Simultaneity in Signed Languages
Form and function
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Simultaneity in atypical signers

Implications for the structure of signed language

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1. Introduction

Because signed languages employ the complex, coordinated movements of multiple articulators, and those articulators can be controlled independently of one another, it is possible for signers to produce separate streams of information simultaneously. The independent control of sign articulators also allows the breakdown of independent articulatory movements in atypical signers. For instance, following damage to one cerebral hemisphere, a signer may experience weakness or loss of movement on one side of the body. As a result, the simultaneity of the movements of the two limbs by typical signers may be disrupted or eliminated in atypical signers, and this disruption of articulatory simultaneity in turn may disrupt the information carried in the linguistic signal.

This paper will explore how simultaneity in signed language is lost or preserved in different groups of atypical signers. No study to date has focused on the breakdown of the types of linguistic (e.g. lexical, syntactic, semantic) simultaneity and linguistic-gestural simultaneity emphasized in this volume, so this paper will review what is known about the breakdown of articulatory simultaneity and suggest how linguistic simultaneity is likely to be affected by particular neural pathologies. The first two sections will examine the physical structure and neural basis of signed language, because these are possible loci of disruption in atypical signing. The following section will discuss specific neural pathologies and how they are likely to impact simultaneity, and the final section will discuss what disruptions to simultaneity can reveal about the structure of signed language.

Modality differences inherent to the physical production mechanism

Speech is produced via the vocal tract, whereas sign uses the hands and arms as its primary articulators. (Secondary sign articulators include the shoulders, trunk, head, mouth, eyes, and eyebrows.) The details of the primary articulators greatly influence the structure of sign and speech in a variety of ways (Klima & Bellugi 1979; Meier 2002). First of all, the primary articulators for sign production are paired: each articulator has a corresponding mirror-image articulator on the opposite side of the body. By contrast, the speech articulators are mostly single organs located along the midline of the body. Additionally, the sign articulators are spread across different parts of the body (e.g. the face and the hands), in contrast to the speech articulators which form a contiguous region in the vocal tract. As a result, the sign articulators can move largely independently of each other; whereas the movement of one speech organ tends to influence or constrain the movements of other speech organs.

The sign articulators consist of groups of large muscles organized around bones, whereas the speech articulators are not arranged in these types of agonist/antagonist configurations. In order for the sign articulators to move, one set of muscles has to relax while the opposing set contracts. Additionally, sign articulators move large distances and have many degrees of freedom: they move easily in all three spatial dimensions and can take an infinite number of paths in moving from one location in space to another. The result of these factors is that movements for sign production are larger and slower than movements for speech (Bellugi & Fischer 1972; Klima & Bellugi 1979). Bellugi & Fischer (1972) measured the durations of individual signs in American Sign Language (ASL) and the durations of individual spoken English words, both produced by hearing speakers from Deaf households. In that study, subjects produced between 4 and 5.2 English words per second, and between 2.3 and 2.5 ASL signs per second.

Speech can be described as a source-filter mechanism with an energy source (the vibrating vocal folds) which generates pulses that are modified by the filter that they pass through (the supralaryngeal vocal tract). By contrast, sign does not have an apparent energy source independent of the excitation of the muscles themselves. In addition, sign is not obviously dependent on or structured by respiration patterns, which could mean that the limitations on utterance length or articulation/pause ratio will be different for the two modalities.

The sign production mechanism is configured such that the articulators that can perform rapid, precise movements (i.e. the hands) are attached at the ends of articulators that make only gross movements (i.e. the arms). The result of this is that signs are composed of precise movements superimposed on the gross movements of the same limb. The two sets of movements (gross and precise) are coordinated to overlap in time. When a typical signer produces a sign with an

internal handshape change and a path movement of the arm, for example, the movement of the hand begins after arm movement has begun, and the two types of movement end at the same time (Brentari, Poizner & Kegl 1995). There is no equivalent coupling of gross and precise movements for the speech production mechanism.

3. Brain, motor control, and language

Little is known about brain function for sign articulation; however, there has been a great deal of research on motor control for the limbs and how it differs from motor control for the vocal tract. So it is possible to extrapolate slightly to discuss how brain function is likely to be different for sign vs. speech, given their primary articulators.

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. At the highest level of the nervous system, there are two cerebral hemispheres on the left and right sides of the brain. The outermost layers of cells in the two hemispheres are known as the cerebral cortex. Motor areas of the cerebral cortex are divided somatotopically, i.e. specific cortical motor areas control movement for specific parts of the body. Cortical areas that control movement are located in the medial and posterior frontal lobe. The primary motor cortex is the main source of movement commands to peripheral muscles; and the supplementary motor area and premotor area, which are just anterior to the primary motor cortex, are activated prior to movement onset and serve a role in movement planning.

The fibres that descend from the cortex can be subdivided into the corticospinal and corticobulbar tracts. The corticospinal tract descends through the cerebral hemispheres and brainstem, and ultimately terminates in the spinal cord. Along the way, in the lower part of the brainstem, the majority of corticospinal fibres cross (or decussate) to the contralateral side of the brainstem (see Figure 1). Consequently, 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 motor nerves in the spinal cord, it projects to 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 tract in almost equal proportions.

There are also structures outside the cerebral neocortex that are involved in the control of voluntary movement. The two largest and most important are the cere-

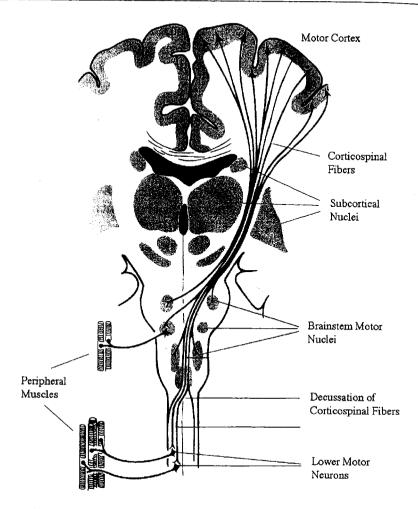


Figure 1. Motor pathways from the primary motor cortex to the muscles (adapted from Brodal 1998)

bellum and basal ganglia. There are no common pathologies associated with the cerebellum in humans, whereas the basal ganglia are affected by both Parkinson's disease and Huntington's disease. Neither the cerebellum nor the basal ganglia are directly responsible for movement generation, but rather for shaping voluntary movement so that it is accurate, natural, well-timed and coordinated (Jueptner, Jenkins, Brooks, Frackowiak & Passingham 1996; Lang & Bastian 2002; Timmann, Citron, Watts & Hore 2001; VanGemmert, Teulings, Contreras-Vidal & Stelmach 1999). Additionally, both structures play a role in modifying muscle tone, balance, and posture, probably through projections to brainstem nuclei. Both the

cerebellum and the basal ganglia receive input from and project back to cortical motor areas.

From this it seems likely that neural damage to the basal ganglia or cerebellum would have the same effect on articulation for sign as for articulation for speech. On the other hand, damage to motor areas of the cortex or to the corticospinal/corticobulbar tracts may have different effects for the two modalities. This follows from the fact that the cerebellum and basal ganglia do not have effector-specific functions, so they tend to affect movements of different parts of the body in the same way. By contrast, specific cortical motor areas can control specific parts of the body. Moreover, damage to motor areas in one hemisphere often has a dramatic effect on the contralateral limb, but less effect on the ipsilateral limb, and also less effect on the vocal tract.

In general, the brain structures most relevant to language are located in the left cerebral cortex. The two language areas identified earliest were Broca's area (left inferior frontal lobe) and Wernicke's area (left posterior temporal lobe) in the posterior and anterior left hemisphere. More systematic analysis of patients' language abilities following stroke later showed that some right hemisphere structures are important for language as well, particularly at the level of discourse. Since new technologies have allowed more precise localization of brain areas relevant to specific cognitive functions, it has become clear that additional cortical areas in the left and right hemispheres are recruited during language production and processing (Stowe, Haverkort & Zwarts 2005). A discussion of particular cortical areas and their role in language is beyond the scope of this paper, but a short list of those areas includes the left inferior frontal gyrus, superior temporal gyrus, posterior temporal lobe, and supplementary motor area, and the left and right supramarginal gyrus and angular gyrus. (For a review of research on the neural basis of language, see Stowe et al. 2005.)

Other studies have suggested that non-cortical areas are also important to language function. In particular, it has been suggested that the basal ganglia play a role in syntax (Arnott, Chenery, Murdoch & Silburn 2005; Lieberman, Friedman & Feldman 1990); however, the evidence for this is somewhat equivocal, and there is no consistent pattern of syntactic deficits in subjects with damage to those areas (Murray & Lenz 2001; Patterson & Bly 1999). In addition, various studies have suggested that parts of the cerebellum are activated during language processing (Fiez, Raife, Balota, Schwarz, Raichle & Petersen 1996), and that damage to the right cerebellum can cause aphasia (Fabbro, Moretti & Bava 2000; Marien, Engelborghs, Pickut & DeDeyn 2000). Anatomically, this makes sense because there are strong connections between the right cerebellum and the left frontal lobe, where many important language areas are located. Because of their importance both to motor control and to other cognitive functions, the role of the cerebellum or the basal ganglia in language as opposed to articulation remains largely unclear.

Signed language and the brain

On the basis of extensive research, it seems clear that neural damage results in the same linguistic deficits in sign and in speech. Several studies have examined ASL signers with various types of brain damage (Corina, Vaid & Bellugi 1992b; Poizner, Klima & Bellugi 1987), in order to determine how the brain processes sign in comparison to speech, given that the two systems 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 issues addressed by studies of signers with brain damage include: the function of traditional language areas and visuospatial cognition areas in processing spatialized grammar; and the relationship between sign aphasia and limb apraxia.

Poizner et al. (1987) studied four Deaf subjects who had either left or right hemisphere damage, in order to determine whether language deficits following stroke would pattern similarly in signed and spoken language. They found a double dissociation in the language and visuospatial abilities of signers with left hemisphere and right hemisphere damage. The signers with left hemisphere damage retained their ability to perceive spatial relations of objects but lost their use of space for signed language grammar; whereas signers with right hemisphere damage lost their ability to perceive spatial relations but retained their ability to use space grammatically.

In addition, Poizner et al. (1987) as well as Corina, Poizner, Bellugi, Feinberg & O'Grady-Batch (1992a) found a dissociation between disruption to language (aphasia) and disruption to symbolic gesture (apraxia). Damage to left anterior cortical areas often causes both of these disorders to occur, and it had been claimed that aphasia and apraxia were the same phenomenon in signed language (Kimura 1981). However, there have been reported cases of Deaf subjects who could understand gestures but not signs (Corina et al. 1992a; Marshall, Atkinson, Smulovitch, Thacker & Woll 2004), indicating that apraxia and aphasia are separate phenomena irrespective of language modality.

Most research on signed language and the brain has focused on signed language as a linguistic or cognitive task; however, a few studies have sought to understand sign 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, Kegl & Poizner 1997; Poizner 1990; Poizner & Kegl 1993), and a more recent study has examined sign production across a broad range of movement disorders (Tyrone 2005).

A few brain imaging studies of healthy Deaf signers have explored questions related to movement or articulation in signing and fingerspelling. One study explicitly designed to investigate articulation explored a basic but neglected issue: whether left-handed signing activates left hemisphere frontal structures and other structures relevant for motor control for speech articulation (Corina, San Jose-Robertson, Guillemin, High & Braun 2003). In principle, given that motor cortical areas in the right hemisphere control movements of the left arm, one might hypothesize that left-handed signing would primarily activate the right hemisphere. In fact, Corina et al. (2003) 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 which suggested that the anterior left hemisphere plays an important role for speech articulation (Dronkers 1996; Wise, Greene, Buchel & Scott 1999). 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.

Simultaneity and specific neural pathologies

Typically, brain injury or stroke affects some portion of one of the cerebral hemispheres, but not both hemispheres. Consequently, injury or stroke may cause weakness or paralysis on one side of the body (i.e. hemiplegia or hemiparesis). When hemiplegia or hemiparesis occurs, the normal simultaneity of limb movements during signing can be disrupted, thereby altering two-handed signs or precluding simultaneous production of two different signs by the two hands. In a recent study of British Deaf signers with movement disorders, one subject with right hemisphere damage had a disruption to simultaneous movements of the two limbs during signing, but simultaneous bimanual movements were not eliminated from his signing altogether. In particular, he produced two-handed signs, but lowered his left hand during signing, while his right hand was not lowered.

In the same study, a subject with left hemisphere damage, whose signing was severely disrupted, produced only one-handed signs (Marshall, Atkinson, Woll & Thacker 2005; Tyrone 2005). While she could copy some signs, when asked to copy a two-handed sign, she would shrug and gesture to her left arm, indicating that she could not produce the sign since she could not move both arms. Deaf subjects with unilateral brain damage whose signing was similarly disrupted by hemiparesis have been reported elsewhere (Atkinson, Campbell, Marshall, Thacker & Woll 2004; Kegl & Poizner 1997; Poizner et al. 1987).

5.1 Left hemisphere damage

Either left or right hemisphere damage can cause hemiparesis and disrupt twohanded signing, as discussed above. Left hemisphere damage can additionally cause aphasia, which is the breakdown of language function. This is so because the brain areas that are most important for both spoken and signed language are in the left hemisphere. The use of space to show grammatical agreement in signed languages causes the verb and its subject or object to be presented in the same lexical item in many cases. Signed language also uses facial expression concurrently with movements of the hands to show grammatical and prosodic information. Because signed languages allow grammatical information to be produced simultaneously with lexical information, if there is a breakdown of grammar, then an aspect of sign simultaneity may be lost as a result. Interestingly, some studies have reported the breakdown of grammatical but not affective facial expression in cases of aphasia (Kegl & Poizner 1997). In other words, signers who have aphasia have exhibited a disruption in manual-facial simultaneity purely resulting from their language deficit, and not from any difficulty coordinating limb movements with facial movements. Additionally, Kegl & Poizner (1997) reported an aphasic subject who preferred to use word order rather than grammatical agreement - which in effect reduces the simultaneous production of lexical and syntactic information.

5.2 Right hemisphere damage

As discussed above, the brain structures most important to language are located in the left hemisphere. As a result, right hemisphere damage does not usually cause aphasia, but it can disrupt prosody, affective facial expression, and pragmatic components of language. Loew et al. (1997) described a signer with right hemisphere damage who experienced a disruption to role-shift in particular. (Role shift is the use of body orientation and gaze to differentiate roles in a narrative.) He had difficulty assuming the perspective of the agents in his discourse, so he continually referred to them in the third person, as points in the signing space. In his case, it was not syntactic simultaneity that was disrupted, but the ability to present multiple viewpoints in his signing.

A recent study in the UK identified a signer with right hemisphere damage who experienced what may have been a deficit in language prosody. In particular, he had an impairment in processing information on the face during signing. On tests of sign perception, he showed difficulty identifying negation when it was presented on the face as eyebrow wrinkling (Atkinson et al. 2004). So in addition to his production deficits, this signer had a deficit in perceiving simultaneous streams of information in signed language, if one stream of information was presented via facial expression. His case is an interesting contrast to the signers with aphasia,

because he exhibited a particular deficit in manual-facial simultaneity irrespective of the nature of the facial information.

There are also sign articulation deficits which result from right hemisphere damage, but these tend to be quite varied. Poizner & Kegl (1993) and Loew et al. (1997) described the effects of right hemisphere damage on one individual's signing. When the subject produced two handed signs, movement initiation was delayed in the left hand relative to the right hand, which Poizner & Kegl (1993) analyzed as a deficit in motor neglect. Given that his simultaneous bimanual movements within signs were so severely disrupted, it is unlikely that he would use many two-handed simultaneous constructions. Poizner & Kegl (1993) do not report their being used at all.

The articulatory deficits of a Deaf signer with right hemisphere damage in the UK ('James') were described in Tyrone (2005). Like the subject reported by Poizner & Kegl (1993), James had incoordination of the two hands during sign production and tended to delay the movements of his weak arm at the onset of two-handed signs. However, his incoordination was not very pronounced. Additionally, he had no difficulties using his affected hand as a base hand in two-handed signs, despite his left-side hemiparesis. This study did not explicitly examine the use of the nondominant hand as a buoy or a placeholder, but based on broader patterns in his sign production, it seems likely that James would be able to produce these and other simultaneous constructions with no difficulty.

5.3 Parkinson's disease

Research on signers with Parkinson's disease suggests that their language deficits are articulatory rather than linguistic in nature (Brentari & Poizner 1994; Poizner 1990). Broadly speaking, signers with Parkinson's disease (PD) tend to hypoarticulate: their sign production is smaller, slower, and prosodically reduced. In particular, signers with PD have reduced and lowered signing space (Loew, Kegl & Poizner et al. 1995; Poizner & Kegl 1992; Poizner & Kegl 1993). Relative to neurologically-intact control subjects, signers with PD 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.

Signers with Parkinson's disease produce signs with more distal articulators; for example, in 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, Kegl & Poizner 1999). Additionally, signers with PD often lax the distal articulators of the hands and wrist during sign production, so that the handshape and orientation of signs are less articulatorily contrastive than they would be normally (Brentari & Poizner 1994; Brentari et al. 1995; Loew et al. 1995).

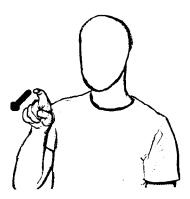


Figure 2. ASL sign ASK

Several studies indicate that signers with PD show a disruption in the simultaneous movement of multiple articulators, including articulators on the same limb (e.g., the fingers and elbow) (Brentari & Poizner 1994; Brentari et al. 1995; Poizner & Kegl 1993; Tyrone et al. 1999). In some cases, signers with PD delete handshape or orientation change from a sign or a fingerspelled word (Brentari & Poizner 1994; Tyrone et al. 1999). In other cases, signers with PD may completely synchronize or completely serialize handshape change and arm movement, rather than produce them in a partially overlapping manner, as control signers do (Brentari et al. 1995). For example, the ASL sign ASK requires a forward movement of the arm while the index finger flexes (see Figure 2). Based on research on ASL, this type of sign seems to be particularly difficult for signers with Parkinson's disease. Interestingly, given that ASL signers with Parkinson's disease sometimes serialize, sometimes synchronize, and sometimes delete distal movements, the normal interarticulator simultaneity in signs can be either increased or decreased, depending on which tendency an individual subject exhibits.

Another atypical signing pattern found in signers with Parkinson's disease was handshape mirroring on the non-active hand in one-handed signs or fingerspelled words (Loew et al. 1995; Poizner, Brentari, Tyrone & Kegl 2000; Tyrone et al. 1999), which effectively creates a type of simultaneity not present in typical signing. For example, in producing the ASL sign BIRD, which is a one-handed sign, a signer with Parkinson's disease produced the sign's handshape and hand-internal movement on both his left hand and his right (Poizner et al. 2000). Signers with Parkinson's disease were not reported to produce fully-articulated two-handed signs in place of one-handed signs. Rather, they produced the movements of the active hand in a reduced form on the non-active hand.

Finally, signers with Parkinson's disease showed a reduction in their use of facial expression (Kegl, Cohen & Poizner 1999). As a result of this, they exhibited less manual-facial simultaneity, for both grammatical and prosodic facial expression.

Unlike signers with aphasia, their facial movement deficit was related to motor control rather than language, and hence it was present for non-linguistic as well as linguistic facial expression.

A recent study of a Deaf man with Parkinson's disease in the UK had somewhat different results from earlier studies in the US (Tyrone & Woll in press). Specifically, the subject in the UK study, 'John', had no more coordination deficits than the control subject, and thus no particular deficit in articulatory simultaneity. In fact, John's signing was relatively similar to that of a typical signer. Static components of his signs were often laxed, which is common during relaxed, informal signing. John had no difficulty with coordination of the two limbs or of proximal and distal articulators on the same limb. In addition, his facial expressions were somewhat reduced, but he was still able to produce the correct oral and facial movements simultaneously with their lexical correlates on the hands. Further study would be needed to determine what differentiates John from other signers with Parkinson's disease whose signing is more severely disrupted.

5.4 Cerebellar damage

While it is clear that the cerebellum serves an important role in motor control, there is still debate as to what exactly it does. In general terms, cerebellar damage causes intention tremor, impairments in movement scaling, and coordination deficits. There has been little research specifically on signed language and the cerebellum, because cerebellar damage is comparatively rare in humans. To date, only one signer with cerebellar damage has been identified and studied (Tyrone 2005).

The subject in that study, 'Robert', experienced severe damage to the right cerebellum following hemorrhaging during an operation to correct an arteriovenous malformation. Broadly speaking, Robert's signing was oversized and uncoordinated. Additionally, his signing, like his limb movement more generally, was slow and often disrupted by intention tremor. In contrast to signers with Parkinson's disease, Robert's movements were proximalized on some signs; in other words, he would produce a sign using articulators proximal to those normally used for its production (e.g. in producing the BSL sign HAMMER, which normally has a repeated downward movement of the wrist, he might instead produce the downward movement from the elbow). In addition, he had an overall pattern of enlarged signing: large movements, distant sign locations, and hyperextended articulators. In a number of ways, Robert's signing was the opposite of what was reported in signers with Parkinson's disease; his signs were large, proximalized, and sometimes included movements which were not required.

Robert's simultaneity difficulties took a variety of forms. In particular, he had difficulty making simultaneous movements of his hands to produce two-handed signs. He was not always able to make his hands begin moving at the same time,

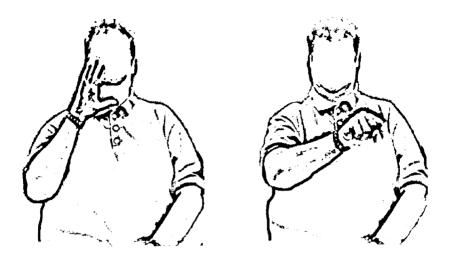


Figure 3. BSL sign BITE

come to the same place, or produce the same movement. His bimanual coordination was so severely disrupted that he did not attempt to produce buoys or place holders, or to produce separate signs simultaneously with the two hands. Because Robert could not suppress involuntary movements during signing, his hands sometimes appeared to be producing different signs even though he did not intend them to be doing so.

Robert also had difficulty with simultaneous movements of the independent articulators on a single limb: both correctly timing separate movements relative to each other and suppressing involuntary movements that emerged during voluntary movement. For example, the BSL sign BITE requires a downward movement of the arm at the same time that the fingers close to make a fist (see Figure 3). What Robert would do instead was to produce the two movements serially, by moving his arm down first, then pausing, then closing his fingers.

Robert had a tendency to produce one-handed signs with two hands without obvious intent to place linguistic emphasis. Unlike signers with Parkinson's disease, Robert would produce a fully two-handed form of one-handed signs, adding a component of simultaneity to signs that would not normally be present. The production of two-handed forms of one-handed signs has also been documented in normal Deaf children in the early stages of acquiring signed language (Cheek, Cormier, Repp & Meier 2001). For example, one child from that study produced a two-handed version of the ASL sign DOG, which is one-handed in its citation form. It may be that the children studied by Cheek et al. (2001) were relying on descending brainstem tracts which enable simple, symmetrical, two-handed movements (Wiesendanger, Kazennikov, Perrig & Kaluzny 1996). The children in that study ranged in age from 5 to 17 months, at which stage the corticospinal tract

and the connection between the cerebellum and cerebral cortex, both of which are necesary for precise, coordinated movements, would not yet be fully developed (Brodal 1998). This could explain the similarity between the children's signing and Robert's signing.

5.5 Progressive supranuclear palsy

Progressive supranuclear palsy is a disease that is similar to Parkinson's disease in pathology and symptomatology, but occurs less frequently. Like Parkinson's disease, progressive supranuclear palsy causes neuronal loss in the brainstem, affecting projections to the basal ganglia and causing movements to be slow and small(or hypokinetic). In addition, progressive supranuclear palsy causes atrophy in the frontal lobes and damages projections from the brainstem to the cerebellum (Cordato, Duggins, Halliday, Morris & Pantelis 2005). One of the characteristic symptoms of the disease, which is used to distinguish it from Parkinson's disease, is the loss of eye movements, or ophthalmoplegia. A recent study on movement disorders and signed language included a British signer with progressive supranuclear palsy, 'Joseph' had severely disrupted facial expression and limited use of eye gaze, which affected both prosodic and grammatical information carried on the face during signing. However, he had no difficulty understanding grammatical or affective facial expressions used by other signers (Tyrone 2005). As a result of his disease symptoms, both his manual-facial and manual-oral simultaneity were drastically reduced in all forms.

Broadly speaking, Joseph's signing was reduced in size and speed, and was characterized by laxed articulation. In this way, his deficits were similar to those reported in signers with Parkinson's disease (Brentari & Poizner 1994; Brentari et al. 1995). In addition, Joseph had great difficulty coordinating multiple sets of articulators during signing. Consequently, he had a reduction in simultaneous movements of the two arms, as well as in the simultaneous movements of multiple articulators on the same arm. However, 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 sign and Parkinson's disease (Brentari et al. 1995; Tyrone et al. 1999), and consistent with models of hand configurations and finger movements (Iberall & Fagg 1996; Schieber 1996). Despite the fact that the fingers can move independently, during sign production in both typical and atypical signers, the fingers seem to move as a unit.

Implications for the structure of signed language

Research on atypical signers consistently suggests that neurological disorders do not disrupt sign and speech substantially differently, whether the deficits being examined are linguistic or articulatory in nature (Tyrone 2005). For users of either spoken language or signed language, for example, left hemisphere damage causes aphasia, right hemisphere damage causes disrupted prosody, and Parkinson's disease causes disrupted articulation, despite the fact that the two modalities use different sets of articulators with different anatomical and physiological properties. This paper has outlined a variety of neural pathologies that can all disrupt simultaneity in signed language; however, each of these pathologies disrupts simultaneity differently, depending on the underlying nature of the sign articulation deficit.

Simultaneity is affected in a variety of ways in atypical signers with aphasia or with movement disorders. Moreover, neither of these categories of disorders affects sign simultaneity in a uniform way. Some aspects of simultaneity do not seem to be inherently linguistic in nature; instead, they provide redundant information in the communication stream which may facilitate language perception or production, although they are not crucial to the linguistic signal. By contrast, there are aspects of simultaneity that are fundamentally linguistic in nature, as evident from the fact that they are preserved in individuals with articulatory disorders but not in those with aphasia. The fact that simultaneity can be either a linguistic or an articulatory phenomenon in signed language suggests that more research should focus specifically on the nature of sign articulation, so that the aspects of the language that result from motor control can be teased apart from those that are linguistic and arbitrary in nature. The idea that sign is by nature more simultaneous and less sequential than speech should be probed further to explore the extent to which this modality difference (assuming it is real) is purely articulatory.

Investigations of atypical language can provide useful insight into language structure, as illustrated by research on atypical signing. For instance, studies in the UK and in the US suggest that particular sets of sign articulators (e.g. the fingers) tend to move simultaneously, while other sets of articulators (e.g. the elbow and fingers) move in a coordinated, overlapping fashion, but slightly asynchronously (Brentari et al. 1995; Tyrone 2005; Tyrone et al. 1999). These findings support the idea that handshape, orientation, movement, and location are the formational primitives of signed language production (Battison, Markowicz & Woodward 1975; Stokoe 1960). Given that the fingers move simultaneously, they might form a single production unit at the level of motor planning. Conversely, since the relative movements of the elbow, wrist, and fingers are slightly asynchronous over the course of a sign's production, they can be modeled as three different production units, namely, movement, orientation, and handshape, respectively.

There is a form of signed language which has not been included here as a type of atypical signing, but it provides an interesting contrast to the forms of atypical signing that have been discussed. Tactile signing is used by people who are both Deaf and blind, and requires signers' hands to remain in contact as signs are produced. As with visually-perceived signing, the physical form of tactile signing can influence its structure in numerous ways. Because Deaf-blind signers must maintain physical contact with an interlocutor during signing, their use of space and of nonmanual articulators is more constrained, which may limit the occurrence of simultaneity in sign production. As a result, Deaf-blind signers develop alternative means of expressing the grammatical information that is usually conveyed by use of space or nonmanual articulators for sighted Deaf signers. For instance, a Deaf-blind signer might use fingerspelling in place of referential pointing (Quinto-Pozos 2002). Similarly, Deaf-blind signers have been reported to use lexical signs rather than facial expressions to mark questions (Collins & Petronio 1998). By contrast, atypical signers whose use of grammatical facial expression is disrupted do not seem to compensate by adding lexical signs for questions. These reported differences between atypical and tactile signing suggest that the former is a disruption to normal sign production which may or may not cause the deletion of simultaneous grammatical information, whereas the latter is an established system which provides a means of encoding grammatical information manually.

7. Directions for future research and methodological development

There remains a great deal that is not known about atypical signing in general, and about the effects of atypical signing on simultaneity in particular. What is known suggests a number of areas of research to be explored. First, it would be interesting to do kinematic studies of simultaneity in typical and atypical signers, in which the relative timing of movements of multiple articulators such as the dominant and non-dominant hands, the trunk, and the head could be compared. In this way, it would be possible to distinguish normal articulatory variation from disrupted signing. Similarly, high speed video recording could be used to investigate the relationship between signing and mouthing during typical and atypical sign production.

Another open research question is whether there are mechanisms that can compensate for loss of simultaneity. It may be that if one channel of information transfer were disrupted (e.g. facial expression), that signers might use another channel to transmit the same information (e.g. lexical signs). In a more basic scenario, if a signer loses the use of one arm, will she attempt to avoid two-handed signs, or will she produce two-handed signs with just one hand?

Finally, this paper has primarily addressed simultaneity as a production phenomenon - it would be interesting to also examine the role of simultaneity in typical and atypical sign perception to determine whether redundant information in the sign stream facilitates or impedes sign perception. It is unclear what aspects of signs or simultaneous constructions can be dropped or reduced and the utterance still be comprehensible. We know from research on typical signers that movements of the non-dominant hand can be deleted from a two-handed sign or added to a one-handed sign in everyday discourse (Battison 1978; Padden & Perlmutter 1987). However, beyond this, it is difficult to say which aspects of a sign (e.g. handshape, relative timing of articulatory movements, nonmanual sign components) can be modified and to what extent for the sign to remain intelligible.

Further exploration of simultaneity and sequentiality in signed language could be facilitated by improved methodologies and measures in sign phonetics. To date, there is no consistent set of measures, methods, or technologies for describing sign productions below the level of the phonological parameter. Development of methodologies for analyzing sign phonetics would allow researchers to better compare natural variation that occurs across phonetic contexts, across signed languages, and across individual signers. It is only by collecting articulatory data from a variety of signers and signed languages that we can learn what is de facto distinct about the structure of signed language, and not simply one possible solution among many for how to organize a language that uses the human hands as its primary articulators.

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