

## A clinician survey of speech and non-speech characteristics of neurogenic stuttering

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### Abstract

This study presents survey data on 58 Dutch-speaking patients with neurogenic stuttering following various neurological injuries. Stroke was the most prevalent cause of stuttering in our patients, followed by traumatic brain injury, neurodegenerative diseases, and other causes. Speech and non-speech characteristics were analyzed separately for these four etiology groups. Results suggested possible group differences, including site of lesion and influence of speech conditions on stuttering. Other characteristics, such as within-word localization of disfluencies and presence of secondary behaviors were comparable across the etiology groups. The implications of our results for the diagnosis of neurogenic stuttering will be discussed.

**Educational objectives:** After reading this article, the reader will be able to: (1) provide a concise overview of the main literature on neurogenic stuttering; (2) discuss the speech and non-speech characteristics of neurogenic stuttering; (3) provide an overview of current clinical practices for intervention with neurogenic stuttering patients and their perceived outcome.

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### 1. Introduction

Neurogenic stuttering is an acquired speech disorder which typically affects adults following neurological disease. Based on published data, this acquired form of stuttering is most often associated with stroke (Jokel, De Nil, & Sharpe, 2007; Sahin, Krespi, Yilmaz, & Coban, 2005; Van Borsel, Van Der Maede, & Santens, 2003), traumatic brain injury (Jokel et al., 2007; Ludlow, Rosenberg, Salazar, Grafman, & Smutok, 1987; Yeoh, Lind, & Law, 2006), neurodegenerative disease (Koller, 1983; Leder, 1996; Mowrer & Younts, 2001), or another neurological event that affects brain function (Byrne, Byrne, & Zibin, 1993; Movsessian, 2005; Perino, Famularo, & Tarroni, 2000; Tsao, Shad, & Faillace, 2004). Although neurogenic stuttering behaviorally appears to have a lot of similarity to developmental stuttering, a number of authors have suggested several features which may differentiate it from developmental stuttering.

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For instance, Helm-Estabrooks (1999) has suggested the following six differential characteristics: (1) disfluencies occur on grammatical words nearly as frequently as on substantive words; (2) the speaker may be annoyed, but does not appear anxious; (3) repetitions, prolongations and blocks do not occur only on initial syllables of words and utterances; (4) secondary symptoms such as facial grimacing, eye blinking, or fist clenching are not associated with moments of disfluency; (5) there is no adaptation effect; and (6) stuttering occurs relatively consistently across various types of speech tasks.

Several case studies of patients with neurogenic stuttering have been reported in the literature, a number of which seem to confirm some or all of the behavioral characteristics suggested by Helm-Estabrooks (1999). For instance, Leder (1996) has described a patient with Parkinson's Disease who showed no specific word fears, no secondary stuttering characteristics, no adaptation effect and who stuttered equally on all types of speech. The patient described by Perino et al. (2000) also stuttered during conversational speech and oral reading, had no secondary stuttering characteristics and no specific word fears. In contrast, other case studies have demonstrated that not all patients with neurogenic stuttering may conform to all of the suggested differential characteristics. For example, Mowrer and Younts (2001) reported on a patient who stuttered more frequently on content words than on function words and whose speech and language problems did not occur when he read aloud. Similarly, Van Borsel et al. (2003) described a patient who was relatively more disfluent during propositional speech. In addition, the six patients with extrapyramidal disease described by Koller (1983) also stuttered mostly during self-formulated speech and presented with a positive adaptation effect. Sahin et al. (2005) discussed two patients with stuttering following an ischemic stroke who both stuttered only on initial sounds and syllables and one of them showed secondary stuttering characteristics.

These case studies seem to indicate that the differential features for neurogenic stuttering, as suggested by some authors, cannot be used unambiguously. Reviewing the existing literature at the time, Ringo and Dietrich (1995) found that five of the reported characteristics included in the criteria proposed by Canter (1971) and Helm-Estabrooks (1993) were reported in a majority of the cases they reviewed: (1) stuttering on grammatical words nearly as frequently as on substantive words (90%); (2) lack of anxiety about stuttering (80%); (3) disfluencies not restricted to initial syllables (75%); (4) absence of secondary symptomatology (70%); and (5) failure to exhibit adaptation effect (60%). Unusual phonemic loci of stuttering events and an inverse relationship between propositionality and disfluency were only reported in a minority of the reviewed case studies. More recent reports seem to suggest that the percentage of patients who show most of these characteristics may even be lower, and that the behavioral characteristics of neurogenic stuttering may indeed be more similar to developmental stuttering than previously suggested, confirming Lebrun's (1997, p. 109) statement that "at times the symptomatology of adult-onset stuttering is undistinguishable from that of developmental stuttering". More recently, Van Borsel and Taillieu (2001) also have argued that one cannot reliably differentiate between the acquired and developmental form of the fluency disorder based solely on the verbal output.

Whether or not acquired and developmental stuttering result in the same or different speech and non-speech characteristics is further complicated by the fact that most observations are based on single clinical case studies. Case studies provide a unique opportunity for detailed descriptions of clinical manifestations of neurogenic stuttering in single patients. However, it is often unclear in case studies whether or not the characteristics observed in that particular patient are typical for the disorder or whether they are reported specifically because they represent an atypical pattern of behavior unique to that individual patient. Consequently, it may be possible that atypical patients become 'the norm' if one were to rely on published case studies only. In addition, not infrequently many case studies are based on post hoc clinical reports and observations and lack the information necessary for a more detailed analysis (De Nil, Jokel, & Rochon, 2007).

In order to circumvent the inherent difficulties associated with case reports, a number of investigators have studied larger groups of patients experiencing neurogenic stuttering. In one such study, Ludlow et al. (1987) gathered information on 10 patients with stuttering following a penetrating brain lesion. Brain lesions were right lateralized in five patients, left lateralized in four patients and bilateral in the remaining patient. The conversational speech of these 10 patients was described as "shot from a gun". Bursts of rapid and unintelligible speech occurred together with long silences without struggle. Uncontrolled repetitions or prolongations were also present and patients were annoyed and surprised when they occurred.

Still other investigators have used surveys of clinicians working with patients with neurogenic stuttering. Survey studies provide a unique opportunity to collect data, albeit indirectly, on a large number of patients representing a wide variety of etiological conditions. Market, Montague, Buffalo, and Drummond (1990) reported on a survey in which they contacted more than 150 speech therapists in the United States. Of those, 100 had previously seen one or more

patients with acquired stuttering. Completed questionnaires were returned for 81 patients. Head trauma and ischemic lesions were the most frequent cause of the stuttering (75%) and lesion sites were often left lateralized (38%). For the majority of the patients the following differential speech characteristics were present: repetitions, prolongations and blocks were not restricted to initial syllables (80%); stuttering occurred on small grammatical words as well as on substantives (79%); the speaker seemed annoyed but not anxious (67%); and secondary speech characteristics were rarely present (68%). Absence of an adaptation effect was reported for 46% of the patients. Data on therapeutic results indicated that traditional treatment procedures for stuttering were also used for acquired stuttering and that almost all the patients recovered to some extent. Stewart and Rowley (1996) aimed at replicating the results from Market et al.'s study. They contacted speech therapists that were members of a special interest group for stuttering in the United Kingdom. Thirty-six therapists (36%) returned a completed questionnaire. Their results on stuttering etiology and outcome of the speech therapy were comparable to those of the previous study by Market et al., but the majority of the differential speech characteristics did not occur as frequently in the British population as in the American population.

While these survey studies have provided interesting information, they also had a number of important limitations. For example, both studies aimed at collecting information on acquired stuttering, without necessarily differentiating between neurogenic and psychogenic stuttering. In addition, information on the specific speech characteristics of neurogenic stuttering was limited and no information on previous speech or language problems was provided. As a result, there is need for a more systematic collection of data on persons with neurogenic stuttering to provide additional and more specific information on the characteristics of this disorder.

The data reported in this study, which was conducted in Flanders, the northern (Flemish speaking) part of Belgium, are based on a systematic survey of clinicians working with patients with neurogenic stuttering. Because Flanders has a population of six million inhabitants, concentrated in a relatively small geographic area, it provides for relatively easy access to speech therapists working in various clinical settings. In addition, obtaining data on a group of Flemish-speaking patients would allow us to gather information on patients with a different linguistic background, thereby providing some cross-linguistic validation of differential characteristics of neurogenic stuttering. The survey was restricted to reports on patients with neurogenic stuttering in order to avoid biasing the results with characteristics of patients with a psychological cause of the stuttering. Specifically, the aims of the present study were to obtain information on: (1) medical conditions and speech and language problems prior to the onset of neurogenic stuttering; (2) the speech fluency behaviors observed in these patients; (3) neurological conditions and the localization of brain trauma; (4) the presence or absence of additional speech and/or language difficulties; and (5) the type and outcome of speech therapy.

## 2. Method

### 2.1. Clinicians

Surveys were obtained by contacting 159 hospitals and 73 rehabilitation centers in Flanders by telephone, and by placing an advertisement in two Flemish professional speech–language pathology journals. Of the institutions that were directly contacted, 164 reported that they had a speech therapist working with a wide variety of patients with neurological disorders, such as stroke, traumatic brain injury, epilepsy and developmental disorders (e.g., ADHD, autism). Of these 164 speech therapists, 95 reported that they potentially had treated patients with neurogenic stuttering during the last five years and were interested in participating in the study. An additional three clinicians were recruited in response to the published advertisement. All therapists reported that they were in charge of working with patients with acquired neurological disorders.

Each of these 98 speech therapists was contacted individually by telephone to provide information on the study and to ask for their consent to participate in the study. To assist the clinicians in selecting patients for the survey, neurogenic stuttering was defined as ‘an acquired neurological communication disorder characterized by an abnormally high amount of repetitions of sound and syllables, prolongations or blocks’. More specific differential speech criteria for patient selection were not provided in order to allow the investigators to obtain information on a wide range of patients. All 98 speech–language therapists agreed to participate and were mailed one or more questionnaires (depending on the number of patients they thought they could report on) together with a cover letter and a pre-stamped, self-addressed envelope. Clinicians who did not return a completed copy of the questionnaire within two months from the mailing were

contacted again by phone. Ultimately, a total of 47 clinicians (48%) returned a total of 65 completed questionnaires. The main reasons that were given for not returning a completed questionnaire were that the speech therapist judged that her or his patient(s) did not have neurogenic stuttering after all, or that they did not have sufficient information on the patient to complete the survey.

## 2.2. Questionnaire

A Flemish custom-developed questionnaire was used to obtain comprehensive information from the clinicians on characteristics of neurogenic stuttering as observed in their patients. This questionnaire was developed using information from previously published surveys (Market et al., 1990; Stewart & Rowley, 1996) as well as additional questions developed specifically for this study. A translated English version of the questionnaire is included in Appendix A.

The questionnaire addressed the following seven topics:

- *Personal and medical information prior to the onset of the stuttering*: gender, date of birth, medical history, previous speech and language problems of the patient and his or her family, and handedness of the patient.
- *Onset of neurogenic stuttering*: age at onset, time period between the first diagnosis of the neurological condition and the first occurrence of stuttering, suddenness of onset, and important psychological events related to stuttering onset.
- *Cause of the stuttering*: stroke, traumatic brain injury, neurodegenerative disease, or 'other' causes.
- *Localization of the neurological injury*: cortical and/or subcortical localization, and lateralization.
- *Disfluency-related characteristics*: speech characteristics, emotions and attitudes, secondary behaviors, and adaptation effect.
- *Co-occurring disorders*: speech and language disorders, neurological disorders, and psychological disorders.
- *Therapy*: type and outcome.

For each of the questions included in the questionnaire, clinicians were asked either to check a box, circle the appropriate answer, or provide a short written response. Clinicians were instructed to use a separate copy of the questionnaire for each patient. Space was provided on the survey to add additional comments or observations which may not have been addressed otherwise.

## 2.3. Analysis

The 65 completed questionnaires were reviewed by the principal author. If one or more questions were not completed, the clinician was contacted again individually to obtain the missing information.

In the final analysis, 7 of the 65 questionnaires were excluded because neurogenic stuttering was diagnosed before the age of 10 and/or no specific neurological event preceding its onset could be identified objectively, which raised questions about differential diagnosis with onset of developmental stuttering. Therefore, the results described in this paper are based on the remaining 58 surveys.

Of the 58 subjects, 6 subjects were reported with pre-morbid stuttering and 1 was reported with pre-morbid cluttering. It was decided nevertheless to retain them in our pool of subjects because their stuttering was reported to have changed significantly following the occurrence of the neurological disorder. For these subjects, information on time lapse between occurrence of the injury and onset of stuttering will be reported as the time period between the occurrence of the injury and the reoccurrence of the stuttering or the change in the stuttering pattern.

The level of detail reported concerning the lesion site differed for all patients. In order to streamline the analysis, the answers to this question were grouped into more comprehensive categories, i.e., 'left', 'right', 'bilateral', and 'unknown'. If a lesion was both left and right lateralized, it was named 'bilateral', irrespective of the size of the lesion in the hemispheres. Similarly, the responses to the questions concerning the locus and type of disfluencies, which were provided by circling 'never', 'sometimes', or 'often', were grouped by combining the responses 'sometimes' and 'often' together under 'yes'. Data on the time period between the neurological injury and the stuttering onset were summarized in the following categories '<1 week', '1–4 weeks', and '>1 month'. For the questions regarding stuttering frequency, the highest reported frequency was included in the analysis, if more than one range of frequency was checked. Finally, the answers consisting of a short written response were summarized into the following categories:

‘yes’, ‘no’, and ‘unknown’. The more detailed information provided in these answers (e.g., type of previous speech and language problems, type of previous neurological disease, and type of co-occurring neurological symptoms) is reported descriptively in Section 3.

For the purpose of our analysis, the patients were grouped into different etiological categories (stroke, traumatic brain injury, neurodegenerative disease, and other). If a patient was reported to have multiple possible stuttering etiologies, he/she was placed into the etiological category that was closest to the stuttering onset. For example, if a patient had a traumatic brain injury followed by a stroke and started to stutter after these events, this patient was classified with the stroke patients. Because of the heterogeneity in our group of patients, the data are reported descriptively using both absolute numbers and percentages.

### 3. Results

Of the 58 patients, 29 experienced a stroke prior to the onset of neurogenic stuttering, 11 patients had a traumatic brain injury, and 9 patients were diagnosed with a neurodegenerative disease such as Parkinson’s or Alzheimer’s Disease. Another nine patients had a variety of isolated conditions, such as brain surgery ( $n = 3$ ), encephalitis ( $n = 2$ ), epilepsy ( $n = 2$ ), use of medication ( $n = 1$ ), or an unspecified cause ( $n = 1$ ). For the purpose of analysis of our survey results, and because of the small numbers in each etiological group, these latter nine patients were grouped together under the category ‘other’. The remainder of the results will be presented for each of the four etiology groups separately.

#### 3.1. Neurogenic stuttering following stroke ( $n = 29$ )

##### 3.1.1. General information

Table 1 lists the general information of all 58 patients in the survey, divided over the four etiology groups. Of the 29 patients who experienced neurogenic stuttering following a stroke, 19 were male and 10 were female. Their mean age at the time of the survey was 69 years (ranging from 36 years to 93 years). A number of patients had experienced previous neurological problems in addition to their stroke, including previous strokes ( $n = 5$ ), Parkinson’s Disease ( $n = 1$ ), and epilepsy ( $n = 1$ ). Four other patients were previously diagnosed with Crohn’s Disease, colon cancer, heart rhythm disorders, or diabetes. The speech therapists indicated that nine of the stroke patients took blood diluting or vasodilating medication or were on antidepressives or Parkinson-related medication prior to the stuttering onset.

Previous speech and language problems were reported to be absent in 16 of the stroke patients. One patient reported to have had articulation difficulties as a child and articulation disorders were also reported in this patient’s family. Three other patients were reported with pre-morbid stuttering, but as was explained earlier, it was decided to retain them in our pool of patients because their stuttering was reported to have significantly changed following the neurological event. One of these patients had a reoccurrence of previously disappeared childhood stuttering, whereas the two other patients were reported with significant changes in their stuttering pattern following the stroke. Of the three patients with previous stuttering, two had family members who stuttered. One patient without a history of pre-morbid stuttering had a son who stuttered during childhood. The large majority of the patients ( $n = 24$ ) were right-handed, two were left-handed, one was ambidextrous, and the hand preference of two other patients was not reported.

##### 3.1.2. Site of the lesion

Information on the lesion site of all patients for each of the four etiology groups is presented in Table 2. Among the 29 stroke patients in the survey group, 17 were reported to have lesions in the left hemisphere, 5 had lesions in the right hemisphere, and 5 showed bilateral lesions. For two patients no localization data was available. The reported lesions were localized to various regions of the brain, including frontal lobe, temporal lobe, parietal lobe, occipital lobe, basal ganglia, pons, and corpus callosum. Lesion localization was not correlated with handedness.

##### 3.1.3. Co-occurring disorders

Strokes often affect multiple functions in the patients and this was also the case in our survey population. Besides the possible presence of neurological and psychological symptoms such as paresis, tremor or coping problems, 27 of the 29 stroke patients were reported to have other communication deficits in addition to their stuttering. These co-occurring

Table 1

Number of patients (percentage in brackets) reported with previous medical problems, use of medication, previous speech and language problems, or familial speech and language problems, for each of the four etiology groups

	Stroke ( <i>n</i> = 29)			Trauma ( <i>n</i> = 11)			Neurodegenerative ( <i>n</i> = 9)			Other ( <i>n</i> = 9)			Total ( <i>n</i> = 58)		
	Yes	No	Unknown	Yes	No	Unknown	Yes	No	Unknown	Yes	No	Unknown	Yes	No	Unknown
Previous neurological problems	7 (24)	14 (48)	8 (28)	0 (0)	10 (91)	1 (9)	0 (0)	1 (11)	8 (89)	3 (33)	3 (33)	3 (33)	10 (17)	28 (48)	20 (34)
Other medical disorders	4 (14)	11 (38)	14 (48)	1 (9)	8 (73)	2 (18)	1 (11)	3 (33)	5 (56)	0 (0)	3 (33)	6 (67)	6 (10)	25 (43)	27 (47)
Use of medication	9 (31)	4 (14)	16 (55)	1 (9)	8 (73)	2 (18)	0 (0)	2 (22)	7 (78)	1 (11)	2 (22)	6 (67)	11 (19)	16 (28)	31 (53)
Previous speech and language problems—patient	4 (14)	16 (55)	9 (31)	1 (9)	8 (73)	2 (18)	2 (22)	6 (67)	1 (11)	1 (11)	4 (44)	4 (44)	8 (14)	34 (59)	16 (28)
Previous speech and language problems—family	4 (14)	13 (45)	12 (41)	0 (0)	6 (55)	5 (45)	0 (0)	1 (11)	8 (89)	1 (11)	2 (22)	6 (67)	5 (9)	22 (38)	31 (53)

Table 2  
Lesion site for number of patients in each of the four etiology groups (percentages in brackets)

	Stroke ( <i>n</i> = 29)	Trauma ( <i>n</i> = 11)	Neurodegenerative ( <i>n</i> = 9)	Other ( <i>n</i> = 9)	Total ( <i>n</i> = 58)
Left	17 (55)	4 (36)	0 (0)	6 (67)	27 (47)
Right	5 (17)	1 (9)	0 (0)	0 (0)	6 (10)
Bilateral	5 (21)	4 (45)	6 (67)	0 (0)	15 (26)
Unknown	2 (7)	2 (9)	3 (33)	3 (33)	10 (17)

communication deficits included one or more of the following disorders: aphasia (mostly Broca's aphasia), aphasic word finding problems, dysarthria, dysphonia, and apraxia of speech.

### 3.1.4. Disfluency-related characteristics

The mean age at onset of neurogenic stuttering in our stroke patients was 67 years, ranging from 35 years to 92 years. The data on the onset of the stuttering are presented in Table 3. For 20 of the 29 stroke patients, neurogenic stuttering was first noticed within the first week after the stroke. One patient had a reported stuttering onset three weeks after the stroke and another patient started to stutter two months after the stroke. Neurogenic stuttering was reported to have a sudden onset for 19 patients, whereas the reported onset was more gradual and became increasingly worse over a time period ranging from a few weeks to a few months for seven of the patients. One patient whose stuttering occurred immediately and suddenly following the stroke, reported that he also was very distressed at the time due to the closure of his favorite swimming pool.

Table 4 lists the information on the stuttering characteristics for the four etiology groups. The reported stuttering frequency varied widely in our stroke patients and ranged from less than 3% to over 50%. The majority of the patients were reported to repeat sounds and syllables and to block during speech. In contrast, whole word repetitions, prolongations and unnatural pauses were reported in approximately one third of the patients. All stroke patients for whom localization of disfluencies was indicated (*n* = 26), stuttered on initial sounds. Word medial disfluencies occurred in 11 patients, whereas final position disfluencies were noted in only 2 patients.

Information on grammatical category was available for 22 stroke patients. All of these patients were disfluent on content words, and all but three were reported to be disfluent on function words. The large majority of our stroke patients (*n* = 23) did not appear to stutter on specific sounds. In addition, 7 of the 13 patients for whom reading adaptation was evaluated were reported to show notable fluency adaptation during successive readings.

Sixteen of the stroke patients were reported to show secondary behaviors such as facial grimaces, associated limb movements, postponement behaviors and avoidance behaviors and 18 showed emotional reactions, such as frustration, irritation, fear, crying, and anger, to their stuttering.

For 12 stroke patients, the stuttering was reported to vary according to the conversational partner or the speaking situations. Nine patients stuttered only during spontaneous speech, whereas the majority of the patients (*n* = 19) stuttered during multiple speech tasks such as reading, singing and automatic speech.

### 3.1.5. Therapeutic intervention

Survey data with respect to reported therapeutic intervention is shown in Table 5. Twenty-three of the stroke patients were reported to have received speech or language intervention. Of those, 18 received therapy specifically aimed at their stuttering problems. Frequently used therapy approaches were: slowing speech rate, breathing therapy, cognitive therapy, fluency shaping, and speaking louder. Nineteen patients were reported to improve as a result of therapy with one reported to have fully recovered from stuttering.

## 3.2. Neurogenic stuttering following traumatic brain injury (TBI, *n* = 11)

### 3.2.1. General information

Ten male and one female patient in our survey started to stutter following a TBI. Their age at the time of the survey varied between 26 years and 82 years, with a mean age of 46 years. As shown in Table 1, for none of the patients neurological problems were reported prior to the TBI. One male patient had suffered from a depression and was on medication for this condition. Previous speech and language problems were reported for 1 patient who experienced

Table 3

Stuttering onset (&lt;1 week, 1 week–1 month, &gt;1 month, unknown) and progression (sudden, gradual, unknown) in number of patients for each of the four etiology groups (percentages in brackets)

Time period between neurological event and stuttering onset	Stroke ( <i>n</i> = 29)			Trauma ( <i>n</i> = 11)			Neurodegenerative ( <i>n</i> = 9)			Other ( <i>n</i> = 9)			Total ( <i>n</i> = 58)		
	Sudden	Gradual	Unknown	Sudden	Gradual	Unknown	Sudden	Gradual	Unknown	Sudden	Gradual	Unknown	Sudden	Gradual	Unknown
<1 week	16 (55)	3 (10)	1 (3)	3 (27)	0 (0)	1 (9)	0 (0)	0 (0)	0 (0)	3 (33)	0 (0)	0 (0)	22 (38)	3 (5)	2 (3)
1 week–1 month	0 (0)	1 (3)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	1 (11)	0 (0)	0 (0)	2 (3)	0 (0)
>1 month	0 (0)	1 (3)	0 (0)	1 (9)	1 (9)	1 (9)	1 (11)	0 (0)	0 (0)	0 (0)	2 (22)	0 (0)	2 (3)	4 (7)	1 (2)
Unknown	3 (10)	2 (7)	2 (7)	1 (9)	0 (0)	3 (27)	0 (0)	4 (44)	4 (44)	1 (11)	1 (11)	1 (11)	5 (9)	7 (12)	10 (17)

Table 4

Number of patients (percentage in brackets) showing different disfluency types, disfluency localization, adaptation, concomitant behaviors, and influencing factors for each of the four etiology groups

	Stroke ( <i>n</i> = 29)			Trauma ( <i>n</i> = 11)			Neurodegenerative ( <i>n</i> = 9)			Other ( <i>n</i> = 9)			Total ( <i>n</i> = 58)		
	Yes	No	Unknown	Yes	No	Unknown	Yes	No	Unknown	Yes	No	Unknown	Yes	No	Unknown
<b>Disfluency types</b>															
Repetitions sound	19 (66)	9 (31)	1 (3)	10 (91)	1 (9)	0 (0)	7 (78)	2 (22)	0 (0)	6 (67)	3 (33)	0 (0)	42 (72)	15 (26)	1 (2)
Repetitions syllable	16 (55)	12 (41)	1 (3)	10 (91)	1 (9)	0 (0)	9 (100)	0 (0)	0 (0)	6 (67)	3 (33)	0 (0)	41 (71)	16 (28)	1 (2)
Repetitions word	10 (34)	18 (62)	1 (3)	4 (36)	7 (64)	0 (0)	8 (89)	1 (11)	0 (0)	2 (22)	7 (78)	0 (0)	24 (41)	33 (57)	1 (2)
Repetitions part of sentence	4 (14)	24 (83)	1 (3)	1 (9)	10 (91)	0 (0)	2 (22)	7 (78)	0 (0)	1 (11)	8 (89)	0 (0)	8 (14)	49 (84)	1 (2)
Prolongations	9 (31)	19 (66)	1 (3)	6 (55)	5 (45)	0 (0)	2 (22)	7 (78)	0 (0)	4 (44)	5 (56)	0 (0)	21 (36)	36 (62)	1 (2)
Blocks	18 (62)	10 (34)	1 (3)	7 (64)	4 (36)	0 (0)	2 (22)	7 (78)	0 (0)	3 (33)	6 (67)	0 (0)	30 (52)	27 (47)	1 (2)
Unnatural pauses	10 (34)	18 (62)	1 (3)	3 (27)	8 (73)	0 (0)	0 (0)	9 (100)	0 (0)	3 (33)	6 (67)	0 (0)	16 (28)	41 (71)	1 (2)
<b>Disfluency localization</b>															
Initial sound	26 (90)	0 (0)	3 (10)	11 (100)	0 (0)	0 (0)	9 (100)	0 (0)	0 (0)	8 (89)	0 (0)	1 (11)	54 (93)	0 (0)	4 (7)
Medial sound	11 (38)	9 (31)	9 (31)	4 (36)	6 (55)	1 (9)	2 (22)	1 (11)	6 (67)	5 (56)	2 (22)	2 (22)	22 (38)	18 (31)	18 (31)
Final sound	2 (7)	16 (55)	11 (38)	2 (18)	8 (73)	1 (9)	1 (11)	3 (33)	5 (56)	1 (11)	6 (67)	2 (22)	6 (10)	33 (57)	19 (33)
Function words	19 (66)	3 (10)	7 (24)	8 (73)	0 (0)	3 (27)	9 (100)	0 (0)	0 (0)	6 (67)	0 (0)	3 (33)	42 (72)	3 (5)	13 (22)
Content words	22 (76)	0 (0)	7 (24)	8 (73)	0 (0)	3 (27)	9 (100)	0 (0)	0 (0)	7 (78)	0 (0)	2 (22)	46 (79)	0 (0)	12 (21)
Specific sounds	3 (10)	23 (79)	3 (10)	2 (18)	9 (82)	0 (0)	2 (22)	7 (78)	0 (0)	3 (33)	6 (67)	0 (0)	10 (17)	45 (78)	3 (5)
<b>Adaptation</b>															
Adaptation	7 (24)	6 (21)	16 (55)	2 (18)	5 (45)	4 (36)	0 (0)	4 (44)	5 (56)	2 (22)	2 (22)	5 (56)	11 (19)	17 (29)	30 (52)
<b>Concomitant behaviors</b>															
Secondary behaviors	16 (55)	11 (38)	2 (7)	7 (64)	3 (27)	1 (9)	4 (44)	5 (56)	0 (0)	5 (56)	4 (44)	0 (0)	32 (55)	23 (40)	3 (5)
Reactions to stuttering	18 (62)	8 (28)	3 (10)	9 (82)	2 (18)	0 (0)	3 (33)	6 (67)	0 (0)	7 (78)	2 (22)	0 (0)	37 (64)	18 (31)	3 (5)
<b>Influencing factors</b>															
Variation persons-situations	12 (41)	9 (31)	8 (28)	5 (45)	3 (27)	3 (27)	0 (0)	4 (44)	5 (56)	3 (33)	4 (44)	2 (22)	20 (34)	20 (34)	18 (31)
Only spontaneous speech	9 (31)	19 (66)	1 (3)	1 (9)	10 (91)	0 (0)	2 (22)	7 (78)	0 (0)	5 (56)	4 (44)	0 (0)	17 (29)	40 (69)	1 (2)

Table 5  
Therapy information for number of patients in each of the four etiology groups (percentages in brackets)

	Stroke ( <i>n</i> = 29)			Trauma ( <i>n</i> = 11)			Neurodegenerative ( <i>n</i> = 9)			Other ( <i>n</i> = 9)			Total ( <i>n</i> = 58)		
	Yes	No	Unknown	Yes	No	Unknown	Yes	No	Unknown	Yes	No	Unknown	Yes	No	Unknown
Speech and language therapy	23 (79)	6 (21)	0 (0)	9 (82)	2 (18)	0 (0)	7 (78)	2 (22)	0 (0)	7 (78)	2 (22)	0 (0)	7 (78)	2 (22)	0 (0)
Stuttering therapy	18 (62)	10 (34)	1 (3)	5 (45)	6 (55)	0 (0)	3 (33)	6 (67)	0 (0)	5 (56)	4 (44)	0 (0)	5 (56)	4 (44)	0 (0)
Improvement in therapy	19 (66)	3 (10)	7 (24)	4 (36)	4 (36)	3 (27)	1 (11)	5 (56)	3 (33)	4 (44)	1 (11)	4 (44)	4 (44)	1 (11)	4 (44)
(Partial) Spontaneous recovery	14 (48)	4 (14)	11 (38)	2 (18)	2 (18)	7 (64)	1 (11)	1 (11)	7 (78)	3 (33)	1 (11)	5 (56)	3 (33)	1 (11)	5 (56)

developmental stuttering that exacerbated following the TBI. Most of the patients were right-handed ( $n=9$ ), 1 was left-handed and the hand preference of another patient was unknown.

### 3.2.2. Site of the lesion

The results for reports on lesion site (see Table 2) indicated that of the 11 TBI patients, 4 had bilateral lesions, 4 showed lesions restricted to the left hemisphere, 1 showed evidence of lesions in the right hemisphere, and for 2 patients no observable injury was reported. The reported injuries comprised multiple regions in each of the four lobes, the cerebellum, the brainstem, and some unspecified subcortical regions.

### 3.2.3. Co-occurring disorders

Similar to stroke, TBI often results in multiple consequences for the patient's functional integrity. Six of our TBI patients showed paresis or tremor and five were reported to have various psychological problems. In addition, 10 of the 11 patients had aphasia, dysarthria, dysphonia, and/or apraxia of speech in addition to their stuttering.

### 3.2.4. Disfluency-related characteristics

The mean age for stuttering onset in the TBI patients was 40 years (ranging from 23 years to 80 years). Four patients started to stutter within the first week following the TBI, while three patients were reported to have started more than one month after the injury. The stuttering occurred suddenly in five patients and more gradually in one patient. For four patients an important psychological event, in addition to the TBI, was reported to have occurred around the time of the onset of stuttering. For one patient, the TBI was the result of an attempted suicide, one patient became a father during that time, another patient was going through a divorce, and the fourth patient was planning to resume work. For these latter two patients, the onset of stuttering occurred more than one month after the TBI.

The majority of the TBI patients ( $n=8$ ) were reported to have a stuttering frequency between 10% and 50%. As shown in Table 4, 10 of the 11 TBI patients showed repetitions of sounds and syllables, 6 showed prolongations and 7 were reported to have blocks during speech. Repetitions of whole words ( $n=4$ ), parts of the sentences ( $n=1$ ) and unnatural pauses ( $n=3$ ) were only reported in a minority of the patients. All of the TBI patients were reported to have disfluencies in word-initial positions, four showed evidence of word-medial disfluencies, and only two were reported to be disfluent on final word segments. In addition, eight patients were disfluent on content as well as function words, and nine were reported not to stutter on specific sounds. Reading adaptation was absent in five of the seven patients for whom this information was provided.

Concomitant behaviors associated with moments of disfluency, similar to those of the stroke group, were reported in seven patients. Emotional reactions to the stuttering, such as frustration, irritation, and fear were reported for the majority of the TBI patients ( $n=9$ ).

For five of the TBI patients, stuttering severity was influenced by either the speaking situation or the conversational partner. One patient was reported to stutter only during spontaneous speech, whereas the other 10 patients stuttered during multiple speech tasks.

### 3.2.5. Therapeutic intervention

Nine of the 11 TBI patients received speech and language therapy (see Table 5). For five of them the stuttering problem was addressed specifically during the therapy sessions by using a variety of therapy techniques such as slowing the speech rate, fluency shaping, stuttering modification, and cognitive stuttering therapy. Four patients were reported to have improved partially during therapy, whereas lack of improvement was reported in four other patients.

## 3.3. Neurogenic stuttering following neurodegenerative disease ( $n=9$ )

### 3.3.1. General information

Of the nine patients whose stuttering was caused by a neurodegenerative disease, eight were male and one was female. Their mean age at the time the questionnaire was completed, was 72 years, with a range from 56 years to 85 years. As shown in Table 1, medical history information was mostly unknown for patients with a neurodegenerative disease. For three patients it was reported that there was no evidence of medical problems prior to the onset of the neurodegenerative disease, while one patient previously suffered from memory problems.

Two patients were reported to stutter before the neurodegenerative disease occurred. For one of these patients, his family reported that he stuttered in the past only when under the influence of alcohol, but that following the onset of the disease his stuttering is present continuously. The other patient was reported to have recovered from a period of stuttering as a child, but his stuttering had reappeared as a result of the neurodegenerative disease. The majority of the patients were reported to be right-handed ( $n = 7$ ), one was left-handed and for one patient the hand preference was unknown.

### 3.3.2. Site of the lesion

Of the nine neurodegenerative patients, six were reported to be diagnosed with bilateral cortical and/or subcortical atrophy, while the lesion site was unknown for the remaining three patients (see Table 2).

### 3.3.3. Co-occurring disorders

Six patients were reported with psychological and/or neurological symptoms such as apathy or tremor. Co-occurring speech and language problems, other than the stuttering, were present in seven patients who showed a combination of either dysarthria, word finding problems, or other aphasic problems.

### 3.3.4. Disfluency-related characteristics

The mean age at stuttering onset for the neurodegenerative patients was 71 years (ranging from 56 years to 80 years). Stuttering onset (see Table 3) was reported to be gradual for four patients and one patient had a sudden onset of stuttering which occurred more than one month after the neurodegenerative disease was diagnosed.

While the reported stuttering frequency varied from 10% to more than 50%, two thirds of the patients ( $n = 6$ ) were reported to have a stuttering frequency of between 10% and 20%. As can be seen in Table 4, the majority of the patients repeated sounds ( $n = 7$ ), syllables ( $n = 9$ ), and words ( $n = 8$ ). Repetitions of parts of the sentence, prolongations, and blocks were reported for two patients, while none were reported with unnatural pauses. All patients were reported to demonstrate disfluencies in the initial position of a word and stuttered on content words as well as on function words. Two patients were said to be disfluent on medial sounds and only one on final sounds. Also, seven patients did not stutter on specific sounds. The four clinicians who commented on reading adaptation reported no such adaptation in their patients.

Secondary behaviors such as facial grimaces, associated movements of the limbs, and avoidance behaviors were reported for four patients and three patients were reported to react with frustration and/or irritation to the stuttering. Of those, two showed secondary behaviors as well as emotional reactions to the stuttering. Influence of the conversational partner or speaking situation on stuttering was absent in four patients with seven patients reported to stutter during multiple speech tasks.

### 3.3.5. Therapeutic intervention

As shown in Table 5, seven patients in the neurodegenerative group received speech and language therapy. Three received stuttering therapy using similar approaches as reported for the stroke and TBI groups. The four remaining subjects received speech and language therapy not specifically related to the stuttering symptoms. The speech of five patients was reported not to improve during the therapy, whereas one patient did show some improvement. This last patient received therapy aimed at slowing his speech rate.

## 3.4. Neurogenic stuttering following other disorders ( $n = 9$ )

### 3.4.1. General information

For nine patients stuttering was reported to start following a variety of other causes, including brain surgery ( $n = 3$ ), encephalitis ( $n = 2$ ), epilepsy ( $n = 2$ ), medication ( $n = 1$ ), and an unspecified cause ( $n = 1$ ). This last patient started to stutter following surgery of the thyroid gland but the exact cause of the stuttering was not determined. The 'other' group consisted of six women and three men and their age at the time of the survey ranged from 39 years to 77 years (mean 55 years). The two patients with epilepsy had prior epilepsy episodes before the onset of stuttering and one patient with a brain tumor had already had a prior brain tumor (see Table 1). None of the patients were reported to have other medical conditions. While information on the use of medication was not provided for most of the patients, one patient was reported to take anti-epileptic medication. One patient was said to have had speech and language problems prior

to the onset of the neurogenic stuttering. This patient previously cluttered and his disfluency problems had exacerbated following brain surgery. One other patient reported to have a nephew who temporarily had developmental stuttering. All patients but one were right-handed.

#### 3.4.2. Site of the lesion

As can be seen in Table 2, six of the nine patients had a lesion restricted to the left hemisphere, whereas the lesion site was not reported for the other three patients.

#### 3.4.3. Co-occurring disorders

Neurological, psychological or speech and language problems co-occurring with neurogenic stuttering were reported for all patients. Eight patients had one or more of the following speech and language problems: word finding problems, aphasia, dysarthria, apraxia of speech, and dysphonia.

#### 3.4.4. Disfluency-related characteristics

At the time the stuttering occurred, the age of our patients with ‘other’ causes ranged from 30 years to 77 years, with a mean age of 50 years. The stuttering onset was reported to be sudden in four patients and more gradual in four patients (Table 3). The stuttering occurred within 1 week for three patients (epilepsy, encephalitis, and unspecified cause), after 10 days for another patient (brain tumor) and after more than 1 month for two other patients (brain surgery and epilepsy). Two patients were reported to have significant psychological factors related to the stuttering onset: one patient who started to stutter following medication had just started chemotherapy, while the patient whose stuttering was reported to be associated with brain surgery had just lost his son.

The reported stuttering frequency ranged from less than 3% to 50%. As can be seen in Table 4, six of the nine patients repeated sounds and syllables, whereas four patients prolonged sounds. Word repetitions ( $n=2$ ) and repetitions of parts of the sentence ( $n=1$ ) were only reported for a minority of the patients. Blocks and unnatural pauses were reported for three of the patients. Eight patients had primarily word-initial disfluencies and medial disfluencies were reported for five of the patients. Only one patient, who started to stutter following brain surgery was reported to have word final disfluencies. Six patients were reported to stutter on content as well as function words. Disfluencies occurred on specific sounds (bilabials or consonant clusters) in three patients, while no sound-specific stuttering was reported for the other six patients. Results on reading adaptation were reported by four speech therapists and two of their patients, who both had brain surgery, showed adaptation during successive readings.

According to the therapists, five of the patients in the ‘other’ category showed evidence of secondary behaviors such as limb movements, facial grimaces, postponement behaviors, and avoidance behaviors associated with the stuttering. Seven of the nine patients were reported to be frustrated with their speech problems and this frustration was combined with feelings of fear in two patients. Situation or conversation partner-specific variations in the stuttering pattern were reported in three patients and five of the nine patients were reported to only stutter during spontaneous speech and not during other speech tasks such as reading, singing, and automatic speech.

#### 3.4.5. Therapeutic intervention

Seven patients received speech and language therapy (Table 5). For five of these patients, at least part of the treatment was specifically focused on their stuttering, using methods such as slowing the speech rate, fluency shaping, stuttering modification, cognitive therapy, voice therapy, and breathing therapy. For one patient, stuttering was reported to be unchanged as a result of treatment, while two were said to show partial recovery and two to have a full recovery.

## 4. Discussion

The purpose of the present study was to expand the available knowledge on neurogenic stuttering by way of surveying speech therapists in hospitals and rehabilitation centers in Flanders. While it is realized that a survey only provides indirect information on neurogenic stuttering, it also has a number of significant advantages. Firstly, a survey allows us to tap into the clinical observations of a large and expert group of speech clinicians working not only with patients with neurogenic stuttering, but also with a broader population of patients with neurogenic communication disorders. Such

a broad clinical perspective provides an important context for differential diagnosis of neurogenic speech disfluencies. Secondly, a survey allows us to obtain detailed information on a relatively large group of patients with neurogenic stuttering and with a wide variety of etiological variables, again contributing to the theoretical and clinical knowledge regarding differential diagnosis.

#### 4.1. Incidence

One of the important issues that remain unanswered is the incidence of neurogenic stuttering in various disorder populations. While the survey approach used in the current study does not allow solid conclusions on the incidence of neurogenic stuttering, some information can be deduced based on the response rate. Of the 164 therapists working with patients with a wide variety of neurological disorders, 44 completed surveys on a total of 62 patients. As such, one fourth (27%) of the speech therapists working with multiple neurological disorders indicated to have worked with one or more patients with neurogenic stuttering during the last five years. This is probably a very conservative estimate as not all clinicians who had observed neurogenic stuttering in their clinical practice completed a survey. In addition, three other therapists responded to an announcement for the study and each of them completed one questionnaire. Based on this result and a return rate of 36% reported by Stewart and Rowley (1996), it is clear that neurogenic stuttering, while not a frequently occurring disorder, is nonetheless a not uncommon disorder in the clinical practice of speech therapists, especially those working with neurological disorders. Nevertheless, it is important that more exact data on the incidence of neurogenic stuttering becomes available. This will allow clinicians and researchers to better identify these patients and provide appropriate clinical intervention. A systematic study aimed at determining incidence of neurogenic stuttering in stroke patients is currently underway in our laboratory.

#### 4.2. Etiology

Of the 58 questionnaires that were included in our analysis, stroke patients were most likely to be diagnosed with neurogenic stuttering, comprising approximately half of the patients on which data were collected. This result is comparable to the finding of Helm, Butler, and Benson's (1978) study that neurogenic stuttering occurred following a stroke in 6 of their 10 patients. In addition to the stroke patients, 11 patients in our survey started to stutter following a TBI, which leads to 69% of the causes being either a stroke or a TBI. Market et al. (1990) and Jokel and De Nil (2003) found very similar results (75% and 71%, respectively) when the stroke and trauma etiologies in their studies are combined. Neurogenic stuttering following a neurodegenerative disease was diagnosed in 16% of our cases, similar to the 18% reported by Jokel and De Nil (2003). In contrast, Market et al. (1990) reported on only one patient (1.2%) with a neurodegenerative disease among their patients. This latter discrepancy may be explained by differences in the selection of the clinicians that were contacted in the respective studies. In addition to these prevalent disorders, it is clear from our study, as well as from other investigations (Helm-Estabrooks, 1986; McClean & McLean, 1985; Meghji, 1994), that neurogenic stuttering may occur from a wide variety of disorders that interfere with normal neurological functions, such as epilepsy, brain surgery, encephalitis, or use of medication.

It has been suggested that neurogenic stuttering can be associated with lesions in almost every region of the brain (De Nil, Rochon, & Jokel, *in press*). This certainly appeared to be the case in our patients, although the lesion sites varied across the etiology groups. The lesions were left lateralized in more than half of the patients in the stroke and 'other' group, while they were mostly bilateral in the neurodegenerative group and frequently either bilateral or left lateralized in the trauma group. These results indicate that left hemisphere involvement is frequently present in neurogenic stuttering. For stroke patients in general, a left cerebral hemisphere injury is also reported more frequently than a right cerebral hemisphere injury (Foerch et al., 2005; Naess, Waje-Andreassen, Thomassen, & Myhr, 2006). However, Foerch et al. reported left hemispheric events in 56% and right hemispheric events in 44% of their patients, while in our survey 17 stroke subjects were reported with a lesion restricted to the left hemisphere, whereas only 5 with a lesion to the right hemisphere. This indicates that the reports of left hemispheric lesions in our stroke patients with neurogenic stuttering are higher than those reported for the stroke population in general. Also, while our data failed to show a clear association between lesion lateralization and neurogenic stuttering characteristics, it cannot be excluded that such a relationship may emerge when more fine-tuned and direct imaging information on lesion site in patients is available.

The speech disfluencies reported for the patients across all four etiological groups in our survey consisted predominantly of repetitions of sounds and syllables. While word repetitions were reported in almost 90% of the patients with

a neurodegenerative disease, this type of speech disfluency was mostly absent in the other etiology groups. Speech blocks seemed characteristic for the stroke as well as for the trauma patients but not for the other two etiology groups. A review of the literature (De Nil et al., *in press*) suggested that stroke patients often repeat syllables and words, while prolongations and blocks are not that frequently reported. In contrast, in our survey, repetitions of sounds and syllables were equally likely to be observed for the stroke patients as were articulatory blocks. Word repetitions and prolongations, on the other hand, occurred to a lesser extent in the stroke group. Jokel et al. (2007) have reported that the nature of disfluencies in stroke patients may differ depending on the complexity of the speech sample, with more complex language material triggering more word repetitions and other disfluencies, characteristic of more language-based disfluencies, while stuttering-like disfluencies are observed more readily in less complex speech tasks. In addition, reports that patients with a neurodegenerative disease often freeze, block or prolong sounds during their speech (De Nil et al., *in press*) do not appear to be consistent with the findings in our survey that repetitions were the predominantly disfluency type in these patients. Finally, the frequent repetitions, prolongations and blocks of the head injury patients in our survey were comparable to the disfluency types of the TBI patients reported in the literature (De Nil et al., *in press*).

Some of the differential characteristics for stroke-induced stuttering, trauma-induced stuttering, and stuttering following extrapyramidal disease identified by Helm-Estabrooks (1986) are supported by our results. Examples of these characteristics are: stuttering always present on initial phonemes in all the etiology groups, possible presence of aphasia in stroke and TBI patients, and low frequency of adaptation in TBI patients. In contrast, other characteristics observed in our survey, such as reports of secondary motor signs in more than half of the stroke, trauma and 'other' group, possible presence of aphasia in patients with a neurodegenerative disease and presence of adaptation in some stroke patients do not seem to support these differential diagnosis guidelines. Taking our results and those from previous studies into account, we believe that there is insufficient evidence at the present time to clearly define differential stuttering characteristics for different stuttering etiologies. To a large extent, this is due to the fact that available descriptions of neurogenic stuttering mostly consist of single case studies and represent a very heterogeneous patient group. Indeed, it is not always clear to what extent these case reports represent the general population, rather than atypical cases which have attracted the attention of the clinician or researcher. We believe that there is an urgent need to obtain more representative and reliable results by systematically collecting and analyzing data on larger, etiologically more homogeneous groups of patients with neurogenic stuttering. A few investigators have already reported on such studies (Ludlow et al., 1987; Jokel et al., 2007), but more studies are needed.

#### 4.3. Differential diagnosis with other communication problems

When assessing neurogenic stuttering, other co-occurring disorders might often be present. De Nil et al. (*in press*) suggested that the presence of other communication problems might hamper the recognition of neurogenic stuttering. The results from our survey showed that aphasic, dysarthric and specific word finding problems were the most likely concomitant communication problems. In our survey sample, apraxia of speech and dysphonia were diagnosed only in a minority of the patients and, consequently, might have less influence when assessing neurogenic stuttering. This observation is in contrast with the reports of Shtremel (1963) and Caplan (1972), both cited in Rosenbek (1980), that neurogenic stuttering is a form of apraxia of speech. Interestingly, the majority of our patients (67%) were reported to have multiple co-occurring speech and/or language disorders, suggesting that acquired stuttering is most likely to occur in patients showing a complex communication and neurological deficiency. This in turn might point to the role of multiple or large lesion sites in the genesis of neurogenic stuttering.

#### 4.4. Differential diagnosis with psychogenic stuttering

Half of our patients were reported to experience an onset of neurogenic stuttering within one month after the onset of the underlying neurological condition, this result is comparable with the reported onset in the two previous survey studies (Market et al., 1990; Stewart & Rowley, 1996). In addition, the onset of stuttering was rather sudden in half of our patient sample. Although the speech therapists were explicitly asked only to complete questionnaires on subjects with neurogenic stuttering, if stuttering was first observed more than one month after the neurological injury, it is difficult to be certain about a clear causal link between the initial neurological event and the onset of stuttering. This is especially the case if additional psychological factors were reported at the time of stuttering onset. For instance, two patients in our survey were reported to start stuttering more than one month after their TBI. At the same time, they experienced potentially

significant psychological stress factors (e.g., divorce) which could have contributed to the onset of acquired stuttering. Five patients for whom stuttering started within one-month self-reported psychological events which they thought could have been related to the stuttering onset. It is not always clear whether these reported psychological events were indeed traumatic for the patients (e.g., closure of a swimming pool) and could have triggered the occurrence of stuttering. Nevertheless, we have to keep in mind that events that may appear quite innocent to an external observer can have significant psychological impact for a patient, which complicates the differential diagnosis between a neurogenic and psychogenic onset of stuttering. In addition, it cannot be excluded that in some patients it is the interaction between both neurological and psychological factors that lead to the onset of stuttering. Clearly, more research is needed to investigate these important but complex issues in greater details in order to provide clinicians with appropriate diagnostic and therapeutic guidelines.

#### 4.5. Differential diagnosis with developmental stuttering

Another question that needs to be addressed is to what extent neurogenic and developmental stuttering represent two distinct speech fluency disorders and whether they can reliably be differentiated.

In our survey, 7 of the 58 patients were reported to have a history of stuttering or cluttering prior to occurrence of the neurological insult. The neurological event triggered the re-occurrence of childhood stuttering in two of those patients and worsened the previously existing stuttering of five patients. These reports confirm an earlier observation by Helm-Estabrooks (1999, p. 255) that “stuttering may occur, worsen or recur in the presence of a neurological dysfunction”. One could argue that patients with a prior history of stuttering should not be included in a study of neurogenic stuttering. However, the fact that a significant change in the patient’s stuttering was reported by either the patient him- or herself, or by the clinician, may provide important information regarding the differential characteristics of acquired stuttering and developmental stuttering. For instance, the speech and secondary behaviors of our seven patients with a prior history of stuttering or cluttering were highly comparable to those of patients without a reported history of developmental stuttering. Of the seven patients, six (86%) repeated sounds and syllables, four (57%) blocked, and three (43%) showed prolongations. Of our total group of 58 patients with neurogenic stuttering, more than 40 (69%) showed repetitions of sounds and syllables, 30 (52%) blocked and 21 (36%) prolonged sounds. Only three (43%) of the patients with previous stuttering were reported to have secondary behaviors and four (57%) were reported to react emotionally to their stuttering. In the total group of patients, these characteristics were reported for 32 (55%) and 37 (64%) patients, respectively. These observations suggest that neurogenic and developmental stuttering may not differ markedly in at least some of their speech and non-speech characteristics.

Of the 27 patients for whom information on speech and language problems in the family were reported, 4 (15%) indicated the presence of stuttering in a family member, whereas 22 (81%) reported the absence of speech and language problems in their family. While this incidence is higher than the 5% expected in the general population (Bloodstein, 1995), it is significantly lower than the 71% previously reported in persons with developmental stuttering (Ambrose, Yairi, & Cox, 1993). While intriguing, the observation of a higher familial incidence in patients with neurogenic stuttering should be interpreted with caution since information on familial speech and language problems was not available for all of our patients, and the reliability and validity of these reports could not be assessed directly.

Some authors have suggested differentiating characteristics between neurogenic and developmental stuttering. One of these characteristics concerns the loci of disfluencies (initial, medial, and/or final) (Helm-Estabrooks, 1999). Based on our data, it appears that clinicians observed that stuttering occurred primarily on initial sounds or word segments. In addition, 22 of the 40 patients for whom this information was provided (55%), stuttered on medial sounds but only 6 patients also stuttered on final sounds. Thus, stuttering was not restricted to initial syllables in more than half of the patients for whom this information was reported. This result is similar to that reported in the literature, with 61% reported by Stewart and Rowley (1996), 80% by Market et al. (1990), and 75% by Ringo and Dietrich (1995). For our four etiology groups, medial disfluencies were more frequently reported than disfluencies on final word segments which occurred rather infrequently. In this respect, it is important to point out that adults with developmental stuttering also have a significant portion of their disfluencies on within-word locations, while disfluencies on final word segments are rare but certainly not absent (Bloodstein, 1995; Natke, Sandrieser, van Ark, Pietrowsky, & Kalveram, 2004). As a result it is doubtful that this characteristic indeed can be used reliably as a truly differentiating the acquired from the developmental disorder.

Another reported differential characteristic between neurogenic and developmental stuttering is that disfluencies occur on grammatical words nearly as frequently as on substantive words (Helm-Estabrooks, 1999). In our survey, almost all the patients in the four etiology groups were reported to be disfluent on content words as well as on function words. Ringo and Dietrich (1995) reported this characteristic in 90% of the cases reviewed in their study, which is similar to the 79% found by Market et al. (1990) and the results in our study. In contrast, Stewart and Rowley (1996) found this characteristic in only half of their patients.

With regard to reading adaptation, approximately half of the stroke and ‘other’ patients from whom such data was reported, were reported to show adaptation, while this was only the case in two of the seven TBI patients. None of the patients with a neurodegenerative disease were reported to show adaptation. In total, 61% of the patients for whom data on adaptation was reported, did not show adaptation during successive reading of a text, while 39% did. Similar results were reported previously by Stewart and Rowley (1996) who found absence of adaptation in 56% of their subjects and Ringo and Dietrich (1995) who reported 60% of the patients not showing adaptation. In other words, the absence of adaptation again does not seem to be a reliable differential criterion.

A fourth differential feature concerns the absence of secondary symptoms associated with moments of stuttering (Helm-Estabrooks, 1999). Approximately half of our patients in the four etiology groups showed secondary behaviors. Absence of these types of behaviors was reported for 40% of our patients, which places this result in between those found by the two previous survey studies. Market et al. (1990) reported a higher proportion of patients in who secondary behaviors were absent (68%), while Stewart and Rowley (1996) reported a lower proportion (22%). Again, the use of secondary behaviors as a differential characteristic between neurogenic and developmental seems uncertain, especially with patients who have a longer history of neurogenic stuttering (De Nil et al., *in press*).

In addition, the majority of the patients in our survey (64%) were reported to react emotionally to their stuttering, although this seems to be less the case in patients with a neurodegenerative disease. Only one third of these patients were reported to react emotionally. Irritation and frustration were frequently reported, whereas only 10% of the patients were reported to be anxious. This provides support for the criterion that the speaker may be annoyed by his disfluencies, but does not appear anxious (Helm-Estabrooks, 1999). Similar results were reported by Market et al. (1990) and Ringo and Dietrich (1995). In contrast, only 17% of the patients included in the study by Stewart and Rowley (1996) showed such reactions.

Influence of the speaking situation or conversational partner was reported for half of the patients in the survey. Patients with an underlying neurodegenerative disorder did not seem as affected by such influences as were the other three etiology groups. In addition, 40 of our 58 patients (69%) were reported to stutter during multiple speech tasks, which supports that stuttering occurs relatively consistently across various types of speech tasks (Helm-Estabrooks, 1999).

#### 4.6. Treatment

A large majority of the patients in our survey received speech and language therapy. If specific stuttering therapy was provided, a combination of multiple therapy techniques was usually reported, as was the case in the two previous survey studies (Market et al., 1990; Stewart & Rowley, 1996). The conclusion by De Nil et al. (*in press*) that “many clinicians start with traditional fluency therapy and complement or replace it with other techniques if necessary”, can be generalized to the Flemish clinicians who participated in the present study. Approximately half of our patients were reported to improve during the therapy, which supports the positive outcome measures found by Market et al. (1990) and Stewart and Rowley (1996), although it is not clear to what extent these improvements were therapy-specific or, in some patients, may have reflected natural neurological recovery.

### 5. Conclusion

Some interesting trends and observations emerged from the clinician survey reported here. Before drawing some general conclusions, it is appropriate to outline briefly some cautions needed in the interpretation of the data. Our data only report characteristics of patients who were tested or treated by a speech therapist. Not all patients with potential neurogenic stuttering are necessarily seen by or referred to a clinician. It is possible, for instance that only the more severe patients were referred. As such, our results do not necessarily apply to those patients who were not seen by a clinician. Secondly, our results also only reflect the perceptions and observations of clinicians, often obtained as a result

of longer term and variable experiences with the patients. They may not always correspond to objectively observed behaviors in the patients at the time of the study. On the other hand, the observations reported by the clinicians may reflect a more typical behavior in these patients, rather than a single snapshot of inherently variable patient behavior. Thirdly, the available data did not allow us to differentiate between transient and persistent stutterers in each etiology group. This could possibly influence the results since differences may exist between these two sub-populations (Helm et al., 1978). Fourthly, given the presence of multiple neurological events in some of the patients reported here, it is not possible to evaluate accurately whether previous neurological conditions and/or an interaction between these conditions may have influenced the stuttering.

The results of the present study were presented separately for four different etiology groups. Our results suggested the presence of possible differences between these groups (e.g., lesion site, influencing factors), while other characteristics were comparable for all etiologies. In addition, no relationship was found between localization or lateralization of the brain lesion and speech and non-speech characteristics. Our results provided support for a number of differential features of neurogenic stuttering, relative to developmental stuttering, such as stuttering on grammatical words as well as on substantive words, annoyance by the speaker but no anxiety and stuttering across various speech types. Other features (e.g., absence of secondary characteristics) did not appear to occur frequently in our patient population. Furthermore, the similarities of our results on Flemish patients with those of previous studies in largely English-speaking populations provide support that most of the characteristics of neurogenic stuttering do not differ for patients with various linguistic backgrounds. However, it is clear that none of the discussed criteria applied to each individual patient. The use of these criteria is therefore only recommended as guidelines instead of strict criteria when assessing neurogenic stuttering. It is important to take all the relevant factors concerning the patient's history, onset of the neurological disease and stuttering, lesion site and speech and non-speech characteristics into account before making a diagnosis. Further systematic collection of data is necessary to shed new light on some aspects of neurogenic stuttering for research as well as for clinical purposes. For instance, detailed information on the incidence of neurogenic stuttering is still lacking as is data concerning the differential diagnosis of neurogenic stuttering and other speech and language disorders. Furthermore, it still remains a question whether neurogenic stuttering is a single diagnostic entity or merely a speech fluency symptom that occurs following various lesions and pathologies but should not be characterized as a separate disorder. We currently are in the process of exploring these and other issues using epidemiological, behavioral, and imaging investigative methodologies.

## CONTINUING EDUCATION

### A clinician survey of speech and non-speech characteristics of neurogenic stuttering

#### QUESTIONS

1. Most of what we know about neurogenic stuttering has come from:
  - a. clinician survey studies
  - b. hypothesis-driven experimental research
  - c. a posteriori analysis of observations from larger groups of patients with neurogenic stuttering
  - d. individual case studies
  - e. longitudinal follow-up studies of patients with neurogenic stuttering
2. Our survey showed that the most prevalent cause of neurogenic stuttering was:
  - a. stroke
  - b. traumatic brain injury
  - c. neurodegenerative disease
  - d. epilepsy
  - e. brain tumor
3. In the group of stroke patients, the onset of the neurogenic stuttering was reported as occurring most frequently:
  - a. suddenly and within one week after the stroke
  - b. suddenly and more than one week after the stroke
  - c. gradually and within one week after the stroke

- d. gradually and more than one week after the stroke
  - e. this information was not reported
4. The results of our survey showed that the four etiology groups did not differentiate significantly on the following aspect:
    - a. external factors influencing neurogenic stuttering
    - b. localization of the neurological injury
    - c. linguistic loci of disfluencies
    - d. patients' emotional reactions to their stuttering
    - e. adaptation
  5. The clinicians in our survey were most likely to use the following treatment approach:
    - a. delayed auditory feedback
    - b. cognitive restructuring
    - c. indirect treatment involving relatives and partners
    - d. breathing therapy
    - e. a variety of stuttering modification and fluency shaping approaches

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## Appendix A

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### Survey Neurogenic Stuttering

This questionnaire was completed by: ..... on: .. / .. / ..

#### **Patient's personal and medical information:**

- gender M / F
- date of birth .. / .. / ..
- previous speech and language problems (developmental stuttering, language delay, etc.)  
yes: ..... / no / unknown  
If the patient already stuttered prior to the injury, did the stuttering worsen / improve /  
reoccur / remain unchanged.
- familial speech and language problems  
yes: ..... / no / unknown
- medical history
  - previous neurological conditions  
yes: ..... / no / unknown

- medication: yes: ..... / no / unknown
- other (migraine, epilepsy, etc.) yes: ..... / no / unknown

**Information with regard to the onset of neurogenic stuttering:**

- Date and/or chronological age at the first occurrence of neurogenic stuttering  
 .. / .. / ..... years
- What was the time interval between the occurrence of the brain lesion and the onset of neurogenic stuttering? ..... days ..... months
- Was the onset of stuttering sudden (it evolved or worsened over a period of < 2 weeks) or gradual (over a period of > 2 weeks)? .....
- Were there any significant environmental events related to the onset of the stuttering (e.g., death of relative, divorce, marriage)  
 yes: ..... / no / unknown

**What is the cause of the neurogenic stuttering?**

- trauma: .....
- stroke
- degenerative disease (Parkinson, Multiple Sclerosis, Alzheimer, etc.): .....
- tumor
- surgery: .....
- medication:.....
- other: .....

**Where is the lesion localized?**

- right hemisphere
- left hemisphere
- subcortical
- cerebellar
- frontal
- parietal
- temporal
- occipital
- medial

Specify if possible: .....  
 .....  
 .....

The patient is left handed / right handed / ambidextrous.

**Does the stuttering co-occur with other disorders?**

- aphasia (Broca’s / Wernicke’s / anomic / conduction / global)
- word finding problems
- dysarthria
- apraxia of speech / dyspraxia
- dysphonia

- other neurological symptoms (hemiparesis, tremor, etc.)  
.....
- psychological disorders (depression, fear, etc.)  
.....

**What are the characteristics of the neurogenic stuttering?**

- repetitions of:
  - sound
  - syllable
  - word
  - parts of utterance
- prolongations
- blocks:
  - audible
  - non-audible
- pauses:
  - audible
  - non-audible
- the stuttering occurs on:
  - initial sounds: never / sometimes / often
  - medial sounds: never / sometimes / often
  - final sounds: never / sometimes / often
  - function words: never / sometimes / often
  - content words: never / sometimes / often
- is an adaptation effect present (less disfluencies with repeated reading of the same text):  
yes / no / unknown
- the following characteristics are present during stuttering:
  - irritation
  - fear
  - frustration
  - other: .....
- the stuttering is observable during:
  - automatic speech (counting, days of the week, etc.)
  - reading
  - singing
  - spontaneous speech
- the frequency of the stuttering during spontaneous speech is:
  - very rare (< 3 %)
  - person specific
  - sporadic (3-10 %)
  - situation specific
  - once per utterance (10-20 %)
  - little variable
  - multiple instances per utterance (20-50 %)
  - almost on each word (> 50 %)
- the stuttering occurs more frequently on specific sounds; these are: .....
- the following secondary behaviors are present:
  - associated movements of the limbs: .....
  - facial grimaces: .....
  - avoidance behaviors (not answering, using other words, whispering, etc.)
  - postponement behaviors (throat clearing prior to speaking, let others speak first, etc.)
  - other: .....

**Therapy**

Which therapy methods were used?

- no therapy
- decreasing speech rate with or without external devices such as DAF, pacing board, etc.  
specify: .....
- medication: .....
- breathing therapy
- stuttering modification
- fluency shaping
- cognitive therapy
- other: .....

What is the result of therapy?

- increase of disfluency
- no change
- improvement of fluency
- complete recovery
- unknown

How much therapy is already given? .....

If the patient’s fluency improved, do you think that other factors may have played a role as well (e.g., spontaneous recovery)?    yes / no

**Do you have some remarks, questions, or suggestions?**

.....  
.....  
.....

*Thank you very much for your cooperation!*

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