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Characteristics and Assessment of Acquired Stuttering: A Clinical Guide

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Characteristics and Assessment of Acquired Stuttering: A Clinical Guide

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Dedication

To my parents, Steve and Carol, who have supported me through my entire journey, both in life and in education. Thank you for teaching me perseverance, strength, and love.

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Abstract

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Disfluencies as a consequence of acquired stuttering can arise subsequent to neurological trauma, psychological stress, and medication use. Acquired stuttering presents as various clinical pictures depending on the etiology of the disfluencies. This leads to difficulties in the assessment, and ultimately treatment, process. Similarly, the onset, characteristics, associated symptomatology, and assessment processes of the disorder differ between individual cases. Thus, there is dispute in the current literature on the characteristics and diagnostic procedures involved with acquired stuttering. This report offers a comprehensive review of the nature and assessment of acquired stuttering, serving to compile and organize the current views of the disorder.

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Introduction

Stuttering is a speech disorder characterized by interruptions in fluent speech, tension, and arrhythmic speech. Secondary behaviors (i.e. extraneous body movements or eye blinks), avoidance behaviors (i.e. word avoidance), and negative emotions associated with disfluent speech may accompany the stuttering. The typical view of stuttering maintains the development of stuttering during childhood and the continuation of stuttering throughout adult life. For the purposes of this report, this type of stuttering will be referred to as organic stuttering, however this type of stuttering is also commonly referred to as developmental stuttering (Jones, Conture, & Walden, 2014; McDearmon, 1968; Van Borsel, 2014). In less common circumstances, stuttering may present as a result of neurogenic injury, psychological trauma, or medicine use and is referred to as acquired stuttering. Similar to organic stuttering, acquired stuttering is a speech disorder characterized by disfluent speech and may be accompanied by the other stuttering characteristics. Individuals with acquired stuttering may show normal disfluencies (whole-word repetitions, phrase repetitions, revisions, interjections) as well as stuttering like disfluencies (sound and syllable repetitions, prolongations, blocks) (Guitar, 2013; Helm, Butler, & Canter, 1980; Theys, van Wieringen, & De Nil, 2008; Van Borsel & Taillieu, 2001; Yairi & Ambrose, 2013).

Organic stuttering and acquired stuttering share many of the same characteristics and may be homologous in primary and secondary stuttering behaviors, necessitating the examination of the disorder to differentiate between the two types of stuttering. The age

and circumstance around the onset of stuttering provide valuable information to the examination and evaluation of acquired stuttering. While organic stuttering presents in childhood, acquired stuttering typically presents after the developmental years (Duffy & Baumgartner, 1997; Krishnan & Tiwari, 2013; Theys, van Wieringen, Tuyls, & De Nil, 2009; Van Riper, 1964).

The etiologies of stuttering, including genetic factors, neurologic trauma, psychological stress, and medication use, each contribute to the unique presentation of acquired stuttering types. Genetic factors are hypothesized in acquired stuttering, but have not yet been reported to impact a significant portion of those with acquired stuttering. Neurologic trauma is a nonorganic, cerebral cause of acquired neurogenic stuttering that occurs after central nervous system damage (Theys et al., 2008; Van Borsel, 2014). Psychogenic stuttering is a form of acquired stuttering caused by psychological or emotional stress that cause disfluent speech (Duffy & Baumgartner, 1997; Theys et al., 2009; Ward, 2010). Medicine induced stuttering is caused by the presence, or excess, of medication which elicits neurochemical responses manifested as disfluencies (Lundgren, Helm-Estabrooks, & Klein, 2010; Van Borsel, 2014). Examining the characteristics of acquired stuttering lends valuable information on the etiology of stuttering.

Associated symptomatology is often present alongside acquired stuttering. Aphasia, dysarthria, and apraxia are common conditions that present simultaneously to stuttering and may contribute to or impact stuttering presentation. Different traumatic experiences lead to different presentations of comorbid symptomatology. Acquired

stuttering commonly occurs in individuals with multifaceted communication problems. Examining the complex nature of communication disorders associated with neurological, psychological, or medicine induced trauma as they relate to acquired stuttering aids in assessment and treatment processes (Helm et al., 1980; Krishnan & Tiwari, 2011; Theys et al., 2008).

Assessment of acquired stuttering is an involved process contingent upon the presence of characteristics of acquired stuttering types and response to trial therapy. General assessment procedures that are used in organic stuttering should be administered during an acquired stuttering evaluation. Examining characteristics present in an individual with acquired stuttering, while specifically looking at the circumstance around the onset of stuttering and the primary and secondary characteristics of stuttering, aid in the evaluation process. Response to trial therapy is a notable diagnostic marker in acquired stuttering. Neurogenic stuttering tends to persist despite fluency treatment, unless the underlying neurologic trauma is remitted. Those with psychogenic stuttering have been reported to have remarkable recovery after fluency or psychiatric treatment. Similarly, medicine induced stuttering tends to resolve with treatment including discontinuing the medicine suspected of inducing disfluencies. Thus, the circumstance under which stuttering improves or does not improve is a telling feature of acquired stuttering assessment (Guitar, 2013; Krishnan & Tiwari, 2013; Lebrun, Bijleveld, & Rousseau, 1990; Van Borsel, 2014; Ward, 2010).

Finally, malingered stuttering is intentional stuttering for the purpose of benefitting from claiming the disorder. Assessing malingered stuttering is an involved

process that includes understanding common stuttering characteristics and evaluating their presence in a person with possible malingered stuttering. Though malingered stuttering is not a genuine form of stuttering, assessing for feigned disfluencies is an important skill for Speech Language Pathologists because of the potential disguise as acquired or organic stuttering (Bloodstein, 1988; Seery, 2005; Shirkey, 1987).

The following report explains the nature, characteristics, associated symptomatology, and assessment of acquired studying. Neurogenic, psychogenic, and medicine induced stuttering are examined in detail, with additional information on malingered stuttering.

Onset of Acquired Stuttering

The onset of acquired stuttering ranges from childhood to adulthood. Acquired stuttering can appear at any age after a neurological, psychological, or medicine related event, though is more likely to occur during adulthood than childhood (Krishnan & Tiwari, 2013; Theys et al., 2009; Van Borsel, 2014; Van Riper, 1964). The onset of acquired stuttering commonly happens suddenly and invariably happens subsequent to a period of time when stuttering was not present (Sabillo, Samala, & Ciocon, 2012; Van Riper, 1964; Ward, 2010).

Organic stuttering initially presents during the language development year, usually between 2 and 6 years old, and begins to appear gradually (Yairi & Ambrose, 2013). Therefore, stuttering that appears after the developmental language years is likely to be acquired stuttering (Chang, Erickson, Ambrose, Hasegawa-Johnson, & Ludlow, 2008; Yairi, 2006). However, neurogenic injury, psychological trauma, or medicine-induced neuronal damage can occur at any age, therefore acquired stuttering can occur during the developmental years if trauma occurs during these years (Perez, Gubser-mercati, & Davidoff, 1996). Additionally, the onset of acquired stuttering can occur later than the initial trauma that caused the stuttering, as seen as a delayed manifestation of disfluencies (Attanasio, 1987). Thus, viewing the circumstances around and before the onset of stuttering is an important factor in determining the etiology of stuttering.

Etiologies of Acquired Stuttering

Acquired stuttering and organic stuttering are both multifactorial disorders and both can precede diverse etiologies. Acquired stuttering results from different types of traumatic incidents, including neurogenic, psychogenic, or drug-induced trauma. These three causes make up the majority of acquired stuttering occurrences (Bhatnagar & Buckingham, 2010; Van Borsel, 2014). Organic stuttering can result from multiple factors that contribute to stuttering development, however unlike acquired stuttering, there is no discernable cause of organic stuttering. Contributing factors of organic stuttering include genetics, language disorders or delays (Ntourou, Conture, & Lipsey, 2011; Yaruss, LaSalle, & Conture, 1998), and neurophysiology (Bhatnagar & Buckingham, 2010).

Though the etiologies of acquired stuttering and organic stuttering differ, the two disorders share a commonality of factors that exacerbate disfluencies. These factors include stressful demands for communication that stem from lifestyle and environmental circumstances. Communication demands may exceed a person's capacity for communication, resulting in a breakdown of speech that is represented as disfluencies (Adams, 1990; Starkweather & Gottwald, 1990). Nevertheless, the factors that exacerbate disfluencies are not interchangeable with the etiologies of stuttering in either disorder. Both acquired and organic stuttering may present alongside, but be independent of, neurological trauma, psychological disorders, or medication use (Alm, 2014; Van Borsel, 2014).

GENETIC COMPONENTS OF ACQUIRED AND ORGANIC STUTTERING

Acquired stuttering has not yet been observed to have a significant genetic component. Organic stuttering, however, has a significant genetic component and familial link. Children with family members who stutter, are more likely to develop a stutter (Duffy & Baumgartner, 1997; Kraft & Yairi, 2011). Individuals who exhibit acquired stuttering may have a genetic predisposition to stuttering in which an aspect of their neuroanatomy increases the likelihood that they will acquire stuttering later in life. Those who appear to have acquired stuttering may be covert stutterers and have hidden their organic stuttering throughout their lifetime. Additionally, neurological trauma, psychological trauma, or medication may have an adverse effect on organic stuttering in which disfluencies recur, change, or increase in severity (Theys et al., 2008; Van Borsel, 2002). After trauma to the brain or specific medication use, disfluencies may become evident in these individuals. Discriminating between the presence of genetic predispositions for stuttering or covert developmental stuttering that resurfaces is a difficult task and one that warrants a comprehensive profile of the individual (Grant, Biousse, Cook, & Newman, 1999; Riggs, Nelson, & Lanham, 1983).

Acquired stuttering can be seen in any gender and is as equally likely to occur in women and men. Gender differences may play a role in acquired stuttering incidence if a particular gender is more likely to sustain neurogenic trauma that results in stuttering. However gender differences are not inherently responsible for disparities in stuttering incidence among men and women (Duffy & Baumgartner, 1997). In contrast, organic

stuttering is 4 times more likely to be seen in males than in females (Craig, Hancock, Tran, Craig, & Peters, 2002; Yairi & Ambrose, 2013).

NEUROGENIC STUTTERING

Neurogenic stuttering is a type of acquired stuttering that results from nonorganic damage to the central nervous system (CNS). Increases in stuttering as a result of CNS damage are associated with many lesion sites. Cerebral damage locations have been reported to be unilateral in either hemisphere, bilateral, focal, and diffuse. However bilateral and diffuse lesions tend to produce more profound and long-term fluency difficulties. Damage that causes stuttering has also been reported in the corpus callosum, in the cerebellum, in the brainstem, and in the frontal, temporal, occipital and parietal lobe. However, acquired neurogenic stuttering predominantly corresponds with damage to the left anterior portion of the brain, an area commonly associated with language. Specific causes of neurologic damage include traumatic brain injury, seizures, encephalitis, Parkinson's disease, neurodegenerative disease, tumor, and stroke (Grant et al., 1999; Krishnan & Tiwari, 2013; Lebrun et al., 1990; Sabillo et al., 2012; Tippett & Siebens, 1991; Van Borsel, 2002).

Neurogenic stuttering can occur at any age after a neurogenic event. Neurogenic stuttering has been reported in a range of ages, from children 3 years old due to damage from rotavirus and encephalitis, to adults 93 years old due to damage from stroke, neurodegenerative disease, or brain injury. Most commonly, however, acquired neurogenic stuttering occurs in adulthood (Theys et al., 2009). In addition to stuttering

that first appears after a neurological event, damage from neurologic events can compound organic stuttering. Disfluencies from organic stuttering can recur, worsen, or change after neurological trauma (Rentschler, Driver, & Callaway, 1984; Theys et al., 2008, 2009).

Disfluencies vary among individuals with neurogenic stuttering. Stuttering frequency has been reported to be present in 3% to 50% of words in different individuals. The majority of individuals present with sound repetitions, syllable repetitions, and blocks during speech, however whole word repetitions and prolongations have also been reported. Disfluencies are largely present on both content and function words, differing notably from organic stuttering, which largely involves disfluencies on content words. Widely, patients are noted to stutter throughout various speech tasks, however some present with stuttering only on spontaneous speech. Secondary behaviors, including facial tension, body movements, and avoidance behaviors have been observed to vary between individuals as well. Additionally, emotional reactions and experiences related to stuttering, including frustration, irritation, fear, anger, and anxiety, are reported to differ. A link between site of neurological trauma and disfluency loci within a word or stuttering behaviors has not been identified; trauma to the same area in the CNS may yield very different clinical pictures. Altogether, disfluencies and secondary characteristics of those with acquired stuttering vary significantly despite similarities in CNS damage (Theys et al., 2008; Van Borsel, Van Lierde, Van Cauwenberge, Guldemont, & Van Orshoven, 1998).

Neurogenic stuttering typically begins close in time to the time of neurologic insult. Disfluencies commonly appear within one week of neurogenic injury. However, stuttering has been reported to appear a few weeks to a few months after the injury, with longer durations between damage and disfluency onset occurring more rarely (Theys et al., 2008; Van Borsel, 2002). When neurogenic stuttering does not begin close to the time of neurological insult, disfluencies are typically related to seizure disorders or epilepsy. Disfluencies may appear as seizures become more complex, severe, or frequent. Additionally, stuttering may begin after subsequent injuries. Disfluencies have been reported to appear after a second stroke or brain injury, but may be related to both traumatic incidents (Helm et al., 1980).

Acquired stuttering has been reported to occur with concomitant neurologic problems. The nature of the disorder permits related complications because brain trauma typically yields difficulties in multiple realms and is affiliated with multiple side effects. Right and left hemiparesis, headaches, tremors, and incoordination are among the commonly co-occurring difficulties (Duffy & Baumgartner, 1997; Theys et al., 2008).

PSYCHOGENIC STUTTERING

Psychogenic stuttering is an acquired fluency disorder related to psychological trauma. Disfluencies in those with psychogenic stuttering are a physical manifestation of acute or chronic emotional problems. The criteria typically used to classify a psychogenic stutter are a change in speech patterns that include disfluencies and are indicative of stuttering, onset of stuttering close in proximity to onset of psychological factors, and no

organic stuttering evidence. History of mental health issues and abnormal secondary behaviors are additional factors that are frequently reported in those suspected of psychogenic stuttering. These features are common in those with psychogenic stuttering, however they have been disputed in the literature and the presence of the characteristics is not invariably indicative of psychogenic stuttering (Duffy & Baumgartner, 1997; Theys et al., 2009; Ward, 2010). Organic stuttering, however, may be exacerbated during emotional turmoil (i.e. getting nervous during a presentation and stuttering more) but the emotional problems do not cause the stuttering.

Psychiatric trauma associated with psychogenic stuttering can be from any form of psychological problem. Common psychiatric diagnoses associated with psychogenic stuttering are conversion reaction, anxiety, depression, personality disorder, and post traumatic stress disorder. Combinations of psychiatric disorders have also been reported (Duffy & Baumgartner, 1997). Emotional stress unrelated to a psychiatric diagnosis has been reported to cause psychogenic stuttering. Psychogenic stuttering is not exclusive to those with mental health disorders. The events that may cause stuttering in an individual should be viewed as the person with stuttering perceives them; events may not appear likely to cause anxiety or stress but may be of particular importance to the individual and cause significant emotional trauma that spurs stuttering (Duffy & Baumgartner, 1997; Ward, 2010).

Symptoms of psychogenic stuttering usually appear around the same time that the psychological stress occurs. However the symptoms may appear later than the psychiatric diagnosis at a seemingly random time or when the psychiatric problem was worsening

(Duffy & Baumgartner, 1997; Helm et al., 1980; Ward, 2010). Disfluencies vary in severity and exist despite normal cognition, vocabulary use, grammar and receptive language functions. Stuttering may develop in a similar manner as organic stuttering, gradually and continuing to develop over time, or there may not be noticeable changes after the initial presentation of disfluencies (Tippett & Siebens, 1991; Van Borsel, 2014). The length of time symptoms of psychogenic stuttering are evident also varies. The disorder can be considered chronic and last for a significant period of time (months or years), or it may subside quickly and last for only days (Duffy & Baumgartner, 1997). However, disfluencies routinely coincide with mental health status of an individual diagnosed with psychogenic stuttering. Stuttering may improve as mental health improves. Likewise, stuttering may worsen with increasing psychiatric demands. For example, if a person is experiencing depression they may be more susceptible to disfluencies during that time, or if a person is in a high anxiety situation, stuttering may increase (Helm et al., 1980; Tippett & Siebens, 1991). If the cause of psychogenic stuttering in an individual is chronic (i.e. a generalized psychiatric disorder), the individual may be prone to a longer recovery period as psychiatrics symptoms come and go. If the cause is unknown psychogenic (i.e. an idiopathic psychiatric disorder), the individual may be less likely to recover, and stuttering is less likely to improve with fluency or psychiatric techniques. However, progress is probable when the specific psychological cause is found and treated (Ward, 2010).

Secondary behaviors exist in those with psychogenic stuttering but have been noted as atypical or "bizarre," and not resembling those of organic stuttering. Abnormal

secondary behaviors noted have included unusual grammatical deviations and multiple repetitions of all phonemes with simultaneous body movements (Duffy & Baumgartner, 1997; Lundgren et al., 2010). However, the literature reports some individuals with more typical secondary behaviors like eye squinting and facial tension (Theys et al., 2009). Those with psychogenic stuttering also have been reported to notice their stuttering, although not experience anxiety associated with their disfluencies. They often report feeling annoyance toward their stuttering or feeling concerned with their communication difficulties, but do not necessarily have stress or anxiety related to their stuttering (Theys et al., 2009; Ward, 2010).

Psychogenic stuttering can happen as a result of an emotional reaction to neurological trauma. The two types of trauma (psychological and neurological) often occur together, warranting an understanding of the two types of stuttering. The disfluencies are not directly resulting from neurologic trauma itself. Rather, they are related to psychological stress that is associated with a neurologic event or that coexists with neurologic disease. Psychogenic stuttering may be rooted in a neurologic event that predisposes an individual to a fluency disorder, but the stuttering is spurred by psychogenic trauma subsequent to the neurologic event (i.e. a person may have a traumatic brain injury, after which no stuttering is noted. Years later, the person may notice stuttering while they are going though a divorce). In these cases, there is stronger evidence of a psychological origin of stuttering than a neurological origin (Duffy & Baumgartner, 1997; Helm et al., 1980; Van Borsel, 2014; Ward, 2010). Additionally, the interaction between psychogenic trauma and neurogenic trauma may cause stuttering

(Theys et al., 2008). Unquestionably, psychogenic stuttering can happen independent of any neurologic injury (Tippett & Siebens, 1991).

Neurogenic diseases reported to have psychogenic implications, which may lead to stuttering, include epilepsy, stroke, degenerative disease, closed head injury, and encephalopathy/encephalitis. Many of these neurologic problems are associated with a significant period of emotional turmoil caused by medical complications, lifestyle changes, and uncertainty about future health and living conditions (Duffy & Baumgartner, 1997).

MEDICINE INDUCED STUTTERING

Medicine induced stuttering is a type of acquired fluency disorder in which stuttering necessarily accompanies the use of certain medications. The stuttering appears in people without a previous history of stuttering and they usually experience disfluencies that parallel the medication use. Disfluencies from medicine induced stuttering share a cause-and-effect relationship with medicine use. The type and severity of disfluencies that appears in medicine induced stuttering depend on the type and dosage of drug that caused the stuttering. Disfluencies from medication use may also manifest uniquely in each individual. Two people taking the same drug may both show signs of acquired stuttering, but exhibit distinct and different disfluencies or stuttering behaviors. Additionally, one person taking a drug may show signs of acquired stuttering, while another person does not (Lundgren et al., 2010; Van Borsel, 2002, 2014).

Acquired stuttering can be manipulated with drug dose. Disfluencies resulting from medications usually increase with increasing drug use and decrease with decreasing drug use (Christensen, Byerly, & McElroy, 1996; Grover, Verma, & Nebhinani, 2012; Murphy et al., 2015; Yadav, 2010). Presence of the drug in a person's system and presence of stuttering are directly correlated. This is the only etiology of stuttering (from both acquired and developmental) that has a removable determinant: the medication. If the drug is removed, then the disfluencies should go away if the stuttering is truly medicine-induced. Additionally, if the same medicine is given to the same person again, the stuttering will likely reappear. This relationship exists because medications can cause perpetual neurochemical abnormalities that exist as long as the medication is in use. Medication induced acquired stuttering is based only in neurochemical irregularities or a disruption of neurophysiology due to neurochemical changes, while organic stuttering has been hypothesized to have a basis in neurochemical and neuroanatomical irregularities (Lundgren et al., 2010; Movsessian, 2005; Van Borsel, 2014).

A variety of drugs have been reported to induce stuttering. Drugs linked to the inception of stuttering include antipsychotics such as Clozapine (Grover et al., 2012; Kumar, Kathpal, & Longshore, 2013; Murphy et al., 2015), Risperidone (Yadav, 2010), Bupropion (Fetterolf & Marceau, 2013), Sertraline (Christensen et al., 1996), and Lithium if given in toxic doses (Sabillo et al., 2012). In contrast, some of the same drugs have been used to treat developmental stuttering by decreasing stuttering severity, including Risperidone (Maguire, Riley, Franklin, & Gottschalk, 2000; Yadav, 2010).

In addition to psychiatric drugs, theophylline, a drug used to treat respiratory diseases has been documented to induce stuttering in individuals. The pathologies that result from theophylline are hypothesized to arise from malfunction of the motor cortex and white matter tracts that connect different cerebral areas. These malfunctions can lead to disruptions in the neurophysiology of speech motor areas and the motor cortex, resulting in stuttering (Gerard, Delecluse, & Robience, 1998; Movsessian, 2005; Rosenfield, McCarthy, McKinney, Viswanath, & Nudelman, 1994).

Associated Speech and Language Symptomatology of Acquired Stuttering

Stuttering commonly occurs with other speech or language disorders. Disorders including aphasia, anomia, dysarthria, and apraxia have been associated with acquired stuttering. All types of acquired stuttering have been reported as related to comorbid symptoms. However, associated symptomatology most commonly occurs with neurogenic stuttering as compared to psychogenic or medicine induced stuttering because neurogenic trauma has the greatest propensity for trauma associated with multiple brain areas and brain functions (Attanasio, 1987; Rentschler et al., 1984; Theys et al., 2008; Van Borsel, 2002). Larger lesions and greater CNS trauma increase the likelihood of additional communication problems present with acquired stuttering (Helm et al., 1980; Krishnan & Tiwari, 2011; Theys et al., 2008). Associated symptoms of acquired stuttering differ from organic stuttering, which most commonly occurs with phonological disorders (Chang et al., 2008; Duffy & Baumgartner, 1997; Ntourou et al., 2011; Yairi, 2006; Yaruss et al., 1998).

Examining the flexible nature of associated speech and language disorders and acquired stuttering aids in assessing and treating acquired stuttering. The presence of disfluent speech does not necessitate an underlying fluency disorder; disfluencies may be present, though they may not be stuttering-like in nature and be more closely related to other speech or language disorders. Additionally, the course of treatment for acquired stuttering varies depending on comorbid symptomatology. Therefore, identifying any

associated symptoms or disorders during evaluation is important for therapeutic processes (Helm et al., 1980; Krishnan & Tiwari, 2011; Theys et al., 2008).

Comorbid speech and language disorders may appear at the same time as stuttering appears, following an onset of neurogenic, psychogenic, or medicine induced trauma. Comorbid symptoms may also begin prior to stuttering but continue to occur simultaneously with stuttering (Van Borsel et al., 1998). Additionally, co-occurring speech or language problems may subside while stuttering does not improve, or stuttering may improve in the presence of abiding speech and language symptoms (Grant et al., 1999).

Aphasia is a language disorder that commonly occurs with acquired stuttering. Aphasia usually presents alongside acquired stuttering as either neurogenic stuttering related to neurological trauma or as psychogenic stuttering related to an emotional reaction to aphasia (Helm et al., 1980; Lundgren et al., 2010; Van Borsel, 2014). Different types of aphasia, including Broca's, Wernicke's, and conduction aphasia, have been reported to occur with acquired stuttering (Van Borsel, 2002). Aphasia of multiple types contributes additional non-stuttering like disfluencies (i.e. whole word repetitions, interjections, revisions) because of word finding or error correcting difficulties that happen alongside stuttering-like disfluencies (Farmer, 1975; Lundgren et al., 2010). Anomia, or word finding difficulties, is a language difficulty that can occur alone or with aphasia, and is reported in association with acquired stuttering. Broca's aphasia and anomia tend to be associated with less severe stuttering, while Wernicke's aphasia and conduction aphasia tend to be associated with more severe stuttering. Additionally,

people with aphasia and stuttering demonstrate more severe stuttering than those with only stuttering (Farmer, 1975).

Broca's aphasia is the most frequently reported associated language disorder to acquired stuttering. Broca's area is in the left anterior portion of the brain, the same area of damage that is most commonly associated with acquired stuttering. Therefore, damage to this area of the brain leads to the possible manifestation of both Broca's aphasia and acquired stuttering (Van Borsel, 2002). Individuals with Broca's aphasia typically present with laborious speech, characterized by the production of slow, strained content words. These difficulties foster additional communication challenges for those who have concurrent acquired stuttering (Farmer, 1975; Lundgren et al., 2010).

Wernicke's aphasia is another aphasia type reported to occur with acquired stuttering. Wernicke's aphasia is characterized by difficulties with comprehension and semantic content in language, despite typical grammar output. Paraphasias (production of unintended sounds, syllables, or words) and neologisms (made up words) commonly occur with Wernicke's aphasia. Attempts to correct paraphasias or neologisms may present as disfluent speech, but should be considered as non stuttering-like disfluencies and different from co-occurring Wernicke's aphasia and stuttering-like disfluencies (Farmer, 1975; Guitar, 2013; Van Borsel, 2014).

Aphasia and anomia often have disfluencies related to the disorders because of word finding difficulties and linguistic errors. Word finding difficulties that create disfluencies should be distinguished from disfluencies that occur alongside, but separate from, aphasias and anomia. Additionally, repetitions during intentional correction of

linguistic errors are common in aphasia and anomia and should be distinguished from stuttering-like disfluencies. These can be distinguished from stuttering by looking at errors in sound production. Stuttering often occurs on the correct production of a sound, while repetitions of sounds, syllables, or words to correct errors occur deliberately on incorrect productions (Guitar, 2013; Van Borsel, 2014).

Dysarthria co-occurring with acquired stuttering may add additional communication difficulties. Problems producing perceptually appropriate rate and volume of speech, alongside fluency difficulties, are common clinical presentations of those with acquired stuttering and dysarthria (Helm et al., 1980; Krishnan & Tiwari, 2011; Theys et al., 2008). Dysarthria may cause a difficulty or inability to tap rhythmically or sing. This creates challenges in stuttering treatment, as treatment sometimes incorporates speaking while tapping and speaking with a particular rhythm (Helm et al., 1980; Krishnan & Tiwari, 2011; Theys et al., 2008). Similar to the presentation of most associated speech and language disorders, different dysarthria types occur with different lesion sites. Therefore, the dysarthric and disfluent characteristics may vary depending on the area and extent of trauma (Van Borsel et al., 1998).

Apraxia is often associated with repetitions of sounds due to articulation and motor speech difficulties and can occur alongside acquired stuttering (Lundgren et al., 2010). Apraxia may cause speech so sound slow and labored, creating additional communication difficulties for those with both acquired stuttering and apraxia. Acquired stuttering should be distinguished from repetitions that occur when an individual is trying to correct motor errors that occur because of apraxia. For example, disfluent speech may

occur because of multiple attempts to correctly articulate a sound by a person with apraxia, however these repetitions should be considered non-stuttering like disfluencies (Chang et al., 2008; Duffy & Baumgartner, 1997; Lundgren et al., 2010; Ntourou et al., 2011; Yairi, 2006; Yaruss et al., 1998).

Pallalia, a disorder that includes disfluencies from repetitions, should be distinguished from acquired stuttering. Pallalia is an extrapyramidal disease characterized by whole word, phrase, or sentence repetitions that are produced with increasing speed (i.e. "speech festination"; most commonly seen as a result of Parkinson's disease) (Duffy & Baumgartner, 1997; Guitar, 2013; Lundgren et al., 2010; Van Borsel, 2014).

Assessment of Acquired Stuttering

There is a strong link between neurogenic, psychogenic, and medicine induced stuttering, rendering the assessment process as challenging. The different types of acquired stuttering share common characteristics in clinical presentation and may be difficult to distinguish from one another. Furthermore, all three types of acquired stuttering can be interrelated. For example, a person who sustains a traumatic brain injury may have psychological stress associated with their recovery and may have to take medication to aid neurological and psychological trauma. Therefore, it is necessary to closely examine the case history and present disfluent characteristics to adequately assess and diagnose the individual (Helm et al., 1980). Additional complications arise from the possibility of a neurologic basis of stuttering that is exacerbated by increased stress or anxiety (Theys et al., 2008, 2009). Key diagnostic questions include asking about the time interval between any suspected or confirmed neurological or psychological trauma. Additionally, individuals should be asked if there were any significant environmental factors, including starting medication use, in the time period around the onset of stuttering (Theys et al., 2008).

Motor disorders often accompany both neurogenic and psychogenic stuttering, and many individuals experiencing trauma have additional sensory and cognitive complaints. It is uncommon that a person does not present with other problems that accompany their disfluencies, adding to the difficulty of diagnosis and etiological explanation (Duffy & Baumgartner, 1997).

GENERAL ASSESSMENT PROCEDURES

General assessment procedures should be administered when assessing any type of acquired stuttering. Gathering a detailed case history and family history of stuttering, language, or learning should be obtained for information on potential symptoms of developmental stuttering during childhood. The case history should also include fluency patterns, changes in fluency patterns over time, and any periods of time in which the individual was fluent. Stuttering is variable, a person may be fluent in one situation and stutter a lot in another, so information of their history of stuttering is essential. Additionally, information on the onset of stuttering is important to collect, such as the age it first occurred and if it occurred after an acute neurologic or psychogenic event, or after medication use (Guitar, 2013).

Standard assessment procedures, like gathering a representative speech sample, finding a disfluency index, examining secondary behaviors, measuring speech rate, and assessing feelings and attitudes about stuttering, should be done during assessment of acquired stuttering. A motor speech examination should be given during the assessment to assess for motor speech disorders that may be influencing the disfluencies (Shipley & McAfee, 2016).

NEUROGENIC STUTTERING ASSESSMENT

Neurogenic stuttering assessment involves examining brain scans to confirm lesions or neurologic trauma. Confirmation of brain trauma around the same time as the

onset of stuttering is a strong diagnostic marker in assessing neurogenic stuttering (Sabillo et al., 2012). If a brain scan is not conducted, or if nothing remarkable is found on a scan, examinations to assess various cognitive abilities may indicate brain damage. Tasks should include probing for language expression and comprehension, memory, orientation, and attention to help identify presence of brain damage. Although rare, MRI scans have been reported to give false negative results. Therefore, additional cognitive testing may be warranted in suspected neurologic damage if brain scans report no damage (Ward, 2010).

Response to Treatment:

Neurogenic stuttering usually persists for a long time and responds poorly to treatment (Tippett & Siebens, 1991; Van Borsel, 2002). Some neurogenic stuttering resolves with common stuttering treatment (i.e. counseling, fluency modification, fluency shaping), however like organic stuttering, improvements in fluency are widely variable. Additionally, stuttering improvement depends on the improvement of the underlying neurological problem. If the lesion site heals or is fixed, stuttering is more likely to resolve. However healing or fixing the lesion site typically takes a significant period of time, so a quick recovery is typically not observed in trial therapy during the assessment period (Ward, 2010).

Associated Non-Speech-and-Langauge Symptomatology:

Assessing non-speech-and-language associated symptomatology aids in evaluation. Confirming the presence of other problems, including headaches, right and left hemiparesis, tremor, and incoordination, contributes to the diagnosis of neurogenic

stuttering. These characteristics are more common among those with neurogenic stuttering than any other type of acquired stuttering (Duffy & Baumgartner, 1997; Theys et al., 2008).

Related Characteristics:

Neurogenic stuttering has been reported to include certain characteristics that are uncommonly present in organic stuttering or other forms of acquired stuttering. The following have been noted as possible diagnostic characteristics of neurogenic stuttering: disfluencies occuring on function words nearly as frequently as on content words; 2) the speaker does not appear anxious, however may exhibit negative emotions towards their stuttering; 3) repetitions, prolongations, and blocks occur on initial, medial, and final syllables of words and utterances; 4) secondary symptoms are not associated with moments of disfluency; 5) there is no adaptation effect; and 6) stuttering occurs relatively consistently across different types of speech tasks. There have been discrepancies in the literature regarding these characteristics. Some have been reported in psychogenic stuttering and organic stuttering, and they are not all always present in neurogenic stuttering. Though the characteristics may occur among a large percentage of neurogenic stuttering, the criteria should be used with caution because they are not always reliable (Krishnan & Tiwari, 2013; Theys et al., 2008; Ward, 2010).

Stuttering typically occurs on content and function words in neurogenic stuttering. This differs from organic stuttering in which disfluencies most often occur on content words. This characteristic has largely been indicated in the literature and may aid in discriminating between neurogenic and organic stuttering, however this characteristic is

also reported in other types of acquired stuttering (Bloodstein, 1988; Guitar, 2013; Theys et al., 2008).

Many individuals have been reported to show emotional reactions, like discontent or annoyance, despite the absence of anxious behaviors associated with stuttering. This supports the idea that they may be displeased with their communication in general but do not have anxiety symptoms associated with their stuttering (Ardila & Lopez, 1986; Helm et al., 1980; Krishnan & Tiwari, 2011; Theys et al., 2008; Tippett & Siebens, 1991). Accounts of individuals reporting or appearing to be anxious have been indicated, however, so this characteristic alone should not be used to determine neurogenic stuttering (Attanasio, 1987; Theys et al., 2008).

Disfluencies have been noted in all syllable positions in neurogenic stuttering. They are most frequently reported to occur in the initial position and then the medial position. While final position disfluencies are present, they are not reported frequently in acquired neurogenic stuttering. Like the other diagnostic characteristics, this characteristic cannot be used in unison to diagnose neurogenic stuttering (Bloodstein, 1988; Guitar, 2013; Theys et al., 2008).

Assessment of secondary behaviors may yield evidence of neurogenic stuttering. Though there is debate in the literature on the relationship of secondary behaviors and neurogenic stuttering, behaviors such as facial tension and extraneous body movements are noted as less frequent in neurogenic stuttering than in organic stuttering. The presence of secondary behaviors should not negate the presence of neurogenic stuttering, however the absence of secondary behaviors offers a more concrete diagnosis of neurogenic

stuttering because the behaviors have been reported as absent in the majority of those with neurogenic stuttering (Helm et al., 1980; Theys et al., 2008; Van Borsel & Taillieu, 2001).

The adaptation effect is noted as variable among people with neurogenic stuttering. The majority of individuals with neurogenic stuttering do not show adaptation during multiple passage readings as they remain consistent after consecutive passage readings (Krishnan & Tiwari, 2013; Rentschler et al., 1984; Theys et al., 2008). A test for adaptation should be included during the assessment process. If no adaptation effect is present, neurogenic stuttering may be indicated. However the results should be applied to the evaluation with caution (Rentschler et al., 1984; Theys et al., 2008).

Gathering speech samples from multiple tasks aids in neurogenic stuttering assessment. Those with acquired neurogenic stuttering have been observed to stutter in multiple speech tasks. Stuttering is generally noted to be relatively consistent among various communication tasks, including spontaneous speech samples and reading aloud. However as previously mentioned, each person who stutters has a unique profile and there are reports of people with acquired neurogenic stuttering that vary among speech tasks (Theys et al., 2008, 2009).

The wide variety of characteristics present in acquired neurogenic stuttering leads to a difficult diagnostic task. Many of the characteristics vary greatly between individuals, so the most salient diagnostic marker is stuttering appearing shortly after a neurological injury. If stuttering occurs close to a neurological trauma, it is likely that the trauma caused stuttering and the stuttering is neurogenic in nature. If the stuttering occurs

a month or more after a neurological insult, it is difficult to link the initial injury and the onset of disfluencies. The patient should be asked the time interval between the neurological event and the onset of stuttering to aid the assessment process. Largely, the literature remains unresolved on concrete characteristics of acquired stuttering. The criteria of characteristics, therefore, is suggested as a guideline in assessing neurogenic stuttering rather than a strict rule of assessment (Helm et al., 1980; Krishnan & Tiwari, 2013; Theys et al., 2008).

PSYCHOGENIC STUTTERING ASSESSMENT

Presence of psychological stress around the onset of stuttering is a significant diagnostic marker in psychogenic stuttering. If disfluencies begin near the time of a psychologically traumatic event, it is probable that the disfluencies are suggestive of psychogenic stuttering (Tippett & Siebens, 1991; Van Borsel, 2002). Additionally, if stuttering worsens during psychiatric behaviors (i.e. stuttering worsening during an anxiety attack), psychogenic stuttering is probable (Helm et al., 1980). Examining neurologic trauma that occurs with psychiatric trauma is an important part of the assessment procedure, however. If the onset of neurological trauma, psychological trauma, and stuttering all occur within the same time period, further factors must be assessed to determine the etiology of stuttering such as trial therapy, mental health testing, examination of secondary behaviors, and examining the adaptation effect (Duffy & Baumgartner, 1997; Guitar, 2013; Helm et al., 1980; Krishnan & Tiwari, 2013; Theys et al., 2009; Tippett & Siebens, 1991; Ward, 2010).

Response to Treatment:

The strongest evidence of psychogenic etiology in acquired stuttering is the response to treatment, necessitating the use of trial therapy during the assessment period. Those with psychogenic stuttering are likely to respond to successful psychological treatment; with improved mental health, improvements in disfluency are likely. Disfluencies are likely to subside or recover completely with one or two behavioral therapy treatment sessions or with the use of psychiatric management strategies during therapy (Duffy & Baumgartner, 1997; Van Borsel, 2002).

Behavioral therapy used during fluency assessments includes psychoeducation, techniques to improve emotions, and rewards. Psychoeducation about traumatic events, negative emotions, or destructive behaviors that the individual is experiencing are discussed at the beginning of therapy. The individual will be taught that emotions are temporary and can be changed. Decreasing negative emotions and destructive behaviors by fostering positive experiences, alternative constructive thoughts, and activities should be a main goal of behavioral therapy in fluency trial therapy. Finally, the individual should receive rewards, that can include praise or a physical object, for engaging in positive behavior (Courtois & Ford, 2009). Results of success with similar treatment approaches are seldom reported with neurogenic stuttering. Therefore, trying behavioral therapy during the assessment period is essential if psychogenic stuttering is suspected, as this is a strong diagnostic marker. This is not to say that all people with psychogenic stuttering will recover quickly with psychiatric therapy (Duffy & Baumgartner, 1997; Theys et al., 2009).

Related Characteristics:

A detailed psychiatric history should be obtained from the individual to assess for current mental health status. Mental health questions should relate to their overall mental health status and current level of stress in their life, relationships, and career. Questions may also be related to daily mood, sleep patterns, energy, orientation, and self harm thoughts. If the person is unable to participate in a mental health assessment, the person's family members or close friends may answer the questions to the best of their ability (Krishnan & Tiwari, 2013).

The absence of neurological factors is a further indication of psychogenic stuttering in adults with acquired stuttering. If brain scans are administered and the results indicate no neuropathology, yet stuttering is still present, this is suggestive that the stuttering is psychological in nature. Furthermore, if stuttering improves despite no neurological improves, psychogenic stuttering may be indicated (Guitar, 2013; Tippett & Siebens, 1991; Ward, 2010). Stuttering that emerges as a result of brain injury may be psychological in nature despite evidence of neurologic damage. Disfluencies can result as an emotional reaction to neurogenic disorders and therefore is best classified as psychogenic stuttering. It is important to distinguish disfluencies that arise as a symptom of an emotional reaction and as a symptom to a neurogenic disorder. A person with aphasia may present with disfluencies, likely to be non-stuttering like disfluencies (word and phrase repetitions, interjections, revisions), which do not result from psychological problems but result from word finding difficulties due to the aphasia. However, the same person may also experience stuttering like disfluencies (sound and syllable repetitions,

blocks, prolongations) that do result from psychological problems. If the person presents with stuttering like disfluencies, they should be assessed for psychogenic stuttering. In assessment of an individual with brain injury, diagnosticians can look for improvement in fluency despite absence of improvement in neurology (Guitar, 2013; Tippett & Siebens, 1991; Van Borsel, 2014).

Examining secondary behaviors is a further method of distinguishing between acquired fluency disorders when psychogenic stuttering is possible. The presence of clear muscular tension is often noted in those with psychogenic stuttering, but not commonly observed in those with neurogenic stuttering. There are also reports of "bizarre" behaviors in individuals with psychogenic stuttering, including agrammatic speech without the presence of aphasia, abnormal body movements (i.e. unusual grammatical deviations, multiple repetitions of all phonemes) and unusual struggle behaviors (i.e. tremor-like arm movements) during disfluent moments (Duffy & Baumgartner, 1997; Guitar, 2013; Lundgren et al., 2010; Van Borsel, 2002; Ward, 2010).

The adaptation effect, or increased exposure to a stimulus leading to decreased anxiety regarding the stimulus, has been reported to help assess acquired stuttering (Starbuck & Steer, 1953). Psychogenic stuttering can be founded in anxiety disorders, therefore improvements have been noted in psychogenic stuttering with adaptation, both to situations and to reading passages, because of less perceived anxiety with each successive stimulus exposure (Theys et al., 2009). However, lack of success using the adaptation effect has also been reported in those with a psychogenic stutter. The

adaptation effect, therefore, should be used with caution when assessing the etiology of acquired stuttering (Helm et al., 1980; Starbuck & Steer, 1953; Theys et al., 2009).

MEDICINE INDUCED STUTTERING ASSESSMENT

Assessing suspected medicine induced stuttering requires a detailed profile of the individual, including medication history and history of any suspected neurological or psychological disorders. Acquired stuttering as a result of medication use is rare, so it is important to assess the profile of the person who stutters because stuttering may happen after drug use but stem from another cause. For example, if a person acquires a stutter after using a drug used to treat psychiatric disorders, the person must be examined to see if the cause of the stuttering is from psychogenic reasons or because of the drug used to treat the psychiatric disorder (Grover et al., 2012). Additionally, because acquired stuttering from drug use is rare, it is possible that the people with medication induced stuttering have a predisposition, or family history, of stuttering and the medication was a catalyst for stuttering (Yadav, 2010).

Response to Treatment:

Many of the drugs that may induce stuttering are of psychotherapeutic nature, it is therefore imperative to assess whether the drug of the psychiatric disorder is causing the stuttering. A reliable method of testing for this is manipulating the drug dosage, or discontinuing the drug, to see if disfluencies are impacted (Christensen et al., 1996; Grover et al., 2012; Murphy et al., 2015; Yadav, 2010). If the person is on multiple medications, especially if the person is on multiple medications that have shown

stuttering as a side effect, it is necessary to manipulate the medications independently and in unison to detect the drug related cause of the stuttering (Sabillo et al., 2012). For example, a person was assessed after a reported drug overdose from Tranxene, a Benzodiazepine used for treatment of psychiatric disorders. The person was noted to have manic-depression, anxiety, and previous suicide attempts. Because stuttering is a proven side effect of both psychological trauma and medication use, and the individual presented with psychiatric problems and used medication that has been associated with acquired stuttering, the individual must be assessed through medication manipulation (and possibly psychiatric behavioral therapy) to find the etiology (Rentschler et al., 1984).

MALINGERED STUTTERING ASSESSMENT

Malingered stuttering is a type of stuttering that is conscious, intentional, and feigned by an individual. Malingered stuttering has not been mentioned in this report yet because it is not an authentic type of stuttering. Rather, a person fakes a stuttering disorder to gain benefit, either by obtaining something (i.e. obtaining insurance benefits) or by avoiding something (i.e. avoiding work responsibilities, avoiding accusation in criminal trial) (Shirkey, 1987; Van Borsel, 2014).

Each person who stutters has unique disfluencies or stuttering behaviors, rendering malingered stuttering difficult to decipher and assess. Assessing for characteristics of both organic stuttering and acquired stuttering types further complicates deciphering authentic stuttering from malingered stuttering. Though specific disfluency characteristics are common among people who stutter and may be difficult for a non-

stuttering individual to replicate, stuttering is idiosyncratic in nature and the possibility of uncommon characteristics in authentic stuttering exists. Therefore, it is difficult to assess malingered stuttering because there are no invariable diagnostic markers of stuttering that are true for every person who stutters (Bloodstein, 1988; Shirkey, 1987).

Malingered stuttering assessment includes a careful analysis of speech samples. Spontaneous speech, multiple readings of passages, and prolonged speech should be evaluated. Speech samples provide information on the position of stuttered sounds or words and the grammatical function of the words (content or function word) that are stuttered. Stuttering is generally most frequent on the initial sound or syllable of a word. Furthermore, stuttering usually appears on content words. Both of these qualities should be present in genuine stuttering. Multiple readings of passages are used to assess adaptation and consistency of disfluencies. The adaptation effect is common among those with organic stuttering and forms of acquired stuttering when they are given multiple opportunities to read the same passage. Additionally, those with organic stuttering tend to have consistent disfluencies, meaning they stutter on the same word when it is said on separate occasions. Prolonged speech, or elongating the duration of the sounds produced, is a beneficial strategy for stuttering that usually mitigates disfluencies. The presence of fluent speech when prolonged is a telling diagnostic marker of stuttering and should be included in assessing for malingered stuttering. Presence and types of secondary characteristics can also be assessed during a speech sample by comparing them to common secondary behaviors in stuttering, including observable facial tension, eye blinking and extraneous body movements. Therefore, assessing speech samples for the

adaptation effect, stuttering consistency, the impact of prolonged speech, and secondary characteristics in suspected malingered stuttering are important facets of assessment (Bloodstein, 1988; Seery, 2005; Shirkey, 1987).

Family history, personal history, and interviews with people who know the individual who is stuttering are further assessment tools. Family history is evaluated to determine if a genetic component to stuttering is present. If a genetic component is present, there may be a smaller likelihood that the person is a malingered stutterer. Obtaining personal history, including school and medical records, provides information on the presence of stuttering during childhood or during a previous period of the individual's life. The absence of stuttering mentioned in these reports does not necessarily reveal that the person is fluent and cannot in itself disprove stuttering. Furthermore, the person could have been a covert stutterer for a significant portion of their life, moderating the reference to stuttering in personal records. As such, records are an important component of stuttering assessment but should not stand alone in evaluation. Interviewing friends, family members, and acquaintances that know the individual provides a reference to compare, by examining symptoms that align or conflict, with the reports of the individual. An additional option is reviewing any previous voice or video recordings of the person to assess for similar disfluencies that are present at the time of evaluation (Bloodstein, 1988; Seery, 2005; Shirkey, 1987).

Assessing speech difficulty and stuttering perception aids in malingered stuttering evaluation to further understand situations when the person has difficulty speaking and the attitudes and challenges related to their speech. Difficult situations commonly

reported by those who stutter are phone conversations, speaking to strangers, saying their name, and speaking in high anxiety situations (i.e., giving a presentation, talking to a person of the opposite gender). Assessing attitudes on communication allows for comparison of common emotions experienced during communication that are felt by people who stutter. Common emotions reported include anxiety in social situations that manifest through being silent, avoiding difficult words, and substituting difficult words for easier words. Sensations felt during stuttering are another evaluation area. Sensations commonly reported by those who stutter include tension in their face and "blocked" vocal chords despite knowing the word they want to say (Bloodstein, 1988; Shirkey, 1987).

Examining the presence of stuttering qualities that are similar to a typical person who stutters, in most or all of the areas previously listed, assists in evaluating malingered stuttering. If a discrepancy in an area is found, the individual should be further assessed. No single trait can verify or refute stuttering, so gathering a comprehensive profile of the individual that includes the traits of typical stuttering contributes to a holistic assessment process. Furthermore, establishing malingered stuttering by inconsistencies found during the assessment process is more feasible than finding concrete evidence of malingered stuttering because of the variability of those who stutter (Bloodstein, 1988; Seery, 2005; Shirkey, 1987).

Conclusion

Acquired stuttering is related to neurophysiological or neurochemical changes in the brain and appears independent of organic stuttering. Types of acquired stuttering include neurogenic stuttering, psychogenic stuttering, and medicine induced stuttering. Acquired stuttering is characterized by disfluent speech accompanied by differing secondary characteristics that depend upon the type of acquired stuttering and the individual. The onset of acquired stuttering is typically seen in adulthood, with few cases developing during childhood. However, any person with neurological trauma, psychological brain changes, or using medication may experience acquired stuttering. Differentiating between the three types of acquired stuttering may be a difficult task because neurological trauma, psychological stress, and medication use often coincide. Examining the circumstances around onset, the characteristics, and the response to treatment can lend beneficial information and evidence used to determine the etiology of stuttering (Duffy & Baumgartner, 1997; Krishnan & Tiwari, 2013; Theys et al., 2009; Van Riper, 1964).

Neurogenic stuttering appears subsequent to CNS damage. Different types of neurological trauma can cause stuttering, including traumatic brain injury, stroke, epilepsy, encephalitis, neurodegenerative disease, and tumors. Disfluency and secondary characteristics vary significantly among individuals with neurogenic stuttering. However, significant portions of individuals display disfluencies within a month of CNS trauma. The strongest diagnostic marker, therefore, is disfluency appearance close in time to

neurological trauma (Grant et al., 1999; Krishnan & Tiwari, 2013; Lebrun et al., 1990; Sabillo et al., 2012; Tippett & Siebens, 1991; Van Borsel, 2002).

Psychogenic stuttering is caused by psychological trauma or emotional stress. A person suspected of psychogenic stuttering should be evaluated for previous or current mental health problems. Mental health problems coinciding with the onset of stuttering suggest psychogenic stuttering. The most salient assessment process for psychogenic stuttering includes trial therapy. The individual should be treated with behavioral therapy during the trial therapy phase and the recovery should be noted. A significant improvement in disfluencies following behavioral therapy provides compelling evidence that the stuttering is psychological in nature (Duffy & Baumgartner, 1997; Theys et al., 2009; Ward, 2010).

Medicine induced stuttering is rooted in the presence or excess of medication use. Neurochemical responses or changes in the brain due to medicine trigger disfluent speech. Medications that have been reported to induce stuttering include antipsychotic drugs and drugs used to treat respiratory infections. Medicine induced stuttering can be assessed through the manipulation of medication use. Disfluencies tend to parallel medication use; with increased medicine use, disfluencies increase and with decreased medicine use, disfluencies decrease (Christensen et al., 1996; Grover et al., 2012; Lundgren et al., 2010; Murphy et al., 2015; Van Borsel, 2014; Yadav, 2010).

Associated speech and language symptomatology is frequently observed with acquired stuttering. Aphasia, dysarthria, and apraxia are common coexisting communication disorders that present alongside acquired stuttering. Co-occurring

communication disorders may impact overall communication effectiveness in an individual with acquired stuttering. Comorbid symptomatology differs among various presentation of acquired stuttering and relates to the specific neurological problems of the individual (Helm et al., 1980; Krishnan & Tiwari, 2011; Theys et al., 2008).

Malingered stuttering is an intentional, feigned type stuttering that is used in order to benefit from a diagnosis of the disorder. Malingered stuttering assessment is a complicated, intricate process that includes comparing common stuttering characteristics seen in organic and acquired stuttering to the disfluency presentation of the individual suspected of malingered stuttering (Bloodstein, 1988; Seery, 2005; Shirkey, 1987).

Acquired stuttering is a complex disorder composed of multifarious presentations and characteristics of disfluencies and behaviors. Understanding typical presentations of the types of acquired stuttering assists in the assessment process, and ultimately the treatment plan. A comprehensive view of the characteristics and assessment of acquired stuttering assists a speech-language pathologist in understanding the complex nature of the disorder.

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