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Sign language aphasia following *right* hemisphere damage in a left-hander: A case of reversed cerebral dominance in a deaf signer?

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Recent lesion studies have shown that left hemisphere lesions often give rise to frank sign language aphasias in deaf signers, whereas right hemisphere lesions do not, suggesting similar patterns of hemispheric asymmetry for signed and spoken language. We present here a case of a left-handed, deaf, life-long signer who became aphasic after a *right-hemisphere* lesion. The subject exhibits deficits in sign language comprehension and production typically associated with left hemisphere damaged signers. He also exhibits evidence of local versus global deficits similar to left-hemisphere lesioned hearing patients. This case represents reversed lateralization for sign language and also may represent reversed lateralization for visuo-spatial abilities in a deaf signer.

Introduction

The classical view concerning cerebral asymmetries held that language dominance was strongly correlated with hand dominance: right-handers were thought to be left-dominant for language, and left-handers to be right-dominant for language (Broca, 1865). Data from a range of sources have shown that the classical view is incorrect in that a majority of left-handers appear to be left-dominant for language, with estimates ranging between 70 and 85 percent (Milner, 1975, Hécaen *et al.*,1981; Naeser and Borod, 1986). However, while a strong version of the classical view is clearly untenable, the fact remains that there is a correlation, albeit weak, between hand-dominance and language dominance: a left-hander is at least an order of magnitude more likely than a right-hander to have right-hemisphere dominance for language.

To date, all existing data on the dominance patterns for language in left-handers have come from studies of hearing speaking individuals. Here we report the first in-depth study of a left-handed deaf signer who presented with aphasia following a right hemisphere lesion. Additional findings from assessment of nonlinguistic abilities suggest that the individual may have reversed dominance. This case is presented in the context of a broader investigation of the effects of unilateral brain injury on neurocognitive organization in deaf signers, which has found hemispheric dominance patterns in deaf signers to be comparable to what is found in the hearing population. In what follows we will first review our findings on lateralization patterns in deaf signers to put the present case in proper context, and then present the case description.

Background: Issues of hemispheric specialization in American Sign Language

American Sign Language (ASL) is one of many sign languages used by deaf populations around the world. It has the formal structure found in spoken languages yet differs significantly with respect to the mode of presentation and to the mode of reception. That is, ASL is based in the visual modality and uses not only temporal relations (as do spoken languages) but also relies heavily at every linguistic level on contrasts of spatial relations to convey linguistic information (Emmorey, 2002).

Hemispheric asymmetry in deaf signers

Our findings over the past two decades have indicated that hemispheric asymmetries for cognitive function in deaf signers are comparable to what has been reported for hearing individuals (Hickok *et al.,* 1996; Hickok, Bellugi *et al.,* 1998, Hickok, Kirk *et al.,* 1998). For example, we have found that left hemisphere damage often produces significant aphasic

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deficits in deaf signers, whereas right hemisphere damage does not (Figure 1). Conversely, right hemisphere damage is more often associated with substantial disruption in spatial cognitive abilities, including hemispatial neglect. When spatial cognitive abilities are affected in left-injured signers, the deficits tend to involve local-level features rather than the global-level deficits most often seen in right-injured signers (Milner 1975); this pattern is identical to that reported among hearing subjects (Lamb *et al.,* 1989; 1990).

The general pattern of hemispheric asymmetry described above holds for right-handed signers (Hickok *et al.,* 1996). There is also some indication that it holds for most left-handed signers as well: Nearly 10% of our left hemisphere damaged subjects (2 of 21) have been left-handed, yet these subjects showed impairments similar to the right-handed group.

Again, in studies up to now, only deaf signers with damage to the left hemisphere have presented with sign language aphasias. In contrast, those with RHD did not display aphasic characteristics; their language profiles were similar to age-matched deaf controls (Poizner *et al.,* 1987; Hickok *et al.,* 1999, Hickok and Bellugi, 2001).

Methods and Results

The case study here is presented against a background of a program of studies with deaf life-long signers with damage to either the left hemisphere or the right hemisphere, a program ongoing at the Salk Institute Laboratory for Cognitive Neuroscience (LCN). The program includes an examination of language abilities, nonlanguage spatial abilities and a neurological examination. The language probes include the Salk Diagnostic Aphasia Examination (SDAE) for Sign Language (a set of tests adapted for ASL from the Boston Diagnostic Aphasia Examination, focusing on sign language production and comprehension) and an additional set of linguistic probes for ASL (designed for the purpose of examining distinctive properties of ASL at a variety of linguistic levels). The assessment of nonlanguage spatial capacities comprises a range of tasks that includes drawings, neglect tests, as well as visual recognition tasks. Over the years, a large group of deaf signers with left or right hemisphere lesions have been studied with this same basic assessment. This program is designed to address the central question of "How is the brain organized for language when the language itself is visuo-spatial, and when language and space are so intimately intertwined?"

Current case study: The case of subject RHD-228

Against this background, we present here the first in-depth study of a subject with a sign language aphasia due to a *right* hemisphere lesion. Subject RHD-228 is a left-handed male who became bilaterally deaf at 18 months of age due to meningitis. At the age of five RHD-228 began attending a residential school for the deaf. Throughout his educational career, including college, ASL was used in and out of the classroom. ASL has been his primary language throughout his life with his immediate family and friends. He taught ASL at community colleges for a number of years.

With respect to RHD-228's handedness, he reported that he was strongly left-handed prior to his stroke. He has a strong familial history of left handedness. At the age of 55, the subject suffered an ischemic infarct to the right hemisphere that left him with serious aphasic symptoms yet caused minimal observable impairment to his visual spatial cognitive abilities.

Lesion site

The MRI images in Figure 2 show that RHD-228 sustained a large unilateral lesion involving his right temporal, parietal, and (to a lesser extent) frontal lobes. The temporal lobe involvement included most of the superior temporal gyrus (including the right hemisphere homologue of Wernicke's area), and middle temporal gyrus. In the parietal lobe the supramarginal gyrus was involved as well as lower portions of the post central gyrus. The lesion continued anteriorly, involving lower portions of the precentral gyrus and undercutting portions of the posterior inferior frontal gyrus.

Neurological examination

Mental Status Examination: The examination was conducted entirely in ASL by way of an ASL interpreter. The subject was alert, with normal attention and in excellent spirits. He generally followed simple commands well.

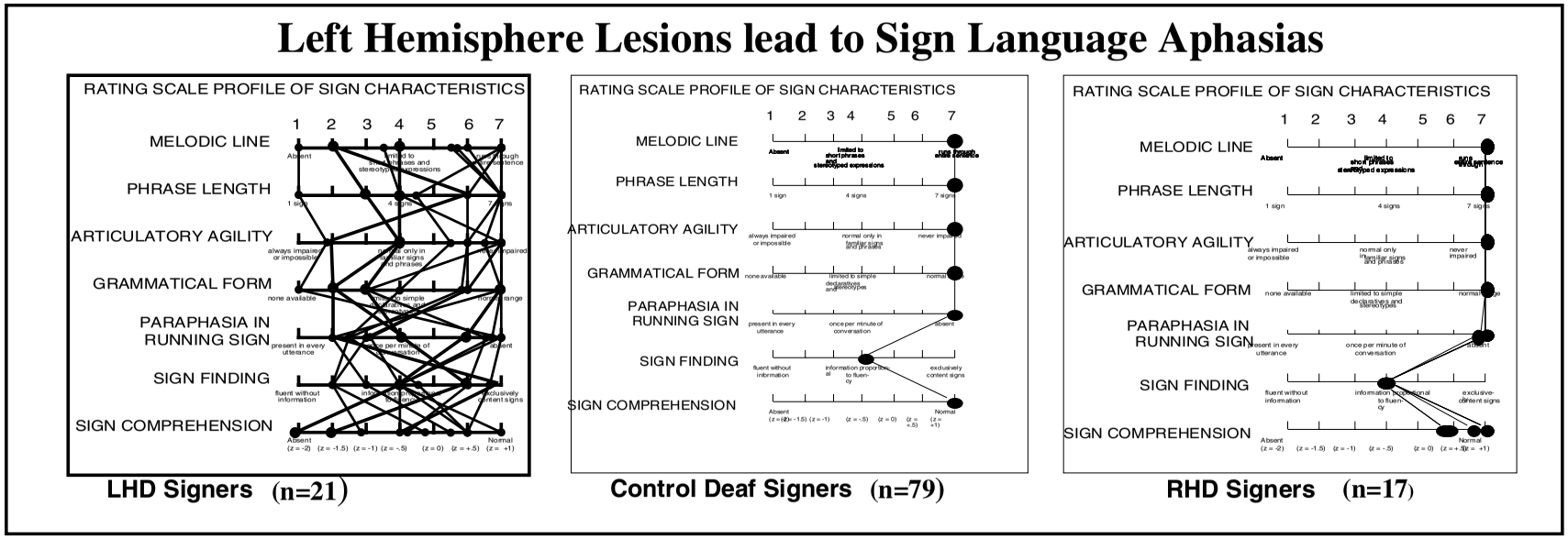
Naming tasks. He named the president correctly. He gave the date (month, day, year), day of the week, and city correctly. When asked to name a wristwatch, he finger-spelled "clock." When asked to name a pen he did so correctly. When asked to name a tie he correctly finger spelled the name. He named six different animals in a minute.

Computational tasks. When asked to subtract 7 from 100 he struggled and eventually signed "92…no 94." When asked to subtract 3 from 20 he first signed "27" and then corrected himself. He was unable to calculate the number of quarters in $1.75 but did correctly sign the number of quarters in $1.00.

Visual spatial tasks. He copied three "nonsense" figures well (he was instructed to remember these for later). He also successfully drew all three "nonsense" figures five minutes later. He copied a picture of a cube but with a 45 degree rotation and mild distortion. His strategy for copying the cube was to first attempt to copy the three inside lines and then draw the outer margin. When asked to draw a clock and set it to 11:10, he drew a circle and placed the numbers correctly on the face of the clock. He then wrote the numbers 11:10 below the clock as if it were a digital clock. He bisected lines without

Comparison of LHD Signers to RHD Signers

SDAE Rating Scale Profiles



LHD Deaf Signers' Impairments in Sign Language Functions

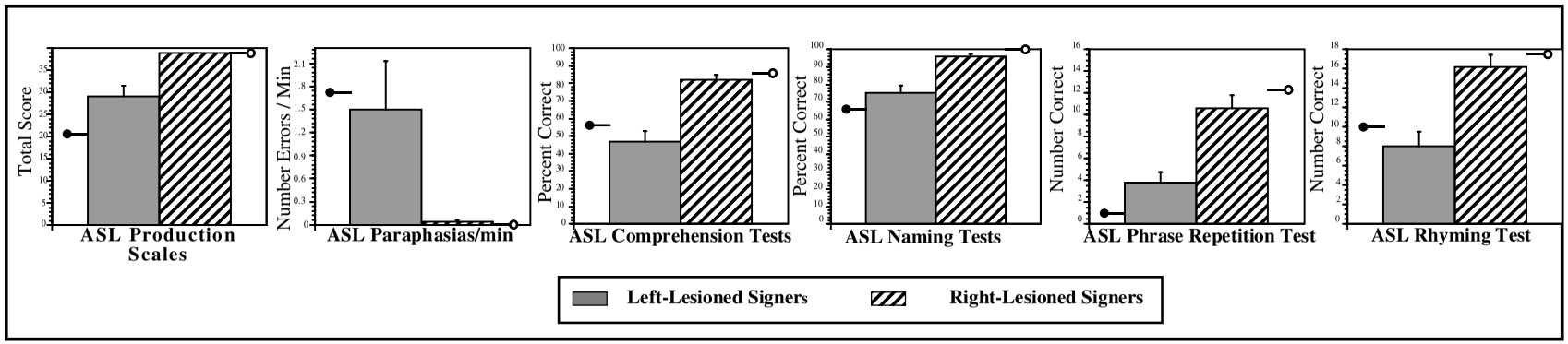


Fig. 1. Presents a composite comparison of two groups of deaf stroke patients and one unimpaired control group. Presented are the profiles of left hemisphere damaged (LHD) deaf signers, right hemisphere damaged (RHD) deaf signers, and normal deaf control signers. These profiles of sign characteristics are from the Salk Sign Diagnostic Aphasia Examination (SDAE)[6] (a measure of sign aphasia that was adapted for ASL from the Boston Diagnostic Aphasia Exam[14]). Compared to the normal signers' profiles, those of the 17 RHD signers showed no impairment in any aspect of ASL grammar. By contrast, the 21 signers with LHD showed a variety of profiles. Taken as a group they showed deficits within all aspects of language performance tapped by the test battery.

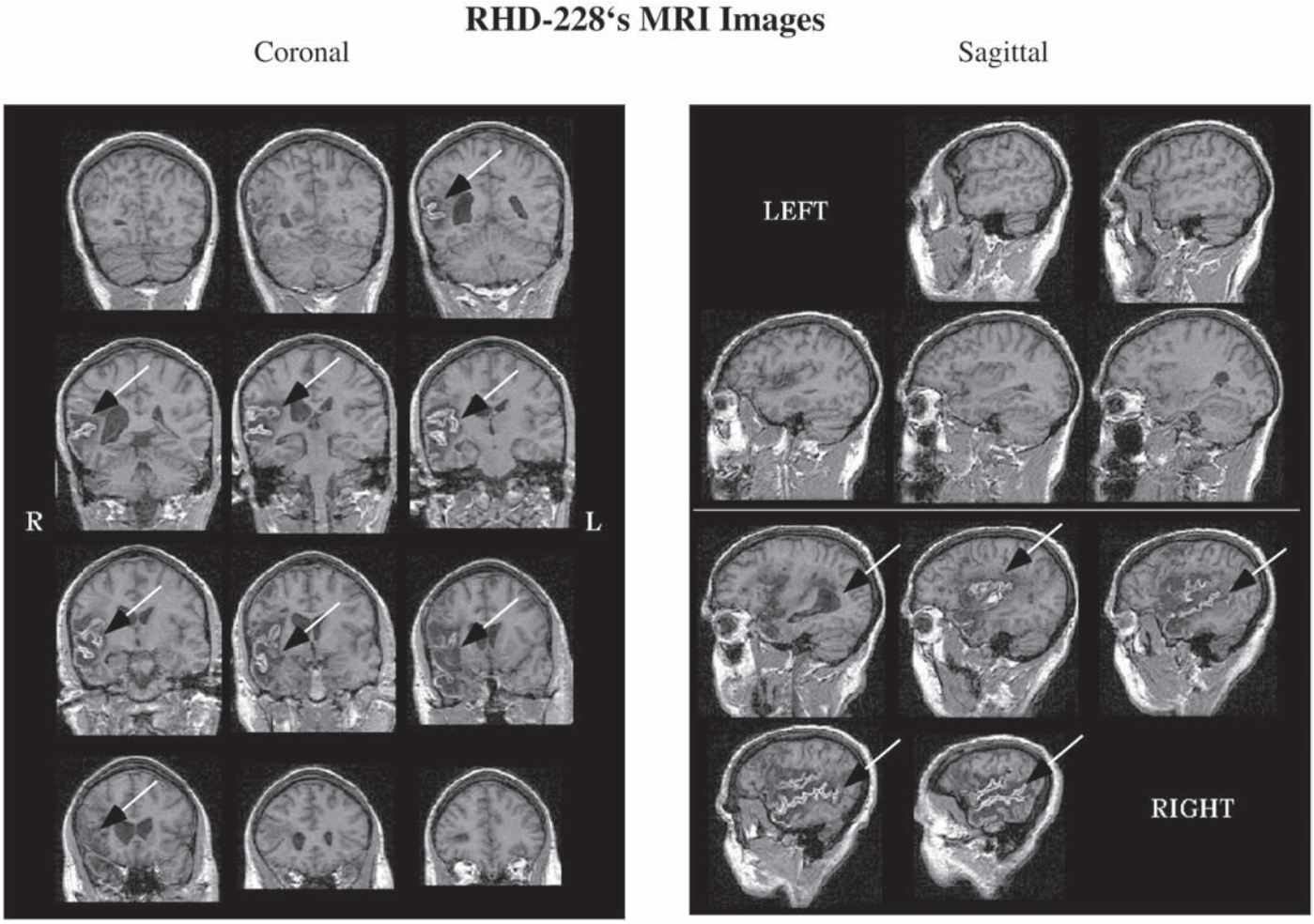


Fig. 2. Magnetic resonance imaging of the brain was performed utilizing the GE Signa superconducting magnet operating at 1.5 Tesla. Coronal and sagittal spin-echo TI-weighted images of RHD-228's brain performed 6 months after his stroke.

significant difficulty and crossed out all the lines on a piece of paper without evidence of neglect. However, with double simultaneous visual stimuli he sometimes reported left-sided stimuli as having been seen on the right, and he often neglected the left-sided stimulus when stimuli were presented bilaterally.

Motor task. There also was at least some mild left motor neglect in that he tended to not use the left side despite its normal strength. With encouragement he was able to show normal strength on the left.

General neurological examination. Visual acuity was J1+ (20/20 equivalent for near vision) when wearing his trifocals. He appeared to be able to count fingers in all four quadrants of each eye. Light touch, sharp and cold sensation were decreased on the left face. Cranial nerves were otherwise normal. There was no ataxia, though rapid movements of index finger on thumb were moderately slowed in a regular fashion on the left. Muscle bulk, tone, and strength were normal in the four extremities. No movement disorder was present. Light touch sensation was decreased on the left body, and position sense was absent on the left index finger and left big toe. Sharp sensation was perceived as tingling and uncomfortable on left body. Light touch, position, and sharp sensation were normal on the right. Muscle stretch reflexes were normal and symmetric except for absent gastrocnemius reflexes bilaterally. Toe responses were withdrawal bilaterally. Posture and gait were normal.

Sign language abilities

We had the opportunity to test RHD-228's language abilities on three different occasions (at eight months, two years and five years after his stroke) with our battery of sign language probes, as well as several tests of nonlinguistic ability.

Comparison subject

To highlight the language performance of our right-lesioned deaf signers, we compare two individuals with similar lesions in

opposite hemispheres: right-lesioned RHD-228 presented here and left-lesioned LHD-108 (Corina *et al.,* 1992), a subject previously reported on. LHD-108 is a 76 year-old right-handed male, congenitally deaf, and a vibrant member of the deaf community. LHD-108 has been chosen because his ischemic infarct was roughly the same size and location in the *left* hemisphere as was RHD-228 in the *right* hemisphere.

Sign language comprehension deficits

Two tests assessed sign language comprehension. The Sign Discrimination test (a 36 item test adapted from the Word Discrimination test of the SDAE) assessed single-sign comprehension, and an ASL Token Test assessed sentence-level comprehension. In the past we have partitioned the ASL Token Test into two sub-parts: the first part includes the "simple" items, namely, those that involve one-clause, one-step commands, and the second part includes the "complex" items, which are multiclause and/or multistep commands (Hickok *et al*.,2002).

Group performance. LHD signers performed more poorly on each of these tasks used. It has been found that (right-handed) right hemisphere-lesioned signers score at ceiling on the simple items, whereas (right-handed) left hemisphere-lesioned signers tend to show impairments even on these simple items. In addition, LHD signers with temporal lobe involvement performed worse on every test when compared to LHD signers without temporal lobe involvement (as well as the RHD signers) (Hickok *et al*., 2002).

Comprehension deficits in RHD-228 and LHD-108. Consistent with the pattern typical of left-lesioned signers, RHD-228 exhibited significant difficulty in the area of sign language comprehension. RHD-228's scores for single sign comprehension (71%), simple sentence comprehension (50%) and complex sentence comprehension (0%) can be seen in Figure 3. When compared to a group study carried out previously by our group, these scores are all in the lower range of scores for left hemisphere subjects with lesions that include the left temporal lobe (this was the group found to have the most significant impairment with regard to sign language comprehension) (Hickok et al., 2002). On all three of these tests, LHD-108, with left hemisphere damage, had exactly the same scores as RHD-228, who has right hemisphere damage.

Additional evidence for a significant sign comprehension deficit in RHD-228 comes from two additional tests. On the "commands" subtest of the SDAE, which involves following one-, two-, and three-step commands, he scored 33%. On the Complex Ideational Material subtest, which involves paragraph-level information, RHD-228 scored 24%. In comparison, LHD-108's scores on these tests were 20% and 36% respectively. Figure 4 shows a comparison of RHD-228's Sign Aphasia Profile to LHD-108's; the two are remarkably similar.

Sign language production deficits

A common symptom of aphasia is paraphasic errors in production. We have previously reported that left hemisphere damaged signers, like hearing aphasics, exhibit paraphasic errors quite frequently in the sign production, whereas right damaged signers do not (Hickok *et al*., 1996). A more recent analysis of paraphasic errors was completed based on transcripts from 37 of our right-handed, deaf brain-injured subjects (this included both RHD and LHD subjects). Errors were categorized into lexical paraphasias (substitution of one sign for another with no semantic relation), semantic paraphasias (substitution of a semantic related sign), phonemic paraphasias (differentiated into orientation, location, movement and handshape), and paragrammatisms (incorrect morphological substitutions). Within the tasks that were transcribed (the Cookie Theft picture and The Paint Story) RHD-228 (during his first testing visit) produced 129 signs, while LHD-108, who was not tested a second time, produced 108 signs.

Group performance

Left hemisphere-damaged signers committed paraphasic errors on 10.06% of signs produced, whereas right hemisphere-damaged signers made errors on only 1.57% of signs produced. This is consistent with previous findings that paraphasic errors in deaf signers are relatively common following left hemisphere damage but not right hemisphere damage.

Types of paraphasic sign error patterns in RHD-228

In contrast to our group of right-handed RHD signers who rarely commit paraphasic errors, RHD-228 displayed a substantial number of paraphasias including semantic, lexical, neologistic and phonemic subtypes.

Lexical paraphasias: RHD-228 made lexical errors in 0.4% of his sign tokens (cf., 0.2% for LHD-108). An example, pre-

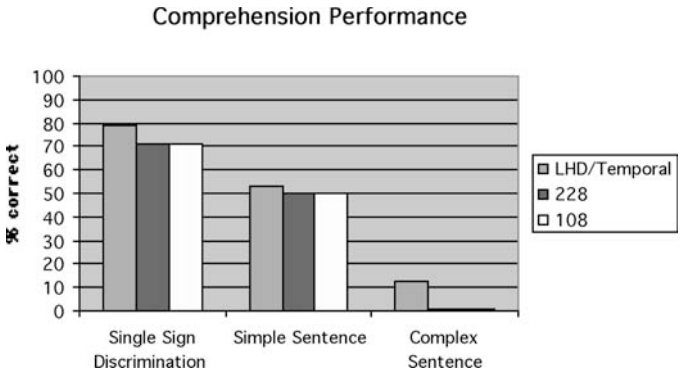


Fig. 3. Scores for RHD-228 and LHD-108 are lower than the mean scores for all deaf subjects with left temporal lobe impairment. Both subjects score 0% for Comprehension of Complex Sentences.

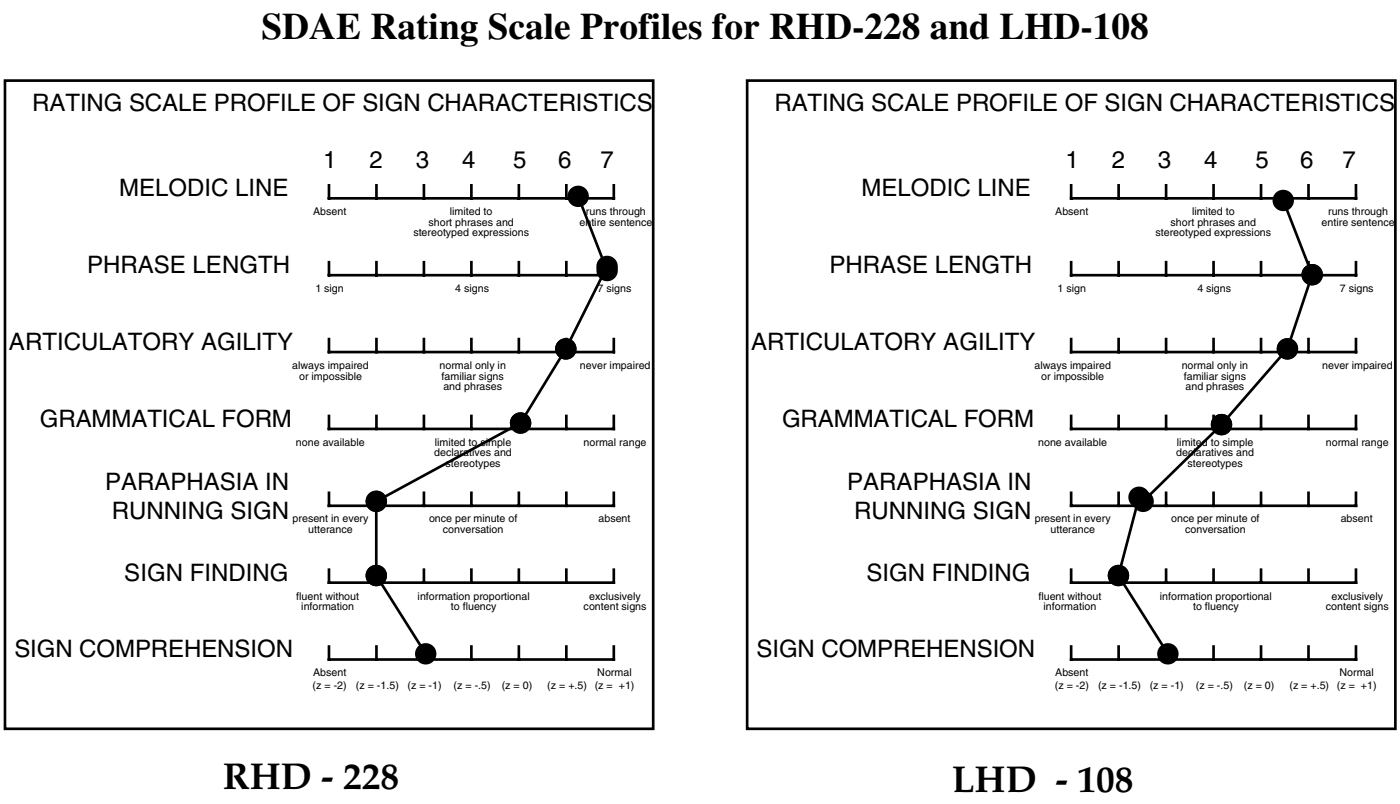


Fig. 4. The similarity of these two profiles combined with the similarity of their lesion sites (though in different hemispheres) makes the two subjects good for comparison.

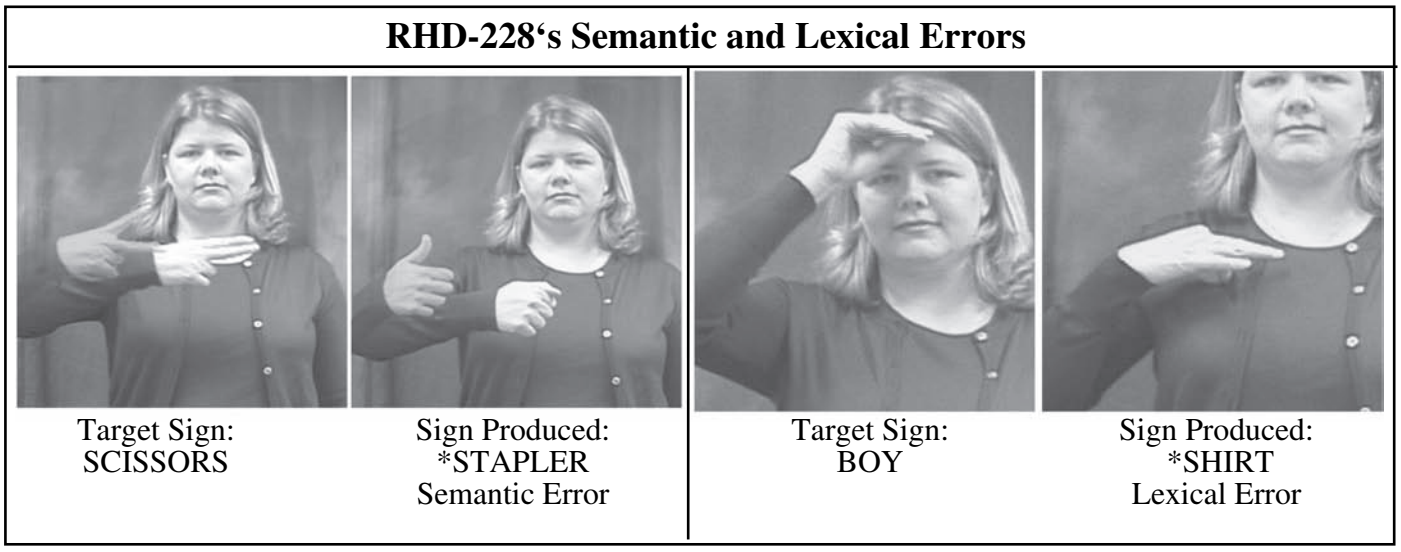


Fig. 5. Examples (illustrated by a model) of RHD-228's Semantic Errors and Lexical Errors.

sented in Figure 5, is his substitution of the sign SHIRT for the intended BOY (all capital letters are utilized as a notation convention representing the English gloss of a lexically signed item).

Semantic paraphasias: RHD-228 made semantic errors in 1.6% of his sign tokens (cf., 0.9% for LHD-108). Examples include production of the sign SPLASH for the target POUR, and STAPLER for SCISSORS (Figure 5).

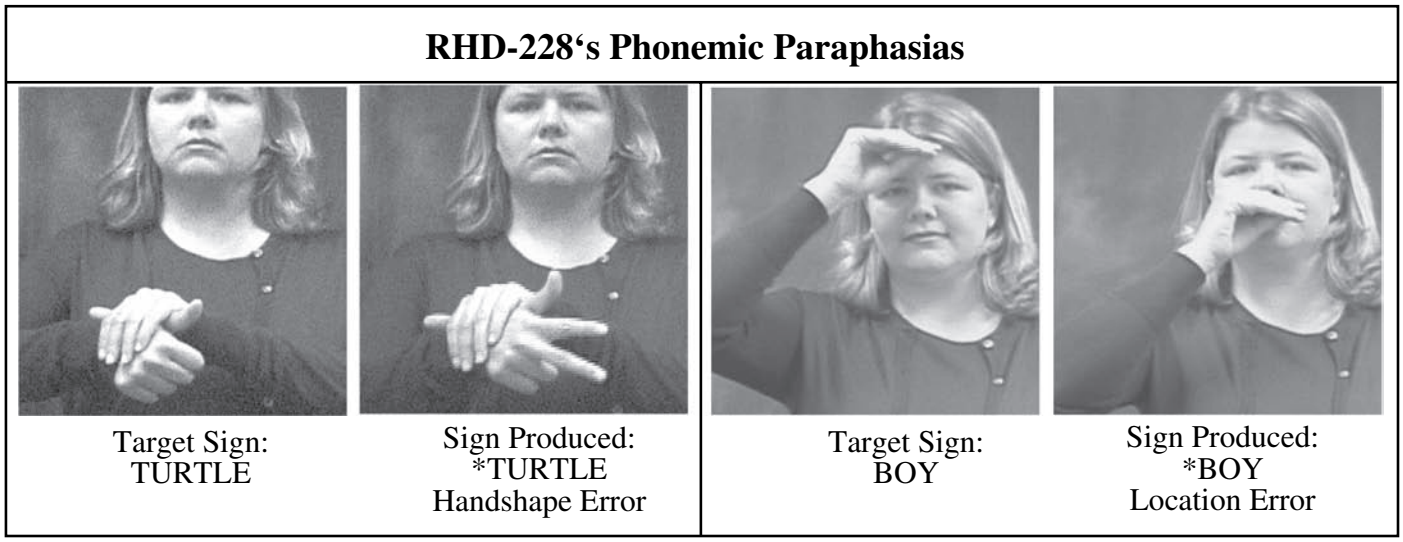


Fig. 6. The figure (illustrated by a model) on the left shows the substitution of a "3" handshape for the "A" handshape that should have been used in the sign TURTLE. The images on the right show RHD-228's placement of the sign BOY at the level of the tip of the nose rather than the correct placement at the forehead.

Phonemic Paraphasias: Phonemic paraphasias in ASL consists of a substitution in one of the major parameters of ASL signs, e.g., (a) hand shape, (b) place of articulation, (c) movement or (d) orientation of the hand. RHD-228 produced phonemic paraphasias in all four of these parameters of various signs during the course of his interview and testing. Within these narratives, RHD-228 made phonemic paraphasias in 11.6% of his signs (LHD-108 made 22.2%).

In the narrative tasks, in terms of the total errors made, RHD-228 and LHD-108 had proportional ratios of phonemic paraphasic errors with respect to the different parameters of ASL signs. For both subjects, handshape errors were by far the most common, accounting for more than 50% of their phonemic errors (RHD-228, 9; LHD-108, 14). An example of a handshape error is presented in Figure 6, along with an example of a location error. Note that both errors result in phonologically permissible non-signs.

Summary of sign language deficits

In summary, this case is unique in our large program of studies of deaf left-and right-lesioned signers. Up to now, we have found that only with left hemisphere lesions are there sign language aphasias. Unlike all the other right-lesioned subjects tested, this subject with a clear right hemisphere only lesion is definitely aphasic for sign language, showing striking deficits in both comprehension and production.

Nonlinguistic spatial abilities

RHD in hearing, speaking individuals often leads to substantial deficits in nonlinguistic spatial abilities. Using a battery of nonlinguistic visual spatial probes which are sensitive to right hemisphere damage, we have found that RHD deaf signers, but not those with LHD, similarly typically have nonlinguistic spatial deficits in a variety of tasks including loss of perspective and disorganization in drawings, left neglect, and may be better at producing local features over global features (Hickok and Bellugi, 2001). In contrast, LHD deaf signers, like hearing subjects with LHD, are generally not impaired in perspective and organization of drawings and are significantly better at reproducing global level features (Hickok, Kirk *et al*., 1998).

RHD-228 was tested for nonlinguistic visual-spatial abilities using several standard assessment tools including the BDAE parietal drawings (Goodglass and Kaplan, 1983), the Rey-Osterrieth Complex Figure (Rey, 1941) and Albert's Line Cancellation Task (Albert, 1973). His performance on these tasks was quite good and within normal limits of our non-brain-damaged deaf elderly controls. Figure 7 presents some sample drawings which demonstrate RHD-228's generally preserved visual-spatial capacity. This degree of preservation of visual-spatial skill is atypical of RHD deaf signers with comparable lesion size and location. In our sample, every RHD signer with a large lesion in the distribution of the middle cerebral artery has presented with significant visual-spatial deficits evident on the basic tasks described above. Thus, RHD-228's performance is a prominent outlier in our sample of RHD signers.

To explore the possibility that RHD-228 might have more subtle visual-spatial deficits that are not readily detected with the probes noted above, we administered the hierarchical figures task modeled after those used by Delis and colleagues (1988). The task involves reproduction of a hierarchical figure with two levels of processing, a global level, such as a large form (e.g., the letter "A") constructed of local forms (e.g. small

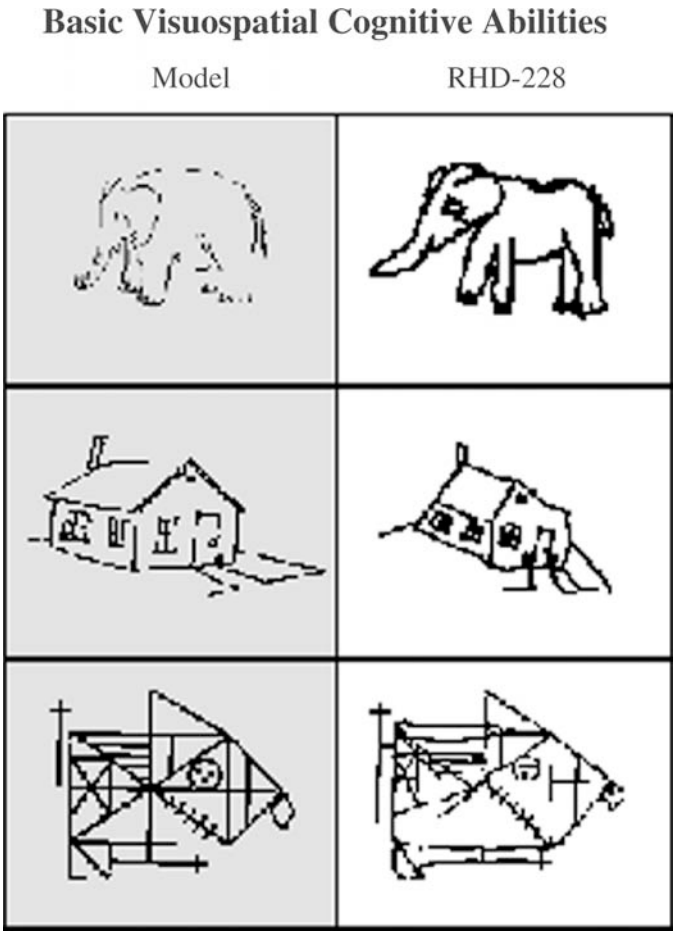


Fig. 7. The left side shows the models of elephant and house from the BDAE parietal drawings and the Rey-Osterrieth Complex Figure. On the right are the renderings produced by RHD-228 copying from the figure. The model was visible during the reproductions.

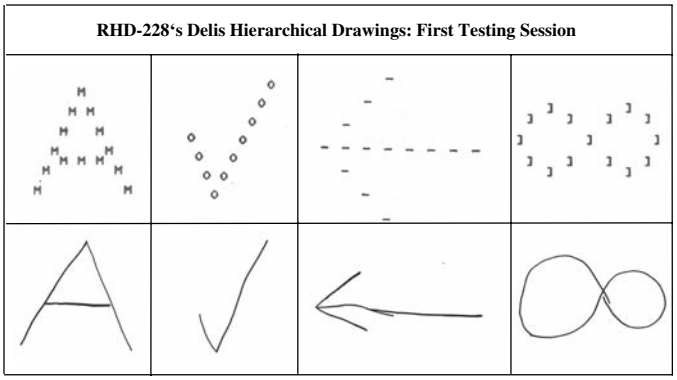


Fig. 8. The top row shows the models that were shown to RHD-228, The bottom row is the actual drawings made while copying from the models. Note the absence of any local elements in his drawings.

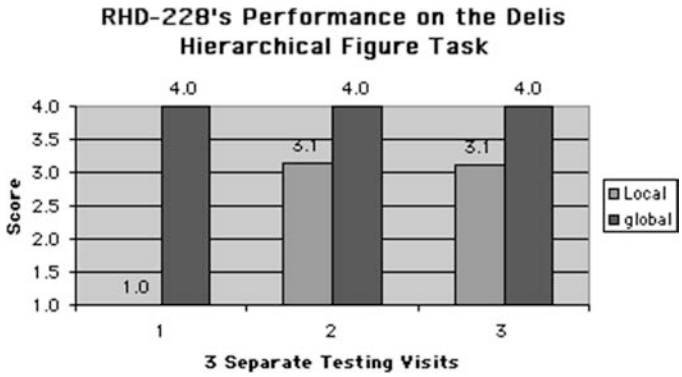


Fig. 9. RHD-228's scores from his three attempts at the hierarchical figures test. His scores clearly show that the only area he has difficulty with is the local features of these drawings.

letter "M's"). The subject views the model for five seconds, after which it is removed, and he is asked to reconstruct it. This test was designed to assess deficits at two levels of processing, the global level and the local level, and has been shown to discriminate left- and right-injured patients reasonably well, with left-damaged patients showing more local-level deficits and right-damaged patients tending toward more global-level deficits (Hickok, Kirk *et al*., 1998). As noted, we have found a similar pattern in our sample of deaf brain-injured subjects. RHD-228 was administered this task on two different occasions. In the first testing visit, this test was administered by having RHD-228 simply copy from the model. For all the drawings given, he represented only the global configuration, omitting any local features in each of the drawings (Figure 8). Using the scoring system devised by Hickok and colleagues (1998), RHD-228 scored perfectly on the global configuration and had the lowest scores possible with respect to the local features (see Figure 9). Upon completion of the items, the examiner asked him to look again and see if he was missing anything. RHD-228 appeared to realize the nature of his errors and the test was given again. He picked up on the local features this time, but still committed some errors. These errors were only in the local features, in that he tended to choose an incorrect symbol to use for the local features.

At another session, this test was performed again. This time the instructions given to him were that he would be shown a picture for five seconds; then it would be taken away and then he could begin drawing. He drew all of the global configurations accurately, but with the smaller (local) features he made a number of errors including perseverative intrusion (of a previous local element) and global figure reiterated as local (Figure 10). In a separate control task administered at the second session, RHD-228 was asked to reproduce individual items that replicated the size and shape of the local features in the hierarchical figures. He performed flawlessly on this task, indicating that his difficulty with local-level features was not simply a result of a low-level visual acuity problem.

In summary, RHD-228's visual-spatial abilities are grossly intact, but subtle deficits involving local-level processing are apparent on closer examination. This pattern is typically associated with left-hemisphere damage rather than right-hemisphere damage.

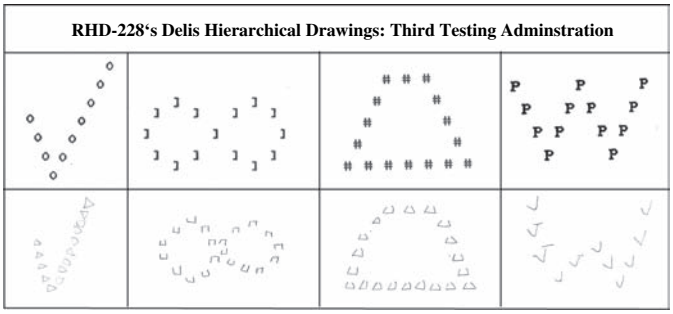


Fig. 10. The models are represented in the top row. Bottom row is actual drawings made after viewing the model for 5 seconds, removing the model, and beginning drawing. The first and third examples show substitution errors, the second example shows an orientation error, and the fourth one is a perseveration error (J's are used in the second model, two items before this one).

Discussion

We have presented the case of RHD-228, a deaf, left-handed, life-long signer, who suffered a moderately large perisylvian infarct in the right hemisphere that resulted in significant deficits in comprehension and production of ASL. This is the first in-depth case of a lifelong deaf signer who has become aphasic following a *right* hemisphere lesion. RHD-228's visual-spatial abilities were grossly intact, which has not been observed in our sample of right-handed RHD signers with similar lesions. This pattern, together with substantial aphasia for sign and largely spared visual-spatial abilities, is typically found in *left*-lesioned signers suggesting that RHD-228 may represent a case of *reversed cerebral dominance*. Closer examination of his visual-spatial skills using hierarchical figures revealed subtle deficits involving local-level features. This is also typical of left-, rather than right-injured patients, providing additional evidence supporting a reversed dominance pattern in RHD-228.

Although we did not have the opportunity to assess RHD-228's calculation abilities extensively, the mental status exam turned up some suggestive evidence of acalculia. In particular, he had difficulty with mental subtraction, and was unable to calculate the number of quarters in $1.75. These difficulties with calculation likely do not reflect pre-stroke deficits as indicated by RHD-228's educational and employment history which required mathematical competence. Acalculia has been associated with damage to the dominant hemisphere (Levin *et al*., 1993). If indeed, RHD-228 has some degree of acalculia as the mental status exam suggested, this would provide additional evidence for reversed dominance in this case.

The one data point that potentially argues against RHD-228 having a complete reversed dominance is the mild left neglect found in the mental status exam. Although he was able to accurately bisect lines, and showed no evidence of neglect on line cancellation tasks, he did sometimes fail to report and/or mis-localized the location of a stimulus under conditions of double simultaneous visual stimulation. Some evidence of left motor neglect was also noted. These findings may indicate that spatial and/or motor attention systems are less asymmetric in RHD-228 than is found in the typical dominance pattern. However, it is quite clear that whatever hemi-attention deficits exist in this patient, they are far less severe than we have observed in our right-handed deaf signers with similar right-sided injuries.

Conclusions

Findings from our sign language aphasia program have shown that hemispheric dominance patterns for language and spatial cognition in deaf signers are comparable to those found in hearing-speaking individuals: left hemisphere-damaged signers often present with aphasia and relatively mild non-linguistic spatial cognitive deficits, whereas right hemisphere-damaged signers often present with more severe spatial cognitive deficits and no aphasia. These conclusions are based on data from predominantly right-handed subjects. The case of RHD-228 demonstrates that right hemisphere dominance for language can occur in deaf, life-long signers, but his history of left-handedness suggests that this dominance pattern is more likely to occur in left-handers, just as one finds in the hearing population. Nonlinguistic testing of RHD-228 revealed a pattern of deficit and sparing that is suggestive of reversed cerebral dominance. If true, RHD-228 would represent an exceptionally rare case indeed.

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