

Understanding Etiology of Hearing Loss as a Contributor to Language Dysfluency and its Impact on Assessment and Treatment of People who are Deaf in Mental Health Settings

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Abstract Working with individuals who are deaf in mental health settings can be complex work, necessitating consideration for the difference in language abilities. These differences include not only the language differences of American Sign Language (ASL) and English, but also the range of heterogeneity within the Deaf Community. Multiple influences such as mental illness, medical conditions, language deprivation and the etiology of deafness can impact how a person acquires and uses language. This article will discuss how various causes of deafness create the potential for specific language dysfluencies with individuals who are deaf in mental health settings. The article will also discuss the use of communication assessments to examine specific language dysfluency patterns and attempt to offer possible corresponding interventions.

Keywords Deaf · Mental health · Dysfluency · Etiology · Language deprivation · Communication assessment

Introduction

Mental illness can have significant impact on an individual's quality of life. Diagnosis and treatment can be complex when the individual with a severe and persistent mental illness is also deaf, and does not share the same cultural and linguistic foundation as the provider, nor the system. In addition to cultural and linguistic differences, a person who is deaf may experience various language influences

which can create dysfluencies. Causalities for language dysfluency can include medical issues, mental illness, language deprivation, and etiological causes of deafness. This paper primarily focuses on the influence of etiology in deaf people who are mentally ill and the subsequent influence of effective and appropriate communication assessments on treatment. Much of this article draws upon the work of the Alabama Department of Mental Health, which has statewide deaf-specific mental health services.

Few states provide statewide mental health services appropriate for deaf or hard of hearing individuals (Gournaris et al. 2013). Those that do are often challenged with budget and staff shortages, as well as the continual struggle to advocate the need for specialized services in a hearing-centric environment. Those programs that do provide deaf-specific programming must also deal with the challenges of service provision across large geographical areas and clients who have complex and uniquely challenging needs. These clients, often referred to as difficult to serve, are labelled with such terms as Low Functioning Deaf (Bowe 1998), Minimal Language Skills (Leigh 1999), Traditionally Underserved and Language and Learning Challenged (Glickman 2009).

The smaller, localized programs are not spared these shortfalls. In a chaotic environment that demands constant vigilance to ensure financial survival, local programs must take care that they generate enough “billable” hours that can meet payroll and keep the business viable. Despite legal requirements for accessible services (*ADA Title III 1990*), programs rarely have time to think systemically about the various peripheral issues related to service provision, such as how dysfluency, its origins and lack of intervention, confound service effectiveness.

Both deafness and mental illness can impact language use and/or acquisition. Certain mental illnesses that cause

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thought disorders also impact language abilities (*Diagnostic and Statistical Manual of Mental Disorders* 2013). Inadequate competency of language affects all areas of life including learning, social relationships, education, employment, rehabilitation and mental health treatment. Dr. Robert Q. Pollard notes that “Psychiatry is unique among the medical fields in that most of the symptoms are conveyed by or through communication, and communication also is the primary method and nature of treatment” (Pollard and Dean 2003). Because clinicians use language and behavioral analysis as diagnostic criteria, those who work with deaf people but do not sign fluently, nor have a thorough understanding of deafness and the parameters of normalcy, can misconstrue the causes and implications of language dysfluency. Subsequently, this lack of understanding can lead to misdiagnoses of psychosis or severe developmental disability, worsen behavioral problems and complicate services to the deaf population.

This paper does not suggest that deafness equates to an inability to acquire and use language fluently. In and of itself, lack of hearing does not create a barrier to the potential to acquire language. Confusion often occurs when uninformed people conflate the ability to speak English—or any other language, for that matter—with cognitive functioning in general.

Lack of exposure to visual language throughout a deaf individual’s lifetime—specifically signed languages—is mentioned by others as an element of the observed phenomena of dysfluency (Mayberry 2002; Crump and Glickman 2011). Inadequate language can exist due to many reasons, including mental illness (Pollard 1998; Thacker 1994, 1998; Trumbetta et al. 2001), medical causes (Klima and Bellugi 1979; Poizner et al. 1987), lack of language exposure/models (Glickman 2007, 2009; Vernon and Andrews 1990), etc. However, the literature pays scant and superficial attention to the etiological cause of deafness in regards to how those specific language patterns of dysfluencies, their impact on the assessments and treatment of mental illness, and even less to modifying treatment approaches.

Regardless, or perhaps because of the pervasiveness of language deprivation, it is critical to consider that in some individuals, the neurological consequences that coexist with the etiology of deafness can have potential impact on language acquisition and use (*Some Causes of Childhood Permanent Hearing Loss* 2006). Some causes of deafness may also create other neurological, medical or psychological problems, any of which can impact language, apart from acquisition (Soren and Druzin 2003), which can be very important to a mental health clinician. How a person became deaf is an important piece of information that helps clinicians understand the clinical presentation of mental illness in deaf people.

In social settings, questions regarding how an individual became deaf are perceived as focusing on a medical or pathological viewpoint, and are not considered culturally appropriate questions. Pursuing that particular line of questioning is often discouraged and may be perceived as being insensitive (Holcomb and Mindess 2008). In a clinical setting, though, “Deaf, How?” can have critical impact on the care and treatment of clients.

People who are hearing are usually born into environments where they are exposed to and learn language. Thus, severe language dysfluency in hearing people often occurs or is assumed to occur as a result of cognitive impairment, traumatic brain injury, or psychosis (Robinson 1991; Crump and Glickman 2011). Language deprivation among hearing people is a rare phenomenon (Gulati 2014). Among congenitally deaf people, however, language deprivation is a more common experience. A small fraction of congenitally, or hereditarily, deaf people are born into families that use some form of visually accessible communication (Karchmer and Mitchell 2004). Another fraction is left essentially with no exposure at all to language (Schaller 1991). The vast majority of people who are deaf fall between those extremes (Pollard 2003).

Being able to tease out where dysfluency is related to lack of language exposure, and where it might relate to neurological sequelae of genetic disease or trauma that also resulted in deafness, is incredibly challenging, even for those who have more experience in, and are more qualified in, looking for and differentiating these causes (Black and Glickman 2005). Glickman (2007, 2009) writes about patterns of language related to deprivation. These common errors include impoverished vocabulary, inability to sequence events in time, lack of indicators related to tense, inadequate story structure, spatial disorganization, unclear references, incorrect syntax, sign repetition, and an increased use of gesture to substitute for poor word development.

In most non-deaf-specific mental health systems, clinicians do not sign fluently, if at all. They are not trained to work specifically with deaf people and rarely have a regular and substantial caseload of deaf people. The idea that severe dysfluency might have a cause other than cognitive disability or psychosis is rarely found in their schema. This is one of the reasons deaf people with mental illness can carry so many varied and often contradictory diagnoses (McEntee 1993). A clinician who does not share the same language as the client will have a difficult time differentiating linguistic patterns associated with language deprivation from those associated with cognitive issues or mental illness. They will assume that the introduction of an interpreter resolves those deficits (Hamerdinger and Karlin 2003; Glickman and Crump 2013; *Interpreting in Mental Health Settings* 2007).

To address those deficits and to reduce misdiagnosis and resulting ineffective treatment, a thorough communication assessment, conducted by individuals trained in language dysfluencies, including the impact of etiologies of deafness, is necessary. This assessment should evaluate the person's language capabilities, so that therapeutic work can be provided in the manner the person can most effectively access. This information should become part of the client's file so that the therapist and interpreter, as well as other professionals working as part of the team are able to have a shared understanding. This practice is required by the Alabama Department of Mental Health and South Carolina Department of Mental Health (*Alabama Administrative Code 2010*; South Carolina 2014).

Specific Etiology-Based Language Patterns

Many etiologies impact language development and use across a broad spectrum. It's also important to note that not every symptom constellation will result in deafness. Additionally, a given symptom set will not impact every person the same way. Not every person who contracts meningitis will become deaf, for example (Richardson et al. 1997). It can be difficult to separate out presumed neurological bases for language deficits from environmental (lack of exposure) deprivation. However, when it is severe, it's reasonable to investigate if there might be underlying neurological compromise, then made worse by inadequate exposure. Hereditary causes are the least likely to produce multiple disabilities, although about 1/3 of those with genetic hearing loss are associated with a syndrome (Usher syndrome, Alport, Waardenburg, etc). In some cases of syndromic deafness, developmental delays, cognitive disabilities, learning problems, and so on—all of which can affect language acquisition and development—will also be present (Smith et al. 1999).

Even deaf children from deaf parents who use sign language can have compromised sign language skills. An example of this is Specific Language Impairment (SLI), which has been shown in two known studies to occur in deaf children at the same rate as hearing children, 5–7% (Mason et al. 2010; Morgan et al. 2007). SLI is diagnosed where a deficit in normal spoken language acquisition is found with no apparent cognitive, social or neurological cause (Leonard 1998). Individuals with SLI may have problems with phonology, grammar (morphology), non-salient morphemes, process linguistic input at a slower rate, have poorer expressive vocabulary, but relatively better receptive vocabulary. Deaf children with SLI may use exaggerated gestures and facial expressions to compensate for poor linguistic competence, use pointing to compensate for poor sentence structures and may use more affective facial

expression rather than using non-manual signals (Morgan et al. 2007).

When an expectant mother is exposed to certain diseases, there may be little or no impact to the mother. Sometimes she will not even be aware that she was exposed and contracted the disease. However, this exposure can have significant impact on the developing fetus. Some examples of such maternal illness or infection include complications as a result of Rh Factor, Rubella, Syphilis, Herpes, Cytomegalovirus (CMV), Toxoplasmosis, Fetal Alcohol Syndrome, Prematurity, Birth trauma, etc. Several of these are grouped together and are referred to by the acronym TORCH Complex (Toxoplasmosis, Other, Rubella, Cytomegalovirus, Herpes) (Soren and Druzin 2003).

Providers in the public mental health system in Alabama are required by state code to have a communication assessment on file for all deaf clients. The assessment tool currently being used is the Communication Skills Assessment developed by Roger Williams and the author of this article (Williams and Crump 2013). As of March, 2016, 265 clients were identified as deaf within the Alabama Department of Mental Health. The top reported deafness-related etiology was Congenital Rubella Syndrome (CRS).

Rubella, or German Measles, results in a well-documented constellation of symptoms referred to as Congenital Rubella Syndrome. During the 1960s in the United States, an epidemic of Rubella occurred, and the large number of children who exhibit symptoms of prenatal exposure to Rubella has often been referred to as the Rubella Bulge, especially in deafness-related literature (O'Donnell 1991). Although not all individuals are impacted in the same way, individuals deafened as a result of Rubella exhibit a variety of symptoms that can progressively worsen throughout their lives.

CRS is interesting to mental health specialists precisely because there are so many symptoms that may be present, manifest later or worsen throughout the entire lifespan of the client. Clinically important emanations of CRS include diabetes, thyroid dysregulation, congenital cardiac problems, intellectual disabilities, autism-like behaviors, dyslexia, developmental delays, cognitive skill problems, visual memory and processing problems, poor balance, dyscoordination, deaf-blindness, renal problems, change in hearing or visual abilities, decline in IQ from childhood, increased premorbid motor and behavioral abnormalities, early menopause, psychological problems and behavioral problems, specifically impulsivity and attention deficits, etc. (O'Donnell 1991).

Language patterns seen in communication assessments of clients known to have Congenital Rubella Syndrome (CRS) suggests there is a predictable set of linguistic abnormalities. These may include brief intermittent periods of language incoherence (similar to, but with a different

origin to incoherence as a psycholinguistic error) in either expressive or receptive language, asymmetrical language in expressive and receptive sign or in written English, use of one modality of sign language expressively and another modality receptively (may use an English-based signing expressively, but understand ASL receptively), signing produced at a slightly slower than normal rate, difficulty learning new vocabulary words, difficulty finding the right word to convey a thought, difficulty expressing and receiving fingerspelled words, some atypical language, comments that diverge from the message, and may copy signs of other people as they are communicating (simultaneously) before responding. As a result, the client may have a need for multiple accommodations (sign language interpreter, captioned materials, modeling, role play, etc.) to assist with re-exposure to material presented.

It is important to emphasize that not all people deafened by Rubella will exhibit all or even any of these symptoms, but the cohort of deaf people born between 1960 and 1965 present for services in the mental health system frequently enough to warrant expressly investigating Rubella as the cause of their deafness. In some cases, later neurological sequelae dysfunction can occur, and as these individuals age, they may experience functional decompensation, such as early onset dementia (O'Donnell 1991), which can impact language and treatment needs.

Another example is Cytomegalovirus, commonly referred to as CMV. CMV is a common and usually harmless form of herpes (to adults) that can cause severe disabilities in newborns, including Cerebral Palsy, vision loss, microcephaly, motor difficulties, developmental delays, mental retardation, learning delays, autism, attention deficit disorder, obsessive compulsive disorder, SLI, and issues with balance. These individuals typically have a shorter attention span, impulse control issues, and a low tolerance for delayed gratification and may also have some significantly different language processing problems (Anderson et al. 1996; Kylat et al. 2006; Dollard et al. 2007).

Approximately 10–15% of individuals exposed to CMV in utero may develop hearing loss. In some cases, babies exposed to CMV will pass a newborn hearing screening yet still soon develop hearing loss. For these children, the hearing loss usually involves one ear initially, and will typically progress to a severe or profound hearing loss. The rate of progression varies, sometimes occurring within a few months, and in other cases it occurs more slowly, taking years to develop. This hearing loss may progress throughout childhood to adolescence and young adulthood. In 10–20% of these children, hearing loss will also involve the other ear. In addition, these children may experience central auditory processing problems, even if their hearing is normal (Anderson et al. 1996; Kylat et al. 2006; Dollard et al. 2007).

Toxoplasmosis can occur through exposure to *Toxoplasma gondii*, a protozoan parasite, and may result in multiple and possibly severe disabilities including vision loss (eye pain, sensitivity to light, tearing of the eyes, blurred vision), brain damage, abnormal enlargement, or microcephaly, seizures, cognitive disabilities, confusion, lethargy, memory loss, weakness on one side of the body, speech and language disorders, global delay on language development, and vocabulary deficits (*Toxoplasmosis Report* 2003).

Another prenatal syndrome reported among deaf recipients of mental health services in Alabama is Fetal Alcohol Syndrome Disorder (FASD). FASD can also cause vision difficulties, impulsivity, and low muscle tone or limbs may be floppy. FASD can cause deficits related to language such as: poor short-term memory, inconsistent memory and knowledge base, poor judgment, information-processing disorder, poor ability to perceive patterns, poor cause and effect reasoning, inconsistent ability to link words to actions, poor generalization ability, and expressive or receptive language disorders. They may have poor spatial awareness, resulting in a failure to cross the midline (e.g., reaching for something to the left with one's right hand) and may have less detailed language than peers. These individuals can repeat information back as if it is understood, when, in fact, the information is not. Concrete examples of this have been reports by parents who say that their children can repeat a rule, and even tell what might happen if it is broken, and then break it a minute later. When the child is reprimanded, they do not understand why the parent is upset ("FASD: The Course," 2007).

Language deficits documented through communication assessments, conducted by Office of Deaf Services Staff, have shown a recurring pattern of expressive skills being superior to receptive skills. In particular, what is being reported is that the deaf client with FASD may exhibit an ability to grasp parts of a concept, but not process the whole message. This, again, is clinically significant in determining whether the dysfluency is developmental in origin, or the result of psychosis.

Infants with a hearing loss who are born prematurely often have physical and psychological ramifications (e.g., developmental delay/cognitive or intellectual disability, cerebral palsy, and learning and emotional disabilities), issues with hyperactivity, distractibility, and restlessness, etc. ("Premature Birth," 2011).

A frequently cited cause of childhood deafness is meningitis (Richardson et al. 1997). There are various forms of meningitis. The literature to date indicates that bacterial meningitis has been correlated to language related issues (dysfluency). Studies that have been conducted have typically excluded deaf individuals, because dysfluency can also occur because of lack of language exposure, prenatal

and perinatal cause or trauma, or other issues (Pentland et al. 2000).

It has been demonstrated that individuals who contract bacterial meningitis may experience delayed language, expressive and receptive abilities may differ, and often, that expressive skills can be superior to receptive skills (Pentland et al. 2000). In this situation, when the clinician or interpreter matches the client's output without knowing that comprehension is impaired, treatment can be confounded.

Additionally, these individuals may struggle with understanding metaphors/idioms and jokes and riddles. They may have difficulty with American Sign Language discourse rules, such as turn taking. They may exhibit impaired inferential reasoning, struggle with sentence assembly or have difficulty comprehending ambiguous sentences. They may not be able to handle figurative language, and have trouble recreating sentences and making inferences. Additionally, short-term memory loss, lower verbal intelligence, and reading difficulties may be present. Acquisition of language and the skills needed to build on what language they do have may be impaired. Also, impaired visuo-spatial functions, hyperactivity, distractibility, impulsivity, and inability to solve non-routine problems have been reported. All of these have practical consequences (Pentland et al. 2000; Schmidt et al. 2006).

As mentioned previously, inferential reasoning can be impaired. This can affect ability to function in society when the deaf person cannot infer others' intentions and appropriately modify their own behavior accordingly. As a result the child may behave inappropriately due to the fact that they have not perceived, or accurately interpreted, another's meaning.

There is a concern that clinicians unfamiliar with deafness and with normative behavior within and outside of the Deaf Community may misdiagnose deaf clients as psychotic based on observed phenomena that is better explained by meningitis.

Children or adults who are deafened by Traumatic Brain Injury (TBI) may also experience dysfluency as a consequence. People who have suffered TBI may have difficulty with understanding or producing language, or with more subtle aspects of communication such as body language (*Traumatic Brain Injury* 2002). Some other complications associated with TBI include impaired attention; disrupted insight, judgment, and thought; reduced processing speed; distractibility; and deficits in executive functions such as abstract reasoning, planning, problem-solving, and multi-tasking (Hall et al. 2005).

Not a lot of attention has been paid to the interplay between cause of deafness and treatment for mental illness. This is unfortunate because a deaf person living with mental illness and manifesting some sort of language dysfluency will more likely have impaired cognitive and

psychological functioning. It is also more likely that clinicians inaccurately diagnose these consumers and they live in more restrictive settings than hearing people with comparable functioning potential (Misiaszek et al. 1985; Pollard 1994; Glickman 2007). Clinicians unaware of the complexities involved, looking at various and often interconnected influences on language and development, will miss subtle cues and indications.

Interpreters Working with Clinicians in Mental Health Settings with Dysfluent Consumers

Interpreters, by dint of education and enculturation may attribute any language dysfluency observed as a consequence of lack of language exposure. Interpreters have not traditionally been explicitly taught to work with deaf people who are dysfluent, let alone develop an understanding of causes of dysfluency and its potential impact on treatment. Subsequently, they may not provide the clinician with an accurate description of what is happening linguistically. As a result of a lack of specialized training, the interpreter may exacerbate problems of misdiagnosis by normalizing language output, therefore leading the clinician to make wrong assumptions, misattributing psycholinguistic errors as normal language variations within the Deaf Community, or utilizing more conservative interpreter techniques and strategies such as voicing in a first person simultaneous method, glossing, etc. that may lead to inaccurate determinations by the therapist (Hamerdinger and Karlin 2003; Glickman and Crump 2013; *Interpreting in Mental Health Settings* 2007).

Clinicians, regardless of their own sign language fluency, who routinely work with deaf people, are not typically trained in language dysfluencies and specifically how causes of deafness may impact language skills. Hearing clinicians, who must rely on interpreters likely not trained in mental health work, are usually not even aware of the interplay of the underlying dynamics involved and may naively trust that the interpreter is conveying all the information necessary. This can be further confounded by adjudging through hearing norms, what information they do receive. The result is inaccurate diagnoses and ineffective treatment (Crump and Glickman 2011; Hamerdinger and Karlin 2003).

Observations and Recommendations

Data from Alabama's Communication Skills Assessment in 2016 has allowed for the assessment of specific language patterns related to etiology and a better understanding of how language dysfluency can confound treatment. Some of the observations thus far include:

- the impact a specific etiology might have on the language development and fluency of a person who is deaf,
- considerations for how this language development and language use have impact on clinical work,
- issues that language or interpreting approaches might have for sign fluent therapists and interpreters in regards to these language considerations,
- strategies for an interpreter to articulate the unique language patterns to hearing clinicians with no cultural or linguistic framework for the discussion, and no understanding of the potential impact that various causes of deafness may also cause on language,
- the root of a deaf client's language dysfluency can impact clinical work with that specific client,
- consideration for strategies that are available for improving language competencies when they are a result of neurological consequence of etiology,
- confluence of multiple causes of language dysfluency which can impact social behaviors, ability to learn, and successful service provision,
- strategization of language instruction and development for clients based on the cause of dysfluency, and
- different approaches which should be developed and structured based on the particular type(s) of causative dysfluency.

Each of these patterns related to etiological presentation of deafness and subsequent co-morbid neurological sequelae and its potential relationship to language dysfluency is a ripe area for further research. In spite of the slow trickle of information on the relationship between cause of deafness and language dysfluency, there is much we do not know regarding patterns of dysfluency, and approaches for expressive and receptive language have yet to be developed into best practice. With the addition of the language disorder diagnosis in the DSM-5 (2013), the ability to accurately assess dysfluency becomes more critical. As discussed in this paper, a communication assessment needs to be conducted by appropriate and highly specialized individuals, and guide all aspects of treatment. An additional recommendation for best practices includes clinicians, deaf or hearing, whether they are sign-fluent or not, being aware of the probability of complex, mitigating factors that influence language patterns and diagnoses. Another recommendation is that interpreters working in mental health settings should be specifically trained for that work with special attention to language dysfluencies.

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