Medical Perspectives in the Treatment of Stuttering

Glyndon Riley
California State University, Fullerton, CA

Gerald Maguire
David Franklin
Tony Ortiz
University of California, Irvine

V an Riper tells the story of a young man who came to him for stuttering treatment. The young man asked if there was a pill to cure stuttering. Van Riper pulled a bottle of pills from his desk and said, “Take one of these pills each day and you will never stutter again.” The young man laughed, so Van Riper said, “You are laughing because we both know the idea of a pill curing stuttering is foolish. Let’s get to work.” If a medication is developed that is useful for people who stutter (PWS), it will not be a “cure” that can work in the absence of other forms of therapy. The same thing can be said about medication for heart disease, schizophrenia, or any other complex disorder. The purpose of this article is to provide a rationale for including some medical aspects of stuttering in our diagnosis and treatment protocols along with established therapies.

The definition we choose for stuttering affects all of our diagnostic and treatment decisions. If we define stuttering using strict behavioral terms, then we diagnose it using objective measures such as percentage of syllables stuttered and duration of the stuttering event. The treatment goal is stutter-free speech. An expanded definition of stuttering may include some cognitive aspects such as avoidance of sounds, words, or situations and some attitudinal aspects such as secondary gains from the stuttering. This expanded definition leads to treatment goals that address these cognitive and attitudinal issues as well as the stuttering behavior.

If we add medical aspects such as neurochemical conditions in the brain to our definition, then the management of these medical issues becomes a routine part of stuttering assessment and treatment.

ABSTRACT: How we describe the various components of a communication disorder determines the protocols used in its diagnosis and treatment. The purpose of this article is to provide a rationale for including some medical aspects in the description of stuttering. For many years, stuttering was considered a learned behavior caused by environmental conditions. Genetic findings provided an impetus to expand this description. Recent brain imaging studies are providing preliminary evidence of some differences between people who stutter and those who do not that expand the description of stuttering even further. Treatment implications and suggested directions for future research are included.

KEY WORDS: stuttering, physiology, medication, adults, children

BACKGROUND

The history of stuttering theory parallels the history of other disorders such as schizophrenia and autism. First, the cause was thought to be in the environment, especially the attitudes and behaviors of the mother. These disorders were labeled “functional” or “emotional,” and mother got most
of the blame for their existence. Certainly stuttering fell into this category in the 1940s and 1950s when it was supposed to start in the ears of the listeners.

Second, genetic evidence began to emerge for each of the disorders that made the environmental cause seem incomplete. In schizophrenia, for example, monozygotic (identical) twins had pair-wise concordance rates of approximately 50%, whereas dizygotic (fraternal) twins and brothers had a concordance rate of approximately 10%. For stuttering, the genetic influence was even stronger. Monozygotic twins had a concordance rate of approximately 60% or more, and dizygotic twins and brothers had a concordance of 20% to 26% (Felsenfeld, 1997 for review). In addition, the fact that stuttering (like most childhood speech and learning disorders) occurs in three times as many boys as girls implies that something in addition to environment is part of the etiology.

Third, neurological findings led to proposed new theories that included medical aspects of the disorder, and treatments were modified to include medical management (especially medications) as part of the overall treatment of each disorder.

Stuttering shares many similarities with Tourette’s syndrome, which is a dopamine-based, basal ganglia disorder (Wolf et al., 1996). Both stuttering and Tourette’s begin in childhood, follow a waxing and waning course, and are made worse by anxiety, and occur in a 4:1 ratio of male to female.

HEMISPHERIC DIFFERENCES

Orton (1927) and Travis (1931), using the just invented electroencephalograph (EEG) technology, developed a theory that speech motor control is overactive in the right hemispheres of PWS so that a definitive left hemisphere control is not established. This theory provided the rationale for studying brain laterality in PWS.

Moore (1991) summarized much of the research that was inspired by the Orton-Travis theory. A wide spectrum of research methods, including dichotic listening, average evoked responses, hemispheric EEG alpha asymmetries, bimanual hand tasks, and sequential finger tapping, concurred in reporting that PWS exhibit differences in hemispheric asymmetries compared with people who do not stutter (PWNS).

Brain Imaging Laterality Findings

Pool, Devous, Freeman, Watson, and Finitzo (1991) reported that cerebral activation in PWS as measured by single-photon emission tomography (SPECT) was greater in the right than in the left hemispheres in selected regions related to speech and language. In addition, PWS had a global reduction in regional cerebral blood flow (rCBF) as compared to matched PWNS. Wood and Stump (1980) had done a SPECT study of stuttering, but they only included one developmental person who stuttered with fluency induced by haloperidol, and their results were difficult to interpret.

Wu et al. (1995) obtained positron emission tomography (PET) scans on PWS and controls using choral reading to induce fluency. They reported that PWS had reduced cerebral activity in the Broca’s and Wernicke’s areas during stuttering and this asymmetry was normalized in the fluent condition.

Fox et al. (1996) obtained PET scans on PWS and controls during a resting condition and during solo reading (stuttering for the PWS) and choral reading (fluent for PWS). During the stuttering condition, the PWS had greater cerebral activation in the right superior lateral premotor cortex. This asymmetry was normalized during the fluent condition. Braun et al. (1997) reported similar differences in PWS during selected speech and language tasks.

Kroll, De Nil, Kapur, and Houle (1997), who also used PET methodology, reported that PWS had increased activation in the left motor cortex that was different from PWNS prior to treatment, but that this pattern was normalized following intensive fluency-shaping treatment.

These five brain imaging studies produced similar but not identical results. See Watson and Freeman (1997) for a comprehensive review of the similarities and differences of the findings. Taken together, these articles indicate that PWS have over-activation in the right primary motor areas or reduced activation in the left primary motor areas (Broadman cortical areas 44, 45) during a stuttering condition that is normalized during a fluency condition. Boberg, Yeudall, Schopflocher, and Bo-Lassen (1983) reported PET scan data showing that stuttering reduction was associated with normalization of hemispheric lateralization. This improvement was maintained for several months following the intensive stuttering therapy program.

A DOPAMINE HYPOTHESIS

Reduced Caudate Functioning

Wu et al. (1995) first reported that people who stutter exhibit a “permanent left caudate hypometabolism that is a possible trait marker for stuttering” (p. 504). Using PET with [18F] deoxyglucose (FDG) as the marker, this study compared four PWS with four controls who did not stutter. The PWS had approximately 50% less FDG uptake than the controls. These results were among the first indications that stuttering was associated with reduced striatal efficiency, which led to the suggestion that dopamine might play some part in this reduction.

We view stuttering as a multidimensional, multiple-risks disorder that includes such aspects as social interactions, emotional reactions, auditory processing, language production, and speech motor programming. Smith and Kelly (1997) described this perspective in more detail. The results of studies that evaluate the possible relation of dopamine to stuttering need to be considered in a context of these parallel, contributing systems. The purpose of this paper is to examine reported and recent, unreported research findings that strengthen or weaken the acceptance of a dopamine hypothesis related to stuttering. As with any
useful hypothesis, the one under consideration can be accepted or rejected based on currently used research designs. Four types of research are reported: (a) PET comparisons of dopamine levels in PWS compared with PWNS, (b) the effects of dopamine blocking medications on stuttering, (c) correlations of stuttering reduction with PET scan changes following medication, and (d) the effects of speech motor training on stuttering.

The hypothesis under consideration states that PWS demonstrate differences in brain chemistry and that one of these differences is excessive dopamine in the striatal (subcortical) regions of the brain. For a summary of early, related findings, see Riley, Wu, and Maguire (1997) and Wu, Riley, Maguire, Najafi, and Tang (1997). Recently, Costa and Kroll (2000) provided an update for physicians who need to apply medical findings to treat PWS. They stated that “research data and the effectiveness of dopamine receptor antagonists in DS [developmental stuttering] seem to support the theory of a hyperdopaminergic origin [of stuttering]” (p. 1849).

In a study by Wu et al. (1997), dopamine levels in the striatum of three PWS (moderate to severe stuttering) were compared with levels in six PWNS. All of the subjects were male. PET used 6-FDOPA as a marker of presynaptic dopaminergic activity. Stuttering subjects showed a 100% to 300% increase in dopamine activation in areas related to the hypothesis under consideration. The authors concluded that “elevated 6-FDOPA uptake in ventral limbic cortical and subcortical regions is compatible with the hypotheses that stuttering is associated with an overactive pre-synaptic dopamine system in brain regions that modulate verbalization” (p. 767). Figure 1 shows the normal controls in the top row and the PWS in the bottom row. The extra brightness in the photo of the PWS indicated that they had more uptake of FDOPA.

Maguire, Gottschalk, Riley, and Franklin (2000) conducted a double-blind, placebo-controlled study of the effects of low doses of risperadone versus a placebo on stuttering using 16 PWS (12 male and 4 female; mean age 40:8 [years:months]). There were no significant differences between groups in age or gender. Stuttering severity ranged from mild to very severe in each group at baseline. The percentage of syllables stuttered (%SS) was reduced from 9.6 to 4.7 (50.4%) by the active medication, as compared with a reduction from 7.0 to 5.1 (27.1%) by the placebo. The mean scores on the Stuttering Severity Instrument–3 (Riley, 1994) were reduced by 7.8 (from 25.3 to 17.5) following the medication and 3.5 (from 24 to 20.5)

Figure 1. Average 6-FDOPA uptakes at 4.1 (MHS 8), 3.4 (MHS 9), and 2.8 (MHS10) above the canthomeatal line for normal controls (top row) and people who stutter (bottom row).
following the placebo (Figure 2). All of the measures except duration reached statistical significance at \( p = < .05 \).

Lavid, Franklin, and Maguire (1999) reported that, in an open clinical trial, stuttering was reduced in three children who stuttered (ages 9–14) following administration of low doses of olanzapine (Zypraxa). The Clinical Global Impression (CGI) judges stuttering to be very mild (1) up to very severe (6). Each child entered the study with a rating of severe (5) or very severe (6) stuttering. All three children ended the study, including 2–5 months follow-up, with very mild stuttering (1).

In another study, Maguire, Wu, Franklin, Ortiz, and Riley (2001) scanned the brains of five PWS using [18F] deoxyglucose FDG as a marker prior to and following administration of low dosages of risperadone. As a group, the PWS demonstrated increased striatal activation following the medication. In addition, the individual responses correlated with the stuttering reduction as measured by the CGI. Correlations in such a small number of subjects should be viewed with caution, but they are in the predicted direction.

A larger study of effects of olanzapine on stuttering and on striatal activation is underway. Twenty-four PWS are involved in a study to test for the effects of this medication on stuttering, glucose uptake, and dopamine activation. So far, the blind has been broken on 12 subjects, 6 on active medication and 6 on placebo. Frequency (%SS) was reduced an average of 8.4 by the medication compared with 4.1 by the placebo. The mean of the three longest stutters was reduced an average of 1.5 by the medication and .5 by the placebo (Figure 3). These results are preliminary and their interpretation must await further data.

Riley and Ingham (2000) reported that children’s stuttering frequency was reduced by approximately 40% following speech motor training (SMT), a treatment that targets motor programming but does not target stuttering behaviors. This reduction was accompanied by an increase in vowel length and a reduction in the intervocalic interval, which suggested that SMT might have been associated with more efficient motor programming. Extended length of utterance treatment resulted in more reduction in stuttering than SMT, but did not change acoustic durations. These results show that changes associated with speech motor programming also reduce stuttering to some extent; therefore, improved activation of the striatal brain region is implied. If so, this mechanism would parallel the findings that dopamine reduction in the striatum is related to reduced stuttering.

During stuttering, cortical speech areas (Wernicke’s and Broca’s) are hypometabolic but elevate in metabolism during induced fluency, possibly as a compensatory mechanism. Stuttering is associated with a persistent hypometabolism of the striatum that is perhaps associated with elevated dopamine activity. This reduction in striatal activity may compromise speech motor programming.

Figure 2. Percentage improvement following a 12-week trial of risperadone (dark bars) or placebo (lighter bars).

Figure 3. Percentage improvement following a 12-week trial of olanzapine (dark bars) or placebo (lighter bars).

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**Note.** %SS = percentage of syllables stuttered, % Time St. = percentage of time spent stuttering, SSI-3 = Stuttering Severity Instrument-3.

Note. %SS = percentage of syllables stuttered.
Medications such as risperadone and early analysis with olanzapine reveal that these medications may improve symptoms associated with stuttering. Pre- and post-medication PET scans reveal striatal activity to be increased after treatment.

**TREATMENT AND RESEARCH IMPLICATIONS**

The novel antipsychotic dopamine-blocking medications such as risperadone and olanzapine are possible effective pharmacological treatments to be used as an adjunct to a comprehensive management of stuttering and warrant further investigation based on these preliminary studies. These changes in cortical and subcortical activation that possibly are related to stuttering occur in the context of social, cognitive, and emotional conditions and need to be viewed as only one part of a very complex, multidimensional process. Even if some medications can be demonstrated to be useful in reducing the frequency and severity of stuttering, they will not provide a total treatment. Rather, each person who stutters needs to work with a speech-language pathologist who specializes in stuttering to work out a comprehensive therapy program in which the use of a given medication may play a part.

**Case Studies of Medical Treatment of Stuttering**

Three case studies illustrate the uneven response of PWS to dopamine-reducing medications. Recall that the amount of stuttering reduction was correlated with the cerebral brain activation changes associated with risperadone. Olanzapine seems to be more promising and has fewer side effects, but these cases illustrate wide variation in responsiveness.

**Case 1.** A young adult female who stutters received 2.5 to 5 mg of olanzapine for 12 weeks. Her stuttering frequency was reduced from 10.9%SS to 1.6%SS, a reduction of 85%. The duration of her longest stutterings was reduced from 4.3 to .5 (90%). This improvement was maintained while she received the same dose of medication for 6 months. This case was representative of half of the participants in the preliminary group in a double-blind, placebo-controlled study. This person reports that the effort she expends on stuttering during conversations is greatly reduced by the medication, but she still engages in the avoidance of certain sounds and words. These avoidance behaviors need to be addressed by appropriate cognitive therapy.

**Case 2.** A young man who stutters received 2.5 to 5 mg of olanzapine for 12 weeks. His %SS was reduced from 12.1 to 10.8 (11%), and the duration of his longest stutterings was reduced from 2.1 to 1.5 seconds (29%). PWS in the placebo group had their %SS reduced an average of 39% and the duration of their longest stutterings reduced 23% so any improvement in stuttering for Case 2 could not be attributed to the active medication. This case was representative of approximately half of the PWS in the study. This person needs treatment designed to meet his individual fluency needs that remain after medication.

**Case 3.** A man who stutters participated in two controlled studies of the effects of dopamine-reducing medications on stuttering. Risperadone reduced his %SS from 13 to 7 and his duration from 13 to 5 seconds. Upon withdrawal from medication, his stuttering frequency increased to 12%. Olanzapine then reduced his %SS from 12 to 7. Duration of the longest stutterings was reduced from 13 to 5 seconds by risperadone and regressed to 11 seconds after the medication was withdrawn. Olanzapine reduced the duration from 11 to 1.5 seconds. Thus, the overall effect of the medication was to reduce stuttering frequency by approximately 46% and severity by approximately 90%. Traditional behavioral, cognitive, and attitudinal stuttering therapy may be useful in helping this individual make maximum use of the fluency gains that he achieved through medication.

Because the effects of medication have been inconsistent, research needs to be designed that will identify conditions that are predictive of the likelihood of a positive response to a given medication. Useful designs might include such variables as:

- genetic history of (a) stuttering, (b) other speech and language conditions, and (c) other medical conditions such as Tourette’s syndrome, depression, bipolar disorder, and so forth (familial patterns of stuttering may possibly overlap with some of these conditions);
- medical history;
- history of family and environmental conditions, including communication style and social history; and
- details of stuttering severity of PWS, including behaviors (%SS, duration, etc.), cognitive issues (avoidance, etc.), and attitudinal issues (using stuttering to manipulate or as a reason for perceived shortcomings).

Because medication seldom provided a comprehensive, complete treatment, research needs to be designed and conducted that includes both traditional (behavioral, cognitive, and attitudinal) stuttering treatment and a selected medication in order to describe the roles of each approach better.

**Implications Regarding Speech Motor Training**

The proposed mechanism by which stuttering improvement is related to a reduction of dopamine in the striatum also can provide a logical paradigm for the reduction of stuttering by SMT that targets speech production at the motor programming level (Riley & Ingham, 2000; Riley & Riley, 1999). Stuttering is reduced an average of approximately 50% by SMT without targeting stuttering behaviors directly. McClean (1997) outlined certain functional components of the motor system and related these elements to the production of fluent and disfluent speech. He included a motor programming phase in his model that may parallel the subcortical regions implicated in the
The dopamine theory presented above. Caruso, Max, and McCloywry (1999) provided a rationale based on motor learning theory that explores the perspective that stuttering should be considered as a motor speech disorder. SMT methods are compatible with motor learning theory in that they operate primarily at a subconscious, automatic level of production rather than at the conscious level. Studies that combine SMT with medications in children may be instructive.

ACKNOWLEDGMENTS

The authors wish to recognize the contributions of each person whose work has contributed to our knowledge of the possible medical aspects of stuttering. Many of these people are included in the reference list. We appreciate the contributions of the people who served as subjects in the University of California, Irvine stuttering research projects. We want to express our appreciation to Sherri Wolff, MA, CCC-SLP for her careful measurements of stuttering frequency and severity.

This research was funded in part by grants from Janssen Pharmaceutical Company and Eli Lilly Pharmaceutical Company and from a significant individual contribution.

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Contact author: Glyndon Riley, Riley's Speech and Language Institute, 218 West Main, Suite 102, Tustin, CA 92780. Email: glynriley@home.com