Translating Recent Research into Meaningful Clinical Practice

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ABSTRACT

The goal of this article is to provide a synopsis of research, both basic and applied, that can improve the evaluation and treatment of stuttering in children and adults, as well as counseling of clients, families, and other professionals who interact with the person who stutters. Relevant basic research has informed genetic contributions to stuttering and possible neurological substrates of the disorder. Several treatment approaches to stuttering have recently been published, with varying degrees of apparent efficacy. There has also been increased attention to therapeutic outcomes that go beyond the components of the specific therapies themselves, which have the potential to improve therapy outcomes. Finally, several subtypes of fluency disorders have been profiled in published reports; these may enable clinicians to better understand less typical presentations of fluency disorder.

KEYWORDS: Stuttering, research, genetics, speech-motor skills, language, diagnostics, therapy, counseling

Learning Outcomes: As a result of this activity, the reader will be able to (1) summarize recent research in the genetic, neurological, and motor bases of stuttering; (2) articulate its relevance to patient counseling; (3) summarize recent research in the earliest stages of stuttering, including factors predicting remission and recovery, assessment concerns, and effective programs for intervention with this age group; (4) evaluate the current research on evaluation and treatments for older age groups, including both traditional and novel approaches; (5) discuss the utility and limitations of pharmacological agents and altered auditory feedback devices for stuttering treatment; and (6) utilize the appropriate new codes for distinguishing subtypes of stuttering in the ICD and DSM revisions.

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ARE WE ANY CLOSER TO UNDERSTANDING WHAT CAUSES STUTTERING?

Among the most common questions that any clinician gets from clients and their families is, "Why do people stutter? What causes stuttering?" Although we do not yet have a definitive answer to this question, we are in fact coming closer and closer to consensus on some of the factors that appear to be involved in the etiology of stuttering. As we continue to accumulate data about the genetic, neurological, and physiological features of stuttering, we also are able to discard many more popular notions about stuttering, such as that it emerges as a response to emotional trauma, that it is learned, or that it is a reflection of emotional or mental disorder. Such information can be very helpful in orienting people who stutter (PWS) and their families more positively and constructively to the problem of treating the stuttering and remove unmotivated feelings of guilt and blame, which often emerge if clients or families are asked to explain their own beliefs about the nature of stuttering.

GENETICS

The notion that stuttering is genetically transmitted has been investigated for many years,[1] because inheritance profiles have been readily observable, even to most clinicians. There has been a large amount of basic genetic research devoted to isolating the specific genetic locus. Candidate genes are being identified in areas of chromosome 12[2–5] but are not found in the majority of PWS. Because different investigations have targeted multiple suspect genes, there is likely to be more than one locus, with stuttering best described as a polygenetic disorder.

Knowing that a disorder might be inherited does not tell us exactly *what* is being inherited; we would like to know how whatever suspect gene(s) do to develop interventions. It is interesting that some of the candidate genes "make sense" in that broader areas of the suspect genome are also implicated in communication and/or motor disorders (missense mutation found in other speech-language pathology disorders, movement disorders). The newest report[3] is exciting because it implicates a very different molecular "suspect," which up until now has only been implicated as a culprit in lysosomal storage disorders, which usually have such catastrophic metabolic consequences that any effect on communication has not been observable using behavioral techniques. Unfortunately for those looking for "quick fixes" such as gene therapy to "turn the suspect gene off" (a comment I saw posted recently on a listserv), this is not likely to happen in any near future. These are not simple sequences that can be turned on/off because they control broad functions necessary for human metabolic function. Blocking such gene activity would be fatal; thus, there are no "knockout" models or simple gene therapies likely in near future.

SO, WHY SHOULD WE CARE?

It actually seems to still be news that stuttering can be inherited rather than learned or acquired through trauma; in some respects, what is newsworthy about the mainstream coverage is a sense of wonder that stuttering might have biological roots.[6,7] However, given clinical experience that many individuals who stutter, as well as their families, seem unaware of the ongoing progress toward identification of "stuttering gene(s)," discussion of emerging research has the potential to diffuse commonly observed parental guilt over behaviors that might have prompted stuttering, or self- or other blame that targets environmental factors as influential in the etiology of stuttering, because none to date have been identified.[8] It is sometimes both amazing and saddening for me to talk to families that describe a fairly clear pattern of stuttering inheritance, with grandparents, uncles, cousins, and so on, but still ask "What did we do to cause this problem?"

BRAIN STRUCTURE AND FUNCTION

Data on the basic neuroanatomy as well as neurophysiology of stuttering are accumulating at a rapid rate. Studies of both cortical structure and function continue to imply that the substrates of speech-language and coordination differ between PWS and those who are

normally fluent. To provide a full list of studies in the last decade would be daunting, and so the reader is referred to *A Handbook on Stuttering*[1] for a more detailed list. However, in the past few years, both anatomic and functional studies converge on a picture that implicates differences in white and gray matter,[9–15] connectivity between speech-language and motor areas, and ensuing functional differences in speech-language processing between PWS and typical speakers.[16–19] Importantly, some of these studies have "reached down" into the pediatric population to support the interpretation that these difference are etiologic (causal), rather than reflective of adaptations to stuttering over the life span.[9,10,15–19] Such work has the ability to shed light on why some therapies work, as well as why some therapies work for some people and not for others. For instance, there are intriguing differences between people who do/don't respond to altered auditory feedback (AAF), such as that provided by SpeechEasy®,[Q1][20] Anne Foundas and colleagues have examined PWS and typically fluent speakers for evidence of the typical cortical asymmetry one normally finds; for most individuals, areas in the left cerebral hemisphere are anatomically larger than those in the right.[20] However, for some, but not all, PWS, this asymmetry is absent; interestingly, it was this subgroup with what we might call anomalous anatomy that appeared to derive the most fluency benefit from AAF. Of course, no one is recommending that candidates for such devices receive expensive brain-imaging procedures—we will suggest some more pragmatic approaches to determining who might benefit from auditory feedback devices later in this article.

TREATMENT AND PLASTICITY

Even as we accumulate information regarding atypical brain anatomy and activation in individuals who stutter, we are also seeing that treatment appears to normalize many of the atypical functional profiles seen in laboratory studies of untreated adults.[21–24] Thus, biology is not destiny. It may very well be that PWS have atypical neuroanatomy, forcing adaptive functional neurophysiological profiles. However, treatment appears to produce both durable results for speech fluency (as shown later in this article) as well as more typical profiles of neurophysiological activation during speaking tasks. This suggests that learning and automatizing new speaking patterns (as well as affective and cognitive approaches to the speaking task) can, in effect, "rewire" the brain's circuitry for speech production.

This is not a trivial notion for some clients, who have viewed recent publicity regarding the neurological substrates of stuttering with mixed emotions. As seen on many Internet chat lists, although most welcome the science suggesting that stuttering has a physiological rather than psychological basis, some are sobered by the information; many of us have been taught that anatomy is permanent, as is anything that reflects neurological damage of some sort. This is not true, as we are now seeing not only in the field of stuttering but also in related fields in our profession, such as aphasia treatment, where treatment does, in fact, produce differences in cortical processing of speech and language.

Finally, clinicians may want to consider reading and providing patients with an excellent, short summary of relatively recent (2004) research on stuttering available through the Public Library of Science (PLoS) on the Internet.[25] Clients can either go to the PLoS site (http://www.plos.org) and use the key word "stuttering," or clinicians can make copies of the short article for distribution to their clients. The article summarizes both historical views of stuttering as well as recent published research on the nature and best treatment of stuttering.

LABORATORY STUDIES OF BEHAVIOR IN PWS

Much as there is a tendency to yawn over "bench" research in many areas of clinical intervention, such controlled investigation has the ability to both reinforce as well as weaken some notions about the underlying problem that produces stuttering behaviors and that may make them difficult to treat therapeutically. For instance, there are continuing findings that PWS differ from people who do not stutter in terms of motor learning and ability to gain automaticity during motor-training

tasks.[18,20,26,27] Examples of research teams working in this area include Luc DeNil and colleagues at the University of Toronto. Such work has the ability to reinforce some clinicians' beliefs that stuttering therapy cannot rest on "quick tricks" or fixes to retrain or reorient old motor (as well as affective and cognitive) behaviors. Rather, if PWS show consistent deficits in motor-learning tasks or dual-task conditions, it is much less surprising that a great deal of practice, including that which takes place in the real, multitasking demands of everyday work, school, and social interactions, would be required to overcome this area of subtle weakness in addition to overcoming years of habit strength in speech behavior as well as responses to the stuttering moment.

An increasing body of data also suggests subtle differences between language (as opposed to speech) processing in PWS; examples of this can be found in work conducted by Chris Weber-Fox and colleagues at Purdue University.[28–30] Using event-related potentials, a measure of the brain's response to linguistic input, researchers have found differences both in the timing and amplitude of responses in adults who stutter, suggestive of very subtle differences in processing language, even when overt speech is not required. Views that stuttering might emerge partially as a response to language encoding difficulty in young children[31] receive support from such research.

One of the less satisfying aspects of models that proposed that stuttering was in fact a problem of language encoding was that it seemed difficult to explain how the actual features of stuttering emerged from this type of deficit; it seems apparent to most of us that the disfluencies in stuttering do not closely resemble other, "typical" disfluencies that we know arise from sentence planning or lexical retrieval difficulties. However, work done by Anne Smith and colleagues at Purdue University[32–34] as well as others[35] show interesting differences between PWS' and typically fluent speakers' speech motor behaviors under conditions of increased linguistic load. Such work has the ability to explain a "missing link"—how numerous reports of subtle language weakness in children and adults who stutter could manifest themselves as a motor speech disturbance. When combined with work suggesting that there is a high degree of comorbidity between stuttering and clinical diagnosis of language impairment in children[36] and that the child's relative level of language development may be a factor in predicting remission and persistence in early stuttering,[37] clinicians have strong research support for the assessment of language skills during stuttering evaluations, as well as mindful planning of the levels of linguistic challenge that may be required as fluency skills are learned, practiced, and generalized to the "real world."

STUTTERING: WHAT ARE THE ROLES OF NATURE AND NURTURE?

The relative contributions of congenital factors and learning to profiles of fluency and stuttering seen in PWS have always been a question that provokes lively discussion. Before commenting on what the status of this debate is in the current literature, it may actually be valuable to define terms in a careful manner. The argument that there must be *environmental* or *nurture* factors at play in the etiology of stuttering is often raised when seeing research reports of a small degree of discordance for stuttering among identical (monozygotic) twin pairs. This, in turn, is often interpreted as supporting a causal or influential role for a host of environmental factors, such as parenting, life experience, and so on, in eliciting stuttering. It is very important to note that when geneticists do talk about environmental factors in mediating between genetic profiles and individual characteristics, they see this as very different than parenting or experience. For a geneticist, the environment may be the womb or gestational/birth history of the child,[1] and they quite typically observe small degrees of discordance for even strongly genetically transmitted disorders among twin dyads. Currently, we have no data to suggest that the experiences/upbringing of PWS differ from those who do not stutter. Most of this work is not very recent, and there is little to contradict it across the history of stuttering research.[1]

Does that mean that a person's experience has little to do with the characteristics of their stuttering profile, whether viewed in terms of

behaviors, affective reactions, or cognitive components? No. Experience does play a role in the evolution of stuttering behaviors and the reactions and adaptations that PWS make to both speaking and their disfluencies, but it does not appear to play a role in the etiology of the disorder.

THE COURSE OF EARLY STUTTERING: RISK FACTORS FOR PERSISTENCE

There are numerous studies across the years on the general incidence and prevalence values for stuttering in multiple populations and age groups; they converge to suggest that close to 80% of children who begin to stutter (as documented by expert clinicians) recover from it, largely between 2.5 and 4.5 years of age.[1] What is relatively newer is the possibility that more, rather than fewer children actually go through a phase of clinically-relevant stuttering; the true value may be closer to 7.5% rather than the 4% generally quoted.[38] This, in turn, would elevate the probability of stuttering remission for a given child.

Additionally, we have been able to identify a handful of factors that can increase or decrease the likelihood of recovery; these are best viewed as factors that merit assessment and parent counseling rather than guide decisions for treatment because they can only predict proportions of outcomes in a cohort of children rather than the outcome for a specific child. These factors were primarily identified in publications from the Illinois Stuttering Research Project, recently collected into a book-length text by Ehud Yairi and Nicoline Ambrose.[37] The risk factors for persistence that were identified include gender (male), later onset of stuttering symptoms (e.g., later than ~2.5 years), a family history of persistent stuttering rather than recovered childhood stutterers, and, to a lesser extent, the child's relative language abilities (as assessed by standardized tests and screenings) at the time of stuttering diagnosis. The sex ratio was somewhat surprising to many of us older clinicians, because we thought of a girl who stuttered as a relative rarity. What we now know is that, closer to stuttering onset, quite a large number of girls stutter, but they are extremely likely to spontaneously recover.

WHAT THERAPIES WORK?

This is clearly the second of the two most frequent questions about stuttering. However, answering it is not at all straightforward, in many opinions. For example, it may come as a surprise to some that there is a certain amount of debate over how one defines a successful outcome. Although several criteria for evaluating successful therapies are not disputed, such as sample size, objective measurement, follow-up, generalization to out-of-clinic situations, naturalness of treated speech, among others,[1] there is some argument about which aspects of the presenting problem should be targeted by the therapy itself, rather than response to improvements in speech fluency. Put more simply, although most would agree that stuttering is composed not only of core and associated motor behaviors, but also of affective and cognitive reactions to both speaking and stuttering, not all agree whether the ABCs, as they are sometimes called, respond to treatment in the same way. For instance, it is possible to treat by targeting only the behavioral features of stuttering (the so-called Bs, the moments of stuttering themselves) to eliminate them. In this case, a desired outcome would be significantly less or no observable stuttering, which in turn could be thought to directly improve any affective or cognitive reactions to stuttering and speaking. In other views, it is difficult to treat the Bs without working on aspects of the As and Cs, and there may be little guarantee that affective and cognitive features will immediately resolve simply because observable stuttering has become less frequent.

One problem in defining the outcomes of therapy is that we had better or more objective measures of behaviors than we did of affective and cognitive features of stuttering, using devices such as the Stuttering Severity Instrument (now the SSI-4), as well as percent stuttered syllables or words in a variety of speaking tasks. The most recent changes to available assessment measures include new and standardized measures of functional impact, as well as cognitive and affective concomitants of stuttering,

along with a wider age range of clients who can be assessed using these measures. For example, the Overall Assessment of the Speaker's Experience of Stuttering (OASES) by Scott Yaruss and Bob Quesal[39–41] measures four different areas of concern for PWS, including functional impacts on lifestyle and cognitive/emotional reactions to stuttering; it is available in both English and Spanish (with many more languages coming), with reference norms from typically fluent speech available to demonstrate (for caseload inclusion or insurance reimbursement purposes) areas of intervention that go beyond a measure of stuttering frequency.

Similarly, the KiddyCAT[42] is a commercially available test of communication attitudes that has been adapted from the older, well-known Communication Attitude Test that is simple enough to be administered to preschoolers, who already show subtle changes in communicative self-concept from their typically fluent peers.[43] Previously, we had no such measure for this youngest age group of clients. The CT[Q2] itself has now been translated and renormed for a large number of language communities, most recently Italian,[44] Swedish,[45] and Pakistani,[46] and has been used as an outcome measure for several therapy outcome reports for both children[47] and adults[48] that will be discussed in the next section of this report.

As part of a more general social sensitivity to the problems of bullying and teasing among school-aged children, there have also been investigations of the functional impacts of these typical but negative social behaviors on young children who stutter,[49] including work by Marilyn Langevin and colleagues,[50] to document common peer reactions to children who stutter. Such measures may be used to augment justifications for active intervention with young children who stutter on the basis of adverse functional impact.

Many clinicians and their patients struggle to document functional limitations imposed by stuttering, and the measures described above provide tangible results, in many cases norm-referenced documentation of the impact that stuttering has on the individual. These can be much more helpful in justifying caseload inclusion or insurance coverage for individuals who stutter, when arguments are made that simple counts of stuttering do not impact educational, vocational, or social function. Many fields have long had quality-of-life indices that quantify the daily consequences of living with an impairment. Recently, research has produced clear data suggesting the adverse impact of stuttering on quality of life, a factor that may provider stronger justification for third-party coverage of stuttering treatment.[51]

Finally, because it has been hard to objectively document variability of fluency during the wide array of vocational, educational, and social settings that PWS may encounter, a novel approach has been investigated by Shelley Brundage and colleagues: virtual reality. Trials of varying levels of simulated "speaking demand" within-clinic, but moderated by virtual-reality, scenarios have been associated with changes in fluency profiles consistent with patients' self-report,[52] suggesting that it may be possible to appraise, treat, and evaluate fluency in a greater number of real-life settings to improve generalization of treatment results.

TREATMENT OF EARLY STUTTERING

By far, the most thoroughly evaluated and most highly published therapy approach for young children who stutter is the Lidcombe Program, which continues to amass positive reports of beyond therapy stuttering rates that fall well below 2% stuttered syllables, within a reasonably short time frame, and with maintenance of gains on follow-up in the overwhelming majority of cases. A cursory search in a major research database shows ~100 references; of these, we reference those with the most summary and follow-up data for those clinicians not yet acquainted with this parent-administered program of verbal contingencies for fluent and stuttered speech.[53–59] Although the program has had its primary implementation and evaluation in Australia, recent reports from the United States,[60] Canada,[61] Germany,[62] and the Netherlands,[63] among other locales, have reported equally good results. The program has also been

adapted for telehealth administration[64] and has been evaluated for application with slightly older children than originally designed for.[65]

Although the Lidcombe Program is clearly the best-documented, most effective intervention approach for early stuttering, some questions remain to be answered. For example, in one analysis of long-term outcome, the reported rate of "relapse" requiring retreatment was 16%, with another 5% of participants who did not complete the original course of therapy.[54] Such numbers do not vary substantively from some estimates of the rate of eventual spontaneous recovery among children who start to stutter, although the age range of children who are treated often falls outside the most dynamic range for recovery as reported in some studies cited earlier in this article, and individual profiles clearly indicate decreases in stuttering rate that are quite clearly aligned with the onset of intervention.

There has only been one "head-to-head" comparison of children's responses to the Lidcombe Program and another intervention approach based on the Demands and Capacities Model of stuttering (roughly based on changing the child's communicative environment and expectations); it did not show an evident advantage for the Lidcombe Program over the alternative therapy,[63] although sample size makes robust comparisons and evaluation against spontaneous recovery rates difficult.

As might be expected, other approaches to stuttering treatment in the early years also report highly favorable outcomes as well, albeit with much less empirical support. Among the treatments that have offered positive outcome data for working with preschool and younger children who stutter are reports detailed (with information about implementation of the therapy approach) by the Palin Centre in London, which customizes all therapy to the parent/child evaluation findings;[66] a family-centered program developed at the Stuttering Center of Western Pennsylvania by Yaruss and colleagues;[67] and additional approaches for younger toddlers who do not show initial responsiveness to Lidcombe therapy, such as a syllable-timed speech fluency-shaping approach examined by the Australian Stuttering Research Centre.[68]

SOME CONCLUDING THOUGHTS ON EARLY INTERVENTION

Given what we are learning about prognostic indicators for persistent stuttering, what the field now needs are some predictors of who responds to therapy and if they respond to one therapy better than another. The high incidence of spontaneous recovery from early stuttering, combined with evidence of relapse even in this younger age group, creates a daunting prospect for evaluation of single therapies, let alone the comparative effectiveness of competing therapies for possible subsets of stuttering children who differ in risk factors for persistence.

TREATMENT OUTCOME DATA FOR OLDER PWS

A positive way to introduce this section is to note that several approaches work well, depending upon how you define "well" (in terms of whether behaviors, affect, and cognitive components of presenting symptoms are equally well ameliorated) and how durable we expect improvements to be. It is fair to say that, as a field of professionals (speech-language pathologists), we are still negotiating what aspects of the speaker's complaint are most important to document as outcomes of the therapeutic process (from both our perspectives and those of our patients) and within what time frame.

Most serious students of stuttering therapy recognize two major philosophical approaches to stuttering treatment that are often integrated with one another; these two major conceptual approaches are often termed *fluency shaping* and *stuttering modification*.[1] What may not be apparent is the relatively unbalanced levels of empirical support for each of these programs, and for what aspects of the patient's profile. The major question of which behavioral components of stuttering therapy appear most efficacious has been the topic of two comprehensive reviews in recent years, including one led by Bothe and colleagues[69] as well as one authored by Herder and colleagues[Q3] for the Campbell Collaborative, reviewed by Law.[70]

It has long been documented that programmed, response-consequated behavioral

"fluency-shaping" interventions do well in general short term, although criteria for inclusion in both reviews excluded some studies that can be considered informative for therapists. There have been a few interesting "twists" on typical fluency-shaping therapy programs; for instance, the Sydney-based Camperdown programs for older teens and adults[71–74] use a model rather than formal instructed changes to speech style. This approach is thought to increase the likelihood of more natural post-therapy speech style.

Other programs pursue a more integrated combination of fluency-shaping and cognitive behavioral therapy techniques. Such programs have begun to publish positive and long-term outcomes data. A primary example would be the Comprehensive Stuttering Program originally designed by the faculty of the Alberta-based Institute for Stuttering Treatment and Research, including Deborah Kully and Marilyn Langevin.[48,75,76] Such programs, which are run on an "intensive" basis (2- to 3-week residential schedule) graduate attendees who can achieve long-term fluency of 0 to 3% stuttered syllables, with associated positive change in OASES scores.

Although stuttering modification programs are historically older approaches to stuttering therapy, outcomes of such programs have not been as frequently reported in the literature. Blomgren and colleagues assessed outcomes for the popular Successful Stuttering Modification program, originally developed by Dorvan Breitenfeld; graduates of the 3-week intensive program were assessed to make better progress on affective and cognitive components of their diagnostic profiles than their measured fluency gains.[77]

There has also been increased attention to the use of cognitive-behavioral therapy principles, used in clinical psychology, to the treatment of individuals who stutter.[78,79] Addition of cognitive-behavioral therapy to a speech-restructuring program aided in reduction of speech-related anxiety, common among individuals who stutter (as well as a wider range of social phobias[80]), but did not appear to improve fluency outcomes, per se.

Finally, there have been some trials of less common approaches to stuttering treatment, such as self-modeling, first discussed by Bray and Kehle.[81–83] This approach, which repeatedly exposes the client to edited samples of their own stutter-free speech, was recently trialed as a relapse intervention for 12 adults (prior studies had involved a smaller number of child participants). Results suggested a substantive reduction in frequency of stuttering (from roughly 8 to ~2% SS[Q4]),[84] and the intervention may hold promise as a "refresher" approach to relapse in previously treated adults or children.

PHARMACOLOGICAL INTERVENTIONS

There has been perennial interest in so called "drugs and devices" that may improve fluency in PWS, either alone or as adjuncts to conventional speech therapies. In general, past drug treatments have not been seen as either effective or sufficiently safe (especially given modest observed gains in fluency).[85,86] Until very recently, the only potential agent with acceptable outcomes for both fluency and limited side effects has been olanzapine, trialed by Gerald Maguire and colleagues.[87] However, in the past few years, there has been a nationwide series of trials of a new drug, Pagoclone[Q5]; the resulting study has been nicknamed "EXPRESS" (for its longer title, Examining Pagoclone for Persistent Developmental Stuttering Study). The 2010 report showed that, in double-blinded administration and evaluation, there was a 20% reduction in %SS over 5% reduction on placebo medication; in a later, open-label trial, in which people were aware that they were taking the active drug, there was a 40% reduction in %SS after 1 year of use, with few reported major side effects. A unique aspect of this drug trial was its scope (over 100 patients at multiple sites, nationwide), as well as joint assessment of the patients in the trial by physicians and specialist speech-language pathologists who could gather objective pre- and post-treatment data garnered from multiple types of assessments. The drug has not yet been approved by the FDA for stuttering treatment and has not been trialed on children or teens. It is also extremely important to note that the drug's developers do not endorse Pagoclone as a stand-alone replacement for

behavioral therapy, but prefer it to be viewed as a potential adjunct to therapy.

AAF DEVICES

I will begin this section by disclosing that I currently consult on study development for the company (Janus) that produces the most widely publicized (and investigated) AAF device, the SpeechEasy©. There has been a recent flurry of reports on the benefits provided by this device, in particular, and others roughly similar in feedback characteristics. They provide feedback of the speaker's speech, altered slightly to produce a timing delay, as well as a change in fundamental frequency. The resulting signal has often been compared with choral speech, well known to produce fluency enhancement for many PWS,[8] particularly in laboratory settings and when reading scripted materials.[88,89] Results of studies combine to suggest that a proportion of users get some measurable benefit from the device, including long-term use, and in a variety of naturalistic settings. However, a real challenge to knowing how many can benefit from such feedback has been the easily observable variability in individual response by study participants. For example, Ryan Pollard and colleagues[90] found no real systematic group gain for conversational speech after 4 months of use (with large individual variability in their sample of 11 adult participants), but in O'Donnell and colleagues' study,[91] 3/7 showed durable improvement. These general patterns are characteristic of other reports in the recent literature: some users show fluency improvement, and some do not; some improvements are durable, some are not.[92–95] It is still not clear how or why the device works to improve fluency, although some have recently proposed that the added signal functions to engage the brain's mirror neuron system, thus "boosting" the effectiveness of speech production.[96] Recent imaging studies have confirmed changes in functional markers of speech processing under AAF.[97,98] As in the case of pharmacological interventions, there are no data on fluency response to AAF in children who stutter.

It is important to note that several PWS show no stuttering reduction under AAF, even in limited laboratory conditions, and thus, before pursuing an AAF option, clinicians should pilot responses using Internet-based free programs and weigh the costs and benefits of trialing the device, as the in-the-ear device must be fitted individually, at substantive cost, with variable coverage by third-party payers.

HOW MUCH DO SPECIFIC THERAPIES MATTER IN FLUENCY THERAPY?

Aside from a few studies that suggest that there may not be large differences in treatment outcomes between some contrasting therapy approaches, there has been increased attention, both in clinical psychology and speech-language pathology, to so-called "common factors" in determining successful outcomes of intervention.[1,99] Such factors may include therapy milieu; client characteristics, including readiness to change[100]; and clinician factors that include expertise, allegiance to the therapy approach being implemented, and ability to create alliance with the client. A widely cited illustrative analysis of how such factors can be disentangled from specific therapeutic agents (in this case, medication) is provided by Bruce Wampold and Jeb[Q6] Brown.[101]

RECENT CODE CHANGES IN DESCRIBING STUTTERING

Although changes to the *International Classification of Disease* (ICD-9-CM) and *Diagnostic Statistical Manual* (DSM; now moving from version IV to V in the coming years) are not strictly recent research findings, impending changes do reflect a subtle shift in how stuttering is conceptualized in the broader medical and psychological/psychiatric literature. Historically, stuttering has had only one code (307.0) under Mental Disorders, in the sequence also coding eating disorders, tics, sleep disorders, eneuresis, soiling, and psychogenic pain. This was unfortunate for numerous reasons, including public perception and inability to track known subtypes of stuttering presentation. After October 2010, there will be four codes for stuttering/fluency disorder in the ICD, with concomitant revisions planned for the DSM. These will include a new primary

default code of 315.35—Childhood onset fluency disorder (stuttering, cluttering), as well as two other new codes: 438.14 fluency disorder due to cerebrovascular accident[Q7] (implemented in October 2009) and 784.52: Fluency disorder in conditions classified elsewhere. The historical 307 code will be reserved solely for adult-onset fluency disorder not covered by these other three codes; we anticipate its use only for cases where stuttering arises outside the normal developmental window and cannot be attributed to obvious organic causes.

HOW TO GET YOUR OWN STUTTERING UPDATES

It should be apparent that there is a tremendous amount of research in stuttering that has the potential to dramatically influence our assessment, therapy, and counseling practices. Keeping up with what can sometimes seem to be a literal torrent of publications can be daunting, but there are some relatively easy solutions to keep abreast of emerging reports. Here's how to keep up with what's out there.

Go to the PubMed website, sponsored by the American Federal government at http://www.ncbi.nlm.nih.gov/pubmed/. This is a free service, available worldwide. It enables searches on any topic and will enable you to establish a preset search that will alert you each time a new article on the topic of stuttering appears in the literature (or is planned for publication). To do this, simply enroll in My PubMed by going to the "my NCBI" logo (located in the upper right corner). To set up your alerts, first run an initial search, using a term such as stutter* OR stammer*, which should allow all reports with either word, and many optional endings (such as stuttering, stutterers, etc.). The initial search will produce thousands of records, but you will be queried if you want to save this search, and you will be given options for delivery of new citations to your email account (e.g., daily, weekly, monthly). Then sit back and wait for information on stuttering to be delivered to your email virtually as soon as it is published. It is obvious that not all practitioners have access to libraries that will have the full text of every article cited. However, the citations usually include a primary author's email address; because authors now receive PDF copies of their articles (even if they are prepublication proofs), you will discover they are more than happy to send you one very quickly.

In sum, there is a lot to learn about stuttering, and we are nowhere close to understanding what we need to know about most effective treatments for specific individuals, in specific contexts, nor why stuttering emerges (and sometimes spontaneously disappears). However, this article has hopefully provided some synopses of major recent findings and will enable readers to go forward with a plan for staying current and informed throughout their professional careers.

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