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Are mirror neurons the basis of speech perception? Evidence from five cases with damage to the purported human mirror system

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Abstract

The discovery of mirror neurons in macaque has led to a resurrection of motor theories of speech perception. Although the majority of lesion and functional imaging studies have associated perception with the temporal lobes, it has also been proposed that the ‘human mirror system’, which prominently includes Broca’s area, is the neurophysiological substrate of speech perception. Although numerous studies have demonstrated a tight link between sensory and motor speech processes, few have directly assessed the critical prediction of mirror neuron theories of speech perception, namely that damage to the human mirror system should cause severe deficits in speech perception. The present study measured speech perception abilities of patients with lesions involving motor regions in the left posterior frontal lobe and/or inferior parietal lobule (i.e., the proposed human ‘mirror system’). Performance was at or near ceiling in patients with fronto-parietal lesions. It is only when the lesion encroaches on auditory regions in the temporal lobe that perceptual deficits are evident. This suggests that ‘mirror system’ damage does not disrupt speech perception, but rather that auditory systems are the primary substrate for speech perception.

INTRODUCTION

Mirror neurons and speech perception have been intimately connected since the discovery of the former in the early 1990s. In the earliest papers describing mirror neurons as cells in the

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macaque motor system that respond both during the perception and execution of action (di Pellegrino, Fadiga, Fogassi, Gallese, & Rizzolatti, 1992; Gallese, Fadiga, Fogassi, & Rizzolatti, 1996), there was speculation about the possible role of these cells in speech perception. For example, Gallese et al. (1996) write, 'neurons with properties similar to that of monkey 'mirror neurons', but coding phonetic gestures, should exist in human Broca's area and should represent the neurophysiological substrate for speech perception' (p. 607). Indeed, the functional interpretation of mirror neurons as cells that form the basis of action understanding (Rizzolatti & Craighero, 2004) appears to have been inspired¹ by Alvin Liberman and colleagues' motor theory of speech perception (Liberman, Cooper, Shankweiler, & Studdert-Kennedy, 1967; Liberman & Mattingly, 1985), which holds that the objects of speech perception are not sounds but articulatory gestures. By the time mirror neurons were discovered, the motor theory of speech perception was all but abandoned by speech scientists (Galantucci, Fowler, & Turvey, 2006), but in the years since, the motor theory has enjoyed an impressive resurgence of interest which has continued to the present: 'Liberman's intuition ... that the ultimate constituents of speech are not sounds but articulatory gestures ... seems to us a good way to consider speech processing in the more general context of action recognition' (Fadiga & Craighero, 2006, p. 489).

In some ways, the existence of a well-known motor theory of perception lent indirect support for the claim in monkeys that mirror neurons were the basis of action understanding. Because of this tight theoretical connection between mirror neurons and speech perception, and because of the extensive behavioral, neurological, and neurophysiological literature on speech, the role of the human mirror system in speech perception has emerged as an ideal test case for the more general claim regarding the functional role of mirror neurons.

Accordingly there has been a number of new studies published in the last several years which have found that motor-related structures are physiologically active during speech perception (Watkins, Strafella, & Paus, 2003; Wilson, Saygin, Sereno, & Iacoboni, 2004; Wilson & Iacoboni, 2006) and that stimulation of motor cortex can produce small modulations in behavioral responses to speech sounds (D'Ausilio et al., 2009; Meister, Wilson, Deblieck, Wu, & Iacoboni, 2007). But these experiments fail to address the critical prediction of the mirror neuron/motor theory hypothesis regarding speech perception, that lesions of the mirror system/motor speech system should cause concomitant deficits in speech perception.

It is well-known that patients with Broca's aphasia often have very large lesions involving the left motor speech system and can have very severe speech production deficits (Naeser et al., 1989), yet are quite capable of processing speech sounds as evidenced by their preserved word-level comprehension (Damasio, 1992; Goodglass, 1993; Goodglass & Kaplan, 1983; Hillis, 2007). Thus, to a first approximation, the existence of Broca's aphasia, in which there is a dissociation between speech production (impaired) and speech comprehension (preserved), constitutes evidence against the motor theory of speech perception (Hickok, 2009b, 2009c; Lotto, Hickok, & Holt, 2009). Previous studies, however, presented group-based data and do not report detailed lesion information, relying instead on clinical diagnosis of Broca's aphasia to infer lesion location.

Here we present data from five cases with lesions involving the human mirror system (Figure 1). Patients were assessed on four word comprehension tests and four speech discrimination tests. In the comprehension tests, subjects were presented with a spoken word and asked to point to the matching picture out of an array of four. In different variants of the

¹Communicated by one of the co-discoverers of mirror neurons, Luciano Fadiga, in a platform lecture at the 2009 Neurobiology of Language Conference, Chicago, IL.

test, the distracter pictures were either all phonemically related, all semantically related, all unrelated, or contained a mixture of distracter types. In the discrimination tasks, subjects heard pairs of syllables and were asked to decide whether the two items were the same or not. The four versions of this test were as follows. One test used real words drawn from the comprehension test items. Another used non-words that were created by changing the vowel of the words used in the words discrimination test. These two tests were matched for onset and offsets of the speech stimuli but were not matched for phonotactic probability (how common a given sequence of sounds is) or neighborhood density (how many similar sounding words there are to a stimulus item). Therefore another word discrimination and non-word discrimination test was created in which words and non-words were matched on these variables.

METHODS

Participants

Five patients (three female) participated in this study (Tables 1 and 2). These patients are a subset of the approximately 60 patients thus far tested in an ongoing large-scale aphasia project directed by G. Hickok. The five patients reported here were the only patients from this sample that fit our inclusion criteria, namely the presence of a brain lesion involving either Broca's region, the inferior parietal lobule, or both. Thus, patients were neither included nor excluded on the basis of performance on the speech perception tasks described below, but rather were selected based on lesion location. Informed consent was obtained from each participant prior to participation in the study, and all procedures were approved by the Institutional Review Boards of UC Irvine, UC San Diego, University of Southern California, and University of Iowa.

Three of these patients had, in addition, damage involving the superior temporal lobe; two did not (Figure 1). Two subjects were non-fluent (1978 & LHDST), one severely so (LHDST). The following is a sample transcription from LHDST describing the 'Cookie Theft' picture from the Boston Diagnostic Aphasia Examination (Goodglass, Kaplan, & Baressi, 2001).

EXP: What can you tell me about this picture?

SUB: Yeah ... yeah

EXP: Do you know what that is?

SUB: Yeah

EXP: If I give you the first sound would that help you? Wa--

SUB: Aws

EXP: Wa—, wa—ter.

SUB: Water

EXP: stoo – [pointing to stool]

SUB: oo—ul, ul

EXP: Stoo—I, good!

SUB: Oh uh yeah.

...

EXP: What about over here? [points to dishes]

SUB: Mm

EXP: Take your time.

SUB: Awgay [Okay] ... ases

EXP: Di—

SUB: Ases

EXP: Dishes. Great!

Lesion mapping

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 brain to a common space in a template brain. To facilitate reliable lesion transfer, with regards to anatomical landmarks, all major sulci of the lesion brain 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 (Damasio & Damasio, 2003). (Note: structural MRIs were used for mapping purposes for LHDST, 1978, 2435, and 2969; 3025's lesion was mapped via a CT scan due to the presence of a metal clip.)

Materials

Patients were tested on four different word comprehension tests and four different syllable discrimination tasks.

The four word comprehension tests used a four-alternative forced choice word-to-picture paradigm. In each test, subjects were presented with a visual display via a computer screen that included depictions of four objects. They then heard a spoken word via headphones and were asked to point to the matching picture. The tests differed in terms of the nature of the distractor pictures (Figure 2). The phonemic foil test had all phonemic distractors; the semantic foil test had all semantic distractors; the unrelated foil test had distractors that were neither phonemically nor semantically related; and the mixed foil test had one phonemic, one semantic, and one unrelated distractor. Each test included 20 trials. Sixteen of the 20 trials in the phonemic foil test had at least one phonemic foil that represented a minimal pair to the target (eight minimal pairs were place of articulator contrasts, four were voicing contrasts, two were manner contrasts, and two trials had both place and voicing minimal pair distractors). Ten of the 20 trials in the mixed foil test had a phonemic foil that represented a minimal pair to the target, four minimal pairs were place of articulation contrasts, four were voicing contrasts, and two were manner contrasts.

The four syllable discrimination tests each involved the presentation of pairs of syllables via headphones. Subjects indicated whether the two syllables were the same or different. In the first test the syllables were minimal phonemic pair words that were used in the

comprehension tests (the same sound files were used) as well as some additional minimal pair words that were not included in the comprehension tests. There were 16 word pairs all together which were presented in four arrangements: A-B, B-A, A-A, and B-B. Thus there were 64 trials in total, half were same trials and half were different trials. Inter-stimulus interval was 1 s. Nine minimal pairs differed by place of articulation, seven by voicing, and one by manner. The second discrimination test involved non-words. The vowel of each word in the above word discrimination test was changed to yield a set of non-words. All of the details were identical to the word discrimination task. Because words and non-words in the above tasks were not controlled for neighborhood density or phonotactic probability, we administered an additional word discrimination test and an additional non-word discrimination test where the word and non-word items were matched for these factors. These tests comprised 40 trials (10 minimal pairs presented in 4 arrangements, A-B, B-A, A-A, B-B); five pairs differed only by place feature, five by voice feature. Signal detection methods were used for analysis of the discrimination tasks. In particular, we calculated an *a*-prime statistic to characterize how well subjects could discriminate same from different pairs (Swets, 1964). Similar to *d*-prime, *a*-prime corrects for response bias but yields values that approximate proportion correct allowing for more straightforward interpretation than *d*-prime.

Items within a test were presented in a fixed random order and the tests themselves were presented in a non-fixed pseudorandom order.

RESULTS

Overall performance

Overall performance on the word comprehension tests was at 97% correct ($SD = 4.56$) across the phonemic, semantic, and mixed foil versions (performance on the unrelated foil version of the word comprehension test was at ceiling across all subjects (no errors) and thus will not be considered further). Overall performance on the word discrimination tests was also quite good, with an *a*-prime of .94 ($SD = 0.08$) (*a*-prime is a measure of discrimination that is corrected for response bias; chance level performance = 0.5, maximum score = 1.0). Performance on the non-word discrimination tests was somewhat lower, with an *a*-prime of 0.898 ($SD = 0.079$). Performance on the neighborhood density and phonotactic probability matched versus non-matched discrimination tests did not differ (word matched vs. non-matched: 94.1 vs. 94.2%, respectively; non-word matched vs. non-matched: 90.0% for each) and so only the discrimination tests that involved stimuli that were used in the comprehension tests (and their corresponding non-words) will be considered further.

Individual subject performance

Figure 3 depicts performance on three comprehension tests and the corresponding word and nonword discrimination tests for each subject. Note that the two subjects with only fronto-parietal lesions scored 100% on the comprehension tests and 95% or better on both the word and non-word discrimination tests. Subjects who had fronto-parietal *and* temporal lobe involvement performed somewhat worse, (but still quite well) on the word comprehension (average = 95%) and the word discrimination tests (average *a*-prime = .91), whereas performance began to fall off on the non-word discrimination test (average *a*-prime = .86).

Some of the comprehension tests did not involve phonemic foils and some of the trials on the tests that included phonemic foils did not include minimal phonemic pair distractors. To provide the strongest test of the phonemic perception abilities of our subjects on our comprehension tests we analyzed only those trials on the phonemic and mixed foil comprehension subtests that included minimal phonemic pair distractors ($n = 25$).

Performance is plotted in Figure 4 for each subject along with their scores on the word and non-word discrimination tests. Note again that the subjects with their temporal lobe spared performed at ceiling on the comprehension and word discrimination tests; one of these two subjects was also at ceiling on the non-word discrimination test while the other performed slightly worse (94.8% correct). Two of the three subjects with temporal lobe involvement performed better than 90% correct (92 and 100%) even with minimal phonemic pair foils, and both performed at better than 96% accuracy ($a\text{-prime} > .96$) on the word discrimination test; non-word discrimination was somewhat depressed however at 88 and 89% accuracy ($a\text{-prime} = .88$ and $.89$). One subject with temporal lobe involvement, subject 2435, had the lowest scores which hovered just under 80% correct for all three tests. Interestingly, this subject's lesion entirely spared frontal cortex, again considered to be the 'core' of the mirror system, and involved posterior parietal and superior temporal regions. Figure 5 shows the average performance of the patients with frontal lesions across the minimal pair comprehension tests and the discrimination tests that used the same words.

DISCUSSION

To summarize the present findings, patients with damage affecting motor cortex (the presumed human mirror system) exhibited high-levels of performance (at or near ceiling) on all receptive speech tasks. This contradicts the prediction of mirror-neuron inspired motor theories of speech perception and supports previous observations from Broca's aphasia indicating that the motor speech system is not necessary for speech perception. Thus, a strong version of the motor theory of speech perception is not viable. In those patients with lesions that extended into the temporal lobe, performance worsened somewhat, although remained well-above chance, even in the patient with complete destruction of the left superior temporal lobe indicating that speech perception is not strongly left dominant. This general pattern of results, in conjunction with previous work, is consistent with the view that (i) temporal lobe structures, rather than motor structures, are the primary substrate for speech perception, and (ii) this system is bilaterally organized (Hickok & Poeppel, 2000, 2004, 2007). Both of these points are discussed more below.

Previous studies indicated that patients with Broca's aphasia, and therefore frontal lesions, were impaired on syllable discrimination tasks. For example, Baker, Blumstein, and Goodglass (1981) report that Broca's aphasics performed at approximately 86% correct on such tasks. While still well above chance, this level of performance appears worse than what we found in our fronto-parietal patients. However, this apparent difference is likely a measurement artifact. Previous studies typically used overall percent correct as their dependent measure. But percent correct is potentially biased in that it reflects both the subject's perceptual discrimination ability and his or her decision criterion (response bias). Signal detection methods (such as calculating $a\text{-prime}$) can correct for response bias to yield a more veridical measure of discriminability. Baker et al. presented their data in sufficient detail to allow calculation of $a\text{-prime}$, which yielded a value of $.96$, indicating that the relatively poor performance of Broca's aphasics in this study was a consequence of response bias rather than perceptual ability. It is worth noting in this context that Broca's region has been implicated in response selection deficits (January, Trueswell, & Thompson-Schill, 2009; MacDonald, Cohen, Stenger, & Carter, 2000; Novick, Trueswell, & Thompson-Schill, 2005). Thus, previous studies may have over-estimated the degree of impairment through the use of a biased dependent measure. See Hickok (2010) for additional discussion and re-evaluation of previous studies.

Some recent influential studies have reported that transcranial magnetic stimulation applied to motor regions in the left frontal lobe of healthy adults can influence speech perception. In one experiment, stimulation of premotor cortex resulted in a decline in subjects' ability to

identify which of three syllables were presented against a noisy background (Meister et al., 2007) and in another, stimulation of lip-related motor cortex facilitated response time to lip-related speech sounds (ba, pa) but not tongue related sounds (da, ta) (all sounds presented in noise), whereas stimulation of tongue-related motor cortex produced the reverse effect (D'Ausilio et al., 2009). Does this not suggest a role for the motor system in speech perception? Possibly, but if there is a demonstrable role for the motor system in speech perception it (i) is only a modulatory role as lesion work shows that the motor system is not necessary for speech perception, (ii) only comes into play under degraded listening conditions, and (iii) has only been demonstrated when subjects are asked to perform a non-natural speech task such as identify meaningless syllables. The latter issue is important because under normal speech processing conditions (e.g., listening for comprehension), we do not perceive speech sounds, we perceive words. Asking subjects to consciously attend to speech sounds recruits additional cognitive mechanisms, such as phonological short-term memory, that are not typically used in ecologically valid situations (Hickok & Poeppel, 2000, 2004, 2007). It may be that the motor system is involved in these meta-linguistic task-specific aspects of performance rather than fundamental processes.

Finally, it is worth noting other sources of evidence against motor theories of speech perception. One is acute deactivation of the entire left hemisphere in patients undergoing sodium amobarbital (Wada) procedures. Anesthesia of the left hemisphere produces complete speech arrest yet leaves speech sound perception proportionately intact (phonemic error rate < 10% in a four-alternative forced choice word-to-picture matching task (Hickok et al., 2008). Importantly, this pattern holds even when fine phonetic discrimination is required for successful comprehension (i.e., when phonemic distractors are included in the choice arrays) (Hickok et al., 2008). This finding, that complete functional disruption of the motor speech system co-occurs with near normal speech comprehension with the left hemisphere anesthetized, suggests that our current results found with left hemisphere damage cannot merely be explained by compensation by the right hemisphere. Rather, our results demonstrate that the motor speech system is not critical for phonemic perception.

It is also worth emphasizing that deactivation of the entire left hemisphere affects not only motor speech areas but also auditory regions. The relative preservation of auditory speech comprehension even when left auditory regions are deactivated has been used as evidence for bilateral auditory cortex organization of speech perception (Hickok & Poeppel, 2000, 2004, 2007; Hickok et al., 2008; Rogalsky, Pitz, Hillis, & Hickok, 2008). Could one similarly argue that the motor speech system is also bilaterally organized? It appears not as deactivation of the left hemisphere function during Wada procedures produces complete functional disruption of the motor speech system (patients are mute). Stimulation of Broca's region is also known to produce speech arrest (Penfield & Rasmussen, 1949). And as noted above, left hemisphere stroke can produce chronic severe speech production deficits. The motor speech system, unlike the auditory speech system, appears to be strongly left dominant. One may still wish to argue that there is a silent motor speech system in the right hemisphere that can support speech perception even when it cannot support production. But this claim is undermined by bilateral lesions to Broca's area (Levine & Mohr, 1979, see case 3) and bilateral damage to the anterior operculum which causes anarthria, that is, loss of voluntary muscle control of speech (Weller, 1993) yet in none of these cases does one find receptive speech deficits.

In sum, previous evidence from aphasia and other neuropsychological sources, together with the present study showing detailed lesion maps and using signal detection methods demonstrates very clearly that the human mirror system/motor speech system is not critical for speech perception.

REFERENCES

- Baker E, Blumstein SE, Goodglass H. Interaction between phonological and semantic factors in auditory comprehension. *Neuropsychologia*. 1981; 19(1):1–15. [PubMed: 7231654]
- Damasio AR. Aphasia. *New England Journal of Medicine*. 1992; 326:531–539. [PubMed: 1732792]
- Damasio, H.; Damasio, AR. The lesion method in behavioral neurology and neuropsychology. In: Feinberg, TE.; Farah, MJ., editors. *Behavioral neurology and neuropsychology*. 2nd ed. New York: McGraw Hill; 2003. p. 71-83.
- D'Ausilio A, Pulvermuller F, Salmas P, Bufalari I, Begliomini C, Fadiga L. The motor somatotopy of speech perception. *Current Biology*. 2009; 19(5):381–385. [PubMed: 19217297]
- di Pellegrino G, Fadiga L, Fogassi L, Gallese V, Rizzolatti G. Understanding motor events: A neurophysiological study. *Experimental Brain Research*. 1992; 91(1):176–180.
- Fadiga L, Craighero L. Hand actions and speech representation in Broca's area. *Cortex*. 2006; 42(4):486–490. [PubMed: 16881255]
- Frank RJ, Damasio H, Grabowski TJ. Brainvox: An interactive, multimodal visualization and analysis system for neuroanatomical imaging. *Neuroimage*. 1997; 5(1):13–30. [PubMed: 9038281]
- Galantucci B, Fowler CA, Turvey MT. The motor theory of speech perception reviewed. *Psychonomics Bulletin Review*. 2006; 13(3):361–377.
- Gallese V, Fadiga L, Fogassi L, Rizzolatti G. Action recognition in the premotor cortex. *Brain*. 1996; 119(Pt 2):593–609. [PubMed: 8800951]
- Goodglass, H. *Understanding aphasia*. San Diego, CA: Academic Press; 1993.
- Goodglass, H.; Kaplan, E. *The assessment of aphasia and related disorders*. 2nd ed. Philadelphia: Lea & Febiger; 1983.
- Goodglass, H.; Kaplan, E.; Barresi, B. *The assessment of aphasia and related disorders*. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2001.
- Hickok G. Eight problems for the mirror neuron theory of action understanding in monkeys and humans. *Journal of Cognitive Neuroscience*. 2009a; 21(7):1229–1243. [PubMed: 19199415]
- Hickok, G. Speech perception does not rely on motor cortex. 2009b. Available: [http://www.cell.com/current-biology/comments/S0960-9822\(09\)00556-9](http://www.cell.com/current-biology/comments/S0960-9822(09)00556-9)
- Hickok G. The role of mirror neurons in speech and language processing. *Brain and Language*. 2009c Advance online publication.
- Hickok G. The role of mirror neurons in speech perception and action word semantics. *Language and Cognitive Processes*. 2010; 25(6):749–776.
- Hickok G, Poeppel D. Towards a functional neuroanatomy of speech perception. *Trends in Cognitive Sciences*. 2000; 4:131–138. [PubMed: 10740277]
- Hickok G, Poeppel D. Dorsal and ventral streams: A framework for understanding aspects of the functional anatomy of language. *Cognition*. 2004; 92:67–99. [PubMed: 15037127]
- Hickok G, Poeppel D. The cortical organization of speech processing. *Nature Reviews Neuroscience*. 2007; 8(5):393–402.
- Hickok G, Okada K, Barr W, Pa J, Rogalsky C, Donnelly K, Barde L, Grant A. Bilateral capacity for speech sound processing in auditory comprehension: Evidence from Wada procedures. *Brain and Language*. 2008; 107(3):179–184. [PubMed: 18976806]
- Hillis AE. Aphasia: progress in the last quarter of a century. *Neurology*. 2007; 69(2):200–213. [PubMed: 17620554]
- January D, Trueswell JC, Thompson-Schill SL. Co-localization of stroop and syntactic ambiguity resolution in Broca's area: Implications for the neural basis of sentence processing. *Journal of Cognitive Neuroscience*. 2009; 21(12):2434–2444. [PubMed: 19199402]
- Levine DN, Mohr JP. Language after bilateral cerebral infarctions: Role of the minor hemisphere in speech. *Neurology*. 1979; 29(7):927–938. [PubMed: 572942]
- Lieberman AM, Mattingly IG. The motor theory of speech perception revised. *Cognition*. 1985; 21:1–36. [PubMed: 4075760]
- Lieberman AM, Cooper FS, Shankweiler DP, Studdert-Kennedy M. Perception of the speech code. *Psychological Review*. 1967; 74(6):431–461. [PubMed: 4170865]

- Lotto AJ, Hickok GS, Holt LL. Reflections on mirror neurons and speech perception. *Trends in Cognitive Sciences*. 2009; 13(3):110–114. [PubMed: 19223222]
- MacDonald AW 3rd, Cohen JD, Stenger VA, Carter CS. Dissociating the role of the dorsolateral prefrontal and anterior cingulate cortex in cognitive control. *Science*. 2000; 288(5472):1835–1838. [PubMed: 10846167]
- Meister IG, Wilson SM, Deblieck C, Wu AD, Iacoboni M. The essential role of premotor cortex in speech perception. *Current Biology*. 2007; 17(19):1692–1696. [PubMed: 17900904]
- Naeser MA, Palumbo CL, Helm-Estabrooks N, Stiassny-Eder D, Albert ML. Severe nonfluency in aphasia: Role of the medial subcallosal fasciculus and other white matter pathways in recovery of spontaneous speech. *Brain*. 1989; 112:1–38. [PubMed: 2917272]
- Novick JM, Trueswell JC, Thompson-Schill SL. Cognitive control and parsing: Reexamining the role of Broca's area in sentence comprehension. *Cognitive Affective Behavioral Neuroscience*. 2005; 5(3):263–281.
- Penfield W, Rasmussen T. Vocalization and arrest of speech. *Archives of Neurology and Psychiatry*. 1949; 61(1):21–27.
- Rizzolatti G, Craighero L. The mirror-neuron system. *Annual Review of Neuroscience*. 2004; 27:169–192.
- Rogalsky C, Pitz E, Hillis AE, Hickok G. Auditory word comprehension impairment in acute stroke: Relative contribution of phonemic versus semantic factors. *Brain and Language*. 2008; 107(2):167–169. [PubMed: 18823655]
- Swets, JA. Signal detection and recognition by human observers. New York: Wiley; 1964.
- Watkins KE, Strafella AP, Paus T. Seeing and hearing speech excites the motor system involved in speech production. *Neuropsychologia*. 2003; 41:989–994. [PubMed: 12667534]
- Weller M. Anterior opercular cortex lesions cause dissociated lower cranial nerve palsies and anarthria but no aphasia: Foix-Chavany-Marie syndrome and 'automatic voluntary dissociation' revisited. *Journal of Neurology*. 1993; 240(4):199–208. [PubMed: 7684439]
- Wilson SM, Iacoboni M. Neural responses to non-native phonemes varying in producibility: evidence for the sensorimotor nature of speech perception. *NeuroImage*. 2006; 33(1):316–325. [PubMed: 16919478]
- Wilson SM, Saygin AP, Sereno MI, Iacoboni M. Listening to speech activates motor areas involved in speech production. *Nature Neuroscience*. 2004; 7:701–702.

Figure 1.

Top left: The hypothesized human mirror system shaded in red (adapted from G. Rizzolatti & M.F. Destro http://www.scholarpedia.org/article/Mirror_neurons). *Bottom left & right panel:* Reconstructed lesions from five stroke patients involving the hypothesized human mirror system. The left panel shows lesions that are restricted to fronto-parietal regions, whereas the right panel shows lesions that additionally involve temporal lobe structures. Areas in red indicate regions of lesion for each subject. For each subject, 3D rendered lateral views as well as coronal and sagittal slices are presented. The crosshairs on the lateral views correspond to the slices and the crosshairs on the slices. The central sulcus of the template brain is marked in yellow on each 3D-rendered view.

Auditory stimulus: "coat"

Phonemic foil test

Semantic foil test

Unrelated foil test

Mixed foil test

Figure 2. Sample stimulus cards from the word-to-picture matching task. [To view this figure in color, please visit the online version of this journal.]

Figure 3.

Proportion correct/A-prime scores for each of the five brain-injured subjects across five speech recognition/perception tasks. Subjects are grouped according to whether there is temporal lobe involvement or not. *Word Comp-Phon*: word-to-picture matching with phonological foils; *Word Comp-Sem*: word-to-picture matching with semantic foils; *Word Comp-Mix*: word-to-picture matching with phonological, semantic, and unrelated foils; *Word Discrimination*: word discrimination task using words from the word comprehension tests; *NW Discrimination*: non-word discrimination task using non-words that were derived by changing the vowel of the word stimuli used in the word discrimination task.

Figure 4.

Proportion correct/A-prime scores for each of the five brain-injured subjects across five speech recognition/perception tasks. Subjects are grouped according to whether there is temporal lobe involvement or not. *Word Comp-min-pairs*: items from word-to-picture matching tests that include at least one minimal pair phonemic foil ($n = 25$). Word Disc & Nonword Disc are the same as in Figure 3.

Figure 5.

Average proportion correct/A-prime scores and standard error bars for the four brain-injured subjects with frontal lesions affecting the hypothesized mirror system. Tests are the same as reported in Figure 3.

TABLE 1

Biographical and general clinical data for the patient sample

patient ID	gender	hardness	age at lesion onset (years)	testing post lesion onset (years)	education (years)	lesion dx	Clinical Diagnosis	Clinical Fluency	Clinical Paraphasia
LHDST	F	R (self-report)	45	12	12	L MCA infarct	Broca's	na*	na
1978	F	R (+100)	49	13	12	L MCA infarct	Broca's	3	3
2435	M	R (+100)	57	8	12	L MCA infarct	Wernicke's	2	3
2969	M	R (+100)	38	6	17	L MCA infarct	Broca's	3	3
3025	F	R (+100)	48	31	16	L MCA aneurysm	Wernicke's	1	3

na, data not available. Clinical Diagnosis, clinical fluency, and clinical paraphasia, respectively = chronic aphasia description, fluency, and severity of paraphasias as estimated by clinician (clinical fluency and clinical paraphasia scales are the following: 1 = normal, 2 = unsure of the presence of a deficit, 3 = abnormal). L MCA = left middle cerebral artery. Handedness scores in parentheses were calculated via the Edinburgh Handedness Inventory.

* LHDST was administered alternative tests concordant with procedures at the UCSD testing site. A comparable test for this item administered was the Western Aphasia Battery (WAB) fluency subtest = 6/15.

TABLE 2

Neuropsychological data for the patient sample

patient ID	WAIS-III Verbal/ Perform	WAIS-III Inform.	WAIS-III vocab	WAIS-III WM index	BNT	MAE Token Test	Benton COWA	Benton Writing	BDAE Read. Comp.	WCST Corr/PE	WCST Cat
LHDST	na [*] /na [*]	na	na	na	29	na [*]	na [*]	na [*]	4	na/na	na
1978	89/81	10	8	69	37	33	9	3	10	70/31	3
2435	94/90	12	6	59	56	35	18	2	10	65/39	1
2969	114/130	14	13	88	58	41	19	1	9	70/15	6
3025	89/110	11	9	61	29	12	15	2	10	na/na	na

na, data not available; WAIS-III verbal/perform., WAIS-III verbal IQ and performance IQ; WAIS-III inform., information subtest; WAIS-III vocab, vocabulary subtest; WAIS-III WM index, working memory index; BNT, Boston Naming Test; MAE, Multilingual Aphasia Examination; COWA, Controlled Oral Word Association Test; BDAE Read. Comp., Boston Diagnostic Aphasia Examination Reading Comprehension subtest; WCST Corr/Per, Wisconsin Card Sorting Task items correct/# of perseveration errors; WCST cat, # of categories generated in WCST.

* LHDST was administered alternative tests concordant with procedures at the UCSD testing site. Comparable tests/scores include: Western Aphasia Battery (WAB) Aphasia Quotient = 59.1; Test of Nonverbal Intelligence (TONI-3) deviation quotient = 94; Test of Oral and Limb Apraxia (TOLA) Composite = 47th percentile; TOLA command subtest = 5th percentile; BDAE Mechanics of Writing subtests; Well formedness of letters = 18/18; Correctness of letter choice = 23/27; motor facility = 12/18.