An Investigation of Sign Dysarthria

Martha Ellen Tyrone

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Abstract

This study explores the nature of sign production in individuals with neurogenic movement disorders. The research goals are to broadly define the phenomenon of dysarthria in signed language; to determine whether anything other than the set of articulators involved differentiates it from dysarthria in spoken language; and to delineate the differences between sign dysarthria and apraxia, and between sign dysarthria and disruption of simple limb movements. In the same way that hearing people may exhibit speech dysarthria in the absence of oral apraxia, deaf signers may, in some cases, exhibit sign dysarthria in the absence of higher level ideomotor impairments. Conversely, just as many movement disorders are more apparent in speech than in simple limb movements, sign dysarthria may also arise in the absence of severe impairment of simple movements, such as reaching or pointing. An ancillary question that this research addresses is the establishment of articulatory measures of sign dysarthria, and of normal signing.

Findings from this study indicate that dysarthria, as distinct from apraxia, aphasia, and loss of simple movement, does manifest itself in sign language, which suggests that speech motor control research should eschew models of dysarthria framed around specific articulators, in favour of those that emphasize patterns of movement. 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 that is necessary for any linguistic system, but that does not make disruptions to it 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.

1 Introduction

This study explores the nature of sign production in individuals with neurogenic movement disorders. While the focus is on a small and atypical group of people, the implications of the research go well beyond the details of their cases to bear on broader issues of motor control, sign language structure, and the biological basis of language. Those implications, the relevant terminology, a short review of previous research on sign language and the brain, the research questions, the design of the study, and the structure of the thesis will be briefly sketched in this chapter.

There have been many studies over the course of many decades indicating that signed languages are full human languages with all the grammatical complexity, sublexical structure, and expressive potential of spoken languages (Fischer, 1988; Klima & Bellugi, 1979; Petitto, 1994; Stokoe, 1960). This study addresses a different issue, and is not concerned with the legitimacy or linguistic status of signed languages; in fact, it takes those questions as firmly resolved. As such, earlier research on the linguistic nature of signed languages was the necessary precursor to the research questions addressed here. From that research, it is clear that, contrary to earlier belief, language can be produced either via an auditory-vocal medium (as in spoken language) or a visual-manual medium (as in signed language). The overarching question that this study then seeks to address is whether there is such a thing that can be described as articulation in signed as well as spoken language; articulation as a modality-independent phenomenon is a new concept and will be explored in depth in this chapter. To put the question another way, are the movements of the hands during signing organised 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?

The subjects in the study are Deaf[1], British signers, who developed a movement disorder as the result of brain damage occurring in adulthood. Subjects were all fluent in British Sign Language (BSL) and used it as their preferred language prior to experiencing any motor control deficits. It is necessary to be explicit on this point, because there is a body of educational research showing that modified sign systems can be used to facilitate learning and communication in children with developmental motor

[1] This thesis follows the convention of using an upper case 'D' in the word 'Deaf' to refer to a cultural group who use a sign language, and a lower case 'd' in the word 'deaf' to refer to the clinical state of being unable to hear. Hence, a deaf individual may or may not be a member of the Deaf community.

or cognitive impairments (Grove, 1990; Grove & Dockrell, 2000). Consequently, because there are two lines of research related to both sign and motor control, their methods and objectives can be easily confused. In effect, this study is the inverse of the educational studies, in that it examines individuals who used BSL prior to any disruption to movement or cognition, and then subsequently experienced a disruption to their sign production as a sequela of neurological damage. Other studies referred to above focused on individuals who use signs or gestures to compensate for a pre-existing movement deficit and would not have used signs except for that reason.

1.1 Sign Language

Sign languages are the natural languages of Deaf people throughout the world. Although the majority of the world's Deaf children are born into hearing families and do not learn sign language from their parents, sign languages are nonetheless languages that have evolved naturally from the interactions of groups of Deaf people. Contrary to popular belief, sign languages were not invented by educators as a pedagogical device; in most cases, sign languages have developed in spite of the efforts of educators rather than because of them. Also contrary to popular belief, there is not a universal sign language common to all Deaf people. While signed languages, like spoken languages, borrow vocabulary from each other, they are nonetheless distinct from one culture or nation to another. Putting aside any question of the universality of grammar, no one would propose that spoken languages should share specific lexical items, morphological devices, or sound inventories on the grounds that those structures are universal. So, assuming that signed languages are prone to the same biological, psychological, and social constraints as spoken languages, there is no reason that they should be alike at the most superficial level. On a related point, sign languages are distinct from gestural communication systems that are used in narrowly-defined situations in which speech is forbidden or somehow difficult to use, like the gesture systems used by Cistercian monks and by the widows in Warlpiri communities (Barakat, 1975; Kendon, 1984).

To clarify some terminology, 'sign language,' 'signed language,' and 'sign' have distinct (but potentially overlapping) meanings. Essentially, they are expressing different perspectives on the same phenomenon and differ in where they place emphasis. Signed language is analogous to the term spoken language, i.e. it highlights the physical structure of the language modality. Similarly, sign, as it is used here, is analogous to speech. There is no exact analogy to the term 'sign language' as it is meant here; however, it is a reference to the form that the language takes, but not necessarily at

the level of articulation. It can also be used, for example, to refer to syntactic or morphological forms that are particular to the sign modality.

A defining feature of signed language is that it uses the hands and arms, rather than the vocal tract, as its primary articulators. While the implications of this basic structural difference between sign and speech have been discussed at length (Meier, 2002; Sandler, 1993), there is still much to do in outlining the full extent of the differences between sign and speech and clarifying which of those are primarily the effect of the set of articulators that the two language modalities use, rather than the effect of perception modality or of abstract linguistic structure. An analysis of the physical structure and the underlying neural basis of sign and of speech is presented in Chapter 2.

1.2 Research on sign language and the brain

Relative to linguistics and other branches of the behavioural sciences, sign language research is an extremely young field, beginning in earnest only in the 1960s and 1970s, with studies by William Stokoe and others (Klima & Bellugi, 1979; Stokoe et al., 1965; Stokoe, 1960). Perhaps because the field emerged when it did, psycholinguistic and neurolinguistic studies of sign language began very early in the history of sign language research. A core group of researchers in the USA were interested in psycholinguistics and the implications of sign language for the neural basis of language, so research projects were established to study ASL signers with various types of brain damage (Corina et al., 1992b; Poizner et al., 1987). The central over-arching question of these and most other studies of sign language and the brain has been: how does the brain process sign in comparison to speech, given that the two use different articulators, different sensory perception channels, and, to some extent, different grammatical structures (e.g. use of word order vs. physical space to express grammatical relations)? Particular questions to be addressed by studies of brain-damaged signers included: does the brain use the traditional language areas or visuospatial cognition areas for processing spatialized grammar?; and can sign aphasia occur independently of limb apraxia, and vice versa? The latter question was motivated in large part by an earlier claim that apraxia and aphasia were one and the same thing in the context of a visual-gestural language (Kimura, 1977).

Poizner, Klima, and Bellugi (1987) were the first to document a dissociation between aphasia and apraxia in Deaf signers. The study included six signers: three with left hemisphere lesions and aphasia, and three with right hemisphere lesions whose

language function remained intact. Of the three signers they reported with aphasia, only one was impaired on pantomime production and imitation; and none of the signers was impaired on pantomime recognition. In addition to documenting a dissociation between sign aphasia and apraxia, Poizner et al. (1987) were the first to compare and contrast cases of anterior, posterior, and mixed aphasia in sign language, and to document that they pattern more or less the same in ASL as they do in English. In both sign and speech, aphasia caused by damage to anterior left hemisphere structures results in phonological disruptions, limited and non-fluent language production, and relatively preserved language comprehension. Aphasia caused by damage to posterior structures usually results in fluent speech or sign that lacks semantic content, and more severe disruption of comprehension. Rather than forming a third distinct category of linguistic deficit, mixed aphasias take various forms and share features of both anterior and posterior aphasia.

Corina et al. (1992a) and Kegl & Poizner (1997) also reported dissociations of aphasia and apraxia in Deaf subjects with left hemisphere lesions. The subject described by Corina et al. (1992a) had poor sign comprehension and fluent, agrammatic sign production, which is typical of language users with a posterior lesion. By contrast, he could produce and understand non-linguistic gestures, as well as imitate sequences of gestures, suggesting that his praxic skills remained largely intact, and his deficit was purely linguistic. The subject reported by Kegl & Poizner (1997) had a left parietal lesion and exhibited severe comprehension deficits and mild sign production deficits, mostly at the level of syntax. Despite his signing deficits, however, he performed normally on tests of pantomime recognition and ideomotor apraxia, including on kinematic measures of joint co-ordination. As with the cases reported by Poizner et al. (1987), and consistent with the tendency in hearing aphasics, these two cases of aphasia without apraxia were both individuals with posterior lesions. Similarly, Hickok et al. (1996) found no significant correlation between aphasia scores and apraxia scores in a group of subjects with left hemisphere damage, as assessed by the Kimura gestures task (Kimura, 1993).

More recently, research on Deaf signers in the UK who had strokes has supported some of the same findings as the early case studies in the USA. Marshall et al. (under review) and Marshall et al. (2004) reported two cases of Deaf subjects with left hemisphere damage and aphasia, who showed differential impairment across sign and gesture tasks. In particular, Marshall et al. (under review) reported a Deaf signer with an anterior lesion who was severely aphasic, with severe comprehension deficits

and no spontaneous language production. Although she showed evidence of apraxia on the Kimura box task and Kimura gesture task (Kimura, 1993), her comprehension of gesture was far better than her comprehension of sign. Marshall et al. (2004) reported another aphasic signer with anomia, good comprehension of single signs, and heavy use of non-linguistic gesture. He showed evidence of apraxia, based on the Kimura box task; however, his comprehension and production of gesture were far better than his comprehension and production of signs. Unlike earlier studies, the UK Deaf Stroke Project was careful to control for the role that iconicity might play for a signer with impaired language ability. To that end, tests of sign language comprehension were designed to include a possible visual distractor, such that if a signer perceived a sign as a gestural representation of a physical object, they might choose the distractor in place of the correct sign. Interestingly, in the two cases reported in Marshall et al. (under review) and Marshall et al. (2004), although both subjects performed better on gesture comprehension tasks than on sign comprehension tasks, neither of them confused BSL signs with gestures representing the object they might be depicting. So although subjects could rely on an iconic strategy to comprehend gestures, they did not apply this strategy to their comprehension of signs.

In addition to documenting the dissociation of aphasia and apraxia in Deaf signers with left hemisphere damage, Poizner et al. (1987) were also the first to report preservation of linguistic function in signers with right hemisphere damage. This was noteworthy because right hemisphere damage, and in particular damage to the right parietal lobe, is known for causing disruption to visuospatial processing; and signed languages rely on spatial relationships between signs to mark grammatical relations. For example, once a sign representing an agent is placed at a particular point in the signing space, verbs and pronouns referring back to that agent have to move toward or away from that point (Sutton-Spence & Woll, 1998; Cormier, 2002). As a result, it is important for a signer to be able to keep track of where signs are located in space in order to know how those signs are related to other signs in the discourse. However, in spite of this demand on visuospatial processing, Poizner et al. (1987) reported two signers with right hemisphere damage and visuospatial processing deficits who could nonetheless understand and produce complex signed sentences. By contrast, the subjects' ability to depict real objects' physical locations relative to each other was severely impaired, suggesting a dissociation between general visuospatial processing and syntactic spatial processing.

Several studies have confirmed the initial finding of Poizner et al. (1987) that

visuospatial processing deficits in right hemisphere damage and syntactic deficits in left hemisphere damage were independent of each other. Hickok et al. (1996) showed a double dissociation between aphasia in subjects with left hemisphere damage and visuospatial deficits in subjects with right hemisphere damage. Other cases of preserved syntax and disrupted visuospatial processing have been reported on an individual basis in subjects with Williams syndrome (Atkinson et al., 2002) and in subjects with right hemisphere damage (Corina et al., 1996; Poizner & Kegl 1992; Loew et al 1997; Emmorey et al., 1996). Additionally, a recent group study of signers with strokes in the UK has shown that subjects with left hemisphere damage are more impaired on grammatical tests in general, whereas subjects with right hemisphere damage are more impaired on reversible locative sentences (e.g. the pen is on the paper) (Atkinson et al., in press). So while both groups had difficulties with sentences that expressed topographic relations between objects, subjects with left hemisphere damage also had difficulties with other sentences, and subjects with right hemisphere damage did not. The authors suggest that processing of topographic space requires an intact left hemisphere as well as right hemisphere.

Although most researchers agree that the left hemisphere serves a more fundamentally linguistic function for both signed and spoken language, there has been some debate in the literature over the role of the right hemisphere in sign language (Emmorey et al., 2002; Hickok et al., 1998; MacSweeney et al., 2002b; Paulesu & Mehler, 1998a, 1998b). This arose in large part from an imaging study suggesting that the right hemisphere showed more activation during processing of ASL than during processing of English (Corina et al., 1996; Neville et al., 1998), which was the first result to challenge earlier findings based on lesion data which had suggested that the right hemisphere was no more important for sign than it was for speech. Neville et al. (1998) found greater recruitment of right hemisphere areas in the processing of ASL by Deaf signers than in the processing of English by hearing signers. However, the stimuli used for the two tasks were videotaped ASL utterances and written English. Paulesu and Mehler (1998a, b) and MacSweeney et al. (2002b) suggested the differential activity could be explained by the presence of prosodic information in the signing task and absence of it in the reading task. In a recent study, MacSweeney et al. (2002b) found that recruitment of right hemisphere areas was equal for BSL processing and the processing of spoken English presented audiovisually, suggesting that the findings of Neville et al. (1998) and Corina et al. (1996) may have been either task- or medium-dependent.

Consistent with research on signers with neurological damage, functional imaging research on healthy signers has supported the idea that the right hemisphere serves an important role in the processing of topographic space in sign language comprehension. MacSweeney et al. (2002a) found that for Deaf subjects topographic sentences caused more activation in both the right and left occipitotemporal junction, posterior middle temporal gyrus, and inferior frontal gyrus. Non-topographic sentences recruited right hemisphere structures less. Similarly, in an imaging study on ASL, Emmorey et al. (2002) found more activation of the right supramarginal gyrus for spatial prepositions than for sentences containing no spatial information. The authors went on to conclude that the right hemisphere was more involved during expression of spatial relations in ASL. Additionally, a behavioural study by Emmorey et al. (1995) supported the idea that there is differential processing of topographic and non-topographic information in sign language, because subjects were better able to remember locations of signs placed topographically than locations of signs placed syntactically. Although this study did not address localization of the function, it did support the dissociation of processing of topographic and non-topographic sentences, which in turn means that the right hemisphere could play an important role in the first case but not the second.

Others have suggested that right hemisphere damage can cause pragmatic or discourse deficits in sign language users, such as inability to process prosodic information related to negation (Atkinson et al., 2004). Loew et al. (1997) reported a subject with a right parieto-occipital lesion, who had intact phonological, morphological, and syntactic function, but exhibited an inability to implement role shift. They proposed that this deficit may be pragmatic in nature, which would complement earlier findings on the role of the right hemisphere in pragmatic function in spoken language. Hickok et al. (1999) also reported two subjects with right hemisphere damage who had discourse level disorders: one had difficulty maintaining topical coherence, and the other had difficulty employing spatial discourse devices (e.g. referring back to antecedents indexically rather than re-using the full noun phrase). Despite their differences in discourse abilities, both subjects from these studies had impairments on non-linguistic spatial tasks, suggesting that spatial discourse function and more general cognitive spatial function are separate.

With the increased availability of functional brain imaging as a research tool, more studies have set out to examine the neural basis of sign in greater anatomical detail. Early research focused exclusively on atypical signers, whose area of brain

damage could only be identified in broad terms (i.e. anterior or posterior regions of one cerebral hemisphere); whereas recent imaging research has placed more emphasis on the parts of the brain that healthy signers use while processing or producing various components of their language. Current techniques in functional brain imaging allow researchers to ask much more narrowly defined questions about which parts of the brain are responsible for sign language function across tasks, individuals, and categories of signers (e.g. hearing vs. Deaf). As a result, researchers have tried to identify areas of activity that might be different across spoken and signed language tasks, with the expectation that left hemisphere language areas will still predominate, and that both hemispheres will serve some role for both modalities. In effect, researchers in these studies were looking for functional differences within a cerebral hemisphere, and often within a lobe or a gyrus.

Many studies have demonstrated the activation of traditional language areas, including the inferior frontal lobe (Levanen et al., 2001; MacSweeney et al., 2002a; MacSweeney et al., 2002b; Neville et al., 1998; Petitto et al., 2000) and the superior, posterior temporal lobe (Braun et al., 2001; MacSweeney et al., 2002a; MacSweeney et al., 2002b; Nishimura et al., 1999), during sign language processing and production. Additionally, researchers have found brain activation in other areas not typically associated with language function, or not typically associated with sign language specifically. One finding that has recurred across several studies is the recruitment of the superior temporal gyrus during sign language processing (Braun et al., 2001; Emmorey et al., 2003; Levanen et al., 2001; MacSweeney et al., 2002a; MacSweeney et al., 2002b; Neville et al., 1998; Nishimura et al., 1999; Petitto et al., 2000). This was of interest because the superior temporal gyrus is an area that has traditionally been associated specifically with auditory function (Brodal, 1998), and yet it seems to have adapted to serve a role in visually-perceived language in Deaf signers. Petitto et al. (2000) found bilateral activation of the superior temporal gyrus in Deaf signers of ASL and Quebecois sign language (LSQ) watching signs and phonologically possible non-signs, and less activation of the same areas in sign-naive subjects watching the same stimuli. In addition, Petitto et al. (2000) found activation of the adjacent left planum temporale in Deaf signers performing a lexical retrieval tasks from sign stimuli and in hearing subjects doing the same task from a written English word. Emmorey et al. (2003) also found bilateral activation of the superior temporal gyrus on a similar lexical retrieval task performed by ASL signers.

A few studies have explicitly examined brain function for sign language in deaf

signers compared to hearing signers, who were usually hearing children of Deaf parents who continued to use sign language regularly in a professional and/or family context (MacSweeney et al., 2002a; Neville et al., 1998; Soderfeldt et al., 1994). Activation of the superior temporal gyrus has been one difference identified across the two groups, with Deaf signers showing more consistent activation than hearing native signers. One study collected data from Deaf signers perceiving BSL, hearing native signers perceiving BSL, and hearing non-signers perceiving English presented audiovisually (MacSweeney et al., 2002b). The authors reported that hearing native signers do not recruit the superior temporal gyrus as much for sign language processing as Deaf signers do, presumably because the area maintains its auditory function in hearing signers rather than adapting to respond to a different type of sensory input, or alternatively because there is greater variation in areas of activity in hearing signers and therefore less of a group effect. Neville et al. (1998) reported a similar finding, but with activation in the more delimited superior temporal sulcus. Additionally, one study that only examined native hearing signers found that they showed more activation of the superior temporal gyrus for speech perception than for sign perception (Soderfeldt et al., 1997). Two other studies found greater activity in superior temporal areas in Deaf signers perceiving individual signs than in hearing non-signers doing the same task (Levanen et al., 2001; Petitto et al., 2000), which both sets of authors attribute to the difference in linguistic experience across the two groups. Despite a few differences in area of activation and task design across these studies, it seems clear from their combined results that secondary auditory areas can take on a non-auditory, linguistic function in Deaf signers.

Another question that has emerged in the study of sign language and the brain is the activity of visual areas during sign language processing. In particular, if auditory areas can adapt to process sign language stimuli, then what role do visual areas serve in sign language processing? The findings related to this question have been less consistent; although many studies have reported activation of visual areas in the posterior temporal and parietal lobes during sign language perception, the details of specific areas, relevant tasks, and differences between subject groups have varied considerably across studies. MacSweeney et al. (2002b) found that, relative to audiovisually-presented speech, sign language perception relies more heavily on posterior visual processing areas. In an ASL lexical retrieval task, Emmorey et al. (2003) found activation of some of the same areas, in particular, the occipitotemporal junction and the inferior temporal lobe. In a study on topographic sentences in BSL,

MacSweeney et al. (2002a) found several visual processing areas (the left and right occipitotemporal junction, the left and right posterior middle temporal gyrus, and the left parieto-occipital sulcus) which were activated more by topographic than by non-topographic sentences, in both Deaf and hearing signers.

In other studies comparing Deaf and hearing signers, the findings are a bit less clear. Neville et al. (1997) found greater activation of temporo-occipital areas in Deaf than in hearing native signers, during performance of a semantic anomaly judgment. Similarly, Soderfeldt et al. (1994) found more activation of the right parieto-occipital junction in Deaf than in hearing signers. By contrast, Levanen et al. (2001) found greater activation of parieto-occipital areas in hearing non-signers than in Deaf signers, during passive perception of signs. While these last two findings do not directly contradict each other since the two hearing groups differed in their language experience, it is also not clear how the two findings can be explained in relation to each other; it is not apparent why hearing subject with no sign language experience in one study would pattern like Deaf native signers but not like hearing native signers from another study. Finally, in a case study of a subject with left occipital damage extending into the posterior section of the corpus callosum, Hickok et al. (1995) found what they described as sign blindness. The subject had intact production but extremely impaired comprehension, which the authors suggested resulted from the disconnection between the language areas of the left hemisphere and the right visual cortex. However, it is not immediately obvious that the deficit did not simply result from damage to left hemisphere visual processing areas.

Several studies have found activation of the left parietal lobe during sign language processing tasks, and some have found activation of the right parietal lobe as well. In an ERP study of Deaf and hearing ASL signers, Neville et al. (1997) found greater activation of both the left and right parietal lobes in native Deaf and hearing signers than in hearing late-learners of ASL. MacSweeney et al. (2002b) also found greater activation of the left inferior parietal lobe during perception of BSL signs than during perception of audiovisually-presented English. Perception of BSL signs also caused activation of the right inferior parietal lobule, but it was much less extensive and more isolated than the left parietal activity. In a related study, MacSweeney et al. (2002a) found more parietal activity while processing topographic as compared to non-topographic sentences, and the difference was greater for Deaf than for hearing signers. In addition to these perception studies, a study of narrative production in ASL and English in hearing native signers found activation of both the left and right parietal

lobes in both English and ASL (Braun et al., 2001). MacSweeney et al. (2002a) proposed that inferior parietal areas may have been engaged more by sign language processing because of their importance for the imagery of hand movements (Gerardin et al., 2000) and hand position discrimination (Hermsdorfer et al., 2001); however, this explanation alone cannot account for the heightened parietal activation during narrative production in both ASL and English, reported by Braun et al. (2001). Neville et al. (1997) suggested that the parietal activation they found in Deaf and hearing native signers was a reflection of grammatical processing for sign language, but did not try to relate that function to previously reported functions of the left or right parietal lobe. It would seem that more research is called for to establish the validity and specificity of parietal lobe activation in sign language processing and to explain how it might be related to other functions of the parietal lobe, i.e., why that area in particular would be well-suited to sign language processing.

Most research on sign language and the brain has focused on sign language as a linguistic or cognitive task; however, several studies have also sought to understand sign language articulation. The majority of these studies collected data from subjects who had motor control disorders, primarily Parkinson's disease, but one research group also described sign articulation in a subject with right hemisphere damage (Loew et al., 1997; Poizner & Kegl, 1993). Research on signers with Parkinson's disease suggests that their language deficits tend to be articulatory rather than linguistic in nature (Brentari & Poizner, 1994; Kegl & Poizner, 1992). Broadly speaking, signers with Parkinson's disease (PD) tend to under-articulate: their sign production is smaller, slower, and prosodically reduced. In particular, PD signers have a reduced and lowered signing space (Loew et al., 1995; Poizner & Kegl, 1992, 1993): relative to neurologically-intact controls, they do not use as much of the space in front of the body to produce signs, even though they are capable of reaching distant locations with their hands and arms. Additionally, signs located in neutral space and on the body are lowered with respect to where they would be produced in citation form (Loew et al., 1995).

On a related but distinct point, PD signers produce signs with distal articulators; for example, on a sign that is normally initiated from the elbow, a signer with PD might produce it from the wrist or from the fingers (Brentari & Poizner, 1994; Tyrone et al., 1999). Poizner & Kegl (1993) described this tendency as a disruption of joint use. Additionally, the distal articulators of the hands and wrist are often laxed during sign production, so that the handshape and orientation of signs are less articulatorily

contrastive than they would be normally (Loew et al., 1995; Tyrone et al., 1999; Brentari et al., 1995; Brentari & Poizner, 1994). In some cases, PD signers also delete handshape or orientation change from a sign or a fingerspelled word (Brentari & Poizner, 1994; Tyrone et al., 1999), which is another form of reduced articulation.

The dynamic as well as the static features of signs are disrupted by Parkinson's disease- Brentari and Poizner (1994) stress this point and conclude from it that an ASL feature inventory should include dynamic as well as static characteristics of signs. Several articles have documented that signers with PD show a disruption in the timing of movements across articulators (Brentari et al., 1995; Tyrone et al., 1999; Poizner & Kegl, 1993; Brentari & Poizner, 1994). For example, if a sign requires both movement and handshape change, signers with PD are more likely to completely synchronise or completely serialise the two movements, rather than producing them in a partially overlapping manner, as control signers do (Brentari et al., 1995). A less frequent dynamic error produced by PD signers, which occurred in both sign and fingerspelling, was handshape mirroring on the non-active hand in one-handed signs (Loew et al., 1995; Poizner et al., 2000; Tyrone et al., 1999). PD signers did not produce a two-handed sign in place of a one-handed sign, but rather replicated the movements of just the active hand on the non-active hand.

To recapitulate, based on existing data, PD signing is monotonous, reduced in size and speed, and characterized by disruption to the relative timing of multiple articulators. As stated above, most research on sign articulation deficits has focused on Parkinson's disease, because of its common occurrence in the elderly and because of its minimal impact on linguistic function. Apraxia, by contrast, often co-occurs with aphasia in both Deaf and hearing individuals, so it can be difficult to differentiate the two disorders; and some have argued that the two are not distinct from each other (Kimura, 1977). No study to date has documented a case of a Deaf signer with apraxia but not aphasia, so it is impossible to assess the effects of apraxia alone on signing as opposed to non-linguistic gesture. The only other neurological disorder to have been examined in terms of articulation is right hemisphere damage. Poizner & Kegl (1993) described the effects of right hemisphere damage on one individual's signing; the same subject is described in Loew et al. (1997). Both articles mentioned that the signer with right hemisphere damage showed movement lagging in his affected left arm during signing. When he produced two handed signs, movement initiation was delayed in the left hand relative to the right hand. Poizner & Kegl (1993) analysed this as a deficit in motor neglect. However, neglect resulting from right hemisphere damage typically

manifests itself spatially rather than temporally: subjects make fewer movements to (i.e. they neglect) the area of space contralateral to the lesion. Movement lagging in the affected limb seems more likely to be the result of mild hemiparesis, which the authors report that this subject shows as well. Because this is the only case study describing sign articulation deficits resulting from right hemisphere damage, more research is required to better understand the nature of the deficit that Poizner & Kegl (1993) identified.

A few recent imaging studies of neurologically-intact Deaf signers have explored questions related to movement or articulation in signing and fingerspelling. In a study of ASL lexical retrieval, Emmorey et al. (2003) found that retrieval of fingerspelled words activated the supplementary motor area-- a region that is important for movement planning. The authors suggested that the movement sequencing demands of fingerspelling may have caused activation of that area, as the subjects rehearsed or simply imagined the requisite fingerspelled words.

In a study more explicitly designed to investigate articulation, Corina et al. (2003) explored a relatively basic but previously unexplored issue: whether left handed signing still activates left hemisphere frontal structures, as well as other structures relevant for motor control for speech articulation. The researchers found left inferior frontal (and right cerebellar) activation during sign production in right handed subjects, even when productions were made with the left hand. This finding is consistent with earlier studies by Wise et al. (1999) and Dronkers et al. (1996) suggesting that the anterior left hemisphere plays an important role for speech articulation. Clearly, in speech it is not possible to test how closely this function is tied to the lateralization of motor control, since speakers cannot control the two sides of the vocal tract completely independently.

To summarize, early research on sign language breakdown in subjects with brain damage following stroke made two important findings; first, even though the right hemisphere serves an important function for visuospatial processing, the use of space for sign language grammar is nonetheless mediated by the language structures of the left hemisphere. The second finding was that apraxia and aphasia could be dissociated, even though the relevant articulators, and sometimes the relevant movements, were the same; apraxia is a gestural and not a linguistic deficit. Brain imaging studies of neurologically-intact signers confirmed the first finding; and recent research on sign language and stroke in the UK has confirmed the second finding as well. Additionally, brain imaging studies found that sign language processing activates auditory processing, visual processing, and higher level association areas, to differing degrees in Deaf and hearing

signers.

Research on sign articulation has revealed that signers with movement disorders show articulatory deficits in the absence of linguistic breakdown, and those deficits pattern similarly to deficits exhibited in non-linguistic movements. In addition, recent imaging studies on sign production and lexical retrieval have indicated that the same areas that are activated during speech articulation are also activated during sign articulation despite the fact that the two modalities use a different set of articulators.

1.3 Articulation as a modality-independent concept

Up until now, articulation has been considered as either synonymous with speech or, in the speech motor control literature, as a component of speech (Darley et al., 1975). To answer the question of whether or not sign movements are like articulation, it is necessary to first consider what articulation is, independent of the particular set of muscles that enact it. At a basic, physical level, articulation is made up of very rapid, complex, co-ordinated movements—in fact, the movements required for speech are the most demanding in the human behavioural repertoire in terms of speed and co-ordination (Fry, 1979; Kent, 1997; Lieberman & Blumstein, 1988), yet notably, not in terms of effort or physical strain. Sign is not produced as rapidly as speech (Klima & Bellugi, 1979); however, apart from speech, it is probably the most co-ordinated, complex motor behaviour that humans engage in spontaneously, on a day-to-day basis, and without explicit instruction. Like speech, sign is also not particularly effortful or tiring for fluent signers.

Similar to being rapid and complex, articulation is multi-dimensional, both in terms of production and perception: the reason that it requires precise co-ordination is in large part due to the fact that it has many physical subcomponents that are independently controlled. Looked at from the perspective of the recipient of the articulatory signal, articulation can be broken down into subcomponents that can be interpreted at least partially independently of one another, as in the case of pitch and loudness of speech. Consequently, articulation is well-adapted to presenting multiple streams of information simultaneously. In addition to being multidimensional and conveying multiple streams of information at once, articulation is also sequential and allows individual movements to be combined serially in very rapid order. In fact, it is more sequential in nature than any other complex human motor behaviour. For these reasons, in terms of its physical structure, articulation is unlike spontaneous, speech-accompanying gesture, which tends to be both holistic and paradigmatic in nature, and

cannot consistently be divided into structural components that can be analysed meaningfully. Nor can spontaneous gestures be combined sequentially in any systematic or meaningful way (McNeill, 1992).

Articulation must have communicative potential that is applicable in a broad variety of contexts; in other words, it must be possible to use articulation to convey complex, specific, new information. Articulation and communication are not the same thing. It is possible both to articulate without communicating and to communicate without articulating. What is unique about articulation is not that it is communicative *per se*, but that it is well-adapted to linguistic communication in particular. What distinguishes articulation from other types of movements is the number and speed of perceptually salient contrasts that it is able to transmit in the information signal. By extension, then, it facilitates linguistic communication by providing an effective means of conveying information.

The form that articulatory productions take is narrowly determined by the physical structure of the relevant effectors as well as by the neural mechanisms controlling their movements. On the first point, regarding the physical structure of the effectors, to say that articulation is determined by the physical structure of its effectors is not the same as saying that use of a given set of effectors is by definition articulation, while use of another set is not articulation. Nonetheless, the shape, size, and orientation of articulatory muscles bear a close relationship to the physical structure of the behavioural output. It only takes slight variation in movement or articulatory configuration to convey a completely different meaning or communicative intent. Articulation as such is not limited to a given set of effectors, but its physical form, and by extension, its perceptual salience, is determined by the actual anatomy and kinematics of its effectors; whereas simpler, non-communicative movements are not by necessity so tightly governed by the effectors that they use. To illustrate the point with a contrast, any skeletal muscle in the body can be gradually stretched to its greatest comfortably-extended length, but whether that is done with the dorsal interosseus or the latissimus dorsi, the behavioural output is effectively identical, both at the level of electrophysiological activity of the muscles and at the level of the communicative potential of the action as a whole. The broader point is that the movements of 'articulators' are highly differentiable and as such they have the capacity to produce a broader range of behavioural output, which can then be modified and combined in a variety of ways to make quick transmission of information (and hence human communication) possible.

Clearly, the example of a non-articulatory movement described above is an extreme; however, the general principle still applies. Unlike other types of movements, articulation combines speed, precision, broad applicability, communicative potential, and behavioural salience. It is unique in that it is simultaneously natural, easy, acquired without instruction, and yet not automatic, reflexive or intuitive in the same way as walking, or chewing, or (in a different respect) crying. Simply put, it is a complex behaviour that must be learned but is learned successfully by all neurologically-intact members of the species.

Additionally, the form that articulation takes is governed in part by the brain mechanisms controlling it. In other words, it is not reflexive, like motor behaviours governed at the level of the spinal cord or brainstem; nor is it automated like walking, chewing, and other rhythmic behaviours governed by brainstem mechanisms. There are rhythmic, automated movements that are a precursor to articulation (MacNeilage et al., 1999; Meier, 2000), but in its fully developed form, articulation is more structurally complex and varied than babbling or similar automated behaviours. That being the case, articulation must be learned, and by extension, can break down at the level of knowledge of how to perform the necessary movements, as opposed to breakdown at the level of weakness, paralysis, or co-ordination (Square et al., 1997), which can affect any and all movement.

Finally, articulation consists of movements that are highly and/or differentially susceptible to breakdown in the context of a movement disorder. It is not uncommon for high-level movement disorders to affect speech but not limb movements (Wertz et al., 1998), or vice versa (Goldenberg et al., 2003), or to actually affect speech in one way and limb movements in a completely different way, as in the well-known case of general movement slowing but rapid speech in Parkinson's disease (Theodoros & Murdoch, 1998b). Traditional descriptions of disruptions to speech motor control have treated it as qualitatively different from disruption to simpler movements of the limbs (Enderby, 1983), and rightfully so; but is speech qualitatively different because of the articulators it employs or because of the motoric demands it imposes? Prior to recent research on sign and movement, these two factors were not dissociable.

1.4 Dysarthria

Broadly defined, dysarthria in hearing populations is a disruption to speech motor control. While it has been known for a long time that speech production could break down in the absence of language impairment, Darley et al. (1969b) were the first

to recognise that there were different forms of dysarthria that could be categorized according to their specific features. Their definition of dysarthria is as follows:

> a collective name for a group of speech disorders resulting from disturbances in muscular control over the speech mechanism due to damage of the central or peripheral nervous system. It designates problems in oral communication due to paralysis, weakness, or incoordination of the speech musculature. p. 246.

There are many important implications of this definition. First of all, as stated above, dysarthria is distinct from aphasia. Although this had been recognized long before, because it can be difficult to distinguish disorders of speech from disorders of language, particularly when the two co-occur, it is a point worth reiterating. Second, and less obviously, dysarthria is distinct from apraxia, which disrupts movement in the absence of weakness, paralysis, or inco-ordination. Dysarthria and apraxia both disrupt movement, but even within that delimitation, they do so at different levels. Additionally, despite being a lower level disorder than apraxia, dysarthria is distinct from speech disruption at the level of the muscle, bone, or tendon. As Darley et al. (1969b) point out, dysarthria results from damage to the nervous system. Finally, according to their definition, dysarthria is differentiable into subtypes that can be analysed methodically and described systematically. It may seem like a minor point, but prior to the research by Darley et al. (1969b), earlier researchers and clinicians had used holistic, impressionistic terms such as 'explosive' or 'hot potato' speech to describe disruptions to speech motor control. So, clearly their research provided an analytical framework that simply had not existed beforehand.

What Darley et al. (1969b) do not state explicitly is that dysarthria may or may not be accompanied by a movement disorder affecting the limbs, or other parts of the body. They take it as a given that speech is unique, and as such, the effects of disrupted motor control on speech may differ from the effects of disrupted motor control elsewhere; the two disruptions are simply not considered to be in the same category. Dysarthria is not treated as a component of a broader movement disorder, but as an independent symptom of a given type of neural damage.

Additionally, for Darley et al. (1969b), dysarthria by definition is a disruption to oral communication. Their choice of the term 'oral communication' is probably intended to convey that the phenomenon they are describing is not fundamentally linguistic in nature; however, at the same time, it does not necessarily affect non-speech oral movements as well as speech movements. Moreover, it does not necessarily affect

all forms of communication—their choice of words is probably also intended to exclude apraxia and other disruptions to gesture. Because they did not consider the possibility of disruption to a linguistic system that does not use the vocal tract, they did not have to allow for what might distinguish dysarthria from other movement disorders apart from the set of effectors that are implicated. As this study is concerned with precisely that question, the definition of sign dysarthria used here is based on the definition put forward by Darley et al. (1969b), but modified to apply to a different set of articulators, while excluding disruption to language and disruption to movement at a simpler level. The working definition of sign dysarthria for purposes of this research is: a disruption of sign production caused by damage to neural mechanisms governing movement. The implications of this definition will be explored in more detail in Chapter 3, which describes the various documented forms of dysarthria.

1.5 Research Questions

The research goals of this study are to broadly define the phenomenon of sign dysarthria; to determine whether anything other than the set of articulators involved differentiates it from dysarthria in spoken language; and to delineate the differences between sign dysarthria and apraxia, and between sign dysarthria and disruption of simple limb movements. In the same way that hearing people may exhibit speech dysarthria in the absence of oral apraxia, deaf signers may exhibit sign dysarthria in the absence of higher level ideomotor impairments. Conversely, just as many movement disorders are more apparent in speech than in simple limb movements, sign dysarthria may also arise in the absence of severe impairment of simple movements, such as reaching or pointing. An ancillary question that this study addresses is the establishment of articulatory measures of sign dysarthria, and of normal signing. The research questions and means of addressing them will be discussed briefly here. Additionally, a full description of the research methodology, including the rationale behind the overall design of the study is presented in Chapter 4.

In order to develop a definition of sign dysarthria, it is necessary to compare subject groups, individual subjects within a group, and linguistic and non-linguistic behaviours of those subjects as assessed by relevant tasks. The first comparison to be made is between Deaf subjects who participated in this study and hearing subjects described in the speech motor control and motor control literature. In particular, it is important to test whether Deaf subjects' performance across tasks resembles what has been reported in hearing speakers with dysarthria, and if so, in what way. Moreover, it

is of interest to know if signers with movement deficits perform similarly to hearing subjects with movement deficits on tasks unrelated to sign or speech. It is remotely possible that frequent use of a conventionalised, complex system of limb movements could influence Deaf signers' performance of movement tasks unrelated to sign; so although comparison of Deaf and hearing subjects on simple movements is not the primary focus of the study, it deserves some consideration in the discussion of dysarthria across the two modalities.

In order to define sign dysarthria broadly enough, it is necessary to test similarities and differences across Deaf signers with a range of movement disorders, in addition to contrasting them with hearing subjects with dysarthria, Deaf control subjects, or Deaf signers with aphasia. Previous research on sign language and motor control has compared movement disorders with linguistic disorders, but few studies have directly compared the nature of articulatory disruption to sign production across subjects with different types of motor control deficits. Comparison of signers with different types of motor control deficits is an important component of this study, because sign is a complex motor act and as such it is apt to break down in a multitude of ways. In part, this study is intended to identify the ways in which sign *can* break down as the result of a movement disorder, above and beyond how signs do break down in one subject or in one type of movement disorder. As in the case of spoken language, it is expected that sign dysarthria will take different forms in subjects with different movement disorders, and those forms can be differentiated along a small set of articulatory measures.

It is only possible to claim that there is such a phenomenon as sign dysarthria if it can be shown that subjects' performance on signing tasks is somehow distinct from their performance on non-linguistic movement tasks. It is for this reason that Deaf signers were tested on a range of linguistic and non-linguistic limb movement tasks. It has traditionally been assumed that speech movement deficits are distinct from movement deficits more generally, simply because a different set of effectors is involved. Given that it is now widely accepted that language can be produced with either the vocal tract or with the hands and arms as primary articulators, it is possible to look for a dissociation between linguistic and non-linguistic limb movements for signers or a strong similarity between motoric disruptions to sign and speech, or possibly both.

To address these research questions, a small number of subjects with different neurogenic movement disorders have been investigated in depth as a series of case studies. The subjects are not intended to represent a uniform group, but rather to

highlight the differences in various possible forms of sign dysarthria. The set of subjects comprises one signer with Parkinson's disease, one with progressive supranuclear palsy, one with cerebellar damage, one with right anterior cerebral damage, and one with left anterior cerebral damage. Additionally, there are control data from two age-matched signers without movement disorders. Individual subjects and the rationale for their inclusion in the study will be discussed in detail in Chapter 4.

Testing was carried out in subjects' homes and recorded on videotape. While there are sign language and motor control research methodologies that capture more spatial and temporal detail, it was decided that videotape would be preferable for two reasons. First, because this study is the first to attempt to describe a range of disruptions to sign articulation, it was necessary to analyse multiple physical aspects of signing at once to determine where and how impairments might manifest themselves. In a sense, the data from kinematic or electromyographic studies are too specific to be useful in addressing this research question. Second, the lack of a body of normative data on the kinematics or physiology of signing would render those types of data on impaired signing relatively meaningless.

Linguistic tests were designed to elaborate the nature of subjects' fingerspelling and signing deficits, and allow comparison of those deficits to each other. (Fingerspelling is a system for borrowing words from spoken language, in which the written letters are represented sequentially by distinct configurations of the hands and fingers. As such, fingerspelling is more rapid and motorically demanding than signing). The linguistic tests also serve as a baseline of performance that can be compared to performance on non-linguistic motor tasks. As discussed above, the comparison of linguistic to non-linguistic data in Deaf signers with movement disorders is critical to the discussion of sign dysarthria. The schema for coding the linguistic data was developed on the basis of research in sign phonology (Brentari, 1998; Friedman, 1976), signing and movement deficits (Brentari et al., 1995; Loew et al., 1995; Tyrone et al., 1999), gesture co-occurring with speech (McNeill, 1992), hand movements (Kimura, 1993; Rothi et al., 1997), and speech motor control (Duffy, 1995; Love & Webb, 2001). Individual signs and fingerspelled letters were coded for the linguistic parameters of handshape, orientation, and location, as well as the non-linguistic parameters of repetition, involuntary movement, bimanual co-ordination, and co-ordination of proximal and distal articulators.

Non-linguistic tasks were designed to give an approximate sense of the nature of subjects' motor control disorders, e.g. inability to appreciate how to use an object as

opposed to the inability to physically manipulate it, or inability to accurately scale a movement as opposed to a loss of range of movement. The tasks consist of: symbolic tool use, pointing to images, copying object manipulation, copying meaningless hand configurations and performing a series of actions on a semi-standardized movement task (Kimura, 1977). The coding scheme for the non-linguistic data was developed on the basis of earlier motor control research (e.g., Kimura, 1993; Wiesendanger et al., 1994; Timmann et al., 2001). Individual movements were coded according to: presence of involuntary movement, targeting errors, co-ordination errors, handshape selection errors, disruptions to integration of proximal and distal movements, and disruptions to movement initiation, continuation or termination. The tasks, procedures, and coding schemes for the linguistic and non-linguistic data are described in greater detail in Chapter 4.

In addition to the specific research questions outlined here, there are broader theoretical and practical implications of research on sign and movement. First, because of the nature of the data, this study and others like it allow a more sophisticated comparison of movements used for language production and other movements of the hands and arms. Past studies have compared speech movements with simpler limb movements and reached conclusions about the fundamental nature of the effectors themselves on that basis (Ackermann et al., 1997; Lieberman, 1995). By examining sign movements and other limb movements, this study is comparing like with like in a way that earlier studies comparing articulatory and non-articulatory movement could not have done. While it is certain that language and gesture, and for that matter, articulation and gesture, are structurally different, gesture is not something accomplished only by the hands, nor is language accomplished solely by the vocal tract. Taking that point into consideration will go a long way toward clarifying the fundamental differences between linguistic and non-linguistic communication, and by extension, the necessary precursors to human language.

1.6 Preview of Findings

It will be argued here that dysarthria, as distinct from apraxia, aphasia, and loss of simple movement, does manifest itself in sign language, which suggests that speech motor control research should eschew models of dysarthria framed around specific articulators, in favour of those that emphasize patterns of movement. 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 that is necessary for any linguistic system, but that does not make disruptions to it 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.

2 The Physical Structure of Sign and Speech

The aim of this chapter is to delineate the motor control mechanisms for speech and sign language in order to identify where they converge and diverge, and thereby allow for speculation regarding differential effects of damage to various components of those mechanisms. Many researchers have pointed out that because speech and sign use different production modalities, they must, by necessity, be perceived differently, at least at the most basic level (Klima & Bellugi, 1979; Poizner et al., 1987), and some researchers have gone further to speculate about the linguistic and behavioural effects of those perceptual differences (Brentari, 2002; Meier, 2002). Though perception modality almost certainly has as much effect on the form (if not the linguistic processing) of speech and sign as production modality, because the focus here is on production, the neurological and physiological basis of language perception will not be discussed.

2.1 Neurology of motor control

The human motor control system is composed of the portions of the brain, brainstem, spinal cord, and peripheral nerves controlling movement, as well as the striated muscles that execute movements. In this discussion, the focus will be on the first and last of these, because they are where the relevant implications for speech and sign production are most likely to emerge.

2.1.1 Pyramidal tract

There is a direct projection from motor areas of the cortex to the spinal cord, which is commonly referred to as the pyramidal tract (Gray, 2000). Although there remains some disagreement on exactly what the pyramidal tract does (i.e. what behavioural phenomena the activity of an individual motor neuron corresponds to), it is generally agreed that it is particularly important for fine-grained, voluntary movement, such as the individuated movements of the fingers necessary for handling objects. There is a broad range of evidence supporting this idea, including the fact that the pyramidal tract is very late to become completely myelinated in human infants (around 2 years of age); and consequently, young infants do not have adequately fine-grained motor control to produce complex, voluntary movements (Brodal, 1998). Additionally, it is not uncommon for precise movements of the affected hand to be disrupted in mild cases of hemiparesis following damage to the motor cortex. Finally, in broader terms, cortical control of movement occurred very late into phylogeny—for most mammals,

movements are controlled through descending brainstem mechanisms that facilitate automatic, over-learned movements like walking or chewing (Kandel et al., 1991).

What is known about the physiology of the pyramidal tract suggests that populations of cortical neurons encode the direction of movement. Additionally, broadly speaking, motor areas of the cortex are divided somatotopically, i.e. sections of motor areas of the cortex control movement for specific parts of the body. As the pyramidal tract descends, it can be subdivided into the corticospinal and corticobulbar tracts. The fibres of the corticospinal tract descend through the cerebral hemispheres and the ventral brainstem. Then in the lower medulla, the majority of fibres cross, or decussate, to the contralateral side of the brainstem and descend from there to the spinal cord (Figure 2.1). In the spinal cord, the corticospinal fibres synapse onto the neurons that form peripheral motor nerves. Because most of the fibres of the corticospinal tract decussate in the medulla, one hemisphere of the brain controls movements for the opposite side of the body. The corticobulbar tract also originates in motor areas of the cortex, but rather than projecting to the spinal cord, projects to the cranial nerve motor nuclei in the brainstem. The cranial nerves arise from these motor nuclei and control the movements of the head, neck, eyes, and vocal tract. Unlike the corticospinal tract, the projections of the corticobulbar tract are largely bilateral. In other words, with few exceptions, both cerebral hemispheres control movements of both sides of the vocal apparatus in almost equal proportions.

Contrary to earlier speculation, the corticospinal and corticobulbar tracts include diffuse projections of fibres from multiple motor and somatosensory areas, not just from the primary motor cortex (Carpenter, 1991). The implications of this are twofold: first, damage to a small area of the cortex can have widespread peripheral motor symptoms, because of the spreading of the fibres in the brainstem and spinal cord; and second, damage to areas of the cortex other than the primary motor area may result in paralysis or paresis of the contralateral limb. Additionally, the somatotopy of the motor areas of the cortex may be less strictly delimited (or more functionally-based) than researchers have assumed (Meister et al., 2003; Rijntjes et al., 1999).

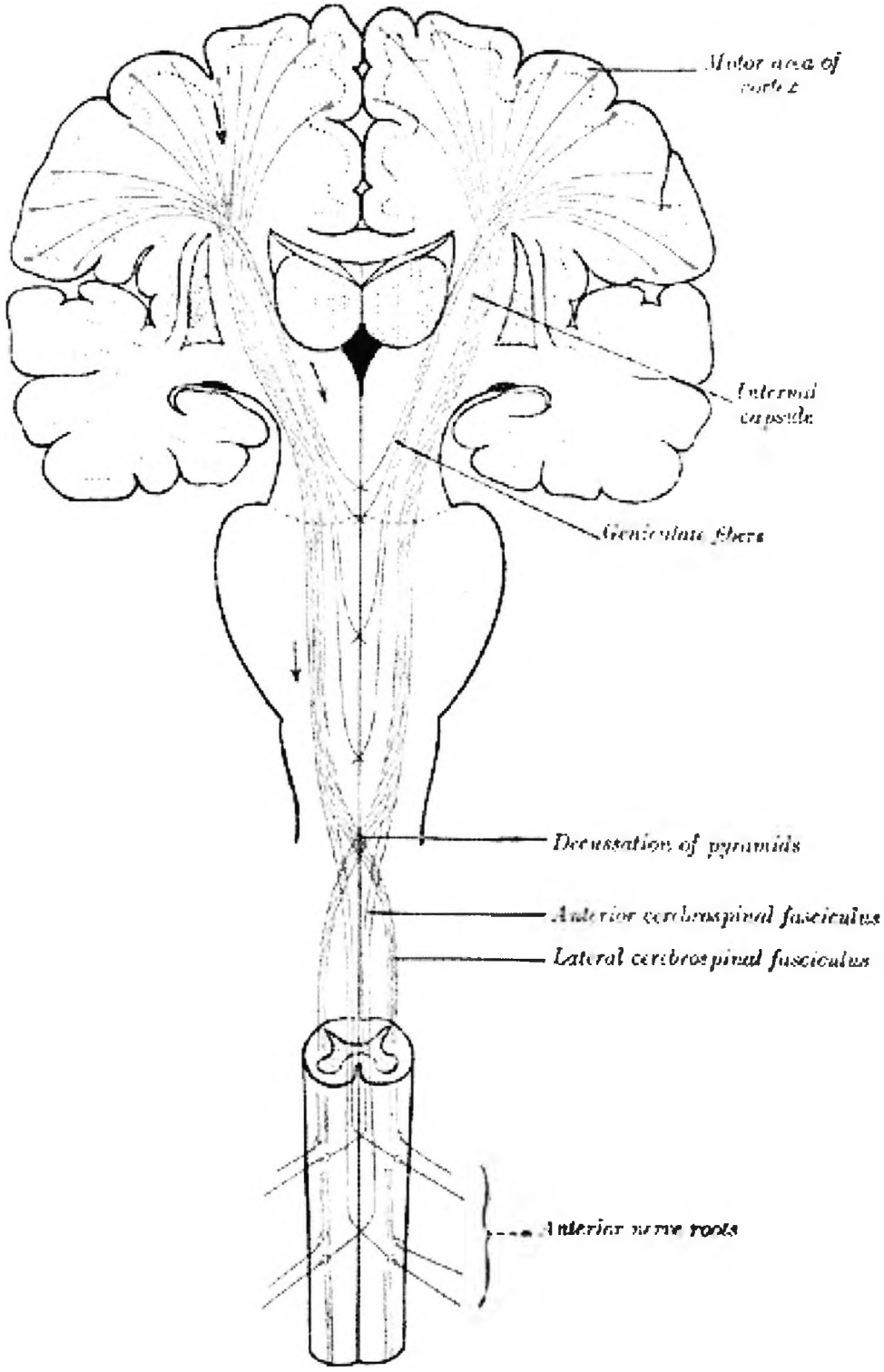

Figure 2.1: The Corticospinal Tract (from Gray (2000))

2.1.2 Extrapyramidal structures

There is far less consensus on the role of non-neocortical structures in controlling movement, quite simply because it is less apparent[2]. Neither the cerebellum nor the basal ganglia is directly responsible for movement generation (i.e. initiating the command to peripheral effectors to move), but rather for shaping voluntary movement so that it is accurate, natural, well-timed and co-ordinated (Brooks, 1986; Hallett & Khoshbin, 1980; Lang & Bastian, 2002). Exactly how either structure accomplishes these functions is not entirely clear. It has been proposed for both that they might integrate or convert relevant sensory information (Jueptner et al., 1996; Lidsky & Brown, 1999; Zia et al., 2000), provide feedforward or feedback information to cortical motor areas (Iansek et al., 1995; Quaia et al., 1999), or select muscle synergies appropriate to the desired task (Jueptner & Weiller, 1998; Timmann et al., 2001). Additionally, both structures play a role in modifying muscle tone, balance, and posture, probably through projections to brainstem nuclei. Anatomically, although both the cerebellum and basal ganglia include somatotopic maps of the body like motor areas of the cerebral cortex, they tend not to function in an effector-specific way; and there is no direct projection from either the cerebellum or the basal ganglia to lower motor neurons in the brainstem or spinal cord (Carpenter, 1991). Both the cerebellum and the basal ganglia do, however, receive (direct or indirect) input from motor areas of the cortex and project back to cortical motor areas via the thalamus (Figure 2.2). Finally, in trying to determine the functions of these structures, it should be remembered that there is no reason to assume a one-to-one relationship between structure and function.

2.1.2.1 Basal ganglia

The basal ganglia comprise a network of subcortical nuclei that are closely linked functionally and electrophysiologically: the caudate, putamen, globus pallidus, and substantia nigra. The first two of these comprise the corpus striatum but are often discussed as independent entities in research on humans. The individual nuclei of the basal ganglia will not be discussed in any detail here; however, earlier studies are described here that do discuss those nuclei individually. The basal ganglia receive input from motor as well as other areas of the ipsilateral cerebral cortex, and project back to

[2] Though there are several subcortical nuclei involved in motor control, the only two brain structures to be considered here are the cerebellum and basal ganglia, both because their functions have been extensively studied and because their related pathologies are not uncommon in humans.

the ipsilateral supplementary motor area via the thalamus (Brodal, 1998) (See Figure 2.2). Furthermore, the basal ganglia's connections to peripheral sensory or motor nerves are extremely indirect: two synapses removed, at best. So, perhaps not surprisingly, there is no tight correlation between muscle activity and neuronal activity in the striatum or the globus pallidus, and even less correlation with activity in the substantia nigra. While it is impossible to analyse normal basal ganglia activity in great detail in humans, single cell recordings in monkeys indicate that globus pallidus neurons fire after movement onset and not consistently beforehand (Brotchie et al., 1991a). Consequently, the basal ganglia have been assumed to have a fairly high-level function in motor control (Brotchie et al., 1991b; Georgiou et al., 1994), removed from the actions of individual effectors, and possibly related to providing task-relevant information to motor areas of the cortex.

Issues related to PD research

A large body of research on basal ganglia function has focused on Parkinson's disease as an indicator of what human motor control may be like in the absence of the basal ganglia. As Marsden (1984) pointed out, it is comparatively rare that an idiopathic condition directly affects such a sharply delimited brain structure; and in the early stages of the disease, the sensorimotor component of the basal ganglia is affected to a greater degree than other components. Nonetheless, Parkinson's disease research has its problematic aspects. First, the motor symptoms of the disease are superficially very disparate, so it is difficult to posit a single motor function underlying all of them (and indeed there may not be one) (Marsden, 1984). Second, the view of Parkinson's disease as a model for motor control without the basal ganglia implies that the disease only exhibits negative symptoms, which is clearly not the case. While analyses of basal ganglia function on the basis of Parkinson's disease research should not be rejected out of hand, these limitations should be given due consideration.

The particular symptoms of Parkinson's disease will be discussed in depth in later chapters on subjects with Parkinson's disease and similar disorders; however, a few clinical findings will be mentioned here for their implications for basal ganglia function, bearing in mind the broader problems with Parkinson's disease as a model for basal ganglia dysfunction. Early research showed that individual ballistic movements of patients with Parkinson's disease are often undershot and slow, and went on to suggest that ballistic movements were selectively impaired (Hallett & Khoshbin, 1980; Marsden, 1982). It is unclear precisely what the neural underpinnings of this

phenomenon are; however, it is consistent with the idea that the basal ganglia are more relevant to pre-planned movements than to modification of movements on the basis of sensory feedback (Wing & Miller, 1984). It was later argued that damage to the basal ganglia differentially impacts movement sequencing (Graybiel et al., 1994; Weiss et al., 1997), particularly in well-learned, automatic tasks (Brotchie et al., 1991a; Iansek et al., 1995). While it is widely agreed that patients with Parkinson's disease show deficits in movement sequencing, it is not clear whether this represents a fundamental function of the basal ganglia, or as Wing & Miller (1984) argued, that the effect of Parkinson's disease on sequencing is secondary to a more general deficit in execution of pre-planned movement.

Low-level functions

Human behavioural and imaging studies have suggested that the basal ganglia are responsible for generation of force (Brown & Marsden, 1999a; Corcos et al., 1996; VanGemmert et al., 1999), or selection of effectors to execute a movement (Jueptner & Weiller, 1998). However, more detailed anatomical and physiological data would not seem to support these findings. First, as outlined above, the basal ganglia do not have very direct contact with elements of the motor system governing the lowest level of muscular activity (Brodal, 1998). In addition to this, though, single cell recordings have shown that the neural activity of the globus pallidus relative to activity in the supplementary and primary motor areas and relative to movement onset is not well-suited to controlling low-level aspects of movement execution (Brotchie et al., 1991a). Jueptner & Weiller (1998) reported that the anterior striatum and dorsolateral prefrontal cortex are active during movement learning, and the posterior striatum and sensorimotor cortex are active during execution of automatic movements. Nevertheless, the relative order of these recorded neural activities and the movements themselves must be taken into account for the functional implications of the data to be clear.

Internal cues/representations

Attempts to classify basal ganglia function at a higher level fall into two groups: those which have proposed that the basal ganglia generate or store internal information related to movement, and those which have proposed that they respond to external stimuli. Interestingly, Gentilucci & Negrotti (1999) took an intermediate view and suggest that the basal ganglia are involved both in storing a plan of action and controlling its implementation. Among those suggesting that the basal ganglia provide

internal triggers for movement, Georgiou et al. (1994) found that Parkinson's disease subjects rely more on external cues in completing a sequential button-press task and conclude from this that the basal ganglia may function to generate internal cues for releasing successive stages of a predefined sequence of movements. While this conclusion does not follow immediately from the empirical finding, it is supported by other human and animal studies (Iansek et al., 1995; Yaguez et al., 1999). On the basis of single cell recordings in monkeys, Brotchie et al. (1991b) proposed that movement is initiated in the premotor or supplementary motor area; then after it has started, the basal ganglia take over running it to completion, and allow transitions from one movement to another.

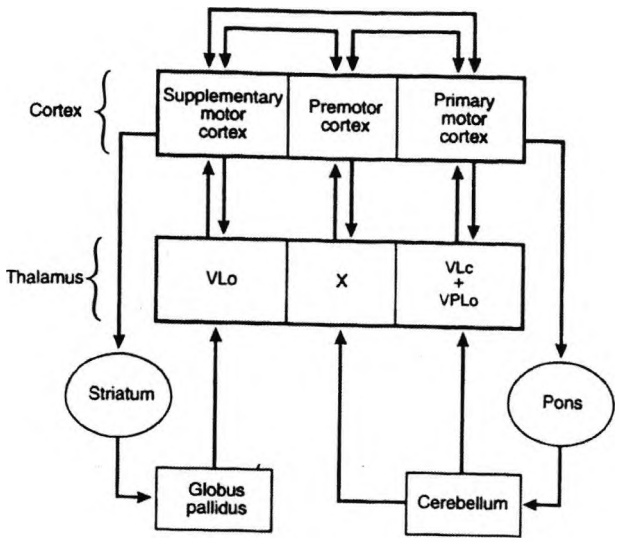

Figure 2.2: Cerebral connections of the basal ganglia and cerebellum (from Kandel et al., 1991)

External/sensory processing

Another school of thought has proposed that the information that the basal ganglia provide to motor areas of cortex is external or sensory, rather than stored internal models or cues. It is not clear whether or not this is a contributing factor, but interestingly, most studies indicating a relationship between basal ganglia function and sensory processing are animal studies. Of course, that may simply be due to the fact that animal studies allow more detailed assessments of the relationship between external stimuli and neural activity.

There is a substantial body of research suggesting a role for the substantia nigra dopaminergic system in coding reward stimuli (Schultz, 1998; Spanagel & Weiss, 1999; Waelti et al., 2001), which clearly would involve the processing of information external to the central nervous system. However, no clear parallel to this function has been found in the human basal ganglia, meaning either that it simply has not been discovered, or that the substantia nigra has taken on a different function in humans, in much the same way that the hippocampus has done.

Studies on sensorimotor function in animals have suggested that the basal ganglia may support sensory-guided movements by responding to behaviourally-relevant sensory information (Lidsky & Brown, 1999), or converting sensory information to egocentric co-ordinates (Graziano & Gross, 1993). However, research on sensory processing in the basal ganglia in humans has been contradictory, with behavioural data indicating that subjects with Parkinson's disease have an impaired sense of still joint position (Zia et al., 2000), and imaging data showing that the basal ganglia are active both during movements that depend on external sensory cues and during those that do not, in contrast to the cerebellum, which is only active during tasks that depend on sensory cues (Jueptner et al., 1996).

2.1.2.2 Cerebellum

The cerebellum receives input from the spinal cord, the brainstem, and indirectly from the primary motor cortex. It projects out to the brainstem, indirectly to gamma motor neurons controlling tone, and indirectly to the motor areas of the cortex (Figure 2.2) (Brodal, 1998). The spinal cord projections to the cerebellum are largely *ipsilateral*, while the cerebrocortical projections to and from the cerebellum are largely *contralateral*; the overall effect of this being that the cerebellum primarily affects movement and co-ordination on the ipsilateral side of the body. In broad terms, the cerebellum is a large structure with many (albeit indirect) connections to the neocortex and to the periphery, and an extremely high ratio of incoming to outgoing fibres: 40 to 1 in humans (Brodal, 1998). Consequently, it is well-situated to integrate information from different sources (e.g. sensory and motor information) and to perform computationally-demanding tasks. Additionally, it is clear from electrophysiological data that there is a tight temporal relationship between cerebellar firing patterns and gamma motor neuron activity (Timmann et al., 2001).

Several potential functions have been proposed for the cerebellum, many of which are similar or potentially co-existent. It is generally agreed that the cerebellum is

involved in motor learning, and possibly in procedural learning more generally (Hikosaka et al., 1999). There are anatomical as well as behavioural data to support both of these theories (DeZeeuw et al., 1998; Thach et al., 1991; VanAlphen et al., 2002). However, it is unlikely that this is the cerebellum's sole function, because individuals with cerebellar lesions show deficits on routine, overlearned tasks such as speech articulation (Ackermann & Hertrich, 2000), as well as on motor learning.

Multijoint movement

One theory that would explain deficits in familiar but complex tasks such as speech is that the cerebellum is important for the co-ordination of multiple effectors at once. Timmann et al. (2001) suggested that cerebellar damage impairs multijoint movements more severely than single-joint movements, but they ascribed this to its role in quickly implementing antagonist muscle activity. Because subjects with cerebellar damage showed variability in finger position while throwing a ball, but not while dropping it, Timmann et al. proposed that the deficit lies in the inability to implement the activity of the antagonist muscles, which are not involved if one is simply dropping a ball against gravity. As with the case of the basal ganglia and selection of appropriate effectors, it seems counterintuitive that a structure with the cortical and peripheral connections and computational capacity of the cerebellum would be chiefly responsible for modulating the activity of antagonist muscles. By contrast, Thach et al. (1992) proposed that the problem with multijoint movements is due to the cerebellum being specifically adapted to coordinating movement by combining multiple, simple motor synergies into more complex ones.

It is worth pointing out that it is unclear if what has been described as a multijoint disorder is literally that or if it is a *multi-effector* disorder, because experimental tasks have focused solely on the limbs. If it is the former, then one would predict that speech would be affected less than limb movements; however, if it is the latter, speech would probably be affected as much, if not more so.

Timing

It has also been proposed that the cerebellum might provide a general timing function, rather than doing anything motor-specific (Keele & Ivry, 1990). In other words, the cerebellum might serve as a sort of pacemaker to correctly time behavioural events relative to each other. Because observable behavioural events necessarily have a motor component, this hypothesis is extremely difficult to test. However, the theory is

supported by anatomical data on the nature of the projection from the inferior olive to the cerebellum (DeZeeuw et al., 1998). Others have taken a more conservative approach and suggested that the cerebellum supports the timing of motor behaviour, rather than behaviour in general (Welsh et al., 1995).

Memory/internal models

Analogous to theories of a basal ganglia internal cuing function, it has been suggested that the cerebellum may contain stored models of learned movements (Lang & Bastian, 2002; Lu et al., 1998) which would allow predictive or feedforward information pertaining to movement to be passed on to motor areas of the cortex. It has also been suggested that the cerebellum serves as the site for internal models of the peripheral motor apparatus and load (Wolpert et al., 1998). In a study by Timmann et al. (2001), subjects with cerebellar damage patterned similarly to untrained control subjects, which supports the claim that the cerebellum is important for the storage of learned motor synergies.

Comparing real and intended consequences/sensory processing

Similarly, it has been proposed that the cerebellum may compare internal models of movement with movement consequences and make appropriate adjustments (Blakemore et al., 2001; Jueptner et al., 1996; Jueptner & Weiller, 1998). So, rather than serving solely a feedforward role, the cerebellum would act as a feedback mechanism as well. Consistent with this, Quaia et al. (1999) proposed a model wherein the cerebellum controls both accuracy and consistency of eye movements, and presumably other movements as well, thus functioning as both a feedforward and feedback mechanism. However, DeZeeuw et al. (1998) argued that the anatomical data do not support this theory, at least not for the projection from the inferior olive to the cerebellum.

2.1.2.3 Summary of extrapyramidal structures

To recapitulate briefly, both the cerebellum and the basal ganglia exchange projections with the motor areas of the cerebral cortex, and hence both structures probably play a high level function in motor control. The cerebellum also receives information directly from the spinal cord, though the basal ganglia do not; and both structures project indirectly to lower motor neurons via brainstem nuclei. Consequently, one would expect damage to either structure to affect multiple effectors, disrupt movement at a high level, and potentially to have a differential effect on movements

that rely on sensory information for their execution. Finally, one might expect damage to the basal ganglia to have a greater impact on sequential or ballistic movements.

2.1.3 Muscles of speech and sign production

The muscles of the oral tract and of the hands are similar in that they both have a high ratio of motor nerve endings to muscle fibres, and also they both have large somatotopic representations throughout the motor structures of the brain. The high proportion of nerve endings and the configurations of the muscles themselves give the hands, the arms and the tongue a wide range of possible movements. But at that point the similarities end. The two systems of muscles differ greatly, particularly in terms of proprioceptive feedback. The muscles of the hands (and arms) all have muscle spindles which send information back to the central nervous system, indicating the degree to which a muscle is stretched. By contrast, most of the facial muscles (including muscles of the lips) do not have spindles (McComas, 1998) but rely on external sensory receptors for proprioceptive feedback. The muscles of the jaw and tongue do have spindles, though their distribution is uneven (Appenteng, 1990; Cooper, 1953). Additionally, the muscles of the hands and arms operate in conjunction with multiple sets of joints, which provides another channel of proprioceptive information to the central nervous system.

The mechanical configuration of muscles, bones and joints also puts loads on the muscles of the limbs, and causes them to be arranged in agonist-antagonist relationships. By contrast, the muscles of speech production only have to displace the mandible, and to a lesser extent, the hyoid bone. The tongue, the lips, the larynx and the velum move almost fully independently of any bone structure (Ackermann et al., 1997). Hence, they carry less weight, collect less proprioceptive feedback, and are unaffected by the activity or inactivity of opposing muscles. Interestingly, in spite of all the mechanical differences, the basic kinematics of simple movements in the two systems are reported to be very similar (Hertrich & Ackermann, 1997; Ostry & Cooke, 1987). Comparisons of more complex movements across the two systems have yet to be made.

2.2 The Mechanics of Speech

The speech production mechanism is divided into three basic components: an energy supply (the lungs/respiratory system), a vibrating sound source (the larynx), and a resonance chamber (the supralaryngeal vocal tract) (Fry, 1979). Pneumatic pressure from the lungs provides a source of energy that is controlled and released by the

opening and closing of the larynx. Because the energy source is crucial to speech and to breathing, speech production units are to some extent framed by respiratory patterns: one can only phonate for so long without having to pause to breathe. Additionally, to produce fluent speech, the three components and their separate articulators must move quickly and with accurate timing relative to the others.

Though the larynx and lungs are necessary for speech production and shape speech at the level of phrasing and prosody, the movements of the supralaryngeal vocal tract do more to differentiate one phoneme from another. Not coincidentally, the supralaryngeal articulators, and the tongue in particular, have a broader range of movement than the larynx or lungs which can only move symmetrically and along one axis.

Regarding the nature of the movements of the supralaryngeal vocal tract, Ackermann et al. (1997) have claimed that speech uses primarily ballistic movements. No one seems to have shown this empirically, but the configuration of the system, including the paucity of spindulated muscle fibres, suggests that it is a plausible hypothesis. One articulator that would probably use a combination of ballistic and guided movements, however, is the jaw. MacNeilage et al. (2000) have suggested that the jaw acts as a primary oscillator in the speech system, as illustrated by the babbling movements of infants, thereby creating a framework for later syllable structure.

2.3 The Mechanics of Sign Compared to Speech

Because signed languages use a visual-gestural production medium, the number of possible sign articulators is extremely high. The chief practical limitations are that articulators be visible to interlocutors and allow a range and speed of movement that facilitate a productive lexicon and minimal memory/perceptual demands. Much of the upper body is used in the articulation of signs, including: the head, torso, shoulders, upper arms, lips, and eyebrows. However, one could argue that the two hands are the primary articulators, because it is their movements that do the most to differentiate one sign from another. Like the supralaryngeal vocal tract, the hands can take on an wide variety of configurations; whereas the shoulders and the larynx cannot. The orientation, internal configuration, movement and location of the hand are the main phonological parameters that have been identified and analysed (Brentari, 1998); and it is worth noting that findings from psycholinguistic research have indicated that these phonological parameters of signs are psychologically real (Dye & Shih, in press; Klima & Bellugi, 1979).

The above description implies that the two hands/arms must function in a co-ordinated manner, or as Meier (2002) stated, "the articulators are paired," but the demands on the system actually go beyond just that. First, it should be pointed out that co-ordination does not imply symmetry. While some signs do require the two limbs to move symmetrically, others require asymmetrical, and consequently more complicated, co-ordination (Wiesendanger et al., 1994; Wiesendanger et al., 1996). While at a phonological level, signs are said to have a symmetry constraint (Battison, 1974; Sutton-Spence, 1999), the two arms can carry out the same movement alternately, without violating this constraint. But purely in terms of movement and co-ordination, this type of sign is more demanding. Moreover, the symmetry constraint only applies to individual signs and does not disallow the production of two signs simultaneously, one on each hand. Similar alternating or simultaneous forms do not arise in spoken language, because the production system is not capable of them (Meier, 2002). Additionally, the movements of the hands must be co-ordinated with those of the other articulators, putting more co-ordination demands on the sign production system. While speech also has to co-ordinate multiple sets of articulators (e.g. the lips, the jaw, the larynx), those articulators are easier to co-ordinate because they do not have as many degrees of freedom in their movements; in fact, some, like the jaw can scarcely move along more than one axis.

Co-ordination of manual and non-manual elements of signs do not necessarily serve to differentiate one sign from another, but rather to produce well-formed syntactic and prosodic phrases. In this last respect, one could draw a parallel between the function of non-manual sign articulators and one function of the larynx and lungs in speech production, namely, the division of extended discourse into prosodic phrases. However, some have argued that non-manual articulators do much more than encode prosody, and that they serve important syntactic and lexical functions (Antzakas & Woll, 2002; Neidle et al., 2000). But also, at a physiological level, the two sets of structures are extremely dissimilar. While the movements of the head and face must be co-ordinated with movements of the hands and arms to form normal, fluent signing, speech itself cannot exist in the absence of laryngeal and respiratory function. There is no analogous single energy source for signing, only the generalized muscle function of all the necessary articulators.

2.3.1 Sign as multichannel production system

Meier (2002) proposed that because of how they are perceived, the vocal-auditory channel is inherently one-dimensional, while the visual-gestural channel is four-dimensional. (As an aside, it should be pointed out that if the latter is four-dimensional, then the former must be at least two-dimensional because it makes use of changes in sound over time.) However, taking as a given the premise that signed language makes use of a multichannel system, the explanation of that could be as much production- as perception-based. While it is true that it is easier to perceive co-occurring visual phenomena than their auditory analogues, it is also true that it is easier to produce multiple, simultaneous articulatory units with effectors that are largely independent of each other, as the two arms are. Patterns of errors in signed language reveal its multichannel structure. Klima & Bellugi (1979) described slips of the hand that pattern basically along the same lines as slips of the tongue in speech: metathesis, anticipation and perseveration of individual phonological features. More recently, though, slips of the hand have been analysed in terms which more clearly illustrate the multichannel structure of signing (Hohenberger et al., 2002). Unlike in speech, two neighbouring signs can combine to form one sign, in what the authors referred to as a fusion error. What is distinct about this is that it is possible for features of two signs to be present simultaneously, one on each hand, which is not possible in speech. These slips reflect the greater physiological independence of individual articulators, which makes it possible for multiple phonological features to be produced at once. Hohenberger et al. (2002) also pointed out that stranding errors, in which two syllables or morphemes swap word-initial positions (e.g. talking Turkish -> turking talkish), do not occur in signed languages because sign segments occur simultaneously rather than sequentially, which supports the idea of sign being a multichannel production system.

2.3.2 Movement speed in sign

Relative to speech, sign uses very large articulators (Meier, 2002), which as described above, bear considerably more weight than speech articulators. Hence, signs are articulated at a much slower rate. Klima & Bellugi (1979) found that ASL/English bilinguals produce spoken words at approximately twice the rate of individual signs, with a mean rate of 4.7 words/second and 2.37 signs/second; although the rate of proposition production is roughly equal for the two modalities. However, there are many additional factors potentially influencing speed of articulation in signed language (Table 2.1). In comparing speech with non-linguistic limb movements, Ackermann et al.

(1997) claimed that speech movements are likely to be quicker because they are smaller: not only that the articulators themselves are smaller, but that movement trajectories are shorter. Although Ackermann et al. (1997) were not examining comparable tasks across the two modalities, the basic principle probably still holds true for sign production as well as non-linguistic limb movements.

Table 2.1: Factors contributing to sign speed

Articulator size
Movement size
Targeting demands / Sensory feedback
Movement complexity

Additionally, the number of possible locations in space that sign articulators can move to is effectively limitless, which could easily be a computational burden and slow down production. Undoubtedly, the linguistic production system employs strategies to reduce the number of possible (or likely) articulatory targets; however, it still seems probable that distinctive articulatory targets in signed language greatly outnumber analogous targets in speech. And as previously mentioned, the sign articulators provide much more sensory feedback (both proprioceptive and visual), allowing movements to be guided rather than ballistic, which also contributes to sign production being slower.

Finally, the mere complexity of sign movements could render sign production slower. It is a well-established principle of psychophysics that there is a trade-off between the speed and precision of movements (Fitts & Peterson, 1964), but there are not yet enough data to compare the physical precision of articulatory targets (either locations in space or configurations of articulators themselves) in sign as opposed to speech (Cormier et al., 1999). It is known, however, that sign movements require the co-ordination of multiple articulators, which unlike speech, are controlled largely independently. Additionally, because sign production is a multichannel system, the signer transmits many separate pieces of information in the production of an individual sign, using multiple movement parameters, which are both computationally and biomechanically demanding.

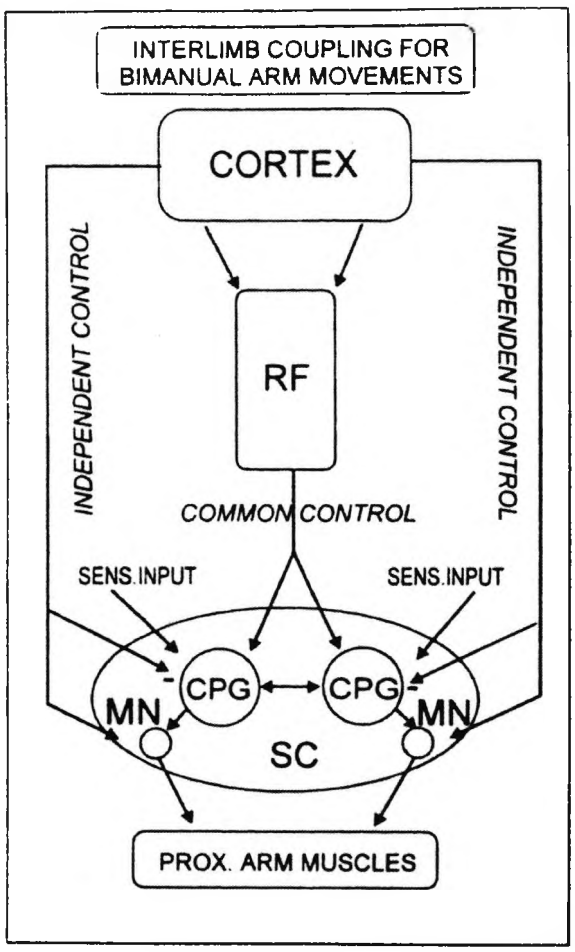

Figure 2.3: Wiesendanger et al. (1996) model of bimanual co-ordination (RF: reticular formation, SC: spinal cord, MN: motor neuron, CPG: central pattern generator)

2.3.3 Oscillators

Meier (2002) referred to the proposal that speech has a predominant oscillator (MacNeilage et al., 2000; Meier et al., 1997) and suggested that signed language probably does not. Meier pointed out that basic, automatic human movements such as walking, chewing, and vocal babbling are oscillatory in nature and reiterated MacNeilage's suggestion that speech articulation might develop from this automatic movement pattern; additionally, he speculated that there is no analogous automatic basis for the development of sign articulation. It is not completely clear what the implications of this claim for adult, synchronic sign production would be; but putting that question aside, it is also not clear that signed language does not have a predominant oscillator similar to the one for speech. Wiesendanger et al. (1996) have outlined a model of bimanual co-ordination that allows for both tight temporal coupling and flexible control of movement (Figure 2.3). According to this model, more complex movements employ

a central command structure, while simple rhythmic movements are co-ordinated at the level of the brainstem or spinal cord. The highest level probably picks the roles that each hand will play in a bimanual task (e.g. to hold or to manipulate an object). It has also been found that proximal limb movements are more tightly synchronized than distal limb movements (DiStefano et al., 1980). The synchronization of proximal movements could be the underlying mechanism for rhythmic bilateral arm movements in infants, not unlike the rhythmic jaw movements described by MacNeilage et al. (2000).

2.3.4 Sensory feedback

One of the most striking inherent contrasts between the two linguistic modalities, and one that seems to have gone completely overlooked, is the nature of sensory feedback to the signer or speaker. Signers can use visual feedback to guide or plan movement, while speakers cannot use auditory feedback to guide speech movements effectively. Also, because proprioceptive feedback from speech is limited, speakers probably cannot make use of it to execute guided, rather than ballistic movements. In effect, feedback is more ongoing for sign than for speech: it is comparatively easy to assess where your hand is at any given instant during signing, either visually or proprioceptively, and alter its position accordingly. By contrast, a speaker cannot hear what her articulators are doing prior to their having done it, except by slowing down considerably and distorting the speech signal. On a related point, anecdotal evidence suggests a greater role for non-proprioceptive sensory feedback in sign than in speech, as illustrated by the effects of Usher Syndrome on signing space.

Perceptual psycholinguistic research has indicated that there is not a lot of form-based language processing in signed language, but rather sign features are perceived more gradiently (Corina & Hildebrandt, 2002). In other words, features of signs are not automatically perceived as fitting into one phonological category or another with no productions seen as being "in between" categories. Corina & Hildebrandt suggested that this may be related to the nature of the output mechanism and how it is consequently perceived. Additionally, they suggested that there may be a more direct mapping between articulatory gesture and perceived form, rendering the articulatory gesture more transparent to signers. There are two other possible interpretations of gradient as opposed to categorical perception of phonological features. First, it may be that phonological features are more variable from one production to another, because signers can easily modify language production (and hence phonological form) online, whereas

speakers cannot, assuming their movements are largely ballistic. Second, sign phonology features could be perceived gradiently for the same reason that spoken language vowels are perceived more gradiently than consonants: they are longer and slower. What Corina & Hildebrandt described as a modality effect may in fact be solely an effect of speed. Sandler (1993) also argued that the movement (in a phonological sense) of a sign is the most sonorous component of the sign syllable, in part because it has perceptual salience by virtue of presenting motion. It should be noted, though, that sign movements also have longer duration than static elements of sign, which in itself could make the movements more salient.

2.3.5 Complexity but redundancy?

Another modality difference between sign and speech is that signers can use different articulators to produce the same sign and still be comprehensible. For example, in the BSL sign THROW (see Appendix A), a signer can move either from the wrist or from the elbow, and not really change how the sign is perceived. Signers are reported to produce the same sign using variably more distal or proximal articulators across multiple productions (Crasborn, 2001). Data from children acquiring sign language as a first language have suggested they frequently use more proximal articulators than the adults from whom they are learning the language (Meier et al., 1998), so it may be that there is not a one-to-one mapping between a sign and its designated articulators. Also, as Brentari (1998) pointed out, the non-dominant hand in a symmetrical two-handed sign can be left out without affecting intelligibility or even well-formedness. So, while the production of signs would seem to require more complex co-ordination, and could consequently be disrupted more easily, there may in fact be enough redundancy in the system that sign intelligibility is actually more difficult to disrupt. Another way of viewing this is that the range of acceptable forms of a sign may be broader than that for acceptable forms of a spoken word, or that the constraints governing acceptability may run along articulatory parameters that have yet to be examined.

2.3.6 Fingerspelling

A discussion of the physical form of signed languages would be incomplete without a description of fingerspelling and its physical form. For the sake of clarity, it should be noted that this discussion of fingerspelling does not include lexicalized forms that contain only a single fingerspelled letter, as in the BSL sign MEMBER, or forms that take on the morphophonological properties of signs, as in the ASL sign A-L-L.

Though fingerspelling can (but does not necessarily) take drastically different forms in different signed languages (Sutton-Spence, 1994), there are commonalities across fingerspelling systems, as well as consistent differences between the structure of fingerspelling and the structure of signing. The structure of fingerspelling is probably shaped to a large extent by its function in signed languages. Fingerspelling systems have developed to allow words to be borrowed from spoken language; what a signer does when fingerspelling is to represent the written form of a word, letter by letter (in the case of an alphabet). Most of western Europe and North America and much of the New World use a one-handed fingerspelling system of European origin, with different hand configurations for the letters of the Roman alphabet. However, Britain and many of its former colonies use a two-handed system, in which the dominant hand makes contact at different points on the non-dominant hand in different configurations to represent individual letters. So while one-handed fingerspelling systems rely primarily on contrasts in handshape, two-handed systems use a combination of contrasts in both handshape and location. It should be stressed that despite the fact that fingerspelling systems are by their nature sequential, there is a lot of articulatory overlap in fluent fingerspelling (in either one-handed or two-handed form), with features of one letter continuing into the next letter or preceding the completion of the previous letter (Akamatsu, 1983; Tyrone et al., 1999; Wilcox, 1992).

Because fingerspelling depicts words approximately according to their written form, the necessary rate of information transfer is often much higher than in the production of signs[3]. Although signs can be broken down into sublexical units, those units are not usually produced serially, and never with more than three units in sequence in one sign. By contrast, fingerspelling consists of sequences of individual units corresponding to ordered sequences of graphemes, which can include many units per production, or per word (Battison, 1978; Wilcox, 1992).

As suggested by the name, fingerspelling systems use only the hands and fingers as their articulators. There is no inherent reason why this must be the case, though it probably facilitates rapid production speed (Sutton-Spence, 1994), but it seems to be consistently true. Because the fingers are relatively small and highly innervated articulators, they can move quickly, so perhaps they could easily meet the demands of a rapid production system. Also, despite the fact that some fingerspelling systems use one

[3] While all fingerspelling systems are grapheme-based, most of the ones studied to date are alphabetical, but ideographic and syllabic fingerspelling systems exist as well (Zhou, 1980).

hand and others use two, they all seem to use highly delimited areas of signing space (Battison et al., 1975), which again would facilitate rapid production. It has been suggested that because of its rapid, serial nature, fingerspelling production might reveal motor control deficits earlier or to a greater degree than sign production (Poizner et al., 2000; Tyrone et al., 1999).

2.4 Concluding remarks

It is not unusual for speech motor control researchers to discuss the relative speed and co-ordination demands of speech movements and limb movements, despite the fact that the two sets of tasks typically being compared are completely different in terms of familiarity, complexity, and difficulty. Ackermann et al. (1997) stated that limb movements are slower than speech movements. Based on the biomechanics of the two motor apparatuses, this is probably true; however, it would be informative to examine limb movements that approximate the speed and complexity of speech. It is also important to tease out the role of sensory feedback in the two modalities, since standardized motor control tasks for the limbs require vision and speech does not. Despite the extent to which it differs in physical structure from speech, sign language production will nonetheless be the most comparable task that uses the hands and limbs.

In ordinary non-linguistic bimanual tasks, the movements of the two limbs are tightly coordinated, not at all points of an action sequence, but at the critical times for accomplishment of the task (Weiss et al., 2000), suggesting that motor tasks are structured in terms of goal achievement rather than the actual dynamics of separate movements. This is likely to be true for sign production as well, but would be an interesting question to explore, given the temporal and co-ordination demands of signing relative to non-linguistic arm and hand movements.

On the basis of the neural systems governing movement and the physical structure of the output mechanism itself, one would predict that bilateral co-ordination is more likely to break down in sign dysarthria, because the two limbs are controlled independently but often have to act either symmetrically or in some more complex form of co-ordination. While speech also requires bilateral co-ordination, its movements can only be symmetrical; besides which, speech output structures get more bilateral innervation and are hence less affected by unilateral cerebral damage.

Damage to the pyramidal tract has the capacity to affect either speech or signing, given its importance for fine-grained movement. Additionally, recent findings contradicting the idea of a strict somatotopic division of motor areas suggest that, for

example, sign language production could be disrupted by damage to areas typically thought of as controlling movements of the mouth (Meister et al., 2003). However, the fact that the speech articulators receive more bilateral input from motor areas of the cortex suggests that speech breakdown following damage to only one side of the brain is more likely to lead to mild or very high level disruption, perhaps at the level of movement schemata. Apart from the question of lateralization of innervation patterns, it remains a possibility that sign could break down independently of speech or vice versa, but it is only the rare case that would allow researchers to identify a lesion affecting one modality but not the other.

Because they are both implicated in complex (i.e. multijoint or sequential) movements, movement timing and co-ordination, the cerebellum and basal ganglia are likely to play an important role in sign language as well as speech production. Also, the fact that neither structure seems to have an effector-specific function renders them both more likely to serve a cross-modal function in language production. The specific role that either structure is likely to play, however, is far from clear.

Duffy (1995) pointed out that speech is motorically complex and susceptible to disruption from neurological injury because it requires symmetric movements of bilaterally innervated structures. Signed language, however, is also motorically complex, in some ways even more so than speech, and requires not only symmetric but also asymmetric movements of *contralaterally*-innervated structures, which can move completely independently of each other. So if the nature of speech renders it highly susceptible to disruption from injury, what can be said of sign's susceptibility to similar disruption? Is the sign production system more robust and less susceptible to breakdown due to its inherent productive flexibility? These are among the most important questions that research on the physical basis of signing can hope to answer.

3 Dysarthric Speech

3.1 Introduction

3.1.1 Dysarthria as motor disorder and neurogenic

Before exploring the nature of sign dysarthria, it is necessary to first describe what has, up until now, been the only recognized form of dysarthria, namely, disrupted speech. Much of the definitive work on describing the fundamental articulatory characteristics of different types of dysarthria was done quite a while ago. As Kent (2000) pointed out, the basic classification scheme currently in use for the dysarthrias is based almost entirely on two papers from over thirty years ago (Darley, Aronson & Brown, 1969a, b). Research on dysarthria over the past decade has focused on correlating dysarthria to other factors, such as brain pathology (Urban et al., 2003; Kluin et al., 2001) or the effectiveness of various treatments (Pinto et al., 2003; Schulz & Grant, 2000); or on exploring non-articulatory measures of the disorder, such as acoustics, neurophysiology or small-scale kinematics, or combinations of those three (Goozee et al., 2001). Various researchers have argued that acoustic, neuroanatomical, and kinematic measures are more relevant, objective, and/or precise than the perceptual measures developed by Darley et al. (1969a, b); however, no one has substantively questioned or reframed those perceptual measures, with the possible exception of Enderby (1983), who described roughly the same measures in more anatomical terms as part of a formalized dysarthria assessment. In other words, no one has developed a framework of different *perceptual* measures, rather than trying to define dysarthria in terms of another category of measures altogether (e.g. acoustic rather than perceptual). Moreover, the measures established by Darley et al. (1969a, b) are still in widespread use among speech and language clinicians and researchers (DeBodt et al., 2002; Ozsancak et al., 2000).

The perceptual measures developed by Darley et al. (1969a, b, 1975) and their relation to other studies and methodologies will be explored later in this chapter. However, it is worth noting that because the primary purpose of this chapter is to describe the different types of dysarthria, where older studies, such as those by Darley, Aronson, and Brown, remain the most relevant, they are the ones that are referred to, rather than newer studies addressing issues that are secondary to basic description and classification of varieties of dysarthria.

Kent (2000) defined dysarthria as "speech disorders that result from neurologic impairments associated with weakness, slowness, or incoordination of the musculature used to produce speech." This definition is consistent with those presented by other

researchers (Hartman & Abbs, 1988; Duffy, 1995). One important aspect of the definition is that it covers a potentially broad range of deficits due to the range, variable extent, and possible combination of specific disruptions that it includes (e.g., mild slowness and severe inco-ordination of the vocal tract mechanism in the absence of muscular weakness). Moreover, Kent (2000), like Darley, Aronson and Brown (1975), explicitly stated that by definition, dysarthria is a neurological symptom. Often this point is not stated outright; however, researchers and clinicians agree that dysarthria stems from some neurological aetiology, either central or peripheral. Furthermore, dysarthria is a disorder of speech, not of language, precisely because it is a movement disorder.

Although they were not the first to recognize that speech could be disrupted in the absence of linguistic or cognitive impairments, Darley, Aronson, & Brown (1969a) were the first to pursue the idea of there being different *varieties* of dysarthria, depending on the type and location of neurological damage, which could be distinguished and categorized, provided that speech was assessed according to suitable parameters. Those parameters are described briefly in section 3.1.2 below. It should be noted that the existence of different types of dysarthria does not imply that a specific type cannot occur in combination with others. In particular, given the fact that the type of dysarthria is determined in part by the nature of the underlying neurological damage, it stands to reason that damage to more than one area will cause a combination of more than one form of dysarthria. Similarly, although it is solely a movement disorder, dysarthria can also occur in combination with other linguistic or cognitive disorders.

For purposes of this thesis, dysarthria is defined to be neurogenic but not psychogenic; hence, there will be no descriptions of mutism caused by psychological trauma, for example. Additionally, while dysarthria can occur as a result of damage to peripheral nerves, those cases will also be excluded from discussion here, first because their implications for non-orofacial articulators are not as great, but also because such cases are very nearly unique in their capacity to affect individual muscles or articulators in isolation. Any dysarthria that is by definition effector-specific, like spasmodic dysphonia, will be excluded from discussion for the same reasons. Finally, the discussion will be limited to the dysarthrias of adulthood, in order to exclude conditions that may impact on an individual's ability to learn the patterns of motor execution necessary for language production, or that may impact on development more generally. However, a description of apraxia of speech is included in this discussion, despite it not being clear whether or not to consider it as distinct from dysarthria. It is included here,

in large part, to explore that question. For that reason, and because of how apraxia of speech has traditionally been defined and typologized, the section on apraxia will take a slightly different structure from the other sections. Within this framework, each of the different forms of motor speech disorders will be summarized, with a description of how they are typically analysed and a discussion of potential problems with those analyses.

3.1.2 Measures of dysarthric speech

Because speech itself is an extremely complex motor act, its measures are by necessity complex and multi-dimensional as well. But apart from the question of complexity, there are many methodologies for measuring speech, because there are many aspects one can measure. The most common approaches are to measure: speech articulators' movements, the acoustic stream generated, or a listener's perception of the speech produced. Most studies have examined one of these sets of measures alone, though a few studies have compared multiple measures to assess how they may be related (DeBodt et al., 2002; Weismer et al., 2001).

In order to precisely measure the movements of articulators during speech, it is necessary to attach instruments to track articulator movements over space and time. However, because most speech articulators remain within an enclosed space, i.e. the vocal tract, it can be difficult to measure their movements precisely. Kinematic systems can be used to measure speech movements (Ackermann et al., 1997), but those that are optoelectronic are only used to record the movements of one or two articulators at once, usually the lips and jaw (Forrest et al., 1989). Another type of kinematic technique is electromagnetic articulography, which uses magnetic coils attached to the tongue to record movements during speech (Goozee et al., 2000; Jaeger et al., 2000). However, this method is not often applied to atypical speakers, and is more often used to assess normal speech processes. As a result, at this point, it is difficult to assess co-ordination of multiple articulators in dysarthria in any detail on the basis of existing literature, which is problematic given that many types of dysarthria appear to disrupt co-ordination specifically. Furthermore, the large amount of data collected in a short period by a kinematic system also dictates that researchers collect very limited sets of data, such as repetition of a single syllable, rather than a closer approximation of natural speech.

Spectrographic recordings can be used to measure the acoustic output of speech production over time. Spectrograms allow measurement of both the absolute values and variability of: pitch, voicing, loudness, spirantization, and segment duration (whether

the segment being analysed is a phoneme, a syllable, or a word) of continuous speech. Unlike direct measures of articulation, spectrographic recording does not interfere with speech movements; however, an acoustic measure can only ever be an indirect measure of those movements. It is not easy, and sometimes not even possible, to infer articulator movement from an acoustic correlate.

Finally, clinicians or researchers can record their observations of someone's speech patterns according to established measures. Unlike the first two methodologies described, perceptual measures of speech do not require the use of especially elaborate equipment. As long as the speaker's voice is audibly recorded and the observer is trained, data can be collected and analysed. Exact measures or the assignment of their values may vary from one institution to another, but broadly speaking, the speech components are the same as those laid out by Darley, Aronson, and Brown (1975):

- Articulation[4]
- Pitch
- Loudness
- Prosody
- Voice quality
- Resonance/nasality
- Respiration

As with acoustic analysis, any of these can be assessed for relative as well as absolute value. In addition to being assessed by these measures, speech is often also described in terms of a holistic, subjective measure of intelligibility. As stated above, many assessments and methodologies have been developed for analysing dysarthria since these categories were created, but no separate set of articulatory measures has been proposed. Moreover, most studies of intelligibility which are implemented by clinicians rely on this framework.

Several studies have tried to compare different measures of dysarthria (acoustic, articulatory, perceptual) for the same subjects. As the number of such studies has increased, researchers have found more contradictions between perceptual and physical data, as well as physical measures that bear no relationship to the functional measures of

[4] It is necessary to include a note about terminology here. Darley et al. use articulation to mean co-ordinated movements of the supralaryngeal articulators. However, the term articulator in this chapter is intended to refer to any effector or set of effectors in the speech production mechanism.

intelligibility and naturalness (Forrest et al., 1991; McHenry, 2001; Weismer et al., 2001; Zeplin & Kent, 1996). In some cases, researchers have noted acoustic results that supposedly reflect components of a motor speech disorder but also contradict perceptual results (Wertz et al., 1998). This highlights the importance of including a variety of measures in the examination and categorization of subjects with dysarthria.

For the reasons outlined, the focus of this chapter will be on perceptual and articulatory measures, though consideration of other types of measures will be included. Ideally, the best way to go about analysing and categorizing types of dysarthria would be to compare perceptual, acoustic, and instrumented articulatory measures with the subject's own perceptions. However, in the absence of such comparative studies, perceptual observations best capture the differences across the dysarthrias as well as the breadth of individual dysarthria types. Additionally, perceptual and articulatory measures are the most comparable to the types of measures to be used in analyses of the sign language data.

There is concern on the part of some researchers that perceptual measures are not sufficiently objective or precise (Kent et al., 2001; Zeplin & Kent, 1996), and clearly they do not reach the level of physical precision of acoustic or kinematic measures. However, what perceptual measures offer is a more holistic analysis of speech which no physical measure can yet accomplish, and which arguably has more bearing on the communicative limitations of an individual with a motor speech disorder. That said, the real difficulties with perceptual measures are twofold: the tendency to conflate perceived behaviour with underlying pathology, and the tendency to incorrectly equate a component of speech with a specific set of articulators.

3.1.3 Disruption to speech vs. disruption to articulators

Because most neurogenic disruptions to speech simultaneously affect more than one component of the physical articulation mechanism, it is worthwhile to focus on how speech as a co-ordinated act is disrupted, rather than looking at the impacts on movements of components of the vocal tract in isolation. It has been suggested that the dysarthrias as a whole have global effects on multiple components of speech, rather than focal effects on individual components (Kent et al., 1998). Similarly, there is often an unclear relationship between dysarthric subjects' performance on speech tasks and on non-speech tasks (Kent et al., 2000; Ziegler & Wessel, 1996), which also suggests that the actions of individual articulators are not always the most important factor to examine in describing a given type of dysarthria. Articulator-specific dysarthria is most

often the result of dysfunction of peripheral nerves, which has been excluded from this analysis as outlined above. When dysarthria is a manifestation of the inability of multiple articulatory systems to act in concert, it would be misguided to focus on the movements, strength, or tone of individual articulators. As Ludlow & Bassich (1983) put it: "Walking is no more the direct result of the strength of the biceps femoris than speech impairment can be directly reflected by the strength of contraction of the orbicularis oris."

In the list of perceptual speech measures above, most of the measures can be (and usually are) equated with a single articulator or set of articulators, even when such equations seem plainly oversimplified (e.g., loudness as a reflection of respiration). The main exception to this is prosody, which by anyone's definition requires the co-ordinated movement of multiple sets of articulators. Another measure, not included in this list, though frequently used by clinicians and researchers, is stress, which also requires the simultaneous interaction of multiple articulators. At least anecdotally, both stress and prosody are psychologically real: it is easy to describe a person's speech as monotonous or flat, in ordinary conversation as well as in a clinical or research context. However, researchers have had limited success in defining exactly what they mean by prosody or stress, not because definitions are not possible, but because they have placed their measures within an anatomical framework that is artificially restrictive. An anatomical framework can be used, but it has to be multi-dimensional, if it is to capture the full range of disruptions to speech motor control.

3.1.4 Disruption to speech vs. damage to the brain

Along similar lines, some characteristics of speech as well as some categories of dysarthria have been described in terms of their underlying pathology rather than their behavioural manifestation. For instance, in some versions of the schema of speech characteristics listed above (e.g., Duffy (1995)), one form of pitch disruption is classified as myoclonus, which is a movement disorder, not a property of sound. Using the two categories of terms indiscriminately is imprecise, because one can only be an indirect measure of the other. While it is true, as Darley et al. (1975) stated, that dysarthria is a neurological symptom and should be categorized as such, that is not to say that a given type of dysarthria should be defined according to its neurological basis to the exclusion of, or even in preference to, other criteria. It should be remembered that broadly speaking, the functioning of the human brain is at least as poorly understood by researchers as the nature of speech motor control, if not more so. Thus,

neurophysiological data should not automatically be interpreted as being more scientific or having greater explanatory value than easily observable behaviour. Second, while researchers are simultaneously trying to better understand the nature of different dysarthrias and the functions of different parts of the brain, to conflate the measures or the terminology of the two fields only serves to make both tasks more difficult. In their review, Kent et al. (2001) provided a summary of documented relationships between lesion site and type of dysarthria, suggesting a more or less one-to-one relationship between the two. This is often not the case. It is not always clear, for example, in the case of unilateral upper motor neuron dysarthria (described below), why damage to exactly the same neural structures results in a specific type of dysarthria for one individual but not for another. As long as dysarthric symptoms are categorized according to neural correlates instead of behavioural symptoms, it will be difficult to address this question.

3.2 Types of Dysarthria

The section that follows will outline the well-known forms of dysarthria in terms of anatomy, accompanying non-dysarthric symptoms, and speech characteristics, with relevant comments included on the clinical and experimental basis for those characterizations. A short list of the speech symptomatology of different types of dysarthria is included in Table 3.1.

3.2.1 Ataxic Dysarthria

Ataxic dysarthria is a manifestation of unco-ordinated movements and hypotonia of the speech muscles. Speech is generally perceived as slow and imprecise, with irregular variations in pitch and loudness, and a "scanning" rhythm (Kent et al., 2000). Ataxia is almost always the result of damage to either the cerebellum, its incoming projections, or both. A few researchers have tried to assign specific speech functions to more narrowly-defined regions of the cerebellum (Ackermann & Hertrich, 2000; Darley et al., 1975; Urban et al., 2003), but such approaches are incomplete, because clinical cases of damage to limited areas of the cerebellum are comparatively rare.

Most researchers agree that ataxia impacts multiple groups of speech articulators at once, rather than specific sets of articulators in isolation (Kent et al., 1997; Murdoch & Theodoros, 1998; Sheard et al., 1991). Additionally, clinical observation has suggested that speech co-ordination and timing are affected to a greater degree than strength or mobility of the speech musculature (Duffy, 1995). A few comparative

studies have suggested that perceptual ratings of ataxic dysarthria reflect a combination of speech components rather than a single acoustic measure (Sheard et al., 1991), and that broad-based measures of intelligibility and naturalness do not correlate with any acoustic measure (Kent et al., 2000; Linebaugh & Wolfe, 1984), even though ataxic subjects get low intelligibility ratings (Liss et al., 2000).

Symptoms

Broadly speaking, the general motor symptoms of ataxia parallel those of ataxic dysarthria. Darley, Aronson, and Brown (1975) probably captured ataxia best in lay terms when they said that finger movements are "clumsy, slow, and fumbling," while more proximal movements are "jerky, irregular, and inaccurate." More specifically, generalized motor symptoms include: reduced muscle tone, slowness, dysmetria, intention tremor, hyporeflexia, dysrhythmia, and dysdiadochokinesia (i.e. impairment of alternating sequences of movements) (Darley et al., 1975). Impairments to specific effectors or specific types of movement include: wide-based gait, postural tremor, and nystagmus (i.e. loss of control of eye movements) (Trouillasa et al., 1997).

Although the cerebellum is best known for its role in movement, it also has non-motor functions, which have not been explored in as much detail. An increasing number of non-motor functions of the cerebellum are being documented, but there is limited agreement about their nature and even their existence. Research has suggested that cerebellar damage may cause disruptions to language as well as speech (Fabbro, 2000; Fiez et al., 1996; Marien et al., 2000) and disruptions to neuropsychological functions such as learning and memory (Lalonde & Botez-Marquard, 2000; Timmann et al., 2002).

Speech characteristics

Despite the general agreement on the nature of ataxic dysarthria, there are disagreements about which speech characteristics are most prevalent. Additionally, even individual measures of speech characteristics appear slightly contradictory, at least superficially. For example, some acoustic and perceptual studies have suggested that subjects with ataxic dysarthria have more variable pitch and loudness (Darley et al., 1975; Kent et al., 1979; Kent et al., 2000), while others have suggested that subjects have a lack of variability in pitch and loudness (Darley et al., 1975; Kent et al., 1979). It may be that all of these analyses are correct, but they are capturing different components of the speech signal. Liss et al. (2000) summarized earlier findings by

saying that ataxic dysarthria is characterized by excess and equal stress, and excessive loudness; in which case, the co-occurrence of equal but excessive values could reflect the presence of both variability and consistency in pitch and loudness. Darley et al. (1975) proposed that there are subtypes of ataxic dysarthria which exhibit distinctive clusters of symptoms; however, later research suggested that pitch and loudness are variable on held syllables but show reduced variability from one syllable to another (Ackermann & Hertrich, 2000; Murdoch & Theodoros, 1998). Because it is unclear what clinical data Darley et al. were relying on, it is impossible to say whether the seemingly contradictory findings represent two subgroups of subjects or two articulatory phenomena, which are perhaps task-specific, in a homogeneous set of subjects.

Other specific characteristics of ataxic dysarthria described by Darley, Aronson, and Brown (1969a) include: exaggerated respiratory movements, harsh voice quality, prosodic excess, excess and equal stress, long syllables and phonemes, reduced rate, imprecise vowels and consonants, and irregular articulatory breakdown. Subsequent studies cite these findings frequently and none has yet contradicted any of them.

3.2.2 PD/Hypokinetic Dysarthria

The defining feature of hypokinetic dysarthria is considered to be a limited range of movement, which manifests itself in monotonous, aprosodic, but rapid speech. By far, the most common form of hypokinetic dysarthria is that resulting from Parkinson's disease (PD) (Theodoros & Murdoch, 1998b); and because it is the most common, it is also the most studied. Hypokinetic dysarthria can also be caused by vascular or traumatic accident, or by any of the other degenerative conditions described below. Unfortunately, early research grouped together all forms of hypokinetic dysarthria, under the assumption that they were all characteristically alike, when in fact they are not (Darley et al., 1969b; Nielsen, 1962). Because documented cases of Parkinson's disease so greatly outnumber those of any other form of hypokinesia, this section will list the different types of hypokinetic disorders, then describe the characteristics of what has been referred to simply as hypokinetic dysarthria, and then outline the characteristics that can differentiate PD and other forms of hypokinetic dysarthria from each other. It should be noted that documented hypokinetic speech characteristics probably primarily represent the speech characteristics of PD, though the two should not be viewed as co-extensive.

Darley, Aronson, and Brown (1969a) associated hypokinetic dysarthria solely with subcortical components of the extrapyramidal system, which they defined as consisting of the basal ganglia and subthalamic nucleus. It should be noted, however, that hypokinetic dysarthria can result from pathologies that affect additional parts of the central nervous system as well, for example, the brainstem and the cerebellum. It is entirely possible that hypokinetic dysarthria is caused specifically by damage to the aforementioned subcortical nuclei, but thus far the data are inconclusive.

Types of hypokinetic disorders

- Parkinson's disease is an idiopathic, degenerative disease of the basal ganglia, affecting between 0.1% and 1% of the population. The disease causes the dopamine-producing neurons in the substantia nigra to deteriorate, which in turn causes a chemical imbalance in the basal ganglia, disrupting its projections to motor areas of the cortex. The characteristic symptoms of PD include resting tremor, slowed movement, muscular rigidity, impaired postural reflexes, movement initiation difficulties, and a general reduction in spontaneous movement.
- Parkinsonism, a condition similar to PD, can occur secondary to encephalitis or head trauma impacting the substantia nigra. The symptoms are essentially the same, though the course and aetiology are different, and research on "Parkinsonian" dysarthria often groups idiopathic Parkinson's disease together with Parkinsonism indiscriminately.
- Progressive supranuclear palsy (PSP) is a degenerative disease causing neuronal atrophy primarily in the brainstem and less in the cerebellum. Though it is rare, it is the most common cause of hypokinetic dysarthria, after PD and Parkinsonism (Duffy, 1995). Its symptoms include reduction in the size and speed of movements, but do not include tremor. Also, unlike PD, one of PSP's characteristic symptoms is progressive loss of eye movement.
- Wilson's disease is a rare metabolic disorder causing degeneration of the lentiform nucleus of the basal ganglia. Characteristic symptoms include proximal arm tremor, slowed movement, axial rigidity, ataxia, and altered facial expression.
- Shy-Drager syndrome is a degenerative disease of the autonomic and central nervous systems, which directly impacts the basal ganglia and cerebellum. Its symptoms include rigidity, slow movement, and resting tremor.

Speech characteristics of hypokinetic dysarthria in general

It is generally agreed that hypokinetic subjects exhibit harsh, breathy voice quality, short rushes of speech, inappropriate silences, variable speech rates, reduced stress, and aprosodia (Darley et al., 1975; Weismer, 1984). Additionally, hypokinetic dysarthria is unique in that one of its characteristics is apparently rapid speech (Netsell et al., 1975). Darley et al. (1975) claimed this was a reflection of acceleration within an utterance rather than a consistently high velocity. Weismer (1984), on the other hand, suggested that hypokinetic speech may be perceived as rapid because individual phonemes are short and acoustic contrasts such as voicing and frication are blurred. In a study that directly compared speech and articulation rates, hypokinetic subjects were found to have a very high speech to pause ratio (Nishio & Niimi, 2001), which could explain why their speech is perceived as rapid. Less commonly reported symptoms of hypokinetic dysarthria include mild hypernasality (Kent & Rosenbek, 1982) and impaired breath support (Theodoros & Murdoch, 1998b).

As in the case of ataxic dysarthria, there are disagreements on the relative importance of specific speech characteristics as well as inconsistencies in the results across studies, perhaps because of changes and improvements in observational techniques or a more careful subcategorization of types of hypokinetic dysarthria. On the basis of perceptual and acoustic data, Darley et al. (1969a) identified imprecise consonants as a characteristic feature of hypokinetic dysarthria, but later clinical as well as perceptual and acoustic data show breathy, harsh voice quality and reduction in pitch variation to be more common than imprecise consonants or vowels in hypokinetic dysarthria. Some researchers have suggested that these speech characteristics may be the result of a problem with the larynx specifically (Hartman & Abbs, 1988; Schulz & Grant, 2000). However, it is unclear how damage to neural structures that influence motor patterns in general would result in dysarthric symptoms confined to a single articulator. What is more likely is that the problem identified as disrupted laryngeal function is a component of a more widespread muscular rigidity that is common in Parkinson's disease. (See Luschei et al. (1999) for a discussion of Parkinsonian rigidity in laryngeal and skeletal muscles.)

There are also apparent inconsistencies in findings related to the pitch and intensity of hypokinetic speech. Some studies have reported uniformly reduced intensity (Hartman & Abbs, 1988; Illes et al., 1988), while others have reported reduced intensity *range* (Darkins et al., 1988; Ludlow & Bassich, 1983; Ludlow & Bassich, 1984). Additionally, early perceptual studies suggested that hypokinetic subjects exhibited

monotonous, low pitch (Darley et al., 1975), and later acoustic studies suggested they exhibited elevated pitch (Goberman et al., 2002; Ludlow & Bassich, 1983; Sanabria et al., 2001). In an attempt to explain this, Duffy (1995) proposed that what listeners were perceiving as low pitch may have instead been monotonous or quiet speech.

Beyond the question of accuracy of specific measures or consistency across measures, there are external factors that could explain some of the inconsistencies in results across studies. First, few researchers have addressed or even mentioned disease stage when collecting data from Parkinsonian subjects. PD is a progressive disease, and it is well-known that some symptoms only emerge in the later stages of the disease (Hoehn & Yahr, 1967). An additional factor that few studies have addressed is the effect of Parkinsonian medication on speech. Though the effects of Parkinsonian medication on motor control in general are significant and well-documented (Duvoisin & Sage, 2001; Fahn et al., 1998), its effects on speech are not clear. Some research has indicated that measures of PD dysarthria improve while subjects are on levodopa (Gallena et al., 2001; Goberman et al., 2002; Sanabria et al., 2001); however, pharmacologic treatment in isolation does not consistently improve dysarthric symptoms of PD (Schulz & Grant, 2000). Studies not explicitly examining the effects of medication have often not controlled for medication status when testing (Liss et al., 2000), so comparing results across studies can be problematic.

To summarize briefly, dopaminergic medication used for the treatment of PD causes patients to experience irregular, uncontrolled movements or dyskinesias of the limb and face muscles. Because of how the medication is metabolized, patients experience on and off phases, in which the side effects are at their worst or non-existent, respectively. Given that dyskinesias are common to many individuals with PD, it is important to know a subject's medication status during testing, and thereby to know whether a deficit being measured is a symptom of the disease or a side effect of medication. It is quite possible that the presence of dyskinesias, or even the temporary abatement of Parkinsonian symptoms, could explain the differences in findings on pitch and intensity in hypokinetic dysarthria.

Finally, an early study suggested that hypokinetic dysarthrics exhibit palilalia, or a tendency to repeat entire words or phrases uncontrollably (Nielsen, 1962). However, Darley, Aronson, and Brown (1975) found that, contrary to what Nielsen had suggested: "The seemingly different phenomenon palilalia... was noted in none of the 32 (hypokinetic) subjects." This likely stems from the fact that most studies up to that point (and even today, to a lesser extent) grouped hypokinetic subjects together, even though

the accompanying dysarthria may take a different form, depending on disease pathology. Interestingly, the few researchers who have directly compared different forms of hypokinetic dysarthria have found palilalia to be one of the symptoms of PSP that can be most easily used to distinguish it from Parkinson's disease (Hartman & Abbs, 1988; Metter & Hanson, 1991; Testa et al., 2001).

Comparisons of different hypokinetic dysarthrias

As stated previously, cases of non-PD hypokinetic dysarthria are not very numerous, either in the literature or in clinical settings. Nonetheless, extant data suggest that there may be characteristic distinctions between different hypokinetic pathologies. PD subjects pattern more or less according to the early description of hypokinetic subjects, with a few exceptions. First, imprecise consonants are not very prevalent in Parkinson's disease (Muller et al., 2001). The grouping together of hypokinetic subjects with and without Parkinson's disease could explain why imprecise consonants and palilalia were identified as a symptom in some studies but not others, as described above. In addition, PD subjects are reported to exhibit reduced intensity, higher pitch, reduced pitch range, reduced stress, and harsh and breathy voice quality, relative to other hypokinetic subjects (Gentil et al., 2001; Hartman & Abbs, 1988; Lu et al., 1992; Muller et al., 2001).

In addition to palilalia, subjects with PSP exhibit imprecise articulation, reduced intensity, low pitch, reduced pitch range, hoarse voice quality, hypernasality, and slow speech rate (Hartman & Abbs, 1988; Lu et al., 1992). The last two characteristics (and possibly also the imprecise articulation) are what most clearly distinguish it from PD. Also, in broader terms, dysarthria is more likely to be a presenting symptom or to appear at all in PSP than in PD (Lu et al., 1992; Muller et al., 2001).

Shy-Drager syndrome and Wilson's disease are rarer than PSP, so it is more difficult to generalize about them. It is also difficult to speculate on how their characteristics may influence perceptions of hypokinetic dysarthria generally, if at all. Bearing those precautions in mind, the most characteristic dysarthric symptoms of Shy-Drager syndrome reported are: reduced pitch range (Ludlow & Bassich, 1983), imprecise articulation, disrupted phonation, slow speech rate, and atrophy of the larynx (Hanson et al., 1983). The last two of these clearly distinguish it from PD, but the muscular atrophy is more striking because it makes Shy-Drager syndrome unique among all central nervous system dysarthrias. Finally, the speech characteristics judged to be most severe in Wilson's disease overlap considerably with those of PD: reduced

stress, reduced pitch variation, reduced intensity variation, and imprecise consonants (Hartman & Abbs, 1988).

3.2.3 Hyperkinetic Dysarthria

While hypokinetic dysarthria's defining quality is a reduction in the magnitude of voluntary movements, hyperkinetic dysarthria does not traditionally refer to excessively large or rapid voluntary movements, but rather to the presence of *involuntary* movements in speech (Darley et al., 1975; Theodoros & Murdoch, 1998a). As a category, hyperkinetic dysarthria is extremely heterogeneous, in part because individuals can present with hyperkinetic dysarthria secondary to a condition causing another, more predominant variety of dysarthria, e.g. dystonia secondary to Parkinson's disease. Common neurological pathologies that can cause primary hyperkinetic dysarthria include Huntington's disease, Tourette's syndrome, cerebral palsy, and essential tremor. Because of its heterogeneity and because it has no direct bearing on the research subjects described in this study, hyperkinetic dysarthria will not be covered in detail in this discussion. A list of the characteristic features of different types of hyperkinetic dysarthria are included in Table 3.1; and a thorough discussion of its clinical manifestation and symptomatology can be found in Theodoros & Murdoch (1998a). It should be mentioned in passing that hyperkinetic movements in the limbs often occur as a side effect of the medications prescribed for Parkinson's disease; however, such pharmacologically-induced dyskinesias have not been documented in speech, so there is no literature to refer to for the variety of hyperkinesia most relevant to subjects in this study.

3.2.4 Spastic Dysarthria

Spastic dysarthria is a manifestation of heightened muscle tone and impairment of skilled movement, usually resulting from bilateral damage to fibres of the pyramidal tract. Speech is perceived as slow, effortful, and imprecise (Darley et al., 1975; Klasner & Yorkston, 2000; Nishio & Niimi, 2001). According to Duffy (1995), spastic dysarthria causes impaired movement patterns rather than muscle weakness, thus simultaneously affecting multiple components of speech. Darley et al. (1975) suggested that spastic dysarthria reflects four components of muscular dysfunction: spasticity (heightened muscle tone), weakness, limited range of movement, and slowness. Unlike the other forms of dysarthria described, there is no common degenerative disease that selectively causes spastic dysarthria; it is often the result of vascular or traumatic

accident, but can also be caused by diseases with diffuse distributions, such as multiple sclerosis or motor neuron disease. Clinically, spastic dysarthria may in some cases be the only indication of bilateral damage following stroke (Duffy, 1995).

Symptoms

Because spastic dysarthria results from pathologies with unpredictable distributions, it is potentially problematic to generalize about accompanying non-speech symptoms. However, given that it is usually the result of damage to motor structures from both cerebral hemispheres, spastic dysarthria is often associated with bilateral limb involvement (Duffy, 1995). The movements of the limbs, as well as the tongue and lips, may be reduced in range and force, with a loss of fine-grained, skilled movement, which is most pronounced in the hands and fingers (Brodal, 1998; Enderby, 1986). Additionally, in some cases, there is a lack of co-ordination of respiration and laryngeal function, causing dysphagia and breathing problems (Enderby, 1986). Spastic dysarthria may be accompanied by generalized hypertonia, hyper-reflexia, and involuntary repetitive muscle contractions in the limbs (Duffy, 1995). Although non-motor symptoms vary greatly according to distribution of damage, Darley et al. (1975) cited frequent occurrences of involuntary, misrepresentative emotional responses, such as laughing or crying.

Speech characteristics

Based on clinical data, Darley et al. (1975) reported that the distinguishing characteristics of spastic dysarthria were slow but regular speech rate, and harsh/strained voice quality. They reported both reduced and excessive stress and prosody, on the basis of both acoustic and perceptual data. A study of Japanese patients reported slow speech with an abnormal articulation rate in spastic dysarthria (Nishio & Niimi, 2001), but this could be due to the difference in syllabic structure in Japanese and English. Additional speech characteristics of spastic dysarthria include: long syllables (Linebaugh & Wolfe, 1984), hypernasality (Enderby, 1986), imprecise consonants and distorted vowels (Klasner & Yorkston, 2000), low pitch and reduced pitch range, reduced intensity range, pitch breaks, and breathy voice quality (Darley et al., 1975). Researchers have also reported generally decreased intelligibility in spastic dysarthria (Klasner & Yorkston, 2000; Linebaugh & Wolfe, 1984).

3.2.5 Unilateral UMN Dysarthria

Unilateral upper motor neuron dysarthria is unique among the central nervous system dysarthrias for being named for the location of a lesion rather than a description of its perceivable qualities. In fact, some researchers have treated unilateral UMN dysarthria as a subcategory of spastic dysarthria, because it results from similar pathologies but with damage to only one side of the brain (Thompson-Ward, 1998). As the name implies, it results from damage to fibres of the pyramidal tract on one side of the central nervous system. It is also unique among the dysarthrias in that even its occurrence is highly variable across patients with identical lesions (Kent et al., 2001).

Symptoms

It is difficult to make broad statements about the general symptomatology of unilateral UMN damage, because it does not often occur in isolation. Additionally, because the most common aetiology is vascular, the region of possible damage is not strictly delimited. However, clinical data have shown that the most frequently occurring non-speech symptoms accompanying unilateral UMN dysarthria include: mild dysdiadochokinesia, hemiplegia/paresis, tactile deficits, clumsy hand syndrome, and unilateral weakness in the lower face, tongue, or palate (less common). Although the non-speech symptoms are variable, it is extremely uncommon for unilateral UMN dysarthria not to be accompanied by other disorders. In fact, the speech characteristics of UMN dysarthria are often masked by more severe manifestations of aphasia, apraxia, or aprosodia (Duffy, 1995).

Speech characteristics

On the whole, unilateral UMN dysarthria is thought to be mild, both in relation to other dysarthrias and in relation to its accompanying non-speech symptoms (Kent et al., 2001). Duffy (1995) suggested that unilateral UMN dysarthria primarily affects articulation and probably reflects muscle weakness and inco-ordination, though it has also been reported to cause dysphonia. Acoustic and perceptual studies have suggested that unilateral UMN dysarthrics exhibit mild articulatory imprecision (Thompson-Ward, 1998), slow speech rate (Nishio & Niimi, 2001), and mild hypernasality (Thompson & Murdoch, 1995).

Table 3.1 Characteristic Dysarthric Symptoms

Spastic: harsh and strained voice quality, reduced/increased stress and prosody, slow speech rate

UUMN: mild hypernasality, slow speech rate, mild articulatory imprecision

Ataxic: exaggerated respiratory movements, harsh voice quality, +/- variability in pitch and loudness, excess and equal stress, long syllables and phonemes, slow speech rate, imprecise consonants and vowels, irregular articulatory breakdown

Hypokinetic:

- *PD*: harsh and breathy voice quality, reduced intensity, high pitch, reduced pitch range, reduced stress, rapid speech rate
- *PSP*: hoarse voice quality, reduced intensity, low pitch, reduced pitch range, hypernasality, slow speech rate, imprecise articulation, palilalia
- *Shy-Drager syndrome*: reduced pitch range, disrupted phonation, slow speech rate, imprecise consonants and vowels
- *Wilson's disease*: reduced pitch variation, reduced stress, reduced intensity variation, and imprecise consonants

Hyperkinetic:

- *Chorea*: harsh and strangled voice quality, variable pitch, mild hypernasality, variable speech rate, imprecise consonants and vowels, irregular articulatory breakdown
- *Dystonia*: audible inspiration, voice stoppages, hypernasality, slow speech rate, imprecise consonants and vowels
- *Myoclonus*: voice fluctuations and arrests, hypernasality, slow speech rate, sporadically imprecise consonants and vowels
- *Essential tremor*: intermittent breathiness, quavering voice quality, intermittent voice arrests, reduced pitch and loudness variability, variable intensity, slow speech rate
- *Tourette's syndrome*: involuntary vocalizations, coprolalia, echolalia, palilalia, and variable speech rate

3.3 Apraxia of Speech

Apraxia of speech (AOS) is fundamentally, qualitatively different from dysarthria, despite also being a speech motor control deficit; however, the differences between the two can be difficult to describe precisely, in part because descriptions of dysarthria tend to focus on individual articulators and acoustic correlates rather than patterns of movement. There is considerable disagreement about the nature of apraxia of speech, and to some extent, even about its existence. AOS is not simply limb apraxia transposed onto the speech articulators, because there is a distinct disorder specifically affecting non-speech movements, known as non-verbal oral apraxia. Limb apraxia and AOS often co-occur clinically, but there have been cases of each independent of the other (Wertz et al., 1998).

In general, apraxia of speech is thought to be a "higher level" speech motor disorder than dysarthria, though researchers have had difficulty setting out criteria that would distinguish the two. Indeed, as Croot (2002) has pointed out, all of the symptoms

that have been reported in AOS have been reported in dysarthria as well. Proposed distinctive criteria of AOS vs. dysarthria include: ability to execute volitional vs. automatic speech (Wertz et al., 1998); impairment of motor speech programming vs. execution (Darley et al., 1975); disruption of speech vs. non-speech movement (Square et al., 1997); variability vs. consistency of speech symptoms (Wertz et al., 1998); symptoms that are progressive vs. stationary (Kent et al., 2001); and disruptions at a phonological vs. phonetic level (Wertz et al., 1998).

Briefly put, the typical, albeit not necessarily distinctive, speech characteristics of AOS include speech initiation problems (Croot, 2002; Wertz et al., 1998), extended aprosodia (McNeil et al., 1997; Wertz et al., 1998), equal stress and reduced intensity (Darley et al., 1975), slow speech rate (Darley et al., 1975; McNeil et al., 1997; Ziegler, 2002b), inco-ordination of multiple articulators (Duffy, 1995), and imprecise articulation (Darley et al., 1975).

Anatomy and Symptoms

Clinically, AOS (or for that matter any form of apraxia) is almost always the result of damage to the left cerebral hemisphere. While neurological lesions rarely affect only a single functionally-defined brain area, Dronkers (1996) reported that the one pathology shared by all cases of AOS reported thus far is damage to the left insular cortex, which is consistent with the finding that the left insula is active during speech articulation (Wise et al., 1999). This finding should be interpreted with caution, however, because it merely identifies the area that was common across multiple cases of AOS and not all areas that could possibly be related to AOS. While AOS can occur in isolation, it is often accompanied by other symptoms correlated with anterior left hemisphere damage, such as aphasia, dysarthria, limb apraxia, right hemiparesis, hypertonia, hyper-reflexia, and somatosensory deficits. Several of these symptoms (particularly the first two) can make AOS more difficult to identify.

Duffy (1995) stated that there are "important conceptual similarities and differences" between AOS and limb apraxia, although he did not elaborate on them. Apart from any structural differences between AOS and dysarthria, AOS is distinct from dysarthria in that there is a related clinical phenomenon that goes by the same name, which is exhibited in another part of the body (i.e. limb apraxia). However, it is difficult to know exactly how to compare limb apraxia and AOS, beyond the difference in affected body parts. Non-linguistic limb movements do not have the structural or sequential demands of linguistic orofacial movements; and non-linguistic orofacial

movements do not have the possible range or combinatorial possibilities of any upper limb movements. As it is typically defined, limb apraxia refers to an inability to produce or copy meaningful gestures, i.e. to match an object or an action to a movement. But what is supposedly retained in AOS is the semantic content of the speech being produced; when this is lost, the patient is not only apraxic but aphasic. So in the case of limb apraxia, there appears to be an impaired semantic function, which in contrast remains intact in apraxia of speech. As a result, the position of AOS between dysarthria and aphasia remains unclear.

Volitional vs. automatic speech

Wertz et al. (1998) defined AOS as "a neurogenic phonologic disorder resulting from sensorimotor impairment of the capacity to select, program, and/or execute in coordinated and normally timed sequences, the positioning of the speech musculature for the volitional production of speech sounds." The idea of a breakdown in volitional but not automatic speech in AOS is appealing, because it is consistent with the pattern of movement breakdown often reported in limb apraxia (Ziegler, 2002a). However, it is not clear precisely what in this definition differentiates AOS from dysarthria. All of the aforementioned forms of dysarthria are neurogenic and sensorimotor; many of them directly impact co-ordination, programming, and sequencing of speech movements, as well as positioning of the speech musculature. As for the differential impairment of volitional as opposed to automatic speech movements, this criterion is scarcely ever mentioned in the context of dysarthria research, so it is difficult to know whether or not dysarthric subjects are impaired on it. Additionally, the claim bears an unclear relationship to the early finding by Darley et al. (1969a) which suggested that AOS subjects were differentially impaired on imitation tasks as opposed to spontaneous production. Depending on the nature of the experimental task, a spontaneous production could be either more automatic or volitional/rehearsed.

Motor speech programming vs. execution

Code (1998) and Wertz et al. (1998) also reiterated the idea proposed by Darley et al. (1969a) that AOS reflects a disruption in speech motor programming, while dysarthria reflects a disruption to execution of speech movements. This theory rests on the observation that individuals with AOS are often unimpaired on low-level measures of muscular ability. However, as previously stated, the nature of a motor speech disorder often bears little relationship to the strength and tone of individual muscles in

the speech production mechanism. The fact that subjects with dysarthria exhibit low level deficits in muscle tone or strength does not by necessity imply that those deficits fully explain their dysarthria. If it did, one would not expect dysarthric subjects to show the types of differential performance on speech compared to non-speech tasks that have been reported (Ziegler, 2002b).

Disruption of speech vs. non-speech movements

Duffy (1995) suggested that one of the distinctive characteristics of AOS is that it does not show symptoms in oral non-speech movements, or a loss of strength, tone, or range of movement in speech musculature. However, Netsell (1975) referred to cases of Parkinsonian dysarthria impacting speech but not non-speech oral movements. Similarly, Ziegler (2002b) reported dysarthric subjects who showed differential performance on linguistic and non-linguistic tasks. Irrespective of these findings, though, a discrepancy between speech and non-speech movements could simply reflect the speed or complexity of the required movements, rather than a qualitative difference in the disorder. Setting disruption to speech but not non-speech movements as a defining quality of AOS is also problematic because it rules out the possibility of non-verbal oral apraxia. It is widely acknowledged that AOS and non-verbal oral apraxia can co-occur, but by this criterion such a case would automatically become defined as dysarthria.

Variability vs. consistency of speech symptoms

Darley et al. (1969a) claimed that dysarthric speech symptoms tend to be present consistently, rather than appearing on some productions but not others; while apraxic speech symptoms present almost arbitrarily on particular productions of a word or syllable but not other productions of the same word or syllable. However, many features of dysarthric speech (in particular of ataxic or hypokinetic speech) do not seem to present consistently, either in perceptual or acoustic data (Ho et al., 1998; Kempler & Van Lancker, 2002). Given the degree of inconsistency in those findings, it is difficult to say that another motor speech disorder is inconsistent in comparison.

Disruptions at a phonological vs. phonetic level

Wertz et al. (1998) claimed that apraxic errors are errors of substitution, while dysarthric errors are errors of distortion. In other words, apraxic errors are phonemic and dysarthric errors are phonetic. First of all, this was contradicted by the acoustic data

from the same study, which revealed distortion errors not shown by the perceptual measures. But more critically, the question is complicated by the fact that apraxia is frequently accompanied by aphasia (Flynn et al., 1999; Nielsen, 1962). As outlined previously, if apraxic errors are phonemic, then what is the difference between it and mild forms of Broca's aphasia?

Symptoms that are progressive vs. stationary

In their review on the relationship between anatomical and behavioural findings on speech movement disorders, Kent et al. (2001) grouped AOS together with progressive dysarthria. On the one hand, this conflates the nature of the movement disorder with the course of the condition. But beyond that, it contradicts earlier findings that AOS is more likely to be transitory and improve with time (Duffy, 1995).

Speculations about anatomy

Because apraxia of speech ostensibly represents a high-level motor speech disorder, researchers have primarily explored questions surrounding the function of parts of the brain associated with AOS, rather than describing the relationship between anatomy and function in terms of pyramidal or extrapyramidal structures, and activation or suppression of components of the motor system. Darley, Aronson and Brown (1975) put forward the idea that AOS results from a problem with the motor speech programmer. Their view is that the motor speech programmer is influenced by input from "sensory feedback, the basal ganglia and cerebellum, the reticular formation and thalamus, the limbic system, and the right hemisphere." Additionally, Duffy (1995) proposed that the motor speech programmer is in the left cerebral hemisphere.

These analyses are problematic for a few reasons. First, as previously implied, the absence of peripheral weakness seems to encourage researchers to look for explanations of AOS outside the bounds of established models of motor control. Although it is possible that the source of AOS does lie outside those models, it would be preferable to search for explanations within that framework first, rather than proposing entirely new functions and a hypothetical anatomical structure, which interacts with nerves, muscles, and the corticospinal tract only very tangentially. Looking beyond the theoretical approach to some details of its logical implications, Duffy's (1995) proposal that the motor speech programmer is in the left cerebral hemisphere is so broad that not much is gained by stating it. All the evidence to date suggests that the left hemisphere plays a greater role in both speech and language than

the right hemisphere; besides which, the left hemisphere as an entirety is not a functionally-meaningful anatomical unit. Finally, on an intermediate level between anatomy and theoretical framework, it is not clear how the motor speech programmer proposed by Darley et al. (1975) relates to either anatomical or theoretical structures, because it is described alternately in terms of one or the other. Consequently, it is unclear what lies within the bounds of the speech programmer and what is structurally or functionally external to it. For instance, are the basal ganglia a part of the motor speech programmer, or do they simply project to it, and what would be the implications of one versus the other? From the opposite perspective, how can possible anatomical correlates of the motor speech programmer be hypothesized, when the structures providing input to it are described in functional rather than anatomical terms?

3.4 Summary

This chapter has explored the varieties of dysarthric speech in order to allow for speculation about similarities and differences between them and possibly analogous forms of sign dysarthria. Up until now, motor control disorders of speech and of movement more broadly have formed two more or less distinct areas of research, and attempts to combine the two will require the basic assumptions of both to be re-examined and perhaps re-framed.

According to traditional definitions, apraxia was a disorder of the hands, and aphasia and dysarthria were disorders of the mouth and vocal tract. While aphasia is no longer thought to pertain exclusively to the mouth/vocal tract, nor apraxia exclusively to the hands, dysarthria is still defined primarily in terms of specific articulators, despite being "a neurological symptom." If the other two disorders can be re-framed in terms of function or behaviour, then it should be possible, and in all likelihood more accurate and informative, to re-frame current models of dysarthria as well.

It is clear that there are significant physical differences in disruptions to motor control for speech and disruptions to motor control for sign. As outlined in the previous chapter, the two language production modalities differ in important ways at every level of the production mechanism, from the projections of the cerebral cortex to the fibres of the relevant muscles. As a result, there will likely be entire categories of dysarthria that can exist in one modality but not the other. However, it is necessary to delineate the inherent differences in the two modalities and not be misled into focusing on differences that are mere artefacts of a theoretical framework or data coding scheme.

Finally, it is hoped that looking at dysarthria in a new modality will yield new insight into the nature of dysarthria in the modality in which it was originally described, and help to integrate speech motor control research into motor control research more broadly. A productive beginning for this would be the establishment of measures that are articulator-independent, and consistent within a particular measurement scheme (rather than, for example, describing a behavioural phenomenon in anatomical terms). For instance, researchers could analyse patterns of voluntary movements, reflexes, and tone in terms of whether or not they are particular to spindulated muscles rather than being particular to the tongue or larynx (Luschei et al., 1999). Similarly, speech movements can be described in terms of their timing and co-ordination demands instead of their particular articulators (Ho et al., 1998). In this way, the implications of research findings would have much larger significance. As a beginning toward that goal, the following chapter will outline the considerations in developing a methodology to investigate sign dysarthria and describe the methodology that was used for this study.

4 Methodology

4.1 Background and Research Issues

One of the most difficult but fundamental tasks in linguistics as well as behavioural science generally is to collect and record informative, representative behavioural data from human subjects. Unlike data examined in the physical and biological sciences, human behavioural data take no long-term physical form, leave no physical trace, and are subject to variation caused by an abundance of uncontrollable factors (physical, social, psychological, or other). Despite the difficulty of the task, methodologies have been devised by linguists to extract as much information as possible about the elusive data produced by humans as language. Above and beyond the difficulties of capturing linguistic data, however, the sign language researcher faces the additional challenge of capturing, interpreting and analysing data in a medium that is under-represented in linguistics. Because of the particular difficulties inherent to research on the physical structure of signed language, the first section of this chapter will discuss methodologies previously used in linguistics, motor control, speech motor control, and sign language research, and their applicability to the research questions posed here. The remainder of the chapter will then describe the methodologies used for this study (including the research subjects, procedures, tasks, measures, and coding schemes) and the rationale behind their use.

4.1.1 Sign language structure and data capture

4.1.1.1 Sign vs. speech

While linguists have well-established techniques for recording and transcribing the data relevant to them, many of these techniques cannot be easily applied to a language that uses no sound and has no commonly-used written form. In signed language research, no equivalent has been developed to parallel representations of the physical form of language (such as spectrographic analysis), written transcription systems (such as international phonetic alphabet), or establishment of relevant units of measure (such as fundamental frequency), which are standardized in spoken language research. Thus, describing the physical form of sign language is a particular difficulty, because there is no commonly agreed upon representation of the physical signal, transcription system, or set of phonetic measures for sign languages.

As outlined in the previous chapter, the physical structure of signed language is strikingly different from that of spoken language. Unlike speech, which uses an auditory/vocal production medium, signed language uses a visual/manual medium, with

the hands and arms as its primary articulators. Because the articulators are much larger, sign uses a large articulatory space relative to speech, which means that sign data must be captured from a broad area of space. Because there are many sign articulators that can act independently of each other, data capture must allow for multiple streams of information to be recorded at once. Sign uses a visual medium, which means that the capture system must be able to capture data that are four-dimensional, with dissociable x, y, and z co-ordinates measured over time. The one parameter along which sign is easier to capture than speech is speed: because signs are produced more slowly than spoken words, the necessary rate of data capture is slightly slower (Klima & Bellugi, 1979).

Given the physical differences between sign and speech, it is likely that the most suitable measures as well as the most suitable data capture techniques will be different for the two modalities. Moreover, the measures for sign are going to be new and often not easily comparable to measures for speech. It is far too easy to fall into the trap of looking for speech analogues in sign language production, which, particularly at the level of articulation, may simply not be there. The fact that researchers have identified the phenomenon of fundamental frequency in spoken language is no reason to search for something in signed language upon which to affix the same label.

4.1.1.2 Data capture techniques

Given the physical structure of signed language, it is worth considering the different methodologies for recording and measuring the movements of the hands and arms, some of which are preferred by motor control researchers, others by sign language researchers, and a few by both. (A short list of the advantages and disadvantages of various movement capture techniques is presented in Table 4.1.) While some motor control research has focused on speech articulation, none has yet examined signed language purely as a movement task, independent of linguistic function. Up to this point, motor control researchers' only interest in hand and upper limb movements has been in non-linguistic contexts, such as pointing or grasping, which are much simpler than signing. Consequently, data capture techniques used by motor control researchers to examine limb movements are well adapted for the sign language medium but not the signal itself, while capture techniques used in phonetics are well adapted to rapid, precise articulation but cannot be applied to sign movements.

Capture techniques used by sign language researchers are more geared towards preserving the linguistic content of the data, irrespective of the technique's suitability to

the signal or the medium. Videotaping is the most widely used capture technique in sign language research (Atkinson et al., 2002; Hickok et al., 1995; Hickok et al., 1999); though it is not widely used in motor control research, outside of research on apraxia (Halsband et al., 2001; Sunderland & Sluman, 2000). The temporal and spatial resolution of videotape are greatly limited (25-30 Hz frame rate, and variable spatial resolution, depending in large part on the researcher), particularly given the speed and precision with which signs and fingerspelled words are produced. Nonetheless, videotaping has its advantages as a capture technique. It is only minimally intrusive relative to other techniques, can be used anywhere (an important consideration for clinical subject groups), and the collected data are easily stored and retrieved. Additionally, the simple fact that videotaping is not spatiotemporally precise means it allows movements to be recorded and described more broadly than techniques that can capture more detail.

Unlike video, kinematic systems record data from markers placed on the body, using basic principles of physics, coupled with rapid processors. Optoelectronic systems use a set of cameras in conjunction with a set of light sources to track movement; for example, the Optotrak™ system (Northern Digital Inc.) uses a set of diodes that emit infra-red light. The diodes are placed on subjects' bodies (usually the hands) and a set of two or more cameras are able to record emissions of light from the diodes and compute the 3D co-ordinates of their locations over time. Optoelectronic systems can be very spatiotemporally precise (450-750 Hz, depending on software; 0.1- 0.15 mm) but because they are optical, they cannot record data when anything opaque comes between a diode and the cameras. This includes instances where the hand changes orientation so that part of the hand itself comes between the diode and the cameras. Another type of optoelectronic system (Vicon™, www.vicon.com), rather than using diodes, uses markers placed on the body that reflect infra-red light emitted by a strobe around the camera lens. The reflected light is received by the electronic cameras, and the locations of the markers are tracked over time. This type of system has lower temporal resolution (60 Hz), though it does have a means of extrapolating a trajectory from one point in space to another, which makes occlusions less problematic.

Optoelectronic systems with diodes are used by speech (Ackermann et al., 1997; Ostry et al., 1996), motor control (Gentilucci & Negrotti, 1999; Lang & Bastian, 2002), and sign language researchers (Brentari et al., 1995; Poizner et al., 1987). Strobed systems are used by fewer researchers on the whole, but the researchers are from both sign language (Cormier, 2002) and motor control research (Krystowiak et al., 2003;

Park et al., 2003). Notably, within motor control research, strobed systems are more often used to measure gait or posture: i.e., movements that are less fine-grained than hand and arm movements (Defebvre et al., 1996). However, optoelectronic systems in general are spatiotemporally precise enough that it is problematic to use them to compare movements from many independent articulators across multiple signed productions. The number of data points generated by such an experimental design is simply too large. Optoelectronic, and kinematic systems more broadly, are very well-suited to comparing the movements of a few markers (e.g., placed on the thumb, wrist, and elbow) during repetitions of the same action, but not for describing how articulators act as a group across several distinct sets of movements.

Kinematic systems can also employ a large magnet in combination with a group of small magnets: the small magnets are attached on the body, and when they move through space, they disrupt the magnetic field generated by the large magnet and their positions can be tracked. Magnetic systems have an advantage over optoelectronic systems in that data are not occluded when the markers face away from the cameras. Magnetic tracking is less temporally precise than the most precise optoelectronic systems (60-120 Hz) (www.polhemus.com). However, there is still the same computational problem of not being able to compare many independent articulators across many productions, because of the spatiotemporal precision of the measurements. Magnetic capture systems are used by both sign language (Bangham et al., 2000) and motor control researchers (Laffont et al., 2000), as well as by phoneticians and speech motor control researchers (Ellis & Hardcastle, 2002; Honda et al., 2002). Magnetic speech capture systems use a methodology called electromagnetometry (EMA), in which magnetic coils are attached to the tongue and palate, while the head remains in a fixed position. However, with a few exceptions (e.g., Goozee et al., 2000), EMA is not in widespread use among researchers investigating speech dysarthria.

Table 4.1: Considerations for different movement capture techniques

Capture Technique	Advantages	Disadvantages	Research Areas
Videotape	can capture multiple streams of data, allows comparison of varied datasets, transportable	spatially and temporally imprecise	sign language, apraxia
Optoelectronic			
LEDs	spatiotemporally precise	data occlusions, applicable only to small set of movements, not transportable	sign language, motor control, speech motor control
Strobe reflectors	spatially precise	minor data occlusions, applicable only to small set of movements, not transportable	sign language, motor control (limited)
Magnetic	spatially precise	applicable only to small set of movements, not transportable	sign language, motor control (limited), speech (limited)
EMG	precise measure of effector activity, tight relationship with neural function	unclear relationship to global output, not transportable	speech, motor control

Electromyography, or EMG, uses electrodes placed on the skin to record electrical activity from muscle fibres. As such, it captures a fundamentally different type of data from all the capture techniques discussed so far: EMG does not record an approximation of the visual representation of a subject's movements, but rather what the muscles themselves are doing. It is the most precise technique in terms of capturing what the effectors or articulators are doing, but the least informative about the global output of the system. EMG has been used in research on motor control (Erimaki & Christakos, 1999; Meunier et al., 2000) and on speech (Strauss & Klich, 2001; Wohlert & Smith, 2002) but not in sign language research. It will only be useful for sign language research when a sufficiently narrow research question about the sign production mechanism arises.

4.1.2 Sociolinguistic aspects of sign language

Because there are implications for experimental design, the sociolinguistics of sign languages will be discussed briefly. As in any linguistic research, one must take into account the linguistic background of the research subjects; and there are particular factors that come into play with sign languages, not because of their physical or grammatical structure, but because of the circumstances in which they are learned and used. First, a large majority of Deaf signers come from hearing families and as a result

do not learn the language from their parents (Sutton-Spence, 1999); most signers acquire sign language relatively late because they have no exposure to it prior to entering school. Additionally, because educational policy has typically discouraged sign language use, deaf children get varying degrees of exposure to sign language from teachers or deaf peers. As a result of being exposed to the language at a range of ages and in a range of environments, different signers' use of the language is extremely varied (Mayberry & Groschler, 1994; Newport, 1990).

The status of sign languages in the industrialized world has improved significantly in the past few decades, but traditionally the stigmatization of sign language in schools and elsewhere has meant that it was used primarily in informal contexts. Hence, there was little in the way of an elite language dialect, which would be emulated and spread through the linguistic community (Battison, 1978). Furthermore, because sign languages are invariably minority languages, irrespective of whether their use is stigmatized, they have typically had no central institutions like universities or the broadcast media to canonize certain forms of the language. Historically, Deaf communities have been relatively isolated from each other linguistically because the language has no written form, no broadcast media presence, and no medium of communication analogous to the telephone. Consequently, there is a great deal of dialectal variation in many sign languages, including British Sign Language. As a result of these factors, age of acquisition, educational policy, linguistic isolation, and access to communication technology, Deaf signers as a linguistic group are extremely diverse.

It is necessary to design linguistic tasks, as well as testing procedures more generally, with these issues in mind. Because of the extent of dialectal variation, it can be necessary to ask subjects to copy signs rather than do a naming task, for example, to ensure that they produce the intended target. For these, as well as other reasons, it is often more useful to design tasks for individual subjects, rather than use a set of standardized tasks, intended to be universal. While the latter approach has the benefit of facilitating statistical comparisons across subjects, it also assumes the subjects are alike except on the measures on which they are being compared, which is often not a valid assumption. Apart from the question of language modality, standardized tests are invariably written in a majority language and cannot always be translated effectively. This is an issue that has been examined in many cultural contexts, but has been insufficiently explored in the case of sign languages. An example of a difficult translation issue particular to sign languages is the problem of how to administer an

apraxia test when a direct translation of the test into sign language can give clues to the correct response.

4.1.3 Atypical sign language users

The final methodological issue for this type of research is the collection and analysis of linguistic data from atypical subjects, in this case, subjects with movement disorders, and in some cases linguistic or other cognitive disorders as well. Particular demands of data collection with atypical users of any language include discriminating movement problems from other problems (e.g. impaired visual processing, cognitive slowing), designing tasks that are reasonable measures of the subject's capabilities, collecting data efficiently in subjects who are apt to tire easily, and picking up important variations from normal production without over-emphasizing variations that are not important.

In addition to these considerations, however, being both an atypical subject and a Deaf signer introduces new complications, the most significant being that sample sizes are inevitably small. Unlike research on hearing people with Parkinson's disease, for example, a study on Deaf subjects with any impairment cannot compare homogeneous subjects on the same individual measure and yield any substantial body of data. Because it is difficult to find many subjects who are both Deaf and also have a cognitive or neurological disorder, studies have to be designed to get the most useful information from a small number of subjects. Finally, a related complication is that there are limited normative data for British Sign Language, which means there is the risk of overinterpreting slight variations in data from atypical signers, simply because those data are examined more carefully than the behaviour of unimpaired signers.

As this is not the first study to investigate atypical signers, it is worthwhile to delineate the topics and methodologies explored by others. As discussed above, virtually all studies of atypical signers have small sample sizes; it is the truly exceptional study that has more than twenty subjects (Atkinson et al., 2002; Emmorey et al., 1996; Hickok et al., 1996). A lot of research on atypical signers takes the form of case studies, which can be very informative if done well. In depth studies of particularly interesting or unique subjects can yield insight that studies with large numbers of "average" subjects cannot (Kegl & Poizner, 1997), because they can reveal precisely what it is that distinguishes the average from the atypical.

One area of research on atypical signers has looked at subjects who are not necessarily deaf, but who have some type of unusual language ability or disability, such

as Landau-Kleffner syndrome (Kegl et al., 1996; Sieratzki et al., 2001), Down syndrome (Woll & Grove, 1996), or linguistic savantism (Morgan et al., 2002), to see how those subjects perform on signed tasks in comparison to spoken or written tasks. These studies were all designed to investigate the effects of language modality on language learning or use. Because of the research question, these studies were able to follow the longstanding tradition in experimental psychology of comparing the same measures on the same tasks across different conditions. The condition in this case is the language modality being tested.

The bulk of research on atypical signers has examined stroke and its effects on signers' linguistic competence (Atkinson et al., 2002). In particular, many studies on sign language and aphasia have focused on distinguishing sign language from gesture (Corina et al., 1992a; Poizner et al., 1987) and from visuo-spatial ability (Emmorey et al., 1996; Hickok et al., 1996; Hickok et al., 1999; Loew et al., 1997), by describing cases of acquired neurological damage that impacted one function independent of the others, thereby documenting that sign language is dissociable from other cognitive functions. This line of research has been put forward as proof that sign language is linguistic rather than gestural in nature, on the assumption that the two are mutually exclusive categories. Unlike studies focusing on typical signers' ability in signed, spoken, or written language, studies on linguistic competence following stroke or injury have usually compared signers' ability on linguistic and non-linguistic tasks. Because the focus in these studies was on showing that sign language ability was independent of other cognitive abilities, the researchers in effect created methodological complications for their studies, since there is no way of directly comparing linguistic and non-linguistic cognition.

Additional research has contrasted the effects of aphasia and movement deficits (other than apraxia) on sign language production (Brentari et al., 1995; Loew et al., 1995; Poizner & Kegl, 1992, 1993). Although these studies examined movement deficits, for the most part, the primary interest was in illustrating deficits in sign production that were fundamentally non-linguistic in nature and contrasting them with aphasia (Poizner & Kegl, 1993). Nonetheless, the shift in emphasis toward movement impacts the research design in a few important ways. First, the measures in these studies tended to be more paralinguistic than linguistic in nature: researchers described non-contrastive variation in handshapes, and quantified pause length (Loew et al., 1995), relative movement onsets of multiple articulators (Tyrone et al., 1999), and ratios of handshape change to limb movement (Brentari et al., 1995). Unlike research comparing

atypical signers' performance on linguistic and non-linguistic tasks, studies on aphasia and movement deficits compared the nature of subjects' performance on the same tasks: usually spontaneous signing. Additionally, these studies used fewer standardized tests of linguistic or cognitive ability, since such measures would miss the behavioural distinctions being described, and standardized tests to detect those distinctions have yet to be developed. Coming back to the earlier point about Deaf signers as an inherently small and diverse subject population, it is not clear how appropriate or useful standardized behavioural measures are for Deaf subjects, particularly when the focus of the research is on an atypical subset of that group. To date, there is too little indication that behavioural measures routinely used for hearing subjects are informative when applied to healthy Deaf signers, much less when they are applied to Deaf signers with neural damage.

To summarize, for the most part, researchers investigating atypical signers have used linguistic tasks (often translated from spoken language) in combination with less rigidly structured conversation or narration tasks to elicit productions, and recorded those productions on videotape for later analysis. Studies contrasting language and movement or language and other cognitive ability have made little direct comparison of scores on standardized cognitive or movement tests, but have instead used more general descriptions of subjects' patterns of performance. Perhaps by necessity, comparisons across tasks and across subjects in these studies tend to be made in broad terms.

4.2 Development of methodology for this study

In addition to the constraints on methodology imposed by doing research on atypical users of a language that is expressed via an under-researched modality, at a more basic level, the methodology has to be appropriate to the research question. The current study differs from most research on atypical signers in that it places greater emphasis on movement itself, and the relationship between disruptions to movement and disruptions to sign production. The purpose of this study is not to show that sign language production and human movement are two separate systems, but rather to examine the effects of movement disorders on sign language production, in light of the effects of those disorders on speech, and in light of the differences in physical structure between speech and sign. Because signed language is rapid, complex and systematized, and because it uses the hands and arms as articulators, it is expected that its relationship to human motor control will be both similar to and different from spoken language's

relationship to human motor control. The question is: how are the relationships similar, and how are they different?

Because the design for this study was created more or less *de novo*, there were several points that had to be considered critically. These include: the breadth and nature of the research question; the schema for the categorization and classification of the data; the physical structure of the data; the type of data likely to be most informative; possible measures of sign data as compared to speech data; and possible measures of sign data as compared to movement data. Given that the study is looking at sign language as movement, it is necessary to compare data collection and analysis methods from phonetics, speech and language therapy, sign linguistics, and motor control research to assess which methods are best applied to the research question and the data.

This is the first study to look at a group of subjects with a variety of movement disorders and try to both describe the broader phenomenon of disruption to sign articulation, as well as capture variations within that phenomenon. As such, the research question seemed better suited to a research methodology and capture technique that are less precise but offer greater generality. The goal was to begin to identify the basic parameters along which sign articulation can be disrupted, so that future studies can pose more sharply delimited research questions about the physical details of those parameters.

While there are sign language capture techniques that provide more spatial and temporal detail than videotape, it was decided that videotape would be preferable for two reasons. First, because this study was intended to describe a range of disruptions to sign articulation, it was necessary to analyse multiple physical aspects of signing at once to determine where and how impairments might manifest themselves. In a sense, the data from kinematic or electromyographic studies are too specific to be useful in addressing this research question. As this is an exploratory study and the first of its kind, it is the necessary precursor to a kinematic study on a related topic. Second, on a smaller but still crucial point, the lack of a body of normative data on the kinematics or physiology of signing would render those types of data on impaired signing relatively uninformative.

With respect to the non-linguistic data, videotaping is by no means the most effective way to capture the physical parameters of relatively simple non-sign movements. However, since linguistic data were collected using videotape, it was decided to use the same data collection procedure for non-linguistic data. Using two different methodologies for comparing measures (e.g. co-ordination of the two arms)

across linguistic and non-linguistic tasks could have easily lead to differences in findings that were purely artefactual to the data collection procedure. Additionally, some of the arguments for using video rather than kinematics or EMG (most notably the inclusion of more subjects) applied to non-linguistic testing as well as linguistic testing.

To restate the research questions outlined in Chapter 1, this study seeks first and foremost to establish a broad definition of sign dysarthria on the basis of observation of sign language users with acquired movement disorders. In so doing, it is necessary to delineate the differences between sign dysarthria and speech dysarthria. No hearing subjects with dysarthria were examined as part of this project, so comparisons between dysarthria in the two modalities will be made on the basis of existing literature. In addition, it is necessary to delineate the differences between sign dysarthria and simpler limb movements, and the differences between sign dysarthria and manual apraxia; these two questions are explicitly addressed in the analyses of the data presented here.

4.3 Subjects

Research subjects will be described individually and in greater detail in later chapters. They are mentioned briefly here to outline their distinctive characteristics and what their inclusion adds to the research. It should be emphasized, first of all, that the subjects are not intended to represent a uniform group, but rather to highlight the differences in various possible forms of sign dysarthria. They are also not intended to be representative of all Deaf signers with acquired movement disorders. Subjects were selected to illustrate a range of disorders, but there is insufficient knowledge of the incidence of acquired neurological impairments among the Deaf to say how this series of case studies relates to it.

Subjects were recruited mostly via the Deaf Stroke Project at City University, which ran concurrently with this study, and both studies obtained the relevant approval from the supervising university ethics panel. Additionally, advertisements for this study in particular were placed in Deaf media outlets (e.g. magazines, websites); and letters were sent to social workers, Deaf clubs, and clinicians in neurology and speech and language therapy. According to medical records obtained from subjects' physicians, all of the subjects had acquired disorders, and none had a developmental neurological disorder. If it was apparent that subjects in the Deaf Stroke Project had a movement disorder, they would typically be referred to this study and considered for inclusion in it. The presence of a movement disorder and the absence of a developmental neurological disorder were the primary criteria for inclusion in this study. Beyond those

criteria, subjects were considered for the contrast that they would show with other signers in the study and with hearing dysarthric subjects who had the same movement disorders, as described in the literature.

4.3.1 Atypical subjects

James is a 79 year old right-handed man who suffered a right hemisphere stroke in the territory of the middle cerebral artery, causing damage to the parietal lobe, primarily subcortically. Scan data revealed that he had had earlier minor cerebrovascular accidents (CVAs), which damaged the left thalamus and the right corona radiata. James's linguistic deficits were solely pragmatic in nature: he had difficulty with conversational turn-taking, staying on topic in conversation, and judging humorous dialogue. His motor deficits included left hemiparesis, affecting the hand most severely. James's case is interesting because of the contrast he showed with the other subjects: his signing was not severely impaired, despite his hemiparesis. Although he could not form handshapes on his left hand and could not move his left arm as well as his intact arm, his signing was otherwise fairly normal. James was included in the study to provide an example of a signer with mild right hemisphere damage and unilateral upper motor neuron dysarthria.

Robert is a 36 year old right-handed man who experienced severe cerebellar damage as the result of both an arteriovenous malformation, and then haemorrhaging subsequent to an operation to correct the malformation. Damage was distributed across the right lateral hemisphere, the spinocerebellum, and a small part of the pons. Robert's grammar and lexicon remained essentially intact: during testing, he was able to respond appropriately to questions and had no difficulty with naming, though his movement disorder made sign articulation difficult for him. His motor deficits included nystagmus, inability to walk, unstable posture, and limb movements typical of ataxia: large, jerky movements, intention tremor, and disruptions to spatial accuracy in voluntary movements. Robert is included in the study to provide an example of ataxic dysarthria in a signer.

Joseph is a 79 year old right-handed man with progressive supranuclear palsy. His language ability was basically intact, except for a mild comprehension deficit. His movement deficits included ophthalmoplegia (loss of eye movement), slow/small limb movements, and a lack of movement generally. He was originally thought to have had a CVA, but his medical record was unclear on this point. With repeated visits by members of the Deaf Stroke Project, it became apparent that irrespective of whether he

had had a CVA, he clearly had a progressive condition. Additionally, his family reported that he had balance problems before he was thought to have had a stroke. Initially, because of his bradykinesia and hypokinesia, it seemed that he may have Parkinson's disease. However, on closer study, it was noted that his eye movements were severely limited and that he exhibited no resting tremor. A consultant neurologist then confirmed the diagnosis of progressive supranuclear palsy. Joseph is included in the study to provide an example of severe hypokinetic dysarthria in a signer, albeit not resulting from Parkinson's disease.

John is a 54 year old right-handed man with Parkinson's disease. His language was very nearly normal, though his production was a bit slow. His primary motor symptoms included resting tremor, and slow and small movements. Additionally, he showed mild cognitive impairment, scoring just below normal on the Folstein Mini-Mental exam (Folstein et al., 1975). Possibly as a result of this, he occasionally had to ask for signs to be repeated. On a related note, he showed slow responses as well as just slow movement, though this was not tested in depth. John is included in the study because he had a milder case of hypokinetic dysarthria, and also because his condition was still sufficiently well-controlled with medication for his symptoms on- and off-medication to be compared.

Maureen is a 72 year old right-handed woman who suffered a left hemisphere CVA, in the territory of the middle cerebral artery, affecting anterior regions of the left cortex. Her spontaneous language was minimal to non-existent, and her naming and copying were severely impaired, as was her language perception. Her motor symptoms included right hemiparesis and severe apraxia. As a result of her aphasia, only her non-linguistic movements were analysed in any depth for this study; there simply were not sufficient linguistic data to include for analysis. Maureen is included in the study because her movement and signing disorders were at a noticeably different level from those of the other subjects. Her impairment was arguably as severe as theirs, if qualitatively different impairments can be compared in such terms, but hers was a representational dysfunction as opposed to a problem of movement execution, co-ordination, or timing.

4.3.2 Control subjects

The primary reason for the inclusion of the left hemiparetic and apraxic/aphasic subjects described above was to provide a contrast to the subjects whose signing is severely impacted by dysarthria itself. However, in addition to including these two

signers for comparison, two healthy, right-handed Deaf signers were also recruited to serve as controls for the subjects with movement disorders. Following standard practice in research on atypical signers, and for the sake of timely data collection and analysis, the number of control subjects included in the study was kept small. For the reasons outlined in section 4.1, finding adequately matched subjects is an exceptionally difficult task for this particular group of subjects, varying as considerably as they do in terms of language experience and dialect. Second, with the number of atypical subjects being so small and the data analysis procedures so time-consuming, the value of three additional control subjects was not estimated to be worth the investment.

A relatively young (38 year old) male signer was recruited as a control for the subject with cerebellar damage, since he was the real outlier in terms of age. The second signer was recruited to be a control subject for the remaining four subjects with movement disorders, and her age was approximately the mean of theirs (70 years old). Both control subjects were Deaf signers from hearing families, who became fully integrated into the Deaf community. Neither control subject had any neurological or movement disorder.

4.4 Procedure

Once subjects had been contacted and procedures explained to them briefly in a letter, researchers affiliated with the study visited them at home. All data were collected at subjects' homes or places of residence, over the course of several visits. For subjects' general comfort and in order to make sign productions as natural as possible, a Deaf research assistant administered both the non-linguistic and linguistic tasks. At the beginning of each testing session, procedures were explained to subjects again and in greater detail. On the first visit, once the procedures and broader research goals had been explained, subjects were asked to give their consent to participate, be videotaped, and allow those videotapes to be shown in academic contexts and to smaller groups for intelligibility judgements (each of these was a separate consent option). (Consent forms are included in Appendix A.) Since data were collected over the course of more than one visit, the order of presentation of the tasks was decided according to the subjects' level of energy, with more difficult or time-consuming tasks saved for when subjects were well-rested. Additionally, subjects were frequently asked if they wanted to take breaks, and consistently given breaks once or twice per session. Because testing was done in multiple sessions, and guided by the subject's energy level, the duration of the entire procedure is extremely variable across subjects and across sessions. On average,

an individual testing session lasted approximately 30 minutes, and the entire session would be recorded on videotape for later analysis.

Background testing

No functional or structural brain imaging data were collected as part of this research; however, where possible, imaging data were obtained from subjects' supervising neurologists. In addition to the linguistic and non-linguistic tasks designed for this study, subjects were given a series of background neuropsychological tests to provide information on their general cognitive ability, non-verbal reasoning, and visuo-spatial processing ability. In order to avoid fatiguing the subjects, tests were given according to where they seemed relevant, and/or where it was possible to administer them. If a subject had an impairment that precluded their taking or being assessed accurately by one of the tests, that test was omitted. Tests included: the Pyramids and Palm Trees test of semantic access (Howard & Patterson, 1992), Folstein Mini-Mental State Exam (Folstein et al., 1975), Raven's Progressive Matrices (Raven & Raven, 2003), WAIS-III Block Design (Wechsler, 1997), a line cancellation test, and the Benton line orientation test (Hamsher et al., 1992).

4.5 Tasks/Materials

Tasks were designed to assess the nature of subjects' movement deficit, and in particular to assess the nature of their deficit on linguistic as opposed to non-linguistic movement. The linguistic tasks were fingerspelling, sign copying, and naming. Additionally, the non-linguistic tasks were designed to probe the nature of subjects' movement disorder, e.g. inability to appreciate how to use an object as opposed to the inability to physically manipulate it. The non-linguistic tasks were: pointing, the Kimura box (Kimura, 1977), handshape copying, reaching to and grasping an object, tool use, and copying object manipulation. To address how tasks were related to each other and how they might relate to specific movement deficits, descriptive characteristics of the tasks, derived partially from the definition of articulation given in Chapter 1, were developed. The tasks used for the study were categorized in terms of whether or not they were: formationally complex, sequential, representational, targeted, co-ordinated, and/or interacting with an external object. These characteristics are described in Table 4.2, and the values of the characteristics for each task are listed in Table 4.3.

Table 4.2: Descriptive characteristics of movement tasks

Formationally complex (henceforth, "complex"): the hand has to take on a specific configuration, not completely determined by the form of an external object or target

Representational: including a symbolic or communicative component

Sequential: consisting of two or more movements in sequence

Co-ordinated: requiring simultaneous or overlapping movements of proximal and distal joints

Targeted: requiring a specific physical interaction with something external to the primary effectors, though not necessarily external to the body

Interacting with an external object: a task that by definition is performed on an external object, rather than being on the body or in an undefined location

Although testing procedures were the same across subjects, there was some variation in the tasks themselves, in order to better adapt them to individual subjects. For example, some of the more difficult linguistic tasks were not presented to the severely aphasic/apraxic subject, because her ability to copy gestures or signs, name images, and read written text were all so severely impaired that asking her to do a task that required those skills would not have been particularly informative. In addition, the left hemiparetic subject's linguistic background did not encourage the use of fingerspelling, so it was decided that the fingerspelling task would not be informative for him, since his pre-morbid performance would not have been comparable to the performance of other subjects. Similarly, the movement task requiring the highest level of co-ordination was not presented to the subject with severe ataxia. A list of which tasks were performed by which subjects is given in Table 4.4.

Table 4.3: Values for characteristics of particular tasks

	Complex	Representational	Sequential	Co-ordinated	Targeted	External Object
Finger-spelling	X	X	X	X	X	
Sign copying	X	X		X		
Naming	X	X		X		
Pointing		X			X	X
Kimura box	X		X	X	X	X
Handshape copying	X					
Reach & grasp			X	X	X	X
Tool use	X	X	X	X	X	X
Object manipulation	X		X	X	X	X

Table 4.4: Tasks performed by atypical and control subjects

	Finger-spelling	Signing	Pointing	Kimura box	Handshape copying	Reach & grasp	Tool use	Object manipulation
James		x	x	x				x
Robert	x	x	x	x		x		x
Joseph	x	x	x					
John	x	x	x	x	x			
Maureen			x	x	x		x	
Christine	x	x	x	x	x			
Graham	x	x	x	x		x		

4.5.1 Linguistic Tasks

4.5.1.1 Fingerspelling

Subjects were asked to fingerspell English words presented to them visually. As this was the most motorically difficult of the linguistic tasks, it was presented to subjects when they were feeling most alert and energetic. Stimuli were presented in the form of an illustration with a typed English word underneath it (the full set of fingerspelling stimuli is included in Appendix A). Illustrations were shown to subjects one by one by the Deaf research assistant, and subjects were asked to fingerspell the word in isolation, in order to minimize the effects of linguistic context or coarticulation across productions. The research assistant was available to prompt or to clarify the stimuli as necessary; however, this was necessary only rarely.

Fingerspelling is a system for borrowing words from spoken language by using the hands and fingers to spell the words, letter by letter (Battison, 1978; Sutton-Spence, 1994), and different sign languages use different fingerspelling systems with different formational properties. Fingerspelling is not a simple cipher, because in production, the movements and handshapes for the individual letters are coarticulated (Tyrone et al., 1999; Wilcox, 1992), and the transitions between letters are crucial to language processing (Akamatsu, 1983). In this regard, charts of fingerspelling systems can be misleading, because they often present letters in isolation, and as if they are static forms. With respect to the two-handed fingerspelling system used in BSL, charts can be additionally misleading, because they typically present fingerspelled forms as if the "non-active" hand does not move, when in fact, it often changes both hand configuration and orientation in the course of fluent fingerspelling (Sutton-Spence, 1994). (There is a BSL fingerspelling chart included in Appendix B, which shows the finger movements of the non-dominant as well as the dominant hand.) It is the rapid rate of production of individual letters in combination with the high degree of coarticulation and the co-ordination demands of independent finger and hand movements that make fingerspelling motorically difficult (Tyrone et al., 1999; Poizner et al., 2000).

The fingerspelling task was included in the study because fingerspelling is an important part of sign language use, and it has greater spatiotemporal and motoric demands than signing (Emmorey et al., 2003; Tyrone et al., 1999). According to the movement categorization criteria listed above, fingerspelling is complex, representational, sequential, co-ordinated, targeted, and requires no interaction with an external object. First, it is *complex* because the hands have to take on particular, arbitrary configurations for productions to be well-formed. Fingerspelling is *representational* because the system is used to convey linguistic information; this was true in the task that was used for this study, as well as being true in Deaf signers' normal day-to-day behaviour. It is *sequential* because the words that it represents are conveyed letter by letter. A signer can of course produce individual fingerspelled letters to refer to specific letters rather than words, but that is not its most common use and was not included in the task for this study. The fingerspelling task is *co-ordinated*, because it requires the movement of multiple joints at once or in an overlapping manner. BSL fingerspelling, unlike most other fingerspelling systems, is *targeted* because in all but one letter, the two hands have to make contact in order for a letter to be produced correctly. Finally, unlike some of the non-linguistic movement tasks described below, fingerspelling requires no *interaction with an external object*; the body

itself is all that is used for fingerspelling production in sighted signers. As a side note, movement tasks that require an external object are almost always targeted, but targeted tasks do not necessarily require an external object, because parts of the body can serve as movement targets.

4.5.1.2 Sign Copying

For the sign copying task, subjects were asked to repeat signs produced by the research assistant. The sign copying task was designed to allow for comparison of specific formational properties of signs (such as co-occurring handshape and movement change, or asymmetrical movements of the two hands) that were predicted to be easier or more difficult to produce. The task consisted of commonly-used signs (a Deaf sign language researcher was consulted to verify that none of the target signs was obscure or uncommon) and had an even distribution of one-handed and two-handed signs. The research assistant produced signs individually from a written list and asked subjects to copy them. In this way, there was no need for subjects to use written English materials, and it could be ensured that all subjects produced the same signs. (A video clip of the target signs being produced is included in Appendix A.) As in the case of the fingerspelling task, signs were produced in isolation rather than in a semantically-appropriate sentence or an arbitrary carrier phrase, in order to avoid coarticulatory effects. If a subject had difficulty understanding or repeating a sign, the research assistant would repeat it, but the subject's difficulty would be noted.

The sign copying task was included in the study so that subjects' sign productions could be compared to their fingerspelling and to their non-linguistic movements, while controlling for linguistic context and variation (phonological, semantic, and dialectal). Like fingerspelling, sign copying is *complex* because it requires the signer to produce hand configurations that are arbitrarily specified. It is *representational* because the signs produced in the task are real and have semantic content; although in the context of testing, signs were not being used to convey information. The sign copying task is not *sequential*, because subjects were not required to produce multiple signs in a single production. Sign copying is *co-ordinated*, because it requires proximal and distal joints to move with specific timing relative to each other. Sign copying can be *targeted*, although it is not necessarily so. Whether or not it is targeted depends on where the sign is located in space: if a sign's location is in neutral space, then it is not targeted; if its location is on the body, then it is targeted. Finally, sign copying requires no *interaction with an external object.*

4.5.1.3 Naming

The naming task was the same as the copying task except that it allowed analysis of subjects' sign production from memory rather than from a model of a sign. While a naming task can also serve as a test of lexical retrieval, it was not analysed for that purpose in this study. Subjects were shown an illustration on a card and asked to produce the corresponding sign. As with the other linguistic tasks, subjects were asked to produce the sign in isolation rather than in a sentence. If the subject could not retrieve the target sign or produced another sign instead, the research assistant would assist by giving an additional description of the illustrated referent or by giving a phonological cue, such as the location of the sign (see discussion of sign phonology in section 4.6.1.1). Target signs were selected to be in common use and to represent an even distribution of one- and two-handed signs. The movement characteristics for the naming task are the same as those described above for sign copying. The naming task is *complex*, *representational*, *not sequential*, *co-ordinated, targeted*, and not *interacting with an external object.*

4.5.2 Non-linguistic Tasks

4.5.2.1 Pointing

For the pointing task, subjects were presented with a sheet of A4 paper with two large illustrations and given a signed description that applied to one but not the other and asked to point to the appropriate picture. Subjects could ask for repetition or clarification if necessary; and only accurate responses were included in the analysis. The purpose of the task was to allow measurement of basic parameters of a comparatively simple, non-sequential movement (see description of behavioural measures for non-linguistic tasks in Table 4.7).

The pointing task is not *complex* because there are no explicit requirements for how the hand(s) should be configured. Typically, subjects used an extended index finger or an extended thumb to point, but that is likely to be an effect of ease or habit, since there were no specified task demands. Additionally, hand configuration did not have to vary in a particular way from one trial to another on the task: subjects could use the same hand configuration throughout the task, and often did so. In the context of this task, pointing is *representational*, because subjects were asked to refer to the image that the researcher described, so there was necessarily a symbolic, communicative component to the task. In other contexts, pointing is not necessarily representational.

The pointing task is not *sequential*, because subjects were not asked to point to multiple images in a given trial. It is not *co-ordinated*, because subjects did not necessarily have to move multiple joints to perform the task; in addition, the proximal and distal joints did not have to move in relation to one another in a specific manner for the task to be performed correctly. It is *targeted*, because subjects had to move toward one image rather than another. The pointing task requires *interaction with an external object*: the movement targets were external to the body.

4.5.2.2 Kimura box

The Kimura box is a well-established motor control task, used to test subjects with apraxia (Kimura, 1977, 1993). The device itself is a wooden box with a column of three distinct manipulanda, or components for subjects to handle (Figure 4.1). In studies of apraxia, subjects are typically asked to imitate a series of three actions on the manipulanda, as produced by the researcher. Each action requires a different handshape, and subjects are typically scored on their ability to adopt the correct handshape for each one. For this study, the task was included in order to assess handshape selection and formation as well as to judge other parameters of movement, such as targeting, speed, and co-ordination, on a complex, sequential, non-linguistic task. The Kimura box task is *complex*, because subjects have to change hand configuration and those configurations are not pre-determined by the shape of the manipulanda. The task is not *representational*, because the movements have no meaningful component. It is *sequential*, because there are three required movements on each trial. The task is *co-ordinated*, because subjects simultaneously move toward the manipulanda while re-configuring their hands, thereby necessitating the movement of proximal and distal joints relative to each other in a specific order. It is *targeted*, because the movements are directed toward specific manipulanda. Finally, the task requires *interaction with an external object*, because the movements are performed on the box.

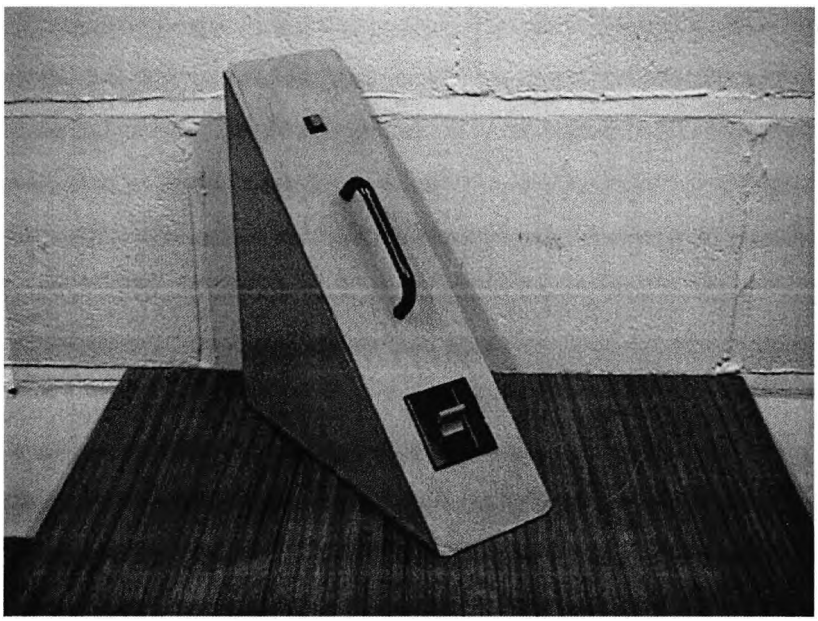

Figure 4.1: Kimura Box (Kimura, 1977, 1993)

4.5.2.3 Handshape copying

For the handshape copying task, subjects were shown drawings of hand configurations, all of which were possible handshapes in BSL, and asked to copy them. Altogether there were 15 pictures, which the research assistant presented to subjects one by one. The primary purpose of the task was to compare production of complex hand configurations in linguistic and non-linguistic contexts. Because no movement was required other than the formation of the handshape, and there was no external target that subjects had to move towards, movements were not coded for targeting or co-ordination (see descriptions of these measures in Table 4.7). The handshape copying task is *complex*, because the required hand configurations are arbitrary but specified. However, it is not *representational*, because even though the hand configurations are possible BSL handshapes, their production in isolation conveys no meaning. The handshape copying task is not *sequential*: subjects produced only one hand configuration in response to each of the stimuli. It is not *co-ordinated*, because subjects can produce all the hand configurations in the same place, thereby avoiding the need to move proximal and distal joints in a co-ordinated way. The task is not *targeted* and requires no *interaction with an external object*: subjects produced the movements at an unspecified location in the space in front of them.

4.5.2.4 Reach & grasp

Subjects were asked to reach to an object placed in front of them, grasp it, move it a short distance forward (approximately 25cm) and set it down. The reach and grasp task was included to allow assessment of movement parameters on a non-linguistic task with moderate co-ordination and targeting demands, but minimal sequencing demands. The task objects were a set of cylinders of different weights and sizes; the cylinders' dimensions are listed in Table 4.5. Reach and grasp is frequently used as a task in motor control research (Gentilucci & Negrotti, 1999); however, the implementation of it was slightly different in this study. Because data were captured on videotape rather than via kinematic systems, subjects were not asked to repeat the task as many times, since the very fine details of the movements were not observable. In terms of movement characteristics, the reach and grasp task is not *complex*: the configuration of the hand(s) is determined solely by the shape of the object to be grasped. It is not *representational*, because the movements have no abstract meaning. It is minimally *sequential*, because subjects had to first grasp the object and then set it down. It is *co-ordinated*, because subjects had to use proximal joints for reaching and distal joints for grasping. The task is *targeted*, because it requires subjects to move toward the objects; and it requires *interaction with an external object.*

Table 4.5: Reach and Grasp Objects

	Diameter (cm)	Height (cm)	Weight (g)
Cylinder 1	8.5	12	12
Cylinder 2	6.3	9.2	15
Cylinder 3	5	17.7	285
Cylinder 4	4.5	11	5

4.5.2.5 Tool use

The tool use task was included in the study to assess subjects' understanding of complex, representational movement as well as to assess their ability to adopt the appropriate hand configurations to manipulate objects. Subjects were presented with household objects and asked to demonstrate how they are used. The objects for this task included: a whisk, corkscrew, peeler, hammer, rubber, screwdriver, cigarette lighter, knife, pepper grinder, spanner, and keys. The tool use task is *complex*: it requires hand configurations that are not determined solely by the shape of the tools, but rather by how they are intended to be used. It is *representational* for a similar reason: subjects have to show how the tool is used, outside the context of its actual use. The tool use task

is minimally *sequential*, because subjects have to grasp an object before demonstrating its use. It is *co-ordinated*, because grasping a tool and illustrating its use require co-ordinated movements of multiple joints. It is *targeted*, because subjects have to move toward a tool and grasp it in order to demonstrate its use. Finally, the tool use task requires *interaction with an external object*, although tool use as an assessment of apraxia often does not require subjects to handle an external object (Halsband et al., 2001; Merians et al., 1999).

4.5.2.6 Object manipulation

The object manipulation task was similar to the tool use task, except that the purely representational component was removed. Instead of being shown an object and asked to demonstrate how to use it, subjects were given a demonstration or illustration of what to do with the object and asked to replicate it. The task was included in order to allow assessment of subjects' performance on a moderately difficult, non-linguistic task, and in particular on a task that could differentiate difficulties in targeting and co-ordination in a non-representational context. The materials for this task included a buckle, screw cap, pen, and a set of plastic toys. The appropriate actions for the first three objects are self-evident. With the set of toys, subjects were given a photograph of the toys arranged in a specific way and asked to place them in that arrangement. The object manipulation task is *complex*, because the hand has to assume a configuration that is not solely determined by the shape of the object. The task is not *representational*, because the movements have no meaningful component. It is *sequential*, because subjects have to grasp an object first, then manipulate it. The task is *co-ordinated*, because grasping a tool and manipulating it requires co-ordinated movements of multiple joints. Like the tool use task, it is *targeted*, because subjects have to move toward an object in order to manipulate it. Finally, by definition, the object manipulation task requires *interaction with an external object.*

4.6 Measures / Coding scheme

Coding schemes were designed to capture observable patterns in the data on the basis of predictions and practices from sign language and motor control research. Because this study focused on subjects' hand and limb movements, they were the only movements that were coded. Disruptions to eye and facial movements that appeared during testing, as well as chronic gait and postural disturbances, were not explicitly coded but were noted and will be described briefly in chapters on individual subjects. In

both the linguistic and non-linguistic tasks, subjects' movements were coded descriptively (in terms of their physical form) and categorically (as erroneous or not) (See Tables 4.6 and 4.7 below). The classification of errors was a difficult issue in the development of the study. Signed and fingerspelled productions were considered to be erroneous when they differed noticeably from the citation form of the sign as represented by the most widely-accepted BSL dictionary (Brien, 1992) and confirmed by a native signing consultant. So a sign would be coded as having an error if the fingers were visibly laxed rather than fully extended, or if a movement was produced with tremor. This coding standard is consistent with the literature on atypical signers (Brentari & Poizner, 1994; Kegl et al., 1999; Loew et al., 1995; Poizner et al., 2000). In the interest of consistency, the same standards were applied to the non-linguistic tasks, so that an observably atypical handshape, direction, location, involuntary movement, etc., in those contexts would similarly be classified as errors. It is common in the motor control literature for performance on a task to be described as disrupted or deficient even when subjects are able to do the task (e.g. grasping an object), if the details of those movements are somehow atypical (e.g. slow or with hesitations) (Ingvarsson et al., 1997). The movement tasks in this study were coded in the same way, although the measures themselves were less precise, due to limitations of the videotape medium.

4.6.1 Linguistic coding

4.6.1.1 Signs

This study was the first to compare formational properties of signs at a phonetic level in subjects with various movement disorders, so it was necessary to design a new coding scheme for describing the data. (A full list of the sign coding parameters is presented in Table 4.6, and a coding sample can be found in Appendix C.) The coding scheme was developed on the basis of measures and coding schemes from previous research on sign language (Brentari, 1998; Stokoe, 1960; Tyrone et al., 1999), gesture (McNeill, 1992; Pedelty, 1987), apraxia (Goldenberg, 1995), and motor control (Castiello et al., 2000; Ghez et al., 1996). The coding scheme began with the linguistic classifications that have been used to describe signs' sublexical structure, which have become known as phonological parameters (Sutton-Spence, 1999). Stokoe (1960) was the first to suggest that signs in American Sign Language could be broken down and differentiated according to three formational properties: handshape, location and movement. Later researchers included orientation of the hand as another parameter (Battison et al., 1975; Friedman, 1976). To give an example of a sign and its

phonological parameters, the British sign DON'T-KNOW has an open-B handshape, palm orientation toward the body with fingers pointing up, location at the forehead, and a single straight movement away from the body (Brennan et al., 1984) (Figure 4.2). The last three parameters are self-explanatory; for the handshape parameter, the label "B" comes from the notational system based on the one-handed manual alphabet used in Europe and North America. (Charts of handshapes, orientations, and locations are included in Appendix B.)

Values for the formational parameters of signs

Descriptions of signs were phonetic rather than phonological, so it was necessary to include values of parameters (especially handshapes) that are not distinctive in the language. Possible handshapes, movements, locations, and orientations were taken from previous research on sign language (Brentari, 1998; Friedman, 1976) and gesture (McNeill, 1992); and where distinctions between those values and what appeared in the data could be discerned, new values were added to the coding scheme. Values for the movement parameter (which was coded in the data as "direction") included the direction, manner, and number of repetitions of movements originating from the wrist or more proximal joints.

Figure 4.2: BSL sign DON'T KNOW (from Brennan et al., 1984)

For describing a sign's location in space, a schema used by McNeill (1992) (Figure 4.3) was adapted to include anatomically-defined reference points for signs that

make contact on the face or contralateral hand, since those sign locations are more finely differentiated. Based on research on ASL and Parkinson's disease (Brentari & Poizner, 1994), the scheme was designed to code relative and absolute locations of signs. To this end, locations that subjects produced were compared to target locations of the sign and coded for their positions relative to the target. Relative locations could be: neutral, medial, (ipsi)lateral, high, low, distal, or proximal, or a combination of the last six. ('Neutral' means that the produced and target locations are the same.)

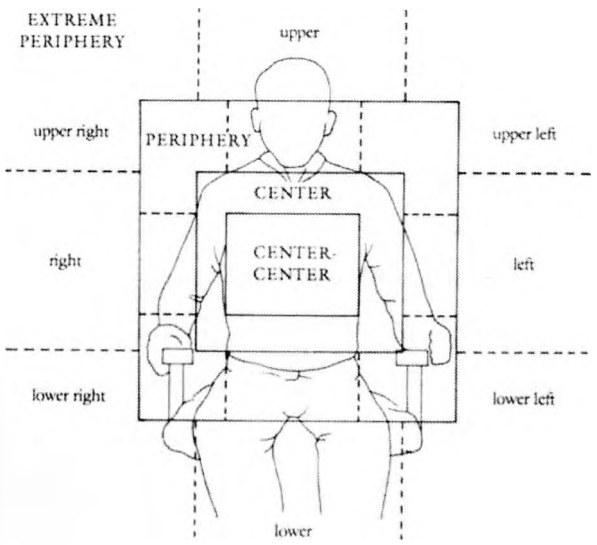

Figure 4.3: Chart of absolute sign locations (From McNeill, 1992)

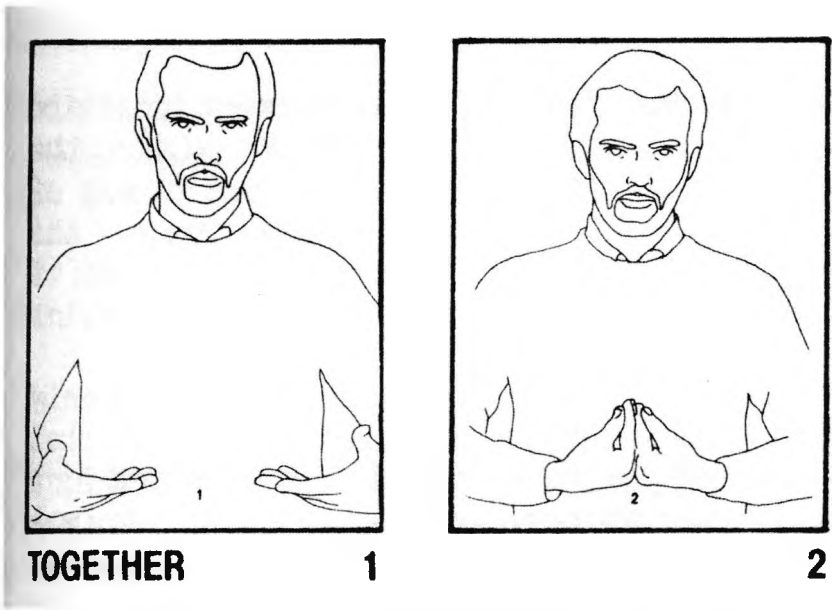

Figure 4.4: BSL sign TOGETHER (from Brennan et al., 1984)

Of the aforementioned formational parameters, movement is necessarily dynamic: by definition, it requires that the articulators move. However, other parameters can be dynamic as well, or require a change from one value to another within a sign. An example of a sign that requires a change in handshape along with an arm movement is the British sign TOGETHER (Figure 4.4). To address this in the coding scheme, descriptions of the initial and final handshape and location for each sign were coded, and if a sign required movement from one location to another, then the movement between the locations was described (e.g., an arc vs. a straight line).

Initially, the coding scheme was designed to code simply for whether or not subjects produced the parameter changes required by the sign, and used a yes-no analysis for handshape and location change. This had to be modified because some subjects added these changes when the target sign did not call for them. For example, on a sign like DON'T-KNOW (Figure 4.2), subjects might add a handshape change even though the sign does not require one. As a result, addition as well as omission of parameter changes was included in the coding of the data.

Co-ordination

Initial analyses of the data revealed that it was necessary to code for the timing of changes in sign parameters relative to each other, because it is possible for a sign to be malformed not just on the basis of the presence or absence of articulators' movements, but on how those movements are related. In the sign TOGETHER, the handshape change and the movement must partially overlap in time for the sign to be well-formed. Consequently, it was noted whether subjects omitted one movement when two were supposed to co-occur, or whether the two co-occurring movements were instead produced sequentially. The latter type of error has been documented in signers with Parkinson's disease (Brentari et al., 1995; Tyrone et al., 1999).

Returning to one point about the differences between the physical structure of sign and of speech, signs can (but do not necessarily) use paired articulators on opposite sides of the body, which are controlled independently of each other. As a result, 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. It was noted whether subjects produced two-handed signs with only one hand (or vice versa), and whether the two hands began together and continued moving together, and in signs with alternating movements, whether they alternated correctly (i.e. not moving simultaneously or asynchronously).

Table 4.6 Sign Coding Parameters

Handshape: a descriptive category for the configuration(s) of the hand(s) in a sign

Handshape error: a closed category to indicate whether a subject's production of *handshape* matches the requirements of the sign target

Handshape change: a closed category to indicate whether a subject's production of handshape change matches the requirements of the sign target

Orientation: a descriptive category for the orientation(s) of the hand(s) in a sign

Orientation error: a closed category to indicate whether a subject's production of *orientation* matches the requirements of the sign target

Orientation change: a closed category to indicate whether a subject's production of orientation change matches the requirements of the sign target

Location (relative): a descriptive category for where subjects place a sign in relation to its target location

Location (absolute): a descriptive category for where the hand(s) are located

Direction: a descriptive category for the direction, number, and manner of the movements of the arms in a sign

Repetition: a closed category indicating whether a sign is repeated in full without pause

Involuntary movement: a closed category indicating whether any involuntary movements occur during sign production

Target features: the phonological parameters that must change in sign production (i.e. handshape, movement, or orientation, or some combination thereof)

His features: the phonological parameters that do change in the subject's production of the sign, in the order that the changes occur

Proximal/Distal Co-ordination Errors: a closed, binary category comparing *target features* and *his features*

Bimanual Co-ordination Errors: a closed category to indicate whether a subject's use of the two hands matches the requirements of the target sign

Finally, movement anomalies that did not fit into any of these categories were noted as they emerged in the data and the coding scheme was modified accordingly. If a subject exhibited a particular type of atypical movement more than twice while signing, the characteristics of that movement would be analysed carefully, on the basis of similarities to speech dysarthria or to other movement disorders. If the characteristics of the atypical movement were easily classified, a new parameter would be added to the coding scheme. For example, one subject (Joseph), when asked to copy a sign, would often produce the sign twice in rapid succession without pausing. Consequently, the parameter 'repetition' was added to the coding scheme.

Once the sign coding scheme was completely formed, it was explained to a second sign language researcher, who then coded fifty percent of the sign data (the first half of all subjects' productions) independently to ensure the reliability of the results.

The second researcher did not know the subjects but was familiar with their neural pathologies. She was a trained sign language researcher and speech and language therapist, and had research experience with children's acquisition of sign phonology, but not with disrupted signing. On the data coded by both researchers, there was a 96.91% agreement rate on both descriptive and categorical coding. A sample of sign coding for one subject is included in Appendix C.

4.6.1.2 Fingerspelling

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. (A chart of the British fingerspelling system is included in Appendix B, and a fingerspelling coding sample in Appendix C.) The fingerspelling data were coded at the level of the individual letter, and most letters have no internal movement; consequently, it was unnecessary to code for handshape change. Also, in BSL, there is much less variability in handshape from one fingerspelled letter to another than from one sign to another, so it is less likely for there to be handshape change or co-articulation across letters[5].

4.6.2 Non-linguistic coding

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 & Webb, 2001; Wing et al., 1996). Because videotaping does not allow detailed measurements of movement speed and trajectory, the coding scheme was designed to describe individual movement errors broadly. Wherever possible, the measures from the sign and fingerspelling coding schemes were used for coding the non-linguistic tasks as well. For example, signing, fingerspelling, and most of the non-linguistic tasks could all be coded for targeting (relative location in the sign and fingerspelling coding schemes) and the presence of involuntary movements. A full list of the measures for the non-linguistic movement tasks is included in Table 4.7, and a coding sample is included in Appendix C.

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[5] Although it has been argued that transitions between letters are important (Akamatsu, 1983), in these data, those transitions were often too quick to code with any reliability.

Table 4.7: Non-linguistic Coding Parameters

Involuntary movement: an involuntary movement that occurs during voluntary movement

Initiation: difficulty or hesitation in beginning a movement

Completion: difficulty completing a task

Pause/Hesitation: cessation of movement during execution of a task

Targeting: inaccurate placement of hand relative to target

Hand configuration: descriptive measure of hand configuration

Hand configuration error: atypical configuration of the hand for the particular task

Speed: excessively slow or rapid movement

Proximal/distal co-ordination: error in relative timing of movements of proximal and distal effectors

Bimanual co-ordination: error in relative timing of movements of the two limbs

4.7 Summary

Many factors had to be taken into account in designing this study, the main ones being: obtaining the most informative data from a varied, atypical group of subjects who are small in number, finding the best medium for capturing the relevant data, and finding ways of comparing data that are different in nature. It has been necessary to compare and combine research methods and equipment from many fields, often in ways that had not been tried. It is hoped that by devising new measures and examining data along new criteria, insight can be gained into the movement and articulatory deficits being described. In turn, it is hoped that this study will introduce narrower research questions to be addressed by precise measurement of sign and movement, and open up new areas of research in both linguistics and motor control research.

5 James: RHD Signer

This chapter examines the case of a signer, James, who had unilateral right hemisphere damage as a result of stroke. His case raises an interesting question about the nature of sign language dysarthria, because a unilateral cerebral infarct often will not cause dysarthria in a hearing speaker, and only rarely will it cause dysarthria in the absence of other symptoms affecting speech or language. Dysarthria following stroke is frequently masked by more severe manifestations of aphasia, apraxia, or aprosodia (Kent et al., 2001). In addition, James's case is interesting because a localized cerebral infarct is unlikely to result in dysarthria if it is located in the right hemisphere. Many have suggested that articulation is governed by left hemisphere structures; consequently, damage to the left hemisphere is more likely to result in dysarthria for spoken language. James's case allows the examination of the question of hemispheric dominance for articulation in signed language as well. Is his a case of dysarthria that would appear in signed but not spoken language, or a different manifestation of the same phenomenon that appears in spoken language? Because of the scarcity of cases of dysarthria from focal right hemisphere damage, it is difficult to generalize about the nature of the resulting form of dysarthria and compare this case to data from the literature. That said, the two most obvious comparisons to be made are with spastic and unilateral upper motor neuron dysarthria. Given the nature of James's neural damage, it is predicted that he will be roughly equally impaired on linguistic and non-linguistic tasks, since unilateral right hemisphere damage typically affects articulation only mildly, if at all.

5.1 Right hemisphere damage

5.1.1 Upper motor neuron dysarthria

UUMN dysarthria

Both the nature and prevalence of unilateral upper motor neuron (UUMN) dysarthria remain extremely unclear (Duffy & Folger, 1996; Hartman & Abbs, 1992). Some researchers treat UUMN dysarthria as a subcategory of spastic dysarthria, because it results from similar pathologies but with damage to only one side of the brain (Thompson-Ward, 1998). However, its severity as well as its actual existence is highly variable across patients with the same lesion (Dronkers, 1996; Kent et al., 2001). Early research suggested that UUMN dysarthria was similar to spastic dysarthria but less severe and affecting fewer peripheral structures (Darley et al., 1975). In particular, with respect to speech, UUMN dysarthria has less effect on respiration, voicing, and intensity, which follows from the fact that the larynx and trachea receive more bilateral

innervation than the tongue and lips. Duffy (1995) suggests that UUMN dysarthria reflects muscle weakness and inco-ordination. Additionally, acoustic and perceptual studies suggest UUMN dysarthrics exhibit mild articulatory imprecision (Hartman & Abbs, 1992; Thompson-Ward, 1998), slow speech rate, and mild hypernasality (Duffy, 1995). Clinical data show that the most frequently occurring non-speech symptoms include: mild dysdiadochokinesia, hemiplegia/paresis, tactile deficits, clumsy hand syndrome, and unilateral weakness in the lower face, tongue, or palate. Judging from past research, these non-speech symptoms as well as slowed and mildly unco-ordinated signing are what one should expect to see in a signer with UUMN dysarthria.

Spastic dysarthria

Since the innervation patterns of the articulators for sign and for speech differ, it is worth briefly outlining the symptoms of spastic dysarthria, on the rationale that unilateral brain damage could cause a deficit to signing that resembles spastic dysarthria. In hearing subjects, spastic dysarthria is usually the result of bilateral damage to upper motor neurons, as in the case of motor neuron disease. However, sign articulators get primarily unilateral innervation, while speech articulators get more bilateral innervation; as a result, unilateral cerebral damage to a Deaf signer could have the same effect as bilateral cerebral damage to a hearing speaker.

Spastic dysarthria was originally described as a manifestation of heightened muscle tone and impaired skilled movement (Darley et al., 1975). According to Duffy (1995), spastic dysarthria causes impaired movement patterns rather than muscle weakness, thus affecting multiple components of speech at once. Its distinguishing characteristics are slow but regular speech rate and harsh/strained voice quality (Darley et al., 1975). Other speech components include long syllables (Linebaugh & Wolfe, 1984), hypernasality (Enderby, 1986), imprecise consonants and distorted vowels. Moreover, unlike UUMN dysarthria, spastic dysarthria is apt to cause disruptions to stress/prosody, pitch, loudness and voice quality. Speech is perceived as slow, effortful and imprecise (Darley et al., 1975).

Darley et al. (1975) suggested that spastic dysarthria reflects four components of muscular dysfunction generally: spasticity (i.e. heightened muscle tone), weakness, limited range of movement, and slowness. Clinical observations of hearing speakers with spastic dysarthria have shown that the movements of the limbs may be reduced in range and force, with a loss of fine-grained, skilled movement, which is most pronounced in the hands and fingers (Enderby, 1986; Darley et al., 1975). So clearly,

there is significant overlap in the symptoms affecting the limbs in spastic and UUMN dysarthria. Based on the descriptions by Darley et al. (1975), the primary differences between the symptoms associated with the two would be that spastic dysarthria is associated with heightened muscle tone, while UUMN dysarthria results in mild inco-ordination.

5.1.2 Sign language and RHD

James's case not only provides an interesting comparison to clinical data on hearing dysarthric speakers, but also to findings from research on signing and right hemisphere damage. While several studies have examined right hemisphere damage (RHD) and sign language, only one has described the effects of RHD on sign articulation (Poizner & Kegl, 1993). In that respect, this study provides an important addition to the body of research. Thus far, most RHD and sign language research has focused on signers' linguistic, visuo-spatial, or other cognitive deficits. A prevalent theme in that area of research has been the absence of grammatical deficits in the signing of RHD subjects in spite of deficits in visuospatial processing thought to be necessary for grammatical function in signed language (Hickok et al., 1996; Poizner et al., 1987; Poizner & Kegl, 1993).

Deaf RHD subjects, like their hearing counterparts, experience impaired visuospatial processing and visual-hemifield neglect (Hickok et al., 1999; Poizner et al., 1987). However, Deaf RHD subjects' language use allows the nature of those deficits to be probed further, since signed language incorporates the physical locations of signs in space into its grammatical structure. Various studies have explored the relationship between visuospatial processing and linguistic use of signing space, with varied results. Some have found that Deaf RHD signers have problems with mental rotation on non-linguistic but not on linguistic tasks (Emmorey et al., 1996); or with topographic, but not linguistic, use of space (Emmorey et al., 1995; Poizner et al., 1987).

More recently, it has been suggested that there are fundamental components of sign language, sometimes described as "paragrammatic," that rely heavily on the right hemisphere. RHD deficits have been reframed as impacting on sign language minimally or indirectly, rather than having no effect on language at all. For instance, the right hemisphere has been shown to play a role in linguistic discourse function (Hickok et al., 1999; Poizner & Kegl, 1993), non-manual role shift (Loew et al., 1997), perception of grammatical facial expression (Atkinson et al., 2004) and production and comprehension of classifiers (Corina & McBurney, 2001).

An earlier case study of a Deaf signer with right hemisphere damage reported that he had a mild articulatory deficit (Poizner & Kegl, 1993). In particular, the subject in that study showed deficits in co-ordinating the two arms over the full course of the production of two-handed signs, which the authors attributed to motor neglect on the subject's affected side. This study is similar in that it reports mild dysarthria in a signer with right hemisphere damage, although the details of the articulatory deficits diverge; similarities and differences in this case and that one will be described in the final section of this chapter.

5.2 James: Background

James is a 79 year old man who was born deaf and grew up in a hearing family. He began learning BSL when he attended an oral day school for the deaf as a child. He continued in secondary school until the age of 15 and worked in various manual trades before retiring. He emphasizes his interest in maintaining good speech and lipreading skills, and many of his family members and social contacts are hearing. Though he has a speech-oriented background, his second wife is Deaf, he had exposure to BSL from a young age, and he participates in events at the local Deaf club.

James suffered a moderate CVA in the territory of the right middle cerebral artery. Brain scans indicate patchy low density in the right hemisphere parietal region, but no evidence of haemorrhaging or swelling, and no midline shift, which suggests that the area of damage is delimited and unilateral. Scans also reveal that James had probably had earlier minor CVAs, which caused slight damage to the left thalamus and the right corona radiata; however, there is no record of neurological symptoms prior to the most recent CVA. James's primary motor deficit is left hemiparesis, affecting the hand most severely. He can move his left arm reasonably well, although it has a limited range of movement, and he cannot change the configuration of his left hand at all. His right side shows no movement deficits that are readily apparent. However, his movements on both sides are slightly slow, including skilled movements and more automatic movements such as sitting, standing, and walking.

5.3 Deaf Stroke Project Findings

5.3.1 Neuropsychological Testing

James was tested on various standardized neuropsychological measures (described in Chapter 4) as a participant in the Deaf Stroke Project. His performance indicated difficulties with abstract, non-linguistic reasoning and visuospatial processing,

and particular difficulties with processing information about faces. (For a more detailed description of James's performance on these tests, see Atkinson et al., in press.) Table 5.1 lists his test scores and indicates which of those represent severe impairment.

Table 5.1: James's Background Assessment

Abstract non-linguistic reasoning	
Ravens Progressive Matrices	15/60*
Pyramids and Palm Trees Test	50/52
Visuospatial	
Wais –III Block Design	24
Line Cancellation	1 omission, left of midline
Benton Test of Line Orientation	26/30
Facial Processing	
Benton Face Recognition	36/54*
Ekman & Friesen Recognition of Facial Expression	44/60
Reduced Warrington Recognition Memory:	
Facial Recognition	3/15*
Car Recognition	10/15

*indicates severely impaired

There are a few details to note about the neuropsychological tests: the Warrington recognition memory test that was used was a shortened and modified version, created by the Deaf Stroke Project. The purpose of the car recognition component of the test is to distinguish recognition memory in general from recognition memory for faces in particular. So in fact, a high score on that component reflects the severity of a low score on the facial component. Also, it should be noted that the Ekman and Friesen test is designed to assess recognition of facial expression; whereas the Benton face test is designed to assess recognition of individual faces.

On the whole, James performed better on the tests of general visuospatial processing than on either the non-linguistic reasoning or the face recognition tests. This is of interest because impaired visuospatial processing is one of the hallmark symptoms of right hemisphere damage. Although he was severely impaired on aspects of non-linguistic reasoning, he did not perform as consistently poorly on those tests as on tests of facial recognition. His specific difficulty with faces was illustrated quite clearly on the Warrington test, as he was able to recall 10 out of 15 cars, but only 3 out of 15 faces.

5.3.2 Deaf Stroke Project Clinical Observations

Members of the Deaf Stroke Project noted that James's spontaneously-produced linguistic deficits were primarily pragmatic in nature. In particular, he did not monitor his interlocutors well: he maintained poor eye contact, had difficulty with conversational turn-taking, told jokes poorly (e.g. placing the punchline first), and his discourse was very repetitive. All of these are typical of RHD language impairments, in spoken as well as signed language (Hickok et al., 1999; Myers, 1997). Specific to his sign production, James used very little fingerspelling, especially relative to other signers of his generation, but based on his own report, this was true before the stroke as well.

Testing also indicated that James's stroke caused minimal disruption to his vocabulary or grammatical ability (Atkinson et al., 2004). He showed no deficits in naming, lexical matching, or sentence comprehension. However, he was impaired at judging grammatical facial expression; and consistent with previous research on sign language and right hemisphere damage (Corina & McBurney, 2001), he had difficulty correctly producing and perceiving classifiers.

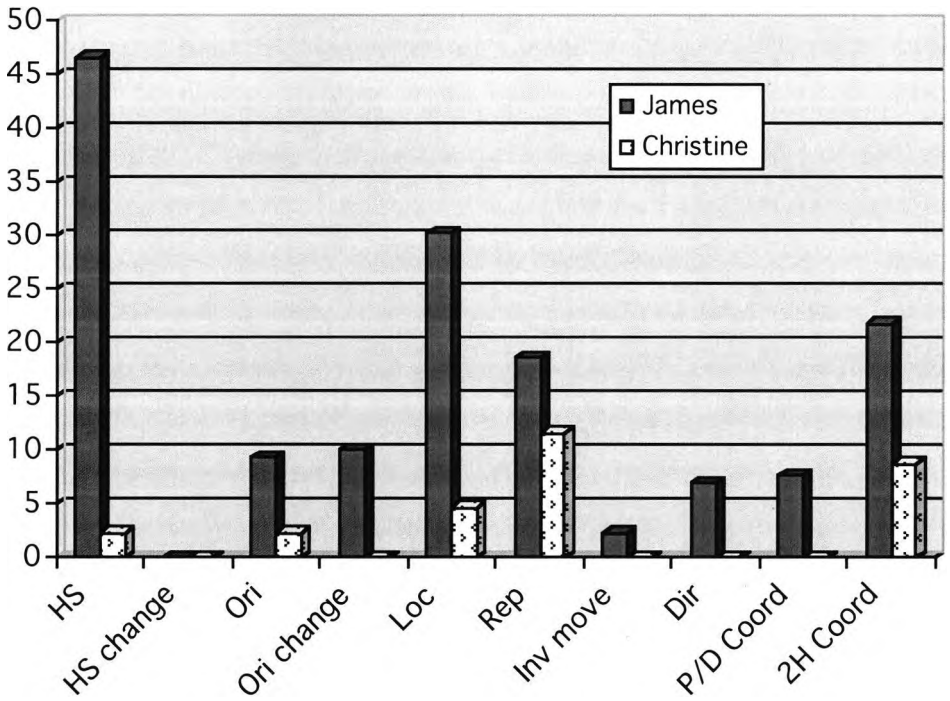
5.4 Signing Task

5.4.1 Methods: Signing

For this study, James performed a naming task in which he produced individual signs corresponding to pictures presented to him by the experimenter; and Christine, the 72 year old female control subject, performed the same task. The coding scheme described in the previous chapter was used to categorize their errors. James had occasional difficulty identifying objects on the task (as did the control subject); however, he had no lexical retrieval errors that might affect articulation. Consequently, no data had to be excluded on that basis. Spontaneous repetitions of the same sign were excluded from analysis, and only the first sign production for each trial was analysed. If repetitions occurred without pause or modification, and for no clear semantic or pragmatic reason, the repetition itself would be considered an error (see discussion of error types in Chapter 4). Though spontaneous sign repetitions were excluded, spontaneous productions that did not match the intended target were analysed.

Figure 5.1: Naming Task: James

errors as % of 43 productions



Key: HS: handshape;
HS change: handshape change;
Ori: orientation;
Ori change: orientation change;
Loc: location;
Rep: sign repetitions;
Inv movement: involuntary movements;
Dir: direction of movement;
P/D Coord: proximal/distal co-ordination;
2H Coord: bimanual co-ordination

A total of 43 individual sign productions was analysed for both subjects. Because this is an exploratory study, it was thought best to employ analysis techniques that transform the data as little as possible; consequently, much of the analysis is qualitative and the data are presented in the form of charts and figures.

5.4.2 Results: Signing

Of all the sign errors that were coded, James had the highest percentage of errors in handshape (46.51% of 43 productions) and location (30.23% of 43 productions), both static components of signs (Figure 5.1). James showed far fewer errors on dynamic components of signs (handshape or orientation change) or their timing relative to each other. No dynamic features were explicitly controlled for in the stimuli; however, several of the target signs required handshape (n=7) or orientation (n=10) change, or required co-ordination of movement at or below the wrist with movement of the arm (n=13). So it is noteworthy that James showed little difficulty producing such complex, co-ordinated movements, given that they occurred often in the stimuli. He showed more difficulty co-ordinating his arms to produce a two-handed sign, though not as much as one would expect given that his hemiparesis: 21.74% of his 23 two-handed signs had errors in bimanual co-ordination, compared to 8.70% for Christine. (There were 43 signs altogether in the task, but 23 required the use of two hands, so the latter was used to calculate percentages of errors.)

The distribution of handshapes in the target signs was not controlled; in other words, signs were not selected to have handshapes that are representative of those in the language, or to show differential ease of production. However, it is doubtful that a more carefully controlled stimulus would have produced a different outcome, since the errors were so pervasive and bore no obvious relationship to particular features of signs. It should be pointed out that James maintained the same handshape on his left hand throughout the testing session; this was not included as a handshape error, since it would have masked the incidence of handshape errors on the opposite hand. By contrast, the distribution of target locations in the stimuli was controlled (24 of the 43 target locations were in the central or lower part of the signing space); and in fact, the target location did affect what James produced. He lowered some target signs with high locations but did not lower other signs.

The only other error that James produced was spontaneous repetition of individual signs; he did this on eight signs (18.60% of 43 productions). To be considered a repetition, a production had to contain (at least) two full iterations of an

entire sign without pause. Additionally, a sign was not counted as a repetition if it was clear that it had been deliberately modified from one iteration to the next (e.g. if the signer changed the orientation of the hand). Christine produced repetitions on five signs (11.67% of her 43 productions) by this definition as well, so the measure of repetition errors probably does not reflect an important difference between the two of them.

Table 5.2 shows the distribution of James's and Christine's errors across individual signs. A couple of patterns in the error distribution are worth pointing out. First, the two signers did not overlap considerably in terms of which signs they produced errors on, even in the categories where they both have errors. So the errors are not determined by the formational properties of the signs, at least not in the same way for both subjects. Second, James had far more errors of lax handshape than of lax orientation; however, the orientation errors all co-occur with the handshape errors, so there may have been a general laxing tendency that was more extreme in some productions than others, and the laxation of the articulators spread to become more proximal (i.e. to affect orientation as well as handshape).

James's location errors all patterned consistently, in that locations were always lowered, and never produced superior, peripheral or medial to the target location. Furthermore, the only signs that were lowered by the right hand were those with high target locations, so James was apparently not lowering his entire signing space uniformly. Also, not all signs with high locations were lowered—but all of those that were lowered had high locations. On some two-handed signs, the left hand did lower a high target location. However, in several of these cases (e.g. RABBIT) the right hand reached the target location, even though the left hand did not, which shows that the right hand was not compensating for the limitations of the left when right-handed signs were produced low. This is supported by the fact that many of the signs that were lowered by the right hand were one-handed.

Table 5.2 Sign Error Distribution: James (x = James; o = Christine)

	HS	HS change	Ori	Ori change	Loc	Rep	Inv Move	Dir	P/D Coord	2H Coord
SOLDIER	x		x							
DOG										x o
GARDEN					x o					
CHAIR										
BANK						x o				
FACTORY								x		x
CHEESE	x									
TREE	x									
AIRPLANE	x									
KING										
FISH	x									
BUS										
SOAP	x									
BISCUIT	x									
FARM						x				
TUNNEL						x				
HAMMER										
RABBIT	x				x	o				
OWL					x					o
ESCALATOR	x									
SUGAR					x					
SHEEP	x		o		x					x
FRIDGE						x				x
ORANGE					x					
CHERRY						o		x		
BREAD	x									
POTATO										
MOUSE	x				o					
FLOWER	x		x		x					
POLICE										
SANDWICH	x									
CLOUD	x									
RAIN										
CHOPS	x									
MAKE	x		x							
BACON						x o				
LIFT	x					x		x		
SHOWER					x	x			x	
APPLE					x					
MEAT	x		x		x	x o				
HORSE	o									
DEER	x				x					
FENCE										

Christine lowered two signs: one with a target location at the nose (MOUSE), and the other with a target location at the shoulder (GARDEN). Because there are only two productions like this, and because one has a high target location and the other does not, it is reasonable to assume that this is a form of relaxed articulation on her part. Also, both of these signs are one-handed, so she produced no symmetrical signs with the two hands in different locations.

Figure 5.2 Clawed-3, target handshape for the sign HORSE

The aspects of sign production that James performed worst on were the static components: the location, orientation, and handshape of a sign. Unlike location, however, handshape and orientation were laxed irrespective of the features of the target sign. For instance, the handshape of a target sign had no effect on whether or not the handshape that James produced was laxed. As a comparison, Christine produced only one lax handshape, which was on a sign with an uncommon handshape (HORSE) (Figure 5.2). If there is any other feature of signs governing production of lax handshape and orientation by James and by Christine, it is not obvious on examination.

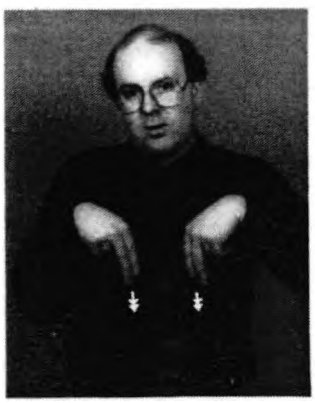


Figure 5.3 DOG

Figure 5.4 FACTORY

(From the British Deaf Association Dictionary of British Sign Language/English)

James produced four signs with disrupted bimanual co-ordination; i.e., the two arms did not begin moving together. Those signs were: FACTORY, DOG, FRIDGE, and POTATO (Figures 5.3 and 5.4). All these signs are low in space and have the same handshape, orientation, and movement in both hands. Christine has two signs with disrupted bimanual co-ordination: DOG and OWL. In both cases, her right hand starts moving long before her left hand begins. It is not clear if there is any type of pattern to James's or Christine's errors—the small numbers make it difficult to judge. It is interesting, however, that all of the signs where errors were exhibited were relatively simple, by almost any measure other than number of hands required. As stated above, James kept the same handshape on his left hand for all the signs. Consequently, when a sign required a handshape change accompanied by a movement, he never produced the handshape change with his left hand. However, this was not included as a bimanual co-ordination error, because it would have masked the incidence of bimanual co-ordination errors at the level of arm movements.

Figure 5.5: James's production of FLOWER, as an example of lax handshape and lax orientation.

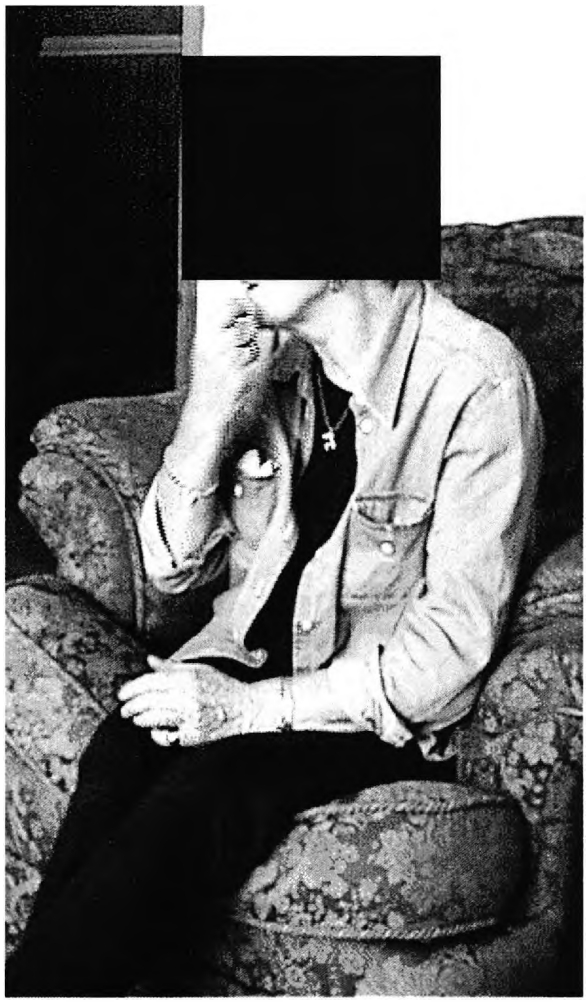


Figure 5.6: Christine's production of FLOWER, with the base of the fingers oriented upwards and the handshape more tightly closed.

5.5 Non-linguistic Tasks

5.5.1 Pointing

5.5.1.1 Methods: Pointing

During the toy placement task, James spontaneously produced many deictic points to the illustrations and to the toys. Because these were fundamentally different in nature from his prehension movements in that task, they were separated out and analysed independently. Altogether he made 30 points to either drawings or objects, and each of these was analysed along the criteria described in the coding scheme for non-linguistic tasks, namely: movement initiation, movement termination, involuntary movement, pausing/hesitation, targeting error, hand configuration, speed, and proximal/distal co-ordination. A complete description of the error categories can be found in Appendix C; however, it is worth elaborating on two error types that may not be clear in the context of pointing. First, although pointing does not require a particular hand configuration, some subjects do choose notably odd configurations of the hand,

rather than the typical default of an extended index finger or thumb (see Chapter 9 for discussion of hand configuration in particular). Second, while there are no specific demands for co-ordination of the proximal and distal effectors in pointing, as outlined in Chapter 4, it is possible for subjects to exhibit a co-ordination deficit by moving their hand to the correct location, pausing, and then configuring the hand to point to the target.

5.5.1.2 Results: Pointing

James had very few errors on the pointing task at all. On the 210 data points that were counted (30 productions analysed along 7 criteria), James produced 4 errors, which were distributed evenly across measures and trials. This was considered to be a low enough error rate to make further analysis uninformative. Christine's performance on the pointing task was similar to James's. She had roughly the same number of errors (n=6), also distributed evenly across measures and trials. Because of the small number of errors and the simplicity of the task itself, it was not thought useful to explore the nature of his errors compared to hers. The only detail worth mentioning is not an error but a behavioural pattern: on nine of the thirty trials, Christine used her left hand rather than her right, which was not a possibility for James. Otherwise, suffice it to say, James and Christine patterned slightly differently but showed significant overlap, and as such, differences between them on this task cannot be attributed to anything other than chance.

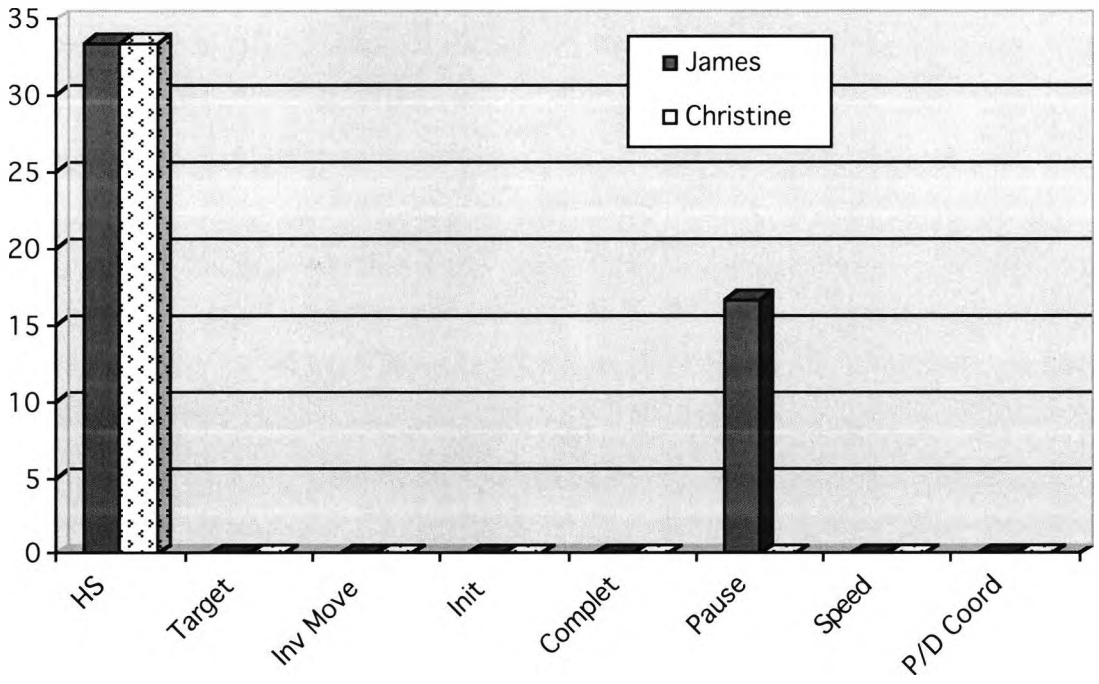
5.5.2 Kimura box

5.5.2.1 Methods: Kimura box

For the Kimura box task (Kimura, 1977, 1993), the procedure was similar to that employed by Sunderland and Sluman (2000). The box has three manipulanda (Figure 4.1) which must be handled in the correct order and using the correct hand configuration and movement. The experimenter demonstrated the sequence of movements slowly and asked the subjects to copy her movements. The objective was to get subjects to produce two complete sequences of correct movements, if possible. The data were coded for targeting, hesitation, and tremor as well as speed of execution and accuracy of hand configuration. Hand configurations were also coded descriptively (according to the set of handshapes shown in Appendix A) as well as being classified as correct or incorrect.

Figure 5.7: Kimura Box: James

errors as % of total movements



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

5.5.2.2 Results: Kimura box

James performed four iterations of the movement sequence, so he had twelve movements to be analysed; whereas Christine performed two iterations of the sequence, so she had six movements to be analysed. Their error rates are plotted in terms of percentages of total movements, so that their performances can be more easily compared (Figure 5.2). James and Christine both had a handshape error rate of 33.33%, which was four handshape errors for James and two handshape errors for Christine. Additionally, though, their errors were different in nature, because all of Christine's errors were cases of handshape laxing. James produced laxed handshapes, but on top of that, three of his four errors were the incorrect selection of a handshape to match what the researcher had produced (e.g., an extended index finger rather than an extended thumb). James also had two pauses during movements, which both co-occurred with his handshape errors.

5.5.3 Toy placement

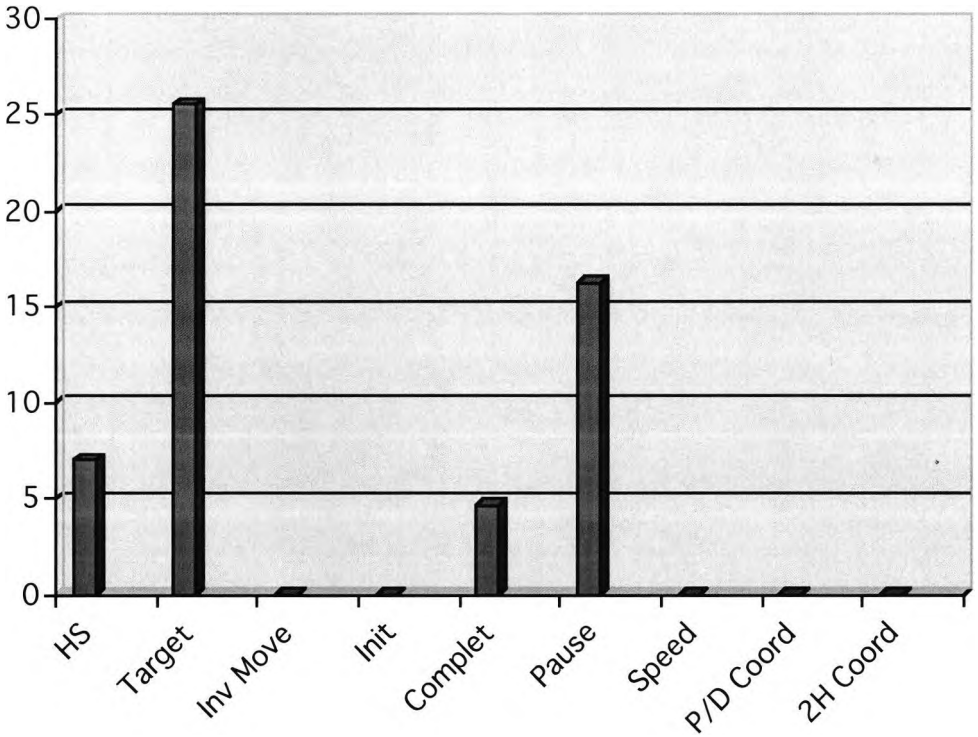
The purpose of the toy placement task was to probe subjects' ability to accurately move to a visual target, to co-ordinate the proximal and distal joints successfully to manipulate objects, and to place objects according to a visual schema. Subjects with right hemisphere damage often experience visuo-spatial neglect that would make such tasks difficult (Hickok et al., 1996; Poizner et al., 1987). More rarely, subjects with right hemisphere damage experience ideomotor apraxia that can disrupt their ability to manipulate objects successfully (Raymer et al., 1999).

5.5.3.1 Methods: Toy placement

James was presented with a set of toys and a series of pictures depicting configurations of those toys and asked to place the toys according to the configurations. The first 43 toy placement tasks were analysed, so that the numbers would be broadly comparable to the data from the signing task. Unfortunately, limits of time and available resources did not allow collection of object placement data from Christine.

Figure 5.8: Toy Placement: James

errors as % of 43 movement sets



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;
2H Coord: bimanual co-ordination

For purposes of analysis, movements are divided functionally in terms of end goals, with movements necessary to achieve a particular goal grouped together as one movement set. For example, if the goal is to move an object from one place to another, then the reach to the initial location, the grasp of the object, and the placement of the object at the second location are grouped together and analysed collectively as one movement set. This framework for analysing movements is consistent with research on symbolic/ideomotor representations of movement (Halsband et al., 2001; Sunderland & Sluman, 2000) as well as research on the kinematics of sequential and bimanual movements (Gentilucci & Negrotti, 1999; Wiesendanger et al., 1996). Kinematic research shows that if an individual grasps an object to push it, the motor plan will be very different from grasping an object to lift it, so for this analysis the grasp is grouped together with the subsequent movement as a task. Exceptions to this rule are when James picks up an object and then pauses to examine it, or to do something else, and then resumes placing the object. In cases like that, the movements are divided into two movement sets. The parameters according to which movement sets are analysed are described in detail in the previous chapter; and the coding of the data from the toy placement task is included in Appendix C.

A list of representative movement sets is presented in Table 5.3, annotated for the features that render them more or less difficult. They are listed approximately according to level of difficulty, however there is not an established means of ranking movement difficulty, so this should be taken only as a rough estimate.

Table 5.3: Typical movement sets and their features

Movement Sets	Formation of hand configuration	Toy as target	Location as target	Force against gravity	Force against toy surface	Bimanual coordination
Place toy			X	X		
Push/slide toy		X	X		X	
Bend toy	X	X			X	X
Pick up toy	X	X		X	X	
Grasp & push/pull toy	X	X	X		X	
Pick up & place toy	X	X	X	X	X	

5.5.3.2 Results: Toy placement

It is difficult to judge bimanual co-ordination from the toy placement task because very few of the movement sets required the use of both hands. Moreover, movement sets that did require two hands were particularly difficult because they are

"asymmetric goal-directed movement sequences" (Wiesendanger et al., 1996). It should be pointed out that James never performed a one-handed movement set with two hands. For example, he never tried to lift a small object with two hands rather than one. On the three movement sets that required both hands, he had difficulty performing them because he could not form the correct hand configuration with his left hand. Fine movements in his right hand were largely preserved: he had only three handshape errors on the task (6.98% of movement sets), and all were on his left hand. James's most common error was a failure to correctly target a toy or location (n=11, 25.58% of all actions) (Figure 5.7). His second most common error was pausing or hesitating during a movement, which happened on 16.28% of all movement sets, or seven times.

5.6 Discussion

5.6.1 Naming

The aspects of sign production that James performed worst on were the static components: the location, orientation, and handshape of a sign. Dynamic components of signs, such as handshape change, orientation change, direction of movement, and proximal/distal co-ordination. Additionally, the static components of handshape and orientation were modified to become more lax, and this was more pronounced for the distal articulators which are required for handshape formation. The laxing in orientation and handshape may be an effect of loss of fine motor control resulting from upper motor neuron damage, which tends to be worst in the fingers and progressively milder in more proximal joints. So James may have been exhibiting a general laxing tendency that was more extreme in some productions than others, and the laxation of the articulators spread to become more proximal (i.e. to affect orientation as well as handshape). However, Christine exhibited articulatory laxing as well, so there may be a more widespread laxing phenomenon related to aging. While articulatory patterns in the signing of elderly subjects have not been researched, there is evidence from motor control research that elderly people show a decline in fine motor control (Contreras-Vidal et al., 1998; Smith et al., 1999), which could easily affect handshape. However, further research would be necessary to determine if this decline is likely to affect signing, and to affect sign handshape in particular.

When James produced errors in the locations of signs, the signs were consistently lowered. He never produced a sign in an incorrect location that was superior, medial, or lateral to the target location. James's lowering of signs was not consistent along any single criterion of the sign, i.e. there was no single feature that

determined that a sign would consistently be produced low. However, the only signs that were produced low were those with high target locations. Furthermore, his lowering of signs was not limited to signs produced with the left hand or to two-handed signs. As an error it probably does not represent a reconceptualization or even simply a uniform lowering of his signing space. Because continuous signing data were not analysed, it is not clear whether low or central signs would be lowered to accommodate James's apparent difficulty reaching high target locations. He was producing one sign at a time, so it seems likely that he would produce individual signs as accurately as possible and without concern for the position of one sign relative to another.

The dynamic error that James produced most frequently on the naming task was bimanual co-ordination. Nonetheless, he had reasonably co-ordinated movements of the two limbs, despite left-sided hemiparesis. Additionally, he had no difficulties using his affected hand as a base hand in two-handed signs. Surprisingly, most of the signs with disrupted bimanual co-ordination were low and symmetrical, i.e. both hands made the same movement, and neither had to move very far. This is especially striking in contrast to the location errors, which only appeared when a sign's target location was high and presumably reflected simply weakness or a limited range of movement.

The final error category from the naming task that should be discussed briefly is sign repetition. Both James and Christine produced several full, identical repetitions of individual signs, without pausing between productions. There is no clear pattern in the formational properties of which signs are repeated and which are not. The only pattern that seems to be noteworthy is in the data that were excluded from this category: namely, both subjects repeat several signs after pausing or after altering the sign slightly as well. As outlined in Chapter 4, repetition of a sign was not counted as an error unless it took place without pause or modification. The original intent of the exclusion criteria for this measure was to capture movements that may be involuntary; however, that is not likely what these two subjects are producing. Consequently, it is predicted that the productions captured by this coding scheme and from these subjects are a subset of a larger phenomenon that is perhaps social or psychological in nature, and beyond the scope of this research. James did not perform a sign copying task in addition to a naming task, so it is impossible to say whether his repetition behaviour is task-specific or not. However, Christine did perform a sign copying task, and in that context, she did not have a high rate of sign repetition. (See Chapter 7 for more discussion of sign repetition.) So it may be that spontaneous repetition of signs, with or without pause or modification, is in some way more likely for a naming task than for a copying task. For

example, it may be that the subjects were trying to produce a perfect token for the former but not for the latter.

5.6.2 Comparisons to past sign language research

In contrast to subjects described in previous research on RHD and signing, James showed no sign language or movement deficits related to visuospatial neglect. Because this study did not examine extended signing, the discourse, grammatical, and prosodic features of his sign productions could not be analysed. Unlike the Deaf RHD signer with articulatory deficits described by Poizner & Kegl (1993), James had only minimal difficulty co-ordinating independent movements of his arms to form signs. Specifically, he only had difficulty co-ordinating the two hands at the onset of movement at the beginning of a sign and not throughout its production. In fact, as described above, James had far greater difficulty with static components of signs. It may be that the signer from that study had a more severe hemiparesis, or it may be that the inability to co-ordinate the two limbs was a manifestation of hemispatial neglect. Two final points should be made about the results from Poizner & Kegl (1993): first, because the signer's articulatory deficit was not the focus of their research, they did not elaborate on her bimanual co-ordination in much detail. Consequently, it is difficult to say what other articulatory deficits may have accompanied it, or precisely how widespread the deficit itself was. Second, it is not clear on what basis Poizner & Kegl (1993) categorize the signer's co-ordination deficit as motor neglect, rather than a movement planning disorder since the two types of movement disruption can co-occur (Laplane & Degos, 1983). Moreover, motor neglect is typically characterized as a spatial rather than a temporal phenomenon (Heilman & Adams, 2003; Laurent-Vannier et al., 2003; Meador et al., 1986), and the latter is what Poizner & Kegl (1993) describe.

Findings from the current study and earlier sign language research support the theory that sign articulation as well as speech may be less disrupted by unilateral right hemisphere damage than by other brain pathologies (Corina et al., 2003; Dronkers, 1996). Although James's signing is disrupted by his right hemisphere infarct, he is only mildly dysarthric. Similarly, according to their report, the signer described in Poizner & Kegl (1993), does not have articulatory disruptions to sign to the same extent as most of the other subjects they report.

Unexpectedly, James patterned similarly to subjects with Parkinson's disease described in earlier studies. Broadly speaking, James's signing was reduced in much the same way as Parkinsonian signing (Loew et al., 1995). He frequently showed slightly

lax handshape in his good hand, and less often lax orientation, and lowering of some signs. However, unlike PD signers described previously (Brentari et al., 1995), he showed no decoupling of proximal and distal movements (e.g. handshape change and arm movement) during signing. Additionally, his sign lowering was not as uniform as theirs: the signers with Parkinson's disease lowered the signing space uniformly, whereas James only lowered signs which had high target locations. Moreover, the signers with Parkinson's disease produced signs from distal articulators, and James did not distalize his signs. More research is necessary to explore the relationships between right hemisphere damage, Parkinson's disease and sign articulation. It is possible that if phonetic measures of signs were refined, greater distinctions between RHD signing and PD signing could be identified. Alternatively, it is possible that by looking at a movement task as demanding and yet familiar as sign language production, this research has identified a functional parallel across motor structures that traditional motor control tasks could not detect.

5.6.3 Non-linguistic tasks

There was not much of note in James's performance on the pointing task. What is worth noting is that James produced far fewer errors on it than on any of the other tasks, linguistic or non-linguistic. Christine also had a small number of errors on the pointing task; and there was no clear pattern in the errors for either of them. In the end, both subjects produced too few errors to merit further examination. It stands to reason that pointing would be easiest, because it uses only one hand, has nearby target locations, requires no hand configuration change, and is not (by definition) sequential.

According to the measures used by Sunderland and Sluman (2000) and Kimura (1993) on the Kimura box task, James had difficulty doing two correct sequences of the three movements in a row. However, by their measures, he was correct on all the movements in the first sequence, so it is not likely that his performance reflects an apraxic deficit. What is more probable is that it represents a memory or perceptual problem, because he consistently had difficulty with the first manipulandum but not with the other two. On the measures added to the task for this study, James was moderately impaired on the Kimura box. His hand configurations were slightly lax and did not consistently match the hand configurations produced by the experimenter. However, he exhibited no involuntary movements or targeting problems. By Kimura's measures, Christine performed at ceiling level. Assessed according to the measures added for this study, she produced two lax hand configurations, but they were still well-

adapted to the relevant tasks and roughly matched the experimenter's hand configurations. Also, Christine's movements from one manipulandum on the box to the next were faster than James's. He tended to hold his position at each manipulandum slightly longer.

James did not have much difficulty with toy placement task in general, although he did have more difficulty with it than with the pointing or Kimura box tasks. He showed no deficits related to hemispatial neglect or visuospatial processing. Additionally, as with the signing task, he produced no involuntary movements. On the more demanding movement sets (e.g. those that required grasp of an object and then its placement), he was more likely to produce targeting errors on toy placement. Not surprisingly, in general, the number of his errors increased with task difficulty: he had more difficulty lifting an object and placing it, compared to grasping and pushing an object, and more still compared to sliding an object along a surface. Perhaps similarly to his performance naming task, James often paused while making a movement on the toy placement task; but the source of this unclear irrespective of whether or not it is the same type of error on the two tasks. In fact, it may not be a motor deficit at all, but something related to visuospatial perception or attention. Further study would have to be carried out to address this question. Finally, it should be mentioned that the errors he made adapting the correct hand configuration for a task are almost exclusively on tasks that required both hands, and it was the left hand that could not assume the necessary hand configuration.

James showed a differential pattern of errors across the non-linguistic tasks, and also between the non-linguistic tasks and the signing task. In general, he performed better on the less complex non-linguistic tasks which had low targeting demands: those that did not require precise movements of distal effectors. The toy placement task required much more complicated movements than either of the other two non-linguistic tasks, and not surprisingly, James produced more errors on it than on the other tasks. In particular, his targeting was far more accurate in pointing than in toy placement. Furthermore, he was able to maintain fine movements and proximal distal co-ordination better in the two more difficult non-linguistic tasks (Kimura box and toy placement) than in signing. The simplest interpretation of James's differential performance on non-linguistic and signing tasks is that signing has greater fine-grained movement demands; and James was particularly impaired on fine motor control.

One of the striking differences between James's and Christine's performance was that on the Kimura box task, they had about the same rate of hand configuration

errors; however, James had a much higher rate of handshape errors than Christine on the naming task. So, on what was essentially the same measure, they performed differentially across two tasks, which reinforces the idea that what James is exhibiting is an exaggeration of a normal tendency (laxing) which is more pronounced on more demanding tasks. Furthermore, on the simplest movement task (pointing), the two subjects performed essentially identically, and at ceiling level. That said, James was uniformly slow in his movements across all the tasks, and probably in his day-to-day behaviour as well. This was the one measure on which he consistently performed differently from the control subject, irrespective of external factors, such as task difficulty.

5.6.4 Comparisons to speech dysarthria research

As an aside, James's English skills remained strong following the stroke; however, his spoken language ability was not explicitly tested for this study. It would have been interesting to compare his articulatory ability across the two modalities, particularly in light of his previously undiagnosed bilateral damage, but since he is a deaf speaker it was difficult to presume what his normal baseline of speech articulation would have been like prior to his most recent stroke.

It is likely that most hearing subjects with the same type of stroke that James had would have had minimal speech motor impairment, at most. In those who did have a speech motor impairment, it would probably present itself as imprecise articulation, slowed speech, and mild disruptions to speech resonance. Given that the dysarthria in such a case would not likely appear in isolation, the hypothetical dysarthric speaker would probably also exhibit mildly impaired co-ordination and weakness in their affected hand and arm. Perhaps James exhibited symptoms that a UUMN dysarthric speaker would also exhibit (e.g. lax hand configurations and reductions in movement displacement) if they were tested on sufficiently-demanding tasks using the hands. In other words, there may be aspects of the movement disorder that would hold true across the two modalities but have remained unidentified because subjects were differentially impaired according to task difficulty rather than according to effector group. The sign errors that James exhibited are similar to the imprecise articulation and loss of fine motor control exhibited in UUMN dysarthria.

It is possible that the symptoms that James exhibited across the tasks are related to his earlier mild contralateral infarct(s). This possibility is supported by the fact that his non-signing, in addition to his signing, movements pattern similarly to the typical

symptoms of spastic dysarthria (i.e. slowed movements, reduced movement range, and possible weakness). Additionally, he had potential indications of heightened muscle tone (reduced movement range and laxed articulation) which could indicate that his mild sign articulation deficit is a type of spastic dysarthria, and also the result of multiple infarcts. More research is required to address these competing hypotheses about the nature of UUMN dysarthria, cross-modal movement disorders, and linguistic vs. non-linguistic movement.

6 Robert: Ataxic Signer

This chapter examines the case of Robert, a signer with severe cerebellar damage (primarily to the right hemisphere) which resulted from a vascular pathology. Robert's is the first case of cerebellar damage to be reported in a sign language user, and consequently, it could have important implications for models of sign articulation and the development of therapy for Deaf stroke victims. Cerebellar damage is thought to cause severe disruptions to co-ordination and targeting, and sign and fingerspelling production have high co-ordination and targeting demands, so it is predicted that sign and fingerspelling will be affected by cerebellar damage to a greater extent than simpler limb movements. Moreover, the right cerebellar hemisphere has been identified as important for speech articulation (Marien et al., 2000; Wise et al., 1999), so it is reasonable to speculate that it might be involved in sign articulation in a similar way. Robert's case allows the same research questions to be asked for cerebellar damage that is asked of the other cases examined here: does the subject show a differential pattern of performance on the linguistic and non-linguistic tasks; and is his pattern of performance similar to what we would expect in a hearing speaker with severe cerebellar damage?

6.1 Cerebellum

6.1.1 Cerebellar function

The importance of the cerebellum for motor control has been recognized for many decades and its nature investigated in depth (Holmes, 1939). However, despite its importance in motor control, there has been less research on human cerebellar function than on the function of many other parts of the brain, probably in large part due to the availability of clinical data. First of all, damage to the cerebellum is relatively uncommon because it is not a frequent site for vascular accidents; the overwhelming majority of strokes occur in branches of the carotid artery and damage the cerebral hemispheres. Perhaps more significantly, there is no common disease affecting the cerebellum; Friedreich's ataxia is the most common, and it only affects 0.002% of the general population. By contrast, basal ganglia damage occurs as the result of both Parkinson's disease and Huntington's disease—two of the most common degenerative neurological diseases occurring in humans. Consequently, data on the function of the human cerebellum are comparatively scarce.

6.1.1.1 Research on the cerebellum and movement

Despite the infrequency of cerebellar damage in humans, there is nonetheless a reasonable body of experimental research suggesting that the cerebellum is important for processing sensory information as it pertains to movement (Jueptner et al., 1996; Ohyama et al., 2003). In order for movements to be executed normally, the nervous system must maintain a sense of where effectors are located relative to each other and relative to movement targets. The cerebellum co-ordinates components of movements, possibly by comparing motor output against both motor plans and sensory feedback and adjusting the output accordingly (Blakemore et al., 2001). It is well placed and configured for this function because it receives input (either directly or indirectly) from the cerebral cortex, the brainstem and the spinal cord. Moreover, its cortical structure is highly dense and regular, making it well-adapted to the high computational demands of online sensorimotor processing (Brodal, 1998).

Looking at motor functions of the cerebellum at a slightly higher level, researchers have uncovered various implications of its role integrating sensory and motor information. Lang & Bastian (2002) suggest that the cerebellum facilitates execution of complex movement in tasks that do not require substantial attentional resources. This is a predictable function given that the cerebellum receives sensory information directly from the brainstem and spinal cord, thereby circumventing higher level information processing structures in the neocortex. Others have suggested that the cerebellum is specifically adapted to combine simple motor synergies into complex synergies (Thach et al., 1992), which could follow from its capacity to integrate large amounts of sensory and motor information rapidly and modify motor output accordingly. Behavioural data show that cerebellar patients exhibit more variability in final finger position in rapid throwing movements (Timmann et al., 2001), which is consistent with the idea that the cerebellum is crucial for integrating sensorimotor information. It is easy to understand how disruption to cerebellar function could cause loss of co-ordination and movement accuracy, if its primary function is to process sensory information and use it to gauge movements.

6.1.1.2 Clinical findings—Ataxia

The pattern of disrupted movement typically resulting from cerebellar damage is referred to as ataxia, which comprises various components. Clinical research suggests that in patients with cerebellar ataxia, voluntary movements are slow, large, and jerky; and often disrupted by tremors (Duffy, 1995). The tremors occur during voluntary

movements of the limbs (as distinct from tremors associated with Parkinson's disease that occur when the limb is still) and are consequently referred to as intention tremors. Additionally, ataxia disrupts more basic motor functions such as posture, gait, and eye movements, rendering them unstable. At a physiological level, patients with ataxia have reduced muscle tone and hyporeflexia. The practical effects of the physiological and psychophysical aspects of ataxia are dysmetria (or spatial inaccuracy), dysrhythmia, and dysdiadochokinesia (or disruption to rapidly alternating movements).

6.1.2 Cerebellum and speech/language

6.1.2.1 Ataxic dysarthria

Broadly speaking, the symptoms of ataxic dysarthria resemble the general movement deficits of ataxia. Ataxic dysarthria is a manifestation of unco-ordinated movements and hypotonia of the speech muscles. Speech is generally perceived as slow and imprecise, with irregular variations in pitch and loudness, and a 'scanning' rhythm (Duffy, 1995). Recent clinical research has tried to characterize the condition according to more discrete, quantifiable measures. It has been proposed that ataxic dysarthria is not a unitary phenomenon, but that there are subtypes, corresponding to different lesion sites in the cerebellum (Kent et al., 1997); however, like any aspect of cerebellar damage, this is difficult to test because of the relative scarcity of clinical data.

Clinical and experimental research indicate that ataxic dysarthria affects many speech articulators at once, rather than individual articulators in isolation (Kent et al., 1997; Murdoch & Theodoros, 1998; Sheard et al., 1991), which is reflected by the inconsistencies between acoustic and perceptual measures of speech components. Subjects with ataxic dysarthria tend to receive low intelligibility ratings (Liss et al., 2000), though these do not correlate with any specific acoustic measures (Kent et al., 2000; Linebaugh & Wolfe, 1984; Sheard et al., 1991). Additionally, clinical observation of articulation suggests that speech co-ordination and timing are affected to a greater degree than strength or mobility of the speech musculature (Duffy, 1995), which is consistent with the idea of a multi-articulator impairment. However, Ackermann & Hertrich (2000) have proposed that deficits in coarticulation related to ataxic dysarthria are the result of reduced articulatory speed rather than a deficit in co-ordination of multiple articulators; but it is difficult to know how their findings relate to aspects of ataxic speech, other than coarticulation.

Despite many reports on specific symptoms of ataxic dysarthria, it is not clear which disrupted speech characteristics are most prevalent. Some acoustic and perceptual

studies have suggested that subjects with ataxic dysarthria have more variable pitch and loudness (Darley et al., 1975; Kent et al., 1979; Kent et al., 2000), which would be consistent with findings from experimental motor control research on variability in arm movements (Timmann et al., 2001). However, other research has suggested that ataxic dysarthria causes too much consistency in pitch and loudness across syllables (Kent et al., 1979); so perhaps, the absolute values of pitch and amplitude are not the most relevant measure. Alternatively, it is possible that increased and decreased variability of pitch and loudness are different versions or components of the same underlying deficit.

Ackermann et al. (1997) address similarities and differences between speech articulation movements and upper limb movements in various central motor disorders. They report that subjects with cerebellar damage show bradykinesia of lip movements in a /p/V/p/ production task. On the basis of this and earlier research suggesting that cerebellar damage only affects velocity profiles on rapid, guided movements, they conclude that lip movements behave like ballistic movements. They suggest that the cerebellum has different functions in speech as opposed to limb movements, arguing that the two types of movement are inherently different. It should be noted, however, that Ackermann et al. were only examining lip movements used to produce plosive consonants; had they looked at lip rounding in transitions from one vowel to another, they may have had a very different result, given that vowels have longer durations and rounding is a more gradual movement. By their reasoning and methodology, differences between speech and limb movements in cerebellar ataxia are as likely to be task-based as effector-based.

6.1.2.2 Cerebellum and language

Although the cerebellum is best known for its role in movement, it has non-motor functions, which have not been explored in as much detail. Small-scale studies suggest that cerebellar damage may cause disturbances to language as well as speech (Fabbro, 2000; Fiez et al., 1996; Marien et al., 2000; Petersen et al., 1989) and disturbances to neuropsychological functions such as learning and memory (Lalonde & Botez-Marquard, 2000). The traditional view that cerebellar damage causes no impairment to cognitive ability has been more or less abandoned, but the nature of the resulting cognitive or linguistic impairment is far from resolved. Because the right cerebellar hemisphere connects (indirectly) to the left cerebral hemisphere, it has been suggested that it has an important role in both language and articulatory function (Corina et al., 2003; Marien et al., 2000; Wise et al., 1999). However, there is also

research suggesting that both the left and right cerebellar hemispheres as well as the vermis are important to language function (Fabbro et al., 2000; Fiez & Petersen, 1998). In fact, there are probably insufficient data at this point to adequately address anatomical correlates of uniquely human behaviour that are internal to the cerebellum.

Ackermann & Hertrich (2000) propose that cerebellar damage impairs speech processing and perception. In particular, they report a disruption to discrimination of voiced and unvoiced consonants in subjects with cerebellar atrophy, which they attribute to the cerebellum's suggested role in perception and production of tempo (Ivry & Keele, 1989; Penhune et al., 1998). Some researchers go beyond this to talk about specifically linguistic functions of the cerebellum, beyond the level of anything sensory or motor, including components of syntax, morphology, and semantics (Fabbro, 2000). Fabbro et al. (2000) describe a small, diverse group of subjects with cerebellar lesions (N=4, one of which is a developmental case) and their resulting linguistic impairments. The researchers examined various components of language production and perception, and claim to have found correlations between language impairments and areas of damage. However, they do not argue that the deficits they describe are independent of the other motor and cognitive deficits that the subjects exhibit. Damage to the cerebellum can affect language indirectly in any number of ways, for example, by disrupting articulation (Kent et al., 2001), sensory processing (Jueptner et al., 1996), or learning (Lalonde & Botez-Marquard, 2000). In their review of neuroimaging studies of reading, Fiez & Petersen (1998) suggest that the bilateral cerebellar activity reported across studies may reflect the sensorimotor demands of the task, since some studies also found bilateral activity during non-linguistic tasks which had a sensorimotor component. As a result, lesion studies must be cautious in assigning differentiable linguistic functions to specific areas of the cerebellum, particularly on the basis of individual cases.

Most other studies of the cerebellum and language have focused on production rather than perception. Petersen et al. (1989) report right cerebellar activity in normal subjects during a lexical retrieval task, in which subjects had to generate nouns to accompany verb stimuli. Additionally, Marien et al. (2000) report a case of reduced spontaneous linguistic output and lexical retrieval deficits in an individual with right cerebellar damage, one year post-morbid. They suggest that it may be similar to frontal dynamic aphasia, and as such, reflect a disruption of cerebellar projections to the left cerebral frontal lobe. These reports, like many others on non-motor functions of the cerebellum, rely primarily on isolated cases and small subject groups, which cannot be

avoided in clinical research on human cerebellar function. The small number of clinical cases combined with the sensorimotor and cognitive demands that accompany most psycholinguistic tests of cerebellar activity means that it is too early to judge the linguistic function of the cerebellum, but the possibility should not be ruled out that cerebellar damage can impact on language as well as articulation.

6.1.3 Sign Articulation

Because damage to the cerebellum is comparatively rare, to date there has been no research on sign language and the cerebellum other than the individual case described here. However, it is worth briefly reviewing studies on other types of atypical sign articulation for the comparisons they provide. Research on atypical sign articulation has focused primarily on Parkinson's disease or cerebral right hemisphere damage. Studies on Parkinson's disease and signing suggest a general reduction in the size and speed of sign production (Brentari et al., 1995; Poizner et al., 2000). Additionally, Parkinsonian subjects show laxing of handshape and orientation (Loew et al., 1995; Poizner & Kegl, 1993), mirroring of handshape on the non-active hand (Brentari et al., 1995), reduction of signing space (Brentari & Poizner, 1994; Brentari et al., 1995), and distalization of relevant sign articulators (Brentari et al., 1995; Tyrone et al., 1999). In a case study on sign and right hemisphere damage, researchers reported that an affected signer showed problems with bimanual co-ordination, in that one hand tended to lag behind the other in two-handed signs (Loew et al., 1997; Poizner & Kegl, 1993).

Another body of research on atypical signers has examined sign articulation in young children, who are not a clinical population but are nonetheless atypical in their signing, relative to fluent adult signers. In their research on the acquisition of American Sign Language as a first language, Cheek et al. (2001) found that young children produced one-handed signs with two hands. In a related study, Meier et al. (1998) found proximalization of sign movements, such that children used a sign articulator more proximal to the body than the articulator in the target form of the sign—the opposite of the pattern seen in signers with Parkinson's disease. The significance of sign movement patterns exhibited by children in relation to the case described here will be examined in greater detail in the discussion section below.

6.2 Robert: Background

Robert is a 36 year old right-handed man who was born deaf into a hearing family. He acquired BSL at age 5 when he attended an oral day school for the deaf. After completing secondary school, he worked as a printer until his health problems began at age 28. There are no other deaf members of his family, and he communicates with his family through speech and lip-reading. His partner and his closest friends are Deaf, and their language of preference is BSL. Prior to his neurological surgery, he was an active member of the Deaf community and participated in Deaf events in his area.

Robert suffered a cerebellar infarct following surgery to correct an arteriovenous malformation, when the surgery caused extensive haemorrhaging. CT scan data reveal that the damage is greatest in the right hemisphere of the neocerebellum but extends into the medial cerebellum as well. Additionally, there is a small area of damage in the pons. His symptoms following surgery included hypotonia, postural instability, nystagmus, and unco-ordinated movements.

6.3 Deaf Stroke Project Findings

6.3.1 Neuropsychological Testing

Robert's performance on neuropsychological tests administered by the Deaf Stroke Project was completely within the normal range of performance by Deaf control subjects. He produced no errors on the line cancellation test for visuospatial neglect. On the Foreshortened Match Test from the Birmingham Object Recognition Battery (BORB) (Riddoch & Humphreys, 1993), which tests the ability to imagine an object from a different perspective and range, he scored 24 out of 25. On the Object Decision test from the BORB, which tests discrimination of real and unreal or impossible objects, he scored 24 out of 32, which despite being low is nonetheless within the normal range.

6.3.2 Clinical Observations and Sign Language Testing

Purely linguistic deficits in Robert's signing were not obvious on cursory observation, though it is possible that they were masked by his more severe movement deficits. He showed no difficulty understanding ordinary conversation, and his production deficits were largely motoric. On explicit testing by the Deaf Stroke Project, he exhibited a mild impairment on comprehension of negatives (both on manual and non-manual forms of negation); however, at the time of testing, his nystagmus was persistent and could have interfered with his performance. On all other linguistic tests, he performed within the normal range.

Robert uses a wheelchair, experiences weakness in both arms and can perform many daily tasks like feeding himself, though he finds them difficult. In early stages of recovery, he had a mild nystagmus which gradually resolved almost completely. Informal observation of his spontaneous non-linguistic movement revealed unsteady posture and head position, difficulty co-ordinating the two hands, and difficulty reaching intended targets in day-to-day movement tasks (e.g. picking up a cup, adjusting his glasses). Additionally, his limb movements are typical of ataxia, with big, irregular movements, and frequent intention tremor.

6.4 Signing Task

6.4.1 Methods: Signing

The signing task given to Robert was the copying task, described in Chapter 4. The experimenter produced individual signs in isolation, and Robert would copy the same signs. The coding scheme described in Chapter 4 was used to analyse the data. Because Robert exhibited no comprehension or visual processing difficulties on this task, and the task did not require lexical retrieval, no data had to be excluded from analysis due to processing considerations. In the few cases in which he repeated a sign, repetitions were excluded from analysis; however, if he repeated a sign in its entirety without pausing, it was counted as an error. Robert's performance on the signing task, as well as on all the other tasks, was directly compared to the performance of Graham, the control subject who is close to him in age and linguistic background. A total of 30 individual sign productions were analysed for each subject. Data are presented in the same format as in the previous chapter.

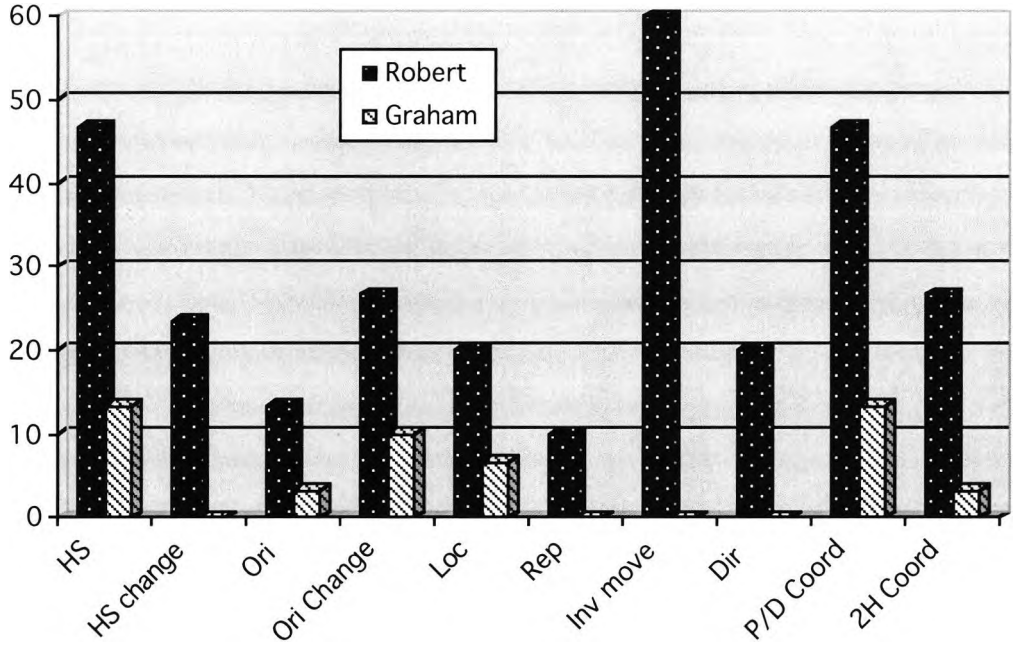
6.4.2 Results: Signing

Robert produced a large total number of errors, a large number of errors per sign, and a broad variety of errors (Figure 6.1). The most frequent was involuntary movement (60% of the 30 signs produced, or 14 signs), which was probably a manifestation of intention tremor. There was no obvious pattern in the distribution of involuntary movements in his signing—they bore no relation to the articulatory complexity of the sign. Involuntary movements were no more likely to be present on signs with (or without) internal handshape or orientation change, with locations in a particular area, or with one vs. two hands in the citation form of the sign. As such, involuntary movement was an error whose occurrence or frequency probably would not

have been influenced by controlling the stimuli for those factors. Graham, the control subject, produced no involuntary movements while signing.

Figure 6.1: Sign Copying: Robert

errors as % of 30 productions



Key: HS: handshape;
HS change: handshape change;
Ori: orientation;
Ori change: orientation change;
Loc: location;
Rep: sign repetitions;
Inv movement: involuntary movements;
Dir: direction of movement;
P/D Coord: proximal/distal co-ordination;
2H Coord: bimanual co-ordination

Figure 6.2: Image of hyperextended handshape

The next most frequent error in Robert's signing was handshape error (46.67% of 30 signs produced). By contrast, 13.33% of Graham's signs (4 signs altogether) included handshape errors. In the case of this error as well as the previous one, there was no clear pattern in the distribution across signs for either subject. The features most likely to influence distribution would probably be target handshape, sign location, or presence of handshape change in the target, and yet none of these seemed to influence whether or not a handshape error was produced. Robert's handshape errors were almost all hyperextended handshapes, that is to say, handshapes in which the selected fingers were extended out from the hand so much that they began to point dorsally along the arm (Figure 6.2). Handshape hyperextension is the opposite of handshape laxing, in that the hand is configured as far from the rest position as possible, and in Robert's case, was probably the result of hypotonia. By contrast, Graham's handshape errors were divided between laxed and hyperextended handshapes, with the majority being laxed.

Equally common in Robert's signing were errors in the co-ordination of the movements of proximal and distal articulators (46.67%, or 14 signs), for example, the elbow and the wrist. Robert had great difficulty moving articulators on the same arm in a co-ordinated manner in the signs that required it; furthermore, he added articulator movements (e.g. handshape change) to signs in which they were not required, which was also counted as a proximal/distal co-ordination error. Similarly, Robert had difficulty co-ordinating movements of the two limbs on two-handed signs, and in some cases, produced one-handed signs with both hands. Both of these were counted as a type of bimanual co-ordination error, which were present in 26.67% of his signs altogether, or 8 signs. By comparison, 13.33% of Graham's productions (4 signs) included proximal/distal co-ordination errors and 3.33% (one sign) included bimanual co-ordination errors. Robert also produced many handshape change errors (23.33%, n=7) and orientation change errors (26.67%, n=8). Errors were included in these categories either when a required change was omitted or an extraneous change was added. On the whole, Robert was more likely to add handshape or orientation changes than to omit them. Only 10% (n=3) of Graham's productions included orientation change errors: in two cases an extraneous change was added, and in one case a required change was omitted.

Additionally, Robert produced several errors in the static components of signs. The most frequent static errors were in direction (Robert: 20%; Graham: 0%); location (Robert: 20%; Graham: 6.67%); and orientation (Robert: 13.33% ; Graham: 0%). The patterns of these errors, like the others, did not seem to be influenced by the formational patterns of the signs themselves. Though Robert produced a range of sign errors, in general he had more trouble with dynamic components of signs than with static components: involuntary movement, co-ordination (proximal/distal and bimanual), handshape change, and orientation change.

Robert's errors in general bore no obvious relationship to the articulatory complexity of the signs he produced: he had as many errors on simple signs as on complex signs. Similarly, his errors did not cluster on particular signs or share an inverse relationship with other errors (Table 6.1). In other words, there were no errors that were consistently complementary or co-extensive with each other in Robert's signing. However, the large number of errors, particularly involuntary movements, makes it difficult to determine much about their distribution.

Table 6.1: Sign Error Distribution: Robert

x = Robert; o = Graham

	HS	HS change	Ori	Ori change	Loc	Rep	Inv Move	Dir	P/D Coord	2H Coord
WIN							x			
BALL	x	x			x		x		x	x
INFORM					x		x		x	
PERSON				x o				x o		
AFTERNOON	x			x	o		x			
HOUSE	x	x		x			x		x	x
THROW	x			x					x	
BITE	x						x		x	
DROP	x o			x			x		x	
FARM				x	x					
ELEPHANT	x	x		o				o	x	
NOTHING		x			x		x		x	x
WOLF			o				x			
ASK		x		x					x	
KERB				x				x	x	x
LOVELY	x o		x				x		o	
TAKE OVER	x						x			
WITH	x o	x				x	x		x	x
UNIVERSITY					x				x	
SWEEP				o		x		o		x
NAME	x	x	x				x		x	
SHELF	x	x	x					x	x	
SCREW			x				x			
TABLE	x		x					x		
SCHOOL			x							
BOWL	x				o	x	x			x
BICYCLE	o									
WEEKEND							x			
HAMMER					x		x	o	x	
TEACHER							x			x

6.5 Fingerspelling Task

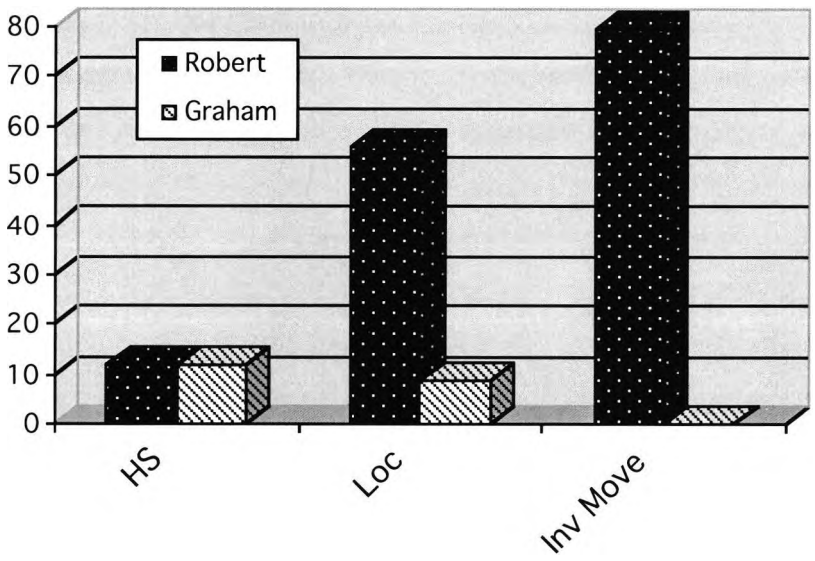
6.5.1 Methods: Fingerspelling

For the fingerspelling task, Robert was asked to copy the experimenter's production of individual fingerspelled words. On a couple of occasions, he had difficulty understanding the experimenter, so she fingerspelled the word again and then he copied it. For each letter in each fingerspelled word, the handshape, location, temporal duration, and presence of involuntary movements were coded. Only a portion of the fingerspelling data collected were analysed at this level of detail; the data to be analysed were selected for word length, variety of letters, variety of handshapes, and variety of locations. In the end, seven fingerspelled words comprising 34 letters

altogether were analysed at this level of detail. Coding categories from the signing task were modified somewhat in order to optimize information capture from BSL fingerspelling. For example, handshape change (as distinct from handshape) was not coded for since it is far less prevalent as a formational feature in BSL fingerspelling; whereas articulator location was coded in much greater detail, since BSL fingerspelling structure facilitates the use of small anatomical landmarks as reference points for location. Additionally, the durations of individual fingerspelled letters were measured, so that broad tendencies in movement speed could be identified.

Figure 6.3: Fingerspelling: Robert

errors as % of 34 letters produced



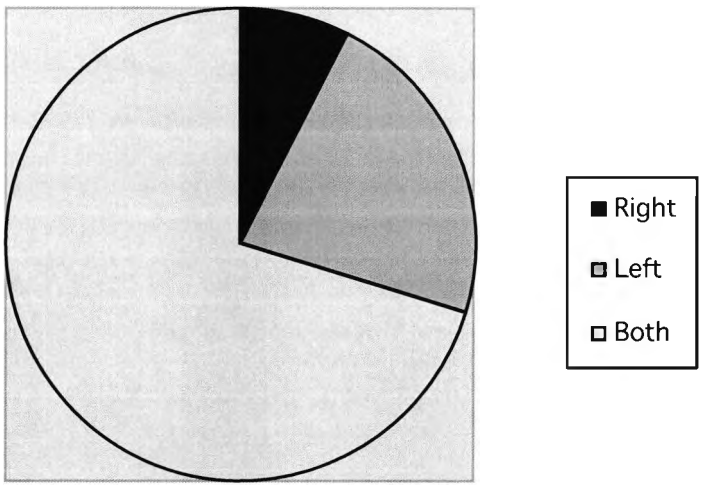
Key: HS: handshape
Loc: location
Inv Move: involuntary movement

6.5.2 Results: Fingerspelling

Involuntary movements were by far the most common of Robert's errors on the fingerspelling task (Figure 6.3). In the production of 34 fingerspelled letters, he made involuntary movements on 79.41%, or 27 letters. As in the case of his signing, all of the involuntary movements were tremors, and all of them co-occurred with voluntary movement. Beyond that, there was no clear pattern in their distribution across letters, except that the one fingerspelled letter that does not require contact between the two

hands (C) also did not elicit tremor. Additionally, in the fingerspelling task, the majority of tremors affected both hands (see Figure 6.4). Perhaps surprisingly, more tremors affected the left hand than the right hand alone, which is striking given that Robert is right-handed and that the damage to the cerebellum is worst in the right hemisphere (which primarily affects the right side of the body). Graham produced no involuntary movements on the fingerspelling task.

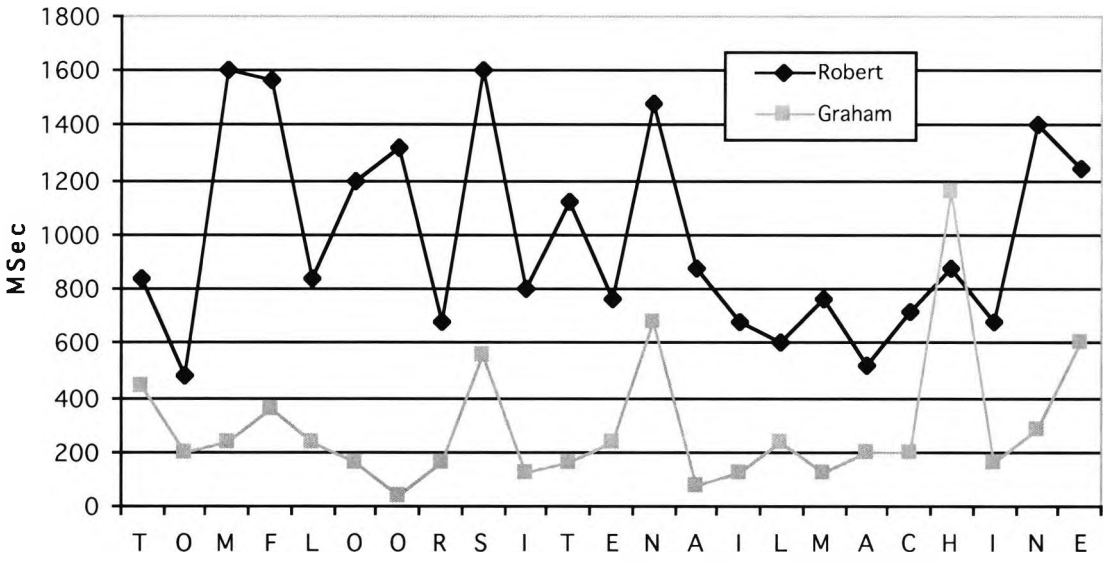
Figure 6.4: Tremor Distribution: Robert



Robert had many location errors on the fingerspelling task: 55.88%, or 19, of all the letters he produced were articulated at an incorrect location (Figure 6.3). However, his errors did not consistently skew in the same direction, nor did they reflect consistent articulatory overshoot or undershoot. Letters that were produced with an incorrect location were very nearly evenly divided according to whether they were proximal (14.71% of all productions), distal (14.71%), or medial (20.59%) to the target location. Graham produced only three location errors, of which, two were proximal, and one was distal.

Robert and Graham did not pattern notably differently in their production of handshape errors on the fingerspelling task. They both produced 4 handshape errors (11.76% of total productions), and both of them produced lax but not hyperextended handshapes as errors, which is in contrast to Robert's performance on the signing task. To differentiate the two subjects any further on this particular parameter for fingerspelling would require a more fine-grained data collection technique, which would allow more detailed measurements of hand configuration.

Figure 6.5: Fingerspelling Durations: Robert



Individual fingerspelled letters had long durations in Robert's production of fingerspelling; in fact, there was very little overlap in the durations of his productions and Graham's (Figure 6.5). It should be noted, however, that the durations of individual letters were extremely varied from one production to another, and that there was no clear source for this variability in the fingerspelling stimuli. The durations of the first letter in a word were typically the longest for both subjects, but beyond that there was no obvious pattern in the durations of individual letters that applied to both subjects. For Graham, fingerspelling durations tended to be related to the proximity of the articulatory targets: when two letters' locations on the hand were close to each other, the duration of the second letter was usually relatively short. By contrast, Robert sometimes took a long time to move from one articulatory location to another, even when those locations were close to each other. Strikingly, sometimes even in the case of a letter that was repeated (i.e. the O in F-L-O-O-R), the durations of the two productions of it were quite long. Beyond these tendencies for the two subjects, there were no inherent characteristics of fingerspelled letters (handshape, location, etc.) that affected their durations.

6.6 Non-linguistic Tasks

6.6.1 Pointing

6.6.1.1 Methods: Pointing

Subjects were asked to point to one of two illustrations in response to a signed utterance produced by the experimenter. The response sheet with two illustrations was placed on a flat surface directly in front of subjects at a comfortable distance. Incorrect responses and hesitations were excluded from analysis, and neither of the subjects responded to the same stimulus twice. A total of 28 responses on the pointing task were analysed for each subject; and pointing was coded according to the scheme described in Chapter 4.

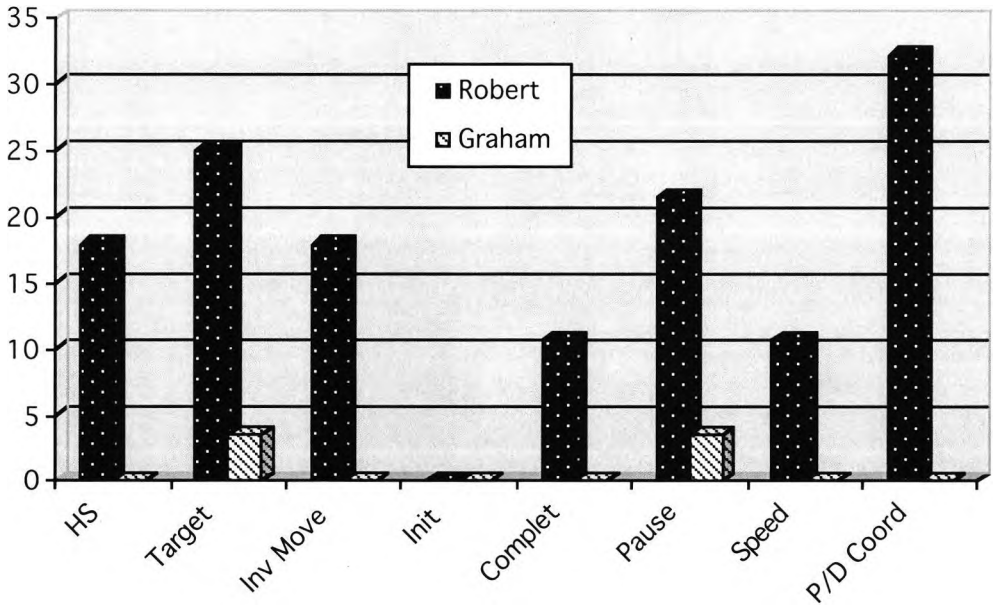
6.6.1.2 Results: Pointing

Robert produced more proximal/distal co-ordination errors than any other error type on the pointing task: 32.14% of the total number of pointing movements, or 9 errors altogether (Figure 6.6). Because all the stimuli on the task were effectively the same (target distances varied only minimally from one item on the task to another), there was no discernible pattern in the distribution of the errors. Unlike signing, pointing requires no change in hand configuration from one production to another. Nonetheless, Robert's proximal/distal co-ordination errors were most often instances in which he moved his hand toward the target, hesitated, configured his hand for a pointing gesture, and then continued moving to the target. He formed the hand configuration again for almost every individual point rather than maintaining a constant hand configuration, and consequently had difficulty co-ordinating the movements of multiple effectors at once. The remaining co-ordination errors were instances in which he produced erratic hand or arm configuration changes while moving toward the target.

Robert's next most common error was targeting, which was present in 25% (or 7) of his pointing movements. He had difficulty accurately bringing his hand to the location of the illustration in front of him. In many cases, he overshot the target and moved his hand too far. Other times, he divided the individual movements into two parts by moving his hand to the sheet of paper and then sliding it the rest of the way to the target. Graham produced almost no errors on the pointing task: one pause with no apparent non-motoric motivation and one targeting error. With such small numbers, it is impossible to say anything definitive about patterns in his pointing errors, with respect to the stimuli or otherwise.

Figure 6.6: Pointing Task: Robert

errors as % of 28 movements



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

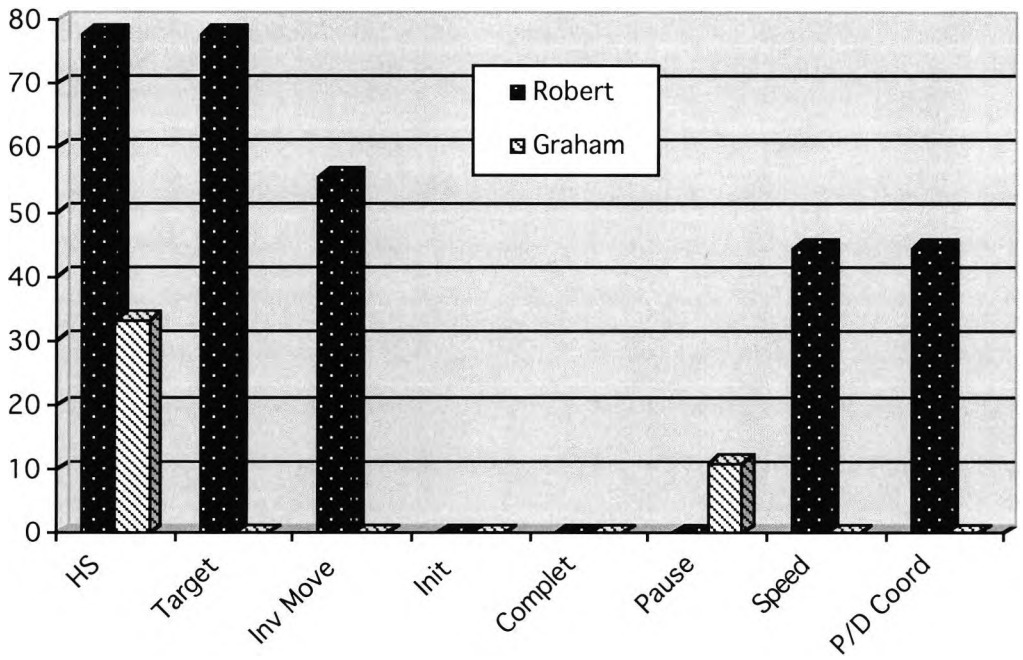
6.6.2 Kimura box

6.6.2.1 Methods: Kimura box

The Kimura box data were analysed according to the criteria set by Sunderland & Sluman (2000) with additional coding for targeting, hesitation, tremor, speed of execution and accuracy of hand configuration. Both subjects performed the specified sequence of movements three times, yielding nine individual movements per subject.

Figure 6.7: Kimura Box: Robert

errors as % of 9 movements



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

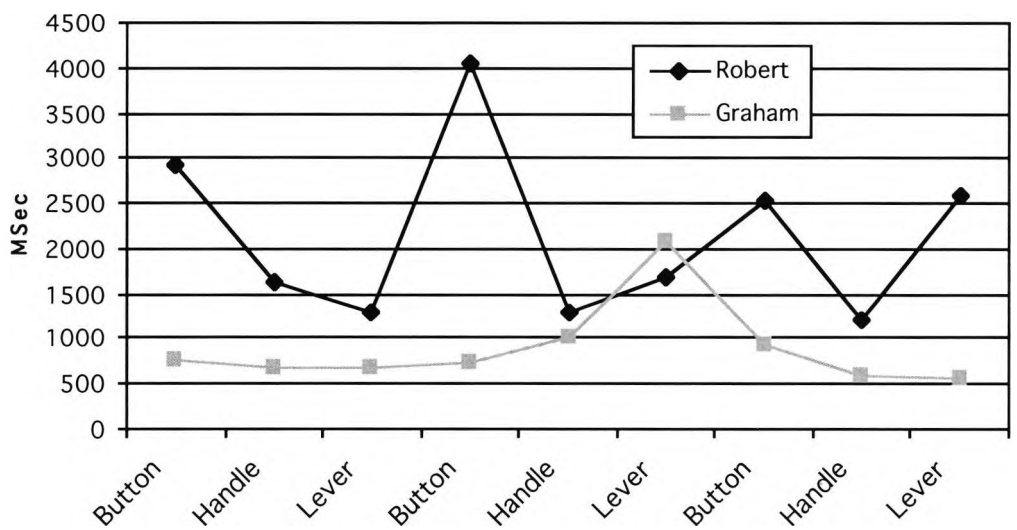
6.6.2.2 Results: Kimura box

As with most tasks, Robert and Graham did not pattern similarly in their distribution of errors on the Kimura box task, and Graham had few errors on the task in general. Robert had a very high rate of hand configuration and targeting errors on the movements he produced for this task: 77.78% of both types of errors (Figure 6.7). By necessity, there was a lot of overlap between these two error types, but they were not completely co-extensive (Table 6.2). There were individual cases of hand configuration errors occurring in the absence of targeting errors and vice versa.

Table 6.2: Distribution of Robert's Kimura Box Errors

Manipulandum	HS	Target	Inv Move	Init	Complet	Pause	Speed	P/D Coord
Button	X	X	X					
Handle	X	X						
Lever	X	X	X				X	X
Button		X	X				X	X
Handle	X	X					X	X
Lever	X	X	X					
Button			X				X	
Handle	X							
Lever	X	X						X

Figure 6.8: Kimura Box Timing: Robert



The distribution of Robert's errors was partially influenced by which manipulandum he was acting on. While almost all of the movements had a hand configuration and targeting error, the two cases in which there was not a hand configuration error was on the button, which is the first manipulandum on the box (see Chapter 4). Additionally, while Robert had no tremors when moving toward the handle, he had a tremor in every movement to the button. Graham had very few errors, so it is difficult to identify a pattern in those he did produce; however, it should be noted that he had no errors when moving to the handle. (The distribution of errors on the Kimura box task and its broader implications will be discussed in greater detail in Chapters 9 and 11).

Robert's movements on the Kimura box task were much slower than Graham's—so much so that there was very little overlap in the durations of their individual movements (Figure 6.8). Additionally, there was more variability in the durations of Robert's movements than in the durations of Graham's movements. Only one of Graham's movements took an exceptionally long time relative to the others, and it was probably an aberration. In fact, Graham's movement durations varied so little that it is difficult to identify any kind of pattern in them, in relation to specific manipulanda or otherwise.

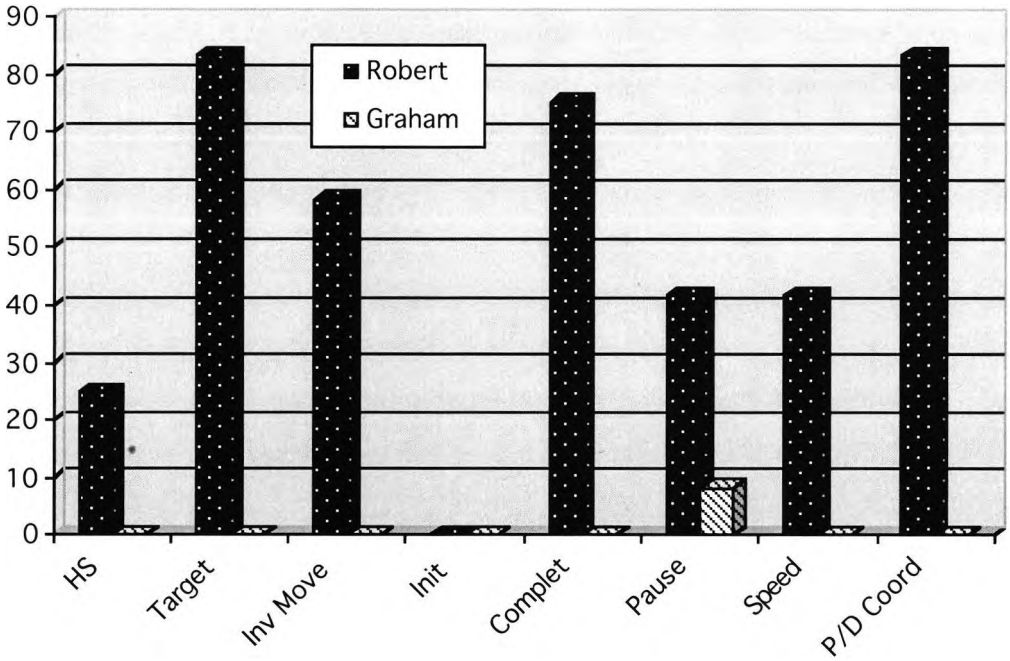
6.6.3 Reach & grasp

6.6.3.1 Methods: Reach & grasp

For the reach and grasp task, three cylinders of different sizes were placed on a table at a comfortable distance in front of subjects. (Cylinder dimensions are listed in Chapter 4.) Subjects were asked to grasp the cylinders individually, move them approximately 25 cm forward, set them down, then move them back to their original locations. In total, both subjects made 12 reach and grasp movements, which were coded for the same parameters as the other non-linguistic tasks, as described in Chapter 4. Timing of individual movements was not measured for this task, since the beginning and ending locations of the movements could not be tightly controlled from one testing session to another.

Figure 6.9: Reach and Grasp: Robert

errors as % of 12 movements



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

6.6.3.2 Results: Reach & grasp

Robert had a large proportion of targeting and co-ordination errors—83.33% of all movements, in both cases—and consequently great difficulty completing individual parts of the task (Figure 6.9). As a result of these and other errors, there were completion errors on 75% of his movements. Also, his hand configurations (errors on 25% of movements) were often not well-adapted to the size and shape of the cylinders, which made completing the grasp difficult. Additionally, he produced many involuntary movements when performing the reach and grasp task (errors on 58.33%, or 7 out of 12 trials). While he was given no explicit instuction on which hand(s) to use, Robert repeatedly used both hands for individual trials on the task. Graham had only one error on the task, which was a pause that had no obvious non-motoric motivation.

6.7 Discussion

6.7.1 Task-by-task analysis

Signing

Broadly speaking, Robert's signing is oversized and unco-ordinated. Additionally, his signing, like his limb movement more generally, is slow and often disrupted by intention tremor. This overall pattern is consistent with the general motor symptoms of ataxia and the characteristics of ataxic dysarthria, which are both characterized by slow, exaggerated movements, and intention tremor superimposed on spontaneous movements of the limbs and the speech articulators. Robert's movements are proximalized on some signs, in other words, he produces a sign using articulators proximal to those normally used for its production (e.g. the wrist instead of the base of the fingers). Similarly, he also produces some signs in locations high and far away from the body, relative to the sign's citation form or how the control signer produces it. In this context, it is important to make a distinction between movement size, location distance, and selected articulator(s): it is not possible to have a small movement to a distant location, so in conditions like Parkinson's disease it is easy to equate movement size with location distance. By contrast, it is possible to have a very large movement to a nearby location, depending on the movement trajectory; and in fact, these are the type of movements Robert often produces. He has an overall pattern of enlarged signing, but it can take a variety of forms: large movements, distant sign locations, and hyperextended articulators.

Robert's inco-ordination takes a variety of forms. In particular, he has difficulty co-ordinating his hands to produce two-handed signs. He is not always able to make his hands begin moving at the same time, come to the same place, or produce the same movement, when required to do so. He also has difficulty co-ordinating the movements of independent articulators on just one limb: both correctly timing separate movements relative to each other and suppressing involuntary movements that emerge during voluntary movement. For example, the sign BITE requires a downward movement of the arm at the same time that the fingers close to make a fist. What Robert does instead is produce the two movements serially, by moving his arm down first, then pausing, then closing his fingers.

Regarding Robert's tendency to produce one-handed signs with two hands without obvious intent to place linguistic emphasis (and bearing in mind that the signs were produced as part of a repetition task and not likely to require emphasis), the only finding similar to this in the adult sign literature suggests that signers with Parkinson's disease occasionally mirror the handshape from a one-handed sign on the non-active hand (Brentari et al., 1995). Without further investigation, it is difficult to speculate on this, since neither the subjects nor the sign productions are very numerous in that study or this one. That said, the signers with Parkinson's disease are never described as fully producing a one-handed sign with both hands; however, that is precisely what Robert did. Similarly, normal Deaf children in the early stages of acquiring individual signs produce two-handed forms of one-handed signs (Cheek et al., 2001). It may be that the children studied by Cheek et al. (2001) were using descending brainstem tracts thought to enable simple, symmetrical, two-handed movements (Wiesendanger et al., 1996). Their sample included children ranging in age from 5 to 17 months, at which stage the connections between the cerebellum and cerebral cortex as well as the corticospinal tract itself, both of which are necessary for precise, co-ordinated movements, would not yet be fully developed (Brodal, 1998).

The measure of bimanual co-ordination was intended to capture subjects' ability or inability to use two hands in a concerted fashion to execute a movement, or in the case of the signing task, a sign. Because it is a relatively broad measure, two subjects can pattern differently on it, despite both showing a deficit. In fact, Robert had a variety of errors that were all classified as bimanual errors, because they were all related to the timing or placement of the two hands relative to each other. In the production of two-handed signs, Robert had difficulty synchronizing movements of the two hands, moving them symmetrically in space, and bringing the hands together in space.

Fingerspelling

The fingerspelling task was particularly challenging for Robert, judging by his own report as well as his performance. He produced a very large number of errors relative to the total number of fingerspelled letters, which may be a reflection of task difficulty. In particular, he had a very high number of involuntary movements. Additionally, Robert produced atypical movement trajectories in going from one fingerspelled letter to another; unfortunately, it was not possible to capture these with any consistency from videotaped data. Finally, Robert produced individual fingerspelled letters very slowly, though, as in the case of signing, not consistently so. Despite his overall difficulty with the task, Robert produced far fewer handshape errors on the fingerspelling task than on the signing or reach and grasp tasks. This may be because BSL fingerspelling does not require as broad a range of handshapes or as many transitions from one handshape to another.

Robert's productions of individual fingerspelled letters were very long, so much so that there was very little overlap in the durations of his productions and Graham's, which parallels the finding of overall articulatory bradykinesia in speech production in cerebellar atrophy (Hertrich & Ackermann, 1999). However, the durations of individual letters were extremely varied from one production to another, and there was no clear source for this variability in the fingerspelling stimuli. In producing a fingerspelled letter, Robert often took a very indirect path to get to the letter's location on the opposite hand. It is apparent that he had a rough sense of where the articulatory target was, because he did not produce one letter in place of another, but he nonetheless had a difficult time getting to the target. This is consistent with the suggestion by Ackermann & Hertrich (2000) that ataxic speakers preserve phonemic vowel length if not voice onset time, and that by extension their articulatory deficit is biomechanical rather than phonological in nature.

With respect to movement trajectories, as well as more broadly, it is informative to look at the transitions from one fingerspelled letter to another, despite the fact that fingerspelling is typically described in terms of just the letters themselves and not the transitions between them. A casual observation made during data analysis was that in transitioning from one fingerspelled letter to another, Robert frequently moved his hands apart, formed the appropriate handshape, and moved his hands together again to form the letter. Unfortunately, this phenomenon was difficult to capture in the absence of clear reference points for describing transitions between letters. Many researchers

have suggested that the transitions between fingerspelled letters are actually more important than the letters themselves in language processing (Akamatsu, 1983; Battison, 1978; Wilcox, 1992); however, the direct mapping between written letters and fingerspelled forms almost certainly facilitates using those forms as the relevant descriptors of the structure of fingerspelling. In future studies focusing on fingerspelling and human movement, it would be productive to code the paths of the individual articulators frame by frame (see Tyrone et al., 1999), in conjunction with a 3D motion capture technique.

Pointing

On the pointing task, Robert showed occasional intention tremor and consistently slow movements relative to the control signer, in the same way as on the linguistic tasks described above. However, Robert's pattern of errors was somewhat different on the pointing task in that he produced far fewer involuntary movements and more co-ordination and targeting errors proportionally. (Targeting errors were not coded as such on the signing or the fingerspelling task, though they are essentially the same as location errors in signing or fingerspelling—both measures describe spatial inaccuracy.)

In some cases, Robert divided individual movements into two parts by moving his hand to the sheet of paper and then sliding it the rest of the way to the target. This is similar to a pattern described by Brooks (1986), in which cerebellar patients break movements down into smaller components and produce the components individually. While Robert had proportionally fewer location errors in signing than targeting errors in pointing, the pattern of breaking movements down into component parts held true across the two tasks and may underlie many of Robert's other types of sign errors, including bimanual and proximal/distal co-ordination errors.

Kimura box

The Kimura box test was originally designed to assess apraxic subjects' ability to correctly manipulate objects according to object shape and size, and according to imitation (Kimura, 1993). Even measured by these standards, without any consideration of laxing or overextension of hand configuration, both subjects made many hand configuration errors. For example, both subjects used a thumb rather than an extended index finger (as the experimenter had demonstrated) to handle the first manipulandum on the box. There were fewer trials on this task than on any of the others; however, Robert had the highest proportion of handshape errors on this task. That said, though, so

did the control subject, so it is difficult to interpret Robert's error pattern for handshape errors. Robert also had great difficulty accurately targeting the location of the manipulanda when moving his hand toward them. Perhaps as a result of targeting difficulty and/or the spatial accuracy demands of the task, he had proportionally more tremors on the Kimura box task than on the pointing task.

As with all tasks, Robert's movements on the Kimura box task were much slower than Graham's and much more variable. Graham's movement durations on the Kimura box task did not vary much at all. However, although Robert's movements varied a lot in duration, he consistently took longer in moving to the first manipulandum, which makes sense given that subjects almost invariably began the movement to the first manipulandum from farther away. So Robert's was a predictable movement pattern, albeit one that was exaggerated.

Reach and grasp

On the reach and grasp task, Robert's main difficulties were with targeting and proximal/distal co-ordination. As a consequence of these errors, Robert had a high proportion of completion errors because the task required a hand configuration change co-ordinated with an arm movement for task completion (i.e. to grasp and set down the cylinder), which the pointing and Kimura box tasks did not require. Also, his hand configurations were often not well-adapted to the size and shape of the cylinders, which made completing the grasp difficult. As with the Kimura box, he produced many tremors when performing reach and grasp movements. Additionally, Robert repeatedly used both hands on the task, which could be a mechanism for dealing with tremor or task difficulty; however, this pattern is also consistent with the pattern from his signing, which seems involuntary. Whether the use of two hands for normally one-handed tasks by individuals with ataxia is common and/or voluntary is a question that should be explored in greater detail.

6.7.2 Comparison of linguistic & non-linguistic tasks

Thach et al. (1992) propose that the role of the cerebellum in motor control is to allow the combination of simple motor synergies into more complex synergies. The movement deficits that Robert exhibited on all the tasks, and his differential pattern of impairment across tasks, certainly support this idea. Robert had particular difficulty with co-ordination, which by definition relies heavily on the combination of motor synergies. Additionally, Robert was differentially impaired on targeted, sequential

movement tasks, such as reach and grasp, the Kimura box and fingerspelling production, which require combining motor synergies in rapid sequence. This could be related to the cerebellum's proposed role in integrating sensory and motor information for movement planning. In addition, Robert exhibited more co-ordination errors specifically on tasks that required integrated movements of proximal and distal effectors: signing, Kimura box, and reach and grasp. This, too, could be explained in terms of sensorimotor integration, which allows movements of independent effectors to be co-ordinated spatially as well as temporally.

Robert's performance on the linguistic and non-linguistic tasks was consistent with the symptoms of both ataxia and ataxic dysarthria: he showed considerable variability in speed and accuracy from one movement to another; he had an overall slowing of his movements; his movements were very unco-ordinated; and he exhibited a lot of intention tremor during voluntary movement. Where past research has suggested a distinction between ataxic speech and limb movements (Ackermann & Hertrich, 2000), namely in target undershoot and overshoot, Robert did not show a consistent pattern, which suggests that earlier findings may have been influenced as much by the task as by the particular effectors. Moreover, many of the movement patterns exhibited by Robert (e.g. prevalence of tremor and hand configuration errors) seem to be task-dependent, which suggests that there is more at play than simply which effectors are selected for a movement. Consequently, more attention should be paid to the demands of the task itself in experiments designed to address the nature of complex movements across modalities. To give a specific illustration, Robert had proportionally more hand configuration errors in signing and on the Kimura box task, probably because they require highly differentiated hand configurations and multiple hand configuration changes, while other tasks (e.g. pointing) do not. This is the type of distinction that would be useful to know in designing kinematic studies of speech and limb movements, because it helps researchers determine which movement patterns are likely to be effector-specific.

6.7.3 Comparison to past sign research

With respect to sign articulation, Robert provides an interesting comparison to past research on Parkinson's disease, right hemisphere damage, and acquisition of sign phonology. First and foremost, his case illustrates that disruptions to sign articulation can take a variety of forms, in the same way as in spoken language dysarthria. The form of either sign or spoken language dysarthria is going to be determined primarily by the

fundamental nature of the particular movement disorder that causes it and its effects on everything from muscle tone to motor programming. Additionally, apparent similarities between Robert's co-ordination deficits and those of a signer with right hemisphere damage studied by Poizner and Kegl (1993) raise interesting questions both for motor function for sign language and neural correlates of motor control more broadly. That subject and Robert both exhibited bimanual co-ordination deficits in signing. Sign articulation may be one of a limited set of movement functions in which the two unrelated co-ordination deficits, ataxia and hemiparesis, can take on similar surface features, and as such, it could serve to highlight the similarities and differences of the two deficits.

Proximalization of sign articulators is a phenomenon that, prior to the current study, had only been reported in children (Meier et al., 1998), and in naive signers (Mirus et al., 2001). It is not entirely clear what the significance of movement proximalization is, however, Robert's signing was similar to that of young children in this and other respects. A related study on sign language acquisition showed that children produced one-handed signs with two hands (Cheek et al., 2001), which is consistent with Robert's pattern of errors. Although articulatory mirroring has been reported in signers with Parkinson's disease (Brentari et al., 1995) children's two-handed versions of one-handed signs are produced at full-size with both hands, as were Robert's; whereas with mirroring in Parkinson's disease, signers produced a very reduced form on the non-active hand, often just a handshape formation.

There is no obvious speech equivalent to the production of one-handed signs with two hands, because speech articulators are not independently controlled on opposite sides of the body's midline. Consequently, there is no parallel to look for in the development of children's speech. However, in addition to sign movements, infants also perform reaching movements with both hands before the age of 6 months. They do not develop deliberate unimanual reaching until between 6 and 8 months of age, at about the same time they develop postural control for sitting (Rochat, 1992). At around the same time, infants' targeting accuracy improves as well (Fallang et al., 2000). It has been suggested that before infants have stable posture, they preferentially reach with two hands in order to maintain balance, which makes Robert's pattern of errors interesting, in light of the fact that he has postural instability. It would be useful to see how broadly this tendency applies in ataxia: are there other tasks in which it is likely to emerge; and, is preferential use of bimanual movement related to posture or to some other factor? Performing one-handed tasks with two hands could be correlated with

specific types of tasks or with degree of task difficulty. It may be that bimanual movements are used when the required movements are complex and rapid, and rely heavily on sensorimotor integration, in much the same way that young or physically inexperienced individuals catch and throw with two hands.

Looked at from a different point of view, performing a task with two hands instead of one could also be a deliberate strategy that subjects employ when they are allowed to take whatever approach to a task they choose. Irrespective of the cause of the behaviour, it may be the way that ataxic subjects would consistently go about grasping an object, if researchers gave them no advance instructions on which hand(s) to use. If such were the case, the implications for all of human motor control research would be enormous, because it would suggest that most experimental designs used with ataxic subjects depend on an unnatural or inherently difficult behaviour.

6.7.4 Sign vs. speech in ataxic dysarthria

Ataxic dysarthria in spoken language is associated with disruptions to speech co-ordination and timing (Duffy, 1995), and irregular variability in phonation and respiration (Hertrich et al., 1998; Kent et al., 1997). Many have suggested that ataxic dysarthria impacts multiple groups of speech articulators at once, rather than specific sets of articulators in isolation (Kent et al., 1997; Murdoch & Theodoros, 1998; Sheard et al., 1991). However, Ackermann & Hertrich (2000) suggest that coarticulatory deficits in ataxic dysarthria are not caused by a problem with temporal co-ordination of articulatory movements, but by an overall slowing of movements, causing articulatory undershoot and perseverative coarticulation due to biomechanical factors. This is not consistent with the pattern in Robert's signing. He had great difficulty with co-ordination of articulatory gestures in particular. There was no clear way to systematically measure the durations of the internal components of signs; however, a few patterns are worth mentioning briefly. In general, Robert had difficulty getting the right speed ratios on a movement: some individual, sign-internal movements were relatively quick, while entire signs had long durations. Contrary to what was suggested by Ackermann & Hertrich (2000), he did not exhibit uniformly slow signing, but rather a great deal of variation in speed from one movement to another and from one sign to another.

Robert's signing pattern was more consistent with what was reported by Kent et al. (1997): that ataxic dysarthric subjects show variability in repetition rates and durations of individual syllables and pauses. Consequently, Robert patterns similarly to

what has been reported for hearing subjects in the dysarthria literature, but there is not complete agreement in the literature on the nature of ataxic dysarthria.

It has been proposed that ataxic dysarthria causes articulatory undershoot (Ackermann et al., 1997; Hertrich & Ackermann, 1999), even though limb ataxia is widely characterized as causing target overshoot (Brooks, 1986). In the fingerspelling task, Robert did not consistently overshoot or undershoot articulatory targets, but rather did both in roughly equal numbers. It is not clear what the full implications of this are for the physical structure of spoken vs. signed language or articulatory vs. non-articulatory movement, but it merits further investigation.

6.7.5 Articulatory targeting

According to Brooks (1986), intention tremor is a manifestation of the breakdown of co-contractions of opposing muscles that occurs when an individual moves toward an intended position. In Robert's case, intention tremor seems to occur when he reaches or gets close to a target. However, there is tremendous variation from one task to another, with fingerspelling showing the most disruption. This suggests that the complexity or sequentiality of the movements, or perhaps the existence of external movement targets, contributes to the prevalence of intention tremor. Brooks' description implies that signing should be more affected by tremor than speech, given that speech articulators, unlike sign articulators, are not configured in agonist-antagonist arrangements. However, there are still laryngeal and respiratory tremors in speech due to ataxic dysarthria, so ataxic tremors cannot result solely from antagonist muscle firing. To complicate matters further, intention tremor seems to be more prevalent in limb movements (signing or non-signing) than in speech (Duffy, 1995), so some sort of biomechanical factors may well be influencing it, though it is not clear what those factors are.

Assuming that intention tremors are caused or at least modified by the existence of movement targets, this raises interesting questions for sign language research: What are "the targets" in signs, and how are they framed by the nervous system? Clearly, a signer must have some articulatory target (i.e. some intended configuration and location of the articulators) when producing a sign, irrespective of that sign's physical form, and irrespective of whether or not there is an external reference point (such as a location on the body, e.g. the nose) for locating the sign. Yet it seems that the presence of a physical reference point on the body or in space influences how movements are executed in the case of ataxia, and possibly in the case of other movement disorders as well. So to some

extent, sign movements are organized in terms of the nature of the articulatory target, and whether or not it depends on something other than proprioceptive feedback from the articulators. But how are proprioceptive, visual and other types of feedback integrated to formulate representations of the structure of signs? The answers to these questions are at the core of the articulatory basis of signed language.

7 Joseph: Signer with PSP

This chapter examines the case of Joseph, a Deaf signer who developed progressive supranuclear palsy (PSP), a hypokinetic disorder similar to Parkinson's disease. His is the first case of PSP to be reported in a sign language user, so it is worth documenting for future reference and possible value in diagnoses. Because there have been no other known cases, Joseph may exhibit signing patterns that have yet to be reported elsewhere. Additionally, his case can be compared to previously reported cases of Parkinson's disease (PD) in Deaf sign language users, because PSP and PD are very similar but tend to result in different forms of dysarthria in hearing subjects. Moreover, dysarthria is one of the most prominent symptoms of PSP in hearing subjects, so it is worth asking whether it is similarly prominent in Deaf signers with the same disorder.

7.1 PSP

Progressive supranuclear palsy (PSP) is a degenerative disease similar to Parkinson's disease, but far less frequently-occurring. Like Parkinson's disease, PSP causes cell death in the substantia nigra pars compacta. However, unlike Parkinson's disease, it also causes cell death in the substantia nigra pars reticulata, thereby further disrupting function throughout the telencephalic basal ganglia. Additionally, PSP causes atrophy, gliosis, and neurofibrillary tangles in the brainstem, basal ganglia, and cerebellar nuclei (although not in the cerebellar cortex) (Metter & Hanson, 1991). Because of its similarity to Parkinson's disease and its comparative rarity, it can be difficult to establish a definitive diagnosis of PSP prior to autopsy. There are, however, clinical differences between the two diseases that are likely to appear on standard medical examination.

Many of the motor symptoms of PSP are similar to those of Parkinson's disease: patients often exhibit bradykinesia, rigidity, facial masking, and dysarthria. Individuals with PSP do not exhibit the prominent Parkinsonian symptom of resting tremor, however; and they tend to have extended rather than stooped posture, with the effect that they fall backward rather than forward. What is likely to be the most distinctive symptom of PSP is ophthalmoplegia, or loss of eye movement, which usually begins as vertical gaze disruption and worsens to affect all eye movement. Finally, while PSP and Parkinson's disease can both cause hypokinetic dysarthria, the dysarthria is typically a more prominent symptom in PSP than in Parkinson's disease, and the form of the dysarthria itself is slightly different (Lu et al., 1992; Theodoros & Murdoch, 1998b).

7.1.1 PSP and dysarthria

Dysarthria resulting from PSP is frequently described simply as a form of hypokinetic dysarthria (Theodoros & Murdoch, 1998b); however, when multiple cases are compared, it becomes clear that PSP dysarthria is actually more varied, which is consistent with the fact that PSP affects multiple neural systems. PSP speech has been judged by trained speech and language therapists to resemble not only hypokinetic dysarthria but also spastic and ataxic dysarthria to differing degrees (Muller et al., 2001), possibly depending on the distribution of neural damage in the individual cases. The relationship between dysarthria subtypes and distribution of damage is a question that merits further research, bearing in mind that assessing the detailed anatomy of a human brainstem disorder is very difficult except on autopsy.

A prevalent symptom of PSP dysarthria is palilalia (Metter & Hanson, 1991), or the tendency to repeat entire words without pausing, as distinct from word-internal hesitations or repetitions in the case of stuttering, and in the absence of a psychiatric or behavioural disorder, such as schizophrenia. In addition to palilalia, subjects with PSP exhibit imprecise articulation, reduced intensity, low pitch, reduced pitch range, hoarse voice quality, hypernasality, and slow speech rate (Hartman & Abbs, 1988; Lu et al., 1992; Metter & Hanson, 1991). Apart from the more complex phenomenon of palilalia, the last two of these characteristics (and possibly also the first) are what most clearly distinguish dysarthria in PSP and in Parkinson's disease. Subjects with PSP, like other dysarthric patients, tend to have a slow speech rate; whereas subjects with Parkinson's disease are unique among all dysarthric subjects in that they exhibit increased speech rate (Metter & Hanson, 1991; Theodoros & Murdoch, 1998b). Additionally, while individuals with Parkinson's disease may exhibit imprecise articulation, it is not usually a prominent symptom as it typically is in PSP (Muller et al., 2001). Metter & Hanson (1991) also suggest that PSP is more likely than Parkinson's disease to affect the timing of speech; they propose that the speech deficits in PSP can be attributed to a broader impairment in co-ordinating the movements of independent articulators relative to each other. Finally, in general terms, dysarthria is more likely to be a presenting symptom or to appear at all in PSP than in Parkinson's disease (Lu et al., 1992; Muller et al., 2001).

7.1.2 PSP vs. PD and sign articulation

The nearest equivalent to research on PSP and sign articulation are the studies on Parkinson's disease, American Sign Language (ASL), and ASL fingerspelling. Although Joseph does not have Parkinson's disease, he does exhibit hypokinetic

symptoms, so a brief review of the research on PD and sign language is useful in part for the contrast it will provide to Joseph's case. Several studies were conducted on American Deaf subjects with Parkinson's disease, which yielded a range of findings. First, Parkinson's disease was found to cause a reduction in the size of signing space (Brentari et al., 1995; Loew et al., 1995; Poizner & Kegl, 1993). Signing space is the area in front of the signer in which the hands move to produce the signs of a language. While some signs are fixed (albeit loosely) to reference points on the body, like the chin, for example, others are articulated in the neutral space in front of the body. In Parkinson's disease, the signing space becomes smaller because individuals' movement patterns change so that they use less of that space. Also, articulatory targets that are referenced to locations on the body become lowered.

Similarly, Parkinson's disease causes distalization of the articulators used to produce ASL signs (Brentari & Poizner, 1994; Poizner & Kegl, 1993) and fingerspelling (Tyrone et al., 1999). In other words, instead of moving the articulators typically associated with a given sign or fingerspelled letter, subjects with Parkinson's disease will use articulators that are more distal to the center of the body. For example, if the form of a sign requires someone to move their wrist, the signer with Parkinson's disease may instead move their fingers. Moreover, the articulators that are chosen are often laxed relative to their typical configurations; instead of being fully extended, an articulator may be only somewhat extended, and closer to the position it would tend toward at rest. This results in the sign parameters of handshape and orientation taking a more laxed form (Brentari et al., 1995; Loew et al., 1995).

Studies on Parkinson's disease and signing have also found disruptions to multi-articulator co-ordination in particular. First, signers with Parkinson's disease were reported to segment movements that were normally simultaneous and produce them sequentially, both in signing and in fingerspelling (Brentari et al., 1995; Tyrone et al., 1999). Additionally, in one-handed signs, and less frequently in fingerspelling, subjects with Parkinson's disease produced reduced forms of signs or letters on the non-active hand as well as on the active hand (Brentari et al., 1995; Tyrone et al., 1999). Producing a two-handed form of a one-handed sign is a common means of placing semantic emphasis, but the two-handed signs produced by signers with Parkinson's disease occurred in the absence of any apparent attempt to emphasize a sign.

If sign articulation in PSP patterns similarly to speech articulation in PSP, then on the basis of research on PSP dysarthria and research on Parkinson's disease and signing, one could predict a few of the probable characteristics of PSP sign dysarthria.

First, PSP might be likely to cause slow signing, a tendency that was evident from the data on Parkinson's disease (Tyrone et al., 1999), and analogous to slow speech production in PSP dysarthria (Lu et al., 1992; Metter & Hanson, 1991). Additionally, PSP might be likely to result in impairments in the co-ordination of independent sign articulators, or what Metter & Hanson (1991) refer to as deficits of timing. One might also predict that signers with PSP would repeat entire signs, since this is one of the typical differences between PSP and PD dysarthria in hearing people. Metter & Hanson (1991) also describe imprecise articulation as a characteristic whose prominence differentiates PSP and Parkinsonian dysarthria. It is unclear what the equivalent to imprecise articulation would be in sign; however, laxing of the configuration of highly-innervated articulators could be a source of imprecise articulation in either sign or speech.

An interesting question that arises from the research on Parkinson's disease and ASL fingerspelling is how movement distalization would manifest itself in the BSL fingerspelling of a signer with PSP. While ASL and BSL fingerspelling are functionally very similar—they are both used to borrow English words—they are structurally very different. (For a full description of the structure of ASL and BSL fingerspelling, see Chapter 3.) In ASL one-handed fingerspelling, it was found that signers with Parkinson's disease produce letters with more distal articulators (e.g. the fingers rather than the wrist) (Tyrone et al., 1999). What remains to be seen is whether locations (as distinct from handshapes) on the non-dominant hand are distalized as a result of hypokinetic disorders in BSL fingerspelling, and whether PSP causes distalization at all. As a broader point, it should be noted that any differences in the results of this study and earlier studies on Parkinson's disease and ASL could be related to the diseases themselves or to the structures of the languages being compared.

7.2 Joseph: Background

Joseph was a 79 year old right-handed man, who was born deaf to hearing parents. There is a history of genetic deafness in his family, however, and he had a deaf brother. He began to learn British Sign Language at age 5 when he entered school and later it became his primary language. He attended a residential oral school for the deaf, left school at the age of 16, and worked most of his adult life as a builder. His wife and one of his children were Deaf, as were most of his friends. Before his health problems late in life, he was an active member of the Deaf community and regularly attended events at the local Deaf club.

Joseph developed a weakness on his left side which began in September 2000, when he was 77. His family originally thought that he had had a CVA then, but no vascular event was formally diagnosed. Joseph's general practitioner reported that he had been admitted to the hospital subsequent to a fall and treated for severe dehydration resulting from dysphagia. The cause of the dysphagia was never determined with any certainty, and no scan was performed to determine whether or not there had been a recent infarct. His records indicate that he had a stroke in September 1999, but no further information about it was available from his doctors.

With repeated visits by members of this project and the Deaf Stroke Project, it became clear that Joseph had a progressive neurological condition, which was becoming worse from one testing session to another. His daughter reported that he and his brother had both been experiencing balance problems, prior to Joseph's clinical event in 2000. Additionally, Joseph had a short career as an amateur boxer, which his daughter suggested might have had some bearing on his later illness. Initially, because of his hypokinesia, it seemed that Joseph might have Parkinson's disease, either in its idiopathic form or as a result of injury. However, on closer study it became apparent that his eye movements were severely limited and that he exhibited no resting tremor, suggesting that his condition was more likely progressive supranuclear palsy than Parkinson's disease. A consultant neurologist confirmed the diagnosis of progressive supranuclear palsy.

7.3 Standardized Assessments and Clinical Observations

7.3.1 Neuropsychological Testing

Because of the extent of his movement deficits, it was difficult to test Joseph on neuropsychological assessments that required movement as a response, particularly when response time was a factor. However, Joseph was tested on the Folstein Mini-Mental State Exam for dementia (Folstein et al., 1975), which was not time-critical and required no non-linguistic complex movements (e.g. handling and arranging objects). His score was 20 out of 30, suggesting mild dementia.

7.3.2 Clinical Observations and Sign Language Testing

For the same reason, it was difficult to conduct formal linguistic testing with Joseph. Critically, in the case of sign language testing in particular, he was difficult to test not only because of his general movement deficits, but also because of his limited eye movements and facial expression. Because facial expression and gaze are crucial

components of BSL grammar and discourse (Sutton-Spence, 1999), judging his comprehension and grammatical abilities in their absence could be problematic. However, on the basis of limited linguistic testing and observations of spontaneous behaviour, Joseph's comprehension of sign language and his productive grammar seemed to be essentially intact, despite his articulatory difficulties. He communicated well with his daughter and had no apparent trouble understanding her, although he often took a long time to respond. Spontaneous signing data and results from explicit linguistic testing over a span of about twelve months suggested that Joseph's signing became progressively smaller and slower as the disease became more advanced. Additionally, in the earlier stages of the disease, he had slightly more eye movement, and more head and lip movement during signing. Unfortunately, because Joseph was not diagnosed as having PSP by his clinicians, it is unclear how advanced his disease status was by the time his sign language was tested; however, his movements were severely impaired from the time he was first visited by the Deaf Stroke Project.

When visited by this project and the Deaf Stroke Project, Joseph was unable to stand or walk and had very little spontaneous movement, either during signing or more generally. One of the most striking aspects of his presentation was his almost complete lack of eye movement and facial expression. In addition, his purposeful hand and arm movements were extremely delayed, slow, and reduced in size. However, he showed no difficulty understanding how to handle and use objects: for example, he could demonstrate how to use a pen, even though it was difficult for him to produce the appropriate movement. Occasionally, he would exhibit intention tremor when he moved, which is not often reported in PSP and may be an indication of cerebellar involvement. Also, unlike most patients with PSP, he had stooped rather than bowed posture.

7.4 Signing Task

7.4.1 Methods: Signing

The design of the task was the same as described in the previous chapter: the experimenter produced signs one at a time and asked Joseph to repeat them. It took him a long time to respond to the explanation of the task at the beginning, but once it was underway, he showed no difficulty understanding the experimenter's signing and copying her productions. In one case, he produced a fingerspelled word rather than the sign, so that production was excluded from analysis. In several cases, Joseph produced a sign twice in quick succession with no pause between productions. These were counted

as repetition errors, but only the first production in each case was analysed in terms of its formational structure. In all, twenty nine productions were analysed and compared to copied sign productions by the control subject, Christine.

7.4.2 Results: Signing

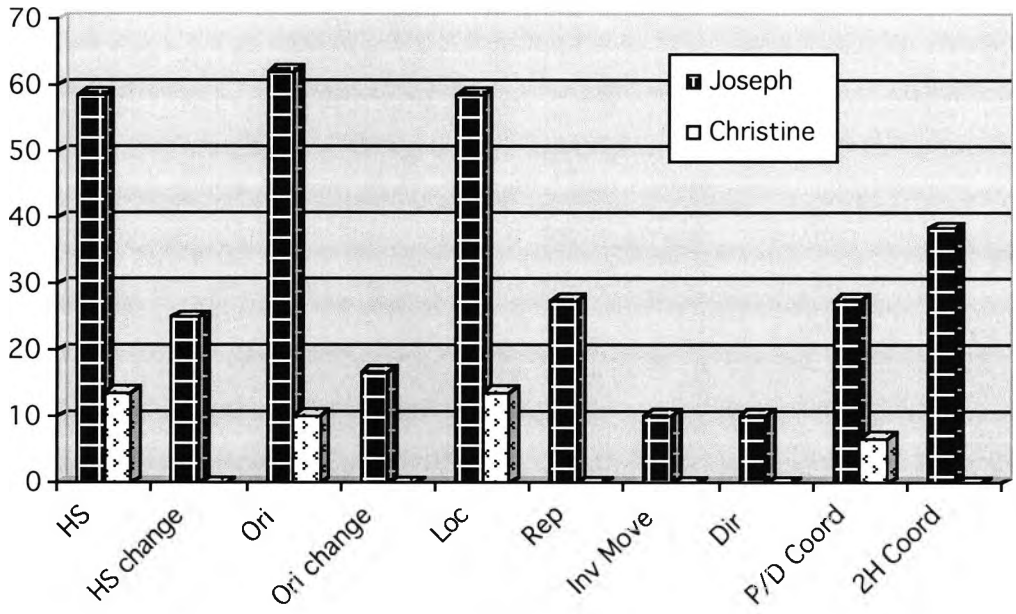
Joseph had an overall high rate of errors on the sign copying task. The distribution of errors in his signing is not very different from the distribution in Christine's signing, although he has far more errors than she does (Figure 7.1). Joseph's most frequent error is in orientation (62.07% of 29 productions), followed closely by handshape and location (for both, 58.62% of 29 productions). These three types of errors are by far the most numerous, and are all static rather than dynamic components of signs. The handshape and orientation errors were manifestations of laxed articulation: the configurations of the articulators were closer to a relaxed or neutral state than they would be in typical signing. Additionally, all but one of the location errors were cases of signs being produced lower than the target location, irrespective of whether the target location itself was high or low. Joseph lowered seven signs that are normally central in citation form, and nine signs that are high in citation form (18 of the 29 target locations are central or low, so the stimuli were not well-balanced). Consequently, his entire signing space is reduced and lowered. Location, handshape, and orientation were the most numerous of Christine's errors as well; she produced location errors in 13.79%, handshape errors in 13.79%, and orientation errors in 10.34% of her signs. As with Joseph, her handshape and orientation errors were both cases of laxing, and half of her location errors were cases of lowering.

The next most common of Joseph's errors is bimanual co-ordination, which was present on 38.46% of two-handed signs (13 of the 29 target signs were two-handed). Joseph had difficulty co-ordinating handshape change with movement from the elbow, in a sign like TOGETHER, for example (Figure 4.4). The next most common are sign repetition and proximal/distal co-ordination (for both, 27.59% of 29 productions). Christine produced no bimanual co-ordination errors and no sign repetition errors on the sign copying task; and 6.90% of her productions included proximal/distal co-ordination errors. Sign repetition was counted as an error only when it was a repetition of the entire sign with no pause between the first and second productions. There was no apparent relation between Joseph's repetition errors and the signs' formational properties. Nor is it likely that repetition served a discourse-level function, since the signs were produced as part of a copying task. It should be noted that Christine did produce sign repetitions

on a separate naming task (as described in Chapter 5), but not on the sign copying task. The few remaining errors that Joseph produced were in handshape change (25% of the 8 signs that required handshape change, or 2 signs) and orientation change (16.67% of the 12 signs that required orientation change, or 2 signs), and involuntary movements and direction (10.34% of 29 productions, or 3 signs). Christine produced none of these errors. (See Appendix C for a full description of error types.)

Figure 7.1: Sign Copying: Joseph

errors as % of total productions



Key: HS: handshape;
HS change: handshape change;
Ori: orientation;
Ori change: orientation change;
Loc: location;
Rep: sign repetitions;
Inv movement: involuntary movements;
Dir: direction of movement;
P/D Coord: proximal/distal co-ordination;
2H Coord: bimanual co-ordination

Table 7.1: Distribution of Sign Errors: Joseph

x = Joseph; o = Christine

	HS	HS change	Ori	Ori change	Loc	Rep	Inv Move	Dir	P/D Coord	2H Coord
WIN	x						x			
BALL			x							x
INFORM	x		x		x					
PERSON					x o	x				
AFTERNOON	x o				x	x				
HOUSE	x		x		x o	x			x	
THROW										
BITE	x		x	x		x		x	x o	x
DROP	x		x		x	x		x	x	
FARM						x				
ELEPHANT		x	x		x				x	
NOTHING	o		x		x o					x
WOLF	x		x		x	x				
ASK			x		x					
KERB	x o		x o						x	
LOVELY	x		x		x					
TAKE-OVER	x				x					
WITH	x	x	x						x	x
UNIVERSITY	x		o		x o	x				
SWEEP							x			x
NAME	x				x					
SHELF	x o				x					
SCREW			x						x o	
TABLE	x		x o							
SCHOOL	x		x		x			x		
BOWL	x		x	x	x				x	
BICYCLE					x					
HAMMER			x				x			
TEACHER			x							

7.5 Fingerspelling Task

7.5.1 Methods: Fingerspelling

For the fingerspelling task, subjects were presented with individual full-page illustrations with corresponding printed text underneath and asked to fingerspell the printed word. (See Appendix A for the full set of fingerspelling stimuli.) The experimenter presented the illustrations one by one and if subjects had any difficulty or misread the word on the first attempt, she would show the illustration again. Because Joseph's pre-morbid signing included a substantial number of fingerspelled words (as is typical of signers of his generation (Sutton-Spence et al., 1990)), and because he performed fewer non-linguistic tasks than the other subjects, a larger sample of his fingerspelling and Christine's fingerspelling was included for analysis (64 letters for each subject). In a few cases, the configurations of the subjects' hands and the orientation of the camera made it impossible to see the dominant hand's location or movement onset, so individual letters were omitted from analysis, while the remaining letters in the same word were included. For each fully visible letter in each fingerspelled word, the handshape, location, temporal duration, and presence of involuntary movements were coded. (See Appendix B for a fingerspelling chart, and Appendix C for a coding sample.)

7.5.2 Results: Fingerspelling

Joseph had far more handshape errors than any other type of error; almost every fingerspelled letter was produced with a lax handshape (93.75% of 64 productions, or 60 letters) (Figure 7.3). He did not produce extraneous handshape changes, nor did he choose handshapes that were phonologically contrastive with or even significantly different from the target. According to Brentari's (1998) phonological model, he consistently produced the selected finger constellation and wrist configuration that matched the target sign, and did not bend, cross, or stack the selected fingers. However, he did produce handshapes that were lax—in other words, the hand and fingers were configured more like they would be at rest, and not fully extended. By contrast, Christine produced handshape errors on 17.19% of the 64 fingerspelled letters (11 letters); like Joseph, all of her handshape errors were lax handshapes.

Joseph also produced many location errors in fingerspelling (59.38% of 64 productions, or 38 letters), of which 15 were proximal, and 23 were distal (two were both ulnar and distal). For a location to be coded as proximalized, the dominant hand had to make contact on the non-dominant hand nearer to the wrist than the target

location. Analogously, for a location to be coded as distalized, the dominant hand had to make contact on the non-dominant hand nearer to the fingertips than the target location. Relative locations on the hand are indicated in Figure 7.2; absolute locations are referenced in terms of anatomical landmarks as described in Appendix C. Subdividing the errors according to target location, all the distalized productions had target locations on the palm, and all the proximalized productions had target locations on the fingers. The stimuli included 37 targets on the fingers, 25 on the palm, and 6 that were off the hand completely (i.e. the fingerspelled letter C). The distribution of Joseph's errors and the target stimuli indicate that a much higher proportion of palm locations were distalized than finger locations were proximalized, so target size does not seem to be a factor in the production of location errors. Christine proximalized 3.13% (or 2) of the letters that she fingerspelled, both on the same letter (A), which has a target location on the palmar thumb.

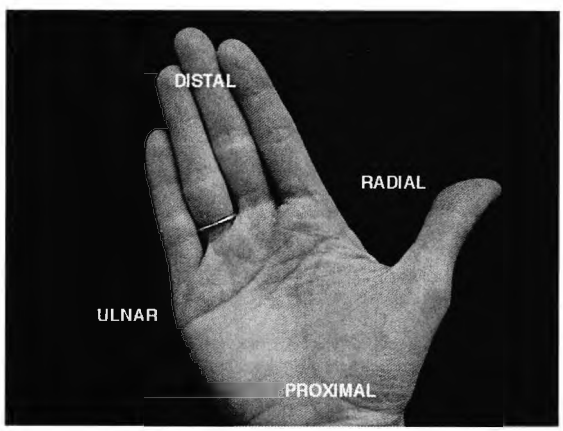
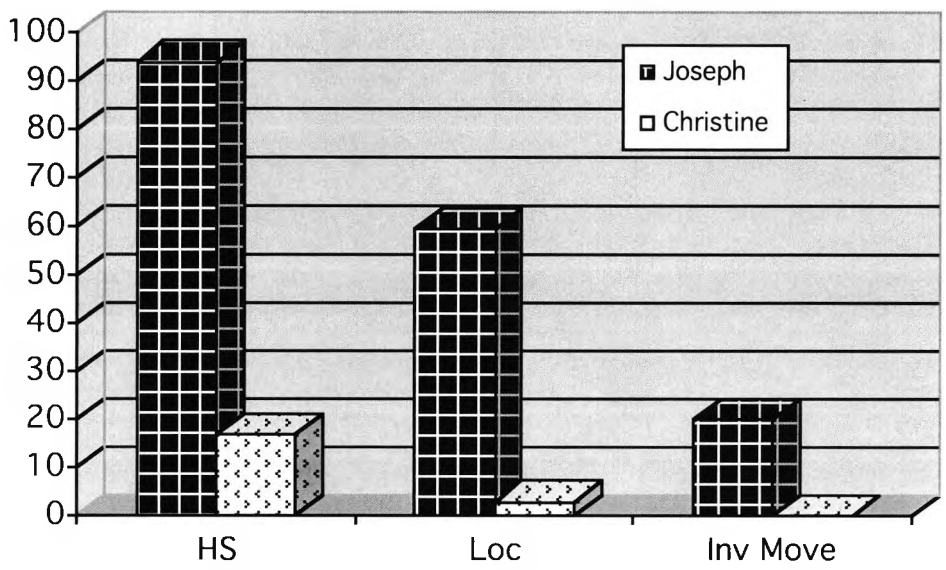


Figure 7.2: Relative locations for fingerspelling

In the fingerspelling task, 20.31% of Joseph's productions (or 13 letters) were accompanied by involuntary movements; while Christine had no involuntary movements on the task (Figure 7.3). Involuntary movements are greatly outnumbered by Joseph's other errors during fingerspelling. There is no clear pattern to which of the letters in the stimuli were more likely to be produced with involuntary movement; however, a higher proportion of the productions of the letters C and A had involuntary movements. C is the only letter that is one-handed, and A is makes contact on the distal thumb of the non-dominant hand (see fingerspelling chart in Appendix B); so it is not clear what features, if any, these letters share that would be likely to induce involuntary movement.

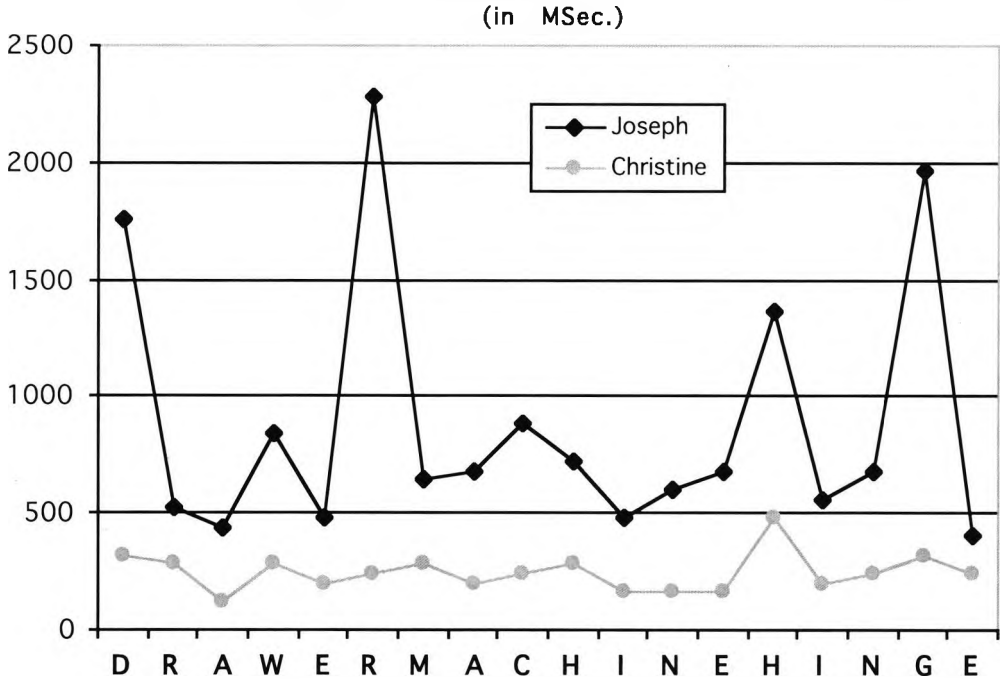
On the whole, Joseph's fingerspelling was slow: individual letters he produced had much longer durations, on average, than those produced by Christine (see Figure 7.4). There was some variation in the durations of different letters (and in different productions of the same letter) in both subjects' fingerspelling. However, the variability in duration from one letter to another was much greater in Joseph's fingerspelling than in Christine's. Nonetheless, there was only minimal indication of which letters were likely to have longer durations for Joseph. Figure 7.4 illustrates the durations of the individual letters from the fingerspelled words, D-R-A-W-E-R, M-A-C-H-I-N-E, and H-I-N-G-E, for Joseph and for Christine. Joseph's movements are slower on letters that require the pronation and supination of the forearm (like G and R), because otherwise he keeps his arms still and only moves his fingers, in which case the articulators do not have to move as far.

Figure 7.3: Fingerspelling: Joseph



Key: HS: handshape
Loc: location
Inv Move: involuntary movement

Figure 7.4: Fingerspelling Durations: Joseph



7.6 Non-linguistic Tasks

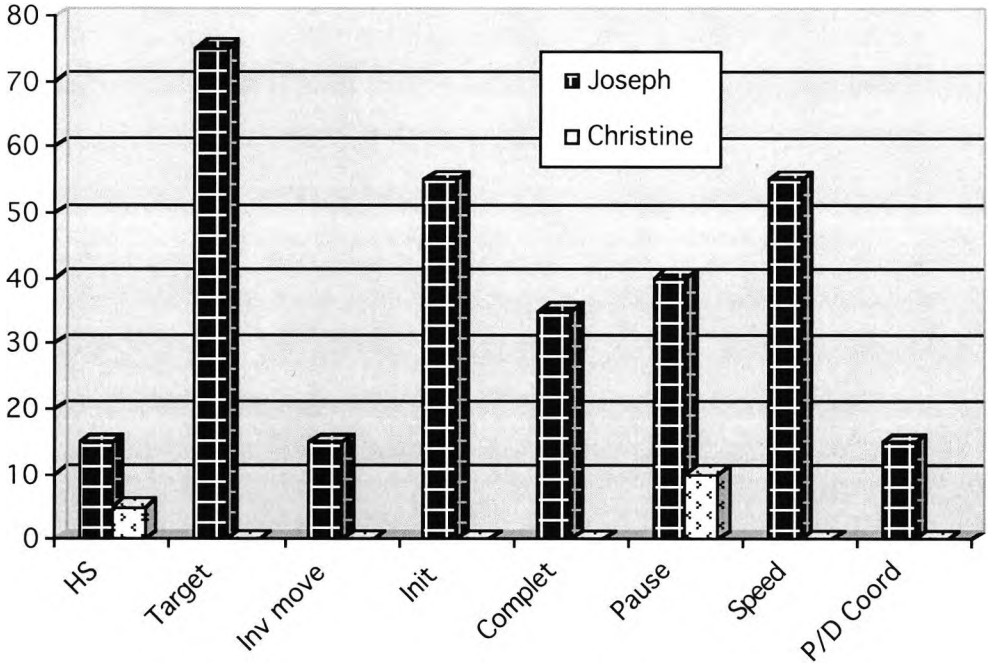
7.6.1 Pointing

7.6.1.1 Methods: Pointing

Subjects were asked to point to illustrations on an A4 sheet of paper in response to a signed utterance produced by the experimenter. The response sheet with the illustrations was placed directly in front of subjects at a comfortable distance. Because Joseph produced only twenty responses, a total of twenty responses on the pointing task were analysed for both subjects; and responses were coded according to the scheme described in Chapter 4. Durations of movements were not directly compared across the two subjects because the exact distances to targets could not be controlled sufficiently during testing for this to be an informative measure. Instead, movement speed was coded somewhat impressionistically and it was noted when movements were exceptionally slow.

Figure 7.5: Pointing Task: Joseph

errors as % of 20 productions



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

7.6.1.2 Results: Pointing

Targeting was the most common of Joseph's errors on the pointing task (Figure 7.5). Of the 20 pointing movements he produced, 75% (or 15) were not targeted accurately, and they were all cases of him not moving far enough to reach the target. By comparison, Christine had no targeting errors. There was no apparent pattern within the stimuli as to which targets Joseph was able to reach accurately and which he was not.

The next most common of Joseph's errors on the pointing task were initiation and speed errors; and Christine produced neither of these. As previously mentioned, Joseph was not only extremely slow once he began to move, but was also very slow to initiate a movement, as these two measures indicate. In addition to difficulty initiating a movement, he had difficulty completing a movement (35% of 20 movements) and often hesitated in the course of a movement (40% of 20 movements). (Completing a movement is distinct from targeting, in that one can successfully stop moving without ending at the correct point, or conversely move along the correct trajectory to reach a target but continue to move past it.)

Joseph showed very little laxing of hand configuration on the pointing task. For most of his responses, his hand was arranged in a fist with the index finger fully extended; in a few cases, his hand was in a fist and he pointed with his thumb. Christine produced one laxed hand configuration, and in two cases, produced a hand configuration with multiple fingers extended.

7.7 Discussion

7.7.1 Task-by-task analysis

Sign Copying

To date, Joseph is the only reported case of a Deaf signer with PSP. Broadly speaking, Joseph's signing was reduced in size and speed, and was characterized by laxed articulation. His sign errors were more prevalent in the static components of signs, namely, handshape, location, and orientation. Moreover, all of those errors were cases of articulatory laxing or sign lowering, which was similar to both the distribution and quality of errors in Christine's signing, but differed greatly in extent. Joseph lowered many signs, but their locations in citation form were equally divided between high and central locations. Thus, it can be concluded that he lowers signs uniformly, rather than having a vertical upper bound beyond which he cannot reach. In general, his sign movements tended toward being too small, lax, and gradual (i.e. not rapidly accelerating or decelerating). As such, his signing deficit resembled an extreme version of the

normal tendencies exhibited in relaxed, informal signing (Kegl et al., 1999), as well as being similar to deficits reported in signers with Parkinson's disease (Brentari et al., 1995; Tyrone et al., 1999).

Joseph had no difficulty with dynamic components of signs in particular, but he did have difficulty co-ordinating multiple sets of articulators during signing. In other words, he had no difficulty with sign-internal movement change as long as those movements were confined to a localized set of articulators. This suggests that at some level, groups of sign articulators (specifically the fingers) are acting together as a unit, which is consistent with findings from studies on Parkinsonian signing (Tyrone et al., 1999), and consistent with models of hand configurations and finger movements (Iberall & Fagg, 1996; Schieber, 1996). Specific sets of sign articulators are probably grouped together in the generation of simple motor synergies during motor programming for signing. It may be that the capacity that Joseph lost was the ability to combine motor synergies to execute more complex movements—a function normally associated with the cerebellum (Thach et al., 1992).

Fingerspelling

Joseph's fingerspelling showed a similar pattern to his signing: his productions were generally laxed and slow, and the space that he used for making fingerspelled letters was reduced. He often made contact on his non-dominant (left) hand in whatever location required the least movement for both hands, so locations on the palm were produced distally and locations on the fingers were produced proximally. (He held his arms more or less in place and only brought his fingers in contact with each other.) Where his fingerspelling differed from his signing was in the distribution of errors; most notably, he exhibited more handshape errors and involuntary movements in fingerspelling than in signing.

Joseph produced proportionally more laxed handshapes when fingerspelling than when signing, despite the fact that there is less handshape variety in fingerspelling than in signing. There are 26 fingerspelled letters in BSL, just as there are in the Roman alphabet; however, many of them are made using the same handshape, placed in different locations on the non-dominant hand. Ten of the letters in BSL are formed with the right hand configured in a closed fist with the index finger extended, what is referred to in the sign literature as a '1' handshape. Altogether, there are only 14 distinct handshapes in BSL fingerspelling, as opposed to 57 distinct handshapes in BSL signs (Brien, 1992). (See fingerspelling chart in Appendix B.) This suggests that the

disruption to Joseph's handshape production was not influenced by the demands of handshape change, since fingerspelling requires few changes in handshape. However, fingerspelling is much more rapid than signing, which could cause more handshape errors.

Joseph's location errors in fingerspelling were not consistently proximal or distal, but they were consistently more centralized, such that the overall articulatory space on the non-dominant (left) hand was reduced. Letters with target locations on the fingers tended to be produced more proximally, and letters with target locations on the palm produced more distally. He had proportionally fewer location errors in fingerspelling than in signing, which suggests that his movement deficit had more to do with range of motion than with targeting, because in fingerspelling the targets are small but near, while in signing, the targets are generally large but sometimes distant.

Pointing

Joseph had considerable difficulty with the pointing task, with extreme delays in his responses, and in some cases, no responses at all. His movements were delayed, slow, effortful, and reduced in size. Rather than lifting his arm, he often placed his hand down on the paper, and slid it toward the intended target. He consistently undershot targets, often paused during movements, and had difficulty ending movements as well. However, his movements were not dysmetric in a general sense because in all the targeting errors he did not move far enough to reach the target, but he never overshot the target. Joseph's errors on the pointing task were related to the timing of individual movements (speed, hesitation, initiation, etc.) rather than being errors of co-ordination.

Proportionally, Joseph had more involuntary movements on the pointing task than in his signing and about the same proportion as in his fingerspelling. It could be that Joseph produces more involuntary movements when the targeting demands of a task are high. This could explain why he had more involuntary movements in pointing and fingerspelling than in signing, since in general, articulatory targets in signing are large. The explanation cannot be quite this simple, because there is no clear pattern to the distribution of involuntary movements *within* the fingerspelling and pointing tasks; but it could begin to account for the disparity across tasks, while a different factor could contribute to smaller scale variation.

Joseph's sign and fingerspelling errors patterned similarly to each other but very differently from his errors on the pointing task. Joseph's pattern was like Christine's on the sign copying and fingerspelling tasks, even though he produced many more errors.

However, Joseph and Christine patterned differently on the pointing task. On the whole, he had a higher proportion of errors on the signing and fingerspelling tasks. Additionally, he had far more handshape errors on the signing and fingerspelling tasks than hand configuration errors on the pointing task, which cannot be explained solely in terms of the hand configuration demands of the different tasks. He produced an extremely high number of lax handshapes on the fingerspelling tasks, when very few handshape changes were required from one letter to another. On the pointing task, Joseph had proportionally more targeting errors, which may be related to the external targets being farther away in that task than in the other two. This explanation seems likely given that his targeting errors were consistently cases of undershoot rather than overshoot.

The category of error that sets Joseph's performance on the signing task apart from his performance on the other tasks, and also sets him apart from any atypical signers reported thus far, is repetition. He does not spontaneously repeat movements on the pointing task or on the fingerspelling task. His spontaneous repetition of signs both during testing and in informal conversation was the original motivation for including it as an error category in the coding scheme. When neither he nor any of the other subjects spontaneously repeated voluntary movements in any of the other tasks, it was removed from the coding schemes for those tasks.

To summarize, compared to the control, Joseph showed a reduction in the size and speed of all his movements. He had fewer location errors in fingerspelling than in signing, suggesting that his movement deficit was more related to range of motion, or difficulty scaling large movements, than to targeting as such. His targeting was more accurate when targets were near than when they were far, irrespective of target size. Joseph did not have large trajectories to nearby locations; by contrast, he kept his trajectories as short and direct as possible, sometimes at the expense of targeting accuracy.

7.7.2 Comparisons to past sign research

In many ways, Joseph's signing resembled that of signers with Parkinson's disease, but with some important distinctions. Like signers with Parkinson's disease, Joseph exhibited laxing and slowing of his signing, as well as reduction of signing space. With respect to slowing, it is interesting to note that this is a symptom that differentiates hearing subjects with PD or PSP dysarthria, and yet does not seem to differentiate Deaf signers with PD or PSP dysarthria, because Deaf signers with

Parkinson's disease do not have atypically rapid sign production (see Chapter 8 for more discussion of festination of sign vs. speech). Unlike signers with Parkinson's disease, Joseph did not exhibit mirroring of one-handed signs on the non-dominant hand. Additionally, he exhibited involuntary movements in his signing and fingerspelling, and palilalia, or repetitions of entire signs, neither of which has been reported in signers with Parkinson's disease.

Joseph's fingerspelling also showed similarities to and differences from the ASL fingerspelling of subjects with Parkinson's disease, in many of the same ways. Like signers with Parkinson's disease, Joseph produced laxed handshapes in fingerspelling. However, it is unclear whether the American subjects had a greater tendency to produce lax handshapes in fingerspelling than in signing, as Joseph did, because the American subjects were not compared across the two tasks. Another pattern that Joseph shared with the Parkinsonian signers was high variability in the durations of individual fingerspelled letters. Finally, Joseph showed a reduction of the articulatory space of his fingerspelling, which is something that cannot be altered in ASL fingerspelling, because ASL letters do not have location. However, this is similar to PD signers' reduction of signing space in ASL, even though it is not possible to do the same in ASL fingerspelling.

Where Joseph's error patterns in fingerspelling differed from the subjects with Parkinson's disease was in distalization and presence of involuntary movements. There was frequent distalization in the handshapes made with the dominant hand in ASL fingerspelling: the fingers would begin their movements from more distal joints than is typical. By contrast, there was no consistent distalization of location on the non-dominant hand in Joseph's BSL fingerspelling. This is probably an effect of the structure of the fingerspelling system as much as a difference in the effects of the two diseases on articulation. While the non-dominant hand is not completely passive in BSL fingerspelling (Sutton-Spence, 1994), it can be viewed as providing articulatory targets that the dominant hand moves toward. As such, the non-dominant hand in two-handed fingerspelling may be compared to signing space in normal signing; and the reduction in the articulatory space on the hand in Joseph's fingerspelling could be related to the reduction in his signing space. Finally, the main distinction between Joseph and the signers with Parkinson's disease was that he produced involuntary movements during fingerspelling, while they did not. This could be an effect of damage to the cerebellum—a mild version of intention tremor—resulting from PSP.

7.7.3 Comparisons to speech dysarthria

The sign repetition errors in Joseph's signing seem very clearly analogous to what is described in PSP speech as palilalia. The form of the repetition is similar across the two modalities in that there is no obvious psychogenic cause, and yet it involves the repetition of an entire semantic unit rather than a phonotactic unit, such as a phone or a syllable. In the case of speech, it is a dysarthric symptom that differentiates PSP from Parkinson's disease. This study raises the possibility that palilalia may differentiate PSP from Parkinson's disease in sign as well, given that previous studies on Deaf signers with Parkinson's disease have reported no similar finding, although Joseph exhibited palilalia many times.

Joseph's proximal/distal co-ordination and bimanual co-ordination errors are consistent with what one would predict from past research on PSP and dysarthria: a particular difficulty in timing and articulatory precision. Clearly, Joseph had an impaired ability to time the movements of separate articulators relative to each other, as indicated by his co-ordination errors; so his case reinforces the view that a timing/co-ordination deficit may underlie the articulatory deficit in PSP speech. Joseph's errors could be related to the suggestion by Metter & Hanson (1991) that PSP causes a deficit in movement timing. Discussing his errors in terms of articulatory imprecision is somewhat more difficult, in part because of the definition of the term as it applies to speech. Articulatory imprecision is essentially a broad term used to describe any problem specific to the supralaryngeal articulators, including imprecise consonants, irregular articulatory breakdown, distorted vowels, and repeated phonemes (Duffy, 1995). If instead what is viewed as distinctive about articulatory precision is that it involves articulators that are highly innervated and have a wide range of motion (unlike the larynx or the velum), then the term could be applied cross-modally. Moreover, that definition would certainly include the types of errors that Joseph exhibited.

Because Joseph had a hypokinetic movement disorder, it was predicted that his errors would pattern similarly to those previously reported in studies of Parkinson's disease and sign articulation, but with a few important differences, given that some of the most notable differences between PSP and Parkinson's disease are evident in dysarthria. In general terms, Joseph's dysarthric symptoms seem very similar to what is reported in PSP speech. In particular, he repeated entire signs without pausing and for no communicative or broader behavioural reason, which seems like a direct parallel to the spoken language phenomenon of palilalia. Additionally, Joseph exhibited a deficit in what could be described as articulatory precision on linguistic but not non-linguistic

tasks, and in the relative timing of the movements of multiple articulators only in signing. While Joseph's case is a very uncommon one, it raises larger questions for the conceptualization of speech motor control. His case illustrates that it would be productive to think about measures of speech motor control (such as articulatory precision) in broader terms as motor control phenomena, in a way that would allow cross-modal application, as well as a more meaningful comparison to motor deficits in general.

8 John: Signer with Parkinson's Disease

This chapter examines the case of John, a signer who developed Parkinson's disease as a middle-aged adult. As outlined in previous chapters, there have been several studies in the USA on the effects of Parkinson's disease on signing in ASL. However, John's case is unique in a few ways: it is the first reported case of a British signer with Parkinson's disease, and of a native signer with Parkinson's disease. It is possible that he could differ from previously-reported cases in any number of ways for either of these reasons. Additionally, John is considerably younger than the PD signers described in earlier studies, so his case could illustrate findings that were age-specific from those studies. The value of analysing John's case lies not only in the contrasts it may show with similar cases, but also in what his case may hold in common with them. As with earlier studies on PD and signing, John's case can help elucidate the role of the basal ganglia in movement and the relationship between the general movement disorder of PD and its specific effects on articulation. Typically, PD dysarthria is less severe than other symptoms of the disease, such as disruptions to gait, balance, and speed of simple limb movements. Finally, John's case provides the opportunity to compare the features of PD dysarthria when a subject is medicated and not medicated (see section 8.1.2 below for discussion of this issue).

8.1 Parkinson's disease

Parkinson's disease is a neurodegenerative disease characterized by the loss of dopaminergic neurons in the substantia nigra pars compacta, which disrupts function of the basal ganglia more generally and severely impairs motor control. It affects between 0.1% and 1% of the population at large and is more prevalent among men than among women, by a 3:2 ratio. The most common symptoms of the disease are resting tremor (as distinct from tremor that occurs during deliberate movement), rigidity, bradykinesia (or slowed movement), impaired postural reflexes, difficulty initiating movement, and a reduction in spontaneous movement including formation of facial expressions. Additionally, dementia and depression are psychological symptoms that tend to begin in the later stages of the disease. Because Parkinson's disease is degenerative and therefore progressive, the symptoms usually multiply and worsen as the disease becomes more advanced. The progression of the disease is so well established that there are standard schemata of Parkinson's disease "stages" used by clinicians to classify a patient's condition based on which symptoms are present and how severe each of them is (Hoehn & Yahr, 1967).

An increasing body of research suggests that there is a genetic component to Parkinson's disease (Bonifati et al., 1995; Nussbaum & Polymeropoulos, 1997; Papapetropoulos et al., 2001; Polymeropoulos et al., 1996). However, most studies that have identified a family pedigree with multiple cases of the disease include a disproportionate number of subjects with the early-onset version (Golbe et al., 1990; Polymeropoulos et al., 1997). Most cases of Parkinson's disease occur 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). In their study of consecutive cases of PD in a neurology clinic, Bonifati et al. (1995) found that the only factor to differentiate familial from sporadic cases was age of onset; familial cases tended to be early-onset, whereas sporadic cases were more likely to occur late in life. More recent research on early-onset PD reveals that a high proportion of those patients have a genetic mutation on a gene hypothesized to be a marker for the disease (Papapetropoulos et al., 2001; Periquet et al., 2003). Consequently, there may in fact be two forms of spontaneously-occurring Parkinson's disease: a heritable form with an early onset, and a non-heritable form that appears in old age, similar to juvenile- and adult-onset diabetes. The symptoms of early-onset and late-onset PD are extremely similar, the chief difference being that patients with early-onset PD are more likely to develop dementia in the early stages of the disease.

Since the disease is related to the lack of a single neurotransmitter, dopamine, it is typically responsive to medication designed to raise dopamine levels. For most patients, the medication is extremely effective for about five years then begins to cause side effects, most notably, dyskinesias, or uncontrolled movements, in the face and limbs. Because of how the medication is metabolized, patients tend to have on-off phases of side effects being at their worst and disease symptoms completely controlled, alternating with the absence of side effects and return of disease symptoms. Consequently, motor behaviour in particular can vary tremendously depending on when patients are examined relative to when they took their medication; so most experimental research controls for medication level when testing subjects.

8.1.1 PD and dysarthria

The dysarthria associated with Parkinson's disease is classified as hypokinetic dysarthria, because like hypokinesia, it is characterized by a limited range of movement, which manifests itself in monotonous, aprosodic speech. Hypokinetic subjects exhibit harsh, breathy voice quality, short rushes of speech, inappropriate silences, variable

speech rates, reduced stress, and aprosodia (Darley et al., 1975). Less commonly reported symptoms of hypokinetic dysarthria include mild hypernasality (Kent & Rosenbek, 1982) and impaired breath support (Theodoros & Murdoch, 1998b). PD dysarthria is unique among the dysarthrias in that one of its characteristics is perceivably rapid speech. Notably, no other type of hypokinetic dysarthria (e.g., from PSP or Shy-Drager syndrome) causes rapid rather than slow speech. Additionally, relative to other subjects with hypokinetic dysarthria, PD subjects are reported to exhibit reduced intensity, higher pitch, and reduced pitch range (Hartman & Abbs, 1988; Lu et al., 1992). To summarize, PD dysarthria primarily affects pitch, loudness, nasality, respiration, and speech rate, in other words, the components of speech least related to the precise, co-ordinated movements of the highly innervated articulators in the anterior vocal tract—what Darley, Aronson, and Brown (1975) referred to as 'articulation.' However, it is unlikely that the effects of PD on speech are articulator-specific *per se*; after all, the general motor symptoms of PD are quite diffuse. What is more likely is that PD speech deficits are particular to certain types of movements or components of movements that make specific demands on the motor control system.

While there have been some studies on the relationship between PD dysarthria and medication status (Gallena et al., 2001; Goberman et al., 2002; Sanabria et al., 2001) or disease stage (Muller et al., 2001), their findings are somewhat unclear. Some research indicates that acoustic (Sanabria et al., 2001), and electromyographic and perceptual (Gallena et al., 2001) measures of PD dysarthria improve while subjects are on dopaminergic medication; however, pharmacologic treatment in isolation does not consistently improve dysarthric symptoms of PD across studies (Schulz & Grant, 2000). Moreover, studies not specifically examining the effects of dopaminergic medication often do not explicitly control for it when testing (Liss et al., 2000; Ludlow & Bassich, 1983), or conversely, control for it in opposite ways, so that either being on medication (Forrest et al., 1989), or being off medication (Ackermann et al., 1997) is taken as the default state, while any deficits described are attributed to the disease itself. As a result, it is difficult to compare findings across studies or to say anything definitive about how the characteristics of dysarthria might vary according to medication status. Regarding disease stage, the development of dysarthria over the course of PD has not been studied systematically; consequently, all that is known is that dysarthria is usually not one of the presenting symptoms of Parkinson's disease but tends to appear later in the course of the disease (Muller et al., 2001).

8.1.2 PD and language

Some researchers have suggested that in addition to disrupting speech motor control, Parkinson's disease may also affect language comprehension and production, in particular syntactic processing (Lieberman et al., 1990; Lieberman et al., 1992; Natsopoulos et al., 1993). This theory has been supported by other lines of research suggesting that syntactic processing in a linguistic context and processing of abstract structure in a non-linguistic context (e.g. event sequencing) share various neural mechanisms, in particular the connection between the striatum and the frontal lobe (Dominey et al., 2003; Lelekov-Boissard & Dominey, 2002). Consequently, if striatal function is disrupted, as in the case of Parkinson's disease, language ability might be impaired as a result. However, there has not been a substantial body of clinical (as opposed to experimental) evidence suggesting that patients with Parkinson's disease exhibit linguistic deficits in the absence of dementia. Additionally, research on syntactic deficits in PD have not consistently controlled for subject age (e.g., Lieberman et al., 1992), which could play a role itself. Murray & Lenz (2001) found no significant difference in language ability of subjects with Parkinson's disease and age-matched controls. Consequently, the impairment previously identified by Lieberman and others could be an effect of age-related cognitive decline rather than Parkinson's disease. Returning to the point about syntax and striatal function, the same study reported significantly reduced production of syntactically complex utterances in subjects with Huntington's disease (HD) but not in subjects with Parkinson's disease (Murray & Lenz, 2001). By contrast, Ullman et al. (1997) reported that HD subjects and PD subjects both show morphosyntactic deficits, but of different varieties: HD subjects over-regularize irregular verbs, but PD subjects produce more morphosyntactic errors in the conjugation of regular verbs. These findings are of interest because although both diseases disrupt striatal function, Huntington's disease does so much more severely and directly. (For a review of studies on syntax and the basal ganglia, see (Patterson & Bly, 1999)). Finally, non-linguistic research has suggested that the observed deficit that PD subjects show in event sequencing was in fact attributable to impairment of a switching mechanism rather than a syntactic deficit (Zalla et al., 1998).

8.1.3 PD and sign language

Because of the high prevalence of the disease, signers with Parkinson's disease have been researched more than signers with almost any other movement disorder. In fact, PD and apraxia comprise the entirety of research on movement disorders and sign

language prior to this project. Early research on sign language and the brain explored aphasia and apraxia in Deaf signers in order to address the similarities and differences between sign and gesture (Corina et al., 1992b; Poizner et al., 1987). Later research on signers with Parkinson's disease focused primarily 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 Parkinson's disease (Brentari et al., 1995; Kegl et al., 1999; Poizner & Kegl, 1992, 1993).

As described in the previous chapter, multiple studies of PD and American Sign Language have examined a range of aspects of sign and fingerspelling articulation and reported their findings. In those studies, signers with Parkinson's disease were reported to have laxed articulation (Brentari et al., 1995; Loew et al., 1995), which is to say that during production of a sign, the configuration or orientation of the hand is closer to how it would be when the hand is at rest. Unlike signers with aphasia, PD signers did not produce handshapes that had an incorrect selection of fingers to be extended (Poizner & Kegl, 1993). Furthermore, as Kegl et al. (1999) point out, laxing of the handshape and orientation of a sign also occurs among typical signers in casual or informal contexts.

One of the most widely reported characteristics of Parkinsonian signing was the reduction and lowering of signing space (Loew et al., 1995; Poizner & Kegl, 1992, 1993), such that signs articulated in neutral space in front of the body were produced closer to each other and lower down in space. Additionally, signs produced in contact with the body were produced at a lower location on the body, e.g., the chin instead of the forehead. A similar, but crucially distinct, characteristic that was reported was the distalization of the active articulators in both sign (Brentari & Poizner, 1994; Poizner, 1990; Poizner & Kegl, 1993), and fingerspelling (Tyrone et al., 1999). In signs that normally require a proximal articulator to move through space, signers with PD would use a more distal articulator (e.g. the wrist instead of the elbow) and make a smaller movement. Notably, this pattern in PD signing encompasses two related, but separate phenomena. Lowering and shrinking of the signing space is a measure of where signs are produced in space, whereas articulator distalization is a measure of which articulators move to form the sign. There is obviously an interaction between these two measures, but they are not identical; it is not possible to make a large movement with a distal articulator, though it is possible to make a small movement with a proximal articulator, or to make a large movement to a nearby location in space (for further discussion of this distinction, see Chapter 6).

Signers with PD are also reported to have impaired co-ordination when signing (Brentari et al., 1995; Poizner et al., 2000; Tyrone et al., 1999), although impaired co-ordination is only rarely associated with PD in the motor control literature (Benecke et al., 1986; Ingvarsson et al., 1997). In signs produced by subjects with PD, the movements of independent sets of articulators were decoupled, so that, for example, movement and handshape change in a sign would be produced either completely serially or completely simultaneously, rather than having partial temporal overlap, as is the case typically. Another type of co-ordination error took place in the transition between signs (or fingerspelled letters) rather than within individual signs: signers with PD would blend distinct handshapes of two signs into each other, such that an intermediate form of the two handshapes would be produced across two-sign utterances (Brentari et al., 1995; Loew et al., 1995). A less frequent co-ordination error, which nonetheless occurred in both sign and fingerspelling, was handshape mirroring on the non-active hand in one-handed signs (Loew et al., 1995; Poizner et al., 2000; Tyrone et al., 1999). Signers with Parkinson's disease occasionally produced the handshape of a one-handed sign on the non-dominant, non-active hand, but did not produce fully-formed two-handed versions of one-handed signs. (See Chapter 6 for discussion of this distinction and its possible underlying neural mechanisms.)

8.2 John: Background

John is a 54 year old right-handed man with Parkinson's disease (PD), who was born deaf into a family with deaf parents and hearing siblings. He was the youngest of four children (the other three were all hearing) and learned British Sign Language from his parents as a native language. He attended an oral residential school for the deaf and left school at the age of 16 to begin working. He worked in various manual trades as an adult, up until the time when his illness made work too difficult. His wife, two of his three children, and many of his friends are Deaf, and he uses BSL as his primary language. Additionally, he and his family are active in the local Deaf community. John was recruited for participation in this study through an announcement placed in the local Deaf news; unlike most of the other participants, he was not involved in the Deaf Stroke Project, because he did not have a stroke, or any pathology that could be mistaken for one.

John developed Parkinson's disease at the age of 48 and was diagnosed and treated promptly. His symptoms are typical of the early stages of the disease and will be discussed in more detail below. He has taken dopaminergic medication for several

years, which controls his Parkinsonian symptoms well but also causes moderate dyskinesias, particularly in his arms and neck. There is no history of the disease in his family, and he suffered no injury likely to cause neural damage, so it is presumed that he has the common, sporadic form of the disease. It should be noted, though, that he developed Parkinson's disease at a fairly young age and he may show some signs of dementia (see below), both of which suggest the early-onset form of the disease. In any case, there are insufficient data to decisively conclude anything about John's disease aetiology.

8.3 Standardized Assessments and Clinical Observations

8.3.1 Neuropsychological Testing

John was given few neuropsychological tests, because he was not recruited by the Deaf Stroke Project, so his case was not followed with the intent of developing and providing therapy. However, he was tested for handedness, using a version of the Edinburgh Handedness Test (Oldfield, 1971), which was modified for sign language users; and dementia, using the Folstein Mini-Mental State Exam (Folstein et al., 1975). The handedness test revealed that John is predominantly right-handed; out of thirteen activities, he reported that he used his left hand only occasionally for one of them. The dementia test is more difficult to interpret because his score was reduced considerably by one question, which he initially had difficulty answering and then refused to attempt. As a result, his score is slightly below normal. Nonetheless, while he was able to interact essentially normally both on and off medication, he did exhibit some mild cognitive slowing in both conditions, in the form of slowed (but correct) conversational responses and occasional inattentiveness.

8.3.2 General Observations

Because John did not take part in the Deaf Stroke Project, and because he had no obvious language impairment, his language ability was not formally tested. On the basis of informal observation and his performance on the sign articulation tasks described below, John had no apparent difficulties in language production or perception, independent of articulatory difficulties. Regarding his general movement patterns, he had resting tremor in his hands and legs; and his voluntary movements were reduced in size and speed, particularly when he was off-medication. When he was on-medication, he exhibited dyskinesias in his arms and especially in his neck. He also reported occasional problems with balance. Despite these difficulties, he is still largely self-

sufficient: he is able to feed himself, stand, walk, and drive a car. When on medication, his gait is slow and festinating, but he has had no problems with falling. He reported no problems swallowing, and his facial expression and eye movements remain intact. It should be noted that his responses as well as his movements were slow; that is to say, there was a differential pattern of slowing on spontaneous movements and movements that are generated in response to an external stimulus, such as a question.

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

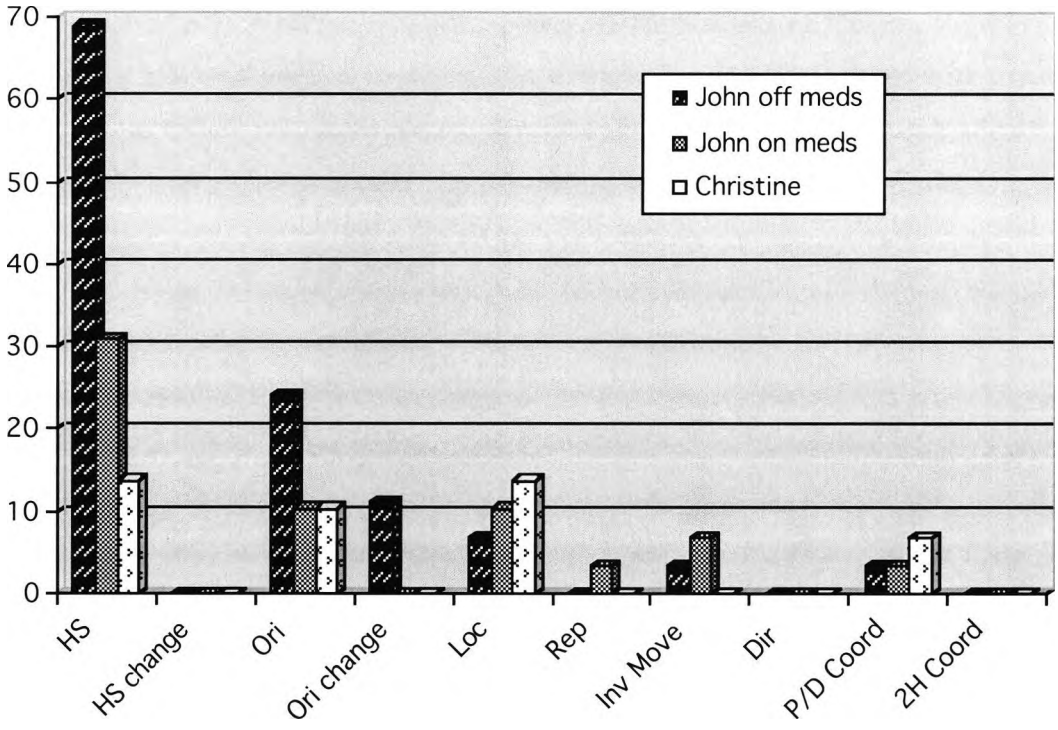
8.4 Signing Task

8.4.1 Methods: Signing

John performed the sign copying task outlined in Chapter 4. The experimenter produced single signs and John repeated her productions. He had no difficulty understanding or executing the task; and all productions were visible and easily codable, so no data were excluded from analysis. John's productions were compared across the two conditions, on and off medication, and to the productions of the control subject, Christine. Altogether, twenty nine productions were analysed for Christine and for John in each of the two conditions. Data were analysed using the coding scheme described in Chapter 4.

Figure 8.1: Sign Copying: John

errors as % of total productions



Key: HS: handshape;
HS change: handshape change;
Ori: orientation;
Ori change: orientation change;
Loc: location;
Rep: sign repetitions;
Inv movement: involuntary movements;
Dir: direction of movement;
P/D Coord: proximal/distal co-ordination;
2H Coord: bimanual co-ordination

8.4.2 Results: Signing

By far, the majority of John's sign errors when he was off medication were errors of handshape. Out of 29 productions, 68.97% (or 20) of them included a handshape error, which is more than three times the number of the next most frequent error. Handshape errors were also the most frequent error when he was on medication, though they were far less frequent in that condition (31.04%, or 9 signs) than when he was off medication. John's next most frequent error off medication was in sign orientation (24.14% of 29 productions), which is an error type that is possible in any sign. Like handshape, orientation is a static component of signs. When John was on medication, his next most frequent errors were location errors and orientation errors (for both, approximately 10.34% of total productions, or 3 signs). In the case of both handshape and orientation errors, both on and off medication, most errors were cases of laxing, such that a sign was produced with a configuration that is closer to how the hand or limb would be configured at rest.

When he was off medication, John omitted an orientation change in approximately 11.11% of target signs that required one; however, only 9 signs required an orientation change, so he only had to produce one error to reach 11.11% of the total. John never added an orientation change where the sign did not require it. Both on and off medication, John had no more than a 7% error rate in any of the other categories. In the off medication condition, those were: location (6.90%), involuntary movement (3.45%), and proximal/distal co-ordination (3.45%). In the on-medication condition, they were: location (10.45%), involuntary movement (6.90%), repetition (3.45%) and proximal/distal co-ordination (3.45%). Interestingly, in the case of location errors, John had a lower rate of errors off-medication than on-medication, and fewer errors than Christine in either condition (13.79% of total productions). The nature of John's and Christine's location errors both varied; that is to say, not all location errors were cases of sign lowering or raising for either of them. Similarly, Christine had more proximal/distal co-ordination errors (6.90%) than John in either condition (for both, 3.45%). However, for all of these measures, the numbers are small enough that it is difficult to interpret them reliably or meaningfully.

Table 8.1: Distribution of Sign Errors: John

X = John off medication, **Δ** = John on medication, **O** = Christine

	HS	HS change	Ori	Ori change	Loc	Rep	Inv Move	Dir	P/D Coord	2H Coord
SHELF	x Δ o									
WOLF			x							
NOTHING	o		x		o					
HOUSE					o					
THROW	x									
UNIVERSITY	x		o		o					
BITE	x								Δ o	
KERB	x o		o							
ASK	x Δ									
CATCH	x Δ				Δ					
INFORM	Δ					Δ				
BALL	x Δ		x							
FARM										
AFTERNOON	x Δ o		Δ							
FIRE	x Δ									
PERSON										
LOVELY										
WITH	x						x Δ			
SWEEP	x									
BICYCLE	x									
SCREW									o	
BOWL	x Δ			x					x	
WEEKEND	x Δ o						Δ			
HAMMER			x		Δ					
TEACHER			x		x Δ					
PICTURE	x		o							
SCISSORS	x		Δ							
ELEPHANT	x		x		x					
NAME	x									

John produced very 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 tremor occurring in the hand. Because dyskinesias occur during voluntary movement, while Parkinsonian tremor typically does not, it is not a great surprise that John had more involuntary movements on medication than off medication.

8.5 Fingerspelling Task

8.5.1 Methods: Fingerspelling

The design of the fingerspelling task is described in detail in Chapter 4: John was presented with individual full-page illustrations with corresponding printed text underneath and asked to fingerspell the printed word. He had no comprehension difficulties or irregular repetitions of letters, so the experimenter did not have to present stimuli more than once, and no data had to be excluded from analysis. For each letter in each fingerspelled word, the handshape, location, temporal duration, and presence of involuntary movements were coded. As with the other subjects, only a portion of the data were coded at this level of detail; the data to be analysed were selected on the basis of word length, variety of letters, variety of handshapes, and variety of locations. Altogether, six fingerspelled words comprising 35 fingerspelled letters were analysed according to these criteria. The fingerspelling coding scheme described in Chapter 4, which was modified from the coding scheme for sign data, was used to analyse John's fingerspelled productions. Also, as before, the durations of individual fingerspelled letters were measured, and a small sample of those are shown in Figure 8.3.

8.5.2 Results: Fingerspelling

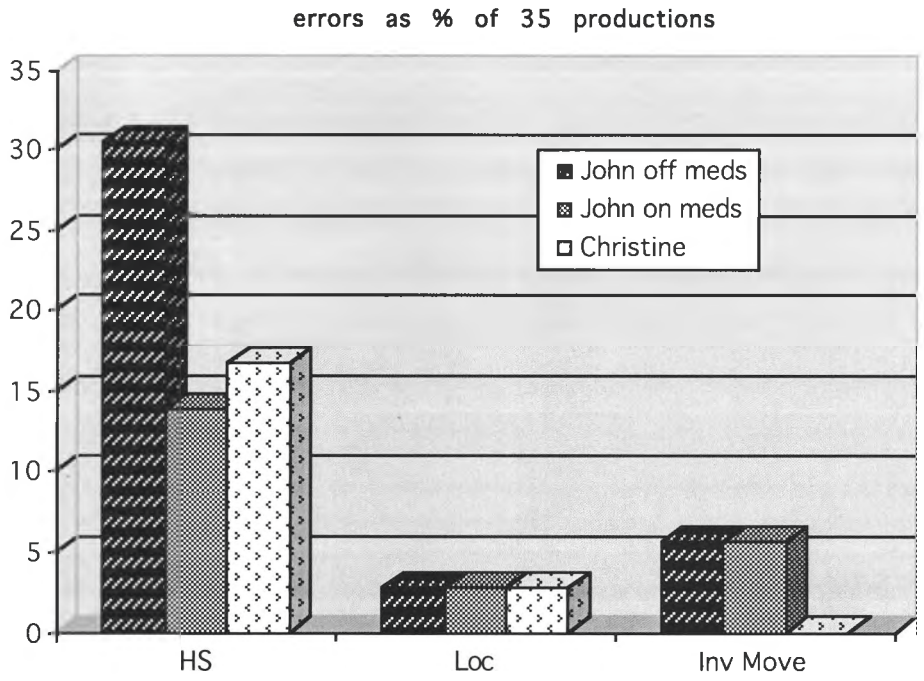
John had fewer errors in proportion to the number of productions on the fingerspelling task as compared to the signing task. However, as with the signing task, most of John's errors on the fingerspelling task were handshape errors (30.56% of total productions or 11 letters, off medication; 13.89% or 5 letters, on medication) (Figure 8.2). His other fingerspelling errors were very few by 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 (2.78%, 1 letter) and equal numbers of involuntary movements (5.56%, 2 letters) in both conditions.

As with the signing task, although John had the same proportions of involuntary movements on the fingerspelling task in the two conditions, the nature of the movements was in fact different. When he was on-medication, the involuntary movements were dyskinesias beginning from the base of his arm that occurred while he was moving. By contrast, when he was off-medication, the involuntary movements were tremors in his hands that occurred between voluntary movements.

The durations of John's fingerspelled letters varied a lot from one letter to another. However, they seem to vary in a predictable fashion and in the same way as the

Christine's fingerspelled productions, although to a greater extent. The first letter of a word consistently had the longest duration for both subjects. This pattern is illustrated in Figure 8.3, 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. Another pattern that can be seen is that the physical proximity of the locations of two letters 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).

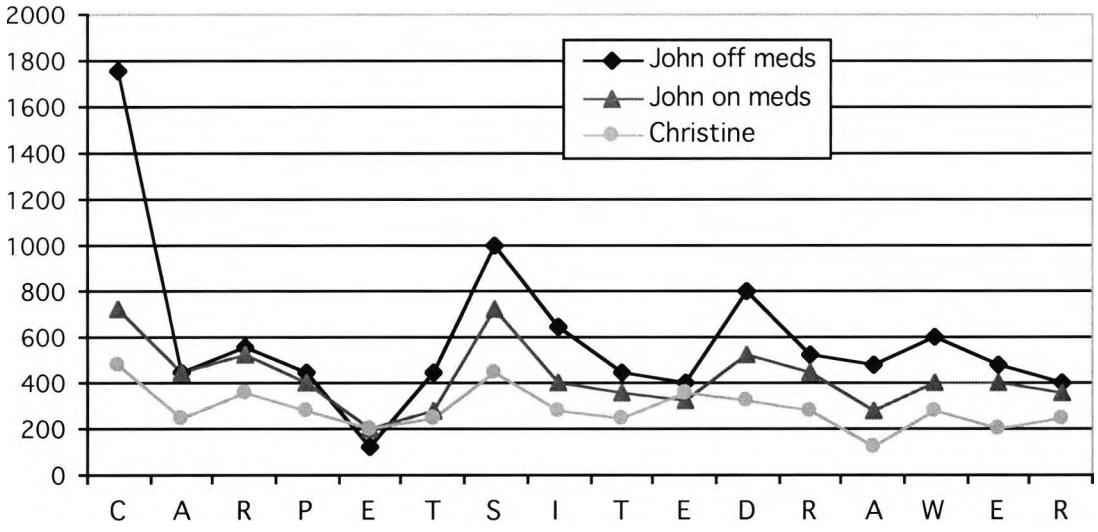
Figure 8.2: Fingerspelling: John



Key: HS: handshape
Loc: location
Inv Move: involuntary movement

Figure 8.3: Fingerspelling Durations: John

(in MSec)



8.6 Non-linguistic Tasks

8.6.1 Pointing

8.6.1.1 Methods: Pointing

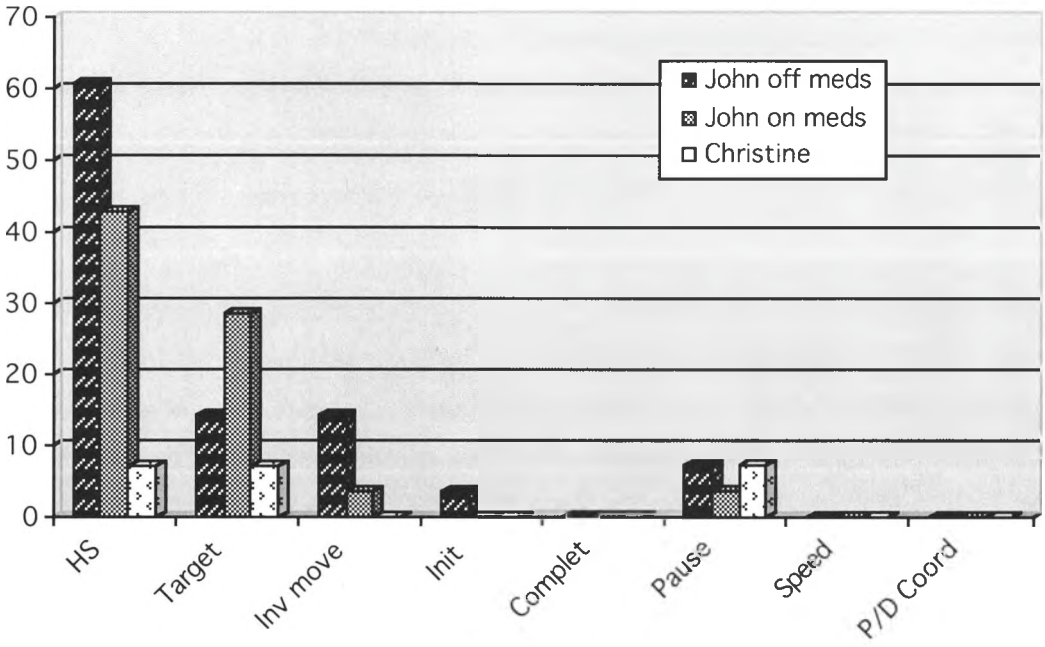
Subjects were asked to point to one of two illustrations in response to a signed utterance produced by the experimenter. The response sheet with two illustrations was placed directly in front of subjects at a comfortable distance. Incorrect responses were excluded from analysis, and neither of the subjects responded to the same stimulus twice. A total of twenty eight responses on the pointing task were analysed for each subject and condition (on- and off-medication); and pointing was coded according to the scheme described in Chapter 4. A coding sample for the pointing task is included in Appendix C.

8.6.1.2 Results: Pointing

As with the previous two tasks, most of John's errors, both on and off medication were in hand configuration (60.71% and 42.85%, or 17 and 12, of total pointing movements, respectively) (Figure 8.4); and they were consistently errors of laxing. Christine had hand configuration errors on only 7.14% (or 2) of total pointing movements. John had no severe timing problems, either with respect to overall speed, or movement initiation, completion, or pausing. Additionally, he had no co-ordination problems, either on- or off-medication.

Figure 8.4: Pointing Task: John

errors as % of 28 productions



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

Targeting errors were John's next most frequent error when he was on medication (28.57%, or 8, out of 28 movements); and targeting errors and involuntary movements were equally the next most frequent errors when he was off medication (for both, 14.29%, or 4). Christine had only 2 targeting errors and no involuntary movements. Perhaps surprisingly, John had twice as many targeting errors when he was on medication as when he was off. By contrast, he had far more involuntary movements off medication than on, although they were not very numerous in either case. Notably, the involuntary movements he produced on the pointing task while off medication occurred during deliberate movement as well as at rest, which is unusual for Parkinsonian tremor in the early stages of the disease. The remaining errors were minimal and did not differentiate John from Christine.

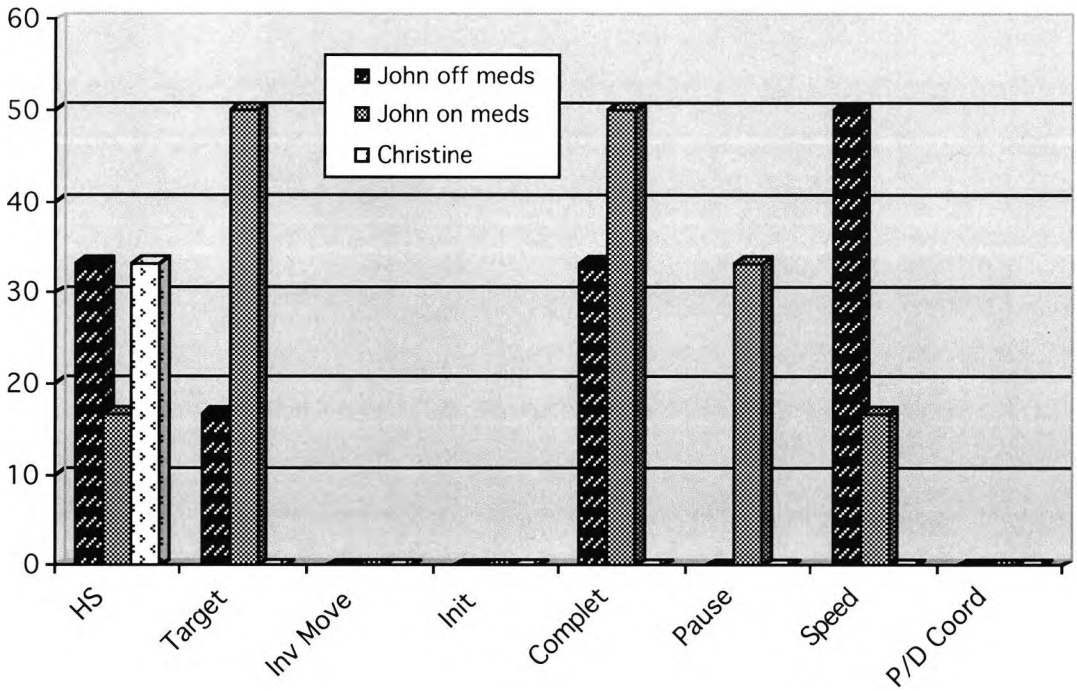
8.6.2 Kimura box

8.6.2.1 Methods: Kimura box

The experimenter presented the Kimura box (see Figure 4.1), 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 Sunderland & Sluman (2000) with additional coding for targeting, hesitation, tremor, speed of execution and accuracy of hand configuration. The test was originally designed to assess apraxic subjects' ability to correctly manipulate objects, according to object shape and size, and according to imitation (Kimura, 1993). The additional categories were added to the coding scheme for this study to allow more detailed assessment of the spatiotemporal aspects of movement as well as their representational and perceptual aspects.

Figure 8.5: Kimura Box: John

errors as % of 6 movements



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

8.6.2.2 Results: 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 8.5). As with the pointing task, 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; though he did have more hand configuration errors off medication (33.33%) than on medication (16.67%). 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 (33.33%). John had a high proportion of completion errors and hesitation errors on this task, which were worse when he was on medication (completion: 50%; hesitation: 33.33%). Additionally, it should be noted that the Kimura box was the only non-linguistic task John performed that required movement sequencing, so it is not easy to separate out the effects of action switching from other mechanically-difficult demands, such as grasping and releasing a handle.

Some of John's errors seem to be specific to individual manipulanda. However, the distribution of his errors is unique. None of his errors under either condition occurred when he was handling the third manipulandum; and there is nothing that would obviously make it easier to handle, except perhaps the fact that it was slightly closer, by virtue of the placement and configuration of the box.

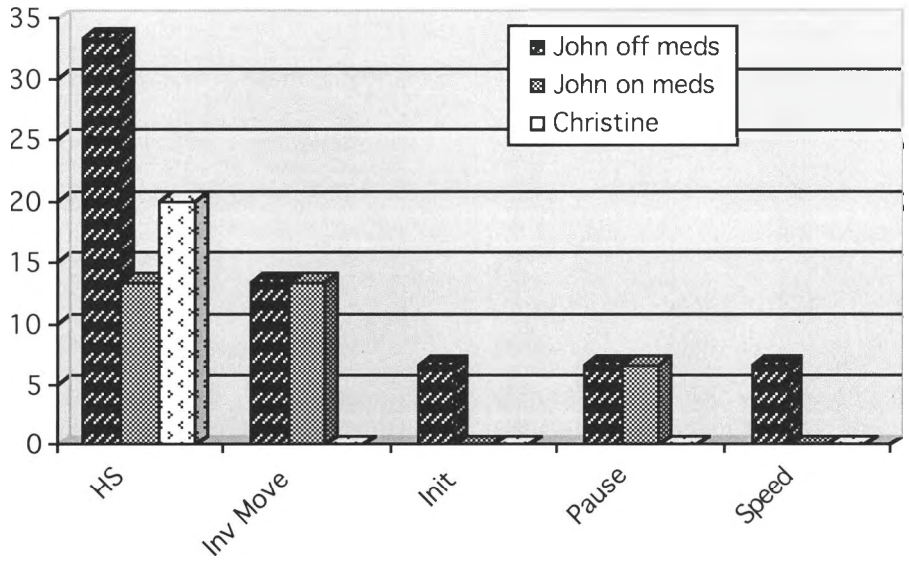
8.6.3 Handshape copying

8.6.3.1 Methods: Handshape copying

The handshape copying task was developed by the Deaf Stroke Project to assess subjects' ability to perceive and produce handshapes in isolation. The handshapes tested were all phonologically possible in BSL, so the test could be used to gauge subjects' ability to produce components of BSL signs, and that result compared to their ability to produce the signs themselves. Subjects were shown 15 individual cards, each depicting a handshape, and asked to produce those handshapes. There were no comprehension problems or spontaneous repetitions of the target movements by either subject, so no data had to be excluded from analysis.

Figure 8.6: Handshape Copying: John

errors as % of 15 productions



Key: HS: Hand Configuration

Inv Move: Involuntary Movement

Init: Initiation

Pause: irregular pause or hesitation

Speed: excessively slow or rapid movement

8.6.3.2 Results: Handshape copying

While both subjects had somewhat unnatural movements, they did not differ that much from each other, particularly when John was on medication. Neither subject repeated any of the handshapes, so there were 15 productions for each subject/condition. As with the other tasks, when John was off medication, he produced more lax handshapes (33.33% of productions, off medication; 13.33%, on medication); however, 13.33% of Christine's handshape productions were laxed as well (Figure 8.6). Both subjects made one handshape error that could be classified as apraxic: they each produced a handshape that structurally did not match the target, as opposed to being simply a laxed or hyperextended version of the same handshape. A handshape that does not match the target might, for instance, have a different number or different configuration of fingers extended or contracted. John made this error while on medication—all of the handshape errors produced when he was off-medication were cases of laxing. Additionally, John produced two involuntary movements during

deliberate movement, under both testing conditions, and a few individual errors of movement initiation, hesitation, and timing, primarily when off medication. These errors only took place once per condition, however, and must be interpreted with caution.

8.7 Discussion

8.7.1 Task-by-task analysis

Signing

Looking at the data qualitatively, John's signing was relatively similar to that of a typical signer. In part, that is because he has good function in both limbs; but additionally, he had fewer errors total on the signing task, and a much higher proportion of them were handshape errors, rather than errors in co-ordination or timing. Although he had more errors when he was off medication, even then, the distribution of errors was roughly the same as when he was on medication, and similar to the pattern of errors produced by Christine, the control signer. His errors differed from hers more in terms of number than in terms of distribution. Most of his errors affected static components of signs and were errors of laxing, i.e. the types of errors that typical signers make during relaxed, informal signing. John had few location errors at all, and those patterned more or less like Christine's location errors, in terms of number and type (i.e. where the produced location was in relation to the target location). He did not have consistent lowering or raising of sign's locations. His signing was observably slow, more so when he was off medication, but not extremely so in either condition. He had no difficulty with co-ordination of the two limbs or of proximal and distal articulators on the same limb, either when he was on or off medication.

John produced very 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 tremor occurring in the hand. Because dyskinesias occur during voluntary movement, while Parkinsonian tremor typically does not, it is not a great surprise that John had more involuntary movements on medication than off medication.

Fingerspelling

John's performance on the fingerspelling task was similar to his performance on the signing task in that his handshape errors greatly outnumbered all other errors, and were more numerous when he was off medication. However, his performance on the fingerspelling task differed in that he had proportionally fewer errors total. Also, the difference between his performance on medication and off medication was not as great on the fingerspelling task as it was on the signing task. The only difference between his performance on medication and off was handshape laxing and slower movements in the latter condition.

It has been 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). Without directly measuring each of those components, it is probably safe to say the same about BSL fingerspelling in comparison to BSL signing. Nonetheless, John was not differentially impaired on the fingerspelling task. He did not have a strikingly 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 Parkinson's disease 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 John's most frequent error across most tasks is in handshape, it could be that the lower demands for handshape in fingerspelling contributed to his overall low rate of errors on the fingerspelling task.

Pointing

John's movements on the pointing task were generally slow, under both conditions. He was slow to respond to the experimenter as well as simply slow to make the required pointing movements. As with most tasks, John's most common error by far, whether he was on or off medication, was in handshape formation. Also, as with other tasks, handshape errors were consistently cases of laxing, and they were fewer when he was on medication than when he was not. When he was on medication, John had more dyskinesias on this task than on the others, especially in his neck and shoulders. Additionally, John had far more involuntary movements off medication than on. Notably, the involuntary movements he produced on the pointing task while off medication occurred during deliberate movement as well as at rest, which is unusual for Parkinsonian tremor in the early stages of the disease. There is no apparent reason why

this should be the case, but it is worth noting for the effect it may have had on his errors. More than on the linguistic tasks, there seemed to be a trade-off between speed and accuracy in the two conditions on the pointing task. John's movements were more accurate when he was off medication, and faster when he was on medication.

Kimura box

John had a high proportion of hesitation errors and completion errors on this task, which may reflect a particular difficulty switching from one action to another, which is a well-established phenomenon in Parkinson's disease (Zalla et al., 1998); however, in his case, they were worse when he was on medication. However, it should be noted that the Kimura box was the only non-linguistic task John performed that required movement sequencing, so it is not easy to separate out the effects of action switching from other mechanically-difficult demands, such as grasping and releasing a handle. In addition to showing a distinct pattern of hesitation and completion errors on this task relative to the other tasks, John also showed a lower proportion of handshape errors than on other tasks. So clearly there is something distinct about the Kimura box task that causes John's movements to take a different structure. That said, however, caution must be exercised in interpreting these findings, because the Kimura box task also had the smallest number of trials of the movement tasks that John performed.

Handshape copying

John did not have many errors on the handshape copying task, relative to the total number of required movements on the task. Moreover, the distribution of his errors was similar to Christine's, and also similar to the distribution of his errors on most other tasks. The majority of his errors both on and off medication were hand configuration errors, and those were mostly cases of laxing. To the extent that John's handshape copying differed from his signing or fingerspelling, Christine's handshape copying differed similarly.

Interestingly, neither John nor Christine performed the handshape copying task very naturally, despite the structural similarity between it and sign production. No explanation of the purpose of the handshape copying task was given in advance; and based on their reactions when the task was explained *post hoc*, neither subject had previously identified the handshapes as possible components of signs. (One of the subjects guessed that they were letters from a foreign fingerspelling system.) Both subjects' movements and use of space in the task seem to reflect the fact that they did

not treat the handshapes as "sign-like." They both produced the target handshapes at distant, high locations, on the peripheral bounds of normal signing space, and moved to those locations very slowly and decelerated very gradually. So even though on this task the target movements and articulator configurations were arguably more sign-like than on any of the other tasks, the movements produced by both the PD and the control signer had a very unnatural quality.

To summarize, John's handshape and hand configuration errors consistently outnumbered his other errors across tasks, with the sole exception of 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 errors of laxing. It is striking that John showed little differentiation in his pattern of errors across tasks, except on the Kimura box. The prevalence of his handshape errors over his other errors remained relatively constant, irrespective of whether a task was linguistic or not, and whether it was motorically difficult or not. For example, the 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 most others.

8.7.2 Comparisons to past sign research

John's movement patterns were similar to those reported from earlier research on signers with Parkinson's disease, though not uniformly so. In his signing, John consistently produced laxed handshapes and sometimes laxed orientations as well. Additionally, his movements were often 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 Parkinson's disease and ASL (Poizner et al., 2000); nor did he produce signs at lowered locations (Kegl et al., 1999). The final important distinction between John and the ASL signers with Parkinson's disease is that he did not show distalization of signs. The subjects from the ASL and Parkinson's disease studies frequently did so, and that finding was one of the most strongly emphasized across publications related to the studies.

One possible explanation for these discrepancies is that earlier studies did not consistently control for the age of the Deaf subjects; consequently, the effects they found could have been an effect of age rather than, or as well as, disease status. Although John did not show a great difference in his signing depending on 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 also be that the deficits they exhibited but John did not were related to the medication itself rather than the disease.

Unlike any of the subjects in the ASL and Parkinson's disease studies (and unlike most subjects in any research on atypical signers), John is a native signer. Due to the lack of data on the qualities of native vs. non-native signing in general, and the lack of data on atypical signers who acquired sign language as a native, it is difficult to speculate about the effects of native language skills on articulation following acquired neural damage. Furthermore, because of the unique situation in which most Deaf signers acquire sign language, they cannot be easily compared to hearing bilinguals who experience neural damage which impacts on their non-dominant or non-native language. Nonetheless, it is not unreasonable to suggest that having native language skills prior to neural damage may work to preserve articulatory ability post-morbid. Further research would be called for to address this question.

8.7.3 Comparisons to speech dysarthria

John exhibited some of the same patterns reported in the speech motor control literature on PD dysarthria. Most notably, because he was in the early stages of the disease, his dysarthric symptoms were not very severe. He had no problems with the co-ordination of the highly-innervated articulators that have the most flexibility of movement (in his case, the hands and fingers; in a hearing person's case, the tongue, jaw, and lips). This is in contrast to the evidence presented by Brentari et al. (1995), which suggested that co-ordination of multiple articulators in particular was impaired in signers with Parkinson's disease. Additionally, he had difficulty initiating movement and had irregular pauses, but less so in signing than in other movement tasks. Similarly, hearing patients with Parkinson's disease do not typically experience movement initiation problems in their speech to the same extent that they do in other movements.

Unlike hearing subjects with PD dysarthria, John did not exhibit anything analogous to festination in sign, though he did exhibit it while walking. Festination is the combined increase in movement in 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 Parkinson's disease that is effector-specific; i.e., it could selectively affect the vocal tract and the legs. What seems more likely, however, is that it is a symptom that impacts on a given type of movement. Speech and gait both seem to be motor systems with an internal oscillator, which sign does not seem to have (MacNeilage et

al., 2000; Meier, 2002); so perhaps festination is particular to this kind of motor system. Similarly, speech and gait movements are effectively constrained to being two-dimensional, which sign movements clearly are not; and this factor could play a role in addition to or instead of the oscillatory mechanism.

Comparison of John's symptoms on and off medication reveal that while his movements were faster when he was on medication, they were not uniformly improved by the medication. In fact, on the pointing task and the Kimura box task, there was a trade-off between speed and accuracy, such that while his movement speed increased on medication, his accuracy simultaneously decreased. Additionally, in some cases, he experienced more involuntary movements while executing a task on medication than off, because his involuntary movements off medication occurred primarily when he was not moving and hence did not interfere with task execution. Finally, without exception across tasks, John's handshape errors were fewer when he was on medication, which is interesting in light of the fact that his movement accuracy was sometimes worse. This supports the idea that movement targets determined by effector configuration and those determined by external reference points are subject to a different set of constraints. Moreover, John's uneven improvement on the signing task when he was on medication was consistent with earlier findings on PD speech dysarthria, which suggested that PD subjects' speech measures improve, but not consistently, and not for all measures (Schulz & Grant, 2000).

The consistency of John's performance across tasks, the similarity of his sign characteristics to the speech characteristics of dysarthric speakers 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 cross-modally it is a symptom that tends not to be severe in the early stages of the disease. However, in addition to showing parallels with hearing subjects with PD dysarthria (no co-ordination deficit, and mild articulatory deficits overall), John patterned differently from those subjects as well. The source of both the similarities and differences in his case and theirs is likely to lie in the movement patterns inherent to the articulatory mechanisms, and those patterns should be examined in greater depth for a better understanding of the nature of both language modalities.

9 Maureen: Signer with Apraxia

This chapter examines the case of Maureen, a Deaf signer who experienced aphasia and apraxia as the result of a left hemisphere CVA. Because of her linguistic deficits, it is solely Maureen's gestures and other non-linguistic movements that were analysed in depth for this study; her production deficit made it impossible to collect enough linguistic data for her signing or fingerspelling to be analysed in terms of articulation. In addition to the mere scarcity of her linguistic production, it would be difficult, perhaps impossible, to determine whether her errors were fundamentally motoric or linguistic (or both) in nature. However, despite the difficulties of analysing articulatory aspects of her signing or fingerspelling, it was thought worthwhile to include her in the study for the analysis of her non-linguistic movement patterns and the contrast they could provide to those of the other atypical subjects. In Maureen's case, the research questions are slightly different: rather than compare her with Deaf or hearing subjects with the same disorder performing similar linguistic tasks, it is of greater interest to describe her movement disorder more broadly, so that in the next chapter her deficits can be compared to those of other atypical signers in this study.

Maureen had a high level movement disorder: she had no impairment in movement timing or dysmetria, but remarkable difficulty with the selection of the correct handshape or movement in producing gestures or meaningless movements. Due to the abstract nature of apraxia, its ambiguous relationship with language and low level movement disruption, as well as the type of data collected from Maureen, this chapter will take a slightly different structure, with a lengthy discussion of the movement disorder itself and a more descriptive analysis of the data.

9.1 Apraxia

Apraxia is generally described as an inability to perform learned, skilled, purposeful movements in the absence of paralysis, weakness or inco-ordination (Liepmann, 1977; Rothi et al., 1991). Because it has no obvious low-level physiological correlates, takes a variety of forms, and is not associated with a single brain region, apraxia's exact nature remains a matter of debate more than a century after it was first documented and described (Liepmann, 1900; Steinthal, 1871). Liepmann divided apraxia into three sub-types: ideomotor, ideational, and limb-kinetic apraxia (Liepmann, 1905, 1920). Ideomotor apraxia affects individual movements as well as movement sequences and is characterized by spatial and temporal errors in movement execution (Rothi et al., 1988). Ideational apraxia causes errors in sequences of movements or

content errors in representational gestures (Ochipa et al., 1992). Limb-kinetic apraxia is qualitatively different from the first two, because it is characterized by the inability to produce fine-grained, individuated finger movements, and probably results from the loss of innervation to proximal effectors from damage to corticospinal neurons, rather than affecting motor programming, stored representations of movements, or sensorimotor integration more generally. For many researchers now, limb-kinetic apraxia is treated as fundamentally distinct from ideomotor and ideational apraxia, which continue to be grouped together (Heilman & Rothi, 1997). It is worth making an additional point about terminology here: although the etymology of the terms does not imply such a distinction, dyspraxia, while sometimes used interchangeably with apraxia (e.g., Lausberg et al., 2003) is used primarily to refer to a developmental disorder similar to acquired apraxia and affecting both gross and fine movements (Morris, 1997). Except where otherwise specified, the discussion in this chapter will address ideomotor apraxia.

Apraxia is most often associated with damage to the left hemisphere, usually to the frontal or parietal cortex (Halsband et al., 2001; Hanna-Pladdy et al., 2001; Liepmann, 1905, 1920), but has been documented in cases of damage to left temporal cortex (Goldenberg et al., 2003), and the right, non-dominant hemisphere (Heilman et al., 1973; Raymer et al., 1999). Additionally, forms of apraxia have been reported to occur in conjunction with subcortical damage (Crosson, 1997), corticobasal degeneration (Leiguarda et al., 2003; Merians et al., 1999), Alzheimer's disease (Capone et al., 2003), PSP (Pharr et al., 2001), Huntington's disease (Hamilton et al., 2003), and disconnection of the two hemispheres (Lausberg et al., 2003). However, in many of these studies, the deficit being described has little in common with earlier definitions of apraxia.

Attempts have been made to correlate individual types of apraxia with specific brain regions. Bearing in mind the complicating factors of variable use of the term apraxia and the prevalence of apraxic subjects with additional semantic or movement disorders, recent research has made some progress towards clarifying the relationship between anatomy and function. Hanna-Pladdy et al. (2001) suggest that subjects with cortical apraxia exhibit deficits in gesture discrimination, imitation, and production following verbal command, while subjects with subcortical apraxia exhibit mild production deficits but retain gesture discrimination and imitation. Halsband et al. (2001) reported a similar dissociation, but between subjects with parietal and frontal lobe lesions, such that parietal lesions disrupted both gesture comprehension and

production, while frontal lesions (specifically damaging supplementary and/or premotor areas) disrupted only production. Since Hanna-Pladdy et al. (2001) did not control for cortical lesion site, these two studies may be tapping into the same phenomenon, particularly in light of the substantial projection from the basal ganglia to the supplementary motor area via the thalamus (Brodal, 1998).

Another study suggests there may be different symptoms of apraxia connected with subcortical damage. In particular, a case study of gesture production suggests that apraxia resulting from corticobasal degeneration results in greater impairment of gestures produced with an object in hand than impairment of gestures produced with an imaginary object (Merians et al., 1999). Studies of left hemisphere apraxia typically report the inverse result, with subjects more impaired when they have to imagine manipulating an object than when they are actually holding it (Halsband et al., 2001).

9.1.1 The nature of apraxia

Beyond the question of symptoms or neural correlates, there are multiple competing theories of the actual nature of ideomotor apraxia, and by extension, of the concept of praxis in human movement. There is still much disagreement about what apraxia is in fact a deficit *of*. There is fairly widespread agreement that ideomotor apraxia is not fundamentally a deficit of motor execution, but rather some sort of deficit in correctly generating a movement matched to the particular demands of an intended goal. Within that categorization, there is room for debate on the level at which the movement is disrupted and the aspects of the goal that are most demanding. There are those who propose that apraxia is a deficit in the storage or retrieval of motor programmes (Jeannerod & Decety, 1995; Poizner et al., 1995; Rushworth et al., 1998; Sirigu et al., 1999), similar to Liepmann's view of apraxia as a disorder of action rather than a disorder of movement or semantic representation (Liepmann, 1905). The main contrastive view places more emphasis on spatiotemporal demands of complex movements and suggests that apraxia is fundamentally a deficit of representation or formation of complex hand or effector configurations and integration of these into movement sequences (Buxbaum et al., 2003; Goldenberg, 1995; Haaland & Harrington, 1994; Hermsdorfer et al., 1996; Kimura, 1993; Sunderland & Sluman, 2000).

9.1.2 Kinematics of apraxia

Although it is defined as a disorder of skilled movement, several studies have suggested that subjects with apraxia exhibit low level deficits as well, including inco-

ordination and reduced speed in the execution of gestures with and without tools (Merians et al., 1999; Poizner et al., 1997). Poizner et al. (1997) suggested that, contrary to previous findings, patients with ideomotor apraxia exhibit deficits on object use as well as pantomime, and those deficits are apparent when one examines the kinematic data. Similarly, Hermsdorfer and Goldenberg (2002) reported impairments in movements of the ipsilateral limb following stroke, not only in terms of apraxia, but also impairments in speed and amplitude on a diadochokinesis task. By contrast, another study reported that apraxic subjects produced normal movement kinematics and endpoint accuracy on simple reaching tasks with vision and from memory (Ietswaart et al., 2001).

Findings related to movement kinematics in subjects with apraxia are important because they may reveal aspects of the disorder that are otherwise imperceptible. In particular, they may indicate that apraxic subjects who seem to show a selective deficit affecting certain types of movement but not others (e.g. pantomime but not tool use), are in fact impaired on multiple types of movement. However, these results do not negate the significance of earlier findings indicating that there are distinct types of apraxia, with differential effects on different movement tasks. The fact that it takes very precise measurement to reveal one type of deficit while the other is readily visible only highlights the fundamentally different nature of the two behaviours. Kimura (1993) makes a similar point with respect to earlier research on movement timing in subjects with left or right hemisphere damage, and also points out that slowing of movements was present in subjects with left hemisphere damage, irrespective of whether or not they were apraxic. Kinematic measures can provide useful information about apraxia, even though its characteristic features are by definition removed from low level movement deficits, but that information must be interpreted in relation to the bulk of research on non-kinematic measures of apraxia, and not as a replacement to it.

9.1.3 Apraxia, effectors and categories of action

Apraxia was originally identified as a high level disorder affecting complex, skilled movements in general, but it was (and mostly still is) typically analysed in terms of the movements of the hands and arms. So while it was examined via a particular set of effectors, it was viewed as a general deficit of skilled movement that was most apparent in the movements of the hands, because they are the effectors used by humans to execute most complex movements. More recently, however, the meaning of apraxia has broadened to include non-skilled, automatic movements; and at the same time,

numerous narrower deficits specific to certain effectors or certain actions have been labeled as apraxia, with a descriptor indicating which effector or action is impaired. In contrast to the original definition, the term apraxia can now be applied to non-skilled or task-specific movements. Consequently, there are now conditions described as dressing apraxia (Fitzgerald et al., 2002), gait apraxia (Della Sala et al., 2002), eyelid opening apraxia (Lamberti et al., 2002), and cough apraxia (Stephens et al., 2003). As Heilman and Rothi (1997) point out, in most cases, these deficits are not related to apraxia as it has been traditionally defined, either psychologically or neurologically. By contrast, two conditions that are both psychologically and neurologically related to traditional manual apraxia are apraxia of speech and oral apraxia, because the supralaryngeal vocal tract, like the hands and arms, allows the production of a broad variety of complex, learned movements, and is used for symbolic and meaningless gesture.

9.1.4 Apraxia of Speech

Apraxia of speech is a disorder of speech articulation, distinct from both aphasia and speech dysarthria, which results from damage to motor structures of the left cerebral hemisphere. Oral nonverbal apraxia, distinct from apraxia of speech, is defined as an impairment of volitional non-linguistic mouth movements while spontaneous movements are retained. The relationship between oral nonverbal apraxia and apraxia of speech is not completely clear, and the two often co-occur; however, there have been cases of each independent of the other (Duffy, 1995). Darley et al. (1975) suggested that apraxia of speech resulted from disruption to the motor speech programmer, which was thought to be located in the left hemisphere. In a review of the literature, Dronkers (1996) reported that the one pathology that patients with apraxia of speech share, irrespective of anything else, is damage to the left insular cortex. However, it has been rightly pointed out that this finding in and of itself is not sufficient to explain the neuroanatomical correlate(s) of apraxia of speech, and the neural basis for the disorder could easily be more narrow, broad, or diverse than Dronkers (1996) suggests (Bennett & Netsell, 1999; Code, 1998; Miller, 2002). Along similar lines, and in support of the idea that apraxia of speech and aphasia are distinct from each other, Wise et al. (1999) found that the left insula, lateral PMA, and basal ganglia are active during articulation, but Broca's area is not. Because of its neural correlates, apraxia of speech may be accompanied by aphasia; however, it is a disorder of speech rather than language, as indicated by preserved linguistic function in cases in which it has occurred in isolation (Wertz et al., 1998).

What distinguishes apraxia of speech from dysarthria is the nature and resulting characteristics of the movement disorder. In contrast to dysarthric speakers, individuals with apraxia of speech exhibit no significant weakness, slowing, changes in muscle tone, or loss of reflex movements in the speech musculature. Moreover, their errors tend to be phonological rather than phonetic in nature, with substitutions of one phoneme for another, difficulty initiating speech, and dysprosody as typical features (Wertz et al., 1998). Broadly speaking, apraxic speech deficits tend to be more variable across productions and more sensitive to the prosodic, articulatory, and discourse context of utterances than dysarthric speech deficits. For instance, apraxia of speech often causes inconsistencies across multiple productions of the same utterance, such that errors are frequent but typologically variable. At the level of articulatory context, physical distance between speech segments influences error production in apraxia of speech more than in dysarthria. Additionally, automatic, imitative, and short productions are more error free than longer, spontaneous productions. Similarly, non-words are more error free than real words (Wertz et al., 1998). All of these taken together support the idea that apraxia of speech is a deficit of motor programming, distinct from dysarthria, which is more often a deficit of motor execution.

The speech characteristics and neural correlates of apraxia of speech have important implications for theories of speech motor control and the relationship between speech and limb movements. First, the deficits that appear in apraxia of speech illustrate that speakers rely on knowledge of where their articulators are at any given time to program speech movements. This is noteworthy because it contradicts the suggestion that speech, unlike limb movements, relies primarily on ballistic movements and therefore open-loop programming (Ackermann et al., 1997). In their description of apraxia of speech, Square et al. (1997) emphasize the importance of sensorimotor integration to rapid, accurate speech production. This is consistent with an earlier model which suggests that somatosensory input may underlie the time critical aspects of orofacial motor control for speech (Gracco & Abbs, 1987). The view that apraxia of speech is a deficit in motor speech programming, caused by inability to integrate sensorimotor information is supported by the finding that apraxia of speech results from damage to the left insular cortex, which projects heavily to the supplementary motor area, an important region for motor programming (Bennett & Netsell, 1999; Dronkers, 1996). In summary, the speech characteristics and neural correlates of apraxia of speech suggest that speech motor control is not as distinct from limb motor control as has often been assumed; both types of motor control require rapid, precise sequences of

movement, rely on sensory feedback and motor programming, and can break down in the absence of low level movement disorders.

9.1.5 Apraxia and aphasia

The relationship between apraxia and aphasia remains an interesting and difficult question, complicated by the fact that the two often co-occur clinically. While it is possible for aphasia to occur in the absence of apraxia and vice versa (Hanna-Pladdy et al., 2001; Heilman et al., 1973; Pedelty, 1987), because the relevant brain areas are near to each other and vascular accidents often do not respect functional boundaries, a large proportion of patients with one deficit also exhibit the other. Though apraxia and aphasia often co-occur, the theory that they are fundamentally the same disorder (as put forward by Kimura & Archibald (1974)) has been more or less abandoned. Indeed, psycholinguistic research has revealed both correlates and dissociations between the characteristics and severity of apraxia and aphasia (Halsband et al., 2001; Hanna-Pladdy et al., 2001), suggesting they are not one and the same disorder. In a fairly large clinical sample, Goldenberg et al. (2003) discovered a closer correlation between disruption to representational gesture and aphasia than between disruption to representational and meaningless gesture production. In other words, in patients who were apraxic and aphasic, their inability to produce representational gestures (e.g. 'waving goodbye') to command was closely correlated to the severity of their aphasia but not to their inability to imitate meaningless gestures. (The authors reject the interpretation that subjects' impairment in producing gestures to command is simply an effect of their not being able to follow verbal instructions.) Along similar lines, in a series of case studies, Pedelty (1987) found similarities not only between the severity of aphasia and impaired gesture production but also between the characteristics of the two disorders. Her research suggests that patients with Broca's aphasia are more likely to have scarce, halting gestures, while patients with Wernicke's aphasia are more likely to have fluidly-moving gestures without much semantic content. In contrast to studies showing correlations between aphasia and apraxia, Heilman et al. (1973) documented a unique case in which a left-handed patient with severe right hemisphere damage experienced apraxia but not aphasia, suggesting that he was left hemisphere dominant for language but right hemisphere dominant for praxis, which is as neuroanatomically distinct as the two functions could be from each other.

Beyond these apparent contradictions, though, the interaction between apraxia and aphasia is arguably more complex and subtle than has often been acknowledged.

For instance, it is sometimes assumed, in the absence of evidence in support or to the contrary, that patients who cannot communicate verbally are nonetheless completely able to do so using gestures of the hands and arms (Goodwin, 2000). Conversely, it is also assumed that if an apraxic subject understands task instructions once, then their performance on the task is at no point likely to be impeded by disrupted communication (Goldenberg et al., 2003). Additionally, as described above, the meaning of the term apraxia is extremely variable from one study, research lab, or hospital to another, which makes comparing it to other deficits considerably more difficult.

9.1.6 Apraxia and sign language

Early research on sign language and the brain was directed toward examining what could differentiate a disorder of language from a disorder of gesture in a manual-gestural language (Corina et al., 1992a; Poizner et al., 1987). Historically, production aphasia had been thought of as a disorder that manifested itself via the vocal tract, while apraxia was thought to manifest itself primarily in the movements of the hands. Consequently, languages that use the hands as their primary articulators present a unique challenge to this paradigm. Inversely, so do complex motor deficits of speech; however, the idea that there is a motor component to speech has traditionally been more widely accepted among researchers than the idea that language could be produced with the hands (Broca, 1861; Stokoe, 1960). As a result, the discovery of a double dissociation between apraxia and aphasia in Deaf signers had enormous implications for psycholinguistics as well as sign language research (Corina et al., 1992b; Poizner et al., 1987).

Given that there are two separate phenomena of apraxia and aphasia for sign language users, the question then arises: what is apraxia of sign? Or perhaps more crucially, is there such a thing as apraxia of sign? First of all, it should be noted that strictly speaking such questions are beyond the scope of this research, because there is only one subject being examined, and she is aphasic as well as apraxic. Consequently, these issues can only be discussed briefly and hypothetically, but they nonetheless merit discussion for the perspective they provide on the case described here and the directions they suggest for future research. The first point to be made is that to uncover a phenomenon like apraxia of sign would require the discovery of a highly atypical case: a signer (preferably native) with left hemisphere damage, intact linguistic ability, intact representational gestures and disrupted sign articulation.

Based on the pattern of deficits in apraxia of speech, the characteristics of apraxia of sign would be articulatory inconsistency, irregular phonemic substitutions (e.g. replacing one handshape with another), and particular difficulty with lengthy, spontaneous signed utterances. It should be noted that many of these have been treated as symptoms of aphasia in sign language (Brentari et al., 1995; Poizner & Kegl, 1993), so analyses of the relevant data would have to be meticulous. Regarding methodology, in order for issues like articulatory inconsistency to be adequately explored in sign, kinematic measures of sign movements would have to be taken over repeated productions. While it is relatively easy to judge speed, co-ordination, and hand configuration from casual observation or videotape analysis, very detailed, precise measures would have to be taken to determine whether or not those values are consistent across multiple productions of the same sign.

Finally, it should be acknowledged that apraxia of sign may simply not exist. The greater temporal and co-ordination demands of speech may imply greater motor programming demands, which would render speech more susceptible than sign to disorders like apraxia. Additionally, apraxia of speech, while similar to limb apraxia, may be effector-specific.

9.2 Maureen: Background

Maureen is a 72 year old right-handed woman who was born into a hearing family and deafened by meningitis at the age of 18 months. She began to learn BSL at the age of 5 when she entered the local oral school for the deaf and has continued to use BSL as her preferred language. She was married twice, both times to Deaf men who were BSL signers. Additionally, she was active in the local Deaf community and had many Deaf friends. At the same time, Maureen's family reports that her pre-morbid use of English was very good; she read newspapers and magazines regularly, and Deaf friends often called upon her to facilitate communication with hearing people. After leaving school, Maureen worked as a hairdresser and in a pub, both jobs that required knowledge and use of English.

At the age of 70, Maureen had an anterior left hemisphere CVA, which CT scan data indicate was in the territory of the middle cerebral artery. Her symptoms following the CVA included right hemiparesis, and severe apraxia and aphasia. She had no spontaneous language production, and her naming and copying abilities on tests of spoken and signed language were severely impaired. Additionally, her language perception was disrupted, with even single sign identification impaired. Maureen's

aphasia and response to therapy are described in greater detail elsewhere (Marshall et al., under review).

9.3 Deaf Stroke Project Testing

9.3.1 Clinical Observations

Following her stroke, Maureen had right hemiparesis, which prevented her from walking or from using her right arm at all, even as a base arm for signing or handling objects. She had some difficulty swallowing, and was consequently on a soft-food diet, but could feed herself. For most aspects of personal care, she required assistance; however, like many individuals with apraxia, Maureen had less difficulty with day-to-day movements than her experimental test results might imply. She could drink tea, turn lights on and off, and put her glasses on and put them away in their case, all with minimal difficulty. Her manipulation of objects outside the context of testing was occasionally clumsy, but more as a result of her contralateral hemiparesis than of her apraxia.

9.3.2 Sign Language Testing

Researchers with the Deaf Stroke Project tested Maureen on a range of linguistic measures to assess the nature of her aphasia and devise appropriate therapy. Generally speaking, both her production and comprehension of written, spoken, and signed language were severely impaired, but her production was comparatively worse. She produced no signs spontaneously and could not name pictures or imitate signs, even when cued. Her sign comprehension was also impaired, even at a very basic level. On a test of comprehension of individual signs, she scored 25/40.

Strikingly, following her stroke, Maureen's speech remained more intact than her sign. Several times during testing, when she was presented with an image and asked to produce the sign for it, she would name it in English instead. What is perhaps even more striking is that phonological cueing for a BSL sign could trigger Maureen's production of the spoken English word, when presentation of the object or image to be named did not do so. Moreover, giving a phonological cue in BSL did not facilitate her production of the BSL sign. Evidently, providing the phonological cue in BSL allowed Maureen to access the linguistic system at some level and generate a response to the information she was given, despite the fact that the cue and the output were not only from different languages but also from different modalities (Marshall et al., under review).

While Maureen was not able to copy signs on command, there were instances during testing by the Deaf Stroke Project when she seemed to be copying handshapes inadvertently. On tests of sign comprehension or copying, she would imitate the experimenter's handshape, which in some cases made it difficult for her to select an object on a naming task, because her hand would be configured incorrectly. Additionally on a few occasions, when Maureen indicated that she could not produce a sign because it was two-handed and she was hemiplegic, the experimenter would suggest that she use the experimenter's hand or limb as the base hand. However, when Maureen tried to do so, she would invariably produce the same handshape that the experimenter was maintaining, irrespective of what the target handshape was. This may be related to the finding by Sirigu et al. (1999) that some apraxics have difficulty differentiating their own hand from the experimenter's hand presented on a monitor, when the two have the same hand configuration.

9.3.3 Neuropsychological and Apraxia Testing

In the course of assessment by the Deaf Stroke Project, Maureen was presented with a series of background neuropsychological tests, some of which she could perform and others not. Because of her communication difficulties, she was unable to do Raven's Progressive Matrices™ (Raven & Raven, 2003) or the Benton line orientation test (Hamsher et al., 1992). On the Pyramids and Palm Trees test of semantic access (Howard & Patterson, 1992) she scored well below normal (39/52). Similarly, on the WAIS-III™ Block Design test (Wechsler, 1997), she had a raw score of 16, which is significantly impaired. By contrast, she performed at ceiling level on a line cancellation task, indicating that she had no hemispatial neglect.

Maureen was tested for apraxia and gesture comprehension and production as well. The Dabul Subtest III (Dabul, 2000), which probes for limb and oral apraxia, was modified so that it could be administered using demonstrations rather than verbal commands, thereby circumventing Maureen's comprehension deficit and the difficulties of translating an apraxia test into a signed language (for discussion of the latter issue, see Chapter 4). As a result, her possible scores were lowered, such that the maximum possible score for either section of the test was 20. Test results indicated that Maureen exhibited both limb and oral apraxia, though the former was much more severe. On the reduced scale, she scored 4/20 on the limb apraxia section, and 13/20 on the oral apraxia section. Similarly, on a test of imitation of meaningless gesture (using only her left, non-hemiplegic hand), she scored 13 out of 24 (Kimura & Archibald, 1974).

The Deaf Stroke Project designed a task to assess Maureen's comprehension and production of transitive gestures, i.e. gestures used to manipulate objects or tools. For the production component of the task, she had to produce the appropriate gesture for a given object, with the object in hand. For the comprehension component, she had to watch the experimenter produce a gesture and choose the correct object to match the gesture by pointing to a picture or an object. On the basis of these measures, her comprehension of gesture was intact, though her gesture production was severely impaired, which is consistent with what has been found in subjects with left hemisphere lesions to the supplementary motor area or premotor area (Halsband et al., 2001).

9.4 Non-linguistic Tasks

Because Maureen had so little spontaneous sign production and only a limited ability to copy signs, she was only tested on non-linguistic tasks for this study. Her movements on non-linguistic tasks were analysed according to the same measures used for other subjects, as well as on more qualitative measures for tests of object manipulation.

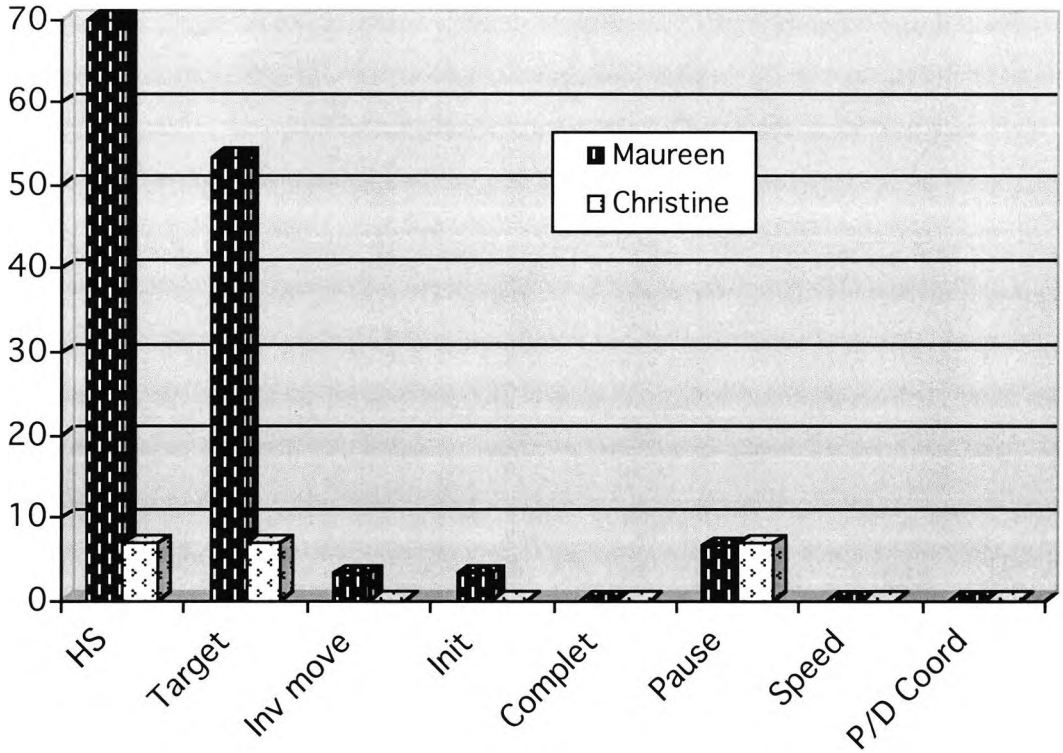
9.4.1 Pointing

9.4.1.1 Methods: Pointing

The design of the pointing task for Maureen was re-structured to allow for her linguistic deficits. Rather than being asked to point to one of a set of images in response to a signed utterance, she was asked to point to one of a set of handshape cards in response to a handshape that was presented to her. Because the timing on the pointing task was measured from the onset of movement rather than from the end of stimulus presentation, the effect of language processing difficulty on movement timing should be minimal. Judging from Maureen's performance, language processing difficulty had no effect on movement speed as it is defined for this study. The first 30 of Maureen's pointing movements were compared to the first 30 pointing movements produced by Christine, the control subject.

Figure 9.1: Pointing Task: Maureen

errors as % of 30 responses



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

9.4.1.2 Results: Pointing

Figure 9.1 shows the distribution of errors for the two subjects on the pointing task, as a percentage of the total number of their productions. Maureen produced many more movement errors than Christine, though she did not produce a broad range of errors. Most of Maureen's errors on the pointing task were hand configuration errors, which were present on 70% (or 21 out of 30) of her pointing movements. Christine had hand configuration errors on only 6.89% (or 2 out of 30) of her pointing movements. Also in contrast to Christine, Maureen's hand configuration errors were related to finger selection rather than laxing or hyperextension. Maureen chose unusual hand configurations for a pointing action. For example, in one case, she extended the index finger, the little finger and the thumb, in a slightly clawed configuration.

Some clarification is necessary when describing hand configuration errors in a task that has no particular demands on how the hand is configured: Maureen was not asked to hold her hand in any specific configuration, but simply to point to the correct pictures. So while there were no explicit demands on how the hand should be configured, other subjects in the study (both control and atypical subjects) almost always used the common hand configuration of an extended index finger with the other fingers flexed, with the thumb sometimes extended and sometimes not.

Maureen's next most common type of error was in targeting. On 53.33% (or 16) of her movements, there was a targeting error, compared to 6.89% (or 2) of the movements produced by Christine. Maureen's targeting errors were consistently cases of undershoot; and many times, she inadvertently caused the card to move as a result of difficulty with targeting.

Table 9.1 illustrates the distribution of errors on the pointing task for both subjects, according to individual movements, in order to show the extent to which errors clustered on individual movements, or conversely, whether errors were decoupled from each other, so that movements that were spatially precise were also slower, for example. Each row in the table represents the individual pointing movements produced by the two subjects in the order they were produced. However, it should be remembered that the two subjects were not performing the same movements in the same order, so comparisons cannot be made across the two subjects for an individual movement. The table is designed as it is solely to allow comparisons of the distribution of errors for each of the subjects individually. Because neither subject had any errors of speed, proximal/distal co-ordination, or movement completion, those categories were left off the table due to formatting considerations.

Table 9.1: Distribution of Pointing Errors: Maureen

Maureen's Errors					Christine's Errors				
HS	Target	Inv Move	Init	Pause	HS	Target	Inv Move	Init	Pause
X						X			
X									
X				X					
				X					
X									X
X	X								
X	X								
X	X								
X			X						
X	X								
X									
									X
X	X								
X	X				X				
X									
	X								
	X								
X	X				X				
X	X					X			
X									
X	X								
	X								
X	X	X							
X	X								
	X								
X	X								
X									

Christine had very few errors on the pointing task; moreover, her errors did not cluster on individual movements. In fact, she did not produce a single pointing movement with more than one type of error. However, because she had so few errors overall, it is not possible to infer that the errors are in complementary distribution. Likewise, it is not possible to infer much about the distribution of Maureen's errors, in her case because there were so many hand configuration errors that they cannot be said to be in either complementary or co-extensive distribution with other types of errors. In the only case in which a comparison is possible, i.e. between Maureen's hand configuration errors and targeting errors, it is not readily obvious that the two bear any relationship to each other.

9.4.2 Kimura box

9.4.2.1 Methods: Kimura box

The experimenter presented the Kimura box (see Figure 4.1), performed an action on each of the three manipulanda in sequence, and asked Maureen to copy her actions, in the same order and using the same hand configurations. The data were analysed according to the criteria set by Sunderland & Sluman (2000), with additional coding for targeting, hesitation, tremor, speed of execution and accuracy of hand configuration. The test was originally designed to assess apraxic subjects' ability to correctly manipulate objects, according to object shape and size, and according to imitation (Kimura, 1993). In the original design of the task, experimenters are supposed to get a subject to produce a series of movement sequences correctly. For this study, the objective was to get subjects to produce two correct sequences in a row. Because Maureen had great difficulty performing the task without errors, she produced many more movement sequences than Christine; so the error and timing analyses compared 6 individual movements for Christine (2 sequences of 3 actions) with 21 individual movements for Maureen (7 sequences of 3 actions). The methodology differed slightly from what was used by Sunderland and Sluman (2000), in that: durations were measured for individual components of the task rather than for the task as a whole; and two, rather than three, correct movement sequences were required.

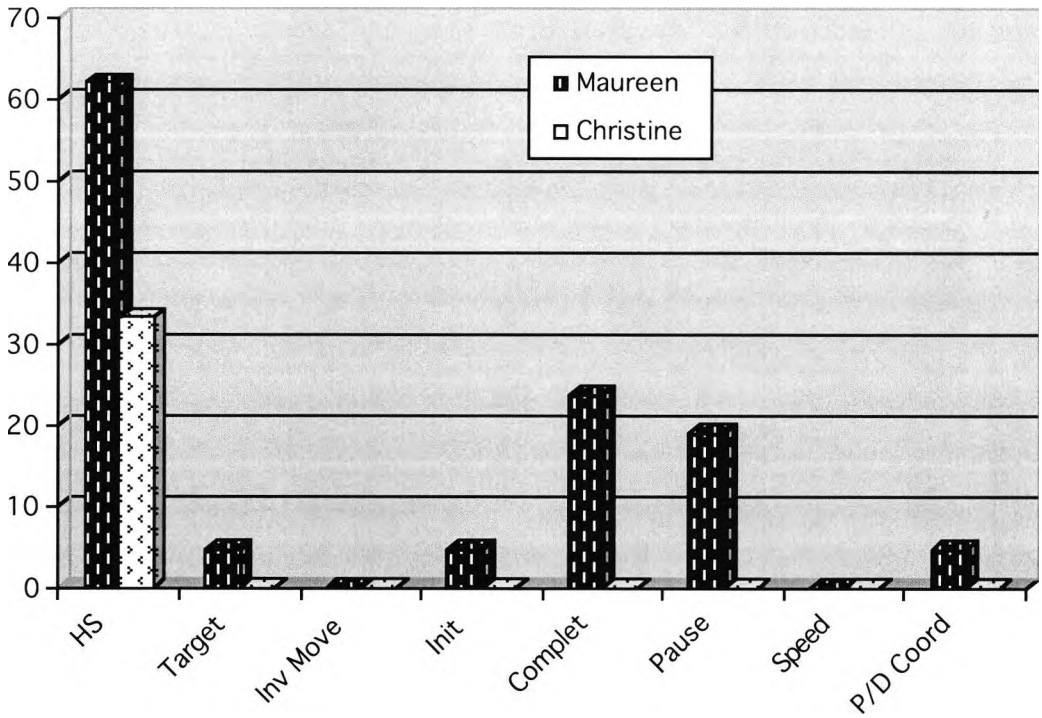
9.4.2.2 Results: Kimura box

On the Kimura box task, Maureen produced many more errors per movement sequence than Christine; and most of her errors were in hand configuration (Figure 9.2). Approximately 61.90% (13 out of 21) of Maureen's actions included a hand configuration error, as opposed to 33.33% (2 out of 6 actions). Additionally, Maureen's hand configuration errors were not laxed or extended hand configurations, but rather errors in the selection of which fingers to extend.

Maureen's next most common error was in action completion. Approximately 23.81% (5 out of 21) of her actions included a completion error, compared to no completion errors for Christine. On this task, a completion error meant that Maureen continued holding, depressing, or otherwise handling a manipulandum for an exceedingly long time before beginning the next action on the task. Unlike her hand configuration errors, her completion errors were not specific to individual manipulanda.

Figure 9.2: Kimura Box: Maureen

errors as % of total responses



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 9.3 indicates the durations of each of the target actions performed by Maureen and Christine in their execution of the Kimura box task. As previously stated, Maureen performed the task many more times than Christine, because she had difficulty producing two correct sequences of three actions. (In fact, she was not able to do so at all, so the experimenter ended the task.) The durations of individual movements on the task were measured from movement onset to contact with the manipulandum. The starting positions for each movement were not controlled, so subjects could begin the first action of the task from anywhere. However, because the three actions were consistently performed in the same order, the second and third actions clearly must have begun from the same locations.

Because of this task design, it is not surprising that the first movement in each sequence would take the longest—while it could be beginning from anywhere, it is most likely beginning from somewhere not on the box, so the hand probably has to travel a greater distance to reach the first manipulandum. Maureen, as well as Christine, consistently took longer to make the first movement in the sequence than either of the other two movements; however, the duration of the third movement for Maureen was variable, sometimes taking longer than the second movement and sometimes not. On both of Christine's two sequences of actions, the movement for each action in turn was shorter in duration. Moreover, there was more variability generally in the durations of Maureen's movements. Additionally, there was no practice effect for Maureen: her performance of the task did not become more rapid with repeated productions. Whereas Christine became noticeably quicker in her second execution of the three movements. That said, it should be noted that Maureen was not slower than Christine in all cases.

Figure 9.3: Kimura Box Timing: Maureen

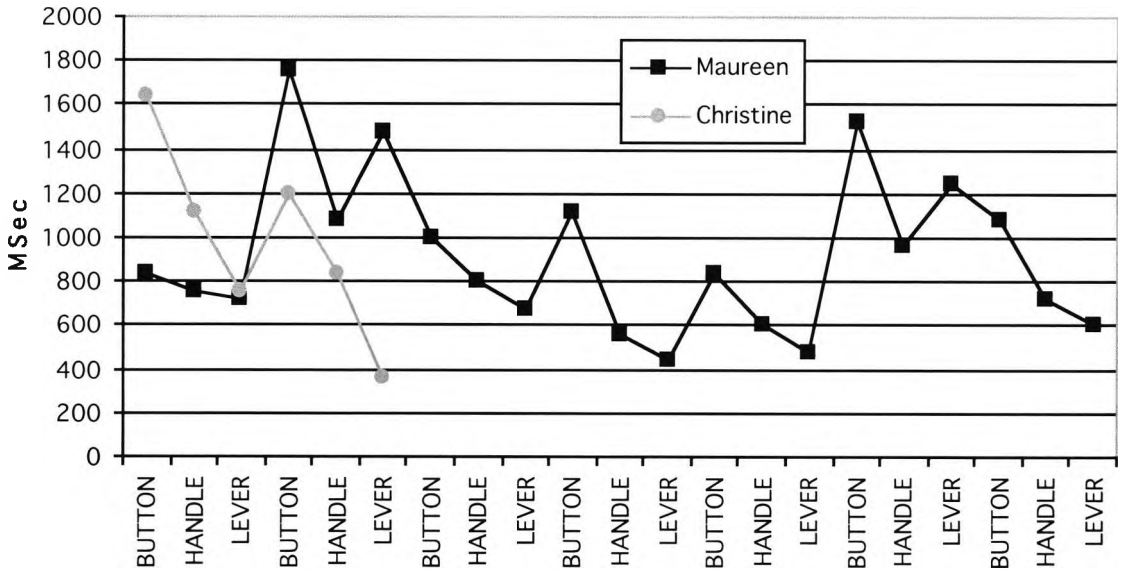


Table 9.2 shows the distribution of Maureen's errors on the Kimura box task. Individual movements on the task were always performed in the same order, so it is possible on this table to see the errors both in relation to each other, and in relation to the specific manipulanda. Because Maureen had so many hand configuration errors, it is hard to judge their distribution relative to other errors. There seems to be a pattern in the distribution of hesitation and completion errors, though, in that they cluster together several times, and also occur primarily in the first two trials of the task, suggesting that the two errors may co-occur and reflect the same underlying deficit. Notably, Maureen's hand configuration errors only occurred on the first and third manipulanda and never on the second. Because Christine had only one type of error (i.e. hand configuration) and because she performed far fewer movements than Maureen, her errors were not included on the table. However, bearing in mind the small numbers, it should be noted that Christine's handshape errors were only on the first manipulandum, and were errors of laxing rather than finger selection. Like Maureen, her errors were specific to individual manipulanda on the box.

Table 9.2: Distribution of Maureen's Kimura Box Errors

Manipulandum	HS	Target	Inv Move	Init	Complet	Pause	Speed	P/D Coord
Button	X							
Handle					X	X		
Lever	X				X			
Button	X				X	X		
Handle					X	X		
Lever				X				
Button	X							
Handle								
Lever	X							
Button	X							
Handle								
Lever	X							
Button	X							
Handle								
Lever	X							
Button	X	X				X		
Handle								
Lever	X							X
Button	X				X			
Handle								
Lever	X							

9.4.3 Handshape copying

9.4.3.1 Methods: Handshape copying

The handshape copying task was developed by the Deaf Stroke Project to assess subjects' ability to perceive and produce handshapes in isolation and in the context of actual signs. The handshapes used as stimuli were all phonologically possible in BSL, so the test can be used to gauge subjects' ability to produce or perceive individual phonological parameters of BSL signs, and that result could be compared to their ability to produce or perceive the signs themselves. Because Maureen had great difficulty with this task, she was not tested on as many individual handshapes as Christine was. In total, Maureen copied 8 handshapes, while Christine copied 15 handshapes.

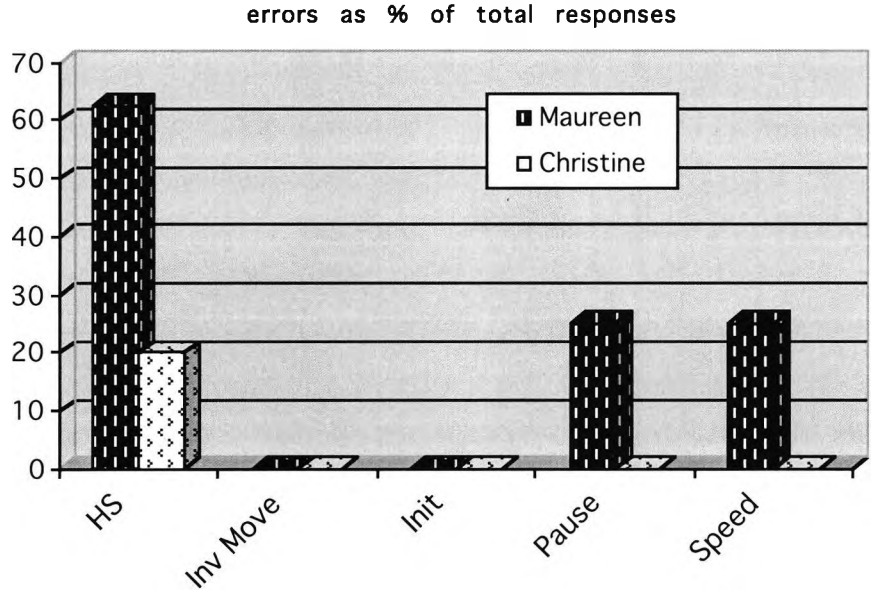
9.4.3.2 Results: Handshape copying

The most common of Maureen's errors on the handshape copying task were in hand configuration, with a particular deficit in selecting the correct configuration of fingers to extend. Altogether approximately 63% (5 out of 8) of her productions included a hand configuration error, compared to 20% (3 out of 15) for Christine (Figure 9.4). Additionally, Maureen had movement slowing and hesitations on 25% (or

2 out of 8) of her productions, while Christine had no errors of either of these types. However, Maureen's errors should be interpreted with caution since the numbers are so small.

Unlike Christine, Maureen did not produce the handshapes at unusually high, distant locations. Christine very clearly placed her hands outside the normal signing space when copying handshapes in isolation. Unfortunately, it is impossible to judge the parameters of Maureen's signing space because she did not produce enough signs; however, she kept her hands within what would likely be the normal signing space when copying handshapes in isolation.

Figure 9.4: Handshape Copying: Maureen



Key: HS: hand configuration;
Inv Move: involuntary movement;
Init: movement initiation;
Pause: irregular pause or hesitation;
Speed: excessively slow or rapid movement

9.4.4 Tool use

9.4.4.1 Methods: Tool use

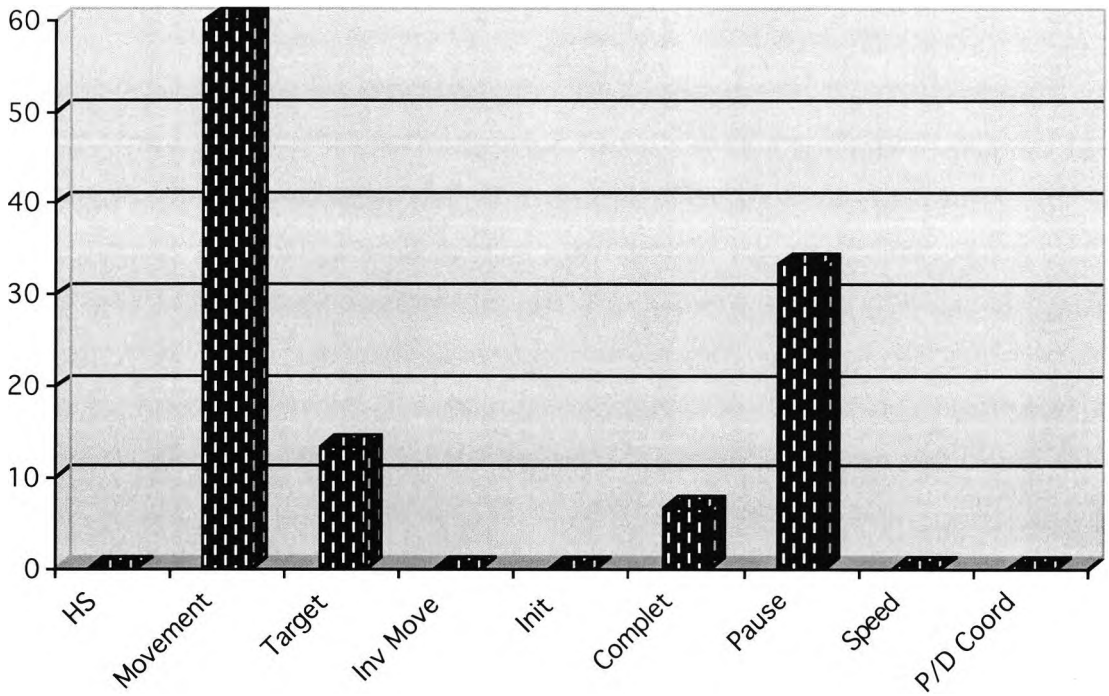
The tool use task was designed to assess Maureen's understanding of tool use as well as her ability to adopt the appropriate hand configurations to manipulate tools. She was presented with household tools by the experimenter and asked to demonstrate how they are used. The tools for this task included: a whisk, corkscrew, hammer, screwdriver, vegetable peeler, clothespeg, pepper grinder, rubber, knife, tin opener, spanner, and keys. For each manipulation of each tool, Maureen's productions were coded descriptively and qualitatively for grasp and movement. Additionally, data were coded for the applicable measures from other tasks: targeting, involuntary movement, initiation, completion, irregular pausing, movement speed, and proximal/distal co-ordination. When Maureen did not perform an action correctly, the experimenter would demonstrate it for her and she would try it again. In total, she performed 15 tool use demonstrations. Control data were not collected for this task, but comparisons can be made to findings from similar studies (e.g., Halsband et al., 2001).

9.4.4.2 Results: Tool use

Maureen produced many errors of movement when manipulating the tools. That is to say, after she picked up a tool, she could not choose the correct movement to illustrate how the tool is meant to be used. On 60% of her actions, she could not produce the movement appropriate to a given tool (Figure 9.5). Also, she would often pause while handling the tools and take a moment to look at them before continuing the task. When asked to manipulate one of the tools, she would most often hold the tool and alternately pronate and supinate her forearm. In the case of the key, this happened to be the correct response, but the key was not the first tool presented to her, and she had used the same movement on the two tools that preceded it. By contrast, she had no deficits of speed, co-ordination, or involuntary movement when manipulating the tools. Strikingly, she had no difficulty configuring her hand correctly and only minimal difficulty targeting the tools to pick them up.

Figure 9.5: Tool Use: Maureen

errors as % of 15 responses



Key: HS: hand configuration;
Movement: movement of tool;
Inv Move: involuntary movement:
Init: movement initiation;
Complet: movement completion;
Pause: irregular pause or hesitation;
P/D Coord: proximal/distal co-ordination

Table 9.3: Distribution of Maureen's Tool Use Errors

Tool	Movement	Movement error	Hand configuration	Hand configuration error	Targeting error	Completion error	Pause
peeler	pronation / supination of forearm	Y	whole hand	N	N	N	N
hammer	pronation / supination	Y	whole hand	N	N	N	N
key	pronation / supination	N	precision grip	N	N	N	N
clothespeg	horizontal figure 8 from elbow	Y	precision grip	N	N	N	N
screwdriver	pronation / supination	N	whole hand	N	N	N	N
pepper grinder	lateral arc from wrist	N	clench	N	N	N	N
spanner	pronation / supination	Y	whole hand	N	N	N	Y
spanner	pronation / supination	Y	whole hand	N	N	N	N
whisk	none	Y	whole hand	N	Y	Y	N
whisk	pronation / supination	Y	whole hand	N	N	N	N
lever corkscrew	lateral arc from wrist	N	whole hand	N	Y	N	Y
rubber	horizontal back & forth from elbow	N	whole hand	N	N	N	Y
knife	medial arc from wrist	Y	whole hand	N	N	N	Y
knife	pronation / supination	Y	whole hand	N	N	N	N
tin opener	lateral arc from wrist	N	whole hand	N	N	N	Y

Table 9.3 delineates the distribution of Maureen's errors relative to each other and across different tools, and includes descriptive coding of the hand configurations she used to grasp the tools and the movements she used to manipulate them. When an individual tool is listed twice in a row, it indicates that Maureen had difficulty with the task the first time and the experimenter demonstrated the gesture for her to copy. In the end, these demonstrations helped only minimally. In one such case, she picked up the tool (specifically, the whisk) but made no attempt to demonstrate its use, so the experimenter demonstrated how the tool is used, and on her second attempt, Maureen did move the tool, but not with the correct movement. This was the only case in which the experimenter's demonstration influenced Maureen's behaviour at all.

9.5 Discussion

9.5.1 Task-by-task analysis

Maureen's performance on the pointing task was typical of her performance on other tasks as well as typical of apraxic movement. She had more hand configuration

errors and targeting errors than any other error type. Moreover, her hand configuration errors were errors in the selection of fingers to be extended, rather than in laxing or hyper-extending the target hand configuration. For example, in some cases, instead of pointing with her index finger, she would point with both her index and little fingers extended. By contrast, she had only minimal timing deficits and no co-ordination deficits.

Maureen had far more targeting errors on the pointing task than on the other tasks, even though the spatiotemporal and psychophysical demands of this task were no greater, and were in some cases less, than on the other tasks. Her targeting errors were consistently cases of undershoot; and many times, she inadvertently caused the card to move. While it is not obvious why Maureen would have more targeting errors on this task than on the others, it may be that her errors result from an unclear understanding of how to interact with the handshape cards. In other words, she may have been treating them as objects to be manipulated rather than images to be referred to. The consistent undershoot in her movements could be explained in terms of a reduced movement range, but in all likelihood that is the type of deficit that would persist across tasks. Moreover, it does not explain why she moved the cards when pointing to them.

On the Kimura box, the main difficulty that Maureen had was with hand configuration. She was repeatedly unable to remember and produce the sequence of hand configurations that the experimenter presented to her to imitate; though her errors were confined to particular manipulanda. She had no difficulty with involuntary movements, or movement slowing. Moreover, she had only minimal difficulty with movement initiation or targeting. According to Sunderland & Sluman (2000), the demands of hand configuration play an important role in subjects' ability to perform the actions on the Kimura box task. They claim that the transition from the second to the third manipulandum is particularly difficult, because at the end of the second action the hand is open with all the fingers extended, and the natural action from that configuration is to depress the lever with the index finger, but the task requires subject to flex the fingers again and depress the lever with the thumb. The authors make no reference to it, but it should be noted that what they claim is the more natural transition from the second to the third manipulandum requires a change in the orientation of the hand, while the transition required by the task (i.e. whole hand grip of the handle to depression of the lever with the thumb) does not.

Another aspect of the task to be considered is the subject's preconceived notion of how the manipulanda should be handled, and how fixed or flexible that notion is.

While many apraxic patients lose their sense of the "obvious" way to handle an object, some objects are more obvious than others, even to unimpaired individuals. The three manipulanda on the Kimura box, in order, are a button, a handle, and a lever (Figure 4.1). For most subjects, there is only one likely action and hand configuration to accompany the handle, namely a whole-hand grasp. By contrast, the first and third manipulanda, the button and the lever, can be manipulated with any of the fingers; and the lever can be raised as well as depressed. Not surprisingly, the most common error made by control subjects in this study is to use the wrong hand configuration on the first or the third manipulandum. Beyond the question of subjects' likely perception of the appropriate movement (or perhaps related to it), whole-hand grip is an easier and more common hand configuration than either of the other two target configurations (Schieber, 1996), which may play a role in subjects' differential performance across manipulanda. The broader point to be made is that a range of factors that could influence subjects' performance must be considered before errors can be attributed to an underlying deficit, in movement sequencing or production of complex hand postures, for example.

With respect to timing on the Kimura box task, it is not surprising that the movement durations would show consistent patterns on the same manipulanda across sequences of the task, with the first movement taking the longest and the next two movements being progressively more rapid. One would also expect a practice effect across sequences, such that the subject became faster with multiple iterations. All of these patterns held true for Christine's performance, and some, but not all, held true for Maureen's performance. Additionally, each corresponding action was shorter on the second sequence than on the first. Maureen consistently took longest to produce the first movement of the sequence, but otherwise, her movement durations were quite variable. Moreover, she did not become faster in repeated sequences, even though she produced several sequences.

On the handshape copying task, Maureen produced many more errors than Christine, but in somewhat different proportions to her performance on other tasks. She had great difficulty judging and replicating the handshapes shown to her. As a result, her productions were prone to hesitations and movement slowing in addition to hand configuration errors. Of all the tasks that Maureen was asked to do, the handshape copying task was the most demanding in terms of hand configuration, because it had an explicit requirement that the hand be configured a certain way; on top of which, there was no object or tool to provide feedback on how the hand should be configured. Consequently, it is likely that her pauses and slow movements were an effect of task

difficulty. In fact, during some of the pauses, she stopped moving and looked at her hand to see how it was configured before proceeding. As discussed above, the control subject also had an atypical movement pattern on the handshape copying task. Specifically, she produced the target handshapes very slowly and outside of her normal signing space. Given that Maureen did not produce handshapes outside of what would likely be her signing space, it may be that she did not draw the same kind of conceptual distinction between producing isolated handshapes and producing signs that Christine seems to have drawn.

Arguably, tool use was the task that most clearly illustrated the nature of Maureen's movement disorder. The task was very demanding in terms of targeting, hand configuration (or grasp), and implementation of a learned movement. However, only the last of these was impaired in Maureen's performance. In contrast to her performance on other tasks, Maureen had no difficulty configuring her hand correctly to grasp a tool; the task only became difficult for her when she had to manipulate the tool in a meaningful way. While it was clear throughout that Maureen's movement deficit was high-level and representational, the tool use task highlighted that point most clearly. Maureen's movement errors on the tool use task seem to reflect either perseveration of particular movements or a limited repertoire of meaningful gestures.

In general, Maureen's movement patterns and errors were relatively similar across all the movement tasks, as well as being consistent with most reports of apraxic movement. Her most common error in all but one of the tasks was hand configuration, specifically selection of the correct or natural set of fingers for a given movement. She had minimal involuntary movements, movement slowing, or reduction of movement range, across tasks.

9.5.2 Comparisons to past research

Maureen had a high level movement disorder, as well as severe aphasia. The extent to which her aphasia and her apraxia were connected remains an open question, just as it does in neuropsychological research more generally. There is almost certainly functional overlap between some forms of apraxia and aphasia, in addition to their having a high rate of co-occurrence. Unfortunately, the severity of Maureen's aphasia made it impossible to test sign articulation to see if she had more movement errors on linguistic than on non-linguistic tasks. It is possible, however, to make some comparisons to previous research on limb apraxia and apraxia of speech.

In contrast to previous findings (Hermsdorfer et al., 1996; Poizner et al., 1995), many of Maureen's simple movements, such as pointing and reaching are quite rapid. Given the difference in research methodologies for those studies and this one, however, it could be that the slowing of movements in subjects with apraxia is detectable using precise measures of movement kinematics but is not noticeable at a functional level. Additionally, because Maureen and Christine were not performing exactly the same movements (i.e. beginning and ending at the same points in space) for most of the tasks, movement durations could only be compared as tendencies rather than direct, one-to-one comparisons; so further research would be necessary to assess Maureen's movement speeds in detail, relative to an age-matched control. What can be said from functionally-based, videotaped movement tasks is that her movements were not obviously slow in the way that the movements of subjects with Parkinson's disease or other hypokinetic disorders are.

One issue that has recurred in investigations of apraxia is the role of the object in facilitating movement and subjects' differential performance on pantomiming an action as opposed to actually executing it. On the whole, Maureen was better at guessing the appropriate movement from the object than from imitation. On the Kimura box task, she consistently had no difficulty producing the appropriate movement on the handle, which was the second manipulandum. While some subjects with apraxia have as much difficulty with this manipulandum as with any other (Sunderland & Sluman, 2000), for an unimpaired subject, the only possible hand configuration to accompany the handle is a whole-hand grasp. By contrast, the first and third manipulanda, the button and the lever, can be manipulated in multiple ways. In fact, the design of the task takes advantage of this flexibility to test subjects' memory of the required actions. Consequently, the fact that Maureen showed a differential pattern of behaviour on the one component of the task that relies less on memory or perception of someone else's hand suggests that she had an easier time forming a movement on the basis of an object than on the basis of a demonstrated or remembered movement. By extension, her apraxia seems to stem more from a disruption to her retrieval of complex motor programmes than to a disruption in online movement planning, since she was able to handle objects appropriately in terms of their shape and size but not able to manipulate them according to a learned movement pattern.

Similarly, on the tool use task, Maureen had no difficulty at all targeting, grasping, and examining the tools, but she was extremely impaired at demonstrating how to use them. One point that may be relevant to this is that on the tool use task, she

was not in fact using the tools but pantomiming how they would be used, albeit with tool in hand. Therefore, the distinction that has been made between pantomime and tool use may not be applicable here; but secondly, it may also not be the important distinction. Subjects may show a differential pattern when asked to pantomime an action with object in hand, compared to pantomiming an action without the object, as was suggested in previous research (Merians et al., 1999). This could be distinct from both pantomiming an action without the object or actually performing the action, which was the contrast explored by Halsband et al. (2001). Maureen seemed able to comprehend stored models of learned movements but not able to retrieve them, indicating perhaps a disconnection between gesture perception and production, similar to conduction aphasia.

Halsband et al. (2001) compared subjects with a range of brain lesions (anterior and posterior, right and left hemisphere) and control subjects on pantomime and tool use. Interestingly, none of their subjects showed an impairment on actual tool use. (Halsband et al. assessed representational movement parameters but not timing or co-ordination.) However, the subjects with left hemisphere damage, but not the controls, were impaired on pantomime, with the left parietal subjects being most impaired. Because they were examining actual tool use rather than demonstrations of tool use, the movement data from Halsband et al. (2001) are not directly comparable, except for what they reveal about controls for the two conditions (pantomime and tool use) and presumably also for the intermediate condition of demonstrated tool use, as well as what they reveal about the lack of prehension errors in the left hemisphere damaged subjects, which is consistent with Maureen's performance.

Wertz et al. (1998) reported that there are cases of apraxia of speech in the absence of limb apraxia. Conversely, there are also cases of limb apraxia in the absence of apraxia of speech, which is what Maureen's case seems to be. Judging from any of a number of measures, she has limb apraxia, which probably affects her signing, in addition to her severe aphasia which obviously affects her signing. What made Maureen highly unusual as a subject, and particularly as a Deaf subject, is that her speech was much closer to normal than her signing, though her language perception and production were impaired in both modalities. She produced no speech spontaneously and very little when prompted, but what she did produce was clearly articulated and without hesitation. However, while she apparently exhibited a mild oral apraxia, it would have been very difficult to determine whether or not Maureen also exhibited apraxia of speech. Apart from the fact that she had no spontaneous language production, she is a

Deaf speaker (albeit a very proficient one), so it is unclear what her articulatory skills were in her pre-morbid condition.

It may be possible in the future to identify a case of apraxia of sign. At the moment it is impossible to say whether or not Maureen has such a condition because it is almost certainly masked both by her aphasia and her manual apraxia. To identify a case of apraxia of sign, it would be necessary to find an individual with normal gesture comprehension and production as well as unimpaired language comprehension and partially intact production. The types of deficits that would suggest apraxia of sign would be substitution of one phonologically possible handshape or movement for another, hesitations or groping behaviour when signing, and atypical sign prosody. However, these are also deficits that have been used to identify signers with aphasia and to distinguish them from signers with movement disorders (Poizner & Kegl, 1993; Brentari et al., 1995), so the criteria for distinguishing sign aphasia and sign apraxia may need to be re-examined and refined.

Apart from paralysis/paresis, apraxia is probably the central movement disorder that is most likely to be articulator-specific, precisely because it does not represent a general deficit in muscle tone or co-ordination and is most likely to result from cortical damage, rather than damage to extrapyramidal structures, which despite being somatotopically-organized, are unlikely to be damaged in a way that would cause effector-specific symptoms. Hence, there may simply not be such a thing as apraxia of sign. It is interesting that this should be the case, given that it was not too long ago that apraxia and sign aphasia were thought to be the same thing.

10 Summary of Findings

The purpose of this study was to investigate a heterogeneous group of Deaf signers with movement disorders to discover the parameters along which sign production is likely to break down, and from that to learn more about the underlying nature of sign language structure, articulation, and human motor control. At the same time, the study was designed to explore how these signers compare to each other, and also how they compare to hearing subjects with analogous movement disorders. Before going on to reach broader conclusions or suggest new areas of research, this chapter will briefly recapitulate the basic findings.

As predicted, the atypical signers described here differed from each other as well as differing in their individual performance across tasks, the details of which will be described below. Some subjects were differentially impaired on signing and/or fingerspelling, as determined by the number of errors per category, per task, relative to the control subject (e.g. Joseph's handshape errors on linguistic vs. non-linguistic tasks). Conversely, some subjects had particular difficulties with non-linguistic tasks (e.g. John's targeting/location errors on linguistic vs. non-linguistic tasks). More interestingly, however, subjects showed qualitative as well as quantitative differences both with each other and across tasks. In other words, the distinctions to be found across subjects, tasks, or measures are not just a question of the number of specific errors but of the type of error (e.g. handshape errors took the form of laxing, hyperextension and incorrect finger selection, across different subjects). These findings support the theory that sign dysarthria is not a single, uniform phenomenon with the same symptoms across signers, but as with speech, dysarthria is a group of disorders that take different forms. Moreover, these findings suggest that the differential impairments that Deaf subjects show across tasks cannot be attributed to the set of effectors used to perform the required movements, because unlike hearing subjects whose speech movements were contrasted with non-linguistic limb movements in previous studies (cf. Ackermann et al., 1997), the subjects in this study used the same effectors for linguistic and non-linguistic movements. So, to go back to the issues discussed at the beginning of the thesis, it seems that sign meets the important criteria that define articulation. In particular, neurogenic movement disorders seem to impact on sign and simpler limb movements differentially in the group of subjects described here. Further large-scale research would be necessary to state anything definitive about the nature of sign dysarthria and sign articulation more broadly.

10.1 Characteristics of Individual Subjects

James, the subject with right hemisphere damage, was included in the study primarily for the contrast he was predicted to show with the other subjects. It was thought that since hemiparesis disrupts movement at a very basic level, he may simply produce one-handed signing, which would provide a baseline of comparison to subjects whose movement disorders were either more subtle or complex in nature. While James is clearly hemiparetic as a result of his stroke, his articulatory patterns were more nuanced than anticipated. In spite of his hemiparesis, he showed only minimal difficulty co-ordinating the necessary movements for sign production, even when the signs he produced were two-handed. His signing was somewhat laxed and lowered; he had no hand internal movement at all on his left (affected) hand, and his movements were slow, but beyond that, he had very little impairment of sign production. Notably, handshape laxing and lowering of signing space were present in his unaffected as well as his affected limb. This suggests that previously reported laxing and lowering of signs in Parkinson's disease could in fact be an effect of age rather than disease pathology, since James is elderly but does not have Parkinson's disease. Moreover, James did not have the sort of severe disruptions to co-ordination between limbs or between articulators on the same limb, which have been reported for other signers with right hemisphere damage (Poizner & Kegl, 1993).

James's articulatory deficits were mild. His dysarthria was similar to what has been reported in hearing subjects with RHD, in terms of severity. In general terms, his pattern of sign dysarthria is consistent with the theory that the left hemisphere is dominant for sign articulation as well as speech articulation (Corina et al., 2003). He showed only minimal disruption to his signing, despite the clear presence of damage to motor pathways in the right hemisphere and resulting hemiparesis. On a related note, James was the only signer to show a clear disruption to sign articulation that depended on the formational structure of the target sign. Unlike other subjects, he lowered signs only when the target location was high and therefore probably beyond his comfortable range of movement. In other words, James's dysarthria was not severe enough to cause fundamental re-structuring of his signing space.

Robert, the subject with cerebellar damage, stood apart from the other atypical subjects in his performance across all tasks. It is a given that the subjects described here comprise a series of case studies and were selected in part for the differences they were likely to show with each other. Nonetheless, focusing on subjects' behaviour in terms of the measures that were applied, it is apparent that Robert diverged noticeably from the

other subjects, not only quantitatively but also qualitatively. In fact, many of the qualitative descriptions of measures throughout the study were necessary solely because he was included as a subject. In terms of quantitative differences, Robert produced a very large number of involuntary movements on both linguistic and non-linguistic tasks. Qualitatively, his movements were large and unco-ordinated, and his hands and fingers were frequently hyperextended. These findings were of interest in part because they highlight the distinctions between measures of movement size, sign location, and articulator proximalization or distalization, thereby providing insight for potential articulatory measures of signed language. Prior to this study, reduction in movement size and articulator distalization had been more or less equated in research on sign and Parkinson's disease (Poizner & Kegl, 1992; Tyrone et al., 1999), because it is impossible to make a small movement to a distant location. However, it is possible to make a *large* movement to a *nearby* location, which is what Robert frequently did.

One of the most intriguing findings related to Robert was his preferential use of two hands in some one-handed signs as well as in a reach and grasp task. Although the cerebellum has been implicated in the co-ordination of complex bimanual tasks (Tracy et al., 2001; Ullen et al., 2003), the preferential use of two hands for presumably one-handed tasks has yet to be documented in any clinical or experimental research on ataxia. Moreover, producing one-handed signs with two hands has only been documented in one group, namely infants acquiring sign language natively (Cheek et al., 2001). Interestingly, preferential two-handed movements have been documented in infants' motor behaviour generally and are thought to be related to the development of postural control (Fallang et al., 2000; Rochat, 1992). It is not clear that the two are related in Robert's case, but it is worth noting that he exhibits postural instability due to cerebellar damage, so he may prefer two-handed movements for the same reason that infants do.

Joseph, the subject with PSP, was included in the study in part for the similarities and contrasts he was predicted to show to signers with Parkinson's disease. In fact, his signing did resemble that of signers with Parkinson's disease in several ways. Like them, Joseph had slow, small movements with laxed articulation. Additionally, he had difficulty co-ordinating the movements of independent articulators. Unlike signers with Parkinson's disease, however, he exhibited palilalia when signing. That is to say, he repeated entire signs without pausing. It should be noted that he had no similar type of error when fingerspelling or producing non-linguistic movements. Like hearing subjects with PSP, his spontaneous repetition of movements was specific

to the production of words. Briefly put, his dysarthria was distinct from what has been reported in PD signing, and parallel to what has been reported about PSP speech.

John, the subject with PD, was also expected to show similarities to other Deaf signers with Parkinson's disease. While he did show some similarities to those signers, he showed notable differences as well. In particular, like Joseph, John's signing was slow and laxed; however, he did not have reduced signing space or distalized articulation. Moreover, John's dysarthria was far less severe than what was reported in previous studies of Parkinson's disease and signing. John's errors were almost completely confined to the static components of signs. Additionally, in some ways, John was more impaired on non-linguistic than on linguistic movement. In particular, he had more targeting errors on the non-linguistic tasks. Unfortunately, there is no way to compare this result to findings from earlier studies on signers with Parkinson's disease, since those subjects' performance on non-linguistic tasks was not reported.

John exhibited mild to moderate dysarthria, which is typical of what is reported in hearing subjects in the mid-stages of Parkinson's disease. Unlike hearing subjects with Parkinson's disease, however, John's articulatory movements like his other movements were slowed. Accelerated, small movements, or festination, have been reported in the speech and gait of many hearing subjects with Parkinson's disease (Hanakawa et al., 1999; Netsell et al., 1975; Theodoros & Murdoch, 1998b); in the case of speech, it has been suggested that movements are not accelerated but that pauses are shorter (Nishio & Niimi, 2001). However, limb movements in PD are most often characterized as slow, or bradykinetic (Brown & Marsden, 1999b; Hallett & Khoshbin, 1980); indeed, slowed limb movements are used as diagnostic criteria of PD (Fahn & Elton, 1987). Festination may be a feature that is contrastive across the signed and spoken modalities. The tendency for subjects with PD to show festination in speech and gait could be related to the existence of an oscillatory mechanism underlying those motor functions. Additionally or alternatively, the difference between sign and speech in terms of festination could be related to the degrees of freedom of movement of the primary articulators for the two modalities. Sign movements take place in three spatial dimensions, whereas speech movements effectively take place in two dimensions.

Maureen, the subject with apraxia and aphasia, was included in the study for the contrast she was expected to show with subjects who had intact language ability but disrupted articulation. Clearly, her aphasia precluded any examination of her sign articulation; however, analysis of her non-sign movements was informative for what it revealed about the nature of her movement disorder in and of itself, and in comparison

to the other subjects. Like some of the other atypical subjects, she exhibited deficits primarily in hand configuration; however, her hand configuration deficits were qualitatively different. While other subjects laxed or hyperextended the fingers, Maureen often selected a different set of fingers to extend or flex. In addition, she had great difficulty with movement tasks when she was required to interpret an object's meaning, but had minimal difficulty locating an object in space, targeting it accurately, and forming the correct hand configuration to handle the object. So for instance, she could pick up a spanner without any difficulty but could not demonstrate how to use it.

Generally speaking, Maureen's movement deficits were fundamentally semantic or representational in nature. Her case compared to the other cases presented here supports the theory that sign dysarthria is not fundamentally representational in nature, but rather impacts on sign articulation specifically. Maureen had representational deficits in non-linguistic movements where other subjects did not. Conversely, other atypical subjects experienced deficits of speed, targeting, and co-ordination in non-linguistic tasks.

10.2 Comparisons across subjects

Now that the basic findings for each subject have been laid out, subjects will be compared to each other in more depth, across measures and across tasks. There were particular patterns of movement deficits that appeared for some subject across all tasks, which differentiated those subjects from others. Additionally, there were cases in which subjects showed a particular behavioural pattern only on one task, or on similar tasks, and these tendencies also served to differentiate subjects from each other. Due to subjects' own deficits and the constraints of subjects' energy and time, not all subjects performed all tasks. However, a certain amount can be gleaned from the direct comparisons that can be made across measures and across tasks for different subjects.

Static vs. dynamic errors

One pattern that emerged in the data was that some signers were consistently worse on static than on dynamic components of movements, or vice versa. The static components of movements were those that did not have to change over the course of the movement (e.g., hand configuration), whereas the dynamic components were those that by definition required change over time (e.g., bimanual co-ordination, or handshape change). James, Joseph, and John, the subjects with right hemisphere damage, PSP, and Parkinson's disease, respectively, all had errors primarily (but not exclusively) in the

static components of signs. However, Joseph had several co-ordination errors as well, which are considered to be dynamic errors. In terms of the static measures used, Joseph's dysarthria is an extreme of the normal imprecision that arises in relaxed, informal signing. By contrast, Robert, the subject with cerebellar damage, showed a different pattern of signing altogether. He had the highest proportion of dynamic errors by far of any subject, across all tasks. In particular, he had difficulty co-ordinating the movements of the two hands as well as the movements of independent effectors on the same limb.

The measure of bimanual co-ordination (a dynamic component of movement) was intended to capture subjects' ability or inability to use two hands in a concerted fashion to execute a movement, or in the case of the signing task, a sign. Because it is a relatively broad measure, two subjects can pattern differently on it, despite both showing a deficit. For James, the only type of bimanual co-ordination deficit he exhibited was initiating movements of the two limbs asynchronously; he had no difficulty co-ordinating movements that were ongoing. Poizner & Kegl (1993) reported a similar bimanual co-ordination deficit in a signer with right hemisphere damage, except more severe and affecting ongoing movement co-ordination as well as co-ordination of movement initiation. Robert's signing was similar to what was produced by these two signers with right hemisphere damage in that they all showed a particular difficulty synchronizing movements of the two hands in two-handed signs. However, Robert had bimanual co-ordination deficits of other varieties as well: e.g., moving the hands asynchronously, moving them asymmetrically, and not being able to bring them together in space in two handed signs.

Movement size, laxing, & distalization

As discussed previously, Robert produced large, unco-ordinated movements with proximal articulators when signing, in contrast to all the other subjects. Additionally, Robert's handshape and orientation errors were from hyperextension rather than laxation. This tendency to hyperextend sign articulators has not been previously documented in any group of signers, clinical or otherwise; although laxed sign articulation has been documented in signers with Parkinson's disease (Loew et al., 1995) and right hemisphere damage (Poizner & Kegl, 1993). James, Joseph and John all patterned somewhat similarly to each other and to other signers with Parkinson's disease, in that they all produced laxed handshapes and orientations. However, John did not have a reduced signing space, as James and Joseph. Additionally, like subjects with

Parkinson's disease and unlike any subjects in this study, Joseph also had articulatory distalization. These cases illustrate that movement size, laxing and distalization are separate phenomena which clustered together in cases of PD sign dysarthria reported previously (Poizner & Kegl, 1992; Poizner et al., 2000), but do not cluster together in all cases of sign dysarthria.

It is worth noting that Robert is much younger than the other atypical subjects. Consequently, a pattern that holds true across the other atypical subjects (e.g. articulatory laxing) could in part be an effect of age. There has been no research to date specifically addressing sign articulation in the elderly; however, findings from motor control research and research on sign and Parkinson's disease suggest that older signers may show articulatory laxing or loss of co-ordination in fine motor control (Contreras-Vidal et al., 1998; Loew et al., 1995; Smith et al., 1999). That said, it is noteworthy that the two control signers were also far apart in age and did not differ noticeably in their motor behaviours.

Lowering/locations

Joseph lowered many signs, but their locations in citation form are equally divided between high and central locations. Consequently, his entire signing space is reduced and lowered. By contrast, James, the signer with right hemisphere damage, typically lowered signs whose target locations were high, but not those whose target locations are not high (see description in Chapter 5); so his signing space seems to have an upper bound that is difficult for him to reach, rather than being wholly reduced and lowered. By contrast, John, the signer with Parkinson's disease, did not have consistent lowering of sign locations. Robert's sign productions were high and lateral when they deviated from the target location; however, more frequently his locations matched the targets, though his movements to them were enlarged.

Repetitions

James, the signer with right hemisphere damage, produced many sign repetitions on the naming task; however, so did Christine, the control subject, even though she produced none on the sign copying task. In the naming task, repeating a sign may serve some sort of discourse function, or may represent attempts at self-correction. Joseph's pattern of sign repetition was distinct from the pattern exhibited by James or Christine, precisely because it was serving no discourse or interactive function. Unlike James and Christine, Joseph produced sign repetitions on a sign copying task in which he did not

have to select the correct sign from memory. Moreover, his sign repetitions were also present in his spontaneous conversations with his daughter and with other signers. The final important point regarding repetitions is that Joseph's repetition errors were consistently produced without modification and without pausing between productions. By contrast, James and Christine produced repetitions both after pausing and without pausing, and in some cases modified the form of the sign on the second production, which suggests that they were exhibiting a qualitatively different behaviour.

Involuntary movements

John, Joseph, and Robert all produced involuntary movements when performing movement tasks; and the involuntary movements took a different form for each subject. John had two types of involuntary movement: tremors caused by the disease, and dyskinesias caused by his medication. His rate of involuntary movements was relatively consistent across tasks but varied in nature depending on whether he was on or off medication. Joseph and Robert had a different type of involuntary movement, which was present only during deliberate movement and not at rest. Proportionally, Joseph had more involuntary movements on the pointing task and on the fingerspelling task than in his signing. It may be that his involuntary movements were similar to Robert's in nature, though not as extreme. Robert produced more involuntary movements when the targeting demands of a task were high. If the same tendency were true in Joseph, it would explain why he had more involuntary movements in pointing and fingerspelling than in signing, since in general, articulatory targets in signing are large. The explanation cannot be quite this simple, because there is no clear pattern to the distribution of involuntary movements *within* the fingerspelling and pointing tasks; but it could begin to account for the disparity across tasks, while a different factor could contribute to smaller scale variation.

Fingerspelling vs. signing

Some subjects patterned differently on the signing tasks as compared to the fingerspelling task, suggesting that their deficit was not specifically linguistic, but rather articulatory. Although both signing and fingerspelling are linguistic, fingerspelling is more rapid and sequential than signing. Additionally, BSL fingerspelling in particular is targeted and co-ordinated because it requires contact between the two hands. At the same time, signing is also potentially demanding for an individual with a movement disorder because the articulatory space is very large, encompassing the torso, upper

limbs, neck, face, head, and area immediately in front of the signer. More research is required to better understand the motoric demands of fingerspelling as opposed to signing.

While Robert showed impairments across all tasks, he was most severely impaired on fingerspelling, which may be due to the fact that targeting was difficult for him and BSL fingerspelling requires the signer to produce movements to relatively small targets. There is also the possibility that his particular difficulty with fingerspelling is due to its sequential nature, in addition to or instead of, its targeting demands. It has been suggested that fingerspelling is more susceptible to breakdown in the case of a movement disorder as a result of its speed and co-ordination demands (Poizner et al., 2000; Tyrone et al., 1999). Robert's case raises interesting new questions about the structure of (two-handed and one-handed) fingerspelling and what its breakdown may mean for sign movement in general.

Joseph had fewer location errors in fingerspelling than in signing, suggesting that his movement deficit was more related to range of motion, or difficulty scaling large movements, than to targeting as such. His targeting was more accurate when targets were near than when they were far, irrespective of target size. By contrast, Robert had the opposite problem: his movements became more erratic and less accurate when the targeting demands of the task were high, but he had no difficulty with large movements, and in fact, produced them when they were not required. Unlike Robert, Joseph did not have large trajectories to nearby locations; he kept his trajectories as short and direct as possible, sometimes at the expense of targeting accuracy.

In contrast to Robert or Joseph, John had fewer handshape errors in fingerspelling than in signing, perhaps because fingerspelling does not require as broad a variety of handshapes as signing does in BSL. In John's case, the contrast between his performance on the two tasks was striking, because his handshape errors greatly outnumbered all other errors on almost all tasks. The three-way contrast across these signers shows that articulatory deficits in sign are varied, and that specific components of the language, such as fingerspelling, are not uniformly more difficult for all subjects.

Non-linguistic tasks

Subjects patterned differently across non-linguistic tasks as well as across linguistic tasks, suggesting that deficits were particular to the demands of the task. As expected, Maureen diverged from the other subjects in terms of which of her non-linguistic movements were most disrupted. She was consistently worse on tasks or

aspects of tasks that had a symbolic component, or that required a specific hand configuration not determined by the shape of an external object. So while she was minimally impaired at pointing, she had great difficulty demonstrating the use of tools. John and James patterned somewhat similarly to each other and to Christine, in that they were both more impaired on tasks that had specific hand configuration demands. However, their hand configuration errors were solely errors of laxing, whereas Maureen's were errors in the selection of which fingers and joints to extend. In addition to having difficulty with complex tasks, John also had difficulty with tasks that were sequential, such as the Kimura box. Robert had difficulty with tasks that were sequential, irrespective of whether the task was complex, and was also differentially impaired on tasks that were targeted.

Non-linguistic vs. linguistic tasks

Finally, some subjects patterned differently across linguistic and non-linguistic tasks. The reason that there is selective impairment of linguistic tasks is because of the particular set of demands imposed by articulation: it is rapid, sequential, complex, and multi-dimensional. The reason that there is selective preservation of linguistic tasks remains unclear, but may be related to the fact that articulation is well-rehearsed and usually acquired at a young age. Regarding this last point, whether or not a subject is a native language user could have implications for articulation following neural damage.

By several of the measures used for this study, including repetitions, co-ordination, and targeting, Joseph was selectively impaired on linguistic tasks. Maureen was also selectively impaired on linguistic tasks but in a different way and for different reasons. She was impaired on any task that was representational and on any task that was linguistic (due to her aphasia as well as her apraxia), whereas Joseph's difficulty was with the spatio-temporal and co-ordination demands of the linguistic tasks. In contrast to Joseph and Maureen, John had more targeting errors on non-linguistic tasks, and a speed/accuracy trade-off which was not present on linguistic tasks. John was also the only subject who was a native signer. Whether or not early acquisition of BSL helped to preserve articulatory function in relation to other motor function in his case is impossible to say; however, the question merits further research.

10.3 Comparisons with speech dysarthria

In addition to showing similarities and differences with each other, subjects in this study showed similarities and differences with hearing subjects with the same

neurological disorders. There were a few clear cases of articulatory deficits that hold true across sign and speech and equally clear cases of deficits that do not, which provides some insight into the structural differences between the two modalities. Additionally, there are categories of error that cannot be compared, simply because the gross anatomical features of the two systems are qualitatively different. The parallels, differences, and incomparable features of sign and speech will be discussed in turn.

Deficits that appear across sign and speech include palilalia, inco-ordination, reduced movement size, and (in some cases) slowed movement. Palilalia is repetition of an entire word without pause which occurs for no apparent reason. It was probably the most striking of the articulatory deficits that can cross modality, because the necessary movements are highly likely to be lengthy, complex combinations of movements, i.e., the combinations of movements necessary to repeat an entire word, as opposed to a syllable or prosodic unit.

Inco-ordination, reduced movement size, and slowed movement (unlike palilalia) may result from low level deficits; however, it is of interest that the same neural pathology can have the same low level behavioural result across the two modalities. Currently, articulatory speech deficits continue to be described as if they were articulator-specific (Ackermann et al., 1997), and the fact that the same deficits appear in sign as well as speech suggests that the articulators are not the relevant variable for differentiating articulation from other movement.

Several subjects (James, Joseph, Robert, and John) had slowed movements when signing; and in all but one case, this tendency paralleled what occurs in hearing subjects with the same movement disorders. (See below for discussion of articulatory speed in signing vs. speech.) Two subjects in particular paralleled hearing subjects with the same disorders but showed a strong contrast to each other. One of the results of the study that was not anticipated in advance was the extent to which the two subjects with hypokinetic movement disorders (Joseph with progressive supranuclear palsy, and John with Parkinson's disease) would diverge from each other. As stated in the methodology chapter, they were included in large part to allow a comparison of two subjects with very similar conditions at different stages of severity, which may be what the findings reflect, though there are other possibilities as well.

Joseph and John did show some similarities to each other, and to other signers with hypokinetic disorders, in that they both exhibited movement slowing and laxed articulation; however, they showed more differences than similarities. In the broadest terms, Joseph was more impaired on signing and fingerspelling than on pointing, while

John was less impaired on the signing and fingerspelling tasks than on the non-linguistic tasks. More specifically, Joseph showed co-ordination deficits, particularly in the relative timing of two sets of independent articulators on one limb; whereas John exhibited very few co-ordination deficits (of the two hands or of proximal and distal articulators) on any of the tasks. The third clear contrast between the two subjects, which is consistent with findings from hearing subjects with dysarthria (Metter & Hanson, 1991), was that Joseph exhibited palilalia, or the spontaneous complete repetition of a sign after producing it once himself. John had no spontaneous, unexplained repetitions of signs or other movements. The contrasts between these two subjects may stem from the fact that Joseph's condition, PSP, affects many brainstem structures, including projections to and from the cerebellum—a structure thought to be important for co-ordination (Thach et al., 1992; Ullen et al., 2003). Whereas John's condition, PD, affects primarily the substantia nigra and its projections to the rostral basal ganglia (Hirsch et al., 1988), whose role in co-ordination is disputed (Ingvarsson et al., 1997; Tracy et al., 2001).

Interestingly, while Joseph and John differed greatly from each other, they both exhibited similarities to hearing subjects with the same diseases, as described in the literature (Lu et al., 1992; Metter & Hanson, 1991; Theodoros & Murdoch, 1998b). Joseph exhibited a more severe form of sign dysarthria, and shows a particular deficit in palilalia, both of which are characteristics used to differentially diagnose PSP and Parkinson's disease. Additionally, Joseph exhibited a co-ordination deficit in his signing, while John did not. One of the characteristic symptoms of PSP dysarthria is co-ordination and timing deficits in the movements of the tongue, lips, and jaw. Those articulators, like the hands and fingers, are highly innervated and have more degrees of freedom than most other components of the speech motor control mechanism. Conversely, John's case parallels what is seen in PD dysarthria in hearing subjects, in that dysarthric symptoms are less severe than other movement deficits, and co-ordination is not selectively impaired. Obviously, because there are only two hypokinetic signers to compare, there could be any number of factors underlying the differences between the two of them, including age, disease, disease stage, and age of language acquisition. However, it is of interest that the distinctions between the two of them are very similar in nature to the typical distinctions between hearing subjects with PD and PSP dysarthria.

There are components of sign and speech that simply cannot be compared, either for subjects with dysarthria or more generally. Particular aspects of the articulators or

their innervation have no analogues across the speech and sign modalities. In speech, these include voicing, nasalization, and respiration. In sign, the main inherent structural contrast with speech is independent co-ordination of the two sides of the body. Sign articulators on opposite sides of the body are controlled independently by the contralateral sides of the brain, whereas speech articulators cannot be controlled completely independently across the midline of the body. While it is worth noting deficits of any of these aspects of articulation and trying to understand their neural bases, there is no reason to look for parallels of all features of one modality in the other modality.

As for patterns that are comparable but nonetheless different across the two modalities, there was nothing resembling speech festination (rapid, small articulatory movements) in the data described here. Festination of speech often occurs in hearing individuals with Parkinson's disease, but did not occur in the sign articulation of John, the signer with Parkinson's disease. Given that John is a single case, this finding could be particular to him, or it could be a type of articulatory deficit not likely to appear in the primary sign articulators, because of a fundamental difference in the structure of the output mechanisms for speech and for sign. Furthermore, this difference (assuming it is a true cross-modal difference) may be related to the existence of an oscillator that provides a structure on which to base the articulatory system. More research would be necessary to clarify whether or not there is an underlying oscillator in sign that has its neurological basis in the brainstem mechanisms that facilitate rapid, automatic bimanual co-ordination.

10.4 Related Issues

Beyond the basic findings, there were additional issues that emerged as the study progressed, which will be addressed briefly. Questions of age, cross-linguistic differences from one sign language to another, and disease stage or injury severity inevitably entered into the comparisons of subjects in this study, and into comparisons of this study with previous studies on sign language and on dysarthria.

Age alone seems to set the atypical subjects apart from each other, though the same is not true for the control subjects. That is to say, mild articulatory laxing was present for all the subjects over the age of seventy and could be explained in terms of an age-related decline in fine motor control. However, there was no apparent difference between the two control subjects (ages 70 and 38), on this or any other measure. Consequently, there may be an interaction between age and clinical status that is

causing movement disorders that appear in old age to have a more debilitating effect, or causing the normal aging process to become accelerated. This could explain why John, the younger subject with Parkinson's disease, bore limited similarity to previously-reported Deaf subjects with Parkinson's disease, because he showed virtually no deficits in co-ordination, which was one of the central findings reported in those studies (Brentari et al., 1995; Poizner et al., 2000). Given that fine motor co-ordination deteriorates with age, it could be that what that research was identifying was an effect of age as much as an effect of Parkinson's disease.

Both this study and an earlier study found movement deficits that are more pronounced in fingerspelling than in signing, despite the fact that two structurally different fingerspelling systems were under investigation in this study and in that one (Tyrone et al., 1999), which raises questions about what is specific to fingerspelling that would make it more susceptible to breakdown. It is difficult to identify a single such feature, and in fact there may not be one: fingerspelling is rapid, sequential, and requires tightly co-ordinated fine motor control. So it could be the combination of any or all of these that makes it a difficult motor task. Additionally, in the case of BSL fingerspelling, precise movement targeting is required as well. Various motor structures have been described as having an important role in motor sequencing: the basal ganglia (Weiss et al., 1997), the supplementary motor area (Haaland & Harrington, 1994), the cerebellum (Harrington et al., 2000; Ullen et al., 2003). The cerebellum and basal ganglia have also been implicated in co-ordination and fine motor control (Ingvarsson et al., 1997; Ullen et al., 2003; VanGemmert et al., 1999). Moreover, the subject in this study who was differentially impaired on fingerspelling had ataxia; in the earlier study on ASL fingerspelling, the subjects who were differentially impaired on fingerspelling had Parkinson's disease. Clearly, more research is required to disambiguate the roles of brain structures and physical movement demands in articulatory deficits that are specific to fingerspelling.

The categorization of errors, and the measurement of subjects' behaviour more generally, was a complex issue in the development of the study. Whether or not a particular aspect of movement was considered an error was defined almost entirely by the coding schemes designed for the study, rather than by any external criteria. As with many tasks used in motor control research, the movement tasks used for this study cannot be performed in a way that is obviously wrong, except at the extremes of behaviour (e.g., pointing to the wrong image). In fact, the tasks are designed so that subjects can perform them in whatever way is natural to them, and their automatic

tendencies for movement can be explored. Within this framework, to call an aberration in speed, trajectory, or co-ordination an error or an impairment in spite of subjects' apparently successful accomplishment of a task (e.g., grasping an object) is consistent with standards in the motor control literature (e.g., Ingvarsson et al., 1997; Lang & Bastian, 2002).

With respect to errors in signing and fingerspelling, up to this point, the general standard for a sign being produced correctly is that it be comprehensible to another signer and the correct lexical choice. The only categorization scheme for the physical structure of signed language is phonological: it is not capable of describing differences across productions which are not contrastive. However, because the articulatory properties of sign and fingerspelling were of interest in this study, new measures had to be applied which had no bearing on the productions' linguistic status. Broadly speaking, signed productions were considered to be erroneous when they differed noticeably from the citation form of the sign. This standard is also consistent with the literature on atypical signers (Brentari & Poizner (1994) alternately use the terms "disturbances" and "deficits."). As a result of setting the citation form as the standard for normal production of a sign, many "errors" were found in the productions of the control signers. However, this approach was chosen deliberately to illustrate the range of phonetic variation in typical as well as atypical signers. In summary, it was with caution that perceived deviations from the norm on the linguistic and non-linguistic tasks were labelled as errors rather than being given a more neutral descriptor, such as "anomalies" or "atypical movements." The term error was chosen for its ease of use and its consistency with current standards in the literature.

Finally, the results of this research drew into question the characteristics of some of the tasks themselves: most particularly the Kimura box. Similarities emerged across typical and atypical signers which raised interesting questions regarding the tasks as opposed to the subjects. Almost all the subjects who performed the Kimura box task showed a differential pattern across the individual components of the task. This would not be of interest by itself except for the explanations that are given for performance on this task, i.e., handshape perseveration (Kimura, 1993), general inability to configure the hand correctly (Sunderland & Sluman, 2000), impaired movement sequencing (Kimura, 1993). For any of these to be viable as an explanation of apraxics' performance on the task, the relative difficulty of individual components of the task would have to be tightly controlled for non-apraxic subjects (whether atypical or typical); and this study indicates that that is not the case.

The handshape copying task raised similar issues, because there was an interesting pattern of movement that held true across atypical and control subjects, which was more a reflection of the nature of the task than of subjects' articulatory abilities. It was striking that both the control subject and the subject with Parkinson's disease performed the handshape copying task very slowly and awkwardly despite the fact that it was the task that was physically most similar to signing. This probably reflects the subjects' conceptualization of sign production as qualitatively different from other types of movements.

10.5 Concluding Thoughts

Findings from this study show that sign dysarthria is distinct from disruption to limb movement in general, as evidenced by the fact that some signers show differential impairment to signing or fingerspelling, or conversely, show differential impairment to limb movements not related to language. However, this is not to say that signers with dysarthria are differentially impaired on signing and fingerspelling because they are linguistic in nature, but rather because they have high articulatory demands, in much the same way that speech articulation does. Moreover, it has fundamentally different demands from simpler limb movements, which may rely on a separate set of neural mechanisms. Sign dysarthria is distinct from disrupted movement generally, but it is also distinct from aphasia and apraxia in a signed language. As with previous studies on sign language and the brain, this study has uncovered sign deficits that are not fundamentally linguistic in nature. Additionally, however, this study has revealed differences in the motor behaviour of apraxic and non-apraxic subjects.

It would be of interest to compare sign production in Deaf subjects with dysarthria and Deaf subjects with apraxia but not aphasia; based on initial indications from this study, it is predicted that those two groups would pattern differentially on several components of sign movements, such as handshape and speed. Psycholinguistics, speech motor control and sign language research would all benefit greatly from a clear delineation of the differences between sign aphasia, apraxia, and dysarthria. It should be mentioned in passing that the fact that the apraxic subject in this study was also aphasic does not by extension mean that apraxia and sign aphasia are synonymous. Several studies have documented Deaf subjects who are aphasic but not apraxic or whose aphasia is far more severe than their apraxia (Corina et al., 1992a; Marshall et al., 2004; Poizner et al., 1987). Moreover, many hearing aphasics are also apraxic, particularly when the relevant neural damage is in the anterior left hemisphere,

and the dissociation between aphasia and apraxia in those cases is widely acknowledged. Apraxia does not become synonymous with aphasia simply by virtue of which articulators are used.

This study has used a well-established methodology in research on atypical populations, the case study, and applied it to a new area of research, the breakdown of sign articulation. The cases outlined here are not assumed to be representative of the movement disorders they exhibit or of British Sign Language users in general. By contrast, they illustrate what is possible in sign language breakdown, not what is typical or likely. Nonetheless, these cases are informative for the contrast they provide with typical cases and the insight they yield into the nature of sign articulation. The results presented here suggest that sign dysarthria is distinct from deficits to simple limb movements, as well as being distinct from apraxia and sign aphasia. Furthermore, sign dysarthria takes a variety of forms which can be classified according to discrete measures of the movements for sign articulation. This study has opened up the possibility of new lines of research related to sign articulation and suggested important future research questions. The implications of the study, its limitations, and directions for future research will be discussed in the following chapter.

11 Conclusions

This study has taken a new approach both to the study of signed language and to the study of motor control for articulation. First, the study is distinct from previous sign language research, in that sign was examined as a movement task, for its own sake, rather than being examined as a linguistic phenomenon. Second, the study is distinct from past research on dysarthria in that subjects with articulatory disorders were compared on linguistic tasks and on an array of complex non-linguistic tasks which use the hands as primary effectors. The results of this study suggest that sign dysarthria is distinct from deficits to simple limb movements, and that sign dysarthria is analysable, non-uniform and not articulator-specific.

The background for this study, its methodologies and its findings have been described in the preceding chapters. This chapter will re-state and address the research questions posed initially, and discuss limitations of the study and directions for future research. The research questions that this study set out to address were:

- Is there a discrete phenomenon, sign dysarthria, which is analogous to dysarthria in speech?
- How should sign dysarthria be defined?
- What are the similarities and differences between sign dysarthria and speech dysarthria?
- What are the similarities and differences between sign dysarthria and apraxia?
- What are the similarities and differences between sign dysarthria and disruption of simple limb movements?

11.1 Discussion of research questions

Sign dysarthria as discrete

This study has presented evidence showing that there are signers who pattern differentially on sign compared to non-linguistic tasks. Additionally, it has presented evidence that signers with movement disorders pattern differently from each other on articulatory measures of sign. The evidence presented suggests that there may be a discrete phenomenon which can be characterized as sign dysarthria. Furthermore, it seems that sign dysarthria is not a single, uniform phenomenon that has the same features across signers. On the contrary, as with speech, dysarthria is a group of disorders of articulation that can take a variety of forms. Moreover, it is clear that the differential impairments that Deaf subjects show across tasks cannot be attributed to the set of effectors used to perform the required movements, because in the tasks analysed

for this study, subjects are using the same effectors for linguistic and non-linguistic movements. This suggests that neurogenic movement disorders might impact on sign and simpler limb movements differentially.

The results of the study also show that some subjects have differential impairments of sign and fingerspelling, with greater disruptions to one component of the language than to the other. In other words, what these signers exhibit is fundamentally articulatory rather than linguistic. Subjects were impaired at producing signs, according to articulatory measures even in cases in which linguistic function was completely intact. Moreover, articulatory impairments were differentiable from each other along specific criteria (handshape, co-ordination, timing, repetition, etc.). By extension from this, it is possible to discuss sign in terms of articulation and begin to explore the nature of sign articulation in more depth to determine precisely what physical, perceptual, and biomechanical factors influence its structure.

Definition of sign dysarthria

Traditional definitions of dysarthria have emphasized that it is motoric, neurogenic, and affects the muscles of the vocal tract (Darley et al., 1969a; Kent, 2000). Additionally, in order to disambiguate dysarthria from apraxia and aphasia, they have also stressed that dysarthria is associated with weakness, slowness and inco-ordination. The proposed definition of sign dysarthria will have to take into account the fact that sign uses a different set of articulators from speech and adjust previous definitions accordingly. A defining feature of signed language is that it uses the hands and arms, rather than the vocal tract, as its primary articulators. Moreover, this study is consistent with past dysarthria research in that it found articulatory deficits which cannot be explained simply in terms of weakness, slowness, and inco-ordination, such as uncontrolled sign repetition. These characteristics of dysarthria should be included as part of its definition. Consequently, it is proposed that the definition of sign dysarthria be: disruption to motoric aspects of sign articulation resulting from neural damage which is associated with weakness, slowness, inco-ordination, or deficits in motor programming. Sign dysarthria manifests itself in the static configurations of the sign articulators as well as the speed and amplitude of their movements, and the co-ordination of those movements with co-occuring movements of other sign articulators. It is hoped that this definition of sign dysarthria might motivate re-examination of current definitions of speech dysarthria and ultimately lead to a definition that is modality-independent.

Sign vs. speech dysarthria

In many ways, Deaf signers with dysarthria exhibit symptoms similar to those exhibited by hearing subjects with dysarthria. Additionally, while some symptoms are present across the two modalities, others may not be, and some simply cannot be because of physical differences in the two systems. There are parallels across the two modalities in errors of speed, movement amplitude, co-ordination and involuntary movements. Sign dysarthria as documented in this study paralleled speech dysarthria in terms of the overall severity of the articulatory disruption in relation to the pathology: PSP and cerebellar damage resulted in severe dysarthria, whereas Parkinson's disease and right hemisphere damage resulted in mild dysarthria. Sign and speech dysarthria are related to similar brain pathologies, Parkinson's disease, PSP, and cerebellar damage, and take similar forms depending on those pathologies.

Sign dysarthria vs. apraxia

This question can only be answered in sufficient depth after the examination of sign data from a Deaf subject who is apraxic but not aphasic. The current study compared an apraxic/aphasic subject with other dysarthric subjects on non-linguistic tasks, so conclusions about the nature of sign apraxia can only be drawn by inference. It is possible to propose, on the basis of this study, that sign dysarthria is not particular to representational components of sign, despite being particular to sign itself. Whereas apraxia in some cases is particular to the representational components of movements in general. The extent to which that tendency manifests itself in sign as well as other movements is a question to be addressed in future research. Moreover, sign dysarthria is not particular to handshape formation but can affect multiple components of sign production, either independently or simultaneously. Sign dysarthria can impact low level aspects of articulation, such as muscle tone or strength. Broadly speaking, dysarthria occurs at a range of levels from motor sequencing to muscle tone, whereas apraxia occurs at the level of meaningful representations of movement.

Sign dysarthria vs. disruption to simple limb movements

There is no single criterion that can distinguish sign dysarthria from disruption to simple limb movements in all cases, which is not surprising given that sign dysarthria takes a variety of forms. What this study suggests, rather, is that sign and non-sign movements may be affected differentially by the same movement disorder in the same

subject, in terms of either severity of symptoms or particular set of symptoms present. However, the research described here relies on a series of case studies, which, as discussed previously, can only suggest what is possible not what is prevalent or likely. Further research is called for to better describe the nature of sign dysarthria and its relationship to disruption of simpler movements.

Articulatory measures of sign

An ancillary question that this study addresses is the establishment of articulatory measures of sign dysarthria, and of normal signing. This study found differences across subjects by using the well-established phonological parameters of signs - handshape, location, movement, and orientation (Battison et al., 1975; Stokoe, 1960) - and describing them in terms of phonetic detail rather than focusing on phonological contrast. These measures were not sufficient to capture the relevant aspects of subjects' articulatory productions, however, and had to be supplemented with more basic measurements of subjects' movements: aspects of speed, distance, and the relative positions/movements of independent articulators. On the basis of this research, it is proposed that articulatory measures of signs should include the aforementioned phonological parameters, described in anatomical terms to the extent possible, and also include absolute and relative measures of the displacement, velocity, and acceleration of the hands, arms, and torso. Additionally, slightly more abstract measures should be included, such as the phonetic symmetry of the two hands and arms when producing sign handshapes and movements. It would also be informative to measure the movements of the facial muscles as they co-ordinate with sign production.

Brain areas for articulation

Although this study did not explicitly measure brain activity, it nonetheless has implications for theories of the neural basis of articulation. The findings from this study are consistent with current models of brain areas for articulation (Dronkers, 1996; Miller, 2002; Wise et al., 1999). These models of dysarthria suggest that critical areas for speech articulation include the left insular cortex and left primary and supplementary motor areas (Wise et al., 1999), the right and paravermal cerebellum (Kent et al., 2001), with involvement of the left posterior temporal and left posterior parietal areas during stages of action planning for speech (Miller, 2002). Areas which may also be involved in articulation but serve a less direct or important role include the

right precentral gyrus (Kim et al., 2003), and the basal ganglia (Theodoros & Murdoch, 1998b).

The findings from this study are consistent with recent findings on brain areas important to sign production, which indicate that the right cerebellum and anterior cerebral motor areas show increased activation during sign production, irrespective of which hand is used for signing (Corina et al., 2003). Nothing conclusive can be said about the relationship between brain anatomy and function solely on the basis of this study. However, it should be noted that the subjects in this study with brainstem and cerebellar damage exhibited a more severe form of dysarthria, while those with right cerebral damage and Parkinson's disease exhibited a milder form of dysarthria, which is what would be predicted by the models described above.

11.2 Limitations of this study

Now that the basic findings and conclusions have been laid out, it is worth considering the limitations of the research, at theoretical, methodological, and technical levels. To begin with, this research follows a long tradition in the behavioural sciences of examining the breakdown of a function to gain insight into the nature and physical basis of that function. In particular, the data were from a set of individual case studies, each of which represented a different disorder. Clearly, there are assumptions that go into such an approach that can be problematic to its interpretation. First of all, it is not at all clear that any of the subjects in this study, or in any case study, is representative of other subjects with the same disorder. Therefore, it is important not to over-interpret findings that are particularly unique or unexpected and treat them as if they automatically supersede past findings. Atypical cases are informative because they illustrate what can happen, but they do not necessarily illustrate what is likely to happen. Another issue to contend with in the examination of breakdown of a function, beyond the question of whether the subjects are representative, is the assumption that the function itself is unitary. This study was able to circumvent that particular issue in part by looking at a diverse group of atypical subjects (signers with movement disorders) and a broadly defined function (sign articulation).

Beyond the purely theoretical issues, there are practical problems with research on atypical subjects, some of which were discussed in the methodology chapter. First, atypical subjects, particularly small populations such as Deaf subjects with acquired neurological disorders, invariably result in studies with small samples, thereby limiting the statistical power of any result as well as drawing into question its generalizability.

Also, research on atypical subjects who are also linguistic or cultural minorities limits the range of behavioural measures that can be applied, because it is unclear how to interpret them relative to the norm. By extension, it is then difficult to make comparisons to other studies which do use more standardized behavioural measures. Clearly, these issues are not particular to this study but hold true for all research on atypical Deaf signers, clinical or otherwise. The critical point is not to extrapolate too far from the results, when they may not be relevant to the "typical" case. However, assuming the research has a theoretical basis and a clear empirical precedent, findings from an atypical subject can reveal a lot about what it means to be typical.

Though there were clear advantages to using a relatively flexible experimental design in this project (for example, the discovery that the signer with cerebellar damage preferentially used two hands for reaching), the research would have benefited from more detailed measures taken on a standardized task. If more were known about the performance of normal Deaf signers on standardized neuropsychological, psycholinguistic, and motor control tasks, then a more narrowly constrained experimental design would have been an obvious choice. However, in the absence of that information, the decision of what experimental design to use was less straightforward. Additionally, had more been understood about the likely symptoms of the participants in advance, or had there been more time to conduct background research precisely to address that question, then measures and narrowly-defined articulatory tasks specific to this study could have been developed and implemented to allow more direct comparisons of individual subjects.

Short of a standardized task, a single task developed for this project, in conjunction with more precise spatio-temporal experimental design and measures, would also have been informative. This would have been easiest to implement if there had either been access to the kinematic equipment and testing facilities on site at a research hospital, or a sufficient number of subjects who could travel to a testing facility. In the absence of such facilities, it would have been necessary at a minimum to do some preliminary testing of subjects to find a range of tasks that would have been informative for an extremely varied but small group, and using a data capture technique that is not extremely precise. As discussed previously, the research questions lent themselves to an experimental design that gains in breadth where it loses in detail. It is hoped that this study will facilitate the development of research questions that can be addressed by more precise measurements and capture techniques.

In terms of consistency across subjects, the study would have benefited from having more tasks that all the subjects performed, while using the same experimental design. The differences in ability across subjects made the experimental design challenging; however, at that level, having more preparation time and more testing time with each subject would have made design of a task that all subjects could perform and that would have been informative for all subjects' motor behaviour both feasible and informative.

Additionally, it is arguable that the signing data would have been more natural if productions had been embedded in a carrier phrase, thereby controlling for the effects of starting and ending points of the sign movements. This was taken into consideration in designing the task, and it was decided that the unnaturalness of the carrier phrase itself would have outweighed its beneficial effects; whereas use of a different semantically viable carrier phrase for each production would have defeated the entire purpose of having a carrier phrase to begin with. Had the articulatory details been measured in very close detail, as in the case of kinematic or EMG recordings, a carrier phrase would have been necessary, irrespective of its semantic appropriateness. However, it was reasoned that since the data capture technique was not very precise, the use of a linguistically unnatural carrier phrase was not justified.

Finally, the study would have benefited from better localization of the lesions or areas of neural damage across subjects, and from a group that had been more uniform in terms of language background. The first issue is effectively beyond the control of researchers who do not have access to brain imaging equipment, because the necessary data are not collected by clinicians in all cases. It would have been possible to include only subjects who had been given brain scans, but it would have required an enormous investment of time. Additionally, it would have been nice to recruit only native signers; unfortunately, they are the minority of Deaf people and an even smaller minority of Deaf people who are elderly. Recruiting along those criteria would have required much more time and a larger sample to draw subjects from. As sign languages gain greater legal recognition internationally (UK-Council-on-Deafness, 2003), it is hoped that these issues will be less problematic for future researchers.

11.3 Directions for future research

While this is the first study to explore the range of disruptions to sign articulation, it has raised many questions while answering others, and raised the possibility of new research areas to be explored. One very broad question to be

addressed is the development of articulatory models of sign language production and perception. Such an undertaking would require a range of approaches and experiments. A few of these are outlined here.

An intriguing aspect of sign articulation that seemed apparent from looking at the data, but which could not be described systematically using this study's methodology was movement trajectory. It clearly emerged as a separate phenomenon from signing space, selected articulator or movement size, but was related to all of these. Moreover, movement trajectory in sign could easily be connected to the idea of sign prosody (Sandler, 1999), as well as to the concept of kinetic melody from motor control research (Freund, 1987). Perhaps, eventually, these two theoretical frameworks could be integrated with data on articulatory measures of movement trajectory to investigate the articulatory basis of movement within and across signs.

Kinematic data from both atypical and typical signers would be extremely useful to the field of sign language research because they would allow researchers to establish measures for normal movement parameters of sign, particularly in terms of speed, acceleration, movement range, and trajectories. There have been a few studies which have collected kinematic data from signers (Brentari et al., 1995; Cormier, 2002; Petitto et al., 2001); however, the focus of these has been primarily linguistic rather than articulatory, and as a result they were not searching for the range of normal variability in signing, but rather for a kinematic pattern that could distinguish one grammatical feature from another, or one group of signers from another. To simply have a sense of the normal range of variability across the same signers and the same productions would make kinematic studies with a linguistic focus much more meaningful. Additionally, to have a sense of normal variability in signing would make it much easier to determine precisely what it is that is "atypical" about atypical signers. Because of the volume of data generated by any one kinematic measure, it would be necessary to conduct multiple studies, each examining only one measure of sign articulation (e.g. sign location) across multiple productions of the same small set of signs.

Another interesting and relevant area to research is the effect of aging on sign articulation (as opposed to its effects on lexicon, grammar, or pragmatics), analogous to the research that has been done on the sign production of young Deaf children of Deaf parents (Cheek et al., 2001; Meier et al., 1998). While there has been research on lexical, grammatical, and pragmatic aspects of elderly people's signing (Blumenthal-Kelly, 1991), there have been no studies on sign articulation in the elderly to determine how it might vary from the articulation in younger adults. Just as sign research on

clinical populations would benefit from more normative data on non-impaired signers, research on signers with disorders that are likely to occur in the elderly (e.g. stroke, Parkinson's disease) would benefit greatly from a larger body of data on signing in elderly subjects who are healthy.

In the context of research on atypical signers, a comparison of multiple signers with the same movement disorder would suitably complement the studies that have preceded it, including this study, which have focused on individual cases. While case studies are an invaluable source of information, and uncover constellations of behaviours that one could never anticipate in advance, a case study can never be taken to be representative of a given disease or deficit. They illustrate what is possible, but not necessarily what is likely. To get a better sense of the nature of any neurological disorder, it is necessary to find multiple subjects who share the same type of neural damage, and the same resulting symptoms and preservation of function. Future group studies on the nature of sign articulation deficits can hopefully serve as a basis for the development of articulatory measures of sign, and ultimately address the nature of the biological constraints on language structure across modalities.

As stated previously, this study cannot directly answer questions about the relationship between brain region and function for sign articulation; however, it confirms previous findings on that topic and on the neural basis of articulation more broadly. It is hoped that future sign language research might examine the neural basis of sign articulation specifically, independent of linguistic function. It would be feasible, for instance, for a study to investigate brain activity during sign production, as opposed to production of phonologically-possible non-signs, as opposed to a reach and grasp task. This type of experiment could begin to address brain function for language relative to articulation, and articulation relative to other co-ordinated movement.

11.4 Final Thoughts

The pioneering work of Darley, Aronson, and Brown revealed that speech breakdown could be described systematically and analytically, and that descriptions implementing a particular, consistent set of measures were useful in differentiating one form of dysarthria from another. By extension, this constitutes empirical evidence that speech is not an unanalysable whole but can be broken down into its constituent parts, which contribute to the underlying form that speech takes. The current research is important because it shows that sign, like speech, breaks down along discrete articulatory measures. Furthermore, sign as well as speech can be analysed in terms of

its component physical structures, and those structures form part of the biological basis of sign language.

There have been a series of studies of both dysarthria and of signed language which have challenged earlier assumptions about the structure of speech and of sign. With respect to the former, prior to the work of Darley, Aronson, and Brown, it had been assumed that the breakdown of speech motor control was a holistic phenomenon which could not be measured along consistent criteria or analysed in terms of its constituent parts. Similarly, there were two basic assumptions about the nature of signed language, which motivated some of the most seminal work in the field of sign language research. First, it was assumed that signs were iconic gestures with no sublexical structure. Then research done by William Stokoe showed that all signs could be differentiated according to a limited set of phonological parameters (Stokoe, 1960). Second, it was assumed that sign language must by necessity be cognitively or linguistically distinct from spoken language and more similar to gesture (Bloomfield, 1933; Kimura, 1977). Later studies then showed that aphasia manifested itself similarly in spoken and signed languages and was distinct from apraxia (Corina et al., 1992a; Poizner et al., 1987; Poizner et al., 1989), indicating that sign languages were neurologically and behaviourally like all other languages.

It is hoped that this study will motivate exploration of a new area of research akin to those founded by the pioneering studies outlined above. What this study suggests is that not only are sign and speech similar linguistically, but they are also similar motorically. The breakdown of sign as movement is qualitatively and functionally similar to the breakdown of speech. This implies that it should be possible to investigate the neural basis of articulation as a broader concept, independent of language modality.

To return to the earlier discussion of what constitutes articulation, on the basis of this study and others, sign meets all the criteria. First, it is rapid and complex, as evidenced by the descriptive measures of this study, as well as by all the linguistic research that came before it. Signers produce many highly co-ordinated articulatory gestures very quickly, as required by any human language. Furthermore, sign is structured in part by the physiology and anatomy of its articulators: as such, its form has similarities to speech because the primary articulators are highly innervated. At the same time, sign is structurally distinct from speech because the articulators are configured differently. Finally, sign allows communication, and is perceptually salient. As stated at the outset, these last two features were not directly addressed by this study

because they had been addressed in earlier studies; however, this research depended on those findings.

In the same way that language is not specific to the aural/oral modality, articulation is not specific to the vocal tract. Sign articulation is rapid, complex, governed by the physical structure of its articulators and by the neural mechanisms governing movement, and differentially subject to breakdown in the context of movement disorders. It should not be surprising that speech and sign would share articulatory properties. Just as there is an interface between the structure of language and the structure of speech, there must by necessity be an interface between the structure of language and the structure of sign, because signed languages are natural human languages.

Bibliography

Ackermann, H., Hertrich, I., Daum, I., Scharf, G., & Spieker, S. (1997). Kinematic analysis of articulatory movements in central motor disorders. *Movement Disorders, 12*(6), 1019-1027.

Ackermann, H., & Hertrich, I. (2000). The contribution of the cerebellum to speech processing. *Journal of Neurolinguistics, 13*, 95-116.

Akamatsu, C.T. (1983). Fingerspelling formulae: A word is more or less than the sum of its letters. In W. Stokoe & V. Volterra (Eds.), *SLR '83: Proceedings of the 3rd International Symposium on Sign Language Research* (pp. 126-132). Silver Spring, MD: Linstok Press.

Antzakas, K., & Woll, B. (2002). Head movements and negation in Greek Sign Language. In I. Wachsmuth & T. Sowa (Eds.), *Gesture and Sign Language in Human-Computer Interaction* (pp. 193-196). Berlin: Springer-Verlag.

Appenteng, K. (1990). Jaw muscle spindles and their central connections. In A. Taylor (Ed.), *Neurophysiology of the Jaws and Teeth* (pp. 96-141). London: Macmillan Press.

Atkinson, J., Marshall, J., Thacker, A., & Woll, B. (2002). When sign language breaks down: Deaf people's access to language therapy. *Deaf Worlds, 18*(1), 9-21.

Atkinson, J., Campbell, R., Marshall, J., Thacker, A., & Woll, B. (2004). Understanding 'not': Neuropsychological dissociations between hand and head markers of negation in BSL. *Neuropsychologia, 42*, 214-229.

Atkinson, J.R., Marshall, J., Woll, B., & Thacker, A. (in press). Testing comprehension abilities in users of British Sign Language following CVA. *Brain and Language*.

Bangham, J.A., Cox, S.J., Lincoln, M., Marshall, I., Tutt, M., & Wells, M. (2000). Signing for the deaf using virtual humans. Paper presented at the IEEE Colloquium on Speech and Language Processing for Disabled and Elderly People.

Barakat, R. (1975). On ambiguity in the Cistercian Sign Language. *Sign Language Studies, 4*(8), 275-289.

Battison, R. (1974). Phonological deletion in American Sign Language. *Sign Language Studies, 5*, 1-19.

Battison, R., Markowicz, H., & Woodward, J. (1975). A good rule of thumb: variable phonology in American Sign Language. In R.W. Fasold & R.W. Shuy (Eds.), *Analyzing Variation in Language* (pp. 291-302). Washington, D.C.: Linstok Press.

Battison, R. (1978). *Lexical Borrowing in American Sign Language*. Silver Spring, MD: Linstok.

Benecke, R., Rothwell, J.C., Dick, J.P.R., Day, B.L., & Marsden, C.D. (1986). Performance of simultaneous movements in patients with Parkinson's disease. *Brain, 109*, 739-757.

Bennett, S., & Netsell, R.W. (1999). Possible roles of the insula in speech and language processing: Directions for research. *Journal of Medical Speech-Language Pathology, 7*(4), 255-272.

Blakemore, S.J., Frith, C.D., & Wolpert, D.M. (2001). The cerebellum is involved in predicting the sensory consequences of action. *Neuroreport, 12*(9), 1879-1884.

Bloomfield, L. (1933). *Language*. Chicago: University of Chicago Press.

Blumenthal-Kelly, A. (1991). Fingerspelling use among the deaf senior citizens of Baltimore. In E.A. Winston (Ed.), *Communication Forum* (Vol. 1, pp. 90-98). Washington, DC: Gallaudet University School of Communication.

Bonifati, V., Fabrizio, E., Vanacore, N., DeMari, M., & Meco, G. (1995). Familial Parkinson's disease: a clinical genetic analysis. *Canadian Journal of Neurological Sciences, 22*(4), 272-279.

Braun, A.R., Guillemin, A., Hosey, L., & Varga, M. (2001). The neural organization of discourse: An H2O-PET study of narrative production in English and American sign language. *Brain, 124*, 2028-2044.

Brennan, M., Colville, M.D., & Lawson, L.K. (1984). *Words in Hand: A Structural Analysis of the Signs of British Sign Language*. Edinburgh: Edinburgh Sign Language Research Project.

Brentari, D., & Poizner, H. (1994). A phonological analysis of a Deaf Parkinsonian signer. *Language and Cognitive Processes, 9*(1), 69-99.

Brentari, D., Poizner, H., & Kegl, J. (1995). Aphasic and Parkinsonian signing: Differences in phonological disruption. *Brain and Language, 48*, 69-105.

Brentari, D. (1998). *A Prosodic Model of Sign Language Phonology*. Cambridge, MA: MIT Press.

Brentari, D. (2002). Modality differences in sign language phonology and morphophonemics. In R.P. Meier, K. Cormier & D. Quinto-Pozos (Eds.), *Modality and Structure in Signed and Spoken Languages* (pp. 35-64). New York: Cambridge University Press.

Brien, D. (Ed.). (1992). *Dictionary of British Sign Language / English*. London: Faber and Faber.

Broca, P. (1861). Remarques sur le siege de la faculte de langage suivies d'une observation d'aphemie. *Bulletin de la Societe d'Anatomie, 6*(2), 330-357.

Brodal, P. (1998). *The Central Nervous System: Structure and Function* (Second ed.). New York: Oxford University Press.

Brooks, V.B. (1986). *The Neural Basis of Motor Control*. New York: Oxford University Press.

Brotchie, P., Iansek, R., & Horne, M.K. (1991a). Motor function of the monkey globus pallidus: Neuronal discharge and parameters of movement. *Brain, 114*, 1667-1683.

Brotchie, P., Iansek, R., & Horne, M.K. (1991b). Motor function of the monkey globus pallidus: Cognitive aspects of movement and phasic neuronal activity. *Brain, 114*, 1685-1702.

Brown, P., & Marsden, C.D. (1999a). Bradykinesia and impairment of EEG desynchronization in Parkinson's disease. *Movement Disorders, 14*(3), 423-429.

Brown, P., & Marsden, C.D. (1999b). Bradykinesia and impairment of EEG desynchronization in Parkinson's disease. *Movement Disorders, 14*(3), 423-429.

Buxbaum, L.J., Sirigu, A., Schwartz, M.F., & Klatzky, R. (2003). Cognitive representations of hand posture in ideomotor apraxia. *Neuropsychologia, 41*, 1091-1113.

Capone, J.G., Della Sala, S., Spinnler, H., & Venneri, A. (2003). Upper and lower face and ideomotor apraxia in patients with Alzheimer's disease. *Behavioral Neurology, 14*(1-2), 1-8.

Carpenter, M.B. (1991). *Core Text of Neuroanatomy* (Fourth ed.). Baltimore, MD: Williams & Wilkins.

Castiello, U., Bennett, K.M.B., Bonfiglioni, C., & Peppard, R.F. (2000). The reach-to-grasp movement in Parkinson's disease before and after dopaminergic medication. *Neuropsychologia, 38*, 46-59.

Cheek, A., Cormier, K., Repp, A., & Meier, R.P. (2001). Prelinguistic gesture predicts mastery and error in the production of early signs. *Language, 77*(2), 292-323.

Code, C. (1998). Models, theories and heuristics in apraxia of speech. *Clinical Linguistics and Phonetics, 12*(1), 47-65.

Contreras-Vidal, J.L., Teulings, H.L., & Stelmach, G.E. (1998). Elderly subjects are impaired in spatial coordination in fine motor control. *Acta Psychologica, 100*(1-2), 25-35.

Cooper, S. (1953). Muscle spindles in the intrinsic muscles of the human tongue. *Journal of Physiology, 122*, 193-202.

Corcos, D.M., Chen, C.-M., Quinn, N.P., McAuley, J., & Rothwell, J.C. (1996). Strength in Parkinson's disease: Relationship to rate of force generation and clinical status. *Annals of Neurology, 39*, 79-88.

Corina, D., Jezzard, P., Clark, V., Braun, A., Turner, R., & Neville, H.J. (1996). Cortical organization for language in native deaf and hearing signers: Part I. American Sign Language. Paper presented at the Society for Neuroscience Conference, Washington, D.C.

Corina, D.P., Poizner, H., Bellugi, U., Feinberg, T., & O'Grady-Batch, L. (1992a). Dissociation between linguistic and nonlinguistic gestural systems: A case for compositionality. *Brain and Language, 43*, 414-447.

Corina, D.P., Vaid, J., & Bellugi, U. (1992b). The linguistic basis of left hemisphere specialization. *Science, 255*, 1258-1260.

Corina, D.P., & McBurney, S.L. (2001). The neural representation of language in users of American Sign Language. *Journal of Communication Disorders, 34*, 455-471.

Corina, D.P., & Hildebrandt, U.C. (2002). Psycholinguistic investigations of phonological structure in ASL. In R.P. Meier, K. Cormier & D. Quinto-Pozos (Eds.), *Modality and Structure in Signed and Spoken Language* (pp. 88-111). New York: Cambridge University Press.

Corina, D.P., San Jose-Robertson, L., Guillemin, A., High, J., & Braun, A.R. (2003). Language lateralization in a bimanual language. *Journal of Cognitive Neuroscience, 15*(5), 718-730.

Cormier, K., Wechsler, S., & Meier, R.P. (1999). Locus agreement in American Sign Language. In G. Webelhuth, J.-P. Koenig & A. Kathol (Eds.), *Lexical and Constructional Aspects of Linguistic Explanation* (pp. 215-229). Chicago: University of Chicago Press.

Cormier, K.A. (2002). *Grammaticization of Indexic Signs: How American Sign Language Expresses Numerosity*. University of Texas, Austin, TX.

Crasborn, O. (2001). *Phonetic Implementation of Phonological Categories in Sign Language of the Netherlands*. Utrecht: LOT.

Croot, K. (2002). Diagnosis of AOS: Definition and criteria. In N. Helm-Estabrooks & N. Bernstein Ratner (Eds.), *Apraxia of Speech: From Concept to Clinic* (Vol. 23, pp. 267-279). New York: Thieme Medical Publishers.

Crosson, B. (1997). Subcortical limb apraxia. In L.J.G. Rothi & K.M. Heilman (Eds.), *Apraxia: The Neuropsychology of Action* (pp. 207-244). Hove, UK: Psychology Press.

Dabul, B. (2000). *Apraxia Battery for Adults*. Austin, TX: Pro-Ed.

Darkins, A.W., Fromkin, V.A., & Benson, D.F. (1988). A characterization of the prosodic loss in Parkinson's disease. *Brain and Language, 34*, 315-327.

Darley, F.L., Aronson, A.E., & Brown, J.R. (1969a). Clusters of deviant speech dimensions in the dysarthrias. *Journal of Speech and Hearing Research, 12*, 462-496.

Darley, F.L., Aronson, A.E., & Brown, J.R. (1969b). Differential diagnostic patterns of dysarthria. *Journal of Speech and Hearing Research, 12*, 246-269.

Darley, F.L., Aronson, A.E., & Brown, J.R. (1975). *Motor Speech Disorders*. Philadelphia: Saunders.

DeBodt, M.S., Hernandez-Diaz, H.M., & Van De Heyning, P.H. (2002). Intelligibility as a linear combination of dimensions in dysarthric speech. *Journal of Communication Disorders, 35*(3), 283-292.

Defebvre, L., Blatt, J.L., Blond, S., Bourriez, J.L., Gieu, J.D., & Destee, A. (1996). Effect of thalamic stimulation on gait in Parkinson's disease. *Archives of Neurology, 53*(9), 898-903.

Della Sala, S., Francescani, A., & Spinnler, H. (2002). Gait apraxia after bilateral supplementary motor area lesion. *Journal of Neurology, Neurosurgery, and Psychiatry, 72*(1), 77-85.

DeZeeuw, C.I., Simpson, J.I., Hoogenraad, C.C., Galjart, N., Koekkoek, S.K.E., & Ruigrok, T.J.H. (1998). Microcircuitry and function of the inferior olive. *Trends in Neurosciences, 21*(9), 391-400.

DiStefano, M., Morelli, M., Marzi, C.A., & Berlucchi, G. (1980). Hemispheric control of unilateral and bilateral movements of proximal and distal parts of the arm as inferred from simple reaction time to lateralized light stimuli in man. *Experimental Brain Research, 38*, 197-204.

Dominey, P., Hoen, M., Blanc, J., & Lelekov-Boissard, T. (2003). Neurological basis of language and sequential cognition: Evidence from simulation, aphasia, and ERP studies. *Brain and Language, 86*(2), 207-225.

Dronkers, N. (1996). A new brain region for coordinating speech articulation. *Nature, 384*, 159-161.

Duffy, J.R. (1995). *Motor Speech Disorders: Substrates, Differential Diagnosis, and Management*. St Louis: Mosby.

Duffy, J.R., & Folger, W.N. (1996). Dysarthria associated with unilateral central nervous system lesions: A retrospective study. *Journal of Medical Speech-Language Pathology, 4*, 57-70.

Duvoisin, R.C., & Sage, J. (2001). *Parkinson's Disease: A Guide for Patient and Family*. Philadelphia: Lippincott, Williams, & Wilkins.

Dye, M., & Shih, S.-I. (in press). Phonological priming in British Sign Language. In L. Goldstein, D. Whalen & C. Best (Eds.), *Papers in Laboratory Phonology 8: Varieties of Phonological Competence*. Berlin: Mouton deGruyter.

Ellis, L., & Hardcastle, W.J. (2002). Categorical and gradient properties of assimilation in alveolar to velar sequences: evidence from EPG and EMA data. *Journal of Phonetics, 30*(3), 373-396.

Emmorey, K., Corina, D., & Bellugi, U. (1995). Differential processing of topographic and referential functions of space. In K. Emmorey & J. Reilly (Eds.), *Language, Gesture, and Space* (pp. 43-62). Mahwah, NJ: Lawrence Erlbaum.

Emmorey, K., Hickok, G., & Klima, E.S. (1996). The neural organization for sign language: Insights from right hemisphere damaged signers. *Brain and Cognition, 32*(2), 212-215.

Emmorey, K., Damasio, H., McCullough, S., Grabowski, T., Ponto, L.L.B., Hichwa, R.D., & Bellugi, U. (2002). Neural systems underlying spatial language in American Sign Language. *Neuroimage, 17*, 812-824.

Emmorey, K., Grabowski, T., McCullough, S., Damasio, H., Ponto, L.L.B., Hichwa, R.D., & Bellugi, U. (2003). Neural systems underlying lexical retrieval for sign language. *Neuropsychologia, 41*, 85-95.

Enderby, P. (1983). *Frenchay dysarthria assessment.* San Diego: College Hill Press.

Enderby, P. (1986). Relationships between dysarthric groups. *British Journal of Disorders of Communication, 21*, 189-197.

Erimaki, S., & Christakos, C.N. (1999). Occurrence of widespread motor-unit firing correlations in muscle contractions: their role in the generation of tremor and time-varying voluntary force. *Journal of Neurophysiology, 82*(5), 2839-2846.

Fabbro, F. (2000). Introduction to language and cerebellum. *Journal of Neurolinguistics, 13*, 83-94.

Fabbro, F., Moretti, R., & Bava, A. (2000). Language impairments in patients with cerebellar lesions. *Journal of Neurolinguistics, 13*, 173-188.

Fahn, S., & Elton, R.L. (1987). Unified Parkinson's Disease Rating Scale. In S. Fahn, C.D. Marsden, D. Calne & M. Goldstein (Eds.), *Recent Developments in Parkinson's Disease* (pp. 153-163). Florham Park, NJ: Macmillan.

Fahn, S., Greene, P.E., Ford, B., & Bressman, S.B. (1998). *Handbook of Movement Disorders*. Philadephia: Current Medicine, Inc.

Fallang, B., Saugstad, O.D., & Hadders-Algra, M. (2000). Goal directed reaching and postural control in supine position in healthy infants. *Behavioural Brain Research, 115*, 9-18.

Fiez, J.A., Raife, E.A., Balota, D.A., Schwarz, J.P., Raichle, M.E., & Petersen, S.E. (1996). A positron emission tomography study of the short-term maintenance of verbal information. *Journal of Neuroscience, 16*(2), 808-822.

Fiez, J.A., & Petersen, S.E. (1998). Neuroimaging studies of word reading. *Proceedings of the National Academy of Sciences, 95*(3), 914-921.

Fischer, R. (1988). Ist die Gebärdensprache der Gehörlosen eine Sprache? *Das Zeichen, 2*(6), 6-14.

Fitts, P.M., & Peterson, J.R. (1964). Information capacity of discrete motor responses. *Journal of Experimental Psychology, 67*, 103-112.

Fitzgerald, L.K., McKelvey, J.R., & Szeligo, F. (2002). Mechanisms of dressing apraxia: a case study. *Neuropsychiatry Neuropsychology and Behavioural Neurology, 15*(2), 148-155.

Flynn, F.G., Benson, D.F., & Ardila, A. (1999). Anatomy of the insula- functional and clinical correlates. *Aphasiology, 13*(1), 55-78.

Folstein, M.F., Folstein, S.E., & McHugh, P.R. (1975). Mini-mental state: A practical method for grading the state of patients for the clinician. *Journal of Psychiatric Research, 12*, 189-198.

Forrest, K., Weismer, G., & Turner, G.S. (1989). Kinematic, acoustic, and perceptual analyses of connected speech produced by Parkinsonian and normal geriatric adults. *Journal of the Acoustical Society of America, 85*(6), 2608-2622.

Forrest, K., Adams, S., McNeil, M.R., & Southwood, H. (1991). Kinematic, electromyographic, and perceptual evaluation of speech apraxia, conduction aphasia, ataxic dysarthria, and normal speech production. In C.A. Moore, K.M. Yorkston & D.R. Beukelman (Eds.), *Dysarthria and Apraxia of Speech: Perspectives on Management* (pp. 147-171). Baltimore, MD: Paul H. Brookes.

Freund, H.-J. (1987). Differential effects of cortical lesions in humans. *Ciba Foundation Symposium, 132*, 269-281.

Friedman, L.A. (1976). *Phonology of a Soundless Language*. University of California, Berkeley, Berkeley, CA.

Fry, D.B. (1979). *The Physics of Speech*. Cambridge: Cambridge University Press.

Gallena, S., Smith, P.J., Zeffiro, T., & Ludlow, C.L. (2001). Effects of levodopa on laryngeal muscle activity for voice onset and offset in Parkinson disease. *Journal of Speech, Language, and Hearing Research, 44*(6), 1284-1299.

Gentil, M., Chauvin, P., Pinto, S., Pollak, P., & Benabid, A.-L. (2001). Effect of bilateral stimulation of the subthalamic nucleus on Parkinsonian voice. *Brain and Language, 78*, 233-240.

Gentilucci, M., & Negrotti, A. (1999). Planning and executing an action in Parkinson's disease. *Movement Disorders, 14*(1), 69-79.

Georgiou, N., Bradshaw, J.L., Iansek, R., Phillips, J.G., Mattingley, J.B., & Bradshaw, J.A. (1994). Reduction in external cues and movement sequencing in Parkinson's disease. *Journal of Neurology, Neurosurgery, and Psychiatry, 57*, 368-370.

Gerardin, E., Sirigu, A., Lehericy, S., Poline, J.P., Gaymard, B., Marsault, C., Agid, Y., & LeBihan, D. (2000). Partially overlapping neural networks for real and imagined hand movements. *Cerebral Cortex, 10*, 1093-1104.

Ghez, C., Cooper, S., & Martin, J. (1996). Kinematic and dynamic factors in the coordination of prehension movements. In A.M. Wing, P. Haggard & J.R. Flanagan (Eds.), *Hand and Brain: The Neurophysiology and Psychology of Hand Movements* (pp. 187-212). San Diego, CA: Academic Press.

Goberman, A., Coelho, C., & Robb, M. (2002). Phonatory characteristics of Parkinsonian speech before and after morning medication: The ON and OFF states. *Journal of Communication Disorders, 35*(3), 217-239.

Golbe, L.I., Miller, D.C., & Duvoisin, R.C. (1990). Autosomal dominant Lewy-body Parkinson's disease. *Advances in Neurology, 53*, 287-292.

Goldenberg, G. (1995). Imitating gestures and manipulating a mannikin-- the representation of human-body in ideomotor apraxia. *Neuropsychologia, 33*, 63-72.

Goldenberg, G., Hartmann, K., & Schlott, I. (2003). Defective pantomime of object use in left brain damage: apraxia or asymbolia? *Neuropsychologia, 41*, 1565-1573.

Goodwin, C. (2000). Gesture, aphasia, and interaction. In D. McNeill (Ed.), *Language and Gesture* (pp. 84-98). Cambridge: Cambridge University Press.

Goozee, J.V., Murdoch, B.E., Theodoros, D.G., & Stokes, P.D. (2000). Kinematic analysis of tongue movements in dysarthria following traumatic brain injury using electromagnetic articulography. *Brain Injury, 14*(2), 153-174.

Goozee, J.V., Murdoch, B.E., & Theodoros, D.G. (2001). Physiological assessment of tongue function in dysarthria following traumatic brain injury. *Logopedics, phoniatrics, vocology, 26*, 51-65.

Gracco, V.L., & Abbs, J.H. (1987). Programming and execution processes of speech movement control: potential neural correlates. In E. Keller & M. Gopnik (Eds.), *Motor and Sensory Processes of Language* (pp. 163-201). Hillsdale, NJ: Lawrence Erlbaum.

Gray, H. (2000). *Anatomy of the Human Body* (Twentieth ed.). New York: Bartleby.com.

Graybiel, A.M., Aosaki, T., Flaherty, A.W., & Kimura, M. (1994). The basal ganglia and adaptive motor control. *Science, 265*(1826-1831).

Graziano, M.S.A., & Gross, C.G. (1993). A bimodal map of space: somatosensory receptive fields in the macaque putamen with corresponding visual receptive fields. *Experimental Brain Research, 97*, 96-109.

Grove, N. (1990). Developing intelligible signs with learning-disabled students: A review of the literature and an assessment. *British Journal of Disorders of Communication, 25*(3), 265-293.

Grove, N., & Dockrell, J. (2000). Multisign combinations by children with intellectual impairments: An analysis of language skills. *Journal of Speech Language and Hearing Research, 43*(2), 309-323.

Haaland, K.Y., & Harrington, D.L. (1994). Limb-sequencing deficits after left but not right-hemisphere damage. *Brain and Cognition, 24*, 104-122.

Hallett, M., & Khoshbin, S. (1980). A physiological mechanism of bradykinesia. *Brain, 103*, 301-314.

Halsband, U., Schmitt, J., Weyers, M., Binkofski, F., Grutzner, G., & Freund, H.-F. (2001). Recognition and imitation of pantomimed motor acts after unilateral parietal and premotor lesions: a perspective on apraxia. *Neuropsychologia, 39*(2), 200-216.

Hamilton, J.M., Haaland, K.Y., Adair, J.C., & Brandt, J. (2003). Ideomotor limb apraxia in Huntington's disease: implications for corticostriate involvement. *Neuropsychologia, 41*, 614-621.

Hamsher, K., Capruso, D.X., & Benton, A. (1992). Visuospatial judgment and right hemisphere disease. *Cortex, 3*, 493-495.

Hanakawa, T., Katsumi, Y., Fukuyama, H., Honda, M., Hayashi, T., Kimura, J., & Shibasaki, H. (1999). Mechanisms underlying gait disturbance in Parkinson's disease: A single photon emission computed tomography. *Brain, 122*, 1271-1282.

Hanna-Pladdy, B., Heilman, K.M., & Foundas, A.L. (2001). Cortical and subcortical contributions to ideomotor apraxia: Analysis of task demands and error types. *Brain, 124*, 2513-2527.

Hanson, D., Ludlow, C., & Bassich, C. (1983). Vocal fold paresis in Shy-Drager syndrome. *Annals of Otology, Rhinology, and Laryngology, 92*, 85-90.

Harrington, D.L., Rao, S.M., Haaland, K.Y., Bobholz, J.A., Mayer, A.R., Binderx, J.R., & Cox, R.W. (2000). Specialized neural systems underlying representations of sequential movements. *Journal of Cognitive Neuroscience, 12*(1), 56-77.

Hartman, D.E., & Abbs, J.H. (1988). Dysarthrias of movement disorders. In J. Jankovic & E. Tolosa (Eds.), *Advances in Neurology* (Vol. 49, pp. 289-306). New York: Raven Press.

Hartman, D.E., & Abbs, J.H. (1992). Dysarthria associated with focal upper motor neuron lesion. *European Journal of Disorders of Communication, 27*(3), 187-196.

Heilman, K.M., Coyle, J.M., Gonyea, E.F., & Geschwind, N. (1973). Apraxia and agraphia in a left hander. *Brain, 96*, 21-28.

Heilman, K.M., & Rothi, L.J.G. (1997). Limb apraxia: a look back. In L.J.G. Rothi & K.M. Heilman (Eds.), *Apraxia: The Neuropsychology of Action* (pp. 7-18). Hove, UK: Psychology Press.

Heilman, K.M., & Adams, D.J. (2003). Callosal neglect. *Archives of Neurology, 60*(2), 276-279.

Hermsdorfer, J., Mai, N., Spatt, J., Marquardt, C., Veltkamp, R., & Goldenberg, G. (1996). Kinematic analysis of movement imitation in apraxia. *Brain, 119*, 1575-1586.

Hermsdorfer, J., Goldenburg, G., Wachsmuth, C., Conrad, B., Ceballos-Baumann, A.O., Partenstein, P., Schwaiger, M., & Boecker, H. (2001). Cortical correlates of gesture processing: Clues to the cerebral mechanisms underlying apraxia during the imitation of meaningless gestures. *Neuroimage, 14*, 149-161.

Hertrich, I., & Ackermann, H. (1997). Articulatory control of phonological vowel length contrasts: Kinematic analysis of labial gestures. *Journal of the Acoustical Society of America, 102*(1), 523-536.

Hertrich, I., Spieker, S., & Ackermann, H. (1998). Gender-specific phonatory dysfunctions in disorders of the basal ganglia and cerebellum: Acoustic and perceptual characteristics. In W. Ziegler & K. Deger (Eds.), *Clinical Phonetics and Linguistics* (pp. 448-457). London: Whurr.

Hertrich, I., & Ackermann, H. (1999). Temporal and spectral aspects of coarticulation in ataxic dysarthria: an acoustic analysis. *Journal of Speech, Language, and Hearing Research, 42*, 367-381.

Hickok, G., Klima, E., Kritchevsky, M., & Bellugi, U. (1995). A case of sign-blindness following left occipital damage in a deaf signer. *Neuropsychologia, 33*(12), 1597-1606.

Hickok, G., Bellugi, U., & Klima, E.S. (1996). The neurobiology of sign language and its implications for the neural basis of language. *Nature, 381*, 699-702.

Hickok, G., Bellugi, U., & Klima, E.S. (1998). What's right about the neural organization of sign language? A perspective on recent neuroimaging results. *Trends in Cognitive Science, 2*, 465-468.

Hickok, G., Wilson, M., Clark, K., Klima, E.S., Kritchevsky, M., & Bellugi, U. (1999). Discourse deficits following right hemisphere damage in deaf signers. *Brain and Language, 66*(2), 233-248.

Hikosaka, O., Nakahara, H., Rand, M.K., Sakai, K., Lu, X., Nakamura, K., Miyachi, S., & Doya, K. (1999). Parallel neural networks for learning sequential procedures. *Trends in Neurosciences, 22*(10), 464-471.

Hirsch, E., Graybiel, A.M., & Agid, Y.A. (1988). Melanized dopaminergic neurons are differentially susceptible to degeneration in Parkinson's disease. *Nature, 334*(6180), 345-348.

Ho, A.K., Bradshaw, J.L., Cunnington, R., Phillips, J.G., & Iansek, R. (1998). Sequence heterogeneity in Parkinsonian speech. *Brain and Language, 64*(1), 122-145.

Hoehn, M.M., & Yahr, M.D. (1967). Parkinsonism: onset, progression, and mortality. *Neurology, 17*, 427-442.

Hohenberger, A., Happ, D., & Leuninger, H. (2002). Modality-dependent aspects of sign language production: Evidence from slips of the hand and their repairs in German Sign Language. In R.P. Meier, K. Cormier & D. Quinto-Pozos (Eds.), *Modality and Structure in Signed and Spoken Languages* (pp. 112-142). New York: Cambridge University Press.

Holmes, G. (1939). The cerebellum of man. *Brain, 62*, 1-30.

Honda, M., Fujino, A., & Kaburagi, T. (2002). Compensatory responses of articulators to unexpected perturbation of the palate shape. *Journal of Phonetics, 30*(3), 281-302.

Howard, D., & Patterson, K. (1992). *Pyramids and Palm Trees: A Test of Semantic Access from Pictures and Words*. Suffolk, VA: Thames Valley Test Co.

Iansek, R., Bradshaw, J.L., Phillips, J.G., Cunnington, R., & Morris, M.E. (1995). Interaction of the basal ganglia and supplementary motor area in the elaboration of movement. In D.J. Glencross & J.P. Piek (Eds.), *Motor Control and Sensory Motor Integration: Issues and Directions* (pp. 37-59). Amsterdam: Elsevier.

Iberall, T., & Fagg, A.H. (1996). Neural network models for selecting hand shapes. In A.M. Wing, P. Haggard & J.R. Flanagan (Eds.), *Hand and Brain: The Neurophysiology and Psychology of Hand Movements* (pp. 243-264). San Diego, CA: Academic Press.

Ietswaart, M., Carey, D.P., Della Sala, S., & Dijkhuizen, R.S. (2001). Memory-driven movements in limb apraxia: is there evidence for impaired communication between the dorsal and ventral streams? *Neuropsychologia, 39*, 950-961.

Illes, J., Metter, E.J., Hanson, W.R., & Iritani, S. (1988). Language production in Parkinson's disease: acoustic and linguistic considerations. *Brain and Language, 33*, 146-160.

Ingvarsson, P.E., Gordon, A.M., & Forssberg, H. (1997). Coordination of manipulative forces in Parkinson's disease. *Experimental Neurology, 145*, 489-501.

Ivry, R.B., & Keele, S.W. (1989). Timing functions of the cerebellum. *Journal of Cognitive Neuroscience, 1*, 136-152.

Jaeger, M., Hertrich, I., Stattrop, U., Schoenle, P.-W., & Ackermann, H. (2000). Speech disorders following severe traumatic brain injury: Kinematic analysis of syllable repetitions using electromagnetic articulography. *Folia Phoniatrica et Logopedica, 52*, 187-196.

Jeannerod, M., & Decety, J. (1995). Mental motor imagery: a window into the representational stages of action. *Current Opinion in Neurobiology, 5*, 727-732.

Jueptner, M., Jenkins, I.H., Brooks, D.J., Frackowiak, R.S.J., & Passingham, R.E. (1996). The sensory guidance of movement: a comparison of the cerebellum and basal ganglia. *Experimental Brain Research, 112*, 462-474.

Jueptner, M., & Weiller, C. (1998). A review of differences between basal ganglia and cerebellar control of movements as revealed by functional imaging studies. *Brain, 121*, 1437-1449.

Kandel, E.R., Schwartz, J.H., & Jessell, T.M. (1991). *Principles of Neural Science* (Third ed.). Norwalk, CT: Appleton & Lange.

Keele, S.W., & Ivry, R. (1990). Does the cerebellum provide a common computation for diverse tasks? A timing hypothesis. *Annals of the New York Academy of Sciences, 608*, 179-207.

Kegl, J., & Poizner, H. (1992). Preservation of syntactic distinctions in a moderate to severe Parkinsonian signer. Paper presented at the CUNY Sentence Processing Conference.

Kegl, J., Baynes, K., Brentari, D., & Poizner, H. (1996). Landau-Kleffner syndrome: modality independence of linguistic competence. Poster presented at the 26th Annual Meeting of the Society for Neuroscience. Washington, D.C.

Kegl, J., & Poizner, H. (1997). Crosslinguistic/crossmodal syntactic consequences of left-hemisphere damage: evidence from an aphasic signer and his identical twin. *Aphasiology, 11*(1), 1-37.

Kegl, J., Cohen, H., & Poizner, H. (1999). Articulatory consequences of Parkinson's disease: perspectives from two modalities. *Brain and Cognition, 40*, 355-386.

Kempler, D., & Van Lancker, D. (2002). Effect of speech task on intelligibility in dysarthria: A case study of Parkinson's disease. *Brain and Language, 80*, 449-464.

Kendon, A. (1984). Knowledge of sign language in an Australian aboriginal community. *Journal of Anthropological Research, 40*, 556-576.

Kent, R., Netsell, R., & Abbs, J.H. (1979). Acoustic characteristics of dysarthria associated with cerebellar disease. *Journal of Speech and Hearing Disorders, 22*, 613-626.

Kent, R., & Rosenbek, J.C. (1982). Prosodic disturbances and neurologic lesion. *Brain and Language, 15*, 259-291.

Kent, R.D. (1997). *The Speech Sciences*. San Diego: Singular.

Kent, R.D., Kent, J.F., Rosenbek, J.C., Vorperian, H.K., & Weismer, G. (1997). A speaking task analysis of the dysarthria in cerebellar disease. *Folia Phoniatrica Logopaedica, 49*, 63-82.

Kent, R.D., Kent, J.F., Duffy, J.R., & Weismer, G. (1998). The dysarthrias: Speech-voice profiles, related dysfunctions, and neuropathologies. *Journal of Medical Speech-Language Pathology, 6*, 165-211.

Kent, R.D. (2000). Research on speech motor control and its disorders: A review and prospective. *Journal of Communication Disorders, 33*, 391-428.

Kent, R.D., Kent, J.F., Duffy, J.R., Thomas, J.E., Weismer, G., & Stuntebeck, S. (2000). Ataxic dysarthria. *Journal of Speech, Language, and Hearing Research, 43*, 1275-1289.

Kent, R.D., Duffy, J.R., Slama, A., Kent, J.F., & Clift, A. (2001). Clinicoanatomic studies in dysarthria: Review, critique, and directions for research. *Journal of Speech, Language, and Hearing Research, 44*, 535-551.

Kim, J.S., Kwon, S.U., & Lee, T.G. (2003). Pure dysarthria due to small cortical stroke. *Neurology, 60*, 1178-1180.

Kimura, D., & Archibald, Y. (1974). Motor functions of the left hemisphere. *Brain, 97*(2), 337-350.

Kimura, D. (1977). Acquisition of a motor skill after left-hemisphere damage. *Brain, 100*, 527-542.

Kimura, D. (1993). *Neuromotor Mechanisms in Human Communication*. Oxford: Oxford University Press.

Klasner, E.R., & Yorkston, K.M. (2000). Dysarthria in ALS: A method for obtaining the everyday listener's perception. *Journal of Medical Speech-Language Pathology, 8*(4), 261-264.

Klima, E., & Bellugi, U. (1979). *The Signs of Language*. Cambridge, MA: Harvard University Press.

Krystowiak, P., Blatt, J.L., Bourriez, J.L., Duhamel, A., Perina, M., Blond, S., Guieu, J.D., Destee, A., & Defebvre, L. (2003). Effects of subthalamic nucleus stimulation and levodopa treatment on gait abnormalities in Parkinson's disease. *Archives of Neurology, 60*(1), 80-84.

Laffont, I., Briand, E., Dizien, O., Combeaud, M., Bussel, B., Revol, M., & Roby-Brami, A. (2000). Kinematics of prehension and pointing movements in C6 quadriplegic patients. *Spinal Cord, 38*(6), 354-362.

Lalonde, R., & Botez-Marquard, T. (2000). Neuropsychological deficits of patients with chronic or acute cerebellar lesions. *Journal of Neurolinguistics, 13*, 117-128.

Lamberti, P., De Mari, M., Zenzola, A., Aniello, M.S., & Defazio, G. (2002). Frequency of apraxia of eyelid opening in the general population and in patients with extrapyramidal disorders. *Neurological Sciences, 23, Suppl 2*, S81-82.

Lang, C.E., & Bastian, A.J. (2002). Cerebellar damage impairs automaticity of a recently practiced movement. *Journal of Neurophysiology, 87*, 1336-1347.

Laplane, D., & Degos, J.D. (1983). Motor neglect. *Journal of Neurology, Neurosurgery, and Psychiatry, 46*, 152-158.

Laurent-Vannier, A., Pradat-Diehl, P., Chevignard, M., Abada, G., & De Agostini, M. (2003). Spatial and motor neglect in children. *Neurology, 60*, 202-207.

Lausberg, H., Cruz, R.F., Kita, S., Zaidel, E., & Ptito, A. (2003). Pantomime to visual presentation of objects: left hand dyspraxia in patients with complete callosotomy. *Brain, 126*(2), 343-360.

Leiguarda, R.C., Merello, M., Nouzeilles, M.I., Balej, J., Rivero, A., & Nogues, M. (2003). Limb-kinetic apraxia in corticobasal degeneration: clinical and kinematic features. *Movement Disorders, 18*(1), 49-59.

Lelekov-Boissard, T., & Dominey, P. (2002). Human brain potentials reveal similar processing of non-linguistic abstract structure and linguistic syntactic structure. *Neurophysiol Clin, 32*(1), 72-84.

Levanen, S., Uutela, K., Salenius, S., & Hari, R. (2001). Cortical representation of sign language: Comparison of Deaf signers and hearing non-signers. *Cerebral Cortex, 11*, 506-512.

Lidsky, T.I., & Brown, L.L. (1999). Behavioural context and a distributed system: metabolic mapping studies of the basal ganglia. *Canadian Journal of Experimental Psychology, 53*(1), 35-43.

Lieberman, P., & Blumstein, S.E. (1988). *Speech Physiology, Speech Perception, and Acoustic Phonetics*. Cambridge: Cambridge University Press.

Lieberman, P., Friedman, J., & Feldman, L. (1990). Syntax comprehension deficits in Parkinson's disease. *Journal of Nervous and Mental Disease, 178*(6), 360-365.

Lieberman, P., Kako, E., Friedman, J., Tajchman, G., Feldman, L., & Jiminez, E. (1992). Speech production, syntax comprehension, and cognitive deficits in Parkinson's disease. *Brain and Language, 43*(2), 169-189.

Lieberman, P. (1995). Manual versus speech motor control and the evolution of language. *Behavioral and Brain Sciences, 18*(1), 197-198.

Liepmann, H. (1900). Das Krankheitshild der Apraxie (motorischen/Asymbolie). *Monatschrift fur Psychiatrie und Neurologie, 8*, 15-44, 102-132, 182-197.

Liepmann, H. (1905). Die linke Hemisphare und das Handeln. *Munchener Medizinische Wochenschrift, 49*, 2322-2326, 2375-2378.

Liepmann, H. (1920). Apraxia. *Ergebnisse der Gesamten Medizin, 1*, 516-543.

Liepmann, H. (1977). The syndrome of apraxia (motor asymboly) based on a case of unilateral apraxia. (A translation from *Monatschrift fur Psychiatrie und Neurologie 1900, 8, 15-44*). In D.A. Rottenberg & F.H. Hockberg (Eds.), *Neurological Classics in Modern Translation*. New York: Macmillan.

Linebaugh, C.W., & Wolfe, V.E. (1984). Relationships between articulation rate, intelligibility, and naturalness in spastic and ataxic speakers. In M. McNeil, J. Rosenbek & A. Aronson (Eds.), *The Dysarthrias: Physiology, Acoustics, Perception, Management* (pp. 197-206). San Diego: College Hill Press.

Liss, J.M., Spitzer, S.M., Caviness, J.N., Adler, C., & Edwards, B.W. (2000). Lexical boundary error analysis in hypokinetic and ataxic dysarthria. *Journal of the Acoustical Society of America, 107*(6), 3415-3424.

Loew, R.C., Kegl, J.A., & Poizner, H. (1995). Flattening of distinctions in a Parkinsonian signer. *Aphasiology, 9*(4), 381-396.

Loew, R.C., Kegl, J.A., & Poizner, H. (1997). Fractionation of the components of role play in a right-hemispheric lesioned signer. *Aphasiology, 11*(3), 263-281.

Love, R.J., & Webb, W.G. (2001). *Neurology for the Speech-Language Pathologist.* Woburn, MA: Butterworth-Heinemann.

Lu, F.L., Duffy, J.R., & Maraganore, D. (1992). Neuroclinical and speech characteristics in progressive supranuclear palsy and Parkinson's disease: a retrospective study (Vol. Paper presented at the Conference on Motor Speech). Boulder, CO.

Lu, X., Hikosaka, O., & Miyachi, S. (1998). Role of monkey cerebellar nuclei in skill for sequential movement. *Journal of Neurophysiology, 79*, 2245-2254.

Ludlow, C.L., & Bassich, C.J. (1983). The results of acoustic and perceptual assessment of two types of dysarthria. In W.R. Berry (Ed.), *Clinical Dysarthria* (pp. 121-147). San Diego: College Hill Press.

Ludlow, C.L., & Bassich, C.J. (1984). Relationships between perceptual ratings and acoustic measures of hypokinetic speech. In M. McNeil, J. Rosenbek & A. Aronson (Eds.), *The Dysarthrias: Physiology, Acoustics, Perception, Management* (pp. 163-196). San Diego: College Hill Press.

Luschei, E.S., Ramig, L.O., Baker, K.L., & Smith, M.E. (1999). Discharge characteristics of laryngeal single motor units during phonation in young and older adults and in persons with Parkinson's disease. *Journal of Neurophysiology, 81*, 2131-2139.

MacNeilage, P.F., Davis, B.L., Kinney, A., & Matyear, C.L. (1999). Origin of serial-output complexity in speech. *Psychological Science, 10*, 459-460.

MacNeilage, P.F., Davis, B.L., Kinney, A., & Matyear, C.L. (2000). The motor core of speech: a comparison of serial organization patterns in infants and languages. *Child Development, 71*, 153-163.

MacSweeney, M., Woll, B., Campbell, R., Calvert, G.A., McGuire, P.K., David, A.S., Simmons, A., & Brammer, M.J. (2002a). Neural correlates of British Sign Language comprehension: Spatial processing demands of topographic language. *Journal of Cognitive Neuroscience, 14*(7), 1064-1075.

MacSweeney, M., Woll, B., Campbell, R., McGuire, P.K., David, A.S., Williams, S.C.R., Suckling, J., Calvert, G.A., & Brammer, M.J. (2002b). Neural systems underlying British Sign Language and audiovisual English processing in native users. *Brain, 125*, 1583-1593.

Marien, P., Engelborghs, S., Pickut, B.A., & DeDeyn, P.P. (2000). Aphasia following cerebellar damage: fact or fantasy? *Journal of Neurolinguistics, 13*, 145-171.

Marsden, C.D. (1982). The mysterious motor function of the basal ganglia. *Neurology, 32*, 514-539.

Marsden, C.D. (1984). Which motor disorder in Parkinson's disease indicates the true motor function of the basal ganglia? *Ciba Foundation Symposium, 107*, 225-241.

Marshall, J., Atkinson, J., Smulovitch, E., Thacker, A., & Woll, B. (2004). Aphasia in a user of British Sign Language: Dissociation between sign and gesture. *Cognitive Neuropsychology, 21*(5), 537-554.

Marshall, J., Atkinson, J.R., Woll, B., & Thacker, A. (under review). Aphasia in a bilingual user of British Sign Language and English: Effects of cross-linguistic cues.

Mayberry, R., & Groschler, R. (1994). The critical period is most critical for first language acquisition: Evidence from American Sign Language. Paper presented at the meeting of the Linguistics Society of America. Boston, MA.

McComas, A.J. (1998). Oro-facial muscles: internal structure, function and ageing. *Gerontology, 15*(1), 3-14.

McHenry, M. (2001). The effect of a dopamine agonist on dysarthric speech production: A case study. *Journal of Communication Disorders, 34*, 397-414.

McNeil, M.R., Robin, D.A., & Schmidt, R.A. (1997). Apraxia of speech: Definition, differentiation, and treatment. In M.R. McNeil (Ed.), *Clinical Management of Sensorimotor Speech Disorders* (pp. 311-344). New York: Thieme.

McNeill, D. (1992). *Hand and Mind.* Chicago: University of Chicago Press.

Meador, K.J., Watson, R.T., Bowers, D., & Heilman, K.M. (1986). Hypometria with hemispatial and limb motor neglect. *Brain, 109*(2), 293-305.

Meier, R.P., McGarvin, L., Zakia, R.A.E., & Willerman, R. (1997). Silent mandibular oscillations in vocal babbling. *Phonetica, 54*, 153-171.

Meier, R.P., Mauk, C., Mirus, G., & Conlin, K.E. (1998). Motoric constraints on early sign acquisition. In E. Clark (Ed.), *Proceedings of the Child Language Research Forum* (Vol. 29, pp. 63-72). Stanford: CSLI.

Meier, R.P. (2000). Shared motoric factors in the acquisition of sign and speech. In K. Emmorey & H. Lane (Eds.), *The Signs of Language Revisited: An Anthology to Honor Ursula Bellugi and Edward Klima* (pp. 331-354). Mahwah, NJ: Lawrence Erlbaum.

Meier, R.P. (2002). Why different, why the same? Explaining effects and non-effects of modality upon linguistic structure in sign and speech. In R.P. Meier, K. Cormier & D. Quinto-Pozos (Eds.), *Modality and Structure in Signed and Spoken Language* (pp. 1-26). New York: Cambridge University Press.

Meister, I.G., Boroojerdi, B., Foltys, H., Sparing, R., Huber, W., & Topper, R. (2003). Motor cortex hand area and speech: implications for the development of language. *Neuropsychologia, 41*(4), 401-406.

Merians, A.S., Clark, M., Poizner, H., Jacobs, D.H., Adair, J.C., Macauley, B., Gonzalez Rothi, L.J., & Heilman, K.M. (1999). Apraxia differs in corticobasal degeneration and left-parietal stroke: A case study. *Brain and Cognition, 40*(2), 314-335.

Metter, E.J., & Hanson, W.R. (1991). Dysarthria in progressive supranuclear palsy. In C.A. Moore, K.M. Yorkston & D.R. Beukelman (Eds.), *Dysarthria and Apraxia of Speech: Perspectives on Management* (pp. 127-136). Baltimore: Paul H. Brookes Publishing Co.

Meunier, S., Pol, S., Houeto, J.L., & Vidailhet, M. (2000). Abnormal reciprocal inhibition between antagonist muscles in Parkinson's disease. *Brain, 123*, 1017-1026.

Miller, N. (2002). The neurological bases of apraxia of speech. In N. Helm-Estabrooks & N. Bernstein Ratner (Eds.), *Apraxia of Speech: From Concept to Clinic* (Vol. 23, pp. 223-230). New York: Thieme Medical Publishers.

Mirus, G., Rathmann, C., & Meier, R.P. (2001). Proximalization and distalization of sign movements in adult learners. In V. Dively, M. Metzger, S. Taub & A.M. Baer (Eds.), *Signed Languages: Discoveries from International Research* (pp. 103-119). Washington, D.C.: Gallaudet Press.

Morgan, G., Smith, N., Tsimpli, I.-M., & Woll, B. (2002). Language against the odds: the learning of BSL by a polyglot savant. *Journal of Linguistics, 38*(1), 1-42.

Morris, M.K. (1997). Developmental dyspraxia. In L.J.G. Rothi & K.M. Heilman (Eds.), *Apraxia: The Neuropsychology of Action* (pp. 245-268). Hove, UK: Psychology Press.

Muller, J., Wenning, G.K., Verny, M., McKee, A., Chaudhuri, K.R., Jellinger, K., Poewe, W., & Litvan, I. (2001). Progression of dysarthria and dysphagia in postmortem-confirmed Parkinsonian disorders. *Archives of Neurology, 58*(2), 259-264.

Murdoch, B.E., & Theodoros, D.G. (1998). Ataxic dysarthria. In B.E. Murdoch (Ed.), *Dysarthria: A Physiological Approach to Assessment and Treatment* (pp. 242-265). Cheltenham, UK: Stanley Thornes.

Murray, L., & Lenz, L. (2001). Productive syntax abilities in Huntington's and Parkinson's diseases. *Brain and Cognition, 46*(1-2), 213-219.

Myers, P.S. (1997). Right hemisphere syndrome. In L.L. LaPointe (Ed.), *Aphasia and Related Neurogenic Language Disorders* (2nd edition ed., pp. 201-225). New York: Thieme.

Natsopoulos, D., Grouios, G., Bostantzopoulou, S., Mentenopoulos, G., Katsarou, Z., & Logothetis, J. (1993). Algorithmic and heuristic strategies in comprehension of complement clauses by patients with Parkinson's disease. *Neuropsychologia, 31*(9), 951-964.

Neidle, C., Kegl, J., MacLaughlin, D., Bahan, B., & Lee, R.G. (2000). *The Syntax of American Sign Language: Functional Categories and Hierarchical Structure.* Cambridge, MA: MIT Press.

Netsell, R., Daniel, B., & Celesia, G.G. (1975). Acceleration and weakness in parkinsonian dysarthria. *Journal of Speech and Hearing Disorders, 40*(2), 170-178.

Neville, H., Bavelier, D., Corina, D., Rauschecker, J., Karni, A., Lalwani, A., Braun, A., Clark, V., Jezzard, P., & Turner, R. (1998). Cerebral organization for language in deaf and hearing subjects: Biological constraints and effects of experience. *Proceedings of the National Academy of Sciences, 95*, 922-929.

Newport, E. (1990). Maturational constraints on language learning. *Cognitive Science, 14*, 11-28.

Nielsen, J.M. (1962). Agnosias, apraxias, speech, and aphasia. In A.B. Baker (Ed.), *Clinical Neurology* (Second ed., pp. 433-459). New York: Hoeber.

Nishimura, H., Hashikawa, K., Doi, K., Iwaki, T., Watanabe, Y., Kusuoka, H., Nishimura, T., & Kubo, T. (1999). Sign language 'heard' in the auditory cortex. *Nature, 397*, 116.

Nishio, M., & Niimi, S. (2001). Speaking rate and its components in dysarthric speakers. *Clinical Linguistics and Phonetics, 15*(4), 309-317.

Nussbaum, R., & Polymeropoulos, M. (1997). Genetics of Parkinson's disease. *Human Molecular Genetics, 6*(10), 1687-1691.

Ochipa, C., Rothi, L.J., & Heilman, K.M. (1992). Conceptual apraxia in Alzheimer's disease. *Brain, 115*, 1061-1071.

Ohyama, T., Nores, W., Murphy, M., & Mauk, M. (2003). What the cerebellum computes. *Trends in Neurosciences, 26*(4), 222-227.

Oldfield, R.C. (1971). The assessment and analysis of handedness: the Edinburgh Inventory. *Medica Neuropsychologia, 9*, 97-113.

Ostry, D.J., & Cooke, J.D. (1987). Kinematic patterns in speech and limb movements. In E. Keller & M. Gopnik (Eds.), *Motor and Sensory Processes of Language* (pp. 223-235). Hillsdale, NJ: Lawrence Erlbaum.

Ostry, D.J., Gribble, P.L., & Gracco, V.L. (1996). Coarticulation of jaw movements in speech production: Is sensitivity in speech kinematics centrally planned? *Journal of Neuroscience, 16*(4), 1570-1579.

Ozsancak, C., Auzou, P., & Hannequin, D. (2000). Dysarthria and orofacial apraxia in corticobasal degeneration. *Movement Disorders, 15*(5), 905-910.

Papapetropoulos, S., Paschalis, C., Athanassiadou, A., Papadimitriou, A., Ellul, J., Polymeropoulos, M.H., & Papapetropoulos, T. (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.

Park, E.S., Park, C., Lee, H.J., Kim, D.Y., Lee, D.S., & Cho, S.R. (2003). The characteristics of sit-to-stand transfer in young children with spastic cerebral palsy based on kinematic and kinetic data. *Gait and Posture, 17*(1), 43-49.

Patterson, M.D., & Bly, B.M. (1999). The brain basis of syntactic processes: architecture, ontogeny, and phylogeny. In B.M. Bly & D.E. Rumelhart (Eds.), *Cognitive Science: Handbook of Perception and Cognition* (pp. 255-318). San Diego, CA: Academic Press.

Paulesu, E., & Mehler, J. (1998a). Response from Paulesu and Mehler. *Trends in Cognitive Science, 2*, 471.

Paulesu, E., & Mehler, J. (1998b). Right on in sign language. *Nature, 392*, 233-234.

Pedelty, L.L. (1987). *Gesture in Aphasia.* University of Chicago, Chicago.

Penhune, V.B., Zatorre, R.J., & Evans, A.C. (1998). Cerebellar contributions to motor timing: A PET study of auditory and visual rhythm reproduction. *Journal of Cognitive Neuroscience, 10*(6), 752-765.

Periquet, M., Latouche, M., Lohmann, E., Rawal, N., De Michele, G., Ricard, S., Teive, H., Fraix, V., Vidailhet, M., Nicholl, D., Barone, P., Wood, N.W., Raskin, S., Deleuze, J.F., Agid, Y., Durr, A., & Brice, A. (2003). Parkin mutations are frequent in patients with isolated early-onset parkinsonism. *Brain, 126*(6), 1271-1279.

Petersen, S.E., Fox, P.T., Posner, M.I., & Mintun, M. (1989). Positron emission tomographic studies of the processing of single words. *Journal of Cognitive Neuroscience, 1*(2), 153-170.

Petitto, L.A. (1994). Are signed languages "real" languages? Evidence from American Sign Language and Langue des Signes Québécoise. *Signpost, 7*(3), 173-182.

Petitto, L.A., Zatorre, R.J., Gauna, K., Nikelski, E.J., Dostie, D., & Evans, A.C. (2000). Speech-like cerebral activity in profoundly deaf people processing signed languages: Implications for the neural basis of human language. *Proceedings of the National Academy of Sciences, 97*(25), 13961-13966.

Petitto, L.A., Holowka, S., Sergio, L.E., & Ostry, D. (2001). Language rhythms in baby hand movements. *Nature, 413*(6851), 35-36.

Pharr, V., Uttl, B., Stark, M., Litvan, I., Fantie, B., & Grafman, J. (2001). Comparison of apraxia in corticobasal degeneration and progressive supranuclear palsy. *Neurology, 56*(7), 957-963.

Pinto, S., Gentil, M., Fraix, V., Benabid, A.-L., & Pollak, P. (2003). Bilateral subthalamic stimulation effects on oral force control in Parkinson's disease. *Journal of Neurology, 250*, 179-187.

Poizner, H., Klima, E., & Bellugi, U. (1987). *What the Hands Reveal About the Brain.* Cambridge, MA: MIT Press.

Poizner, H., Bellugi, U., & Klima, E.S. (1989). Sign-language aphasia. In F. Boller & J. Grafman (Eds.), *Handbook of Neuropsychology* (Vol. 2, pp. 157-172). Amsterdam: Elsevier.

Poizner, H. (1990). Language and motor disorders in Deaf signers. In G.R. Hammond (Ed.), *Cerebral Control of Speech and Limb Movements* (pp. 303-326). North-Holland, NY: Elsevier.

Poizner, H., & Kegl, J. (1992). Neural basis of language and motor behaviour: Perspectives from American Sign Language. *Aphasiology, 6*(3), 219-256.

Poizner, H., & Kegl, J. (1993). Neural disorders of the linguistic use of space and movement. In P. Tallal, A. Galaburda, R. Llinas & C. von Euler (Eds.), *Annals of the New York Academy of Science, Temporal Information Processing in the Nervous System* (Vol. 682, pp. 192-213). New York: New York Academy of Sciences Press.

Poizner, H., Clark, M.A., Merians, A.S., Macauley, B., Rothi, L.J.G., & Heilman, K.M. (1995). Joint coordination deficits in limb apraxia. *Brain, 118*, 227-242.

Poizner, H., Merians, A.S., Clark, M.A., Rothi, L., & Heilman, K.M. (1997). Kinematic approaches to the study of apraxic disorders. In L.J.G. Rothi & K.M. Heilman (Eds.), *Apraxia: The Neuropsychology of Action* (pp. 93-110). Hove, UK: Psychology Press.

Poizner, H., Brentari, D., Tyrone, M.E., & Kegl, J. (2000). The structure of language as motor behavior: Clues from signers with Parkinson's disease. In K. Emmorey & H. Lane (Eds.), *The Signs of Language Revisited: An Anthology to Honor Ursula Bellugi and Edward Klima* (pp. 509-532). Mahwah, NJ: Lawrence Erlbaum.

Polymeropoulos, M., Higgins, J., Golbe, L., Johnson, W., Ide, S., DiIorio, G., Sanges, G., Stenroos, E., Pho, L., Schaffer, A., Lazzarini, A., Nussbaum, R., & Duvoisin, R. (1996). Mapping of a gene for Parkinson's disease to chromosome 4q21-q23. *Science, 274*, 1197-1199.

Polymeropoulos, M., Lavedan, C., Leroy, E., Ide, S., Dehejia, A., Dutra, A., Pike, B., Root, H., Rubenstein, J., Boyer, R., Stenroos, E., Chandrasekharappa, S., Athanassiadou, A., Papapetropoulos, T., Johnson, W., Lazzarini, A., Duvoisin, R., Di Iorio, G., Golbe, L., & Nussbaum, R. (1997). Mutation in the alpha-synuclein gene identified in families with Parkinson's disease. *Science, 276*(5321), 2045-2047.

Quaia, C., Lefevre, P., & Optican, L.M. (1999). Model of the control of saccades by superior colliculus and cerebellum. *Journal of Neurophysiology, 82*, 999-1018.

Raven, J., & Raven, J. (2003). Raven Progressive Matrices. In R.S. McCallum (Ed.), *Handbook of Nonverbal Assessment* (pp. 223-237). New York: Kluwer.

Raymer, A.M., Merians, A.S., Adair, J.C., Schwartz, R.L., Williamson, D.J., Rothi, L.J., Poizner, H., & Heilman, K.M. (1999). Crossed apraxia: implications for handedness. *Cortex, 35*(2), 183-199.

Riddoch, M.J., & Humphreys, G.W. (1993). *Birmingham Object Recognition Battery.* New York: Psychology Press.

Rijntjes, M., Dettmers, C., Buchel, C., Kiebel, S., Frackowiak, R.S.J., & Weiller, C. (1999). A blueprint for movement: Functional and anatomical representations in the human motor system. *Journal of Neuroscience, 19*(18), 8043-8048.

Rochat, P. (1992). Self-sitting and reaching in 5--8-month-old infants: the impact of posture and its development on early eye-hand coordination. *Journal of Motor Behavior, 24*(2), 210-220.

Rothi, L.J.G., Mack, L., Verfaellie, M., Brown, P., & Heilman, K.M. (1988). Ideomotor apraxia: error pattern analysis. *Aphasiology, 2*, 381-388.

Rothi, L.J.G., Ochipa, C., & Heilman, K.M. (1991). A cognitive neuropsychological model of limb praxis. *Cognitive Neuropsychology, 8*, 443-458.

Rothi, L.J.G., Ochipa, C., & Heilman, K.M. (1997). A cognitive neuropsychological model of limb praxis and apraxia. In L.P.G. Rothi & K.M. Heilman (Eds.), *Apraxia: The Neuropsychology of Action* (pp. 29-50). Hove, UK: Psychology Press.

Rushworth, M.F.S., Nixon, P.D., Wade, D.T., Renowden, S., & Passingham, R.E. (1998). The left hemisphere and the selection of learned actions. *Neuropsychologia, 36*, 11-24.

Sanabria, J., Ruiz, P.G., Gutierrez, R., Marquez, F., Escobar, P., Gentil, M., & Cenjor, C. (2001). The effect of levodopa on vocal function in Parkinson's disease. *Clinical Neuropharmacology, 24*(2), 99-102.

Sandler, W. (1993). A sonority cycle in American Sign Language. *Phonology, 10*, 243-279.

Sandler, W. (1999). Prosody in two natural language modalities. *Language and Speech, 42*(2&3), 127-142.

Schieber, M.H. (1996). Individuated finger movements: rejecting the labeled-line hypothesis. In A.M. Wing, P. Haggard & J.R. Flanagan (Eds.), *Hand and Brain: The Neurophysiology and Psychology of Hand Movements* (pp. 81-98). San Diego, CA: Academic Press.

Schultz, W. (1998). Predictive reward signal of dopamine neurons. *Journal of Neurophysiology, 80*, 1-27.

Schulz, G.M., & Grant, M.K. (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.

Sheard, C., Adams, R.D., & Davis, P.J. (1991). Reliability and agreement of ratings of ataxic dysarthric speech samples with varying intelligibility. *Journal of Speech and Hearing Research, 34*, 285-293.

Sieratzki, J.S., Calvert, G.A., Brammer, M.J., David, A.S., & Woll, B. (2001). Accessibility of spoken, written, and sign language in Landau-Kleffner syndrome: a linguistic and functional MRI study. *Epileptic Disorders, 3*(2), 79-90.

Sirigu, A., Daprati, E., Pradat-Diehl, P., Franck, N., & Jeannerod, M. (1999). Perception of self-generated movement following left parietal lesion. *Brain, 122*, 1867-1874.

Smith, C.D., Umberger, G.H., Manning, E.L., Slevin, J.T., Wekstein, D.R., Schmitt, F.A., Markesbery, W.R., Zhang, Z., Gerhardt, G.A., Kryscio, R.J., & Gash, D.M. (1999). Critical decline in fine motor hand movements in human aging. *Neurology, 53*(7), 1458-1461.

Soderfeldt, B., Ronnberg, J., & Risberg, J. (1994). Regional cerebral blood flow during sign language reception: Deaf and hearing subjects with deaf parents compared. *Sign Language Studies, 84*, 199-207.

Soderfeldt, B., Ingvar, M., Ronnberg, J., Eriksson, L., Serrander, M., & Stone-Elander, S. (1997). Signed and spoken language perception studied by positron emission tomography. *Neurology, 49*(1), 82-87.

Spanagel, R., & Weiss, F. (1999). The dopamine hypothesis of reward: past and current status. *Trends in Neurosciences, 22*, 521-527.

Square, P.A., Roy, E.A., & Martin, R.E. (1997). Apraxia of speech: Another form of praxis disruption. In L.J.G. Rothi & K.M. Heilman (Eds.), *Apraxia: The Neuropsychology of Action* (pp. 173-206). Hove, UK: Psychology Press.

Steinthal, P. (1871). *Abriss der Sprachwissenschaft.*Unpublished manuscript, Berlin.

Stephens, R.E., Addington, W.R., & Widdicombe, J.G. (2003). Effect of acute unilateral middle cerebral artery infarcts on voluntary cough and the laryngeal cough reflex. *American Journal of Physical and Medical Rehabilitation, 82*(5), 379-383.

Stokoe, W., Casterline, D., & Croneberg, C. (1965). *A Dictionary of American Sign Language on Linguistic Principles.* Washington, D.C.: Gallaudet University Press.

Stokoe, W.C. (1960). *Sign Language Structure: An Outline of the Visual Communication Systems of the American Deaf.* Silver Spring, MD: Linstok Press.

Strauss, M., & Klich, R.J. (2001). Word length effects on EMG/vowel duration relationships in apraxic speakers. *Folia Phoniatrica Logopaedica, 53*(1), 58-65.

Sunderland, A., & Sluman, S.-M. (2000). Ideomotor apraxia, visuomotor control and the explicit representation of posture. *Neuropsychologia, 38*(7), 923-934.

Sutton-Spence, R., Woll, B., & Allsop, L. (1990). Variation and recent change in fingerspelling in British Sign Language. *Language Variation and Change, 2*, 313-330.

Sutton-Spence, R. (1994). *The Role of the Manual Alphabet and Fingerspelling in British Sign Language.* Unpublished PhD Thesis, University of Bristol, Bristol.

Sutton-Spence, R. (1999). The influence of English on British Sign Language. *International Journal of Bilingualism, 3*(4), 363-394.

Testa, D., Monza, D., Ferrarini, M., Soliveri, P., Girotti, F., & Filippini, G. (2001). Comparison of natural histories of progressive supranuclear palsy and multiple system atrophy. *Neurological Sciences, 22*(3), 247-251.

Thach, W.T., Goodkin, H.P., & Keating, J.G. (1991). Inferior olive disease in man prevents learning of novel eye-hand synergies. *Abstracts of the Society for Neuroscience, 17*, 1380.

Thach, W.T., Goodkin, H.P., & Keating, J.G. (1992). The cerebellum and the adaptive coordination of movement. *Annual Review of Neuroscience, 15*, 403-442.

Theodoros, D.G., & Murdoch, B.E. (1998a). Hyperkinetic dysarthria. In B.E. Murdoch (Ed.), *Dysarthria: A Physiological Approach to Assessment and Treatment* (pp. 314-336). Cheltenham, UK: Stanley Thornes.

Theodoros, D.G., & Murdoch, B.E. (1998b). Hypokinetic dysarthria. In B.E. Murdoch (Ed.), *Dysarthria: A Physiological Approach to Assessment and Treatment* (pp. 266-313). Cheltenham, UK: Stanley Thornes.

Thompson, E.C., & Murdoch, B.E. (1995). Disorders of nasality in subjects with upper motor neuron type dysarthria following cerebrovascular accident. *Journal of Communication Disorders, 28*, 261-276.

Thompson-Ward, E.C. (1998). Spastic dysarthria. In B.E. Murdoch (Ed.), *Dysarthria: A Physiological Approach to Assessment and Treatment* (pp. 205-241). Cheltenham, UK: Stanley Thornes.

Timmann, D., Citron, R., Watts, S., & Hore, J. (2001). Increased variability in finger position occurs throughout overarm throws made by cerebellar and unskilled subjects. *Journal of Neurophysiology, 86*, 2690-2702.

Timmann, D., Drepper, J., Maschke, M., Kolb, F.P., Boring, D., Thilmann, A.F., & Diener, H.C. (2002). Motor deficits cannot explain impaired cognitive associative learning in cerebellar patients. *Neuropsychologia, 40*(7), 788-800.

Tracy, J.I., Faro, S.S., Mohammed, F.B., Pinus, A.B., Madi, S.M., & Laskas, J.W. (2001). Cerebellar mediation of the complexity of bimanual compared to unimanual movements. *Neurology, 57*(10), 1862-1869.

Trouillasa, P., Takayanagi, T., Hallett, M., Currier, R.D., Subramony, S.H., Wessel, K., Bryer, A., Diener, H.C., Massaquoi, S., Gomez, C.M., Coutinho, P., Ben Hamida, M., Campanella, G., Filla, A., Schut, L., Timmann, D., Honnorat, J., Nighoghossian, N., & Manyam, B. (1997). International Cooperative Ataxia Rating Scale for pharmacological assessment of the cerebellar syndrome. *Journal of the Neurological Sciences, 145*(2), 205-211.

Tyrone, M.E., Kegl, J., & Poizner, H. (1999). Interarticulator co-ordination in Deaf signers with Parkinson's disease. *Neuropsychologia, 37*, 1271-1283.

UK-Council-on-Deafness. (2003). *Government Statement - 18th March 2003*. Retrieved 23 July 2004, 2004

Ullen, F., Forssberg, H., & Ehrsson, H.H. (2003). Neural networks for the coordination of the hands in time. *Journal of Neurophysiology, 89*(2), 1126-1135.

Urban, P.P., Marx, J., Hunsche, S., Gawehn, J., Vucurevic, G., Wicht, S., Massinger, C., Stoeter, P., & Hopf, H.C. (2003). Cerebellar speech representation: Lesion topography in dysarthria as derived from cerebellar ischemia and functional magnetic resonance imaging. *Archives of Neurology, 60*, 965-972.

VanAlphen, A.M., Schepers, T., Luo, C., & DeZeeuw, C.I. (2002). Motor performance and motor learning in Lurcher mice. *Annals of the New York Academy of Sciences, 978*, 413-424.

VanGemmert, A.W.A., Teulings, H.-L., Contreras-Vidal, J.L., & Stelmach, G.E. (1999). Parkinson's disease and the control of size and speed in handwriting. *Neuropsychologia, 37*, 685-694.

Waelti, P., Dickinson, A., & Schultz, W. (2001). Dopamine responses comply with basic assumptions of formal learning theory. *Nature, 412*, 43-48.

Wechsler, D. (1997). *Wechsler Adult Intelligence Scale - III*. New York: Psychological Corporation.

Weismer, G. (1984). Articulatory characteristics of parkinsonian dysarthria: segmental and phrase-level timing, spirantization, and glottal-supraglottal coordination. In M. McNeil, J. Rosenbek & A. Aronson (Eds.), *The Dysarthrias: Physiology, Acoustics, Perception, Management* (pp. 101-130). San Diego: College Hill Press.

Weismer, G., Jeng, J.Y., Laures, J.S., Kent, R.D., & Kent, J.F. (2001). Acoustic and intelligibility characteristics of sentence production in neurogenic speech disorders. *Folia Phoniatrica Logopaedica, 51*(1), 1-18.

Weiss, P., Stelmach, G.E., & Hefter, H. (1997). Programming of a movement sequence in Parkinson's disease. *Brain, 120*, 91-102.

Weiss, P., Jeannerod, M., Paulignan, Y., & Freund, H.-J. (2000). Is the organisation of goal-directed action modality specific? A common temporal structure. *Neuropsychologia, 38*(1136-1147).

Welsh, J.P., Lang, E.J., Suglhara, I., & Llinas, R. (1995). Dynamic organization of motor control within the olivocerebellar system. *Nature, 374*, 453-457.

Wertz, R.T., LaPointe, L.L., & Rosenbek, J.C. (1998). *Apraxia of Speech in Adults: The Disorder and its Management*. San Diego: Singular.

Wiesendanger, M., Kaluzny, P., Kazennikov, O., Palmeri, A., & Perrig, S. (1994). Temporal coordination in bimanual actions. *Canadian Journal of Physiology and Pharmacology, 72*, 591-594.

Wiesendanger, M., Kazennikov, O., Perrig, S., & Kaluzny, P. (1996). Two hands- one action: The problem of bimanual coordination. In A.M. Wing, P. Haggard & J.R. Flanagan (Eds.), *Hand and Brain: The Neurophysiology and Psychology of Hand Movements* (pp. 283-300). San Diego: Academic Press.

Wilcox, S. (1992). *The Phonetics of Fingerspelling*. Amsterdam: John Benjamins.

Wing, A.M., & Miller, E. (1984). Basal ganglia lesions and psychological analyses of the control of voluntary movement. *Ciba Foundation Symposium, 107*, 242-257.

Wing, A.M., Haggard, P., & Flanagan, J.R. (1996). *Hand and Brain: The Neurophysiology and Psychology of Hand Movements*. San Diego: Academic Press.

Wise, R.J.S., Greene, J., Buchel, C., & Scott, S.K. (1999). Brain regions involved in articulation. *Lancet, 353*, 1057-1061.

Wohlert, A.B., & Smith, A. (2002). Developmental change in variability of lip muscle activity during speech. *Journal of Speech Language and Hearing Research, 45*(6), 1077-1087.

Woll, B., & Grove, N. (1996). On language deficits and modality in children with Down Syndrome: a case study of twins bilingual in BSL and English. *Journal of Deaf Studies and Deaf Education, 1*(4), 271-278.

Wolpert, D.M., Miall, R.C., & Kawato, M. (1998). Internal models in the cerebellum. *Trends in Cognitive Sciences, 2*, 338-347.

Yaguez, L., Canavan, A.G.M., Lange, H.W., & Homberg, V. (1999). Motor learning by imagery is differentially affected in Parkinson's and Huntington's diseases. *Behavioural Brain Research, 102*, 115-127.

Zalla, T., Sirigu, A., Pillon, B., Dubois, B., Grafman, J., & Agid, Y. (1998). Deficit in evaluating pre-determined sequences of script events in patients with Parkinson's disease. *Cortex, 34*(4), 621-627.

Zeplin, J., & Kent, R.D. (1996). Reliability of auditory-perceptual scaling of dysarthria. In D.A. Robin, K.M. Yorkston & D.R. Beukelman (Eds.), *Disorders of Motor Speech: Assessment, Treatment, and Clinical Characterization* (pp. 145-154). Baltimore: Brookes.

Zhou, Y. (1980). The Chinese finger alphabet and the Chinese finger syllabary. *Sign Language Studies, 9*, 209-216.

Zia, S., Cody, F., & O'Boyle, D. (2000). Joint position sense is impaired by Parkinson's disease. *Annals of Neurology, 47*, 218-228.

Ziegler, W., & Wessel, K. (1996). Speech timing in ataxic disorders: Sentence production and rapid repetitive articulation. *Neurology, 47*, 208-214.

Ziegler, W. (2002a). Psycholinguistic and motor theories of apraxia of speech. In N. Helm-Estabrooks & N. Bernstein Ratner (Eds.), *Apraxia of Speech: From Concept to Clinic* (Vol. 23, pp. 231-244). New York: Thieme Medical Publishers.

Ziegler, W. (2002b). Task-related factors in oral motor control: Speech and oral diadochokinesis in dysarthria and apraxia of speech. *Brain and Language, 80*, 556-575.