



Theory and therapy in stuttering: A complex relationship

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ABSTRACT

There are many treatments currently available for stuttering, for both children and adults. These range from direct interventions intended to reduce the severity and/or frequency of the speech behaviors of stuttering, to those intended to alleviate the anxiety and other mental health issues that can accompany the disorder. However, as there are little supporting data for many of these treatments, there is little consensus about which to use. Another way to evaluate stuttering treatments is to explore the extent to which they address the cause of the disorder. However, the cause of stuttering is not yet known. In this theoretical paper, a 3-factor causal model is presented, to which the mechanisms thought to be driving different treatments are then aligned. The model is innovative, in that it attempts to explain moments of stuttering. It is argued that all causal factors must be operating at each moment of stuttering. The model is intended as a new way of looking at cause, and how treatments may address cause. It is hoped this will stimulate discussion and lead to further lines of inquiry.

Educational objectives: The reader will be able to: (a) describe the P&A 3-factor causal model of moments of stuttering; (b) state how indirect direct stuttering treatments relate to cause, according to the P&A model; (c) describe how direct stuttering treatments relate to cause, according to the P&A model; (d) state the purpose of cognitive behavior therapy; and (e) describe at least one suggestion for further research arising from the P&A model.

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1. Introduction

There are currently many treatments available for people who stutter, across the life span (Bloodstein & Bernstein Ratner, 2008). However, most of these treatments have not yet been shown to be effective and there is currently little consensus about the best way to treat stuttering (Bloodstein & Bernstein Ratner, 2008). It is of interest, then, to ponder on the extent to which current treatments, at least those that are intended to improve fluency, address the cause of stuttering. However, as yet the cause of stuttering is poorly understood.

In this theoretical paper, the complex relationship between theory and therapy for stuttering is explored. When talking about stuttering, theory is typically taken to mean causal theory, in the scientific sense. This will be the focus of the discussion here. The paper covers the following: (1) a brief overview of stuttering treatments and causal theories of stuttering, and (2) the presentation of a new causal model of stuttering which is intended to increase understanding of how treatments relate to cause.

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2. Overview of treatments for stuttering

There is a long history of treatments for stuttering, dating back to the use of rhythmic speech in the 3rd century BC by the Greek philosopher Demosthenes (Packman, Onslow, & Menzies, 2000). Over the last 60 or so years, the approach to treatments for stuttering has become more professional than in previous times, in that treatment methods have been published in reputable journals and books and outcomes of some treatments have been measured and reported (for some overviews of treatments for stuttering, past and present, see Bloodstein & Bernstein Ratner, 2008; Guitar & McCauley, 2010; Ingham, 1984; Packman et al., 2000; Shapiro, 1999; Van Riper, 1973). These treatments cover a broad spectrum, from those intended to enhance fluency to those that address the psychological (including cognitive) and/or social concomitants of stuttering. Many treatments include procedures for both. Further, the approach to treatments for stuttering varies across the life span, with treatments for preschoolers being quite different from those for adolescents and adults.

For the purposes of this paper, fluency treatments have been categorized as direct and indirect. Direct treatments are those where procedures focus on changing spoken language, while indirect treatments are those that aim to enhance fluency by changing features of the environment thought to be impacting fluency. A number of treatment approaches include both. This categorization is typically used with interventions for children who stutter (for example, see Guitar & McCauley, 2010), but has wider applications. Treatment procedures that address the psychological and/or social concomitants of stuttering, such as social anxiety and avoidance, may be incorporated into fluency approaches or may stand alone, such as cognitive behavior therapy. Interestingly, drugs have been trialed for the treatment of stuttering in both adults and children and fall into both camps, with some targeting speech production and others targeting anxiety (for reviews see Bothe, Davidow, Bramlett, Franic, & Ingham, 2006; Boyd, Dworzynski, & Howell, 2011). The aims of various treatments will be discussed further later in this paper.

3. Overview of causal theories of stuttering

The cause of stuttering is as yet not fully understood. In order to fill this gap in understanding, many causal theories have been proposed (for reviews and discussion of causal theories, see Bloodstein & Bernstein Ratner, 2008; Packman & Attanasio, 2004; Yairi & Seery, 2011). As with treatments for the disorder, theories on the cause of stuttering date back centuries. However, in the main these theories are not testable in the sense that a scientific theory can be tested, which goes some way to explaining why so many are currently still considered viable. In other words, if a theory or model is not couched in operational terms and so cannot be tested, it can never be shown to be wrong (Packman & Attanasio, 2004).

There are many ways of categorizing causal theories. For example, Packman and Attanasio (2004) discussed them under the categories of speech motor control, systems control modeling, cognitive and linguistic processing, multifactorial and anticipatory struggle, while Bloodstein and Bernstein Ratner (2008) talked about "theories of etiology versus concepts of the moment of stuttering" (p. 39). As suggested by Bloodstein and Bernstein Ratner's classification, it has long been suggested that causal theories be categorized according to whether they explain the underlying cause of stuttering or the cause of individual moments of stuttering. These are sometimes referred to as the distal and proximal causes. According to Bloodstein and Bernstein Ratner, some causal theories address both.

Most causal thinking at the present time is multifactorial. Two influential multifactorial models theories are the Demands and Capacities model (e.g. Starkweather, 1987; Starkweather & Givens-Ackerman, 1997; Starkweather & Gottwald, 2000) and the Dynamic Multifactorial model (Smith & Kelly, 1997). According to the Demands and Capacities model, stuttering is the result of the interaction of intrinsic and environmental factors and occurs when the demands for fluency are greater than the capacity to produce it. None of these factors are necessarily abnormal and, "there is no single etiology, but as many etiologies as there are stories of stuttering development" (Starkweather & Givens-Ackerman, 1997, p. 24). Similarly, according to the Dynamic Multifactorial model, "stuttering emerges from the complex, nonlinear interaction of many factors. No single factor can be identified as 'the cause' of stuttering" (Smith & Kelly, 1997, p. 209).

There can be little doubt that stuttering is multifactorial and highlighting this, as these two models do, has done much to aid thinking about the cause of the disorder. However, the way these two models have been presented means that they cannot be falsified. It is also the case, as will be discussed below, that the findings of brain imaging research that have emerged since the models were formulated, are suggesting that there are structural differences in the brains of people who stutter and that these may be a necessary condition for stuttering to occur. Clearly, however, even if further research establishes unequivocally that such brain anomalies are present in people who stutter, such anomalies are not sufficient to cause stuttering. They do not explain why some syllables are said with struggle and tension while others are said fluently.

A causal model is now presented that is multifactorial in nature but that describes the factors operationally. It is based on the reasoning that the question to be addressed by any causal model is not the general one, what causes stuttering, but rather the specific one, what causes individual moments of stuttering. It is argued here that distinguishing between what are termed "distal cause" and "proximal cause" is misleading, because it is the case that all causal factors must be operating at every moment of stuttering. The Packman and Attanasio 3-factor causal model of moments of stuttering (P&A model) is the first multifactorial explanation of stuttering that attempts to model the necessary and sufficient conditions for a moment of stuttering to occur.

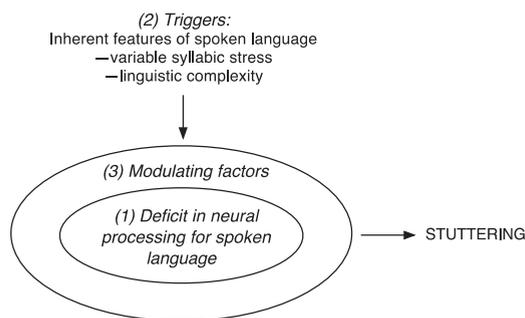


Fig. 1. The Packman and Attanasio 3-factor model of moments of stuttering.

4. The Packman and Attanasio 3-factor causal model of moments of stuttering

The P&A model is shown in Fig. 1. This was first presented at the American Speech and Hearing Association Convention in Philadelphia, in November 2010 (Packman & Attanasio, 2010). The three factors are, (1) a deficit in the neural processing underpinning spoken language, which renders the speech production system unstable and prone to perturbation, (2) triggers, which are some inherent features of spoken language that increase the motoric task demands on that system, and (3) modulating factors, which determine the triggering threshold. The three factors are now explained in more detail.

4.1. Impaired neural processing

There is now growing evidence from brain research that people who stutter have a deficit in the neural processing underpinning spoken language (Chang, Erickson, Ambrose, Hagesawa-Johnson, & Ludlow, 2008; Chang, Horwitz, Ostuni, Reynolds, & Ludlow, 2011; Cykowski, Fox, Ingham, Ingham, & Robin, 2010; Watkins, Smith, Davis, & Howell, 2008). This research has shown both functional and structural abnormalities, but the precise nature of this neural processing problem is as yet unclear.

An important recent development in brain research, using Diffusion Tensor Imaging (DTI), suggests there may be a deficit in white fiber transmission in people who stutter (e.g. Chang et al., 2011; Cykowski et al., 2010). According to Kochunov et al. (2010), "Cerebral white matter consists of axonal bundles that connect proximal and distal brain regions and create large-scale neural networks facilitating complex behaviors" (p. 1109). In their study, Chang et al. (2011) concluded, "Our data provide strongest support for deficient left hemisphere inferior frontal to premotor connectivity as a neural correlate of stuttering" (p. 2507).

Drawing on their DTI findings and on recent genetic research, Cykowski et al. (2010) hypothesized that the impairment in brain function in stuttering involves a deficit in the myelogenesis in the white fibers of the brain, in the areas responsible for spoken language.

Cykowski et al. (2010) stress the tentative nature of their hypothesis and, indeed, given the exclusion criteria in their study, it is not based on a representative sample of people who stutter. However, the hypothesis has some explanatory power; that is, it can explain some of the things we know about stuttering. First, this myelin sheath starts to develop in the first few years of life after birth and children typically start to stutter in the third and fourth years of life. Hence, if the hypothesis is correct, it suggests that in children who start to stutter, the development of myelin is defective or delayed in relation to normal language development and hence interferes with speech production. Second, according to this hypothesis, transmission still occurs, but it is impaired. This can explain, to a large extent, why stuttering does not occur on every syllable. Third, it explains the high rate of natural recovery. The hypothesis predicts that stuttering will remit for most children as the development of the myelin sheath finally completes. For some children, however, myelogenesis remains incomplete and so stuttering continues into later life. Fourth, it suggests that the extent to which the myelin sheath is impaired will influence the baseline severity of stuttering. Fifth, since the structure of white matter is under strong, but not clear, genetic control, this goes some way towards explaining why there is not yet a clear genetic model of stuttering. Whether this hypothesis is supported by further research remains to be seen. However, whether or not it is supported, there is increasing evidence from recent brain research that people who stutter have a deficit in neural processing of some sort. The presence of such a deficit is critical/central to the P&A model.

4.2. Triggers

As discussed earlier stuttering does not occur on every syllable, so there must be a trigger for each moment of stuttering. The P&A model suggests that these triggers consist of certain inherent features of spoken language. They are more likely to trigger stuttering because they are associated with increased motor demands. These increased demands perturb the already unstable system responsible for the production of spoken language and hence trigger moments of stuttering. Of course, the idea that stuttering is associated with various linguistic features is not new. However, what is novel here is that language

is not necessarily impaired in people who stutter but rather there are inherent features of language that, when realized in speech, trigger stuttering.

The first of these proposed triggers is variable syllabic stress. This idea was integral to the development of the Variability model or Vmodel (Packman, Onslow, Richard, & van Doorn, 1996). This model was developed to explain why the novel speech pattern known as prolonged speech is so effective in reducing stuttering. This speech pattern has been used extensively and is still widely used for instating stutter free speech in behavioral treatments for older children and adults (for example, see Ingham, 1984; Langevin et al., 2006; O'Brian, Onslow, Cream, & Packman, 2003). As it has been taught, historically, the speaker slows the speech rate and prolongs sounds and words, shaping this towards natural sounding speech, which can then be used in everyday situations (see Ingham, 1984; Packman et al., 2000). However, an acoustic analysis of vowel duration indicated that using prolonged speech actually reduced variation in stress from syllable to syllable (see Packman et al., 1996). This was an unexpected finding, as clients are not taught to do this when learning prolonged speech. The Vmodel, then, explains the reductions in stuttering that occur with prolonged speech as due to reduction in the variability in contrastive syllabic stress. Reduction in the variability of syllabic stress is also a feature of syllable-timed or rhythmic speech, which is an even more powerful stuttering suppressant, or fluency enhancing condition (see Ingham, 1984; Packman et al., 2000). Reducing the variability in emphasis from syllable to syllable in both these speech patterns simplifies speech production considerably and so reduces the motoric task demands on an unstable speech production system.

The second trigger identified in the P&A model is linguistic complexity. Linguistic complexity has been an area of interest in the study of stuttering for decades, with research findings suggesting that stuttering tends to occur in utterances that are linguistically more complex (Bloodstein & Bernstein Ratner, 2008). The P&A model attributes this to the motoric task demands that this complexity places on the unstable speech system. Linguistic complexity renders speech movements unstable (Kleinow & Smith, 2000) and these authors concluded with the hypothesis that "linguistic complexity is one factor that contributes to the disruptions of speech motor stability characteristic of stuttering" (Kleinow & Smith, 2000, p. 548). In the P&A model, then, linguistic complexity, like varying syllabic stress, is an inherent feature of normal spoken language that triggers stuttering in speakers whose neural processing for speech is compromised by inefficient transmission in the connective white fibers.

The question arises, then, whether there is a relationship between the production of variable linguistic stress and linguistic complexity. It may be that increased linguistic complexity may involve increased variability in contrastive syllabic stress. This is an empirical question waiting to be answered.

4.3. Modulating factors

In the P&A model, the triggering mechanism is modulated by intrinsic factors. The main one is physiological arousal. According to the P&A model, the level of physiological arousal in an individual can alter the threshold at which a moment of stuttering is triggered. Here, physiological arousal refers to the readiness of the body to react to stressful internal and external stimuli. For example, a competitive tennis player may do a perfect backhand shot in practice but mistime it when in competition. Or a violinist may play Bach wonderfully in rehearsal but make errors when performing in public. These two examples relate to performance anxiety, but arousal can increase with excitement or anticipation or fear. Similarly, there is considerable evidence that stuttering severity can vary according to communicative context (Ingham, 1984). However, there is unlikely to be a straightforward linear relationship between arousal and stuttering severity, within individuals. For example, if a person uses a fluency enhancing technique, such as prolonged speech, an increase in arousal may result in them paying more attention to it, hence increasing their control over their stuttering. This is another empirical question raised by this model.

The availability of cognitive resources during communication has also been shown to have an effect on stuttering (see Metten et al., 2011). Metten et al. (2011) found that stuttering increased when a competing linguistic task diverted cognitive resources away from speaking. Interestingly, dual tasking also interfered with speech production for the normally fluent participants in the control group, who had many more normal disfluencies in this condition. According to the P&A model, then, dual- or multi-tasking where the tasks share resources can lower the threshold at which stuttering is triggered. Interestingly, it is also the case that stuttering may reduce during dual tasking where the secondary task does not share cognitive resources (Arends, Povel, & Kolk, 1988; Vasic & Winjen, 2005).

In this model, the modulating factors are considered to be unique for each individual. For example, in a study of 140 adults seeking treatment for their stuttering, Iverach et al. (2011) found wide variation in scores on the Unhelpful Thoughts and Beliefs About Stuttering scale and on a range of psychological tests. Modulating factors will likely be influenced by, among other things, individual experiences (for example, teasing during childhood), anxiety, fear of negative evaluation and stuttering severity, all in turn possibly influenced by individual differences in emotional reactivity (Walden et al., 2012) and resilience (Craig, Blumgart, & Tran, 2011).

Environmental stressors are seen as important in the P&A model, just as they are in the Demands and Capacities model. However, in the P&A model the individual's perceptions of, and/or reactions to, potential environmental stressors are also important in determining the threshold at which stuttering is triggered. For example, one person who stutters may be highly anxious when talking in a group, whereas another may not. Modulating factors, then, can be seen as the major contributor to the variability of stuttering within individuals, across communicative contexts.

To summarize the P&A model, an underlying deficit in neural processing is the necessary condition for stuttering to occur, while features of spoken language trigger moments of stuttering. Together, these form the necessary and sufficient conditions for a moment of stuttering to occur. In the model, differences in stuttering severity across individuals can be attributed to differences in the extent of the neural processing deficit across individuals, while variability of stuttering within individuals, across communication contexts, can be attributed to the range and potency of modulating factors in individuals.

5. Do therapies for stuttering address cause?

It would seem logical that behavioral treatments for stuttering – in this context this refers to treatments that aim to reduce stuttering – should in some way address cause. If that is indeed the case, then it should be possible to explain, theoretically at least, how current behavioral treatment approaches fit the P&A model. This is a theoretical exercise and does not imply that because a treatment addresses cause it must therefore be efficacious. That is an empirical question to be answered by clinical trials.

5.1. An overarching consideration

Before addressing the extent to which individual treatments for stuttering address cause, the question of whether any treatment for stuttering can change/improve the deficit in neural processing that is hypothesized to underpin it needs to be considered. It would seem we simply do not know enough about the nature of that deficit at this stage to speculate seriously on this. Brain plasticity is thought to facilitate the forming of new neural pathways for speech and language after brain injury and stroke (Kleim, 2011). However, it is not at all clear that plasticity could accommodate the formation of the presumably large new networks required to support the fluency that adolescents and adults can acquire as a result of speech restructuring treatments. Indeed, all the research suggests that the improvements in fluency that come as the result of speech restructuring treatments are difficult to maintain (see Bloodstein & Bernstein Ratner, 2008; Iverach, Jones, et al., 2009).

The brain is more plastic in young children than in adolescents and adults. However, if Cykowski et al's (2010) hypothesis is correct, the idea of forming new and intact neural networks in very young children in response to fluency enhancement may not be tenable, although this is open to conjecture (Martin Sommer, personal communication, September 2011).

The proposal that therapies for stuttering primarily address Factors 2 and 3 in the P&A model is now considered. As proposed, Factor 2 comprises features of spoken language that can trigger stuttering and Factor 3 comprises intrinsic factors that can alter the triggering threshold.

5.2. Behavioral treatments

Therapies that are intended to ameliorate the behaviors of stuttering can be categorized as direct and indirect, although many therapy programs integrate these two approaches (see Guitar, 2006). We suggest that direct therapies address stuttering triggers (Factor 2 in the P&A model) while indirect therapies address intrinsic modulators (Factor 3 in the P&A model).

5.2.1. Direct treatment procedures

The primary aim of direct behavioral procedures is to modify the production of spoken language in ways that ameliorate stuttering. This is done by participants reducing speech rate, stretching speech sounds, modifying the use of the voice, reducing variability of syllabic stress and reducing utterance length and/or linguistic complexity (for overviews see Bloodstein & Bernstein Ratner, 2008; Guitar, 2006). As referred to above in the description of the development of the P&A model, reducing the variability of contrastive syllabic stress is thought to be an active agent in prolonged speech and rhythmic speech, two of the most widely used fluency enhancing treatments for adults who stutter. Treatments based on rhythmic speech have also been developed recently for children who stutter (Andrews et al., 2012; Trajkowski et al., 2011). According to the P&A model, then, these changes can be seen as reducing the frequency with which the relevant features of spoken language trigger stuttering.

Another treatment procedure aimed at changing speech production is altered auditory feedback (AAF) (for a review see Lincoln, Packman, & Onslow, 2006). The developers of the Speech Easy AAF device hypothesize that AAF reduces stuttering via the activation of mirror neurons (Kalinowski & Saltuklaroglu, 2006) while Ingham, Moglia, Frank, Ingham, and Cordes (1997) hypothesized that AAF stimulates temporal lobe activity. Hence, it could be said that AAF addresses Factor 1 (brain function) in the P&A model. However, Kalinowski and Saltuklaroglu report that many users of AAF also need to use speech-restructuring techniques such as prolongation, gentle onsets and light articulatory contacts. These procedures, then, would address the triggering factors. In conclusion, given that current understanding of the mechanisms underpinning AAF is unclear, it is currently unclear how AAF fits the P&A model.

A biofeedback treatment developed by Ingham et al. (2001) also alters speech production by reducing short periods of phonation. It has been suggested by the developers of the program that short periods of phonation trigger stuttering. Whether this program also results in reduced variability of syllabic would be an interesting area for further research.

The speech restructuring treatments discussed above clearly address speech production directly. However, operant treatments also aim to change speech production but without instruction to do so (for examples, see Hewat, Onslow, Packman, & O'Brian, 2006; Ingham, 1984; Onslow, Packman, & Harrison, 2003). For example, in the Lidcombe Program of early intervention (Onslow et al., 2003), the parent gives verbal contingencies for the child's stutter-free speech, such as "good talking", "that was smooth", and less frequently for stuttering, such as "that was a bumpy word". This is a direct treatment, in that it focuses on the child's speech, but there is no specific direction to children to change how they produce spoken language. However, since there is evidence that the program has an effect on stuttering frequency over and above that of natural recovery (Jones et al., 2005; Lattermann, Euler, & Neumann, 2008), presumably children are changing some aspect of their spoken language. However, despite research, the actual mechanism is unknown (Bernstein Ratner, 2005; Hayhow, 2011) although it appears fluency is not achieved at the cost of reduced linguistic complexity (Bonelli, Dixon, Bernstein Ratner, & Onslow, 2000; Lattermann, Shenker, & Thordardottir, 2005).

Reducing the frequency of the trigger of linguistic complexity can be seen as the aim of two other direct treatments for children who stutter, namely Extended Length of Utterance and Gradual Increase in Length and Complexity of Utterance (for a review see Davidow, Crowe, & Bothe, 2004).

5.2.2. Indirect treatment procedures

Indirect treatment procedures intended to increase fluency, typically focus on environmental or internal factors that are thought to be affecting the severity of stuttering (for examples see Guitar & McCauley, 2010). In children this usually involves having parents change various aspects of the child's environment that are seen to be stressful, and changing the way they communicate with their child (see Bloodstein & Bernstein Ratner, 2008; Guitar, 2006). Parent-Child Interaction Therapy (see Millard, Nicholas, & Cook, 2008) is an example of a predominantly indirect treatment. While procedures vary across treatments, according to the P&A model they can be seen to be primarily directed at the modulating factors. That is, by reducing time pressure and other environmental pressures, they can be seen to be reducing physiological arousal and/or cognitive load. In the P&A model, it is proposed that the primary role of such procedures is to raise the threshold at which individual moments of stuttering are triggered. It could be argued that such indirect procedures reduce linguistic complexity and/or other features of spoken language and hence reduce the frequency of triggering, but this argument is difficult to sustain, given Millard et al.'s (2008) description of PCI therapy. Rather, Millard et al. (2008) report that direct therapy approaches – which would address the triggering mechanism in spoken language – may be introduced after PCI for children who continue to stutter.

5.3. Other therapies

Drug therapies and psychological treatments are listed separately as they do not include behavioral procedures intended to modify speech production. However, it seems that they are intended to act on physiological arousal. In any event, clinical trials have yet to demonstrate efficacy for any drug (Bloodstein & Bernstein Ratner, 2008; Bothe et al., 2006; Boyd et al., 2011).

Psychological therapies for stuttering (for a review see Bloodstein & Bernstein Ratner, 2008) are now addressed, with emphasis on cognitive behavior therapy (CBT). Many adults who stutter have anxiety disorder and qualify for a diagnosis of social phobia (see Iverach, O'Brian, et al., 2009) and CBT aims to change the unhelpful thoughts and fear of negative evaluation that people have in relation to social situations. The rationale for this is that reducing negative thoughts and fears will lead to reductions in anxiety. The first CBT program for stuttering to be developed from a psychological model has been trialed with adults who stutter (Menzies et al., 2008).

However, while measures in this trial indicated that social anxiety reduced to normal limits after CBT, this had no effect on participants' stuttering. This is in line with findings that the relationship between anxiety and sympathetic nervous system activity is not straightforward (Alm, 2004). It may also be that the CBT-induced reduction in social anxiety reduced participants' desire to (1) control their stuttering, which is typically achieved by using various techniques, and/or (2) conceal their stuttering by avoiding various words or speaking situations that they consider to be problematic. In other words, it may be that the CBT treatment results in people worrying less about their stuttering and its effects in social situations.

We can speculate, then, that reducing arousal in preschoolers who stutter, by reducing for example environmental stressors, may have a positive effect on stuttering because these children have yet to adopt the complex avoidance and control strategies of older children and adults. So perhaps this modulating component of the model works differently according to age. In any event, the relationship between anxiety and stuttering remains an intriguing one. More research involving physiological markers of stress would seem to be a fruitful way to investigate this further (for example, see Ortega & Ambrose, 2011).

5.4. Stuttering is complex

The P&A model addresses cause in a somewhat linear way and, of course, this oversimplifies matters somewhat. Viewing stuttering through the lens of complexity (non-linear dynamics) provides a more holistic view of the disorder (Packman & Kuhn, 2009). This perspective is not a causal model but rather a framework for understanding the complexity of stuttering. The non-linear complexity analysis in this report locates stuttering within the broadest framework possible. The initial

condition, a glitch in neural processing, can lead to the self re-organization of other related dynamic systems, which in turn influence others. More specifically, the glitch can lead to changes in mind and body, which in turn affect and are effected by the context in which communication is occurring, including the situation, the purpose of the interaction, the communicative partner, and so on. Society's attitudes to stuttering are important here also.

So, while in the P&A model a treatment is depicted as *addressing* one component of cause, when viewed through the lens of complexity the effects are likely to be much wider. While therapies may address one or perhaps more features of the causal model, in assessing outcomes their effects should be measured across many domains.

It is also possible that if a therapy is shown to have an effect, this effect may not be due to the mechanism modeled but is in fact due to other factors. The Lidcombe Program is a case in point. It is known that the program has an effect over and above that of natural recovery but it cannot be deduced from this that parental contingencies are in fact the active treatment agent. Is it possible, for example, that stuttering may reduce to the same extent if the parent and child simply sit down and talk for prescribed periods each day, with the parent praising the child for achievements other than fluent speech. Indeed, there may be active treatment agents that are common across treatments. This intriguing question can only be answered empirically, with clinical trials in which the effects of individual components of treatments are investigated and entire treatments compared.

The latter is a critical point. For example, a study comparing Lidcombe Program with treatment based on the Demands and Capacities model (Franken, Kielstra-Van der Schalk, & Boelens, 2005) found no difference in stuttering after 12 weeks. However, as reported by the authors this was a pilot study. It was not a valid comparison of outcomes for the two interventions, because the Lidcombe children did not receive a full treatment "dose". It is known from previous research (see Kingston, Huber, Onslow, Jones, & Packman, 2003) that, at that time, 50% of children were taking more than 11 weekly clinic visits to complete Stage 1 of the Lidcombe Program. As Franken et al. acknowledge, only two of the 11 children in their Lidcombe group had completed Stage 1 by 12 weeks.

6. Conclusion

The P&A model attempts to explain, comprehensively, why moments of stuttering occur. As alluded to already, the model is multifactorial and can be seen as describing the demands and capacities underpinning stuttering, just as the Demands and Capacities model does. However, the P&A model propose that a neural processing deficit is a necessary (albeit not sufficient) condition for stuttering to occur. This deficit, along with proscribed triggers, comprises the necessary and sufficient conditions for a moment of stuttering to occur (provided a trigger is above threshold). While the modulating factors will be different in each individual, they can be measured and are hence amenable to research. As well as having considerable explanatory power, the P&A model provides a framework for exploring the extent to which various treatments for stuttering address the cause of the disorder. The model also suggests avenues for further research.

It is stressed again that the P&A model presented in this paper is just that: a model. A model is simply a proposal of how things might work. The model is open to testing and will be modified, or indeed rejected, in the face of conflicting evidence. Such evidence might be, for example, that a neural processing deficit is *not* a necessary condition for stuttering to occur. An important role of theory building in the study of stuttering is to open up discussion and prompt new lines of enquiry (Packman & Attanasio, 2004). It is hoped that the model presented in this paper will do that.

CONTINUING EDUCATION

Theory and therapy in stuttering: A complex relationship

QUESTIONS

- (1) The P&A 3-factor causal model:
 - a. Provides an explanation of the causal mechanism of moments of stuttering
 - b. Explains other models of stuttering
 - c. Has been disproved
 - d. Is true
- (2) The Vmodel:
 - a. Is never used with people who stutter
 - b. Has been shown to reduce anxiety in people who stutter
 - c. Always reduces stuttering
 - d. Has not been investigated in a clinical trial with people who stutter
- (3) Cognitive behavior therapy:
 - a. Was not based on evidence
 - b. Explains the effects of intonation on stuttering
 - c. Proposes that variable syllabic stress triggers stuttering
 - d. Does not explain the effects of any treatments for stuttering

- (4) The Lidcombe Program
- Is a treatment for adults who stutter
 - Is delivered in the speech clinic
 - Is delivered by parents
 - Is not supported by evidence from clinical trials
- (5) According to the P&A model:
- Treatments using syllable timed speech reduce one of the triggers for stuttering
 - Indirect treatments for stuttering are the most effective
 - Early intervention for stuttering is not recommended
 - Operant treatments for stuttering address psychological factors

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References

- Alm, P. A. (2004). Stuttering, emotions and heart rate during anticipatory anxiety: A critical review. *Journal of Fluency Disorders*, 29, 123–133.
- Andrews, C., O'Brian, S., Onslow, M., Harrison, E., Packman, A., & Menzies, R. (2012). Syllable-timed speech treatment for school age children who stutter: A Phase I trial. *Language, Speech and Hearing Services in Schools*, 43, 359–369.
- Arends, N., Povel, D. J., & Kolk, H. (1988). Stuttering as an attentional phenomenon. *Journal of Fluency Disorders*, 13, 141–151.
- Bernstein Ratner, N. (2005). Evidence-based practice in stuttering: Some questions to consider. *Journal of Fluency Disorders*, 30, 163–188.
- Bloodstein, O., & Bernstein Ratner, N. (2008). *A handbook of stuttering* (6th ed.). Clifton Park, NY: Delmar.
- Bonelli, P., Dixon, M., Bernstein Ratner, N., & Onslow, M. (2000). Child and parent speech and language following the Lidcombe Program of early stuttering intervention. *Clinical Linguistics and Phonetics*, 14, 427–446.
- Bothe, A. K., Davidow, J. H., Bramlett, R. E., Franic, D. M., & Ingham, R. J. (2006). Stuttering treatment research 1970–2005: II. Systematic review incorporating trial quality assessment of pharmacological approaches. *American Journal of Speech-Language Pathology*, 15, 342–352.
- Boyd, A., Dworzynski, K., & Howell, P. (2011). Pharmacological agents for developmental stuttering in children and adolescents: A systematic review. *Journal of Clinical Psychopharmacology*, 31, 740–744.
- Chang, S.-E., Erickson, K. I., Ambrose, N. G., Hasegawa-Johnson, M. A., & Ludlow, C. L. (2008). Brain anatomy differences in childhood stuttering. *Neuroimage*, 39, 1333–1344.
- Chang, S.-E., Horwitz, B., Ostuni, J., Reynolds, R., & Ludlow, C. (2011). Evidence of left frontal inferior–premotor structural and functional connectivity deficits in adults who stutter. *Cerebral Cortex*, 21, 2507–2518.
- Craig, A., Blumgart, E., & Tran, Y. (2011). Resilience and stuttering: Factors that protect people from the adversity of chronic stuttering. *Journal of Speech, Language and Hearing Research*, 54, 1485–1496.
- Cykowski, M. D., Fox, P. T., Ingham, R. J., Ingham, J. C., & Robin, D. A. (2010). A study of the reproducibility and etiology of diffusion anisotropy differences in developmental stuttering: A potential role for impaired myelination. *Neuroimage*, 52, 1495–1504.
- Davidow, J. H., Crowe, B. T., & Bothe, A. K. (2004). Gradual increase in length and complexity of utterance and extended length of utterance treatment programs for stuttering: Assessing the implications of strong but limited evidence. In A. K. Bothe (Ed.), *Evidence-based treatment of stuttering: Empirical bases and clinical applications* (pp. 201–230). Mahwah, NJ: Lawrence Erlbaum Associates.
- Franken, M.-C. J., Kielstra-Van der Schalk, C. J., & Boelens, H. (2005). Experimental treatment of early stuttering: A preliminary study. *Journal of Fluency Disorders*, 30, 189–199.
- Guitar, B. (2006). *Stuttering: An integrated approach to its nature and treatment*. Philadelphia, PA: Lippincott Williams & Wilkins.
- Guitar, B., & McCauley, R. (2010). *Treatment of stuttering: Established and emerging interventions*. Baltimore, MD: Lippincott Williams & Wilkins.
- Hayhow, R. (2011). Does it work? Why does it work? Reconciling difficult questions. *International Journal of Language and Communication Disorders*, 46, 155–168.
- Hewat, S., Onslow, M., Packman, A., & O'Brian, S. (2006). A Phase II clinical trial of self-imposed time-out treatment for stuttering in adults and adolescents. *Disability and Rehabilitation*, 28, 33–42.
- Ingham, R. J. (1984). *Stuttering and behavior therapy*. San Diego, CA: College-Hill Press.
- Ingham, R. J., Kilgo, M., Ingham, J. C., Moglia, R., Belknap, H., & Sanchez, T. (2001). Evaluation of a stuttering treatment based on reduction of short phonation intervals. *Journal of Speech, Language and Hearing Research*, 44, 1229–1244.
- Ingham, R. J., Moglia, R. A., Frank, P., Ingham, J. C., & Cordes, A. K. (1997). Experimental investigation of the effects of frequency altered auditory feedback on the speech of adults who stutter. *Journal of Speech, Language and Hearing Research*, 40, 361–372.
- Iverach, L., Menzies, R., Jones, M., O'Brian, S., Packman, A., & Onslow, M. (2011). Further development and validation of the Unhelpful Thoughts and Beliefs about Stuttering (UTBAS) scale: Relationship to anxiety and social phobia among adults who stutter. *International Journal of Language and Communication Disorders*, 46, 286–299.
- Iverach, L., Jones, M., O'Brian, S., Block, S., Lincoln, M., Harrison, E., et al. (2009). The relationship between mental health disorders, stuttering severity and treatment outcome among adults who stutter. *Journal of Fluency Disorders*, 34, 29–43.
- Iverach, L., O'Brian, S., Jones, M., Block, S., Lincoln, M., Harrison, E., et al. (2009). Prevalence of anxiety disorders among adults seeking speech therapy for stuttering. *Journal of Anxiety Disorders*, 23, 928–934.
- Jones, M., Onslow, M., Packman, A., Williams, S., Ormond, T., Schwartz, I., et al. (2005). Randomised controlled trial of the Lidcombe Program of early stuttering intervention. *British Medical Journal*, 331, 659–661.
- Kalinowski, J. S., & Saltuklaroglu, T. (2006). *Stuttering*. San Diego, CA: Plural Publishing.
- Kingston, M., Huber, A., Onslow, M., Jones, M., & Packman, A. (2003). Predicting treatment time with the Lidcombe Program: Replication and meta-analysis. *International Journal of Language and Communication Disorders*, 38, 165–177.
- Kleim, J. A. (2011). Neural plasticity and neurorehabilitation: Teaching the new brain old tricks. *Journal of Communication Disorders*, 44, 521–528.
- Kleinow, J., & Smith, A. (2000). Influences of length and syntactic complexity on the speech motor stability of the fluent speech of adults who stutter. *Journal of Speech, Language and Hearing Research*, 43, 548–559.
- Kochunov, P., Glahn, D. C., Lancaster, J. L., Winkler, A. M., Smith, S., Thompson, P. M., et al. (2010). Genetics of microstructure of cerebral white matter using diffusion tensor imaging. *Neuroimage*, 53, 1109–1116.
- Langevin, M., Huinck, W. J., Kully, D., Peters, H. F. M., Lomheim, H., & Tellers, M. (2006). A cross-cultural, long-term outcome evaluation of the ISTAR Comprehensive Stuttering Program across Dutch and Canadian adults who stutter. *Journal of Fluency Disorders*, 31, 229–256.

- Lattermann, C., Euler, H. A., & Neumann, K. (2008). A randomized control trial to investigate the impact of the Lidcombe Program on early stuttering in German-speaking pre-schoolers. *Journal of Fluency Disorders*, 33, 52–65.
- Lattermann, C., Shenker, R., & Thordardottir, E. (2005). Progression of language complexity during treatment with the Lidcombe Program for early stuttering intervention. *American Journal of Speech-Language Pathology*, 14, 242–253.
- Lincoln, M., Packman, A., & Onslow, M. (2006). Altered auditory feedback and the treatment of stuttering: A review. *Journal of Fluency Disorders*, 31, 71–89.
- Menzies, R., O'Brian, S., Onslow, M., St Clare, T., Packman, A., & Block, S. (2008). An experimental clinical trial of a cognitive behavior therapy package for chronic stuttering. *Journal of Speech, Language and Hearing Research*, 51, 1451–1464.
- Metten, C., Bosshardt, H. G., Jones, M., Eisenhuth, J., Block, S., Carey, B., et al. (2011). Dual tasking and stuttering: From the laboratory to the clinic. *Disability and Rehabilitation*, 33, 933–944.
- Millard, S. K., Nicholas, A., & Cook, F. M. (2008). Is parent–child interaction therapy effective in reducing stuttering? *Journal of Speech, Language and Hearing Research*, 51, 636–650.
- O'Brian, S., Onslow, M., Cream, A., & Packman, A. (2003). The Camperdown Program: Outcomes of a new prolonged-speech treatment model. *Journal of Speech Language and Hearing Research*, 46, 933–946.
- Onslow, M., Packman, A., & Harrison, E. (Eds.). (2003). *The Lidcombe Program of early stuttering intervention: A clinician's guide*. Austin, TX: Pro-Ed.
- Ortega, A. Y., & Ambrose, N. G. (2011). Developing psychological stress profiles for school-age children who stutter. *Journal of Fluency Disorders*, 36, 268–273.
- Packman, A., & Attanasio, J. S. (2004). *Theoretical issues in stuttering*. New York: Psychology Press.
- Packman, A., & Attanasio, J. S. (2010, November). A model of the mechanisms underpinning early interventions for stuttering. In *Seminar presentation at the annual convention of the American Speech-Language and Hearing Association* Philadelphia, USA.
- Packman, A., & Kuhn, L. (2009). Looking at stuttering through the lens of complexity. *International Journal of Speech-Language Pathology*, 11, 77–82.
- Packman, A., Onslow, M., & Menzies, R. (2000). Novel speech patterns and the control of stuttering. *Disability and Rehabilitation*, 22, 65–79.
- Packman, A., Onslow, M., Richard, E., & van Doorn, J. (1996). Syllabic stress and variability: A model of stuttering. *Clinical Linguistics and Phonetics*, 10, 235–263.
- Shapiro, D. A. (1999). *Stuttering intervention*. Austin, TX: Pro-Ed.
- Smith, A., & Kelly, E. (1997). Stuttering: A dynamic, multifactorial model. In R. F. Curlee, & G. M. Siegel (Eds.), *Nature and treatment of stuttering: New directions* (2nd ed., pp. 204–217). Boston, MA: Allyn & Bacon.
- Starkweather, C. W. (1987). *Fluency and stuttering*. Englewood Cliffs, NJ: Prentice-Hall, Inc.
- Starkweather, C. W., & Givens-Ackerman, J. (1997). *Stuttering*. Austin, TX: Pro-Ed.
- Starkweather, C. W., & Gottwald, S. R. (2000). The demands and capacities model II: Clinical implications. *Journal of Fluency Disorders*, 15, 143–157.
- Trajkowski, N., Andrews, C., Onslow, M., O'Brian, S., Packman, A., & Menzies, R. (2011). A phase II trial of the Westmead Program: Syllable-timed speech treatment for pre-school children who stutter. *International Journal of Speech-Language Pathology*, 13, 500–509.
- Van Riper, C. (1973). *The treatment of stuttering*. Englewood Cliffs, NJ: Prentice-Hall.
- Vasic, N., & Winjen, F. (2005). Stuttering as a monitoring deficit. In R. Hartsuiker, R. Bastiaanse, A. Postma, & F. Winjen (Eds.), *Phonological encoding and monitoring in normal and pathological speech* (pp. 226–247). Hove, UK: Psychology Press.
- Walden, T. A., Frankel, C. B., Buhr, A. P., Johnson, K. N., Conture, E. G., & Karrass, J. M. (2012). Dual diathesis-stressor model of emotional and linguistic contributions to developmental stuttering. *Journal of Abnormal Child Psychology*, 40, 633–644.
- Watkins, K. E., Smith, S. M., Davis, S., & Howell, P. (2008). Structural and functional abnormalities of the motor system in developmental stuttering. *Brain*, 131, 50–59.
- Yairi, E., & Seery, C. H. (2011). *Stuttering: Foundations and clinical applications*. Upper Saddle River, NJ: Pearson Education, Inc.

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