

Stuck on the Tip of My Thumb: Stuttering in American Sign Language

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## INTRODUCTION

Stuttering is a disorder that has been with humankind for all of recorded history. Stutterers have been tortured and discriminated against for centuries. They have been poked and prodded like lab rats for decades. There have been numerous studies done on stuttering and the frequency of stuttering. It is estimated that roughly 10.5 million Americans suffer from stuttering and an additional 50 million suffer from stuttering around the world (Williams, 2003). It is also estimated that 75% of children who stutter will “grow out of it” before they reach adulthood. Speech and language disorders comprise the largest portion of the disabled community, approximately 5-10% of that community ([www.geocities.com/stutteringhelp](http://www.geocities.com/stutteringhelp)). There is genuine effort underway to understand stuttering better, its causes, and its reach. So far, one area that has not been researched is the existence and the symptoms of stuttering in ASL. Manual communication offers unique opportunities and insight into languages and their effects on the human mind (or vice versa). Within the past two decades, psycholinguists have been able to determine scientifically that ASL is a language. Although there is a significant amount of research on ASL and the brain, much more remains to be done. There is however, one undeniable benefit to the comparisons of English and ASL and their effect on the brain: a better understanding of what language is and why it works.

As general knowledge increases in the comparisons of the spoken and signed languages of the world, there must now be a new direction in this research. That direction is the research of articulation disorders common to both forms of language. I believe a starting point rests with stuttering.

During the course of this thesis, I will summarize the volumes of information on stuttering research, and use this necessarily simplified synopsis to do something that has never before been attempted: isolate and identify the core symptoms of stuttering in ASL. I will attempt to see whether well-connected members of the Deaf community know sign language using individuals who demonstrate these newfound stuttering behaviors and incorporate their observations into the hypothetical list of symptoms. I will do all this, with the intention of proposing and stimulating interest in alternative avenues of research to be explored by professionals in the field.

During the first part of this paper I will discuss the controversy surrounding an actual clinical definition of stuttering, review the symptoms of stuttering in spoken languages, and discuss theories of the etiology of stuttering. In the second part of this paper I will explain the methodology of my study and summarize my results in the form of a single list of ASL stuttering symptoms. During the third, and final, part of this paper I will discuss what forms of stuttering may exist in ASL, I will discuss the inadequacies with the study, and I will discuss the alternative avenues of research that must be pursued.

## DEFINING STUTTERING

To define, according to Merriam-Webster's Online Dictionary (2003), is "to determine or identify the essential qualities or meaning of, to discover and set forth the meaning of (as a word), to fix or mark the limits of, to make distinct, clear, or detailed especially in outline, characterize, or distinguish." Any quest to attain the level of distinction required for a solid definition of stuttering in spoken language will encounter

problems. First of all, “stuttering,” as commonly used, has a broad definition. Hulit, the author of “Straight Talk on Stuttering,” is used frequently in these next pages because the manner in which he explains stuttering easily can be used to begin developing a list of ASL symptoms. Hulit (1996, p. 28) explains, “all experts would agree that stuttering is properly used to describe a speech disorder characterized by disrupted rhythm or disfluency.” However, Hulit continues, this would mean that all people would have to be stutterers, because “at one time or another, [all people] experience disrupted fluency . . . The mere fact that someone occasionally repeats sounds, syllables, or words does not make that person a stutterer. Stuttering goes beyond normal fluency failures, and therein lies the rub. If we had a solid handle on what constitutes normal speech, we would be in a better position to define stuttering.” Hulit (1996, p. 29) maintains, “Whatever lines [one uses to demarcate stuttering from other speech behaviors] are largely arbitrary.” Experts have tried to agree on what is stuttering and what is not, [and] their agreement has been very poor, not appreciably better than when laypersons have attempted the task.”

Stuttering, according to Hulit (1996), as used in the literature, includes speech disorders directly caused by various neurological conditions such as Down’s syndrome, mental retardation, cerebral palsy, and epilepsy more frequently than those who do not have neurological damage. There is one primary difference that can be made between those diagnosed with known neurological conditions and stutterers. First, those neurological conditions listed, and many others that are not, cause disfluency in speech that is constant. A stutterer’s is not. A stutterer’s speech is largely impacted by stress and environmental factors. Hulit (1996) states

“At the risk of making a differentiation that is too simplistic, the neurologically normal stutterer’s problem is heavily perceptual and the neurologically damaged individual’s problem is heavily motoric. Although both of these individuals have fluency problems that combine perceptual and motoric components, the neurologically normal stutterer is likely to do well when he *thinks* speaking will be easy and very poorly when he *thinks* speaking will be difficult” (p. 30).

Another definition, outlined by Robert Quesal (1998) is:

Stuttering is a disorder of fluency characterized by various behaviors that interfere with the forward flow of speech. While all individuals are disfluent to some extent, on the surface what differentiates stutterers from nonstutterers are the frequency of their disfluency and/or the severity of their disfluency. However, the other factor that differentiates stutterers from nonstutterers is that almost invariably the disfluencies that the stutterer regards as “stutters” are accompanied by a feeling of loss of control. It is this loss of control, *which can’t be observed or experienced by the listener*, that is most problematic for the stutterer.

The general commonalities in the definition of stuttering can be identified within these two definitions: frequency or severity of disfluency in speech and the stutterers’ personal feelings of loss of control. Thus, the subjective judgements involved make it rather difficult for science to say definitively whether a person is a true stutterer or not. Western science depends largely on definitive problems and definitive answers. Stuttering itself is a definitive problem, but the question looming is who definitively has the problem.

This research is the first ever to develop a list of stuttering symptoms in American Sign Language. Given the fluid nature of the spoken stuttering, however, it becomes difficult to juxtapose stuttering in a spoken language to stuttering in a non-spoken language by measuring exact symptoms. Given that nature, a flexible diagnostic criterion for the evaluation of stuttering symptoms in ASL will be used. Traditionally, three primary diagnostic criteria are used for diagnosing stuttering in spoken language. The three primary behaviors used to identify stuttering are repetition, prolongations, and

blocks. Given that the list of symptoms for stuttering is the first list created, not to mention very hypothetical, only two symptoms will be used to identify people who exhibit stuttering-like behaviors.

## SYMPTOMS OF SPOKEN STUTTERING

Hulit (1996) describes the behaviors of stuttering in two categories: “core behaviors of stuttering” and “behaviors superimposed on the core behaviors.” Core behaviors of stuttering are the initial culprits of stuttering. These include repetitions, closure, and prolongation. As the stuttering develops, additional behaviors are superimposed on the core behaviors, which we will address later.

The first of the core behaviors includes *repetitions*. A stutterer will, for example, repeat the letter “t.” (This is usually not a single repetition but a series of fast-moving repetitions of a sound, syllable or word.) For example, the stutterer might have problems saying the word “train.” Instead of “train,” the stutterer would probably produce a word much more like “t-t-t-t-t-t-t rain.” Repetitions could also happen for whole words, although most repeated words are one syllable. An example of a repeated word would be “I-I-I-I-I-I-I will go to the store.” These repetitions, according to Hulit (1996), are geared toward two purposes: “(1) finding the appropriate placement for the speech structure, and (2) finding the proper degree of pressure or muscular tension for the production of the target sound, syllable or word” (p. 49). Thus, the same, presumably, could be true in ASL in that a person seeking to articulate correctly a specific sign could engage in a similar kind of repetition to ascertain a proper manual articulation.

The second core behavior is the *block*, or technically known as the closure. During a blockage event, the stutterer, as described by Hulit (1996), “feels as though he cannot get the sound out at all, as though the air is stuck in his mouth or throat” (p. 50). During stuttering in spoken languages, the causes of the “self-imposed” restriction of air flow in a block are locking the lips shut, pressing the tongue against the roof of the mouth (specifically the alveolar ridge), or straining the vocal cords so much that air cannot pass through (Hulit, 1996, p. 51). In English, the block is most often produced when trying to create sounds that involve some closure in their pronunciation. Blocks are very often inaudible to the listener. These sounds that cause blocks are b, p, d, t, g, or k (Hulit, 1996). Presumably, during a stuttering event in ASL, the stutterer could “self-impose” a restriction of muscle movements, or extended holding of muscles to generate a similar block in ASL.

The third, and final, core behavior is *prolongation*. This occurs when a stutterer is trying “unnaturally” to force a word out. These most often occurs with the sounds s, z, sh, f, or v (Hulit, 1996). This involves an abnormal restriction of airflow through the larynx, but not a complete stoppage like in a block. Thus the stutterer exaggerates and prolongs the “natural” sound. For example, the stutterer might encounter problems when saying the word “forest.” Instead of saying “forest,” the word would be produced much like ffffffffOREST, although, unlike the repetitions, all the “f’s” would be blended into one prolonged sound. Presumably, it is possible for the prolonged syllable to be created in ASL in the form of a prolonged handshape.

Superimposed behaviors often constitute the more bizarre aspects of stuttering. These include a *vocal fry* (a ticking noise produce by the stutterer because of excessive

hypertension on the vocal folds), *complemental air* (using the air normally required for speaking a sentence on one word), and the *tremor* (“a rhythmic vibration of a muscle or muscle groups,” [Hulit, 1996, pg. 54]). Given the facts that communication in American Sign Language is visual in nature and that it does not require breathing regulation, the only superimposed behavior that would be applicable is the tremor.

There is a long list of symptoms that are associated with stuttering. However, only three of them are “core” symptoms. Those “core” symptoms are the focus of this research. However, there may be other more “minor” stuttering symptoms that could also be demonstrated in ASL. The Cincinnati Children’s Hospital’s website provides a lengthy list of the symptoms of stuttering:

#### Symptoms

The following are symptoms of stuttering:

- Repetitions of whole words, mostly one syllable, multiple times
- Repetitions of a syllable in a word, mostly the first syllable (i.e. ba-ba-banana)
- Holding out a sound or syllable (prolongation)
- Tremors (small movements) in the muscles around the mouth or jaw
- Changes in pitch or loudness of the voice
- Adding a schwa (i.e. buh/buh/buh/baby)
- Avoiding or refusing to talk because of fear of stuttering
- Struggling to speak
- Abnormal breathing

These are the widely accepted symptoms of stuttering that can be observed by a person other than the stutterer himself. These symptoms will be the basis of observing stuttering in ASL. If stuttering in all spoken languages reveals these same symptoms, then it will be necessary in future research to assess American Sign Language by the same characteristics to determine whether stuttering exists in ASL.

## PREVIOUS STUDIES

There have also been two studies that attempt to determine the rate of incidence of stuttering in Deaf individuals (Silverman & Silverman, 1971; Montgomery & Fitch, 1988). These studies, however, do not specify symptoms to look for in ASL, do not hypothesize what the symptoms might look like, and do not provide an operational definition of stuttering. These studies also do not take into account such variables as mild Tourette's Syndrome and cerebral palsy, which may very well exhibit stuttering-like behavior in individuals with one of the disorders. The rate of incidence in these studies was so low, .012, that it is very likely that stuttering may be attributed to disorders such as the ones previously mentioned. Because of these inadequacies, which are fundamental to the research, I have decided not to use them as sources for this thesis.

## ETIOLOGY OF STUTTERING

Wendell Johnson fashioned the views of stuttering for much of the 20<sup>th</sup> Century. He believed that the only difference between stutterers and non-stutterers is that stutterers were labeled as such at an early age. He believed that stuttering could be defined by the struggle, facial contortions, and disordered breathing required to speak. To him, these symptoms occurred only appeared after the child was labeled a stutterer. He believed that "stuttering is what one does in order not to stutter" (Hulit, 1996, p. 29). Today most experts disagree with Johnson. Current theories of the causation of stuttering may be

categorized in four general dynamics: auditory dysfunction, psycholinguistic dysfunction, learned behavior, and neurological dysfunction.

### *AUDITORY DYSFUNCTION*

The first dynamic, auditory dysfunction, is based on the disturbed auditory feedback theory. We all follow our speech patterns through feedback. This dynamic credits the cause of stuttering in the neurologically normal stutterer to a time delay from when the person first emits the sounds associated with a word to the time it is processed in the brain. Supporters of this dynamic cite evidence that stuttering can be replicated in people who are normally fluent. (Such tests include using headphones to produce delayed auditory feedback. This delay is a fraction of a second.) There have been many tests conducted in an attempt to isolate factors in producing delayed auditory feedback. No factor has been positively identified.

Because no single factor has been positively identified, if indeed stuttering is an auditory disorder, it would be ideal for the research to focus on those individuals who are profoundly deaf and have no chance of being exposed to this problem. Given that American Sign Language itself is a non-spoken language (relying on visual expressions of language rather than auditory expressions), it would appear that even those individuals who could hear would benefit from using sign language as an alternative language.

In fact, an interesting observation can be made in the plight of an unknown Australian woman who grew up stuttering and writes of her experience:

In 1999 I was diagnosed with cancer. I did the normal course of chemotherapy and radiation. But almost a year later I was told the conventional treatment was not working -- I was dying. Alas, there was one tiny ray of hope: a whopping six-week long continual dose of (what I called) high-voltage chemotherapy. I had nothing to lose, I was already dying and dammit, I would try anything not to die! Period. My sons, only 8 and 10 needed a mom and a dad. So I agreed. The good news: I am alive and in remission. The bad news: I am deaf. The high dosage of chemotherapy was 'ototoxic', or toxic to the cochlea and inner ear functions. While not an uncommon side effect of many chemotherapies, it is not one of the side effects people expect (like hair and weight loss).

It is almost a year since I became deaf. Do I stutter? No. You cannot stutter if you cannot hear yourself. Was it worth the trade-off? Absolutely not, uh uh, not a chance, no way Jose. I will never hear my children's voices or my husband tell me he loves me. Yes, we are learning American Sign Language, so we do communicate. But the world of magnificent sounds and music is gone forever for me (personal communication, Dr. Robert Lee Williams).

Perhaps an evaluation of stuttering in a “silent” language could render some clues as to whether stuttering is auditorily based.

### *LEARNED BEHAVIOR*

This dynamic argues that stuttering is a result of learned or conditioned behavior. Operant behaviors are behaviors that are either increased or decreased depending on their consequences (frequently referred to as the “carrot and the stick” principle). Operant behavior has two general “methods” of obtaining consequences: through presentation of a stimulus (both pleasant and adverse) or through a withdrawal of stimulus (both pleasant and adverse). Punishment by withdrawal (taking away a pleasant stimulus) has been proven to reduce stuttering by putting people in time-out as punishment for stuttering. A study by Haroldson, Martin, and Starr (1968) demonstrates that after 15 1-hour sessions (first 40 minutes with time-out and the last 20 minutes without time-out) the subjects' stuttering behavior decreased by a factor of 88%. It is important to note that no follow-

up studies were conducted on the individuals to demonstrate whether they had relapsed back into stuttering—and this research was conducted entirely in a laboratory, where day-to-day factors and stressors were minimal.

Punishment by presentation of adverse stimuli has also been championed as a possible solution. Flanagan, Goldiamond, and Azrin (1958) published the first study of operant behavior. Their controversial experiment included presenting bursts of noise at 105 dB and 6000 Hz when a subject stuttered. This study produced reductions of 31, 43, and 61% for 3 subjects, and virtually eliminated it for the other. The effects of this experiment have never been replicated, although Biggs and Sheehan (1969) conducted a study that could neither replicate the results nor challenge the findings. Martin and Siegel (1966) presented electric shock as punishment for stuttering. Costello and Ingham concluded that the initial tests were “impressive and unambiguous” (p. 195). In a later study in 1975 also headed by Martin to replicate the first results, the results were “much less impressive and somewhat ambiguous.”, Many studies have presented ambiguous results when presenting adverse stimuli: Daly and Cooper (1967); Williams and R. B. Martin (1974); and Hegde (1971); in a study by Janssen & Brutten (1973) stuttering actually increased as the shock treatment progressed.

Negative reinforcement (withdrawing unpleasant stimuli) has also been researched. Because of the nature of stuttering, withdrawing the unpleasant stimuli results in increased fluency. Increasing the rate of stuttering in individuals has little clinical value, but its use to researchers and clinicians is significant. It is significant because the ability to manipulate the frequency of stuttering, whether for better or for worse, yields new insights into the causes and characteristics of stuttering. In one study,

Flanagan et al. (1958), an adverse stimulus was continuously presented, (sound of 6000 Hz and 105 dB), and was halted for five seconds when the person stuttered. This study showed a 30% increase in stuttering behavior. In a later study, Goldiamond (1962; 1965), continuous shock was administered and halted for ten seconds following a stutter. This produced an increase in the percent of the words stuttered [%WS]. The first subject initially had a 37% WS and at the end of the study had a 55% WS. The second subject saw only a small increase in stuttering.

The final operant conditioning method is positive reinforcement (presentation of a pleasant stimulus). The studies related to positive reinforcement are very confusing. One study, Lanyon and Barocas (1975), was a study to determine the effects of stuttering-contingent monetary gain/loss on the subjects. This study produced reductions in stuttering. (This is rather confusing because one would assume that the presentation of money would increase stuttering and not reduce it.) They hypothesize that both monetary gain and loss resulted in an increased self-awareness of the stuttering which had an overriding adverse effect—increasing the rate of stuttering, thus making this punishment by presentation of unpleasant stimulus. Additional studies are confounded by problems such as faked stuttering (presumably for additional monetary gain), inconsistent monetary rewards, and, according to Costello and Ingham (1984), results were predicted based on the researcher's "philosophical leanings" (p. 206), thus suggesting experimenter bias.

In conclusion, these studies all indicate that stuttering can be manipulated, either to increase stuttering or decrease it. Many people use these results to indicate that stuttering is a result of natural child disfluency. That is, if a child attempts to avoid disfluency; he/she inadvertently creates more. However, these studies merely point to a

change in symptomatic behavior. There have been no studies to demonstrate the effects of the subjects changed behavior with known physical conditions associated with stuttering including brain differences and laryngeal function. Deaf people have never been labeled stutterers as a result of childhood disfluency, given the fact that stuttering has never been diagnosed in ASL.

### *PSYCHOLINGUISTIC DYSFUNCTION*

The third dynamic—and the primary focus of this paper—is psycholinguistic. The first inquiry into a possible relationship between linguistic features and stuttering was made by Brown in 1937 and eventually expanded to include other researchers by 1942. According to Wingate, in 1938 Brown made the remarkable discovery that “almost all stuttering occurred on the stressed syllable of a word—regardless of the grammatical or structural characteristics of the word” (1975, p. 47). These results have been corroborated by additional research (Hejna, 1972; Wingate 1972). All of their original studies, according to Wingate (1975) yielded comparable findings; however, the idea was abandoned because of lack of interest in psycholinguistic comparisons. Recently however, the idea has become more popular and additional research is being conducted. There is one finding that has appeared with astounding regularity.

“Stuttering is related to the grammatical class of words: there is apparently more stuttering on ‘content’ words (nouns, verbs, adjectives, adverbs) than on ‘function’ words (articles prepositions, conjunctions, auxiliaries). It is also pretty well documented that stuttering occurs on longer words, on less familiar words, and on words occurring towards the beginning of utterances. It is also quite well documented that more stuttering occurs on consonants than on vowels, although some equivocal findings are reported” (Wingate, 1975, 46).

The two general findings revolve around one basic problem: consistency with problems on certain classes of words. Within a certain class of words, such as content words or long words, one would think it most promising to look when attempting to notice stuttering in American Sign Language:

“Analysis of the major dimensions of the linguistic features associated with increased stuttering reveals that there is considerable overlapping among them. Content words clearly tend to be longer than function words; they are also regularly less familiar than function words; they also occur more frequently near the beginning of utterances. This overlap suggests that the seemingly separate dimensions actually reflect a common quality. It might well suggest that the higher incidence of stuttering on certain words is more an expression of the ease with which a word is said. For example, longer and less familiar words are not likely to be produced with as much facility as short and familiar words. Thus, the difference in stuttering on content words, as compared to function words, might be explained as some function of the difficulty level of the speech act rather than in terms of the meaning of the word or a reaction to its communicative value” (Wingate, 1975, 48).

If it were indeed a psycholinguistic dysfunction, the chances of it showing up in sign language would be noticeable. From these findings, we can conclude that the longer/more complex a word is, and the less familiar the word is, the more likely that there will be problems with the word. This, again, gives us another avenue to look for.

### *NEUROPHYSIOLOGY OF STUTTERING*

There have been many PET scans that have showed that stutterers use both brain hemispheres. In fact, according to a Mankato.com website,

A 1995 PET scan study by Fox and Ingham found that when nonstutterers spoke, brain activation was higher in the left hemisphere than in the right in areas that control the muscle movements necessary for speech -- and also in the auditory areas that process incoming language formation. In stutterers, however, this dominance occurred in the right hemisphere. This difference persisted even

during chorus reading (which induces fluency in most stutterers). This suggests an inherent difference in the way nonstutterers and stutterers process and output language.

Similar findings have also been published by NIDCD (National Institute of Deafness and Other Communication Disorders). This would seemingly reinforce the theory that has been floating around for 20+ years that the words must “first get out of the brain before they can get out of the mouth.” For example, Foundas (2001) found that the planum temporale is larger in the left hemisphere of stutterers than most adults. This, according to Foundas, reduced interhemispheric size in adults who stuttered. This reduction Foundas hypothesized would be the cause of stuttering in spoken language. She also found that other speech language production areas of the brain are larger in adults who stutter than non-stuttering adults, such as extra-gyri and atypical planar symmetries and have a double diagonal sulcus). Thus, there is evidence that suggests there may be a biological cause to stuttering in spoken languages.

There has also been extensive research on language in the brain in Deaf people as well. The results of this research (Emmorey, 2003) indicate that Deaf people use different areas of the brain for both the processing of language and the production of language from hearing people. One of the core foundations in psycholinguistics is the existence of both a phonological loop and a visual-spatial loop. The phonological loop is primarily responsible for language production and comprehension in spoken individuals-- (memory is also believed to be, in large part, phonological in nature). The visual-spatial loop is much more oriented towards non-language aspects such as processing the layout of a room, or remembering how to walk to from your car to your office. Emmorey summarizes research on language and ASL to maintain two basic fundamentals. The first

is that deaf people, although using both the phonological and visual-spatial loops, have a significant additional portion of their brain dedicated to the visual-spatial processes. The second part is that production of ASL and the production of speech are in separate, but adjacent areas of the brain.

Thus, the two languages utilize similar areas of the brain for linguistic functions, and different parts of the brain for production functions. These similarities and differences could be identified and researched to isolate possible causes of stuttering in American Sign Language. For example, if the speech production areas of both spoken and signed stutterers are enlarged and/or ambiguous, that would provide compelling evidence towards a neurological cause of stuttering.

#### COMMONALITIES IN STUTTERING SYMPTOMS ACROSS LANGAUGES

There are two fundamental stuttering commonalities that can be seen when examining stuttering across languages. The first commonality is that stuttering exists in all known spoken languages ([www.cyh.com](http://www.cyh.com), 2002). The second commonality is that stuttering does not just occur in language-oriented settings, but can occur when people are playing a wind instrument as well (Meltzer, 1992; Silverman and Bohlman, 1988). From these two commonalities, there are 5 common characteristics that can be drawn. The first is that in all of these examples, stuttering is auditory in nature. All languages and wind instruments seriously studied up until this point have auditory properties to which stuttering could be attributed. If stuttering is found to exist in ASL, stuttering could not be auditory in nature, given that ASL is, for all functional purposes, a silent language. The second characteristic is that they all involve the same speech-production

areas of the brain. This is an important difference between spoken languages and ASL. The part of the brain involved with language production is separate, but adjacent to where spoken language production occurs. It is documented in Emmorey that language areas of the brain are very similar. Both English speakers and ASL signers exhibit similar aphasia symptoms if Broca's Area or Wernicke's Area are damaged. These areas are, however, separate from the actual language production areas. Thus, if stuttering were a result of a difference in the brain, it would be hypothetical that an individual could stutter solely in manual communication, solely in verbal communication, or in both. Another important characteristic is that stuttering in both spoken language and wind instruments involve the same muscles/organs to produce the sound with. If stuttering were to be the result of a failure to adequately "coordinate" all the separate entities involved with speech production, ASL would provide a different mechanism through which to express language. The final commonality I will draw is that stuttering in both wind instruments and spoken language are problems with initiation. In both of these commonalities, the greatest likelihood of an instance of stuttering is during the first syllable of a word or the first breath of a note. Given that stuttering appears to be a problem related to initiation, it seems reasonable to conclude that if stuttering were to appear in ASL it would also present itself as a problem of initiation.

Of all these characteristics the only one that applies to possible stuttering in ASL is the failure of initiation. Any possible discovery of stuttering in ASL in any other way would cast serious doubt on the other commonalities/characteristics and/or redefine the nature and understanding of stuttering.

## METHODOLOGY

This compilation of symptoms was developed by interviews with 10 well-connected members of the deaf community who are on the faculty of Gallaudet University. It was determined that restricting the interviews to faculty would avoid any possible problems with the Institutional Review Board (IRB) for two reasons. The first reason is that asking faculty for their professional opinion does not require approval by the IRB. The second reason why using the professional opinion of faculty members was important for this survey is that there could not be, in the limited time allotted, an adequate questionnaire developed for mass-use and approved by the IRB. This paper is largely designed to develop a list of possible symptoms of stuttering in ASL, a task of which there are no known previous attempts, and, as a result, any IRB approved questionnaire would probably be out-dated at the end of the first interview. It was therefore reasoned that seeking professional opinions first, from faculty who are well versed in ASL and faculty members who are knowledgeable about communication disorders, would be the most effective and logical first step to take in attempting to assemble the list.

The interviews consisted of three parts: a brief summary of basic stuttering information, a presentation of a list of symptoms and examples, and 3 pre-prepared questions for the faculty to answer. Each interview was expected to be 20 – 30 minutes in length, and the faculty members were provided with an email address to contact me if any additional ideas or comments occurred to them after the interview was concluded.

The brief summary of basic stuttering symptoms consisted of my providing information related to the nature of stuttering. The information included a brief overview

of such facts as the two commonalities of language and their subsequent characteristics, the initiation problem of stuttering, an explanation of the ambiguity of the clinical definition of stuttering, and an overview of the core symptoms of spoken stuttering.

During the second part of the interview, participants were presented with lists of symptoms, derived from the list of core symptoms in spoken language, that stuttering might resemble in ASL. This list provided explanations of the stuttering symptoms as well as an example of what such a disfluency MIGHT resemble in ASL. The faculty members were then asked to read over the list of symptoms, and, if they were not clear on an explanation or example to ask.

During the final phase of the interview, the faculty members were asked three fundamental questions, as well as common follow-up questions, similar to the following.

1.) Do you think the examples of stuttering provided are adequate given the nature of the disfluency described? If yes, can you think of any additional examples that might be different in nature from the example provided? If no, why do you feel the example was not adequate and can you think of an example that you would find satisfactory? 2.) Do you know any persons who are “healthy” in that they are not diagnosed with cerebral palsy, Tourette’s Syndrome, or any other neurological disorder that exhibits symptoms similar to these? If so, which symptoms did they exhibit and can you demonstrate how the symptoms were presented in ASL for that individual? 3.) Can you think of any other symptoms that could be related to a stuttering disorder in ASL? If so, what is the symptom and can you think of an example?

#### HYPOTHETICAL SYMPTOMS OF STUTTERING IN ASL

Of a list I compiled prior to beginning the interview process, 8 hypothetical symptoms of stuttering were supported during the interview process. Many of the examples were also modified / improved during the course of the interviews. One additional hypothetical symptom was proposed by one of the faculty members I interviewed and I have added it in this thesis as well. The surviving list of 8 hypothetical symptoms was compiled from spoken symptoms and characteristics I described in the literature review. I also draw on my own experience as a (spoken) stutterer in attempting to relate the spoken symptoms to ASL.

The first symptom is “inconsistent interruptions in sign and fingerspelling.” This is a broad symptom, however it underlies one of the important characteristics of stuttering: inconsistency. No patterns are consistent with stuttering; no one word is always stuttered upon; and no word is always produced fluently / fluidly. Due to the intricacy and complexity of fingerspelling, this *may* be more associated with fingerspelling gestures rather than sign gestures. Signed or fingerspelled gestures, for the purposes of this paper, are either whole or parts of signed linguistic communication. No example was provided for this symptom due to the broad nature of the symptom.

The second symptom provided was that “symptoms most often occur at the beginning of the sign gesture.” This would occur in the first (possibly second) fingerspelled gesture in a word. This could also occur in the first (possibly second) signed gesture in a phrase or sentence. Again, no example was provided for this symptom due to the generalized nature of the symptom.

The next symptom is “hesitation of sign movement.” This involves involuntary hesitations prior to or during signing and/or fingerspelling. I hypothesize this will be a

hesitation that occurs at the beginning of a sign. All other features of the sign (handshape, orientation, location, etc) will be accurate with the exception of the involuntary pause. An example would be having the two hands in proper location of the sign “START” prior to the initiation of movement. There would be an involuntary hesitation between the time the sign is completely formed and the time the movement necessary to complete the sign begins. It may not be visible in the video; however, there is muscular tension in the forearm prior to the contraction that actually initiates the sign that causes the hesitation.

The fourth symptom provided is “repetition of sign movement.” Many monosyllabic signs in ASL are repetitive by their nature (i.e. CHAIR). Thus, repetition of sign movement could only be observed in multi-syllabic signs and/or as irregular repetition movement in monosyllabic signs. An example of monosyllabic sign would be the name-sign “DAVID” as a “d” sign across the chest repeatedly. This would be a repeating monosyllabic sign; therefore, the repetition movement would have to be irregular. It is important when viewing the video to recognize the inconsistent repetition in the sign. An example of a multi-syllabic sign would be “GRANDMOTHER.” A possible stuttering symptom in grandmother would be repeating the initial gesture of the dominant hand without stretching out the dominant arm as the normal sign progresses.

The fifth symptom provided is exaggerated / prolonged signs. This is the extension of the time necessary to complete the sign appropriately. A variety of strategies could be used to accomplish this task. The sign, or possibly a whole phrase, would be signed very slowly to increase the time necessary to complete the sign. Another possible strategy for accomplishing this would be to make the sign significantly

larger than “normal.” An example of this would be signing house with completely outstretched arms and making each of the two movements of each arm in house, (the “roof” and the “walls”) extremely long in comparison with other signs with no intentional linguistic or storytelling significance.

The sixth symptom provided is “unusual body movements completely unrelated to linguistic communication.” This may include involuntary “extra-movements” during either sign or fingerspelling. This unusual meaning must carry no intentional linguistic significance (i.e. moving the elbows erratically during signed communication). An example of this would be unusual elbow moving during the sign “PARENTS.”

The seventh symptom is “fluidity of the sign.” This would be a sign that is choppy and unnaturally gated or timed. There may also be unusual orientation/movement in the sign itself. An example of this would be fingerspelling “dog” with a “d” that incorporates a half-circular movement prior to signing “o.”

The last of the eight symptoms originally provided is “inappropriate muscular tension associated with sign production.” This, presumably, would occur in the arms and hands of an ASL signer. This would, most likely, lead to an involuntary block or stoppage in the fingerspelling or signing. An example of this would be “TRAIN-GONE.” The significant “block” occurs at the very beginning of the signs with the fingers being locked/stuck in position before proceeding with the rest of the sign.

The ninth symptom, which was added during the course of the interviews, was adding a schwa. (You will recall that an example of adding a schwa in spoken language is buh/buh/buh/baby.) Several interviewees felt the addition of a schwa in the hypothetical symptoms serves in helping to identify persons who stutter in ASL. The

addition of a schwa would mean that gestures would be included prior to a sign that serve no storytelling meaning—although they may have linguistic significance that is not necessary for the context. The gestures could also have no linguistic significance at all. An example of this would be “winding” up the arm prior to signing “THROW.” This winding up would be adding additional time needed to overcome the initiation problem.

#### DEVELOPMENTAL STUTTERING IN ASL?

During the interview process, I asked those who I interviewed whether they knew of anyone who exhibited such symptoms while they were signing. Determining this would be most helpful in attempting to encourage further research in the area. A few said that they had exhibited two or more of the symptoms. For (spoken) stuttering, three core behaviors are used to identify stuttering: repetition, prolongations, and blocks. A person having three of these symptoms is widely considered a clinical stutterer. This list of symptoms is hypothetical and I am only interested in finding out whether there are “possible” cases of stuttering, rather than definitive cases. Because of these factors, a lesser standard was applied for the identification of possible stutterers. To be identified as a possible stutterer in this research, a person must exhibit only two of the listed ASL stuttering behaviors and the behaviors must be inconsistent. This lesser standard was adopted in consultation with Dr. Greg Snyder, the Speech Pathology Department’s stuttering expert.

When asked whether they had seen this type of behavior before, the interviewees mentioned the names of several people. When the interviewees were asked to identify the symptoms of these individuals, they described at least two or more of the listed

behaviors. I have been advised not to mention how many or which of the listed symptoms they exhibited in an effort to preserve total anonymity—especially given the fact that IRB has not been involved with this research thesis. The names of those given are being held until a process is approved by IRB for the Speech Pathology Department to contact and meet with these individuals.

There does, however, seem to be strong indications that people do stutter in ASL.

#### ACQUIRED STUTTERING IN ASL?

During the interview process, there were many examples / names of individuals mentioned of people with Cerebral Palsy or other neurological conditions. According to plosbiology.com (2004), acquired stuttering occurs in people with definitive brain damage (intracerebral hemorrhaging, stroke, head trauma, lesions to the brain). Developmental stuttering symptoms are inconsistent and largely prevalent at the beginning of sentences, long and/or meaningful words, or other syntactically complex statements. Internal mechanisms, such as nervousness or feeling a loss of control also contribute to developmental stuttering.

Acquired stuttering is very consistent.

Descriptions of many individuals who are ASL users with a neurological condition were largely consistent with the symptoms described above. Again, the large difference between these stutterers and other potential stutterers was the level of consistency. People with these disorders struggled throughout the sentence, regardless of complexity or other variants. Again, I will refrain from going into further detail related with these individuals because IRB has not approved a process for interviewing these

interviews, and total anonymity must be preserved until such research can be appropriately conducted. There is some research that indicates that stuttering is a symptom of Cerebral Palsy in spoken language (Woods et al., 1997). This symptom may be far more pronounced in the Deaf community given the different motor processes involved with the production of ASL.

#### FUTURE STARTING POINTS FOR RESEARCH

There is much work that can be done in this field. Additional research is needed in the refinement of the symptoms in ASL. It may be found that there are be omitted symptoms or symptoms in this list that are not related with stuttering. There would also need to be a clinical diagnosis of those individuals who “stutter” in ASL. Although very rare, it would prove very beneficial to examine stutterers who both speak and know ASL. Do they stutter in both languages or only one? It was discussed in the literature review that hearing children regularly go through periods of disfluency. This same finding could be assessed in Deaf children to see if it remained true. Do Deaf children acquiring ASL go through phases of disfluency? There would also need to be a more comprehensive and accurate attempt to measure the rate of incidence in ASL. There is significant evidence that demonstrates that a stutterer’s brain and a “normal” person’s brain are different. There is also evidence that a Deaf person’s brain is significantly different than that of a hearing person. How does an ASL stutterer compare with a spoken language stutterer?

This finding raises more questions than it answers.

## INADEQUACIES WITH STUDY

There were several major points that were not included in the research of this thesis. First of all, there is no formal diagnostic criterion in place for diagnosing stuttering in ASL. Therefore, this was the first attempt to identify symptoms of stuttering in ASL, and the diagnostic criteria needs much testing / refinement before it can pass scientific muster.

The second inadequacy with the research is that I was unable to interview any of the individuals who potentially stutter in ASL. This eliminates any ability to gauge adequately “internal mechanisms” of diagnosing stuttering such as feeling a loss of control or being more fluent when one thinks he/she will be more fluent.

Third, this research did not attempt to examine the effect of situational circumstances on the stutterer. Fluency is, in large part, determined by situational circumstances and an individual’s emotional reaction to them. (For example, people who stutter in spoken language are very likely to be significantly less fluent if they are nervous.) This research did not attempt to identify this sort of reaction in potential ASL stutterers.

Last, this research did not incorporate a list of what “grammatical stuttering” might resemble. After the interview process was concluded, I realized that this was a significant possibility. ASL has a large dependence on facial expressions and visual-

spatial usage. In retrospect, it might be possible for stuttering to exist in these new dimensions of language that were previously completely foreign to stuttering research. Grammatical stuttering, I believe, will be most prevalent in those individuals with Cerebral Palsy of other neurological conditions, and if it is present in those conditions, I believe it is possible for an individual who has developmental stuttering to also display these words. Stuttering particularly occurs in content words or sentence of significant syntactical complexity. In ASL syntax often involves extensive use of facial expressions. I believe it is possible for the syntactical complexity issue to be demonstrated in ASL through facial expressions (Karniol 1995; Natke et al. 2002), as cited in plosbiology.com (2004). If expressed in ASL, grammatical stuttering could be observed directly, instead of indirectly (through stuttering of words in sentences of grammatical complexity).

## PURPOSE

The declared purpose of this paper was to develop a list of hypothetical symptoms and collect sufficient evidence to further research in this field as well as to point out alternative avenues in stuttering research. In an e-mail to me on April 5<sup>th</sup>, 2004 Dr. Greg Snyder said as a result of this research we are “standing on a goldmine,” and the department was “most excited” about this new line of research. The alternative avenues of research this paper proposes are: to explore further the existence and properties of acquired stuttering and developmental stuttering in American Sign Language; to explore the existence of grammatical stuttering in a language where its symptoms are directly expressed (as opposed to spoken languages where syntactic complexity contributes to

stuttering only through the secondary means of causing stuttering with words); and, finally, by satisfying the first two avenues, to demonstrate a broader correlation between spoken articulation disorders and signed articulation disorders that must be more fully explored. On April 16<sup>th</sup>, 2004 I received another communiqué from Dr. Snyder on behalf of the Department confirming that they had applied and were expecting to receive three to four grants to continue this research concept.

I believe the purpose of this paper was achieved.

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