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Disfluency Clusters in Speakers With and Without Neurogenic Stuttering Following Traumatic Brain Injury

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Abstract

Purpose: Analyze the characteristics and rate of disfluency clusters in adults with and without neurogenic stuttering after traumatic brain injury (TBI).

Method: Twenty adults with TBI participated in this study, including 10 with neurogenic stuttering (Group B) and 10 without -stuttering (Group A). Disfluency clusters in speech samples were classified into three types: Stuttering-like (SLD), other (OD), and mixed (MIX).

Results: Speakers with and without neurogenic stuttering produced the same mean number of disfluency clusters. In addition, the mean length of clusters did not differ between these speaker groups although the longest clusters did. The most frequently occurring cluster type for people with neurogenic stuttering was MIX and OD for people without stuttering. Although the speakers in Group A produced stuttering-like disfluencies, these never occurred together to form a SLD type cluster. For Group B, the starter units of the clusters were usually stuttering-like disfluencies, while for Group A, the starter units were mostly interruptions.

Conclusions: Compared to non-stuttering speakers, stuttering after TBI did not increase the number of clusters, but rather lengthened them. In speakers with neurogenic stuttering, the number and length of clusters were related to the manifestation of other communication deficits, not to the frequency of stuttering-like disfluencies. Still, SLD clusters occurred only in those people with neurogenic stuttering. These findings raise questions about the nature of both neurogenic stuttering and the dynamics of disfluency clustering.

Keywords: Disfluencies, Disfluency Clusters, Fluency, Neurogenic Stuttering, Traumatic Brain Injury

1. Introduction

1.1. ***The Controversial Nature of Neurogenic Stuttering***

Disfluencies are part of typical speech regarded as fluent (Bortfeld, Leon, Bloom, Schober, & Brennan, 2001; Fox Tree, 1995); however the frequency of disfluencies tends to increase with some acquired neurological conditions, such as Parkinson's disease (Goberman, Blomgren & Metzger, 2010; Juste, Sassi, Costa, & de Andrade, 2018), stroke (Van Lieshout, Bose, Square & Steele, 2007; Yairi, Gintautas & Avent, 1981), and traumatic brain injury (TBI) (Jokel, De Nil & Sharpe, 2007). These conditions may induce sound, syllable or word repetitions which are considered the key features of neurogenic stuttering (NS) **(**Lundgren, Helm-Estabrooks & Klein, 2010; Theys, van Wieringen, & De Nil, 2008; Van Borsel, 2014). Similarly to developmental stuttering (DS), phenomena such as adaptation effect, secondary behaviors, and word initial stutters have been reported in people with NS (De Nil, Theys, & Jokel, 2017; Jokel et al., 2007). However, NS differs from DS in that people with DS produce more prolongations and blocks than people with neurogenic stuttering (PWNS) and PWNS are more likely to stutter in tasks that are often found to be fluency inducing, for example reading (De Nil et al., 2017; Guitar, 2014). In DS, stuttering frequencies vary in different tasks and they seem to vary also in NS (Tani & Sakai, 2011; Theys, van Wieringen, Sunaert, Thijs, & De Nil, 2011). Theys with her colleagues (2011) reported that PWNS produced the highest stuttering frequency (percentage of stuttered syllables) in a conversational speech task (M = 6%, range 0.6 - 19.4%), somewhat less in a monologue task (M = 4.1%, range 1.0 - 10.9%), and the lowest frequency in a reading task (M = 2.6%, range = 0.0 - 10.3%).

Both the incidence and the prevalence of NS remain unknown, but the condition generally is considered a rare disorder (Cruz, Amorim, Beca, & Nunes, 2018). NS has been associated with damage in all the lobes of the cerebral hemispheres (Grant, Biousse, Cook & Newman, 1999;

Krishnan & Tiwari, 2011; Lundgren et al., 2010; Theys, De Nil, Thijs, van Wieringen, & Sunaert, 2013), as well as in the corpus callosum (Hamano et al., 2005), brainstem (Balasubramanian, Max, Van Borsel, Rayca, & Richardson, 2003), cerebellum (Tani & Sakai, 2011), and thalamus (Van Borsel, Van Der Made & Santens, 2003; Levine & MacDougall, 2016). Interestingly, many studies have reported a strong relationship between stuttering and basal ganglia- circuits (Burghaus et al., 2006; Kono, Hirano, Ueda, & Nakajima, 1998; Nebel, Reese, Deuschl, Mehdorn, & Volkmann, 2009). For example, Theys et al. (2013) examined lesion locations of 20 stroke patients with NS and located the pathology in the cortico-basal ganglia cortical loop.

In Alm's (2004) review, stuttering seems to result from lesions in the basal ganglia and its circuits because the basal ganglia provide internal timing cues that are important for subsequent movements. Further, Levine and MacDougall (2016) proposed that word-initial repetitions are produced because the first component of a word is generated outside the basal ganglia while motor progression to the next sequence is prevented because the basal ganglia subsequently fail to provide the necessary cue for the upcoming movements. In addition, lesions in the thalamus may cause stuttering indirectly by disrupting the transmission of signals from the basal ganglia to the cortex because the role of the thalamus is to relay information to cortical regions. In a recently published review, neural circuits related to DS contain both an auditory-motor cortical loop, guided by the sensory cortex, that enables speech motor planning and execution, and a basal ganglia-thalamocortical loop, guided by cerebellum, that provides the initiation and timing of speech sequences (Chang, Garnett, Etchell, & Chow, 2018). Although these findings and theories seem promising, it can be difficult to determine the affected neural pathway of NS because lesions in one structure may disrupt the function of other structures.

1.2.Stuttering After Traumatic Brain Injury

Traumatic brain injury (TBI) is defined as an impairment in brain function due to an external physical force that damages brain tissue, with the primary pathology manifesting itself, for example, as contusion, hemorrhage, and diffuse axonal injury (Strasberg, Johnson, & Parry, 2016). The impaired brain function in TBI typically includes difficulties in higher cognitive functions (e.g. attention, executive functions, memory, and language) (McDonald, Togher, & Code, 2014). Therefore, it is not surprising that individuals with TBI have multifaceted communication problems, described as "cognitive-communication disorder" (Turkstra, Coelho, & Ylvisaker, 2005). For example, discourse skills in speakers with TBI have been described as inefficient and impoverished (Davis & Coelho, 2004). Patients with TBI also show impaired capacity in organizing and structuring information and the flow of thought (Hagan, 1984). In addition, deviant speech patterns in patients with TBI have been attributed to aphasia (Lundie, Erasmus, Zsilaveczm, & van der Linde, 2014; Theys et al., 2008; Togher, McDonald, & Code, 2014), apraxia of speech (Yadegari, Azimian, Rahgozar, & Shekarchi, 2014; Ziegler, 2008), and dysarthria (Cahill, Murdoch, & Theodoros, 2000; Wang, Kent, Duffy, & Thomas, 2005) These deviations typically result in linguistic and motor disfluencies (see Section 1.3).

Neurogenic stuttering (NS) after TBI is seldom mentioned in the literature and the majority of studies on NS concern stuttering after stroke (Lundgren et al., 2010). In a study by Tani and Sakai (2011), three patients out of five had started to stutter after a stroke (hemorrhage or infarction), one after bleeding at a cerebral arteriovenous malformation, and one after a traffic accident, that is, after traumatic brain injury. The last mentioned individual's brain imaging showed lesions in the right pons and both right and left putamen, and his speech disorder consisted of stuttering with dysarthria. His most common stuttering types were part word, syllable, and word repetitions as well as consonant prolongations. His stuttering frequency was highest in a reading task (15.3%), less severe both in a conversational (8.4%) and an explanatory speech task (8.3%), and mildest in a sentence repetition task (2.8%) which is somewhat contrary to what Theys et al. (2011) reported (see above).

Adding to the confusion are data reported by Jokel, De Nil and Sharpe (2007) who investigated single speech disfluencies in adults with NS after stroke (n=6) and after TBI (n=6). A mild-to-moderate task effect was noticed in the mean disfluency frequencies (per 100 syllables) for both the stroke group (reading 24% vs. automatic speech 11% vs. conversation 21%) and in the TBI group (reading 37% vs. automatic speech 34% vs. conversation 32%). The highest dysfluency frequencies in a reading task were also found by Tani and Sakai (2011, see above) but not by Theys et al. (2011; see above) who found the conversation task to produce most of the dysfluencies in NS. The variable results are most probably explained by methodological differences, for example, in the selection of participants in different studies. In Jokel et al. (2007) study, the TBI group of six subjects was very heterogenous: two had a background of language learning disability, three reported a positive family history of stuttering and four were diagnosed with posttraumatic stress disorder. Thus, one may ask if these subjects were "pure" representatives of NS.

Jokel et al. (2007) further divided the disfluencies into less typical (multi-unit word repetitions, sound and syllable repetitions, prolongations, and blocks), more typical (hesitations, interjections, revisions, interrupted words or phrases, repeated words and/or phrases), and other disfluencies (pauses, substitutions, omissions, additions). The two groups (NS after a stroke or TBI) did not differ from each other in a conversational task in terms of more or less typical disfluencies but they performed differently in reading and automatic speech tasks. Most of the more typical disfluencies in these two tasks were produced by the stroke patients (44% and 30%, respectively) while the TBI patients produced most of the less typical disfluencies (44% and 57%, respectively). Thus, other disfluencies were also frequent in both speaker groups. In addition, Lundie et al. (2014) reported repetitions (i.e. less typical disfluencies) to be the most commonly observed disfluencies in four cases with NS after TBI; one of them had a previous history of DS. However, more typical disfluencies such interjections, silent pauses, broken words, revisions and starters were also noted. Therefore,

disfluencies that are not typically considered features of stuttering appear to be important when characterizing the overall speech patterns of patients with NS.

In a retrospective study of 309,675 U.S. Iraq and Afghanistan veterans, 235 were diagnosed with NS (Norman, Jaramillo, Eapen, Amuan, & Pugh, 2018). The likelihood of an NS diagnosis was greater for veterans with concomitant TBI and post-traumatic stress disorder than for veterans without these diagnoses. This result points to a complication in understanding the etiology of NS in that psychological factors may exacerbate a fluency disorder resulting from neurological damage per se. The psychological sequelae of brain damage are not easily distinguished from the effects of disruption of the neural circuits that control speech fluency.

The studies of NS (for example Jokel et al., 2007; Lundie et al., 2014; Tani & Sakai, 2011) have focused on disfluency features occurring separately, however, not all disfluencies occur in solitude but rather in clusters containing several disfluencies (Bona, 2018; LaSalle & Huffman, 2015; Robb, Sargent, & O´Beirne, 2009). Although disfluencies occurring in clusters seem to indicate more severe speech planning difficulties than single disfluencies (Bona, 2018; Robb et al., 2009), information is lacking on the occurrence of disfluency clusters in NS.

1.3.Neurogenic Stuttering and Co-morbid Symptomatology

Neurogenic stuttering (NS) may remain undiagnosed, as different brain injuries lead to different presentations of co-morbid symptomatology, such as NS that is associated with aphasia, dysarthria or apraxia (Tani & Sakai, 2011; Krishnan & Tiwari, 2011). In a review by De Nil and colleagues (2017), data on co-morbid speech symptoms of 95 stroke patients were examined. NS as the only concomitant was reported in 28% of the cases, and the rest of the patients had either aphasia and/or dysarthria associated with NS. In addition, apraxia of speech has also been reported to occur with NS (Theys et al., 2011).

In the presence of comorbid speech and language disorders, it may be difficult and sometimes even impossible to differentiate between certain types of speech and language difficulties and stuttering (De Nil et al., 2017; Lundgren et al., 2010). In fact, the characteristics of NS are known to be etiology-specific (De Nil, Rochon & Jokel, 2009). One way to approach different speech phenomena is to consider the predictions and implications of speech production theories – such as Hickok´s (2012) hierarchical state feedback control (HSFC) model and Levelt's model of speech production (1989) (Figure 1).

Figure 1 around here

Figure 1. Levelt's (1989) and Hickok's (2012) speech production model. (See also Alm, 2004; Duffy, 2005, pp. 58-66; Theys, De Nil, Thijs, van Wieringen, & Sunaert, 2013)

According to both models, speaking starts with activation in the conceptual system (ideational level) which then activates the word level, or "lemma" (Hickok, 2012; Levelt, 1989). In the HSFC model, the lemma then activates in parallel the sensory and motor sides of the higher-level loop responsible for syllabic level analysis. The higher-level loop, in turn, activates also in parallel the sensory and motor sides of the lower-level loop responsible for phonemic level analysis. In Levelt's model (1989), phonological and grammatical encoding seems to correspond to the higher and lower level loops of the HSFC model. The systems in the HSFC model not only get feedback from motor acts but also predict movements yet to take place. These inverse corrections and forward predictions are coordinated by the Sylvian parietotemporal area in the higher-level loop, and by the cerebellum in the lower-level loop. After and possibly during the performance, acoustic feedback enters the higher-level loop and somatosensory feedback is directed to the lower-lever loop.

Apraxia of speech (AOS) is a condition described either as a phonological, motor or cognitive disorder (Kent, 2000). Interestingly, AOS sometimes manifests together with NS although speech features in AOS are often difficult to distinguish from stuttering behavior (Theys et al., 2011; De Nil

et al., 2017). Recently, stuttering-like disfluencies have been systematically analyzed in people with AOS (Bailey, Blombgren, DeLong, Berggren, & Wambaugh, 2017). In this study, the frequencies of stuttering-like disfluencies varied from 0.0 to 17 %, and interestingly, these frequencies remained relatively stable over time in speakers with AOS. The pathology behind AOS seems to be located in ventral premotor cortex and anterior insula (Dronkers, 1996; Hillis et al., 2004) whereas in NS, the cortico-basal ganglia-cortical loop (Theys et al., 2013) and circuits through the basal ganglia to putamen (Alm, 2004) have been noted to have an important role. Based on the HSFC model (Hickok, 2012; Figure 1), AOS reflects problems in the higher-level loop, that is in access to motor phonological codes resulting in variable error patterns and effortful speech in speakers with AOS. According to Levelt's (1989) model, AOS arises from problems in the articulation region where the phonetic plan is retrieved from a prearticulatory buffer, initiated and then executed (Deger & Ziegler, 2002; Kent, 2000; Peters, Hulstijn, & van Lieshout, 2000).

Neurogenic stuttering (NS) is often accompanied by aphasia (Baumgartner & Duffy, 1997, Theys et al., 2008). In fact, stuttering-like symptoms have been associated with most of the classic aphasia types (Lundgren et al., 2010). Hickok (2012) gives an example of conduction aphasia stating that it follows an impairment in the higher-level loop. That means that conduction aphasia reflects an impairment in the internal state feedback control between sensory and motor coordination – and manifests as phonological substitutions, many self-corrections, and difficulty in voluntarily repeat others' speech. In general, individuals who have aphasia with NS show higher stuttering frequency compared to non-aphasic individuals who stutter (Theys et al., 2011). De Nil and colleagues' (2017) have speculated if co-morbid linguistic difficulties may burden motor planning and execution processes and induce NS. The same issues have been discussed relative to DS (see Ambrose, Yairi, Loucks, Seery, & Throneburg, 2015; Guitar, 2014). Also in AOS, syntactically complex or long utterances are more difficult to produce than short and simple ones (Kent, 2000).

It is, however, plausible that PWNS after traumatic brain injury (TBI) suffer from typical cognitive impairments associated with TBI, and, thus, from disorganized language processing due to these cognitive disorders (McDonald et al., 2014). These disturbances are reflected through language use and manifest as irrelevant utterances, word-finding difficulties and problems in ordering words and propositions in utterances (Hagan, 1984). In fact, accessing words from semantic memory, that is, difficulties in word finding, naming and word fluency tasks, are the most common findings in people with TBI, and naming difficulty seems to be the most persisting problem during recovery (McDonald et al., 2014). Sentence level processing also appears to be affected by brain injury, and, for example, complex syntactic processing has been associated with increased pause time (Ellis & Peach, 2009). Therefore, disorganized language processing may produce specific disfluency patterns for speakers with NS after TBI.

Dysarthria has also been observed to co-occur with NS (Helm, Butler, & Canter, 1980; Krishnan & Tiwari, 2011). Approximately 60% of patients with TBI are estimated to have dysarthria early after onset, and approximately 10% chronically (Cahill et al., 2000; Wang et al., 2005). Dysarthria itself often affects multiple dimensions of spoken language, like voice, intelligibility and prosody (Kent, 2000). In the HSFC model (Hickok, 2012), dysarthria is considered as a low-level motor disorder because the errors are predictable and consistent due to conditions such as muscle weakness or abnormal tone. Hickok (2012) writes that cerebellar-cortical circuits control the lower level, considered as the phonetic level (Figure 1). In Levelt´s model (1989), dysarthria is considered a disorder of the motor execution level where speech is articulated. Like NS, different dysarthria types also occur, given different lesion sites, and therefore, the disfluencies may vary depending on the location and extent of trauma (Van Borsel, Van Lierde, Van Cauwenberge, Guldemont, & Van Orshoven, 1998). In Table 1, some guidelines are given to help clinical decision making between NS and other acquired speech disorders affecting fluency.

Table 1 around here

Table 1. Distinguishing neurogenic stuttering from acquired fluency reducing conditions

1.4. Disfluency Clusters

Disfluency clusters are aggregates of disfluencies that occur within a word or in adjacent words (Robb et al., 2009; Silverman, 1973). Existing studies of disfluency clusters are mostly based on children with DS (LaSalle & Conture, 1995; LaSalle & Huffman, 2015; Sawyer & Yairi, 2010) and adults with persistent stuttering (Robb et al., 2009) or cluttering (Bona, 2018; Myers, Bakker, St. Louis, & Raphael, 2012; Myers, St. Louis, & Faragasso, 2008). Previous studies have shown that grammatical complexity influences DS by increasing stuttering frequency as well as the number and length of disfluency clusters (LaSalle & Conture, 1995, Logan & LaSalle, 1999; Robb et al., 2009). Research on NS has been focused on the location, frequency, and form of single disfluencies (Jokel et al., 2007; Lundgren, et al., 2010; Tani & Sakai, 2011), and there are no published studies on disfluency clusters as they relate to NS.

In nonstuttering adults, increased processing load is associated with higher disfluency frequency levels (Bortfeld et al., 2001; Shriberg, 1996). In addition, long utterances are more likely to be nonfluent compared to short utterances. Kleinow and Smith (2000) examined syntactic complexity and length in the speech motor stability in both adults with and without persistent stuttering. Speech motor stability of people with stuttering decreased with increases in the length and syntactic complexity of utterances. Smith, Sadagopan, Walsh, and Weber-Fox (2010) similarly found that adults with stuttering showed less consistent inter-articulator coordination, compared to their fluent peers, and this effect was larger with increased length and phonological complexity. Therefore, linguistic processes contribute to the probability of breakdown in the speech motor system.

As TBI can cause various cognitive impairments that can be seen in communication either secondarily (word-finding difficulties, irrelevant utterances, impaired syntactic processing) or

primarily (dysarthria and stuttering) (Jokel et al., 2007; McDonald et al., 2014; Tani & Sakai, 2011; Togher et al., 2014) it is important to analyze not only stuttering phenomena or single disfluencies, but the possible strings of disfluencies that influence the communicative performance of PWNS. Probably the most common way to categorize disfluency clusters is based on disfluency taxonomy (e.g., Ambrose & Yairi, 1999; Yaruss, 1998). A cluster that includes at least two *stuttering-like-disfluencies* (sound and syllable repetitions, prolongations, and blocks) in the same or adjacent words is called a SLD-cluster, and a cluster including at least two *other disfluencies* (interjections, revisions, interruptions, and repetitions of words and phrases) in the same or adjacent words is called an OD-cluster (Hubbard & Yairi, 1988; Colburn, 1985; LaSalle & Huffman, 2015; Robb et al., 2009). In more recent studies (Robb et al., 2009; Sawyer & Yairi, 2010), a MIX-type cluster was added to the cluster classification. As expected, MIX clusters contain both OD- and SLD-type disfluencies.

La Salle and Conture (1995) found out that children with stuttering produced "pure" SLD-clusters when their nonstuttering peers did not show stuttering-stuttering clusters at all. It was also interesting that children with stuttering produced significantly more stuttering-repair clusters, than their fluent peers. In fact, the majority of disfluency clusters in fluent children are the OD-type (Colburn, 1985; Wexler & Mysak, 1982); however, in those children who stutter, the SLD-types (Hubbard & Yairi, 1988) or MIX-types dominate (LaSalle & Conture, 1995). Robb and colleagues (2009) investigated disfluency clusters in 10 adults who stuttered (M=35 years). The percentage of the disfluencies ranged from 9% to 31% (M=19%). The group mean for the number of clusters in adults was 14.4 (SD=5.5) which is less than that reported for children in previous studies (e.g., LaSalle & Conture, 1995; Logan & LaSalle, 1999). The cluster types were, in order of decreasing frequency: MIX, OD, and SLD. Of all the clusters, 76% had two elements, 19% had three elements, and only 5% had over four elements.

Myers with her colleagues (2012) investigated disfluencies and disfluency clusters in fluent adults and adults with developmental cluttering (PWC). The mean number of clusters did not differ

between these speaker groups statistically although PWC produced more clusters (M=5.02, SD=4.60) than did their fluent peers (M=3.2, SD=2.41). The most common disfluency cluster type was OD for both groups, including interjections, revisions and word repetitions. Adults with cluttering had statistically significantly more revisions and word repetitions in their clusters than did their fluent peers. In a very recent study by Bona (2018), disfluency clusters in nine adults with cluttering (PWC) were compared to typical speakers (n=9). In a 300-syllable speech sample, typical speakers produced a mean of 4.8 disfluency clusters (SD = 4.2) while PWC had 6.9 clusters (SD = 3.5) but the difference between the groups was not significant. Among typical speakers, there were two persons who did not produce disfluency clusters at all. Interestingly, in the typical speakers group 24.5% of the clusters consisted of more than two disfluent units, whereas in PWC the proportion was 36.2%.

A correlation with stuttering frequency and the number of disfluency clusters was found by LaSalle and Conture (1995), as mentioned, and Logan and LaSalle (1999) reported a correlation between grammatical complexity of utterances and disfluency clusters children with DS. In adults who stutter, fluent utterances have been reported to be significantly shorter than utterances with single disfluencies or disfluency clusters (Robb et al., 2009). Currently, we lack data on disfluency clusters in PWNS. Results based on children and adult stutterers suggest that disfluency clusters reflect difficulties in motor (SLD-clusters) and linguistic formulation and expression (OD-clusters), with mixed clusters indicating that both motor and linguistic formulation and expression processes are involved (Robb et al., 2009). Therefore, analyzing disfluency clusters in people with specific difficulties in both language formulation and motor coordination has the potential to increase our knowledge not only of NS, but also of the total speech production process.

1.5.Purpose of the Present Study

Previous studies of NS have observed only stuttered disfluencies (Tani & Sakai, 2011) or different disfluency types as single units (Jokel et al., 2007; Lundie et al., 2014). Not all disfluencies, however, occur in solitude but in clusters containing several disfluencies, also of various types (Bona, 2018; LaSalle & Huffman, 2015; Robb et al., 2009). When the disfluencies occur in clusters, they probably influence to the listener more than disfluencies occurring as singletons (Sawyer & Yairi, 2010). However, disfluency clusters are an integral part of disfluent speech, and it is likely that speakers who produce many disfluencies also tend to present a high number and a large spread of disfluency clusters (Logan & LaSalle, 1999). In addition, disfluencies occurring in clusters may indicate more severe speech planning difficulties than single disfluencies (Bona, 2018; Robb et al., 2009). NS has been considered a speech motor disorder (Balasubramanian, Cronin, & Max, 2010; Tani & Sakai, 2011), but it is rarely manifested in isolation from other communication disorders (Lundgren et al., 2010). The analysis of disfluency clusters has potential to expose the role of linguistic, cognitive and motor components of speech in PWNS. Therefore, the aim of this study is to increase our understanding of NS as well as the factors that contribute to the variability of its symptoms by analyzing the types and frequencies of disfluency clusters as well as their relationships to other communication disorders after TBI. The analysis utilized in this study is intended to offer a new perspective to the nature of NS, speech production itself, and possible fluency-improving strategies. The following research questions were thus addressed:

1. What are the types and rates of disfluency clusters produced by speakers with neurogenic stuttering?
2. Do the clusters in persons with neurogenic stuttering differ from those produced by non-stuttering peers with TBI?
3. What features affect the quantity and/or quality of the disfluency clusters found in both speaker groups?

2. Methods

2.1.Participants

The participants (N=20) consisted of two groups of speakers with communication problems following traumatic brain injury (TBI): 10 speakers without NS (Group A) and 10 with NS (Group B). To recruit participants with and without NS after TBI, flyers were sent to the Finnish Brain Injury Association and to speech-language pathologists (SLPs) working in neurological departments all over Finland, who distributed the flyers to candidate participants who then self-recruited themselves to the study by contacting the first author (*initials*). Study participation was voluntary, and all subjects provided an informed written consent before inclusion. Before data collection, the participants were asked to bring all relevant medical reports related to their brain injury as well as available reports from speech-language pathologists.

A total of 40 volunteers were evaluated during years 2014-2016. Of the volunteers, 18 subjects had NS reported in the recruiting SLP's report (see Section 2.1.2 Group B). In the interview and data collection session, eight of those volunteers were excluded because NS described in the recruiting SLP's report was not verified by the first author (also a SLP). Then, from remaining 22 subjects without stuttering (see Section 2.1.1 Group A), 10 were included to Group A. A total of 12 subjects were excluded based on unsuitable etiology (n=9; tumor, stroke, poisoning), or because of missing information from neuroimaging (n=3). The ethics of the consent forms, flyers, and experiment procedures were approved by the Faculty of Social Sciences in the University of Tampere.

2.1.1. Group A

Ten subjects with traumatic brain injury verified with CT or MRI and who did not stutter (see criteria below) comprised Group A (GA). Other inclusion criteria were age over 18 years, Finnish as the mother tongue, communication deficit followed by TBI, no communication disorders before the injury, nor bilingualism or hearing loss. The participants' mean age was 46.3 years (SD=11.6, range

34–64 years), and they were all right-handed. The typical educational background for this group was secondary education (6/10), while three participants had completed their Bachelor's degree and one participant had elementary school education only. The mean time since onset of the injury was 9.4 years (SD=5.1, range 2-19 years). Four subjects had received speech therapy during recent years (0-3 years before data collection for this study), and six subjects in an earlier phase in their medical history (+4 years ago before data collection for this study).

To verify stuttering in the participants, the frequency of stuttering-like disfluencies was calculated according to Guitar (2014) in the three tasks of sentence repetition, spontaneous speech and narrative speech. When the disfluency frequency was less than 3% in every speech task, the participant was defined not to stutter. The participants in GA showed, however, various communication disorders due to their injury and suffered from multifaceted cognitive deficits (Table 2). Aphasia and motor speech disorders had been assessed using the Finnish version of the Western Aphasia Battery (Pietilä, Lehtihalmes, Klippi & Lempinen, 2005) or the Boston Diagnostic Aphasia Examination (Laine, Koivuselkä-Sallinen, Hänninen, & Niemi, 1997), and various motor speech tasks by those SLPs who recruited the candidates for this study. Their reported findings were confirmed during the interview and speech data collection by the first author. In addition, the guidelines described in Table 1 were used to distinguish certain nonfluent conditions from NS.

Neurological and neuropsychological data were collected from the medical files with the participants' permission. The TBI severity classification by responsible neurologists was based on the Glasgow Coma Scale (*Brain Injuries: Current Care Guidelines*, 2017). All participants had one of their significant others (spouse, friend, aide, adult child) with them during the test and interview session that was performed by the first author to confirm the validity of the information gathered.

Table 2 around here (crosswise)

Table 2. Characteristics of participants in Groups A and B

2.1.2. Group B

Ten participants with NS after CT or MRI verified TBI comprised Group B (GB). Firstly, **NS** was diagnosed if the frequency of stuttering-like disfluencies was higher than 3% during one or more of the three speech tasks mentioned earlier (Guitar, 2014; Theys et al., 2011). In addition, the onset of stuttering after TBI had to be reported in the participant's medical file or SLP report. Thirdly, all participants had to have self-identified the onset of their stuttering. Other inclusion criteria were age over 18 years and Finnish as the mother tongue, no communication disorder before the TBI, bilingualism, nor hearing loss. Apraxia of speech, cluttering, echolalia, and palilalia were ruled out by the first author according to the guidelines in Table 1.

Seven of the participants were male, and three were female. Their mean age was 40.9 years (SD=15.9, range 19-61 years), and all were right-handed. Their educational backgrounds varied from a Bachelor's degree (4/10) and secondary education (3/10) to elementary school only (3/10). The mean time since onset of the injury was 8.1 years (SD=4.4, range 3-18 years). Seven subjects had received speech therapy during recent years (0-3 years before data collection for this study), and three subjects in an earlier phase in their medical history (+4 years ago before data collection for this study).

Three participants in GB had NS with aphasic symptoms, another three dysarthria with NS while the rest four had NS as the only communication disorder (Table 2). All participants suffered from multifaceted cognitive deficits due to their injury. Aphasia and speech motor disorders were assessed using the Finnish version of the Western Aphasia Battery (Pietilä et al., 2005), and various motor speech tasks by those SLPs who recruited the candidates for this study. As with Group A, communication deficits reported by SLPs for Group B were confirmed during the interview and speech data collection. In addition, neurological and neuropsychological data were collected from the medical files with the participants' permission. These ten participants also had one of their significant

others (spouse, friend, aide, adult child) with them in the test and the interview session to confirm the validity of the information gathered.

2.2. Procedures

2.2.1. Speech Sample

Three speech samples (sentence repetition, spontaneous speech, and narrative speech) were audio recorded in the interview session from each participant using a Zoom H2 –device (Zoom Corporation). The microphone was positioned approximately 20 cm from the subject's mouth. For this study, speech fluency was evaluated on the basis of the narrative discourse samples.

The rationale for the picture-elicited story generation task was to provide for some similarity of the content in the speech samples and, thus, to enable comparison between samples and participants. The story generation task was expected to guide the participants in maintaining the same topic, contrary to spontaneous speech task where speakers, especially those with TBI, may have difficulties in maintaining the clues for discourse (Biddle, McCabe & Bliss, 1996). The fictional narratives of adults with TBI have been described to be reduced in coherence, completeness and fluency (Biddle et al., 1996). Therefore, it was anticipated that the narrative story task increased the linguistic load related to semantics and syntax (imposed constraints on lexical and sentence structures) as well as to pragmatics (creating coherent story to the listener, using narrative styles), which were expected to reveal new aspects of stuttering and dynamics of disfluency clusters in PWNS after TBI.

Participants were asked to generate a story based on the 9-frame comic strip *Ferd'nand* by Henning Dahl Mikkelsen (year unknown). The same narrative task has been commonly used in speech and language studies in Finland (see Korpijaakko-Huuhka & Lind, 2012; Moore & Korpijaakko-Huuhka, 1996) as well as in clinical settings.

2.2.2. Analyses of the Disfluencies

The speech samples were transcribed orthographically using Praat-software (Boersma & Weenink, 2005). The speakers produced stories of varying length (range 101–356 syllables, mean =164.6, SD=61.7). The following disfluency types were identified and grouped into two classes based on Ambrose and Yairi (1999): (a) stuttering-like disfluencies (SLDs) including repetitions (sound, syllable and part-word), prolongations and blocks, and (b) other disfluencies (ODs) including interjections, word repetitions, phrase repetitions, interruptions, and revisions.

The total disfluency frequency (i.e., instances of SLDs and ODs per 100 syllables) was counted using the transcripts. Because half of the participants (GA) were not diagnosed as stutterers, the concept of "frequency of stuttering-like disfluencies" was adopted instead of "stuttering frequency", as in Bailey and colleagues (2017). The term "stuttering-like disfluencies" in association with neurological conditions has been used also by De Nil, Theys and Jokel (2017). Frequencies for ODs and SLDs were determined, and the most common disfluency types were thus established.

2.2.3. Analysis of Disfluency Clusters

A disfluency cluster was defined as the occurrence of two or more disfluencies in the same word and/or consecutive words (Colburn, 1985; Sawyer & Yairi, 2010). We classified the disfluency clusters into the same three types as Sawyer and Yairi (2010): (a) SLD-type clusters involving the occurrence of two or more consecutive SLDs (e.g., repetition followed by prolongation, like "*so-so-somethiiing*"); (b) OD-type clusters involving the occurrence of two or more consecutive ODs (e.g., interruption followed by interjection and phrase revision, like "*the scure um I mean scarecrowthing*"), and (c) MIX-type clusters involving the occurrence of both SLD- and OD-types (e.g., repetition followed by interjection, like "*ma-ma-man um tries to*". A disfluency cluster started when at least two disfluencies occurred adjacent to each other and ended when the speaker: (a) managed to continue

fluently (e.g., either linguistically, like "*umm (1.0) th (0.4) I mean (0.9) well she (0.2) no (0.1) he (0.2) took the car (0.6) and smashed it*", or articulated the utterance as "*b-b-boy umm bo-bo-boy um-um-um bo(˙0.3)y boy goes to school*"), or when the speaker (b) abandoned the message and started a new utterance on a different topic (e.g., two disfluency clusters separated by a long pause and a topic change, like "*Ta-ta-take (0.2) take (0.1) ta-ta-takes (0.3) umm (0.9) the gu (1.0) no the girl (0.4) um (1.1) gi-gi-girl taaake (6.1) theee:n (0.3) theeen the bi-bi-birds came*").

The total number of clusters and the number of each cluster type per speaker were determined. Next, the average length of clusters was calculated based on the number of disfluent units occurring in a cluster (e.g., three units in a cluster: "*he um he wou-wou-would go*). For each participant, cluster patterns were analyzed by observing the most common disfluency types and any possible repeated trends.

2.2.4. Reliability

The inter-judge reliability of the disfluency cluster analysis was estimated using Fleiss's kappa. Interrater reliability (IRR) between the investigator (1st author) and the rater (a qualified speech and language pathologist) was high: For the location of disfluency clusters, 100%; for their length, 94.8%; for SLD-type, 94.4%; OD-type, 92.2%; and for MIX-type, 96.0%.

2.2.5. Statistical Analysis

The data management and analyses were performed using nonparametric tests with SPSS 24.0 (2016). A series of *t*-tests (Mann-Whitney U) were used to evaluate the differences between the speaker groups according to (a) the type of disfluency cluster (SLD/OD/MIX); (b) the total number of clusters; (c) the mean length of clusters; (d) the unit starting a cluster, (e) total disfluency frequency; (f) frequency of stuttering-like disfluencies; (g) frequency of other disfluencies; and (h) disfluency type. Further, a Chi-Square test was used to determine a possible difference between the longest

clusters. Finally, a series of correlated analyses with Spearman's rank correlation coefficient were performed for each group to evaluate the relationship between the clusters and the disfluencies.

3. Results

3.1. Disfluency Frequencies and Types

The descriptive statistics of speech variables in the two speaker groups are presented in Table 3. The speakers in GA with aphasia, cluttering, dysarthria, and/or apraxia of speech had in a mean disfluency frequency of 15.5% (SD = 10.02). Interruptions and interjections were the two most common disfluency types for GA. The number of interjections correlated with disfluency frequency in this speaker group statistically (r_s = .847, p = .002). In this group, speakers with aphasia and apraxia of speech (n = 3) had the highest disfluency frequencies (M = 28%), and these disfluencies were mostly interjections and interruptions. The frequency of stuttering-like disfluencies varied from 0 to 2.8% per 100 syllables.

The speakers with NS in GB presented with almost twice as many disfluencies (M = 27.5%, SD = 17.08) as Group A (GA). The two most common disfluency types were interjections and stuttering-like sounds and syllable repetitions. The frequency of stuttering-like disfluencies varied from 3.4% to 27.4% and the most common SLD type was stuttering-like repetition, although prolongations and blocks also occurred. As expected, the high disfluency frequency related to the frequency of stuttering-like disfluencies (r_s = .770, p = .009).

Table 3 around here (crosswise)

Table 3. Disfluency frequencies and types in different speaker groups

3.2. Disfluency Clusters

The mean number of clusters was the same in both groups (see Table 4). The frequency of stuttering-like disfluencies correlated statistically with the average length of the disfluency clusters (r_s = .530, p = .016), but not with the frequency of disfluency clusters (r_s = .332, p = .118). In GA, the most common cluster type was OD (76.6%), and the number of OD-clusters also correlated statistically with the total number of clusters (r_s = .885, p = .001). The remaining clusters (23.4%) were of the MIX type. Although there were stuttering-like disfluencies, they never occurred together to form a SLD type cluster. Typically, a disfluency cluster started with an interruption (46%) or an interjection (27.2%). Initial revisions (12.7%), word repetitions (10.9%) and stuttering-like repetitions (3.2%) were less frequent. A wide variation in the lengths of these clusters was found (Table 4). Of all the clusters, 23.8% had two disfluency units, 42.8% had three units, 27.0% had four units; however, only 6.4% had over five units. The longest cluster contained 14 units for participant A10, and it was the MIX-type (see Sample 1 in Appendix 1).

Table 4 around here (crosswise)

Table 4. Disfluency clusters

Interestingly, different speech disorders profiled differently based on the clusters in GA (see Table 5). Speakers with dysarthria (subgroup a1; n = 3) had no MIX-type clusters, and they also had the smallest number of disfluency clusters. Further, the longest clusters in this subgroup were shorter than those in other subgroups. Aphasic symptoms increased the number of disfluency clusters: Speakers with aphasic symptoms with or without dysarthria (subgroup a2; n = 3), had on average 6.6 clusters. The longest clusters in this subgroup were on average 4.6 units long. These speakers did not have MIX-type clusters. Speakers with both aphasic symptoms and apraxia of speech (subgroup a3; n = 3) had the highest number of clusters and also the longest ones, with the majority being the MIX-

type. One speaker diagnosed with cluttering (participant GA2) had a total disfluency frequency of 10.5%, with three clusters having a mean length of 2.7 units. This speaker, however, had only MIX-type clusters.

Table 5 around here

Table 5. Disfluencies based on the subgroups of communication disorders

In GB (Table 4), the most common cluster type was MIX (42.9%), and the number of MIX-clusters correlated strongly with the total number of clusters (r_s = .869, p = .001). Cluster type SLD also correlated with the number of clusters (r_s = .708, p = .022), and stuttering-like disfluencies most frequently (46.1%) started a new cluster followed by, in order of decreasing frequency, interruption (22.2%), interjection (19%) and word repetition (12.7%). The mean length of the clusters in GB was 4.1 units. Only 15.9% of the clusters consisted of two disfluency units, but 34.9% had three units, 20.6% four units, and 28.6% had over five units. Participant GB2 produced the longest cluster (18 units), and it was the MIX type (see Sample 2 in Appendix 1).

Dividing the speakers in GB into three subgroups revealed that speakers with NS only (subgroup b1; n = 4) had the smallest number of clusters, and their longest clusters were also the shortest compared to other subgroups of GB (see Table 5). Speakers with NS and aphasic symptoms (subgroup b2; n = 3) produced on average 7.3 clusters, and their longest ones consisted of approximately 6.3 units. Surprisingly, speakers with NS and dysarthria (subgroup b3; n = 3) had the highest number of clusters (M = 8) and also the longest clusters (M = 9.6 units). Most commonly a cluster started with stuttering-like repetition (50%) in “pure” NS (n = 4) when compared to other speakers (n = 6), whose clusters started either with an interjection (33.3%) or an interruption (33.3%).

3.3. Differences in Disfluency Clusters Between Speaker Groups

The two groups differed from each other in multiple ways on the Independent-Samples Mann-Whitney U Test. The GB differed from GA, having both a higher disfluency frequency ($U(18) = 81.000$, $Z = 2.343$, $p = .019$, $r = .523$) and a higher frequency of stuttering-like disfluencies ($U(18) = 97.000$, $Z = 3.360$, $p = .000$, $r = .811$), as supposed. The clusters in GB were mostly the SLD ($U(18) = 80.000$, $Z = 2.802$, $p = .005$, $r = .626$) and MIX-type ($U(18) = 80.000$, $Z = 2.323$, $p = .020$, $r = .519$) differently from GA where the speakers produced statistically more OD clusters ($U(18) = 15.000$, $Z = -2.698$, $p = .007$, $r = -.603$). Interestingly, speakers with NS in GB had statistically more word repetitions ($U(18) = 88.500$, $Z = 2.982$, $p = .003$, r = .666) compared to speakers in GA.

In addition, the disfluency type that started a cluster differed from the groups statistically. GB produced statistically more cluster-initial stuttering-like repetitions ($U(18) = 87.500$, $Z = 3.106$, $p = .002$, $r = .694$) than GA did, and GA produced more cluster-initial interruptions when compared to GB ($U(18) = 20.500$, $Z = -2.307$, $p = .021$, $r = .515$). According to the Chi-Square test, speakers with NS in GB had statistically more clusters that contained more than five units than did the speakers in GA ($X^2(1, n = 22) = 8.001$, $p = .003$).

4. Discussion

Clusters of disfluencies have been explored in the stuttered speech of children (LaSalle & Conture, 1995; LaSalle & Huffman, 2015; Sawyer & Yairi, 2010) and adults (Robb et al., 2009) but not, as far as we know, in speakers with NS. Therefore, the aim of the present study was to examine the characteristics of disfluency clusters as well as the differences and similarities in disfluency clusters between speaker groups with and without NS. The data were based on a narrative speech task, and the samples were considered semi-spontaneous speech with some constraints on lexical and grammatical structures due to the use of pictures to elicit the stories. The first main finding of this research is that the mean number of clusters was equal for the two speaker groups (M = 6.3). This

result may indicate that NS in and of itself does not increase disfluency clustering compared to other acquired speech and language disorders. In other words, it seems that linguistic and cognitive demands induce disfluency clusters rather than stuttering itself in acquired neurological conditions. This is contrary to what has been found in studies of DS in children where stuttering itself has been found to increase clustering of disfluencies (Hubbard & Yairi, 1988; LaSalle & Conture, 1995). In addition, our subgroup analyses revealed that the number of clusters was higher only when the speakers with NS had either aphasia or dysarthria as a concomitant disorder – regardless of the frequency of their stuttering-like disfluencies. The second main finding of this study was that the disfluency clusters were statistically longer in PWNS than in participants in GA. In fact, the disfluency clusters appear to be longer in PWNS compared to adults with DS. For example, 75% of the disfluency clusters consisted of only two disfluency units in adults with DS in a study by Robb and colleagues (2009), whereas participants of Subgroup b1 (Table 5; participants with NS solely) had on average 5.5 units in their disfluency clusters. Interestingly, the disfluency frequencies were still almost the same for these groups (DS: 19%, NS: 20.5%). A third main finding was that stuttering-like disfluency clusters were found only in samples from PWNS but not in those with cluttering, apraxia of speech, dysarthria or aphasia.

4.1.Theoretical Explanations for Disfluency Clusters

It is not clear how or why the speech production system breaks down to make disfluencies co-occur or concatenate. Studies that have focused on DS suggest that the production of a single disfluent item may cause anxiety, which then further increases the risk of a new stutter (Still & Griggs, 1979). This aspect, however, may not be the case in PWNS, as they are not reported to show such strong secondary behaviors as people who have DS (De Nil, 1999), despite their documented negative communication attitudes (Jokel et al., 2007).

The production of disfluency clusters may also be explained by a motor-based theory where disfluencies are considered to result from a coordination breakdown between speech articulators (Hubbard & Yairi, 1988). When a disfluency occurs, the speech system fails to restore itself, which then leads to overflowing clustering behavior. This theory does not fit perfectly with disfluency clusters in NS, however, because, contrary to DS, etiologies and comorbid communication disorders vary (Ciabarra, Elkind, Roberts & Marshall, 2000; Theys et al., 2008). In fact, in the present study, 22% of the disfluency clusters in PWNS started with interruptions, and 19% started with interjections, which are understood as reflecting linguistic planning or formulation difficulties (Wexler & Mysak, 1982). In addition, as much as 27% of the disfluency clusters in PWNS were purely of the OD-type. Logan and LaSalle (1999) combined the linguistic aspects from a *covert repair hypothesis* (Postma & Kolk, 1993) with Hubbard and Yairi's (1988) motor-based theory to propose that disfluency clusters may be a speaker's repeated attempts to repair linguistic errors before they are produced.

Developmental stuttering (DS) has been explained by models of sensorimotor integration in speech production (Hickok, Houde, & Rong, 2011; Max, Guenther, Gracco, Ghosh, & Wallace, 2004). Hickok et al. (2011) proposed in their *state feedback control model* that in persons who stutter, the mapping between the internal model of the vocal tract and the sensory system is 'noisy', so that the forward sensory prediction of speech gestures tends to be inaccurate. As a result, the system is caught in a predict-and-correct loop that is based on inaccurate error signals that result in stuttering. Max et al. (2004) took a somewhat different view, hypothesizing that stuttering results from either unstable or insufficiently activated internal models or an overreliance on afferent feedback that leads to instabilities because of delays occurring in the feedback loop. These theories of DS may be relevant to NS as well. If the neural disturbance responsible for NS either erodes the models of speech production or contaminates the mapping between the models and the sensory system, disfluencies may possibly result. The frailty of the system may be further compromised by difficulties in language

formulation, which could then disrupt the ongoing interplay between the feed forward and feedback signals.

Based on recent findings of the basal ganglia-cortical circuits involvement in NS (Burghaus et al., 2006; Nebel et al., 2009; Theys et al., 2013), the factor of timing in NS may be important (Alm, 2004) as the basal ganglia have a special role in providing timing cues for forward prediction and upcoming movements (Max et al., 2004). In the HSFC model (Hickok, 2012; Figure 1), the possible "location" for NS could then be somewhere in the lower-level loop where the somatosensory targets are involved. However, from the perspective of auditory targets, one may consider NS a deficit in the higher-level loop as altered auditory feedback (AAF) may either decrease the number of disfluencies (Krishnan & Tiwari, 2013; Marshall & Neuburger, 1987), or increase it - or have no effect at all on disfluencies (Balasubramanian & Max, 2008; Balasubramanian et al., 2010; Van Borsel, Drummond, & Pereira, 2010). In this study, based on the symptomatology, three different subgroups of PWNS were found. A concluding hypothesis could be that, because of the co-morbid symptomatology and varying etiology (De Nil et al., 2017; Lundgren et al., 2010; Theys et al., 2011), NS can be "located" in different levels in the hierarchy, as well as in the transfer of information between the lower and higher level loops in the HSFC model, rather than exclusively in any one loop.

4.2. Characteristics of Disfluency Clusters after Traumatic Brain Injury

In typical speakers, 75.5% of clusters and in adults with DS, 75% of the clusters were found to consist of two disfluency units (Bona, 2018; Robb et al., 2009). However, in the current study, the occurrence of clusters with two disfluency units was much lower – 15.9% for speakers with NS, and 23.8% for speakers without NS. This means that speakers with NS produced longer clusters more frequently than did adults with DS. One explanation for this difference could be the many cognitive deficits related to TBI, such as changes in attention, executive functions, problem solving, and

memory (McDonald et al., 2014), which were also reported in the participants of this study. One empirical example of the possible interplay between speech production, linguistic planning, and other cognitive processes is what we call 'abandoned' clusters, that is, failures of the speaker to return to either linguistically or articulatory fluent speech after a disfluency cluster (Appendix, Samples 3-5). We suppose that the of aphasic word-finding problems and dysarthria in Group B (GB) as well as aphasic and apraxic symptoms in Group A (GA) may have increased the length of the clusters (see Table 5); thus, additional memory and executive functions were possibly required to maintain the idea of the message and finish the utterance despite the disruptions. When the cognitive-linguistic system, however, was dysfunctional after TBI, the return to smooth operation was not possible. Admittedly, these hypotheses are risky and are based on very heterogeneous speakers and limited data. Therefore, observations relating to these "abandoned" clusters need to be further studied in a larger data setting, in different speech tasks and by analyzing the outcomes of the various disfluency clusters displayed in NS.

The role of different communication problems in the production of disfluency clusters also warrants further discussion. As seen in Table 5, the longest clusters were observed in three participants with aphasic symptoms and apraxia of speech (AOS) in GA (Subgroup a3) and also in three participants with dysarthria and NS in GB (Subgroup b3). That speakers with AOS in GA produced the highest quantities of clusters (amount and length) was not surprising because AOS is characterized by the production of sound distortions and substitutions with iterative attempts to repair the errors in actual speech output (Wambaugh, Duffy, McNneil, Robin, & Rogers, 2006). Besides AOS, these speakers had aphasic symptoms, mostly word finding difficulties, and many disfluent features (e.g., hesitations, long silent word finding pauses, word repetitions, revisions) which can easily cluster together during the word-search (Basso, 2003; Harmon, Jacks, Haley, & Faldowski, 2015). Therefore, it was unexpected that speakers with aphasia and NS (subgroup b2) did not have the longest clusters while speakers with dysarthria and NS did. Nevertheless, speakers with both NS

and aphasia (subgroup b2) had the highest stuttering frequencies compared to other subgroups, supporting the results from Theys and colleagues (2011).

Dysarthria may cause difficulty in producing speech at an appropriate rate, accuracy, or volume (Duffy, 2005). Dysarthria has been observed in speakers with NS (Helm et al., 1980; Krishnan & Tiwari, 2011), and it is a common deficit following TBI (Cahill et al., 2000; Wang et al., 2005). When dysarthria co-occurs with fluency difficulties like stuttering it is not surprising to see very long disfluency clusters because dysarthria itself causes various changes in voice, intelligibility, and especially in prosody (Kent, 2000). However, in this study, the frequency of stuttering-like disfluencies correlated positively with average length of the disfluency clusters. Therefore, in the future, we should gather more information about different subgroups of subjects with NS, as it may lead to more developed diagnostic processes as well as specific therapeutic strategies (Theys et al., 2013).

4.3. Features Related to Disfluency Clusters and Disfluency Types

In this study, speakers with NS (GB) differed from their non-stuttering peers (GA) with respect to the types of disfluency clusters. Participants in GB produced statistically more stuttering-like and mixed clusters (SLD and MIX, respectively) than did speakers in GA. In fact, speakers in GA did not have SLD-clusters at all. These results are in line with previous studies that demonstrated that people without stuttering produce OD-clusters (LaSalle & Conture, 1995) and people with DS produce SLD- and MIX-clusters (LaSalle & Conture, 1995; Sawyer & Yairi, 2010). Interestingly, adults with persistent stuttering in the study by Robb et al (2009) had most typically MIX-type clusters, secondly OD type clusters, and only thirdly, SLD clusters, while in the present study, SLD clusters were more frequent than OD-clusters in speakers with NS.

Robb and his colleagues (2009) distinguished between “stalling" and “advancing” clusters based on the theory by Howell and Au-Yeung (2002). From this perspective, the OD clusters are stalling clusters which occur because the upcoming word is difficult, either semantically (difficulty in retrieving a word) or in articulation (difficulty in retrieving or constructing a coherent motor plan or difficulty in executing it). In contrast, the SLD clusters can be seen as advancing clusters, meaning that the word itself is somehow difficult to produce and, therefore, is stuttered. If we consider clustering behaviors with respect to stalling and advancing clusters (Robb et al., 2009; Howell & Au-Yeung, 2002), we can hypothesize that the OD-clusters may be produced “voluntarily” as a fluency-maintaining strategy, and SLD clusters to be produced “involuntarily” wherein the disfluencies are pathologically following each other. Then the MIX clusters in speakers with TBI may possibly represent an overload in the processes of speech planning and execution wherein the stalling and advancing disfluencies blend together and are also affected by the attempts to repair what can be repaired. The distinction between voluntary and involuntary responses in different cluster types needs neuropsychological evidence. Future research could clarify this issue by analyzing both the function and the outcome of the disfluency cluster, as well as each speaker’s attempts to “repair” stuttered words and utterances. If there are voluntary/involuntary aspects in these clusters, it would be important to know what types of rehabilitation might reduce these clustering phenomena.

Finally, comments should also be offered about the disfluencies in NS. In this study, PWNS produced statistically more word repetitions compared to speakers in GA. In addition to word repetitions, PWNS had an abundance of interjections in their speech (35.2%). In fact, interjections and word repetitions have been considered as part of NS in previous studies (Van Borsel & Taillieu, 2001; Van Borsel et al., 2003). Wingate (2001), however, objected to the inclusion of word repetitions in the taxonomy of SLDs because whole-word repetitions are widely regarded as aspects of normal speech. In the current study, word repetitions were excluded from the frequency of stuttering-like disfluencies, and therefore, it will be well worth studying how the results of this study may change if

word repetitions had been included in SLDs. As a final point, total disfluency frequency was significantly higher in speakers with NS than for speakers in GA who did not stutter. Still, the mean number of clusters was the same. As we already know that frequently occurring single disfluencies influence the listener, it is likely that the long strings of disfluencies in clusters also have an impact (Sawyer & Yairi, 2010). Therefore, any future studies related to PWNS should investigate how the length and frequency of clusters influence the severity ratings, as well as the communication effectiveness, instead of only observing the effects of stuttering phenomena.

4.4.Limitations of the Study

In this study, several different analyses were done on speech samples from a relatively small number of people. Both groups had only 10 speakers, and the speakers in these groups were heterogeneous, based on the severity of brain injury, time since the injury, and the communication deficits. In addition, for eight participants only limited information about the injury types was available their medical reports, often only a mention about CT or MRI scan, which hinders discussion about the possible associations of lesion sites and behavioral findings in this study. However, localization of lesion was not the main purpose of this study. In fact, because NS is relatively rare, most studies report data from a small number of participants. In natural clinical settings, the expectation is that most TBI patients, who have communication deficits, do not show clear symptoms of a single disorder, but more likely demonstrate a combination of different symptoms' occurring differently in tasks that have a different cognitive load. Because of these multifaceted phenomena, total disfluency frequency was used as a measure instead of observing only stuttering-like disfluencies. The total disfluency count incorporated ODs and SLDs, thereby capturing the disfluencies resulting not only from NS, but also from other speech and language difficulties, such as aphasia, AOS and dysarthria that made the speech sound disfluent. In the case of language difficulties,

some of the OD clusters were similar to word finding problems, which may have been the reason for clustering. This study did not use a control group of nonstuttering adults without neurological condition, which may be a useful direction for future studies.

The use of the term “stuttering-like disfluencies” (SLD) instead of “stuttering” may be puzzling to some readers as the concept SLD in association with neurological speech disorders is quite new (see Bailey et al., 2017; De Nil et al., 2017). As mentioned in the Methods section herein, we felt that when examining speech samples from some speakers without a formal diagnosis of stuttering, the term SLD was more objective when analyzing disfluencies. Because NS may not be a distinct disorder (Lundgren et al., 2010) we wanted to use the term SLD to emphasize that we are only observing symptoms in speech, not diagnosing disorders.

Some studies have measured disfluency frequencies based on words (Robb et al., 2009) and some based on syllables (Jokel et al., 2007). Because of the special morphosyntactical structure of the Finnish language (see Helasvuo, 2008), syllables are more commonly used instead of words in Finnish studies. Moore and Korpijaakko-Huuhka (1996) explain that because the base forms of words are built upon with affixes, a Finnish word is often equivalent to phrase in English. This is seen in the following example: the English clause “I wonder if I could throw myself into an adventure?” (10 words) translates into Finnish as “Heittäytyisinköhän seikkailuun” (two words). Because of these differences in the language structures between English and Finnish, some of the cross-language comparisons are limited.

Aspects of the task used in this study also need further consideration. In this study, disfluency and disfluency cluster analysis was performed on a semi-spontaneous speech sample based on a 9-frame comic strip depicting a little man (Ferd’nand by Henning Dahl Mikkelsen) in his garden, first sowing seeds and later chasing birds with a scarecrow (hence the “scarecrow story”). Semi-spontaneous speech refers to speech elicited by situational pictures or retell-stories (Prins & Bastiaanse, 2004). The story generation task required speakers to generate utterances instead of

simply repeating or reading them, and this “scarecrow story” has been commonly used both in clinical and research settings in Finland (see Korpijaakko-Huuhka & Lind, 2012; Makkonen, Korpijaakko-Huuhka, Ruottinen, Puhto, Hollo, Ylinen & Palmio, 2016; Makkonen, Ruottinen, Puhto, Helminen & Palmio, 2018). Although this task may have restricted lexical and grammatical choices, it enabled more reliable comparisons between subjects compared to unpredictable spontaneous or totally fictional narratives. The semi-spontaneous story-generation task most probably enhanced the speakers with TBI to maintain the clue of the story while in fictional narratives, speakers with TBI tend to leave out information seen as gaps in the stories and omitted implicit and explicit propositions (Biddle et al., 1996).

The task chosen naturally affects the speech outcome. It is known that both OD- and SLD-disfluencies increase as syntactic complexity increases (Silverman & Ratner, 1997) and that people with DS tend to produce more disfluencies during their self-generated versus prepared utterances (Logan, 2001) indicating that engaging in linguistic formulation increases disfluencies compared to a specific task, such as sentence repetition. Robb, along with his colleagues (2009), and Logan and LaSalle (1999) found that greater syntactic complexity increased the number of disfluency clusters. The scarecrow story task was anticipated to increase number of disfluencies as well semantic- and syntactic difficulties in creating narrative-like utterances as seen in fictional stories from speakers with TBI (Biddle et al., 1996). Therefore compared to sentence repetition or monologue from familiar topic, this narrative task possibly revealed a different sight of speech process and dynamics of disfluency clusters in PWNS. Thus, in the future, we are aiming to explore the nature of disfluency clusters in NS by analyzing data from the two other tasks (sentence repetition and spontaneous speech) that were not included in this study. In addition, data from dialogues need to be considered in the future to see if some fluency enhancing strategies exist that influence disfluency clusters in PWNS.

5. Conclusion

Based on this study, neurogenic stuttering (NS) is characterized by three special features. First, the disfluency clusters were longer in speakers with NS than in nonstuttering speakers. Second, although the clusters were longer, NS itself did not increase the number of clusters. Third, in NS, stuttering-like disfluencies (SLD) most commonly initiated clusters of MIX- and SLD-types whereas SLDs never clustered together in the speech of nonstutterers. These features may have potential diagnostic value. As to possible therapeutic interventions, our results suggest that a more holistic approach to communication skills would be attempted instead of only trying to reduce stuttering. This suggestion is based on the observation that the most frequently produced cluster type in NS was mixed including interruptions, interjections, word repetitions, revisions and stuttering-like disfluencies. The different disfluency features in these clusters are supposed to reflect the role of linguistic, cognitive, and motor components of speech production. The results of this study open a window for further discussion of the origin and dynamics of disfluency clusters, as well as the true nature of NS.

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Tables and Figures

Figure 1. Simplified models of Levelt's theory of speech production (1989) and Hickok's theory of hierarchical state feedback control (2012) (See also Alm, 2004; Duffy, 2005, pp. 58-66; Theys, De Nil, Thijs, van Wieringen, & Sunaert, 2013)

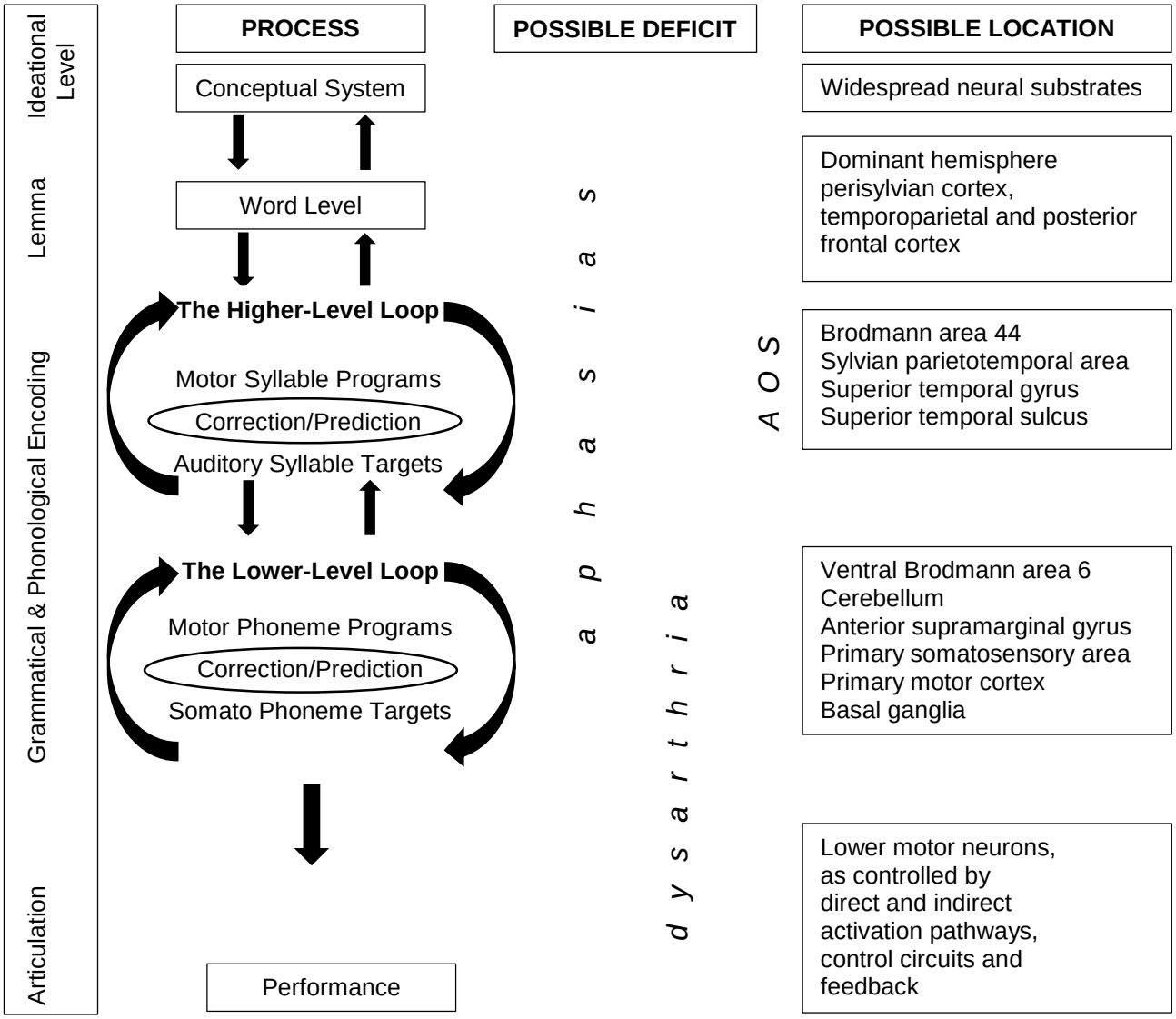

Table 1. Distinguishing neurogenic stuttering from acquired cluttering, palialia, echolalia and apraxia of speech (See De Nil, Theys, & Jokel, 2018; Duffy, 2005, pp. 358-359, 417, 427; Lundgren et al., 2010; Myers, Bakker, St. Louis, & Raphael, 2012; Tani & Sakai, 2011).

Fluency disorders	Distinction
Cluttering vs Neurogenic stuttering (NS)	In cluttering, speech rate is systematically rapid, but in NS, speech rate can be slow, normal or abnormal, but systematically not rapid. In cluttering, there are excessive amount of typical disfluencies (interjections, revisions, word and phrase repetitions, unfinished words and utterances), but in NS, stuttering behavior is pronounced and mostly manifesting initially in words. In cluttering, the most striking features are collapsed and deleted syllables in speech, which are not features in NS.
Palilalia vs Neurogenic stuttering	In palilalia, speaker is repeating involuntary his/her own words, which is not a feature in NS, where word initial sound-, syllable- and part word repetitions are dominant characters.
Echolalia vs Neurogenic stuttering	In echolalia, speaker is repeating involuntary others' utterances. This is not a feature in NS.
Apraxia of Speech (AOS) vs Neurogenic stuttering	In AOS, speaker makes compensatory efforts to correct his/her phonological errors and these error-revisions may contain multiple revision/interruption units. In NS, there may be phonological errors due to aphasia, but there are no variation in the error patterns like in AOS that makes the speech sound effortful In AOS, initiation and production is systematically laboured. NS can be co-morbid with dysarthria, and in those cases, the speech can be slowly produced, but the reason in those cases is the muscle weakness.

Table 2. Characteristics of participants in Groups A and B

ID	Age	Sex	Injury	Severity of TBI	Neuroimaging	Aphasic symptoms	Neurogenic stuttering	Dysarthria	Cluttering	Apraxia of speech	Cognitive deficits			
											AT	PS	EF	Mem
A1	39	M	Fall	moderate	expansive contusions in the parietal and occipital lobes (CT)			x						x
A2	41	M	MVA	mild	CT				x		x			x
A3	34	M	PA	moderate	CT	x		x				x		x
A4	50	M	MVA	very severe	contusions in the apex of the temporal lobe, frontal lobe and parietal lobe (MRI)	x				x			x	x
A5	36	M	Fall	moderate	CT			x					x	x
A6	35	M	Fall	moderate	cerebral edema, hemorrhage bilaterally in parietal lobes (MRI)	x					x			x
A7	43	M	PA	mild	hemorrhage in the occipital area			x						x
A8	60	F	MVA	severe	MRI	x		x				x	x	x
A9	61	M	MVA	very severe	contusion in frontotemporal area and deep parts of the brain (CT)	x				x		x		x
A10	64	M	Fall	very severe	expansive subdural hemorrhage, cerebral edema, contusions in pons area (MRI)	x				x		x		x
B1	59	M	Fall	severe	contusions in frontobasal area (MRI)	x	x				x			x
B2	22	M	MVA	moderate	CT		x	x				x		x
B3	26	M	MVA	very severe	CT		x	x					x	x
B4	61	F	MVA	moderate	cerebral edema, hemorrhage in the frontoparietal lobe and putamen (MRI)		x						x	x
B5	30	M	MVA	mild	diffuse axonal injury, mild contusion in right frontal lobe (MRI)	x	x					x		x
B6	56	M	MVA	moderate	CT		x				x		x	x
B7	19	M	PA	mild	subdural hemorrhage, contusion in the left parietal lobe (CT)		x	x						x
B8	42	F	Fall	moderate	CT		x							x
B9	59	F	PA	very severe	contusions in the deep parts of the brain, in frontal lobe and left temporal lobe (MRI)	x	x					x		x
B10	42	M	MVA	moderate	contusions in basal ganglia area and left frontal lobe (CT)		x					x		x

Note. Columns include participant identification (ID; A = GA, B = GB), age in years (at time of testing), sex (M = male; F = female), type of injury (MVA = motor vehicle accident, PA = physical abuse), severity of the injury, neuroimaging results (CT (computer tomography) or MRI (magnetic resonance imaging = neuroimagined head trauma, but information of the lesions missing), type of communication disorder, and type of cognitive deficit (AT = changes in attention, PS = changes in problem-solving, EF = changes in executive functioning, Mem = changes in memory).

Table 3. Disfluency frequencies and disfluency types in different speaker groups

ID	Total disfluency frequency (OD+SLD)	OD frequency	SLD frequency	The most common disfluency type (percentage of all disfluencies)
A1	8.9	8.9	0.0	Interruption (60%)
A2	10.5	10.3	0.2	Interruption (69%)
A3	9.8	9.8	0.0	Revision (39%)
A4	39.7	38.4	1.3	Interjection (92.3%)
A5	7.0	7.0	0.0	Revision (52%)
A6	12.4	10.0	2.4	Revision (50%)
A7	8.5	7.8	0.7	Interruption (34%)
A8	14.1	14.1	0.0	Interruption (66.6%)
A9	23.5	23.5	0.0	Interruption (55%)
A10	20.9	18.1	2.8	Interruption/interjection (60.8%)
Group Mean	**15.5 (SD 10.02)**	**14.8 (SD 9.75)**	**0.7 (SD 1.41)**	**Interruptions (38.1%)** **Interjections (31.7%)** **Revisions (20.9%)** **Stuttering-like repetitions (5%)** **Word repetitions (4.3%)** **Phrase repetitions (0%)**
B1	27.5	15.7	11.8	Revision (33.3%)
B2	47.3	31.6	15.7	Interjection (75.4%)
B3	27.6	14.4	13.2	Interjection (37.5%)
B4	23.6	5.8	17.8	Stuttering-like repetition (66.6%)
B5	10.9	7.5	3.4	Interjection (35%)
B6	15.9	12.3	3.6	Word repetition (47.4%)
B7	13.8	9.9	3.9	Revision (33.3%)
B8	26.6	9.4	17.2	Stuttering-like repetition (55.5%)
B9	66.0	38.6	27.4	Interruption (33.3%)
B10	15.8	7.0	8.8	Stuttering-like repetition (46.7%)
Group Mean	**27.5 (SD 17.08)**	**15.2 (SD 11.06)**	**12.3 (SD 7.69)**	**Interjections (35.2%)** **Stuttering-like repetitions (26%)** **Interruptions (12.8%)** **Word repetitions (12.1%)** **Revisions (9%)** **Phrase repetitions (2.9%)** **Prolongations (1.4%)**

Note. Columns include participant identification (ID; A = GA, B = GB)

Table 4. Disfluency clusters

ID	Number of clusters	The most common cluster type and its percentage of all clusters	Mean length of a cluster (units)	Disfluency that starts a cluster	Longest cluster type and number of its units	Disfluencies in the longest clusters
A1	4	OD (100%)	2.3 (2-3)	Interruption / Interjection	OD (3)	IR+R+I
A2	3	MIX (100%)	2.7 (2-4)	Interruption	MIX (4)	SLR+WR+R+I
A3	9	OD (100%)	3 (2-4)	Interruption	OD (4)	I+WR+I+R
A4	5	MIX (60%)	4.2 (2-8)	Interjection	MIX (8)	SLR+I+SLR+WR+I+IR+I+R
A5	2	OD (100%)	3.5 (3-4)	Interruption	OD (4)	IR+I+R+I
A6	5	OD (100%)	2.8 (2-4)	Interruption / Word repetition	OD (4)	WR+I+IR+R
A7	7	OD (100%)	3.4 (3-4)	Interruption	OD (4)	WR+R+I+R
A8	6	OD (100%)	3.2 (2-6)	Interruption / Interjection	OD (6)	I+IR+R+IR+R+R
A9	8	OD (87.5%)	3.5 (2-7)	Interruption / Revision	OD (7)	IR+R+WR+R+IR+I+R
A10	14	MIX (57.1%)	4.1 (2-14)	Interruption	MIX (14)	SLR+WR+IR+SLR+R+I+IR+R+R+R+I+IR+R+R
Group Mean	**6.3 (SD 3.46)**	**OD (76.6%) MIX (23.4%) SLD (0.0%)**	**3.4 (SD 1.76)**	**Interruption: 46% Interjection: 27.2% Revision: 12.7% Word repetition: 10.9% Stuttering-like repetition: 3.2%**	**5.8 (SD 3.29)**	**Revisions (36.4%) Interjections (20%) Interruptions (20%) Word repetitions (12.1%) Stuttering-like repetitions (8.6%)**
B1	7	MIX (42.9%)	4.3 (3-8)	Stuttering-like repetition	MIX (8)	(SLR+P)+WR+I+P+SLR+WR+I
B2	8	MIX (50%)	6.4 (2-18)	Interjection	MIX (18)	WR+SLR+WR+IR+SLR+R+WR+I+B+R+ WR+I+R+(SLR+B)+I+IR+R
B3	11	SLD (72.7%)	3.8 (3-6)	Prolongation	SLD (6)	(SLR+P)+SLR+SLR+(SLR+SLR)
B4	4	SLD (50%)	4.0 (2-8)	Word repetition	SLD (8)	P+SLR+SLR+P+(SLR+P)+SLR+P
B5	3	OD (66.6%)	2.6 (2-3)	Stuttering-like repetition	OD (3)	IR+R+R
B6	5	MIX (60%)	3.2 (2-5)	Word repetition	OD (5)	WR+I+R+WR+I
B7	5	OD (60%)	3.8 (3-5)	Word repetition / Interruption	MIX (5)	WR+I+SLR+WR+I
B8	5	MIX (60%)	3.2 (2-5)	Interjection	MIX (5)	I+(SLR+B)+SLR+I
B9	12	MIX (41.6%)	4.0 (2-8)	Interruption	MIX (8)	SLR+I+SLR+WR+IR+R+R+SLR
B10	3	MIX (66.6%)	3.6 (3-7)	Interruption	MIX (4)	I+IR+R+SLR
Group Mean	**6.3 (SD 3.16)**	**MIX (42.9%) SLD (30.1%) OD (27.0%)**	**4.1 (SD 2.36)**	**Stuttering-like disfluency: 46.1%** (SLR: 23.8% + P: 17.5% + B: 4.8%) **Interruption (22.2%) Interjection (19%) Word repetition (12.7)**	**7.0 (SD 4.73)**	**Stuttering-like repetitions (30.0%) Interjections (18.5%) Revisions (17.1%) Word repetitions (15.7%) Prolongations (10%) Interruptions (4.4%) Blocks (4.3%)**

Note. Columns include participant identification (ID; A = GA, B = GB), clusters (stuttering-like disfluency cluster = SLD, other disfluency cluster = OD, mixed disfluency cluster = MIX), disfluency types (interruption = IR, revision = R, stuttering-like repetition (sound-, syllable-, or part word) = SLR, word repetition = WR, prolongation = P, and block = B). Disfluency types in brackets (x+x) are disfluencies within a word.

Table 5. Disfluencies based on subgroups of communication disorders

	Subgroup a1	**Subgroup a2**	**Subgroup a3**	**Subgroup b1**	**Subgroup b2**	**Subgroup b3**
Mean of the total disfluency frequency	8.1%	12.1%	28.0%	20.5%	34.8%	29.6%
Mean frequency of the SLDs	0.2%	0.8%	1.4%	11.9%	14.2%	10.9%
Mean frequency of the ODs	7.9%	11.3%	26.6%	8.6%	20.6%	18.7%
Mean of the total number of clusters	4.3	6.6	9	6	7.3	8
Mean length of the longest clusters	3.6	4.6	9.6	5.5	6.3	9.6

Appendix. Speech samples 1-5 from the disfluency clusters

Sample 1. The longest disfluency cluster in Group A: Speaker A10 with symptoms of aphasia and apraxia of speech after traumatic brain injury

Original Finnish transcription (length of the pause noted in brackets):

pa-paprikoita (0.4) paprikoita (0.2) ker (0.2) ku-kurnitsoja (0.9) kurpitsoja (1.4) ää (0.7)
SLR WR IR SLR R I
sele (0.2) seleettejä (0.4) selaatteja (0.4) salaatteita (1.1) ö (0.2) sele (0.2) sataatteta (0.3) salaatteja (0.7) kaikkea hyvää kasvaa
IR R R R I IR R R fluent continuation

Translated English transcription (length of the pause noted in brackets):

pe-peppers (0.4) peppers (0.2) pem (0.2) pu-pumtins (0.9) pumpkins (1.4) um (0.7)
SLR WR IR SLR R I
sele (0.2) seleds (0.4) selededs (0.4) saleds (1.1) er (0.2) sele (0.2) salededs (0.3) salads (0.7) so many nice things are crowing
IR R R R I IR R R fluent continuation

Sample 2. The longest disfluency cluster in Group B: Speaker B2 with neurogenic stuttering and dysarthria after traumatic brain injury

Original Finnish transcription (length of the pause noted in brackets):

ukko (1.0) ukko (1.2) hät-hät-hätyttää (0.6) hätyttää (2.0) kor (0.3) va-va-variksia (0.8) eiku korppeja (0.7) korppeja niiku (0.9)
WR SLR WR IR SLR R WR I
ka´(0.2)us (0.4) kauas (0.6) kauas (1.3) öö (1.1) siis pois pe-pellolt´(0.3)a (2.0) ö (0.7) sar (0.3) peltomaalta pois (1.0) mutta sitte mies
B R WR I R SLR B I IR R fluent continuation

Translated English transcription (length of the pause noted in brackets):

man (1.0) man (1.2) cha-cha-chase (0.6) chase (2.0) ra (0.3) cro-cro-crows (0.8) I mean ravens (0.7) ravens well (0.9)
WR SLR WR IR SLR R WR I
a'(0.2)ay (0.4) away (0.6) away (1.3) um (1.1) I mean away from the fi-fiel´(0.3)d (2.0) er (0.7) cer (0.3) away from the arable land (1.0) but then the man
B R WR I R SLR B I IR R fluent continuation

Sample 3. Abandoned disfluency cluster: Speaker B1 with neurogenic stuttering and symptoms of aphasia after traumatic brain injury

Original Finnish transcription (length of the pause noted in brackets):

ki-ki-kihhhveli (0.4) kihveli (1.4) öö (2.0) tooolla (0.8) sa-saa (0.1) saa (0.9) ää (6.0) pihalla on (0.2) ku-kurpitsoja
SLR P WR I P SLR WR I rejection, and a new start

Translated English transcription (length of the pause noted in brackets):

du-du-dussstpan (0.4) dustpan (1.4) um (2.0) whiiit that he (0.8) ge-gets (0.1) gets (0.9) um (6.0) there are (0.2) pu-pumpkins on the yard
SLR P WR I P SLR WR I rejection, and a new start

Sample 4. Abandoned disfluency cluster: Speaker B4 with neurogenic stuttering after traumatic brain injury

Original Finnish transcription (length of the pause noted in brackets):

keeeppei (0.3) jo-joita (0.3) lai-lai-laittaa (0.5) riiistiin (0.4) pe-peeellolle (0.4) pys-pystyyn (1.4) kossska (3.4) ja sitte (0.3)
P SLR SLR P SLR P SLR P rejection, and a new start

Translated English transcription (length of the pause noted in brackets):

stiiicks (0.3) whi-which he (0.3) se-set up as (0.5) croossed up to (0.4) fi-fiiield (0.4) up-up (1.4) becaaause (3.4) and then (0.3)
P SLR SLR P SLR P SLR P rejection, and a new start

Sample 5. Abandoned disfluency cluster: Speaker B9 with neurogenic stuttering and symptoms of aphasia after traumatic brain injury

Original Finnish transcription (length of the pause noted in brackets):

harak-harak-harakkoja (1.0) ää (0.5) le-lentää (0.2) lentää (0.4) mö (0.1) kohti peltoo (0.2) kohti kasvimaata (1.3) et-et-et-et (4.1) noo sit täs ruudus
SLR I SLR WR IR R R SLR rejection and a new start

Translated English transcription (length of the pause noted in brackets):

mag-mag-magpies (1.0) um (0.5) fli-flies (0.2) flies (0.4) ca (0.1) towards the field (0.2) towards the kitchen garden (1.3) fo-fo-fo-for (4.1) okay in this frame
SLR I SLR WR IR R R SLR rejection, new start

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