12 Acquired Stuttering

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Introduction

Stuttering is a speech fluency disorder characterised by the occurrence of *stuttering dysfluencies*. These dysfluencies are the core behaviours of stuttering and include part-word repetitions, single syllable word repetitions, prolongations and blocks. Stuttering can also be associated with secondary behaviours and negative affective and cognitive thoughts. In the literature, the term stuttering is often used as a synonym for developmental stuttering. However, not all stuttering is developmental in origin.

Stuttering can also have an onset following cerebrovascular injuries, traumatic brain injuries, neurodegenerative conditions and emotional traumas. This has been described in the literature as late-onset ~~stuttering, adult-onset stuttering, stuttering associated with acquired neurogenic disorders,~~ among other terms (De Nil et al., 2017). For the purpose of this chapter, we will use the wording *acquired stuttering* to differentiate it from *developmental stuttering*. This terminology is preferred as acquired stuttering can also have an onset in childhood due to a neurological or psychological trauma, although such conditions are more likely to appear later in life (Theys & De Nil, in press).

The International Classification of Disease has categorised the different types of stuttering (World Health Organization, 2018; see Table 12.1). In this chapter, we will refer to childhood onset fluency disorder (F80.81) as *developmental stuttering*, to stuttering following neurological events (I69) or disorders (R47.82) as *acquired neurogenic stuttering* and to stuttering following emotional trauma (F98.5) as *acquired functional stuttering*. The term functional has gradually replaced the term psychogenic, as it allows focusing on the behavioural symptoms that are present, rather than on the presumed underlying aetiology of the speech problem (Edwards et al., 2014). Further, it should be noted that stuttering can also be *malingered*, as opposed to *acquired*. Malingering is the feigning of a condition, typically for financial or some other type of gain (Bass & Halligan, 2014) and is classified in the ICF as Z76.5.

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Table 12.1 International Classification of Disease classifications for stuttering

Code	*Description*	*Inclusion*
F80.81	childhood onset fluency disorder	• stuttering; cluttering • childhood onset
F98.5	adult onset fluency disorder	• stuttering • adult onset • other emotional/psychogenic
I69	fluency disorder (stuttering) following cerebrovascular disease	• stuttering, dysfluency • stroke, traumatic brain injury, vascular/ circulatory disease
R47.82	fluency disorder in conditions classified elsewhere	• stuttering, dysfluency • Parkinson's, Tourette's

Current State of the Art

Acquired stuttering can be neurogenic or functional in origin. Our knowledge of these acquired types of stuttering has significantly increased – and changed – over the past 20 years. We will provide a brief overview of the current state of knowledge here, but readers are referred elsewhere for more detailed recent overviews (De Nil et al., 2017; Duffy, 2020; Theys & De Nil, in press).

Approximately half of the cases of acquired neurogenic stuttering are caused by stroke. This is followed by traumatic brain injuries and neurodegenerative diseases such as Parkinson's and Alzheimer's disease (Lundgren et al., 2010; Theys et al., 2008). The onset of acquired neurogenic stuttering has also been linked with many other conditions that may influence brain functioning, including deep brain stimulation and use of medication (Brady, 1998; Picillo et al., 2017). Most people with acquired neurogenic stuttering will not have a history of developmental stuttering, but it is possible that pre-existing stuttering may re-occur following neurological conditions (Helm-Estabrooks, 1999).

Detailed prevalence data is sparse, but one study showed that 5% of 319 stroke patients presented with >3% stuttering dysfluencies in the acute phase following stroke. While some recovered from their stuttering, follow-up after 6 months showed that stuttering persisted in eight of 14 patients who were re-assessed (Theys et al., 2011). Studies on Parkinson's disease suggest that the prevalence ranges from 4–57% (Hartelius, 2015; Whitfield et al., 2018), with the large variability possibly due to differences in disease progression.

Acquired functional stuttering can occur as a psychological reaction to stress or trauma. It often occurs without evidence of an underlying neurological disease, although 20 of 69 patients with functional stuttering in Baumgartner and Duffy's (1997) study had evidence of neurological disease. While precise prevalence data is again not available, stuttering occurred in 53% of 30 patients with functional speech and voice disorders (Baizabal-Carvallo & Jankovic, 2015).

Contrary to previous beliefs, these prevalence numbers indicate that acquired stuttering is not rare. However, clients with acquired stuttering may not always receive referrals for speech therapy support for their fluency problems.

Traditionally, a number of features were suggested to differentiate acquired neurogenic stuttering from functional and developmental stuttering in adults (i.e., consistency of stuttering across speech tasks, dysfluencies not restricted to content words or word-initial positions and absence of anxiety, secondary symptoms and adaptation effect, Helm-Estabrooks, 1999). However, use of these criteria may lead to underdiagnosis as these features do not apply to all – or some even to most – clients with acquired neurogenic stuttering (Market et al., 1990; Stewart & Rowley, 1996; Theys et al., 2008). Recent evidence shows that stuttering characteristics vary depending on the underlying aetiology of acquired neurogenic stuttering, and it is therefore important not to overgeneralise findings across aetiologies. For example, dysfluencies in neurogenic stuttering following stroke occur almost always in initial position (De Nil et al., 2017) and ~~there is no difference in~~ location of within-utterance dysfluencies ~~between acquired neurogenic and~~ developmental stuttering (Max et al., 2019). Reading adaptation occurs in about half of the clients with stuttering following stroke and TBI (De Nil et al., 2017; Jokel et al., 2007) and in at least two thirds of people with stuttering following Parkinson's disease (Whitfield et al., 2018). Most reports across all acquired neurogenic stuttering aetiologies indicate increased fluency when singing. Approximately half of the cases in the literature on neurogenic stuttering following stroke report presence of secondary behaviours (e.g., facial grimacing, fist clenching), and more than two thirds show negative speech-associated emotions (Theys & De Nil, in press). A similarly high occurrence of secondary behaviours and emotions has been reported following traumatic brain injury, but these behaviours seem to occur less frequently in clients with an onset of stuttering following neurodegenerative conditions (Theys et al., 2008).

Acquired functional stuttering may present with characteristics similar to those seen in developmental and acquired neurogenic stuttering. However, the speech characteristics are often described as atypical. Atypical may refer to the pattern of dysfluencies (e.g., very consistent on each syllable), the location of the dysfluencies (e.g., word-initial as well as word-final), the variation in dysfluencies (e.g., change in dysfluency types throughout a conversation) and suggestibility (e.g., change in stuttering severity consistent with clinician suggestion of task difficulty). People with acquired functional stuttering seem to adapt less, with one in nine clients showing reading adaptation in Baumgartner and Duffy's (1997) study. The most striking feature of acquired functional stuttering is the ability to achieve a very rapid (1–2 sessions) improvement in fluency (in 70% of cases) (Baumgartner & Duffy, 1997). However, as with acquired neurogenic stuttering, the stuttering characteristics can vary widely across individuals ~~with acquired functional stuttering~~.

Differential Diagnosis

The information presented earlier shows that – besides the core stuttering dysfluencies – there are no defining characteristics that apply to each person with acquired stuttering. The diagnostic process will therefore need to be tailored to each individual client, taking underlying conditions and co-occurring speech, language and cognitive problems into account. If the stuttering occurs later in life, in a person who previously spoke fluently, the acquired as opposed to developmental onset of stuttering is usually clear. More important is the differentiation between neurogenic and functional aetiologies of acquired stuttering as these will have direct implications for treatment. Another important differentiation to make is that between acquired neurogenic stuttering following events such as stroke and traumatic brain injury, where an improvement in symptoms can be expected, and stuttering associated with neurodegenerative conditions, where a progressive worsening of the stuttering needs to be anticipated.

Many clients with acquired stuttering will present with a complex combination of symptoms. In addition to a comprehensive fluency assessment, assessment for other communication and cognitive difficulties may be necessary. All stuttering/fluency assessments should begin with collection of accurate case history information, an assessment and description of speech characteristics across ~~varying~~ tasks of length, complexity and settings and assessment of attitudes about stuttering. This needs to be followed by trialling of potential therapeutic interventions, such as fluency-inducing tasks (e.g., prolonged speech, singing, pacing, reading adaptation, delayed auditory feedback). This type of complete evaluation can help in both differential diagnosis and planning of intervention. An example of a comprehensive fluency profile is provided in the online resources.

Treatment Options

If the assessment results uncover that stuttering is perceived as a significant problem by the client, specific treatment for the stuttering needs to be provided. This does not always happen, as the cases described in sections ~~5 and 6~~ were initially not referred for stuttering therapy, despite stuttering being their most prominent and disabling communication problem.

Before starting stuttering therapy, it is important to note that some clients with acquired stuttering following stroke may recover spontaneously, and sometimes more pressing medical issues need to be prioritised. Due to the often-complex presentation of problems, a multidisciplinary approach may be needed. In some cases, adjusting medication (Brady, 1998) or deep brain stimulation parameters (Picillo et al., 2017) may be sufficient to alleviate the stuttering. Other important considerations include quality of life and inclusion of family members in decision-making.

There are no evidence-based speech treatments that have been developed specifically for acquired neurogenic stuttering. However, our clinical experiences indicate that an individualised approach is necessary and a variety of speech therapy approaches have been reported to be successful. These include fluency shaping, stuttering modification, rhythmic speech or slowing down the speech rate. Success with altered auditory feedback and pacing strategies has also been described (Theys & De Nil, in press).

For people with acquired functional stuttering, discussing the absence of a neurological problem that may hinder progress is often helpful, as is directly addressing the underlying psychological problem (e.g., with psychological counselling or using cognitive strategies) and using fluency-inducing techniques to help clients 'find' their fluent speech again. In clinical settings, this is accomplished through accurate assessment of all information gathered and reported, followed by a frank and honest debriefing with the client and their family. As described earlier, rapid recovery occurs often, and clients may need support in explaining such a rapid change to family and others in their environment (Duffy, 2020).

Many of these considerations are reflected in the two cases presented here.

Case # 1

MB was presented to one of the authors (JT) at a university speech and hearing clinic. MB initially came to the clinic with reported symptoms of aphasia. She was subsequently referred for a fluency evaluation during the course of her initial visit.

MB was in her early 50's when she had a sudden onset of stuttering. The stuttering started two months before the initial evaluation, after a spell of dizziness and weakness, with brief loss of consciousness. When she awoke, she could not speak at first. When she finally could speak, she presented with severe stuttering. There was no stuttering in her history prior to this point in time and an MRI revealed no new damage. She previously had a diagnosis of breast cancer at age 40 and had two subsequent bouts with cancer. Four months prior to her stuttering onset, she received chemotherapy and carried a post-therapy diagnosis of chemo-induced neuropathy.

MB's fluency evaluation (using the format in Appendix 12.A) revealed several key findings. These included: 10% stuttered syllables during word tasks and 13% during conversation. Stuttering types included part-word repetitions and blocks that ranged between 1–3 seconds in duration. Stuttering dysfluencies occurred on content and function words and in phrase initial and phrase medial points. No secondary behaviours were noted. Fluency-inducing tasks including prolonged speech, mouthed speech, whispering and singing resulted in no change in stuttering. She was administered the Overall Assessment of the Speaker's Experience of Stuttering (Yaruss & Quesal, 2006) and showed a mild, but significant reaction to her stuttering. One item of note was her substituting of words when she feared stuttering. During the

evaluation (and during subsequent early therapy sessions) she often cried when she stuttered. In addition, she showed mild word finding difficulties. She scored in the low, but normal range of the Boston Naming Test (Goodglass et al., 1983). A sample of her speech at this time is provided in Audio 12.1.

MB was enrolled in therapy. Fluency enhancing ~~therapies~~ (prolonged speech, increased pauses) were the dominant philosophy during the initial three months of therapy with little success and significant frustration. This appears in Audio 12.2. After a thorough case review, it was decided that there was a significant emotional component attached to her stuttering, and her therapy was modified to more of a stuttering modification approach where her therapy emphasised education about stuttering, decreasing word substitutions and building communication confidence. At this point in time, she was made aware that her diagnosis of stuttering was modified from *neurogenic* to "*psychogenic*". Although initially upset, MB was counselled to understand that she could indeed control her speech and that it was not due to neurogenic limitations. She embraced this view with counselling from the clinician and was dismissed from therapy two months after the shift in diagnosis and treatment paradigm. At the time of dismissal, MB demonstrated less than 1% stuttering dysfluencies at word tasks and 2% during conversation tasks (Audio 12.3). Her OASES score was very mild.

In summary, this case shows how – despite the diagnosis of chemotherapy-induced neuropathy – MB fit the updated criteria for acquired functional stuttering. Proper differential diagnosis allowed for more of a counselling and acceptance method of intervention with a successful outcome within a few months ~~of intervention~~.

Case # 2

Similar to Case #1, DB was referred to a university speech and hearing clinic to participate in aphasia groups. However, she decided not to attend these groups due to embarrassment about her speech problems and was referred to one of the authors (CT) for a fluency assessment.

DB was 79-years-old when she had a left occipital infarct. This was followed by an additional left total anterior circulation stroke one month later and multiple post-stroke epileptic seizures. She spoke fluently before these events, as can be seen in Video 12.1. Following the strokes and seizures, DB was diagnosed with receptive and expressive aphasia, apraxia of speech, speech dysfluencies and cognitive problems. Our initial stuttering assessment took place 5 months following onset of the seizures.

During two baseline assessment sessions, DB presented with 41% and 39% stuttered syllables during spontaneous speech, respectively. Her stuttering dysfluencies consisted of repetitions of sounds, syllables, monosyllabic words and blocks. An example of her pre-treatment speech can be seen in Video 12.2. During the assessment sessions, a number of different treatment techniques were trialled. Her speech fluency increased markedly during

singing, unison speech and repetition. This is illustrated in Video 12.3. DB also presented with secondary behaviours, such as clenching her jaw and fists during stuttering dysfluencies. She reacted verbally to her dysfluencies and reported to be frustrated and embarrassed about 'getting stuck', which had led to social isolation.

One-hour treatment sessions were started, once per week. She completed two 10-week treatment blocks, with a 5-month break in between due to personal events in the client's life. During the assessment sessions, it became evident that the combination of speech, language and cognitive problems required a stuttering treatment approach that that would require minimal cognitive demands. The paced speech approach was most successful, especially when supported with visual and tactile feedback given by a pacing board, and visual and auditory guidance given by the clinician. The client was encouraged to tap a square on a laminated six-square pacing board, with her index finger, for each syllable she produced. As she spontaneously started tapping a square for each word rather than syllable, we adjusted our approach to what came most natural to her. During the training phase, frequent modelling of pacing board use was provided. The clinician demonstrated using the pacing board and then tapped along with the participant. When the participant was able to use the technique independently without clinician modelling, external guiding from the clinician was gradually removed.

Once the pacing technique was implemented successfully, it was complemented with low-level cognitive restructuring to address the negative emotions and attitudes around communication. Strategies were implemented to recognise and reduce frustration. These included pausing, self-imposed time out, relaxation through deep breathing and easy onset. Next, naming tasks were introduced as word finding problems were the second most frequent cause for interruptions in speech fluency following the stuttering dysfluencies. We focused on names and relationships of DB's family members as talking with and about her family were priorities for her. At the start of the second treatment block, a goal of increased participation in community activities was set following a shared goal-setting approach. Treatment focused on skill transfer and generalisation, and conversation partner training was implemented. This included instructed demonstration of the techniques to ensure that the conversation partners would continue to provide support and reminders outside of the therapy sessions, when needed.

During the first treatment block, DB's stuttering frequency during spontaneous speech decreased from 41% to 24% stuttered syllables. An example of DB using the pacing technique at the end of this treatment block can be seen in Video 12.4. During the treatment break, DB reported not to have worked on her speech as she had been faced with significant personal loss. Her stuttering frequency had increased to 66% at the start of the second treatment block. However, it quickly reduced following re-introduction of the techniques and returned to 22% during the final session.

As DB's stuttering frequency decreased throughout both treatment blocks, her reactions to the stuttering also decreased. As the second treatment block progressed, DB began attending social activities in the retirement village as well as weekly gym classes. She also commented on improvement in her confidence and increased willingness to engage with others. Overall, the treatment resulted in a significant improvement in DB's quality of life.

Summary and Clinical Implications

Acquired stuttering is characterised by the occurrence of stuttering dysfluencies in a person's speech, and these can be neurogenic or functional in origin. The stuttering dysfluencies are the core characteristic of acquired stuttering, and presence of other characteristics (e.g., emotional reactions) can vary depending on the underlying aetiology and client characteristics. Differential diagnosis between neurogenic and functional stuttering needs to be attempted – although this may need to be adjusted later on as more information becomes available. A detailed assessment session is also needed to provide information regarding potential treatment strategies.

The two cases presented here both had a complex medical history and were initially not referred for stuttering therapy, despite the stuttering being their most significant communication problem. For both clients, different treatment approaches were trialled and gradually adjusted over the course of the treatment. While both had a different underlying aetiology of their acquired stuttering (neurogenic versus functional), they showed a large and clinically meaningful reduction in stuttering dysfluencies and a significant improvement in quality of life following treatment.

These cases illustrate the importance of recognising acquired stuttering dysfluencies as a problem that may require specific stuttering treatment, and such treatment should be provided upon the client's request. Despite the shortage of evidence-based therapy information in the literature, the cases presented here show that individualised treatment approaches can lead to positive outcomes.

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