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The Role of the Left Frontal Operculum in Sign Language Aphasia

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Abstract

Broca's area has long been implicated in aspects of speech production. But does this region play a role in the production of signed language in prelingually deaf individuals? In this report, we describe our findings in a patient, congenitally deaf and a native user of American Sign Language, who suffered an ischemic infarct involving the left frontal operculum. Our patient presented with an acute expressive aphasia that subsequently resolved, and a chronic deficit predominantly characterized by frequent phonemic-like paraphasias. We conclude that the left frontal operculum does, in fact, play a role in the production of signed language.

Introduction

Since Broca's time, the left frontal operculum, in particular Brodmann's areas 44 and 45, has figured prominently in attempts to determine the anatomy of speech production (Mohr, 1976). While recent studies have shown convincingly that lesions restricted to the frontal operculum do not lead to a lasting, severe speech production deficit (Mohr *et al.*, 1978; Tonkonogy and Goodglass, 1981; Alexander *et al.*, 1990), evidence from the acute postictal syndrome as well as evidence from cortical stimulation (Penfield and Roberts, 1959; Ojemann, 1983) and functional neuroimaging (Petersen *et al.*, 1988; Hinke *et al.*, 1993; Rueckert *et al.*, 1994) confirms at least some role for Broca's area in speech production.

In this report, we describe our findings in the case of a congenitally deaf native user of American Sign Language (ASL), who suffered an ischemic infarct involving the left frontal operculum and the inferior portion of the primary motor cortex. To be sure, part of the long-standing enthusiasm for the hypothesis that the frontal operculum is crucially involved in speech production derives from the intuitive appeal of its anatomical position anterior to motor cortex controlling the musculature involved in speech. Thus, a major question in the present study is, what is the role of this region in the production of a language produced with the hands, rather than with orofacial articulators? To the extent that the language production system is plastic and self-organizing, one might expect the functional analog of Broca's area to be shifted superiorly so that it is anterior to the sensory-motor representation for hand/arm in the case of a deaf signer. Conversely, similarities in the functional neuroanatomy of speech and sign production would suggest that there are constraints on the extent to which neural organization for language production is a self-organizing system, that is, one which is driven by sensory-motor inputs.

A primer on sign language

Like spoken languages, signed languages of the deaf are formal, highly structured linguistic systems, passed down from one generation to the next, with a rigid developmental course, including a critical period for acquisition (Newport and Meier, 1985; Newport, 1991). Signed languages have emerged independently of the language used among hearing individuals in the surrounding community: American Sign Language (ASL) and British Sign Language, for example, are mutually incomprehensible, despite the fact that English is the dominant spoken language in both surrounding communities.

Signed and spoken languages, however, share all the underlying structural complexities of human language. That is, all natural human languages have linguistic

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structure at phonological, morphological, and syntactic levels, and signed languages are no exception. At the phonological level, research has shown that, like the words of spoken languages, signs are fractionated into sublexical elements, including various recurring handshapes, articulation locations, and limb/hand movements, among other features (Perlmutter, 1992; Corina and Sandler, 1993). Further, comparison of two different signed languages (ASL and Chinese Sign Language) reveals that there are even fine-level systematic phonetic differences leading to an 'accent' when native users of one sign language learn another (Klima and Bellugi, 1979; Poizner *et al.*, 1987). At the morphological level, ASL, for example, has developed grammatical markers that serve as inflectional and derivational morphemes; these are regular changes in form across classes of lexical items associated with systematic changes in meaning (Klima and Bellugi, 1979). At the syntactic level, ASL specifies relations among signs using a variety of mechanisms including (i) sign order, (ii) the manipulation of sign forms (usually verbs) in space, where different spatial relations between signs have systematic differences in meaning, and (iii) a small set of grammaticized facial expressions that are used to mark questions, topicalized sentences, and conditionals (Liddell, 1980; Lillo-Martin and Klima, 1990; Lillo-Martin, 1991).

In sum, ASL has developed as a fully autonomous language with grammatical structuring at the same levels as spoken language and with similar kinds of organizational principles. Yet the surface form that this grammatical structuring assumes in a visual-spatial language is deeply rooted in the modality in which the language developed.

Sign language aphasia

A number of recent case studies (Poizner *et al.*, 1987; Bellugi *et al.*, 1989; Corina *et al.*, 1992; Poizner and Kegl, 1993; Hickok *et al.*, 1995) as well as a relatively large group study (Hickok *et al.*, 1996) of deaf stroke patients argue strongly that language is predominantly a function of the left cerebral hemisphere, independent of language modality. Unilateral left cerebral hemisphere lesions in deaf signers often lead to frank sign language aphasias, with clinical syndromes resembling those found in hearing aphasics. For example, one deaf patient, who suffered a large left frontal lesion, presented with non-fluent telegraphic sign production and relatively spared sign comprehension (Poizner *et al.*, 1987). Other left-lesioned signers have been reported with fluent sign production marked by frequent phonemic paraphasias and grammatical errors, and poor sign comprehension (Bellugi *et al.*, 1989). In contrast, none of the right hemisphere-lesioned signers reported to date have had aphasia (Poizner *et al.*, 1987; Bellugi *et al.*, 1989; Poizner and Kegl, 1993). Their signing was grammatically complex and well formed, and their comprehension intact (Hickok *et al.*, 1996). Some of these same patients, however, showed severe visuospatial deficits, as revealed through distorted and fragmented drawings, poor performance on line-orientation tasks and visuo-constructional tasks, and hemispatial neglect.

Thus, the neurology of signed language (and spatial cognition) in deaf individuals appears to be similar to that of hearing individuals, at least at the hemispheric level. We now turn to the present case.

Case RS

A 78-year-old, right-handed woman suffered sudden onset of right facial droop and expressive aphasia. Her medical history was remarkable for hypertension, atherosclerotic heart disease, cardiomegaly, obesity, and deafness. She had been deaf from birth and had a family history of deafness in both parents, one grandmother, and two children. She had acquired ASL in the home from her parents and other family members, and through social contacts, just as a hearing child would have acquired a spoken language.

According to hospital records, at the time of admission she was awake, alert, and oriented to person and place. She appeared to understand questions addressed to her, but was unable to respond. Neurological examination revealed an 'expressive' aphasia (subsequent interviews with family members suggested that RS's production was acutely non-fluent and effortful), a right visual field defect, right central facial weakness, and right arm 'clumsiness'. (Right arm clumsiness would not have explained her expressive aphasia because grammatically complex and correct signing can be produced by the non-dominant hand in normal signers.) She was able to follow most commands.

We first examined RS approximately 4 months after her stroke. We found no evidence of a visual field defect or upper limb motor dysfunction. Her language output was normal in terms of grammatical complexity, phrase length, and rate of production. She complained of occasional 'word' (i.e. sign) finding difficulties which we also noted during conversational signing. RS also produced relatively frequent 'phonemic' paraphasias (i.e. substitutions of sublexical sign components; in one sample we noted 1.4 such errors per minute of uninterrupted signing) and occasional semantic paraphasias. Articulation was normal.

On the visual confrontation naming and 'sign discrimination' tests – adapted for ASL from the Boston Diagnostic Aphasia Examination (BDAE) (Goodglass and Kaplan, 1976) – RS made no errors. Comprehension was largely spared as revealed by our ASL adapted version of the Token Test: RS scored 34 out of 44 correct, compared with a mean of 37 correct among our control group of signers with right hemisphere strokes ($n = 6$; score range = 33–43), and a mean of 19 correct in another group of signers with left hemisphere stroke ($n = 12$, including RS; score range = 4–39). Repetition was mildly impaired: RS correctly repeated 8 out of 16 signed sentences compared with a mean of 3.9 in our signers with left hemisphere strokes ($n = 13$; range = 0–10) and a

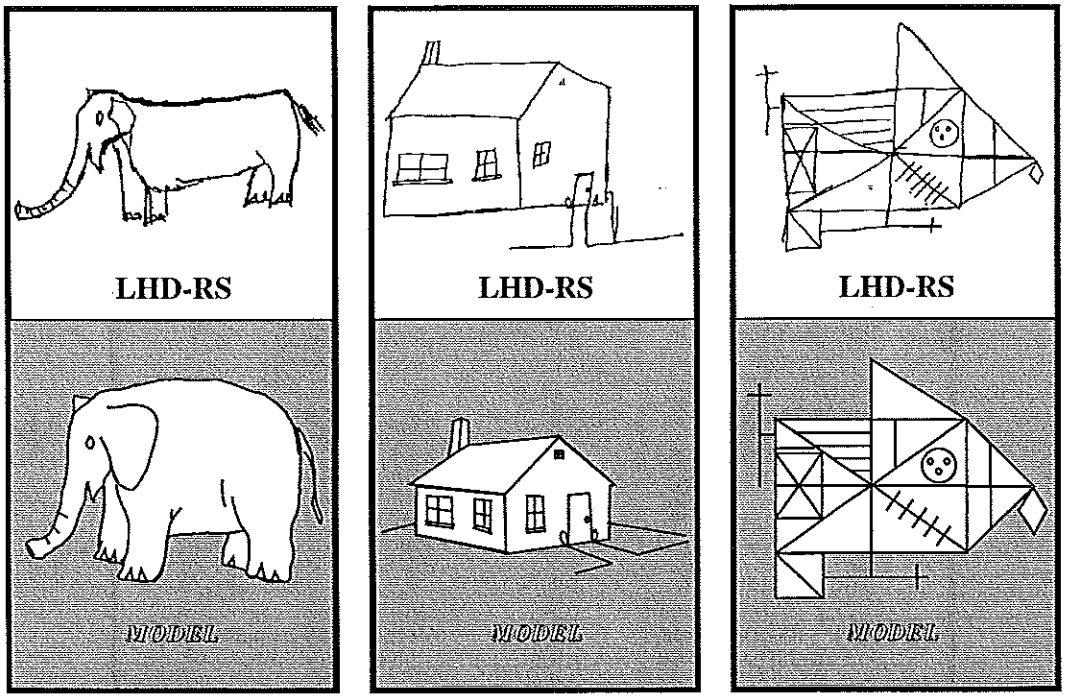

Fig. 1. Sample drawings (copy) from the BDAE drawing task and Rey-Osterrieth task.

mean of 11.6 among our signers with right hemisphere strokes ($n = 7$, range = 6–16).

Limb apraxia was assessed using the apraxia test from the BDAE supplementary non-language test section (Goodglass and Kaplan, 1976), as well as an apraxia test for non-representational movement (Kimura, 1993). RS performed without error on these tests. Visuospatial and visuoconstructional abilities were tested using BDAE drawings and the Rey-Osterrieth Complex Figure (Osterrieth, 1944). She performed well on both tasks (Fig. 1).

A unique aspect of RS's phonemic paraphasias was that they all appeared with two-handed signs (unlike the other cases of sign aphasia we have seen in which there were also paraphasias on one-handed signs). More specifically, her phonemic errors involved coordinating the two hands to make a sign appropriately. For example, on signs that require the two hands to assume different handshapes and/or move independently, RS might incorrectly mirror the movement of one hand with the other. In other cases, on signs where both hands must move simultaneously, RS might incorrectly fail to move one of her hands. In still other instances, on signs where the appropriate movement of one hand was relative to the position of the other, RS might produce an incorrect relational movement in the sense that the movement itself was correct, but it was not carried out correctly with respect to its spatial relation to the other hand. Examples of some of these errors are given in Fig. 2A.

In addition to her paraphasias, we noted an unusual and frequent feature in her signing. On one-handed signs (there are both one- and two-handed signs) she often inadvertantly mirrored the movement and handshape of the dominant (right) signing hand with an identical but smaller movement of the non-signing, non-dominant hand. This movement of the non-dominant hand typically was articulated out of the normal signing space (Fig. 2B), and occurred at a frequency (in one sample) of 1.1 per min of uninterrupted signing. These movements appear to be different from the typical manifestation of mirror movements in that RS's mirror movements were bilaterally asymmetric (the movement of the non-dominant hand was reduced in amplitude), they occurred equally in proximal and distal muscle groups, and there was no evidence of hemiparesis. In contrast, mirror movements are typically bilaterally symmetrical, occur predominantly with distal movements, and are generally associated with hemiplegia or hemiparesis (Herzog and Durwen, 1992). Another difference between RS's mirror movements and published descriptions of mirror movements is that RS did not mirror any non-linguistic movements, such as during apraxia testing. We tabulated the total time that RS was engaged in the production of one-handed non-linguistic motor gestures, and found no mirror movements over a total span of 5.68 mins. Thus, to distinguish RS's movement 'errors' from the standard definition of mirror movements we will henceforth refer to these as 'shadowing'.

We examined RS again at approximately 11 months and again at 3 years after her stroke. Over this time period, there was improvement in her repetition ability (15/16 correct), but we again noted several phonemic paraphasias (again only on two-handed signs) and shadowing of one-handed signs, suggesting that these symptoms were fairly stable.

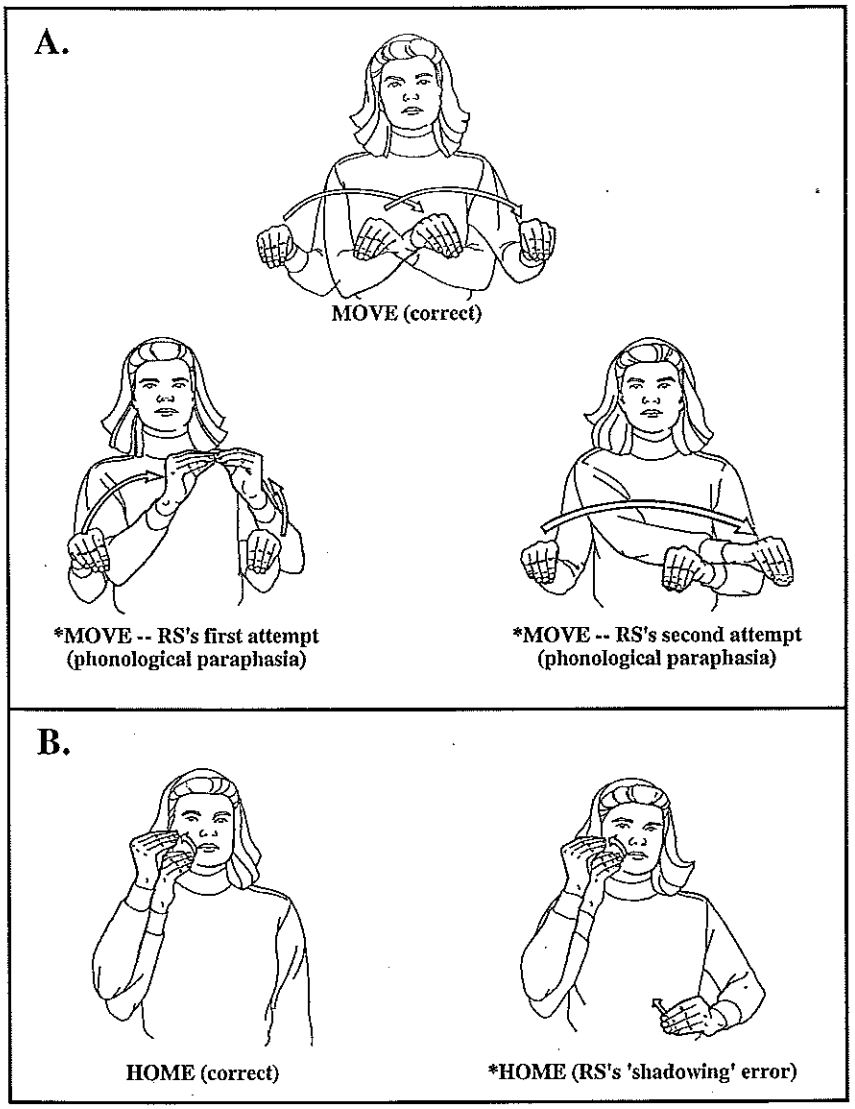

Fig. 2. (A) Examples of phonemic paraphasias involving bimanual coordination. The correct form is shown at the top. In the first example, both hands incorrectly articulate mirror movements (bottom left). In the second example, one hand fails to move at all. (B) Example of 'shadowing' during sign production. The correct (non-shadowed) form is on the left.

Approximately 11 months after her stroke we obtained a brain MR scan suitable for 3D reconstruction using Brainvox (Damasio and Frank, 1992) (T1-weighted, coronal, 3D volume SPGR, slice thickness = 1.5 mm). The 3D reconstruction demonstrated a cortical lesion involving inferior motor cortex with minimal extension posteriorly into the somatosensory cortex (Fig. 3A). The cortical lesion extended anteriorly to involve most of the pars opercularis, and inferiorly it appeared to involve the anterior, superior insula. The middle frontal gyrus was intact. Subcortically, the lesion undercut most of the pars triangularis, and involved subcortical white matter deep to lower motor cortex and posterior inferior frontal gyrus extending medially to nearly the frontal horn of the lateral ventricle but sparing the medial subcallosal fasciculus (Naeser *et al.*, 1989), caudate, and paraventricular white matter.

For direct comparison with previous lesion analyses carried out by Alexander and colleagues (1990), we also were able to analyse para-axial slices generated from the same data set using Brainvox. Fig. 3B shows tracings of the relevant oblique axial slices. Note that there was minimal involvement (if any) of para- and periventricular white matter (PVWM). In contrast, all of Alexander and colleagues' patients with both frontal operculum involvement and lasting aphasia (including frequent phonemic errors) had lesions that involved PVWM adjacent to the anterior superior portion of the body of the ventricle. Similar cases have been identified by Mohr and colleagues (1978) and by Tonkonogy and Goodglass (1981). Patients with similar frontal operculum lesions but with spared PVWM presented with a transient aphasia fitting the criteria for transcortical motor aphasia.

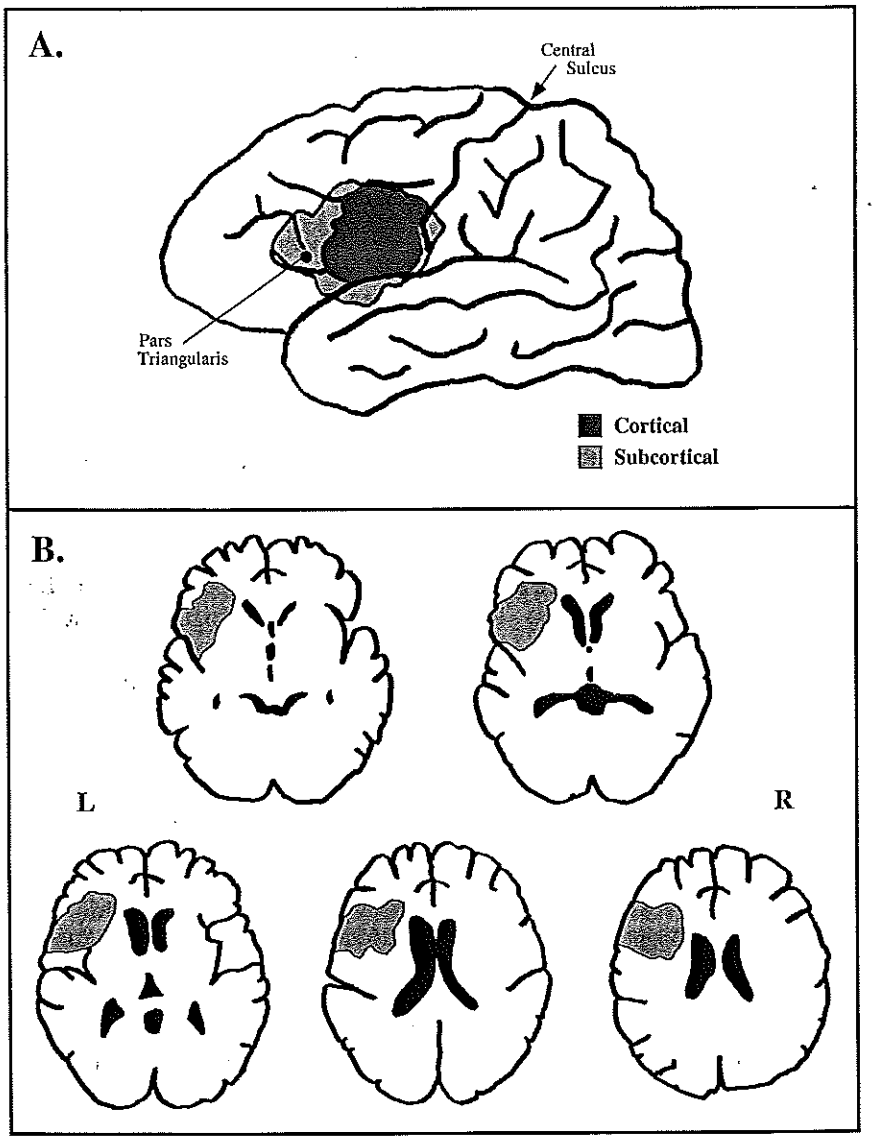

Fig. 3. Tracings of brain reconstructions from MRI generated using Brainvox software. (A) Tracing from a 3D reconstruction of RS's brain showing the location of the cortical and subcortical extent of the lesion relative to sucal and gyral landmarks. Subcortical extent of the lesion was mapped by tracing the lesion on each slice and projecting it to the surface of the 3D reconstruction. (B) Tracings from oblique axial slices showing the deep extension of the lesion.

Discussion

To summarize the main characteristics of the case: RS presented with an acute 'expressive' aphasia that resolved into normal sign production except for relatively frequent phonemic paraphasias, occasional sign-finding difficulties, and semantic paraphasias. Her sign comprehension and repetition abilities were largely spared. In addition, RS often 'shadowed' one-handed signs with her non-dominant hand. RS's lesion involved the posterior aspect of the frontal operculum, inferior motor cortex, and white matter deep to these regions. PVWM was spared.

The finding of chronic and significant disruption in aspects of sign production demonstrates that the left frontal operculum does indeed play a role in sign language production. The involvement of this brain region in language processing, then, appears to be independent of modality. This is the primary conclusion of the present paper. In what follows we (i) examine the similarities and differences between this case and reported cases of hearing patients with a similar lesion, and (ii) consider some possible explanations for the observed differences.

There are several reports of hearing/speaking patients with a lesion similar to that of RS (Mohr *et al.*, 1978; Tonkonogy and Goodglass, 1981; Alexander *et al.*, 1990). These reports describe a clinical course that is, for the most part, similar to that observed with RS: an acute non-fluent aphasia or mutism and occasionally some chronic, but mild, word-finding problems and/or semantic paraphasias. There is no indication, however, that such a lesion in a

hearing/speaking patient would produce a syndrome that includes chronic phonemic paraphasic errors as it did in RS. Rather, Alexander *et al.* (1990) showed that frequent paraphasic errors occurred only when the lesion extended into PVWM, which in RS's case it did not.

Thus, clinical-anatomic correlations based on data from hearing/speaking patients do not appear to apply wholesale to the present case. There are at least three possible explanations for this discrepancy. The first is that the difference is simply a function of natural individual variation in the functional organization of the relevant language-related systems. This explanation assumes that there is no qualitative difference in the function of the left frontal operculum with respect to processing signed versus spoken language, and predicts that one should be able to find hearing patients with a chronic syndrome much like that of RS. The second possibility is that there is a small, but real, qualitative difference in the neural organization for signed versus spoken language that produced the difference in the observed syndromes for RS and hearing patients. This possibility suggests a greater role for the left frontal operculum in sign language production as compared to spoken language. The third possibility is that the differences can be explained in terms of peripheral differences between manual and oral articulation of language. This possibility requires some elaboration.

Recall that RS's phonemic paraphasias were rather specific to signs that involved the coordination of the two hands. As noted above, this is not characteristic of phonemic paraphasias we have observed in other deaf signers where errors are often noted along a range of phonemic parameters (e.g. handshape, place of articulation, hand orientation). This fact, together with the observation of RS's shadowing behavior, suggests that her sign errors may in fact be the result of a more general disorder of bimanual motor coordination rather than true phonemic paraphasias. In fact, what is characteristic of virtually all RS's production errors (both 'paraphasic' and shadowing errors) is that she has difficulty performing non-symmetric limb/hand movements. (Again, as detailed above, RS's deficit in this respect does not seem to fall under the definition of mirror movements.)

This generalized characterization of RS's deficit seems at first quite plausible because there is evidence from both animal work (e.g. Brinkman, 1984) and human lesion data to suggest that damage to frontal lobe structures can cause a chronic bimanual coordination deficit. For example, Freund and Hummelsheim (1985) report a series of patients with either left or right premotor lesions who exhibit a chronic bimanual coordination deficit. The deficit shows up predominantly in the coordination of proximal muscle groups, as in performing alternating windmill movements with the arms or pedalling motions with the legs. However, these movements can be executed with each limb on its own. Bimanual alternating movements of distal muscle groups (e.g. fingers and hands) were spared. Because many of RS's sign paraphasias involved errors in the coordinated movement of proximal muscle groups, and because her lesion involves inferior premotor cortex, one might ask whether her deficit can be characterized as a non-specific 'premotor syndrome'.

As noted above, however, RS's coordination difficulties did not show up on non-linguistic limb movement tasks. In addition, she had no difficulty in everyday bimanual tasks such as tying shoelaces, buttoning a sweater, or knitting. In order to assess further the possibility of a generalized bimanual coordination deficit we asked RS to perform alternating limb movements of the type known to be disrupted following premotor lesions. RS showed no deficits in producing bilateral symmetric or alternating (i) windmill movements of the arms (in both the forward and backward directions), (ii) flexion and extension of the elbow, (iii) flexion and extension of the wrist, and (iv) finger tapping (with the index fingers). In fact, in our total sample of >5.5 min of RS performing the above-mentioned non-linguistic manual tasks, we did not see any examples of bimanual incoordination, including shadow-type movements. In contrast, we noted 10 instances of bimanual incoordination (including both paraphasic-like errors and shadowing errors) during a 4.4 min sample of sign language production.

We also did not see any of the other sorts of deficits that have been reported to be associated with premotor lesions, for example, contralateral proximal muscle weakness (particularly involving arm elevation, Freund and Hummelsheim, 1985) and (at least in the case of left hemisphere lesions) disrupted rhythm production, and resulting severe non-aphasic dysgraphia (Freund, 1989). During testing for bimanual coordination, RS had no difficulty raising either arm above her head during the windmill task. Rhythm production, as evidenced by the temporal movement pattern in RS's handwriting, was normal.

There is, therefore, no evidence of a premotor syndrome, or, more specifically, a domain-general bimanual coordination deficit. To the extent that RS's deficit can be characterized as one of bimanual coordination, it appears to be specific for sign language production. Further, the specificity of the deficit rules out the possibility that coordination is disrupted simply as a result of muscle weakness.

While it is difficult to draw firm conclusions on the basis of a single case, we would like to propose the following as one possible explanation of our results: (i) RS's production errors, both the shadowing and the phonemic paraphasia-like errors, result from the same underlying deficit, namely some form of bimanual incoordination. The argument for this hypothesis is simply one of parsimony: the same behavior, a tendency to perform similar bimanual movements when distinct movements are intended, can account for both error types. (ii) Based on the fact that RS did not have any difficulty performing non-linguistic bimanual

tasks, we conclude that her bimanual coordination deficit is specific to the production of sign language. From these conclusions we hypothesize that (iii) the left frontal operculum in deaf sign language users is involved in (among other things perhaps) bilateral coordination of motor output *in the service of language*.

Returning now to the question of possible differences in the neural organization of signed and spoken language, and assuming our reasoning to be on the right track, one might ask whether in deaf sign language users the left frontal operculum takes on the role of bimanual coordination for language production *in addition to* whatever functions it normally has, or whether it subserves a similar function for speech production. Again, the answer to this question will have to await further investigation, but it does seem at least feasible that this region could play a role in bilateral coordination for speech. One might speculate that the reason chronic deficits are not identified for speech is because the speech articulators are largely hidden from view, and phonetic contrasts in speech are not (for the most part) achieved by movement in the left-right dimension, or with left-right asymmetry of movement. (The exception in English is the /l/, as in '*l*ap,' which, in some speakers, is articulated by raising one side of the tongue.) Thus, if there were a mild coordination deficit involving the speech articulators, it would likely manifest as occasional dysarthric production rather than phonemic-like paraphasias. This contrasts with signed language where left-right asymmetries in movement of the hands and arms often signal phonemic contrasts. We might speculate further that acute expressive aphasia or mutism often reported following Broca's area lesions, and also noted in RS's acute syndrome, might be a consequence of disrupting the normal coordinated control of the speech articulators, although the mechanism of recovery of coordinated speech is unclear.

In summary, the present case demonstrates that in individuals who are deaf from birth, the left frontal operculum does in fact participate in the production of signed language. Compared with what has been reported for hearing patients with similar lesions we found both similarities and differences in the resulting syndrome. Whether the differences (i) represent normal variation in functional organization, (ii) indicate a qualitative difference in neural organization, or, as we have suggested, (iii) can be explained in terms of peripheral differences in motor articulation of signed versus spoken language, is a question for future work. Unanswered questions notwithstanding, the fact that the left frontal operculum is involved in aspects of sign language production suggests that there are indeed constraints on the extent of plasticity in the neural systems underlying language production.

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Abstract
Broca's area has long been implicated in aspects of speech production. But does this region play a role in the production of signed language in prelingually deaf individuals? In this report, we describe our findings in a patient, congenitally deaf and a native user of American Sign Language, who suffered an ischemic infarct involving the left frontal operculum. Our patient presented with an acute expressive aphasia that subsequently resolved, and a chronic deficit predominantly characterized by frequent phonemic-like paraphasias. We conclude that the left frontal operculum does, in fact, play a role in the production of signed language.

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Primary diagnosis of interest
Aphasia

Author's designation of case
RS

Key theoretical issue
- What is the role of Broca's area in the production of signed language? More generally, to what extent is the neural organization of language affected by experience?

Key words: Broca's area; aphasia; language; sign language; neural plasticity

Scan, EEG and other related measures
MRI with 3D surface reconstruction

Standardized assessment
Sign language adapted Boston Diagnostic Aphasia Examination and Token Test, Rey-Osterrieth Complex Figure.

Lesion location
- Left posterior inferior frontal gyrus and lower motor cortex.

Lesion type
Ischemic infarct

Language
English