Speaker Profiling Persons with Communication Disorders

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Abstract

This paper examines speaker profiling and the large, heterogeneous communication disordered population. Speaker profiling is addressed as it pertains to the deaf community, persons with immature and regressive speech, adult stuttering, psychogenic voice irregularities, and neurogenic communication disorders. The type and severity of a communication disorder can be an important consideration when speaker profiling, and people with communication disorders have certain traits and characteristics that can provide valuable forensic information.

INTRODUCTION

Speaker profiling, a branch of criminal profiling, is the use of deductive reasoning to reach logical conclusions about a suspect or perpetrator based on his or her speech and language patterns. Tanner (2007), Culbertson and Tanner (2005), and Tanner and Tanner (2004) propose that speaker profiling addresses 1) voice prints, 2) accent and dialect analysis, 3) the speech of intoxicated persons, 4) deception detection using voice stress analysis, and 5) forensic interviewing and interrogation. This paper addresses speaker profiling of suspects or perpetrators with communication disorders.

According to Rubin (2000), in industrialized countries, the percentage of people with communication disorders ranges between 5% and 10%. In non-industrialized countries, the incidence and prevalence of communication disorders vary greatly because of vague and imprecise definitions of what constitutes a communication disorder. With the population of the United States at approximately 300 million, a reasonable estimate suggests that 30 million Americans have a communication disorder. Bello (1995) reports that roughly 1 in 6 Americans have a communication disorder, yielding about 50 million persons with speech, voice, language, and hearing disorders. In this paper, speaker profiling is addressed as it pertains to the deaf community, persons with immature and regressive speech, adult stuttering, psychogenic voice irregularities, and neurogenic communication disorders.

SPEAKER PROFILING: DEAF PERSONS

The complete loss of hearing in both ears is rare; most individuals have some residual hearing and can sense air vibration. In the deaf community, there are two social and political philosophies concerning deafness: Oralists and Manualists. The “Oralists,” believe deafness is a disability that limits social interaction and vocational opportunities. When the disorder is detected and evaluated, parents ascribing to this philosophy place their children in special education programs, and take advantage of all resources available to minimize the social, educational, and psychological effects of the communication disorder including hearing aids and cochlear implants. Children are also taught lip reading and given intensive speech therapy to learn how to talk. Deaf individuals ascribing to the Oralists Philosophy seek full integration into society and view deafness as a treatable disability.

In contrast to the Oralists Philosophy, “Manualists,” do not want communication inclusion with the hearing-world. They believe manual communication, finger spelling and sign language, is the natural way for deaf persons to communicate. They are content with being members of the deaf community and do not feel stigmatized. They embrace sign language, which is a true language. Manualists believe that deaf persons are a repressed linguistic minority, similar to other repressed social groups, and many are active politically and socially.

When speaker profiling deaf individuals, a distinction must be made about deaf persons with regard to when the deafness occurred, and whether they ascribe to the Oralists or Manualist Philosophies. Those persons with a postlingual onset deafness, after the development of speech and

language, are likely to have the same social and psychological makeup as others with major communication disorders. Because of their communication disorder, people with postlingual deafness are likely to feel inferior to the normal population regarding their disability, suffer similar negative reactions by society, and experience frustration at their difficulties communicating (Tanner, 2003). However, it is unlikely that these individuals will identify strongly with the larger group of deaf persons, particularly those ascribing to the Manualist Philosophy.

If the speaker is born deaf, or if it occurred shortly thereafter, the effects of the disorder on speech and language development will be all-inclusive. Because no speech sounds will have ever been heard by prelingually deaf person, their speech attempts will be profoundly distorted. Congenitally deaf speakers, if they have speech at all, will have speech sound production that is nasal, monopitch, and distorted. Most speakers with prelingual deafness will identify with the Deaf Culture more than with the hearing population. Speakers who are Manualist and identify with the Deaf Culture are likely to share many of the beliefs, attitudes, and behaviors of other alleged repressed social groups. They tend to seek out mates and friendships within their own social milieu, resent outside social, political, and educational intrusion, lack trust in outsiders, and suspect the motives of many persons in the hearing world.

SPEAKER PROFILING: PERSONS WITH IMMATURE AND REGRESSIVE SPEECH

The most common types of immature speech are "lisping," substituting the "th" for the /s/ speech sound such as occurs in "thee" for "see," and "lalling," substituting the /w/ for /r/ speech sound such as occurs in "wabbit" for "rabbit." Developmentally, 75% of children have mastered correct production of /r/ and /s/ speech sounds in the initial, medial, and final positions of words by 4 years, 6 months, and 4 years, 9 months respectively. Ninety percent of children have mastered these speech sounds in two of three positions of words by 6 years-of-age (Tanner, Culbertson, and Secord, 1997).

Lisping in adult males has been associated with effeminate homosexuality. The motion picture industry often uses speech stereotypes to develop characters and to advance plots and storylines (Tanner, 2001). Hollywood continues to propagate the stereotype of the effeminate male homosexual as one with the immature speech pattern of a lisp. Notwithstanding general public perceptions, there are no valid and reliable scientific studies showing a link between lisping and male homosexuality. There is no scientific evidence that males who are homosexuals lisp more frequently than the general population, lisp more than heterosexuals, nor is lisping in a male child suggestive of homosexual tendencies. With regard to lalling, cartoons, movies, and televison sometimes use the /w/ for /r/ substitution for its humor value and to portray a character as being chronically befuddled, e.g., Warner Bros.' cartoon character, Elmer Fudd (Tanner, 2003). As with the lack of scientific evidence about lisping, there is no evidence showing adults who substitute the /w/ for /r/ speech sound have lower intelligence nor are they more bewildered than adults with normal articulation.

There are three reasons why an adult may talk with some of the speech characteristics of a child. First, he or she may have developed the speech disorder as a child and it was never discovered. Although rare in industrialized countries, some children with speech disorders slip through the system and become adults with communication disorders. Second, some children have their communication disorder identified and evaluated. Even so, the treatment is completely or partially unsuccessful. Not all communication disorders are amenable to successful treatment, and some children do not respond to therapies. Third, because of the psychological defense mechanism and coping style of regression, some adults revert to childlike speech, or for social reasons, they prefer to talk abnormally. They have the ability to talk normally, but consciously or subconsciously engage in childlike speech.

There are several speaker profiling assumptions that can be made about immature and regressive speech in adults. Adult speakers with longstanding articulation errors such as lisping and lalling will have likely spent years in speech therapy as school children. As such, part of their identity and self-concept will be that of a communication-disordered individual. However, in these cases, the therapies will have been unsuccessful in significantly minimizing or removing the disorders. Accompanying the identity and self-concept of these adult communication-disordered persons will be the sense of inferiority in speaking situations shared by most persons with a history of speech, voice, language, and hearing disorders, but also at least some guilt for the negative therapy outcomes. Often, adults with lisping and lalling will have had a long history of suffering negative social reactions to their immature speech patterns.

Regression is a retreat to a more secure and comfortable

level of adjustment (Stuart, 1998). This psychological defense and coping style reduces anxiety by allowing the person to become more dependent, and to return to thoughts, attitudes, and behaviors that he or she has outgrown. People utilizing the psychological defense and coping style of regression seek and find comfort in dependent relationships. Regression to more secure and comfortable thoughts, attitudes, and behaviors may include changes from adult to childlike speech patterns. Speaker profiling of adults with childlike speech patterns involves addressing possible reasons for their regression and immature speech production. Aronson (1990) notes that regressive speech patterns can involve phonatory and resonatory systems and include aberrant articulation with reduced mouth opening. "Regressive speech serves the purpose of relieving the person from the responsibility of relating to others on an adult plane. It says, in effect, that the person does not wish to be regarded as an adult with the responsibilities for mutual interaction that an adult relationship entails" (Aronson, 1990, p. 139).

Adult-onset of lisping and lalling are likely the result of the psychological defense and coping style of regression, or an attempt by the speaker to create a new identity. When a period of mutism accompanies or precedes the adult onset of lisping and lalling, and when associated with psychological trauma and shock, they are likely a manifestation of hysteria. A hysterical psychological disorder involves extreme, volatile emotions, and is often accompanied by attention-seeking behavior such as the adult-onset of lisping or lalling.

Some individuals may lisp for social reasons. As noted above, while there is no demonstrated scientific link between lisping and male effeminate homosexuality, some male adults may lisp because it is part of an acquired identity. Because of society and the motion picture industry's linking of lisping and effeminate male homosexuality, they may speak with a lisp to portray their sexual orientation, preference, and identity. Just as some people wear types of clothing to show affiliation with certain groups, a lisping male may be showing his association with the homosexual subpopulation by his speech patterns. In this sense, the lisp is a nonverbal form of communication showing a desire for social inclusion into the male homosexual subpopulation.

SPEAKER PROFILING: ADULT PERSONS WHO STUTTER

Stuttering has afflicted speakers presumably since humans began to talk. Stuttering occurs in all languages, although it is more prevalent in some cultures than in others. Approximately 1-3 percent of the U.S. population currently stutters, and upwards of 5% of Americans report periods of their lives where they stuttered. Occurring mostly in males, stuttering ranges from mild inconveniences for some, and for others, devastating disabilities that have and continue to significant psychological and social effects on their lives. Analysis of this speech disorder can provide valuable insight into the personality of the speaker, and contribute substantially to his or her profile.

Because everyone has disfluent speech, i.e., occasional repetitions, prolongations, and hesitations in their ongoing speech, clearly defining and describing true stuttering is important. True stuttering is a combination of defective speech production and the stutterer's reaction to the disorder. Generally, stuttering consists of too many sound, word, and phrase repetitions, prolonging or stretching-out utterances, blocks in the smooth flow of speech, and the speaker's reactions to them. The reactions include visible avoidance and escape behaviors as the speaker tries to force his or her speech mechanism into functioning and to get out of the stuttering moment. Most confirmed stutterers report anxiety and associated negative emotions before, during, and after the moment of stuttering. For many, particularly those with long-standing and severe stuttering, the disorder affects their personalities, vocational choices, self-concepts, socialization, and self-esteem. "Stuttering affects a person in many ways, but one of the most critical social activities an adult individual undertakes is that of finding a partner and maintaining an intimate relationship." (Linn and Caruso, 1998, p. 12).

Besides the visible, eye-squints, hand slaps, jaw tremors, etc., and the audible repetitions, prolongations, and blocks, the frequent bouts of anxiety and associated negative emotions experienced by the stutterer are pivotal to their psychological reactions to speaking. Fear, dread, guilt, and apprehension are common associated negative emotions to speaking, be they speeches to large audiences or one-to-one acts of communication. Although stuttering authorities differ on whether there is a clinically definable "stuttering personality," most agree that stuttering is aversive and negatively affects the person suffering from it. Studies of large samples of stutterers compared to normal-speaking subjects have found that, as a group, people who stutter are not dramatically affected psychologically by the disorder. However, research using the Minnesota Multiphastic Personality Inventory (MMPI) shows that stutterers fall

within the range of normal and resemble troubled persons seeking counseling.

During forensic interviews and interrogations, the severity of the stuttering will increase as the stress levels increase. Stuttering is often a barometer of how much stress and anxiety the speaker is experiencing. As stress and anxiety increase during forensic interviews and interrogations, there will be consequent increases in the number of repetitions, duration of prolongations, and the length and severity of blocks.

For many stutterers who have undergone extensive therapy, the treatment outcome is "control" of the dysfluencies rather than a complete "cure" for the disorder. Many adults who stutter control their stuttering by being relaxed during speaking, having light, easy, articulatory contacts, and a slow, purposeful, singsong manner of talking. Additionally, for the majority of people who stutter, the more nonthreatening the audience, the more improved is their fluency. For example, most stutterers can talk fluently to pets, babies, and aloud to themselves while having more problems with police officers, lawyers, and interrogators. Simply repeating what has been spoken often results in normal fluency; it is likely that a person who stutters will be able to repeat a confession when stated by an interrogator. When speaking in unison with another subject, the stutterer is also likely to be fluent. Ambient noise such as a loud interview and interrogation room, or an office in a precinct with high background noise levels are likely to cause some stutterers to be more fluent. Miscellaneous factors such as illnesses, alcohol intoxication, and fatigue may also affect the fluency levels of some people who stutter.

Adult stutterers are likely to suffer from reduced self-esteem, particularly in speaking situations. They are also likely to be socially awkward. Typically, adult stutterers have had years of suffering the pangs of rejection and ridicule. They will have the self-concept of a stutterer and the accompanying reduced self-esteem about social interaction that accompanies it. The seeds for this learned inferiority will have been planted early in their lives on playgrounds and in classrooms, and propagated by family, friends, and society's reaction to stuttering in adulthood.

In the not-so-distant past, some authorities believed that stuttering is a manifestation of a psychological disorder, and that to cure it without the benefit of psychotherapy could cause the patient to be a danger to self or others. It was thought that stuttering is a safe expression of a deep-rooted psychological disorder and if removed without a psychotherapist providing another alternative, the person who stuttered would resort to a dangerous alternative expression of his or her psychological distress. Although symptom substitution is a well-established psychiatric phenomenon, there are no scientific studies showing that it applies to stuttering. "The scientific literature has never reported a case where eliminating stuttering resulted, directly or indirectly, in injury to anyone. No incident has been reported in which a person was cured of stuttering and replaced it with a destructive or harmful psychological substitute" (Tanner, 2003a, p. 37).

SPEAKER PROFILING: PERSONS WITH PSYCHOGENIC VOICE IRREGULARITIES

The voice production mechanism is highly sensitive to musculoskeletal tension. As a result, an increase in anxiety and consequent elevation in physical tension as a response to stress can affect a person's pitch, loudness, and voice quality. While psychological factors do not cause cleft lip and palate, vocal paralysis, and laryngeal cancers, many voice disorders are either completely or partially psychogenic in nature. Voice disorders, such as screamer nodules (noncancerous growths on the vocal cords) and chronic laryngitis (longstanding inflammation of the vocal cords) occur, at least in part, because of the effects of external or internal stressors and the person's attempts to deal with anxiety. For example, hysterical aphonia, the complete loss of voice due to a conversion reaction is wholly psychogenic in nature, while voice disorders related to vocal strain and abuse, such as vocal nodules and contact ulcers, are partially psychogenic.

Vocal nodules in adults are more common in females and associated with vocally abusive behaviors. However, there is usually a psychogenic component involving the speaker's reaction to stress. "They are talkative, socially aggressive, and tense, and have acute or chronic interpersonal problems that generate tension, anxiety, anger, or depression. Even when the nodule may be the sole result of abuse from singing or other strenuous vocal activity, it is often found that these were not the only factors responsible for the vocal abuse; these patients had also entered a period of their lives in which concomitant emotional stress had surfaced" (Aronson, 1990, pp. 125-126). Vocal contact ulcers are ulcerations, breaks in the tissue of the vocal folds, and are associated with pain and a foreign body sensation in the throat. Contact ulcers occur more frequently in males than females, possibly due to some males talking in an

unnaturally lower pitch to appear more masculine and powerful. Chemical exposure, heavy smoking and alcohol consumption, and vocal abuse have been associated with contact ulcers. Approximately one-third of voice disorders can be traced to some degree of increased musculoskeletal tension and voice abuse (Cooper, 1973).

As noted above, hysterical aphonia is the complete loss of voice not due to organic, physical factors. Most authorities on hysterical aphonia attribute it to the conversion of significant emotional distress into a physical symptom, i.e., the loss of voice. Hysterical aphonia usually manifests itself in the patient's remarkable ability to voice during laughing, humming, coughing, and throat clearing, and his or her reverting to whispering during speech. Symbolically, patients with hysterical aphonia present with loss of voice to represent problems in social interaction, usually involving family members and significant others in sexual relationships. A hysteria-based psychogenic voice disorder is an explicit signal sent by the person to seek medical, psychological, and family support for an underlying psychological turmoil.

For speaker profiling purposes, persons with voice disorders resulting totally or partially from psychogenic factors can be divided into two groups: tension-based and hysteria-based. The tension-based group consists of individuals with increased musculoskeletal tension due to their attempts to deal with external or internal stressors. In this group of subjects, psychogenic factors partially contribute to the voice disorder, however, vocal strain and abuse are equally or more important to their development. Vocal nodules and contact ulcers are the primary voice pathologies occurring in this group of speakers. The voice qualities of hoarseness and intermittent breaks in the ability to produce voice are the usual perceptual features of tension-based voice disorders. Some individuals will also have voice fatigue, particularly late in the day.

Typically, the person with a vocal nodule is a talkative, socially aggressive, middle-aged female. According to Aronson (1990), she suffers from persistent interpersonal problems that generate musculoskeletal tension, anxiety, depression, or anger. It is likely that she is also a singer without formal training. The typical person with a vocal contact ulcer is a talkative, socially aggressive, middle-aged male. "The classic profile of the contact ulcer patient is a male in his forties who uses his voice intensively in his daily life and is either a lawyer, teacher, minister, actor, or salesman. In personality, he is tense and hard-driving, and is often under chronic stress" (Aronson, 1990, p. 128).

SPEAKER PROFILING: PERSONS WITH NEUROGENIC COMMUNICATION DISORDERS

Neurogenic communication disorders are a large group of speech, voice, and language disorders caused by brain damage and neuromuscular impairments. The role of brain damage and neurological impairments in antisocial personalities, psychopaths, and criminal behavior is well-established. Smith and Kling (1976) review the association of dyssocial behavior with brain function including the role of frontal and temporal lobe lesions in aggressive behavior, violence, and impulse disorders. Lykken (1995) discusses genes, evolution, and brain dysfunction in the development of the antisocial personality, psychopaths, and perpetrators of violence. Stoff, Breiling, and Maser (1997) provide a comprehensive overview of brain damage and neurological dysfunction in antisocial behavior.

TRAUMATIC BRAIN INJURY

The majority of individuals with traumatic brain injuries have communication disorders either as a direct result of damage to the speech and language centers, indirectly as a consequence of reduced or disordered awareness, or a combination of factors. Regardless of the specific manifestations of the brain damage, accurate profiles can be drawn for the typical adult person likely to suffer a traumatic brain injury. First, according to Hickey (1997), the presumptive causes of TBI are motor vehicle accidents (50%), followed by falls (21%), and assaults or other type of violence (12%). Kraus and Sorenson (1994) show that the age group of 15-24 is at the highest risk for traumatic brain injury. All studies show that single males are at more risk for TBI than married persons or females. Alcohol and drugs are involved in more than 50% of traumatic brain injuries. Typically, the traumatic brain-injured person has a poor education and is employed, if at all, in a low-paying risky job. Although there are no studies showing traumatic brain injured persons have lower intelligence, they are likely learning disabled which has contributed to academic and other learning frustration throughout school.

A patient with TBI induced amnesia may be suffering from memory loss for events occurring before the injury, retrograde amnesia, or have difficulty remembering new information, anterograde amnesia, or both. The person with retrograde amnesia may have selected memory deficits, including committing crimes and specific events related to them, or complete loss of memory for weeks, months, years,

and even decades. The person with anterograde amnesia will have problems remembering events since the traumatic brain injury including forensic interviews and interrogations, statements made about a crime, and even meeting forensic interviewers and investigators. Memory loss and disorientation go hand-in-hand. The person with disorientation may be completely confused about time, place, person, and predicament, or partially disoriented to one or more of aspects of reality. Reports from persons with posttraumatic amnesias must be considered suspect because of memory loss and disorientation.

Most persons with significant traumatic brain injuries have impaired "metacognition" or "thinking about thinking." According to Gillis (1996), they have difficulty recalling information, organizing, planning, and monitoring behaviors, and problems with inhibition. Metacognition is sometimes referred to as mental executive functioning and has been linked to damage to the frontal and temporal lobes.

The speaker profile of a person with significant traumatic brain injury and communication disorder(s) is a young unattached learning-disabled male earning low wages and working in a risky job with reality testing, memory, and orientation problems. He also is likely to be a risk-taker, engages in substance abuse, and has probably been repeatedly hospitalized for traumatic head injuries. While the traumatically brain-injured person may be consciously deceptive, it is also likely that he cannot accurately remember events related to a crime or incident.

NON-TRAUMATICALLY INDUCED NEUROGENIC COMMUNICATION DISORDERS ARISING FROM FRONTAL LOBE DAMAGE

The psychological reactions associated with non-traumatically induced neurogenic communication disorders has been studied extensively. Tanner (2003b), Gordon, et al. (1996), Tanner and Gerstenberger, (1996), Gainotti, (1989), Robinson, et al., (1988), Lipsey, et al., (1986), Robinson, (1986), Robinson, et al., (1985), Gordon, et al., (1985), Sackeim and Weber, (1982), Sackeim, et al, (1982), Robinson and Benson, (1981), Gasparini, et al., (1978), Black, (1975), Weinstein and Puig-Antich, (1974), Gainotti, (1972), Weinstein, et al., (1966), and others. Neuroscientists have discovered that many of the psychological reactions caused by brain injury are similar to the psychological reactions seen in emotional disturbances not caused by brain damage. Consequently, many non-traumatically induced brain injured speakers show many of the psychological reactions displayed by normal, psychologically disturbed persons.

Frontal lobe brain damage, as is also seen in the expressive language disorder of aphasia (loss of language due to brain insult), has been linked to dyssocial, aggressive, violent, impulsive, and other behaviors. Many authors have tied damage to the frontal lobes of the brain with the propensity for violence, impulsivity, aggression, hostility, and many other potentially antisocial behaviors. Some have concluded that most violent offenders have frontal lobe irregularities or brain damage, and may have inherited the propensity for criminal behavior. However, regarding non-traumatically induced frontal lobe damage and cooccurring neurogenic communication disorders, there are four typical psychological concomitants: emotional lability, perseveration-echolalia, anxiety-depression, and catastrophic reactions. These reactions are likely to be part of a speaker profile for a person with apraxia of speech (deficits in purposeful planning of speech motor acts), spastic dysarthria (paralysis of the speech production mechanism), and/or expressive aphasia arising from non-traumatically induced frontal lobe damage.

A speaker with spastic dysarthria may be emotionally labile. Emotional lability, sometimes called "pseudobulbar emotional lability" is usually seen as unwarranted and uncontrolled crying. Technically, emotional lability refers to wide swings in emotions and can include uncontrolled laughing and other emotional behaviors. A speaker with emotional lability usually does not have "inappropriate" emotions; his or her emotions are "exaggerated." Once the emotional response is set into motion, it is often difficult to stop. When a speaker has spastic dysarthria and is highly emotional, his or her emotions are likely to be partially or completely exaggerated as a result of the brain and neurological injury.

Perseveration is the tendency to continue an activity for a longer time than is warranted by the significance of the stimulus prompting it. Echolalia, a manifestation of perseveration, is the automatic repeating of what has been said. Both are associated with damage to the frontal lobes of the brain. A subject with frontal lobe brain damage who cannot seem to break from a topic or automatically repeats what has been spoken is likely suffering from the effects of the brain and neurological damage, and is not necessarily being evasive, deceptive, or uncooperative for self-serving reasons.

When a subject has left frontal lobe brain damage and

accompanying neurogenic communication disorders, he or she is more likely to suffer from anxiety-depressive disorders than a person who has had right hemisphere or temporal lobe damage. When a person has brain injury to the anterior (front) part of the left hemisphere, he or she is likely to endure anxiety-depression that will be long-lasting. However, if the brain damage is in the posterior part of the left hemisphere, the subject is likely to display indifference, lack of awareness of his or her disabilities, and even euphoria. It also appears that the smaller the brain lesion, the more likely the subject will be aware of his or her disorder, and suffer more anxiety, frustration, and depression. For some yet-to-be-determined reason, females have more strokes involving the anterior part of the brain, and males suffer more from strokes affecting the posterior region. Consequently, speakers with nonfluent, predominantly expressive aphasia are likely to anxious-depressed women, while speakers with fluent, predominantly receptive aphasia are likely to be indifferent, unaware, and euphoric men.

A catastrophic reaction can be described as an anxiety attack, and the more nonfluent the speech output, the more likely the subject will suffer from it. A subject with nonfluent aphasia has trouble remembering words for expression and difficulty producing them. Speech is produced with hesitations, struggle, revisions, and complications, and because the understanding parts of the brain have not been damaged, the speaker is aware of the nonfluencies and errors. Consequently, nonfluent aphasia is frustrating, especially when an aphasic person is placed in a situation where important and critical speaking is required such as a forensic interview and interrogation. Some aphasic persons may suffer from one or more catastrophic reactions in these situations. When too much pressure builds, they may strike out physically and verbally. Most catastrophic reactions occur when stimuli overwhelm the speaker and rapid responses are required of him or her. The subject with expressive aphasia who strikes out when under pressure to respond verbally is doing so, at least partially, because of the brain and neurological damage.

NON-TRAUMATICALLY INDUCED NEUROGENIC COMMUNICATION DISORDERS ARISING FROM TEMPORAL LOBE DAMAGE

Neuroscientists have linked temporal lobe brain damage to dyssocial, aggressive, violent, impulsive, and other behaviors. Temporal lobe brain damage causes receptive aphasia often with jargon output. Kling (1976) provides a review of animal and human behavioral changes, particularly aggression, in temporal lobe damage. Tardiff (1997), and others, have found organic brain disease affecting the temporal lobes to result in a propensity for violence. Temporal lobe epilepsy has been associated with purposeless violence as have brain infections, diseases, and strokes. Temporal lobe damage and many accompanying antisocial behaviors are well-accepted aspects of traumatic brain injury. The amygdala, part of the reticular activating system which is deep in the temporal lobe, appears to play an important role in regulating violent, aggressive, and other antisocial behavior. This is not to say that patients with receptive aphasia are necessarily violent, aggressive, and antisocial; they simply have damage in similar regions of the brain. However, there are three non-traumatic induced temporal lobe damage and cooccurring neurogenic communication disorders psychological concomitants: denial of disability, projection, and jargon-confabulation. They are likely to be a part of the speaker profile for a person with predominantly receptive aphasia and auditory processing disorders.

When a subject produces aphasic jargon speech, he or she is likely to have damage to Wernicke's and adjacent areas of the left temporal-parietal lobes. Denial of disability, anosognosia, is also a frequent occurrence in many persons with this type of neurogenic communication disorder (Weinstein et al., 1966; Weinstein and Puig-Antich, 1974, Tanner, 2006b). Denial of disability and other aspects of the communication situation account for the persistent meaningless speech in many persons with aphasic jargon. The subject with persistent partial or complete jargon output is engaging, at least in part, in the psychological coping style and defense of denial, and does not appreciate the significance of the communicative situation. Many denying persons with persistent jargon also engage in the coping style and psychological defense of projection.

Projection is the attributing of one's own intolerable thoughts and feelings to another person. Projection, in the extreme, plays an important role in the genesis of psychotic behaviors and delusions. The denying, projecting subject with aphasic jargon speech denies that he or she is communicatively impaired, that communication is malfunctioning, and additionally, projects the problems onto the listener. Many subjects with aphasic jargon output utter nonsense and act as if the listeners would simply try harder, they would understand the perfectly normal attempts at communication. Because of the neurogenic communication disorder, denial, and projection, most if not all, statements

made by the subject is meaningless for legal and forensic purposes.

Confabulation is more than just lying; it is remarking about an event without consideration of the facts related to it, and in the extreme, disregard of the reality associated with an event. During forensic interviewing and interrogation, confabulation is the speaker giving answers to questions with little or no regard to their truthfulness. The subject engaging in confabulation makes up false stories, often to fill in unknown gaps in a real occurrence, and may or may not be self-serving. Confabulation is related to the coping style and psychological defense of fantasy and a form of psychological escape. It can also be reporting of excessive, wish-fulfilling daydreaming that has become so chronic that the speaker believes the events and confuses true and false memories. The subject with partial jargon aphasia may confabulate as part of denial-projection and disregard of the facts surrounding an event. A confabulating subject may be reporting a false memory where he or she remembers an earlier traumatic experience that had been repressed. Regardless of the organic or functional causes of confabulation, by definition, the veracity and accuracy of a confabulating speaker's reports are suspect.

SUMMARY

With approximately one-in-ten persons having a communication disorder, there is forensic value in examining the psychological and social makeup of speakers from this subpopulation. Although generalities are difficult to make about the heterogenous communication disordered population, many speakers suffer from reduced self-esteem, particularly in speaking situations. Some members of the Deaf Community consider themselves a repressed minority due to their adherence to sign language and rejection of other forms of communication. Some speakers engage in immature and regressive speech for identification purposes and to avoid having mature relationships. Speakers who stutter, especially those with adult-onset, may have underlying psychological reactions causing or contributing to the dysfluencies. Many voice disorders are psychogenic in origin. Speakers suffering from neurogenic communication disorders have predictable psychological reactions related to type of brain damage and sites of lesions.

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References

r-0. Aronson, A. (1990). Clinical voice disorders: An interdisciplinary approach. New York: Thieme.
r-1. Bello, J. (1995). Hearing loss and hearing aid use in the United States. Communication facts. Rockville, MD: American Speech-Language-Hearing Association.
r-2. Black, F. (1975). Unilateral Brain Lesions and MMPI Performance: A Preliminary Study. Perceptual and Motor Skills, 40: 87-93.
r-3. Brownell, R. (2000). Expressive One-Word Picture Vocabulary Test (3rd ed). Novato, C.A.: Academic Therapy Publications.
r-4. Cooper, M. (1973). Modern techniques of vocal rehabilitation. Springfield, Ill.: Charles C. Thomas.
r-5. Duffy, J. (1995). Motor speech disorders. St. Louis: Mosby.
r-6. Fuller, G. N. and Goodman, J.C. (2001). Practical review of neuropathology. Philadelphia: Lippincott, Williams, and Wilkins.
r-7. Gainotti, G. (1989). The Meaning of Emotional Disturbances Resulting from Unilateral Brain Injury. In Emotions and the dual brain, G. Gainotti, and C. Caltagirone (Eds). New York: Springer-Verlag.
r-8. Gainotti, G. (1972). Emotional Behavior and Hemisphere Side of the Lesion. Cortex, 8: 41-55.
r-9. Gasparini, W., Satz, P., Heilman, K., and Coolidge, F. (1978). Hemispheric Asymmetries of Affective Processing as Determined by the Minnesota Multiphasic Personality Inventory. Journal of Neurology, Neurosurgery and Psychiatry, 41: 470-473.
r-10. Gillis, R., & Pierce, J. (1996). Mechanism of traumatic brain injury and the pathophysiologic consequences. In R. Gillis (Ed), Traumatic brain injury rehabilitation for speech-language pathologists. Boston: Butterworth-Heinemann.
r-11. Gordon, W., Hibbard, M., and Morganstein, S. (1996). Response to Tanner and Gerstenberger. In Forums in Clinical Aphasiology, C. Code (Ed). London, UK: Whurr Publishers.
r-12. Gordon, W., Hibbard, M., Egelko, S., and Diller, L. (1985). The Multifaceted Nature of the Cognitive Deficits Following Stroke: Unexpected Findings. Archives of Physical Medicine and Rehabilitation, 66: 338.
r-13. Hall, B.J., Oyer, H.J., and Haas, W. H. (2001). Speech , language, and hearing disorders: A guide for teachers. Boston: Allyn & Bacon.
r-14. Herrmann, M. and Wallesch, C. (1989). Psychosocial Changes and Psychosocial Adjustments with Chronic and Severe Non-Fluent Aphasia. Aphasiology, 3(6): 513-526.
r-15. Hickey, J. (1997). Craniocerebral injuries: In J. Hickey (Ed), The clinical practice of neurological and neurosurgical nursing (4th ed.). Philadelphia: Lippincott.
r-16. Johnson, W. (1930). Because I stutter. New York: Appleton-Century-Crofts.
r-17. Kling, A. (1976). Frontal and Temporal Lobe Lesions and Aggressive Behavior. In W. Smith and A. Kling (Eds). Issues in brain/behavior control (pp. 11-22). New York: Spectrum Publications.
r-18. Kraus, J. and Sorenson, S. (1994). Epidemiology. In J. Silver, S. Yudofsky, and R. Hales (Eds) Neuropsychiatry of traumatic brain injury. Washington, DC: American Psychiatric Press.
r-19. Linn, G.W., & Caruso, A.J. (1998, July-September).

Perspectives on the effects of stuttering on the formation and maintenance of intimate relationships. Journal of Rehabilitation, 64(3): 12-14.
r-20. Lipsey, J., Spencer, W., Rabins, P., and Robinson, R. (1986). Phenomenological Comparison of Poststroke Depression and Functional Depression. American Journal of Psychiatry, 143:4.
r-21. Lykken, D.T. (1995). The antisocial personality. Hillsdale, N.J.: Lawrence Erlbaum Associates, Publishers.
r-22. Martin, F. and Clark, J. (2003). Introduction to audiology (8th ed.). Boston: Allyn & Bacon.
r-23. National Center for Health Statistics. (1988). Current estimates from the National Health Interview Survey, United States, 1988. Vital and health statistics, Series 10, No. 173 DHHS Publication No. (PHS) 89-1501.
r-24. Owens, R., Metz, D.E., & Haas, A. (2000). Communication disorders: A life span perspective. Boston: Allyn & Bacon.
r-25. Robinson, R., Lipsey, J., Bolla-Wilson, K, Bolduc, P., Pearlson, G, Rao, K., and Price, T. (1985). Mood Disorders in Left-Handed Stroke Patients. American Journal of Psychiatry 142:12.
r-26. Robinson, R., Boston, J., Starkstein, S. and Price, T. (1988). Comparison of Mania and Depression After Brain Injury: Causal Factors. American Journal of Psychiatry, 145:2.
r-27. Robinson, R. (1986). Depression and Stroke. Psychiatric Annals, 17(11): 731-740.
r-28. Robinson, R., and Benson, D. (1981). Depression in Aphasic Patients: Frequency, Severity, and Clinical-Pathological Correlations. Brain and Language, 14: 282-291.
r-29. Ruben, R.J. (2000). Redefining the survival of the fittest: Communication disorders in the 21st century. Laryngoscope,
110: 241-245.
r-30. Sackeim, H., Greenberg, M., Weiman, A., Gur, R., Hungerbahler, J., and Geschwin, N. (1982). Hemispheric Asymmetry in the Expression of Positive and Negative Emotions: Neurological Evidence. Archives of Neurology, 39: 210-218.
r-31. Sackeim, H. and Weber, S. (1982). Functional Brain Asymmetry in the Regulation of Emotion: Implications for Bodily Manifestations of Stress. In Handbook of Stress, L. Goldberger and S. Breznitz (Eds). New York: Macmillan.
r-32. Sacks, O. (1990). Seeing voices: A journey into the world of the deaf. New York: Vintage Books.
r-33. Smeltzer, D., Nasrallah, H., and Miller, S. (1994). Psychotic disorders. In J. Silver, S. Yudofsky, and R. Hales (Eds) Neuropsychiatry of traumatic brain injury. Washington, DC: American Psychiatric Press.
r-34. Smith, W. L. and Kling, A. (1976). Issues in brain/behavior control. New York: Spectrum Publications.
r-35. Stoff, D.M., Breiling, J., and Maser, J.D. Handbook of antisocial behavior. New York: John Wiley & Sons.
r-36. Stuart, G. (1998). Self-Concept Responses and Dissociative Disorders. In Principles and Practice of Psychiatric Nursing (6th ed.), G. Stuart and M. Laraia (Eds). St. Louis: Mosby
r-37. Herrmann, M., Barrels, C., and Wallesch, C. (1993). Depression in Acute and Chronic Aphasia: Symptoms, Pathoanatomical-Clinical Correlations and Functional Implications. Journal of Neurology, Neurosurgery, and Psychiatry, 56(6): 672-678.
r-38. Tardiff, K. (1997). Evaluation and Treatment of Violent Patients. In D. Stoff, J. Breiling, and J.D. Maser (Eds). Handbook of antisocial behavior (pp. 445-453). New York: John Wiley & Sons.
r-39. Tanner, D. and Gerstenberger, D. (1996). Clinical Forum 9: The Grief Model in Aphasia. In Forums in Clinical Aphasiology, C. Code (Ed). London: Whurr Publishers
r-40. Tanner, D. (2001). Hooray for Hollywood: Communication disorders and the motion picture industry. ASHA Leader, 6(6): 10.
r-41. Tanner, D. (2003a). Exploring communication disorders: A 21st century introduction through literature and media. Boston: Allyn and Bacon.
r-42. Tanner, D. (2003b). Eclectic perspectives on the psychology of aphasia. Journal of Allied Health, 32, 256-260.
r-43. Tanner, D. (2006a). Case studies in communication sciences and disorders. Upper Saddle River, N.J.: Pearson Merrill Prentice Hall.
r-44. Tanner, D. (2006b). An advanced course in communication sciences and disorder. San Diego: Plural Publishing.
r-45. Tanner, D. (2007). Redefining Wernicke's area: Receptive language and discourse semantics. J Allied Health, 36:63-66.
r-46. Tanner, D., Culbertson, W., and Secord, W. (1997). The developmental articulation and phonology profile (DAPP). Oceanside, C.A.: Academic Communication Associates.
r-47. Van Riper, C., & Erickson, R. (1996). Speech correction (9th ed). Boston: Allyn & Bacon.
r-48. Weinstein, E. and Puig-Antich, J. (1974). Jargon and its Analogues. Cortex, 10:75-83.
r-49. Weinstein, E., Lyerly, O., Cole, M., and Ozer, M. (1966). Meaning in Jargon Aphasia. Cortex, 2: 165-187.
r-50. Wise, R.J.S., Greene, J., Büchel, C., & Scott, S.K. (1999). Brain regions involved in articulation. Lancet, 353: 1057-61.
r-51. Yeoman, B. (1998, November/December). Wrestling with words. Psychology Today, 31(6): 42-47.

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