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Neural Basis of Action Understanding: Evidence from Sign Language Aphasia

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Abstract

Background—The neural basis of action understanding is a hotly debated issue. The mirror neuron account holds that motor simulation in fronto-parietal circuits is critical to action understanding including speech comprehension, while others emphasize the ventral stream in the temporal lobe. Evidence from speech strongly supports the ventral stream account, but on the other hand, evidence from manual gesture comprehension (e.g., in limb apraxia) has led to contradictory findings.

Aims—Here we present a lesion analysis of sign language comprehension. Sign language is an excellent model for studying mirror system function in that it bridges the gap between the visual-manual system in which mirror neurons are best characterized and language systems which have represented a theoretical target of mirror neuron research.

Methods & Procedures—Twenty-one life long deaf signers with focal cortical lesions performed two tasks: one involving the comprehension of individual signs and the other involving comprehension of signed sentences (commands). Participants' lesions, as indicated on MRI or CT scans, were mapped onto a template brain to explore the relationship between lesion location and sign comprehension measures.

Outcomes & Results—Single sign comprehension was not significantly affected by left hemisphere damage. Sentence sign comprehension impairments were associated with left temporal-parietal damage. We found that damage to mirror system related regions in the left frontal lobe were not associated with deficits on either of these comprehension tasks.

Conclusions—We conclude that the mirror system is not critically involved in action understanding.

The neural basis of human action understanding is the subject of much debate. One view, inspired by research on mirror neurons – cells in the dorsal stream of the macaque monkey that fire both during action execution and action observation (Rizzolatti & Craighero, 2004) – is that motor simulation provides the basis for understanding the actions of others. The alternative view is that the motor system is not critical for action understanding but instead is dependent on sensory systems in the temporal lobe. This view is grounded in both monkey and human research showing that the superior temporal sulcus contains neurons (or regions in the case of humans) that are selective for biological action detection (Grossman et al., 2000; Grossman, Battelli, & Pascual-Leone, 2005; Grossman, Blake, & Kim, 2004;

Perrett, Mistlin, Harries, & Chitty, 1990; Perrett et al., 1985). This view is also consistent with broader models of the organization of dorsal and ventral processing streams, which hold that the dorsal stream supports motor control whereas the ventral stream supports recognition (Hickok & Hauser, 2010; Hickok & Poeppel, 2007; Milner & Goodale, 1995).

Neuropsychological data is particularly important for assessing these models because it provides evidence regarding causal links between neural systems and cognitive function. Such work has been reported in two domains: (1) manual gesture recognition in apraxic and non-apraxic patients (Buxbaum, Kyle, Grossman, & Coslett, 2007; Nelissen et al., 2010; Pazzaglia, Smania, Corato, & Aglioti, 2008) and (2) speech perception and word comprehension in aphasia (Hickok, Costanzo, Capasso, & Miceli, 2011; Hickok et al., 2008; Rogalsky, Love, Driscoll, Anderson, & Hickok, 2011; Rogalsky, Pitz, Hillis, & Hickok, 2008). Data from aphasia has demonstrated that damage to motor speech systems does not have a substantial effect on speech recognition. Data from the gesture recognition literature, however, is equivocal. One large group study showed a correlation between apraxic deficits and gesture recognition scores as well as a correlation between Broca's area damage and gesture recognition (Pazzaglia et al., 2008). But that same study clearly demonstrates dissociations between execution and recognition as well as dissociations between Broca's area damage and recognition (Hickok, 2009) and questions about the task used in that study have also been raised (Kalenine, Buxbaum, & Coslett, 2010). Moreover, two additional large-scale studies implicated temporal lobe structures in gesture recognition rather than the mirror system¹. One examined the relation between cortical atrophy and gesture discrimination in primary progressive aphasia and reported that cortical thickness in posterior superior temporal and temporal-parietal junction regions were significantly correlated with gesture discrimination performance (Nelissen et al., 2010). Another study examined gesture comprehension in left-hemisphere damaged chronic stroke patients and found that deficits were strongly related to posterior middle temporal regions (Kalenine et al., 2010).

Although neuropsychological studies of gesture execution and recognition are often discussed in relation to mirror neuron-based theory, the relation between the theory predictions and the behaviors assessed in the neuropsychological experiments is not always clear. For example, mirror neurons in macaque monkeys do not respond to pantomimed actions (Gallese, Fadiga, Fogassi, & Rizzolatti, 1996), yet assessments of apraxic patients (a focus of the neuropsychological debate since the earliest mirror neuron papers) often involve the execution (sometimes to imitation) and recognition of pantomimed actions. Even in neuropsychological studies designed specifically to assess action understanding, there has been variation in the type of task used. Pazzaglia et al. used a yes-no discrimination task with real objects on some trials (is the action correct or incorrect?), whereas Kalenine et al. used a matching task in which subjects were asked to match a verbal stimulus (e.g., the spoken or written word "sawing") to the corresponding pantomimed video clip of the action from a set of two sequentially presented clips. The former task has been criticized on grounds that it can be performed without real understanding of the action (e.g., using familiarity judgments) (Kalenine et al., 2010) whereas the latter requires action-based inferences from pantomimes, the processing of linguistic information, and a working memory component. Consequently, the neural basis of action understanding, or even precisely what constitutes action understanding in terms of manual gestures, remains unclear and as a result so does the empirical status of the mirror neuron theory of manual action

¹The typical anatomical description of the proposed human mirror system is a frontal-parietal circuit consisting of ventral premotor cortex (Brodmann areas 44 & 6) and the anterior portion of the inferior parietal lobe (Brodmann area 40) (Rizzolatti & Craighero, 2004).

understanding. (For a recent review of the relevant literature see (Roby-Brami, Hermsdorfer, Roy, & Jacobs, 2012).)

Here we assess the predictions of the mirror neuron theory of action understanding from a unique perspective, that of the signed languages of the Deaf. Signed languages sit at a potentially informative intersection between manual actions and language. It is potentially informative because manual action control and understanding is the domain of monkey mirror neurons whereas language is a prominent domain to which mirror neuron function has been generalized in humans. And the evolutionary bridge between these domains has been claimed to be manual gesture communication (Corballis, 2010; Rizzolatti & Arbib, 1998).

Broca's area has been suggested as a region of particular importance in bridging manual actions and language. Fadiga colleagues recently reviewed evidence for Broca's area involvement not only in language but also in non-linguistic actions (Fadiga, Craighero, & D'Ausilio, 2009) and Rizzolatti and Arbib have argued explicitly that language systems evolved "atop" of manual action systems within Broca's area (Rizzolatti & Arbib, 1998). Given these sorts of arguments, one would expect that if anything is dependent on mirror/motor simulation mechanisms in Broca's area, it would be signed language. Thus, signed language allows one to study this action-language bridge as directly as possible.

Previous functional imaging work has indeed demonstrated that Broca's area is activated during sign language execution and observation (Buchsbaum et al., 2005; Emmorey, Mehta, & Grabowski, 2007; Kassubek, Hickok, & Erhard, 2004; McGuire et al., 1997; Neville et al., 1998; Petitto et al., 2000; San Jose-Robertson, Corina, Ackerman, Guillemin, & Braun, 2004) and a case report of a deaf native signer of American Sign Language with a lesion largely restricted to Broca's area provided neuropsychological evidence for a causal role of this region in sign production (Hickok, Kritchewsky, Bellugi, & Klima, 1996). However, the question of whether damage to Broca's area causes receptive sign language deficits has not previously been addressed beyond the observation that left frontal lesions have been associated with Broca's aphasia for sign language, which by definition involved relatively preserved comprehension despite non-fluent sign production (D. Corina, 1998; Poizner, Klima, & Bellugi, 1987).

Here we report neuropsychological data that bridges the gesture recognition and language perception domains of investigation. We studied the sign language comprehension ability of a group of deaf American Sign Language (ASL) signers who had suffered unilateral focal brain injury to assess whether damage to the motor system, Broca's area in particular, is predictive of sign language comprehension deficits as the motor simulation account would predict.

Method

Participants

Thirty-four Deaf, life-long, right-handed ASL signers with focal brain injury comprised our database from which to sample. Subjects were excluded from the present study if they had bilateral lesions, if the lesions were unmappable due to quality of the scan, or if the only available scan was acquired in acute stages of the brain injury. Twenty-one cases remained after applying these exclusion criteria resulting in a study sample of 11 left-hemisphere damaged signers and 10 right-hemisphere damaged signers. All had some experience with written English, but this was not formally assessed. Table 1 presents relevant biographical information.

Procedure and Tasks

Neuropsychological testing was carried out in a quiet room by a native Deaf signer of ASL. Tests included a range of language and spatial cognitive measures administered as part of our basic neuropsychological assessment. The language measures included an ASL-adapted version of the Boston Diagnostic Aphasia Exam (BDAE; Goodglass & Kaplan, 1976). Sessions were videotaped for later scoring by a native Deaf signer of ASL.

For the present study we focus on two comprehension subtests of our ASL- adapted version of the BDAE, “sign discrimination” and “commands”. The sign discrimination test consists of 36 items sampling from several categories including objects, actions, letters, colors, shapes, and numbers (six items each). A single sign is presented to the subject and the subject is asked to point to the picture that matches the sign from an array of 18 items. The pictures are black and white line drawings. The first half of the trials use one 18-item picture array, and the second half use a second 18-item array. Within each array, items are grouped by category, as are the signs presented. Correct answers receive one point each. The commands test requires subjects to follow eight signed commands of increasing difficulty, ranging from one-step commands (“point to the ceiling”) to multi-step commands (“put the pencil on the paper, then put it back”). Two points were given for a correct response, one point for a partially correct response, and zero points for a completely incorrect response, or no response.

Brain image analysis

Neuroanatomical analyses were based on clinical scans obtained for each subject with their permission. T1-weighted MRI images were used if available ($n=10$); if not, CT scans ($n=7$), and T2-weighted MRI images ($n=4$) were used. Lesion mapping was performed according to MAP-3 lesion analysis methods, using Brainvox software (Frank, Damasio, & Grabowski, 1997). This method entails a transfer of the lesion, as presented on the clinical scans, to a common space in a template brain. To facilitate reliable lesion transfer with regards to anatomical landmarks, all major sulci visible on the lesioned brain's images were identified in both the lesion brain and the template brain. The template brain's slices were then resliced to match the orientation and thickness of the slices in the native image space of the lesioned brain. The area of lesion visible on each native space slice was manually mapped to the corresponding slice in the template brain, respecting identifiable anatomical landmarks (H. Damasio & Damasio, 2003). Overlap and subtraction maps of lesioned voxels across subgroups of subjects were generated using Brainvox software. Figure 1 displays the distribution of lesions on the template brain for our patient sample.

We then used two approaches to analysis, an anatomical approach and a behavioral approach. For the anatomical approach we identified those cases with lesions involving frontal motor-related structures but without substantial temporal lobe involvement ($N=6$) and compared these with cases that had involvement of temporal lobe structures ($N=3$). The three cases with temporal lobe lesions were a variable group: one had a small lesion centered on the mid-superior temporal sulcus region, one had a moderate temporal parietal lesion, and one had a larger fronto-temporo-parietal lesion. Comprehension performance between the groups on the two tasks was then compared, with particular emphasis on the performance of the cases with frontal involvement.

In the behavioral approach we partitioned the left hemisphere subjects into those cases who performed on the commands test within 1 s.d. ($N=4$) of the mean of the control (RHD) group and those cases who performed more than 2 s.d. below ($N=6$) the mean of the control group. The lesion distribution of the spared (unimpaired) group was then subtracted from the impaired group to identify regions associated with sign language comprehension.

impairment. A similar approach was not used for sign discrimination performance due to the near ceiling performance of the left hemisphere patients in this task.

Results

Anatomical approach

Lesion overlap maps for those subjects with frontal lobe involvement are presented in Figure 2. A majority of these lesions overlap in the posterior inferior frontal lobe (roughly BA 44/6) and anterior insula. Thus regions thought to be part of the human mirror system were substantially affected¹. Average performance (number correct, maximum = 36) on the sign discrimination test for the three groups of patients were: RHD signers = 34.55, left frontal lobe damaged signers = 35.25, left temporal lobe damaged signers = 28.67. For the signed commands test average performance (number correct, maximum = 16) for the three groups of patients were: RHD signers = 12.3, left frontal lobe damaged signers = 10.67, left temporal lobe damaged signers = 3.3 (Figure 3).

Behavioral approach

A subtraction of the lesions in the impaired minus unimpaired command comprehension groups revealed a relatively wide region of maximal difference centered primarily on left mid to posterior peri-Sylvian cortex (Figure 4). Or put differently, it is not the case that the lesions in patients with sign command comprehension deficits are concentrated in frontal motor-related regions.

Discussion

We assessed the neural basis of manual gesture comprehension through the window of signed language. Deaf, life-long signers with focal left or right hemisphere brain injury were assessed on their ability to comprehend signs and signed sentences. Our focus was to determine whether damage to the “human mirror system” (the Broca’s area portion in particular) caused deficits in the ability to understand manual gestures in the form of natural sign language. We found no evidence to support this possibility. Signers with lesions involving Broca’s area and surrounding areas were effectively at ceiling in their ability to comprehend individual signs and performed comparably to right hemisphere damaged signers on signed sentence instructions (the commands test). Participants with left temporal lobe lesions, on the other hand, showed evidence of comprehension impairments, primarily on the command test where the distribution of scores for the temporal lobe group were largely non-overlapping with those for the left frontal or right hemisphere lesion groups (although the temporal lobe results must remain tentative given the low sample size). A comparison of the lesions associated with impaired versus spared comprehension performance on the commands test did not implicate left frontal regions.

This finding largely supports a previous lesion-based study of the neural correlates of sign comprehension in Deaf, brain damaged signers which found that comprehension of individual signs was largely preserved following left hemisphere lesions and that comprehension deficits on sentence-level signed stimuli were primarily associated with temporal lobe lesions (Hickok, Love-Geffen, & Klima, 2002). It is also consistent with case reports of signers with Broca’s aphasia-like deficits including non-fluent, effortful sign production but with preserved sign comprehension (Poizner, Klima, & Bellugi, 1987). In fact, one of the present cases (LHD03) had a large left frontal lesion associated with a severe non-fluent aphasia (Figure 5). This patient’s comprehension scores were the best scores in the sample of left hemisphere damaged signers (sign discrimination = 36/36, sign command test = 13/16).

The subtraction map comparing impaired versus spared sign comprehension did not identify Broca's area or any frontal lobe region as a site of substantial overlap in those cases with impairments. Instead, lesions in the inferior parietal and superior temporal lobe of the left hemisphere showed the most lesion overlap in signers who were impaired on the comprehension tasks. Based on this result, it is unclear whether the critical region is in the temporal lobe, the parietal lobe, or both and consequently, we cannot rule out the possibility that the parietal lobe portion of the proposed mirror system plays a role in sign comprehension (MacSweeney, Capek, Campbell, & Woll, 2008). Precisely the same ambiguity exists in the literature on non-linguistic gesture recognition, as noted in the introduction. Resolution will have to await future research.

It is worth pointing out that, despite some apparent similarities in the overall pattern of lesions implicated in signed language and non-linguistic gestural processing, these two abilities are quite dissociable. As Corina and Knapp note in their recent review, there have now been several reports of sometimes severely impaired ability to sign with the preserved ability to gesture (including pantomime) non-linguistically (D. P. Corina & Knapp, 2006). The authors go on to point out that this complicates the picture regarding a single gesture production-comprehension mirror network, if one does exist.

More broadly, the present finding is consistent with the literature on spoken language aphasia. It has long been established that lesions to left frontal regions cause speech production deficits that are far more severe than comprehension deficits, a defining feature of Broca's aphasia, which is associated with left frontal lesions (Damasio, 1992; Dronkers & Baldo, 2009; Hillis, 2007). Comprehension deficits do occur following left frontal lesions, but these are typically limited to syntactically complex sentences, and have little effect on word or simple phrase level comprehension (Caramazza & Zurif, 1976; Dronkers, Wilkins, Van Valin, Redfern, & Jaeger, 2004; Hickok et al., 2011; Rogalsky et al., 2011).

Although research on manual gesture recognition in the hearing population is equivocal, as noted in the introduction, the present findings are consistent with a recent study of 43 left hemisphere damaged individuals for which there was not an association between damage in the inferior frontal gyrus and the ability to recognize manual gestures. This was true when recognition required discriminating between the target and a semantic foil (e.g. sawing versus hammering motion) as well as between the target and a "formal" or "spatial" foil (sawing versus the same arm motion with the wrong hand configuration) (Kalenine et al., 2010). These authors similarly concluded that the inferior frontal gyrus is not particularly involved in action recognition, and that mirror neurons are not necessary for action understanding (Buxbaum & Kalenine, 2010).

Taken together, the weight of the evidence from speech, manual gesture comprehension, and sign language comprehension argues rather strongly against the view that the mirror system is critically involved in action understanding.

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Figure 1. Lesion distribution on the template brain for our patient sample. Warmer colors indicate areas of greater overlap. For reference, the inferior frontal sulcus is marked in cyan, and the central sulcus is indicated in red.

Figure 2.

Overlap map of cases with left frontal lobe damage (i.e. damage to the proposed human mirror system), but no substantial temporal lobe involvement. The crosshairs are focused on the maximum area of overlap. The behavioral performance of this left frontal damage group is compared to the other sub groups in Figure 3.

Figure 3. Performance on the (A) single sign and (B) sign command comprehension tests of the left frontal, left temporal, and right hemisphere damaged groups. Each white circle represents an individual participant's performance.

Figure 4. Subtraction map of lesions of the participants with impaired minus unimpaired performance on the commands test. A 3-D rendered surface view and axial slices are shown. Warmer colors and black indicate more impaired participants have lesion in that area.

Figure 5.
3-D rendered surface view and orthogonal slices of participant LHD03's large left frontal lesion as mapped onto the template brain. This lesion includes frontal regions proposed to be part of the human mirror system.

Table 1

Biographical data of participants.

	Age of Sign Exposure	Age of Onset of Deafness	Sex	Handedness	Age at Testing	Approximate Lesion Location	Lesion Etiology
Left Hemisphere Lesioned:							
LHD01 *	6	5	m	r	81	frontal-par	Ischemic Infarct
LHD03 *	0	0	f	r	37	frontal	Ischemic Infarct
LHD05 *	13	0	m	r	45	temp-par	Hematoma
LHD06 *	0	0	m	r	77	frontal-temp-par	Ischemic Infarct
LHD08	6	2	f	r	64	medial occ	Ischemic Infarct
LHD09 *	7	<1	m	r	29	frontal-par	Hematoma **
LHD10 *	0	2	f	r	79	inf-post frontal	Ischemic Infarct
LHD11 *	9	<1	f	r	73	frontal-par	Ischemic Infarct
LHD12 *	11	0	f	r	79	frontal-temp-par	Ischemic Infarct
LHD13	5	0	f	r	37	sup temp	Hematoma **
LHD14	9	<1	f	r	81	insula	Ischemic Infarct
Right Hemisphere Lesioned:							
RHD01 *	12	0	f	r	71	front-temp-par	Ischemic Infarct
RHD02 *	9	5	m	r	82	temp-par	Ischemic Infarct
RHD03 *	5	0	m	r	60	front-temp-par	Ischemic Infarct
RHD04 *	0	0	f	r	61	sup front-par	Tumor **
RHD05 *	0	n/a	f	r	38	sup par-occ	Hematoma **
RHD06 *	0	0	m	r	74	front-temp-par	Ischemic Infarct
RHD07	11	2	f	r	78	frontal-par	Ischemic Infarct
RHD08 *	7	<1	m	r	74	frontal-temp-par	Ischemic Infarct
RHD09	6	3	f	r	83	temp-par	Ischemic Infarct
RHD10 *	0	0	f	r	78	temp-par-occ	Ischemic Infarct

All ages are in years, par = parietal, temp = temporal, occ = occipital, inf = inferior, post = posterior, sup = superior.

* indicates participation in Hickok, et al. 2002.

** indicates surgical intervention.