

## Discussion

# How effective is therapy for childhood stuttering? Dissecting and reinterpreting the evidence in light of spontaneous recovery rates

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### Abstract

*Background:* Similar positive results (e.g. immediate decreases in stuttering frequency and a 60–80% recovery rate from stuttering) have been reported for numerous therapeutic protocols for treating childhood stuttering, many of which have been diametrically opposite in their orientations and implementations. For example, Johnson advocated indirect treatments that simply advocated refraining from drawing any negative attention to childhood disfluencies as persistent and chronic stuttering was thought to progress via negative parental reactions to normal disfluencies. In contrast, direct interventionists sought immediately to eliminate stuttered speech patterns by training ‘corrected’ speech models that usually involved some form of prolonged speech. However, reports from speech and language therapists around North Carolina, USA, suggest much lower recovery rates in the children they treat (i.e. 13.9% over a median therapeutic period of 3 years, which to the present authors is an indicator of therapeutic inefficiency and ineffectiveness).

*Aims:* The discrepancy between these recovery rates calls for a re-examination of the efficacy of stuttering therapy for children, especially in light of recent statements from some therapies suggesting that therapy might be curative in nature.

*Main contribution:* Spontaneous and complete recovery (removing all overt and covert markers of the pathology) occurs in 60–80% of all children who display incipient stuttering behaviours. As such, it appears that many claims of therapeutic success in children who stutter are confounded by the possibility of spontaneous recovery during the testing and intervention period. Simply put, it is impossible to discriminate between recovery that would occur naturally over

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time, and what may have been simply accelerated via therapy. Based on stable prevalence rates and the data in the present paper, it is suggested that therapy does little to boost recovery rates from incipient stuttering. Therapy can provide 'inhibitory' symptomatic relief with varying degrees of success with respect to decreasing stuttering severity and the need for continued therapy. However, it must be made clear that curing stuttering is not a likely outcome of therapy, although successful management can decrease the severity of the problem. It is argued that all forms of stuttering inhibition, including those at work during spontaneous recovery, are all mediated by the degree of mirror neuron engagement in the brain.

*Conclusions:* It is proposed that in children who stutter, the best source of relief from stuttering is in the effective and efficient engagement of mirror neurons via methods that best replicate choral speech. In order to induce natural sounding, fluent speech, it is suggested that one uses primarily derivations of choral speech such as altered auditory feedback. Motoric techniques might also be used synergistically to provide supplementary sources of mirror neuron engagement.

*Keywords:* MeSH terms: child/preschool, speech therapy/methods, stuttering/therapy, fluency, inhibition, mirror neurons.

### **Overview of childhood stuttering**

Repetitions and prolongations of speech sounds are the primary overt signatures of developmental stuttering that arise in about 5% of children between the ages of 2 and 6 (Bloodstein 1995). Repetitions are the first behavioural manifestations of the disorder (Van Riper 1973, Yari *et al.* 1993, Bloodstein 1995, Zebrowski 1995, Silverman 1996, Mansson 2000), seemingly emanating from nowhere and for no apparent purpose. Even if displayed for the briefest periods, witnessing these incipient stuttering behaviours often evokes a salient fear of disordered communication in parents and other adults who may be listening (Ratner 2004). The fear lays not so much in the repetitive behaviours themselves, which are typically free flowing, effortless, produced without the child's awareness and do little to impede communication. The fear lays more in the knowledge of the advanced forms and the progressive nature of the stuttering syndrome may be forthcoming if the child producing these easy incipient behaviours is among the prevailing 1% (Bloodstein 1995, Mansson 2000) of the population who do not undergo natural recovery processes, whereby stuttered speech patterns spontaneously self-remit, removing all traces of pathological existence. That is, those who are truly considered to be recovered from the malady of childhood stuttering must meet the following criteria: they are no longer seen as persons who stutter, either by themselves or by others (e.g. parents, teachers or friends). In addition, they no longer have a propensity to generate involuntarily part-word repetitions or any other overt stuttering behaviours (e.g. prolongations or blocking). Furthermore, they do not display any avoidance behaviours associated with the fear of stuttering, nor do they use any types of imposed controls to avoid stuttering (Finn *et al.* 1997).

For those who do not remit spontaneously, stuttering is a complex and dynamic disorder that evolves over time and is characterized by a progressive nature. The progression of stuttering is not linear, as evidenced by the wide variety of outcomes and rates of progression. It is the non-linearity of its progression that makes it so

insidious. Simply put, seemingly innocuous incipient stuttering behaviours may at almost any time be replaced by more severe forms in those who are prone to continue stuttering. Epidemiological evidence indicates that up to 80% of children who display incipient stuttering behaviours undergo spontaneous recovery, regardless of whether any therapy is administered and regardless of the type of therapy administered (Yairi and Ambrose 1992, 1999, Yairi *et al.* 1993, Kalinowski *et al.* 2002). However, no signature behaviour exists to delineate on an individual basis those who will spontaneously remit from those who will not (Kalinowski and Saltuklaroglu 2004). In addition, no calculus or computational method exists to differentiate those who will develop mild forms of stuttering from those who will develop moderate or severe forms of the pathology. Simply put, even the mildest of incipient behaviours can be fear evoking for parents and caregivers listening to the child and appear to be a harbinger of bad tidings (i.e. lifelong stuttering).

With this in mind, it is not surprising that therapy has focused on the elimination of repetitions in an effort to halt the progression of a disorder where the calculus for progression is unknown. Therapies varying fundamentally in theoretical orientations have been used. Most have claimed similar success rates, making the therapeutic picture appear optimistic at first glance, though suspect upon more thorough examination. For example, Johnson (1955) did not directly treat the child who stutters *per se*. Instead, he advocated counselling the parents to ignore stuttering behaviours and let the repetitions freely occur without any social punishment or stigma. He suggested that the 'attention' given to normally occurring childhood non-fluencies could lay pathological foundations on an otherwise normally functioning speech system. In contrast, direct therapists or interventionists have typically viewed repetitions as reflections of temporal problems in the speech motor system that can be simply unlearned or compensated for via peripheral speech retraining (e.g. slowed speech or droned speech, gentle onsets, etc.) when attended to in a clear and precise manner (Ingham 1984, Packman *et al.* 2000). Both these diametrically opposed therapeutic milieus and their various offshoots have made numerous claims of therapeutic success claiming recovery rates in the range of 80% (e.g. Culp 1984, Shine 1984, Onslow *et al.* 1994, Craig *et al.* 1996). However, any claims of therapeutic success in stuttering children are immediately clouded by the 80% chance of natural spontaneous recovery that most often occurs between 1 and 3 years post-onset (Yairi *et al.* 1999), suggesting that no therapeutic milieu for children can be sure of providing a curative agent for recovery from stuttering. In fact, we have argued that spontaneous recovery is probably the only way in which stuttering is ever 'cured' (Kalinowski and Saltuklaroglu 2004). Thus, it appears that therapeutic techniques might be providing stuttering children with varying levels of relief from overt stuttering symptoms (i.e. methods of inhibiting stuttering; Saltuklaroglu *et al.* 2002); yet in the vast majority of children, it does not appear to be sufficiently powerful so as to remove permanently all pathological markers in children whose stuttering is not remitted by spontaneous natural processes. This statement is supported in the accompanying paper. It provides self-reported data from school-based speech therapists who treated stuttering children in North Carolina, USA. The 101 speech therapists sampled treated 2036 stuttering children. Although many children were still within an age range in which spontaneous recovery should still help 'boost' their recovery statistics, the median recovery rate for the children treated in this study was only 13.9% over a median therapeutic period of 3 years, which to the authors is not an indicator of high success and

suggests inefficiencies in the policies and procedures in the treatment of children who stutter.

The questions that arise are where the faults lie in our delivery of stuttering therapy and how they can be corrected to serve stuttering children better. First, speech and language therapists cannot be blamed for the inefficiencies in helping children recover from stuttering. According to our questionnaires, based on the tools available, most speech therapists used an appropriate battery of behavioural and counselling techniques thought to ameliorate or eliminate stuttering. Therefore, most speech therapists more than adequately fulfilled their professional obligations of using the appropriate methods made available to them. They simply appear to be ill equipped for putting children on the path to recovery (Cooper and Cooper 1996). Second, children who stutter cannot be blamed for failing to implement therapeutic techniques. They must not be victimized. After all, stuttering is defined as an 'involuntary' speech disorder (World Health Organization 1977) and no amount of parental counselling, speech retraining, relaxation or attitudinal changes can make it otherwise. Thus, the inefficiencies must lie in the tools themselves. To understand better why current therapies are doing little to improve upon nature's recovery rates from childhood stuttering, a historical perspective is presented on how these therapies were adopted in children as an extension of adult-based stuttering therapy, followed by our explanations of the mechanism by which these therapies have operated. These therapeutic techniques will also be discussed in relation to other well-known 'fluency enhancers' such as choral speech (Cherry and Sayers 1956, Bloodstein 1995, Kalinowski and Saltuklaroglu 2003). The paper will conclude by explaining parsimoniously the addressed phenomena in terms of common neural mechanisms and suggesting methods for serving better the population of stuttering children.

### Evolution of stuttering therapy in children

One of the first documented attempts to remove stuttering behaviours was by Demosthenes (384–322 BC), the Greek orator who blamed his stuttering on a weakened system. To remove the overt stuttering events from his speech, he spoke with pebbles in his mouth. Later Professor Dieffenbach (1792–1847) of Prussia suggested that overt stuttering events manifested as a result of an enlarged tongue. His form of remediation was surgically removing a v-shaped wedge from the tongue to make it smaller (Silverman 1996). Both Demosthenes and Dieffenbach had similar results. Stuttering was immediately reduced while speech was distorted by the pebbles in the mouth or by the aberrant speech patterns caused by the pain inflicted from the surgery. However, once the pebbles were removed or the tongue healed, stuttering invariably resurfaced. Both Demosthenes and Dieffenbach had found the 'fools gold' in stuttering amelioration. This short-lived, tenuous, unstable, odd-sounding, yet relatively stutter-free speech that they induced would be mistaken in therapeutic circles for truly fluent speech for centuries to come. Recently, however, these characteristic features have been re-examined and the label 'pseudofluency' has been aptly appropriated to such speech patterns (i.e. short-lived, tenuous, effortful and unnatural sounding) to differentiate them from the truly fluent speech (i.e. long-term, stable, effortless and natural sounding) of those who do not stutter (Dayalu and Kalinowski 2002). Finding the Holy Grail in stuttering amelioration (i.e. replacing stuttered speech with truly fluent speech over the long-term) has proven

more challenging than most expected and the disorder has remained highly resistant to long-term remission (Bloodstein 1995, Kalinowski and Saltuklaroglu 2004).

Bogue (1919) offered a different solution to the previous structural anatomists. He suggested that stuttering was due to incorrectly learned speech patterns and advocated treatment at his specialized institute. He claimed that he could teach those who stuttered to unlearn stuttering behaviours via his intensive therapeutic course. Bogue would attempt to 'tear out' improper methods of speech production, supposedly replacing them with the correct, natural methods and then supposedly establish normal coordination between the brain and the muscles of speech. What Bogue was actually teaching was speaking to a self-imposed rhythm, a condition that results in immediate reduction in stuttering symptoms, yet sounds highly unnatural and does not translate into fluent, natural sounding speech across speaking situations. Thus, like those who preceded him, Bogue was seemingly unable to deliver on his promises of long-term stuttering remediation to those who prescribed to his methods.

As Freudian psychology gained popularity, stuttering began to be associated with the manifestation of neurotic behaviours. It was thought, and still is in some psychoanalytical circles, that stuttering was a psychological defence mechanism. For example, Fenichel (1945) stated that that stuttering was 'a pregenital conversion neurosis where an inner conflict is converted to the external'. Travis (1957), on the other hand, espoused the notion that 'the stutterer cannot speak because he has unspeakable feelings'. However, Travis's transcripts suggest a later onset of neurosis than those of Fenichel. Indeed, many of the psychoanalysts (e.g. Coriat 1928, Barbara 1954) held different opinions with respect to the onset time and the site of the neurosis associated with stuttering. More, importantly, few achieved success in reducing the overt manifestations of the disorder. For example, Brill (1923) reported only being able to help four of 69 patients he had treated. With the lack of success or viable alternatives within this paradigm, the field of stuttering began to reject psychoanalysis, although it did leave a lasting impression.

In a clearly transitional period, Johnson (1959), father of the semantogenic or diagnosogenic theory, believed that a child with normal non-fluencies and normally functioning central nervous and motor systems could be morphed into a 'stutterer' if proper nurturing was not provided during a critical period. That is, if primary caregivers (typically the mother) responded negatively to any speech abnormalities during this period, a child may be 'labelled' and could be turned from the path of fluency to that of stuttering. Although this orientation clearly displayed psychoanalytical roots (e.g. the strong parental influences and lasting disability), Johnson leaned towards a semantic view of the disorder. Indeed, the difference between his view and those of the psychoanalysts may only exist on a semantic level. However, from his viewpoint, if stuttering truly was a result of the stigma induced by a mother's labelling lips, the intervention protocol was clear: therapy must be targeted towards the mother. Mothers were taught to ignore all speech aberrations in their children and refrain from drawing any negative attention to their speech. They feared applying the stuttering label, thought to be sufficient to etch permanently disruptive speech behaviours into the psyche or neuronal firing patterns of a young child. This paradigm dominated the treatment of stuttering in children from the 1940s to the early 1980s. Caregivers and therapists alike feigned ignorance of stuttering behaviours, simply pretending that the disorder did not exist in young children. Stuttering was remitted in approximately 60–80% of the children (Johnson 1955), as would be expected from natural spontaneous recovery rates.

For the 20% or more of children who continued to stutter, Johnson began to advocate direct therapy, but only in their school-aged years. To him, it was clear that their mothers had failed in their attempts to refrain from negative labelling and the child now needed direct intervention. Johnson's approach to treating children was essentially the same as his methods for treating adults. His theories were littered with paradoxical statements such as 'stuttering is what you do to avoid stuttering' (Johnson and Knott 1936). With this in mind, he taught methods of easy stuttering such as the 'bouncing' technique which was essentially easy, voluntary stuttering. Bouncing was used with the intention of providing those who stutter with a means of overcoming their fears and tendencies to avoid stuttering (Johnson 1959). Interestingly, like other motoric methods that would follow, these techniques have been found to have some direct inhibitory capacity over stuttering, rather than simply being a means of fear reduction (Saltuklaroglu *et al.* 2004a). Johnson's approach to working with stuttering children and their caregivers remained seminal and the gold standard for decades. Speech therapists refrained from administering direct therapy to stuttering children until they were, by all standards, confirmed stutterers with little probability of recovery.

However, with the growth of behaviourism in psychology and the growing popularity of the speech motor dynamics in stuttering, the 1980s ushered in a new approach to the treatment of stuttering in children that advocated direct therapy. Unlike the underpinnings of the Johnsonian approach, this new research appeared to demonstrate an organic defect in those who stutter, suggesting that it was time for new strategy that was more proximal to the believed aetiology. Under Johnsonian dogma, this was heresy. In stark contrast to ignoring stuttering behaviours in children, the removal of syllabic repetitions and their later emerging derivatives was to become the direct target of stuttering therapy. It appeared as if time, repeated failures with (i.e. 20% or more children who do not recover), and an inability to determine therapeutic outcome had left no option but to try alternative methods. However, though the new theoretical orientation was radically different from previous ones, the new therapeutic options were only incrementally different. Children were simply to be treated by similar methods as those that had been applied to adults for years. In other words, it was simply time to point out that children were cognisant of their stuttering or that the 'emperor has no clothes'.

Thus, since the 1980s, speech therapy for children who stutter has centred primarily on direct intervention via retraining peripheral speech motor system, an approach that has been popular in the treatment of adult stuttering since the 1970s (e.g. Goldiamond 1965, Ryan 1974, Perkins *et al.* 1976). Stuttering is overtly characterized by disruptions in the respiratory, laryngeal and articulatory systems. Under this paradigmatic umbrella, these disruptions were assumed to be the result of a temporal planning or executions deficits (Caruso *et al.* 1988) that may or may not be specific to the speech motor system. Speech was 'taught' in a similar manner to other motor skills requiring precise muscular coordination (e.g. skating, riding a bicycle or playing a musical instrument). This approach acknowledged a compromised speech motor system and allowed for temporal leeway to compensate and or retrain the system. It was assumed that 'correct' speech patterns were best learned by implementing slow rates of speech, sometimes up to two seconds per syllable (Webster 1979). Implementing these slowed rates caused speech to have a highly unnatural or 'droned' quality, a condition that results in speech that is highly conspicuous and unnatural sounding, yet tends to immediately eliminate most, if not

all, overt stuttering behaviours (Saltuklaroglu *et al.* 2004b). The notion underlying these therapeutic procedures was that producing speech at a slower rate via droning allows the system more temporal freedom to enable it to produce fluent utterances. It was hoped that during this period of droned speech some form of long-lasting re-stabilization or integration of the 'corrected' speech model would occur. Nobody thought that using 'droned' speech may be the equivalent of using one long 'prolongation', similar to those that characterize true stuttering behaviours (Kalinowski and Dayalu 2002).

As the direct interventionists dictated, stuttering behaviours were to be removed via the systematic imposition of droned or novel speech patterns that were incrementally shaped over time via simple operant conditioning. Children were often able to emulate easily the new speech patterns that were relatively free from repetitions and secondary behaviours. Like Johnsonian type therapists, direct interventionists claimed a fair amount of success in the attack on incipient stage stuttering (Ryan 1971, Culp 1984, Shine 1984, Onslow *et al.* 1994, Craig *et al.* 1996). However, unlike the Johnsonian approach, direct therapy placed the onus of control on the child and the clinician. As such, this direct approach became popular among speech therapists and has been readily implemented in a variety of settings including the public school, where early intervention is the logical choice for treating any 'at risk' child. However, three clear problems began to surface. First, the novel speech patterns possessed an artificial nature (Kalinowski *et al.* 1994), in that they were difficult to produce spontaneously, required considerable effort and sounded highly unnatural or 'droned' (i.e. pseudofluent), making generalization very difficult and relapse common (Craig and Hancock 1995). In fact, as with adults, the ameliorative effects of motoric based speech therapy appear to wear off in children with attempts to make post-therapeutic speech sound more natural (Saltuklaroglu *et al.* 2004b). Second, the recurring role of natural recovery continued to present a confounding element for anyone making claims of therapeutic success in stuttering children (Yairi and Ambrose 1999, Kalinowski and Saltuklaroglu 2004). Lastly, it should be further noted that the amelioration of overt stuttering can be rather easy in the short term. Success in the long-term can be a much more nebulous affair, especially when no evidence exists to support any change in overall prevalence rates. Specifically, in adult therapy, Bloodstein examined 162 studies of therapy efficacy, using numerous intervention modalities. Nearly 95% demonstrated a significant reduction in stuttering frequency. Nonetheless, relapse to previous stuttering behaviours is often the natural sequence events, regardless of the type of therapy used.

### **Choral speech**

The most compelling phenomenon in stuttering is 'choral speech', when two speakers produce the same material simultaneously. It has been known for centuries that while under choral speech conditions, all stuttering behaviours are conspicuously absent from the speech of those who would otherwise stutter (Cherry and Sayers 1956, Bloodstein 1995, Kalinowski and Saltuklaroglu 2003). This nearly universal phenomenon has intrigued researchers and clinicians, and appeared to have little explanatory power as to the nature of stuttering. Choral speech was often used simply to demonstrate to persons who stutter that they had the innate capacity to generate natural sounding, fluent utterances that were free from

repetitions and all other forms of stuttering. Furthermore, choral speech has been used in brain imaging studies to demonstrate ‘normalization’ of the stuttering brain during truly fluent speaking episodes (Fox *et al.* 2000, Salmelin *et al.* 2000, Ingham 2001). The inhibitory mechanism that operates to suppress repetitions, even in the most severe of those who stutter, was unknown. Bloodstein (1995) speculated a distraction theory. Others have suggested reduced communicative load, the inducement of novel patterns of vocalization (Wingate 1969) and the provision of an external timing mechanism (Sommer *et al.* 2002). However, various permutations of choral speech have displayed robust yet differential levels of stuttering inhibition depending on stimuli used (e.g. Howell *et al.* 1987, Hargrave *et al.* 1994, Kalinowski *et al.* 2000, Kalinowski *et al.* 2004a). Thus, the distraction theory and the other post-hoc explanations lose credibility. Instead, it appears that the robust inhibition of stuttering achieved through choral speech may be the result of a centrally based neural mechanism that pairs speech perception and production as interchangeable forms of action recognition and execution (Kalinowski and Saltuklaroglu 2003). This same mechanism may also be fundamental to nature of all overt stuttering behaviours as well as potent procedures for inhibiting stuttering.

### Synthesizing the knowledge

To best explain the nature of stuttering the pieces of the puzzle examined above must fit together parsimoniously. Thus, the phenomena that require assimilating are as follows. First, 5% of children show signs of stuttering between the ages of two and six, with the primary symptom being part-word repetitions. Second, natural spontaneous recovery occurs in nearly 80% of these children, leaving the 1% of the population who comprise the documented prevalence rate for stuttering. Third, choral speech inhibits repetitions from the speech of those who stutter’s speech by 90–100%. Fourth, Johnsonian types of therapy have claimed similar levels of success as direct intervention methods, even though the orientations and methodologies of the two approaches are diametrically opposed. All of these phenomena need be integrated or explained for a parsimonious understanding of the nature childhood stuttering and why therapeutic intervention appears to do little to alter the natural course of the pathology.

### *Epidemiological evidence*

Let us begin by addressing the epidemiological data in light of the therapeutic interventions and the data in the accompanying paper. It appears that no evidence exists for speech therapy ever ‘curing’ stuttering in children. The incidence and prevalence rates of the disorder have remained stable over time, regardless of the approach taken towards remediation. These figures appear to be consistent regardless of the therapeutic intervention or despite what any small data sample may suggest (e.g. Ryan 1981, Culp 1984, Shine 1984, Boberg and Kully 1994, Onslow *et al.* 1994, Wagaman *et al.* 1995, Craig *et al.* 1996, Elliott *et al.* 1998, Harris *et al.* 2002). In the accompanying manuscript, the 13.9% median recovery rate among children by the respondents can be explained by two possibilities. Most likely, they were those for whom natural recovery occurred during their period of therapeutic intervention. However, it is also possible that therapeutic intervention accounted for a minimal

number of children that were 'on the edge of recovery'. That is, the use of some 'inhibitory' techniques along with natural processes may have been sufficient to push a few children with milder symptomatology 'over the edge' and into the realm of recovery. For the remaining 86.1% of the children who reportedly did not recover, it is possible that the use of therapeutic techniques promoted varying levels of stuttering inhibition and some relief from the symptoms, possibly inducing some overall decrease in severity levels.

But what price was paid for these minor improvements? Most children who stutter in the state school systems undergo therapy once, twice or thrice a week, spending about 3 years on the caseload of one therapist, and then possibly being transferred to another caseload. They are often noticeably removed from their regular classrooms, trained to use unnatural sounding speech patterns that are often embarrassing to use beyond the clinical setting, and are often destined to underachieve in one or more facets of life due their fear of stuttering. So again, we surmise that natural spontaneous processes are probably the only reliable, efficient, and effective means by which stuttering is 'cured' and children truly recover and become 'normally' fluent speakers according to the previously specified criteria. The power of spontaneous recovery is seen in that it can 'cure' even the most severe cases of childhood stuttering. In contrast, though the effects of therapy cannot be clearly delineated in any child who recovers, it is most likely that therapy may simply help accelerate the path to recovery or provide varying degrees of relief from stuttering symptomatology in those who do not recover.

The findings in this study are consistent with what we have inevitably learned about stuttering since the time of Demosthenes: regardless of intervention, stuttering continues to progress in most children who are not 'blessed' with the path of natural recovery. Therapies have taken on a variety of forms including punishment for stuttering, tongue surgeries and oral prosthetics, parental counselling, behavioural speech retraining that attempts to guide the central nervous system into fluid, if not natural, continuous motion, and almost everything in between. However, the rate of progression from incipient to adult stutterer continues relatively unabated, as do the inevitable consequences and penalties of lives impeded.

#### *Incipient stuttering, choral speech and shadowed speech*

Interestingly, however, the overt behaviours produced during the incipient stages of stuttering may be fundamental to natural spontaneous recovery processes. As previously stated, incipient stuttering is primarily characterized by part-word or syllabic repetitions. Eighty percent of children displaying these incipient behaviours recover, suggesting that producing these fundamental stuttering units may play a substantive role in the recovery process (Kalinowski *et al.* 2004a). Simply put, rather than being the stuttering 'problem', the production of simple, free-flowing, repeated syllables may indeed serve to 'release' the central nervous system from the involuntary stuttering block (Kalinowski *et al.* 2000). In other words, the disruptive behaviours that have been the consistent target for elimination via therapeutic protocols may in reality be peripheral compensations for a central pathology that appear to induce full recovery in up to 80% of stuttering children. In order to understand why these behaviours are 'special' or fundamental to recovery, we must

examine them in reference to the most powerful of all known fluency enhancers: choral speech (Kalinowski and Saltuklaroglu 2003).

The potency of choral speech for inducing immediate fluency in almost any child or adult who stutters is undisputed. Without any training at all, fluency can be induced in even the most severe cases of stuttering, suggesting direct and potent over-riding of the central stuttering block. Recently this seemingly 'magical' effect has been explained by the engagement of 'mirror neurons'. Mirror neurons are thought to be primitive neuronal substrates that allow for motoric gestures such as speech to be immediately recognized and replicated. Thus, they have been found to fire similarly both during both during action recognition and execution (Rizzolatti and Craighero 2004), providing the neurophysiological link for the long hypothesized parity of speech perception and production (Lieberman and Mattingly 1985). This innate neural linkage may also be fundamental to the human capacity to imitate, a specialized human ability that is performed fluently (Skoyles 1998). As such, when two speakers speak in unison, one is simply imitating the others. Thus, for the person who stutters, the second speaker provides an exogenous framework of speech information that can be fluently imitated by the person who stutters via mirror neuron engagement (Kalinowski and Saltuklaroglu 2003). A growing body of data is continuing to demonstrate that inhibitory mechanism is endowed with substantial flexibility. Robust levels of stuttering inhibition have been achieved when the choral signal is manipulated with respect to its temporal and linguistic synchrony with the intended utterance (e.g. Howell *et al.* 1987, Kalinowski *et al.* 2000, Saltuklaroglu *et al.* 2003, Kalinowski *et al.* 2004a).

One example of temporal flexibility is shadowed speech, a close relative of choral speech. In choral speech, the two utterances are produced in near synchrony, whereas in shadowed speech, the person who stutters repeats or directly imitates a preceding utterance. This shadowing or direct imitation has also been found to induce high levels of stuttering inhibition comparable to those achieved by choral speech, again demonstrating the fluency enhancing properties of imitation via mirror neuron activation. Thus, upon re-examination, overt part word repetitions may be described as 'endogenous' forms of shadowed speech, whereby the same speaker simply shadows himself or, self-imitates for the purposes of reinstating fluent speech by accessing the mirror neuronal mechanism. Self-imitation is more likely to be successfully accomplished if the sequence is short (Kalinowski *et al.* 2004a). For this reason, syllabic repetitions are optimal as syllables are the smallest and most fundamental units of acoustic speech (Studdert-Kennedy 2000). Under these auspices, stuttering behaviours may be best described as a form of choral speech as both employ forms of gestural imitation and may provide access to mirror systems for reinstating fluency. The difference is that 'true' choral speech uses an exogenous model for imitation and therefore does not require the production of self-imitated behaviours (i.e. overt stuttering) that are disruptive to communication. With this focus, we will now examine how motoric therapies fit into this schemata.

#### *Explaining motoric techniques*

Perhaps best exemplifying the enigmatic nature of stuttering is the consistent finding that it is highly amenable to short term symptom reduction (i.e. the reduction or elimination of core stuttering behaviours such as repetitions and prolongations), yet

remains overwhelmingly resilient to long-term remission and complete recovery. As it happens, almost any substantial deviation from normal speech production can immediately result in a decrease in overt stuttering symptomatology (Bloodstein 1995, Saltuklaroglu *et al.* 2004b). Examples of such motoric deviations include singing (Glover *et al.* 1996), whispering (Perkins *et al.* 1976), adopting a foreign accent (Bloodstein 1995), imposing a rhythm, and *any* other motoric alteration that significantly alters the manner in which speech is produced and perceived. Commonly implemented operant therapeutic techniques such as prolonged speech (Ingham 1984, Webster 1980), 'airflow control' (Schwartz, 1977), 'continuous phonation' (Shames and Florence, 1980), and 'light articulatory contacts' (Cooper and Cooper 1985) also qualify as examples of motoric alterations that induce an immediate reduction in overt stuttering events. As such, the amenability to immediate symptom reduction appears to have been exploited for centuries by countless practitioners seeking to reduce or eliminate overt stuttering symptoms. The continuing problem is that despite ubiquitous reports of immediate reductions in stuttering events upon the implementation of any therapy that induces altered speech forms, no magical 'cookbook' has ever emerged to allow those who stutter to produce fluent, natural sounding, stable, and relatively effortless speech that is indistinguishable from those who do not stutter and is stable over the long-term. After all, the speech mechanism is a closed system with limited degrees of freedom. Most of the motoric alterations to speech production have already been tried with similar results: short-term improvements followed by a relapse to stuttering (Craig and Hancock, 1995) suggesting that the problem is more complex than a simple matter of motoric speech retraining. For example, relapse from behavioural speech retraining protocols continues to exceed 70% in adults and teenagers, most likely because the deviation from normal speech required to remove stuttering behaviour fails to meet acceptable standards for speech naturalness (Kalinowski *et al.* 1994), is difficult to implement and often highly tenuous (Dayalu and Kalinowski 2002).

Obviously, motoric interventions provide a contrived means of temporarily removing overt stuttering behaviours. However, it has been argued that by replacing stuttered speech patterns with droned and highly unnatural speech patterns, one conspicuous behaviour is simply being replaced by another (i.e. repetitions for one long prolongation). In other words, the tenuous and unstable speech patterns that often result from operant motoric therapies (Dayalu and Kalinowski 2002) may actually be considered to be simply altered or 'denatured' forms of stuttering (Saltuklaroglu and Kalinowski 2002) and certainly not the true speech of those who are by definition, recovered or have never stuttered. The use of motoric techniques appears to be simply masking the pathology. Thus, it appears as if those devising therapeutic interventions for stuttering have simply been baffled by a complex and dynamic pathology that is capable of continually resurfacing like the heads of the hydra.

Perhaps the temporary reduction in stuttering that is observed upon the implementation of any motor 'strategy', 'technique', or 'trick', can also be explained via mirror neuronal engagement. Volitionally imparted and substantial deviations from normal speech production may be considered a form of imitation and usually require a decrease in speech rate that provides a 'redundancy' of gestural (speech) information or more gestural information per unit of time. This may be analogous to increasing the number of frames per second in a movie (film) sequence in order to improve the clarity of the output. As such, therapeutic techniques carry no more

weight for fluency enhancement than speaking with a foreign accent, whispering, singing, or speaking in rhythm. There is no 'correct' speech pattern. The only requirement for a motoric strategy to qualify as a stuttering inhibitor is that it deviates sufficiently from normal speech to engage mirror systems. It is precisely for this reason that motoric methods alone have failed to yield long-term remission. Operantly derived therapeutic speech appears only to reduce the need to produce overt stuttering behaviours when it is possesses its characteristic unnatural or droned quality. Relapse to stuttering occurs with attempts to impose naturalness upon therapeutic speech (Saltuklaroglu *et al.* 2004b). That is, the chance of engaging mirror neurons diminishes as the deviation from normal diminishes and therefore, it is unlikely that naturalness can imposed upon therapeutic speech without the resurfacing of overt stuttering.

### Management of stuttering

The amenability of childhood stuttering to short-term symptom reduction, coupled with the high possibilities of spontaneous recovery, might have grossly inflated the claims of recoveries by therapeutic interventions. Only when epidemiological evidence shows complete recovery beyond 80% in therapeutic populations and overall prevalence rates begin to drop below 1% can any true attribution of recovery be given to therapeutic procedures. However, the epidemiological evidence, the history of resilience in those who do not spontaneously recover and the very nature of stuttering continue to suggest that we are far from finding a curative agent that extends beyond what nature offers. Simply put, stuttering therapy appears to be missing its mark.

For now, instead of gambling on nature's odds of recovery from a disabling pathology with no known aetiology, the more realistic goal in the treatment of stuttering might begin by managing its symptoms efficiently and effectively. Three possible means of reducing or eliminating overt stuttering behaviours have been described: the use of sensory-based methods such as choral speech and its derivatives; the implementation of motoric techniques such as therapeutic techniques, droning, singing, whispering, etc.; and producing overt stuttering behaviours such as part-word repetitions. Although on the surface these methods may appear distinct, it should not be surprising that at the central level, these methods probably converge in mirror neuronal engagement for stuttering inhibition. This makes sense, considering the end result of each procedure is the inhibition of stuttering: it is logical that a single mechanism unites their effects. Thus, the goal for symptom management is clear: children who stutter should be provided with a means of engaging efficiently and efficiently mirror neurons for fluent speech. The measure of efficiency may be found in the time and effort taken to engage mirror systems, whereas the measure of effectiveness might be found in the level of fluency, speech naturalness and stability of the resultant speech over time. The question is how to make optimal use of this innate mechanism to best provide children who stutter the ameliorative benefits they deserve.

The commonality among all methods that have effectively reduced or eliminated stuttering is the engagement of mirror neurons (Saltuklaroglu *et al.* 2004b). Although the present paper has described three means by which this is achieved, choral speech remains the benchmark for efficient and effective symptom reduction (Kalinowski

and Saltuklaroglu 2003), yet it is difficult to implement functionally in its 'raw' form. However, electronically derived permutations of choral speech have allowed those who stutter to receive many of the ameliorative benefits of choral speech without the constant need of a speech accompanist. For example, the use of altered auditory feedback (AAF), such as delayed auditory feedback (DAF) and frequency altered feedback (FAF), has been clearly documented to substantially (e.g. above 80%) to reduce stuttering across a variety of speaking situations such as reading (Howell *et al.* 1987, Hargrave *et al.* 1994), during conversation (Stuart *et al.* 2004), speaking to an audience (Armson *et al.* 1997) and speaking on the telephone (Zimmerman *et al.* 1997). The inhibitory effects of DAF over stuttering have been documented since the 1950s (Bloodstein 1995), but only recently have they been revisited and become known as permutations of choral speech. In fact, the use of DAF lost considerable favour during the domination of the direction interventionists and the speech motor paradigm, when it was believed that DAF was only useful using long delays (e.g. 200 ms and above) as a means of reducing speech rates motorically and instilling droned speech patterns (Costello-Ingham 1993).

Although the use of altered auditory feedback alone is a powerful permutation of choral speech, its effects may be limited in some people who stutter. Using AAF requires speech output by the user so that it can be 'fed back' in a choral manner to engage mirror systems and induce fluent speech. Stuttering characterized by difficulties initiating speech or long silent blockages may not be as amenable to the ameliorative benefits of AAF. For this reason, we advocate using synergistic protocols that simultaneously employ multiple channels of engaging mirror systems. For example, Stuart *et al.* (2004) demonstrated that the use of an inconspicuous device that provides AAF can incur long-term natural sounding and fluent speech when used in combination with minor motoric alterations. Training on this protocol required approximately 3 hours; stuttering was reduced by over 80%; and the resultant speech was perceived as being highly natural by naïve listeners. Most importantly, the results appear to be withstanding the test of time. The powerful inhibitory effects over stuttering appear to be derived by using relatively subtle motoric changes to enhance the electronically derived choral effect. In other words, the simultaneous use of sensory- and motor-based methods of engaging mirror systems appears mutually complementary and may produce better access to mirror systems than using either method in isolation. The use of these protocols is also supported in self-report data from 105 users who reported substantial decreases in stuttering symptoms when using combinations of active and passive protocols (Kalinowski *et al.* 2004b).

We continue to be inundated with strategies that might provide some temporary forms of inhibition, yet continue to show strong evidence of ineffectiveness when examined over the long-term in reference to the epidemiological evidence. By adopting criteria for recovery that include the removal of all stuttering symptoms and a quality of speech that is afforded to normally fluent speakers, we can see that our therapeutic procedures have done little to boost the recovery statistics of stuttering children. However, all does not appear to be lost. Even those who stutter severely are in possession of an innate neural mechanism that can be readily engaged to allow for fluent speech. Behavioural methods alone appear to be inadequate for rendering treatments that children who stutter can use effectively and efficiently as, in order to improve speech naturalness, they advocate the eventual relinquishing of the processes responsible for inhibiting stuttering (Saltuklaroglu *et al.* 2004b). We

suggest using electronic derivations of choral speech (e.g. AAF) as primary agents for reducing stuttering. Intermittent use of behavioural methods may provide additional access to mirror neuronal systems and reflexive, natural sounding and stable fluency. With a better understanding of mirror neuronal stuttering inhibition and further technological advances, it is anticipated that better renditions of the choral effect that rely even less on motoric supplementation will arise, thus relieving children who stutter from both the burden of stuttering and the onus of control for an involuntary pathology whose path has always been primarily charted by nature.

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