anyone else to define it as a neurological entity. He showed that apraxia was primarily a condition that localised to the dominant (usually left) hemisphere. This has been subsequently confirmed [1–4,7] and in particular, lesions of the supramarginal gyrus and underlying white matter of the left parietal lobe have been implicated. Furthermore, although apraxia and Wernicke's aphasia can co-exist, they also

0306-9877/$ - see front matter 
doi:10.1016/j.mehy.2007.05.017

occur independently, showing that apraxia is not due to incomprehension of verbal commands.

Liepmann [4] went on to propose that visual analysis of a complex action lead to the formation of a ''movement formula'', which was essentially a visual image of the action stored in the left parietal cortex. The theory was that this three-dimensional representation of the activity would be translated elsewhere into an ''innervatory pattern'', which was then fed to the relevant motor area [3,7]. Others were to refine this model to suggest this translation took place in the premotor cortex [1—3] However, few well-documented cases of ideomotor apraxia caused by premotor damage have been reported [2].

## Mirror neurons

''Mirror neurons'' were first reported in the premotor cortex of macaque monkeys [8]. These neurons fire, both when a monkey performs a specific action, but also when the monkey simply watches another monkey carry out the same action. This was the first description of a neural mechanism that allowed a ''direct matching between the visual description of an action and its execution'' [9]. There is now evidence, from functional imaging studies, to suggest that mirror neurons not only exist in man but are present in several locations, including the inferior parietal lobule (IPL) [10,11].

We have previously proposed [12,13] that the term ''mirror neuron'' be extended beyond its original meaning of motor maps being mapped on to visual maps. The booba-kiki effect [12] for example, in which the overwhelming majority of people (regardless of language, alphabet or culture) will attach the names kiki to a spiky jagged shape and booba to a gelatinous amoeboid shape, which implies some mirror neuron-like skill may be involved in abstracting the common denominator between sounds and shapes (Fig. 1.) Perhaps any type of cross-domain mapping- fundamental to the brain's functional organisation may rely on such a process.

## The inferior parietal lobule

The mirror neuron population within the inferior parietal lobule of macaques has recently been studied using microelectrodes [14,15]. The majority of the mirror neurons in the macaque IPL discharge not in response to an isolated motor act, but in response to a number of acts combined together to produce an action [14,15]. An example might be a neuron that fires in association to the act of food being grasped and eaten but that does not fire when the food is grasped and placed in a container. Another neuron nearby might perform in exactly the opposite manner. The mirror neurons in the IPL have been described as forming ''prewired chains coding the whole action'' [14].

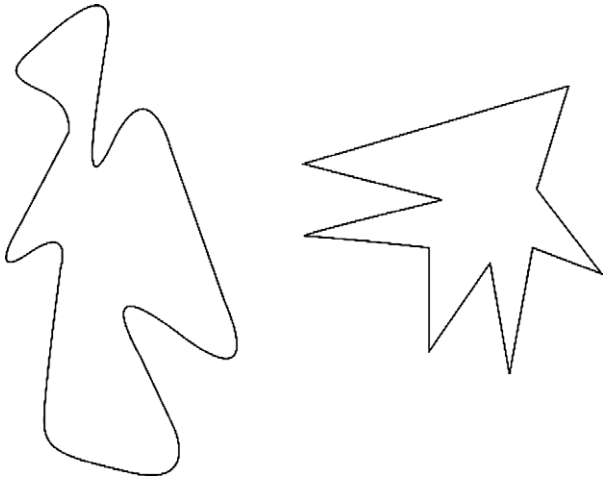

**Figure 1** Which is booba and which kiki?.

The IPL, even more so than the rest of the cortex, underwent an accelerated enlargement in the phylogenetic line leading to the great apes and hominids- splitting into the supramarginal and angular gyri [16]. What could have been the selection pressure driving this? We would suggest that the answer to this lies in the location of the IPL at the junction of the parietal, temporal and occipital lobes. This makes it ideally situated to perform the kinds of cross-modal abstraction (proprioception/hearing/vision) required for motor praxis and mirror neuron-like computations. Indeed the IPL has long been known to play a role both in the processing of visual input [14] and in motor control of the upper-limb [14,16,17].

Prehension must have had a tremendous survival value in our early arboreal primates. When reaching for, say, an oblique branch its brain would have to match the visual input from the retinae with a sequence of entirely different proprioceptive signals being processed in separate brain areas. Despite being superficially dissimilar both inputs do have the common denominator of, in this case, ''obliqueness'' and it is this that the mirror neuron system is able to extract.

## Our hypothesis

Based on the above it is our hypothesis that mirror neurons within the human supramarginal gyrus form interconnected patterns that encode learned,

skilled actions. We postulate that Liepmann's ''movement formula'' and ''innervatory pattern'' both exist and are actually complementary functions of the same networks of mirror neurons within the supramarginal gyrus. It is either direct damage to these movement engrams or their disconnection from the premotor cortex, which is the neurological basis of apraxia.

In our scheme the premotor cortex, although likely involved in further ''fine-tuning'' and imitation of simple gestures (which is often preserved in ideomotor apraxia [4]), is not the area, where the visual representation of the action is converted into a motor innervation. This is because no such conversion occurs, since the neurons responsible are one and the same. Consistent with this view, patients with ideomotor apraxia cannot tell if someone else is performing an act correctly or not.

The nature of the mirror neuron system in the IPL, which seems to predispose to the linking of individual acts to form a movement sequence [14,15] would thus facilitate the coordinated and fluid execution of a complex sequence of actions. Apraxia, conversely, refers to a breakdown in this fluidity — a discord within Luria's ''kinetic melody'' [18].

We go on to suggest that the mirror neuron system within the IPL (originally having evolved for the kind of cross-modal abstraction required for prehension and praxis) was subsequently exapted to also perform the more abstract types of re-conceptualisation, such as metaphor, that lie at the heart of human cognition. In ''Juliet is the sun'', for example, Shakespeare juxtaposes two seemingly unrelated ''concepts'' to reveal the common denominator of radiance and warmth; just as the primate we talked about above extracted the common denominator of ''obliqueness''. Evolution often works this way; taking advantage of structures that originally evolved for other functions. Consequently, any monkey can reach for a peanut but only a human can reach for the stars or even understand what that means [13].

## Testing the hypothesis

Consistent with these ideas, as described by Gerstmann, lesions in the angular gyrus of the left IPL often produce deficits in abstractions, such as arithmetic and mapping the ordinality and cardinality of numbers in spatial coordinates [16,19,20] However, one of us (VSR) recently examined several patients with apraxia from left supramarginal lesions, and found they had a disproportionate difficulty in understanding not only the actions of others, but in correctly interpreting action-based metaphors such as, ''He hit the nail on the head''.

If such abilities depend on the mirror neuron system in the IPL, then one can make two predictions. Firstly, given the reduced mirror neuron function in autistic children [13], they should show symptoms typical of apraxia. This has never been tested although it is known that they are poor at imitation. Conversely, patients with apraxia may have difficulty not only in miming and in interpreting other's actions [1—4] but also, like autistics, in ''theory of mind'' tasks; that is in attributing complex types of intentionality to other people.

## Conclusion

We hypothesise here that normal limb praxis is mediated by the mirror neuron system in the supramarginal gyrus. In addition to proposing a new and potentially testable explanation for the century-old neurological affliction of apraxia, we go on to suggest that this same cross-modal abstraction mechanism, which first evolved in primates to guide accurate prehension, was then exapted to allow such uniquely human abstractions as metaphor and arithmetic.

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