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# SIGN PHONETICS AND THE MOTOR SYSTEM: IMPLICATIONS FROM PARKINSON'S DISEASE

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#### **Abstract**

This study reports the first documented case of a Deaf native signer of British Sign Language with Parkinson's disease (PD) and describes the effects of the disease on his signing, fingerspelling, and non-linguistic movements. Previous studies have described the disruption to signing caused by PD, emphasizing the distinctions between Parkinson's disease, whose effects are largely motoric, and aphasia, which is fundamentally linguistic in nature. The current study adds to the existing body of research by examining a native user of a signed language other than American Sign Language, who is significantly younger than most individuals with PD, including those described in earlier studies. For one or more of these reasons, the pattern of disrupted signing described here is very different from any that has been described to date, which raises new questions for the study of human linguistic and motor systems, and how they interact to shape sign language structure.

Language can be produced either via an auditory-vocal medium (as in spoken language) or a visual-manual medium (as in signed language). This study seeks to address whether there is such a thing that can be described as 'articulation' in signed as well as spoken language. In other words, are the movements of the hands during signing organized and structured in a comparable way to speech movements, or are they structured more like other, non-linguistic hand movements, such as gesturing, pointing, or picking up an object? In order to address this question, a Deaf British Sign Language user with Parkinson's disease (PD), "John," was studied as part of a larger study on sign language and motor control (Tyrone 2005), and his performance across linguistic and non-linguistic movement tasks was compared. This study examines the breakdown of sign production, or sign dysarthria, to gain a better sense of sign language structure more generally.

Previous research on American Sign Language (ASL) has contrasted motoric disruptions to sign production resulting from PD with fundamentally linguistic disruptions, such as aphasia (Brentari et al. 1995). The case described here supplements the field of sign phonetics in several ways: it is the first reported case of an individual with PD who is a native signer, or who uses a sign language other than ASL. He might differ from other subjects with PD for either of these reasons. John is also much younger than the Parkinsonian signers described previously, so his case could illustrate findings from those studies that were age-specific. Moreover, John's case can help elucidate the relationship between the general movement disorder of PD and its specific effects on articulation.

#### 1 Parkinson's disease

PD is characterized by the degeneration of dopamine-producing cells projecting to the basal ganglia: a large network of subcortical nuclei, which are important for motor control. The most common symptoms of the disease are resting tremor (as distinct from tremor during movement), muscular rigidity, bradykinesia (or slowed movement), impaired postural reflexes, difficulty initiating movement, and reduced spontaneous movement (including facial movement). Dementia and depression are additional, psychological symptoms that tend to begin later in the course of the disease. Because PD is degenerative and progressive, symptoms often multiply and worsen as the disease becomes more advanced. PD is typically responsive to medication, but for most patients, the medication begins to cause side effects, particularly dyskinesias (uncontrolled movements) in the face and limbs, after about five years. Because of how the medication is metabolized, patients tend to have on-off phases of severe side effects and controlled disease symptoms, alternating with no side effects and return of disease symptoms. Consequently, motor behaviour can vary greatly depending on when patients are examined relative to when they take medication.

There may be a distinct, genetically-inherited form of PD, which has an early onset (Nussbaum and Polymeropoulos 1997, Papapetropoulos et al. 2001). Most cases of PD begin when the patient is between 50 and 80 years of age; a case is described as early-onset if it occurs at or before age 45 (Periquet et al. 2003). The symptoms of early- and late-onset PD are similar, the main difference being that patients with the early-onset form are more likely to develop dementia earlier in the course of the disease.

# 1.1 PD and spoken language

Typically, disruption to speech motor control, or dysarthria, resulting from PD is less severe than other symptoms of the disease, such as disruptions to gait, balance, and the speed and magnitude of simple limb movements. Dysarthria associated with PD is characterized by a limited range of movement, which manifests itself in monotonous, quiet, aprosodic speech, with harsh, breathy voice quality (Darley et al. 1975, Hartman and Abbs 1988). Parkinsonian dysarthria is also unusual in that it causes perceptibly rapid speech, while other movements tend to be slowed (Theodoros and Murdoch 1998). Even though PD affects speech differently from other movements, it is unlikely that the speech deficits are articulator-specific, given that the general motor symptoms of the disease are quite diffuse. Parkinsonian speech deficits may instead be particular to certain types of movements or movement combinations that make specific demands on the motor control system.

Some researchers have suggested that in addition to disrupting speech motor control, PD may also affect language comprehension and production, particularly syntactic processing (Lieberman et al. 1990, Lieberman et al. 1992, Natsopoulos et al. 1993). However, there has not been substantial clinical (as opposed to experimental) evidence suggesting that patients with PD exhibit linguistic deficits in the absence of dementia (Murray and Lenz 2001, Patterson and Bly 1999). Additionally, research on syntactic deficits in PD has not consistently controlled for subject age (e.g., Lieberman et al. 1992). Murray and Lenz (2001) found no significant difference in language ability of subjects

with PD and age-matched controls. Consequently, the impairments identified by Lieberman and others could be an effect of age-related cognitive decline rather than PD. (For a review of studies on syntax and the basal ganglia, see Patterson and Bly 1999.)

# 1.2 PD and signed language

Because of the high prevalence of the disease (0.1% to 1% of adults), signers with PD have been researched more than signers with other movement disorders. Early research on sign language and the brain explored aphasia and apraxia in Deaf signers in order to address similarities and differences between sign and gesture (Corina et al. 1992b, Poizner et al. 1987). Research on signers with PD has tended to focus on illustrating the differences between disruption to language and disruption to articulation in a signed language by outlining the characteristics of sign errors in individuals with aphasia and with PD (Brentari et al. 1995, Corina 1999, Kegl et al. 1999, Poizner and Kegl 1992, Poizner and Kegl 1993).

Studies of PD and American Sign Language have reported a range of findings on sign and fingerspelling articulation. Firstly, signers with PD exhibited laxed articulation (Brentari et al. 1995, Loew et al. 1995, Tyrone et al. 1999), i.e., the configuration or orientation of the hand resembled its shape when resting. Laxing also occurs among typical signers in casual or informal contexts (Kegl et al. 1999). Unlike signers with aphasia, PD signers did not produce handshapes that had an incorrect selection of fingers (Poizner and Kegl 1993).

Signers with PD exhibited reduction and lowering of signing space (Loew et al. 1995, Poizner and Kegl 1992, Poizner and Kegl 1993) and lowering of sign locations on the body. A related characteristic of Parkinsonian signing was articulator distalization in both sign (Brentari and Poizner 1994, Poizner 1990, Poizner and Kegl 1993), and finger-spelling (Tyrone et al. 1999). In signs that require a given articulator to move, signers with PD often use a more distal articulator (e.g., the wrist instead of the elbow) and make a smaller movement. This set of deficits in Parkinsonian signing encompasses two related, but separate phenomena. Lowering and shrinking of signing space is a measure of where signs are produced, whereas distalization is a measure of which articulators move to form the sign. There is an interaction between the two measures, but they are not identical; it is possible to make a small movement with a proximal articulator, or to make a large movement to a nearby location in space.

Signers with PD had impaired co-ordination during sign production (Brentari et al. 1995, Poizner et al. 2000, Tyrone et al. 1999), although impaired co-ordination is infrequently associated with the disease in the motor control literature (Benecke et al. 1986, Ingvarsson et al. 1997). In signs produced by subjects with PD, the movements of independent articulators were decoupled, so that movement and handshape change in a sign would be produced either completely serially or simultaneously, rather than having partial temporal overlap. Another type of co-ordination error occurred in the transition between signs: signers with PD would blend distinct handshapes of two signs into each other, so that an intermediate form of the two handshapes would be produced (Brentari et al. 1995, Loew et al. 1995). The final type of co-ordination error reported was handshape mirroring on the non-dominant hand during production of one-handed signs (Brentari and Poizner 1994).

#### 2 Methods

# 2.1 Subjects

John is a 54 year old right-handed man with PD, who was born deaf into a family with deaf parents and hearing siblings. He is the youngest of four children (the others are hearing) and learned British Sign Language (BSL) from his parents as a native language. He attended an oral residential school for the deaf and left school at age 16 to begin work. He worked in various manual trades until his illness made work impossible. His wife, two of his three children, and many of his friends are Deaf, and he uses BSL as his primary language. In addition, he and his family are active in the local Deaf community. John was recruited for participation through an announcement in the local Deaf news. A healthy, 70 year old Deaf woman, Christine, was included in the study as a control subject. She is not a native signer, but her husband and children are Deaf, and she is active in the local Deaf community. John and Christine gave written informed consent to participate in the study, which was approved by the ethics committee at City University London.

John developed PD at the age of 48 and was diagnosed and treated promptly. He has taken medication for several years, which controls his Parkinsonian symptoms but causes moderate dyskinesias, particularly in his arms and neck. John developed PD at a fairly young age and shows signs of mild dementia, both of which are symptomatic of the early-onset form of the disease. However, there are insufficient data for firm conclusions about John's disease aetiology. John has resting tremor in his hands and legs; and his voluntary movements are reduced in size and speed, particularly when he is off-medication. He also reports occasional problems with balance. When on medication, his gait is slow and festinating, but he has not experienced falls. He reports no problems swallowing, and his facial expression and eye movements are normal. John has no difficulty in language production or perception, other than articulatory deficits.

John is right-handed, as measured by a version of the Edinburgh Handedness Test (Oldfield 1971), modified for sign language users. Additionally, he shows mild dementia, according to the Mini-Mental State Exam (Folstein et al. 1975). Although his interactions with others were basically normal, he exhibited mild cognitive slowing, in the form of slow conversational responses and occasional inattentiveness.

John was tested under two conditions: on- and off-medication. Consequently, his performance on all tasks was compared both to the control subject and to his own performance in the other condition. As discussed above, medication status can affect motor behaviour, particularly in subjects who have taken medication for many years. These effects have been studied clinically and experimentally, though they are not always controlled for in studies on speech; this is the first time they have been examined in the context of sign language production.

### 2.2 Procedure

Testing was carried out in subjects' homes, and stimuli for all tasks were presented by a Deaf research assistant. For one testing session, John had been asked in advance not to take his morning dose of medication; then he resumed his normal medication schedule after testing. The tasks included signing and fingerspelling as well as a series of non-

linguistic tasks: the Kimura Box (Kimura 1993), pointing, and handshape copying. The first three tasks will be discussed here; the other non-linguistic tasks have been described in depth elsewhere (Tyrone 2005). For the signing task, the research assistant produced single signs which the subjects repeated. Altogether, twenty nine productions were analysed for Christine, and for John with medication and without. For the fingerspelling task, the subjects were presented with individual full-page illustrations with corresponding printed text underneath and asked to fingerspell the printed word in BSL, which uses a two-handed alphabet. For each letter in each fingerspelled word, the handshape, location (that is, where the dominant hand makes contact on the non-dominant hand), temporal duration, and presence of involuntary movements were coded. Altogether, six finger-spelled words comprising thirty five fingerspelled letters were analysed according to these criteria.

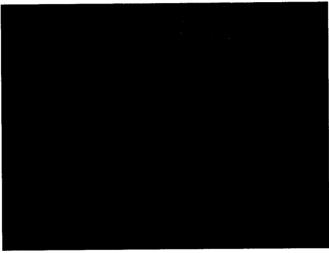


Figure 1: Kimura Box

The Kimura Box test was originally designed to assess apraxic subjects' ability to correctly manipulate objects according to shape and size, and to imitate movements (Kimura 1993). For this study, it was used to generate complex, targeted movements that could be compared to subjects' signing movements. The research assistant showed subjects the box (Figure 1), performed an action on each of the three manipulanda in sequence, and asked subjects to copy her actions, in the same order and using the same hand configurations. Both subjects performed the specified sequence of movements twice, yielding six individual movements per subject and condition to be analysed. The data were analysed according to the criteria set by a previous apraxia study (Sunderland and Sluman 2000), with additional coding for targeting, hesitation, tremor, speed of execution and accuracy of hand configuration. These categories were added to the coding scheme to allow more detailed assessment of the spatiotemporal aspects of movement as well as their representational and perceptual aspects.

#### **Table 1 Sign Coding Parameters**

Handshape: compares configuration(s) of the hand(s) to target handshape(s) from the citation form of the sign

Handshape change: compares change in handshape to target handshape change Orientation: compares orientation(s) of the hand(s) to target orientation(s)

Orientation change: compares change in orientation to target orientation change

Location (relative): compares placement of a sign to its target location

Direction: compares direction, number and manner of movements of joints alo

Direction: compares direction, number, and manner of movements of joints above the wrist to their target movements

Repetition: indicates whether a sign is repeated in full without pause

Involuntary movement: indicates whether involuntary movements occur

Proximal/Distal Co-ordination: compares co-ordination of the articulators on one limb with target movements of articulators

Bimanual Co-ordination: indicates whether use of the two hands matches the target sign

# 2.3 Analysis

The coding scheme was developed on the basis of previous research in sign phonology, motor control, and gesture. The coding scheme uses the phonological parameters developed by Stokoe and others to describe signs' sublexical structure: handshape, movement, location, and orientation (Battison et al. 1975, Friedman 1976, Stokoe 1960). Additionally, when a sign-internal change was required to any of those parameters, subjects' ability to time and co-ordinate that change was coded. Signs can use paired articulators on opposite sides of the body, so it was necessary to code for bimanual co-ordination: namely, whether or not both hands were used in a sign, and if so, whether or not they were co-ordinated. The remaining coding parameters were adopted on the basis of past research on disrupted signing and gesture (Brentari et al. 1995, Pedelty 1987, Tyrone et al. 1999).

The coding scheme for the fingerspelling data is a reduced and slightly modified form of the scheme used to code sign data: both the number of possible formational parameters and the values they can take are more limited. The coding scheme for the non-linguistic tasks combines the measures used by motor control researchers with those used by clinicians to assess movement disorders (Duffy 1995, Love and Webb 2001, Wing et al. 1996). Wherever possible, the measures from the sign and fingerspelling coding schemes were also used for coding the non-linguistic tasks.

# 3 Results

# 3.1 Signing

By far, the majority of John's sign errors were in handshape. Of 29 productions when he was off medication, 68.97% (or 20) of them included a handshape error, which is more than three times the number of the next most frequent error type (Figure 2). Handshape errors were also the most frequent error when he was on medication, though they were far

less frequent (31.04%, or 9 signs) than when he was off medication. John's next most frequent error was in sign orientation, which is a static component of signs. Most of his handshape and orientation errors, as well as Christine's, were cases of laxing. Laxing is a type of hypoarticulation, such that the sign articulators fall short of achieving the target hand configuration (Figure 3). For a sign that requires a flat handshape, this would mean not extending the fingers fully; for a sign that requires a closed fist, this would mean not flexing them fully.

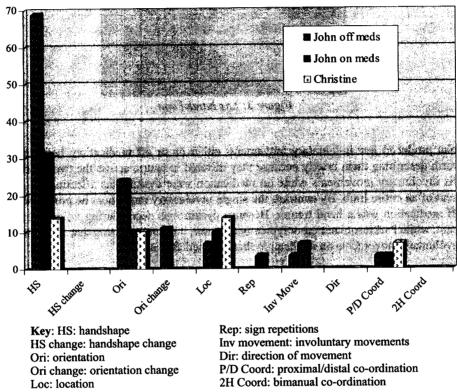


Figure 2: Signing Task (errors as % of 29 productions)

When John was on medication, his next most frequent errors were in location. Interestingly, in the case of location errors, he had a lower rate of errors off-medication than on-medication, and fewer errors than Christine in either condition. The nature of both subjects' location errors varied; i.e., not all location errors were cases of sign lowering or raising. John had a few other errors, but most were infrequent: orientation change, location, involuntary movement, repetition and proximal/distal co-ordination errors.

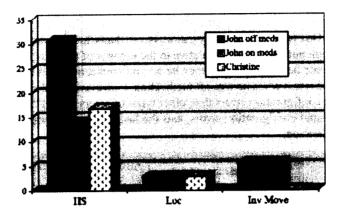


Figure 3: Lax handshape

John produced few involuntary movements either on or off medication; however, it is worth describing them briefly because they differed in nature across the two conditions. His involuntary movements while on medication were dyskinesias affecting the movement of an entire limb. By contrast, the single involuntary movement he produced when off medication was a hand tremor. Because dyskinesias occur during voluntary movement, while Parkinsonian tremor typically does not, it is not surprising that John had more involuntary movements on medication than off medication.

# 3.2 Fingerspelling

John had proportionally fewer errors on the fingerspelling task than on the signing task. As with the signing task, most of his errors on the fingerspelling task were in handshape (30.56% of total productions or 11 letters, off medication; 13.89% or 5 letters, on medication) (Figure 4). His other fingerspelling errors were few in comparison. When he was on medication, his performance was not very different from Christine's. Though he had far more handshape errors off medication than on medication, he had equal numbers of location errors and equal numbers of involuntary movements in both conditions. His handshape errors on the fingerspelling task were similar to those on the signing task, in that they were all cases of articulatory laxing.



Key: HS: handshape Loc: location

Inv Move: involuntary movement

Figure 4: Fingerspelling Task (errors as % of 35 productions)

The durations of John's fingerspelled letters varied from one letter to another. They varied in the same way as Christine's fingerspelled productions, but to a greater extent. The first letter of a word consistently had the longest duration for both subjects. This pattern is illustrated in Figure 5, which shows the durations of the letters in the words, C-A-R-P-E-T, S-I-T-E, and D-R-A-W-E-R, for Christine, and for John, both on- and off-medication. Additionally, the physical proximity of the letters' locations generally reduced the duration of the movement from one letter to another (e.g., the movement from P to E in C-A-R-P-E-T).

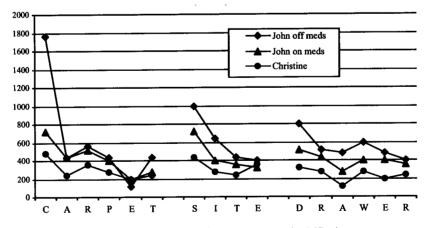
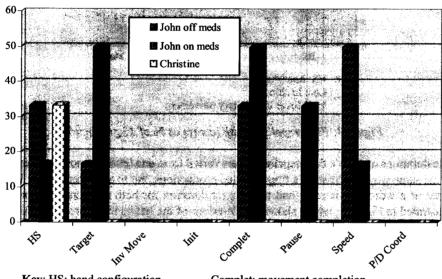


Figure 5: Fingerspelling Durations (in MSec)

#### 3.3 Kimura Box

On the Kimura Box task, John had many more errors than Christine, both on and off medication, and a high number of errors relative to the number of actions (Figure 6). John had more targeting and hesitation errors when he was on medication, and more hand configuration and speed errors when he was off medication. However, he did not have proportionally more hand configuration errors than other types of errors. All of Christine's errors on this task were hand configuration errors, and she had as many of them as John did when he was off medication.



Key: HS: hand configuration Target: movement targeting Inv Move: involuntary movement Init: movement initiation Complet: movement completion
Pause: irregular pause or hesitation
Speed: excessively slow or rapid movement
P/D Coord: proximal/distal co-ordination

Figure 6: Kimura Box Task (errors as % of 6 movements)

# 4 Discussion

In qualitative terms, John's signing was relatively normal. Most of his errors affected static components of signs and were cases of laxing, i.e., the types of productions that typical signers make during relaxed, informal signing. He had fewer errors on the signing task, and a high proportion of them were handshape errors, rather than errors in co-ordination or timing. The distribution of his errors was roughly the same on- and off-medication, and similar to the pattern of errors produced by Christine. His errors differed from hers in terms of number but not distribution. John had few sign location errors, and these patterned similarly to Christine's errors, in terms of number and type (i.e., in the relationship between the target location and the location produced). His signing was observably slow, especially when he was off medication, but not extremely so in either condition.

John performed similarly on the fingerspelling task and the signing task, in that his handshape errors greatly outnumbered all other errors, and were more numerous when he was off medication. However, he had proportionally fewer errors in total on the fingerspelling task than on the signing task, and his performance on medication and off medication did not differ greatly on the fingerspelling task. On both tasks, his handshape errors took the form of laxing, unlike the handshape errors reported in subjects with aphasia, which were often phonological substitutions (Corina 1999, Corina et al. 1992a).

Researchers have observed that ASL fingerspelling is more rapid and has smaller articulatory targets and greater sequencing demands than ASL signing (Poizner et al. 2000, Tyrone et al. 1999). The same is true of BSL fingerspelling in comparison to BSL signing. Nevertheless, John was not differentially impaired on the fingerspelling task. He did not have a noticeably different pattern of errors on the fingerspelling and signing tasks, and he had proportionally fewer errors when fingerspelling than when signing. Consequently, the particular difficulty that signers with PD are reported to have with fingerspelling was not evident in his case. Given that BSL fingerspelling does not require as many different handshapes as BSL signing, and that John's most frequent error across most tasks is in handshape, it could be that the lesser demands for handshape contrasts in BSL fingerspelling contributed to his overall low rate of errors on this task. Alternatively, it could be that by virtue of its being two-handed, BSL fingerspelling requires a type of co-ordination that is not directly impacted by PD.

John had a high proportion of hesitation errors and completion errors on the Kimura Box, which may reflect the difficulty switching from one action to another which is frequently reported in PD (Zalla et al. 1998). In addition, John showed a lower proportion of handshape errors on the Kimura Box task than on other tasks. Some caution must be exercised in interpreting these findings, because the Kimura Box task had the smallest number of trials of the tasks that John performed.

To summarize, John's handshape and hand configuration errors consistently outnumbered his other errors, except on the Kimura Box task, in which he had a higher proportion of hesitation and completion errors. Moreover, like the control subject, his handshape errors were consistently cases of laxing. Strikingly, John's handshape errors occurred irrespective of whether a task was motorically difficult. For example, the small size of the articulatory targets and the relative speed of individual movements in fingerspelling did not seem to impair John's overall performance; in fact, he had fewer errors on that task than on the others.

John exhibited some of the same patterns reported in the speech motor control literature on PD dysarthria. Because the disease was not advanced when he was tested, his dysarthric symptoms were not severe. The comparative mildness of Parkinsonian dysarthria relative to other movement deficits in the early stages of the disease is a widely-reported phenomenon. This is the first study to suggest that it is true across sign and speech modalities. Moreover, John had no articulatory co-ordination deficits in his signing or finger-spelling. This is in contrast to the findings of Brentari et al. (1995), which suggested that articulatory co-ordination in particular was impaired in signers with PD, but consistent with findings from speech motor control, which suggest that Parkinsonian dysarthria does not strongly impact co-ordination in particular. John also had irregular pauses and difficulty initiating movement, but less so in signing than in other movements. Similarly, hear-

ing patients with PD do not typically have movement initiation problems in speech to the same extent that they do elsewhere.

John did not exhibit anything similar to festination in sign, though he did exhibit it in walking. Festination is the combined increase in movement speed and decrease in movement amplitude that PD patients often experience in both speech and gait. Perhaps this is one of the few symptoms of PD that is effector-specific; i.e., it could selectively affect the vocal tract and the legs. However, this seems unlikely. The basal ganglia project broadly throughout motor areas of the cerebral cortex, affecting multiple descending motor tracts, including the corticobulbar tract, which controls speech movements, and the corticospinal tract, which controls complex limb movements. There are no direct projections from the basal ganglia to the spinal cord or to the motor nerves, so damage to the basal ganglia, as in the case of PD, is unlikely to affect individual effectors in isolation. It seems likely that festination would instead affect particular types of movement. Speech and gait are effectively constrained to being two-dimensional in terms of motor planning—for those two tasks there is only minimal lateral movement—whereas sign movements make extensive use of three spatial dimensions. Moreover, speech and gait both make consistent use of cyclic movements, and it is not clear that sign does so (MacNeilage et al. 2000, Meier 2002).

Comparison of John's symptoms on and off medication reveal that while his movements were faster when he was on medication, they were not improved on all measures. John's uneven improvement on the signing task when he was on medication is consistent with earlier findings on PD speech dysarthria, which suggested that PD subjects' speech may improve, but not consistently, and not on all measures (Schulz and Grant 2000). The consistency of John's performance across linguistic tasks, the similarity of his sign characteristics to the speech characteristics of hearing subjects with PD, and the mildness of his articulatory deficit compared to other signers with PD all support the idea that PD dysarthria is present cross-modally, and that it tends not to be severe in the early stages of the disease in either modality.

In a variety of ways, John's signing breaks down similarly to dysarthric speech resulting from PD, whereas his non-linguistic limb movements are more like the non-linguistic limb movements of hearing subjects with PD. In other words, the type of impairment depends on the function for which the articulator is used and not on the articulator itself. In his signing, John often produced laxed handshapes and sometimes laxed orientations. Additionally, his movements were slow during signing and other movement tasks. However, he did not exhibit the co-ordination deficits that were so strongly emphasized in the research on PD and ASL (Poizner et al. 2000), nor did he produce signs at lowered locations (Kegl et al. 1999). One final important distinction between John and the ASL signers with PD is that he did not show articulator distalization.

One explanation for these discrepancies may be that earlier studies did not consistently control for the age of the Deaf subjects; consequently, their results could have been related to age rather than, or as well as, disease status. Although John's signing was not greatly affected by his medication status, the ASL studies did not control for medication status at all, and subjects were on medication when tested (Brentari et al. 1995), so it could be that the deficits they exhibited were related to the medication rather than the disease.

Unlike subjects in the ASL and PD studies (and unlike most atypical signers described previously), John is a native signer. Due to the lack of data on native vs. non-native signing in general, and the lack of data on atypical signers who acquired sign language natively, it is difficult to speculate about the effects of native language skills on articulation following neural damage. In addition, because of how most Deaf signers acquire signed language, they cannot be easily compared to hearing bilinguals who experience neural damage which impacts their non-dominant or non-native language. Nonetheless, it may be that having native language skills prior to neural damage works to preserve articulatory ability post-morbidly.

Although John's case is unusual, it reveals a great deal of what is unknown about signed language to date and suggests new areas of research to be explored. First and foremost, there is a serious lack of research on the normal range of phonetic variation in typical sign production. Without such basic data, any study of atypical signing is severely limited in its analyses and conclusions. To date, a substantial proportion of the kinematic studies of sign production have focused on atypical signers (Brentari et al. 1995, Poizner et al. 1987) or infants (Petitto et al. 2001). While these studies are inherently valuable, they would be much more informative if there were a substantial body of normative data to which they could be compared. Furthermore, given that most sign language users (unlike the subject described here) do not acquire their primary language natively, it would also be worthwhile to compare the kinematics of native and non-native signing.

This study supports earlier findings on PD and sign production but suggests that the primary sign deficit caused by PD is articulatory laxing. Findings from this study suggest that dysarthria, as distinct from loss of simple movement, does manifest itself in sign language. However, just as dysarthria is not articulator-specific, it is also not fundamentally linguistic in nature. The reason that dysarthria can occur in either a vocal or a manual language modality is because both use very rapid, complex, co-ordinated movements. The movement speed and complexity facilitate the rapid information transfer necessary for any linguistic system, but that does not mean that these disruptions are inherently linguistic. One would predict that subjects with dysarthria would also be impaired at any task with similar motor demands, but since few normal activities require such a high level of movement precision, deficits manifest themselves primarily in speech or sign.

# 5 References

- Battison, R., H. Markowicz and J. Woodward. 1975. A Good Rule of Thumb: Variable Phonology in American Sign Language. In *Analyzing Variation in Language*, eds. R. W. Fasold and R. W. Shuy, 291-302. Washington, D.C.: Linstok Press.
- Benecke, R., J.C. Rothwell, J.P.R. Dick, B.L. Day and C.D. Marsden. 1986. Performance of Simultaneous Movements in Patients with Parkinson's Disease. *Brain* 109: 739-757.
- Brentari, D. and H. Poizner. 1994. A Phonological Analysis of a Deaf Parkinsonian Signer. Language and Cognitive Processes. 9(1): 69-99.
- Brentari, D., H. Poizner and J. Kegl. 1995. Aphasic and Parkinsonian Signing: Differences in Phonological Disruption. *Brain and Language* 48: 69-105.

- Corina, D.P. 1999. Neural Disorders of Language and Movement: Evidence from American Sign Language. In *Gesture, Speech, and Sign*, eds. L. S. Messing and R. Campbell, 27-43. New York: Oxford University Press.
- Corina, D.P., H. Poizner, U. Bellugi, T. Feinberg and L. O'Grady-Batch. 1992a. Dissociation between Linguistic and Nonlinguistic Gestural Systems: A Case for Compositionality. *Brain and Language* 43: 414-447.
- Corina, D.P., J. Vaid and U. Bellugi. 1992b. The Linguistic Basis of Left Hemisphere Specialization. *Science* 255: 1258-1260.
- Darley, F.L., A.E. Aronson and J.R. Brown. 1975. *Motor Speech Disorders*. Philadelphia: Saunders.
- Duffy, J.R. 1995. Motor Speech Disorders: Substrates, Differential Diagnosis, and Management. St Louis: Mosby.
- Folstein, M.F., S.E. Folstein and P.R. McHugh. 1975. Mini-Mental State: A Practical Method for Grading the State of Patients for the Clinician. *Journal of Psychiatric Research* 12: 189-198.
- Friedman, L.A. 1976. *Phonology of a Soundless Language*. PhD Thesis, University of California, Berkeley.
- Hartman, D.E. and J.H. Abbs. 1988. Dysarthrias of Movement Disorders. In *Advances in Neurology*, eds. J. Jankovic and E. Tolosa, 289-306. New York: Raven Press.
- Ingvarsson, P.E., A.M. Gordon and H. Forssberg. 1997. Coordination of Manipulative Forces in Parkinson's Disease. *Experimental Neurology* 145: 489-501.
- Kegl, J., H. Cohen and H. Poizner. 1999. Articulatory Consequences of Parkinson's Disease: Perspectives from Two Modalities. *Brain and Cognition* 40: 355-386.
- Kimura, D. 1993. Neuromotor Mechanisms in Human Communication. Oxford: Oxford University Press.
- Lieberman, P., J. Friedman and L. Feldman. 1990. Syntax Comprehension Deficits in Parkinson's Disease. *Journal of Nervous and Mental Disease* 178(6): 360-365.
- Lieberman, P., E. Kako, J. Friedman, G. Tajchman, L. Feldman and E. Jiminez. 1992. Speech Production, Syntax Comprehension, and Cognitive Deficits in Parkinson's Disease. *Brain and Language* 43(2): 169-189.
- Loew, R.C., J.A. Kegl and H. Poizner. 1995. Flattening of Distinctions in a Parkinsonian Signer. *Aphasiology* 9(4): 381-396.
- Love, R.J. and W.G. Webb. 2001. Neurology for the Speech-Language Pathologist. Woburn, MA: Butterworth-Heinemann.
- MacNeilage, P.F., B.L. Davis, A. Kinney and C.L. Matyear. 2000. The Motor Core of Speech: A Comparison of Serial Organization Patterns in Infants and Languages. *Child Development*. 71(153-163.
- Meier, R.P. 2002. Why Different, Why the Same? Explaining Effects and Non-Effects of Modality Upon Linguistic Structure in Sign and Speech. In *Modality and Structure in Signed and Spoken Language*, eds. R. P. Meier, K. Cormier and D. Quinto-Pozos, 1-26. New York: Cambridge University Press.
- Murray, L. and L. Lenz. 2001. Productive Syntax Abilities in Huntington's and Parkinson's Diseases. *Brain and Cognition* 46(1-2): 213-219.
- Natsopoulos, D., G. Grouios, S. Bostantzopoulou, G. Mentenopoulos, Z. Katsarou and J. Logothetis. 1993. Algorithmic and Heuristic Strategies in Comprehension of

- Complement Clauses by Patients with Parkinson's Disease. *Neuropsychologia* 31(9): 951-964.
- Nussbaum, R. and M. Polymeropoulos. 1997. Genetics of Parkinson's Disease. *Human Molecular Genetics* 6(10): 1687-1691.
- Oldfield, R.C. 1971. The Assessment and Analysis of Handedness: The Edinburgh Inventory. *Medica Neuropsychologia* 9: 97-113.
- Papapetropoulos, S., C. Paschalis, A. Athanassiadou, A. Papadimitriou, J. Ellul, M.H. Polymeropoulos and T. Papapetropoulos. 2001. Clinical Phenotype in Patients with Alpha-Synuclein Parkinson's Disease Living in Greece in Comparison with Patients with Sporadic Parkinson's Disease. *Journal of Neurology, Neurosurgery, and Psychiatry* 70(5): 662-665.
- Patterson, M.D. and B.M. Bly. 1999. The Brain Basis of Syntactic Processes: Architecture, Ontogeny, and Phylogeny. In *Cognitive Science: Handbook of Perception and Cognition*, eds. B. M. Bly and D. E. Rumelhart, 255-318. San Diego, CA: Academic Press.
- Pedelty, L.L. 1987. Gesture in Aphasia. PhD Thesis, University of Chicago.
- Periquet, M., M. Latouche, E. Lohmann, N. Rawal, G. De Michele, S. Ricard, H. Teive, V. Fraix, M. Vidailhet, D. Nicholl, P. Barone, N.W. Wood, S. Raskin, J.F. Deleuze, Y. Agid, A. Durr and A. Brice. 2003. Parkin Mutations Are Frequent in Patients with Isolated Early-Onset Parkinsonism. *Brain* 126(6): 1271-1279.
- Petitto, L.A., S. Holowka, L.E. Sergio and D. Ostry. 2001. Language Rhythms in Baby Hand Movements. *Nature* 413(6851): 35-36.
- Poizner, H. 1990. Language and Motor Disorders in Deaf Signers. In *Cerebral Control of Speech and Limb Movements*, ed. G. R. Hammond, 303-326. North-Holland, NY: Elsevier.
- Poizner, H., D. Brentari, M.E. Tyrone and J. Kegl. 2000. The Structure of Language as Motor Behavior: Clues from Signers with Parkinson's Disease. In *The Signs of Language Revisited: An Anthology to Honor Ursula Bellugi and Edward Klima*. eds. K. Emmorey and H. Lane, 509-532. Mahwah, NJ: Lawrence Erlbaum.
- Poizner, H. and J. Kegl. 1992. Neural Basis of Language and Motor Behaviour: Perspectives from American Sign Language. *Aphasiology* 6(3): 219-256.
- Poizner, H. and J. Kegl. 1993. Neural Disorders of the Linguistic Use of Space and Movement. In Annals of the New York Academy of Science, Temporal Information Processing in the Nervous System. eds. P. Tallal, A. Galaburda, R. Llinas and C. von Euler, 192-213. New York: New York Academy of Sciences Press.
- Poizner, H., E. Klima and U. Bellugi. 1987. What the Hands Reveal About the Brain. Cambridge, MA: MIT Press.
- Schulz, G.M., & M.K. Grant. 2000. Effects of Speech Therapy and Pharmacologic and Surgical Treatments of Voice and Speech in Parkinson's Disease: A Review of the Literature. *Journal of Communication Disorders* 33(1): 59-88.
- Stokoe, W.C. 1960. Sign Language Structure: An Outline of the Visual Communication Systems of the American Deaf. Silver Spring, MD: Linstok Press.
- Sunderland, A. and S.-M. Sluman. 2000. Ideomotor Apraxia, Visuomotor Control and the Explicit Representation of Posture. *Neuropsychologia* 38(7): 923-934.

- Theodoros, D.G. and B.E. Murdoch. 1998. Hypokinetic Dysarthria. In *Dysarthria: A Physiological Approach to Assessment and Treatment*, ed. B. E. Murdoch, 266-313. Cheltenham, UK: Stanley Thornes.
- Tyrone, M.E. 2005. An Investigation of Sign Dysarthria. PhD Thesis, City University London.
- Tyrone, M.E., J. Kegl and H. Poizner. 1999. Interarticulator Co-ordination in Deaf Signers with Parkinson's Disease. *Neuropsychologia* 37: 1271-1283.
- Wing, A.M., P. Haggard and J.R. Flanagan. 1996. *Hand and Brain: The Neurophysiology and Psychology of Hand Movements*. San Diego: Academic Press.
- Zalla, T., A. Sirigu, B. Pillon, B. Dubois, J. Grafman and Y. Agid. 1998. Deficit in Evaluating Pre-Determined Sequences of Script Events in Patients with Parkinson's Disease. *Cortex* 34(4): 621-627.